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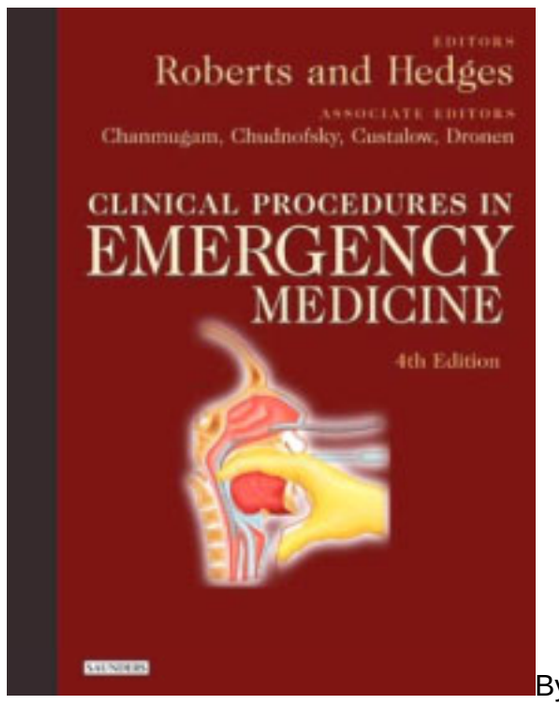

By OkDoKeY

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Appendix: Commonly Used Formulas and Calculations

CLINICAL PROCEDURES IN EMERGENCY MEDICINE

4th Edition

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Notice

Emergency Medicine is an ever-changing field. Standard safety precautions must be followed but as new research and clinical experience broaden our knowledge, changes in treatment and drug therapy may become necessary or appropriate. Readers are advised to check the most current product information provided by the manufacturer of each drug to be administered to verify the recommended dose, the method and duration of administration, and contraindications. It is the responsibility of the treating physician, relying on experience and knowledge of the patient, to determine dosages and the best treatment for each individual patient. Neither the publisher nor the author assumes any liability for any injury and/or damage to person or property arising from this publication.

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To Michael P. Spadafora, MD (1953–1999). A great guy whose talent, charisma, and friendship will be missed by many. You taught me more than I ever taught you, Michael, and life's just not the same without you.

To David K. Wagner, MD, Lewis R. Goldfrank MD, and Jerris R. Hedges, MD. Their prowess, prescience, and dynamism were obvious the first time I met them. And to Michael I. Greenberg, MD. As a toxicologist he was first my student, then my colleague, and now my mentor.

J.R.R.

This Fourth Edition of Clinical Procedures in Emergency Medicine *is dedicated to all the future generations of emergency physicians. As our specialty becomes an international discipline, this text in English, Italian, and Spanish is now found in many lands. Countless practitioners have expanded their knowledge and shared this information with their trainees. If a copy of this text can make a profound difference in at least one new emergency physician's career, we have served the future well.*

J.R.H.

This book is dedicated to all those who practice emergency medicine and to those that support them in their endeavors.

A.S.C.

To my wife Marcy, without a doubt the very best thing that has ever happened to me. Her kindness, patience, and devotion to family is surpassed by none. To my children, Adam, Arielle, and Allison, whom I love more than any words could ever express. And to my mother Eleanor, whose strength and courage in the face of adversity is an inspiration to everyone lucky enough to know her.

C.R.C.

To my son, Nicholas, and in memory of my daughter, Lauren; to Peter Pons, my favorite teacher; and to our residents so that when they are far away from this place of learning, they may confidently pick up the scalpel and perform these lifesaving procedures without hesitation—both quickly and competently.

C.B.C.

This effort is dedicated to the idealistic spirit that drives emergency physicians to seek excellence in the care of their patients. It is my hope that this book will be a valuable tool in their quest to bring order to the chaos of life in the ED.

I could not work on projects such as this without the support and unfailing generosity of my wife, Beverly. I offer my thanks to Bev for all she has given to me and to the specialty of Emergency Medicine.

S.C.D.

How This Book Should be Viewed by the Practicing Clinician

The editors and authors of this textbook strongly believe that the complex practice of medicine, the vagaries of human diseases, the unpredictability of pathologic conditions, and the functioning and responses of the human body cannot be defined, explained, or rigidly categorized by any written document. Therefore, it is *not* the purpose of this text to serve as an *authoritative source* on any medical condition or clinical intervention, nor an attempt to define a *standard of care* that should be practiced by all clinicians. We provide the physician with a literature-based database, and a reasonable clinical guide that is combined with practical suggestions. We offer a general reference source on a variety of conditions and procedures that may confront clinicians who are experienced in emergency medicine practice. This text cannot replace physician judgment, cannot describe every possible aberration or clinical scenario, and cannot define rigid standards for clinical actions or procedures. Every medical encounter must be individualized and every patient must be approached on a case-by-case basis. Some of the procedures described are common, while others are uncommon, rarely encountered, or best performed, given specific circumstances, by another practitioner with different training, experience, or logistical constraints. The procedures described herein do not constitute the expertise or the knowledge base to be possessed by all clinicians. Finally, many of the described complications associated with implementing complex medical and surgical procedures may be encountered, even when every aspect of the intervention has been performed correctly.

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Foreword

The emergency physician has the unique responsibility of offering his or her skills at all times to all people (young and old, friendly and hostile, rich and poor). No other health providers are always collectively there at the entrance to the hospital. As emergency physicians, our responsibilities have grown and our horizons have been expanded because of our commitment to people. We have built a system that creates a caring environment from the home to the street and to the hospital, and a system that also integrates firefighters, police officers, paramedics, nurses, clerks, students, pharmacists, and physicians into this caring service. Each new clinical problem and each creative intervention has led to innovations in thought and technical advances. The Fourth Edition of Roberts and Hedges' text, *Clinical Procedures in Emergency Medicine*, takes another step in the pursuit of excellence in the provision of that care. The authors' detailed critical analyses of the studies pertinent to the use of each technique allow for a rigorous approach as to how, why, and when each procedure is indicated.

The past 30 years in the history of emergency medicine have seen a remarkably rapid evolution in care. Organized medicine has often been criticized for its inability to change thought patterns and approaches to care, but the ability to change current patterns is the recognized strength of emergency physicians. We have undertaken our responsibilities, created new relationships, and developed new perspectives on clinical medicine in an area where previously no one dared to serve. In the past, medical providers have also been criticized for not evaluating their clinical techniques and technology effectively. This text exemplifies and describes the tremendous progress in thought and technology that mark the success of emergency medicine in America today.

The rapid growth of prehospital care, the ever-increasing roles of emergency care, and the diversity of clinical issues and research dilemmas in emergency medicine have led to the development of a new type of physician in the emergency department. This text defines the breadth of academic and clinical emergency medicine and the enormous technical skill and intellectual responsibility required by each emergency physician.

These chapters are written by emergency physicians and other physicians working closely with emergency patients who have highly specialized knowledge in particular aspects of emergency medicine. Almost a third of these authors are new contributors to this edition. A reevaluation of the clinical and academic roles of the emergency physician has led to the refinement of this Fourth Edition. As the basic science and clinical practice of emergency medicine have further developed, this book has grown to represent a complete view of our specialty. This text offers a balanced analysis of the interventions at our disposal in the emergency department for the care of those with urgent and emergent problems. The authors attempt to simplify and clarify while focusing on knowledge and process with regard to the equipment we use in the environment where we practice. This text permits any practitioner the opportunity to perform his or her first emergency procedures with a foundation that emphasizes evidence and limits bias and ignorance.

This text has filled a void in medical practice. Procedural interventions in the emergency department had previously been largely undefined and certainly inadequately analyzed. The emergency physician who is trained in these techniques can develop the requisite technical skills and combine them with the warmth and humanity essential to render concerned, committed, and compassionate emergency care. Knowledge of these skills and their indications, as well as the risks and benefits of practice, will permit emergency physicians to achieve the highest level of service and will foster their potential to initiate quality research.

This book is also about motivating physicians to appreciate the clinical norms and expectations of our procedures. The editors have recognized for years many of the problems defined in the report *To Err is Human* released by the Institute of Medicine of the National Academy of Sciences in 1999. This text has moved the physician from anecdote to a rigorous analysis of procedures. The reader will not only feel more secure about performing an essential procedure, but he or she will also become more confident about not performing a procedure that entails more risk than benefit to an individual patient. The editors and authors have attempted to enhance education and limit the errors of commission as well as omission while improving the safety and occupational health of the emergency physician.

This book attempts to prepare the clinician for his or her role in the emergency department. Recognizing that the emergency department environment is by definition unpredictable and often chaotic, these authors have prepared us to change the human response in an attempt to make errors more difficult to commit.

Understanding the remarkable spectrum of responsibility of the emergency physician is our essential task. We shall succeed as health providers if we understand our patients and their needs, the pathophysiology of emergency medicine and its therapeutics, and our procedures and their pitfalls. The Fourth Edition of Roberts and Hedges' *Clinical Procedures in Emergency Medicine* provides enough thought-provoking information about medical technology to prepare the emergency physician to care for the emergency department patient in a humane and intellectually sound manner.

Although few physicians other than emergency physicians will use all the techniques and technology detailed in this text, many other physicians can and will profit immensely from its use. The techniques are well defined, well illustrated, and well referenced by clinicians who obviously use them daily. This text remains unique with respect to the depth and breadth with which the editors and authors critically evaluate the tools of our trade. The two leaders of our field, Roberts and Hedges, have been joined by four new associate editors. The addition of these respected emergency physicians

expands the text's excellent foundation of editorial contributions and ensures continued successful presentation of procedural techniques to help guide our clinical care.

The understanding and application of the principles defined in this edition should be considered essential for each emergency physician in his or her attempt to continuously improve the delivery of the best possible health care to our patients.

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Section I - Vital Signs and Patient Monitoring Techniques

Chapter 1 - Vital Signs Measurement

Diane L. Gorgas

Documentation of temperature, pulse, respiration, and blood pressure is generally recommended for all emergency department (ED) patients, except those with the most minimal complaints. These measurements provide a unique, objective, capsule assessment of the patient's clinical condition. Vital signs indicate the severity of illness and may dictate the urgency of required intervention. Although a single set of values may suggest disease, the greatest utility of vital signs is their measurement over time. Deteriorating vital signs are an important indicator of a deteriorating physiologic condition, whereas improving values provide reassurance that an unstable patient is responding to therapy. Hence, when a patient undergoes treatment over an extended time, selected vital signs, particularly previously abnormal ones, should be repeated. In some circumstances, the monitoring of select vital signs should be continuous.

Vital signs should be measured and recorded at intervals dictated by the patient's clinical state (e.g., before and after fluid resuscitation, invasive procedures, or administration of medications with cardiopulmonary effects) or with any sudden change in the patient's clinical status. In addition, an abnormal vital sign can direct the clinician toward a group of diagnoses or a particular organ system for further evaluation. An abnormal vital sign may constitute the patient's entire complaint, as in the febrile infant, or be the only indication of the potential for serious illness, as in the patient with resting tachycardia. For these reasons, accurate determination and interpretation of vital signs are mandatory. Unfortunately, in many EDs, vital signs are not recorded reliably, accurately, [1] or with optimal frequency.[2] This can lead to delayed diagnosis or misinterpretation of the severity of an illness or injury.

Assessment of a patient's status and vital signs should begin in the prehospital setting in cases where Emergency Medical Service (EMS) transport is involved. EMS transport-induced stress can alter vital signs because of epinephrine and norepinephrine surges that commonly occur during transport. This has been shown to lead to increased heart rates of >10%.[3] Although prehospital vital signs need to be interpreted carefully, they should still be obtained and in the vast majority of situations, they are. The exception occurs in the pediatric population, especially those younger than 2 years of age. The lack of routine measurement of vital signs by EMS personnel in this group is largely due to the paramedic's or technician's lack of confidence in accurately measuring vitals signs in newborns, infants, and toddlers. [4]

In the ED, the accurate assessment and management of abnormal vital signs must reflect the priorities of resuscitation. Determination of airway patency with respiratory rate (RR) and pattern assumes first importance. Establishing the presence and quality of an arterial pulse is the second vital sign to be assessed, followed by blood pressure. Blood pressure and pulse are often evaluated in conjunction, as a measure of blood volume. Although body temperature is the last vital sign to be monitored during resuscitation, it has special importance for patients suffering from thermal regulation failure (see Chapter 67 and Chapter 68). The current chapter is organized according to the priorities of patient resuscitation and evaluation.

Additional "vital signs" recently introduced into emergency medicine are pulse oximetry, capillary refill, and the analogue or similar pain scale. The use of pulse oximetry is discussed subsequently (see Chapter 2). Capillary refill in general is considered part of the assessment of overall perfusion and most closely linked to circulatory volume and blood pressure in children. In accordance, capillary refill is covered under the blood pressure section. Assessment of pain as a vital sign is gaining acceptance. Mental status has also been proposed as a vital sign, as it can be viewed as a summation of measurable vital signs (blood pressure, heart rate, RR, and temperature). Significant aberrations in any of these quantifiable vital signs will cause mental status changes.

BACKGROUND

Early pulmonary medicine was dominated by the concepts of Herophilus (4th century B.C.) and Galen (131 to 200 A.D.), whose belief in the humoral theory of medicine dictated that the lungs functioned as a cooling device and site for generation of body humors. The pulmonary circulation was correctly described in the 13th century by Ibnan-Nafis; however, his observations passed unnoticed.

Respiratory physiology did not progress until the significance of the pulmonary circulation was recognized by Harvey in 1628. It was not until the 1700s that advances in physics and chemistry allowed the identification of gases involved in respiration. [5]

Sphygmology, or palpation of the pulse, was first appreciated by Herophilus. He believed that interpreting the pulse required a knowledge of both music and geometry and defined the characteristics of the pulse as size, frequency, force, and rhythm. Chinese clinicians (2nd century B.C.) timed the pulse by the RR of the examiner, believing that 4 pulsations/respiration was normal for adults. The study of pulses was greatly influenced by Galen, who expanded the subject into a rather complex and obscure art form, writing 18 books on the subject.[6]

Blood pressure was first measured directly in 1733 by Hales, who recorded the arterial pressures in a mare by cannulation with a brass pipe and a blood-filled glass column.[7] Frank used large-bore catheters connected to a rubber membrane in a 1903 manometer. [8] The invention of inflatable cuff manometers (Riva-Rocci, 1896) and the discovery of the arterial phase sounds (Korotkoff, 1905) allowed for the development of indirect blood pressure measurement. [7] [8]

The earliest recorded references to fever are from 6th century B.C. Akkadian cuneiform inscriptions, which appear to have adapted an ancient Sumerian icon of a flaming brazier to denote both fever and the local warmth of inflammation in a single ideogram. Clinical thermometry was introduced by Sanctorius in 1625. Mercury column thermometers were introduced by Fahrenheit in 1714. Although their routine use was supported by Boerhaave, thermometry was not established as routine clinical practice until the 1870s. [9]

TABLE 1-1 -- Normal Values for Vital Signs of Infants and Children (Mean ± SD)

	Age				
Parameter	***0–2 mo***	***3–12 mo***	***1–6 yr***	***7–12 yr***	***13–18 yr***
Breaths/min	—*	—*	24 ± 3	19 ± 2	17 ± 3
Pulse/min	126 ± 20	131 ± 20	88 ± 9	70 ± 8	64 ± 7
Systolic BP†	72 ± 10	95 ± 15	93 ± 13	100 ± 10	112 ± 12
Diastolic BP	51 ± 9	53 ± 10	55 ± 10	63 ± 10	67 ± 10

*For data on children 0 to 36 months, see Table 1-2 .
†As an estimate, for children 1 to 10 years: 2 × age (in years) + 90 mm Hg = 50th percentile for systolic BP.

NORMAL VALUES

The range of normal, resting vital signs for specific age groups must be recognized by the clinician to enable identification of abnormal values and their clinical significance. Normal ranges for vital signs also may be influenced by sex, race, pregnancy, and residence in an industrialized nation. These ranges have not been validated in ED patients, who may have many reasons for vital sign abnormalities, including anxiety; pain; and other forms of distress, in addition to altered physiology from disease states.

Published vital sign norms for children are not as well accepted as for adult patients. Table 1-1 and Table 1-2 report normal vital signs for children by age group as mean and standard deviations. In Table 1-1 , the values for pulse and blood pressure for 0- to 2-month-olds are adapted from studies of newborn populations (i.e., younger than 7 days).[10] [11] [12] During the newborn period, normal arterial blood pressure rises rapidly. Values for pulse and respiration in children older than 3 years reflect an average of male and female values for 0- to 1-, 3-, 9-, and 16-year-old populations. [13] The values for blood pressure reflect an average of male and female values for the 1- to 6-month and 3-, 9-, and 16-year-old populations. [12] Newer studies have reassessed reference values for RRs in children. [14] [15] [16] [17] [18] Table 1-2 reflects the age-related changes and the effect of the state of wakefulness in the RRs of children up to 3 years of age. [15] Hooker and colleagues measured resting RRs in pediatric ED patients up to age 18 years. [14] They noted considerable patient variability and somewhat higher RRs than are shown in Table 1-2 .

For the adult population, normal values for blood pressure are well established. Although there is an increase in systolic blood pressure with age, normotensive or normal systolic blood pressure is defined as 90 to 140 mm Hg, and normotensive or normal diastolic blood pressure is defined as 60 to 90 mm Hg. Although most patients have similar blood pressures in both arms, Pesola and coworkers found that 18% of their hypertensive

TABLE 1-2 -- Normal Respiratory Rates (Breaths/Min) for Children to Age 3 Years (Mean ± SD)

Age (mo)	Awake	Asleep
0–<2	48.0 ± 9.1	39.8 ± 8.7
2–<6	44.1 ± 9.9	33.4 ± 7.0
6–<12	39.1 ± 8.5	29.6 ± 7.0
12–<18	34.5 ± 5.8	27.2 ± 5.6
18–<24	32.0 ± 4.8	25.3 ± 4.6
24–<30	30.0 ± 6.2	23.1 ± 4.6
30–36	27.0 ± 4.1	21.5 ± 3.7

Adapted from Rusconi F, Castagneto M, Gagliardi L, et al: Reference values for respiratory rate in the first 3 years of life. Pediatrics 94:351, 1994.

population[17] and 15% of their normotensive population [16] had a difference of >10 mm Hg in systolic blood pressure between arms.

The normal limits for resting heart rate of 60 beats/min and 100 beats/min were established by consensus in 1928 by the New York Heart Association. [19] Recent data indicate that 45 beats/min and 95 beats/min better define the heart rate limits of normal sinus rhythm in adults of all ages. Spodick recommends that the operational definition for the limits of resting heart rate in adults should be 50 beats/min and 90 beats/min. [19] [20] This view is widely supported among cardiologists. [21]

There is currently no consensus on what constitutes a normal adult RR.

Most studies on RR support 16 to 24 breaths/min as the norm for adults.

Pregnancy results in alteration of the normal adult values for pulse and blood pressure. RR is unchanged, although the physiologic hyperventilation of pregnancy is well recognized. This is a result of increased tidal volume and decreased residual and expiratory reserve volumes. [22] Resting pulse rate increases through pregnancy to 10% to 15% over baseline values. Norms for systolic and diastolic blood pressure are dependent on patient positioning. When the pregnant patient is sitting or standing, systolic pressures are essentially unchanged. Diastolic pressures decline until approximately 28 weeks' gestation, when they begin to rise to nonpregnant levels. When the pregnant patient is in the lateral decubitus position, both systolic and diastolic pressures decline until the 28th week and then begin to rise to nonpregnant levels (Table 1-3).[23]

RESPIRATION

Respiratory frequency reveals only part of the clinical picture. The pattern, effort, and volume of respiration may be more indicative of altered respiratory physiology. An abnormality in respiration may be a primary complaint or a manifestation of other systemic disease.

Physiology

Breathing is initiated and primarily controlled in the medullary respiratory center in the brainstem. The respiratory center is modulated by the pneumotaxic and apneustic centers in the pons. The pneumotaxic center limits the length of the inspiratory signal and therefore can greatly increase or decrease RR.

In addition to being modified by other areas of the brainstem, the medullary respiratory center is modified by voluntary centers in the cerebral cortex; pulmonary stretch receptors of the airways; type J or juxtapulmonary capillary

TABLE 1-3 -- Vital Signs During Pregnancy in the Lateral Decubitus Position (Mean ± SD)

	Trimester		
Parameter	***1st***	***2nd***	***3rd***
Pulse rate (beats/min)	77 ± 2	85 ± 2	88 ± 2
Systolic BP (mm Hg)	98 ± 2	91 ± 2	95 ± 2
Diastolic BP (mm Hg)	53 ± 2	49 ± 2	50 ± 2

Adapted from Katz R, Karliner JS, Resnik R: Effects of a natural volume overload state (pregnancy) on left ventricular performance in normal human subjects. Circulation 58:434, 1978. By permission of the American Heart Association.

receptors of the pulmonary capillaries; arterial baroreceptors of the carotid sinus; and receptors found in skeletal muscle, tendons, and joints. Central and peripheral chemoreceptors also influence RR.[24]

Indications and Contraindications

Generally, ED patients should have their RR documented during their evaluation. Repeated assessment and documentation of the patient's respiratory status are indicated in patients who present with an abnormal RR or a complaint referable to the airway or breathing.

The only contraindications to a careful measurement of RR are the scenarios of respiratory distress, apnea, or upper airway obstruction that require immediate therapeutic intervention. A measurement of RR and effort should be performed as soon as patient care demands allow it in these circumstances.

Observation and palpation of chest movement are the techniques most frequently used to monitor respiratory frequency and amplitude noninvasively. Discussion of inductive plethysmography and other noninvasive monitors are beyond the scope of this chapter. Periodic manual measurements as described later generally suffice for ED patients.

Respiratory status in both adults and children plays a crucial role in determining the overall assessment of illness. Although it is a sensitive yet nonspecific indicator of respiratory dysfunction, the RR can also predict nonpulmonary morbidity. Several prehospital- and hospital-based illness or injury severity scores feature RR as a cardinal value. An prehospital RR <10 or >29 is associated with a major injury in 73% of children. [25] Other studies have linked abnormal RRs to inhospital mortality and level of care required in the ED. [26] [27] Using tachypnea alone as a predictor for pulmonary pathology, infants with a RR of >60 are found to be hypoxic 80% of the time.[28]

Procedure

RR is the number of inspirations per minute. Generally, it is best measured with the patient unaware that breathing is being observed because awareness makes the patient conscious of the breathing pattern, which may alter the rate. Commonly, examiners count respirations while appearing to count the pulse. The RR is most accurately determined by counting for a full minute. Because the frequency is much less than the pulse, and breathing is less regular, an inaccurate measurement is more likely to occur if a 15-second interval is used.

Infants, in addition to being principally nasal breathers, are predominantly diaphragmatic breathers, and an infant's RR is easily determined by observing or palpating excursion of the chest or the abdominal wall. [29]

Complications

There are no inherent complications from measuring respiration by observation. Problems related to the measurement of RR are generally due to failure to recognize a patient in obvious respiratory distress or failure to monitor RR in a patient who may be at risk for respiratory depression (e.g., in the case of sedative-hypnotic or narcotic overdose).

Interpretation

Respiratory Rate

A limited number of studies have examined RRs. Hutchinson evaluated RRs in 1897 healthy males at rest and found that 91% had RRs between 16 and 24 breaths/min. He also noted that 30% had exactly 20 breaths/min.[30] Hooker and colleagues note that current texts vary considerably in their definitions of a normal RR and cite published values that range from 8 to 20 breaths/min. [31]

Hooker and associates, in a study that specifically investigated normal RRs in an ED, measured RRs in 110 afebrile ambulatory patients without respiratory complaints (53 females and 57 males).[31] They report a mean rate of 20.1 breaths/min. For patients whose RR was measured again before release from the ED, no significant difference was noted between initial and subsequent RRs. When analyzed by gender, females had a mean RR of 20.9 breaths/min and males had a mean RR of 19.4 breaths/min, a statistically significant difference. The researchers concluded that a normal RR in the adult patient population was 16 to 24 breaths/min. [31] This study also suggested a significant variability in the accurate measurement of RR by different examiners. Rates obtained by nurses versus medical students varied significantly, as did those obtained by medical students versus residents versus attending clinicians. [1]

Other studies have provided additional information on normal resting and sleep state RRs in children younger than 7 years. [14] [15] [16] [17] [18] RRs obtained with a stethoscope were higher than those obtained by observation (mean difference, 2.6 breaths/min in awake and 1.8 breaths/min in asleep children). Smoothed percentile curves demonstrated a larger dispersion at birth (5th percentile, 34 breaths/min; 95th percentile, 68 breaths/min), while at 36 months of age (5th percentile, 18

breaths/min; 95th percentile, 30 breaths/min) dispersion was less.

RR will generally increase in the presence of fever. It is often difficult to determine if tachypnea is a primary finding or simply associated with hyperpyrexia. Taylor et al.[32] studied 572 children younger than 2 years of age, 42 (7%) of whom were subsequently diagnosed with pneumonia and found that age-appropriate limits for resting tachypnea in the presence of fever could be defined. A sensitivity and specificity of 74% and 77% for pneumonia was achieved when children 6 months of age had a RR >59/min, those aged 6–11 months had a RR >52/min and those 1–2 years had a RR >42/min. Therefore, even in the face of physiologic compensation for fever, an interpretation of RR alone can help predict the presence of pulmonary disease.

Respiratory Pattern and Amplitude

Abnormal respiratory patterns may be characteristic of metabolic or central nervous system pathologic conditions. Hyperventilation and hypoventilation may result from an

extensive differential diagnosis including primary pulmonary disorders, such as pneumonia or chest wall pain. Respiratory disturbances also occur secondary to other disease processes. For example, *Kussmaul respiration* describes the hyperventilation pattern seen in diabetics with ketoacidosis. Abnormal respiratory patterns in adults can be used in differential diagnosis or in determining the location of central nervous system lesions.

The recognition of subtle tachypnea can be difficult in the emergency setting, although this can be the solitary harbinger of disease. Measurement of an accurate RR in this patient population is crucial. Another instance of pathology that can confuse the routine measurement of RR is diaphragmatic breathing or retractions. The variability in counting respiratory effort versus effective respirations is generally not appreciated in a single recorded value.

Respiratory patterns in children must be observed carefully. In infants, periodic breathing, which may be normal, must be distinguished from apnea. By definition, periodic breathing consists of three or more respiratory pauses >3 seconds in duration, with <20 seconds between pauses. There is no associated bradycardia or cyanosis. This contrasts with apnea, which is a particular problem in preterm infants. Apnea is defined as a respiratory pause of >20 seconds. It may be associated with bradycardia and hypoxia.[29] Periodic breathing and apnea are believed to be disorders on a continuum, both stemming from abnormal physiologic control of respiration. However, periodic breathing is considered a benign disorder, whereas infants with symptomatic apneic episodes resulting in an apparent life-threatening event (ALTE) are thought to be at increased risk for sudden infant death syndrome. [33]

PULSE

The pulse is examined primarily to establish cardiac rate and rhythm. However, palpation of peripheral pulses yields clues to cardiac disease, such as aortic insufficiency, and information about the integrity of the peripheral vascular supply. Doppler ultrasound provides a noninvasive method of assessing blood flow in the ED. It has utility in the location of a pulse, in the assessment of fetal heart tones beyond the first trimester, for evaluation of peripheral lower extremity vascular insufficiency, and for the evaluation of blood pressure in infants or in patients with low-flow states.

Physiology

Blood flowing into the aorta with each cardiac cycle initiates a pressure wave. Blood flows through the vasculature at approximately 0.5 m/sec; however, pressure waves in the aorta move at 3 to 5 m/sec. Therefore, palpated peripheral pulses represent pressure waves, not blood flow.

Indications and Contraindications

The evaluation of pulse presence and rate is indicated in most patients who present to the ED. Patients with minor complaints, believed unlikely to be related to a circulatory problem, do not require this measurement. The necessity of repeated evaluations is dictated by the clinical complaint and status of the patient. Detailed pulse assessment is essential in all patients with potential peripheral vascular insufficiency. Although an association between the absence of a radial pulse (or the absence of both radial and femoral pulses) and hypotension has been demonstrated for hypovolemic trauma patients, the variability in individual response prohibits the use of this parameter as an absolute gauge of blood pressure.[34] No contraindications exist to assessment of pulse, but a few cautionary notes about the examination of the carotid pulse should be kept in mind: Concurrent bilateral carotid artery palpation should be avoided, as this maneuver could endanger cerebral blood flow. In addition, massage of the carotid sinus, found at the bifurcation of the external and internal carotid arteries at the level of mandible angle, may result in reflex slowing of the heart rate (see Chapter 11). To avoid inadvertent carotid sinus massage, the carotid pulse should be palpated at or below the level of the thyroid cartilage. A rare risk of precipitating a cerebrovascular event by *vigorous palpation* of the carotid artery is present in adults with atherosclerotic disease. This risk may be minimized by prior auscultation of the carotid artery. If a bruit is present, the carotid pulse may be gently palpated, but avoid vigorous palpation.

Equipment

Assessment of the pulse may be performed by the clinician at the bedside with any timepiece that has a second-hand display. This allows simultaneous assessment of all characteristics of the pulse: its rate, rhythm, gross perfusion pressure, and upstroke. If continuous monitoring is deemed necessary, bedside cardiac monitors can constantly monitor heart rate and rhythm and may be more accurate indicators of a perfusing rhythm than cardiac auscultation. Pulse oximetry (see Chapter 2), although primarily intended to measure oxygen saturation, also may be used to monitor the pulse rate. In a critical care situation, more sophisticated invasive monitoring techniques are available (see Chapter 20) for arterial pressure measurement and rate assessment.

Procedure

Pulses are palpable at numerous sites, although for convenience the radial pulse at the wrist is routinely used. The examiner should use the tips of the first and second fingers to palpate the pulse. The two advantages to this technique are (1) the fingertips are quite sensitive, enabling the pulse to be easily located and counted, and (2) the examiner's own pulse may be erroneously counted if the thumb is used instead of the first and second fingers. Pulses are also easily palpated at the carotid, brachial, femoral, posterior tibial, and dorsalis pedis arteries. Palpation of the pulse at the brachial artery may facilitate the appreciation of pulse contour and amplitude. It is located at the medial aspect of the elbow and is more easily palpated when the elbow is held slightly flexed.[35] Pulse rate is ideally determined by counting the pulse for 1 minute, particularly if any abnormality is present. Common convention in acute care settings is the counting of a *regular* pulse for 15 seconds and multiplying the resultant number by 4 to determine beats/min.

In newborns, direct heart auscultation and umbilical palpation are the methods of choice to determine heart rate. Instantaneous changes in newborn heart rates are best

indicated for the resuscitation team by the examiner tapping out each heartbeat.[36] In unstable children, palpation of central arteries, particularly femoral and brachial pulses, is recommended over palpation of more peripheral arteries.

In most circumstances the palpated heart rate will approximate the actual heart rate within 2%.[37]

Interpretation

Pulse Rate

Individual physiology must be considered in pulse interpretation. In infants and children, pulse rate must be interpreted with reference to age. Pulse varies with respiration, increasing with inspiration and slowing with expiration. This is known as a *sinus dysrhythmia* and is physiologic.

Although bradycardia is defined as a heart rate of <60 beats/min in adults, a well-conditioned athlete may have a normal resting heart rate of 30 to 40 beats/min.[38] As discussed earlier, a redefinition of bradycardia to <50 beats/min and tachycardia to >90 beats/min has been proposed based on a normal healthy population.[19] [20] [39] [40] These definitions of normal represent the 95% of a population and do not speak to an individual's normal baseline rate.

The clinician must consider whether an abnormal pulse rate is a primary or secondary condition. The examination of the entire set of vital signs is instrumental in discerning the cause for the abnormal rate. For example, hyperthermia causes a sinus tachycardia. Drug fever, typhoid fever, and central neurogenic fever are suggested when no corresponding tachycardia is found in a patient with elevated body temperature. Hypothermia, with its reduced metabolic demands, may be associated with bradycardia.

Clinical evaluation of the patient with an abnormal pulse rate dictates a consideration of medications the patient may be taking or the presence of a mechanical pacemaker. Digitalis compounds, ß-blockers, and antidysrhythmics may alter the normal heart rate and the ability of this vital sign to respond to a new physiologic stress. These cardioactive medications may be causing the patient's heart rate abnormality.

Heart Rhythm

In addition to determining the pulse rate, information about the regularity of the pulse is obtained during palpation. An irregular pulse suggests atrial fibrillation or flutter with variable block, and accurate assessment of the pulse should be obtained by auscultation of apical cardiac sounds. The apical pulse is frequently greater than the peripheral pulse, reflecting inadequate filling time and stroke volume, with resultant non-transmitted beats. A greater pulse deficit generally reflects more severe disease.

Pulse Amplitude and Contour

Amplitude and contour of the pulse are generally assessed simultaneously. Figure 1-1 compares normal and abnormal pulse amplitudes and contours. Accurate examination and description provide additional clinical information. Superimposition of one pathophysiologic state on another may modify the pulse. For example, sepsis may manifest with variable pulse amplitude, depending on the stage in the development of the disease at which the patient presents. Early in sepsis, cardiac output increases and vascular

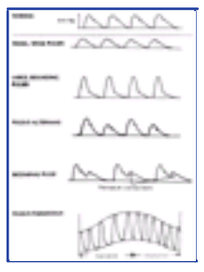

Figure 1-1 Examples of abnormalities of the arterial pulse compared with the normal pulse. The normal pulse pressure is approximately 30 to 40 mm Hg. The pulse contour is smooth and rounded. (The notch on the descending slope of the pulse wave is not palpable.) *(From Bates B: A Guide to Physical Examination and History Taking, 4th ed. Philadelphia, JB Lippincott, 1987.)*

resistance decreases, causing bounding pulses. In advanced sepsis or septic shock, falling cardiac output and increased vascular resistance are seen, and pulses are diminished.[41]

Definable age-related pulse amplitude and contour changes can be identified. Aging changes in the arterial pulse are explained by both an increase in arterial stiffness with increased pulse wave velocity and progressively earlier wave reflection. This leads to increased pulse amplitude in the elderly in all commonly measured sites (carotid, femoral, and radial).[42] In addition to these age-related changes, pulse wave analysis may be useful in determining arterial stiffness and the likelihood of atherosclerotic disease.[43] Although intriguing, the routine measurement of pulse amplitude is not reproducible by simple palpation but instead requires instrumentation not available in EDs.

Pulses During Cardiopulmonary Resuscitation

Palpated "femoral pulses" during chest compression may represent either forward arterial blood flow or "to-and-fro" movement of blood from the right heart to the venous system. A carotid pulse is preferred when assessing the adequacy of chest compressions during cardiopulmonary resuscitation (see Chapter 17).

ARTERIAL BLOOD PRESSURE

Changes in arterial blood pressure over time may indicate the success of treatment or the worsening of the patient's overall condition. An abrupt reduction in the patient's arterial blood pressure usually indicates the need for immediate intervention or reconsideration of therapy. The technique of arterial cannulation and direct intra-arterial blood pressure monitoring is discussed elsewhere (see Chapter 20). The current section discusses indirect blood pressure monitoring. Discussions of the specific use of the Doppler device for pulse and blood pressure measurement and the measurement of orthostatic blood pressure and pulse changes follow this section. As noted earlier, despite an association between the absence of a radial pulse (or the absence of both radial and femoral pulses) with hypotension, in the setting of trauma, the variability in individual response prohibits the use of this parameter as an absolute gauge of blood pressure. [34]

Physiology

Arterial blood pressure indicates the overall state of hemodynamic interaction between cardiac output and peripheral vascular resistance. Arterial blood pressure is the lateral pressure or force exerted by blood on the vessel wall. Arterial blood pressure indirectly measures perfusion, where blood flow equals the change in pressure divided by resistance.[8] However, because peripheral vascular resistance varies, a normal blood pressure does not confirm adequate perfusion. [44] Mean arterial blood pressure can be estimated by adding one third of the pulse pressure (i.e., the difference between the systolic and diastolic blood pressure) to the diastolic pressure.[8] [45]

Indications and Contraindications

Blood pressure measurement and documentation are essential for most ED patients seen for the first time. Patients with minor ambulatory complaints not related to the cardiovascular system may not receive blood pressure measurements in the ED. Patients with hemodynamic instability need frequent blood pressure monitoring.

In children, there is a significant amount of variability regarding standard situations requiring blood pressure measurement. In general, the younger the patient, the less likely a blood pressure will be obtained. [46] In newborns, infants, and even toddlers, capillary refill is frequently substituted for a standard blood pressure measurement, although viewing these tests as equivalent can lead to significant errors.

In low-flow states, Doppler measurement of blood pressure may be obtained rapidly. Repeated measurements will provide an evaluation of the adequacy of resuscitation in patients whose blood pressure cannot be auscultated by standard techniques. Placement of a catheter for direct intra-arterial measurement of blood pressure has a higher risk of complications, but it may be performed safely in the ED (see Chapter 20). In particular, direct measurement of arterial pressure during pulseless electrical rhythms may help to discriminate between severe shock and an otherwise non-resuscitatable status. [47] [48] [49] Alternative noninvasive devices for continuous blood pressure measurement are being introduced clinically, including the use of radial artery tonometry via a sensor applied over the radial artery in conjunction with an inflatable upper arm cuff. [50]

Relative contraindications to specific extremity blood pressure measurement include an arteriovenous fistula, ipsilateral mastectomy, axillary lymphadenopathy, lymphedema, and circumferential burns over the intended site of cuff application.

Equipment

The equipment required for indirect blood pressure measurement includes a sphygmomanometer (cuff with inflatable bladder, inflating bulb, controlled exhaust for deflation, and manometer) and a stethoscope or Doppler device (for auscultation) or an oscillometric device. [51] [52] [53] [54] A common practice in the prehospital and interhospital transport setting is to forego auscultatory blood pressure measurement with a stethoscope and instead obtain systolic values only via palpation of the first Korotkoff sound. This practice, although sometimes the only feasible method of obtaining any value in a noisy environment, poses significant potential for errors. In a study of critically ill patients transferred between hospitals, palpated systolic blood pressures underestimated manometric values by nearly 30%. [37] According to the American Heart Association Guideline, to ensure an accurate reading, the sphygmomanometer cuff should be of an appropriate size for the patient. The width of the bladder should be at least 40% of the distance of the limb's midpoint (i.e., from the acromion process to the lateral epicondyle). This published figure of ideal width, when studied in a validation review, may be higher up to approximately 50%. [55] The length of the bladder should be 80% of the midarm circumference or twice the recommended width.[45]

Manometers in common use are either an aneroid or a mercury gravity column. Both types of manometers are convenient for bedside use, although the mercury gravity column must be placed vertically to ensure accurate measurements. An aneroid manometer uses a metal bellows that elongates with the application of pressure. This elongation is mechanically amplified, transmitting the motion to the indicator needle.

Manometers require annual servicing. Mercury columns may require the addition of mercury to bring the edge of the meniscus to the zero mark. The air vent or filter at the top of the mercury column should also be checked for clogging. The aneroid manometer should be calibrated against a mercury column at least yearly. If the aneroid indicator is not at zero at rest, the device should not be used. [56]

Automatic sphygmomanometers may improve physiologic monitoring with alarm and self-cycling capabilities. They offer indirect arterial blood pressure measurement with little pain and lack the risks associated with invasive arterial lines. [57] Various manufacturers use oscillometric (Dinamap 845, Applied Medical Research, Tampa, FL), Korotkoff sound (Pressurometer, Avionics, Irvine, CA); and ultrasonic (Arteriosonde, Hoffmann-LaRoche Co, Nutley, NJ) techniques. Oscillometric blood pressure monitors detect motion of the blood pressure cuff transmitted from the underlying artery. A sudden increase in the amplitude of arterial oscillations occurs at systolic pressure and mean arterial pressure, and an abrupt decrease occurs at diastolic pressure.

Dinamap blood pressures obtained in 29 hemodynamically stable children (mean age, 18 months) demonstrated a smaller mean error (systolic, -0.24 vs 3.26 mm Hg) than auscultatory measurements in 20 children (systolic, -1.65 vs 6.68 mm Hg) using direct radial arterial pressures for comparison. [58] Park and Menard also found less variability with the Dinamap vs the auscultatory method in children. [59]

These results are not, however, generalizable to the neonatal population, where errors are commonly encountered even when exhaustive measures are taken to control the environment. [60]

In adult patients, numerous studies have focused on reliability of auscultory vs automated blood pressure measurements. Mercury column vs Dinamp readings showed increased disparity at systolic blood pressures >140 mm Hg, the range where accuracy should be most rigorously sought to correctly identify hypertension. In general, automated blood pressure readings yielded higher systolic blood pressures and lower diastolic values. [61] The range of error in automated devices was on average 4.0 to 8.6 mm Hg.[62] Unfortunately, these studies represent populations without critical illness and do not reflect accuracy of readings in the extremes of hypertension and hypotension. This makes generalization to an ED population difficult.

Procedure

Indirect blood pressure measurements may be obtained at the patient's bedside by palpation, auscultation, Doppler, or oscillometric methods. The technique is

straightforward and accurate when well-maintained, calibrated equipment is used by practitioners who follow accepted standards. The patient may be lying or sitting, as long as the site of measurement is at the level of the right atrium and the arm is supported. [45] [51] Interestingly, unless the arm is kept perpendicular to the body, measurements will be 9 to 14 mm Hg higher regardless of body position.[52] Hence, allowing the arm to be parallel to the body when supine, but supporting the arm perpendicular to the body when measuring the blood pressure in either the sitting or standing position may create a pseudo drop in blood pressure.

Palpation of arterial blood pressure requires cuff inflation to 30 mm Hg above the level at which a palpable pulse disappears. Once properly inflated, the examiner should palpate directly over the artery and deflate the cuff at 2 to 3 mm Hg per second. The initial appearance of arterial pulsations is reported as the palpable blood pressure. The same technique may be used with the Doppler device with the Doppler auditory signal replacing the palpated pulse. Arterial pressure measurement by palpation and Doppler yields only systolic blood pressure estimates. The Doppler method is preferred when obtaining blood pressures from infants. [63]

When auscultating the blood pressure at the brachial artery, the blood pressure cuff is applied about 2.5 cm above the antecubital fossa with the center of the bladder over the artery.[51] The bell of the stethoscope is applied directly over the brachial artery but with as little pressure as possible. [64] The systolic arterial blood pressure is defined as the first appearance of faint, clear, tapping sounds that gradually increase in intensity (Korotkoff phase I), whereas the diastolic blood pressure is defined as the point at which sounds disappear (Korotkoff phase V). [51] [64] In children, phase IV defines the diastolic blood pressure (Fig. 1-2). [12] Phase IV is marked by a distinct, abrupt muffling of sound when a soft, blowing quality is heard.

Measurement by auscultation over the brachial artery is preferred because of accepted standardization of measured values. Alternate sites include the radial, popliteal, posterior tibial, or dorsalis pedis arteries, although any fully compressible extremity artery may be used. Studies correlating direct and indirect blood pressure measurements have demonstrated a good correlation between these methods. [65] [66] [67]

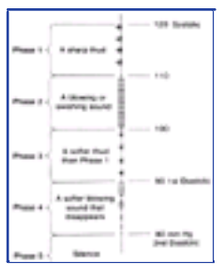

Figure 1-2 Korotkoff sounds. Systole—first audible sound. Diastole—sound disappears. *(From Burnside JW, McGlynn TJ: Physical Diagnosis, 17th ed. Baltimore, Williams & Wilkins, 1986.)*

There are situations in which it may not be feasible to obtain upper extremity blood pressure measurements because of patient access issues, particularly those encountered in the prehospital setting. Forearm measurements may be more easily obtained, and show fair correlation to standard upper extremity values (within 20 mm Hg in 86% of systolic measurements and 94% of diastolic measurements).[68] Alternatively, noninvasive finger blood pressure measurements have shown promise when compared to standard upper extremity readings. The overall discrepancy in an ED study was 0.1 mm Hg with a standard deviation of ±5.02 mm Hg when comparing finger blood pressures and invasive mean arterial blood pressure via radial artery cannulation. [69]

The accuracy of palpatory, Doppler, and oscillometric methods has also been demonstrated. [70] [71] [72] [73] However, when phase I and V Korotkoff sounds are used, indirect methods typically underestimate systolic and diastolic pressure by several mm Hg. [66] [74] In addition, during shock, palpatory and auscultatory methods underestimate simultaneous direct arterial pressure measurements.[75] The flush method, in which return of color after deflation of the cuff is used for estimating blood pressure in infants, may underestimate systolic blood pressure by up to 40 mm Hg. [74] This method is unreliable and is not recommended.

Complications

Complications of indirect blood pressure measurement are minimal when the proper procedure is followed. Inadvertent prolonged application of an inflated blood pressure cuff may

result in false elevation of diastolic pressure and in ischemia distal to the site of application, with attendant complications. [8] [53] Invasive blood pressure monitoring is associated with a number of potential problems (see Chapter 20).

Interpretation

Normal blood pressure increases with decreasing distance from the aorta. Blood pressure tends to increase with age and is generally higher in males. Individual factors that influence blood pressure include body posture, emotional or painful stimuli, environmental influences, vasoactive foods or medications, and the state of muscular and cerebral activity. Exercise and sustained isometric muscular contraction increase blood pressure in proportion to the strength of the contraction. [8] A normal diurnal pattern of blood pressure consists of an increase throughout the day with a significant, rapid decline during early, deep sleep. [76]

Normal lower limits for systolic blood pressure for infants and children can be estimated by adding 2 times the age (in years) to 70, with the result expressed in mm Hg. The 50th percentile for a child's systolic arterial blood pressure from 1 to 10 years of age can be estimated by adding 2 times the age (in years) to 90 mm Hg. Children older than 2 years are considered hypotensive when systolic blood pressure is <80 mm Hg. [77] Children, in particular, are able to maintain mean arterial blood pressure until very late during shock.[78] Thus the finding of a normal blood pressure in a child with signs of poor perfusion should not dissuade the practitioner from appropriate treatment. Adults are considered hypotensive if the systolic blood pressure is <90 mm Hg. When accompanied by signs of shock, immediate treatment is indicated. In patients with shock, blood flow cannot be reliably inferred from heart rate and blood pressure values. [79] [80]

Hypertension

Adults are hypertensive if either the systolic or the diastolic pressure consistently exceeds 140 or 90 mm Hg, respectively. [81] [82] The applicability of population norms for hypertension in a stressful emergency situation is controversial. One should not make diagnostic or therapeutic decisions based solely on an abnormal initial measurement. Patients with hypertension require repeat measurement to assess whether ED therapy is required. Because sustained hypertension may be seen in more than a third of initially hypertensive ED patients, careful evaluation and follow-up are required. [83]

The phenomenon of white coat hypertension (WCH) is defined as the persistent elevation of blood pressure in the clinical setting only. In order to strictly fit this definition, patients must have a normal ambulatory blood pressure outside of the clinical setting. WCH should not be confused with the white coat effect (WCE), a finding of increased blood pressure values in nearly all patients within the clinical setting. The white coat effect is used to characterize exaggerated hypertension in a normally hypertensive patient, frequently pushing moderately controlled hypertension readings into significantly higher readings while in the clinical setting. The prevalence of WCH is between 20% and 94%, depending on the frequency of clinical setting reassessment. [84] [85] [86] Patients in whom WCH or the WCE is most likely to occur are women, nonsmokers, and the elderly.[86] [87] It is unclear whether these patients who have isolated clinical setting hypertension (WCH) are at increased risk for developing hypertension and subsequent end-organ damage. [88]

Measurement Errors

Erroneous blood pressure measurements may result from several factors.[82] [89] Falsely low blood pressure may be caused by using an overly wide cuff, by placing excessive pressure on the head of the stethoscope, or by rapid cuff deflation. [90] [91] Falsely high blood pressure may be caused by the use of an overly narrow cuff, anxiety, pain, tobacco use, exertion, an unsupported arm, or slow inflation of the cuff.

In 470 unselected adults, investigators at Duke University found no spurious effect of cuff size in 350 patients weighing <95 kg and with an arm circumference <35 cm. However, in 120 patients weighing >95 kg and with an arm circumference >35 cm, the use of a large cuff reclassified 33% of those with systolic hypertension to borderline, 62% of those with borderline systolic hypertension to normal, and 79% of patients with borderline diastolic hypertension to normal. [92] Of note, 41% of

adults observed at the University of Pittsburgh required nonstandard size cuffs, and use of small cuffs was associated with a mean error of 8.5 and 4.6 mm Hg in the systolic and diastolic pressures, respectively. [93] Other studies have confirmed relatively high rates of inappropriately diagnosed hypertension among obese patients based on erroneous cuff size.[94]

Hypotensive patients have unreliable Korotkoff sounds. However, Doppler measurements are well correlated with direct arterial systolic pressure in hypotensive patients.[95]

An auscultatory gap can be appreciated in hypertensive patients and may mislead the examiner. It is heard during the latter part of phase I and should not be confused with diastolic readings. Auscultation until the manometer reading approaches zero should prevent misinterpretation. In patients with aortic insufficiency or hyperthyroidism, in persons who have just finished exercising, and in children younger than 5 years of age, the measurement of diastolic blood pressure should occur at Korotkoff phase IV.

Irregular heart rates also may interfere with accurate blood pressure determination. A second or third reading, with 2 minutes of deflation between recordings, should be used to obtain an average when premature contractions or atrial fibrillation is present. Ultrasonic methods may be more accurate during shock states and in infants for systolic blood pressure measurement.[85] [95]

Dewar demonstrated that hemiplegic patients may exhibit different blood pressures in affected and unaffected arms. [96] A flaccid extremity tended to yield lower systolic and diastolic pressures, whereas a spastic extremity tended to yield higher values than the extremity with normal motor tone. Although these differences are generally small, it is preferable to monitor blood pressure in the unaffected limb.

As noted earlier, numerous errors may occur in the measurement of accurate blood pressure. The only way to combat them is to first be cognizant of practices contributing to them. Unfortunately, few nurses can identify causes of potentially erroneous readings. In a study examining nurses' ability to accurately obtain readings, proper techniques in obtaining systolic blood pressure could be identified 61% of the time, diastolic blood pressure 71%, and an auscultatory gap 54%. Nurses were able to correctly determine faulty equipment 58% of the time, assess cuff size 57%, determine appropriate

inflation pressure 29%, determine appropriate deflation rate 62%, and determine correct arm positioning only 14%. [97]

Pulse Pressure

Increased pulse pressure (i.e., =60 mm Hg) is commonly observed in anemia, exercise, hyperthyroidism, arteriovenous fistula, aortic regurgitation, and patent ductus arteriosus. A narrowed pulse pressure (=20 mm Hg) may be a manifestation of hypovolemia, increased peripheral vascular resistance, or decreased stroke volume.

Differential Brachial Artery Pressures

The presence of systolic blood pressure differences between arms is suggestive of advanced focal atherosclerosis, coarctation of the aorta proximal to the left subclavian artery, aortic dissection, other aortic arch syndromes, or other vascular processes preferentially affecting one extremity. Although most patients have similar blood pressures in both arms, Pesola and coworkers found that 18% of their hypertensive population [17] and 15% of their normotensive population[16] had a difference of >10 mm Hg in systolic blood pressure. Others report that only 1.4% of elderly patients had a systolic brachial blood pressure difference of >10 mm Hg, although 6.5% had a difference exceeding 7.5 mm Hg.[98] Panayiotou noted that for most stroke patients, the difference between the paretic and normal arm was only 4 to 5 mm Hg.[99] However, differences of 9 to 12 mm Hg were noted for some patients. Hence, differential brachial blood pressures must be interpreted within the clinical context of the patient's presentation.

Pulsus Paradoxus

Normal respiration decreases the systolic blood pressure by approximately 10 mm Hg during inspiration. Pulsus paradoxus occurs when there is a >12 mm Hg decrease in the systolic blood pressure during inspiration. Pulsus paradoxus may occur in patients with chronic obstructive pulmonary disease, pneumothorax, severe asthma, and pericardial tamponade.[100] Other conditions such as an atrial septal defect, aortic insufficiency, and poor left ventricular compliance have been associated with pulsus paradoxus without pericardial fluid.

To measure a paradoxical pulse, the patient should be lying comfortably, at a 30° to 45° angle, and breathing normally in an unlabored fashion (unusual conditions in a patient suspected of cardiac tamponade, severe asthma or chronic obstructive pulmonary disease, or pneumothorax). [101] The blood pressure cuff is inflated well above systolic pressure and is slowly deflated until one first hears the systolic sounds that are synchronous with expiration (Fig. 1-3). Initially, one will hear the arterial pulse only during expiration, and it will disappear during inspiration. The cuff is then further deflated until arterial sounds are heard throughout the respiratory cycle. A paradoxical pulse can be palpated if it is very large. During palpation the pulse may completely disappear during inspiration. When present, this technique is a quick bedside confirmation of the possibility of severe tamponade. Palpation for this purpose is best done at peripheral arteries, such as the radial or femoral.

An alternative approach to measurement of pulsus paradoxus is to use a finger arterial pressure monitor (Finapres; Ohmeda, Englewood, CO) and to subtract the peak systolic blood pressure during expiration from the lowest systolic

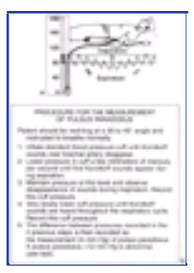

Figure 1-3 *A*, Measurement of pulsus paradoxus. Note that the systolic pressure varies during the respiratory cycle. *(From Stein L, Shubin H, Weil M: Recognition and management of pericardial tamponade. JAMA 225:504, 1973. Copyright 1973, American Medical Association. Reproduced by permission.) B*, Technique for the measurement of pulsus paradoxus.

blood pressure during inspiration. [102] The pulsus paradoxus obtained with this technique was found to have less variability (when compared to intra-arterial measurements) than with manual measurements. Furthermore, pulsus paradoxus obtained using the finger pressure monitor correlates well with the pulmonary index score in asthmatic children.[103] Changes in the pulsus paradoxus were found to correlate with other markers of clinical status and admission decisions.

If the difference between these inspiratory and expiratory pressures is >12 mm Hg, the paradoxical pulse is high. [104] Most patients with proven tamponade have a difference of =20 to 30 mm Hg during the respiratory cycle.[105] [106] This may not be true of patients with very narrow pulse pressures (typical of advanced tamponade), who have a "deceptively small" paradoxical pulse of 5 to 15 mm Hg. The relative decrease in pulsus paradoxus occurs because the paradoxical pulse is a function of actual pulse pressure, and the inspiratory systolic pressure may be below the level at which diastolic sounds disappear. [101] For this reason, the ratio of paradoxical pulse to the pulse pressure is a more reliable

measure. A paradoxical pulse >50% of the pulse pressure is abnormal.[101]

Pulsus paradoxus has been correlated with the amount of impairment of cardiac output by tamponade. In uninjured patients with pericardial effusion, a pulsus paradoxus >25 mm Hg (in the absence of relative hypotension) was both sensitive and specific for moderate or severe versus mild tamponade. [104] A similar study of

right ventricular diastolic collapse by echocardiography found that an abnormal pulsus paradoxus had a sensitivity of 79%, specificity of 40%, positive predictive value of 81%, and negative predictive value of 40%.[107] The absence of a paradoxical pulse does not rule out tamponade (see Chapter 16).

In the pediatric population, pulsus paradoxus has been studied to determine the disease severity of obstructive and restrictive pulmonary disease,[108] most commonly asthma. A value of 15 mm Hg or greater correlates well with clinical score, peak expiratory value, flow rate, oxygen saturation, and subsequent need for admission.[109]

Despite the disease entities that a widened pulsus paradoxus may suggest, it is a difficult task to perform adequately using only a syphgmomanometer. In a study by Jay et al., emergency clinicians and critical care specialists were unable to reliably measure pulsus paradoxus in a trained reference subject either by palpation or by syphgmomanometer. The variance of actual versus measured pulsus paradoxus was greater with increasing pulsus paradoxus values into the pathologic range, lowering significantly the positive predicative value of the test.[110] The author's conclusion was that new aids should be developed and used to reliably predict this important vital sign.

Shock Index

The ratio of the pulse rate over the systolic blood pressure has been suggested as a measure of clinical shock. The shock index (SI) has a normal range of 0.5 to 0.7. A number of clinical scenarios have been studied using the shock index as a predictor of severe illness or injury. A shock index >0.85 to 0.90 suggests acute illness in medical patients, as well as a marked increase in potential for gross hemodynamic instability in a trauma patient.[111] [112] [113] [114] In a study evaluating first-trimester pregnancy, those patients with a shock index >0.83 were 15 times more likely to be diagnosed with an ectopic pregnancy in the ED.[111] [115] However, some studies have found that the presenting pulse rate alone had nearly the same predictive power for severity of illness as the shock index. Further, Rady and coworkers demonstrated that although the SI appeared to correlate with left ventricular stroke work index, it had little correlation with systemic oxygen transport in hemorrhagic and septic shock.[112]

DOPPLER ULTRASOUND FOR EVALUATION OF PULSE AND BLOOD PRESSURE

Principles of Doppler Ultrasound

Doppler ultrasound is based on the Doppler phenomenon: The frequency of sound waves varies depending on the speed of the sound transmitter in relation to the sound receiver. Doppler devices transmit a sound wave that is reflected by flowing erythrocytes, and the shift in frequency is detected. Frequency shift can only be detected for blood flow >6 cm/sec.

Indications and Contraindications

Doppler ultrasound is commonly used in the ED for the measurement of blood pressure in low-flow states, evaluation of lower extremity peripheral perfusion, and assessment of fetal heart sounds after the first trimester of pregnancy. Doppler sensitivity allows the detection of systolic blood pressure down to 30 mm Hg in the evaluation of a patient in shock. In the patient with peripheral vascular disease in whom there is concern about the adequacy of peripheral perfusion, the ankle/brachial index provides a rapidly obtainable, reproducible, and standardized assessment. [116] Fetal heart sounds provide a baseline assessment of any patient with =12 weeks' gestation in whom there is possible abdominal trauma or fetal distress due to a pregnancy complication.

The use of Doppler ultrasound in the evaluation of deep venous thrombosis is a valuable tool; however, it requires specific training and experience to attain proficiency. Discussion of this topic is beyond the scope of this chapter.

Equipment

A nondirectional Doppler device has a probe that houses two piezoelectric crystals. One crystal transmits the signal and the other receives it. Reflected signals are converted to an electrical signal and fed to an output that transforms them to an audible sound. Two commonly used Doppler units are the pocket Doppler stethoscope (model BF4A, Medsonics, Inc, Los Altos, CA) and the ultrasonic Doppler flow detector (model 811, Parks Medical Electronics, Aloha, OR) (Fig. 1-4 and Fig. 1-5).

Probes with a frequency of 2 to 5 MHz are best for detecting fetal heart sounds. Frequencies of 5 to 10 MHz are appropriate for limb arteries and veins. The probes should be monitored periodically for electrical damage and integrity of the crystal. Sphygmomanometers used in conjunction with the Doppler device should be calibrated periodically, as described in the section on blood pressure evaluation.

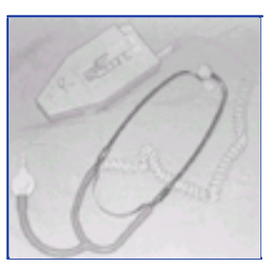

Figure 1-4 Pocket Doppler stethoscope (model BF4A). *(Courtesy of Medsonics Inc, Los Altos, CA.)*

Figure 1-5 Ultrasonic Doppler flow detector with speaker and probes (model 811). *(Courtesy of Parks Medical Electronics, Aloha, OR.)*

Procedure

The Doppler probe is placed against the skin using an acoustic gel as an interface. The gel ensures optimal ultrasound signal transmission and reception and protects the crystals. In an emergency, water-soluble lubricant (e.g., Surgilube or K-Y jelly) may be substituted for commercial acoustic gel. The probe is angled at 45° along the length of the vessel to optimize frequency shifts and signal amplitude.

In the evaluation of peripheral perfusion, a sphygmomanometer cuff is placed proximal to the arterial pulse and inflated. The probe is placed over the arterial pulse and the cuff is slowly deflated. The pressure at which flow is first heard is the systolic pressure.

In the evaluation of peripheral vascular disease, the ankle/brachial index is determined. Both brachial arteries are examined at the medial aspect of the antecubital fossa. The probe is angled until the most satisfactory signal is obtained. The cuff is inflated and slowly deflated until the systolic pulse is heard. The procedure is repeated for the posterior tibial and dorsalis arteries of both lower extremities.

In the evaluation of fetal heart tones, variable positioning of the fetus may require examination at several locations over the uterus and angling of the probe to search for the optimal signal. It is best to begin in the mid-suprapubic area and to explore the uterus via angulation of the probe. Once tones are located, the probe can be moved along the abdomen to reach a position closer to the origin of the sound. Fetal heart tones are distinguished from placental flow by the discrete quality of the fetal heart tones and the rate of placental flow, which matches the maternal pulse.

Interpretation

As noted earlier, in low-flow states, Doppler ultrasound can detect a blood pressure as low as 30 mm Hg. The ankle/brachial index of each limb is calculated by dividing the higher systolic pressure of the posterior tibial or the dorsalis pedis artery of the limb by the higher of the systolic pressures in the brachial arteries. In normal individuals, the index should be >1.0. Patients with claudication have values between 0.6 and 0.8. Values <0.5 imply severe impairment and are consistent with rest pain or gangrene. [116] When the lower extremity has been amputated or is itself injured, brachial/brachial indices can be used (i.e., the systolic blood pressure of the injured or diseased upper extremity compared to the other. Patients with ankle/brachial index values of =0.9 have been found to have increased cardiovascular morbidity and mortality. [117] One study of 323 penetrating extremity wounds found the ankle/brachial index (or the brachial/brachial index) of <0.9 to be 72.5% sensitive and 100% specific for the 29 vascular injuries. [118] Segmental lower extremity pressure measurements may help to identify the level of obstruction (Fig. 1-6 Fig. 1-7 Fig. 1-8). [119] Obese patients, diabetic patients, or patients with calcified vessels that are not compressible may have abnormally high systolic pressures (e.g., 250 to 300 mm Hg) and indices that do not accurately reflect flow.

Normal fetal heart tones should be between 120 and 140 beats/min. Fetal tones may be heard as early as the 12th week of gestation.

ORTHOSTATIC VITAL SIGNS MEASUREMENT

Orthostatic vital signs are used to evaluate patients with fluid loss, hemorrhage, syncope, or autonomic dysfunction. They are also used to assess the patient's response to therapy. The practitioner is often concerned with the accurate detection of

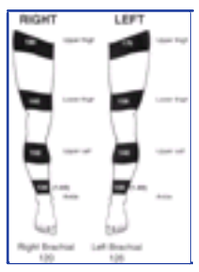

Figure 1-6 Typical pressures in a normal subject. Findings, based on resting pressures, show no evidence of occlusive disease of the large- or medium-sized arteries. Significant findings (normal) are as follows: (1) Ankle-to-brachial pressure index =1.0. (2) All pressure gradients <30 mm Hg. (3) Upper thigh pressure at least 40 mm Hg above brachial pressure. *(From Doppler Evaluation of Peripheral Arterial Disease: A Clinical Handbook. Fredericksburg, VA, Sonicaid, Inc. Reproduced by permission.)*

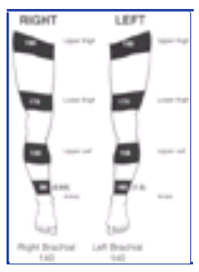

Figure 1-7 Typical pressures in a patient with obstruction of the popliteal or tibial arteries. Significant findings are as follows: (1) Ankle-to-brachial pressure index <0.9 in right leg. (2) Abnormally high gradient from ankle to below knee and again from below to above knee in right leg. (3) Upper thigh pressures are 50 mm Hg higher than brachial pressures, consistent with normal flow at the aorta-iliac level. Findings are suggestive of a right popliteal occlusion or an anterior and posterior tibial occlusion, or both. *(From Doppler Evaluation of Peripheral Arterial Disease: A Clinical Handbook. Fredericksburg, VA, Sonicaid, Inc. Reproduced by permission.)*

acute blood loss or volume depletion. When the clinical syndrome of shock exists, assessment of a blood volume deficit poses little difficulty. It is preferable, however, that volume loss be detected before loss of physiologic compensation and clinical shock occurs. This section addresses the utility of orthostatic vital signs in the detection and monitoring of acute volume depletion.

Many techniques have been advocated to assess volume status. Unfortunately, most procedures lack a database against which to judge their reliability. Recommended methods include evaluation of: skin color; skin turgor; skin temperature; supine, serial, and orthostatic vital signs; neck vein status; transcutaneous oximetry; and hemodynamic monitoring (e.g., monitoring of central venous pressure) (see Chapter 22). Serial vital sign measurements have been used for assessing blood loss, but they do not reliably detect small degrees of blood loss. [120] [121] [122] *Up to 15% of the total blood volume can be lost with minimal hemodynamic changes or any alteration of the supine vital signs.*[120] A decrease in the pulse pressure occurs with acute blood loss, [122] but the patient's baseline blood pressure values are often unknown. Clinical examination of neck veins adds useful information but is less precise than measurement of central venous pressure. Most clinicians use skin color, temperature, and moisture as a reflection of skin perfusion and sympathetic tone but not as an accurate guide to circulatory volume, because the vasomotor tone of the skin is affected by numerous diseases as well as by

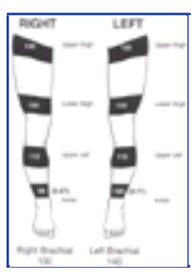

Figure 1-8 Typical pressures in a patient with obstruction of the abdominal aorta or bilateral iliac obstruction. Significant findings are as follows: (1) Ankle-to-brachial pressure index <0.9. (2) All segmental gradients <30 mm Hg. (3) Both upper thigh pressures relatively low with respect to brachial pressure. Findings are suggestive of severe aorto-iliac occlusive disease. *(From Doppler Evaluation of Peripheral Arterial Disease: A Clinical Handbook. Fredericksburg, VA, Sonicaid, Inc. Reproduced by permission.)*

emotional and environmental factors. Capillary refill has been advocated as an ideal noninvasive test for hypovolemia, but it has not been found to be accurate in adults [123] (see the following discussion regarding its use for children).

The ideal test for determining volume status would rapidly and accurately detect volume depletion of 5% or more using a noninvasive technique. At present, no such test exists. Orthostatic vital signs meet the criteria of being noninvasive and easily used at the bedside. However, *in patients with acute blood loss of <20% of total blood volume, orthostatic vital signs lack both sensitivity and specificity*.[124] Further, ethanol ingestion exaggerates postural pulse changes, thus mimicking the hemodynamic changes seen with acute blood loss. [125]

The medical literature is replete with unsubstantiated claims regarding what constitutes a positive or negative orthostatic test, and its value for estimating volume status may be overstated. Some authors[126] [127] have stated that postural hypotension or postural tachycardia occurs with varying degrees of hypovolemia, but they do not define specific criteria for a positive test. Other sources (without documentation) have perpetuated the notion that relatively small changes in the orthostatic blood pressure or the pulse are reliable in detecting hypovolemia. For example, Hayes and Briggs state erroneously that "a decrease of 10 mm Hg or more on assuming the sitting position indicates significant hypovolemia." [128]

The next section discusses the physiologic compensatory mechanisms that are activated by hypovolemia and postural tilting and the clinical use of orthostatic vital signs to detect acute volume loss.

TABLE 1-4 -- Homeostatic Mechanisms in Hemorrhagic Shock

Sympathetic reflex compensation
Arteriolar vasoconstriction
Venous capacitance vasoconstriction
Increased inotropic and chronotropic cardiac activity

Central nervous system ischemic response
Selective increase in cerebral and coronary perfusion by means of local autoregulation
Increased oxygen unloading in tissues
Restoration of blood volume
Renin-angiotensin-aldosterone axis activation
Antidiuretic hormone secretion
Transcapillary refill
Increased thirst resulting in increased fluid intake
Increased erythropoiesis

Physiologic Response to Hypovolemia

Acute blood loss decreases the pressure gradient between the venules and the right atrium. A fall in this pressure gradient decreases venous return. [129] As a result, cardiac output falls, and clinical manifestations of shock ensue. Several homeostatic mechanisms are initiated by acute blood loss (Table 1-4). The dominant compensatory mechanism in shock is a reduction in the carotid sinus baroreceptor inhibition of sympathetic outflow to the cardiovascular system. This increased sympathetic outflow results in several effects: (1) an arteriolar vasoconstriction, which greatly increases total peripheral vascular resistance; (2) a constriction of venous capacitance vessels, thereby increasing venous return to the heart; and (3) an increase of heart rate and force of contraction, which helps to maintain cardiac output despite significant volume loss. [130] These sympathetic reflexes are geared more for the maintenance of arterial pressure than for the maintenance of cardiac output (Fig. 1-9). The value of sympathetic reflex compensation is illustrated by the fact that 30% to 40% of the blood volume can be lost before death occurs while these reflexes are intact. When the sympathetic reflexes are absent, loss of only 15% to 20% of the blood volume may cause death. [130]

Several other reflexes maintain cardiac output in the presence of volume loss. The central nervous system ischemic response stimulates the sympathetic nervous system after the arterial pressure falls below 50 mm Hg and is responsible for the second plateau on the arterial pressure curve (see Fig. 1-9).[130] Other compensatory mechanisms that tend to restore the blood volume to a normal level include the release of angiotensin and antidiuretic hormone, which cause arteriolar vasoconstriction and conservation of salt and water by the kidneys. [131] A fluid shift from the interstitium to the intravascular space occurs, helping to restore blood volume over a longer period (1 to 40 hours). [132] [133]

When blood loss results in anemia, part of the loss in oxygen-carrying capacity is countered by an increase in tissue oxygen extraction. [134] Finally, the lost red blood cell mass is slowly replaced by erythropoiesis.

Several investigators have examined the changes in blood pressure and pulse that occur in the supine patient with blood loss. [120] [121] [122] Collectively these studies have shown

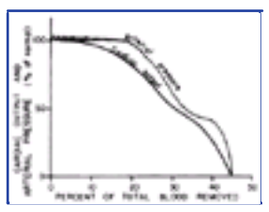

Figure 1-9 The effect of hemorrhage on cardiac output and arterial pressure. *(From Guyton AC: Textbook of Medical Physiology, 6th ed. Philadelphia, WB Saunders, 1981. Reproduced by permission.)*

variable individual hemodynamic responses to acute blood loss of up to 1 L. The frequent inability to detect significant volume loss with supine vital signs and the observation that patients with acute volume loss frequently develop syncope on rising led to the investigation of the use of orthostatic vital signs to detect occult hypovolemia.

Physiologic Response to Postural Changes

When an individual assumes the upright posture, complex homeostatic mechanisms compensate for the effects of gravity on the circulation to maintain cerebral perfusion. These responses include (1) baroreceptor-mediated arteriolar vasoconstriction, (2) venous constriction and increased muscle tone in the legs and the abdomen to augment venous return, (3) sympathetic-mediated inotropic and chronotropic effects on the heart, and (4) activation of the renin-angiotensin-aldosterone system.[135]

These compensatory mechanisms preserve cerebral perfusion in the upright position with minimal changes in vital signs. Currens found that when normal subjects stand, the pulse increases by an average of 13 beats/min, systolic blood pressure falls slightly or does not change, and diastolic pressure rises slightly or does not change.[136] These changes have been confirmed by others. [124] [125] [126]

In patients with *vasodepressor syncope*, the normal compensatory reflexes that preserve cerebral perfusion with postural changes are altered. The normal increased sympathetic tone on standing is paradoxically inhibited, and an exaggerated enhancement of parasympathetic activity (bradycardia) occurs, which can lead to syncope.[137]

Few data exist on the effect of acute blood loss on postural vital signs. One early study [122] looked at 23 young adult volunteers from whom 500 to 1200 mL of blood was withdrawn. They found no reliable change in the postural blood pressure, but a consistent postural increase in the pulse of 35% to 40% was noted after a 500-mL blood loss. In the 6 subjects who were bled approximately 1 L, only 2 were able to tolerate standing; each of them had a postural increase in pulse of >30 beats/min. The other four subjects experienced severe symptoms on standing, followed by a marked bradycardia and syncope if they were not allowed to lie down.

Knopp and colleagues[124] phlebotomized 450 to 1000 mL of blood from healthy volunteers. By using the criterion of a pulse increase of 30 beats/min or the presence of severe symptoms (syncope or near syncope) during a supine-to-standing test, they distinguished accurately between a 1000-mL blood loss and no blood loss. Changes in blood pressure and pulse were not evaluated in the symptomatic subjects. In this study population of 100 normal healthy volunteers with acute blood loss, the sensitivity and specificity using the aforementioned criteria for detecting a 1000-mL blood loss (Table 1-5) were both 98%, giving an accuracy of 96% (2% false-negative results and 2% false-positive results). The investigators were unable to consistently detect a blood loss of 500 mL by using these criteria. In a similar study by Kosowsky and coworkers,[138] the change in heart rate with postural changes after a 500-mL phlebotomy was more discriminatory for the blood loss than blood pressure changes or the change in bioimpedance-based stroke index changes. Of note, these authors found a heart rate change of =30 beats/min was 13.2% sensitive and 99.5% specific for the 500-mL blood loss vs a heart rate change of =20 beats/min that was 44.7% sensitive and 95.4% specific. Hence, the finding of a significant pulse rise, although insensitive for a 500-mL blood loss, was relatively specific in these healthy adult blood donors.

In the most recent meta-analysis of orthostatic vital signs, the authors concluded that a large postural pulse change (>30 beats per minute) *or* severe postural dizziness precluding the completion of vital sign measurements are required to clinically diagnose hypovolemia secondary to acute blood loss. However, the analysis demonstrates that orthostatic vital signs are often absent after *moderate* amounts of blood loss, significantly limiting the test's sensitivity (22%) in this scenario. [139]

Variables Affecting Orthostatic Vital Signs

Many conditions affect the compensatory mechanisms that allow us to assume the upright posture (Table 1-6).[135] Because of decreased vasomotor tone, limited

chronotropic response, and other factors, the elderly have a higher incidence of orthostatic

TABLE 1-5 -- Summary of Orthostatic Tilt Testing*

Test Procedure
1. Blood pressure and pulse are recorded after patient has been supine for 2–3 min
2. Blood pressure, pulse, and symptoms are recorded after patient has been standing for 1 min; the patient should be permitted to resume a supine position immediately should syncope or near-syncope develop
Positive Test
1. Increase in pulse of 30 beats/min or more in adults, *or*
2. Presence of symptoms of cerebral hypoperfusion (e.g., dizziness, syncope)

*The predictive ability of orthostatic vital signs to assess volume status is often *overestimated* in clinical practice. This suggested *guide* is based on the ability of the pulse change and patient symptoms to distinguish between no acute blood loss and a 1000 mL acute blood loss in healthy, previously normovolemic volunteers (sensitivity of 98% for detecting 1000 mL acute blood loss). [124] *This guide may not be applicable to elderly patients, sick children, medicated patients, and those with autonomic dysfunction.*

hypotension, which can lead to syncope and fall-related injury, [140] although carotid sinus hypersensitivity may play a greater role in geriatric syncope than orthostasis does.[141] Note that drugs that antagonize the normal autonomic compensatory mechanisms can also produce orthostatic changes. These changes can be severe enough to produce frank syncope, especially in the elderly. However, in one study of *euvolemic* adult volunteers, orthostatic changes in patients with diabetes or in those using various antihypertensive agents were similar to changes in normal adults. [142] Patients with hypertension may also have abnormal vasomotor responses to tilt testing, demonstrating more instability in small studies. [143]

Even in normal subjects, passive tilting generates a high incidence of orthostatic syncope. [144] Patients with chronic anemia (and a compensated blood volume) seem to have the same postural response as normal subjects.[145] Most of the conditions that affect postural blood pressure regulation involve a pathologic condition that affects the sympathetic nervous system. Orthostatic hypotension caused by autonomic insufficiency is usually not accompanied by tachycardia, whereas the orthostatic hypotension produced by acute volume depletion is commonly accompanied by a pronounced reflex tachycardia.

As noted in Table 1-6 , many conditions, diseases, and medications have been implicated as causing abnormal orthostatic vital sign changes. Most variables have been poorly studied. In the elderly, orthostatic vital sign changes in *volume-depleted* patients have not been studied. In studies of *normovolemic* nursing home patients, orthostatic hypotension has largely been attributed to autonomic dysfunction and its prevalence has been stated at anywhere from 8% to 40%. [146] In a study of patients 65 years or older, 28% had a drop in systolic blood pressure of >20 mm Hg. The study results showed no increased incidence of orthostatic hypotension among patients with chronic cardiovascular disease, disability, body mass index, or medications, although many of these comorbidities would seem intuitive to increase the risk of orthostasis.[147] Normovolemia orthostatic hypotension in the elderly has been loosely linked to long-term cardiovascular mortality and the risk of subsequent cerebrovascular accidents, but these results have not been consistently observed. [148] Ethanol ingestion exaggerates postural pulse changes up to 8 hours following ingestion,[125] thus mimicking the hemodynamic changes seen with acute blood loss. However, in the setting of ethanol intoxication and trauma, one must be vigilant for associated occult hemorrhage and not reflexively assign tachycardia to being a result of intoxication alone.

The utility of orthostatic vital signs in children has been questioned. Horam and Roscelli found that healthy adolescents had heart rate changes of 21.5 ± 21.2 beats/min with orthostatic measurements made after 2 minutes of standing.[149] They found similar variation in the systolic blood pressure change (+19 to -17 mm Hg). Bergman and colleagues found that 25% of clinically normovolemic children had a postural increase in pulse of >20 beats/min and 11% had a postural fall in systolic blood pressure of >20 mm Hg.[150] However, children with fever and diarrhea were included in this "normal" study group. Another study comparing mildly dehydrated children with normal children found a significant difference in the orthostatic rise in pulse between the two groups. [151] Using near-syncope or a change in heart rate of >25 beats/min, orthostatic vital signs have a specificity of 95%, a sensitivity of 75%, and a predictive

TABLE 1-6 -- Classification of Disorders of Postural Blood Pressure Regulation

I. Poor postural adjustment
A. Tall, asthenic habitus
B. Advanced age
C. Physical exhaustion
D. Prolonged recumbency
E. Prolonged weightlessness
F. Pregnancy
G. Gastrectomy
II. Orthostatic hypotension
A. Secondary orthostatic hypotension
1. Endocrinologic-metabolic disorders
a. Diabetes mellitus
b. Dopamine-ß-hydroxylase deficiency
c. Primary amyloidosis
d. Primary and secondary adrenal insufficiency
e. Pheochromocytoma
f. Primary aldosteronism with marked hypokalemia
g. Porphyria
2. Central and peripheral nervous system disorders
a. Intracranial tumors (parasellar and posterior fossa)
b. Idiopathic paralysis agitans
c. Wernicke's encephalopathy
d. Multiple cerebral infarctions
e. Brainstem lesions
f. Tabes dorsalis
g. Syringomyelia

h. Traumatic and inflammatory myelopathies
i. Guillain-Barré syndrome
j. Chronic inflammatory polyradiculoneuropathy
k. Peripheral neuropathies
l. Familial dysautonomia (Riley-Day syndrome)
3. Miscellaneous disorders
a. Ciguatera fish poisoning
b. Electrolyte disturbance
c. Hypochromic anemia
d. Hypovolemia
e. Medications
i. Alcohol
ii. Antihypertensives with central effects (methyldopa, clonidine)
iii. Antihypertensives with peripheral effects (prazosin, hydralazine, and guanethidine)
iv. Calcium-channel blockers
v. Diuretics
vi. Insulin
vii. Levodopa
viii. Marijuana
ix. Narcotic agents
x. Nitrates
xi. Psychotropic agents (tricyclic antidepressants, phenothiazines, monoamine oxidase inhibitors, minor tranquilizers)
xii. Sympatholytics
xiii. Sympathomimetic agents (prolonged use)
xiv. Vasodilators
xv. Vincristine sulfate
f. Extensive surgical sympathectomy
g. Chronic hemodialysis
h. Anorexia nervosa
i. Hyperbradykininism
B. Primary or idiopathic orthostatic hypotension
1. Idiopathic orthostatic hypotension (Bradbury-Eggleston syndrome)
2. Idiopathic orthostatic hypotension with somatic neurologic deficit (Shy-Drager syndrome)

Adapted from Thomas JE, Schirger A, Fealey RD, et al: Orthostatic hypotension. Mayo Clin Proc 56:117, 1981. Reproduced by permission.

value of 92% in detecting mild clinical dehydration in children. [151] No difference in orthostatic blood pressure was found between normal and dehydrated children. Considering resting tachycardia as a positive sign of dehydration increased the predictive value of the test. [152] The investigators concluded that in the appropriate clinical setting, an orthostatic increase in pulse >25 beats/min constitutes a positive tilt test, and an orthostatic pulse increase of <20 beats/min constitutes a negative test for hypovolemia.[151]

Another complicating factor in interpreting orthostatic vital signs is the development of *paradoxical bradycardia* in the presence of blood loss. Bradycardia in the face of hemorrhage has generally been considered a preterminal finding of irreversible shock, but bradycardia has been documented in hypovolemic, yet conscious, trauma patients. Several early studies [121] [122] noted that when orthostatic syncope occurred, it was accompanied by hypotension and often bradycardia. Many central nervous system factors can contribute to *vagalmediated* syncope in ED patients with acute traumatic blood loss. These factors include pain, the sight of blood, stress, and nausea. Several investigators [153] [154] have described women with hemoperitoneum secondary to ruptured ectopic pregnancy who were hypotensive but did not have tachycardia. Jansen reviewed other cases of this relative bradycardia that occurred in hypotensive patients with acute intraperitoneal bleeding and postulated a parasympathetic mechanism triggered by the presence of free blood in the peritoneal cavity. [155] This bradycardia may be reversed with atropine, but *aggressive fluid replacement is the treatment of choice* because anecdotal reports mention serious ventricular arrhythmias from atropine used in this setting. [156] Paradoxical bradycardia has also been described in patients with abdominal or thoracic trauma or arterial bleeding from extremity wounds. [156] This paradoxical bradycardia may be more frequently associated with rapid and massive bleeding, whereas patients with a more gradual blood loss tend to have a more typical tachycardiac response. When the patient's clinical presentation is consistent with volume loss or shock, the clinician should not allow the absence of tachycardia to change the assessment.

Indications and Contraindications

When the volume status of a patient is assessed by use of orthostatic vital signs, several points should be remembered. Many factors influence orthostatic blood pressure including age, preexisting medical conditions, the use of medication, and autonomic dysfunction (see Table 1-6). Data relating the effect of *blood loss* to orthostatic vital signs are limited to phlebotomized healthy volunteers. Great care must be used when extrapolating these data to patients with anemia, dehydration, or painful trauma. The clinician must consider

the clinical condition of the patient as well as the orthostatic vital signs in evaluating a patient for volume depletion.

Orthostatic vital signs are indicated as part of the evaluation of any patient with known or suspected volume loss or a history of syncope, except in the presence of the following contraindications: The use of orthostatic vital signs is unnecessary and dangerous in a patient with supine hypotension or the clinical syndrome of shock. Orthostatic vital sign evaluation also is contraindicated in patients with a severely altered mental status, in the setting of possible spinal injuries, and in patients with lower extremity or pelvic fractures.

The use of medications that block the normal vasomotor and chronotropic response to orthostatic tests also represents a contraindication to use of this test for assessment of volume status. However, when the patient's volume status is believed to be adequate and the clinician seeks to determine if specific medications may have affected the patient's ability to respond to postural changes, the test may be useful. In the latter situation, the primary finding may be the feeling of near-syncope with little or no change in vital signs.

Orthostatic vital signs are often used to assess a patient's response to therapy. In patients receiving intravenous rehydration therapy, serial orthostatic vital signs are

widely used to judge the end point to therapy before release. Johnson and colleagues used this technique to demonstrate that the individual orthostatic vital signs response to saline infusion in women with hyperemesis gravidarum was associated with other measures of rehydration, including weight gain and decreased urine-specific gravity.[157] Although the individual improvement in orthostatic vital signs in response to rehydration was of clinical value, the presenting orthostatic vital signs were considered insufficient as the sole indicator of clinical dehydration in this population.

Technique

Once the decision to obtain orthostatic vital signs has been made, blood pressure and pulse are recorded after the patient has been in the supine position for 2 to 3 minutes (see Table 1-5). The patient should rest quietly. No painful or invasive procedures should be performed during the test. Anxiety, fever, and other causes of resting tachycardia may make the test uninterpretable.[150]

The patient is then asked to stand, and the examiner should be prepared to assist the patient if severe symptoms or syncope develop. A supine-to-standing test is more accurate than a supine-to-sitting evaluation. Knopp and coworkers found that the supine-to-sitting test was not reliable for detecting 1000 mL of blood loss (55% false-negative results).[124] If severe symptoms develop (defined as syncope or extreme dizziness requiring the patient to lie down) on standing, the test is considered positive and should be terminated. If the patient is not symptomatic, the blood pressure and pulse should be recorded after the patient has been standing for 1 minute. This interval resulted in the greatest difference between the control and 1000-mL phlebotomy groups in the study by Knopp and colleagues. [124]

Complications

The possible complications of orthostatic vital sign assessment can be avoided if the aforementioned contraindications and precautions are remembered. Complications include syncope with a resulting fall and injury and the possibility of exacerbating an existing fracture or spinal cord injury.

Interpretation

Criteria for positive orthostatic vital sign changes are either tachycardia greater than a specific threshold (see later) or symptoms of cerebral hypoperfusion (e.g., near-syncope).[152] Although blood pressure changes may be seen, they are too variable to be an indicator of blood volume loss. Although specific population-based thresholds for pulse rate and blood pressure changes have some value for identifying patients at high risk for significant volume loss, great individual variability limits the use of this technique as a screening test. That is, a volume loss of 500 mL (and occasionally more) may be associated with a negative orthostatic vital sign assessment (see later).[138] [158] However, the use of serial measurements to ascertain the response to therapy of patients considered at risk for volume loss appears to have clinical utility.[157]

In the setting of possible blood loss, if the patient has a pulse rise of 30 beats/min or severe symptoms and if other complicating factors have been excluded, then blood loss is highly likely (2% false-positive rate). [124] [138] When evaluating the patient with moderate blood loss, there appears to be no advantage to using the shock index as a marker of hypovolemia vs previously published tilt test criteria. [159] The presence of a negative test indicates only that an acute blood loss of 1000 mL is unlikely (2% false-negative rate); a blood loss of 500 mL cannot be excluded (43% to 87% false-negative rate). [124] [138] [160]

In children, postural near-syncope or an orthostatic pulse increase of 25 beats/min may be a predictor of mild dehydration. The accuracy of these criteria is increased by the addition of resting tachycardia.[151] However, one cannot quantify the amount of volume depletion with this test in children.

Criteria for significant orthostatic blood pressure changes cannot be definitively set for the following reasons: (1) in the study by Knopp and coworkers, a lack of correlation between blood pressure in the phlebotomy and control groups was seen [124] ; (2) a large variability in postural blood pressures has been found in the adult ED population[161] [162] ; (3) results of studies using passive tilt tables cannot be extrapolated to the bedside use of orthostatic vital signs; (4) studies using healthy patients with acute blood loss may not reflect orthostatic changes that are seen in the elderly or those with chronic bleeding, dehydration, and various other medical problems; and (5) many studies of orthostatic changes never used a criterion standard (measurement of actual volume loss) in their determinations. Because of lack of agreement about the degree of postural blood pressure change that constitutes a positive test result, the most reasonable definition may be any postural fall in blood pressure that results in symptoms of cerebral hypoperfusion. [163]

CAPILLARY REFILL

The capillary refill test is a measurement of the time interval from the release of nailbed or soft-tissue pressure (sufficient to blanch the nailbed or superficial soft tissue) until the return to normal coloration. Delayed capillary refill is an indication of reduced skin turgor, often as a result of volume depletion. Measurement of the capillary refill time interval

appears to be somewhat accurate in children, but its accuracy in assessing dehydration and reduced perfusion in adults is highly suspect. [123] [164]

Physiology

Skin elasticity is the characteristic that allows skin to spring back to its original shape after it has been deformed. One sign of decreased elasticity is skin tenting. The normal fullness of blood vessels or capillaries is referred to as *turgor vitalis*. The presence of normal skin turgor is a sign of adequate circulatory perfusion, because the speed of refilling the capillary bed after compression is responsible for the return of color to the skin. [36]

Indications and Contraindications

There are no specific contraindications to performing capillary refill measurements in ED patients. However, the capillary refill time interval should not be obtained in a dependent extremity, a recently burned or injured extremity, or at the site of an infection or acute injury. Frequent monitoring of capillary refill is useful in accessing responses to rapid fluid resuscitation in children. Because capillary refill is available without additional equipment and takes only a few seconds to perform, it is a useful bedside assessment of perfusion and dehydration.

Procedure

The preferred sites for performing capillary refill are the nailbed, the thenar surface of the palm, and the heel. The current standards are best developed for capillary refill obtained at the nailbed. [164] Regardless of the site chosen, the extremities should be positioned at about the level of the right atrium. The minimum pressure necessary to produce blanching yields the most reproducible values. Timing can be performed with a stop watch or simply by counting out "one-thousand-one, one-thousand-two" for approximation of the interval. The relative apparent simplicity of the test notwithstanding, significant interobserver reliability has been noted in obtaining measurements.[165] A repeated measurement should be obtained at the same location as the initial test, because alternate sites may have different capillary refill times.

Interpretation

The normal capillary refill interval increases with age, degree of dehydration, and degree of hypoperfusion. Hypothermia, hyponatremia, congestive heart failure, malnutrition, and edema all increase the capillary refill interval. Environmental conditions, which can falsely alter capillary refill, are the ambient temperature [165] and the quality of ambient lighting. [166] Fever does not appear to prolong or shorten capillary refill time. [167]

The main difficulty in interpreting the capillary refill interval is that normal values in healthy patients fall into a wide range. In 30 normal infants from 2 to 24 months of age, the mean capillary refill interval was 0.8 ± 0.3 seconds. Measurements obtained from the nailbed were more reproducible than those from the heel. Dehydration was strongly correlated with a capillary refill time of >3 seconds and suggested a fluid deficit of >100 mL/kg. [164] The presence of delayed capillary refill >2 seconds when combined with any two or more of absent tears, dry mucous membranes, or ill general appearance can predict clinical dehydration (>5% deficit of body weight) in children (age 1 month to 5 years) with a 87% sensitivity and 82% specificity. [167]

The role of serial capillary refill interval measurements for assessing the response to rehydration in adults is unknown. However, the test does not appear to be useful for assessing acute blood volume loss. In adults, the capillary refill interval was found to be less sensitive and less specific than orthostatic vital signs for detecting a 450-mL blood loss during blood donation. [123]

TEMPERATURE

Accurate measurement of body temperature is an essential part of clinical medicine. When taken in the context of other vital signs, abnormalities of core body temperature are excellent guides to the severity of illness.

Detection of abnormal body temperature facilitates proper diagnosis and evaluation of presenting complaints. [168] [169] [170] [171] [172] [173] [174] [175] [176] The inability to maintain normal body temperature is indicative of a vast number of potentially serious disorders, including infections, neoplasms, shock, toxic reactions, and environmental exposures.[168] [171] Fever in neutropenic, immunocompromised, and intravenous drug-abusing patients may be more reliable than laboratory tests or physician assessment in diagnosing serious illness. [171] Infants are particularly sensitive to thermal stress and may demonstrate lower body temperatures during asphyxia or necrotizing enterocolitis.[172] [173] [174] Normalization of body temperature following intervention may have important prognostic and therapeutic implications. [171]

Physiology

Under normal conditions, the temperature of deep central body tissues (i.e., core temperature) remains at 37 ± 0.6°C (98.6 ± 1.08°F). [175] [176] Core body temperature can be maintained within a narrow range while environmental temperature varies from as much as 13 to 60°C (55 to 140°F), [177] whereas surface temperature rises and falls with environmental and other influences. Maintenance of normal body temperature requires a balance of heat production and heat loss. Heat loss occurs by radiation, conduction, and evaporation. Approximately 60%, 18%, and 22% of heat loss, respectively, occurs by these methods. Heat loss is increased by wind, water, and lack of insulation (e.g., clothing). Sweating, vasodilation, and decreased heat production serve to decrease temperature, whereas piloerection, vasoconstriction, and increased heat production serve to increase body temperature. Heat production is increased by shivering, fat catabolism, and increased thyroid hormone production.

Temperature control occurs by feedback mechanisms operating through the preoptic area of the hypothalamus. Heat-sensitive neurons in this area increase their rate of firing during experimental heating. Receptors in the skin, spinal cord, abdominal viscera, and central veins primarily detect cold and provide feedback to the hypothalamus, which signals an increase in heat production. Stimuli that change the core body temperature result in reflex changes in mechanisms that increase either heat loss or production. [177]

Indications and Contraindications

Clinicians generally measure body temperature to determine if it is outside the normal range and as an indication of pathologic conditions that can affect core body temperature. Because actual core body temperature measurement requires the placement of invasive monitors, such as an esophageal or a pulmonary artery probe, clinicians commonly use estimates of core body temperature, which conveniently and safely assess abnormalities of core temperature. Unfortunately, all noncore body sites and methods have inherent accuracy limitations, which clinicians have come to accept in assessing most patients.

Oral temperature measurement requires a cooperative adult or child, generally older than 5 years. Patients who are grossly uncooperative, hemodynamically unstable, septic, or in respiratory distress (with a RR >20) require another method of temperature measurement. [178] This group includes children younger than 5 years and patients who are intubated.

Special techniques of measuring actual core body temperature may be indicated in certain patients (e.g, those with profound hypothermia, frostbite, or hyperthermia). Measurement of core body temperature is indicated in these individuals because it accurately measures treatment effects. This is the group of patients who will also benefit most from continuous temperature measurements.[179]

Measurement Sites

Core Body Temperature

It has been demonstrated that the following sites accurately reflect core body temperature and its changes: esophageal (in the distal third of the esophagus), the tympanic membrane (using *direct thermistor contact* at the anterior inferior quadrant of the tympanic membrane),[180] [181] and the pulmonary artery temperature.[182]

Other sites may represent core body temperature under certain conditions. For example (1) the rectum, when the temperature is obtained at least 8 cm from the anus using an indwelling thermistor and the body temperature is relatively constant, and (2) the bladder when measured with an indwelling thermistor. [128] [183] Data on core temperature in pediatric patients is limited and it is unclear if bladder, rectal, or oral temperature is a good measurement of core body temperature in children. [184]

Peripheral Body Sites Approximating Core Body Temperature

Oral temperature measurement with a digital electronic probe is commonly used for ambulatory patients. [185] Advantages include convenience, timing, safety, and availability. Disadvantages include various factors that affect clinical accuracy and sensitivity. Electronic oral temperature probes must be covered with disposable covers, although these have been shown not to be completely effective in preventing probe contamination with microorganisms. [186] Although there are no absolute contraindications for oral temperature assessment, patients with factors shown to produce unreliable results (see later) require temperature measurement at other sites.[178] [187] [188]

Rectal temperature is often considered the criterion standard of body temperature for ambulatory patients and is often routine for children younger than 3 years. [189] Advantages include accuracy, sensitivity, and availability. One intensive care unit study found rectal probe temperatures to demonstrate limited variability or bias when compared with pulmonary artery temperatures.[190] Disadvantages include longer intervals for measurement, safety concerns, and inconvenience. Neutropenia and recent rectal surgery represent relative contraindications to rectal temperature measurement. [191] Placement of a rectal probe thermometer may produce autonomic changes in patients with acute myocardial infarction. [192]

Body temperature measured as a function of infrared radiation (IR) detected from the ear, including the auditory canal and tympanic membrane (TM), is easy to use, hygienic, convenient, and quick. [193] The noncontact IR ear thermometer has been studied under a variety of clinical conditions. [194] Concerns have been raised over the accuracy of these thermometers in screening for fever in children younger than 3 years of age. [195] [196] Romano and colleagues found the Thermoscan Pro-1 IR thermometer to perform similar to rectal probe temperature, but the FirstTemp IR thermometer displayed considerably more variability. [190]

Intensive care studies have compared infrared thermometry to pulmonary artery core temperatures and found a sensitivity of detecting fever to be 58% and a specificity of 94%. Double ear thermometry (measuring values at both right and left TMs and calculating the mean) increased the sensitivity and specificity to 61% and 95%.[197] Increased variability of tympanic temperatures vs oral temperatures was also found in the critical care population. [198]

In the ED setting, infrared tymponometers have undergone limited scrutiny. In 100 children, TM, rectal, and axillary temperatures were obtained with statistically significant differences noted. [199] In a similar ED-based study, 95 geriatric (older than 60 years of age) patients had oral, TM, and rectal temperatures measured with fevers missed with both oral and tympanic readings, as compared to rectal readings. [200] In another study of 100 adult ED patients, the TM and rectal temperatures showed generally good agreement, but the TM temperature missed 4 of 10 patients with a fever (>38.5°C). [201] The authors also noted that the temperature difference between TM and rectal temperature readings was greater in the presence of cerumen impaction. Special patient populations deserve separate attention. First, when a neonatal population is examined, significant variability is noted with TM temperatures, making rectal thermometers the standard. [202] Second, a theoretical

disadvantage of TM temperatures might be a falsely elevated estimate of the core temperature in the presence of otitis media. However, in one study tympanic thermometers accurately reflected oral temperatures in children with otitis media. [203]

Although not a likely ED concern, prehospital providers who might wish to measure IR tympanic temperature at low ambient temperatures should be aware that below 24.6°C the TM readings will greatly underestimate core temperature. [204] EMS personnel should also be aware that in a cohort of exhausted marathon runners, rectal and IR tympanic temperatures have only moderate correlation.[205] Hence when hyperthermia or hypothermia is clinically suspected and the IR tympanic temperature does not confirm an abnormal temperature, a rectal temperature should be considered.

Axillary and tactile temperature assessment have been demonstrated to be unreliable and insensitive. They should not be used as screening methods for core temperature abnormalities in the ED.[206] [207] [208] Similarly, the use of liquid crystal chemophototropic strips applied to the skin of the forehead

are not accurate for single measurement or fever screening. [209] [210] Single-use Tempa-DOT thermometers, which show increasing temperature dot darkening with increasing temperature, have been adopted by many EDs. The sensitivity of these thermometers for fever identification remains to be determined. The most rudimentary method for temperature measurement, parental assessment by tactile touch, is associated with a measured fever approximately 75% of the time. [211] Clinician estimation of fever is almost identical (70%). [212]

Equipment

Mercury-in-glass thermometers remain popular despite requiring longer equilibration times and having cumbersome cleansing requirements. Electronic methods of temperature measurement are based on the thermocouple principle. Modern electronic thermometers signal once extrapolation of the temperature-time curve occurs. [212A] Current in vitro standards call for an accuracy of ±0.1°C (±0.18°F) over the range of 37 to 39°C (98.6 to 102.2°F). [213] Thermistor probes (i.e., small thermocouples with instantaneous readouts) for esophageal and vascular temperature measurement provide continuous temperature readouts when attached to a potentiometer. [9] Thermistor products are available for esophageal, bladder, and rectal probes (Mallinckrodt Medical, Inc, St. Louis; or Yellow Springs Instruments, Yellow Springs, OH) with appropriate readout monitors.

Noncontact IR ear thermometers were introduced in 1985. These thermometers were initially used only in hospitals, but they are now sold over the counter for home use. The IR ear thermometers work by incorporating an IR sensor in the field of view of the IR emissions from the ear. Ear IR thermometers generally detect naturally occurring IR emissions over a brief time period, generally <1 sec. The emissions are converted to an electrical analog signal, which is digitized and analyzed by a microprocessor with resultant digital display. [214] The primary determinant of radiation emission is the temperature of blood perfusing this area, and the warmest spot in the auditory canal is the TM. Thus, current IR ear thermometers are operator-dependent in the same fashion that otoscopy requires proper positioning to enable the examiner to "see" the TM.[215]

Various IR ear thermometers are available commercially (Table 1-7) with varying operating temperature ranges, features, and reported accuracy. Clinical data assessing relative performance of the many commercial devices are limited. In one study comparing many of these IR thermometers, the Pro-1 and FirstTemp gave significantly higher readings. [215]

Procedure

Temperature measurement begins with body site selection. Consideration should be given to the accuracy of using a site to reflect core temperature, the site sensitivity to temperature changes, convenience, time required, safety, and site availability. [191] [216]

When reusable probes are used, the thermometer end should be covered with a probe cover. Temperature measurement continues with insertion of the temperature probe and probe equilibration with the temperature of local body tissues. Proper placement of temperature probes significantly influences results for oral, rectal, esophageal, and vascular temperatures.[175] [217] [218]

Sublingual oral temperatures should be obtained in either right or left posterior sublingual pocket, with the mouth closed. [217] The patient should be sitting upright or lying, holding the base of the probe with one hand.[185] Rectal temperatures require removal of clothing, lubrication of the probe, and gloving of personnel. They should be obtained with the patient relaxed in the left or right lateral decubitus position. Gentle insertion to 3 to 5 cm should ensure accurate, atraumatic results. [175]

Axillary temperatures are frequently used for neonatal patients in incubators because of convenience. The technique is not indicated unless other sites are unavailable.[207] [219]

Although not commonly used, temperature measurement in a freshly voided urine specimen can validate temperature measurement at other body sites. A nomogram of expected urinary temperatures has been derived from measurements of oral and urinary temperatures in 55 subjects (Fig. 1-10).[220]

Placement of an esophageal catheter for measurement of core body temperature is similar to placement of a nasogastric or orogastric tube (see Chapter 41). In this case, the distal tip of the esophageal catheter contains a thermistor to measure body temperature. In normal adults, the catheter is inserted approximately 34 cm deep into the esophagus, with the objective of locating the probe tip at the level at which the esophagus is between the aorta and the left atrium. The catheter is connected to a potentiometer and allowed to equilibrate briefly before a body temperature reading is taken.

For pulmonary artery (PA) catheter assessment of core body temperature, a PA or thermistor-tipped catheter is placed into the PA. A detailed discussion of the placement of PA catheters is beyond the scope of this chapter. Once a PA catheter is in the correct position, the temperature value may be obtained by using the potentiometer attached to the distal thermistor. Because of the risks associated with catheter placement, PA temperatures are generally reserved for patients with another clinical indication for PA pressure monitoring.

Complications

Complications associated with axillary, oral, ear IR, and liquid crystal thermometers are rare or unreported. Tympanic membrane perforation and pain have been reported as complications of thermistor probe placement in the auditory canal. Complications associated with rectal temperature measurement are extremely rare, but include rectal perforation, pneumoperitoneum, bacteremia, dysrhythmias, and syncope. [191] [216] Falsely low but supranormal rectal temperature measurements may be seen during shock. [221] Rectal temperatures also may lag behind core temperature changes.

Complications associated with esophageal temperature measurement include those associated with placement of an orogastric or nasogastric tube (see Chapter 41). Confirmation of accurate placement can be verified with a chest radiogram. In addition, spurious temperatures may arise from an improperly calibrated potentiometer, a damaged thermistor, or a proximal esophageal location. Mechanical ventilation or thoracotomy can falsely lower measured esophageal core temperatures.

Interpretation

Normal values for body temperature are affected by the following variables: (1) site and methods used for measurement, (2) perfusion, (3) environmental exposure, (4) pregnancy,

TABLE 1-7 -- Commercial Infrared Ear Thermometers*

Brand Name	Manufacturer[§]	Temperature Range (°C)[¶]	Read Modes	Response Time (sec)	Continuous Mode	Charge Use Time	Warranty	Anti-Theft	Offsets (°C)[£]		
Genius	IMS	16–34	O.R.C.Cal.S	2	Yes	10,000 uses	3 yr	No	O=0.80	R=1.10	C=0.74
Diateck-9000	Diateck	24–42	O.R.T.S	3	No	20,000 uses	1 yr	Yes	O=3.30	C=1.20	R=1.15
LighTouch[†]	Exergen	16–54	T	1	Yes	4000 uses	Lifetime	No	None	R=1.20	—
Pro-1	Thermoscan	26–42	O.R.C.T	1	No	1500 uses	1 yr	Yes	O=0.30	—	R=1.15
HM-1[‡]	Thermoscan	34–42	O.R	1	No	1500 uses	1 yr	No	O=0.30	—	—
Pro-LT	Thermoscan	34–42	O.R.T	1	No	10,000 uses	1 yr	No	O=0.30	—	—
CoreChec	IVAC	25–43	C	2	No	8000 uses	3 yr	Yes	—	R=1.15	C=1.83

O, Oral; R, rectal; C, core; Cal, calibration; T, tympanic; S, surface.

**Adapted from Lewinter JR, Terndrup TE, Williams TM, Knopp RK. Vital Sign Measurement. In Roberts JR and Hedges JR. Clinical Procedures in Emergency Medicine. 3rd Ed. 1998, WB Saunders, Philadelphia, PA. p. 1186.*

§IMS, Intelligent Medical Systems, San Diego, CA; Diateck, Diateck Corp, San Diego, CA; Exergen, Exergen Corp, Newton, MA; Thermoscan, Thermoscan Inc, San Diego, CA; IVAC, IVAC Corp, San Diego, CA.

¶Thermometers listed meet laboratory standards of the American Society for Testing and Materials (±0.1°C).

£ Temperature value, which is added to unadjusted infrared ear thermometer reading to estimate body temperature at specific sites.

†A "pediatric" infrared ear thermometer (Pedi-Q) is also available from this manufacturer.

‡Not intended for professional use.

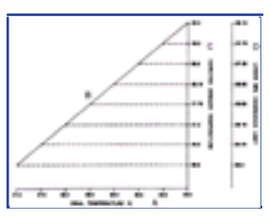

Figure 1-10 Relationship between oral and urinary temperatures constructed as a nomogram. Urinary temperatures are consistently within 1 to 1.5°C of simultaneously obtained oral temperatures and within 2°C of rectal temperatures. To use the nomogram, locate the oral temperature on the horizontal axis (*A*) and draw a perpendicular line to intersect the diagonal border of the graph (*B*). Follow the dotted horizontal line to the right to determine the expected urinary temperature on the longitudinal axis (*C*). The far right-hand scale (*D*) provides a 99% confidence level for the lower range of expected urinary temperatures and finding a value below the 99% confidence level is essentially diagnostic of a factitious fever. *(From Murray HW, Tuazon CU, Guerrero IC, et al: Urinary temperature: A clue to early diagnosis of factitious fever. N Engl J Med 296:23, 1977. Reproduced by permission of the New England Journal of Medicine.)*

(5) activity level, and (6) time of day. Clinicians must interpret body temperature with knowledge of the range of normal values at the intended site of measurement. Although the core body temperature remains nearly constant (37.0 ± 0.6°C or 98.6 ± 0.18°F), the surface temperature rises and falls with changes in ambient temperature, exercise, and time of day. The definition of fever varies by the site of measurement and is defined by a temperature >2 standard deviation (SD) above the mean. Fever has been defined as an oral temperature =37.8°C (=100.0°F),[222] a rectal temperature =38.0°C (=100.4°F),[223] or an IR ear temperature =37.6°C (=99.6°F).[224] Based on the measurement of temperatures in normal, healthy infants, Herzog and Coyne recommend that fever should be defined as rectal temperature =38°C in infants younger than 30 days old; =38.1°C in infants 30 to 60 days (1 month); and =38.2°C in infants 60 to 90 days old (2 months).[225] Hypothermia has been defined as a core body temperature <35°C (<95°F), whereas hyperthermia has been defined as a core body temperature >41°C (>105.8°F), with accompanying symptoms and signs.[226] A useful nomogram and formulae for conversion of °C to °F are provided in Figure 1-11 .

Temperature probes that require the transfer of heat energy from local tissues to the temperature probe require a period of equilibration and reliable tissue contact at the intended body site. Acceptable equilibration times for mercury-in-glass thermometers for oral, rectal, and axillary sites are 7, 3, and 10 minutes, respectively. Used in a predictive mode, electronic digital thermometers generally require 30 seconds for oral or rectal temperature equilibration. The predictive mode uses temperature changes vs time to predict an equilibration temperature.

Normal ranges and suggested febrile thresholds for common body sites and methods should be considered in the interpretation of temperature values (Table 1-8). The interpretation of temperature measurements during clinical assessment must consider the use of antipyretics, level of activity, pregnancy, environmental exposure, and patient age. The duration of antipyresis with acetaminophen or aspirin is 3.5 to 4 hours. When both drugs are given together, the duration of action may be extended up to 6 hours.[227] Body temperature is increased during sustained exercise, during pregnancy and during the luteal phase of the menstrual cycle. Temperature also increases in later afternoon during diurnal variation. Body temperature is generally reduced with advanced age.

The interpretation of temperatures obtained with pulmonary artery or esophageal thermistors is generally straightforward. Comparison of measured values to the expected normal range should be performed to determine if the patient has an abnormal core body temperature. In addition to improper placement of the thermistor, sources of temperature error include an improperly calibrated potentiometer or thermistor, damaged thermistor, or improper placement. The biomedical staff at the institution should periodically verify readout and calibration of these instruments.

Figure 1-11 Temperature conversion scale. To change Celsius (centigrade) to Fahrenheit, multiply the Celsius temperature by 9/5 and add 32. To change Fahrenheit to Celsius, subtract 32 from the Fahrenheit number and multiply by 5/9.

TABLE 1-8 -- Normal Ranges and Suggested Febrile Thresholds for Human Body Temperature (in Healthy Resting Patient)

Body Site	Type of Thermometer	Normal Range (°C)	Fever (°C)
Core[*]	Electronic	36.4–37.9	38.0
Oral	Mercury-glass, electronic	35.5–37.7	37.8
Rectal	Mercury-glass, electronic	36.6–37.9	38.0
Ear	Infrared emission	35.7–37.5	37.6[†]

*Temperature obtained with a properly positioned pulmonary artery, esophageal, or tympanic membrane thermistor.

†For unadjusted ear temperature using Thermoscan Pro-1 (Thermoscan, Inc, San Diego, CA).

Oral temperature measurements are affected by ingestion of hot or cold liquids, [191] tachypnea,[228] and cold ambient air.[229] Smoking appears to result in little change in oral temperatures.[188] [191] Therefore, before taking an oral temperature, the examiner should inquire about these features and possibly delay taking the temperature. Also, Erickson found a 2.7°C (4.9°F) reduction of oral temperature measurement when the probe was placed under the tip of the tongue instead of under the posterior sublingual pocket. [187] When using a mercury-glass thermometer, optimum placement time was found to be 7 minutes for oral temperatures in children. [230] Given the extrapolation that occurs with rapid reading thermocouple devices and IR detectors, it is not surprising that the sensitivity of these devices (whether oral or tympanic) for fever (as detected by oral mercury thermometers) is only 86% to 88%. [231] Hence, many practitioners have adopted the adage that when a temperature is suspected or crucial in decision making, but not evident with an oral thermocouple probe or IR tympanic thermometer, measurement with a glass, mercury thermometer is indicated.

Axillary temperatures obtained in 108 children by Kresch had a sensitivity of only 33% and a specificity of 98% for fever. [207] Ogren obtained similar results using an electronic thermometer in the axilla.[232] Hence, axillary temperatures should not be used to screen for fever.

When rapid changes in body temperature occur, oral and tympanic temperature measurements appear to be more reliable than rectal temperature. In 20 adults examined during open-heart surgery, oral temperatures showed a better correlation with blood temperature during rapid cooling and rewarming. [218] In 12 adults, the average rectal temperature lag during warming was 5.3 minutes during water immersion and 3.8 minutes during exercise. [233] Sublingual (oral) temperature delay (1.3 and 0.9 minutes for the 2 experiments, respectively) was less than auditory canal delay (1.1 and 4.1 minutes, respectively) in temperature response.

Infrequently, ED patients require constant monitoring of temperature (e.g., in cases of hypothermia or hyperthermia). This can usually be performed using a bladder or esophageal probe attached to a potentiometer. Patients with indwelling central venous or pulmonary arterial catheters may have electronic thermistors inserted into the central circulation to measure core body temperature. As noted earlier, rectal temperature measurements are less desirable for monitoring patients undergoing rapid core temperature changes. Periodic IR tympanic temperature monitoring may represent one useful option in the hypothermic patient. [234]

The interpretation of ear IR temperatures requires a knowledge of the mode of thermometer operation and ambient temperature. Cerumen occlusion of the ear canal may produce a false low reading.[235] Most IR ear thermometers have different modes that allow users to predict the equivalent temperature at other body sites. IR ear thermometers appear moderately sensitive for fever.[195] [196] If these devices are used, the clinician must be aware of the potential for a false low temperature. When in doubt, the measurement should be repeated with a more standard method.

CONCLUSION

Vital signs must always be interpreted in relationship to each other to obtain a more complete clinical picture. All vital signs are subject to errors in measurement and therefore must be verified when the initial result does not match the clinical presentation. Abnormal vital signs may lead the clinician to a diagnosis, and abnormalities should be explained within the context of the patient's illness.

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Chapter 2 - Use of Monitoring Devices for Assessing Ventilation and Oxygenation

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Ensuring delivery of oxygen (O_2) to the cell is the primary critical action in emergency medicine. Without O_2 to fuel cellular energy production, the cells ultimately falter and the organism dies. Except during cardiopulmonary bypass, tissue perfusion depends on an adequate inspired O_2 content, ventilatory effort, alveolar gas exchange, blood O_2 carrying capacity, and cardiac output. This chapter covers emergency department (ED) assessment of spontaneous ventilation and O_2 delivery and noninvasive means of monitoring and improving inspired O_2 concentration and spontaneous ventilation.

BACKGROUND

Respiratory illness has been poorly understood until recent times. The Talmud, the ancient law book of the Israelites, blames the etiology of asthma-like illness on a malignant spirit. Later, Celsus (AD 25), an encyclopedist of the late Roman period, noted a favorable prognosis for a respiratory illness if the "expectoration is white as if mucus from the nose, but unfavorable if sputum is purulent, and accompanied by fever," descriptions that are consistent with chronic bronchitis and pneumonia. Celsus recommended bleeding, purgatives, emetics, and diuretics; this therapy was less preferable perhaps to his prescription for phthisis (tuberculosis), for which he recommended a leisurely sojourn down the Nile and drinking tea and honey. [1]

The Greek word asthetaμa signified panting and was applied generally to difficult breathing and respiratory illness; the term eventually gave rise to the word *asthma*. The earliest comprehensive distinction between asthma and other respiratory diseases came from Aretaeus of Cappadocia, who first recognized and recorded the chronic recurrent nature of the disease. [2]

TABLE 2-1 -- Causes of Respiratory Failure*

Neurologic	Muscles and Chest Wall	Oropharynx	Lower Airway	Lung Parenchyma	Heart
Drug overdose	Myopathy	Foreign body	Tracheobronchitis	Adult respiratory distress syndrome	Pulmonary edema
Stroke	Myasthenia gravis	Laryngospasm	Tracheal stenosis		
Central hypoventilation	Kyphoscoliosis	Tonsillar hypertrophy	Bronchospasm	Emphysema	Mitral stenosis
Guillain-Barré syndrome	Flail chest			Pneumonia	
Head trauma				Interstitial pneumonitis	
Poliomyelitis					
Botulism					

*Disease at any level of the respiratory system, central or peripheral nervous system, bellows mechanism, or heart may cause respiratory failure.

In 1698, Sir John Floyer wrote the first book devoted entirely to asthma and recorded the first description of pulsus paradoxus. Atropine therapy began in England in 1802, and in 1830 John Eberle deduced that "it is highly probable, therefore, that asthma consists essentially in a peculiar irritation of the pneumogastric nerves (vagus), in consequence of which the smaller bronchial tubes and air-cells are thrown into a state of spasmodic constriction." [3]

The American Thoracic Society statement on asthma in 1962 is an often-quoted definition of the disease: "asthma is a disease characterized by an increased responsiveness of the trachea and bronchi to various stimuli and manifested by a widespread narrowing of the airways that changes in severity either spontaneously or as a result of therapy." The term *asthma* is not appropriate for bronchial narrowing, which results solely from widespread bronchial infection; from destructive diseases of the lung, as in pulmonary emphysema; or from cardiovascular disorders.

Asthma is the most common chronic disease of childhood and among the most frequent complaint of adults, resulting in 2 million outpatient visits per year; it is also the most common cause of absence from school and work.[4] Asthma, depending on one's definition, affects between 7 and 20 million people in the United States and is especially prevalent among those living below the poverty level. [5] The National Center for Health Statistics estimates that asthma affects 9.7 million people in the U.S. population and chronic obstructive pulmonary diseases (COPDs) afflict up to 14 million adults, with similar economic consequences. [6] Although many investigators and studies distinguish between asthma (pure reactive airway disease) and COPDs, which include chronic bronchitis (airway inflammation with increased mucus secretion) and emphysema (airway destruction and loss of airway elasticity), clinically the distinction is blurred by the similarities in ED management. In fact, the current literature cites studies that can document little to distinguish the response of either entity to bronchodilators. [7] These diseases must, however, be separated from other causes of dyspnea and respiratory distress (Table 2-1), many of which are associated with wheezing.

PULMONARY FUNCTION TESTING

Airway maintenance and breathing are given primacy in the ABCs of emergency medicine. Clinical assessment always begins with the patient's ventilatory function. The clinician notes quickly the patient's mental status, level of distress, skin color, character of effort, use of accessory muscles, presence of diaphoresis, lung sounds, and vital signs. In conjunction with this clinical overview, a brief clinical history provides

the clinician with sufficient information on which to initiate therapy.

Unfortunately, the clinician's initial clinical impression of the patient's ventilatory status is based on imprecise subjective findings that may not detect serious illness in all patients. The ability of experienced clinicians to detect compromised pulmonary function when compared with pulmonary function testing seems only moderately better than chance alone.[8] In the study by Godfrey and coworkers, the sensitivity of clinical impression did not improve when the clinicians underwent training on the common and more subtle signs of respiratory distress.[8] Patients also appear superior to clinicians in predicting their own pulmonary function [9] and in assessing day-to-day variation in disease, using pulmonary function testing as the standard. [10]

Regardless of the initial clinical presentation, results of treatment in a subjectively asymptomatic patient with reactive airway disease will reach, at best, only 40% to 50% of predicted normal pulmonary function and 60% to 70% when all abnormal physical signs have resolved. [11] This potentially undetected degree of dysfunction may contribute to recrudescence. Objective measures of pulmonary dysfunction serve both to quantify results of therapy and as possible predictors of admission.

Spirometry has been used for decades by pulmonary specialists to assess airway limitation. The spirometric measurements were originally validated based on comparisons with clinical and body plethysmographic data. The terminology of pulmonary function testing was derived from the various measured subsegments of spirometry (Fig. 2-1). More recently, inexpensive handheld electronic meters have replaced formal spirometry in many clinical settings. These devices accurately measure or calculate the peak expiratory flow rate (PEFR), forced expiratory volume in 1 second (FEV_1), forced vital capacity (FVC), and percent FEV_1/FVC.[12] The Wright peak flow meter (Fig. 2-2) was originally designed by Wright and McKerrow for use in their pneumoconiosis unit in

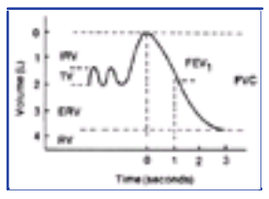

Figure 2-1 Time-forced vital capacity (FVC) is the volume of gas forcibly expelled following a maximal inspiration. Forced expiratory volume in 1 second (FEV_1) is the volume of gas expelled during the first second of the forced expiration. The other lung volumes obtainable are the tidal volume (TV), which is the volume of gas moved during quiet respiration; the inspiratory reserve volume (IRV), which is the volume of gas that can be inspired in addition to the tidal gas volume; and the expiratory reserve volume (ERV), which is the volume of gas that can be forcibly expired at the end of a tidal expiration. Some gas cannot be expired and remains in the chest. This is known as the residual volume (RV).

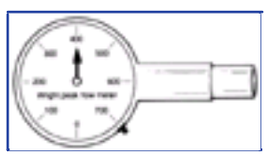

Figure 2-2 Wright peak flow meter. After resetting the dial to zero, the patient inhales fully and exhales forcefully through the disposable paper mouthpiece. The best of three attempts is recorded. A tight seal of the lips around the mouthpiece is required.

1959. Subsequently, development of compact, less complex, and less expensive calibrated spring-mechanism peak flow meters have allowed for widespread use in acute care and home settings (Fig. 2-3). These peak flow meters also have been successfully used as an adjunct in the assessment of pediatric patients. In the acute clinical situation, PEFR meter readings correlate well with formal spirometry, offer simplicity, and reduce the need for patient cooperation. [13]

Indications and Contraindications

In the ED, acute respiratory diseases such as asthma and chronic lung diseases make up the bulk of the situations requiring the objective assessment of ventilatory status. ED pulmonary function testing with either a peak flow meter or spirometer provides objective data on pulmonary status and patient response to therapy. These tests may assist the clinician in determining patient disposition [14] and may facilitate patient transfer at the time of admission by providing an objective and reproducible measure of the patient's failure to improve. Studies have found excellent correlations between PEFR and FEV_1, [13] [15] as well as between Wright and mini-Wright meters.[16] One investigation has evaluated PEFR for aiding differentiation of congestive heart failure from chronic lung disease in the patient with moderate to severe dyspnea.[17] Although opportunities will be infrequent, clinicians evaluating neuromuscular diseases affecting ventilatory function, such as the Guillain-Barré syndrome, may find these techniques useful in both initial and ongoing assessment. Near or true respiratory arrest may be the only true contraindication to obtaining these measurements because of limited patient cooperation and delay of immediate therapy.

Figure 2-3 Mini-Wright peak flow meter. The indicator arrow is moved back to zero. The patient inhales fully and then exhales forcefully through the disposable paper mouthpiece. The best of three attempts is recorded. Patient cooperation and a tight seal of the lips around the mouthpiece are required.

Equipment

A *spirometer* is a tube connected to a bellows-type device that communicates with a recording device. The subject breathes in and out through an orifice, causing expansion and contraction of the bellows, which in turn activates the recorder that traces a curve corresponding to the lung volume. This traditional volume method is complex and cumbersome. Handheld electronic spirometers now available use sensing devices either to translate the pressure of exhalation (e.g., Respiradyne, Kendall Healthcare Products Company, Ocala, FL, Fig. 2-4) or to detect the number of rotations of a small turbine (e.g., Pocket Spirometer, Micro Medical Instruments, Rochester, NY) by an optical system. Both systems are self-calibrating, take little practice to use, and can calculate PEFR, FEV_1, and FVC. Some systems, such as the transducer-based Respiradyne, also give additional calculated information such as percent FEV_1/FVC and forced mid-expiratory flow rate ($FEF_{25-75\%}$). Results are displayed digitally and maintained in memory until cleared.

Peak flow meters are simple mechanical devices that use the force of exhalation to rotate or push a membrane-coupled measuring arm or spring-loaded piston to statically record a position of maximum flow. Several brands of peak flow meters are commercially available including the Pulmo-Graph (De Vilbiss Healthcare Inc., Somerset, PA), Assess (HealthScan Products Inc., Cedar Grove, NJ), Wright Pocket (Ferraris Medical International, Holland, NY), and the Mini-Wright (Clement Clark International, Columbus, OH). Low-flow units designed for children are also available.

Procedure

The operation of peak flow meters and electronic spirometers is similar in many ways. Disposable mouthpieces are inserted

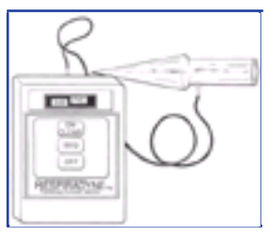

Figure 2-4 The Respiradyne portable spirometer. The device is turned on or "cleared" from the last effort. The patient exhales forcefully into the handheld mouthpiece following maximal inhalation. The "sequence" button permits selection of the desired spirometry measurements. The best of several tries should be recorded.

or attached. If the mouthpiece is to be reused by patients sharing a common meter (during serial evaluations), the "mouth" end should be identified with pen or tape to limit the possibility of spreading infection between patients. The electronic devices must be switched on for a 30-second self-calibration period. Both the handheld spirometers and the peak flow meters can be operated with the same respiratory maneuver: a maximal inhalation followed by a maximum forced expiration into the mouthpiece. The key is to have the patient exhale as rapidly as possible in order to achieve a brief, forceful exhalation rather than a slow, prolonged exhalation. Three attempts are standard, and the highest value is recorded, provided the best two of three readings are within 10% of each other. [18] Children as young as 3 years have consistently achieved the level of cooperation necessary to perform PEFR testing. [19]

Interpretation

Although intraindividual peak flow variability is low, [20] variability between different brands of calibrated spring mechanism peak flow meters may be significant. Flow measurements of four brands studied at low to medium flows differed by as much as 100 L/min, [21] and accuracy waned with age of the device. [22] This information should be considered when interpreting PEFR values both clinically and in the literature. Likewise, measurement of serial PEFRs should be done on the same meter. Altitude minimally affects PEFR interpretation; readings at 1400 m underestimate PEFR by 5.3% to 6.9%. [20] As with FEV_1 measurement, patient size affects PEFR and is most important when interpreting readings in children. Most charts of normal PEFRs in pediatric patients are based on height. As age and height are also related, some investigators have developed age-PEFR charts. [14] [23] Having a measuring tape secured to the wall alongside a chart of normal values in an accessible area enables the clinician to quickly interpret the PEFR of a child. Table 2-2 lists approximate peak flow and spirometric values for various degrees of obstruction in adults and Figure 2-5*A* and *B* demonstrate percentile charts of PEFR vs height in boys and girls. These charts were constructed from data obtained by Carson and coworkers in 2752 healthy children in Dublin. [23] Normal PEFRs do vary in children based on a number of variables including race, geographic location, and local environment. [23] [24] [25]

The use of PEFR measurements to predict the need for admission early during ED treatment found that in severely compromised adults (i.e., with initial PEFRs <16% of the predicted value), an improvement in PEFR <60 L/min after the first epinephrine treatment indicated the need for admission. [26] Similarly, an initial FEV_1 of 0.6 L and a post-treatment

TABLE 2-2 -- Approximate Values for Spirometry and Peak Flow for Various Degrees of Obstruction in Adults

	FEV_1 (L)	FEV_1 /FVC (%)	Peak Flow (L/min)
			550–650 (males)
Normal	4.0–6.0	80–90	400–500 (females)
Mild	3.0	70	300–400
Moderate	1.6	50	200–300
Severe	0.6	40	100

Peak flow and spirometry values vary with height, sex, and distance above sea level; men and taller persons in general have larger flow rates. The reduced barometric pressures found at higher elevations also increase airflow.

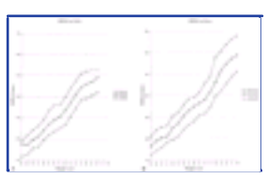

Figure 2-5 Peak expiratory flow rate vs height for girls (*A*) and for boys (*B*). These figures were constructed from Carson and coworkers' unsmoothed data obtained from 2752 healthy children in Dublin. *(From Carson JWK, Hoey H, Taylor MRH: Growth and other factors affecting peak expiratory flow rate. Arch Dis Child 64:96, 1989.)*

FEV_1 of 1.6 L were predictive of the need for inpatient therapy. [27] PEFR values of <100 L/min initially and <300 L/min after treatment were predictive of the need for inpatient therapy in a follow-up study. [15]

These studies have been challenged due to lack of blinding. A subsequent study found that neither the initial PEFR reading nor the response to the first bronchodilator treatment was helpful in predicting admission. [28] In fact, in this double-blind study, with strict admission criteria and with the additional use of nebulized bronchodilators, <50% of those requiring admission were detected by initial measurements. However, measurements performed later in therapy, just before release, were more accurate in predicting relapse. Similarly, in a series of 83 COPD patients, a post-treatment FEV_1 <40% of predicted value identified patients requiring admission (i.e., at high risk for relapse [>30%] if sent home). [29]

As with adults, initial spirometric values are weakly associated with pediatric asthma admission decisions. [30] [31] However, children with PEFRs >60% of expected *after* treatment with nebulizer therapy can be considered in the category "admission probably unnecessary," and those with PEFRs <40% can be considered in the category "admission probably necessary." [14]

These studies indicate that when initial flow rates are extremely poor, admission is likely, because a brief course of therapy in the ED is unlikely to provide sufficient improvement. However in patients with less severe flow rates, the decision to admit should be based on the patient's response to therapy (which is dependent in part on the form of therapy and the duration of the therapy chosen). Prediction of the need for admission based on PEFR and FEV_1 values obtained shortly after presentation is largely unsuccessful because of patient response variability and the option of more prolonged, aggressive ED therapy.

Hence, in adults, initially low flow rates (PEFR <100 L/min) and spirometry values (FEV_1 <1.0 L) identify sick patients with the potential need for admission or close follow-up. Patients with PEFR or FEV_1 values of <40% of predicted normal values after bronchodilator therapy probably require admission. Also, one can reserve blood gas analysis for patients with poor initial flow measurements or lack of improvement in pulmonary function (or hypoxia based on oximetry) and hence reduce the expense and morbidity of evaluation for reactive airway disease. [32] [33]

PULSE OXIMETRY

Pulse oximetry has made a significant contribution to noninvasive monitoring of oxygenation in a wide variety of clinical situations. Pulse oximetry requires no special training, is noninvasive, is inexpensive, gives continuous real-time estimates of arterial saturation in the range of 80% to 100%, and provides an early warning of diminished perfusion while avoiding the discomfort and risks of arterial puncture. As a result, it has become the standard of care during procedures requiring general anesthesia,[34] and it is gaining wide acceptance in pre-hospital care, [35] neonatal and pediatric critical care, [36] adult ED care,[37] and other clinical areas. [38] [39]

Background

Oximetry was first developed in Germany in 1932 by Nicolai, Kramer, and Matthes. The principle of oximetry is based on the Beer-Lambert law, which states that the concentration of an unknown solute dissolved in a solvent can be determined by light absorption. Early oximeters relied exclusively on this spectrophotometric principle, using a finger or an ear as a curette containing hemoglobin, compressing these tissues to obtain a bloodless baseline absorption, and warming them to obtain a signal from maximally arterialized tissue.

Pulse oximetry, combining the principles of optical plethysmography and spectrophotometry, was invented by Aoyagi and coworkers in 1974. [40] However, it was not until 1980 that the pulse oximeter reached modern form, consisting of a probe and an onboard computer. [41] The probe, set into a reusable clip or a disposable patch, is made up of two photodiodes producing light at 660 nanometer (nm) (red) and at 900 to 940 nm (infrared), and a photodetector, which is placed across a pulsatile vascular bed such as the finger or ear (Fig. 2-6). These particular wavelengths are used because the absorption characteristics of oxyhemoglobin and reduced hemoglobin are quite different at the two wavelengths. The majority of light is absorbed by connective tissue, skin, bone, and venous blood. The amount of light absorbed by these substances is constant with time and does not vary during the cardiac cycle. A small increase in arterial blood occurs with each heartbeat, which results in an increase in light absorption (Fig. 2-7). By comparing the ratio of pulsatile and baseline absorption at these two wavelengths, the ratio of oxyhemoglobin to reduced hemoglobin is calculated.

Because the pulse oximeter uses only two wavelengths of light, it can only distinguish two substances. As a result, pulse oximeters measure "functional saturation," which is the percentage of oxyhemoglobin compared to the sum of the oxyhemoglobin and reduced hemoglobin. The disadvantage of functional saturation is that the denominator does not include other hemoglobin species that may be present, such as carboxyhemoglobin and methemoglobin. The advantage is that the use of only two wavelengths in the oximeter reduces

Figure 2-6 *A*, Pulse oximeter sensors attached to digits. *B*, Pulse oximeter sensors attached to the dorsum of the nose and the earlobe. Only one site is measured at a time.

device cost, size, and weight. The CO-oximeter, one example of a commercially available in vitro oximeter and the standard by which pulse oximetry is calibrated, uses four or more wavelengths, measures the "fractional saturation," and is able to measure additional hemoglobin species.

The arterial O_2 saturation (SaO_2) measures the large reservoir of O_2 carried by hemoglobin, 20 mL of O_2/100 mL of blood, compared with the arterial O_2 partial pressure (PaO_2), which only measures the relatively small amount of O_2, approximately 0.3 mL of O_2/100 mL of blood, dissolved in the plasma. The SaO_2 correlates well with the PaO_2, but the relationship is nonlinear and is described by the oxyhemoglobin dissociation curve (Fig. 2-8). For the hypoxic patient, small changes in SaO_2 represent large changes in the PaO_2, because these SaO_2 values fall on the steep portion of the curve. Conversely, measurements of SaO_2 are relatively insensitive at detecting significant changes in PaO_2 at high levels of oxygenation because these SaO_2 values fall on the plateau portion of the curve.

Currently available pulse oximeters are accurate and precise when saturations range from 80% to 100%. [42] [43] This range is satisfactory, because for most patients an O_2 saturation of 80% is as much an urgent warning as one of 67%.

Clinical Utility

Pulse oximetry has been suggested as an additional vital sign. [39] By providing the clinician with either continuous or frequent intermittent estimated SaO_2 measurements, pulse oximetry offers a more physiologic means of assessing the adequacy of oxygenation than the arterial blood gas. The latter must be drawn from an arterial source and quickly transported under specific conditions to a machine that is often distant from the patient's bedside. With pulse oximetry, the test results are available at the bedside without an invasive arterial procedure. Note that as described earlier, measurement of SaO_2 is determined from blood gas measurements coupled with knowledge of actual hemoglobin levels in a patient's blood. The SaO_2 measurement is estimated using pulse oximetry (SpO_2). In this chapter we equate SaO_2 and the two measures and an SpO_2. We address potential differences in the interpretation

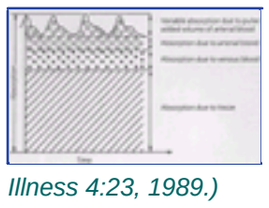

Figure 2-7 Factors influencing light absorption through pulsatile vascular bed. *(From McGough EK, Boysen PG: Benefits and limitations of pulse oximetry in the ICU. J Crit Illness 4:23, 1989.)*

section in the next section. A detailed review of pulse oximetry is provided elsewhere. [44]

All recommended uses of pulse oximetry fall into one of two broad categories. First, as a real-time indicator of hypoxemia, continuous monitoring of pulse oximetry can be used as a warning system, as many (but not all) adverse patient events are associated with arterial desaturation. [45] The second broad use of pulse oximetry is as an end point for titration of therapeutic interventions.

Although limited data address the clinical efficacy of routine monitoring of pulse oximetry in intensive care or ED settings, several studies have addressed operating suite and recovery room use that allow extrapolation. [46] [47] [48] These studies reported a decreased incidence and duration of desaturation episodes, fewer adverse impact events in the recovery room, and a shortened time to discovery of hypoxia. Therefore, routine monitoring of pulse oximetry in the critically ill patient population should result in fewer episodes of severe arterial desaturation and more rapid recognition of the adverse physiologic events that produce arterial desaturation. Patient outcomes should be improved with therapeutic interventions resulting from immediate knowledge of an unfavorable SaO_2. [49]

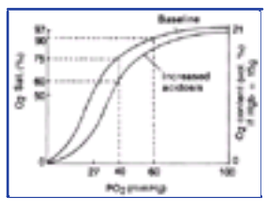

Figure 2-8 Oxyhemoglobin dissociation curve.

Indications

Some clinical applications of pulse oximetry is indicated are shown in Table 2-3 . Of note regarding assessment of respiratory illness severity in children, clinically unrecognized hypoxia (SaO_2 <92%) has been documented by pulse oximetry in preschool children with respiratory symptoms.[50] Also, an initial SaO_2 <91% in children with bronchospasm, regardless of subsequent improvement with ED management, has been associated with increased morbidity and need for admission.[51] As an additional "vital sign", SaO_2 monitoring affords the opportunity to assess both oxygenation and perfusion. This screen is helpful in those patients with suspected sepsis.

Procedure

The location for the probe is determined by the clinical situation and available probes. A reusable clip-on probe makes the digits easily accessible. Little is needed in the way of preparation, and

TABLE 2-3 -- Clinical Applications of Pulse Oximetry

During emergency airway management
Assess need for further airway management
Assess adequacy of preoxygenation before endotracheal intubation
Monitor ventilator and FiO_2 changes
Provide early indicator of ventilator dysfunction
Assist routine weaning of O_2 therapy, but not mechanical ventilation
Oxygenation monitor
Monitor patients with lung dysfunction for unexpected hypoxic events
Monitor during procedures including systemic sedation
Monitor oxygenation during interhospital and intrahospital transfer
Assess acute childhood asthma
Additional "vital sign"

as mentioned subsequently, even nail polish may generally be left on. Other sites include the earlobe, the nasal bridge or septum, the temporal artery, and the foot or palm of an infant. Tape and splints can be used to secure the digit probe and to minimize motion.

After placement of the probe, the machine is turned on. The computer analyzes the incoming data to identify the arteriolar pulsation and displays this in beats per minute; newer devices also display a pulse plethysmograph. Simultaneously, O_2 saturation is displayed on a beat-to-beat basis. Some machines have hard copy capability and can provide paper documentation of the patient's status. If the oximeter fails to detect pulsatile flow, either the reading will not display or the SaO_2 value will be given with a poor signal quality warning, depending on the machine. It is valuable to evaluate serial measurements and to verify that measurements correlate with other clinical markers.

Interpretation

Patients with good gas exchange have O_2 saturations between 97% and 100%. When the SaO_2 falls below 95%, hypoxia is present, although patients with COPD may live in this range. Oxygen saturations below 90% represent relatively severe hypoxia. Children with SaO_2 values <92% often require admission for O_2 and additional therapy.[50] [51] Although hypoxia is common with pneumonia, room-air SaO_2 values =98% have been reported in 45% of children with pneumonia.[52] In adults and older children with reactive airway disease, as with PEFR and spirometry, a low isolated early measurement of SaO_2 does not mandate admission because of the wide variability of response to therapy.[53] [54] However, low SaO_2 readings should be heeded as important clinical warning signs.

Although pulse oximetry represents a significant advance in noninvasive monitoring, clinicians must recognize and understand its limitations. Pulse oximetry measures O_2 saturation. In comparison to arterial blood gas determination or end-tidal CO_2 (carbon dioxide) monitoring, which is discussed later, pulse oximetry provides no direct information on pH or $PaCO_2$ levels, and only indirectly assesses the adequacy of ventilation. Witting and Lueck have empirically demonstrated that a room-air SaO_2 value =97% strongly rules against hypoxemia and moderate to severe hypercapnia.[55] Their validated study of patients with respiratory complaints receiving arterial blood gas analysis found good discrimination with a room-air SaO_2 value =96%. For hypoxia (PaO_2 <70 torr), this value was 100% sensitive (95% CI 95% to 100%) and 54% specific (95% CI 45% to 64%). For hypercapnea ($PaCO_2$ >50 torr), this value was 100% sensitive (95% CI 70% to 100%) and 31% specific (95% CI 25% to 38%). Kelly and colleagues found a cut-off of a room-air SaO_2 value =92% as more accurate for identifying hypoxia in chronic obstructive pulmonary disease patients.[56]

During both conscious sedation and general anesthesia, monitoring of ventilation is a more desirable goal for prevention of hypoxia than simple pulse oximetry. Hypoventilation and resultant hypercapnia may precede a decrease in hemoglobin O_2 saturation by many minutes. Furthermore, supplemental O_2 may mask hypoventilation and CO_2 retention by delaying the eventual O_2 desaturation for which pulse oximetry monitoring is designed to recognize. One animal study demonstrated that in preoxygenated animals, airway obstruction could be detected within 10 seconds using capnography, but SaO_2 values did not change during the 180-second study periods.[57] Several other pulse oximeter limitations have been noted and are summarized in Table 2-4 .

Effects of Dyshemoglobinemias

Carboxyhemoglobin results in falsely high SaO_2 estimates of hemoglobin O_2 saturation.[58] Low quantities of methemoglobin (MetHb) will reduce a pulse oximeter reading by about half the actual MetHb percentage.[59] However, even large quantities of MetHb (>10%) can result in a stable pulse oximeter reading of 85% regardless of the actual SaO_2 . In summary, in patients with methemoglobinemia or with elevated carboxyhemoglobin levels, the reading on the pulse oximeter does not accurately depict quantitative hemoglobin O_2 saturation changes.[60] [61] The pulse oximeter will variably *underestimate* the percentage of abnormal hemoglobin; a CO-oximeter is required for confirmation of these conditions and quantitative analysis.

Fetal Hemoglobin

Newborn full-term infants can have up to 75% of total hemoglobin in the form of fetal hemoglobin.[62] Pulse oximeters remain accurate in the presence of fetal hemoglobin.[63] However, a CO-oximeter will erroneously interpret the carboxyhemoglobin level to be elevated, and the oxyhemoglobin level to be artificially reduced.[64] Therefore, when fetal hemoglobin levels are high, CO-oximetry readings should not be used to confirm pulse oximetric readings. It should be noted that

newborns normally have up to 5% carboxyhemoglobin.

Low Perfusion

To function properly, pulse oximeters require a pulsating vascular bed. Hypotension with vasoconstriction, hypothermia, or the administration of vasoconstricting drugs may reduce the pulsatile component to <0.2% of the total signal. At this level, the true signal cannot be distinguished from background noise. Under these conditions, many pulse

TABLE 2-4 -- Factors Affecting Pulse Oximetry Readings

Severe anemia: Satisfactory readings obtained down to hemoglobin level of 5 mg/dL
Motion-artifact: See text regarding probe sites
Dyes: Transient effect unless methemoglobinemia results
Light-artifact: Minimize by covering probe with opaque material
Hypoperfusion: Inadequate pulse signal will display
Electrocautery: Minimize by increasing the distance of the sensor from the surgical site
Deep pigmentation: Use fifth finger, earlobe, or other area of lighter pigmentation
Dark nail polish: Remove with acetone or place sensor sideways on digit
Dyshemoglobinemias (e.g., carboxyhemoglobin and methemoglobin): Falsely elevate saturation reading
Elevated bilirubin: Accurate up to bilirubin level of 20 mg/dL in adults; no problem reported for jaundiced children
High saturation: Pulse oximetry not useful for monitoring hyperoxemia in neonates
Fetal hemoglobin: No effect on pulse oximetry; falsely reduced CO-oximetry readings
Venous pulsations: Artificially lower O_2 saturation; choose probe site above the heart
Dialysis graft (A-V fistula): No difference from contralateral extremity unless fistula produces distal ischemia

oximeters display a message indicating an inadequate pulse signal. A change in the location of the sensor to an area with higher perfusion, such as an earlobe, may improve the pulse signal. [65]

Intravenous Dyes

A number of dyes and pigments interfere with the accuracy of pulse oximetry. [66] [67] Methylene blue, the treatment for methemoglobinemia, absorbs light at 660 nm, similar to the absorption of reduced hemoglobin, and can artificially lower pulse oximeter saturation readings to as low as 1%. Low readings also can be seen with other intravenous dyes such as indigo carmine, indocyanine green, and fluorescein, although the rapid clearance of these agents minimizes the phenomenon. [68] [69]

Bilirubin

Hyperbilirubinemia does not affect the accuracy of pulse oximetry measurements. However, hyperbilirubinemia may have an effect on absorption at the lower wavelengths used by CO-oximeters, resulting in a discrepancy between pulse oximeter and CO-oximeter readings. [70]

Skin Pigmentation

Pulse oximeter accuracy is somewhat reduced by deeply pigmented skin. This effect is likely due to a shift in the light-emitting diode's output spectrum as the light output is increased. This effect is small and results in only a slight decrease in accuracy over a large number of samples. [70] Placing the probe on an area of lighter pigmentation, such as the fifth finger or an earlobe, has been suggested as a means to minimize this effect.

Nail Polish

Conflicting data exist about the effect of nail polish on the accuracy of pulse oximeter determinations. Mounting the probe side-to-side on the finger was found to be as accurate as readings on uncovered nails. [71] This technique also circumvents the problem of only partial placement of the probe because of very long fingernails, which may cause a low O_2 saturation reading. An alternative solution to the problem of nail polish is to remove it with acetone. The accuracy of SaO_2 readings in the setting of synthetic nails is unknown. If a poor signal is obtained in the setting of a synthetic nail, either the synthetic nail should be removed or an alternative site for placement used.

High Saturation

Because the O_2 dissociation curve plateaus at saturation levels >90%, a large increase in PaO_2 results in a small increase in saturation. Therefore, an error of a few percentage points could represent a large error in PaO_2. This is inconsequential for most adult patients but is of extreme importance for neonates at risk of retinopathy caused by hyperoxemia.

Venous Pulsations

Increased venous pulsations resulting from right heart failure, tricuspid regurgitation, or placement of a tourniquet or blood pressure cuff above the probe can interfere with accurate readings and lead to artificially lower O_2 saturations, because the pulse oximeter interprets any pulsatile measurement as arterial. [72] Placing the probe on a site above the heart may improve accuracy. Some pulse oximeters have the capability to synchronize pulsations at the probe site to electrocardiographic (ECG) signals, thus enhancing the signal-to-noise ratio.

Anemia

Because pulse oximeter measurements depend on light absorption by hemoglobin, they become less accurate and reliable in conditions of severe anemia. However, accuracy is not diminished until the hemoglobin content is <5 mg/dL. [73] [74]

Ambient Light

As the pulse oximeter's photodetector is nonspecific, high-intensity ambient light can produce interference. Surgical, fluorescent, and heating lamps are the common sources of this interference. This problem can be corrected by wrapping the probe with a light barrier, such as a dark cloth or other opaque material. [65]

Motion

Motion of the probe can produce considerable artifact and inaccurate pulse oximetry readings. However, this phenomenon is transient and seldom influences

assessment. Correlating a pulse oximeter signal with an ECG waveform[70] or using alternate probe sites, such as the ear or toe, also may reduce motion artifact.

Probe Site

The finger is the most common probe site used for adult pulse oximetry. If the finger is inaccessible or unsuitable, other probe sites, such as the earlobe, nose, and forehead (using reflectance instead of transmittance), may be used. It should be noted, however, that the forehead and nasal bridge probes may be less accurate than the finger and ear probes.[70] In infants and small children, an adhesive sensor unit is preferred. Probes also can be secured in place over the heel or lateral foot using a gauze or Coban wrap. Common sites of attachment include the great toe, the heel, and the lateral aspect of the foot.

Electrocautery

Electrical interference from devices such as electrocautery also can impair the accuracy of pulse oximetry. This interference can be reduced by increasing the distance between the surgical site and the probe.

Conclusions

Pulse oximetry is a widely available and relatively inexpensive technology that provides an easy, noninvasive, and generally reliable method to monitor oxygenation. As measurements are continuous, pulse oximetry allows for earlier detection of clinically unsuspected hypoxic episodes than does intermittent arterial blood gas analysis. Frequent measurements should lead to earlier corrective measures with the prevention of adverse consequences.

CARBON DIOXIDE MONITORING

Capnometry is a noninvasive method used to assess the adequacy of ventilation by measuring the CO_2 concentration of exhaled air. With an intact pulmonary circulation, CO_2 is present in tracheal but not esophageal gas efflux. The absence of CO_2 in an endotracheal tube indicates esophageal intubation, technical malfunction, circulatory arrest, circuit disconnection, or intraluminal/extraluminal tube obstruction. The continued presence of CO_2 in the expired air confirms tracheal intubation. In addition, capnometry provides a measure of systemic

metabolism and circulation that may be continuously monitored. Although the measurement of $PaCO_2$ via arterial blood sampling is the most direct method of evaluating ventilation, it is invasive, costly, and provides only intermittent data.

Capnography, introduced into clinical practice by Smalhout and Kalenda in the mid-1970s, [75] displays the CO_2 measured by capnometry graphically with respect to time throughout the respiratory cycle. The capnogram is the displayed waveform of breath-to-breath variations in the expired CO_2 concentration, and characteristic variations in its shape are associated with specific abnormalities. [76]

Indications

Verification of Endotracheal Tube Placement

In an emergency situation, the clinician desires a quick and reliable method to verify correct placement of an endotracheal tube (ETT). Traditional clinical methods of evaluating ETT placement (breath sounds, chest movement, epigastric auscultation), although adequate in the majority of circumstances, are not fail-safe. [77] Although direct cord visualization reliably assesses ETT passage into the trachea, such visualization is not always possible. Additionally, an ETT may become dislodged even after correct placement as a result of head movement or patient positioning. Numerous studies in animals and humans under controlled and emergency conditions support the use of CO_2 measurement in expired air as a means of ETT placement confirmation.[60] [77] [78] [79] This technique is described in more detail in Chapter 4 .

Systemic ("Conscious") Sedation

Many procedures performed in the ED are facilitated by sedation. The use of hypnotic doses of benzodiazepines can result in respiratory depression, and opioid analgesics predictably reduce ventilatory drive and the normal response to both hypoxia and hypercarbia. Moreover, the combination of benzodiazepines and opioids substantially increases the incidence of hypoxemia and apnea as compared with the effects of either agent alone. [80]

Monitoring of ventilation, perfusion, and oxygenation increases the safety of sedation. As mentioned earlier, pulse oximetry indirectly assesses ventilation. Although hypoventilation, hypercarbia, and apnea ultimately lead to a decrease in O_2 saturation on pulse oximetry, hypoxia is preceded by a rise in end-tidal CO_2 partial pressure ($PetCO_2$).[81] [82] Hence, combining exhaled CO_2 monitoring with pulse oximetry should further enhance the safety of systemic sedation.

Nasal cannula (side-stream) CO_2 analyzers have made continuous $PetCO_2$ monitoring possible during systemic sedation. Nasal cannula-derived $PetCO_2$ values correlate well with $PaCO_2$ in awake, spontaneously breathing patients and may be as accurate as those obtained from an ETT. [82] Two beneficial features of the $PetCO_2$ monitor are immediate evidence of changes in the respiratory pattern by observation of the capnographic waveform and an audible apnea alarm. This latter feature is especially valuable after completion of emergency procedures when personnel are less likely to be at the bedside and patient stimulation is minimized.

Cardiopulmonary Resuscitation and Shock Resuscitation

The role of $PetCO_2$ for monitoring the effectiveness of cardiac compressions, estimating the potential for cardiac resuscitation, and detecting restoration of spontaneous circulation is discussed in more detail in Chapter 17 . A proposed related use is the difference between $PaCO_2$ and $PetCO_2$ (i.e., the $P(a\text{-}et)CO_2$ gradient) as a measure of under-resuscitation in traumatic shock. [83]

Mechanical Ventilation

Many emergency clinicians manage ventilated patients past the initial resuscitation phase while they are awaiting patient transfer to a critical care unit or during further diagnostic testing. Capnography can noninvasively and continuously monitor for ventilation failure (e.g., discontinuities in the ventilator circuit, CO_2 rebreathing) and assess changes in mechanical support, including weaning from the ventilator. [84]

Contraindications

In the critically ill patient, capnography may not be an adequate substitute for arterial blood gas sampling during adjustments of or weaning from mechanical ventilation. In such patients, $PetCO_2$, when used, should be correlated with $PaCO_2$ measurements via arterial blood gas analysis. That is, $PetCO_2$ values should be used as a supplement to, rather than as a replacement for, $PaCO_2$ values.

In spontaneously breathing patients, side-stream analyzers with nasal cannula sampling techniques must be used with caution, because ambient air may be entrained and therefore dilute $PetCO_2$ measurements. Moreover, the accuracy of monitoring $PetCO_2$ by this method is critically dependent on the relative placement of the sampling tube in the patient's naris or nasopharyngeal airway relative to the source of supplemental O_2 . The presence of mouth breathing also can dilute $PetCO_2$ values when nasal cannulas are used.

In children with fast ventilatory rates and low tidal volumes, a side-stream analyzer with a slow sampling flow rate underestimates the $PetCO_2$. Sampling flow rates of 150 mL/min yield consistently accurate estimates of $PaCO_2$ and an acceptable capnogram in neonates, infants, and children. [85] Adult-size disposable end-tidal CO_2 detectors are not recommended in children who weigh <15 kg, because their small tidal volumes may be diluted in the large (38 mL) dead space of the device, resulting in failure to detect CO_2 appropriately. [78] [86]

Equipment

Of the two quantitative methods of CO_2 gas analysis used in hospitals, mass spectrometry and infrared absorption spectrophotometry, only the latter is used commonly in the intensive care unit and ED. Qualitative CO_2 -sensitive colorimetric technology also is available. A product comparison of the many commercial capnography devices has been published elsewhere. [87]

The two types of infrared capnometers used with intubated patients are:

1. The mainstream (in-line) capnometer, which consists of a transducer housed in an airway connector and placed in-line with the patient's breathing circuit (Fig. 2-9). The transducer contains the infrared light source and photodetector, heated to approximately 40°C to prevent condensation. Mainstream sampling allows

the capnometer to directly assess the patient's respiratory circuit. Hence, gas analysis is nearly instantaneous (<500 msec). The major disadvantage is the weight associated with the airway connector attached to the

proximal end of the ETT. Examples of these devices are the Nellcor Stat-CAP N60B and Ultra-CAP N6000B (Nellcor Inc., Pleasanton, CA). The former device is a portable, battery-operated instrument that provides continuous qualitative readings. The latter device is a bedside monitor that displays both a continuous waveform and numeric information.

2. The side-stream or diverting capnometer actively withdraws gas from the patient's airway via a sampling tube, and the concentration of CO_2 is measured in the machine rather than in the ETT (Fig. 2-10). The major advantage of side-stream analyzers is their applicability to nonintubated patients. Their major disadvantage is a slower response time, because expired gas must be transported to the capnometer itself for analysis. Slower response time results in dispersal of the gas bolus due to convection and diffusion during transit through the sampling catheter. These factors result in an underestimation of $PetCO_2$, particularly in children who have high ventilatory rates and small tidal volumes. Response times of side-stream analyzers are dependent on the aspiration flow rate, sampling tube length, and tubing diameter. To achieve predictable $PetCO_2$ values and CO_2 waveforms, the response time of the analyzer must be less than the respiratory cycle of the patient.[88] The optimal gas sampling flow rate is considered to be 50 to 200 mL/min, which ensures that the capnographs are reliable in both children and adults.[89] Despite a water trap, side-stream analyzers also are affected by secretions, saliva, and water condensation.

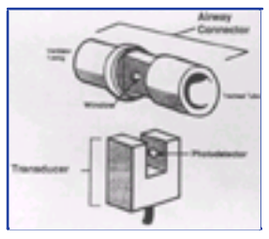

Figure 2-9 Schema of mainstream analyzer used for capnography. Transducer to measure CO_2 is placed in-line of airway connector. Infrared light source (not indicated) resides opposite photodetector in transducer. *(From Szalfarski NL, Cohen NH: Use of capnography in critically ill adults. Heart Lung 20:363, 1991.)*

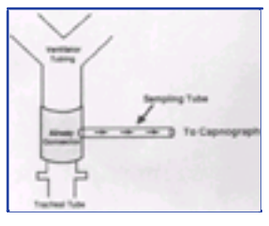

Figure 2-10 Schema of side-stream CO_2 sampling technique in capnography. *(From Szalfarski NL, Cohen NH: Use of capnography in critically ill adults. Heart Lung 20:363, 1991.)*

Qualitative, lightweight, disposable end-tidal CO_2 detection devices are based on colorimetric changes. A pH-sensitive chemical indicator is enclosed in a plastic housing and is connected to the gas stream between the ETT and the ventilator. The concentration of CO_2 in ambient air or the esophagus is <0.3%. These devices change color when the concentration of CO_2 is higher than 4% or 5.4%. The color varies as the CO_2 level increases and decreases between expiration and inspiration. The devices are designed to function for approximately 2 hours. The response time of the device is sufficiently fast to detect changes of CO_2 breath by breath.[90] In addition to their portability and single-patient use, these devices have the advantage of not requiring electrical power. Originally, the disposable CO_2 detectors were only available for attachment to ETTs (EASY-CAP and Pedi-CAP, Nellcor Inc., Pleasanton, CA). Chemical CO_2 detection devices, built into disposable bag-valve ventilation devices, are now available for ED use (CAPNO-FLO, Kirk Specialty Systems, Carrollton, TX). Both systems have components for adult and pediatric patients.

Procedure

For mainstream capnometers, simply place the airway connector in-line between the ETT and respiratory circuit. For side-stream or diverting capnometers, position the sampling tube in the naris via nasal cannula, nasopharyngeal tube, or other adapters available with particular machines. After sufficient warm-up time, a continuous recording of the capnogram or a digital display of a $PetCO_2$ value will display depending on the type of machine used. The chemical CO_2 detection devices are ready to use after attachment to the ETT (EASY-CAP, Pedi-CAP), or they come as a component of the bag-valve device (CAPNO-FLO).

Interpretation

Various factors result in either increased or decreased (or even absent) $PetCO_2$ (Table 2-5). With regards to the measurement device, a continuous recording of the capnogram is more advantageous than simply a digital display of a $PetCO_2$ value.

TABLE 2-5 -- Various Factors That Influence $PetCO_2$

	Factor			
Effect on $PetCO_2$	***CO_2 Production***	***Pulmonary Perfusion***	***Alvelor Ventilation***	***Technical/Mechanical Errors***
Increased				
Fever	Sodium bicarbonate	Hypoventilation	Rebreathing	Leaks in breathing system
Thyrotoxicosis	High cardiac output	Bronchial intubation	Exhausted CO_2 absorber	Faulty ventilator
Malignant hyperthermia	Hypertension	Parital airway obstruction	Inadequate fresh gas flow	Faulty valves
Decreased or Absent				
Hypothermia	Hypovolemia	Hyperventilation	Partial airway obstruction	Circuit disconnection
Low cardiac output	Pulmonary embolism	Apnea	Tracheal extubation	Sampling tube leak
Hypotension	Cardiac arrest	Total airway obstruction		Malfunction of ventilator

A normal capnogram has four phases (Fig. 2-11). At the onset of exhalation (point B) respiratory gases are free of CO_2, because they represent the anatomic dead space. The steep rising slope of segment B–C represents the emptying of rapidly exchanging alveoli. Segment C–D represents the emptying of uniformly ventilated alveoli, where the concentration of exhaled CO_2 is nearly constant. True end-tidal CO_2 ($PetCO_2$) is the highest concentration measured during the alveolar plateau (point D) and immediately precedes the onset of the next inspiratory cycle. Inspiration is represented by the rapidly descending limb of the capnogram (segment D–E) and, unless significant CO_2 rebreathing occurs, return of the CO_2 concentration to near zero (baseline).

At the end of exhalation, the composition of expired air closely reflects that of alveolar air. Under normal conditions, the alveolar CO_2 reflects the CO_2 present in mixed venous blood ($PvCO_2$), which equilibrates with the resultant value for arterial blood ($PaCO_2$). Under normal conditions, only a small gradient (<6 mm Hg) exists between $PaCO_2$ and $PetCO_2$.[91] The $P(a\text{-}et)CO_2$ gradient is increased with age, COPD, pulmonary embolism, decreasing cardiac output, hypovolemia, and anesthesia

itself.[92] [93] [94] In the paralyzed and ventilated patient without underlying lung disease, the

Figure 2-11 The four phases of a normal capnogram. Exhalation begins at point *B*. The steep ascending limb (segment *B–C*) represents rapidly exchanging alveoli. Segment *C–D* is the alveolar plateau representing uniformly ventilated alveoli, with a nearly constant CO_2 concentration. $PetCO_2$ is the highest point on the alveolar plateau (point *D*) and immediately precedes the next inspiration. Segment *D–E* rapidly descends to the original baseline (zero in normal subject) during inspiration (segment *A–B*). *(Courtesy of Novametrix Medical Systems, Inc., Wallingford, CT.)*

$PetCO_2$ can be used to estimate the $PaCO_2$ and thus guide ventilation in the head-injured patient. [95]

The principal determinant of the size of the $PaCO_2$ -$PetCO_2$ gradient ($P[a\text{-}et]CO_2$) is the matching of ventilation and perfusion (V/Q). The major cause of V/Q mismatch is dead space ventilation, in which lung units have more ventilation than perfusion. As a result, the end-tidal gas contains less CO_2, and $PetCO_2$ values are lower, causing large differences in the $PaCO_2$ -$PetCO_2$ gradient.[94] [95] [96] [97] In this situation, the alveolar plateau (see Fig. 2-11, segment C–D) will not be flat, but rather will have a slow, rising expiratory upstroke (Fig. 2-12). In a normal patient, the alveolar plateau is flat because the expired gas from all lung units have similar V/Q relationships.[93] If the patient has significant maldistribution of ventilation, exhaled gas will have varying concentrations of CO_2, because the CO_2 concentration from some airways will be low, representing dead space ventilation, whereas the CO_2 concentration from others will be high. Only when a distinct alveolar plateau is present can it be assumed that true alveolar gas is being sampled and analyzed. [84]

In the setting of under-resuscitation of traumatic shock, the $P(a\text{-}et)CO_2$ gradient will be elevated. Tyburski and colleagues

Figure 2-12 Capnogram of patient with adult respiratory distress syndrome and pneumonia. Lack of a normal alveolar plateau is evident. *(From Szalfarski NL, Cohen NH: Use of capnography in critically ill adults. Heart Lung 20:369, 1991.)*

noted that a postoperative $P(a\text{-}et)CO_2$ gradient of >9 torr was associated with death in multiple-trauma patients. [83]

Intubation Assessment

False-positive capnographic tracings associated with esophageal intubation that closely mimic successful ETT intubation may occur in patients who have recently ingested carbonated beverages or antacids and those receiving mask ventilation with resultant gastric insufflation of CO_2-containing gases. [98] However, after 3 to 6 esophageal ventilations, $PetCO_2$ concentration rapidly declines as gastric CO_2 is washed out and diluted with fresh gas (Fig. 2-13).[99] [100] Tracheal ventilation will, of course, continue to produce a normal waveform.

After initial successful ETT placement and confirmation, changes in the CO_2 waveform can provide immediate detection of tube-related adverse events. When the ETT is partially obstructed, the capnogram will have a slow upstroke without a significant change in the $PetCO_2$ (Fig. 2-14).[84] [99] Bronchospasm and COPD exacerbations produce obstruction to expiratory gas flow, thus increasing the slope of the expiratory upstroke. With inadvertent extubation, the capnogram shows a sudden drop in $PetCO_2$ (the same change that occurs when the tube becomes completely obstructed or malpositioned in the esophagus) (Fig. 2-15 and Fig. 2-16). [99] ETT cuff leaks yield low $PetCO_2$ values because of the partial escape of the expired gas around the tube.

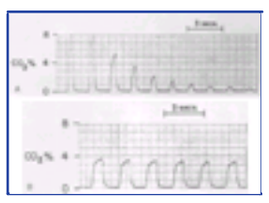

Figure 2-13 Esophageal capnogram after ingestion of carbonated beverage. *A*, CO_2 is clearly present as the esophagus is ventilated. The CO_2 concentration rapidly dissipates to the nonphysiologic range. *B*, The normal tracheal capnogram obtained simultaneously from the same experimental animal is shown. *(Modified from Garnett AR, Gervin CA, Gervin AS: Capnographic waveforms in esophageal intubations: Effect of carbonated beverages. Ann Emerg Med 18:387, 1989.)*

Monitoring Ventilation

Capnography is useful in diagnosing and monitoring maldistribution of ventilation during mechanical ventilation. In patients with a large V/Q mismatch, the capnogram will have a continued rise throughout the expiratory phase (see Fig. 2-12) and will lose the normal alveolar plateau. When this waveform occurs, the measured end-tidal CO_2 may not represent a good estimate of $PaCO_2$. However, by monitoring the slope of the expiratory phase on the capnogram, the efficacy of therapy can be evaluated. For example, in the case of asthma, as bronchospasm resolves, the slope of the expiratory phase will become less steep and the alveolar plateau better defined as a result of improved distribution of ventilation. [84] [101]

Furthermore, as the $P(a\text{-}et)CO_2$ gradient is sensitive to increases in dead space, this gradient can be used to evaluate changes and trends in dead space ventilation in critically ill patients with significant intrapulmonary shunting. [102] [103] It also can be used as a guide to determine the best positive end-expiratory pressure (PEEP) level for optimal oxygenation. Because a low $P(a\text{-}et)CO_2$ gradient implies the optimal V/Q relationship, the level of PEEP that produces the lowest $P(a\text{-}et)CO_2$ gradient is the level at which optimal alveolar recruitment occurs. [79]

In well oxygenated animals subjected to airway occlusion or extubation, changes in capnography occur considerably more quickly than vital sign changes or pulse oximetry changes.[57] The observational study of Miner and colleagues demonstrated that patients undergoing procedural sedation could demonstrate $PetCO_2$ >50 torr, an absolute change of >10 torr, or an absent waveform before a change in pulse oximetry. [104]

Excluding airway mishaps, ventilator setting changes, and circuit discontinuities, a sudden drop in end-tidal CO_2 may be an early indicator of shock or pulmonary embolism.[75] [105]

A dip in the plateau (curare cleft) (Fig. 2-17) indicates a spontaneous respiratory effort during mechanical ventilation [106] [107] and may be an early clue to the waning effects of a paralytic agent. Capnometry may also be especially useful in assessing the adequacy of hyperventilation in the

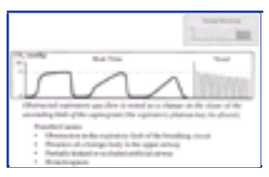

Figure 2-14 Partial endotracheal tube occlusion and other obstructions in the breathing circuit or airway. Note slurred upstroke of CO_2 waveform and absence of expiratory plateau. *(Courtesy of Novametrix Medical Systems, Inc., Wallingford, CT.)*

hemodynamically stable patient with head trauma. Other capnography patterns and their clinical significance are demonstrated in Figure 2-18 .

OXYGEN THERAPY

Earlier sections have dealt with ventilation and assessment of oxygenation. Delivery of O_2 is impaired in many clinical situations. Neurologic dysfunction is an early sign of hypoxemia. Otherwise-healthy individuals begin to experience short-term memory loss, euphoria, and impaired judgment when the PaO_2 level approaches 55 mm Hg. Progressive loss of cognitive and motor function, increasing tachycardia, and other physical signs occur when values are between 30 and 55 mm Hg. When levels are <30 mm Hg, the patient loses consciousness. PaO_2 is

Figure 2-15 Malposition of endotracheal tube in the esophagus. Note loss of capnographic waveform. *(Courtesy of Novametrix Medical Systems, Inc., Wallingford, CT.)*

a function of the fraction of inspired O_2 tension (FiO_2), the alveolar ventilation, and the relative distribution of both ventilation and perfusion in the lung. Supplemental O_2 can increase the amount of O_2 dissolved in blood enough to deliver one third of the body's resting metabolic requirements. This section reviews the noninvasive delivery of supplemental O_2.

Indications

Oxygen therapy is generally indicated when hypoxia is present (PaO_2 <60 mm Hg or SaO_2 <90% to 95%). Clinical situations that are commonly associated with hypoxia and generally benefit from supplemental O_2 include pulmonary disease, cerebrovascular accidents, gastrointestinal bleeding, shock, and trauma. Supplemental O_2 also is indicated when hypoxia is anticipated from a procedure such as intubation or

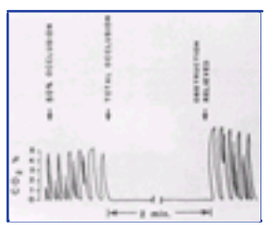

Figure 2-16 Occlusion of the endotracheal tube. Note that occlusion of a properly placed tube yields the same flat capnogram as esophageal intubation or accidental extubation. *(From Murray IP, Modell JH: Early detection of endotracheal tube accidents by monitoring carbon dioxide concentration in respiratory gas. Anesthesiology 59:345, 1983.)*

before a procedure in which intravenous analgesia may cause respiratory depression.

High altitude also results in hypoxia. At 7000 ft the barometric pressure is 564 mm Hg, the atmospheric partial pressure of O_2 is 116 torr, and the arterial partial pressure of O_2 (with healthy lungs) is approximately 63 torr. Commercial airlines maintain cabin pressures at levels between 5000 to 8000 ft at most cruising altitudes, and close to 10,000 ft temporarily under certain circumstances. Patients with chronic hypoxia may require supplemental O_2, and they must contact the airline before flying to make appropriate arrangements (the reader is referred elsewhere for a review of air travel and

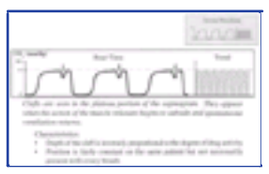

Figure 2-17 Capnogram of patient receiving assist-control ventilation and vecuronium paralyzation. *(Courtesy of Novametrix Medical Systems, Inc., Wallingford, CT.)*

O_2 therapy).[108] Oxygen is specifically indicated for carboxy-hemoglobinemia; 100% O_2 reduces the half-life of carboxy-hemoglobin from a mean of 5 hours to 1 hour; hyperbaric (more than 1.0 atm) O_2 can lessen this time by half again. However, discussion of hyperbaric O_2 therapy is beyond the scope of this chapter.

Contraindications

Paraquat turns O_2 into free radicals, thus making O_2 a substrate of the poison. Similar toxins in this group include bleomycin, cyclophosphamide, doxorubicin, ozone, and nitrous dioxide.[109] [110] Hence, O_2 therapy in the absence of documented hypoxia is contraindicated for this group of toxins.

Fears of ventilatory shutdown in the presence of CO_2 retention have made clinicians hesitant to give supplemental O_2 in amounts greater than 2 L/min. However, if hypoxia remains after low-flow O_2 is initiated, the O_2 delivery must be increased. Continuous monitoring of spontaneous respiration is required in this setting, and preparations to perform intubation should be made to assist the patient's ventilation if indicated. Note that inspired O_2 levels may actually rise with low-flow O_2 therapy as respiratory effort wanes, because less room air is entrained with shallow breaths; significant exacerbation of ventilatory failure has been documented with even 1 L/min of O_2 by nasal cannula.[111] However, a high CO_2 level is not a contraindication to appropriate O_2 therapy.

Equipment

The two most common sources of O_2 for hospital patients are wall outlets linked to a large reservoir of liquid O_2 and O_2 tanks (green by convention) of various sizes, listed in Table 2-6 . Both provide outlet pressures in the range of 30 to 50 psi. An approximation of the amount of time left in a given tank at a given flow rate may be calculated using the following formula:

Minutes of O_2 = gauge pressure (psi) × cylinder factor/flow rate (L/min)

Figure 2-18 *A–F,* Various abnormal capnography patterns and their causes. *(Courtesy of Novametrix Medical Systems, Inc., Wallingford, CT.)*

Cylinder factors are shown in Table 2-6 . Alternatively, a simple computer program that can estimate the duration of O_2 remaining in a tank based on the previous variables is available. [112] The numerous methods available to deliver O_2 from the source to the patient are listed in Table 2-7 .

Nasal cannulas can provide low-flow O_2 supplementation when accurate control of O_2 concentration is unimportant, as inspired concentrations vary considerably depending on patient tidal volume and inspiratory flow rate. [113] Rough guidelines for the *theoretical maximum* inspired O_2 concentration related to O_2 flow rates are listed in Table 2-7 . Importantly, the actual inspired O_2 will be less than the theoretical

TABLE 2-6 -- Oxygen Cylinders (Approximate Values)

Cylinder Type	Volume (L)	Cylinder Factor	Hours of O_2 at 2 L/min
D	450	0.20	3.5
E	650	0.28	5.0
G	5600	2.41	44.0
H, K	6900	3.14	58.0

maximum, due to mouth breathing, poor fitting devices, and inaccurate flow settings. Similarly, some mask systems are designed to be used without Venturi inserts, and package inserts indicate the range of inspired O_2 concentration based on O_2 flow rates. As with nasal cannulas, inspired O_2 concentrations vary considerably (may be upward of 10% greater than predicted by package inserts) depending on the patient's breathing pattern (rate and tidal volume).

Venturi systems should be used in patients with CO_2 retention for whom tight control of inspired oxygen is required. A partial nonrebreathing mask incorporates a 200 mL reservoir that effectively delivers an inspired FiO_2 of 60% to 80% in most patients. The system uses a 200 mL reservoir bag, initially filled with O_2 . Inspiration at high flow rates generally empties the reservoir and exhalation fills it with the first 200 mL of expired gas, which is mostly derived from the dead space of the upper airway and thus contains a high FiO_2 . A true nonrebreathing mask contains 2 types of valves and is capable of delivering close to 100% O_2 . One valve prevents exhalation gas from entering the reservoir bag. The second type has two valves on the mask that allow exhalation, concurrently preventing ambient air from entering the

TABLE 2-7 -- Oxygen Delivery Systems

System	Flow Rate (L/min)	FIO_2* (% Oxygen)	Advantages	Disadvantages
Nasal cannulas	1	25	Simple, comfortable, inexpensive, allow eating and drinking	Can cause local irritation and drying of mucous membranes (primarily for flow rates >4 L/min)
	2	29		
	3	33		
	4	37		
	5	41		
	6	45		
Simple oxygen masks	>5 L/min	35–50	Deliver higher flow rates than nasal prongs	Must be removed for airway care, eating, and drinking
Masks with reservoir bag	Up to 15 L/min	60	Higher FIO_2 at lower flow rates	Risk of atelectasis and oxygen toxicity (with prolonged use)
Venturi masks	4	24–28	More precise control of final oxygen concentration	Same as above
	6	31		
	8	35–40		
	10	50		
Reservoir nebulizers, including CPAP, T-tubes	40+ L/min	60–90	Can deliver increased humidity, positive pressure	Risk of barotrauma

*The *theoretical maximum* FIO_2 . In clinical practice the FIO_2 will be lower.

inhalation circuit. To comply with safety regulations designed to prevent asphyxiation should the O_2 supply be interrupted, many manufacturers remove one of the valves located on the mask, somewhat limiting the concentration of O_2 delivered.

Procedure

Supplemental O_2 should be given to increase the PaO_2 to between 60 and 80 mm Hg or between 90% and 95% SaO_2 . In attempting to deliver high concentrations of O_2 to a nonintubated patient, the challenge is to provide enough O_2 at flow rates sufficient to meet the patient's demands. A simple method of assessing the adequacy of O_2 delivery with a medium flow system is to add water mist to the O_2 (through a humidifier) and to visually observe the pattern of mist flow at the mask. Mist should escape from the side holes of the mask during both inspiration and expiration. If mist is not visualized to escape during inspiration, ambient air is being inspired by the patient (effectively reducing inspired O_2 concentration) and a higher flow system is required. Bedside pulse oximetry can provide prompt monitoring of changes in SaO_2 , allowing adjustments to be made without repeated arterial blood gas determination. Pulse oximetry values generally equilibrate within 5 minutes of an oxygen delivery adjustment. [114]

Evidence of CO_2 retention, such as decreasing mental status and failing respiratory drive, indicates the need for an arterial blood gas determination to document the $PaCO_2$; however, the decision to intubate generally should be made on clinical grounds. The need to humidify bedside low-flow O_2 is unproven. [110] [115] Table 2-8 lists the steps required to provide O_2 from cylinders.

Complications

Three distinct areas of risk accompany supplemental O_2 use: (1) respiratory dysfunction, (2) cytotoxic injury, and (3) physical hazards. Respiratory dysfunction results from CO_2 retention and atelectasis. Variations in CO_2 level provide the main stimulus to breathe in normal subjects. Patients with COPD have a decreased sensitivity to CO_2 levels secondary to chronic exposure to higher CO_2 levels, and hypoxia provides backup support for the respiratory drive. Oxygen given in sufficient amounts can remove the remaining chemical stimulus to respiration and has the potential to cause respiratory shutdown.

The complete abolition of the hypoxic drive has been reported to require a PaO_2 of 200 mm Hg. However, administration of 100% O_2 to 22 patients with COPD in acute respiratory distress (mean baseline blood gas values: $PaCO_2$ = 65 mm Hg, PaO_2 = 38 mm Hg) for 15 minutes resulted in only a transient decrease in minute ventilation.[116] The lowest values for minute ventilation were reached between 20 and 180 seconds from onset of inhalation, followed by a slow rise over the next 12 minutes to within 93% of the control value. The decrease was the result of falls in both tidal volume and respiratory rate; however, after 15 minutes, these parameters had returned to baseline levels. Despite little difference in

TABLE 2-8 -- Operation of Oxygen Cylinders

1. Secure the tank in an upright position so that it will not move or fall while being manipulated.
2. Remove the cylinder seal ("E" tank) or cylinder cap ("G," "H" tank).
3. Turn the cylinder valve on and off quickly to clear ("crack") the valve. On the "E" tank, this is done with a wrench; on the "G" or "H" tank, it is done with the cylinder handle.
4. Check the yoke to ensure that it is compatible for use with oxygen and place it on the cylinder. Make sure the fittings are compatible.
5. Tighten the yoke, making certain that any necessary gasket is in place.
6. Close the needle valve.
7. Slowly open the cylinder valve until the pressure maximizes.
8. Observe that the cylinder contains an adequate supply of gas.
9. Connect the desired secondary delivery system (e.g., nasal cannula).
10. Open the needle valve so that the desired oxygen flow registers on the flow meters.

these parameters at the 15 minute point, $PaCO_2$ had risen a mean of 23 mm Hg, and PaO_2 had risen a mean of 225 mm Hg.[117] The mechanism leading to atelectasis is less clear. Elevated O_2 levels may affect underperfused or underventilated pulmonary segments by decreasing hypoxic vasoconstriction. The increased perfusion could lead to greater absorption of the remaining gas, destabilizing the alveolar units and bringing on collapse. [118]

Cytotoxic damage, theoretically secondary to free radical production, leads to tracheobronchitis and adult respiratory distress syndrome (ARDS) manifested by pulmonary edema and focal lung collapse with pulmonary fibrosis as a long-term consequence. The mechanism of damage has been shown experimentally to include oxidation of carbohydrates with disruption of cell surface receptors, DNA-RNA alterations, lipid peroxidation, and protein denaturation. Administration of an inappropriately high level of O_2 does not usually produce consequences within the time frame of the ED visit. The risk of O_2 toxicity depends on several factors, including O_2 tolerance (the state of biologic resistance to O_2-induced damage, which is itself dependent on antioxidant defenses, age, and nutritional and hormonal factors), concentration of O_2 delivered, and duration of treatment. The goal is to deliver the least O_2 required to achieve adequate tissue levels. During resuscitation and most emergency care, 100% O_2 can be delivered safely to most patients without fear of cytotoxic injury. Healthy adult volunteers have received 100% O_2 for up to 6 hours without evidence of pulmonary injury. Except in special circumstances, such as paraquat poisoning, O_2 at concentrations of =50% is safe for 2 to 7 days. [110]

Physical risks associated with O_2 therapy include trauma associated with tank explosions, fire hazard, local irritation, and drying of mucous membranes.

NONINVASIVE PRESSURE SUPPORT VENTILATION

Noninvasive pressure support ventilation can be used to deliver increased airway pressure in several modes: (1) pressure increases solely during inspiration (i.e., delivery of inspiratory positive airway pressure [IPAP]) to supplement ventilatory mechanics; (2) continuous steady positive airway pressure maintained throughout the ventilation cycle (i.e., expiratory positive airway pressure [EPAP], also known as continuous positive airway pressure [CPAP]) to improve alveolar oxygen exchange; or (3) a combination of both modalities to achieve both goals. During noninvasive pressure support ventilation, a tight, well-fitting mask is placed over the patient's mouth and nose or just over the nose.

Mask CPAP treatment of cardiogenic pulmonary edema was first described more than 50 years ago[119] and has been shown to be a useful adjunct in the treatment of acute cardiogenic pulmonary edema. Mask CPAP results in early physiologic improvement and reduces the need for intubation and mechanical ventilation in patients with pulmonary edema.[120] [121] [122] It also reduces total hospital costs for patients with severe cardiogenic pulmonary edema. [123] Use of mask CPAP to deliver PEEP has aided the treatment of other forms of respiratory failure including those due to pulmonary infections, trauma, and obstructive lung disease. Historically, patients with respiratory failure secondary to *Pneumocystis carinii* pneumonia who required mechanical ventilation had in-hospital mortality rates as high as 86% to 94%; however, those who were judged to require mechanical ventilation, but who were instead treated with mask CPAP, had in-hospital mortality rates of 22% to 55%. [124] [125] [126] Mask CPAP may be efficacious in the treatment of respiratory failure secondary to other pulmonary infections. [127] [128] Further, mask CPAP (either by full-face or nasal mask) may obviate the need for intubation and mechanical ventilation in acute exacerbations of COPD. [129] [130] [131]

The general use of IPAP for respiratory failure has recently been reviewed. [132] The use of combined IPAP and EPAP in the ED and critical care setting appears promising.[133] [134] [135] [136] This technique is also known as bi-level positive pressure ventilation, because it allows the clinician to deliver different inspiratory and expiratory pressures. This technology offers the opportunity to both support mechanical ventilation and improve oxygen exchange through an enhanced functional residual capacity. In essence, a mask device provides the ventilation and PEEP that was once possible only with the use of tracheal intubation and a standard ventilator.

Indications and Contraindications

Noninvasive pressure support ventilation is indicated to treat impending ventilatory failure and avoid intubation and standard mechanical ventilation, with their associated morbidity and mortality. Noninvasive pressure support ventilation seems best applied to patients whose respiratory failure is expected to quickly respond to medical therapy, as continuous long-term mask CPAP or ventilation requires close attention.

Noninvasive pressure support ventilation for acute respiratory failure requires an alert patient capable of protecting the airway and handling secretions. Other contraindications include an inability to obtain a good mask fit, cutaneous irritation from the mask, and inability to cooperate with the therapy. Finally, intubation and standard ventilation is preferred for patients who require total ventilatory support, because the mask may slip and effective ventilation may cease.

Equipment

Although numerous systems that provide CPAP are on the market, they are infrequently used in the ED. A small, noninvasive bi-level positive pressure ventilation system (BiPAP System, Respironics Inc., Murrysville, PA) that permits use of a nasal (rather than facial) mask alone *in lieu* of tracheal intubation may be most useful in the ED.[136] Therefore, an overview of the use of the BiPAP System is given in the next section. The advantage of the BiPAP System is that it supplies air or O_2 at pressure and flow rates that are suitable for assisted *inhalation and expiration* by sensing the patient's spontaneous breathing efforts and automatically adjusting to compensate for variations in ventilatory requirements, even in the presence of airway leaks. *Importantly, this device is not intended to be a life support ventilator, because it is used only to temporarily augment spontaneous breathing.* Regardless of the system used, close attention to manufacturer guidelines is advised.

Procedure

Before initiation of this technique, the patient must be informed of the purpose of the nasal mask and cooperate.

It helps to discuss an alternative therapy should this technique not meet the patient's needs and to reassure the patient that the operator will stay with him or her until a comfort level with the mask and ventilator system is obtained. Baseline vital sign and oxygenation measurements are made.

The patient should be treated in a closely monitored setting where his or her vital signs and respiratory status are closely monitored.

Despite intervention with this system, emergent intubation and mechanical ventilation may become needed. Equipment for airway support (e.g., O_2 source, tubing, bag-valve mask, laryngoscope, suction equipment, and various airways) should be immediately available.

The BiPAP System components are assembled and connected to oxygen—generally at the same flow settings as the patient is currently receiving (generally at 10 to 15 L/min). The pressure tubing is attached to the airway pressure monitor and the pressure monitor is turned on; the main system is then turned on and a ventilation mode selected. Generally, the system will be used in the spontaneous ventilation mode (i.e., to support spontaneous ventilations) with an initial EPAP setting of 3 to 5 cm H_2O and an IPAP setting of 8 to 10 cm H_2O.

Ideally, a mask sizing gauge (Fig. 2-19) is used to choose a mask size that does not place direct pressure on the bridge of the nose, the lateral *ala nasi*, the inferior nasal septum, or the lip. The mask may be initially held in place by the operator as the patient adjusts to the ventilatory support. With the mask in place, the BiPAP System settings are modified to optimize patient ventilatory status (Fig. 2-20). As the patient becomes more comfortable, the mask is secured in place on the face using self-adhesive binding straps (Fig. 2-21). If time permits, the headgear straps may be loosely attached before placement and the mask slipped over the head as a unit; the straps are then tightened.

Figure 2-19 Use of template to size nasal mask for BiPAP System. *A*, The nasal mask sizing gauge is placed over the patient's nose. The size that comes close to, but does not touch, the nose in three locations is selected. *B*, The sites to avoid direct contact: (1) just above the junction of the nasal bone and cartilage; (2) on the sides of both nares; and (3) just below the lowest point of the nose, above the lip. *Remember to use the smallest size mask that will sufficiently cover the nasal area.* Too small a mask will produce skin discomfort or injury. Too large a mask will increase air leak. *(Courtesy of Respironics Inc., Murrysville, PA.)*

The patient is encouraged to breathe with the mouth closed. The facial mask fit should be adjusted for comfort and to minimize air leak, especially about the eyes. When the patient has accepted the mask and the clinical status has stabilized, the patient may be permitted to speak and even to eat small amounts.

Adjustments in the EPAP and IPAP settings are generally made in 2 cm H_2O increments. Near optimal settings can generally be attained within 10 minutes. Periodic blood gas measurements should be coupled with ongoing vital sign and pulse oximetry measurements. When available, a CO_2 device side-flow catheter can be placed under the nasal mask for monitoring exhaled CO_2 levels. Increases in IPAP settings generally increase tidal volume and decrease CO_2 levels, whereas

increases in EPAP generally increase functional residual capacity and increase O_2 levels. The concentration of inspired oxygen also can be modified by changing the O_2 flow rate. High levels of EPAP or IPAP can induce PEEP-related reductions in cardiac preload. Further, although the system can adjust for some air leak about the mask, higher pressures require a tighter mask fit and can increase patient discomfort.

If the patient's hypoxic drive is diminished, decreased spontaneous ventilatory effort may be noted. If this situation occurs, the BiPAP System should be changed to the "spontaneous/timed" mode, which permits spontaneous breaths but imposes a mandatory ventilation if an extended ventilatory pause is noted. When initiating this mode, the clinician generally sets the respiratory rate at 10 breaths/min. At this setting, when a breath does not occur within 6 seconds of the preceding breath, a mandatory breath is provided.

If the patient requires a brief period of hyperventilation to help coordinate ventilatory efforts, the BiPAP System can be

Figure 2-20 Control panel of BiPAP System. Note control setting for ventilation mode in lower left corner (see text for use of settings). For spontaneously breathing patients in respiratory distress, the "spontaneous" mode is generally selected and IPAP and EPAP settings selected. *(Courtesy of Respironics Inc., Murrysville, PA.)*

Figure 2-21 BiPAP System nasal mask in place on patient. *(Courtesy of Respironics Inc., Murrysville, PA.)*

set to the "timed" mode and a respiratory rate of 15 to 20 breaths/min can be initiated. In this mode, the %IPAP time must be set by the operator. A %IPAP of 30% produces an inspiratory-to-expiratory (I:E) ratio of 1:2.3, whereas a %IPAP of 40% produces an I:E ratio of 1:1.5.

Aerosolized medications can be delivered either through in-line "T-pieces" in the BiPAP System circuit or through standard mouthpiece units (see following section).

Complications

Complications from this therapy include facial irritation, abrasion, or even facial necrosis; conjunctivitis due to mask air leak; aspiration; and gastric distention. A wound care dressing on the bridge of the nose may reduce skin abrasion. [135] Nasogastric tubes have been used to relieve gastric distention, although a nasal mask system is less likely to produce gastric distention. Additionally, although the pressures used are generally low, all of the complications of positive pressure ventilation may be seen with this technique.

Conclusions

Positive pressure ventilation modalities offer the promise of averting the need for intubation and mechanical ventilation in certain groups of patients with acute respiratory failure. The procedure for instituting these therapies is relatively simple and may even have potential in the pre-hospital setting. Additionally, although best studied in adults, these therapies have been applied to pediatric patients, [137] [138] and pediatric masks are available commercially.

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Section II - Respiratory Procedures

Chapter 3 - Basic Airway Management and Decision-Making

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Although it is common knowledge that airway management is the first priority in the management of any seriously ill or injured patient, its importance is often overlooked. Failure to treat airway management as the top priority can lead to serious errors in patient care and often presages a cycle of patient deterioration and misguided therapeutic intervention. Even when its importance is fully recognized, airway management can be one of the most difficult components of a resuscitation. Because of the sheer variety of airway difficulties that are possible, even the most skilled resuscitator can find the task challenging. *Simply stated, it may be impossible to adequately manage an airway in an emergency situation in a timely enough fashion to prevent deterioration or assure a successful resuscitation.* Blood, loosened teeth, vomitus, swollen or distorted landmarks, immobility of the jaw or cervical spine, and variations in anatomy all may present formidable barriers to successful management. When airway obstruction occurs in conjunction with reflex clenching of the jaws and possible cervical spine injury, conventional airway management tools may be ineffective. Time pressures imposed by the need to avoid cerebral anoxia force difficult decisions such as whether the neck can be moved, whether paralyzing agents should be administered, or whether a surgical airway is needed. Tools must be immediately available, skills must be well-honed, and decision-making must be sharp if optimal emergency airway management is to occur. Massive neck and upper airway swelling from angiotensin-converting enzyme (ACE)-inhibitor angioedema can essentially prohibit a successful airway, even by surgical means.

Some solutions to the many obstacles faced in emergency airway management are presented in this and the following chapters. Minimally invasive approaches to airway establishment and emergency airway decision-making are described first in the current chapter with more advanced techniques covered in following chapters. Decision algorithms are presented to help assemble the pieces of the airway management puzzle into a logical framework.

DECISION-MAKING IN AIRWAY MANAGEMENT

The resuscitator must have many tools at hand to deal with the acutely compromised airway. It is important to be proficient in many different techniques and to tailor their use to the needs of the individual patient. Rescuers should practice potential scenarios before facing patients with a compromised airway in the clinical situation. Failure to do so may lead to unnecessarily aggressive management in some situations or to irreversible hypoxic injury as a result of unnecessary hesitation in others. Deciding on who requires intervention in the field, and who can be supported adequately enough until definitive hospital management can be obtained, is a formidable task for even the most skilled clinician.

Several parameters must be assessed quickly before an airway management choice can be made. The parameters to be considered are as follows:

1. Adequacy of current ventilation
2. Potential for hypoxia
3. Airway patency
4. Need for neuromuscular blockade (muscle tone, teeth clenching, severe obstructive pulmonary disease, or asthma)
5. Cervical spine stability
6. Safety of technique and skill of the operator

Consideration of these factors should guide the clinician in selecting the optimal technique. This initial choice is often straightforward. Difficulty may arise precipitously when the initial choice fails. Time then becomes critical as the risk of irreversible hypoxic injury rises. Anxiety increases and error potential compounds under these circumstances. Forethought and practice are invaluable when making these decisions.

Schemata offered in Figure 3-1 and Figure 3-2 outline a logical approach to airway management. Figure 3-1 presents the nonsurgical approaches with end points of either success or the decision to pursue surgical management. Note that a prolonged duration of hypoxia may hasten a decision in favor of surgical airway management. Figure 3-2 addresses choices once the decision has been made to manage the airway surgically. Consideration of patient condition, security of the airway approach, and the invasive nature of the procedure are factors to be weighed in the final decision.

ESTABLISHMENT OF AIRWAY PATENCY

The first concern in the management of a patient in critical condition is adequacy of the airway. Partial or complete airway obstruction must be overcome quickly. In some cases, such as an airway obstructed by a tongue, simple maneuvers will suffice. In other cases, particularly those in which multiple agents are combining to block the airway, the task will be formidable. The tongue, dentures, swollen or distorted tissues, blood, and vomitus are common obstructing agents that compromise airway patency. Clearing obstructing agents may be made more difficult by muscular activity due to reflex stimulation or patient efforts to improve oxygenation. Moreover, the neck motion required for suction and intubation must be carefully managed in the setting of potential cervical spine instability.

The wide availability of pulse oximetry monitors has greatly improved our ability to monitor oxygenation for patients at risk of airway or ventilatory compromise. [1] These monitors are accurate under most conditions, [2] and clinically subtle deterioration can be quickly recognized using the monitors. They are standard equipment in emergency departments (EDs), intensive care units, and operating rooms. Pulse oximetry is discussed in more detail in Chapter 2 .

Airway Maneuvers

Partial or complete airway obstruction resulting from *lax musculature* and *tongue occlusion* of the posterior pharynx may be overcome by a variety of maneuvers, including the neck-lift and head-tilt method, the jaw-thrust method, and the chin-lift method. In a study of 120 anesthetized patients whose airways

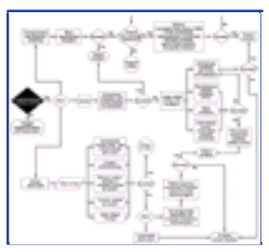

Figure 3-1 Nonsurgical airway management algorithm.

were obstructed by their tongues, Guildner concluded that the chin lift was the easiest to perform and produced the greatest airway patency of the three methods tested. [3] The chin-lift method has been shown conclusively to increase the airway diameter in pediatric patients. [4] Both the chin-lift and jaw-thrust methods have the additional advantage that neck extension is unnecessary (Fig. 3-3 , Table 3-1 and Table 3-2).[3]

Partial airway obstruction in the patient with a decreased level of consciousness is commonly due to posterior displacement of the tongue. This may be recognized readily in the presence of snoring or stridor, but an apneic patient or one who is moving minimal air may not exhibit any audible evidence of airway obstruction. Either a jaw-thrust or chin-lift maneuver should be performed on every unconscious patient. When uncertain about cervical spine status, the neck must be maintained in the neutral position. If the patient was found with a flexed or extended neck, the neck should first be restored to neutral position with gentle longitudinal traction. The chin-lift or jaw-thrust method is then performed. These maneuvers usually clear airways obstructed as a result of lax pharyngeal musculature or posterior displacement of the tongue. The neck-lift and head-tilt maneuvers described in cardiac life support courses should not be used when cervical spine injury is suspected, because the extension of the spine produced during the maneuver endangers the spinal cord.

Partial or complete airway obstruction can be the result of upper airway hemorrhage, accumulation of the patient's own secretions, foreign body aspiration, vomitus, or fractured dentition. If foreign body aspiration is suspected, the rescuer should perform a subdiaphragmatic abdominal thrust (Heimlich maneuver). [4] When stability of the spine is a concern, application of the subdiaphragmatic thrust should be limited to the supine method described for unconscious victims. Potential risks of a subdiaphragmatic thrust include stomach rupture, esophageal perforation, and mesenteric laceration, compelling the rescuer to weigh the benefits of its application. [5] [6] [7] [8] [9] [10]

Figure 3-2 Surgical airway management algorithm.

The Chin-Lift Maneuver

To perform the chin-lift maneuver, the rescuer places the tips of the fingers, volar surface superiorly, beneath the patient's chin. The jaw is lifted gently forward. The patient's mouth is opened by drawing down on the lower lip with the thumb of the same hand. [3] Mouth-to-mouth resuscitation or other means of positive-pressure ventilation is provided if the patient is not ventilating spontaneously.

The Jaw-Thrust Maneuver

The jaw-thrust maneuver is the second choice, again because neck extension is not necessary. Forward traction on the mandible is achieved by using two hands to grasp the mandibular rami and pull them forward.

The Subdiaphragmatic Abdominal Thrust

The subdiaphragmatic thrust is a method to relieve a completely obstructed airway. The technique was popularized by Dr. Henry Heimlich and is commonly referred to as the *Heimlich maneuver*.[11] The technique is most effective when a solid food bolus obstructs the larynx. Although a subject of controversy, a role for the maneuver has not been found for the resuscitation of near-drowning victims. [12]

The conscious patient with an obstructed airway exhibits increased respiratory effort, anxiety, aphonia, and, occasionally, cyanosis. In the conscious patient, the maneuver is performed with the rescuer positioned behind the upright patient. The rescuer's arms are circled about the patient's midsection with the radial side of the clenched fist placed in the epigastrium of the patient. Care is exercised to position the fist midway between the umbilicus and the xiphoid of the patient. After proper positioning, the rescuer grasps the fist with the opposite hand and delivers an inward and upward thrust to the abdomen. A successful maneuver will cause the obstructing agent to be expelled from the patient's airway by the force of air exiting the lungs. [13]

An unconscious, supine patient must be handled differently: The rescuer kneels next to the patient's pelvis facing cephalad. The palmar bases of the hands are

placed in an

Figure 3-3 Illustration of maneuvers for opening the airway. *A,* Neck lift. *B,* Chin lift. *C,* Jaw thrust. *(From Guildner CW: Resuscitation—opening the airway: A comparative study of techniques for opening an airway obstructed by the tongue. JACEP 5:588, 1976. Reproduced by permission.)*

overlapping fashion on the epigastrium at the same spot as that used in the upright patient. Inward, upward thrusts are delivered in this fashion with the same objective. [13]

Abdominal thrusts are relatively contraindicated in pregnant patients and others with protuberant abdomens. A chest compression identical to chest compressions delivered during cardiopulmonary resuscitation (CPR) may be used instead. Chest compressions may create greater peak airway pressures than a Heimlich maneuver, although this is controversial.[14] A combined (simultaneous) chest compression and subdiaphragmatic abdominal thrust may produce even higher peak airway pressures. Hence, a combined maneuver should be considered in the case of total airway obstruction that is unresponsive to simple Heimlich maneuver. [15]

Visceral injury can occur with the Heimlich maneuver.[5] [6] [7] [8] [9] [10] Excessive force or improper technique may be responsible in such cases. Nonetheless, the technique can be life-saving

TABLE 3-1 -- Subjective Evaluation of Effectiveness of Techniques on Patients Not Making Any Respiratory Effort (n = 120)

	Neck Lift		Chin Lift		Jaw Thrust	
Effectiveness	***No.***	**%**	***No.***	**%**	***No.***	**%**
Total obstruction						
Unable to ventilate	7	5.8	—	—	1	0.8
Partial obstruction						
Inadequate ventilation	8	6.7	2	1.7	2	1.7
Partial obstruction						
Adequate ventilation but with difficulty	58	48.3	9	7.5	23	19
Good airway						
Easy ventilation	47	39.2	109	90.8	94	78

From Guildner CW: Resuscitation—opening the airway: A comparative study of techniques for opening an airway obstructed by the tongue. JACEP 5:588, 1976. Reproduced by permission.

and should be used when needed. Attention to proper execution may limit complications.

Positioning

Positioning the patient who has sustained multiple trauma can be a problem. Spinal injury and airway access priorities dictate that the patient should be kept in the supine position while immobilized on a backboard. Turning the patient on the side allows upper airway hemorrhage, secretions, and vomitus to drain externally rather than to collect in the patient's mouth, which can lead to aspiration and airway obstruction. However, there is evidence suggesting that rotating the patient to the lateral decubitus position may not prevent aspiration. [16]

Guidelines for patient positioning must take into account the status of the patient's spine and the use of gravity to enable secretions to drain rather than accumulate in the airway. The following is a judicious approach to airway management in a patient *with spontaneous respiration*:

1. Initial airway maintenance accomplished by the chin-lift maneuver and the application of cervical stabilization (see Chapter 47).
2. Immobilization of the patient on a spinal backboard.
3. With the position of the neck controlled, transportation of the patient on the side to facilitate airway drainage.

Suctioning

Patient positioning and airway opening and clearing maneuvers are often inadequate to achieve the degree of airway

TABLE 3-2 -- Effectiveness of Techniques for Opening Airway in Patients with Complete Respiratory Obstruction Who Are Making Spontaneous Respiratory Effort (n = 30)

	Neck Lift		Chin Lift		Jaw Thrust	
Effectiveness (Tidal Volume)	***No.***	**%**	***No.***	**%**	***No.***	**%**
0–50 mL	13	43.3	—	—	1	3.4
50–250 mL	9	30	2	6.7	3	10
250–400 mL	6	20	7	23.3	7	23.3
>400 mL	2	6.7	21	70	19	63.3

From Guildner CW: Resuscitation—opening the airway: A comparative study of techniques for opening an airway obstructed by the tongue. JACEP 5:588, 1976. Reproduced by permission.

patency desired. Ongoing hemorrhage, vomitus, and particulate debris often require suction to clear and maintain the respiratory passage. Three basic types of suctioning tips are available (Fig. 3-4). Each is suited to different types of airway obstruction problems.

Dental tip suction is most useful for clearing particulate debris from the upper airway. Vomitus is most readily cleared with this tip because it is least likely to become obstructed itself by particulate matter. The *tonsil tip (Yankauer)* suction device is used most effectively to clear upper airway hemorrhage and secretions. Its design is

intended to prevent the obstruction of its tip by tissue and clot. The rounded tip is also less traumatic to soft tissues. The catheter tip works well for suctioning the trachea and bronchi through a tracheal tube, but it is inferior to the other tips for suctioning the oropharynx.

The dental tip device should be used during the resuscitation period and should be ready at the bedside. The dental tip allows rapid clearing of both particulate matter and hemorrhage, thereby expediting airway control. A limiting feature of many suction catheters is the diameter of the tubing. Large particulate emesis may obstruct the standard ¼-inch diameter catheter.[17] A ?-inch diameter suction catheter (Conmed Corp) has been shown to significantly decrease suction time of viscous and particulate material, potentially decreasing the risk of aspiration. [18]

Stabilization of the patient with multiple injuries may involve use of all three types of suction tips. The tonsil or dental tip should be attached to the suction source during the interval between patient evaluations because it is most likely to be the one needed on short notice. All suction apparatus must be immediately available, and everyone participating in a resuscitation should be familiar with the suction equipment and *know how to turn it on* during an emergency. In the resuscitation rooms, suction equipment should always be connected and ready to operate. Interposition of a suction trap at the base of the dental tip suction device prevents clogging of

Figure 3-4 Three types of suction tips: dental, tonsil, and catheter. *(From Clinton JE, Ruiz E: Trauma Life Support Manual. Minneapolis, MN, Hennepin County Medical Center, 1982.)*

the tubing with particulate debris. A trap that fits directly onto a tracheal tube has been described; use of this device still allows effective suctioning during intubation (Fig. 3-5).[19]

No specific contraindications to airway suctioning exist. Complications of suctioning may be avoided by anticipating problems and providing appropriate care before and during suctioning maneuvers. Nasal suction is seldom required to improve oxygenation (except in infants), because most adult airway obstruction occurs in the mouth and oropharynx. Vigorous nasal suction can induce epistaxis and further complicate an already difficult airway. Epistaxis may be avoided by limiting the force applied during suctioning. Vasoconstrictor drops or spray constrict the nasal mucosa and may reduce the injury potential in patients who require repeated nasopharyngeal suctioning.

Prolonged suctioning should be avoided because it may lead to significant hypoxia, especially in children. *Suctioning should not exceed 15-second intervals*, and the provision of supplemental oxygen before and after suctioning should be routine (see also Chapter 7). Naigow and Powasner found that suctioning consistently induced hypoxia in dogs and that it was best avoided by hyperventilation with high-concentration oxygen before and after suctioning. [20]

Figure 3-5 The Ruben suction booster, which is designed to allow high-capacity suctioning through the endotracheal tube during intubation. Schematic diagram: *A,* Tracheal tube connection. *B,* Connection to suction. *C,* Introducer opening in the closed position. *D,* Opening that is kept closed when suction is needed through the tracheal tube. Note: All suction should be done under direct vision. *(From Ruben H, Hansen E, MacNaughton FI: High-capacity suction technique. Anaesthesia 34:349, 1979. Reproduced by permission.)*

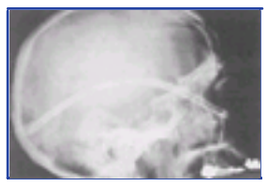

Figure 3-6 Intracranial intubation. Lateral skull x-ray showing nasogastric tube placed into brain through skull fracture. *(From Clinton JE, Ruiz E: Trauma Life Support Manual. Minneapolis, MN, Hennepin County Medical Center, 1982.)*

Extreme care should be exercised when a basilar skull or facial fracture is suspected, because communication between nasal and intracranial cavities may exist and allow the inadvertent placement of nasal suction tubes in the cranial cavity (Fig. 3-6).

Generally, it is best to perform suctioning *under direct visual inspection* or with the aid of the laryngoscope. Forcing a suction tip blindly into the posterior pharynx can injure tissue or convert a partial obstruction to a complete obstruction.

Artificial Airways

Indications and Contraindications

Once the airway has been opened through various maneuvers and suctioning, the patient may require further temporary support to maintain airway patency. The semiconscious patient who is breathing with an adequate rate and tidal volume at the time of the chin-lift maneuver may develop hypoxia because of recurrent obstruction if the maneuver is discontinued. Oxygen supplementation and an artificial airway may be all the support that is necessary. The use of an artificial airway also allows more efficient use of rescuer skills and relief from fatigue that is caused by the continuous application of chin-lift or jaw-thrust maneuvers.

Positive-pressure ventilation with a bag-valve-mask (BVM) device may be necessary to bolster the patient's inadequate ventilatory effort or to provide total ventilation in cases of apnea. By maintaining airway patency, artificial airways may facilitate both spontaneous and bag-mask ventilation.

Airway Placement Technique

The simplest artificial airways are the oropharyngeal and nasopharyngeal airways (Fig. 3-7). Both are intended to prevent the tongue from obstructing the airway by falling back against the posterior pharyngeal wall. The oral airway also may prevent teeth clenching. The oropharyngeal airway may be inserted by either of two procedures. One approach is to insert the airway in an inverted position along the patient's hard palate. When it is well into the patient's mouth, the airway is rotated 180 degrees and advanced to its final position along the patient's tongue, with the distal end of the airway lying in the hypopharynx. A second approach involves performance of a jaw-thrust maneuver, either manually or with a tongue blade, and the simple advancement of the airway into the mouth to its final position. No rotation is performed when the airway is placed in this manner. Once inserted, the oral airway may have to be taped in place to prevent expulsion by the patient's tongue. The oropharyngeal airway will keep the mouth partially open and prevent clenching of the teeth, which can obstruct an orogastric or orotracheal tube.

The nasopharyngeal airway is likely an overlooked technique to assure a patent airway in an otherwise stable patient. The soft tube is placed by gently advancing the airway into a nostril, directing the tip along the floor of the nose toward the nasopharynx. When in final position, the flared external end of the airway should rest at the nasal orifice. Either of these two airways provides airway patency similar to that in a correctly performed chin-lift maneuver, but the nasal airway may be better

tolerated by the semiconscious patient. Since the nasal tube is about the same size as an endotracheal tube, some clinicians place the nasopharyngeal airway to gently dilate the nasal passages before attempting blind nasotrachael intubation, but this technique is neither standardized nor well studied (Fig. 3-8).

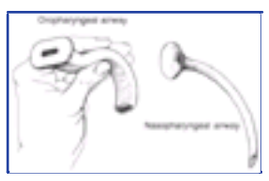

Figure 3-7 Simple artificial airways: oropharyngeal and nasopharyngeal. *(From Clinton JE, Ruiz E: Trauma Life Support Manual. Minneapolis, MN, Hennepin County Medical Center, 1982.)*

Figure 3-8 Some clinicians place a nasopharyngeal tube to dilate the nasal passage prior to blind nasotrachael intubation.

Complications

Few complications are encountered with the use of oral or nasal airways. The oropharyngeal airway may cause airway obstruction if during its placement the tongue is pushed against the posterior pharyngeal wall. Care in placement will prevent this. In the semiconscious patient with intact reflexes, the gag reflex may stimulate retching and emesis. If gagging is a persistent problem, the airway should be removed and a nasal airway or tracheal intubation should be considered. If the patient with airway compromise is comatose and lacks a gag reflex, the oropharyngeal airway *should not be used as a definitive airway*; tracheal intubation should be used instead.

The nasopharyngeal airway may offer an advantage over the oropharyngeal airway in that the nasopharyngeal airway is less likely to induce gagging. When placing a nasopharyngeal airway, care must be exercised not to induce epistaxis, and extreme caution is indicated in patients with a suspected basilar skull fracture or facial injury. All potentially unstable patients with oral or nasal pharyngeal airways should be observed constantly, because these devices are temporary measures and cannot substitute for tracheal intubation.

BAG-VALVE-MASK VENTILATION

Indications and Contraindications

Although the BVM method of ventilation appears to be simple and effective, it can be difficult to perform correctly. BVM ventilation should be used by experienced individuals who are able to ensure a tight mask seal in situations requiring positive-pressure ventilation. The BVM often is used with an oropharyngeal or nasopharyngeal airway in place.[13] Predictors of difficult BVM ventilation are shown in Table 3-3 .[21]

Inexperience is a relative contraindication to the use of a BVM. A rescuer who is not skilled with the BVM will achieve much better ventilation with mouth-to-mouth or mouth-to-mask breathing than with a BVM. Concern regarding transmission of infectious diseases has reduced the willingness of the lay public and health professionals to perform mouth-to-mouth ventilations.[22] However, only 12 cases of infectious

TABLE 3-3 -- Risk Factors for Difficult Mask Ventilation [21]

Presence of beard
Body mass index >26 kg/m^2
Lack of teeth
Age >55 yr
History of snoring

disease transmission from mouth-to-mouth or mouth-to-tube ventilation have been confirmed in the last 30 years, making CPR ventilation a relatively safe procedure (Table 3-4).[23]

Although BVM ventilation may provide excellent respiratory support in the anesthetized, paralyzed patient in the operating room, the device frequently is of marginal value during CPR, during an ambulance run, or in the combative patient. The three major problems encountered with BVM ventilation are inadequate tidal volumes, inadequate oxygen delivery, and gastric distention. A tight mask seal is mandatory to prevent loss of tidal volume and to ensure oxygen delivery during ventilation. Achieving a tight mask seal requires excellent procedural technique, and is much easier to achieve in the controlled setting with an anesthetized, paralyzed patient. Another hazard of BVM ventilation occurs when vomitus, blood, or other debris is present in the mouth or pharynx. The foreign material may be insufflated down the trachea if it is not cleared before ventilation.

Ventilation Technique

Achieving adequate tidal volume with BVM ventilation requires a tight mask seal and appropriate compression of the bag. The ideal tidal volume for BVM ventilation is 5 to 6 mL/kg, or approximately 500 mL for an adult.[24] A variety of mask configurations are available to facilitate a tight seal, but none substitutes for the practiced skill of the rescuer. For the single rescuer, only one hand can be used to achieve the seal because the other must squeeze the bag. The rescuer must apply pressure anteriorly while simultaneously lifting the jaw forward. The thumb and index finger provide anterior pressure while the fifth and fourth fingers lift the jaw. In pediatric patients the E-C clamp technique is used: The thumb and index finger form a "C" while providing anterior pressure over the mask, while the third, fourth, and fifth fingers form an "E" to lift the jaw (Fig. 3-9). *Dentures generally should be left in place to help ensure a better seal with the mask.*

It has been suggested that effective BVM ventilation during CPR requires two hands and, therefore, two rescuers. [25] We suggest using the two-rescuer technique (see Fig. 3-9*B*)

TABLE 3-4 -- Reported Infections (Since 1965) Acquired by Mouth-to-Mouth or Mouth-to-Tube Ventilation During CPR [23]

Organism	Cases
Mycobacterium tuberculosis	1
Neisseria meningitides	4
Shigella sonnei	1
Salmonella infantis	1
Helicobacter pylori	1
Herpes simplex virus	3
Neisseria gonorrhoeae	1

Figure 3-9 Bag-valve-mask ventilation is very difficult for one person to do *(A),* and it frequently fails to deliver adequate tidal volumes, especially during cardiopulmonary resuscitation. With the two-person method *(B),* one person uses both hands to hold the mask firmly against the face and extend the head. The other person uses both hands to squeeze the bag. Dentures are generally left in place to help provide a better-fitting mask. *(From Jesudian MC, Harrison RR, Keenan RL, Maull KI: Bag-valve-mask ventilation: Two rescuers are better than one. Crit Care Med 13:122, 1985.)*

whenever it is practical. The presence on the BVM device of a pop-off valve may further frustrate ventilation efforts in the patient with reduced compliance.

All BVM devices should be attached to a supplemental oxygen source (with a flow rate of 15 L/min) to avoid hypoxia. A significant problem with the BVM method is the low percentage of oxygen achieved with some reservoirs. The amount of delivered oxygen is dependent on the ventilatory rate, the volumes delivered during each breath, the oxygen flow rate into the ventilating bag, the filling time for reservoir bags, and the type of reservoir used. The commonly used corrugated tube reservoir is dependent on ventilatory technique and does not alert the rescuer to changes in oxygen flow. A 2.5-L bag reservoir and a demand valve are the preferred supplementation technique during BVM ventilation.[26]

Pediatric BVM devices should have a minimum volume of 450 mL. Pediatric and larger bags may be used for ventilation of infants with the proper mask size, but care should be taken to administer only the volume necessary to effectively ventilate the infant. Pop-off valves should be avoided because airway pressure under emergency conditions may often exceed the pressure of the valve.[13] BVM ventilation may be the preferred method of prehospital airway support in children under the age of 12 years. Although the final outcome of out-of-hospital pediatric resuscitation is generally poor, Gausche et al. [27] reported that neurologic outcome and ultimate

survival rates of prehospital pediatric resuscitations by Emergency Medical Service (EMS) providers with BVM ventilation were as good as with tracheal intubation.

Complications

Hypoventilation often occurs because of improper technique including poor sealing of the mask, failure to achieve airway patency, and delivery of insufficient tidal volume. Proper training is necessary to avoid these errors. Gastric distention can occur if air is insufflated down the esophagus, increasing the risk of regurgitation and aspiration. When assistance is available, the application of firm posterior pressure on the cricoid ring helps reduce gastric inflation during BVM ventilation. [28] [29] The technique must be used carefully in infants, whose airway is more pliable and subject to obstruction with excessive cricoid pressure. Even with proper BVM technique, aspiration can occur. The rescuer must be vigilant to recognize complications early and take corrective action. Under most situations, however, some gastric dilation will occur even with strict attention to detail. Minor gastric distention should not be considered substandard under the setting of prolonged BVM ventilation.

INTERMEDIATE AIRWAYS

Intermediate airways are those interventions that go beyond the maintenance of a patent airway. They represent a midpoint between airway establishment and true airway control by maneuvers such as tracheal intubation and tracheotomy. Many devices are available and no one technique is universally used. No agreed-upon standard of care has been promulgated for these devices, and their use varies by location and clinician experience. The devices described in this section allow ventilation across the larynx but do not involve complete airway control. These devices include the esophageal obturator airway (EOA), the esophageal gastric tube airway (EGTA), the laryngeal mask airway (LMA), and the esophageal-tracheal Combitube (ETC) airway (Sheridan Catheter Corp., Argyle, NY). Two are designed to occlude only the esophagus (EOA and EGTA), one (LMA) seals the larynx at the hypopharynx level, and one (ETC) offers the versatility of use whether placed into the esophagus or the trachea. There are several newer supraglottic ventilation devices, including the cuffed oropharyngeal airway (COPA), the airway management device (AMD), and the laryngeal tube (LT). Each is designed for use in the unconscious patient who requires positive-pressure ventilation. The esophageal cuff or seal built into these devices reduces gastric content aspiration. The EOA and EGTA have a relatively high complication rate and therefore are becoming less common as other ventilation devices are becoming available.

Esophageal Obturator Airway and Esophageal Gastric Tube Airway

The EOA and the EGTA maintain airway patency in ways similar to the oral and nasal airways, but they also protect the airway by occluding the esophagus to reduce gastric distention

and regurgitation. The face mask permits use of these airways as positive-pressure ventilating devices. Air insufflated through the airway traverses the upper airway before crossing the larynx and entering the trachea. Ventilation from the EOA exits the airway through numerous ports in its hypopharyngeal portion (Fig. 3-10*A* and *B*). Ventilation from the EGTA is identical to mask ventilation, with the addition of esophageal occlusion. A port is available on the EGTA to vent the stomach. The attractiveness of the EOA and the EGTA for use in the apneic patient stems from their retention of much of the simplicity of the artificial airway with the addition of an important feature of more complicated airways—some protection against regurgitation and reduction of gastric distention.

Indications and Contraindications

The EOA and EGTA are indicated when positive-pressure ventilation is needed but neither BVM ventilation nor tracheal intubation can be performed safely, effectively, and rapidly. The EOA can be placed more quickly than a tracheal tube, [30] [31] and there are fewer intubation failures with the EOA than the tracheal tube. [32] The EOA and EGTA cannot be used in the awake patient with an intact gag reflex, and they are not available in pediatric sizes. They are relatively contraindicated in the presence of active oropharyngeal bleeding, suspected esophageal injury, caustic ingestion, or a history of esophageal disease. As a precaution against pressure-related complications, it is recommended that these devices be left in place for no longer than 2 hours. It must be recognized that the EOA and the EGTA are *temporary* forms of airway control, most suitable for use in out-of-hospital settings.

Placement of EOA/EGTA

The head is in the neutral position during placement of the EOA and the EGTA. Neck motion is unnecessary. The rescuer

Figure 3-10 *A,* Esophageal obturator airway. Correct placement of the esophageal airway with the cuff inflated in the esophagus caudad to the bifurcation of the trachea. *(From Clinton JE, Ruiz E: Trauma Life Support Manual. Minneapolis, MN, Hennepin County Medical Center, 1982.) B,* Esophageal obturator airway. *(From Jacobs LM: The importance of airway management in trauma. J Natl Med Assoc 80:873, 1988.)*

grasps and pulls the jaw forward. At this point, the rescuer inserts the assembled airway with the mask attached. The obturator tip is directed into the patient's posterior pharynx with gentle, steady pressure. The obturator is advanced down the esophagus until the mask rests flush against the face of the patient. Figure 3-10*A* illustrates the correct position at placement. The cuff should lie in the esophagus just distal to the carina of the trachea. The rescuer postpones inflation of the balloon until proper position is confirmed. The patient is ventilated with a tight mask seal on the face, and the lungs are auscultated. For effective ventilation, the mask seal must be tight. Breath sounds should be audible bilaterally. Unilateral breath sounds or failure of auscultation should lead the rescuer to reassess the airway placement. Pneumothorax or hemothorax may explain unilateral sounds, as may inadvertent main stem bronchus intubation. Tracheal intubation will result in the absence of breath sounds. The possibility of bronchial or tracheal intubation requires removal and replacement of the airway. Once satisfactorily placed, the esophageal balloon is inflated with 20 to 25 mL of air.

Complications

The complications of the EOA and EGTA are well reported. Hypercarbia from inadequate ventilation occurs more commonly with EOA than tracheal intubation. [33] [34] Unrecognized tracheal intubation may occur in 2.9% to 5% of patients with up to a 100% mortality due to airway occlusion. [31] [35] Esophageal injury may also occur, ranging from small lacerations in 8.5% of patients [32] to esophageal rupture.[36] [37] [38]

Under ideal circumstances, tracheal intubation should be performed before removal of the EOA, because vomiting often occurs following deflation of the balloon and EOA removal. If the EOA cuff has been overinflated, it may partially occlude the trachea and make intubation difficult. In such

cases, the balloon is partially deflated to facilitate tracheal intubation.

The Laryngeal-Mask Airway

The LMA (Intavent International SA, Henley-on-Thames, England) functions intermediately between an oropharyngeal airway and an endotracheal tube. It was developed for use in the operating room as an alternative for endotracheal intubation, but it has also been recommended for use in difficult intubations and for rescue ventilation in emergency resuscitations. [39] [40] The LMA is considered a temporary adjunct until tracheal intubation can be established. [39] Since the introduction of the original LMA, several variations have become available. The original LMA, which is available in both reusable (LMA Classic) and disposable (LMA Unique) varieties, consists of a tube fitted with an oval mask, rimmed with an inflatable cuff (Fig. 3-11*A*). The mask is intended to reside

Figure 3-11 Laryngeal mask airway (LMA). *A*, inflated LMA outside of the body. *B*, LMA in place with cuff overlying larynx. *C*, LMA placement into the pharynx. *D*, LMA placement using the index finger as a guide. *(From Basket PJF, Brain AIJ: The use of the LMA. In Basket PJF, Brain AIJ (eds): Cardiopulmonary Resuscitation Handbook. London, Intavent Research, 1994.)*

in the hypopharynx rather than on the face. It is inserted digitally until its tip meets resistance in the upper esophageal sphincter. The cuff is then inflated, forming a seal around the glottic opening (see Fig. 3-11*B*). The result is a relatively secure airway. However, it cannot be considered to protect against gastric regurgitation. Leakage of the hypopharyngeal mask allows aspiration of emesis and gastric distention may occur with misplacement.

One variation, the Proseal LMA, has a parallel drainage tube attached to the airway tube that is designed to reduce gastric insufflation and allow gastric drainage by a nasogastric tube, potentially decreasing the risk of aspiration. [41] Another variation, the intubating LMA (LMA Fastrach) is designed to facilitate blind tracheal intubation while allowing continuous positive-pressure ventilation. [42] The LMA and the intubating LMA (ILMA) have become part of the standard airway armamentarium for many rescuers, both in the hospital and prehospital setting. [43] Although the standard LMA can

act as a conduit for tracheal intubation, there are several advantages to the ILMA. [44] Insertion of the ILMA is easier than the standard LMA when the head and neck must be maintained in the neutral position, [45] and the ILMA allows for passage of a larger tracheal tube (up to 8-0). [40] Thus far, the Proseal LMA's role in the setting of difficult intubation or rescue ventilation has not been studied, and it is mainly used in the operative environment.

Indications and Contraindications

The LMA is indicated for patients requiring an airway who cannot be endotracheally intubated or cannot be ventilated with a BVM. The most frequently cited example is a patient whose anatomy prevents visualization of the larynx. Contraindications include the inability to open the patient's mouth, vomiting, or the need for high pulmonary inflation pressures. [40]

Placement of LMA

After selecting the appropriate size (Table 3-5) [40] the LMA is checked for possible air leaks by inflating and deflating the cuff. If the patient has a gag reflex, deep oropharyngeal topical anesthesia or conscious sedation must be administered. Although the sniffing position is preferred for the standard LMA, placement is 95% successful when the neck and head are held in the neutral position, as would be necessary with cervical spine immobilization. [45] The posterior surface of the mask is lubricated and the mask is oriented so its opening faces the tongue. With the index finger of the dominant hand placed on the proximal aspect of the mask, the mask is inserted into the mouth, firmly against the hard palate (see Fig. 3-11*C*). The index finger (or thumb) may also be used as a guide during advancement (see Fig. 3-11*D*). With one smooth motion, the mask is advanced until resistance is encountered. With the tip of the mask thus seated in the upper esophageal sphincter, the cuff is inflated. The lungs are auscultated to confirm correct placement.

Preparation for ILMA placement is similar to LMA placement. The preferred patient position for ILMA is with the head and neck in a neutral position. [46] The posterior surface of the mask is lubricated, and the mask is oriented so the opening is facing the tongue. While holding the ILMA by the handle, slide the mask along the hard palate and then downward until resistance is felt at the base of the hypopharynx. The cuff is then inflated, and position is confirmed by auscultation of the lungs. [40]

After successful placement of the LMA, several methods are available to achieve subsequent endotracheal intubation. The

TABLE 3-5 -- Laryngeal-Mask Airway (LMA), Disposable LMA, and Intubating LMA Size Recommendation Based on Weight [40]

Weight	LMA	Disposable LMA	ILMA
<5 kg	1	—	—
5–10 kg	1.5	—	—
10–20 kg	2	—	—
20–30 kg	2.5	—	—
30–50 kg	3	3	3
50–70 kg	4	4	4
70–100 kg	5	5	5
>100 kg	6	—	—

Note that only the standard LMA is available for patients <30 kg.

first method is simply to pass an appropriately sized endotracheal tube down the lumen of the LMA, rotate the tube 90 degrees so that the tip easily passes through the fenestrations, and advance it through the larynx to the trachea (Fig. 3-12). Success rates vary widely from 0% to 93% depending on operator experience and technique. [47] [48] [49] The second method involves the use of a tracheal tube exchanger or gum elastic bougie passed blindly down the lumen of the LMA and into the trachea. The LMA is then removed and an endotracheal tube is passed over the tracheal tube exchanger. [50] [51] [52] The more flexible bougie may be preferable, as the tube exchanger has been shown to pass into the esophagus in up to 70% of attempts. [53] The third and most dependable method of intubation with an LMA in place is via a fiberoptic scope. A lubricated, appropriately sized endotracheal tube is mounted over a fiberoptic scope, and this combination is advanced through the lumen of the LMA out through the mask and through the larynx. The scope is then removed, but the LMA may be left in place with the cuff deflated. If the LMA must be removed after a tracheal tube has been successfully placed through it, pass a tracheal tube exchanger down the tube, remove the tracheal tube/LMA combination, and replace it with a tracheal tube. After any manipulation of the LMA or with any tracheal tube placement, ventilation of the lungs must be confirmed.

Blind tracheal intubation via an ILMA has a success rate of greater than 90%. [42] [54] [55] [56] [57] Either the silicone ETTs which accompany the ILMA or a standard ETT may be used for tracheal intubation. First, the tube should be thoroughly lubricated and then advanced into the ILMA. If the ETT advances past the 15 cm mark with minimal resistance, it likely is entering the trachea. It should then be advanced to appropriate depth and the cuff should be inflated. Resistance may be encountered at 15 cm due to inappropriate seating or size of the ILMA. If the ETT cannot be passed blindly after repositioning the ILMA, a fiberoptic scope may be passed to guide tracheal intubation. Confirmation of tube position should be

Figure 3-12 Blind passage of an endotracheal tube through the laryngeal mask airway. *(Courtesy of the Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

standard procedure. Following tracheal intubation, the ILMA may be removed by deflating the ILMA cuff and withdrawing the ILMA over the ETT. A flexible rod at least 20 cm long should be used to stabilize the ETT during ILMA removal. [58]

Complications

Although the LMA works well in most cases, this airway has several significant drawbacks. Aspiration is always a possibility, because the cuff does not provide a watertight seal. Laryngospasm can occur if adequate anesthesia is not achieved. A significant air leak around the cuff may occur when high airway pressures exist, leading to poor ventilation. Finally, while success rates in the operating room range from 94% to 98%, [59] success rates in difficult emergency airway management vary between 76% and 100% depending on the background and training of the clinician. [60] [61]

Conclusion

The LMA is a blindly placed intermediate airway that should be considered in patients who require establishment of an emergency airway but cannot receive endotracheal intubation. The technique is quick and simple, and requires a minimum amount of training. [61] [62] Perhaps the best feature of the LMA and ILMA is the ability to blindly transition from an intermediate airway to definitive airway control, providing an invaluable resource in the setting of the difficult intubation.

The Esophageal-Tracheal Combitube

The ETC is an intermediate airway device that can be placed blindly and rapidly. [63] It allows for effective ventilation and oxygenation when placed in either the esophagus or the trachea. The ETC is successfully placed in 72% to 95% of patients by prehospital providers. [64] [65] [66] The device has two lumina running parallel to each other. One is perforated at the level of the pharynx and occluded at the distal end, similar to the EOA. The second lumen is open at the distal end, resembling an endotracheal tube. The device has two balloons: a proximal pharyngeal balloon that occludes the oropharynx by filling the space between the base of the tongue and the soft palate and a smaller, distal cuff that serves as a seal in either the esophagus or trachea (Fig. 3-13).[67] [68] The Combitube has compared favorably with the endotracheal tube with respect to ventilation and oxygenation in cardiac arrest situations. [63] [69] While it may provide some degree of protection from aspiration, [70] complete airway control should not be assumed.

Indications and Contraindications

The ETC is superior to the EOA because no face mask seal is necessary and the risk of complications is lower. [67] The ETC should be used in an unresponsive person who requires an airway when tracheal intubation is not successful or practical. The ideal situation for ETC use is in the prehospital setting by a rescuer not skilled in tracheal intubation. The ETC should not be used in patients with an intact gag reflex and is not recommended in patients younger than 16 years or shorter than 5 feet tall. It is contraindicated in suspected caustic poisonings or proximal esophageal disorders.

Placement of ETC

The device is held in the dominant hand and gently advanced caudally into the pharynx while the nondominant hand grasps the tongue and jaw between the thumb and index finger. The tube is passed blindly along the tongue to a depth that

Figure 3-13 Esophageal-tracheal Combitube (ETC) airway. *(From Frass M, Frenzer R, Zdrahal F, et al: The esophageal tracheal Combitube: Preliminary results with a new airway for cardiopulmonary resuscitation. Ann Emerg Med 16:770, 1987.)*

positions the printed rings on the proximal end of the tube between the patient's teeth or alveolar ridge. [71] If resistance is met in the hypopharynx, the tube should be removed and bent between the balloons for several seconds to facilitate insertion. [71] After insertion, the pharyngeal balloon is filled with 100 mL of air, and the distal cuff is subsequently filled with 10 to 15 mL of air. The large pharyngeal balloon serves to securely seat the ETC in the oropharynx and to create a closed system in the case of esophageal placement. Because up to 91% of placements are esophageal, [66] ventilation is begun through the longer (blue plastic) connector associated with the esophageal lumen. Chest rise and good breath sounds without gastric insufflation confirm effective placement in the esophagus. However, gastric insufflation without breath sounds and chest rise indicate a tracheal positioning of the tube and require changing the ventilation to the shorter (clear plastic) tracheal lumen. Auscultation of breath sounds over the lateral lung fields confirms endotracheal placement of the Combitube. If the tube is in the esophageal position, gastric suctioning can be accomplished by passing a catheter through the open lumen into the stomach while the patient is being ventilated via the other port. [66]

An alternative method to identify position is to attach an aspirating device (see Chapter 4) to the tracheal or clear plastic shorter tube. The inability to easily aspirate air confirms esophageal placement, necessitating ventilation via the longer blue esophageal tube. In the patient with ventilatory effort, CO_2 detector devices also may be useful.

A patient who has been successfully resuscitated with an ETC positioned in the esophagus should ultimately receive a definitive airway. The steps required to place a tracheal tube in this setting are detailed in Chapter 4 but consist generally of deflating the large pharyngeal balloon and, with the distal balloon still inflated, intubating around the ETC.

Complications

Inappropriate balloon inflation and incorrect ETC placement can lead to air leaks during ventilation. The most common placement error is an improper insertion angle. A more caudal, longitudinal direction is recommended, as opposed to an anteroposterior direction of insertion. Another caveat is

that the ETC must be maintained in the true midline position during insertion to avoid blind pockets in the supraglottic area, which prevent passage of the tube. [66] Attention to the ring markings on the tube at the level of the incisors ensures proper positioning of the tube. One must remember to first inflate the oropharyngeal balloon before inflating the distal balloon. Although unlikely, esophageal injury is theoretically possible with overinflation of the distal balloon.

Cuffed Oropharyngeal Airway

The cuffed oropharyngeal airway (COPA) is a standard oropharyngeal airway with a cuff at the distal end and a standard 15 mm connector at the proximal end. [72] When inflated, the cuff is seated in the hypopharynx. When used with a BVM, the COPA allows for greater tidal volumes than either a standard oropharyngeal airway or a BVM alone.[73] The role of the COPA for resuscitation has not been fully defined. Rees has shown that basic life support providers can use the COPA effectively with minimal instruction. [74] [75] The COPA is indicated for the unresponsive patient who may need ventilation assistance when no other airway is available or practical. Relative contraindications include vomiting and an intact gag reflex.

The COPA is inserted in a similar fashion to a standard OP airway. With the cuff deflated, it is placed in an inverted fashion along the hard palate. It is then rotated 180 degrees while advancing toward the base of the tongue. The cuff is then inflated. The COPA may be secured with either tape or an accompanying strap.

Complications of the COPA include airway obstruction and minor trauma to the pharynx. [72] [73] The COPA should not be considered a definitive airway.

Laryngeal Tube and Airway Management Device

The laryngeal tube (LT) and airway management device (AMD) are separate intermediate airways with a similar design. Both are single-lumen tubes with oropharyngeal and esophageal cuffs and a ventilation port between the cuffs. [76] The AMD has the capability of gastric decompression by partially deflating the esophageal cuff, then passing a nasogastric tube through the lumen. Ventilation and gastric decompression cannot occur simultaneously. [77] The LT has a single port that inflates both cuffs.

The LT and AMD are currently under investigation to determine their clinical efficacy and practicality. Early studies show mixed results. Fifty clinicians and nurses were able to correctly place an LT and adequately ventilate a mannequin in 95% of attempts. [78] However, in an observational study of the AMD, experienced anesthesiologists were unable to establish an airway in 10/104 patients in whom an AMD was attempted. [77]

The LT and AMD have similar indications as the LMA and Combitude. They are indicated in the unresponsive patient who requires airway control but in whom tracheal intubation is difficult or impractical. They are contraindicated in awake patients, caustic ingestions, and in patients with known proximal esophageal disease.

The LT and AMD both are inserted blindly, and both can be inserted with the head and neck in a neutral position. The tube is advanced until the indicator marks are at teeth level, and the cuffs are inflated. The AMD oropharyngeal cuff should be inflated prior to the esophageal cuff. Placement should be confirmed by routine methods.

Complications of the AMD may include airway obstruction, glossal venous congestion, coughing, and minor oropharyngeal trauma. [77] [79] LT complications are predicted to be similar, but have not been described in the medical literature. Neither the AMD nor the LT should be considered a definitive airway, and aspiration is a potential complication. The AMD and LT should be left in place until the trachea is intubated.

SPECIAL CONSIDERATIONS

Cardiac Arrest

Mouth-to-mouth and BVM ventilation may suffice for out-of-hospital care with short transport times or for the initial few minutes of ventilation in cardiac arrest. However, optimal BVM ventilation during CPR is impossible. Mouth-to-mouth and BVM ventilation are adequate and effective in the anesthetized or paralyzed patient with an empty stomach in the absence of chest compression, but they are inadequate for prolonged ventilation in the patient in cardiac arrest. Proper BVM ventilation is probably harder to master than tracheal intubation, and prolonged attempts during CPR usually only distend the stomach and give the uninitiated a false sense of security. While BVM ventilation will suffice for the first few minutes of a resuscitation, patients in cardiac arrest should be orotracheally intubated when logistically feasible. No specific timeline or standard can be advanced because of the varying clinical circumstances surrounding cardiac arrest. If orotracheal intubation is impossible due to lack of skill or a difficult airway, an LMA or ETC may be placed until definitive airway control can be established. Most cardiopulmonary arrests are not associated with cervical spine injury. When there is suspicion of cervical injury, the following precautions should be followed.

Potential Cervical Spine Injury

Any patient who has sustained a significant injury has the potential for cervical spine injury. Approximately 1.5% to 3.0% of initial survivors of all types of major trauma seen in EDs have significant cervical spine injury. [80] It is interesting to note that this prevalence is not increased in the setting of significant head injury. [81] Also, gunshot wounds to the head that do not involve direct neck penetration are not considered high risk for concomitant cervical spine injury. [82] [83]

In patients with multiple injuries, the possibility of cervical spine injury warrants caution when considering tracheal intubation involving the use of the laryngoscope. If the patient needs tracheal intubation, an assistant should provide in-line manual stabilization of the neck while minimizing axial traction or lateral neck motion. When done cautiously, oral intubation of the unconscious spinal cord-injured patient may be as safe as other techniques, including intubation with fiberoptic guidance. [84] Multiple studies in patients with a cervical spine injury have not demonstrated neurologic deficits following orotracheal intubation. [85] [86] [87]

Note that mouth-to-mouth and BVM ventilation frequently require some degree of neck extension to open the airway. A cadaver study demonstrated increased neck motion with BVM ventilation when compared to various intubation

techniques, including oral intubation, lighted stylet-guided oral intubation, and nasotracheal intubation. [88] BVM techniques may, therefore, be less desirable than the other methods of ventilating the patient with a potential cervical spine injury. However, this may be the clinician's only immediate option.

Potential Epiglottitis/Supraglottitis

Epiglottitis is often considered a disease of children between the ages of 2 and 8 years, but it is being recognized in adults with increasing frequency. [89] [90] Currently epiglottitis is quite rare in children in the United States because of the use of immunizations against *Haemophilus influenzae*, the most common etiology. Adults are currently at greater risk for epiglottitis than are children in this country.

The classic scenario is that of an adult or child sitting upright, drooling, or spitting up oral secretions rather than swallowing. The voice may sound muffled. There is a history of a relatively abrupt onset of a sore throat that rapidly becomes more painful. Children commonly present with a high temperature, but adults usually are only mildly febrile. The disease is especially treacherous in children because of their small airways and their tendency to panic when an oral examination or insertion of an intravenous (IV) line is attempted. Small children are most calm when allowed to sit on a parent's lap. An oxygen mask with a flow rate of 10 L/min should be held by the parent several centimeters from the child's face. If the child is using accessory muscles to breathe, every attempt should be made to keep the child calm. If a lateral radiograph of the neck taken on inspiration can be obtained without disturbing the child, it will often establish the diagnosis. On radiography, the inflamed epiglottis often appears thickened and rounded. The hypopharynx is dilated above the obstruction.

In cases of respiratory compromise, an epiglottitis protocol should be implemented rapidly. The otolaryngologist, anesthesiologist, and respiratory therapist must be notified. A preestablished protocol can save many minutes of time otherwise spent trying to reach all of the personnel needed to manage this critical emergency. [91] When a child is suspected of having epiglottitis based on history and clinical presentation, the safest course of action to establish the airway should be pursued. The emergency clinician should accompany the child at all times and be prepared to intervene until the airway is secure. Otolaryngologist notification should be included in the protocol because a tracheostomy may be necessary. [91] [92] When operating room space or personnel are not available immediately, ED personnel must be prepared to manage the airway.

If the child lapses into a coma or stops making ventilatory efforts, the first step is to attempt to force oxygen past the obstruction by using mouth-to-mouth respiration or a BVM apparatus. Because the obstruction is edematous supraglottic tissue and epiglottis, positive-pressure ventilation often can displace the edema enough to allow adequate ventilation. Moving the patient to a prone position may cause the epiglottis to fall forward, causing decreased airway resistance and improved ability to ventilate the patient. [93] If this is unsuccessful, the emergency clinician should attempt oral intubation. However, a normal larynx will not be visible because of the edema. The operator should attempt to pass an endotracheal tube through the slit-like opening that remains for the supraglottic airway. An assistant can compress the chest to force bubbles through the airway, as a means of locating the airway. [94] The assistant can also palpate the larynx and the trachea to detect the tube's entry into the trachea. If orotracheal intubation fails, the intubator should go directly to transtracheal needle ventilation (TTNV) (see Chapter 6). The obstruction of epiglottitis is mainly inspiratory, so there should be no difficulty with chest hyperinflation with intermittent TTNV. This method should ease subsequent orotracheal intubation, because the path of the airway should be readily apparent as exhaled gases pass through it.

In general, cricothyrotomy and tracheostomy are not feasible in the emergency setting for small children with epiglottitis because of technical and procedural complexities. It is recommended that all children with acute epiglottitis receive tracheal intubation. [95] If the child is not in distress, an IV line can be established before intubation for appropriate drug administration, although some operators prefer to delay IV placement until after inhalation anesthesia.

Adults and cooperative older children with suspected epiglottitis can be examined directly. It is good practice to visualize the epiglottis and the vocal cords of the stable older patient with laryngeal tenderness who is complaining of a severe sore throat or difficulty swallowing. A mirror, fiberoptic scope, or a right-angle scope can be used to do this (see Chapter 65). In epiglottitis, the pharynx and tonsils usually do not appear inflamed, a finding that might otherwise explain the symptoms. Adults with epiglottitis do not always need to be intubated if rigorous monitoring can be accomplished, a skilled intubator is immediately available, and the patient is not in distress. [89] Orotracheal intubation for epiglottitis is not as difficult in adults as it is in small children. Transtracheal needle ventilation can also be used in adults who are difficult to intubate.

Jaw Clenching

Hypertonus induced by neurologic dysfunction is a common complicating factor of airway management, especially in the patient with multiple injuries, drug overdose, or seizures. Jaw clenching may be a lethal complication when it prevents clearing of blood, vomitus, or foreign bodies in the airway. No more difficult airway problem exists than occlusion of the nasal and oral passages by vomitus while the patient's teeth are tightly clenched. Respiratory efforts may lead to severe aspiration, and although the hypertonus gradually gives way as the brainstem becomes progressively hypoxic, the cerebrocortical hypoxic insult sustained in the process may be irreversible. Various disease states can lead to a similar scenario in which the jaws are clenched in the presence of upper airway hemorrhage or the accumulation of secretions.

Jaw clenching and cervical spine injury can, of course, occur together. At times, the blind nasotracheal route of intubation may be adequate for airway management while minimizing the risk of further spine injury. However, at least a small degree of spontaneous air movement should be present for the blind nasotracheal approach to be successful. Although a serendipitous success may occur in the apneic patient, it is recommended that time not be wasted on this approach in the completely apneic patient.

Neuromuscular blocking agents are generally an effective means to overcome jaw clenching in the breathing patient. Both neuromuscular depolarizing and nondepolarizing agents may be administered intravenously to induce paralysis and

allow orotracheal intubation. Neuromuscular blocking agents and their administration for intubation are discussed in Chapter 5 .

Apnea with Airway Obstruction

Despite the many nonsurgical approaches to tracheal intubation discussed in this chapter, the patient who is apneic secondary to deep airway obstruction may be served best by a surgical airway. When maneuvers to relieve airway obstruction are unsuccessful and direct laryngoscopy is not possible or cannot rapidly alleviate the obstruction and permit ventilation, the operator should rapidly move to a surgical airway approach (see Chapter 4).

CONCLUSION

Airway management is the most fundamental aspect of emergency care. Every rescuer must know basic airway maneuvers and be able to use them instinctively. When basic maneuvers fail, airway management rapidly becomes more complex. Familiarity with the ingenious intermediate airway devices can often reverse a deteriorating situation and provide the rescuer with a temporary solution to an airway dilemma. When basic and intermediate maneuvers fail, complexity, risk, and exigency mount. Choices become more critical and complications more likely. Advance consideration of situations represented in the airway management algorithms is a wise practice for the emergency clinician. It may hasten accurate decision-making when time becomes critical. In this chapter, we have described basic and intermediate airway techniques and offered a logical schema for their use in the patient with an acutely compromised airway. Subsequent chapters deal with the more advanced airway techniques of tracheal intubation and cricothyrotomy.

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Chapter 4 - Tracheal Intubation

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Although some patients simply cannot be successfully intubated in the emergency department (ED) by any clinician, most patients can be intubated. Therefore, tracheal intubation is generally considered the most definitive means of airway control. As discussed in Chapter 3 , the decision to tracheally intubate must consider the patient's physiologic status, anticipated patient care needs, clinician experience, and features related to preparation for the procedure. This chapter discusses the indications for tracheal intubation in greater detail as well as the preparation for intubation and the key steps and modifications of the actual procedure. Chapter 5 discusses the pharmacologic adjuncts (including pharmacologic paralyzing agents) for facilitating tracheal intubation and their administration. Chapter 5 discusses more invasive means of establishing an airway and ventilating the patient. The general decision-making that is relevant to these techniques has been presented in Chapter 3 and should be reviewed before proceeding with this chapter.

GENERAL PREPARATION

Preparation is the key to successful airway management. Two general areas of preparation should be addressed before undertaking the first attempt at definitive airway management in a clinical setting. The first is mental and physical preparedness. The second is the assembly of essential intubation equipment. Note that the following discussion describes the ideal situation. Often patients require intubation under less than ideal circumstances, and lack of formal or sequential training should not deter attempts at intubation when the patient is in extremis. The best way to learn intubation is to perform the procedure in the emergency setting under supervision of an experienced clinician. As with any medical procedure, there is no classroom instruction or textbook that teaches all of the nuances of the procedure that are encountered in real life, or that can produce the milieu in the ED.

Mental and physical preparation comes from reading about the procedures, discussing the principles and details with instructors, practicing the techniques on intubation mannequins or in the animal laboratory, and finally performing the technique under supervision in a controlled clinical setting. Studies addressing various approaches to tracheal intubation are generally performed under optimal conditions (i.e., with equipment available and appropriate preparatory training). Also, often hidden within the study findings are individual learning curves. Therefore, it is overly optimistic to expect to match the success reported in the literature when first attempting a new intubation technique. However, the goal of preparation is to be as high on the learning curve as possible before the first clinical application of a new intubation technique. Further, continued rehearsal and application of the techniques that have been learned are important for skill maintenance.

Each approach to tracheal intubation has a preferred training format. Orotracheal intubation, for example, may be simulated with a mannequin, whereas retrograde intubation is best learned using an animal or cadaver model. Orotracheal intubation is likely to be successful on the first attempt, whereas considerable practice is required for facile use of the scope for fiberoptic intubation. In preparation for managing critical airway problems, maximal hands-on training is desirable.

The second general area of preparation is material preparedness (i.e., the immediate availability of all essential equipment required to optimally perform the airway maneuvers that are within the capabilities of the care provider). This may be accomplished by the wall-mounting of essential resuscitation equipment. [1] Alternatively, dedicated adult and pediatric airway carts or tackle boxes may be used for placement of the equipment in an open, organized, and labeled manner that can be regularly checked (Fig. 4-1).[2] Seldom used but potentially life-saving equipment should be clearly identified in an easily accessible location such as a dedicated difficult airway cart. The worst moment to realize that a vital piece of equipment is missing is when a patient's life depends on it. The importance of this concept cannot be overstated. Technical expertise cannot substitute for the lack of essential equipment.

In airway management, failure has ominous consequences. Mental, physical, and equipment preparation maximizes the chances of success.

AIRWAY ANATOMY

Requisite for a discussion of airway management procedures is a common understanding of airway anatomy and its terminology (Fig. 4-2*A* and *B*). The following terms are used frequently in this chapter:

Arytenoid cartilages
—the paired cartilages forming the posterior aspect of the laryngeal inlet.
Nasal Cavity
—from the external nares to the choana.
Nasopharynx
—from the end of the nasal cavity (choana) to the level of the soft palate.
Oropharynx
—soft palate to the upper border of the epiglottis.
Hypopharynx (laryngopharynx)
—epiglottis to the lower border of the cricoid cartilage.
Vallecula
—the space at the base of the tongue formed posteriorly by the epiglottis and anteriorly by the anterior pharyngeal wall.
Laryngeal inlet
—the opening to the larynx bounded anterosuperiorly by the epiglottis, laterally by the aryepiglottic folds, and posteriorly by the arytenoid cartilages.
Piriform fossae (recesses)
—the pockets on both sides of the laryngeal inlet separated from the larynx by the aryepiglottic folds.
Corniculate cartilage
—the posteromedial portion of the arytenoid cartilage.
Cuneiform cartilage
—the anterolateral prominence of the arytenoid cartilage.
Glottis
—the vocal apparatus, including the true and false cords and the glottic opening.
Glottic opening (rima glottidis)
—the opening into the trachea as seen from above through the vocal cords.

Figure 4-1 Adult airway cart. Equipment and materials are visible, labeled, and accessible. *(Concept and design by Dr. Ernest Ruiz, Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

OROTRACHEAL INTUBATION

Indications and Contraindications

Any clinical situation in which a definitive airway is necessary, and limited neck motion is permissible, is an indication for orotracheal intubation. Many of these situations, including cardiac arrest, airway compromise in infection and trauma, and airway obstruction are discussed in detail in Chapter 1 . Most orotracheal intubations are accomplished using a direct laryngoscope. An unstable cervical spine injury is a relative contraindication to direct laryngoscopy, but should not preclude definitive airway management when clinically indicated.

Equipment

Laryngoscope

Facility in the use of the direct laryngoscope is a prerequisite for orotracheal intubation. Various adult and pediatric blade sizes are available. There are two basic blade designs—curved (MacIntosh) and straight (Miller and Wisconsin). Slight variations in laryngoscopic technique follow from one's choice of blade design, which is often a matter of personal preference. The tip of the *straight blade* goes under the epiglottis and lifts it directly, whereas the *curved blade* fits into the vallecula and indirectly lifts the epiglottis via the hyoepiglottic ligament to expose the larynx. Special blades designed for the anterior larynx include the Siker and the Belscope (Avulunga Pty Ltd, New South Wales, Australia).

Each blade type has advantages and disadvantages. The straight blade is often a better choice in pediatric patients, in patients with an anterior larynx or a long floppy epiglottis, and in individuals whose larynx is fixed by scar tissue. It is less effective, however, in patients with prominent upper teeth, and it is more likely to break teeth. Use of the straight blade is also more often associated with laryngospasm due to its stimulation of the superior laryngeal nerve, which innervates the undersurface of the epiglottis. A straight blade may inadvertently be advanced into the esophagus and initially present one with unfamiliar anatomy until it is withdrawn. The blade has a light bulb at the tip that may slightly hamper vision. The wider, curved blades are helpful in keeping the tongue retracted from the field of vision, allowing for more room in passing the tube in the oropharynx, and they are generally preferred in uncomplicated adult intubations. Aside from patient considerations, some clinicians prefer the curved blade because they find it requires less forearm strength than the straight blade.

Tracheal Tubes

The standard adult endotracheal (ET) tube measures approximately 30 cm in length. Tube size is typically printed prominently on the tube and is based on the *internal diameter* (ID), measured in millimeters. The range is from 2.0 to a 10.0 mm, increasing in increments of 0.5 mm. The outer tube diameter is 2 and 4 mm larger than the internal diameter.[3] Tubes are also imprinted with a scale in centimeters that indicates the distance from a tube's distal tip.

Adult men generally accept a 7.5 to 9.0 mm orotracheal tube, whereas women can usually be intubated with a 7.0 to 8.0 mm tube. In most circumstances, tubes smaller than these should not be used because airway resistance increases as tube size decreases. However, in emergency intubations, particularly if a difficult intubation is anticipated, many clinicians choose a smaller tube and change to a larger tube later. Although generally an acceptable practice, it should be avoided in the burn patient because swelling may prohibit subsequent tube placement. For nasal intubation, a slightly smaller tube than would be used for orotrachael intubation (by 0.5 to 1.0 mm) is chosen.

Correct tube size is especially important in the pediatric population (Table 4-1). Because most patients younger than

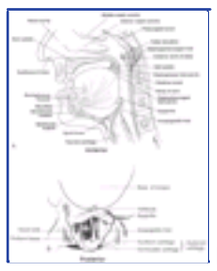

Figure 4-2 *A*, Lateral (sagittal) view of the upper airway. *(A from Snell RS, Smith MS (eds): Clinical Anatomy for Emergency Medicine. St Louis, Mosby-Year Book, 1993, p 16.) B*, View of larynx, epiglottis, and vocal cords as seen with laryngoscope (not shown) in place.

8 years are intubated with an uncuffed tube, adequate tube size is necessary to provide a good seal between the tube and the upper trachea. A cuffed tube is used in children with decreased lung compliance who may require prolonged mechanical ventilation. In a child, the smallest airway diameter is at the cricoid ring rather than at the vocal cords, as in adults. Hence, a tube may pass the cords but go no farther. Should this occur, the next smaller sized tube should be passed.

In infants and children, the following formula is a highly accurate method for determining correct tracheal tube size:

Tube size = [4 + age (years)]/4

For most clinical situations, however, using the width of the nail of the little (5 th) finger as a guide is sufficiently accurate and has been shown to be more precise than finger diameter (Fig. 4-3).[4]

A standard tracheal tube uses a high-volume, low-pressure cuff to avoid pressure necrosis of the tracheal lining. A clinical test for determining correct cuff inflation is to slowly inject air until no air leak is audible while the patient is receiving bag-tube ventilation. This usually occurs with 5 to 8 mL of air if the proper-sized tracheal tube has been selected. Many clinicians use the tension of the pilot balloon as a guide to cuff inflation; slight compressibility with gentle external pressure indicates adequate inflation for most clinical situations. For long-term use, cuff pressure should be measured and maintained at 20 to 25 mm Hg. Capillary blood flow is compromised in the tracheal mucosa when the cuff pressure exceeds 30 mm Hg. In emergency situations, the balloon may simply be inflated with 10 mL of air and adjusted when the patient's condition has stabilized.

Adult ET tubes will accept a standard adaptor on which the ventilator tubing will fit. Pediatric tubes require a special adaptor with a distal end small enough to accommodate the small tube sizes.

Preparing for Intubation

Before intubating, the following steps should be taken in chronologic order: (1) confirm that the required intubation equipment is available and functioning; (2) position the patient correctly; (3) assess the patient for difficult airway; (4) establish intravenous (IV) access, time permitting; (5) draw up essential drugs; and (6) attach the necessary monitoring

TABLE 4-1 -- Tracheal Tube Sizes for Average Patients*

Age	Size (Fr)	Internal Diameter (mm)	Equivalent Tracheotomy Tube Size
Premature			
Newborn	12	2.5	00
6 mo	16	3.5	00–0
1 yr	20	4.5	0–1
2 yr	22	5.0	1–2
4 yr	24	5.5	2
6 yr	26	6.0	3
8 yr	28	6.5	4
10 yr	30	7.0	4
12 yr	32	7.5	4
14 yr	34	8.0	5
Adult			
Female	34–36	7.0–8.0	5
Male	36–40	7.5–9.0	6
Special cases			8–10

Modified from Applebaum EL, Bruce DL: Tracheal Intubation. Philadelphia, WB Saunders, 1976.

*A slightly smaller size may be required for nasotracheal intubation.

devices. In the haste of the moment, it is a common error to fail to position the patient properly or to proceed with the procedure before the proper equipment is assembled and checked. Simple omissions, such as failing to restrain the patient's hands, removing dentures, or misplacing the suction device, can seriously hamper the performance of the procedure. One suggested preintubation checklist is presented in Table 4-2 .

In addition to the preparation necessary for optimum patient care, the clinician should also minimize exposure to potentially infectious materials (see Chapter 71). Generally, the clinician should wear gloves and eye/mouth protection to guard against exposure to patient secretions.

The ET tube cuff should be checked for leaks by inflating the balloon before attempting intubation. The tube is prepared for placement by passing a flexible stylet down the tube to increase its stiffness and enhance control of the tip of the tube. *The stylet should not extend beyond the eyelet of the tube.* The tube is then bent in a gradual curve with a more

Figure 4-3 Pediatric endotracheal tube size estimation using fingernail width of the 5th finger.

TABLE 4-2 -- Suggested Preintubation Checklist

1. An assistant should be in the room watching the cardiac monitor, blood pressure, and O_2 saturation while observing the patient for signs of decompensation. The assistant should be instructed to inform the operator if more than 30 sec have elapsed without ventilation.
2. An IV infusion should be running properly. Oxygen should be administered to the patient.
3. Draw the necessary drugs (e.g., atropine, lidocaine, paralyzing agent, induction agent).
4. Attach the bag-valve-mask to an oxygen source (rate of 15 L/min).
5. If used, a stylet should be inserted properly into the tracheal tube.
6. Check the integrity of the balloon on the tracheal tube.
7. Have tape or commercial tube stabilizer available.
8. Check laryngoscope light source. Have a second light source, a selection of blades, and additional endotracheal tubes available.
9. Turn on the oral suction device and place the suction tip under the mattress to the left of the patient's head. Prepare the catheter suction for postintubation use.
10. Place the syringe to inflate the endotracheal tube balloon on the stretcher to the right of the patient's head. An option is to attach a syringe to the pilot balloon of the endotracheal tube.
11. If the patient is awake, restrain the hands.
12. Remove the patient's dentures (delay this action until immediately before intubation if patient is being bag-mask ventilated).
13. Check the cardiac monitor leads and the rhythm strip immediately before the intubation attempt.
14. Check for optimal head positioning: neck slightly flexed and head extended on the neck (conditions permitting). May be facilitated by placing a towel under the patient's occiput to raise it 10 cm.
15. Have an aspiration device for esophageal detection or an end-tidal CO_2 detector at the bedside.
16. Radiology department should be alerted for the postintubation chest radiograph.

acute angling in the distal one-third to more easily access the anterior larynx. The tip and cuff of the tube are lubricated with viscous lidocaine or a water-soluble gel.

The patient should be positioned to optimally align the oral, pharyngeal, and laryngeal axes (Fig. 4-4). The desired position was aptly described by Magill to make the patient appear to be "sniffing the morning air," with the head extended on the neck and the neck slightly flexed relative to the torso. A small towel under the occiput (to raise it 7 to 10 cm) may facilitate positioning in the adult, but not in the child. Positioning of the head and neck is a critical step; suboptimal head positioning may be a common reason for intubation failures.

The Difficult Airway

Most difficult intubations are predictable if the clinician has time to evaluate the patient before securing the airway. Unfortunately, most intubations in the emergency setting are not elective, nor do they lend themselves to careful evaluation for their degree of difficulty. Unlike the anesthesiologist who evaluates the airway and intubation nuances on the night before surgery, the emergency clinician must often proceed with an intubation that only turns out to be "difficult" when

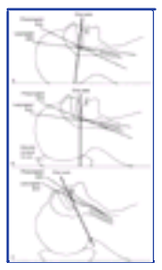

Figure 4-4 Head positioning for tracheal intubation. *A,* Neutral position. *B,* Head elevated. *C,* "Sniffing" position, with flexed neck and extended head. Note how flexing the neck and extending the head to line up the various axes allows for intubation. This position creates the shortest distance and straightest line between the teeth and vocal cords.

the patient cannot be readily intubated. Perhaps the most frequently encountered condition associated with a difficult intubation is the agitated or combative patient. Fortunately, this condition can often be readily eliminated through pharmacologic intervention (see Chapter 5). The classic parameters that predict a difficult intubation include a history of previous difficult intubation, prominent upper incisors, limited ability to extend at the atlanto-occipital joint, [5] poor visibility of pharyngeal structures when the patient extends the tongue (Mallampati classification, or the tongue/pharyngeal ratio) (Fig. 4-5*A*),[6] limited ability to open the mouth (suggested by a space less than three fingerbreadths between upper and lower incisors), [7] a limited direct laryngoscopic view of the laryngeal inlet, [7] and a short distance from the thyroid notch to the chin with the neck in extension (Fig. 4-5*B*). [8] In emergency airway management, many of these predictors are not obtainable. An extensive history is rarely available, the patients are frequently uncooperative, and the presence of trauma limits movement of the neck. Fortunately, some of the key predictors are apparent simply by observing the external appearance of the patient's head and neck.

Patients with neck tumors, thermal or chemical burns, traumatic injuries to the face and anterior neck, angioedema, infection of the pharyngeal and laryngeal soft tissues, or previous operations in or around the airway suggest a difficult intubation because distorted anatomy or secretions may compromise visualization of the vocal cords. Facial or skull fractures may further limit airway options by precluding nasotracheal intubation. Patients with ankylosing arthritis or developmental abnormalities, such as a hypoplastic mandible or the large tongue of Down syndrome, are difficult to intubate because neck rigidity and problems of tongue displacement can obscure visualization of the glottis.

Besides these obvious congenital and pathologic conditions, the short, thick neck poses the greatest difficulty for performing orotracheal intubation. Obese patients with a "short" neck are common and present some of the most difficult airways in the ED. In such individuals, the larynx is anatomically higher and more anterior, which makes it harder to visualize the vocal cords. These individuals are easily identified by observing the head and neck in profile. A mentum to hyoid distance less than three fingerbreadths also suggests a difficult airway. In such patients, apply laryngeal pressure and consider using the straight blade. Use of other options, including nasotracheal intubation, may be required. Facial hair can make forming a tight seal with the bag mask difficult and should alert the clinician that attempting to adequately ventilate the patient by that means may be difficult, if not impossible.

It should be emphasized that some patients, despite normal-appearing anatomy and the absence of a complicating history, are unexpectedly difficult to intubate. One must be prepared for this rare but inevitable occurrence.

Procedure

Adults

Direct laryngoscopy.

The clinician is stationed at the patient's head (Fig. 4-6). The patient is generally supine *with the head at the level of the clinician's lower sternum*. Attempting to intubate a patient on a low stretcher is a common error. To maintain the best mechanical advantage, the clinician keeps his or her back straight and does not hunch over the patient; any bending should occur in the knees. The left elbow is kept relatively close to the body and flexed to provide better support. In the severely dyspneic patient who cannot tolerate lying down, direct laryngoscopy can be performed with the patient seated semi-erect and the laryngoscopist on a step stool behind the patient. [9]

The laryngoscope is grasped in the left hand with the blade directed toward the patient from the hypothenar aspect of the clinician's hand. The patient's lower lip is drawn down with the right thumb, and the tip of the laryngoscope is introduced into the right side of the mouth. The blade is slid along the right side of the tongue, gradually displacing the tongue toward the left as the blade is moved to the center of the

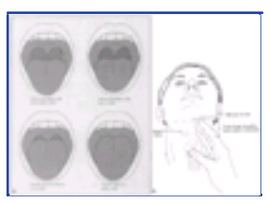

Figure 4-5 *A*, Mallampati classification predicting difficulty of intubation based on visibility of intraoral structures: Intubation should pose no difficulty in patient in class I or II, moderate difficulty in class III, and severe difficulty in class IV. *(A from Whitten CE: Preintubation evaluation: Predicting the difficult airway. Emerg Med 21:111, 1989.) B*, A difficult intubation also may be expected if the distance from the mentum of the chin to the hyoid bone in an adult is less than three fingerbreadths when the head is extended. Patients with a shortened distance often have a hypoplastic or poorly developed mandible or a short, thick neck, resulting in an anterior larynx that may be best approached with the straight blade and cricoid pressure.

mouth. If the blade is initially placed in the middle of the tongue, the tongue will fold over the lateral edge of the blade and obscure the airway. *Placing the blade in the middle of the tongue and failing to move the tongue to the left are two common errors preventing visualization of the vocal cords* (Fig. 4-7*A–C*).

As the blade tip approaches the base of the tongue, the clinician exerts force along the axis of the laryngoscope handle, lifting upward and forward at a 45° angle. The epiglottis should come into view with this maneuver. It may help to have an assistant retract the cheek laterally to further expose the laryngeal structures. Bending the wrist should be avoided because it can result in dental injury if the teeth become a fulcrum for the blade.

The step following visualization of the epiglottis depends on which laryngoscope blade is used. With the curved blade, the tip is placed into the vallecula, the space between the base of the tongue and the epiglottis. Continued anterior elevation of the base of the tongue and the epiglottis will expose the vocal cords. If the blade tip is inserted too deeply into the vallecula, the epiglottis may be pushed down to obscure the glottis. [6] When using the straight blade, the tip is inserted under and slightly beyond the epiglottis, directly lifting this structure. The jaw and larynx are literally suspended by the blade. If the straight blade is placed too deeply, the entire larynx may be elevated anteriorly and out of the field of vision. Gradual withdrawal of the blade should allow the laryngeal inlet to drop down into view. If the blade is deep and posterior, the lack of recognizable structures indicates esophageal passage; gradual withdrawal should permit the laryngeal inlet to come into view. The use of the curved and straight laryngoscope blades is illustrated in Figure 4-7*D* and *E* .

Proper neck positioning and pressure (cephalad, dorsally, and rightward) on the larynx by an assistant will facilitate visualization and intubation of an anterior larynx. The "BURP" (backward, upward, rightward pressure) maneuver consists of displacing the larynx as far posteriorly and superiorly as possible and slightly to the right. Use of this technique allows greater visualization than simple backward pressure or no pressure in patients with incompletely visualized airways on initial

inspection.[10] If the vocal cords are not visualized or are incompletely visualized, the clinician may wish to apply external laryngeal manipulation, also called bimanual laryngoscopy. To perform this maneuver, the laryngoscopist holds

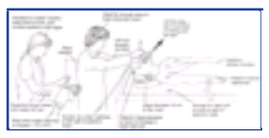

Figure 4-6 Proper positioning of clinician, patient, and assistant for tracheal intubation.

the laryngoscope in the standard fashion and places the right hand on the patient's thyroid cartilage to determine the best position of the larynx from the intubator's perspective. The thyroid cartilage is manipulated until optimal visualization of the vocal cords is obtained. An assistant then holds pressure in the same position and the clinician places the ET tube as described later. Levitan demonstrated in a videographic study that use of this technique by novice intubators improved percentage of glottic opening scores by an average of 57%.[11] This is demonstrated in Figure 4-8 .

If the vocal cords are still not seen, a tracheal tube introducer may be used (Smiths Industries Medical Systems, Keene, NH). This device, also known as the "elastic gum bougie," is a long, semirigid introducer that is placed, using the laryngoscope, through the laryngeal inlet and into the trachea. [12] The tracheal tube is then passed over the introducer and the introducer is withdrawn. If resistance is met in passing the tracheal tube, rotate the tube 90° counterclockwise and advance the tube. A study of 200 patients in the anesthesia literature reported a 96.5% success rate for gum bougie insertion within 2 attempts. The success rate for passing the ET tube over the bougie was 99% within 2 attempts.[13] A curved or "coude tip" bougie is best suited for aiding in difficult intubations. The curved tip provides tactile feedback as it passes along the tracheal rings. The straight tipped gum bougie should be reserved for changing of ET tubes. [14]

Tube passage.

Once the vocal cords have been visualized, the final step is tube passage under direct vision through the vocal cords and into the trachea. The tube is held in the clinician's right hand and introduced from the right side of the patient's mouth. The tube is advanced toward the patient's larynx at an angle, not parallel with or down the slot of the laryngoscope blade. This way, the clinician's view of the larynx is not obstructed by the hand or the tube until the last possible moment before the tube enters the larynx. If the patient is not chemically paralyzed, the tube should be passed during inspiration, when the vocal cords are maximally open. It enters the trachea when the cuff disappears through the vocal cords. The tube is advanced 3 to 4 cm beyond this point. It is not enough to see the tube and the cords; the tube must be seen passing through the vocal cords to ensure tracheal placement.

When the vocal cords are stimulated, laryngospasm—the persistent contraction of the adductor muscles of the vocal cords—may prevent passage of the tube. Inadequate anesthesia is often the cause. Pretreatment with topical lidocaine decreases the likelihood of this occurring. Lidocaine, 2% or 4%, can be sprayed directly on the cords. An infrequent but effective route for achieving tracheal anesthesia is via transtracheal puncture, injecting a bolus of 3 to 4 mL of lidocaine through the cricothyroid membrane. Laryngospasm is usually brief and often followed by a gasp. The clinician should be ready to pass the tube at this moment. Occasionally, the spasm is prolonged and needs to be broken with sustained anterior traction applied at the angles of the mandible—the jaw lift. At no time should the tube be forced because permanent damage to the vocal cords may result. Consideration should be given to using a smaller tube. Prolonged, intense spasm may ultimately require muscle relaxation with a paralyzing drug (see Chapter 5). The pediatric patient is far more prone to laryngospasm than an adult. [15] In a child, if vocal cord spasm prevents tube passage, a chest thrust maneuver may momentarily open the passage and permit intubation. [16]

Positioning and securing the tube.

The ET tube should be secured in a position that minimizes both the chance of inadvertent endobronchial intubation and the risk of extubation. The tip should lie in the midtrachea with room to accommodate neck movement. Because tube movement with both neck flexion and extension averages 2 cm, the desired range of tip location is between 3 and 7 cm above the carina. [17]

On a radiograph, the tip of the tube should ideally be 5 ± 2 cm above the carina when the head and neck are in a neutral position. On a portable radiograph, the adult carina overlies the fifth, sixth, or seventh thoracic vertebral body. If the carina is not visible, it can be assumed that the tip of the tube is properly positioned if it is aligned with the third or fourth thoracic

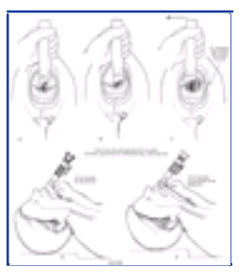

Figure 4-7 Common problems encountered with the laryngoscope: *A,* Laryngoscope blade under the middle of the tongue, with the sides of the tongue hanging down and obscuring the glottis. *B,* Tongue not pushed far enough to the left, obscuring the glottis. *C*, Correct blade position with tongue elevated and to the left. *D,* Use of the curved (MacIntosh) laryngoscope blade. *E,* Use of the straight (Miller) blade.

vertebra. In children, the carina is more cephalad than in the adult, but it is consistently situated between T3 and T5. In children, T1 is the reference point for the tip of the ET tube.[18]

An estimate of the proper depth of tube placement, before radiograph confirmation, can be derived from the following formulas, the lengths representing the distance from the tube tip to the upper incisors in children[19] [20] and from the upper incisors[21] or the corner of the mouth[22] in adults:

Children: Tracheal tube depth (cm) = age (years)/2 + 12
Adults: Tracheal tube depth (cm) = 21 cm (women)
Tracheal tube depth (cm) = 23 cm (men)

Figure 4-8 The epiglottis is elevated and visualization of the vocal cords is improved by pressure on the hypoepiglottic ligament and external laryngeal manipulation. *(Courtesy*

of Richard M. Levitan, MD. Airway Cam Technologies Inc., Wayne, PA. Used by permission.)

In adults, this method has been shown to be more reliable than auscultation in determining the correct depth of placement. [21]

The cuff is inflated to the point of minimal air leak with positive-pressure ventilation. In an emergency intubation, 10 mL of air is placed in the cuff, and inflation volume is adjusted after the patient's condition is stabilized.

After tracheal tube placement, both lungs are auscultated under positive-pressure ventilation. Care is taken to auscultate laterally because midline auscultation may lead to an erroneous impression of tracheal placement when the tube is actually in the esophagus. With the tube in position and the cuff inflated, the tube is secured in place. Commercial ET tube holders, adhesive tape, or umbilical (nonadhesive cloth) tape can be attached securely to the tube and around the patient's head (Fig. 4-9 and Fig. 4-10). The tube should be positioned in the corner of the mouth, where the tongue cannot expel it. This position is also more comfortable for the patient and allows for suctioning. A bite-block or oral airway to prevent ET tube crimping or damage from biting is commonly incorporated into the system used to secure the tube.

Unintentional extubation can have disastrous consequences, particularly if the patient was difficult to intubate initially. Care should be taken to secure the endotracheal tube immediately after placement has been confirmed. Orotracheal intubation is associated with a higher rate of unplanned extubation than is nasotracheal intubation. [23] During transport of the intubated patient, one person should be designated to tend to the ET tube to avoid accidental extubation. Inadequate sedation is another risk factor for accidental extubation. [23] The use of chemical or physical restraints may be necessary to prevent extubation in the agitated or confused patient.

Obstetric Patients

Pregnant patients present unique challenges in airway management, and knowledge of the changes that occur in the anatomy and physiology of the obstetric patient is important to ensure safe care of the mother and her unborn child. Functional residual capacity is significantly reduced in pregnancy due to the enlarged uterus, which displaces the abdominal organs and elevates the diaphragm. This decline in functional residual capacity is most pronounced in the supine position, [24] causing the rapid desaturation seen in the preintubation period. Adequate preoxygenation is therefore of great importance in the pregnant patient. Airway management can also be complicated by the development of hypotension in the supine position as a result of compression of the vena cava by the gravid uterus. This supine hypotension can be prevented by maintaining a pregnant patient at a minimum tilt of 15° to the left. In the trauma setting, this position can be maintained by placing a wedge-shaped foam pad or blankets under the long spine board.

Airway anatomy is also altered in pregnancy. Engorgement of oropharyngeal and nasal mucosa leads to easily provoked bleeding with manipulation. The generalized edema of pregnancy does not spare the structures of the airway and the clinician should be prepared to use smaller ET tubes (6.0 to 7.0). [25] [26] Advanced gestation has been shown to correlate with higher maternal Mallampati scores. [27] Equipment for dealing with difficult airways should be ready when ET intubation of the patient is anticipated. If emergency Caesarean sections are performed in the ED, contingent plans for the maintenance of anesthesia and the care of neonates should also be made.

Pregnant patients are at higher risk for aspiration during emergency airway procedures. The relaxed tone of the lower esophageal sphincter and delayed gastric emptying contribute to this risk. The principles of rapid sequence intubation with maintenance of cricoid pressure should be followed if at all possible when intubating the obstetric patient.

Infants and Children

Appreciation of the anatomic differences between children and adults is helpful when intubating the pediatric patient (Table 4-3 and Fig. 4-11). Infants' proportionately larger heads naturally places them in the "sniffing position," so a towel under the occiput is rarely necessary. The large head can even result in a posterior positioning of the larynx that prevents visualization of the vocal cords; a small towel under the child's shoulders should correct this problem. The head also may be floppy, and it can be stabilized by an assistant during intubation. The child's increased tongue-to-oropharynx ratio and shorter neck hinder forward displacement of the tongue and, coupled with a U-shaped epiglottis, can make visualization of the glottis difficult. Consequently, direct laryngoscopy in the infant and young child is generally best performed with a straight blade: Miller size 0 for premature infants, size 1 for normal-sized infants, and size 2 for older

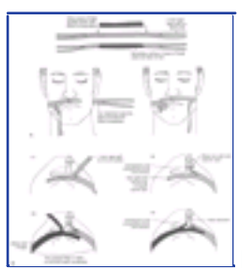

Figure 4-9 Two views (*A* and *B*) of technique for taping the tracheal tube. It is important to secure the tracheal tube properly. The method illustrated can be replaced by using a commercial holder or tracheostomy cloth tie. Avoid taping the lips.

children. The infant's larynx lies higher and relatively more anterior. One can have an assistant lightly apply laryngeal pressure, or the clinician can use the little finger of the hand holding the laryngoscope blade for this purpose (Fig. 4-12). If no laryngeal structures are visible after laryngeal pressure, the blade should be gradually withdrawn. Inadvertent advancement of the blade into the esophagus is a common error.

Confirmation of Tracheal Intubation

Clinical Assessment

The best assurance of tracheal placement is for the clinician to see the tube pass through the vocal cords (Table 4-4). Absent or diminished breath sounds, vocalization, increased abdominal size, and gurgling sounds during ventilation are clinical signs of esophageal placement. However, esophageal placement

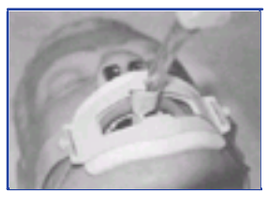

Figure 4-10 This disposable endotracheal tube holder provides a firm anchor on which to tape the tube, while also serving as a bite block. *(Courtesy of Precision Medical, Northampton, PA.)*

is not always obvious. One may hear "normal" breath sounds if only the midline of the thorax is auscultated. The presence of condensation of the ET tube as a means of confirming tracheal placement may also be misleading. Blinded observers noted condensation of the ET tube during ventilation in 23 out of 27 esophageal intubations in an animal model. [28] One way to clinically assess tracheal placement after a ventilation or during spontaneous respiration is to note whether air is felt or heard to exit through the tube following cuff inflation. If the tidal volume is adequate, the exit of air should be obvious. It is important to note that when an appropriately sized tube is placed in the trachea, the patient cannot groan, moan, or speak. Any vocalization suggests esophageal placement.

Asymmetrical breath sounds indicate probable main stem bronchus intubation. Due to the angles of takeoff of the main bronchi and the fact that the carina lies to the left of the midline in adults, right main stem intubation is most common and is indicated by decreased breath sounds on the left side. When asymmetrical sounds are heard, the cuff should be deflated and the tube withdrawn until equal breath sounds are present. Bloch and colleagues report accurate pediatric tracheal positioning if after noting asymmetrical breath sounds, the tube is withdrawn a defined distance beyond the point at which equal breath sounds are first heard—2 cm in children younger than 5 years and 3 cm in older children. [29]

Esophageal Detector Device

An aspiration technique used to determine ET tube location was first described by Wee in 1988. [30] The technique takes advantage of the difference in tracheal and esophageal resistance to collapse during aspiration to locate the tip of the tracheal tube. Following intubation, a large syringe is attached to the end of the ET tube and the syringe plunger is withdrawn. If the tube is correctly placed in the trachea, the plunger will pull back without resistance as air is aspirated from the lungs. However, if the tracheal tube is in the esophagus, resistance is felt when the plunger is withdrawn, because the pliable walls of the esophagus collapse under the negative pressure and occlude the end of the tube. Another device using the same principle as syringe aspiration is the self-inflating bulb (e.g., Ellick device).

In the initial study conducted in the operating room setting, [30] the tube placement was correctly identified in 99 of 100 cases (51 esophageal, 48 tracheal). The result was considered equivocal in the remaining case. That tube was removed and found to be nearly totally occluded with purulent secretions. Slight resistance was noted in one patient with a right main stem intubation; resistance decreased when the tube was pulled back. Before use, the esophageal detector device must always be checked for air leaks. If any connections are loose, the leak may allow the syringe to be easily withdrawn, mimicking tracheal location of the tube.

A guideline to follow when using the aspiration technique is to apply constant, slow aspiration to avoid tube occlusion from tracheal mucosa drawn up under high negative pressure. If the tracheal tube is correctly placed, 30 to 40 mL of air can be aspirated without resistance. If air was initially aspirated and then some resistance is encountered, the tracheal tube should be pulled back between 0.5 and 1.0 cm and partially rotated. This takes the tube out of the bronchus, if it has been placed too deeply, and changes the orientation of the bevel if the tube has been temporarily occluded with tracheal mucosa. Air is easily aspirated if the tube was in the trachea, but repositioning will make no difference if the tube was in the esophagus. The syringe aspiration technique can be used before or after ventilation of the patient. Continuous cricoid pressure should be applied pending tube confirmation. Inflation of the tube cuff has no effect on the reliability of the test. [31] This device is reliable, rapid, inexpensive, and easy to use. [32] A squeeze-bulb aspirator can be used as an alternative to the syringe technique. [33] [34] The bulb is attached to the ET tube and squeezed; if the tube is in the esophagus, it is often accompanied by a flatus-like sound followed by absent or markedly delayed refilling. Insufflation of a tube in the trachea is silent, with instantaneous refill. An early study with the Ellick evacuator bulb device reported that 87% of esophageal intubations were identified. [33] A later study using a slightly different bulb device (Respironics, Murrysville, PA) found that all 45 esophageal intubations were detected. [34] The device is cheap, easy to use, and operable single-handedly in <5 seconds. [33] [34] The bulb should not be used in freezing temperatures due to a loss of elasticity.

Confusion may occur if the esophageal tube is tested more than once, because subsequent inflations may be silent. On repeated assessments, a false-positive refilling of the bulb may occur due to instillation of air during the first attempt. This observation has led to a recommendation that the bulb be compressed before it is attached to the ET tube. Delayed though complete refilling may occur with bronchial tube placement or in the more pliable pediatric airway. The bulb suction modification of the aspiration technique has not been studied as thoroughly as the syringe technique.

End-Tidal CO_2 Detector Devices

A high level of CO_2 in exhaled gas is the physiologic basis for capnography and the principle on which end-tidal CO_2 ($ETCO_2$) detectors were developed (see Chapter 2). The most commonly available devices for emergency use are colorimetric indicators, which correspond to CO_2 levels flowing through the device when placed on the tracheal tube adapter. The typical device displays opposite colors (e.g., yellow and purple) to indicate the low level of CO_2 in esophageal gas versus the higher level in gas exhaled from the respiratory tree. Handheld quantitative or semiquantitative electronic CO_2 monitors are also available.

TABLE 4-3 -- Comparison of the Airway in the Adult and Child

Comparison	Child	Adult	Clinical Consequences or Adjustments for Child
Head	Proportionately larger (up to about age 10 yr)	Proportionately smaller	Child naturally in sniffing position when supine. Do not place towel under occiput; may benefit from elevation of shoulders. Large head may be "floppy," requiring assistant to hold head still during intubation.
Teeth	Easily knocked out	Stable unless decay or trauma is a factor	Teeth may be knocked out and aspirated or forced into trachea.
Tonsils or adenoids	Large and friable	Generally not a problem	Nasotracheal intubation in child may cause excessive bleeding. Adenoid or tonsil tissue may plug endotracheal tube or cause airway obstruction from aspiration.
Tongue	Relatively larger	Relatively smaller	Difficult to displace the tongue anteriorly in child. Consider using straight blade.
Larynx	Opposite C2, C3	Opposite C4–C6	The more superiorly located larynx, the "anterior" larynx, is more difficult to visualize. Consider using straight blade.
Epiglottis	U-shaped, shorter, stiffer	Flatter, more flexible	Epiglottis more difficult to manipulate in child, may fold down and obstruct view with curved blade. Consider using straight blade.
Vocal cords	Concave upward. Anterior attachment of cords lower than posterior, creating a slant	Horizontal	Concave shape does not affect intubation, but it may affect ventilation. For partial airway obstruction or to break laryngospasm, consider positive pressure ventilation with jaw lift to open arytenoids. Anterior superior slant of the vocal cords may cause the endotracheal tube to hang up on anterior commissure as it passes into larynx. Rotate tube 90° counterclockwise. Overextension of the neck may cause partial airway obstruction due to airway collapse.
Length of trachea	Relatively shorter	Relatively longer	Short trachea increases likelihood of main stem bronchus intubation. Follow the formula for correct depth of placement (cm depth = 0.5 age (yr) + 12) measured from the corner of the mouth. Double black line on endotracheal tube should pass just beyond the cords.
Airway diameter	Relatively smaller; smallest diameter is at the cricoid ring	Relatively larger; smallest diameter is between vocal cords	Laryngoscope-induced trauma, edema, and foreign material will significantly alter airway diameter. Be gentle. Extremes of flexion or extension may kink airway. If trouble with bag-valve-mask ventilation, reassess the degree of head flexion or extension. Cricoid pressure may cause complete airway obstruction. Endotracheal tube may pass through cords but be too large to pass through cricoid ring. If unable to pass into trachea, use the next smaller tube.
Residual lung capacity	Relatively smaller	Relatively larger	Child may become hypoxic more quickly than adult. Closely monitor O_2 saturation and avoid prolonged periods without ventilation.

A multicenter study of a colorimetric device demonstrated an overall sensitivity of 80% and a specificity of 96%. [35] In patients with spontaneous circulation and the tracheal tube cuff inflated, the sensitivity and specificity rose to 100%. The poor sensitivity seen in cardiac arrest (69%) is due to the fact that low exhaled CO_2 levels are seen in both very-low-flow states and in esophageal intubation. The device must therefore be used with caution in the cardiac arrest victim. Levels of CO_2 return to normal after return of spontaneous circulation in these patients. Further, colorimetric changes may be difficult to discern in reduced lighting situations, and secretions can interfere with the color change. Regardless of the monitoring device, patients in cardiac arrest should be ventilated for a minimum of 6 breaths before taking a reading. Otherwise, recent ingestion of carbonated beverages can result in spuriously high CO_2 levels with esophageal intubation. [36] Colorimetric changes do not rule

out glottic positioning of the ET tube tip. Adequate ventilation and oxygenation may be achieved in the glottic position, but the risk of aspiration in the absence of a protected airway and the potential for further tube dislodgement remain. Glottic positioning may be difficult to detect clinically. The only signs may be persistent cuff leak or dimished chest rise with ventilation. Radiographic evidence or direct visualization confirms the diagnosis. [37]

Figure 4-11 Sagittal sections of the neck of an adult (*A*) and an infant shortly after birth (*B*). Different vertebral levels in these age groups are shown. Note that in children, the neck is shorter and the larynx is located more cephalad. *(From Snell RS, Smith MS (eds): Clinical Anatomy for Emergency Medicine. St Louis, Mosby-Year Book, 1993, p 16.)*

Comparison of Detector Devices

In the setting of spontaneous circulation, both syringe aspiration and $ETCO_2$ detection are highly reliable means of excluding esophageal intubation. An animal study comparing these techniques with clinical assessment and measuring the speed and accuracy of tube placement determination demonstrated that both the syringe esophageal detector device and $ETCO_2$ detection were highly accurate, approaching 100%. [38] The

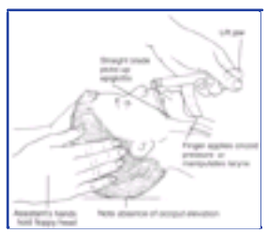

Figure 4-12 Oral intubation in a child, using a straight blade. The proportionately large, floppy head of the child may present some difficulty, and an assistant may be required to hold the child's head straight.

esophageal detector device was more rapid with determination in 13.8 seconds vs 31.5 seconds for $ETCO_2$ detection. The detector device remained accurate when air was insufflated into the esophagus for 1 minute, simulating unrecognized esophageal placement. Clinical assessment alone yielded an alarming 30% rate of failure to identify esophageal intubation. In the setting of cardiac arrest, the aspiration method is more reliable than CO_2 detection, because its accuracy is not dependent on the presence of blood flow.

The sonomatic confirmation of tracheal intubation (SCOTI) device is a battery-operated, handheld monitor designed to confirm ET tube location. It is a third-generation esophageal detection device and operates based on acoustic differences in air in the rigid trachea and collapsible esophagus. A trial of the device in both ED and surgical patients reported a sensitivity and specificity of 93% and 98% respectively. [39] However, experience with the device is necessary in order to appropriately interpret its readings. Also, the device must be calibrated before each use. The SCOTI device is not reliable in spontaneously breathing patients.

At present, there is no perfect device for the confirmation of ET tube placement in all situations. The clinician should be aware of the limitations of each device and ideally should rely on information from multiple sources of information to ensure correct placement of the ET tube.

Complications

Failure to achieve adequate ventilation and oxygenation is the most serious complication of tracheal intubation. The potential for hypoxia exists just before intubation as more conservative methods are attempted and fail, during a difficult intubation when ventilation is halted for intubation attempts, and after intubation when esophageal intubation goes

TABLE 4-4 -- Assessing Proper Tube Placement

Test	Interpretation
Observe tube pass through the vocal cords	Accurate way to ensure placement; if in doubt, look again after intubation
Auscultation of breath sounds over the chest	May be misleading, especially if only midline is examined; listen in both axillae
Auscultation over the stomach	Gurgling indicates esophageal placement
Condensation (fog) forms inside tube with each breath	Quite reliable
Observe chest rise with positive pressure and fall with release	Generally reliable but may be absent in patients with small tidal volume or severe bronchospasm
Feel air exiting from end of tube following inflation	Reliable
Air remains in lung after end of tube is occluded and exits when occlusion is removed	Reliable but one may "ventilate" a closed area of the esophagus
Ask patient to speak; listen for moaning or other sounds	If tube is in proper place, no sound is possible
Chest radiograph	Generally reliable but can be misleading
End-tidal CO_2 measurements	CO_2 not persistently detected if esophagus is intubated
Aspiration technique	Tracheal location with patent tube if 30–40 mL of air is aspirated without resistance; probable esophageal location if unable to aspirate syringe easily or delayed bulb refill
Fiberoptic bronchoscope	Reliable if tracheal rings are seen down endotracheal tube
Lighted stylet down endotracheal tube	Reliable if transillumination seen in low midline neck

undetected. Because irreversible cerebral anoxia occurs within minutes, conservative airway management maneuvers should be limited to 2 to 3 minutes; failure to achieve adequate oxygenation should lead to a quick decision to intubate.

As a guide, intubation attempts should be limited to the amount of time a single deep breath can be held. This is especially important in a child, because the functional residual capacity of a child's lungs is less than that of an adult. Historically, the maximum recommended duration of an intubation attempt in an apneic patient has been 30 seconds, followed by a period of bag-valve-mask ventilation before intubation is attempted again. Longer intubation attempts are permissible, however, when guided by accurate data from an oxygen saturation monitor. Oxygen saturation may remain in the normal range for minutes, especially in patients who have been adequately pre-oxygenated. Assuming optimal preoxygenation to a saturation >98%, attempts at intubation should be halted for bag-mask ventilation whenever the O_2 saturation drops below 92%.

Assessment of tube location is the top priority immediately after its passage. The best assurance of tracheal placement is for the clinician to see the tube pass through

the vocal cords. Techniques to assess tube placement are discussed earlier. Another method of reliably determining tracheal tube location uses the fiberoptic scope. Passage of the scope through the tube with visualization of tracheal rings confirms ET placement as well as the position within the trachea. The placement of a lighted stylet down the tracheal tube and successful transtracheal illumination also reliably predicts ET placement. [40]

The clinician should be aware of the potential for vomiting following removal of a tube from the esophagus. Cricoid pressure should be applied during tube removal and maintained until intubation is successful. Alternatively, the first tube can be left in the esophagus to serve as temporary gastric venting until tracheal intubation is achieved. Suction must be readily available should vomiting occur.

Although seldom associated with serious complications, unrecognized positioning of the ET tube tip in the right mainstem bronchus may cause hypoxia as well as unilateral pulmonary edema.[41] A chest radiograph should be taken shortly after the intubation to confirm tube positioning. Endobronchial intubation was clinically unrecognized without a chest film in 7% of prehospital intubations in one study. [42] Persistent asymmetrical breath sounds after correct tube positioning suggests unilateral pulmonary pathology (e.g., main stem bronchus obstruction, pneumothorax, or hemothorax).

Prolonged efforts to intubate may also cause cardiac decompensation. Pharyngeal stimulation can produce profound bradycardia or asystole; when feasible, an assistant should follow the cardiac rhythm throughout the intubation. Atropine should be available to reverse vagal-induced bradycardia that may occur secondary to suctioning or laryngoscopy. Prolonged pharyngeal stimulation also may result in laryngospasm, bronchospasm, and apnea.

Complications may also result from the application of cricoid pressure. Cricoid deformation is proportional to the force applied during the application of cricoid pressure. This deformation may result in expiratory obstruction in up to 56% of patients at 44 Newtons but only 2% at 30 Newtons. [43] However, BURP maneuvers may increase obstruction even at low pressure.

One should check for loose or missing teeth before and after orotracheal intubation. Any avulsed teeth not found in the oral cavity warrant a postlaryngoscopy chest film to rule out aspiration of a tooth. Swallowed teeth are of no consequence. Broken teeth are the most common complication of laryngoscopy. [44] Laceration of the mucosa of the lips, especially the lower lip, may also occur. Tracheal or bronchial injuries are rare but serious, usually occurring in infants and the elderly as a result of decreased tissue elasticity. [45]

Vomiting with aspiration of gastric contents is another serious complication that can occur during intubation. Case reports of both ARDS and chronic lung disease are thought to be due to the aspiration of activated charcoal. [46] [47] Patients who are obtunded, at risk for seizures, or vomiting should be considered for tracheal intubation before the administration of activated charcoal.

Controversy exists regarding exacerbation of cervical spine injuries during orotracheal intubation in the trauma patient. A cadaveric study of intubation under fluoroscopy showed that the greatest degree of motion occurs at the

occiput-C1 junction and decreases with each sequential interspace. [48] Manual in-line immobilization did not significantly limit motion at any level in this study. Neither manual in-line immobilization nor traction significantly reduced motion at a surgically created ligamentous injury at the C4–C5 level. Complete immobilization of the cervical spine likely cannot be completely achieved by means readily available in the ED, but attempts should still be made to minimize movement with manual in-line stabilization and use of proper intubation technique. However, concern for a cervical spine injury should not deter the clinician from securing a patient's airway when clinically warranted. Exacerbation of a cervical spine injury remains largely a theoretical concern, while in contrast, inadequate ventilation and failure to adequately secure the airway occurs regularly in the emergency setting.

Intubation may also be complicated by a persistent air leak. This is generally caused by failure of either the cuff or pilot balloons or by positioning the cuff balloon between the vocal cords. If the cuff balloon is leaking, the tracheal tube must be replaced (see Changing Tracheal Tubes later in this chapter). If the pilot balloon is determined to be leaking, however, this can usually be remedied without changing the tube. [49] An incompetent one-way balloon valve can be fixed by placing a stopcock into the inflating valve. Reinflation of the cuff followed by shutting off the stopcock should solve the problem. If the leak involves the pilot balloon itself, or if the distal inflation tube has been inadvertently severed, cut off the defective part and slide a 20-ga catheter into the inflation tube. Then connect the stopcock to the catheter, inflate the cuff, and close the stopcock.

Tracheal stricture used to be a significant late complication of long-term intubation with low-volume high-pressure cuffs. The standard use of high-volume low-pressure cuffs has markedly decreased the incidence of this complication. [50] Tubes with high-pressure cuffs are obsolete and should be avoided.

Summary

Orotracheal intubation is the primary method of definitive airway management. In the comatose patient, it is usually accomplished rapidly and without difficulty. The easy intubation is frequently successful in the hands of the novice; the difficult intubation often proves challenging even for the experienced clinician. Rapid-sequence induction has increased the use of orotracheal intubation as the first-line approach in various clinical situations and settings (see Chapter 5). Once the patient's breathing and protective reflexes are removed, however, the clinician has the supreme responsibility of safely reestablishing them. A mastery of the technique of orotracheal intubation is essential.

MODIFIED OROTRACHEAL INTUBATION

Intubation with an Intermediate Airway in Place

Esophageal Obturator/Gastric Tube Airway in Place

The unconscious patient requiring ventilatory assistance may benefit from temporary use of the esophageal obturator airway (EOA) or similar device, as described in Chapter 3 . Although the EOA may provide effective ventilation, it is, at best, a temporary measure. An ET tube generally provides more safe, convenient, and effective airway control. Therefore, the EOA should be replaced as soon as the patient's clinical condition permits and personnel skilled in ET intubation are available.

Removal of the EOA from the esophagus often results in gastric regurgitation. Therefore, ET intubation must be performed around the EOA to protect the patient from aspiration. The process is begun by hyperventilating the patient through the EOA. The EOA mask is then removed, and the EOA tube is moved to the left side of the patient's mouth. Laryngoscopy and intubation are then performed in the usual fashion. The EOA balloon may cause resistance to passage of the tracheal tube, requiring the volume of the EOA balloon to be reduced. After passage of the orotracheal tube, the clinician deflates the EOA balloon completely and slides it out of the patient's esophagus. If resistance is met, the clinician must be sure that the esophageal cuff has been deflated completely.

Esophageal-Tracheal Combitube (ETC) in Place

Combitubes placed in the esophagus will generally require replacement with a tracheal tube. The inflated pharyngeal balloon prevents tracheal intubation around this airway. This proximal balloon must be deflated before attempting tracheal intubation. If intubation is still not possible, the ETC may need to be removed; the stomach should first be emptied via a gastric tube placed through the esophageal port of the airway. Suction is readied, the distal balloon is deflated, and the patient is quickly intubated.

Laryngeal Mask Airway in Place

The trachea can often be intubated with the laryngeal mask airway (LMA) left in place. The various approaches to intubation with an LMA in place are discussed in Chapter 3 (see Figs. 3-11).

Bullard Laryngoscope

A recent development for intubating the difficult airway is the Bullard laryngoscope, an anatomically shaped rigid fiberoptic laryngoscope that provides an indirect view of the larynx (Fig. 4-13). Because no manipulation of the neck is necessary, it is especially well suited for the patient with potential cervical spine injury. Indeed, in the anesthetized patient, the Bullard laryngoscope has been found to cause less head extension and cervical spine extension than the conventional laryngoscope. [51] The addition of an intubating stylet attached to the laryngoscope has resulted in increased ease and speed of intubation, and the technique appears to be effective regardless of the patient's head and neck anatomy.[52] Because alignment of the oropharyngeal and laryngeal axes is not required, the Bullard laryngoscope offers the advantage provided by a conventional fiberoptic scope but requires less training to gain proficiency in its use. [53]

Indications and Contraindications

The Bullard laryngoscope is indicated in patients with anticipated difficult airways who require definitive airway control. It can be used in awake as well as unresponsive patients.[54] Marked impairment of mouth opening is a contraindication to the use of a laryngoscope. However, because the Bullard laryngoscope follows the contour of the mouth and hypopharynx, only 2 cm of occlusal opening is necessary for the

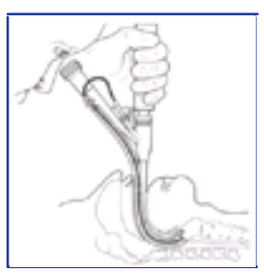

Figure 4-13 Bullard laryngoscope. Anatomically shaped laryngoscope visualizing the glottis; tracheal tube is mounted on the attached stylet for easy tracheal passage. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

introduction of the scope and an ET tube. The laryngoscope itself requires only 6 mm of mouth opening for insertion. [55]

Procedure

The technique for introducing the Bullard laryngoscope blade is similar to that for direct laryngoscopy. The clinician, standing at the patient's head, opens the mouth with the left thumb while holding the head stable. As the scope blade is introduced into the oropharynx, the handle is rotated to follow the curve of the hypopharynx until the handle is fully vertical. The tip of the blade can be used to lift the epiglottis, but visualization of the larynx is usually possible without this maneuver. Only minimal force is exerted along the axis of the handle. Intubation of the larynx can be accomplished using a styletted ET tube or an ET tube with a directional tip (Endotrol; Mallinckrodt, Critical Care, Glens Falls, NY). The technique is generally successful when using the new Bullard intubating stylet. [52]

Awake intubation using the Bullard laryngoscope can be performed comfortably using topical anesthesia and light IV sedation. [54] Adult and pediatric Bullard laryngoscopes are available, and the scope has been used successfully in neonates. [53] The Bullard scope can also be used in conjunction with nasotracheal intubation to visualize proper tube placement.

Complications

The major difficulty in using the Bullard laryngoscope is the inability to visualize the larynx because of blood, emesis, or secretions. Another reason for failure is the inability to place the blade tip under the epiglottis. [52]

Summary

The Bullard scope is useful in the difficult airway uncomplicated by blood and excessive secretions. In the all-too-common setting of blood and secretions, however, the inability to visualize the vocal cords significantly limits the utility of this device in emergency airway management.

NASOTRACHEAL INTUBATION

Nasotracheal intubation was first described by Magill in the 1920s. The tube may be placed blindly or with the aid of a laryngoscope or bronchoscope. Blind nasotracheal intubation can be one of the more technically demanding airway approaches, with the outcome being heavily dependent on the skill and experience of the clinician. The primary advantage of the blind technique is that it minimizes neck movement and does not require opening the mouth.

General Indications and Contraindications

Nasotracheal intubation is technically more difficult than oral intubation, but it has definite advantages. It is especially suitable for the patient with a short, thick neck or other anatomic characteristics that would make orotracheal intubation difficult. It also requires minimal preparation.

Blind nasotracheal intubation is possible with the patient in the sitting position, a distinct advantage when intubating the patient with congestive heart failure who cannot tolerate lying flat. In fact, patients in respiratory distress are the easiest to intubate blindly because their air hunger results in increased abduction of the vocal cords, which facilitates tube entry into the trachea.

A nasotracheal tube has advantages that extend beyond the immediate difficulties of airway control. The patient cannot bite the tube or manipulate it with the tongue. Oral injuries may be cared for without interference by the tube. A nasotracheal tube is more easily stabilized and generally easier to care for than an orotracheal tube. It is better tolerated by the patient, permitting easier movement in bed, and produces less reflex salivation than do oral tubes.

Nasal intubation should be avoided in patients with severe nasal or midface trauma. In the presence of a basilar skull fracture, a nasotracheal tube may inadvertently enter the cranial cavity. [56] The technique should be avoided in patients in whom thrombolytic therapy is being considered. Nasal intubation is relatively contraindicated if the patient is taking anticoagulants or is known to have a coagulopathy. Because the sound of air passing though the vocal cords guides proper tube placement, nasotracheal intubation is not recommended in patients without spontaneous respirations.

Blind Placement

Blind nasotracheal intubation is the most common form of nasotracheal intubation in the emergency setting. Danzl and Thomas reported a success rate of 92% in a large series of ED patients, but success rates are highly dependent on clinician skill. [57]

Indications and Contraindications

Any patient requiring airway control who has spontaneous respirations is a candidate for blind nasotracheal intubation. Specific indications that favor this approach over others are (1) short, thick neck, (2) inability to open the mouth, (3) inability to move the neck, (4) gagging or resisting the use of the laryngoscope, and (5) oral injuries.

Apnea is the major contraindication to blind nasotracheal intubation. Attempts to place the tube without respirations as a guide are futile. Relative contraindications include basilar skull fracture and nasal injury. [56] [58] Furthermore, significant bleeding may occur if the patient is receiving anticoagulants or has a coagulopathy. Blind nasotracheal intubation should be avoided in patients with expanding neck hematomas. Patient combativeness, if not controlled with sedation, is also a contraindication.

Some would argue that inability to open the mouth is a relative contraindication, because emesis may be induced and the vomitus could not be cleared. The clinician must exercise judgment in the individual case and be prepared to use neuromuscular blocking agents or bypass the upper airway with a surgical technique if such a complication develops.

Procedure

The patient is placed in the "sniffing" position with the proximal neck slightly flexed and the head extended on the neck. In preparation for intubation, the clinician constricts the nasal mucosa of both nares, using either 0.25% to 1.0% phenylephrine drops, oxymetazoline (Afrin) spray, or 4% cocaine spray. Topical anesthesia of the nares, oropharynx, and hypopharynx with lidocaine spray (10%) is also indicated if time permits. If available, cocaine is ideal because it is both a vasoconstrictor and an anesthetic—caution is necessary in hypertensive patients. The most patent nostril is chosen. In the cooperative patient, this can be determined simply by occluding each nostril and asking the patient which one is easier to breathe through. The most patent nostril can also be identified by direct vision, or by gently inserting a gloved finger lubricated with viscous lidocaine, full length into the nostrils. If time is not an issue, an effective method to dilate the nasal cavity and administer the anesthetic is to pass a lidocaine gellubricated nasopharyngeal airway (nasal trumpet) into the selected nostril. This airway is left in place for several minutes, and progressively larger trumpets are introduced.

After preparation of the nostril, a well-lubricated 7.0 or 7.5 ET tube is inserted along the floor of the nasal cavity. The tube is not directed cephalad, as one might expect from the external nasal anatomy, but rather is directed straight back toward the occiput, along the nasal floor. Twisting the tube may help bypass soft tissue obstruction in the nasal cavity. It is sometimes recommended that the tube's bevel be oriented toward the septum to avoid injury to the inferior turbinate. However, such an event is rare. At 6 to 7 cm, one usually feels a "give" as the tube passes the nasal choana and negotiates the abrupt 90° curve required to enter the nasopharynx. This is the most painful and traumatic part of the procedure and must be done gently. If resistance persists despite continued gentle pressure and twisting of the tube, the passage of a suction catheter down the tube and into the oropharynx may allow for successful passage of the tube over the catheter. [59] If this fails, the other nostril should be tried. In an attempt to avoid this difficulty from the outset, a controllable-tip tracheal tube (Endotrol, Mallinckrodt Medical Inc, St Louis) may be used. The tube allows the clinician to increase the flexion of the tube, facilitating passage past this tight curve. One study found that the Endotrol tube enhanced first-attempt success with blind nasotracheal intubation. [60] A study of paramedic-performed blind nasotracheal intubation reports success rates of 58% using standard ET tubes versus 72% success with directional tip control ET tubes. [61]

As the tube advances through the oropharynx and hypopharynx and approaches the vocal cords, breath sounds from the tube become louder and fogging of the tube may occur. At the point of maximal breath sounds, the tube is lying immediately in front of the laryngeal inlet. The tube is most easily advanced into the trachea during inspiration, when the vocal cords are maximally open. As the patient begins to breathe in, the tube is advanced in one smooth motion. If a gag reflex is present, the patient usually coughs and becomes stridulous during this maneuver, suggesting successful tracheal intubation. The absence of such a response should alert the clinician to probable esophageal passage. If there is a delay in advancing the tube, oxygen can be added to the end of the tube to increase inspired oxygen. Once the tube is in the trachea, vocalization should cease. Persistent vocalizations suggest esophageal intubation. Breath sounds coming from the tube and tube fogging are other signs of ET placement. Reflex swallowing during blind nasotracheal intubation may direct the tube posteriorly toward the esophagus. If this occurs, the conscious patient should be directed to stick out the tongue to inhibit swallowing and prevent consequent movement of the larynx. Application of laryngeal pressure may also help avoid esophageal passage.

Following intubation, both lungs are auscultated while positive-pressure ventilation is applied. If only one lung is being ventilated, the tube is withdrawn until breath sounds are heard bilaterally. The optimum distance from the external nares to the tube tip is about 28 cm in males and 26 cm in females. [62] After verification of tracheal placement, the cuff is inflated and the tube is secured.

Technical Difficulties

The nasotracheal tube may slide smoothly through the hypopharynx and into the trachea on the first pass. Unfortunately, this is not always the case; in an operating room series, the first attempt was successful in <50% of cases. [63] When the initial pass is unsuccessful, there are 4 potential locations of the tip of the tube: (1) anterior to the epiglottis in the vallecula, (2) on the arytenoid or vocal cord, (3) in the piriform sinuses, or (4) in the esophagus.

Observation and palpation of the soft tissues of the neck during attempted passage of the nasotracheal tube are helpful in finding the misplaced tube. Before reattempting placement, the tube is withdrawn slightly; it is not removed from the nose, because this will create additional trauma to the nasal soft tissues. The possibility of cervical spine injury must be kept in mind when considering corrective maneuvers. Any maneuver that moves the neck significantly should not be used if alternatives are available. Methods for achieving success when difficulties with tube placement are encountered include the following.

Anterior to the epiglottis.

Difficulty advancing the tube beyond 15 cm or palpation of the tube tip anteriorly at the level of the hyoid bone suggests an impasse anterior to the epiglottis in the vallecula. Withdrawing the tube 2 cm, decreasing the degree of neck extension, and readvancing the tube will frequently remedy this problem.

Arytenoid cartilage and vocal cord.

Contrary to the classic teaching, [64] recent studies have demonstrated a propensity for a nasotracheal tube, when placed through the right nares, to lie posteriorly and to the right as it approaches the larynx. [55] [65] It is not surprising, then, that the most common obstacles to advancement of the nasotracheal tube are the

Figure 4-14 Common problem with blind tracheal tube passage through the larynx. *A,* Tip of tube caught on the arytenoid cartilage. *B,* Rotation of tube 90° counterclockwise orients the bevel of the tip posteriorly and allows passage into the larynx. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, MN.)*

right arytenoid and vocal cord. No data are available on the common obstacles encountered if the tube is placed in the left nares. If the tube appears to be hanging up on firm, cartilaginous tissue, withdraw the tube 2 cm, rotate it 90° counterclockwise, and readvance the tube. This maneuver orients the bevel of the tube posteriorly and frequently results in successful passage (Fig. 4-14). Another technique is to pass a suction catheter down the tube; it often will pass through the larynx without difficulty and the tube can then be advanced over the catheter (Fig. 4-15). [66] [67]

Piriform sinus.

Bulging of the neck lateral and superior to the larynx indicates tube location in a piriform sinus. The tube should be withdrawn 2 cm, rotated slightly away from the bulge, and readvanced. An alternate method is to tilt the patient's head toward the side of the misplacement and reattempt placement. [68]

Esophageal placement.

Esophageal placement is indicated by a smooth passage of the tube with the loss of breath sounds. The larynx may be seen or felt to elevate as the tube passes under it. Assisted ventilation will usually produce gurgling sounds when the epigastrium is auscultated. The tube should be withdrawn until breath sounds are clearly heard, and passage should be reattempted while pressure is applied

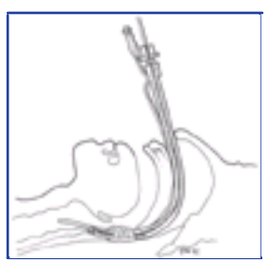

Figure 4-15 Use of suction catheter to aid in passage of nasotracheal tube caught at laryngeal inlet. The suction catheter is passed down the tracheal tube and into the trachea. The tracheal tube is then passed over the suction catheter, and the catheter is removed. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

to the cricoid. Increased extension of the head on the neck during placement may help. If attempts continue to result in esophageal misplacement, the following maneuver may result in successful tracheal intubation: from the precise point at which breath sounds are lost, the ET tube is withdrawn 1 cm. The cuff is inflated with 15 mm of air, resulting in an elevation of the tube off the posterior pharyngeal wall and angling it toward the larynx. The tube is then advanced 2 cm; continued breath sounds indicate probable intralaryngeal location. At this point, the cuff is deflated and the ET tube is advanced into the trachea (Fig. 4-16). This technique may be particularly useful in the patient with cervical spine injury, because it requires no manipulation of the head or neck. [69] This maneuver, when used on the first pass in 20 patients in the operating room, was successful in 75% of cases. [63] One should bear in mind, however, that these patients were paralyzed and thus did not experience the laryngospasm that may be encountered in a breathing patient. The use of topical anesthesia is recommended. Alternatively, if a controllable-tip ET tube (Endotrol) is used, the tip can be flexed anteriorly to help avoid esophageal placement. [60] Remember that the tip is very responsive to pulling on the ring. A common mistake is to exert too much force on the ring, resulting in the tube curling up short of the larynx, thus preventing tube advancement. There has been a case report of an Endotrol tube "kinking" at the point of sharpest curvature, causing difficulty with suctioning but no problems with ventilation. [70]

Laryngospasm.

Laryngospasm is common when attempting nasotracheal intubation. It is usually transient. The tube is withdrawn slightly and the clinician should wait for the patient's first gasp; advancement of the tube at this precise moment is frequently successful, as the vocal cords are widely abducted during forced inhalation. Laryngeal anesthesia should

Figure 4-16 Use of tracheal tube cuff inflation to aid in nasotracheal intubation. *A,* Tracheal tube is pulled back after esophageal passage. *B,* Once breath sounds are heard, cuff is inflated with 15 mL of air and is readvanced into the laryngeal inlet. Once seated in the inlet, the cuff is deflated and the tube is advanced into the trachea. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

also be assessed, and if IV and nebulized lidocaine have already been administered without success, transcricothyroid anesthesia (e.g., 2 mL of 4% lidocaine) should

be considered.[71] Occasionally, a jaw lift is necessary to break prolonged spasm. Another option is to use a smaller tube.

Placement Under Direct Vision

This technique combines elements of oral and nasotracheal intubation. The indications and precautions are similar, and the importance of considering cervical spine injury is identical. Likewise, the need for jaw opening by physical or pharmacologic means is unchanged. This method is preferred to orotracheal intubation if the presence of an orotracheal tube might interfere with the repair of an oral injury. It is also useful when blind nasotracheal intubation has failed.

Preparation of the nose and nasopharynx and passage of the tube into the oropharynx are the same as described for blind nasotracheal intubation. It is with the introduction of the laryngoscope that the technique changes.

Laryngoscopy, as described with orotracheal intubation, is used to visualize the vocal cords and the tip of the ET tube. With the Magill forceps in the right hand, the ET tube is grasped proximal to the cuff (to avoid damage to the balloon) and directed toward the larynx (Fig. 4-17). An assistant advances the tube gently while the clinician directs the tip into the larynx and trachea. Cricoid pressure may facilitate the passage. Often the larynx can be manipulated sufficiently with the laryngoscope so that the clinician can advance the tube with the right hand and guide it between the cords without using the Magill forceps. Occasionally, the natural curve of the tracheal tube guides it through the cords without any manipulation. The cuff is inflated, and both lungs are auscultated to ensure ventilation. When placement is satisfactory, the tube is secured.

Complications

Epistaxis is the most common complication of nasotracheal intubation. However severe epistaxis was encountered in only 5 of 300 cases reported by Danzl and Thomas.[57] Tintinalli and Claffey reported severe bleeding in 1 of 71 cases and less serious bleeding in 12 others. [72] Bleeding is usually not a problem unless it provokes vomiting or aspiration, a serious potential problem in obtunded patients with a clenched jaw or a

Figure 4-17 Nasotracheal intubation with the aid of a laryngoscope and Magill forceps. Note that the forceps do not pull the tube—they only serve to guide the tip of the tube through the vocal cords while an assistant advances the tube. The cuff is frequently damaged if it is grasped.

decreased gag reflex. Other immediate complications include turbinate fracture, intracranial placement through basilar skull fracture, retropharyngeal laceration or dissection, and delayed or unsuccessful placement. [56] [73] [74] [75] Complications may be minimized by selection of a smaller tube and by gentle technique.

Sinusitis in patients with nasotracheal tubes is common and can be an unrecognized cause of sepsis. [76] Rare but potentially fatal delayed complications include mediastinitis following retropharyngeal abscess [77] and massive pneumocephalus.[78]

Because most complications occur during tube advancement through the nasal passage and proximal nasopharynx, the complications of blind nasotracheal intubation and placement under direct vision are largely the same. However, retropharyngeal laceration and esophageal intubation are more of a threat in blind placement techniques because they are more likely to go unrecognized. [72] One unique problem associated with nasotracheal intubation is damage of the tube cuff with the Magill forceps.

Summary

Nasotracheal intubation is being used less frequently than in the past because clinicians are increasingly comfortable using oral intubation in the patient with potential cervical spine injury. In addition, emergency clinicians frequently use paralytics to facilitate orotracheal intubation. Nevertheless, nasotracheal intubation remains an effective and potentially life-saving approach to the difficult airway and should be a dependable part of the armamentarium of all clinicians who are active in emergency airway management.

GUIDED INTUBATION TECHNIQUES

Digital Intubation

Digital intubation uses the index and middle fingers to blindly direct the ET tube into the larynx. It is particularly well adapted to the prehospital situation in which a trapped victim cannot be positioned for intubation. A prehospital series of 66 digitally intubated patients demonstrated an 89% success rate. [79]

Indications and Contraindications

Digital intubation is indicated in the deeply comatose patient whose larynx cannot be visualized and who has a contraindication to nasotracheal intubation. Advantages include speed and ease of placement, immunity to anatomic constraints and other difficulties visualizing the larynx, and little neck movement. Contraindications are primarily precautions to protect the clinician: Digital intubation should not be attempted on any patient who presents a significant risk of biting. This includes the calm and awake patient as well as the agitated patient.

Procedure

The patient's head and neck are placed in neutral position. The clinician stands at the patient's right side, facing the patient. The clinician's left index and middle fingers are introduced into the right angle of the patient's mouth and are slid along the surface of the tongue until the epiglottis is palpated. The tip of the epiglottis is palpated at 8 to 10 cm from the corner of the mouth in the average adult. The use of a stylet in the tube is optional; the largest reported series had good success without a stylet.[79] For the clinician with short fingers or a patient with an anterior larynx, a stylet is advantageous. If a stylet is used, it is placed in the tube and bent into the form of an open "J" with the distal end terminating in a gentle hook. A lubricated tube is introduced from the patient's left between the tongue and the rescuer's two fingers (Fig. 4-18). The tube is cradled between two fingers and the tip is guided beneath the epiglottis. Gentle anterior pressure directs the tube into the larynx. If the clinician has sufficiently long fingers, they can be placed posterior to the arytenoids, acting as a "backstop" for the tube to both avoid esophageal passage and to assist in laryngeal placement. [80] If a stylet has been used, it is withdrawn at this time while simultaneously advancing the tube. An alternative to using a stylet for directing the tube anteriorly is to select an ET tube with a controllable tip (Endotrol, Mallinckrodt Medical Inc, St Louis).

A variation on the technique of digital intubation has been described for intubating the newborn. [81] Only the index finger is used to guide the tube into the larynx. The end of the tube is bent and both the tube and the finger are moistened with sterile water. The index finger of the nondominant hand follows the tongue posteriorly to easily palpate the epiglottis and paired arytenoids. The thumb of the same hand may be

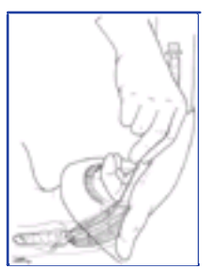

Figure 4-18 Digital intubation. The tracheal tube is cradled between the index and middle fingers and guided into the glottic opening. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

used to apply cricoid pressure to steady the larynx. The ET tube is held in the dominant hand and advanced using the non-dominant index finger as a guide (Fig. 4-19). The tube snugs up (encounters subtle resistance) as it enters the trachea, and palpation of the tube through the trachea provides further confirmation of correct placement. A styletted tube, shaped in the form of a J, is usually desired until familiarity with the procedure is achieved.

Complications

The risk of esophageal intubation is always present and, being a blind procedure in deeply comatose or cardiac arrested patients, the potential for esophageal misplacement is increased. If used in patients with a gag, induction of emesis with aspiration is a possibility. A high incidence of left main stem intubations was noted in a cadaveric study, [82] but clinical confirmation is lacking. The greatest risk seems to be to the clinician, whose fingers may be bitten.

Summary

While most recent experience with digital intubation in adults has been prehospital, there is no reason why it should be confined to this setting. The majority of moribund ED patients who defy orotracheal intubation are never given a trial of digital intubation. This omission undoubtedly deprives some patients of expeditious airway management.

Lighted Stylet Intubation

This technique uses a battery-operated lighted stylet that is placed in an ET tube and used to guide the tube into the trachea by transilluminating the soft tissues of the neck. First described in 1957 by MacIntosh and Richards, [83] it was designed to aid in managing the difficult airway. It has also been shown to be a useful means of determining the position of the tracheal tube. [40] Some newer devices incoporate fiberoptic capabilities or audio feedback to facilitate intubation. [84]

In the operating room, the Tube Stat lighted stylet (Concept Corp, Clearwater, FL) has been 99% to 100% successful. [85] [86] The requirement that the overhead lights be dimmed during the procedure has limited its use in most emergency settings. In a small prehospital study, 88% of patients were successfully intubated by clinicians using a lighted stylet.[87] The majority of the failures occurred in the setting of bright sunlight and in patients who had vomited. A

Figure 4-19 Digital intubation in the neonate. The tube is guided using only the index finger to palpate the epiglottis and laryngeal inlet. A stylet is optional. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

newer device (Trachlight, Laerdal, Inc, Starger, Norway) with a brighter light source and adjustable length, appears to have solved this problem. In a series of 96 patients, many with a history of difficult intubation, all but 1 were successfully intubated in ambient light with this device using either the oral or nasotracheal route. [88] Consistent with other series, the only failure was in a morbidly obese patient. Several studies have shown that time to intubation is equivalent or shorter using a lighted stylet vs standard orotracheal intubation. [84]

Indications and Contraindications

The patient with a difficult airway in whom direct laryngoscopy has failed is a candidate for light-guided tracheal intubation. A multiple trauma patient with airway

bleeding is a prime example. The patient who has been pharmacologically paralyzed and cannot be intubated with direct laryngoscopy is another example. The lighted stylet may also be helpful in successfully completing a difficult nasotracheal intubation. One advantage of this technique over nasotracheal intubation is that it can be used in the apneic patient.

Because lighted stylet intubation is a blind approach, it should be avoided in patients with expanding neck masses and patients with airway compromise presumed due to a foreign body. Massive obesity is the most common cause for failure with this technique because of the difficulty transilluminating the generous soft tissue.

Preparation

The function of the bulb of the lighted stylet should be checked before use. The patient's head should be placed in a neutral or, if cervical spine injury is not a concern, the sniffing position. The awake patient should have the oropharynx and hypopharynx sprayed with lidocaine and sedation should be administered as indicated.

Procedure

The lubricated lighted stylet is inserted into a tracheal tube 2.5 mm or larger (pediatric stylets are available) until the bulb lies just distal to the side port but not protruding from the end of the tube.[84] This unit is bent in the shape of a hockey stick with a 90° curve beginning just proximal to the tube cuff. The clinician stands at the head of the patient. When this is not possible, the patient can be approached from either side. The tongue is grasped with gauze and pulled forward. Another means of exposing the oropharynx is to grasp the jaw between

Figure 4-20 Lighted stylet intubation. Use of the Trachlight for endotracheal intubation using transillumination of soft tissues as a guide to placement. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

the thumb and the fingers (Fig. 4-20). The light is turned on and the unit is inserted into the mouth, following the curve of the tongue into the oropharynx. A transilluminating glow indicates the location of the tube tip. Application of cricoid pressure may enhance transillumination. [40] The overhead light should routinely be dimmed if feasible. Positioning is optimal when the glow emanates from the midline at the level of the hyoid bone. Holding the lighted stylet steady, the tube is slid off and advanced into the trachea. If the glow is located elsewhere, the unit should be withdrawn 2 cm or cocked back and repositioned as indicated by the light. If no light is seen, the tube is in the esophagus and should be pulled back, laryngeal pressure applied, and, if necessary, the head extended slightly. After passage, the tube should be checked for correct positioning and then secured.

Complications

Earlier reports noted complications resulting from an equipment failure and lost bulbs, but these technical problems have been corrected. No complications have been noted in the recent literature, but this may only reflect the limited use of this technique in uncontrolled settings. A blinded study of pathologic specimens did not show any evidence of burn injury in the tracheal mucosa in cats intubated with the Trachlight. [84]

Summary

Lighted stylet intubation is a safe, rapid, and highly successful method that has a definite place in the management of the difficult airway. Improvements in the device have made its use more practical in most settings in which emergency airway management is performed.

Intubation Over a Fiberoptic Bronchoscope

Use of the flexible fiberoptic bronchoscope as an aid to tracheal intubation is an alternate method of in airway management in the ED. In this setting, success approximates 80%, with the most common cause of failure being the inability to visualize the glottis secondary to blood and secretions. [89] [90]

Flexible fiberoptic endoscopy is often the best method for intubating the awake patient with a difficult airway. It can be accomplished using the nasal or oral route and is better tolerated than direct laryngoscopy. It also may be effective in the comatose patient when more conventional methods have failed. It provides excellent visualization of the airway and permits the evaluation of the airway before tube placement. The greatest obstacle to success is obscured visualization secondary to blood, secretions, or fogging. The expense of the equipment, its fragility, and the time required to achieve technical proficiency are additional drawbacks.

Indications and Contraindications

Common indications for emergency fiberoptic intubation include the unstable cervical spine, expanding neck masses, upper airway infection, facial and airway burns, and anticipation of a difficult intubation due to abnormal anatomy. It may also be helpful in guiding blind nasotracheal intubation that is initially unsuccessful.

Contraindications to fiberoptic nasotracheal intubation are similar to those for nasotracheal intubation: severe midface trauma and coagulopathy. Although there are no clear contraindications to fiberoptic orotracheal intubation, active airway bleeding and vomiting are relative contraindications because successful fiberoptic intubation is rarely achieved in these settings. If the clinician is inexperienced in fiberoptic intubation, apnea is another relative contraindication to its use.

Equipment

Fiberoptic scopes are graded according to their external diameter (in millimeters). A practical size for an intubating scope is 3.5 mm. Although it is physically possible to pass a 4.0 mm (0.5 mm larger) tracheal tube over the scope, the fit is quite tight. As a rule, the tracheal tube should be at least 1 mm larger than the intubating scope. The size of the working channel—the port to which suction or oxygen is applied and through which fluid or catheters may be passed—is another important dimension when evaluating fiberoptic scopes. Large working channels are desirable.

Procedure

The optimal positioning of the neck is in extension, as opposed to the cervical flexion desired when using direct laryngoscopy. Extension allows for better visualization of the glottis by elevating the epiglottis off the posterior pharyngeal wall. This is especially pertinent in the comatose patient who lacks the muscle tone necessary to maintain an open airway. Problems with the tongue and soft tissues falling back and obscuring fiberoptic scope view are effectively managed by applying a jaw lift or pulling the tongue forward and away from the soft palate and posterior pharyngeal wall. This maneuver also moves the epiglottis away from the posterior pharyngeal wall, facilitating exposure of the cords. Extending the head on the neck may accomplish the same objective.

Nasotracheal approach.

The nasal approach is preferred to the oral approach because the angle of insertion allows for easier visualization of the larynx and because patient cooperation is not as critical. Also, in the unconscious patient, the tip of the scope is less likely to impinge on the base of the tongue with a nasal approach.

The nose is prepared using a vasoconstrictor and topical anesthetic agent as described for nasotracheal intubation. Using an aerosolized anesthetic agent, it is important to obtain sufficient hypopharyngeal anesthesia to minimize gagging and laryngospasm once the procedure begins. The well-lubricated ET tube may be placed in the nostril first, and the scope passed through it, or the ET tube can be mounted over the scope and the scope first passed through the nostril. The advantage of the former is that it avoids the possibility of nasal secretions covering the scope and obscuring the view. Disadvantages are that nasotracheal placement may cause bleeding and, in some patients, the tube may not pass easily into the nasopharynx.

The most patent nostril is prepped and the ET tube is advanced until it makes the bend into the nasopharynx in the manner described under nasotracheal intubation. If negotiating this bend is difficult, a well-lubricated fiberoptic scope can be placed through the tube and into the oropharynx to serve as a guide for the ET tube. Once the tracheal tube is in the oropharynx, thorough oropharyngeal suctioning should be performed before introducing the scope into the ET tube. The fiberoptic scope is then advanced toward the larynx; the epiglottis and vocal cords are seen with little or no manipulation of the tip of the fiberoptic scope in 90% of patients. [91] As the scope is advanced, the cords are kept in view by frequent minor adjustments of the scope tip.

In the comatose or obtunded patient, the tongue and other soft tissue may obscure the view of the larynx; this can be alleviated by having an assistant pull the tongue forward or apply a chin or jaw lift. The scope is advanced through the larynx to the level of the midtrachea and the ET tube is passed over the firmly held fiberoptic scope into the trachea (Fig. 4-21). It is helpful to remember that in adults the average distance from the naris to the epiglottis is 16 to 17 cm; if the scope has been advanced much beyond this distance and the glottis is still not seen, the scope is probably in the esophagus. [92] If the scope meets resistance at about this same level and only a pink blur is visible, the scope tip is probably in a piriform sinus; transillumination of the soft tissues may be present to confirm this as well as to indicate what corrective maneuvers are necessary.

The greatest impediment to successful fiberoptic intubation is the inability to visualize the larynx because blood or secretions have covered the optical element and cannot be removed. The best time to suction is before introducing the fiberoptic scope, actively suctioning the oropharynx just before scope insertion. Once the scope is in place, minor secretions can be suctioned through the fiberoptic suction port. Significant blood and secretions, however, are best removed by insufflation of oxygen through the suction port and out the tip of the scope, serving simultaneously to remove blood and secretions, defog the tip, and increase the inspired O_2 content. The setup required for insufflation should be immediately available, if not already attached to the suction port before scope insertion. Once the scope has entered the trachea, difficulty may be encountered in advancing the ET tube into the trachea. The tip of the tube most commonly catches on the right arytenoid cartilage or vocal cord; withdrawing the tube 2 cm, rotating it counterclockwise 90°, and readvancing the tube should result in successful tracheal intubation (see Fig. 4-14).

Orotracheal approach.

Oral fiberoptically guided intubation is indicated when contraindications to nasal intubation are present, the most common being severe midface trauma, or when the clinician is more comfortable with this approach. The oral approach is more difficult than the nasal approach because the path of the scope is less defined by the surrounding soft tissue and the tip of the scope is more likely to impinge on the base of the tongue or vallecula. Attention to keeping the scope in the midline and elevating the soft tissue by pulling the tongue forward or applying the jaw lift will minimize this difficulty. Another disadvantage of the oral approach is that the oropharyngeal axis is not as well aligned with the larynx as is the nasopharyngeal axis and thus more scope manipulation is required to visualize the larynx.

The drawbacks of the oral approach can be minimized by using an oral intubating airway. This adjunct resembles an oropharyngeal airway but is longer and has a cylindrical passage through which the fiberoptic scope and tracheal tube are passed. The tip of this airway lies just cephalad to the epiglottis and assures midline positioning and a predictable place from which to advance the scope (Fig. 4-22).

The patient must be adequately anesthetized or obtunded to minimize the gag reflex. Topical anesthesia is achieved by

Figure 4-21 Fiberoptic nasotracheal intubation. Note use of the fiberoptic scope as a guide over which the tracheal tube is passed. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

spraying a 4 or 10% solution of lidocaine into the oropharynx. A degree of laryngeal and tracheal anesthesia may be achieved by a transoral spray using the laryngeal tracheal anesthetic (LTA) set. EMLA cream is also safe and effective in achieving topical anesthesia of the airway. [93] Time permitting, the tongue should first be anesthetized using a small globule of EMLA on a tongue depressor left in place for 1 to 2 minutes. Next, a cotton tip applicator may be used to apply the cream to the remainder of the oropharynx. This procedure should be repeated until the gag reflex is greatly diminished or absent or a total 4 grams of cream are used. This dose has been shown not to result in lidocaine toxicity. A well-lubricated fiberoptic scope, premounted with an ET tube, is then placed through the oral intubating airway and the trachea is fiberoptically intubated. The ET tube is advanced over the scope into the trachea, frequently requiring the same counterclockwise manipulation as described with the nasal approach. After successful intubation, the intubating device can be left in place as a bite block, or it can be removed over the ET tube after removal of the tube adapter. Some oral intubating airways can be removed from the mouth without disconnecting the ET tube adapter.

Complications

Complications of fiberoptic orotracheal intubation include prolonged intubation attempts as well as vomiting and laryngospasm in the underanesthetized patient. Oxygen saturation monitoring should alert the clinician to hypoxemia from prolonged intubation attempts. Most complications seen with fiberoptically guided nasotracheal intubation are associated with passing the ET tube through the nasopharynx. Epistaxis is most common, followed by other nasopharyngeal injuries. A rare but potentially significant complication may result if on blind advancement of the fiberoptic scope through the ET tube, the tip of the scope exits through the distal side port. [94] Attempts at passing the ET tube through the larynx will fail because the tube tip, now extending off the midline, will catch on the laryngeal structures. This complication is avoided if the scope is introduced before tracheal tube placement.

Summary

The primary advantage of fiberoptic intubation is its ability to negotiate difficult airway anatomy. It is noninvasive and well tolerated. Its major limitation in the emergency setting is lack

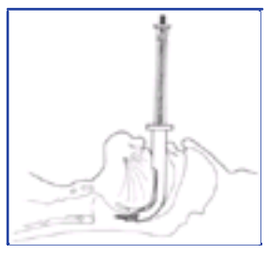

Figure 4-22 Fiberoptic orotracheal intubation. A fiberoptic scope is passed through a hollow oral airway device to help position the scope and tube and to protect them from being bitten during fiberoptic oral intubation. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

of visibility in the presence of blood and secretions. The fiberoptic scope requires more practice than other methods of airway management; the first experience using the scope should not be in the setting of an emergency airway problem. Once familiarity and facility with the scope are acquired, fiberoptic intubation can be used early in the management of the difficult airway rather than as a last resort after repeated failed attempts using conventional techniques.

Magnetically Guided Orotracheal Intubation

Magnetically guided orotracheal intubation is a novel technique of guided ET intubation. The technique uses a strong magnet over the cricothyroid membrane to guide a standard ET tube containing a stylet with a magnetized tip. A study of novice intubators reports an overall success rate of 80% using this technique on a mannequin in a rigid cervical collar. [95] The same study reports a 67% rate when the vocal cords were not visualized. A study of anesthesiologists using a similar technique in elective surgical patients reports a 92.5% first attempt success rate and 100% success within 2 attempts. [96]

Indications and Contraindications

Magnetically guided intubation may be a useful adjunct for the intubation in situations where the vocal cords are difficult to visualize due to anatomic limitations of the patient or the presence of blood or vomitus in the airway. Its use has not been studied in patients with pacemakers or implantable debrillators and should be avoided in this population.

Procedure

A flexible stylet with a magnetic tip is inserted into a standard ET tube. A laryngoscope is then inserted into the oropharynx in the standard fashion. The epiglottis must be visualized to prevent the tip of the ET tube from entering the vallecula. The vocal cords, however, do not necessarily need to be visualized for successful intubation. The ET tube is then placed in the oropharynx with its tip posterior to the epiglottis. A second magnet is then placed over the cricothyroid membrane. The ET tube is then advanced, with magnetic attraction drawing the tip anteriorly through the vocal cords. [95]

Complications

No specific complications of magnetically guided intubation have been reported. The risk of unrecognized esophageal intubation always exists. Again, the effect of the magnets on pacemakers and implantable defibrillators is unknown and use of this technique should be avoided in these patients.

Summary

Magnetically guided orotracheal intubation has not been widely studied, but appears to be a safe and effective adjunct in management of the difficult airway.

Retrograde Intubation

Retrograde orotracheal intubation is a technique of guided ET intubation using a wire or catheter placed percutaneously through the cricothyroid membrane or high trachea and exiting through the mouth or nose. An ET tube is then passed over this guide and advanced through the vocal cords into the trachea. Introduced by Butler and Cirillo in 1960, [97] the technique has undergone several recent modifications that have enhanced its value as a means of establishing a definitive airway when more conventional techniques have failed.

Indications and Contraindications

Retrograde intubation is indicated when definitive airway control is required and less invasive methods have failed. Indications include trismus, ankylosis of the jaw or cervical spine, upper airway masses, unstable cervical spine injuries, and maxillofacial trauma. It can be used to convert transtracheal needle ventilation (see Chapter 6) into a definitive airway. Its successful use has been described in a 1-month-old with developmental abnormalities. [98] It is particularly helpful in the trauma patient with airway bleeding that prevents visualization of the glottis. [99]

Contraindications to this procedure include the availability of a less invasive means of airway control and inability to open the mouth. A relative contraindication is the apneic patient who cannot be effectively ventilated using the bag-valve-mask; in this setting it is advisable to first establish transtracheal needle ventilation (see Chapter 6) before attempting retrograde intubation or to go directly to cricothyrotomy.

Equipment

Materials include the following: (1) local anesthetic and skin preparation materials, (2) 18-ga needle, (3) 60 cm epidural catheter-needle combination or 80 cm (0.88 mm diameter) spring guidewire (J-tip preferred), (4) hemostat, (5) long forceps (e.g., Magill) for grasping wire in pharynx, (6) ET tube of appropriate size, (7) syringe for tube cuff, and (8) materials for securing the tube. A prepackaged alternative is the Cook Retrograde Intubation Set (Cook Critical Care, Bloomington, IN), which also contains a sheath.

Procedure

Three anatomic landmarks must be located by palpation: the hyoid bone, thyroid cartilage, and cricoid cartilage. The skin

overlying the cricothyroid membrane is prepped and anesthetized. Next, the lower half of the cricothyroid membrane is punctured with a needle directed slightly cephalad. The bevel should also face cephalad. Air is aspirated to confirm needle tip position within the lumen of the larynx (see also Chapter 6). An alternative entry point is the high trachea, usually through the subcricoid space, using the same steps as described for the cricothyroid membrane.

The syringe is removed and the wire is then passed through the needle and advanced until it is seen in the patient's mouth, with the help of the laryngoscope, or until it exits the nose. If the wire is found in the hypopharynx, it is grasped with the Magill forceps and drawn out through the mouth. The needle is removed from the neck and the end of the wire is secured at the puncture site with a hemostat. The oral end of the wire is then threaded in through the ET tube side port—not the end of the tube—and advanced up the tube until it can be grasped by a second hemostat. Threading the wire through the side port allows the tube tip to protrude 1 cm beyond the point at which the wire enters the larynx. The wire is then pulled taut and moved back and forth to ensure that no slack remains.

The ET tube is then advanced over the wire until resistance is met. This is the most critical point in the procedure; because this is a blind technique, it may be difficult to determine whether the tube has entered the trachea or is hung up on more proximal structures. If the ET tube has successfully passed through the vocal cords and it is being restricted by the guidewire as it traverses the anterior laryngeal wall, one should feel some caudally directed tension on the wire at its laryngeal insertion point. If this does not occur, the tip of the ET tube may be proximal to the vocal cords, either in the vallecula, the piriform sinus, or abutting the narrow anterior aspect of the vocal cords. If in doubt, pull the tube back 2 cm, rotate it 90° counterclockwise, and readvance the tube. This will usually result in successful passage through the larynx. [92] When satisfied that the tube has entered the trachea, the tube should be stabilized and the guidewire pulled out through the mouth. The tube is then

advanced farther into the trachea.

The classic method of retrograde intubation, as described earlier, has undergone modifications that facilitate passage of the ET tube through the glottis. A significant advance has been the addition of a plastic sheath that is passed antegrade over the wire until it meets resistance where the wire penetrates the laryngeal mucosa (Fig. 4-23).[100] This sheath needs to be stiff enough to effectively guide an ET tube, yet small enough to easily pass through the vocal cords without impinging on supraglottic or glottic structures. Once the sheath comes to rest against the anterior laryngeal wall, the wire is withdrawn from the mouth and the sheath is advanced. With the sheath well within the trachea, the ET tube is passed over the sheath. Any resistance that may be encountered at the arytenoids or vocal cords can usually be remedied by pulling the tube back 1 to 2 cm and rotating it counterclockwise 90°. One advantage of the antegrade sheath is that it lies freely in the larynx, allowing for a more posterior passage through the widest distance between the cords. The wire, however, pulls the ET tube anteriorly toward the narrow commissure of the vocal cords and is more likely to result in impingement of the tube on the cords. Also, the use of the sheath permits unrestricted advancement of the ET tube, whereas a wire entering the larynx 1.0 to 1.5 cm below the vocal cords prevents the tube from advancing more than this distance before removal of the wire.

If no sheath is available, consider placing the needle inferiorly in the subcricoid space, thereby increasing the distance the ET tube can be advanced before being stopped by the wire.[101] This will decrease the likelihood of dislodging the ET tube tip when the guidewire is withdrawn.

Up to this point, blind retrograde intubation has been described. A further modification of the technique allows for visualization using a fiberoptic scope. [102] In addition to the scope, an extra-long guidewire (e.g., 125 cm, 0.025 cm Teflon-coated J-wire) is also required. The procedure is the same as previously described up to the point at which the wire is withdrawn from the mouth. At that point, with an ET tube mounted on a lubricated fiberoptic scope, the long guidewire is passed retrograde up through the end of the fiberoptic scope and out the suction port. The fiberoptic scope is then advanced over the guidewire and through the cords, coming to rest against the anterior laryngeal wall (Fig. 4-24). The wire is withdrawn from the suction port and the scope is advanced into the trachea. The ET tube is then slid off the fiberoptic scope, and visualization guarantees correct ET placement. The scope is then withdrawn and the lungs are auscultated.

Complications

The complications of retrograde intubation are largely related to cricothyroid membrane puncture (see Chapter 6). Hemorrhage is minimized by taking care to puncture the cricothyroid membrane in its lower half (to avoid the cricothyroid artery). Subcutaneous emphysema may occur, but it is of no clinical significance because no air is insufflated during this technique. A small incidence of soft tissue infection is reported with translaryngeal needle procedures, but this can be minimized by ensuring that the wire is withdrawn from the mouth rather than the neck.

The final complication, the failure to achieve intubation, has been mitigated by the addition of the antegrade sheath over the wire.

Summary

Retrograde intubation is an underused technique for achieving ET intubation in a patient who cannot be intubated by less aggressive means. It is more invasive than fiberoptic intubation but requires less skill. Whereas retrograde intubation usually takes several minutes to complete, [80] the patient can undergo bag-mask ventilation through much of the procedure. Recent modifications in the technique guarantee this method a prominent place in the management of the difficult airway, particularly when active bleeding compromises the airway.

CHANGING TRACHEAL TUBES

The tracheal tube with a leaking cuff is a vexing problem, especially if the original intubation was difficult. A method of replacing the tube without losing control of the tracheal lumen is preferred. This can be achieved by passing a guide down the defective tube, withdrawing the tube while leaving the guide in place, and introducing a new tube over the guide and into the trachea.

A number of different guides have been described (e.g., simple nasogastric tubes, 18 Fr Salem sump tubes [Table 4-5], feeding tubes), but they are poor substitutes for a designated tube exchanger such as the TTX "tracheal tube exchanger" (Sheridan Catheter Corporation, Argyle, NY) or a similar commercially available device. The advantages of the

Figure 4-23 Retrograde intubation using guidewire and antegrade sheath. *A*, Placement of a needle through the cricothyroid membrane (note: needle bevel is oriented cephalad). *B*, Placement of the J-wire directed cephalad through the translaryngeal needle. *C*, Passing antegrade sheath over the guidewire. *D*, Removal of guidewire with advancement of sheath into trachea. *E*, Advancement of tracheal tube over sheath into trachea. *F*, Removal of the sheath. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

Figure 4-24 Fiberoptic scope as an aid in retrograde intubation. Once the retrograde wire (long) is placed and retrieved superiorly, the wire is threaded through the tip of the fiberoptic scope and out through the suction port. The scope is then advanced until it passes through the vocal cords, stopping at the anterior laryngeal wall. The wire is withdrawn through the suction port, the fiberoptic scope is advanced into the trachea, and the tracheal tube advanced over the scope into the trachea. The fiberoptic scope is then withdrawn. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

designated tube exchanger are that it is stiff enough to prevent dislodgment when the ET tube is introduced, it is ready to use without modification, it has a printed scale to aid in determining depth of placement, and if replacement is prolonged, the patient may be oxygenated using the exchanger and wall oxygen.

Procedure

Before the procedure, the patient is properly sedated or restrained. The patient is hyperventilated before placing the guide through the existing tube. The guide is lubricated and advanced into the defective tube so that it is well within the tracheal lumen (adults, 30 cm). While applying cricoid pressure (Sellick maneuver), the defective tube is withdrawn over the guide, and care is taken not to dislodge the guide when removing the tube. The replacement tube is then slid over the guide and is gently advanced into the trachea (Fig. 4-25). At this juncture, it may be helpful to perform a jaw thrust or chin lift to facilitate passage through the pharynx. Resistance may be encountered at the laryngeal inlet or vocal cords; if this occurs, withdraw the tube 1 to 2 cm, rotate it 90° counterclockwise, and readvance it. With the tube clearly in the trachea, remove the guide, inflate the cuff, and ventilate the patient. After correct placement has been verified, the new tube can be secured.

Benumof describes a technique in which a bronchoscope with an ET tube jacketed over the proximal end is first passed into the trachea with the defective ET tube still in place.[103] A tube exchanger capable of jet ventilation is then passed through the defective tube. The defective tube can then be withdrawn and the new ET tube can be passed over the bronchoscope. The tube exchanger can be withdrawn once placement of the new tube is confirmed. This technique permits direct visualization of the placement of a new tube and a fail-safe means to ventilate the patient should difficulties arise.

Complications are related to the time required to change the tube. A successfully performed procedure can be accomplished within 30 seconds. Laryngeal injury from forcing the guide or the tube is a possibility to consider when replacing a tube.

CONCLUSION

Airway management in the critically ill or injured patient with acute airway compromise is a most demanding task for the emergency clinician. Mastery of oral and nasotracheal

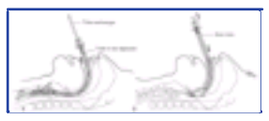

Figure 4-25 Replacing damaged tracheal tube. *A,* Placement of tracheal tube exchanger down damaged tube and deep into trachea (*open arrow*) followed by removal of tracheal tube (*solid arrow*). *B,* Replacement tube is passed over tube exchanger into trachea, followed by removal of tube exchanger. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

TABLE 4-5 -- Procedure for Changing an Orotracheal Tube Using a Salem Sump Nasogastric Tube as a Guide

1. Cut off the proximal 6- to 8-cm flared end of an 18 Fr Salem sump nasogastric tube.
2. Test the balloon or cuff of the new tracheal tube. An 8.0-mm tracheal tube should be used. Remove the proximal adapter from the new tracheal tube.
3. Sedate the patient and restrain the hands as necessary. Preoxygenate as much as possible.
4. Lubricate the entire length of the nasogastric tube with water-soluble (K-Y, Lubrifax) lubricant.
5. Advance the nasogastric tube as far as possible into the trachea through the existing tracheal tube.
6. Deflate the balloon of the existing tracheal tube and remove it. *Now only the nasogastric tube remains in the trachea, to be used as a guiding stylet.*
7. Thread the proximal end of the nasogastric tube through the distal end of the new tracheal tube. This is facilitated by the use of a hemostat.
8. Advance the new tracheal tube over the guide until the nasogastric tube exits through the proximal opening in the new tracheal tube.
9. Grasp the exiting nasogastric tube, have an assistant *lift the patient's jaw*, and advance the tracheal tube over the guide into the trachea. If resistance is encountered, rotate the tube 90° counterclockwise.
10. Remove the guide.
11. Replace the adapter on the new tracheal tube, inflate the cuff, and ventilate the patient. Confirm proper placement of the new tube.

intubation and a facility with various guided intubation techniques, plus competence with the surgical approaches, is necessary to meet all emergency situations that might occur. Preparation (including mastery of technique), advance preparation of equipment, and experience in clinical decision-making are essential. Scenario visualization is one means of practicing the difficult decisions that will afford the patient the most expeditious and safest means of airway control available under the circumstances.

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Chapter 5 - Pharmacologic Adjuncts to Intubation

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Endotracheal intubation in the acute care setting presents a challenge distinct from that associated with intubation of the fasted, premedicated patient in the operating room (OR). The emergency department (ED) patient is frequently uncooperative and unstable and may have medical problems that are completely unknown to the treating clinician. Often within a matter of minutes, the clinician is expected to simultaneously accomplish many formidable tasks: assess and control the airway, as well as to diagnose and manage other life-threatening problems.

In 1979, Taryle and colleagues reported that complications occurred in 24 of 43 patients intubated in a university hospital ED. They called for improved house officer training in endotracheal intubation, as well as "more liberal use of the procedures used in the OR, such as sedation and muscle relaxation." [1] Since the report of Taryle and others, training programs in both critical care and emergency medicine have been greatly expanded, resulting in significant improvement in the expertise of clinicians who provide acute airway management.[2] Simultaneously, the use of established pharmacologic adjuncts to intubation previously available only in the OR has increased.[3] [4] [5] [6] [7] [8] [9] In addition, new drugs that are potent, rapid-acting, and safer have been developed, giving the clinician greater ability to tailor therapy to specific clinical problems.[10] [11] Because of these developments, clinicians now may not only concentrate on the manual skill of intubation, but also skillfully use drugs to achieve specific objectives. These objectives may include (1) immediate airway control necessitating induction of anesthesia and muscle relaxation; (2) provision of analgesia and sedation to the awake patient; and (3) minimization of intubation adverse effects, including systemic and intracranial hypertension.

This chapter reviews the pharmacology and use of the drugs that are currently available to facilitate intubation in the acute care setting.

RAPID-SEQUENCE INDUCTION OVERVIEW

In the critically ill patient who may be hypoxic, hemodynamically unstable, agitated, uncooperative, and at risk of further deterioration, it is frequently necessary to gain immediate control of the airway. In recent years the technique of rapidly inducing anesthesia (rapid-sequence induction [RSI]) as a means of airway control has gained broad acceptance among emergency clinicians. RSI as practiced in the ED is a modification of the process initially described in the anesthesia literature and used to minimize the risk of aspiration in patients with full stomachs who require anesthesia. There is also a subtle but significant modification of the intent of RSI as practiced in the ED. Whereas anesthesiologists have used RSI to intubate patients requiring anesthesia, emergency clinicians commonly use RSI to induce anesthesia in patients requiring intubation.

Airway control in the ED is obviously not the same as planned intubation prior to general anesthesia in the OR. While some techniques and medications are similar, ED airway control cannot simply be accomplished by "calling anesthesia." The anesthesia team may serve as backup, but it is not standard that airway control await the arrival of an off-site team. What is standard is that the total process is accomplished with the personnel and resources available in the ED, with the prudent use of anesthesia specialists in select cases and for procedures and equipment not available in all settings. As described by Stept and Safar, [12] general anesthesia RSI includes 13 steps, several of which are not practical or relevant to the ED setting. In the ED, RSI is begun by placing the patient on 100% oxygen, for at least 2 to 3, and ideally 5, minutes. The intent is to denitrogenize the lungs and build an oxygen reserve that will last several minutes should intubation prove to be difficult. Under optimal conditions, breathing 100% oxygen for 3 minutes has been demonstrated to maintain acceptable oxygen saturation for 8 minutes. [13] The same study demonstrated that 4 maximal breaths of 100% oxygen from a face mask maintained acceptable saturation for 6 minutes. Comparable results should not be expected in the ED setting because of differences in the underlying health and cooperation of the patient population. While preoxygenation should be maintained for the longest period practical prior to beginning intubation, the ideal situation and circumstances are not always present, and clinical judgment is the deciding factor for this portion of RSI.

During the period of preoxygenation, the airway should be assessed to determine the likelihood of a difficult intubation while establishing an intravenous (IV) line, placing the patient on cardiac and pulse oximetry monitors and assembling all necessary equipment for oral intubation and potentially cricothyrotomy. A defasciculating dose of a non-depolarizing neuromuscular blocking agent is commonly recommended 2 to 3 minutes prior to administration of succinylcholine, although the advantages of this time-consuming step are poorly documented and generally overstated. General anesthesia is then rapidly induced with one of the agents discussed later in this section, followed immediately by muscle paralysis with succinylcholine at a dose of 1.5 to 2.0 mg/kg. Cricoid pressure is applied by an assistant as consciousness is lost. After the onset of adequate relaxation, orotracheal intubation is performed, and correct tube placement is verified. Cricoid pressure can then be released and ventilation with 100% oxygen begun. Mask ventilation prior to intubation is unnecessary if adequate preoxygenation occurs. In fact, mask ventilation should be avoided because of potential gastric distention and passive regurgitation. Mask ventilation should be used only if adequate oxygenation cannot be ensured and then should be performed gently in association with cricoid pressure. If a first intubation attempt is unsuccessful, bag-mask ventilation with cricoid pressure may temporize until a repeat orotracheal attempt or an alternative approach to intubation is made. In the event of failure to expeditiously accomplish orotracheal intubation, cricothyrotomy generally should be performed in order to secure the airway. The protocol for ED-based RSI is described in Table 5-1 .

Endotracheal intubation and RSI have also expanded beyond the ED into the prehospital setting. Using an RSI protocol of a sedative plus paralytic for non-cardiac arrest patients, success rates at intubation in the field have ranged from 92% to 98%, [3] [14] [15] [16] which is similar to rates achieved in the ED.[15] As in the ED setting, without a full complement of medications, prehospital intubation becomes significantly more difficult with success rates dropping to approximately 60%[14] [17] largely due to patient combativeness, preexisting muscle tone, and intact airway protective reflexes.

The impact of prehospital intubation on outcome remains controversial. Gausche and colleagues compared prehospital bag-mask ventilation and endotracheal intubation for critically ill and injured pediatric patients. [17] Their protocol did not include the use of sedatives or paralytics and had a 57% intubation success rate. A total of 820 subjects were analyzed for survival and neurologic outcome. The two groups were statistically equivalent (26% to 30% survival with 20% to 33% of those having a good neurologic outcome). Rates of vomiting, aspiration, and airway trauma were likewise similar. This study was performed in an urban environment with short Emergency Medical Service (EMS) transport times, which may limit its generalizability. Nonetheless, it has raised important and as yet unanswered questions

TABLE 5-1 -- Rapid-Sequence Induction Protocol

1. Preoxygenate (denitrogenize) the lungs by providing 100% oxygen by mask. If ventilatory assistance is necessary, bag gently while applying cricoid pressure.
2. Assemble required equipment:
• Bag-valve-mask connected to an oxygen delivery system
• Suction with Yankauer tip
• Endotracheal tube with intact cuff, stylette, syringe, tape
• Laryngoscope and blades, in working order
• Cricothyrotomy tray
3. Check to be sure that a functioning, secure IV line is in place.
4. Continuously monitor the cardiac rhythm and oxygen saturation.
5. Premedicate as appropriate:
• *Fentanyl:* 2 to 3 µg/kg given at a rate of 1 to 2 µg/kg/min IV for analgesia in awake patients
• *Atropine:* 0.01 mg/kg IV push for children or adolescents (minimum dose of 0.1 mg recommended)
• *Lidocaine:* 1.5 to 2 mg/kg IV over 30 to 60 seconds
6. Induce anesthesia with one of the following agents administered intravenously: thiopental, methohexital, fentanyl, ketamine, etomidate, or propofol. Apply cricoid pressure.
7. Give succinylcholine 1.5 mg/kg IV push (use 2 mg/kg for infants and small children).
8. Apnea, jaw relaxation, and/or decreased resistance to bag/mask ventilations (use only when pre-RSI oxygenation cannot be optimized by spontaneous ventilation) indicate that the patient is sufficiently relaxed to proceed with intubation.
9. Perform endotracheal intubation. If unable to intubate during the first 20-second attempt, stop and ventilate the patient with the bag-mask for 30 to 60 seconds. Follow pulse oxymetry readings as a guide.
10. Treat bradycardia occurring during intubation with atropine 0.5 mg IV push (smaller dose for children; see item 5).
11. Once intubation is completed, inflate the cuff and confirm endotracheal tube placement by auscultating for bilateral breath sounds and checking pulse oxymetry and capnography readings.
12. Release cricoid pressure and secure endotracheal tube.

about the necessity of prehospital endotracheal intubation if the airway is able to be adequately managed by other means.

In contrast, Winchell and Hoyt, in a retrospective review of 1092 blunt trauma patients with a Glasgow Coma Scale (GCS) score of less than 9, showed that prehospital intubation reduced mortality from 36% to 26% with the impact most pronounced among the most severely injured. [18] As with the previous study, this one also evaluated orotracheal intubation without any pharmacologic adjunct with 66% of subjects able to be intubated in the field.

Bochicchio and colleagues compared brain-injured patient outcomes in patients with and without prehospital RSI. [18A] They found that patients receiving prehospital intubation versus those intubated at the hospital had a higher mortality rate and more ventilator days. Although the equivalence of the patient groups upon paramedic arrival is unknown, this study does suggest that prehospital RSI and intubation may adversely affect outcomes. Further prospective evaluation with evaluation of prehospital physiology and notation of preexisting aspiration will be required for resolution of this prehospital controversy.

The two general types of drugs used most commonly as part of RSI protocols are anesthetic agents and neuromuscular blocking agents. These are discussed in greater detail in the following section.

ANESTHETIC AGENTS

A number of diverse drugs are routinely used in the ED to induce anesthesia prior to intubation. These include thiopental, methohexital, ketamine, etomidate, and propofol. Midazolam and fentanyl may also be used, but these two agents are more commonly used at low doses to facilitate procedural sedation (see Chapter 34). The choice of a particular anesthetic agent depends to a great extent on the experience and training of the clinician, patient hemodynamic factors, and to a certain extent on institutional protocols governing use of these agents. Drugs commonly used and their doses are listed in Table 5-2 .

Barbiturates: Thiopental and Methohexital

The barbiturates, particularly thiopental, have been the traditional agents used for RSI in the OR. These agents are used less often in the ED setting because of their reputation as cardiorespiratory depressants. Of these two agents, methohexital is used more commonly in the ED because of its extremely rapid onset and short duration of action.

Pharmacology

Following IV injection, the ultrashort-acting barbiturates bind rapidly to plasma proteins, particularly albumin. Unbound

TABLE 5-2 -- Recommended Anesthetic Doses for Rapid-Sequence Induction

Drug*	Dose
Thiopental	3–5 mg/kg IV
Methohexital	1–3 mg/kg IV
Fentanyl	5–15 µg/kg IV
Ketamine	1–2 mg/kg IV
Etomidate	0.3 mg/kg IV
Propofol	2 mg/kg IV

*Any one of these can be used before the administration of a neuromuscular blocking agent to induce anesthesia (see text).

barbiturate quickly accumulates in highly vascular organs, reaching peak brain levels in as short a time as 50 seconds. The drugs then diffuse from the brain, ultimately reaching equilibrium between the intracerebral and plasma concentrations. Degradation occurs primarily in the liver, producing inactive metabolites that are excreted in the urine or gut, depending on the drug used. Single-pass hepatic clearance is substantially higher for methohexital than for thiopental, which accounts for the former drug's shorter duration of action. The period of anesthesia following a single IV dose of methohexital is 4 to 6 minutes, compared with 5 to 10 minutes for thiopental. [19] [20]

Barbiturates are central nervous system (CNS) depressants that are capable of producing mild sedation to deep coma. They do not block afferent sensory impulses to a significant extent and therefore should be used in conjunction with an analgesic agent such as fentanyl if a painful procedure is to be performed. However, it is common practice to intubate patients who have received only barbiturates. [20]

The advantages of barbiturates as adjuncts to intubation include their high potency, rapid onset, and short duration of action, traits they share with fentanyl and midazolam. The barbiturates are also known to reduce cerebral metabolism and oxygen consumption and, secondarily, cerebral blood flow and intracranial pressure (ICP). [21] [22] For this reason, thiopental is considered the agent of choice for anesthesia induction and maintenance in patients with elevated ICP. Some have stated that thiopental is the drug of choice to temporarily anesthetize the hypertensive patient with a head injury before intubation. It has not been proved, however, that barbiturates exert a protective effect on the CNS when used for a short period of time during RSI. Moreover, their use in trauma patients may lead to systemic hypotension and impaired cerebral perfusion pressure that may offset the theoretic advantages of barbiturate therapy.

Dose

The recommended dose of thiopental is 3 to 5 mg/kg intravenously administered as a 2.5% solution over 60 seconds. Normal saline should be used as a diluent. Methohexital is given at 1 to 3 mg/kg intravenously over 30 to 60 seconds.

Adverse Effects

It has been stated that barbiturates are "fatally easy" to use. [20] This is an overstatement that reflects improper use of the drugs more than an inherent danger associated with their proper use. The most significant complication of barbiturate therapy is depression of the vasomotor center and myocardial contractility leading to significant hypotension. One study showed an average decrease in mean arterial pressures during RSI with thiopental to be 40 mm Hg. [23] This may be particularly pronounced in the presence of hypovolemia or cardiovascular disease.

Barbiturates also depress the brainstem respiratory centers when given rapidly or in large doses. This effect may be accelerated by simultaneous treatment with opioids. Patients with asthma or chronic bronchitis may experience bronchospasm. Laryngospasm may occur in patients who were anesthetized lightly with barbiturates during manipulation of the upper airway. Laryngospasm usually responds to positive-pressure ventilation or paralysis with succinylcholine. In addition, the high pH of the barbiturate solution can cause tissue necrosis following extravascular administration and severe pain, vessel spasm, and thrombosis following intra-arterial infusion.

Etomidate

Etomidate is an ultrashort-acting nonbarbiturate hypnotic agent that has been used successfully as an anesthesia induction agent in Europe since the mid-1970s and in the United States since 1983. Only recently has it been used as an adjunct to intubation in the ED. A potentially significant benefit of etomidate in the emergency setting is its lack of cardiode-pressant effects. [24] [25] Several smaller, case series have now demonstrated its safe and effective use in ED RSI, [26] [27] [28] [29] [30] and extensive experience exists with its use in anesthesia induction for both pediatric and adult patients.

Pharmacology

Etomidate is a carboxylated imidazole that is both water- and lipid-soluble. The drug rapidly accumulates in vascular organs, reaching peak brain concentrations within 1 minute of IV infusion. [31] Sleep is produced within 1 arm-brain circulation time and lasts less than 10 minutes following a single bolus infusion. [32] Redistribution of the drug is quite rapid (distribution half-life, 2.6 minutes), which accounts for the short duration of action. Etomidate is rapidly hydrolyzed in the liver and plasma,

forming an inactive metabolite excreted primarily in the urine. [31]

Etomidate acts on the CNS to stimulate ?-aminobutyric acid receptors and depress the reticular activating system. After IV infusion, etomidate produces electroencephalographic (EEG) changes similar to those produced by barbiturates as patients pass rapidly through light to deep levels of surgical anesthesia. Because etomidate has no analgesic activity, [31] it should be used in conjunction with an analgesic such as fentanyl when painful conditions are being treated; although, as with barbiturates, it is commonly used as a sole sedative agent for intubation. Etomidate decreases cerebral oxygen consumption, cerebral blood flow, and ICP but appears to have minimal effects on cerebral perfusion pressure. [33] At therapeutic doses, etomidate is characterized by hemodynamic stability without significant changes in mean arterial pressures, [25] [26] [29] although a slightly increased heart rate may be observed. [34]

Dose

The recommended dose is 0.3 mg/kg intravenously. There is virtually no accumulation of the drug, and anesthesia may be maintained through repeated or continuous dosing.[35]

Adverse Effects

The most common side effects of etomidate are nausea and vomiting, pain on injection, and myoclonic jerks. [36] Pain on

injection occurs in up to two thirds of cases. Use of a large vein, simultaneous saline infusion, and opioid premedication are reported to reduce this side effect. [37] Myoclonic activity has been reported in about one third of cases and is believed to be caused by disinhibition of subcortical activity rather than CNS stimulation. Furthermore, myoclonic activity does not represent seizure activity. [31] However, myoclonic jerks may be misinterpreted as seizure activity.

Since first reported in the early 1980s, prolonged infusions of etomidate have been associated with suppression of adrenal function. When used for sedation over periods of days, the drug is linked with increased mortality in critically ill and injured patients presumably due to insufficient cortisol production. [38] [39] Suppression of adrenal function is due to competitive enzyme inhibition by etomidate during the cortisol biosynthesis pathway. Several small case control studies have attempted to examine the impact of singles doses of etomidate on adrenal function. These have shown some degree of altered function that may persist for up to 12 to 24 hours after even a single dose. However, as yet, no evidence exists to suggest that this is clinically significant. [28] [30] [40]

Ketamine

Pharmacology

Unique among anesthetic agents currently in use, ketamine produces a dissociative anesthesia characterized by excellent analgesia and amnesia despite the appearance of wakefulness. As a drug that is potent and relatively safe and possesses a rapid onset and brief duration of action, ketamine fits the profile of a drug that could be used effectively to facilitate intubation. It does, however, possess a number of pharmacologic properties that limit its use to selected circumstances.

Ketamine is a water- and lipid-soluble drug with rapid penetration into the CNS. Like the barbiturates, ketamine accumulates rapidly in highly vascular organs and then undergoes redistribution. The half-life of redistribution from plasma to peripheral tissues is 7 to 11 minutes, and the half-life of elimination is 2 to 3 hours. Degradation occurs primarily in the liver. [41]

Unlike other anesthetic agents that depress the reticular activating system, ketamine acts by interrupting association pathways between the thalamocortical and limbic systems. Characteristically, the eyes remain open, and patients exhibit spontaneous, although not purposeful, movements. Increases in blood pressure, heart rate, cardiac output, and myocardial oxygen consumption are seen—effects that are most likely mediated through the CNS. In vitro studies indicate that ketamine is a myocardial depressant, but the CNS-mediated pressor effects generally mask the direct cardiac effects. [42] [43] Respirations are initially rapid and shallow after ketamine administration, but they soon return to normal.

Other features of ketamine anesthesia include increased skeletal muscle tone, preservation of laryngeal and pharyngeal reflexes, hypersalivation, and relaxation of bronchial smooth muscle. ICP is increased, most likely as a consequence of increased cerebral blood flow. [41]

Ketamine has been recommended for anesthesia induction in children because of its relative safety and the infrequency of postanesthesia emergence reactions in this group. There is no evidence, however, that it offers any advantage over commonly used agents. Ketamine also has been recommended for the unstable critically ill patient, because it does not depress the cardiovascular system. [44] [45] This recommendation is too vague to be useful to the clinician, and it ignores the fact that ketamine is potentially harmful in patients with cardiac ischemia (because it can increase myocardial oxygen consumption) or acute intracranial pathology (because it can increase ICP). Ketamine may be useful during hemorrhagic shock because of its cardiostimulatory effect. Its administration to patients in shock has been reported to cause a fall in blood pressure only when the shock state has been prolonged. [46] [47]

The most promising use of ketamine as an intubation adjunct has been in the setting of acute bronchospastic disease. Ketamine relaxes bronchial smooth muscle either directly, through the enhancement of sympathomimetic effects, or through the inhibition of vagal effects. Ketamine is commonly considered as an adjunct to intubation in the decompensated asthmatic patient, and the intramuscular (IM) route may be considered if IV access is impossible. Ketamine also increases bronchial secretions, which may decrease the incidence of mucous plugging commonly reported in autopsies of asthmatic patients. [48] Clinical studies have demonstrated a reduction in airway resistance and an increase in pulmonary compliance that occur within minutes of ketamine administration. [49] [50] L'Hommedieu and Arens[9] reported prompt improvement in respiratory acidosis in five asthmatics intubated with ketamine and succinylcholine.

Dose

The recommended dose of ketamine before intubation is 1 to 2 mg/kg administered intravenously over 1 minute. Anesthesia occurs within 1 minute of completing the infusion and lasts 5 to 10 minutes. A small dose (0.5 to 1 mg/kg) may be given 5 minutes after the initial dose if there is a need to maintain anesthesia. The simultaneous administration of succinylcholine and midazolam is recommended to provide adequate muscle relaxation and to decrease the incidence of postanesthesia emergence reactions, respectively. The IM dose for intubation has not been well studied, but a suggested dose is 4 mg/kg. Onset of action occurs within 2 to 3 minutes. The deltoid muscle is theoretically the preferred site because of its good vascularity.

Adverse Effects

A side effect that has greatly limited the use of ketamine is its tendency to produce postanesthesia emergence reactions, a characteristic that it has in common with the structurally similar drug phencyclidine. The reactions may be marked and distressing to the patient; symptoms include floating sensations, dizziness, blurred vision, out-of-body experiences, and vivid dreams or nightmares. The reported incidence of these reactions varies from 5% to 30%. [51] [52] They are less common in children than in adults.

Of the drugs that have been evaluated for their ability to suppress postanesthesia emergence reactions, the benzodiazepines show the most promise. Both diazepam and lorazepam are useful, but the latter is more effective, most likely owing to its enhanced amnestic effect. [53] [54] [55] [56] Midazolam, likewise, is highly effective at doses of 0.07 mg/kg,[57] and may be the preferred agent as it has potent amnestic effects and a short duration of action. White [58] reported a 55% incidence of postemergence dreaming in patients receiving ketamine and complete suppression of dreaming with the addition of midazolam. Evidence also suggests that midazolam may inhibit the cardiostimulatory effects of ketamine.

Although ketamine produces excellent analgesia and is relatively safe, its use as an agent to facilitate intubation is somewhat limited. The widely held belief that aspiration does not occur with ketamine because of preservation of pharyngeal and laryngeal reflexes is incorrect. [55] [60] Moreover, ketamine does not relax skeletal muscle. The production of desired intubation conditions often requires the simultaneous administration of a paralytic agent, thereby removing any upper airway reflexes.

Propofol

ED experience with propofol is limited, and it is uncertain whether it will have a significant role as an adjunct to intubation in this setting.

Pharmacology

Propofol is an alkylphenol sedative-hypnotic recently introduced for induction and maintenance of general anesthesia. The drug has no analgesic activity, but it does have an amnestic effect. It produces dose-dependent depression of consciousness ranging from light sedation to coma. Propofol is a highly lipophilic, water-insoluble compound that undergoes rapid uptake by vascular tissues, including the brain, followed soon afterward by redistribution to the muscle and fat. The drug is metabolized by the liver and excreted in the urine. [61] [62] [63]

Dose

Following an induction dose of 2 mg/kg intravenously, hypnosis occurs within 1 minute and lasts for 5 to 10 minutes. A smaller dose (1 to 1.5 mg/kg) is recommended in the elderly and when simultaneously administering other CNS depressants. Because propofol has a short duration of action and patients rapidly regain consciousness, repeat bolusing is not a practical way to maintain a desired level of anesthesia or sedation. [61] [62] [63] A slow drip infusion of 3 to 5 mg/kg per hour titrated to effect is the preferred technique. Conscious sedation may be achieved using a drip infusion beginning at 6 mg/kg per hour and *decreasing* the rate as the desired level of sedation is obtained. [64] [65]

Adverse Effects

Side effects of propofol include direct myocardial depression causing a moderate fall in blood pressure, particularly in the elderly, in hypovolemic patients, and when administered simultaneously with opioids. Propofol reduces cerebral blood flow and may cause mild CNS excitation activity (e.g., myoclonus, tremors, hiccups) during anesthesia induction. Pain on injection occurs commonly, even when the drug is infused slowly. [61] [62] [63]

NEUROMUSCULAR BLOCKING AGENTS

Overview

Neuromuscular blocking agents (NMBs) are the standard agents used to achieve muscle relaxation for intubation, because they permit complete airway control and greatly simplify visualization of the vocal cords. This is particularly important when intubation must be performed quickly under less than ideal circumstances. Sedatives may be used to provide some muscle relaxation, but this requires that they be administered rapidly and in large doses, risking depression of the cardiovascular system. The combination of a paralytic agent and a sedative or analgesic agent is generally superior to the use of either agent alone. A 1999 report showed an 18% failure-to-intubate rate with sedative alone compared with a 0% failure rate for sedatives plus paralytics. Procedural complication rates such as significant airway trauma and aspiration were also markedly higher in the group receiving sedation only. [66]

The only absolute contraindication to the use of NMBs is the inability to manage the airway once the patient becomes apneic. Although not absolutely contraindicated, it is relatively inhumane to paralyze and intubate an alert patient. A sedative or an analgesic agent should always be administered simultaneously if the patient is able to perceive pain.

Controversy surrounds the use of NMBs to facilitate intubation in the patient who may have a cervical spine injury. Despite claims to the contrary, there is no evidence that orotracheal intubation performed during in-line stabilization, with or without paralysis, is dangerous. Cadaver studies [67] [68] [69] purporting to demonstrate the dangers inherent in this practice simply do not simulate the condition of a living patient accurately. [70] Conversely, no studies prove conclusively that orotracheal intubation following the use of NMBs is a safe practice, but it has been performed for a number of years in ORs without reported detrimental effects, even in patients with unstable cervical spine fractures. [71] [72]

NMBs are classified as either depolarizing or nondepolarizing. Depolarizing agents mimic the action of acetylcholine (ACh), producing a sustained depolarization of the neuromuscular junction during which muscle contraction cannot occur. Nondepolarizing agents competitively block the action of acetylcholine at the neuromuscular junction and prevent depolarization. NMBs in common use and their dosages, onset, and duration of action are listed in Table 5-3 .

Depolarizing Agents

Succinylcholine

Pharmacology.

The standard depolarizing agent currently in use is succinylcholine, which was introduced in 1952. It has a chemical structure similar to that of acetylcholine and is therefore able to depolarize the postjunctional neuromuscular membrane. Administration is followed by a brief period of muscle fasciculation that corresponds to the initial membrane depolarization and muscle fiber activation. Unlike acetylcholine, which is released in minute amounts and hydrolyzed in milliseconds, succinylcholine requires several minutes for significant hydrolysis to occur. During this time, the neuromuscular membrane remains depolarized, but the muscles relax and will not contract again until the neuromuscular end plate and adjacent sarcoplasmic reticulum return to the

TABLE 5-3 -- Commonly Used Neuromuscular Blocking Agents

Agent	Dose (mg/kg)	Onset (min)	Duration (min)
Succinylcholine	1.5	1	3–5
Pancuronium	0.1	2–5	40–60
Vecuronium	0.1	3	30–35
	0.25	1	60–120
Atracurium	0.5	3	25–35
Mivacurium	0.15	2–3	15–20
Rocuronium	1.0	1–1.5	30–110

resting state and are again depolarized. Relaxation proceeds from the small, distal, rapidly moving muscles to the proximal, slowly moving muscles. The diaphragm is one of the last muscles to relax.[73]

Succinylcholine is rapidly hydrolyzed in the serum by the enzyme pseudocholinesterase. Only a small amount ever reaches the neuromuscular junction, and that portion is quickly drawn back into the serum by a concentration gradient produced by serum clearance. The duration of action of a single dose is 3 to 5 minutes. Relaxation may be maintained by repeated IV injections or a constant infusion. Prolonged or repeated use of the drug, however, may enhance its effects at either the vagal or sympathetic ganglia. Vagal stimulation may result in bradycardia and hypotension, as well as other muscarinic effects. These effects may be seen even at normal doses, particularly in children. [74] For this reason atropine pretreatment is recommended in all small children and in adults receiving multiple doses. [75] Prolonged neuromuscular blockade is uncommon but may occur in patients with reduced pseudocholinesterase levels.

Repeat dosing may also produce desensitization blockade in which the neuromuscular membrane returns to the resting state and becomes resistant to further depolarization and succinylcholine. [76] [77] Clinically this is indicated by an unsustained contraction in response to a tetanic stimulus and response to a test dose of edrophonium. [78] In general, there is little need for repeated doses of succinylcholine. If paralysis in excess of 3 to 5 minutes is desired, agents such as vecuronium or pancuronium should be used.

Succinylcholine, despite its many potential side effects, currently is the most frequently used agent for neuromuscular blockers in RSI due to its rapid onset and offset and reliable muscle relaxation characteristics.

Dose.

The recommended dose of succinylcholine is 1 to 1.5 mg/kg given intravenously. It is better to err on the side of too large a dose, thereby guaranteeing complete relaxation and avoiding the need for repeat dosing. Current evidence also suggests that dosage calculations, at least in adolescents, should be based upon actual as opposed to lean body mass due to alterations in both volume of distribution as well as pseudocholinesterase activity. [79] Neonates and infants require a slightly higher dose of succinylcholine (2 mg/kg intravenously), due to their higher volume of distribution. [80] *It is crucial that succinylcholine be administered as a rapid bolus. Slow administration may lead to incomplete relaxation. Use of a rapid 20- to 30-mL saline flush following IV administration may enhance its desired effect.*

Adverse effects.

There are a number of potential adverse effects of succinylcholine use. These include muscle fasciculations and their side effects, hyperkalemia, stimulation of

autonomic ganglia, malignant hyperthermia, prolonged apnea, histamine release, and elevation of ICP.

As noted previously, muscle fasciculations accompany the initial depolarization of the neuromuscular membrane. They may be prevented by preadministration of a subparalytic dose (0.01 mg/kg) of pancuronium or vecuronium. Fasciculations are most prominent in muscular adolescents but are uncommon in children. Their most frequent side effect is deep, aching muscle pain that may last for several days. [81] Fasciculations of the abdominal wall may elevate intragastric pressure and cause regurgitation of stomach contents. This is an uncommon complication that most often follows overzealous bag-mask ventilation before intubation. Distention of the stomach with air and failure to perform the Sellick maneuver (i.e., firm cricoid pressure to occlude the esophagus during airway management procedures) are more likely to cause vomiting than are muscle fasciculations alone. It is also important to note that oropharyngeal manipulation of the non-paralyzed patient is far more likely to cause vomiting than are succinylcholine-induced fasciculations.

Other reported but distinctly uncommon side effects of muscle fasciculations include elevations of intraocular pressure (IOP) and displacement of skeletal fractures or joint dislocations. The clinical significance of a transient rise in IOP in a patient with a penetrating eye injury is unknown. There has never been a reported case of vitreous expulsion occurring during rapid-sequence intubation with succinylcholine, despite its widespread use in open eye surgery. [82] Indeed, paralysis will prevent spontaneous patient motor activity such as coughing or gagging, both of which are associated with a greater risk of vitreous expulsion. The degree of elevation of IOPs with administration of succinylcholine is reported to range from 3 to 8 mm Hg. [34] [83] [84] [85] Blinking alone may raise IOP by 10 to 15 mm Hg, whereas more vigorous activity such as coughing, gagging, or crying may elevate IOPs by up to 70 mm Hg. [34] Although there may be theoretical advantages to using a nondepolarizing agent in patients with penetrating eye injuries, [86] a necessary intubation using succinylcholine should never be delayed or avoided because of this theoretical concern. [87]

The precise mechanism by which succinylcholine causes hyperkalemia is unknown, but it is thought to occur secondary to the asynchronous depolarization of muscle cells and resultant cellular injury. Hyperkalemia is associated with increased extrajunctional muscle acetylcholine receptors that develop secondary to prolonged diseases of the neuromuscular system. Susceptibility may occur within 5 to 7 days and persist indefinitely. Hyperkalemia is not prevented with defasciculating doses of nondepolarizing agents. The elevation in serum potassium may occur in normal patients following standard doses, but is typically clinically inconsequential. Elevations of less than 0.5 mmol/L (mEq/L) are seen following a 1 mg/kg dose. [88] In certain pathologic states the hyperkalemic response may be as much as 5 mmol/L. These conditions include late severe burns, [89] major muscle trauma,[90] spinal cord injury, muscular dystrophy, and other upper motor neuron diseases. [91] [92] These large elevations occur only in patients who have had significant tissue injury or muscle denervation for several days or weeks before succinylcholine use. Succinylcholine is not contraindicated in the acute initial management of patients with these injuries. Succinylcholine is also not contraindicated in normokalemic patients with renal failure as the magnitude of rise in serum potassium is the same as in patients with normally functioning kidneys. [93] A recent retrospective review of succinylcholine use in 38 operative cases with moderate pre-RSI hyperkalemia (5.6 to 7.6 mmol/L) suggests that the risk of hyperkalemia-related complications may be lower than feared. [94] Regardless, succinylcholine is best avoided (given other equally effective pharmacologic options exist) in the setting of known or suspected preexisting hyperkalemia (e.g., renal failure patients not receiving regular dialysis or demonstrating a wide-QRS complex.

Malignant hyperthermia is a rare complication with an autosomal dominant inheritance pattern. It occurs in approximately 1 in 15,000 children and 1 in 50,000 adults. [95]

The clinical syndrome consists of high fever, tachypnea, tachycardia, cardiac arrhythmias, hypoxia, acidosis, myoglobinuria, and impaired coagulation. Unabated muscle contractions mediated by abnormal calcium channels are believed to be the physiologic basis for this condition. [96] Treatment includes aggressive cooling measures (see Chapter 68), volume replacement, and correction of hypoxia and acid-base and electrolyte abnormalities. Dantrolene sodium, a direct-acting skeletal muscle relaxant, has been shown to be effective in reducing the muscle hypermetabolism that causes the fever. [97]

An associated abnormal response to succinylcholine is isolated masseter spasm. [98] Barlow and Isaacs reported two cases in which masseter spasm was the first abnormality noted in fatal episodes of malignant hyperthermia. [99] Masseter spasm may be more common in patients with neuromuscular disorders such as myotonia congenita.

Concern has also been raised over the use of succinylcholine in the pediatric population due to rare case reports of hyperkalemic cardiac arrests following administration to children with undiagnosed myopathies. Succinylcholine is currently recommended for use in pediatrics only under emergency circumstances when the airway must be rapidly secured. [80]

Prolonged apnea may occur because of decreased pseudocholinesterase levels (e.g., in hepatic disease, anemia, renal failure, organophosphate poisoning, pregnancy, chronic cocaine use, advanced age, bronchogenic carcinoma, or connective tissue disorders) or, more commonly, because of the inheritance of an atypical pseudocholinesterase present in about 0.03% of the population. This atypical enzyme has both a decreased affinity for the succinylcholine molecule and a decreased ability to hydrolyze it. The period of apnea is therefore increased from 3 to 5 minutes to up to several hours. Patients with markedly decreased levels of normal cholinesterase experience only a two- to threefold increase in the duration of apnea. [100] Patients with cocaine intoxication may experience prolonged muscle relaxation if given succinylcholine, because cocaine is competitively metabolized by the cholinesterases.

The magnitude and significance of the increase in ICP that occurs with succinylcholine use remain controversial. [101] [102] [103] [104] [105] Increases in the range of 5 to 10 mm Hg have been reported by several investigators, but other researchers have shown no increase, nor is there evidence of neurologic deterioration associated with these transient elevations in ICP. Mechanisms that have been proposed to explain the elevated ICP include (1) a direct effect of fasciculations, (2) an increase in cortical electrical activity with a resultant increase in cerebral blood flow and blood volume, and (3) sympathetic postganglionic stimulation. Minton and colleagues have demonstrated that pretreatment with vecuronium (0.14 mg/kg) reduces the rise in ICP following succinylcholine administration from mean values of 5 mm Hg to 1 mm Hg.[106] It has been postulated that nondepolarizing blockade prevents muscle spindle firing and the increase in cortical activity that may lead to increased ICP. Pretreatment with a nondepolarizing agent may not be practical when intubation must be performed rapidly; furthermore, the dose that has been shown to be effective is itself a paralyzing dose and would obviate the need for succinylcholine. Limited studies have thus far been performed to evaluate the significance of this rise in ICP in a brain-injured human patient population. These have thus far shown no significant change in EEG or ICPs with succinylcholine; however, the small size of the studies limits the ability to draw conclusions about clinical outcomes. [80] [107] [108]

At present, questions concerning the safety of succinylcholine in the setting of acute intracranial pathology do not have clear answers. The drug has been used widely and successfully in this setting, and its continued use is supported by this experience. The very real risk of airway compromise and secondary cerebral insult due to hypoxia from delayed or failed intubation must always be weighed against the uncommon and theoretical harmful effects mentioned earlier.

Nondepolarizing Agents

Nondepolarizing agents act in a competitive manner to block the effects of acetylcholine at the neuromuscular junction. Drugs in this class include pancuronium, atracurium, vecuronium, mivacurium, rocuronium, and *d*-tubocurarine. These drugs, particularly the intermediate-acting agents vecuronium and atracurium, have fewer side effects than succinylcholine and have the potential for reversal. However, they generally have longer onset and duration of action than succinylcholine, making them less attractive choices for rapid intubation. *In most instances, succinylcholine remains the agent of choice to facilitate emergency intubation*, and nondepolarizing agents are indicated to maintain paralysis after intubation. Of note, the new nondepolarizing agent, rocuronium, has an onset of action within 1 to 1.5 minutes, which may permit its use for rapid-sequence intubation when succinylcholine is contraindicated.

Because nondepolarizing agents act competitively, increasing the concentration of acetylcholine may reverse their effects. Cholinesterase inhibitors such as neostigmine or edrophonium may be used, but not until some spontaneous signs of reversal are seen. Thus, the concept of reversal is of limited clinical importance in the ED. When reversal is required, neostigmine 0.02 to 0.04 mg/kg is given by slow IV push. Additional doses of 0.01 to 0.02 mg/kg may be given in 5 minutes if reversal is incomplete, but the total dose should not exceed 5 mg in the adult. Atropine 0.01 mg/kg (with a *minimum* dose of 0.1 mg for children and a maximum dose of 1 mg for adults) should be given concurrently with neostigmine to block its cholinergic effects. [109] [110] [111]

Of the nondepolarizing agents, pancuronium has been used the most widely, but its comparatively slow onset of action, long half-life, and greater incidence of side effects make it less than ideal for ED use. Vecuronium and atracurium are preferable in most instances when a longer duration of paralysis is required.

Pancuronium

Pancuronium is an aminosteroid derivative that is primarily excreted in the urine within 1 hour of IV administration. [111] Classified as a long-acting agent, its onset and duration of action are dose-related.

Dose.

Following the recommended 0.1 mg/kg IV dose, paralysis occurs within 2 to 5 minutes and lasts approximately 60 minutes. Paralysis may be maintained safely by repeated bolus or drip infusion. Because the effects of the drug are cumulative, repeating the original dose significantly lengthens the duration of paralysis.

Adverse effects.

Relatively few adverse effects are associated with the use of pancuronium. Many patients experience an increase in heart rate, blood pressure, and cardiac

output because of the vagolytic effect of the drug. Ventricular tachycardia and severe hypertension have been reported but are quite rare. [112] [113] Pancuronium may cause histamine release that results in bronchospasm or anaphylactic reactions. [114] Prolonged paralysis may also occur, primarily in patients with myasthenia gravis or with significant impairment of renal function. One consensus panel recommends pancuronium for maintaining paralysis, except in patients with cardiac disease or hemodynamic instability.[115] The panel recommended vecuronium for the latter patient groups.

Vecuronium, Atracurium, Mivacurium, and Rocuronium

Vecuronium and atracurium are intermediate-acting agents with an onset of action of about 3 minutes and a duration of action of 30 minutes. Mivacurium has an onset of action of 2 to 3 minutes and a duration of action of 15 to 20 minutes. Rocuronium has an onset of action within 1 to 1.5 minutes and a duration of action of 20 to 75 minutes (longer in geriatric patients). These drugs represent significant advances over pancuronium in that they have minimal cardiovascular effects, cause little histamine release (with the exception of mivacurium), [80] and lack cumulative effects.[116]

Dose.

The recommended doses of vecuronium, atracurium, mivacurium, and rocuronium are listed in Table 5-3 . Use of a larger than recommended dose will hasten the onset of action but greatly prolong the period of paralysis. For example, vecuronium at a dose of 0.25 mg/kg intravenously will cause paralysis in as little as 1 minute, but the period of paralysis will last 1 to 3 hours. [117] [118] Because a rapid onset of action comparable to succinylcholine is achieved at high doses of intermediate-acting agents, they may be used as the sole agents to facilitate intubation, particularly if a long period of paralysis is desired after intubation. However, the use of succinylcholine prior to intubation and an intermediate-acting agent at a normal dose after intubation provides rapid intubating conditions and better control over the duration of paralysis.

Rocuronium, a structural analogue of vecuronium, is emerging as an alternative for RSI when succinylcholine is contraindicated. At doses of 0.6 to 1.2 mg/kg, rocuronium consistently provides good to excellent intubating conditions within 1 minute of administration. The duration of action is dose-dependent ranging from 20 to 75 minutes.[119] [120] OR- and ED-based studies have demonstrated its clinical utility and safety in RSI protocols. [27] [119] [120] [121] [122] [123] [124] [125] Other newer and shorter-acting agents are currently under development and study. One promising agent (rapacuronium) has been withdrawn from the market because of associated bronchospasm.[126]

Paralysis induced by vecuronium or atracurium may be maintained by repeat bolus or drip infusion. Unlike both pancuronium and succinylcholine, there are no side effects specifically related to repeated dosing in the ED. A repeated dose of 0.01 to 0.02 mg/kg of vecuronium will extend the period of paralysis 12 to 15 minutes.

ANALGESIA AND SEDATION

Overview

An alternative to the induction of anesthesia in patients requiring intubation is the use of local anesthetic and sedative agents in the conscious patient. The availability of relatively effective and safe anesthetic agents such as etomidate make this a less attractive alternative than in the past, but clinicians may find maintaining some degree of wakefulness desirable in specific patients. Laryngoscopy in the awake patient has been likened to the "mouth being held open with a wrench." [127] Nasotracheal intubation is also an extremely unpleasant experience. The upper airway is richly innervated by sensory branches of the fifth, seventh, ninth, and tenth cranial nerves. In addition to pain fibers, there are stretch receptors that stimulate coughing and gagging reflexes with even minor airway manipulation. It is therefore essential that adequate analgesia be provided before intubation in all but the most extreme circumstances. Treatment options include topical application of anesthetic agents to the pharyngeal and tracheal mucosa and IV infusion of analgesic or sedative agents.

Local Anesthesia

Local or topical anesthesia techniques may be used in patients who are awake, either in place of—or as a supplement to—IV analgesia or sedation. They are particularly useful as adjuncts to nasotracheal intubation but do not generally provide the degree of analgesia or relaxation desirable for orotracheal intubation. In addition, the time required to achieve good topical anesthesia may limit the usefulness of these techniques in emergency situations.

Topical anesthesia may be achieved by direct application using laryngoscopy, by cricothyroid membrane puncture, or by inhalation of a nebulized anesthetic.

Direct Application Using Laryngoscopy

Achieving anesthesia of the oral and pharyngeal mucosa is a relatively simple procedure using commonly available agents such as 4% lidocaine or benzocaine-tetracaine-butamben-benzalkonium (Cetacaine). Achieving anesthesia of the hypopharynx is more difficult, because optimal results require application of the anesthetic to the epiglottis and vocal cords under direct vision.

This procedure begins with spraying the tongue and pharynx with a topical agent. Note that use of atomization devices which attach to standard syringes (e.g., Mucosal Atomization Device [MAD], Wolfe Tory Medical, Inc., Salt Lake City, UT) can provide effective drug dispersal without a forceful spray. The more forceful pressurized canister sprays commonly provoke a cough reflex. After allowing at least 2 to 3 minutes to permit numbing of the tongue and pharynx, the epiglottis and vocal cords are visualized using a laryngoscope and are sprayed directly with the anesthetic agent. Even if laryngoscopy is not used to facilitate this procedure, it is at best an unpleasant experience. It is also time-consuming because of the inherent delay associated with mucosal absorption of an anesthetic agent. An alternative is percutaneous injection of an anesthetic agent into the trachea at the level of the cricothyroid membrane. [128] [129]

Cricothyroid Membrane Puncture

The cricothyroid membrane is identified in the trapezoidal space between the cricoid and thyroid cartilages. After appropriate skin preparation with an alcohol or povidone-iodine (Betadine) swab, the overlying tissue and membrane are punctured with a 22-gauge needle in the midline and just above the superior border of the cricoid cartilage. Care should

be taken to maintain the needle in the midline at all times to avoid injury to the recurrent laryngeal nerves. The needle should be advanced until air can be aspirated, indicating placement of the tip in the trachea. A volume of 2 mL of 4% lidocaine is then injected rapidly. Alternatively, 3 to 4 mL of 1% or 2% lidocaine, as used for local anesthesia, may be injected if the 4% concentration is not available. Typically this will precipitate a cough, which adequately distributes the anesthetic over the upper trachea, vocal cords, and epiglottis.

Nebulized Anesthesia

This simple and painless technique can be used to facilitate awake intubation when the patient's condition is stable enough to permit a several-minute delay. [130] The anesthetic is delivered using a standard nebulizer and face mask connected to an oxygen source that delivers 4 to 8 L/min. A volume of 4 mL of a 4% lidocaine solution is nebulized over about 5 minutes. Bourke and colleagues reported achieving consistently good topical anesthesia using this technique, although their patients were often premedicated with opioids, sedatives, or both. [131]

Intravenous Analgesia and Sedation

The IV infusion of analgesic or sedative agents either alone or in combination can be used to facilitate relatively painless intubation. Depending on the clinical situation encountered, the doses of these drugs may be titrated to produce alterations of consciousness ranging from light to deep sedation. When used to facilitate orotracheal intubation, these drugs are frequently administered in conjunction with succinylcholine, thus producing a state of complete muscle relaxation, good analgesia, reduced anxiety, and loss of recall for the event. Ideally, drugs used for this purpose are characterized by high potency, rapid onset of action, short half-life, and minimal potential for cardiorespiratory depression.

The most commonly used classes of drugs for sedation prior to intubation are opioids and benzodiazepines. Anesthetic agents previously discussed, in particular the barbiturates, may also be used as sedatives, although in lower doses than required for RSI.

Opioids (Fentanyl)

Although any of several opioids administered IV could be used to induce sedation, only fentanyl will be discussed because it possesses significant advantages over other opioid agents.

Pharmacology.

Fentanyl is a synthetic opioid related to the phenylpiperidine family. Since its introduction in 1968, fentanyl has been used widely in a variety of settings, often replacing meperidine as the agent of choice for rapid short-term analgesia. [132] [133] [134] [135] [136] Its favorable pharmacologic properties include a highly lipophilic nature, rapid serum clearance, high potency, and minimal histamine release. [137] [138] [139] [140] Fentanyl crosses the blood-brain barrier rapidly, producing analgesia in as little as 1 to 2 minutes. Serum levels decline rapidly from peak concentrations because of extensive tissue uptake. [141] [142] Unlike morphine, the brain concentration of fentanyl falls in conjunction with the serum level. The duration of analgesic action is 30 to 40 minutes, although at high doses a second peak of activity may be seen several hours later because of the release of the bound drug from tissue stores. Fentanyl is about 50 to 100 times as potent as morphine. [143] This unique combination of potency and short half-life permits the administration of numerous small doses that can be titrated to the desired clinical effect. Similar to other opioids, fentanyl is competitively reversed with naloxone or nalmefene.

Dose.

The relative safety of fentanyl permits considerable latitude in dosing. When used as a primary anesthetic agent for major surgical procedures, doses ranging from 50 to 100 µg/kg produce minimal side effects.[144] Comparatively tiny doses produce sedation, and 3 to 5 µg/kg, given at a rate of 1 to 2 µg/kg per minute, is generally an effective analgesic dose. More rapid administration will cause greater depression of the level of consciousness. Mostert and coworkers reported successful awake intubation in 99 of 103 patients who were administered an average cumulative dose of 3.7 µg/kg. [137] Most of these patients were able to follow commands, and many recalled the events surrounding the intubation. A small percentage could not be intubated even after receiving 500 µg of fentanyl.

Larger doses, perhaps up to 25 µg/kg, may be needed to produce ideal intubating conditions, although if given rapidly, 10 µg/kg is usually adequate. However, even this lower dose is more likely to produce anesthesia than sedation and it may cause a longer period of unresponsiveness than is desirable. It is preferable to use a low dose of fentanyl (2 to 3 µg/kg) for analgesia combined with a paralytic agent (e.g., succinylcholine) to produce adequate muscle relaxation and a sedative (e.g., midazolam) to reduce anxiety and produce amnesia for the event.

Adverse effects.

Unlike other opioids, fentanyl causes little or no histamine release, and its use is seldom associated with emesis or hypotension. It is probably the safest opioid to use in the hypovolemic patient. Fentanyl also has significantly fewer emetic effects than other opioids. Adverse effects that have been reported with fentanyl are few and primarily follow the rapid IV infusion of very large doses. Like other opioids, fentanyl may cause rigidity of the skeletal musculature, including the chest wall and diaphragm. Typically this occurs with doses in excess of 15 µg/kg, but it has also been reported with doses as low as 10 µg/kg and may be related to too rapid administration.[137] [145] The muscular rigidity may be prevented or treated with standard doses of succinylcholine or naloxone. [146] Grand mal seizures have also been reported but are very uncommon.[147] [148] [149] Chudnofsky and colleagues[8] reported a complication rate of less than 1% in 841 ED patients treated with fentanyl. The most common complication was respiratory depression, and it generally occurred when fentanyl was given in combination with other CNS depressants.

Benzodiazepines (Midazolam)

Pharmacology.

The benzodiazepines are a widely used class of drugs characterized by anxiolytic, hypnotic, sedative, anticonvulsant, muscle relaxant, and amnestic effects. Several of these properties make the benzodiazepines ideal adjuvant agents for intubation, particularly when used in combination with opioids. It is important to remember that benzodiazepines do not have analgesic effects. Although they may produce excellent sedation and impair the patient's memory of an unpleasant experience, they will not prevent the pain associated with intubation. Diazepam has been used widely to facilitate intubation, but its use has been supplanted in recent years. Diazepam has a variable onset of action and a long elimination half-life, and it causes significant infusion site pain and frequently phlebitis when given intravenously.

Midazolam has, to a great extent, replaced diazepam as a preoperative sedative agent. [150] [151] Midazolam is also used widely as an anesthesia induction agent, even in high-risk elderly and cardiac patients. [152] [153] [154] Compared with diazepam, the primary advantages of midazolam include a twofold increase in potency, a shorter half-life, and a lessened potential for cardiorespiratory depression. Midazolam possesses a unique imidazole ring that is stable and water-soluble in an acid medium but highly lipophilic at physiologic pH.

Because it does not require suspension in propylene glycol, midazolam is not a tissue irritant. It causes minimal pain on injection, is rarely associated with phlebitis, and can be given intramuscularly when a very rapid onset of action is not required. The highly lipophilic character of the drug permits rapid accumulation in the CNS with onset of sedation in as little as 1 to 2 minutes. Rapid penetration into fatty tissue coupled with extensive binding to plasma proteins causes a prompt fall in serum levels after IV administration. This may account for the paucity of side effects outside the CNS. The half-life of elimination is 1 to 4 hours and is dependent on release of the drug from adipose tissue and protein-binding sites. The period of sedation following a single IV dose is considerably shorter. Emergence from a 0.15 mg/kg dose occurs in 15 to 20 minutes.[155]

Clinical experience using midazolam for sedation before the performance of surgical and dental procedures or as an adjunctive agent for the induction of anesthesia is considerable. Baker and Gordon reported the use of midazolam to achieve conscious sedation in 400 ambulatory surgery patients. [151] Used in combination with either fentanyl or ketamine, midazolam was considered to be both effective and safe.

Dose.

The recommended dose for moderate sedation with midazolam is 0.05 to 0.1 mg/kg given in 1-mg boluses and not exceeding 2.5 mg over 2 minutes. Doses up to 0.1 mg/kg are often needed to produce good conditions for intubation. All patients receiving midazolam should be monitored closely, and skilled personnel prepared to manage the airway should be present.

Adverse effects.

Although midazolam was initially touted to be free of cardiorespiratory side effects, recent experience suggests that the potential adverse effects of midazolam are quite similar to those of other benzodiazepines. A small increase in heart rate is seen frequently, as is a small decrease in systolic blood pressure. [156] Changes in blood pressure may be exaggerated in the presence of hypovolemia. [157] Cardiac index and coronary artery blood flow are generally not affected. Respiratory depression may occur even at standard doses but most often follows rapid administration of an excessive dose. Respiratory depression is also more likely to occur in debilitated or elderly patients and in those simultaneously receiving opioids. Reports of fatalities soon after midazolam's introduction in the United States prompted changes in the recommendations for its use. These included lowering of the dosage schedule, reduction of the speed of administration, and careful patient monitoring during administration. [158] The effects of midazolam are rapidly reversed by the administration of the benzodiazepine antagonist flumazenil. One case report has also implicated midazolam in the development of laryngospasm, which, as with respiratory depression, responded to treatment with flumazenil. [159]

Limited information is available on the use of midazolam to facilitate ED intubation. Wright and colleagues reported its use in 289 ED patients, 20 of whom were undergoing intubation.[160] The overall complication rate was 1.4%, including 2 cases of hypotension and 2 cases of respiratory depression. In every case, patients had received other drugs in combination with midazolam that may have been responsible for the observed adverse effects. Most patients in their study (71%) also received fentanyl. The midazolam-fentanyl combination readily permits titration to the desired level of sedation, and has a rapid onset and short duration of action. [161] [162] A study by Silivotti and colleagues of various induction agents for endotracheal intubation suggested that use of midazolam alone may be associated with suboptimal intubating conditions and increased difficulty with the performance of endotracheal intubation. [23] In the prehospital setting, hypotension with midazolam was found to be dose related[163] and thus should be used cautiously in patients with hypovolemia or traumatic brain injury, or both.

PREVENTING THE COMPLICATIONS OF INTUBATION

Overview

Numerous reports have highlighted physiologic responses to trachael intubation, and have attempted to define their immediate or long-term adverse effects and to offer interventions to ameliorate potential organ injury. While it is certain that intubation and adjunct medications will alter reflexes, ICP, blood pressure, and pulse rate, and may induce cardiac rhythms disturbances, the actual clinical consequences of these commonly observed changes are largely unknown. Clinical experience suggests that most transient alterations in physiology that occur with ED intubation produce no specific or readily documented long-term sequelae, or are often consequences that cannot be easily monitored or cannot be prevented. The prudent clinician is aware of the potential adverse effects of intubation and is cognizant of potential methods to minimize them. Most importantly, however, it is the careful monitoring of the post intubation milieu that will guide specific interventions. Overzealous attempts to suppress those physiologic responses that normally accompany airway manipulation may be counterproductive and are to be avoided. Although it would be desirable to provide airway control under the best of circumstances and with the least amount of injury to the patient, at this juncture the ideal approach to the physiologic responses to intubation are simply unknown. Most information has been extrapolated from experimental animal models or from the anesthesia experience, but similar issues may not apply to the milieu of the ED experience. The following discussion serves as a general clinical guide to the previous considerations, but at this juncture no specific standard of care can be promulgated.

The Pressor Response

The pressor response to stimulation of the pharynx, larynx, and trachea was first described by King and coworkers in 1951. [164] This reflex, mediated by the sympathetic nervous system, consists of a transient increase in blood pressure and pulse rate. The stretching of the hypopharynx that occurs with laryngoscopy is the most common precipitant of the pressor response, but any manipulation of the upper airway, including

nasotracheal intubation or suction, may elicit a potent response. [165] [166] [167] An increase in plasma catecholamines, including adrenaline and noradrenaline, is found in association with the pressor response. [168] [169]

Considerable variation exists in the magnitude and duration of the pressor response. Studies of young healthy normotensive subjects have shown that an average increase of 20 to 25 mm Hg in mean arterial pressure occurs with laryngoscopy and intubation. [170] [171] [172] [173] [174] The magnitude of the response increases as the duration of the stimulus increases, reaching a peak at 45 seconds. Data from controls in several studies of patients with a broad spectrum of medical problems reveal blood pressure increases ranging from 14 to 48 mm Hg, with an average of about 30 mm Hg. Similarly, the increase in heart rate ranges from 8 to 45 beats/min, with an average of approximately 30 beats/min. Typically these elevations last less than 5 minutes. The magnitude of the pressor response may be increased in hypertensive patients and those with cardiovascular disease, even if the underlying hypertension is adequately controlled before intubation.

In addition to sinus tachycardia, a number of dysrhythmias have been reported following intubation. These are primarily ventricular in origin and include ectopic beats, bigeminy, and, occasionally, short runs of ventricular tachycardia. Bradyarrhythmias have been reported uncommonly. Electrocardiographic changes suggestive of ischemia have been reported, particularly in patients with dramatic increases in blood pressure. [175] [176] In 1977, Fox and colleagues reported two patients, [177] both of whom deteriorated after induction of anesthesia and orotracheal intubation. This report has been widely quoted as evidence that the pressor response should be prevented. However, no studies have reported comparative data and none have established a direct relationship between the response and subsequent clinical deterioration in a large patient population. It is also unclear that attenuation of the pressor response will prevent dysrhythmias or electrocardiographic evidence of ischemia, although intuitively it is prudent to avoid sudden increases in blood pressure in unstable patients with acute cardiac or vascular disease.

Multiple studies have evaluated procedures to pharmacologically block the pressor response. Lidocaine has been the most extensively evaluated, but the results of these studies are inconclusive. [172] [178] [179] [180] [181] Although it appears that lidocaine given at 1.5 to 2 mg/kg may blunt the response, it is not clear that the reductions reported (10 to 15 mm Hg and 20 beats/min) are of any clinical significance. Other drugs, including thiopentone, sodium nitroprusside, labetalol, nitroglycerin, verapamil, nifedipine, clonidine, fentanyl, sufentanil, etomidate, and magnesium have shown variable responses. [182] [183] [184] [185] [186] [187] [188] [189] [190] [191]

Of these drugs, fentanyl may be the most effective. It completely suppresses the pressor response at anesthetic doses of 50 µg/kg, [192] [193] but considerably smaller doses may also be effective. Two studies [171] [173] have shown marked suppression of the response at doses of 5 to 6 µg/kg, although in both studies patients also received 5 mg/kg of thiopental. Fentanyl has also been shown to blunt the pressor response when administered in conjunction with etomidate. [190] The 1998 SHRED study evaluated the pressor response to RSI comparing thiopental, fentanyl, and midazolam as induction agents. [23] Midazolam, which was also associated with the poorest intubating conditions and most attempts required for intubation, showed a mean heart rate increase of 17 beats/min. Thiopental, likely due to its direct myocardial depressant and venodilatory effects, decreased mean arterial pressures by about 40 mm Hg. Fentanyl recipients maintained a relatively neutral hemodynamic profile during intubation. Use of paralytics during intubation did not appear to alter the hemodynamic response associated with each sedative agent. A recent study isolated the effect of laryngoscopy from the effect of tube passage into the trachea. Adachi and colleagues found that pretreatment with 2 µg/kg of fentanyl could blunt the hemodynamic effects of tracheal tube passage, but not the hemodynamic effects of laryngoscopy. [194]

It is important to note that even those studies demonstrating blunting of the pressor response failed to demonstrate that this provided any real benefit to the patients. It is likely that the pressor response is innocuous in the vast majority of patients, but it may be exaggerated and potentially harmful in the presence of preexisting hypertension and cardiovascular disease [177] or in patients with other vascular comorbidities such as esophageal varices in whom sudden changes in hemodynamics may be detrimental. [195] In addition, the pressor response may contribute to the rise in ICP that follows laryngoscopy, and therefore it is potentially harmful in patients with intracranial pathology. Administration of lidocaine or fentanyl to blunt the pressor response is appropriate in these subsets of patients.

Intracranial Hypertension

Physical stimulation of the respiratory tract by maneuvers such as laryngoscopy, tracheal intubation, and endotracheal suctioning is commonly associated with a brief rise in ICP. The exact mechanism responsible for this rise in ICP is unknown. One potential mechanism is the coughing and gagging that frequently follows manipulation of the upper airway and subsequent transmission of intrathoracic pressure to the cerebral circulation. An alternative explanation is the catecholamine release that accompanies laryngoscopy, causing a rise in mean arterial pressure and cerebral perfusion pressure. A small rise in ICP has also been reported following administration of succinylcholine. The value of using pretreatment with defasciculating doses of NMBs to prevent ICP rises is unknown. [196]

Although the exact significance of a transient rise in ICP is unknown, it is logical to assume that it may be detrimental in patients with head trauma or intracranial hypertension. A number of drugs including lidocaine, succinylcholine, and the majority of the anesthesia induction agents have been studied to determine whether their use prevents this response. Many of the existing clinical data are not particularly relevant to the ED setting because they are derived from patients in various stages of general anesthesia, often incorporating a wide variety of drug combinations and doses.

Lidocaine may be an effective agent either because it prevents coughing associated with airway manipulation or because it blunts the pressor response and the resultant rise in cerebral perfusion pressure. At a dose of 1.5 mg/kg intravenously, lidocaine suppresses coughing induced by citric acid inhalation [197] and at 2 mg/kg intravenously, it suppresses coughing associated with intubation. [198] However, studies have demonstrated conflicting results regarding lidocaine's ability to suppress the rise in ICP that follows airway manipulation. [198] [199] [200] [201] Optimal results have been demonstrated in paralyzed patients, suggesting that paralysis may be the best

TABLE 5-4 -- Sample Protocol for Intubation for a Head-Injured Adult Patient

1. Preoxygenate with 100% O_2 for 2–3 minutes.
2. Administer 1.5–2 mg/kg lidocaine.
3. Administer 0.01 mg/kg vecuronium (OPTIONAL).
4. Sedate with 3–5 µg/kg fentanyl.
5. Induce anesthesia with 0.3 mg/kg etomidate.
6. Paralyze with 1.5 mg/kg of succinylcholine.
7. Apply cricoid pressure and perform intubation.
8. Maintain postintubation analgesia and sedation.
9. Maintain paralysis if indicated (vecuronium 0.1 mg/kg).

method to suppress coughing and resultant ICP elevations during intubation. [202] Thus, the use of lidocaine to prevent coughing may have a role only when paralysis is not an option. In the paralyzed patient, lidocaine's effectiveness is uncertain, but it may be beneficial in blunting the pressor response and, secondarily, ICP elevations.

There is good evidence that deep general anesthesia prevents the rise in ICP associated with intubation, although depending on the drug used, anesthesia may compromise cardiovascular performance and critically reduce cerebral blood flow. [203] [204] [205] Consequently, the ideal anesthetic agents to facilitate intubation of patients with acute intracranial pathology may be those that have minimal effects on cardiovascular performance such as etomidate or fentanyl. Etomidate has been demonstrated to prevent changes in both cerebral perfusion pressure and ICP following tracheal intubation of patients with space-occupying intracranial lesions. [33]

At the present time, the clinical consequences of intubation-induced physiologic changes are not thoroughly understood, nor is the role of drugs in preventing these changes clear. Despite this lack of data, it may be prudent to attempt to protect patients at theoretic risk. The approach outlined in Table 5-4 is recommended.

CONCLUSION

This chapter has described a number of pharmacologic adjuncts that permit a much more sophisticated approach to intubation of the critically ill patient. As the expertise of clinicians practicing in the acute care setting increases, it is appropriate that they incorporate these adjuncts into their airway management protocols. The agents discussed in this chapter are representative of drugs currently used to facilitate intubation in U.S. EDs.

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Chapter 6 - Cricothyrotomy and Translaryngeal Jet Ventilation

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Although intubation is the usual method of definitive airway control, clinical situations occur in which intubation is difficult, impossible, or contraindicated. Under these circumstances, cricothyrotomy may be the quickest, easiest, safest, and most effective way to obtain an airway. [1]

Cricothyrotomy is the procedure by which an opening is made in the cricothyroid membrane (Fig. 6-1) to establish an airway. Cricothyrotomy is also known as a laryngostomy, laryngotomy, cricothyrostomy, or coniotomy. *Surgical* cricothyrotomy is the use of a blade to create the opening in the cricothyroid membrane. Once access to the airway is created, oxygen is administered through the opening in the cricothyroid membrane. A needle or small cannula also can be passed percutaneously (i.e., *needle* cricothyrotomy) through

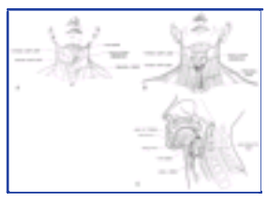

Figure 6-1 Anatomy of the neck. *A*, Surface anatomy of the neck, showing important external landmarks. *B*, Anterior view of the neck, showing various internal structures (overlying superficial skin and structures removed to show cricothyroid membrane). *C*, Lateral view of the neck, showing various structures.

the cricothyroid membrane to permit *translaryngeal jet ventilation*. Cricothyrotomy procedures differ from *tracheostomy,* in which entry into the airway is at a lower point (i.e., between the tracheal rings).

An emergency cricothyrotomy allows oxygenation and prevents hypoxemia and its complications, including anoxic encephalopathy and death. Management of the airway is of paramount importance, because the clinician has only 3 to 5 minutes in which to obtain an airway and achieve effective ventilation to prevent the complications of hypoxemia.[2]

BACKGROUND

The importance of securing an airway has been recognized since ancient times. Egyptian tablets dating from 3600 B.C. depict a surgical tracheostomy. [3] Historical records give credit for the first tracheostomy to Asclepiades of Prusa in 124 B.C. [4] In the 2nd century A.D., Galen suggested tracheostomy as the treatment for emergency management of airway obstruction[5] and Antyllus performed a tracheostomy with an incision between the third and fourth tracheal rings. [6]

In the United States, tracheotomies were a known procedure even in colonial times. In 1796, tracheotomies were advocated as a possible treatment for drowning victims.[7] Perhaps the first recorded "successful" case of a surgical cricothyrotomy was in 1852. The cricothyrotomy was successful, but the patient died later from airway stenosis, a complication of the procedure.[8] In 1886, the tracheostomy

mortality rate was about 50%, with a high complication rate from airway stenosis, which was usually fatal. [9]

Although tracheotomies and cricothyrotomies were successful and life saving, they were generally done only on severely hypoxic, terminally ill patients, thus contributing to the high morbidity and mortality of the surgical procedure. The fear of complications with the surgical procedure led to a reluctance of the clinician to actively intervene until the patient was near death. "Certainly the delay due to this great dread of tracheostomy was itself largely accountable for the fatality attending its performance."[10]

In 1909, Jackson described the surgical technique for tracheostomy and the factors that are critical for successful tracheostomy or cricothyrotomy. [11] These factors, which are still important today, include (1) obtaining the best airway control possible before surgery; (2) using local anesthesia initially rather than sedation or general anesthesia, which can lead to respiratory arrest in patients with an already compromised airway (although sedation and general anesthesia may be acceptable if the patient is already intubated); (3) emphasizing careful, precise surgical technique with good exposure; (4) using an inert, appropriate-sized and shaped tracheostomy tube; and (5) ensuring meticulous postoperative care, which helps avoid contamination or infection and other complications. He noted a surgical mortality of approximately 3%, which is similar to that reported in current series.

Jackson achieved worldwide recognition and patient referrals regarding cricothyrotomy complications. In 1921, Jackson published an investigation of 200 patients referred to him with tracheal stenosis.[12] The study was biased in that patients who did well and had no surgical complications (e.g., no laryngeal stenosis) were not referred to Jackson. Many of the patients had stenosis because of their underlying medical condition and not because of the procedure. Factors relating to surgical technique and postoperative care were not controlled, including aseptic technique, location of the opening in the larynx, and size and type of the tracheostomy tube. Jackson condemned cricothyrotomy because of the complication of subglottic stenosis.

Years later, Brantigan and Grow reported the results of cricothyrotomy on 655 patients. [13] Only 8 patients (0.01%) developed airway stenosis, and none of the patients developed chronic subglottic stenosis. Major vessel hemorrhage and operative misadventures were absent, and the complication rate, which included minor problems, was only 6.1%. The literature supports their conclusions that cricothyrotomy is a safe and effective procedure for airway management with relatively few complications.[14] [15] [16] When examined at bronchoscopy, no signs of laryngeal stenosis or damage have been noted in patients with cricothyrotomy. [17] Animal studies have documented a normal larynx after cricothyrotomy.[18]

ANATOMY

Knowledge of the anatomy of the neck and the upper airway is essential when performing a cricothyrotomy. The essential anatomic landmarks include the hyoid bone, the thyroid cartilage, the cricoid cartilage, and the tracheal rings (see Fig. 6-1).

The hyoid bone is located midway between the mental protuberance of the mandible and the third cervical vertebra. The midpoint of the body of the hyoid is also the transverse midpoint of the neck and can function as an anchor for stabilizing the airway in an edematous neck. The thyroid cartilage and the cricoid cartilage, both part of the larynx, and the tracheal rings are the major supporting structures of the airway. The trachea is the downward continuation of the larynx. Cartilaginous rings that are deficient posteriorly support the trachea. Posteriorly, the trachea lies against the esophagus.

The thyroid cartilage consists of two approximately quadrilateral-shaped laminae of hyaline cartilage that fuse anteriorly to form the laryngeal prominence. This structure calcifies with age. Above the laryngeal prominence is the superior thyroid notch, where the two laminae are separated. The anterior superior edge of the thyroid cartilage, the laryngeal prominence, is known as the Adam's apple and is usually easily seen in men. Except in the infant, the markedly obese patient, or the patient with massive neck edema, the laryngeal prominence of the thyroid cartilage is usually easily recognized and is palpable. It is probably the most important landmark in the neck when performing a cricothyrotomy.

The cricoid cartilage is the only circumferential ring in the larynx. It is shaped like a signet ring with the shield located posteriorly. The cricoid cartilage forms the inferior border of the cricothyroid membrane. The thyroid cartilage forms the superior border of the cricothyroid membrane.

The highly vascular thyroid gland lies over the trachea at the level of the second and third tracheal rings. If the tracheal rings or the thyroid gland is encountered when performing a cricothyrotomy, then the clinician is too low in the neck and must redirect the incision superiorly toward the cricoid and thyroid cartilages. Avoid the thyroid gland, because marked bleeding may occur if it is injured.

The cricothyroid membrane is a dense fibroelastic membrane located between the thyroid cartilage superiorly and the cricoid cartilage inferiorly; the cricothyroideus muscles bound it laterally. The cricothyroid membrane covers an area that is trapezoidal in shape. The average size of the cricothyroid membrane in the adult is approximately 22 to 30 mm wide and 9 to 10 mm high. Palpating a notch, a slight indentation or dip in the skin, inferior to the laryngeal prominence, can identify the cricothyroid membrane. The cricothyroid membrane is located approximately 2 to 3 cm below the laryngeal prominence in an adult.

Several anatomic and physiologic features make the cricothyroid membrane an excellent choice for gaining access to the airway. The membrane is immediately subcutaneous in location. It does not calcify with age. It has no overlying muscles or fascial layers and no major arteries, veins, or nerves are in the region. Although the right and left cricothyroid arteries, branches of the right and left superior thyroid arteries, respectively, transverse the superior part of the cricothyroid membrane (i.e., nearer the thyroid than the cricoid cartilage) and anastomose in the midline, these vessels generally have not been found to be of clinical significance when performing a cricothyrotomy.

Anatomic Variations

The larynx has a few anatomic features that vary according to age and sex. In children, the larynx is much higher than it is in the adult. The larynx descends from approximately the level of the second cervical vertebra at birth to the level of the fifth or sixth cervical vertebra in the adult.

The laryngeal prominence, the angle at which these two laminae of the thyroid cartilage meet anteriorly, varies from

90 to 120°. This angle is smaller in the male, which makes the laryngeal prominence meet at a more acute angle, or a sharper, more prominent point, than in the female.

In infants, the prominent structures in the anterior neck are the hyoid bone and the cricoid cartilage. The laryngeal prominence does not develop until adolescence and young adulthood.

A few vascular anomalies in the neck may result in a major artery crossing the midline of the neck. This is usually not a problem, because these vascular anomalies are almost always located lower in the neck.

SURGICAL CRICOTHYROTOMY

Indications

There are several potential indications for cricothyrotomy (Table 6-1). The most common indication is inability to perform endotracheal intubation because intubation is either contraindicated or cannot be done easily and quickly. This decision is always best made by the clinician at the bedside using clinical judgment to weigh the individual circumstances, resources and expertise. This procedure can be a formidable task in children, and in the struggling hypoxic patient.

Clinical conditions in which endotracheal intubation is difficult at best and is often impossible may include significant bleeding of any of the structures in the upper airway (e.g., massive oral, nasal, or pharyngeal hemorrhage), massive emesis or regurgitation, masseter spasm, clenched

TABLE 6-1 -- Indications for Surgical Cricothyrotomy

Failure of oral or nasal endotracheal intubation
Massive oral, nasal, or pharyngeal hemorrhage
Massive regurgitation or emesis
Masseter spasm
Clenched teeth
Structural deformities of oropharynx, congenital or acquired
Stenosis of upper airway (pharynx or larynx)
Laryngospasm
Mass effect (cancer, tumor, polyp, web, or other mass)
Airway obstruction (partial or complete)
Nontraumatic
Oropharyngeal edema
Laryngospasm
Mass effect (cancer, tumor, polyp, web, or other mass)
Traumatic
Oropharyngeal edema
Foreign body obstruction
Laryngospasm
Obstruction secondary to a mass effect or displacement
Stenosis
Traumatic injuries making oral or nasal endotracheal intubation difficult or potentially hazardous (relative)
Maxillofacial injuries
Cervical spine instability
Need for prolonged intubation
Need for definitive airway during procedures on face, neck, or upper airway
Laryngeal surgery
Oral surgery
Maxillofacial surgery
Laser surgery
Bronchoscopy

teeth, spasm of the larynx or pharynx (or both), laryngeal stenosis, and structural deformities of the upper airway (e.g., congenital or acquired abnormalities or deformities of the oropharynx). Whenever several unsuccessful attempts at endotracheal intubation or a rescue airway procedure cause an inordinate delay in airway control and oxygenation, cricothyrotomy is indicated to prevent cerebral anoxic damage. [19]

Obstruction of the upper airway is another indication for cricothyrotomy. [20] It has been stated that patients who are completely obstructed or in extremis are best managed by establishing an airway via the cricothyroid membrane. [21] When simple airway procedures are unsuccessful, cricothyrotomy should be undertaken without delay.

When maxillofacial, cervical spine, head, or soft tissue neck injuries are present, several factors may prevent intubation. These factors include (1) gross distortion of structures, (2) airway obstruction, (3) disruption of upper airway structures, (4) massive emesis, (5) significant hemorrhage, (6) patient discomfort, and (7) the possibility of aggravating existing or suspected injuries that would result in additional damage. In such patients, cricothyrotomy is an excellent alternative for obtaining definitive control of the airway. [22]

Cricothyrotomy is an option (1) for patients with a known cervical spine injury or a high probability of cervical spine injury in whom definitive airway control is needed before the cervical spine can be assessed and (2) for patients with certain head injuries, especially basilar skull fractures or cribriform plate fractures. Gerling and associates have demonstrated that cricothyrotomy can be done with only 1 to 2 mm of anterior-posterior displacement in an unstable cadaver model. [23] Hence, in trauma patients with known or anticipated cervical spine injury, cricothyrotomy may minimize movement of the neck during airway maneuvers. [24] Alternatively, careful *in-line stabilization of the neck with minimal movement during direct laryngoscopy is a relatively safe alternative during emergent intubation of the trauma patient*. [25] [26] [27]

Cricothyrotomy has several advantages over tracheostomy when prolonged intubation is needed (Table 6-2). [28] [29] Cricothyrotomy has been used for definitive airway control before elective surgery of the face, head, and neck. [30] Translaryngeal jet ventilation has become a routine method of ventilation during procedures (both endoscopy and surgery) on the upper airway. [31] This ventilation technique allows better access to and visualization of the upper airway for the surgeon or endoscopist than is possible with intubation from above the larynx, which often obscures and limits the surgical field. It also decreases the chance of flames or a "miniexplosion," which may occur during laser surgery or during other procedures that require flammable anesthetics or other gases. [32]

Cricothyrotomy vs Tracheostomy

The decreased incidence of complications with cricothyrotomy compared with that of tracheostomy is due at least partly to anatomy. [33] Anatomic considerations make tracheostomy a relatively complicated and difficult procedure. Many delicate complex structures in the neck lie in close proximity to the trachea. Less encroachment on the mediastinum occurs with a cricothyrotomy than with a tracheostomy, because the cricothyroid membrane is farther away from the mediastinum and other critical structures. Early complications, including

TABLE 6-2 -- Advantages of Surgical Cricothyrotomy Over Tracheostomy

Advantages due to anatomic considerations
Immediate subcutaneous location (vs deep dissection)
Absence of critical structures overlying cricothyroid membrane
Easily seen landmarks, recognizable from the surface anatomy
Less chance of esophageal injury (circumferential cricoid cartilage vs deficient tracheal cartilage posteriorly)
Farther away from mediastinum, dome of pleura (less encroachment on thoracic structures)
Overall advantages
Easier to do, faster, safer
Does not need to be done in an operating room
Less need for hyperextension of the neck
Better cosmetic appearance of scar (shorter, less adherent)
Decreased incidence of late complications (e.g., swallowing problems, voice disturbances, fistulas, erosion of innominate artery)
Decreased incidence of early complications (e.g., pneumothorax, pneumomediastinum, mediastinal perforation, esophageal injury) because of less encroachment on the mediastinum and other critical structures
Can be done quickly by nonsurgeons
Requires a minimum of instruments

pneumothorax, pneumomediastinum, and mediastinal perforation, occur less often with cricothyrotomy. Because the tracheal cartilage, unlike the cricoid and thyroid cartilage, is deficient posteriorly, the chance of damage to the posterior structures (e.g., esophagus) lying immediately behind the airway is greater with a tracheostomy. The incidence of late complications, including fistulas, erosion of the innominate vessels, swallowing problems, and voice disturbances, is less with cricothyrotomy.

Cricothyrotomy is preferred over tracheostomy for definitive emergency management of the airway when intubation is impossible or contraindicated. The few exceptions are in children 5 years old or younger (although some recommend needle cricothyrotomy with translaryngeal jet ventilation rather than tracheostomy) and for transection of the trachea with retraction of the distal trachea into the mediastinum.

Contraindications

Cricothyrotomy has relatively few contraindications (Table 6-3). Cricothyrotomy should not be done in patients who can be quickly, easily, and safely intubated. Transection of the trachea with retraction of the distal end is a contraindication to cricothyrotomy. A fractured larynx or other significant damage to the larynx or cricoid cartilage is another contraindication. [34]

Factors shown to be associated with subglottic stenosis include prolonged intubation, underlying laryngeal disease, and younger age (e.g., infants and small children).[35] Some believe that these conditions are relative contraindications to cricothyrotomy and that a cricothyrotomy should be converted immediately to a surgical tracheostomy.[36] Current studies indicate that subglottic stenosis is not a common complication of cricothyrotomy, even in the presence of laryngeal pathology, and that patients can have a cricothyrotomy for months without having subglottic stenosis. These considerations (i.e., prolonged intubation and underlying

TABLE 6-3 -- Contraindications to Surgical Cricothyrotomy

Absolute contraindications
Endotracheal intubation can be accomplished easily and quickly, and no contraindications to endotracheal intubation are present
Transection of trachea with retraction of distal end into the mediastinum
Fractured larynx or significant damage to the cricoid cartilage or larynx
Relative contraindications*
For infants and toddlers (<5 yr) transtracheal ventilation may be preferred over surgical cricothyrotomy
Bleeding diathesis
Patients with massive neck edema (may use modified technique for these patients)
Acute laryngeal disease

*Relative contraindications may be overlooked in the true emergency situation because it is more important to obtain an airway and to avoid hypoxemia.

laryngeal disease) are no longer absolute contraindications to cricothyrotomy.

A bleeding diathesis is not an absolute contraindication to emergency cricothyrotomy, because it is easier to obtain hemostasis with a cricothyrotomy than with a tracheostomy. Cricothyrotomy has been used successfully in patients requiring airway control following thrombolytic administration. [37] Loss of cervical landmarks with massive neck edema makes the procedure more difficult, although one approach using a measured estimation of the cricothyroid membrane location in this setting is promising.[38] These relative contraindications may be overlooked in a true emergency (i.e., when obtaining an airway is imperative).

Equipment

In the out-of-hospital setting, the only essential items are a sharp blade and a hollow tube to maintain the airway. In the emergency department (ED), the optimal instruments needed for surgical cricothyrotomy should be easily accessible. A sterile tray containing all the necessary instruments in an organized fashion should be part of the standard equipment stocked in the major resuscitation area (Table 6-4). Prepackaged percutaneous kits (e.g., Melker Emergency Cricothyrotomy Kit, Cook Critical Care, Bloomington, IN) using a wire-guided approach to aid location of the cricothyroid membrane and thus direct the skin incision are available.

TABLE 6-4 -- Equipment for Surgical Cricothyrotomy Tray

Scalpel with No. 15 blade and scalpel with No. 11 blade
Tracheal dilator (Trousseau dilator) or spreader
Two hemostats
Scissors
Tracheal hook
Needle holder
Tracheostomy tube (appropriate-size Portex or Shiley tubed—No. 5–6 in an adult)
25-ga needle and syringe containing lidocaine with epinephrine (for local anesthesia)
Preparation solution
Sterile gauze pads
Sterile tracheal suction catheter
Suture or circumferential tie (to secure tracheostomy tube in place)

An appropriate-sized tracheostomy tube should be selected (Table 6-5). Secretions obstruct a small tube more easily, whereas a large tube may damage the surrounding cartilage and lead to a fractured larynx. In an adult, a tracheostomy tube with an 8-mm internal diameter (ID), such as a No. 5 Portex or Shiley tube, is usually an appropriate size (see Table 6-5).

If an appropriate-sized tracheostomy tube is not available immediately, an endotracheal tube can be used as is or modified for use as a tracheostomy tube (Fig. 6-2). The uncuffed (proximal) end of the endotracheal tube is cut to an appropriate length (see Fig. 6-2*A*). The adapter is then attached to the cut end (see Fig. 6-2*B*), and the modified endotracheal tube is inserted like any tracheostomy tube. After insertion, the cuff of the endotracheal tube should be inflated, if possible.

The essential supplemental equipment for airway management, specifically supplemental oxygen, suction, and a bag-valve device, should be readily available.

Procedure

Preparation and Positioning

In a truly emergent situation, rapid airway access is critical, and there may not be enough time even for local anesthesia. Because of the dangers of respiratory depression in a patient who may have a compromised airway already, sedation and general anesthesia may be contraindicated. Ketamine is a potential alternative for some struggling patients whose agitation hampers the procedure.

The patient is positioned to expose the neck and its landmarks. If there are no contraindications, such as known or suspected cervical spine injury, the *patient's head should be hyperextended. Extension of the neck aids identification of the anatomy and control of the cricoid space*.

The cricothyroid membrane is located by identifying the dip or notch in the neck below the laryngeal prominence. The cricothyroid membrane is bounded by the thyroid cartilage superiorly and the cricoid cartilage inferiorly. The landmarks are most easily found by placing the index finger on the prominence of the thyroid cartilage and slowly palpating downward until the finger "drops off" the thyroid cartilage

TABLE 6-5 -- Tracheostomy Tube Sizes

Age	Internal Diameter of Tracheostomy Tube (mm)	Holinger or Magill Tube Size	Internal Diameter of Endotracheal Tube (mm)
Premature (<1.8 kg; <4 lb)		000	2.5
Premature (=1.8 kg; =4 lb)		00	2.5
Newborn	2.5	00 or 0	3.0
0–6 mo	3.5	0	3.5
6–12 mo	4.0–4.5	1	4.0
1 yr	4.5–5.0	1 or 2	4.5
2 yr	5.0	2	5.0
4 yr	5.5	3	5.5
6 yr	6.0	4	6.0
8 yr	6.5	4	6.5
10 yr	7.0	4	7.0
12 yr	7.5	5	7.5
14 yr	8.0	5	7.5
Adult female	8.0–8.5	5	7.5–8.5
Adult male	8.5–9.5	6	8.0–9.0

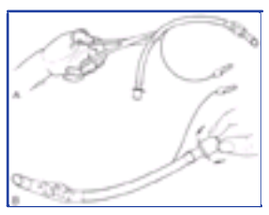

Figure 6-2 Modification of an endotracheal tube for use as a tracheostomy tube. *A*, An appropriate-sized endotracheal tube is cut to an appropriate length. *B*, An adapter is attached to the cut end before the modified endotracheal tube is inserted into the larynx via the opening in the cricothyroid membrane.

and onto the cricoid membrane (Fig. 6-3*A*). When the tip of the finger is in the proper position, the hard cricoid cartilage may be palpated with the fat pad of the index finger. If time permits, local anesthesia and skin preparation with povidone-iodine may be used. The skin and subcutaneous tissue immediately above the cricothyroid membrane may be infiltrated using lidocaine with epinephrine.

Incision and Tube Placement

A skin incision is made over the cricoid membrane. A midline longitudinal (vertical) skin incision about 3 to 4 cm long is made for an emergency cricothyrotomy. For an elective cricothyrotomy, a more cosmetic 2-cm transverse (horizontal) skin incision can be made. A longitudinal skin incision is preferred during an emergency,

because if the skin incision is too high or too low, a longitudinal skin incision merely needs to be extended, thus saving time and avoiding a second incision. As an option, one can insert a 20- to 22-ga needle through the cricothyroid membrane and confirm intra-airway positioning by aspirating air. The needle can be left in place to serve as a guide for the surgical procedure and then removed before insertion of the tracheostomy tube. Commercial kits containing guidewires for this approach are available and in cadaver models have resulted in smaller, more accurate skin incisions. [39]

The larynx is stabilized by holding it between the nondominant thumb and middle finger or by using a tracheal hook. After the skin incision, a short horizontal (transverse) stabbing incision about 1 cm long is made in the lower part of the cricothyroid membrane (i.e., nearer the cricoid cartilage than the thyroid cartilage to avoid the cricothyroid arteries). The stabbing incision is made in such a way that only the tip of the scalpel blade enters the trachea (see Fig. 6-3).

Curved Mayo scissors are inserted beside the scalpel blade and then spread horizontally to widen the space. An alternative method is to widen the opening by using a scalpel handle and turning the scalpel 90° ("longitudinally" with respect to the patient) instead of using curved Mayo scissors. The scalpel handle then is removed and a tracheal (Trousseau) dilator or curved hemostat is inserted into the incision site in

Figure 6-3 The procedure for surgical cricothyrotomy. *A*, Locate the cricothyroid membrane. The insert shows further anatomic details of the region. *B*, A longitudinal skin incision is made over the cricothyroid membrane. *C*, The larynx is stabilized with the thumb and middle finger or a tracheal hook (held in the nondominant hand) while an incision is made in the cricothyroid membrane. *D*, The surrounding anatomy of the neck is shown, with the incision being made in the cricothyroid membrane. *E*, After the incision in the cricothyroid membrane is widened using hemostats, curved Mayo scissors, or the blunt end of the scalpel, the tracheostomy tube is inserted between the curved hemostats or tracheal dilator. *F*, Lateral view, showing insertion of the tracheostomy tube.

the cricothyroid membrane. The dilator is then opened to enlarge the opening in the cricothyroid membrane.

When attempting to insert the tracheostomy tube, the larynx may be displaced posteriorly. Posterior displacement of the larynx may make it difficult or impossible to insert the tracheostomy tube. Stabilization of the larynx (preferably with a tracheal hook) is important to lift and hold the larynx anteriorly in proper anatomic position so that this potential problem can be avoided. The tracheostomy tube is inserted between the tracheal dilator blades. Alternatively, curved hemostat blades may be used (see Fig. 6-3*E*). The dilator or hemostat is then removed. The cuff of the tracheostomy tube is inflated. One cadaver study suggests that insertion of the tracheostomy tube during use of the tracheal dilator limits tube passage more than when the tracheal hook alone is used for laryngeal stabilization. [40]

An alternative to use of the tracheal hook for stabilization of the trachea indirectly by elevation of the larynx has been described by Brofeldt and colleagues. [41] In their technique, the tracheal hook is placed in the opposite direction (i.e., aligned caudally rather than cephalically) to allow elevation of the cricoid ring and overlying structures. The operator stands to the left of the patient's neck and faces the patient's thorax as if performing an oral endotracheal intubation. Using this technique, Brofeldt and colleagues have advocated a rapid, four-step approach composed of (1) palpation of the cricoid membrane using external landmarks, (2) horizontal stab through skin and cricothyroid membrane together using a No. 20 scalpel, (3) tracheal hook traction applied caudally at the cricoid cartilage, and (4) intubation (Fig. 6-4). A further modification of the technique using a "Bair Claw" device (Fig. 6-5) rather than a tracheal hook to stabilize the cricoid ring has been found to reduce the time to procedural completion by 18 seconds. [42]

In an adult, a No. 5 or 6 Shiley tracheostomy tube is usually an appropriate size (No. 5 for an adult female and No. 6 for an adult male). If edema is present, a smaller (No. 4) Shiley tube may be used. Other tube size guidelines are provided in Table 6-5 .

If a tracheostomy tube is not available immediately, then an endotracheal tube can be used as a temporary airway device. The endotracheal tube can be replaced later when an appropriate-sized tracheostomy tube is available.

The lungs are ventilated, and proper tube placement should be ensured. The tracheostomy tube is secured in place via a circumferential tie around the neck or by suturing. After confirming proper tube placement, the tracheostomy tube may be attached to a ventilator or a bag-valve device. Flexible connector tubing is recommended to avoid excessive forces on the tracheal wall during mechanical ventilation.

Precautions

The skin incision and the incision in the cricothyroid membrane should not be made too far laterally (see Fig. 6-1 and Fig. 6-3*A*). A central incision will decrease the chance of hemorrhage due to laceration of vessels. The skin incision should not be made too far caudally in the neck, thus avoiding hemorrhage from and damage to the thyroid gland.

The stabbing incision should be made in the lower half of the cricothyroid membrane (i.e., nearer the cricoid cartilage than the thyroid cartilage) in an attempt to avoid bleeding from the cricothyroid arteries, which course superiorly across the cricothyroid membrane near the lower edge of the thyroid cartilage.

Transection of the cricoid cartilage or tracheal rings should be avoided. These structures help maintain the stability of the laryngeal lumen. Injury to these structures may predispose to late complications, including subglottic stenosis.

The scalpel should be directed in a caudal direction, so that injury to the vocal cords is avoided. The vocal cords are located above the cricothyroid membrane and are protected by the thyroid cartilage on three sides. When a cricothyrotomy is being done, the needle or scalpel should be directed posteriorly at a 90° angle to the cricothyroid membrane (see Fig. 6-3).

The clinician should avoid making a blind stab with the scalpel in the region. Controlled, anatomically placed incisions will minimize hemorrhage and injury to any of the adjacent structures.

The handle of the scalpel should be held in such a way that only the tip of the scalpel blade can enter the trachea during the stab incision through the cricothyroid membrane. For control, the clinician should place the thumb and index finger low on the handle of the scalpel (just above the scalpel blade). A controlled stab incision should help avoid injury to the posterior wall of the trachea, esophagus, and other posterior structures by limiting the depth of scalpel insertion.

Placement of too large a tracheostomy tube through the cricothyroid membrane should be avoided. Placement of an oversized tracheostomy tube can lead to a fractured larynx.[26] In the adult, the average size of the cricothyroid membrane is about 2 to 3 cm wide and 1 cm high. Thus, the appropriate tracheostomy tube in an adult is one with an 8- to 9-mm internal diameter (a No. 5 in an adult female and a No. 6 in an adult male) unless edema is present, in which case a smaller size (No. 4) is used.

The Patient with Massive Neck Swelling

One method of surgical cricothyrotomy uses the hyoid bone as a landmark and an anchor to stabilize the mobile airway in a patient with massive neck edema. [43] [44] The horizontal midpoint of the body of the hyoid is also the midpoint of the neck. The key measurement in this technique is distance from the angle of the mandible to

the mental protuberance of the chin (line *A'* on Fig. 6-6). Distances can be measured using a piece of suture or a string. This distance from the angle of the mandible to the mentum is divided in half.

A point is identified on the anterior midline of the neck below the chin (point *C*), which equals line *B'* or one half the length of line *A'* (see Fig. 6-6). A needle is placed at a point *C* and directed toward the midline so that it will reach an imaginary line connecting both angles of the mandible. The tip of the needle should hit the hyoid bone. A large-bore (e.g., 18-ga spinal needle) should be used, as the needle must be long enough to pass through massive neck swelling and hit the hyoid bone. If the hyoid bone is not found, the angle of the needle should be adjusted.

When the hyoid bone is found, the needle is left in position, and a No. 11 blade is inserted along the needle tract until the hyoid bone is reached. A skin hook is placed alongside the scalpel's tract and under the hyoid bone to retract the hyoid superiorly and anteriorly. A longitudinal skin incision is made inferiorly from the skin hook with care taken to stay in the midline. The hyoid bone acts as a fulcrum, holding and stabilizing the larynx in position, so that the incision will stay in the midline despite massive swelling of the neck. The incision

Figure 6-4 Rapid, four-step cricothyrotomy technique. *A,* Step 1: Palpation (location of cricoid membrane externally). *B,* Step 2: Incision (horizontal incision of skin and soft tissues through cricoid membrane). *C,* Step 3: Traction (caudal traction on cricoid ring). *D,* Step 4: Intubation (passage of tracheal tube). *(Reproduced from Brofeldt BT, Panacek EA, Richards JR. An easy cricothyrotomy approach: the rapid four-step technique. Acad Emerg Med 3:1060, 1996.)*

is extended caudally over the thyroid cartilage until the cricothyroid membrane is exposed. The remainder of the procedure follows the routine steps for cricothyrotomy.

The only additional supplies needed for this modification of cricothyrotomy in patients with massive neck edema are the suture (or string) to measure the distance from the angle of the mandible to the mentum of the chin and an 18-ga spinal needle to find the hyoid bone.

Infants and Children

Cricothyrotomy, as with tracheostomy, is technically much more difficult in infants and children than in adults and may have a higher rate of problems or complications. Because the laryngeal prominence is not well developed until adolescence and young adulthood, the most prominent structures in the anterior neck of the infant or child are the hyoid bone and the cricoid cartilage. In children younger than 5 years old, the cricoid is the narrowest part of the airway, and the cricothyroid membrane is quite small. Furthermore, the larynx is higher, relatively less accessible, and smaller in the child than in the adult.

Some advocate tracheostomy rather than cricothyrotomy in infants and children (when endotracheal intubation is contraindicated or impossible), because of the difficulty in palpating and identifying the important landmarks of the neck, and because of the small diameter of the cricoid cartilage. Others note that emergency surgical cricothyrotomy has fewer complications than emergency tracheostomy in infants and children. It has been suggested that a cricothyrotomy be converted to a tracheostomy when the infant or child is stabilized, to reduce the possibility of subglottic stenosis.

Complications

The incidence of complications for an elective surgical cricothyrotomy is approximately 6% to 8%. The complication rate for an emergency cricothyrotomy ranges from 9% to 31%.[45] [46] The complication rate for emergency cricothyrotomy compares favorably with that for tracheostomy, which has an average complication rate of approximately 45%.[47] The mortality rate for cricothyrotomy of 0.15% is less than that for tracheostomy, which is 1.6% to 5.0% and as high as 16% in one series. [48] [49] [50] In general, there has been a higher incidence of complications with cricothyrotomy in infants and children than in adults. This is also true for tracheostomy. Tumor, inflammation, other masses, certain congenital anomalies, and significant trauma may distort the anatomic features of the neck and airway, creating additional difficulty in performing a cricothyrotomy and increasing the complication rate for this procedure.

Minor complications are more common than serious complications (Table 6-6). Early complications are more

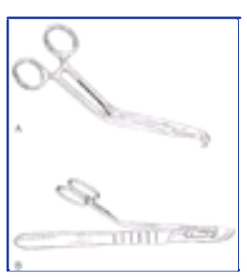

Figure 6-5 "Bair Claw" device. *A,* Device shown without scalpel. During the tracheal tube intubation procedure, the device is used as a caudally directed tracheal hook with slight spreading of the arms of the hook. In this manner the device both stabilizes the distal trachea and provides exposure of the lumen. *B,* Device shown attached to scalpel. During the initial incision, the device is attached to the No. 20 scalpel and passively placed through the cricothyroid membrane during the initial horizontal incision. The "claws" of the device are then released and the scalpel removed. The device is then rotated into position and used as outlined above. *(Davis DP, Bramwell KJ, Hamilton RS, et al. Safety and efficacy of the rapid four-step technique for cricothyrotomy using a Bair Claw. J Emerg Med 19:125, 2000.)*

frequent than late complications. In one series of patients who had emergency cricothyrotomies, the overall incidence of complications was 23% with no long-term complications.[51] Complications may be more common when the procedure is performed under suboptimal conditions. In one Emergency Medical Service (EMS) system, EMS personnel used surgical cricothyrotomy in 56 of 376 patients requiring emergency airway intubation. [52] In 79% of those receiving cricothyrotomy, prior orotracheal intubation attempts were unsuccessful (mean of 1.9 attempts per patient). There were six failed cricothyrotomy attempts, one case of excessive bleeding, and one episode of patient agitation. Generally the cricothyrotomy procedure was judged to be acceptable (64%) or functioning marginally (16%) upon arrival at the hospital.

Under ideal conditions, the most common complications are bleeding, incorrect site of tube placement, unsuccessful tube placement, and prolonged procedure time (see Table 6-6). The overall procedure time for cricothyrotomy should be <3 minutes. It is not unusual for surgical cricothyrotomy to be completed in 30 to 60 seconds. Cadaver studies have suggested that the rapid, four-step technique allows a more rapid intubation over standard surgical cricothyrotomy with a similar complication rate.[39] [42] [53]

Bleeding from the procedure is usually minor and occurs early. Packing the cricothyrotomy site with gauze can usually

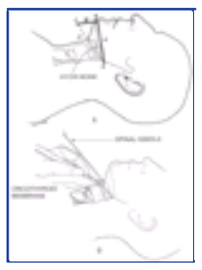

Figure 6-6 Modification for a patient with a massive neck swelling (see text). *A*, Landmarks in the neck. *B*, The procedure for a patient with a massive neck swelling uses a spinal needle to find and identify the hyoid bone; the skin incision is then made in the midline, moving inferiorly from the midpoint of the hyoid bone past the thyroid cartilage until the cricothyroid membrane is exposed. Care is needed to *avoid* a high incision through the thyrohyoid membrane above the thyroid cartilage.

control such bleeding. It is unusual for bleeding vessels to require ligation. Persistent hemorrhage is usually attributable to vessels located at the edges of a transverse skin incision. This complication is minimized if a longitudinal skin incision is made. [54] Potential bleeding may be minimized by making the stab incision in the lower part of the cricothyroid membrane near the cricoid cartilage to avoid the cricothyroid arteries. Major bleeding generally has not been a problem. [55] [56] One cadaver study noted a high number of small vessels in the region of the cricothyroid membrane that are at risk during a cricothyrotomy. [57] Hence, hemorrhage could still be a problem even if the technique of cricothyrotomy is done correctly and all the guidelines for the procedure are followed. Even a small amount of bleeding could be life-threatening if it occurs endotracheally, with resultant pneumonitis or suffocation. [58] The mortality from acute bleeding during a tracheostomy has been attributed to airway obstruction from the blood and not from volume loss. [59] In the management of postcricothyrotomy or post-tracheostomy bleeding, the airway is protected if the tracheostomy tube is in place and the cuff is inflated. If the airway is protected, then hemorrhage control (i.e., local

TABLE 6-6 -- Complications of Surgical Cricothyrotomy

Immediate or early complications
Common
Bleeding, hematoma
Incorrect tube placement
Unsuccessful tube placement
Prolonged procedure time
Subcutaneous emphysema
Obstruction
Infrequent
Esophageal perforation
Mediastinal perforation
Pneumothorax, pneumomediastinum
Vocal cord injury
Laryngeal fracture or disruption of laryngeal cartilage
Aspiration
Late complications
Most common
Obstructive problems
Voice changes or dysphonia
Infections
Late bleeding
Persistent stoma
Subjective feeling of lump in the throat
Infrequent complications
Subglottic or glottic stenosis
Tracheoesophageal fistula
Tracheomalacia

pressure on the bleeding vessels and, if necessary, ligature) can be done in a controlled fashion.

An incorrectly placed transverse skin incision may lead to bleeding and incorrect or unsuccessful tube placement. Too low a skin incision may lead to tube placement between the tracheal rings. In an emergency, a longitudinal skin incision is preferred, because a misplaced longitudinal skin incision, whether too high or too low, can be easily extended. [60] Davis and colleagues suggest that injury to the cricoid ring by a tracheal hook represents a theoretical risk with the rapid, four-step technique of Brofeldt and colleagues. [61] Yet in another cadaver study of the rapid, four-step technique, the use of the "Bair Claw" device was not associated with cricoid injury. [42]

Other less common complications include infection and airway obstruction. [62] An indwelling tube will become colonized by bacteria even if meticulous care is taken, and infection can result. Infection can occur in the structures or tissues of the neck and in the tracheobronchial tree. Potential infections include cellulitis, perichondritis, abscess in the incision site, and laryngotracheitis.

Acute airway obstruction can occur with tracheostomy or cricothyrotomy. In a correctly placed tube, the most common cause of acute airway obstruction is a mucus plug. A mucus plug that causes obstruction should be cleared from the airway by careful suctioning, using sterile technique (see Chapter 7). If this cannot be accomplished, then the entire tracheostomy tube may need to be changed. Less frequently, acute airway obstruction can be caused by overinflation of the tracheostomy balloon with herniation of the balloon over the tip of the tracheostomy tube or by retrograde intubation of the pharynx. [63] The tracheostomy tube can be passed into a subcutaneous fascial or tissue plane instead of into the trachea. This complication can occur during insertion of the tracheostomy tube or, more commonly, during a tube change, especially during the immediate postoperative period.

Long-term complications following cricothyrotomy are unusual. [64] The major long-term complication is a change in voice or dysphonia. [65] Laryngeal damage may occur secondary to a laryngeal fracture caused by using a tracheostomy tube that is too large for the size of the cricothyroid membrane. Dysphonia can also be caused by direct injury to the vocal cords.

Other late and rare complications include a suture sinus, a subjective feeling of a lump in the throat, a persistent stoma, chondritis of the thyroid or cricoid cartilage, and subglottic or glottic stenosis. Swallowing problems have been reported in tracheostomy patients [66] but not in cricothyrotomy patients.

Airway stenosis is a potential complication whenever a tube is placed in the airway. Airway stenosis can occur with intubation, cricothyrotomy, or tracheostomy. The most common cause of subglottic stenosis is endotracheal intubation and not cricothyrotomy or tracheostomy. [67] The pathophysiology of subglottic stenosis involves

mucosal ulceration and damage secondary to a tube eroding the mucosal surface and laryngeal disease along with bacterial colonization of the region. Excessive cuff pressures, frequent tube motion, and rigid tubes that are anatomically incorrect contribute to mucosal damage. The use of flexible, anatomically correct tubes with low-pressure cuffs and flexible tubing between the tube and the ventilator will minimize mucosal damage.

Prolonged endotracheal intubation followed by tracheostomy or cricothyrotomy is associated with a higher incidence of subglottic stenosis than with either endotracheal intubation or the surgical procedure (tracheostomy or cricothyrotomy) alone. [68] The airway stenosis produced by an indwelling tube is generally located at the cuff site of the tube. If airway stenosis occurs with a standard tracheostomy, the stenosis occurs quite distal in the trachea, thus making it difficult to reach during surgical repair. Early aggressive surgical management of granulation associated with mucosal injury has been successful in limiting chronic airway stenosis. [69] There are other theoretical complications of cricothyrotomy. These include pneumothorax, pneumomediastinum, major vessel hemorrhage, tracheoesophageal fistula, and esophageal perforation. These complications are seen with tracheostomy but have not yet been reported as complications of cricothyrotomy. [70] [71]

The complications of asphyxia, including dysrhythmias and cardiac arrest, during the performance of tracheostomy or cricothyrotomy are generally due to the lack of adequate oxygenation and not to a vagal reaction. Because cricothyrotomy is one of the easiest, quickest, and safest ways to achieve definitive airway control and oxygenation, the complications of hypoxemia should occur less commonly than with tracheostomy when performed in critically ill patients.

PERCUTANEOUS TRANSLARYNGEAL JET VENTILATION (NEEDLE CRICOTHYROTOMY)

Overview

Needle cricothyrotomy is a percutaneous technique in which a needle is placed through the skin, the subcutaneous tissue, and the cricothyroid membrane without using a formal incision. This airway access procedure is *ideally* followed by intermittent

high-pressure administration of oxygen through the cannula in the cricothyroid membrane (*translaryngeal jet ventilation*). Only when a sufficiently large catheter (generally =3 mm ID) is inserted, can a low-pressure ventilation source (e.g., bag-valve device) be used to partially support ventilation in the adult. However, a low-pressure ventilation source can be effective for ventilation through a 12- to 14-ga IV catheter in very small infants (5 kg) and may partially support ventilation in larger infants without complete airway obstruction.

The technique of puncture of the cricothyroid membrane is relatively simple and has many uses, ranging from obtaining sputum samples to giving local anesthesia before intubation to ventilating the patient. [72] [73] Whether the lungs can be ventilated adequately via a cannula in the cricothyroid membrane has been debated. We now know that percutaneous translaryngeal ventilation can be an effective and relatively safe procedure for obtaining and maintaining an airway for extended periods of time provided that appropriate equipment and techniques of ventilation are used. [74]

Historically, oxygen was simply passed through the needle or cannula in the cricothyroid membrane (*apneic oxygenation*). In this way, hypoxia was treated, and tissue oxygenation was generally adequate. Clinical studies and animal experiments noted that although the partial pressure of O_2 (pO_2) was adequate, CO_2 was retained, and the elevated partial pressure of CO_2 (pCO_2) led to respiratory acidosis. The oxygenation could be maintained for only about 30 minutes because of the severe hypercapnia and resultant respiratory acidosis, even when the hypoxia was treated successfully. [75] This procedure, whereby oxygen is run continuously through a needle or cannula in the cricothyroid membrane, is referred to as *translaryngeal oxygenation*.

The solution to avoiding a rise in pCO_2 and respiratory acidosis is to ventilate the patient, allowing time for both inhalation and exhalation, thus simulating normal respiration. [76] Intermittent bursts of oxygen provide sufficient "tidal volume" and time for exhalation, thus avoiding hypercapnia and respiratory acidosis. *Translaryngeal jet ventilation* is the ventilation of the lungs by intermittent bursts of oxygen through a needle or cannula in the cricothyroid membrane. The technique can supply adequate ventilation up to high degrees of airway obstruction. Percutaneous *transtracheal jet ventilation* is the same procedure except that the cannula is placed through the larynx into the trachea.

Physiology

The idea of gas insufflation via an opening in the trachea was described in the early 17th century, [77] although the current usage of the technique has only become popular in the last 25 years. A lack of understanding of the differences between transtracheal oxygenation and ventilation and a failure to standardize the technique itself (with regard to such factors as pressure, volume, intermittent vs continuous administration of air vs oxygen, size of opening in the membrane, and location of opening in the airway) has led to conflicting reports in the literature and to many misconceptions. [78]

Studies as early as 1909 showed that animals could survive for approximately 30 minutes when oxygen was given via a needle placed in the cricothyroid membrane. [79] Reed and coworkers, in 1954, showed that animals could be kept alive for a longer period of time with a normal pH if they were ventilated with intermittent bursts of compressed air instead of a continuous flow of oxygen. [80] The key to avoiding respiratory acidosis and maintaining a normal pH was the use of a method allowing for adequate ventilation, not just a continuous inflow of oxygen or air (as with apneic oxygenation).

A popular misconception is that percutaneous transtracheal ventilation is a temporizing procedure, good for only 30 minutes at best. Animal experiments and clinical studies have demonstrated that transtracheal ventilation is an effective, quick, fairly simple, and safe way to obtain and maintain an airway for a prolonged period of time. [81] Percutaneous translaryngeal ventilation with an adequate-sized cannula, appropriate ventilation pressures, sufficient tidal volumes, and adequate exhalation times can provide adequate ventilation for extended periods. [82]

Transtracheal jet ventilation has been used extensively as a means of ventilation during surgery and procedures of the upper airway. [83] [84] Translaryngeal jet ventilation may be used even with partial airway obstruction. [85] [86] Percutaneous translaryngeal jet ventilation is a rapid procedure for obtaining airway control in both elective and emergency situations in patients of all ages and in many clinical situations. [87] [88] [89]

The advantages of needle cricothyrotomy with translaryngeal jet ventilation over surgical cricothyrotomy include (Table 6-7) faster performance (may take only 10 seconds), easier performance, less equipment and smaller surgical setup required, no need for an assistant, smaller scar, less bleeding, expulsion of oropharyngeal secretions and other particles or small objects from the proximal trachea, less tracheal erosion, and less frequent subglottic or glottic stenosis (see Table 6-6). Some of the advantages are a result of the smaller size of the opening and the percutaneous technique.

The disadvantages of percutaneous transtracheal jet ventilation are (1) incomplete control of the airway, with a greater potential for aspiration than with a cuffed tracheal tube; (2) likelihood of barotrauma (subcutaneous emphysema or pneumothorax) if exhalation is inadequate and airway pressure is elevated; and (3) inability to perform adequate suctioning through a percutaneous catheter setup. Airway protection during transtracheal ventilation is attained by positioning the patient to allow drainage of secretions away from the larynx during expiration, so upward gas flow through the larynx causes secretions and blood, for example, to be blown away from the larynx. [90]

TABLE 6-7 -- Advantages of Percutaneous Translaryngeal Ventilation vs Surgical Cricothyrotomy

Faster (i.e., may take as little as 10 sec vs 30 sec to 3 min for surgical cricothyrotomy)
Less bleeding (due to smaller opening, smaller incision)
Simpler technique, easier to perform
Does not need an assistant, can be done by nonsurgeons
Requires fewer instruments
Lesser need for extensive surgical setup (lighting, equipment, and preparation)
Smaller scar (stoma is only as large as the size of the tube)
Less tracheal erosion
Less subglottic or glottic stenosis
Forces oropharyngeal secretions out of the proximal trachea
May force foreign body out of proximal trachea with a partial airway obstruction
Can be performed in patients, of all ages

Indications

Indications for needle cricothyrotomy with translaryngeal jet ventilation are similar to those for surgical cricothyrotomy. Cricothyrotomy is indicated in any situation in which intubation is contraindicated or cannot be performed (see Table 6-1). Failure to achieve endotracheal intubation in a timely fashion, and a subsequent inordinate delay in definitive airway control and oxygenation, is an indication for needle or surgical cricothyrotomy to prevent hypoxemia.

Percutaneous translaryngeal ventilation can be used in a patient with airway obstruction, but it requires an adequate-sized catheter or catheters. [91] [92] Translaryngeal ventilation has been recommended as the procedure of choice for airway control and oxygenation during surgery and procedures on the upper airway. [93] Percutaneous translaryngeal ventilation has been used as an elective procedure and in patients of all ages in an emergency (even premature infants), for extended periods of time.[94]

Contraindications

The absolute contraindications to surgical or needle cricothyrotomy are (1) the ability to accomplish endotracheal intubation easily and rapidly; (2) retraction of the distal end of the trachea into the mediastinum after tracheal transection; and (3) occurrence of known significant direct damage to the cricoid cartilage or larynx (see Table 6-3).[95]

Known complete airway obstruction requires an adequate-sized catheter or catheters to avoid barotrauma and hypercarbia. Barotrauma with high pressures occurring in the trachea can lead to complications such as a pneumothorax or pneumomediastinum, or both, during percutaneous transtracheal ventilation. [96] If intratracheal pressures =20 cm H_2O are maintained, the complication rate of percutaneous transtracheal ventilation is low. If complete upper airway obstruction is present, surgical cricothyrotomy is preferred over percutaneous translaryngeal ventilation, although percutaneous translaryngeal ventilation could be used with partial or complete airway obstruction provided appropriate-sized catheters are used. [97]

Relative contraindications to percutaneous translaryngeal ventilation are different from those for surgical cricothyrotomy. Age younger than 5 years has been mentioned as a relative contraindication to surgical cricothyrotomy. Age is not a factor in percutaneous translaryngeal ventilation. Some authors recommend percutaneous translaryngeal ventilation as the invasive airway procedure of choice in infants and young children, especially in an emergency. [98]

Equipment

Translaryngeal jet ventilation requires three components: an oxygen supply at 50 psi, an in-line one-way "valve" to allow for the intermittent administration of oxygen, and a needle or cannula with a bore =13 or 14 ga in the cricothyroid membrane (Table 6-8).

Oxygen Supply

The oxygen supply must be at a pressure of 50 psi to deliver enough volume to ventilate the lungs rapidly between periods of exhalation through the glottic opening. An oxygen source of 50 psi can be obtained by attaching a connector to the piped

TABLE 6-8 -- Equipment for Needle Cricothyrotomy and Translaryngeal Jet Ventilation

Oxygen source
High-pressure oxygen source at 50 psi
Deliver 100% oxygen at 20 bursts/minute with I:E = 1:4
Manual jet ventilator device
High-pressure tubing (to be attached to cannula or catheter and oxygen at other end with the manual jet ventilator device in the middle)
Manual jet ventilator device (Y-connector or push-button device) to allow for ventilation with inhalation and exhalation
Cannula
Large bore (=13- or 14-ga needle) with a Teflon or plastic cannula or, alternatively, one of the commercial cricothyrotomy devices, or 9 Fr catheter

I, inhalation; E, exhalation.

oxygen wall line found in any ED, attaching the tubing to the flush valve of an anesthetic machine, or attaching the oxygen tubing directly into an oxygen tank line (regulator) but not to the flow valve of the oxygen cylinder. The oxygen tanks in ambulances or other patient transport vehicles for prehospital care should have two components: (1) an outlet that leads directly into the cylinder line, and (2) a simple, easy-to-connect device that allows the oxygen hose to be quickly attached to the outlet.

Stewart recommends unscrewing the demand valve connection from the oxygen regulator of the tank and rapidly connecting the oxygen hose that has a nipple in place for fitting into the outlet. [77] He suggests leaving a small wrench fastened to the oxygen hose to allow a rapid transfer.

Two key points should be remembered: (1) high-pressure tubing should be used throughout the system, and (2) all connections and attachments should be well secured to avoid any disconnection under high pressure. Ligatures or other fasteners may be helpful in securing the attachments.

"Valve" Apparatus

It is essential to have the proper attachment for delivery of oxygen to the cannula or needle. Whatever the device or valve apparatus selected, it is critical to have all of the equipment ready, all of the connections available, and everything easily accessible. The apparatus can be gathered together and placed in one bag or kit and should be stored with the other airway management equipment.

First, high-pressure tubing goes from the oxygen source at 50 psi to a manually controlled device, which is then connected via high-pressure tubing to the cannula or needle in the cricothyroid membrane (Fig. 6-7). Stewart[78] recommends the manual valve by Instrumentation Industries (Bethel Park, PA), although others such as the Manujet by VBM (Germany) are also available.[77] A list of other "valve" apparatus options with varying costs ($5 to $200) has been published elsewhere. [99]

Note that standard commercial demand valves deliver only 60 cm H_2O (approximately 0.9 psi).[95] Systems using makeshift bag-valve assemblies (Fig. 6-8) do slightly better, but may deliver as little as 12 to 15 L/min of oxygen using 14- and 13-ga catheters, respectively.

Cannula or Needle

The third piece of equipment required is the cannula. A 13- or 14-ga needle catheter with a length of about 2 cm (or 1 inch) or a 3-mm ID cannula is preferred for percutaneous

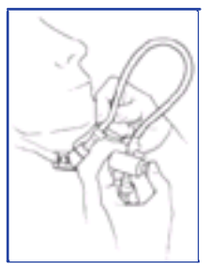

Figure 6-7 The equipment for translaryngeal ventilation. One-way valve used to control ventilation. Note that tubing is attached at the neck to the hub of the cricothyrotomy device, or if a tracheostomy is in place, the insufflation catheter can be passed into the tracheostomy tube.

translaryngeal ventilation. The size of the needle or cannula is critical. [100] Too small a cannula will not allow for sufficient ventilation, causing hypercapnia and even hypoxemia to develop.

Modifications for the cannula have been suggested: side holes, a slight curve, and a plate or flange. The advantage of side holes is that they will help keep the end or point of the cannula away from just one area of the tracheal wall. Thus, the full force of the 50 psi oxygen source is not directed at just one area of the laryngeal wall but is distributed in the larynx. The shape of the cannula may facilitate its initial placement or enhance complications. A fixation plate, flange, or side handle helps secure the cannula into place. A snug fit of the cannula will help prevent subcutaneous emphysema and ventilation of overlying soft tissue.

The use of a large-bore cricothyrotomy catheter (9 Fr ID with 13 Fr outside diameter [OD] and length of 6 cm) has been recommended instead of a 14-ga IV catheter. The large-bore cricothyrotomy catheter has several advantages: less chance of kinking or plugging up, improved mechanical structure, and the ability to be used with jet ventilation or bag-valve device ventilation. [101] [102]

Commercial Kits

Cannulas designed for cricothyrotomy or percutaneous translaryngeal ventilation are commercially available (Table 6-9). Several studies suggest that the "commercial" kits may have a higher complication rate due to a higher puncture/insertion force or a curved shape with more frequent posterior perforation, or both. [103] [104] [105] [106] [107] Placement of a commercial device may take longer than the traditional surgical cricothyrotomy. [108] [109] [110] One study suggests that procedural inexperience may be the primary factor causing delays whether with a percutaneous, wire-guided approach (Arndt Emergency Cricothyrotomy Catheter set, Cook Critical Care, Bloomington, IN) or via traditional surgical cricothyrotomy. [111] The use of

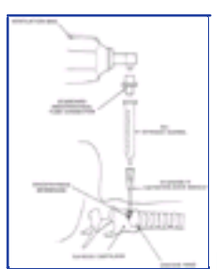

Figure 6-8 A simple setup for translaryngeal ventilation using standard equipment found in any emergency department. This setup is inadequate for adults. High-pressure (50 psi) ventilation systems (see Fig. 6-7) are optimal. Even with the pressure relief valve on the bag-valve device turned off, only a suboptimal pressure can be developed. However, this technique may be satisfactory in infants and small children.

one commercial cricothyrotomy device (Pertrach) is shown in Figure 6-9 .

Ventilation

Ventilatory parameters may be critical in preventing barotrauma and significant hypotension and in eliminating hypoxemia and hypercarbia. Animal studies suggest that the minute volume as reflected by a short inspiratory and a prolonged expiratory phase is an important parameter. [89] [97] [98]

Bag-valve devices should not be used for ventilation of adults or children unless a large catheter (3 mm ID) has been passed. The clinician should verify that adequate tidal volumes are being delivered with this technique by monitoring for hypoxemia.

Procedure

The actual technique of needle cricothyrotomy with translaryngeal jet ventilation is fairly simple (Fig. 6-10). The anatomy, landmarks, and several of the steps in the technique are identical to that for surgical cricothyrotomy (see Fig. 6-1 and Fig. 6-3).

A small 3- to 5-mL syringe containing 1 to 2 mL of sterile normal saline or water is attached to a large-bore needle (13 or 14 ga). If lidocaine will be injected, then 1 to 2 mL of 1% lidocaine without epinephrine should be in the syringe.

TABLE 6-9 -- Commercial Devices

Commercial Cricothyrotomy Devices
There are many commercial cricothyrotomy devices. A few of them are listed below. Mention is not intended to imply endorsement of these devices.
1. NU-TRAKE and PEDIA-TRAKE are distributed by International Medical Devices, Inc., PO Box 408 Canoga Park, 19355 Business Center Drive, Suite 8, Northridge, CA 91324, or PO Box 408 Canoga Park, CA 91305; phone (818) 701-5433 or (800) 522-LIFE (outside California)
2. Pertrach: Pertrach, Inc., 900 Davisson Run Rd, Suite 301, Clarksburg, WV 26301, phone (304) 624-7122 or (800) 736-3194
3. Abelson cricothyrotomy cannula: Gilbert Surgical Instruments, 115 Harding Ave, PO Box 458, Bellmawr, NJ 08031, phone (609) 933-2770
4. Quick Trach emergency cricothyrotomy device for adults and infants: VBM Medizintechnik GMBH, D-7247 Sulz am Neckar, Germany, phone (07454)6211; telefax (07454)4953
5. Portex Mini Trach Kit
6. Melker emergency transcricothyrotomy catheter set: Cook Critical Care, PO Box 489, Bloomington, IN 47402; phone (800) 457-4500
Commercial Transtracheal Puncture Devices
Also designed for transtracheal puncture, the Trans-Cricothyrotomy device is distributed by VBM Medizintechnik (same address and phone as above)
Commercial Manual Ventilator Devices
1. Manual valve that allows for the flow of oxygen when the control button is pushed. Manual Valve, by Instrumentation Industries, Bethel Park, PA
2. Manujet: a manual jetting device by VBM Medizintechnik GmbH, Bruhlstrasse 10, 0–727 Sulz am Neckar, Germany

If the patient is responsive, 1 to 2 mL of lidocaine may be injected into the larynx to prevent reflex coughing when the needle or cannula enters the larynx.

While the dominant hand (usually the right) holds the syringe, with the needle directed caudally at <45° to the skin, the other hand (nondominant) holds and stabilizes the larynx. The thumb and middle fingers of the nondominant hand stabilize the cricoid cartilage, and the index finger palpates the cricothyroid membrane.

The needle is inserted through soft tissues, the skin, and the cricothyroid membrane (see Fig. 6-10). While negative pressure is exerted on the barrel of the syringe, the needle is inserted through the cricothyroid membrane into the larynx. Air bubbles in the fluid-filled syringe signify entry into the larynx. After entering the larynx, the cannula is advanced into the larynx, and then the needle is removed. The cricothyroid membrane should be punctured in the inferior aspect (i.e., nearer the cricoid cartilage than the thyroid cartilage) to avoid the cricothyroid arteries (see Fig. 6-3*A*).

If there is much resistance to the needle's or catheter's passage through the skin, subcutaneous tissue, or cricothyroid membrane, kinking or bending of the catheter may occur unless a stiffer catheter is used. A small nick in the skin may be needed to facilitate passage through the dermis into the subcutaneous tissue. A percutaneous dilational or Seldinger guidewire technique may result in fewer complications. [112] [113]

The cannula is secured by suturing it to the skin or by placing a circumferential tie around the neck. The oxygen source is connected to the cannula. It is critical that the proximal end of the cannula be snug or tightly fitting and securely held around the puncture wound opening. If it is not securely held in place, subcutaneous emphysema will result or the cannula may be dislodged from the larynx, or both. A trial of several bursts of oxygen flow is recommended to make certain that the cannula is correctly placed and the setup is working and ventilating properly.

The hypoxic patient should receive 100% oxygen in intermittent bursts <50 psi at a rate of 20 bursts per minute. For children, 30 psi has been recommended. The percentage of inspired oxygen concentration can then be adjusted, depending on blood gas results. The inspiratory phase or insufflation with the burst of oxygen should last approximately 1 second, and the expiratory phase long enough to allow for adequate exhalation (2 to 9 seconds has been suggested).

Precautions

The location of the needle puncture (or incision) in the skin and cricothyroid membrane is critical. The preferred location is near the midline in the inferior aspect of the cricothyroid membrane, just above the cricoid cartilage. The needle (or scalpel) is directed at an approximately 45° angle to the skin in a caudal direction. The key landmarks in the neck should be identified before inserting the needle-cannula combination into the cricothyroid membrane.

Too small an opening in the cricothyroid membrane leads to hypoxemia, hypercapnia, and respiratory acidosis. In an adult, a 13- or 14-ga needle or larger bore with a 3-mm ID is needed to provide an adequate-sized opening.

Use of a bag-valve device or a demand valve resuscitator apparatus to ventilate a patient through a translaryngeal cannula is generally inadequate. Using a 50 psi oxygen source, up to 89 L/min can be delivered through a 13-ga cannula, and up to 72 L/min can be delivered through a 14-ga cannula. [95] Additional side holes or ports in the cannula to allow for the additional egress of oxygen into the trachea and to limit the obstruction of the cannula by mucus plugs are desirable.

During the insufflation phase of percutaneous translaryngeal ventilation, secretions in the upper airway are blown out of the mouth and nose. It is recommended that personnel stand clear of the patient's face to avoid being sprayed with secretions when oxygen exits through the patient's glottis and pushes oropharyngeal secretions out the glottis and then out the nose and mouth. This may be a useful side effect if partial upper airway obstruction is present, as with a foreign body (e.g., a bolus of meat or a peanut), because some of the delivered volume of air exiting through the patient's glottis might dislodge such a foreign body. The risk of aspiration is decreased or eliminated by lowering the head. The proximal end of the cannula should be snug against the puncture wound to minimize localized subcutaneous emphysema and should be secured in place to prevent the cannula from being dislodged.

Complications

The exact incidence of complications with percutaneous translaryngeal ventilation is not known, but it is thought to

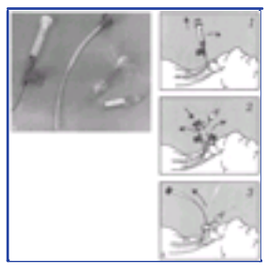

Figure 6-9 *A*, Commercial device used in performing a cricothyrotomy with a modified Seldinger technique. The adult Pertrach percutaneous emergency airway provides a means of rapidly inserting a tube into a patient's trachea (through either the cricothyroid membrane or trachea) in certain clinical situations. It provides an adequate airway that can, by its standard 15-mm adapter, be used to allow the patient to breathe. The unit allows full control of the airway with inflation of the cuff. The pediatric device is provided uncuffed in three sizes for infants and children of various sizes. The 15-mm adapter is part of each unit. (Tracheostomy *only* is recommended in children and infants.) *B*, Method of establishing an airway with the Pertrach cricothyrotomy kit: (1) The 14-ga needle is inserted into the trachea through a cricothyroid membrane or upper trachea. Placement in the airway is verified by drawing air. The syringe is removed. (2) The dilator is placed to its hilt into the tracheostomy tube, and both are inserted as a single unit. The leader of the dilator is inserted into the trachea through the needle, which is then split and removed. The dilator and trachea tube are inserted into the trachea. Note that a small skin incision at the puncture site is needed to facilitate passage of the tracheal tube. (3) The dilator is removed. The cuff can then be inflated and a respirator applied. *(Courtesy of Pertrach, Inc., Clarksburg, WV 26301.)*

Figure 6-10 The procedure for transtracheal ventilation. *A*, The cricothyroid membrane is punctured with the needle or catheter aimed caudally at approximately a 45° angle. *B*, The inner needle has been removed, and the catheter is hooked up to a manual in-line device and then to a source of oxygen. A valve device or tubing modification as shown in Figure 6-7 is also required.

be low, considering that the complication rate of translaryngeal puncture alone is in the range of 0.03% to 0.8% (Table 6-10).[114]

Bleeding can occur at the site of the needle puncture, but this is usually not a major problem. Hemoptysis may occur but is infrequent. Fatal hemorrhage secondary to transtracheal aspiration for sputum cultures has been reported, [56] although no bleeding fatalities secondary to percutaneous transtracheal ventilation have been reported.

Minor bleeding is the most common complication of a surgical cricothyrotomy, whereas subcutaneous emphysema is the most frequent complication of percutaneous translaryngeal ventilation. With percutaneous translaryngeal ventilation, subcutaneous emphysema will occur if egress of gas is prevented by inadequate exhalation (e.g., with occlusion of the mouth and nose or with inadequate cannula size). The development of subcutaneous emphysema with percutaneous transtracheal ventilation is often immediate and dramatic. If this complication does occur, the subcutaneous emphysema may be gently squeezed away from the midline and the trachea can often be recannulated and successfully ventilated. Subcutaneous emphysema can be decreased or avoided by making sure that the flange or hub of the cannula fits securely against the skin.

Barotrauma secondary to high airway pressures may occur if percutaneous translaryngeal ventilation is used when

TABLE 6-10 -- Complications of Percutaneous Translaryngeal Jet Ventilation

Common
Subcutaneous emphysema—most common (less occurrence if there is a "secure" fit at the skin)

Kinking of the catheter
Blockage or obstruction of the catheter
Coughing (in a conscious patient)
Infrequent
Bleeding (minor), hematoma
Infections
Aspiration
Incorrect or unsuccessful catheter placement
Prolonged procedure time
Persistent stoma
Subjective feeling of a "lump in the throat"
Pneumatocele
Serious, rare complications
Barotrauma (secondary to high airway pressures, more common with complete airway obstruction)
Pneumothorax
Pneumomediastinum (less occurrence if high airway pressures are avoided, and not performed with complete airway obstruction)
Mediastinal perforation
Esophageal perforation
Dysphonia or voice changes (secondary to vocal cord injury, laryngeal fracture, or disruption of laryngeal cartilage)
Potential or theoretical complications (not yet commonly associated with percutaneous translaryngeal jet ventilation, although reported with tracheostomy and other airway procedures)
Subglottic/glottic stenosis
Tracheoesophageal fistula
Damage to laryngotracheal mucosa (such as tracheobronchitis)
Swallowing problems

complete airway obstruction is present. Such barotrauma could lead to serious complications, including pneumothorax, pneumomediastinum, and subcutaneous emphysema.

Another side effect that can occur in the conscious patient is coughing with each burst of oxygen. A few milliliters of lidocaine may be injected into the larynx to help prevent coughing when the cannula enters the larynx. The initial instillation of lidocaine solution will precipitate a brief coughing episode, but further coughing should be minimized. However, because the patient undergoing cricothyrotomy in the ED is almost always critically ill and is rarely conscious, coughing is usually not a problem.

Percutaneous translaryngeal ventilation for an extended period of time can dry the membranes of the airway unless appropriate humidification is obtained. Techniques or devices for humidification are available. [115]

One complication that may occur with needle cricothyrotomy is kinking of the catheter as it enters the neck. This problem can be overcome if a stiffer catheter is used. A 14-ga (1 ¼-inch) catheter or any device approximately 2 cm long from the hub or flange of the cannula has been recommended as the best length, because it puts the tip of the catheter in the middle or midstream of the airway.

Infection, ranging from cellulitis, perichondritis, or laryngotracheitis, is a possible complication of percutaneous translaryngeal ventilation, but it has not been reported in most series. Careful technique and follow-up care should help prevent these complications of infection and obstruction by blockage of the tube.

Damage to the laryngotracheal mucosa, including necrotizing tracheobronchitis, from percutaneous translaryngeal ventilation is a theoretical complication that has not been reported. Most studies have concluded that percutaneous translaryngeal ventilation or high-frequency jet ventilation may be performed "without undue risk of tracheobronchial injury."[116]

Blockage or obstruction of the tube from bleeding or from a mucus plug occurs infrequently, although such blockage can cause acute airway obstruction. Additional small holes near the tip of the catheter may help to prevent this problem.

A pneumatocele caused by incorrect needle placement is a rare and benign complication that can be treated by aspiration with a needle. Misplacement of the cannula can lead to tracheal, mediastinal, or esophageal perforation if the needle is advanced too far. Other complications that occur with surgical cricothyrotomy could occur with needle cricothyrotomy, such as damage to the laryngeal cartilage, which may cause dysphonia or voice changes.

CONCLUSION

Cricothyrotomy is a simple, reliable, rapid, and effective means of achieving airway control and ventilation. It has relatively few complications. The value of cricothyrotomy as a life-saving procedure in emergency situations has been clearly demonstrated, and its usefulness has been well established in emergency care. Both surgical cricothyrotomy and needle cricothyrotomy with translaryngeal jet ventilation can be used for an extended period of time if appropriate ventilation factors are maintained. Cricothyrotomy has also gained acceptance as an elective procedure for surgical access to the airway. In many instances, cricothyrotomy is the airway procedure of choice.

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Chapter 7 - Tracheostomy Care and Tracheal Suctioning

Kathleen A. Neacy

TRACHEOSTOMY CARE

Tracheostomy patients may present to the emergency department (ED) with respiratory compromise, recent and long-term postoperative complications related to the tracheostomy, or, most commonly, with complaints unrelated to the tracheostomy itself. The emergency clinician's evaluation, based on the clinical scenario, includes assessment of the tracheostomy site's appearance and function, appropriate routine care, cleaning and suctioning of the apparatus and site, and initial management of life-threatening complications. This chapter will review tracheostomy equipment, routine suctioning and hygiene, early and late postoperative complications, and procedures and techniques helpful in managing routine and emergent tracheostomy problems. In addition, tracheal suctioning techniques will be reviewed.

Background

Tracheostomy has been performed since 2000 B.C. and is one of the oldest reported surgical procedures in the medical literature. Mortality associated with tracheostomy is low. Most tracheostomies are performed electively, with a postoperative complication rate of 10% to 40%. [1] Emergent tracheostomy is indicated under certain circumstances, such as laryngeal trauma, when emergency orotracheal intubation or cricothyrotomy may be complicated or contraindicated. The complication rate for emergent tracheotomy is several-fold higher than reported with elective tracheostomy. [2]

Elective tracheostomy can be performed in the operating room, using an open technique, or at the bedside in the intensive care unit, using a percutaneous dilation technique (PDT). The surgical site is located between either the first and second, or the second and third tracheal rings. With open tracheostomy, the anterior trachea is generally sutured to the skin until tract maturation occurs, 4 to 5 days postprocedure. PDT eliminates the need to transport critically ill patients to the operating room. In addition, the use of blunt dissection and dilation, combined with a tightly fitting tracheostomy tube, contributes to a lower postoperative bleeding rate. [3] The smaller stoma created with PDT lead to complications such as difficult replacement following displacement in the immediate postoperative period, and stomal occlusion or stenosis.

Newer procedures reviewed in this chapter include transesophageal puncture for voice restoration, minitracheostomies for tracheal suctioning, and transtracheal catheter placement for low-flow oxygen administration.

Indications for Tracheostomy

Commonly accepted indications for tracheostomy include chronic respiratory insufficiency, airway obstruction, and impaired secretory clearance. Patients with chronic respiratory insufficiency who require prolonged ventilation often undergo tracheostomy to avoid long-term complications of orotracheal intubation, including laryngeal dysfunction or subglottic stenosis. Causes of upper airway obstruction that may result in tracheostomy include head and neck cancers, trauma, infections, angioedema, and foreign bodies. Long-term pulmonary toilet can be enhanced with elective tracheostomy in patients who have impaired cough reflexes, increased secretions, or ciliary dysfunction. [4] In the pediatric population, tracheostomy is performed most frequently for upper airway obstruction, ventilator dependence, laryngeal diversion, and central hypoventilation syndrome. [5]

Indications for emergency tracheostomy are rare, as orotracheal intubation or cricothyroidotomy are most often used for emergency airway management. Laryngeal trauma, edema of the oropharynx, fractured mandible, and uncontrolled upper airway hemorrhage are reported indications for emergency tracheostomy. [6]

Tracheal Anatomy, Physiology, and Surgical Considerations

The adult trachea is approximately 10 to 12 cm in length. Forming the anterior and lateral walls of the trachea are 18 to 22 incomplete cartilaginous rings. A fibromuscular sheet completes the posterior wall, which lies anterior to the esophagus. The interior diameter of the adult trachea is approximately 12 to 25 mm, and is lined with mucosa, covered by respiratory epithelium. [4] Blood supply to the trachea comes from branches of the inferior thyroid artery. The innominate (bracheocephalic) artery lies in proximity to the tracheostomy stoma. From its origin at the aortic arch, it courses between the sternum and the anterior trachea, veering right at the sternomanubrial joint. The location of the innominate artery is important, as erosion of the anterior tracheal wall can lead to a major life-threatening bleed from this artery (see *Complications, Bleeding* later).

The recurrent laryngeal nerve supplies innervation to most intrinsic laryngeal muscles and innervates the mucosa below the vocal cords. Efferent vagal fibers stimulate bronchoconstriction, mucosal secretions, and vasodilation. Efferent sympathetic fibers of the pulmonary plexus stimulate tracheal bronchodilation and vasoconstriction.

The upper airway, including the oropharynx and nasal passages, filter particulate matter, humidify inspired air, and aid in expectoration of secretions. These functions are reduced in patients with tracheostomy. [7] Placement of a tracheostomy bypasses humidification and results in formation of thick, dry secretions. [9] Normal mucociliary clearance is often impaired due to increased viscosity of respiratory secretions, underlying chronic illness, and respiratory infections, particularly due to mycoplasma or viral pathogens. [10] Tracheostomy weakens the anterior tracheal wall, allowing possible tracheal collapse. The cough mechanism, also important in tracheal secretory clearance, is often blunted in tracheostomy patients. [10] Normally, the epiglottis and vocal cords close to trap air in the lungs and raise intrathoracic pressure prior to a cough. Patients with a tracheostomy tube are generally unable to generate sufficient pressure to initiate a strong cough and facilitate airway clearance. [8] Physiologic PEEP (positive end-expiratory pressure) is eliminated in patients unable to maintain a closed glottis.

Immune responses are often blunted in patients with tracheostomies due to underlying illnesses, chemotherapy, or

acquired immunosuppression. Pulmonary macrophage and polymorphonuclear cell function in particular are impaired. [10]

Evaluation of the Tracheostomy Patient

Independent of a patient's presenting complaint, a thorough assessment of the airway, respiratory status, and indwelling device should be undertaken in any ED patient with a tracheostomy and respiratory complaint. Tube occlusion, dislodgment, or fracture should be considered. The clinician should verify snug placement of the tracheostomy tube, the presence of blood or secretions, integrity of the skin at the tracheostomy site, and the patient's ability to speak or mentate. A rapid physical examination should help differentiate between tracheostomy complications and other etiologies as the basis of a patient's problem.

Historical components of the evaluation include the indication for tracheostomy placement, the length of time from tracheostomy to ED presentation, and previous complications. Other historical considerations that may be helpful in the ED evaluation include planned or existing voice prosthesis surgeries, previous bleeding

complications or strictures, and whether permanent tracheostomy or planned decannulation of the tracheostomy site is anticipated. Changes in the efficacy of home ventilator or tracheostomy care may help identify potential problems, including increased oxygen use, increased suctioning, equipment failure, or skill in out-of-hospital tube changes.

Complications of Tracheostomy

Obstruction

Any tracheostomy patient who presents with respiratory distress should first be assumed to have a partially or completely obstructed tracheostomy tube. [11] Improper maintenance and poor attention to routine tracheostomy care in the outpatient setting are common, exemplified by the observation that a plugged tube accounted for 30% of ED presentations for respiratory distress in one review. [12] Plugging occurs most commonly with dried respiratory secretions and, less often, with blood or aspirated materials. Secretions may act as a ball valve, allowing air in, but restricting outward ventilation. [4] Granuloma formation just distal to the tube may also cause obstruction. Some patients and caretakers simply do not understand the principles of tracheostomy care, and fail to routinely clean the tube, or fail to even understand the function of an inner cannula.

Emergent management includes high-flow oxygen, and preparing for immediate tube exchange (see *Tube Exchange Technique*, later). Obstructions at the exterior tracheal tube opening can be manually removed. The inner cannula should be removed and cleaned of dried secretions. If no inner cannula is in place, the tracheostomy tube can be suctioned to remove obstructing plugs (see *Suctioning*, later). Instillation of normal saline to loosen thick secretions can be helpful with a critical patient, but is no longer recommended in routine suctioning. [13] [14] If evidence of obstruction persists, the outer tube should be removed and replaced. Persistent respiratory insufficiency despite these maneuvers should lead the clinician to consider other etiologies for the patient's complaints. [11]

Prior to hospital discharge, Rumback and colleagues evaluated tracheostomy patients with a trial of occlusion of their tracheostomy catheter with the tracheal cuff deflated.[15] If the patient developed significant hemodynamic changes (>15% change in respiratory rate, heart rate, or blood pressure), oxygen desaturation of <90% or other signs of respiratory distress or diaphoresis while under observation, they were investigated for potentially obstructive tracheal lesions (e.g., granulation tissue, tracheal stenosis, tumor, edema). Surgical interventions or downsizing of the tracheal tube were undertaken before the patient was released. A modification of their protocol in the ED after the clearing of secretions and resolution of obstructive symptoms will identify patients at risk for subsequent airway obstruction and for whom further otolaryngologic consultation is warranted.

Dislodgment

Tracheal tube displacement can occur when traction is placed on the tube, especially when it is manipulated for ambu-bag or ventilator connections. Long tracheostomy tubes can be dislodged inferiorly, causing the tip to abut the mucosal wall of the trachea, or obstruct at the level of the carina. The clinician should determine that the tube flanges rest snugly at the skin. Lateral neck x-rays may reveal that the tracheostomy tube opening abuts the anterior tracheal wall or obstructs the tracheal lumen. Gentle manipulation of the tube or repositioning may resolve the obstruction. Since flexion of the neck can cause downward displacement of the tube as much as 3 to 4 cm,[6] gentle extension of the neck may restore air flow. Tracheal tubes connected to respirators should be secured with tube supports or holding arms.

False Passage

Creation of a false lumen can occur during tracheal tube replacement, or during repositioning of a dislodged tube. Subcutaneous air, crepitus, or distortion of the anterior neck landmarks may indicate placement of the tracheostomy tube into a soft tissue lumen anterior to the trachea. Abdominal distention after bagging may indicate tracheal tube placement through a tracheoesophageal (TE) fistula. Expeditious removal and replacement of the tracheostomy tube are required if a false passage is suspected.

Fracture

Tracheostomy tube fractures occur infrequently, and are more common with metal tubes. Tube fracture is presumed secondary to metal fatigue and interactions with respiratory secretions. Tube fractures occur most frequently at the juncture of the flange and the tube connection. [16] Lateral neck radiographs may show air outlining the cannula at the flange.[16] A fractured tube fragment may displace inferiorly, obstructing the tracheal lumen. Management should include replacement of the tube and possible bronchoscopy for retrieval of the tube fragment, if present. [17] Cardiac complications have been reported with aspiration of tracheostomy tubes. [17]

Tracheal Cuff Complications

Complications related to the tracheostomy tube cuff include cuff perforation, resulting in poor seal and increased aspiration risk; over-inflation, causing pressure on or impingement of the esophageal lumen; and distention of the cuff distal to the tracheal tube, causing obstruction of the tracheal tube opening. Mucosal injury is much less common since the use of low-pressure cuffs became standard. Pain with ventilation or swallowing, inadequate oxygenation despite correct tube placement, or presence of gastric secretions in the tracheostomy tube may indicate cuff problems. Appropriate measures include verification of appropriate inflation pressures (18 to 23 mm Hg),

and cuff position. If these symptoms persist, the tube should be replaced.

Bleeding

Bleeding complications account for 20% of ED tracheostomy presentations. [12] Minor bleeding occurs in up to 30% to 40% of patients and 0.2% to 10% having massive, or life-threatening bleeding complications. [4] [6] [18] Sources of bleeding include small, superficial blood vessels at the tracheostomy site, granulation tissue, thyroid vessels, anterior jugular veins, and the bracheocephalic (innominate) artery. Bleeding from esophageal or gastric sources may present as tracheal bleeding if a TE fistula is present,[4] or if the patient has aspirated blood. Erosion of a major vessel from the cuff or tip of the tube is responsible for 10% of all tracheostomy hemorrhage, and for most tracheostomy-related deaths. The innominate artery is the most commonly involved vessel. [19] Thyroid artery bleeding occurs in 5% of patients during the immediate postoperative period, but rarely accounts for ED presentations.

Early bleeding, within the first 4 weeks postoperatively, is most commonly incisional. However, 85% of tracheoinnominate artery fistula bleeds are reported within the first postoperative month.[4] Tracheoinnominate artery fistula occurs in approximately 2% of patients with tracheostomies, and has a 50% to 75% mortality rate, despite identification and emergent management. [6] [12] The innominate artery crosses from left to right as it moves superiorly, and lies immediately anterior to the trachea at the level of the superior thoracic inlet. Risk factors for developing a tracheoinnominate artery fistula include placement of the tracheostomy stoma below the third tracheal ring, caudal migration of the tracheostomy tube from leverage on the tube, and presence of a more cephalad-coursing innominate artery, the latter more prevalent in thin, young patients. [12] Pressure exerted by the distal tracheostomy tube or cuff causes erosion of the anterior tracheal wall into the vessel, and subsequent bleeding. Brisk bleeding from the tracheostomy, hemoptysis, or a history of either complaint should alert the clinician to the possibility of a life-threatening bleed. Many patients experience a "sentinel" bleed hours or days before a catastrophic bleed. Some patients may report only a new cough or retrosternal pain. [19] Any history or evidence of 10 mL or more of blood should be presumed arterial.

Visualization of the bleeding site can be attempted in stable patients. A bleeding innominate artery will be located at or below the sternal notch, in the anterior tracheal wall. If significant tracheal bleeding is present, hyperinflation of the tracheostomy tube cuff should be attempted to compress the artery against the sternal wall. If this is unsuccessful, the patient should have an endotracheal tube inserted through the oropharynx, with the cuff positioned at the level of the upper sternum, and hyperinflated. Digital pressure applied through the tracheal stoma, compressing the anterior tracheal wall against the sternum, should be done for continued bleeding (Fig. 7-1). Digital pressure is considered the most reliable technique to stop hemorrhage [12] and may provide control of bleeding during transport to the operating room. Emergent surgical consultation is mandatory.

Management of Minor Bleeds

Incisional bleeding or bleeding from granulation tissue is usually confined to the skin surrounding the stoma. Bloody secretions issuing from the tracheostomy tube may represent

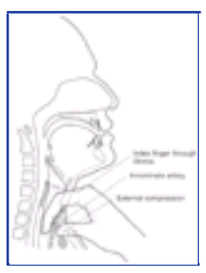

Figure 7-1 Control of innominate artery bleed by digital pressure. When major bleeding occurs and a cuffed tracheostomy tube is present, overinflation of the tube cuff may temporize (see text). When unsuccessful or a cuffed tube is not available, use the illustrated maneuver; digital pressure should be applied to the anterior tracheal wall through the tracheostomy. The index finger is placed within the trachea and then pulled against the anterior tracheal wall, allowing the airway to remain partially open. The artery is compressed between the index finger and the thumb—placed over the anterior neck. Digital compression of the innominate artery is a temporizing procedure, until definitive (operative) management of the bleed is obtained.

diffuse tracheitis, rundown bleeding from the skin or thyroid, or superficial tracheal ulceration from suctioning or tracheal tube pressure. Examination of the stoma site and tube gauze may indicate the source and volume of blood loss. The tracheal lumen may be visualized with a nasopharyngoscope. The proximal trachea and inner stoma can also be visualized by inserting a small pediatric laryngoscope blade into the stoma. Superficial erosions should be differentiated from active bleeding. Clots in the trachea should not be disturbed, as this may increase the rate of hemorrhage.

Incisional or stomal bleeding can usually be controlled by cauterization or packing the wound with petroleum jelly gauze. [6] After examination and management of minor bleeding, the tracheostomy tube should be replaced. Careful suctioning following tube replacement should confirm resolution of superficial bleeding, or identification of secondary sources of bleeding.

If stomal bleeding or intratracheal sites do not account for bleeding, other sites should be considered. Gastrointestinal bleeding may be identified by placement of an NG (nasogastric) tube. The nasopharynx and oropharynx should also be examined for possible bleeding sources. Mucosal bleeding from radiation therapy may occur above the level of the tracheostomy stoma and be present in tracheal secretions.

Infection

Infections are common in patients with tracheostomies and account for approximately 50% of ED presentations. [12] Underlying medical conditions, impaired host defense mechanisms, and poor nutrition all increase susceptibility to infection. Additional contributing factors include contamination of the tracheostomy stoma, the presence of an indwelling device, and exposure to flora colonizing ventilator tubing.

Hackeling reported 18 ED patients diagnosed with tracheostomy-related infections. Four had paratracheal cellulitis, and 14 were diagnosed with bronchitis, tracheitis, or pneumonia. The infectious agents cultured most frequently in tracheostomy-induced cellulitis included *Staphylococcus aureus, Pseudomonas* species, and *Monilia*.[12] Because some degree of aspiration occurs in 65% to 85% of all patients with tracheostomies, [20] enteric organisms are commonly cultured from the sputum of tracheostomy patients. Accurate identification of infectious organisms from carefully obtained sputum samples is essential in this population. Colonization of the stoma is common, but infection is unusual. Peristomal cellulitis can usually be treated with oral outpatient antibiotics. The most feared complications from cellulitis are mediastinitis, mediastinal abscess, and paratracheal abscess. Pain with breathing or swallowing or signs of systemic infection should be investigated for these complications, usually by obtaining a computed tomography (CT) scan.

In Hackeling's review, all tracheostomy patients with infections other than cellulitis were admitted for intravenous antibiotics. Depending on the patient's presentation, a cautious approach to management of infections in this population seems warranted.

Tracheal Stenosis and Tracheomalacia

Tracheal stenosis and tracheomalacia are late complications of tracheostomy, and often present weeks to months after decannulation. Tracheal stenosis can occur at the subglottic area, stoma, or cuff. [6] Pressure on the tracheal lumen from the tracheostomy tube or cuff can cause epithelial destruction, tracheitis, ulceration, persistent inflammation, and subsequent stenosis. [6] Rigid tube systems with excessive motion and pressure points can lead to stenosis at the stoma site. [6] Cough, retained secretions, and progressive dyspnea on exertion usually herald the onset of clinically significant stenosis. Symptoms become evident when the tracheal diameter is narrowed by 50% to 75%. Stridor occurs when the tracheal lumen is =5 mm. [6]

Weakening of the tracheal cartilages from pressure necrosis may cause luminal widening and tracheomalacia. Tracheostomy tube loosening can result, as can tracheal collapse. Pediatric patients are less able to tolerate cartilaginous weakening and tracheomalacia. Cuff over-inflation and tracheal wall dilation can be detected on chest radiography. [6]

Tracheal stenosis can be accurately identified in the ED using laryngoscopy. [12] CT scans are not sensitive in detecting stenosis. Definitive diagnosis is obtained by bronchoscopy, with operative dilation of the stenosis, or resection of granulation tissue being the most common treatment.

Relief of any associated respiratory compromise is problematic because a high-grade stenosis may make endotracheal intubation difficult or impossible. If attempts to intubate fail due to this type of obstruction, temporizing measures can be taken while awaiting transport to the operating room. The patient should be placed on high-flow humidified oxygen and positioned with the head elevated. Application of nebulized bronchodilators or racemic epinephrine may be helpful.

Tracheoesophageal (TE) fistula

Early TE fistulas can occur if a puncture wound or small laceration is made in the anterior esophagus during tracheostomy surgeries. Delayed TE fistulas can occur from a poorly fitting tracheostomy tube impinging on an NG tube, through the common wall of the anterior esophagus. Symptoms of a TE fistula include persistent leak around the cuff, abdominal distention, or evidence of aspiration. The clinician may be able to auscultate breath sounds simultaneously over the lung fields and epigastrium. Bronchoscopy or swallowing studies can aid in making the diagnosis.

Routine Tracheostomy Care

Routine tracheostomy care is focused on maintenance of a patent tube through removal of respiratory secretions. Most tracheostomy tube obstructions occur from the accumulation of dried secretions at the tube tip or within the inner cannula. Intermittent or continuous inhalation of humidified air decreases mucus viscosity and reduces the accumulation of secretions.

The routine cleaning of the tube and inner cannula can effectively reduce accumulation of dried secretions. The inner cannula fits snugly into the tracheostomy tube, and can be easily removed without disturbing tracheostomy tube placement. It should be soaked in hydrogen peroxide solution for 10 to 15 minutes and scrubbed lightly with a soft brush to remove encrusted secretions. [9] The tracheostomy tube flanges should be cleaned with dilute hydrogen peroxide if dried blood or secretions have accumulated. All airway equipment should be thoroughly rinsed in sterile saline before reinserting, as hydrogen peroxide may cause mucosal irritation and increased tracheal secretions. [8]

Suction alone is the preferred way to initially remove excessive secretions. Instillation of normal saline into the tracheostomy tube to loosen secretions is no longer

initially recommended for routine suctioning. Saline and mucus are immiscible. The common practice of instilling 5 to 10 mL of sterile saline may potentially hamper oxygenation, especially during suctioning. Instillation of saline elicits a cough in most patients, usually preceded by forceful inspiration that may draw loose airway material and organisms into the lower respiratory tract. Dislodgment of tube encrustations by a similar mechanism may have the same undesirable effect. For these reasons, instillation of saline for routine tracheostomy care is discouraged. [13] [14] [21] However, the use of saline for thick secretions not responding to routine suction may be needed. The administration of supplemental oxygen can generally control any mild hypoxia associated with aspiration of the small amount of saline used to soften the thick secretions.

Stomal wound care is enhanced by changing tube ties that have been contaminated with secretions, by routine cleansing of the tube flanges, and by using precut tracheostomy gauze. Loose fibers from hand-cut gauze may induce inflammatory changes at the stomal site. [8]

Changing a Tracheostomy Tube

Indications

Tracheostomy tract maturation is generally completed by 5 days postoperatively. Since most tracheostomy patients who

present to the ED do so beyond this time frame,[12] routine changes of most tracheostomy tubes can be safely done in the ED. Indications for tracheostomy tube exchange, electively or emergently, include cuff rupture or leak, peritubal leak caused by tracheomalacia, complete or partial tube occlusion, or conversion to an alternate tube style (e.g., unfenestrated to fenestrated). [22] Routine cleaning of the tube also requires removal and exchange, often done by the patient at home every few weeks.

Few contraindications to tube exchange exist. The clinician should consider whether further tissue trauma or hemorrhage may occur as a result of the procedure [22] and anticipate possible complications, including loss of the airway, before undertaking the tube exchange.

Equipment and Setup

Tracheostomy tubes may be made of metal (Jackson), plastic (Shiley), silicone, or nylon. The main components are an outer cannula, used to secure the tube to the skin, an inner cannula, which has a standard 15-mm respiratory connector for attachment to a bag-valve device or ventilator tubing, and an obturator, the smooth blunt end of which facilitates tracheostomy tube insertion (Fig. 7-2). The outer cannula is the more permanent portion of the tube, as it remains in place unless there are complications. Air exchange is accomplished through the lumen of the inner cannula. *It is imperative to note that the inner cannula contains the adapter for a standard ambu bag. Without the inner cannula, effective ventilations cannot be maintained since a seal cannot be obtained between the ambu bag and the outer cannula.* The inner cannula is removable, and fits snugly into the outer cannula. The inner cannula is the portion of the tube that is routinely removed, cleaned of secretions, and replaced to provide the

Figure 7-2 Tracheostomy tube *(top)* with inner cannula and attached ambu bag adaptor *(middle)* and obturator *(bottom)*.

airway. An obturator has a rounded or cone-shaped distal end, and fits snugly inside the inner cannula, generally extending several millimeters beyond its distal end. The obturator is solid and has no lumen; therefore, it has no purpose except as an aid in tube passage.

The size of the tracheostomy tube is usually stamped on the flange of the outer cannula, and indicates the tube's inner diameter (Table 7-1). The tube is secured in place by ties that tether the flange and should be tightened so that one finger can be placed snugly between the tracheostomy ties and the patient's skin.

Cuffed tracheostomy tubes are used for patients on long-term mechanical ventilation, or those at risk of aspiration. In addition to preventing ventilatory volume loss during positive pressure ventilation and reducing the risk of aspiration, inflation of the cuff prevents diversion of expired air across the vocal cords; therefore, speech is not possible with cuffed tubes.

Most low-volume, high-pressure cuffs have been replaced by high-volume, low-pressure cuffs that reduce mucosal injury and the risk of tracheal erosion or stenosis.[23] The cuff is inflated by attaching a syringe to the Leur-lok port at the proximal end of the pilot balloon. Cuff inflation can be verified by palpating the pilot balloon. The cuff pressure can be determined by connecting the Leur-lok port to a manometer. Recommended cuff pressures are between 18 and 23 mm Hg. [5] [24]

Uncuffed tubes can be used in patients with adequate ventilatory effort, who are awake and at low risk of aspiration. Depending on the size of the tracheostomy tube and how much of the tracheal diameter the tube fills, air can bypass the tracheostomy tube and be transmitted across the vocal cords. Digital occlusion of the tracheostomy, or use of specialized valves attached to the tube opening, can occlude expired air from the tracheostomy, and enhance voice production. Certain tracheostomy tubes have single or multiple fenestrations, which allow air to be transmitted over the vocal cords. Fenestrations are generally located at the superior, posterior arch of the tracheostomy tube and are sometimes also found on the inner cannula.

In an emergency, the proper sized replacement tracheostomy tube may not be available. In such instances a standard *endotracheal* tube may serve as a temporary replacement. Usually a smaller tube than would be used for orotracheal intubation is used. In the adult, a 6- to 7.5-size tube may be accommodated by the stoma and trachea. This tube should not be advanced more than is necessary to provide ventilation. The cuff may be cautiously inflated to assure a proper seal.

TABLE 7-1 -- Tracheostomy Tube Recommended Sizes

Age	Size	Inner Diameter (mm)	Outer Diameter (mm)
Premature	00	3.1	4.5
Newborn-3 mo	0	3.4	5.0
3–10 mo	1	3.7	5.5
10–12 mo	2	4.1	6.0
13–24 mo	3	4.8	7.0
2–9 year	4	5.0	8.5
10–11 year	6	7.0	10.0
12-older	6 or larger		
	8	8.5	12.0
	10	9.0	13.0

Adapted from Mullins et al: Airway resistance and work of breathing in tracheostomy tubes. Laryngoscope 103:1367, 1993.

Very short tracheal tubes that maintain the tracheostomy opening, but do not protrude into the tracheal lumen, are available. Known as tracheal buttons, these devices are used during weaning to maintain stoma patency. Tracheal buttons can become displaced into the tracheal lumen if not correctly tethered, and may become clogged with secretions.[25] Speaking valves, such as the Passy-Muir (Passy-Muir, Inc., Irvine, CA) or the Shiley-Phonate (Mallinckrodt Medical, St. Louis) may be present. These devices generally clip or twist on to the 15 mm coupling of the tracheostomy tube or inner cannula. Speaking valves should be removed before changing a tracheostomy tube.

Procedure

Tracheostomy supplies and adjunct airway equipment should be at the bedside before beginning the procedure. The flange on the patient's indwelling tracheostomy tube will have a size stamped on it; replacement tubes available should include this size and one or two sizes smaller. Component parts should be inspected prior to use. They should fit together easily, and if a cuff is present, it should be inflated to check for leaks. A nasopharyngoscope or red rubber catheter may facilitate tube exchange if a modified Seldinger technique is used (Fig. 7-3). Additional airway equipment, including endotracheal tubes, laryngoscope, suction catheters, and oxygen should be at the bedside, in case difficulties in tube exchange are encountered. A list of recommended equipment is outlined in Table 7-2 . Soft

Figure 7-3 Changing a tracheostomy tube. *A,* Before the old tube is removed, a small red rubber catheter (or other guide catheter) is passed into the proximal trachea. *B,* The tracheostomy tube has been removed over the catheter, and only the catheter remains in the trachea. The catheter serves as a guide for easy and atraumatic insertion of a new tube. Note that the neck should be slightly hyperextended. *C,* A new tracheostomy tube, without the obturator is threaded over the guide catheter; once the tube is in place, the catheter is removed. Similarly, if the tracheostomy tube has already been removed, the catheter may be passed through the stoma before a new tube is advanced. Note that an obturator or inner cannula is *not* used when changing a tube with this technique.

restraints or anxiolytic medication may be used at the clinician's discretion.

Ideally, the patient should be preoxygenated prior to tube exchange, either by providing supplemental oxygen with a face mask applied over the tracheostomy stoma or by manual bagging. However, patients with significant tube obstruction require immediate relief of the obstruction, and preoxygenation may not be possible. [11] The patient should be positioned with the neck slightly *hyperextended*. Flexion of the neck may misalign tissues and hamper attempts at tube passage. Adults may sit or lie down. Small children should be placed supine. A towel roll under a child's shoulders may facilitate mild hyperextension.

The new tracheostomy tube should be lubricated with water-soluble lubricant prior to insertion. The patient's cuff should be deflated, and the indwelling tube should be removed in one fluid movement, following the arch of the tube. *With the obturator inserted,* the new tube should be inserted smoothly, with the same sweeping motion. The operator's wrist motion should simulate the curve of the tube as it advances (Fig. 7-4). Tracheal hooks or a dilator may help to hold the stoma open. The obturator is immediately removed and the inner cannula placed inside the tube (if desired). No attempt to force placement should be made, and the clinician should stop insertion if resistance is met. Creation of a false passage into the soft tissue planes of the neck or posterior trachea is possible with potentially devastating consequences.

TABLE 7-2 -- Bedside Equipment for Tracheostomy Tube Changes

Tracheostomy Tube
Size matched to patient's indwelling tube
1–2 sizes smaller
Obturator in place
Oxygen source
Suction catheters
Water-soluble lubricant
Ambu bag
10-mL syringe (to deflate and reinflate cuff, if present)
Visualization of tracheal lumen
Tracheal hook
Light source (nasopharyngoscope)
Pediatric laryngoscope
Guide for tube placement
Red rubber catheter
Fiberoptic laryngoscope
Securing tube
Cloth umbilical tape
Precut tube gauze
Intubation supplies for emergent orotracheal intubation

A modified Seldinger technique can aid in tube placement. A small-diameter (14 French [Fr] or smaller) red rubber catheter may be threaded through the indwelling tracheostomy tube, and held in place while the original tube is removed. [26] A new tube, without an obturator, may then be threaded over the catheter until seated in the trachea. Weinmann and Bander reported a modified Seldinger technique using an airway exchange catheter that allows jet or bag ventilation into the trachea during tube exchange.[22] This adjunct provides intratracheal oxygen delivery if hypoxemia is a threat.

If a tube is being replaced after accidental dislodgment, visualization of the stoma may be more difficult. Insertion of a pediatric laryngoscope blade with light source may assist in exploration of the wound and airway visualization. [12]

Following tube insertion, the obturator is immediately removed, and, if appropriate, the inner cannula is inserted. Note that the obturator occludes the tube's lumen, and it should be immediately removed. The patient can, however, breathe normally with only the outer cannula in place. Tracheal tube ties should be applied with the patient's neck in slight flexion. The tethers should be tightened enough so that only one finger can fit between the patient's neck and the tube tie.

Confirmation of correct tube exchange is obtained by patient reassessment, and auscultation of adequate inspiratory and expiratory breath sounds. The clinician should be able to feel, as well as hear, breaths out of the tube when it is correctly placed. [12] Visual confirmation of placement can be done by inserting a nasopharyngoscope through the tube. Visualization of tracheal rings verifies intraluminal tracheal position.

TRACHEAL SUCTIONING

Tracheal suctioning removes secretions or aspirated material from the upper airway in patients whose cough is impaired, or in whom an artificial airway is in place. Tracheal suctioning can be performed through an orotracheal or tracheostomy tube, through a minitracheostomy in the cricothyroid

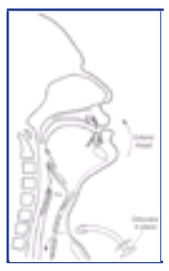

Figure 7-4 The tracheostomy tube should be inserted with a gentle rocking motion of the wrist. Insertion should follow the curve of the tube, as diagrammed above. If any resistance is met during insertion, the clinician should discontinue insertion and identify the obstruction. A pediatric laryngoscope or other light source may be helpful in visualizing the tracheal lumen prior to insertion. *(Adapted from Hackeling et al: Emergency care of patients with tracheostomies: A 7-year review. Am J Emerg Med 16:681, 1998.)*

membrane, or through the nasopharynx. Indications, basic procedures, and complications are similar with each technique.

Indications and Contraindications

The accumulation of secretions or aspirated material in the trachea impairs gas exchange and promotes atelectasis. Patients prone to increased volume of secretions are those with obstructive pulmonary diseases, bacterial infections, and pulmonary edema. Patients with a decreased level of consciousness, respiratory muscle weakness, pain, or an artificial airway often have cough reflexes that are ineffective in clearing airway secretions. [27]

The primary indication for tracheal suctioning is to remove secretions in order to enhance oxygenation, or to obtain samples of lower respiratory tract secretions for diagnostic tests.[11] In the ED, tracheal suctioning is usually performed following endotracheal intubation to clear the airway of aspirated material or secretions.

Bronchopulmonary toilet is indicated when secretions are visible in the endotracheal tube or at the orifice of the tracheostomy tube. The presence of coarse rales, rhonchi or tubular breath sounds, acute or worsening dyspnea, or arterial oxygen desaturation may indicate the need for tracheal suctioning. [11] Patients should be suctioned when clinical

symptoms warrant rather than routinely according to a predetermined schedule. [27] [28] [29]

Relative contraindications to tracheal suctioning include severe bronchospasm, which may worsen with suctioning, and persistently raised intracranial pressure, which is exacerbated by suctioning.[28] Bronchodilators, sedatives, or paralytics may alleviate the symptoms mentioned previously. Tracheal suctioning should be undertaken with caution in patients with cardiovascular instability, due to the risk of inducing dysrhythmias. However, when oxygenation is significantly impaired, expeditious suctioning should be done using techniques that minimize these potential complications. [11]

Equipment

Suction equipment should be assembled at the patient's bedside, and the patient should be placed on a cardiac monitor and pulse oximeter. Standard resuscitation medications and equipment should be immediately available.

Suction catheters are available in different lengths, diameters, and configurations of distal ports. The diameter of the catheter should not exceed one-half of the internal diameter of the tracheal tube. An approximate Fr gauge size is usually twice the tracheal tube size number; for example, a number 12 Fr catheter would be appropriate for suctioning a size 6 tracheostomy tube. A catheter that is too small will not adequately remove secretions, and one that is too large will obstruct airflow around the catheter during insertion. Evacuation of airway gases by an oversized catheter may result in alveolar collapse and hypoxemia.

Catheter tips are designed to maximize removal of secretions without causing mucosal invagination and trauma. Tips may have single or multiple side ports, usually proximal to the distal tip. Coude tips are available that allow selective suctioning of mainstem bronchi. Most authors agree that mucosal injury from suctioning is not related to the tip design, but to vigorous or extensive suctioning at high vacuum pressures. [8] [28]

A closed system airway encases a suction catheter in a sterile sheath attached to ventilator tubing, which allows tracheal suctioning to be performed without interrupting ventilatory support. In high-risk patients, including those requiring PEEP to maintain oxygenation, continued ventilatory support may reduce the risk of suction-induced hypoxia and dysrhythmias.[28] Closed system catheters are reusable, and changed approximately every 24 hours. They have not been shown to increase the incidence of nosocomial pneumonia.[28]

Suction tubing and vacuum settings should be checked before the procedure. Optimal vacuum settings should provide effective removal of secretions while minimizing mucosal trauma. Current recommendations are 60 to 80 mm Hg for infants, 80 to 120 mm Hg for children, and 120 to 150 mm Hg for adults.[28] [30] To assess suction pressures, the clinician can obstruct the catheter by kinking the distal segment and simultaneously occluding the side thumb port while the catheter is attached to wall suction. Catheter patency can be checked by suctioning sterile saline through the catheter prior to tracheal suctioning.

Additional equipment helpful for nasotracheal suctioning includes nasal trumpets, water soluble lubricant, and topical anesthetic agents. Sterile sputum traps can be used if sputum samples will be sent for microbiology or cytology studies.

Procedure

Sterile technique should be used throughout all suctioning procedures. Equipment should be inspected prior to use (see earlier). Unless contraindicated, elevate the head of the patient's bed to maximize diaphragmatic excursion.

All patients should be preoxygenated prior to suctioning. For nonventilator-dependent patients, application of humidified air through a face mask placed over the tracheostomy is recommended.[9] The rate of oxygen flow should be 10 to 15 L/min through the face mask. Patients suctioned through a minitracheostomy should have the face mask placed over the nose and mouth, as should patients suctioned nasotracheally. The face mask can be moved to the mouth only for nasal insertion of the catheter. The patient should take 5 to 10 deep breaths of 100% oxygen before suctioning, and between aspirations. [27]

Ventilated patients can be preoxygenated by increasing FiO_2 (to 1.0), tidal volume and respiratory rate, or some combination of these. [8] [31] Most ventilators require a 1 to 2 minute "washout" period before new FiO_2 levels are reached, so it may be necessary to bag the patient just before suctioning. Four to 10 breaths should be delivered before each suctioning pass, and following completion of the procedure.

Hyperinflation and hyperventilation carry the risk of over-distention lung injury. The clinician should anticipate possible complications, such as pnuemothorax during

preoxygenation.

Catheters should be inserted into the trachea smoothly, without applying suction. [30] The catheter is advanced until resistance is met, usually at the carina, and withdrawn 1 to 2 centimeters. Irritation of the carina typically elicits a vigorous cough reflex, which should be allowed to subside before proceeding. Suction is applied as the catheter is withdrawn, and should be limited to 10 to 15 seconds' duration. Some authors recommend intermittent application in place of continuous suctioning.[30] Gentle rotation of the catheter as it is withdrawn may facilitate removal of secretions. The catheter can be flushed with sterile saline between passes, and the patient should be oxygenated between suctioning attempts. No more than three suction passes should be performed in succession.

The common practice of instilling saline into the tracheal tube to elicit a cough or loosen secretions is no longer supported for hospitalized patients. [29] [30] The viscosity of airway secretions does not change with saline instillation. [13] Systemic hydration and application of humidified air will decrease mucus viscosity more effectively. In addition, instillation of saline can have a detrimental effect on oxygenation, which may last up to 5 minutes postprocedure. [14] Theoretically, the cough stimulated by saline instillation may drive secretions or microorganisms into the lower airway. [13] However, one survey demonstrated that 70% of respiratory therapists and 20% of nurses continue to routinely instill saline prior to suctioning. [21]

The recommendations presented earlier also do not address the ED patient with a critical airway obstruction due to inspissated secretions. Of note, Hudak and colleagues studied oxygen saturation and amount of secretions suctioned, with and without saline instillation prior to suctioning. The mean weight of secretions removed increased after instillation of saline, but it is not clear whether mucus or recovered saline accounted for the increased weight. They reported no instances of oxygen desaturation as a result of saline instillation prior to suctioning. [32] The usual instillation of 5 to 10 mL of saline is consistent with volumes infused during

endotracheal drug administration, which are generally well tolerated. Nonetheless, when saline instillation is used for unusually thick secretions in a patient with respiratory symptoms, supplemental oxygen is advised.

Clotted blood or other thick secretions that are not removed by suctioning may be retrieved by extraction with a No. 6 Fr Foley catheter. The catheter is passed beyond the occluding material and inflated. The material is pulled ahead of the catheter during its withdrawal. [11]

Patients should be monitored throughout the procedure for signs of cardiac dysrhythmias or hypoxia, and the procedure should be stopped if either is evident (see *Complications*, later). Causes of hemodynamic instability should be addressed before continuing with suctioning. However, if marked respiratory distress is presumed secondary to significant tracheal obstruction, expeditious suctioning should be done to reduce the obstruction.

Nasotracheal Suctioning Techniques

Nasotracheal suctioning has been used to avoid intubation intended solely for removal of secretions. [33] It should be instituted if audible evidence of secretions in the central airway persist despite a patient's best cough effort. Infants with thick nasopharyngeal secretions may also benefit from tracheal suctioning if deep nasal suctioning does not clear the upper airway. Suspicion or evidence of epiglottis or croup are absolute contraindications to the procedure; occluded nasal passages, nasal bleeding, laryngospasm, and coagulopathy are relative contraindications. [33]

A nasopharyngeal airway ("trumpet") is placed in the adult patient's nare to decrease nasal mucosa irritation and to help direct the suction catheter toward the airway. Topical anesthetic jelly can be applied to the nasal mucosa for patient comfort. The patient's reflex to swallow the catheter may be reduced by asking the patient to stick his or her tongue out or manually applying traction to the tongue of a patient unable to voluntarily comply. The catheter is advanced through the nare before attaching it to wall suction. When the catheter is properly positioned over the airway, the clinician should be able to hear air movement through the proximal catheter. The catheter is advanced into the trachea until resistance is met, and withdrawn 1 to 2 centimeters. Endotracheal suctioning is completed as described earlier. [33]

Minitracheostomy Suctioning Procedures

The minitracheostomy ("minitrach") was designed to improve tracheal hygiene in patients with intact cough reflexes, normal ventilatory function, and vocalization. The minitrach serves as a small port solely for suctioning secretions. Commonly, a 4 mm indwelling, cuffless cannula is inserted through the cricothyroid membrane into the trachea. Patients suctioned through a minitrach are at lower risk for the gagging and aspiration associated with blind endotracheal suctioning because they are able to maintain laryngeal and glottic function. [27] Because of small airway diameters, the minitrach is rarely used in children.

The technique used to suction through a minitrach is the same as that for tracheostomy suctioning. The smaller port size may require that smaller catheters be used. Most patients with minitrachs are decannulated before discharge from the intensive care unit, and uncommonly present to the ED.

Complications of Suctioning

Complications that occur during or after suctioning are relatively common, and can result in significant morbidity. Fortunately, most complications can be anticipated, and simple maneuvers can reduce their incidence and severity.

Hypoxemia from suctioning may be transient, but has the potential to cause increased intracranial pressure (ICP), dysrhythmias, or death. Cerebral hypoxia during suctioning may contribute to the genesis of intracerebral hemorrhage in neonates. [34] A number of factors contribute to suctioning-related hypoxia, including interruption of mechanical ventilation, aspiration of air from the respiratory tract, and suctioning-related atelectasis. In-line suction catheters can be used with ventilator-dependent patients, to allow continuous oxygen delivery and positive pressure ventilation during suctioning. Catheters should be sized appropriately (see procedure described earlier) to reduce the evacuation of airway gases during suctioning, and help prevent atelectasis. Suctioning passes should be limited to 10 to 15 seconds' duration and to only three suction passes in succession. The patient should also be reoxygenated between suctioning passes and after the procedure. Reducing or discontinuing routine saline instillation into the trachea before routine suctioning is also recommended. [14]

Measurements of arterial oxygen saturation may not be sufficient to assess hypoxia following suctioning. It has been shown that oxygen consumption increases during suctioning despite insignificant changes in oxygen saturation. This increase in oxygen consumption was more marked in patients who displayed a vigorous cough, agitation, or resistance to suctioning. [35]

Dysrhythmias due to suctioning may be caused by hypoxia, increased myocardial oxygen consumption, vagal stimulation, or catecholamine release. Vagal stimulation caused by suctioning or rough movement of the endotracheal tube can cause bradycardia and possible hypotension. Bradycardia in the setting of hypoxia potentiates ventricular dysrhythmias, including ventricular fibrillation. Nebulized or intravenous atropine is recommended for bradycardias, and can be used as pretreatment in patients at risk for bradycardia, including infants. Digoxin enhances vagal activity and may potentiate the vagal stimulation of endotracheal suctioning. [28] Sympathetic stimulation may occur as a result of hypoxia, pain, or stress of the procedure. Pain medication, anxiolytics, or preparation of the patient for the procedure may blunt the sympathetic response. Suctioning should be terminated immediately if any new dysrhythmia is evident.

Increase in ICP during suctioning is well documented. [36] [37] [38] The theoretical risks of increased ICP include decreased cerebral perfusion pressure in patients with compromised cerebral blood flow, inducing cerebral hypoxia, worsening cerebral edema, and possibly contributing to intraventricular hemorrhage in neonates. [31] [34] [35] There is no common agreement on the underlying cause of increased ICP during suctioning. [39] Mean ICP increases occur in a stepwise fashion with repeated suctioning; this effect is reversed if manual hyperventilation is extended to 60 seconds between suctioning passes. [39] Kerr suggested that decreases in Pa_{CO2} can minimize suctioning-induced ICP elevation, and advocated increasing the rate of manual hyperventilation from 12 breaths/min to 30/min. This study demonstrated a transient decrease in Pa_{CO2} with this rate, with a corresponding

decrease in ICP during suctioning.[31] Long-term reduction in Pa_{CO2} is not advocated. Mean arterial blood pressure also increases with suctioning, but this may be protective of cerebral oxygenation by increasing the cerebral perfusion pressure. Cerebral vasodilation after preoxygenation has been suggested as a mechanism that maintains cerebral oxygenation, even when transient increases in ICP occur during suctioning. [37] Vigorous coughing and increases in intrathoracic pressure are possible causes of increase in ICP. Instillation of lidocaine into the trachea can help suppress coughing. Pretreatment with fentanyl, or thiopental has not shown a significant effect in blunting the increase in ICP during suctioning. However, intravenous lidocaine (1.5 mg/kg), intratracheal lidocaine (2 mL of 4% solution) or temporary paralysis may attenuate ICP increases in patients with severe head injury. [11] In patients without evidence of increased ICP, routine pretreatment with drugs to prevent increased ICP is probably unnecessary. [11] Preoxygenation and hyperventilation are simple maneuvers that should be carried out in all patients.

Atelectasis can occur when airway gases are suctioned rapidly. This complication is reduced by choosing a suction catheter that is less than one-half the diameter of the artificial airway, and by controlling the time and suction pressure applied during suctioning (see earlier, *Procedure*). Postprocedure hyperventilation can treat suction-related atelectasis. [11]

Mucosal injury is a common complication of tracheal suctioning. Invagination of the mucosa into the side ports of the catheter occurs during suctioning, causing tracheal mucosa to become denuded, edematous, and more likely to bleed. Mucosal damage also interferes with mucociliary transport. Tracheitis, indicated by blood-streaked secretions or a persistent hacking cough, can occur as the result of frequent or improperly performed suctioning. [11] To reduce tracheitis, 1 mL of 1% lidocaine may be instilled into the trachea.

Suctioning technique appears to be more important than catheter tip design in reducing mucosal injury. Suctioning should be performed only when clinically indicated to reduce repeated airway trauma. The length of time suctioning is performed should be limited to 10 to 15 seconds, and appropriate vacuum pressures used. Bronchoconstriction may be triggered by suctioning. Cessation of the procedure, application of positive pressure ventilation, or use of bronchodilators should help resolve this complication.[11]

Sudden death has been reported during tracheal suctioning, and is likely due to one or more of the complications discussed here. Cardiopulmonary resuscitation equipment and medications should be available to the clinician before the procedure is initiated. [11]

Transesophageal Puncture for Voice Restoration

Transesophageal puncture (TEP) has evolved into the most widely used and accepted technique for voice rehabilitation. Originally developed in the 1980s, it can be done as a primary or secondary procedure, following laryngectomy or pharyngeal surgeries.

A puncture site is instrumented through the anterior esophagus and posterior tracheal wall, and the TEP prosthesis is inserted after dilation of the wound. Production of speech with TEP is similar to that of esophageal speech. The mucosa vibrate in segments of the pharyngeal esophagus resulting from air flow.

TEP Complications

Operative and immediate postoperative complications of TEP are infrequent. [40] Long-term complications include stomal stenosis, aspiration of the prosthesis, fistula leakage, TEP necrosis, and swallowing impairment. Reported infectious complications associated with TEP include deep neck abscess, aspiration pneumonia, and cervical cellulitis. [41]

While few TEP complications are life-threatening, the practitioner should be aware of several considerations with this device. Thick or inspissated secretions, especially after radiation therapy, or food may accumulate above the TEP, causing airflow obstruction in the upper airway. Acute changes in voice production or decreased ability to speak warrant consideration of prosthesis dislodgment, occlusion, or erosion due to infectious etiologies, especially fungal. [42] Esophageal edema causing dysphagia and loss of TEP speech has been reported [42] and should be differentiated from other etiologies of esophageal obstruction. The clinician should ascertain the presence of TEP by history or medical records, assess airway and esophageal patency, and determine if the patient has experienced changes or difficulties in voice production. Infectious complications should be sought and treated in the ED, maintaining a high degree of suspicion for fungal etiologies. In stable patients, management of prosthesis complications most often can be referred to the specialist, generally an otolaryngologist. Communication with the specialist should include consideration of prosthesis leak, dislodgment, obstruction, or infections.

Transtracheal Oxygen Delivery Systems

Low-flow oxygen administration is prescribed for patients who have adequate ventilatory function, but suffer from chronic hypoxia. Patients with chronic obstructive pulmonary disease, pulmonary fibrosis, sleep apnea, [43] lung cancer, and alpha-1 antitrypsin deficiency are often candidates for outpatient use of supplemental oxygen.[44] [45] Traditionally, supplemental oxygen has been delivered by nasal cannula. Although technically simple, nasal cannula use has numerous side effects including nasal mucosa drying and epistaxis, ear discomfort, and contact dermatitis from oxygen tubing, [44] and dry throat.[45] Delivery of oxygen via nasal cannulae is also very inefficient because it occurs only during inspiration, and oxygen must traverse the anatomical dead space of the nares and hypopharynx.

Transtracheal oxygen (TTO) delivery systems can be used in place of nasal cannula to enhance the efficiency of oxygenation, reduce complications, and improve patient comfort and compliance.[46] Low-flow oxygen (2 to 10 L/min) is delivered directly into the trachea through a narrow (7 to 11 Fr) catheter. Typically, the catheter sits in the trachea with its tip 1 to 2 cm above the carina.[44] The catheter can have single or multiple distal ports for oxygen flow.

TTO is administered throughout all phases of the respiratory cycle [43] and directly into the trachea, bypassing the upper airway dead spaces. For these reasons the required oxygen flow rates are decreased, often by 50% or more.[46] Gas mixture in the distal trachea is more effective in eliminating carbon dioxide. Clinically, these systems reduce the work of breathing and exertional dyspnea.[44] The catheter is held in place by a thin band or necklace through two openings in the flange. The catheter stoma is generally created at the base of the neck, and can be hidden under clothing.

The surgical procedure is often done as an outpatient, under local anesthesia. Initially, a small stent is placed percutaneously into the anterior neck, and replaced with a TTO catheter when the tract matures, usually in 1 to 2 weeks.[44] Once the tracheocutaneous fistula has epithelialized, the catheter may be inserted and changed safely by the patient at home. Early catheter changes may be done in the clinician's office over a guidewire, if the integrity of the stoma is questionable.

Early complications (within 3 months of the procedure) occur in approximately 30% of patients [44] [45] and include bleeding at the stoma, infection, pneumothorax, costochondritis,[47] ejection of the catheter from coughing,[48] and inability to replace the catheter after dislodgment. [45] Pneumomediastinum and sudden death were reported as possible early complications. [49] Late complications include mucus plugging, bleeding, infections, and hemoptysis [44] [45] [48] and occur in >75% of patients.[45]

Regular maintenance includes cleaning and changing the catheter. One milliliter of sterile saline is inserted into the catheter, and a cleaning rod is inserted as far as possible. The cleaning rod is inserted and removed three times to remove secretions from the catheter lumen. Catheters are changed according to manufacturer's recommendations, from twice daily to once every 2 weeks, or as needed. The stoma should be cleaned twice daily, and inspected for signs of infection. All catheter maintenance procedures should be done while the patient is given supplemental oxygen by nasal cannula.

Dyspnea or increased cough may indicate catheter obstruction by mucus or from kinking, or that the catheter tip is located cephalad to the stoma. A whistling sound from the oxygen tank humidifier may indicate obstruction within the catheter or oxygen tubing. Cellulitis around the stoma, subcutaneous air, or dislodgment of the catheter should be evident on patient examination.

In the ED, the catheter can be cleaned or replaced if obstruction is suspected. A catheter change should be done by modified Seldinger technique if the stoma tract has not healed or appears infected. Water-soluble lubricant should be used for catheter changes. If changing the catheter does not relieve obstruction, and the patient's airway is intact, increasing the intrathoracic pressure may help increase the force of the cough. The patient may sit upright, holding a pillow to his or her abdomen, and coughing forcefully after three deep inspirations. This maneuver may help mobilize secretions or small mucus plugs in the airway. Dyspnea and cough

should lessen with effective removal of obstructions.

Bleeding complications at the catheter site can be managed with gauze packing or cauterization, if minor. If significant bleeding is identified or suspected, emergency consultation by a specialist should be arranged, and definitive airway management applied as clinically indicated. Management of skin and pulmonary infections is similar to that discussed for tracheostomy care.

TRANSTRACHEAL NEEDLE ASPIRATION

Transtracheal needle aspiration is an infrequently performed procedure used to obtain tracheal sputum samples for microbiology. It offers the advantage of providing access to lower respiratory secretions in patients for whom accurate and timely identification of infectious agents is desired. Cultures of lower respiratory tract secretions obtained by transtracheal needle aspiration are more predictive of pulmonary infection than are those obtained from expectorated washed sputum. [11] Transtracheal aspiration specimens have been shown to be especially useful in diagnosis of unusual pulmonary infections, including those caused by anaerobic bacteria, tuberculosis bacilli, *Aspergillus, Pneumocystis carinii*, and hospital-acquired and partially treated pneumonias. [11]

Indications and Contraindications

The primary clinical indication to perform transtracheal needle aspiration is to obtain sputum samples from patients unable to generate an adequate expectorated sample. This population may include obtunded patients or those with underlying pulmonary or neurologic illnesses that limit ability to cough. Several contraindications to the procedure exist. These include the presence of bleeding diatheses, distorted anterior neck landmarks, hypoxia, and the patient's inability to remain supine and cooperate with the procedure. Aspiration of sputum from patients with chronic bronchitis may yield false-positive cultures. [11]

Equipment

Commercially available intravenous catheter sets that contain an introducer needle, a J-wire guide, and tracheal catheter are available. The catheters are generally 15 to 20 cm in length, and 16- to 18-ga in diameter. An additional 10 to 50 mL syringe is needed to collect the sputum sample. Previously discussed recommendations to limit the use of saline for sputum collection also may apply to this procedure. However, the literature does not provide data or specific recommendations regarding saline instillation for this procedure.

Procedure (Fig. 7-5)

The patient should be placed on a cardiac monitor and given supplemental oxygen. Cardiac resuscitation equipment should be readily available. Sterile procedure, including skin preparation and draping, should be used. The patient should be supine, with a small towel or pillow between the shoulder blades, to allow full extension of the neck. The cricothyroid membrane should be identified by palpation, and a small weal of local anesthetic injected into the skin above this landmark.

The introducer needle is inserted into the trachea through the cricothyroid membrane, with the needle tip directed inferiorly. Aspiration of air through the needle confirms intratracheal placement. The guidewire is subsequently threaded

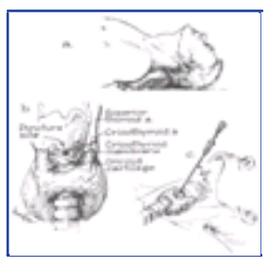

Figure 7-5 Transtracheal aspiration. *A*, Position of patient. *B*, Anatomic landmarks. *C*, Technique of puncture. The Intracath needle or J-wire introducer needle is inserted just above the cricoid cartilage through the cricothyroid membrane with its bevel up at a 45° angle to the skin. *(From Eknoyen G: Medical Procedures Manual. Chicago, Year Book Medical Publishers, 1981. Reproduced by permission.)*

through the needle, and the needle removed. The catheter can then be advanced over the guidewire. A sterile syringe is applied to the catheter, and sputum is aspirated from the tracheal lumen with gentle suction. A small aspirate amount is suitable for analysis. After sputum has been obtained, the catheter is removed, and direct pressure is applied to the puncture site. The patient should avoid therapies that stimulate coughing for 24 hours after the procedures. [11] Antitussive medications may be helpful.

Complications

Complication rates for transtracheal needle aspiration are low, and can be managed by similar techniques described elsewhere in this chapter. Cardiac dysrhythmias may be induced by vagal stimulation or hypoxia. Preoxygenating the patient should ameliorate hypoxia, and atropine may be required for bradydysrhythmias. Some patients develop subcutaneaous emphysema, which is usually self-limiting and can be minimized by reducing the amount and force of the patient's cough. Digital pressure will control most bleeding that develops. [11]

CONCLUSION

In the infrequent instances in which tracheobronchial secretions are not obtainable from expectorated sputum and when accurate bacteriologic diagnosis is essential to care of the patient with a pulmonary infection, transtracheal needle aspiration is a relatively safe, practical method for obtaining a sample. The procedure has low morbidity, high yield, and high diagnostic accuracy. [11]

SUMMARY

Knowledge of tracheal procedures, especially those related to tracheostomy care, is essential for the emergency clinician. Management of tracheostomy related hemorrhage, infection, and airway obstruction are important emergency skills. Awareness of newer tracheal and esophageal voice devices and their potential complications will also help guide the care of patients with these devices. Although transtracheal suction is rarely used today, it provides an important diagnostic alternative for patients with unusual pulmonary infections who do not require intubation.

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Chapter 8 - Mechanical Ventilation

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Initiating mechanical ventilation (MV) in the emergency department (ED) is an integral part of emergency medicine practice. However, increasing evidence holds that the manner by which human lungs are mechanically ventilated may be as deleterious as helpful. [1] The traditional view of MV as little more than a formulaic prescription that will fit virtually all patients equally well is to be discarded as a gross misunderstanding of pulmonary pathophysiology at best. Every ED clinician should embrace the firmly established paradigm of pulmonary protective ventilation and oxygenation strategies as a cornerstone of care. This chapter will address indications for MV, new strategies for safe ventilation and oxygenation, survival advantages that accrue from such interventions, complications of positive-pressure ventilation, and advanced modes of MV. One key to providing excellent ED care is to recognize when airway control and MV are warranted.

INDICATIONS FOR MECHANICAL VENTILATION

There are wide-ranging reasons for patients to require MV in the ED, and there are no absolute contraindications. Many time-honored indications for invasive ventilation are now identified as appropriate indications for noninvasive ventilation and will be addressed later. Current indications for endotracheal intubation may be separated into several categories based on the urgency of the airway need: (Emergent, Urgent, Delayed, and Elective). Emergent intubation is for patients who require immediate airway protection and MV on arrival to the ED. These patients tend to be *in extremis*, either suffering from shock with decreased oxygen delivery (DO_2) or having an absent or unprotected airway. These problems may cause hypoxemia or hypercarbia with acidosis, or both. Urgent intubation is for patients who require assistance within the first few minutes of arrival. It is used in patients who have impending airway loss. Patients who have increased work of breathing with worsening hypoxia and rising CO_2, or those with emerging injury complexes that may compromise the airway or thoracic cage may also require urgent intubation.

Delayed intubation is for patients who are stable for the initial and secondary assessment, but then require mechanical assistance. This scenario may arise for numerous reasons, including progression of the disease process despite therapy or inadequate improvement with treatment. Transport to a noncritical care area with a patient who has potential for airway loss is another reason for delayed intubation to occur. [2] More ED clinicians are facing the challenge of patients requiring delayed intubation because of increasing demands on the health care system. Patients are spending more time in the ED before admission due to the shortage of critical care beds and appropriately trained nurses. [3] This health care crisis allows the ED doctor to see these patients when they decompensate due to disease progression or treatment failure (when they normally would have already been in the intensive care unit [ICU]).

Elective intubation is for patients who will require airway protection and MV to facilitate care of organ systems besides the respiratory tract. A common example is for airway control for an invasive procedure. This last category is not common in the ED. If a patient needs to leave the department for a test or for transfer to another institution and there is potential for airway compromise during the transfer/procedure, then elective intubation is indicated. Emergency clinicians are acute care airway specialists and it is their responsibility to ensure patient safety.

In order to initiate and sustain appropriate MV, it is important that the ED clinician understand basic principles, standard equipment, goals, settings, and monitoring strategies for patients on MV.

BASIC PULMONARY PHYSIOLOGY

Minute Volume and Alveolar Ventilation

The volume of air that moves in and out of a patient's lungs per minute is termed the minute volume ($?V_E$). $?V_E$ is the product of tidal volume (V_T) and respiratory frequency or rate (f):

$?V_E = V_T \times f$

Tidal volume can be further broken down into alveolar volume (V_A) and dead space volume (V_D):

$V_T = V_A + V_D$

In healthy young persons, the anatomic dead space is accounted for by the trachea and the larger airways and is approximately 2.2 mL/kg of lean body weight. In disease states, in addition to the anatomic dead space, there is also a variable amount of "pathologic" dead space corresponding to ventilated alveoli and respiratory bronchioles that are not adequately perfused. The sum of the anatomic and pathologic dead spaces is often referred to as the physiologic dead space.

Alveolar minute ventilation ($?V_A$) is the product of rate times V_T minus dead space:

$?V_A = (V_T - V_D) \times f$

$?V_A$ and the rate of CO_2 production by the body ($?V_{CO_2}$) determine the partial pressure of CO_2 in the alveoli ($PACO_2$), which is approximately equal to the systemic arterial CO_2 tension ($PaCO_2$). This relationship is as follows:

$PaCO_2 ? PACO_2 = k \times (?V_{CO_2} / ?V_A)$

The value of the constant (k) is 0.863 when the partial pressure of CO_2 is measured in millimeters of mercury at 37°C saturated with water vapor. $?V_{CO_2}$ is measured in milliliters per minute, and $?V_A$ is measured in liters per minute. Understanding basic pulmonary physiology is essential to understanding how to initiate MV; it ensures that the method of gas delivery meshes with the patient's underlying physiology to avoid ventilator-induced lung injury. Clinicians must also understand the basics of ventilator operation and order writing.

EQUIPMENT—STANDARD OPTIONS

Regardless of which ventilator one uses, a limited number of standard features are common to each of them. This discussion explores machine features and settings.

Control Mechanisms

Volume-Cycled.

With this target, the ventilator seeks to deliver a preset amount of gas. The time of gas flow is determined by the set volume (V_T), flow (?Q), and the waveform of gas delivery (see later). When the set V_T is reached, gas flow is terminated and expiration passively begins. An advantage is that this method delivers a constant V_T. Unfortunately, it does not take into account dynamic changes in lung compliance, which may alter the ability of the lung to accept delivered gas in gas-exchanging alveoli. This is the most common mode used in the United States.

Pressure-Cycled.

With this target, the ventilator alters gas flow to achieve and maintain airway pressure at a preset level for the duration of a preset inspiratory time (T_i). Gas flow is terminated when the preset change in pressure is achieved. The pressure is maintained with a variable or intermittent ?Q for the set T_i. Gas flow is adjusted to not exceed a set pressure limit (i.e., pressure-limited or pressure-controlled ventilation). One problem with this method is that the volume received by the patient is variable. The delivered volume is determined by the patient's lung and chest wall compliance, the airway resistance, as well as the T_i and pressure target. An advantage is that airway pressures are tightly managed to limit or eliminate alveolar overdistention and to reduce ventilator-induced lung injury. [5] In the setting of hypoxemia, the inspiratory time may be increased quite precisely to increase the mean airway pressure and thus oxygenation; this strategy is much more difficult, if not impossible, to manipulate using volume-cycled ventilation.

Modes

Controlled Mechanical Ventilation/Assist Control (CMV/AC).

This ventilator mode provides breaths, known as machine breaths, at a preset rate. If the patient tries to breathe faster than the set rate, he or she can initiate additional breaths known as spontaneous breaths. In volume-cycled ventilation (VCV), the spontaneous breath receives the same V_T that is set for the machine breath, regardless of how much gas the patient wants to receive. For the patient to trigger the ventilator to initiate flow for a spontaneous breath, the airway pressure must decrease by a preset amount below PEEP. The amount of decrease necessary to open the inflow valve is the sensitivity setting. This is typically 0.5 to 2 cm H_2O pressure. The higher the sensitivity, the greater the work of breathing required to trigger a breath.

Intermittent Mechanical Ventilation (IMV)/Synchronized IMV (SIMV).

This ventilator mode provides breaths at a preset rate (machine breath) similar to the AC mode. The patient can initiate an additional spontaneous breath, but only receive a spontaneous V_T that reflects the depth and time spent in inspiration. For each of these nonmandatory (i.e., spontaneous) breaths, the patient receives no support from the ventilator and has a high work of breathing. Thus, SIMV is typically partnered with Pressure Support Ventilation (PSV—see later) to aid in spontaneous breathing support and to overcome the intrinsic resistance of breathing through long tubes and the endotracheal tube with a diameter smaller than the patient's native airway. The synchronized version of IMV allows the ventilator to coordinate spontaneous and machine breaths to prevent it from delivering a scheduled breath on top of a spontaneous breath (excess V_T delivered) or during exhalation from a spontaneous breath (exhalation compromised by positive airway pressure). Both conditions were a problem with the original IMV mode. This could lead to elevated airway pressures, alveolar overdistention, and biotrauma. [4]

Each of these modes (CMV/AC and IMV/SIMV + PSV) may be combined with either a volume target or a pressure target to achieve the desired minute ventilation (see above).

Continuous Positive Airway Pressure (CPAP).

CPAP is another positive-pressure, spontaneously breathing mode. It is most commonly used in isolation for those with chronic obstructive pulmonary disease (COPD) or obstructive sleep apnea via a tight-fitting nasal or full face-mask. In patients with an endotracheal tube (ETT) or tracheostomy tube, CPAP is typically used as a weaning mode in combination with PSV. The CPAP level when transitioning from IMV/PSV or AC mode should be the PEEP level that was being used. Reducing the CPAP below the previous PEEP may result in loss of alveolar recruitment, atelectasis, hypoxia, and an increased work of breathing.

Adjunct Ventilator Settings

Oxygen.

The percentage of inspired O_2 (FIO_2) is set in a range from 21% (room air; not generally indicated) to 100%. In the ED it is common to start at 100% FIO_2 to ensure adequate oxygenation and titrate the FIO_2 down to nontoxic levels ($FIO_2 < 0.60$) following the SaO_2 via the pulse oximeter (SaO_2 90% ? pO_2 60 torr). In theory, placing the patient on 100% FIO_2 may lead to alveolar collapse due to absorption of all the oxygen in that alveolus. Recall that room air is principally N_2 mixed with O_2 and a number of inert gases. If there is only O_2 delivered to an alveolus, and that O_2 is absorbed, there will be no nonabsorbed gases to keep open the alveolus. This is known as absorption atelectasis and leads to V/Q mismatch when present. Thus, some practitioners recommend using 95% O_2 as the upper limit of FIO_2.

PEEP.

This is the pressure in the airway at the end of exhalation. PEEP helps keep the large noncartilaginously supported airways and the smaller alveoli open to prevent collapse, atelectasis, and hypoxia at the end of expiration. The required ventilation to compensate for this triad commonly worsens lung compliance and is associated with ventilator-induced lung injury. The useful PEEP range is from 3 to 20 cm H_2O.[6] In general most patients should be started on a PEEP of 5 cm H_2O. This is considered a physiologic level. PEEP can be increased by 3 to 5 cm H_2O every 10 to 15 minutes as needed/tolerated for patients who remain hypoxic. The initial goal is to reduce the FIO_2 to nontoxic levels ($FIO_2 < 60\%$). This goal is coming under increasing scrutiny as new information challenges the timeframe and concept of O_2-induced lung injury at FIO_2 levels >0.6. [7]

PEEP is used to increase functional residual capacity (FRC) and move the zero pressure point of each alveolar unit more proximal in the airway so as to prevent early alveolar collapse.[8] By so doing, PEEP increases the available number of alveolar units that can participate in gas exchange. The

primary effect of PEEP on gas exchange, however, is to improve oxygenation, not CO_2 removal. CO_2 clearance is rather efficient and will be well preserved in situations where oxygenation is not. By opening one alveolar unit, the tendency of the adjacent unit is to open as well (i.e., alveolar codependency; Fig. 8-1).[9] Excessive PEEP will compromise hemodynamics. Therefore, there are two primary questions to ask when using PEEP to augment oxygenation: What is the "optimal PEEP," and is the current amount of PEEP compromising the patient's hemodynamics?

There are several ways to determine the "optimal PEEP." One way is to increase the PEEP until there are no longer increases in the pO_2. This method, however, may result in several untoward events. First, oxygen tension may steadily increase, but pCO_2 may increase as well from alveolar overdistension. With overdistension, the alveolar pressure may exceed pulmonary arteriolar pressure and actually decrease pulmonary blood flow and CO_2 clearance. Second, alveolar overdistension may increase total intra-thoracic pressure and therefore diminish venous return and hence cardiac output. Third, decreased venous return may result in cerebral venous hypertension as indicated earlier. The "optimal PEEP" for one organ system may be deleterious for another. For example, the optimal PEEP for ideal oxygenation may be the worst PEEP for cerebral venous drainage.

A better method of determining the "optimal PEEP" relies on measuring oxygen delivery and oxygen consumption. PEEP may be increased until there is no further increase in oxygen delivery, and no increase in oxygen consumption.[10] This method does require a pulmonary artery catheter to be in place for determination of $?DO_2$ and $?VO_2$. In many institutions, the application of PEEP above 10 cm H_2O pressure in a

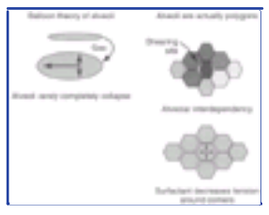

Figure 8-1 Alveolar interdependence. Note that alveoli are not round in shape; instead, they are polygons. Polygons have corners and may have two opposing surfaces that may adhere to one another via surface tension. Surfactant works to reduce this surface tension and allow alveoli to open with reduced shear stress at the junction of closed and open alveoli. Alveoli are connected via the pores of Kohn. This allows opening alveoli to pull a relatively closed alveolus open while equalizing pressure between adjacent alveoli. The central alveolus on the right is fairly closed in the upper diagram, but is pulled open by its neighbors as they expand and accept gas.

multiply injured patient necessitates a pulmonary artery catheter being placed to make such determinations regarding oxygen extraction. An alternative is to increase PEEP until a complication of PEEP occurs (e.g., pCO_2 elevation, hypotension), and then reduce PEEP if needed (inability to tolerate hypercapnia), or expand the patient's intravascular volume to combat decreased venous return. The authors favor the placement of a pulmonary artery catheter in these situations to accurately guide therapy and know the patient's $?DO_2$ and $?VO_2$ status.

Another excellent method of determining the "optimal PEEP" is guided by assessing changes in plateau pressure with changes in PEEP. As PEEP is increased from a minimal level, the patient's peak airway pressure as well as plateau pressure will increase by the amount of the applied PEEP. However, when the "optimal PEEP" for the lung units is achieved, the plateau pressure will no longer increase; in fact, as the lung is optimally recruited, the peak and plateau pressure may decrease as there is more volume of lung available to receive a set V_T. Once this level is exceeded, however, there will be further increases in plateau pressure beyond the incremental increase in PEEP as the units overdistend. Therefore, the clinician must readily identify the plateau in the plateau pressure trend. The same relationship may be displayed graphically in the dynamic pressure-volume loop (Fig. 8-2). The lower limb of the loop represents the pressure required to open the alveolar units.[11] In the absence of PEEP (or inadequate PEEP), this limb is prolonged and flattened and has an inflection point far to the right of the origin of the loop (Fig. 8-3). As PEEP is progressively increased, the inflection point travels to the left. When the "optimal PEEP" is achieved, there will be a rapid upstroke of the loop as the vast majority of the functional lung units are already open and ready to be ventilated (see Fig. 8-2). This strategy is known as the "open lung model" of MV.[11]

PEEP is not without untoward side effects, and increased levels of PEEP can lead to hemodynamic compromise.[12] This occurs from increased intrathoracic pressures leading to cardiac compression and collapse, principally of the right

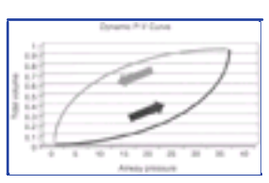

Figure 8-2 Dynamic pressure-volume loop. Note that as soon as there is delivered pressure to the airway, there is an increase in measured tidal volume. The lower arrow denotes inspiration while the upper arrow indicates exhalation. This indicates that the airways are open and do not need to be forced open by increasing the pressure in the airway. If this latter case were true, then the P-V loop would initially be flat along the X-axis.

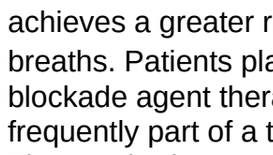

Figure 8-3 Inadequate PEEP and the P-V loop. Compare this curve to that in Figure 8-2 . Note that the loop is initially flat (lower segment) along the X-axis. Once the airway pressure is high enough to open the alveolar units, each increase in airway pressure is matched by a corresponding increase in tidal volume.

atrium. It is imperative that the patient be adequately resuscitated as volume depletion compounds this problem. Desired levels of PEEP simply may not be possible because of deleterious effects on cardiac output.

Pressure Support Ventilation (PSV).

This mode augments spontaneous ventilation in the IMV/SIMV and CPAP modes. There is gas flow during inspiration for each spontaneous breath to help the patient overcome the resistance of the circuit and to achieve an acceptable V_T. The range is from 0 up to 35 cm H_2O pressure; some ventilators may deliver PSV that achieves a greater range. The average starting point is 10 cm H_2O. PSV is adjusted as needed so that the spontaneous V_T approximates the set V_T for mandatory breaths. Patients placed on the combined mode of CPAP/PSV *must* be spontaneously breathing. PSV-based ventilation must *never* be combined with neuromuscular blockade agent therapy, or death from apnea, hypercarbia, and hypoxia will result. PSV is commonly used to aid in weaning from IMV-based ventilation and is frequently part of a transition strategy from IMV to CPAP.[13] In this way, PSV is used to eliminate the work of breathing improved by the ETT and the ventilator tubing. The required amount of pressure support to overcome the tube-induced resistance has been well documented (Table 8-1).[14] It is imperative not to lower the PSV during weaning below that required to overcome the resistance imposed by the diameter of the ETT or tracheostomy tube as the work of breathing may precipitously rise. Unlike AC or SIMV, PSV does not have a preset T_i. In fact, the time at which the gas flow terminates for each PSV breath is determined by an algorithm that in most older ventilators is not manipulable.

For instance, a patient breathing on CPAP/PSV on a Puritan-Bennett 7200 ventilator would have his or her maximal inspiratory flow required to achieve the set pressure support level measured. As the patient's airways progressively fill with gas, the patient draws in gas at a slower rate, and the machine needs less of a flow rate to maintain the airway pressure. When the machine flow rate decreases to 25% of the previously measured maximal inspiratory flow rate, all gas flow ceases. This termination occurs whether the patient is done inspiring or not. This algorithm may lead to significant patient-ventilator dysynchrony.[15] Newer ventilators, such as the Hamilton Gallileo, allow user adjustment for such dyssynchrony. A common resolution using such a PSV device is either patient sedation or an increased level of pressure support so that the patient receives his or her desired amount of gas before gas flow termination.

Inspiration:Expiration (I:E) Ratio.

The normal I:E ratio in a spontaneously breathing, nonintubated patient is 1:4.[14] Intubated patients commonly achieve I:E ratios of 1:2. Shorter ratios may lead to

decreased exhalation by compromising exhalation time (T_e). In its extreme form, inverse ratio ventilation (IRV), the normal pattern of breathing is reversed. There is a longer time spent in inhalation to allow a longer time for oxygenation. Longer inspiratory times allow for better matching of alveolar regional time constants. [16] The decrease in expiratory time can lead to air trapping, elevated airway pressures, and rising pCO_2. These problems lead to hypercapnia, respiratory acidosis, and auto-PEEP.[16] Auto-PEEP is additional pressure that is generated within the airways from trapped gas that should have been exhaled, but for various reasons (commonly obstruction to exhalation such as COPD), was not.

Auto-PEEP can cause hemodynamic instability secondarily to decreased venous return just like high levels of PEEP. [17] Auto-PEEP may be detected in two ways: (1) evaluating the flow-time trace or (2) disconnecting the patient from the ventilator and listening for additional exhaled gas after an exhalation has already occurred. [11] The flow-time trace will demonstrate that the exhalation is not yet completed before the next breath has been initiated (Fig. 8-4). Auto-PEEP is a real potential when one initiates IRV for the management of hypoxemic respiratory failure. One can initiate IRV most easily in pressure cycled ventilation (PCV) where the operator sets the T_I directly. With VC ventilation, one can achieve a similar gas delivery by adjusting the flow rate of each breath to adjust the I:E ratio; that the Q needs to be adjusted on a breath-by-breath basis makes this impractical.

?Q.

This is the rate of gas delivery (L/min). The range of flows that can be achieved by current ventilation is from 10 to 160 L/min. Common flow settings are from 40 to 75 L/min. The higher the rate, the faster the ventilator will reach its set volume or pressure. A faster rate allows for a longer exhalation time, but due to the shortened inspiratory time, hypoxia can result. A slower rate allows for longer time in inhalation and improved oxygenation but a shortened expiratory time, which may lead to retained CO_2 and auto-PEEP from inadequate T_e (Fig. 8-5).

Waveforms

Square.

Once the maximal inspiratory ?Q is achieved, the gas flow is constant until the set volume is delivered. When that point is reached, the gas flow is terminated. This waveform is best for patients with COPD and those suffering from head injury because gas delivered with this waveform allows for a longer T_e and lower mean airway pressure (P_{aw}-mean). The longer T_e is beneficial for patients with restrictive airway disease such as COPD. Spending a longer time in exhalation allows for improved venous drainage from the brain. The drawback to this waveform is an increased P_{aw}-peak, often requiring a lower V_T or ?Q. This can lead to inadequate alveolar recruitment in patients with acute lung injury.

Decelerating (Ramp).

Once the maximal inspiratory flow is reached, the rate of gas delivery immediately begins to slow in a preprogrammed fashion. Therefore, relative to the

TABLE 8-1 -- Initial Ventilator Settings

Type of Ventilation	Comments
Volume Cycled Ventilation (volume set, pressure varies)	
Mode: SIMV	If patient is paralyzed: IMV = AC ventilation
Rate: 10–12/min	Target normal $?V_E$
V_T : no acute lung injury: 10 mL/kg acute lung injury or ARDS: decrease V_T : 6–7 mL/kg	Increased survivorship in ARDS with decreased V_T ventilation
FIO_2 : 0.4–0.95	Titrate to pO_2 > 60 mm Hg
PEEP: 5–20 cm H_2O pressure	Titrate to pO_2 > 60 mm Hg and FIO_2 = 0.6
PSV: 10–35 cm H_2O pressure	Target V_T spont = V_T set
Waveform: decelerating ramp	Use square wave form in COPD only
Peak Airway Pressure: <35 cm H_2O pressure	(Noted on dial on ventilator) Peak pressure >35 cm H_2O associated with lung injury
Sigh: 0–2/min	Optional—use with decreased respiratory rate to maintain alveolar recruitment
Temperature: 35°C	Not efficacious in rewarming or cooling
Flow Rate (?Q): start at 60 L/min (range 45–75)	Titrate to P_{aw}-peak and pO_2
	Decrease ?Q for hypoxemic patients
	Increase ?Q for patients with air hunger
Pressure Cycled Ventilation (pressure set, volume varies)	
Mode: SIMV	If patient is paralyzed: IMV = AC
Rate: 10–12/min	Decrease rate to allow longer time to exhale and to increase CO_2 clearance
PC: 2/3 of prior peak airway pressure or prior plateau pressure	Titrate to peak airway pressure-V_T curve (hysteresis curve)
T_i : start at 2 sec and increase to increase mean airway pressure and pO_2	Increased T_i will lead to IRV: will need heavy sedation and/or paralysis
FIO_2 : 0.4–0.95	Titrate to pO_2 > 60 mm Hg
PEEP: 5–20 cm H_2O pressure	Titrate to pO_2 > 60 mm Hg and FIO_2 = 0.6
PSV: 10–35 cm H_2O pressure	Target V_T spont = V_T set
Waveform: decelerating ramp	Use square wave in COPD only
Temperature: 35°C	Not efficacious in rewarming or cooling
Note:	
1. Use inspiratory time cycled PVC to precisely control I:E ratio. IRV is used to manage hypoxia but may lead to hemodynamic instability owing to decreased venous return and decreased cardiac output.	
2. Best to titrate ventilator settings to the shape of the pressure-volume curve (need ventilator with a graphics package, a.k.a. "open lung model").	
Special Circumstances	
1. Severe acute lung injury—consider permissive hypercapnia. If able to achieve pO_2 > 60 mm Hg on FIO_2 = 0.6, the pCO_2 may be allowed to be >40 mm Hg if pH > 7.25. Further attempts to raise $?V_E$ to decrease pCO_2 may induce additional lung injury.	

2. Asthma—defect is decreased gas flow. In conventional ventilation use higher flow rate and lower respiratory rate to allow more time for exhalation.
3. Traumatic brain injury—Do not lower pCO_2 < 35 mm Hg, as it may induce severe cerebral vasoconstriction and lead to cerebral ischemia. The goal is normal pCO_2 : 35–40 mm Hg. Acceptable to hyperventilate for a patient with an acute herniation syndrome as a bridging maneuver for definitive therapy.
4. PEEP—used to raise alveolar recruitment and increase pO_2 in patients with hypoxemic respiratory failure. Caution: excessive PEEP can lead to hypotension from diminished venous return. Initial treatment of this hemodynamic instability is with volume replacement and lowered PEEP if possible.
AC, assist control; **ARDS,** acute respiratory distress syndrome; **FIO_2 ,** percent of inspired oxygen; **I:E,** inspiratory:expiratory; **IMV,** intermittent mechanical ventilation; **IRV,** inverse ratio ventilation; **MAP,** mean arterial pressure; **P_{aw} -peak,** peak airway pressure; **PEEP,** positive end-expiratory pressure (5 cm H_2O is considered physiologic); **PSV,** pressure support ventilation; **PC,** pressure control (setting); **?Q,** flow rate; **SIMV,** synchronized intermittent mechanical ventilation; **T_i ,** time in inspiration; **?V_E ,** minute ventilation; **V_T ,** tidal volume.

square waveform, longer time is spent in inhalation to deliver the set V_T or achieve the target pressure, which allows for improved oxygenation. This waveform also achieves a lower P_{aw} -peak and higher P_{aw} -mean.

Sine Wave.

Not useful in critically ill patients.

Accelerating.

This is the flow pattern for neonatal gas intake and is generally not used in adult ventilation unless one must use the Siemens Servo 900 ventilator, which has only two options: square or accelerating.

Sighs.

This is a large single breath or large multiple breaths, both designed to help maintain alveolar recruitment by ventilating a patient on a periodic basis at close to vital capacity. There is controversy regarding the utility of the sigh option with regard to alveolar overdistention. [18]

Pause.

This is a variable used on the Siemens Servo 900 ventilator to alter the I:E ratio. This is technically complex for anyone not working with ventilator setup on a daily basis. It is much easier to directly adjust the T_i/T_e on more modern ventilators. A pause is useful to determine the Plateau Pressure on the Puritan Bennett 7200 or the Infrasonics Adult Star ventilator (as a point measurement). If a short pause is used to measure plateau pressure, the authors suggest no

Figure 8-4 Identifying auto-PEEP with the flow-time trace. The X-axis is time while the Y-axis is flow rate in L/min. Deflections above the X-axis indicate inspiration, while deflections below represent exhalation. In this example, the flow pattern is ramp (decelerating). Note that as the respiratory rate increases (decreased time for exhalation), the flow has not yet returned to baseline (a), indicating incomplete exhalation when compared to the following breath (b).

longer than 0.5 seconds as the pause duration; remove the pause when the measurement is completed.

NEW MODES

Airway Pressure Release Ventilation (APRV)

APRV is essentially a high level CPAP mode that is terminated for a very brief period. The CPAP level may be as high as 40 or more cm H_2O pressure. The long time during which the high-level CPAP is maintained achieves oxygenation while the short release period achieves CO_2 clearance (Fig. 8-6). The long time during which the high-level CPAP is present results in substantial recruitment of alveoli of markedly different regional time constants at rather low gas flow rates and lower airway pressures (by comparison with conventional ventilation strategies). The establishment of

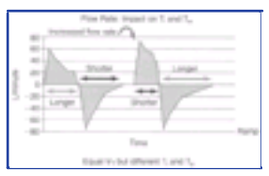

Figure 8-5 Effect of flow rate on inspiratory and expiratory times. Note that as the flow rate changes, there are corresponding alterations in the effective time for inspiration and exhalation. Deflections above the X-axis (time) indicate inspiration, while those below indicate exhalation. The delivered tidal volume for each cycle is the same, but the inspiratory and expiratory times are different.

intrinsic PEEP by the short release time enhances oxygenation. CO_2 clearance is aided by recruitment of the patient's lung at close to total lung capacity (TLC); elastic recoil creates large-volume gas flow during the release period. This is a fundamentally different mode from cyclic ventilation. This mode allows the patient to spontaneously breathe during all phases of the cycle. This mode is enabled to succeed by having a floating valve that is responsive to the patient's needs regardless of the location within the respiratory cycle. In other words, the patient is allowed to breathe in or out during the high-level CPAP phase as well as during the release phase. Accordingly, the sequence is called a phase cycle; there is no set inspiratory or expiratory time, and no readily identifiable respiratory rate in the traditional sense. During the high-CPAP phase, a patient may exhale 50 to 200 or more mL of gas as his or her lung volume becomes full of gas; this is not a full exhalation and the release of excess gas should not be counted as a breath.

Given the spontaneous nature of the mode, there should be virtually no need for continuous infusions of neuromuscular blocking agents in patients placed on this mode of ventilation[19] ; exceptions to this observation do occur for the management of ICP but not for oxygenation or clearance of CO_2 . This may result in a shorter length of ICU stay and a reduced incidence of prolonged neuromuscular blockade syndrome. Furthermore, since patients may be ventilated at lower airway pressures than using cyclic ventilation, there is a reduced need for pressor support of hemodynamics to ensure oxygen delivery. [19] Moreover, there is a reduced sedative need as patients are more comfortable on this spontaneous mode than on cyclic ventilation. [19]

Hemodynamic assessment using a pulmonary artery catheter in patients on APRV has been investigated. The pulmonary artery occlusion pressure (PaOP) must be read at the middle or end of the release phase to maintain the fidelity of the reading. Reading the PaOP at any other point in the cycle will give a significantly different value by comparison with the end expiratory reading obtained using pressure-cycled ventilation. [20] Transport of patients on APRV with a P_{aw} -high (sustained peak P_{aw}) > 20 cm H_2O pressure should be with the patient attached to the ventilator instead of being hand ventilated. [21] Hand ventilation is unable to match the manner of gas delivery and pressure dynamics that the patient requires. Attempts at hand ventilation, even with an appropriately set PEEP valve, are frequently complicated by unexpected hypoxemia and hemodynamic instability.

Proportional Assist Ventilation

Ventilators that are capable of performing in this mode will be able to assess on a breath-by-breath basis how much work of breathing support the patient needs to achieve the targets and goals that the clinician sets. [22] The unique features of this type of ventilation promise to reduce inadvertent airway injury, and, in many ways, serve as a self-weaning ventilator mode. As the patient requires less support, the ventilator delivers less support. Current data are lacking to determine if this will realize a shortened length of ventilator support for those with acute respiratory failure.

Permissive Hypercapnia

As stated, excessive P_{aw} -peak may be quite detrimental. One means of limiting P_{aw} -peak, and thereby offer protection from

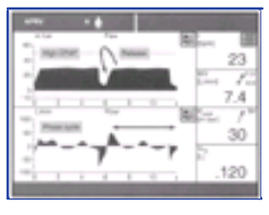

Figure 8-6 Airway Pressure Release Ventilation (APRV)—airway pressure-time and flow-time traces. Note that the peak airway pressure (P_{aw} -high) is maintained for a long period. This phase establishes oxygenation (T_{high}). There is a short period of release when most CO_2 is cleared (T_{low}). Note that the bottom trace is flow over time. The combined time for the T_{high} and T_{low} is known as a phase cycle. Note that the number of phase cycles is not the respiratory rate as patients breathe within the entirety of the T_{high} . As the release phase is initiated, the flow rate is identified as negative and is of a high rate (here approximately 7.5 L/min), consistent with significant alveolar recruitment. During the high CPAP phase, the patient is allowed to exhale (negative deflections on the flow-time trace). Thus, APRV is quite dissimilar from traditional cyclic ventilation. This unique mode is made possible by a floating valve system.

the trauma of ventilation, is to decrease the delivered V_T until an acceptable and less deleterious P_{aw} -peak is achieved (=35 cm H_2O). However, changes in P_{aw} -peak may alter the pH-pCO_2 balance. If the pH is =7.25, and the patient can tolerate the elevated pCO_2 while still remaining well oxygenated, then the ?V_E is not increased. Alternatively, the f may be decreased in similar fashion, but usually not <8 breaths per minute. This paradigm is known as permissive hypercapnia and the concept represents a major departure from previously accepted tenets of MV, which mandated that MV should always achieve a normal pCO_2 .[24] Clearly, many patients can safely tolerate pCO2 elevations that have in the past been thought to be harmful. A slower respiratory rate reduces the shear forces active across the alveolar common walls by allowing for fewer openings and closings per minute. [25] The side effect of such a rate reduction is greater time for exhalation that may lead to alveolar collapse and increased shear stress at the junction of open and closed alveoli. Nonetheless, a greater T_e helps prevent auto-PEEP, alveolar overdistention, and hemodynamic embarrassment. Likewise, a reduced V_T also helps prevent alveolar overdistention.

While these features appear very attractive, permissive hypercapnia is not entirely benign. An elevated pCO_2 triggers cerebral vascular vasodilatation, which leads to increased cerebral blood flow and possible elevated intracranial pressure (ICP). [26] Increased ICP > 20 mm Hg can be detrimental in patients suffering from head injury or cerebral ischemia.[27] In patients with acute lung injury complicating traumatic brain injury or stroke, such a management strategy is optimally accompanied by a measure of cerebral perfusion to evaluate for hyperemia, or ICP monitoring to assess for intracranial hypertension from increased CO_2 tension.[28]

Hypercapnia also shifts the oxyhemoglobin dissociation curve to the right, leading to increased early unloading of O_2 at the tissue level. Hypercapnia also creates an acidosis that may initiate myocardial depression, dysfunction of pH dependent enzyme kinetics, and distorted cellular metabolism. [29] Severe acidosis, pH <7.2, may be effectively countered by using an $NaHCO_3$ infusion. This proper intravenous infusion can be created by mixing 1 liter of D_5W and 150 mEq of sodium bicarbonate, creating a sodium content similar to that of lactated ringers solution (130.4 mEq/L). Alternatively, if 150 mL of D5W are removed before adding the 150 mL of $NaHCO_3$

, then the resultant Na^+ concentration is 150 mEq/L and approximates 0.9% normal saline solution. A common additional repair of the increased pCO_2 if the pH is <7.2 is to increase the respiratory rate to increase the $?V_E$ while maintaining the "lung protective low tidal volume." This strategy is quite similar to the ARDSNet protocol that is commonly used for volume ventilation of patients with acute lung injury or ARDS (see later). [30]

Prone Positioning

Multiple authors have proposed gravity as an effective aid in lung recruitment for those patients with severe or refractory hypoxemia. [31] [32] Several studies have identified benefits in terms of increased pO_2 following pronation. [31] [32] Several areas remain unresolved. It is unclear who will maximally benefit (up to one third have no benefit), how many proning cycles per day and for what duration are most beneficial, and for how long to continue pronation once it has been initiated. What is clear is that chest geometry is critical in successful pronation [31] (Fig. 8-7). Patients with ovoid chests have little benefit from pronation as there are relatively equal lung volumes that exchange the superior and inferior positions when the patient is placed prone. Patients whose thoracic cage is more triangular have a greater volume of lung posteriorly (while in the supine position) that is able to exchange with a smaller volume of anterior lung. This patient population benefits from pronation.

Similarly, patients with small cardiac volumes benefit more than those with large volumes as there is more lung behind the heart to recruit with the pronation maneuver (see Fig. 8-7). It is quite clear that pronation is a challenging and potentially dangerous maneuver in a patient with invasive lines and an endotracheal tube in place, although no increase in unintentional extubations was noted in a study by Gattinoni et al. [31] This risk of tube or catheter dislodgement or malposition is compounded by the altered hemodynamics in a patient who is not yet volume-replete, especially after trauma. Thus, pronation cannot be recommended as first-line therapy for

Figure 8-7 Chest geometry and prone position. Note that ovoid chest geometry results in equal volumes above and below the transverse axis leading to no change in recruited lung volume with pronation. Triangular chest geometry leads to a significant increase in lung volume with prone positioning. This is by virtue of the large volume of lung that lies posterior to the transverse plane with the patient in the supine position. Increases in cardiac volume reduce the available retrocardiac lung volume and impair the effectiveness of prone positioning with regard to pulmonary recruitment.

hypoxemia in the ED. Instead, it is best reserved for the more controlled environment found in the ICU. Moreover, it is the author's experience that pronation use has been virtually eliminated since using APRV as the rescue mode of choice for refractory hypoxemia.

VENTILATOR ORDER GOALS

Minute Ventilation ($?V_E$).

This is the amount of gas delivered to a patient over a minute. It is calculated by multiplying the patient's respiratory rate by the tidal volume ($f \times V_T$) for patients without spontaneous breaths. It is conveniently determined by the ventilator and can be read directly for those with and without a spontaneous component to their $?V_E$. The need for $?V_E$ varies with the patient's condition, body mass, co-morbidities and acid-base status. For example, a patient who has a metabolic acidosis from diabetic ketoacidosis needs an elevated $?V_E$ to decrease pCO_2 and acutely buffer the acidosis. Thus, the emergency provider should determine what $?V_E$ range the patient will need and set the f, V_T, or PCV/T_i to compensate for the increased metabolic acid load. A normal $?V_E$ is 7 to 10 L/min.

Spontaneous Minute Ventilation.

This is the $?V_E$ derived from spontaneous breathing. During weaning, progressive increases in this parameter are expected as mandatory breaths are decreased or eliminated.

Spontaneous Tidal Volume.

See SIMV and PSV earlier.

Peak Airway Pressure (P_{aw}-peak).

This is the maximum amount of reflected pressure in the patient's airway. This peak occurs during inspiration—an important concern because of the well-documented relationship of elevated airway pressure (and volume) causing biotrauma. [4] Excessive P_{aw}-peak (>35 cm H_2O) commonly leads to alveolar overdistention and injury, causing release of inflammatory mediators and complications including pneuomothoraces, pneumatoceles, pulmonary interstitial emphysema, pneumomediastinum, acute lung injury and acute respiratory distress syndrome (ARDS). The peak airway pressure and alveolar overdistension are best evaluated using the pressure-volume curve, looking to abrogate any increases in airway pressure that are not accompanied by increases in delivered volume (Fig. 8-8). Increases in airway pressure without accompanying increases in V_T lead to a plateau of the P-V curve, known as the "bird's beak" profile. This profile is a reasonable indicator of alveolar overdistension and airway injury.

Plateau Pressure.

This is the pressure reflected from the airways once the full set volume or targeted pressure change has been achieved. It is a reflection of pulmonary compliance, airway resistance and elastance. It is not directly manipulable, but may be impacted by V_T, ?Q, PCV, T_i, and PEEP. It does provide a basis for the initiation of other modes of ventilation and is quite useful in that regard (see APRV earlier).

Mean Airway Pressure (P_{aw}-mean).

The area under the pressure-over-time curve (Fig. 8-9) may be calculated and represents the mean airway pressure. The P_{aw}-mean correlates most closely with the achieved pO_2 in volume-cycled or pressure-cycled ventilation modes. The longer the T_i, the greater the P_{aw}-mean. When a patient has hypoxemia and the clinician wants to change the ventilator orders, *it is important to not reduce the P_{aw}-mean as a result of the change in therapy* since a decreased P_{aw}-mean consistently leads to a decrease in pO_2.

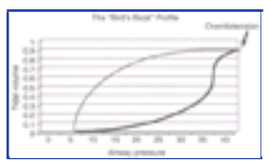

Figure 8-8 Alveolar overdistension is reflected in the increase in airway pressure without any concomitant increase in tidal volume. This pressure-volume curve pattern approximates a "bird's beak" profile.

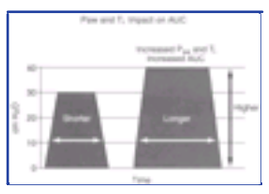

Figure 8-9 Mean airway pressure and the pressure-time trace. Note that the greater the maximum airway pressure and the longer the T_i, the greater the area under the curve (AUC) described by the positive-pressure (inspiratory) limb of the respiratory cycle. The increase in mean airway pressure (area under the curve) is the principal correlate of oxygenation in volume or pressure-cycled ventilation.

COMPLICATIONS OF MECHANICAL VENTILATION

Pneumothorax.

Pneumothorax unassociated with thrauma in a mechanically ventilated patient typically stems from alveolar overdistension (continuous or episodic) that leads to alveolar rupture and escape of gas into the pleural space. [36] Patients on positive-pressure ventilation should have their pleural space drained to prevent progression to a tension pneumothorax with hemodynamic compromise. Loculated pneumothoraces may be successfully drained percutaneously under ultrasound or CT guidance. Successful drainage of airspace disease leads to enchanced liberation from MV. [23] Pneumothorax or tension pneumothorax may also result from aggressive and incorrectly performed bag-valve-mask ventilation. Patients with intrinsic lung disease such as COPD or asthma are more prone to pneumothorax than the average patient due to the abnormal structural integrity of their alveolar air spaces. [37]

A simple pneumothorax may be drained by surgical tube thoracostomy with a small-bore tube (24 French [Fr]), a commercially available pneumothorax kit (Arrow), or a pigtail catheter placed into the pleural space using the Seldinger technique (see Chapter 10). Each of these catheters should be placed to a chest drainage collection unit that incorporates a water seal chamber as well as a variable suction control. Persistent airleaks initially require continuous suction (usually 20 cm H_2O suction) to evacuate the pleural space and promote coaptation of the visceral and parietal pleurae. Reduction of suction and placement on water seal alone follows the resolution of the air leak. Chest tube removal may proceed directly from water seal if there is no pneumothorax on chest film, or may follow a test period of tube clamping and subsequent radiographic evaluation. The authors favor a 4-hour period of clamping as a recurrent pneumothorax is easily treated by unclamping a tube instead of placing a new one.

Not all patients with a pneumothorax require invasive techniques to evacuate air from the pleural space. It is important to recognize that small pneumothoraces occurring in *spontaneously breathing patients* (i.e., negative-pressure ventilation) may be reevaluated in 4 to 6 hours with a repeat CXR, and drained only if they are expanding. This option is *not* advised for patients on any form of positive-pressure ventilation as a simple pneumothorax may rapidly become a tension pneumothorax with subsequent hypotension and death. Tension pneumothoraces may be recognized by tachycardia, hypotension, elevated P_{aw}-peak (if mechanically ventilated, tachypnea if not), jugular venous distension (if not intravascularly depleted), thoracic resonance by percussion *on* the affected side, diminished or absent breath sounds *on* the affected side, and tracheal deviation *away from* the affected side. Clearly, not all signs or symptoms are present in all patients and treatment is dictated by the patient's clinical condition.

Certain patients develop loculated pneumothoraces or fluid collections. If the collections are either single *or* immediately adjacent to one another *and* readily identified, they may be drained using ultrasound guidance at the bedside. [38] However, the loculations are frequently in inaccessible areas, or are difficult to image with ultrasound. Therefore, CT scanning of the thorax provides precise anatomic definition of the presence and number of loculated collections, as well as a guide for the interventional radiologist. The authors have successfully used CT-guided drainage of loculated pleural collections (air and fluid) to assist weaning of head-injured patients from mechanical ventilator support. [23]

Biotrauma.

This term refers to the self-sustaining process of lung injury from MV that follows alveolar overdistension or rupture, alveolar hypoperfusion, and repetitive shear stresses across alveolar walls. Originally this problem was thought to be from too much pressure (barotrauma). [39] Current principles hold that elevated airway pressures are a straightforward reflection of excess volume delivered to a lung that cannot accept that much gas (i.e., volutrauma: excess volume is delivered). [13] When this process is active in a patient on MV, it is termed volume induced lung injury. Lung injury is an inhomogeneous process with areas of normal lung immediately adjacent to quite diseased and injured segments. [40] Thus, the healthy and compliant segments with shorter regional time constants will readily accept gas, while their neighbors with reduced compliance and longer regional time constants will not. The end result is overdistension of the compliant segments, alveolar injury, and the liberation of inflammatory cytokines, chemokines, and activation of endothelin and arachidonic acid pathways, as well as the expression of adhesion molecules along the vascular endothelium. [4] This leads to infiltration of inflammatory cells, their destructive lysosomal enzymes, and the induction of toxic oxygen metabolites. Avoiding this inflammatory cascade is an intelligent means of protecting a patient's lungs from volume induced lung injury. Such a notion has given rise to lung-protective ventilator strategies based on the low tidal volume ventilation (6 to 7 mL/kg body weight).

Hemodynamic Compromise.

In all circumstances, the volume of venous return exactly matches the cardiac output volume. Any process that impedes venous return will decrease the available volume that establishes cardiac output. For patients on positive-pressure ventilation, each gas delivery increases the intrathoracic pressure while exhalation decreases that pressure. Therefore, venous return principally occurs during exhalation. If the ventilator orders are constructed in such a way as to lead to increased intrathoracic pressure during exhalation, then venous return will be

reduced. Variables that can lead to this circumstance are increased PEEP, auto-PEEP, and inverse ratio ventilation. Recall that venous return depends not only on a relatively negative pressure within the thoracic cavity, but relies on a sufficient amount of time for flow into the thoracic vasculature and right side of the heart. Thus, significantly high respiratory rates may compromise venous return as well. An additional untoward side effect of impaired venous return is cerebral venous hypertension from impeded venous drainage. Since there are no valves between the cerebral parenchyma and the right atrium, increased pressure on the right atrium reduces cerebral venous flow and may contribute to cerebral ischemia in patients with traumatic brain injury or stroke, especially in the patient with compromised systemic hemodynamics. Such patients are prone to watershed infarction; cerebral venous hypertension may increase this risk.

Ventilator Associated Pneumonia.

The association between the duration of endotracheal intubation and the promotion of pneumonia is quite clear. In fact, the likelihood of developing a pneumonia is four times greater for patients in a surgical ICU than those in a medical ICU. [41] Endotracheal intubation for >12 hours increases the risk threefold. [41] Early pneumonias (<72 hours post intubation) are typically with community-acquired pathogens and may be adequately treated with American Thoracic Society (ATS) Class A or B agents. [42] Late pneumonias (>72 hours) typically stem from nosocomial pathogens that may be resistant to community antibiotics. [42] Such patients need to have empiric coverage for *Pseudomonas*, methicillin-resistant *Staphylococcus aureus* (MRSA), and the other SPACE microbes (*Serratia*, *Pseudomonas*, *Acinetobacter*, *Citrobacter*, and *Enterobactericiae*). Empiric coverage for fungi are not warranted except in special circumstances (recrudescent pneumonia in a patient already on broad-spectrum antibiotics for >7 days with negative cultures; solid organ transplant patient after implanatation for >4 months; poly-site positive fungal cultures or fungemia). Unequivocally, clinical estimation of pneumonia is correct at best 33% of the time. [43] The most sensitive and specific test to diagnose pneumonia in a patient with a radiographic infiltrate, fever, leukocytosis, and purulent secretions is bronchoscopy and bronchoalveolar lavage with quantitative cultures. [44] This strategy provides strong evidence of the exact pathogen(s), eliminates treating nonpathogenic microbes that are upper airway colonizers, and provides confidence in withholding antibiotic for the diagnosis of "no pneumonia," as many other diagnoses can present with a similar clinical picture (Fig. 8-10).

Nosocomial pathogens commonly have multiple resistance profiles, typically plasmid-mediated. Resistance pressure from the use of third-generation cephalosporins has led to the establishment of Vancomycin resistant enterococci (VRE) as well as extended spectrum ß-lactamase producing (ESBL) organisms of which *Klebsiella* is the prototype. [45] Plasmid-mediated resistance to fluoroquinolones parallels the rise of ESBL-producing organisms. [46] Empiric antibiotic selection should be derived from each hospital's local antibiogram based on likely pathogens. A ß-lactamase inhibitor combination paired with an aminoglycoside and vancomycin are the authors' empiric agents of choice based on their local antibiogram for Ventilator associated pneumonia. Should the reader's microbiology lab identify an ESBL-producing pathogen, the appropriate antibiotic class of choice is carbapenem. [47] Carbapenems consistently demonstrate excellent efficacy in eradicating ESBL-producing

microbes.

Figure 8-10 Confounders in the diagnosis of pneumonia. Fever, leukocytosis, radiographic infiltrate, and sputum production do not necessarily indicate the diagnosis of pneumonia. Multiple other causes should be considered as well so that one does not apply antibiotics when there is no infectious agent to address. ALI = acute lung injury, ARDS = acute respiratory distress syndrome.

ADJUNCTIVE THERAPIES

$ß_2$ -Agonists.

These agents stimulate ß-adrenergic receptors in bronchial smooth muscle, and induce muscle relaxation. This reduces airway resistance and improves gas flow through the conducting airways.[48] The $ß_2$ -agonists also inhibit MAST cell degranulation, leading to ameliorated immune stimulation of the reactive airway. The most widely used agent in ICUs in the United States is albuterol. This agent may be administered via a side port of the ventilator circuit using a metered dose inhaler (MDI; cost-effective). Alternatively, albuterol may be delivered by placing an in-line nebulizer device between the circuit and the ETT, or on a side port on the ventilator tubing's inspiratory limb (ventilator tubing-dependent). Many patients may develop bronchoconstriction and wheezing when mechanically ventilated without a preexisting history of reactive airways disease. $ß_2$ -agonists should be administered to patients who have poor air movement, wheezing, or both. A physiologically appropriate means of detecting and following bronchospasm is the Peak-plateau gradient. A normal gradient is <4 cm H_2O pressure; increased values indicate increased airway resistance. The efficacy of treatment with $ß_2$ -agonists, intravenous magnesium, or diuresis may be assessed by following the changes in this gradient.

Acetylcholine Antagonists.

The main utility of this class of agents is to dehydrate secretions although these agents may also block cholinergic-mediated bronchospasm. The most common agent is ipatropium bromide (Atrovent), an atropine derivative that has twice its biopotency on an eqimolar basis. Ipatropium is commonly prescribed in combination with $ß_2$ -agonists every 4 to 8 hours. As with albuterol, ipatropium may be administered by MDI or nebulizer. Ipatropium is rarely used in isolation for the therapy of bronchospasm.

Mucolytics.

The prototype for this class is n-acetylcysteine (NAC). NAC is believed to reduce the adhesion of mucous strands to each other as well as the luminal surface of the alveoli and larger airways. It may be administered by nebulizer or lavage routes; no MDI equivalent is available. NAC has the unfortunate side effect of inducing mucosal

inflammation in an unpredictable fashion when used for >24 hours. However, for the first 24 hours, NAC may provide significant benefit in liberating densely inspissated secretions from dependent portions of the airways.

Recruitment Maneuvers.

These maneuvers are designed to apply consistent but well-regulated pressure to partly or completely closed alveoli to reintroduce gas into those segments.[49] The targeted segment(s) are those with poor compliance and long regional time constants. The area of interest is placed in a nondependent position (i.e., for left lower lobe benefit, place the left side of the patient up and right side down), and the patient is hand-ventilated using a bag-valve device attached to the endotracheal tube. An in-line pressure monitor is needed. The patient will usually require sedation to comply with the maneuver—fentanyl and midazolam are ideal. The clinician then applies pressure to the bag to achieve 35 cm H_2O pressure and holds it for 4 to 6 seconds, after which the patient is allowed to exhale. This cycle is repeated for up to 5 minutes. A second examiner listens to the area of interest for an increase in breath sounds. The recruitment may be terminated when there are good breath sounds on two consecutive maneuvers. Recruitment maneuvers may be combined with chest physiotherapy for added benefit; chest physical therapy should, in general, precede the recruitment maneuver.

Bronchoscopy.

See nosocomial pneumonia earlier.

Bronchoscopy/Bronchoalveloar Lavage (BAL).

With MV, the normal bacterial, viral, and secretion clearance mechanisms of the mucociliary elevator are compromised. Accordingly, excellent pulmonary toilet is required to prevent secretion impaction, atelectasis, pneumonitis, pneumonia, intrapulmonary shunt, or ventilation perfusion mismatch. Despite seemingly adequate nursing or respiratory therapist care of a patient's airways, atelectasis, mucus plugging, and segmental or subsegmental airway obstruction and collapse may occur. Several initial maneuvers are indicated, including: alveolar recruitment, chest physiotherapy, postural drainage, and aerosolized bronchodilator therapy. Frequently, these maneuvers resolve the elevated P_{aw} -peak and hypoxemia that are the markers of complications. When these initial therapies fail, a more invasive approach is warranted.

The traditional approach to clearance of inspissated secretions is therapeutic bronchoscopy. The adult flexible fiberoptic bronchoscope has an outer diameter of 3.3 mm and a working channel for suctioning of 2.5 mm. Therefore, an ETT of size =8.0 is ideal as it will permit easy passage of the bronchoscope and allow for adequate MV of the patient. However, airway mucosal irritation is a powerful sympathetic stimulant. Tachycardia, systemic hypertension, and bronchospasm commonly complicate therapeutic bronchoscopy. In the setting of intracranial hypertension, the clinician must take steps to blunt any potential sympathetic stimulation. Mucosal irritation may be minimized by careful bronchoscopic technique that avoids impacting and suctioning the sidewalls. Additionally topical or systemic lidocaine will also blunt mucosal irritation. Preprocedure ß-blockade with a relatively short-acting agent like esmolol will blunt the tachycardia and elevated dP/dt that accompanies heightened sympathetic tone. This will diminish any increase in cerebral blood flow that accompanies sympathetic discharge. Adjuvant therapy with narcotic analgesia, with a short-acting agent like fentanyl, will enhance sedation and ameliorate pain from mucosal injury. When these measures fail, significant sedation and cerebral protection may be achieved with cautious administration of barbiturates like sodium pentothal. Pentothal therapy may also be complicated by systemic hypotension. Alternatively, for very short procedures, etomidate is an excellent and powerful sedative that has the unique advantage of inducing diminished ICP. Thus, excellent intravenous access for fluid or inotrope administration is mandatory when using barbiturates. A postprocedure CXR is indicated to assess the results of the bronchoscopy, and to assess for complications such as a pneumothorax or ETT malposition.

When a specimen is obtained, it should be examined by Gram stain as well as culture for bacteria, viruses, fungi, or AFB when indicated. These results will help guide initial antibiotic therapy if the patient's clinical condition indicates infection (i.e., mucosal erythema, leukocytosis, fever, hypotension). The culture results are *qualitative* only and serve to identify which organism(s) are present, but not the bacterial burden. Accordingly, the utility of such results has been derided as being no more useful that an aspirate obtained by a closed suction system. However, a closed suction system does not allow directed suctioning of a particular side of the airway. In fact, the right side is more frequently suctioned than the left based on the straighter geometry of the right mainstem bronchus. To combat the geometry, "steerable" suction catheters that allow for directed lavage and suctioning are available. Furthermore, the suction catheter is enclosed within a sleeve that protects it from contamination during passage through the ETT and upper airways.

With both the "steerable" catheter system and therapeutic bronchoscopy, the techniques may be modified so as to allow for *quantitative* assessment of the bacterial/viral/fungal burden. The technique is called bronchoalveolar lavage, and relies on wedging the tip of the fluid instillation/suction catheter into a broncho-pulmonary segment, instilling a known amount of fluid (usually 180 mL of NSS in 60-mL aliquots), and recovering that fluid for analysis. An adequate recovery is >50% of the instillate volume. Moreover, there are criteria for the diagnosis of infection (bacteria >300 CFU/mL).[50] The criteria are liberalized (>500 CFU/mL) if the lavage and aspirate were performed in a larger airway such as the bronchus intermedius, instead of a segmental orifice such as the superior segment

of the lower lobe.

Another modality that may be useful in the diagnosis of pulmonary infection is the bronchoscopically directed "protected brush biopsy." [51] In this technique, the bronchoscope is advanced into the area of interest, and a sheathed brush is advanced through the working channel into the airway. Then the brush is extruded and worked back and forth against the airway to "biopsy" adherent microbes and airway mucosa. The brush is withdrawn into the sheath, and the entire assembly is withdrawn. The brush is then cut off and incubated in culture media. This technique has numerous advantages in that upper airway secretions may be suctioned without fear of contaminating the specimen and obtaining spurious results. By comparison, the standard BAL technique requires that the operator guide the bronchoscope into the affected region without suctioning so as to not contaminate the subsequently aspirated lavage sample. The downside to protected brush biopsy is that mucosal injury, bleeding, and pneumothorax occur more commonly than with bronchoscopically directed or steerable catheter BAL. Regardless of the technique used, the clinician must match the sample results with the patient's clinical picture.

Gastric Content Aspiration and Pneumonia.

Additional consideration is needed to avoid gastric acid blockade in patients who require prolonged intubation and MV. Gastric acid inhibition has, in some studies, been associated with a higher rate of nosocomial pneumonia than in patients who received ulcer prophylaxis with sucralfate alone. [52] It is believed that the gastric acid milieu destroy refluxed bacteria and that most nosocomial pneumonias occur because of aspiration of gastric contents. It is important to recall that aspiration may occur simply by "wicking" of gastric secretions along an indwelling nasogastric tube that stents open the upper and lower esophageal sphincters, as well as by vomiting and passage of gastric contents along the sides of the cuffed endotracheal tube. Aspiration of gastric acid with intubation (the most common scenario) does not require antibiotic therapy, nor is it improved by the administration of glucocorticoids or the use of immediate bronchoscopy (unless there is large airway obstruction). The clinical syndrome of sterile gastric content aspiration is Mendelson syndrome.

Heliox Therapy.

The interface of gas with airways creates a certain amount of friction. Heavier gases lead to greater amounts of friction than lighter gases. The more friction generated, the greater the work of breathing for a given gas. Severe asthma commonly entails significant work of breathing and may lead to respiratory failure from respiratory muscle fatigue. Altering the gas composition from N_2:O_2 to He:O_2 provides for a lighter gas that requires less work of breathing. Different percentage mixtures of He:O_2 (Heliox) are prepared and commercially available (e.g., 70% O_2 and 30% helium). Successful resolution of impending respiratory failure has been achieved using this strategy. [53] *Note that this is not a commonly used therapy, but an important adjunct to have available when the need arises.*

Negative-Pressure Ventilation.

This unique mode of ventilation is best achieved using the Hayak Oscillator, a device produced in Israel. Its appearance is quite similar to a Cuirasse vest, but the driving negative-pressure source is quite different. The Hayak Oscillator features independent controls for the application of negative pressure as well as positive pressure, frequency of cycling between negative and positive pressure (inspiration and expiration), a chest physiotherapy mode for sputum expectoration (useful for those with cystic fibrosis), as well as a cardiopulmonary resuscitation mode. It is not widely used in the United States, but has demonstrated use in the cystic fibrosis patient population and during upper airway surgery, when an indwelling ETT would be a significant obstruction. [54]

SPECIAL TOPICS

Asthma.

Fortunately, most patients with asthma are easily managed with combination therapy such as ß $_2$ agonists, acetylcholine antagonists, and glucocorticoids. A true management challenge is the critically ill asthmatic. These patients are different from others with asthma exacerbation in that they require intubation and MV. [33] Unlike many other disease states, asthmatics are not immediately improved by positive-pressure ventilation; asthmatics often become acutely worse before any improvement from intubation and ventilation is realized. After intubation, the asthmatic's P_{aw} -peak is usually elevated. This leads to various problems including, but not limited to, early termination of a volume cycled breath (excessive airway pressure limiting the breath), impaired gas exchange (increased V_D/V_T), and the induction of "biotrauma" (see later). A slower respiratory rate allows for a longer time in exhalation. A prolonged T_e is essential for the patient with restrictive disease. In volume-controlled ventilation, a lower respiratory rate with a low V_T , as in the ARDSNet protocol may be used for the management of life-threatening asthma. [30] With pressure-controlled ventilation (as discussed earlier), the set pressure may generate an inadequate V_T based on the restrictive component of the exacerbated asthma. It is essential that bronchodilator and anti-inflammatory therapy (i.e., glucocorticoids) be pursued in conjunction with positive-pressure ventilation for an optimal outcome.[34] A diligent search should be undertaken to discern any potential triggers (e.g., infection) that may be eliminated to hasten recovery and limit the duration of MV. Appropriate sedation is critical to ensure adequate gas exchange; it enables the patient to "synch" with the ventilator and not trigger early volume cycled breath termination. If sedation alone is inadequate to reduce the restriction imposed by the chest wall or intra-abdominal contents, pharmacologic relaxation is then indicated (although uncommonly required). Heliox therapy has also been used with success for the failing asthmatic as a means of avoiding intubation in select patients (see Heliox section).[35] APRV has been used for severe life-threatening asthma; insufficient data are currently available to recommend this as front-line therapy. Its role may be as a salvage mode for asthmatics with refractory hypoxemia.

Ventilator Weaning Protocols and Pathways

A well-designed weaning protocol is an invaluable aid in reducing the length of stay in the ICU. An appropriate protocol will enable the respiratory therapist and bedside nurse to initiate the weaning process each day before clinician evaluation. Computer order entry may create an ICU admission data set that automatically activates such a protocol once the entry criteria are met (i.e., the cause of respiratory failure is improving or has been eliminated, $FIO_2 < 0.50$, PEEP < 10 cm H_2O, and no pressors other than dopamine at <5 µg/kg per minute or Epi or NorEpi at < 0.05 µg/kg per minute). A ventilator pathway to chart and modify the progress of each patient through his or her MV needs is a useful tool. Such a pathway allows clinicians to regularly review a patient's progress along what would be considered a "usual course" for someone requiring MV. Deviation from this course should prompt an investigation into the cause(s). A pathway is also an excellent tool to use as a platform for quality assurance and improvement review.

Neuromuscular Blockade

Neuromuscular blocking agents are used to induce muscular paralysis for various reasons including, but not limited to, reducing peak airway pressures during MV, reducing total body oxygen consumption, protecting life-sustaining indwelling devices, and placing an artificial airway. Agent selection entails consideration of factors identical to those surrounding analgesic and sedative selection. Commonly used agents include pancuronium, vecuronium, and cisatracurium. All may be given by bolus or continuous infusion. Only pancuronium and vecuronium have active metabolites and reportedly result in prolonged neuromuscular blockade in some patients following cessation of drug therapy. [55] Furthermore, aminoglycosides, for

instance, may potentiate the effect of neuromuscular blocking agents, thus reducing the amount of drug necessary to achieve the desired paralysis. [56] The authors prefer cisatracurium for neuromuscular blockade as it undergoes Hoffman elimination in the plasma and is therefore independent of renal or hepatic metabolism. However, the authors also rarely use neuromuscular blockade outside of the operating room except when placing an endotracheal tube.

Paralysis is commonly titrated to an effect monitored by a peripheral nerve monitor applied over the ulnar or other peripheral nerve distribution. [57] No blockade results in four twitches of the adductor pollicis muscle resulting from four supra-maximal triggering stimuli; complete blockade yields no response. A common goal of blockade is enough agent to result in two twitches out of a "train of four." Another goal of twitch monitoring is to avoid over-paralysis, to diminish the risk of prolonged neuromuscular blockade following withdrawl of the agent. If, however, zero twitches are required to achieve the goals of therapy, then the monitor cannot monitor over-paralysis at all. Additionally, if feasible, many clinicians allow patients to emerge from paralysis once each 24-hour period to perform a neurologic assessment and help ensure return of neuromuscular function following cessation of drug therapy. There are no data to support this practice as a preventive measure, but it seems to make intuitive sense. There is a growing trend to avoid chemical relaxation throughout the United States; chemical relaxation is rare in the European Union for patient management in the critical care arena.

The myriad potential complications of neuromuscular blockade have been described in detail in standard anesthesia texts. However, two important complications deserve mention: prolonged paralysis syndrome [55] and the polyneuropathy of critical illness. [58] The postparalysis syndrome is diffuse motor weakness associated with elevated creatine kinase levels (MM fraction), and preserved sensory nerve function on electromyography and nerve conduction velocity testing. By comparison, critical illness polyneuropathy involves both sensory and motor nerves, and is less frequently associated with neuromuscular blocking agents as an etiologic cause. Critical illness polyneuropathy is believed to be principally related to the underlying disease and carries a less favorable prognosis for recovery than postparalysis syndrome. There are some data implicating the amino-steroid structure of vecuronium and pancuronium in the pathogenesis of either of the complications mentioned previously by drawing a parallel between the neuromuscular blockade polyneuromyopathies discussed earlier and those identified in patients on long-term steroid regimens. However, data are currently inconclusive as to the exact etiology of the syndromes discussed earlier.

Sedation

Patients on MV commonly require some sedation, which can be provided in an intermittent bolus fashion or by continuous infusion. Clinician monitoring of the depth and adequacy of sedation is feasible in an interactive patient. When patients require sedation for agitation control, are mechanically ventilated, or are chemically relaxed, the sedative assessment ability of the clinician is severely impaired. Additionally, since the use of pharmacologic paralysis presents the external appearance of a quiet, restful patient, it is important to have some means of titrating sedation to an appropriate level. The authors favor using a modified single

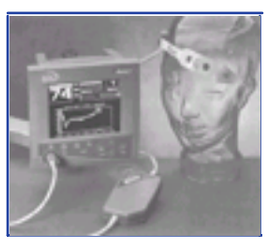

Figure 8-11 The Bispectral Index monitor (Aspect Medical, Nantucket, MA). This is a pole-mounted device that attaches to the patient's forehead and provides a modified single-lead electroencephalogram whose power spectrum undergoes a Fourier transformation to yield a numeric representation of the level of sedation.

lead electroencephalogram montage known as the Bispectral index (Fig. 8-11). This device integrates a power spectrum of the coherence of electrical activity of the monitored areas of the brain and translates the information into an analog value ranging from 0 to 100. [59] Lower numbers indicate deeper levels of sedation. This device has been successfully used in the operating room to monitor and titrate the level of benzodiazepine or propofol sedation for surgical procedures. [60] Further

experience is being gained in the ICU titration of therapy in pharmacologically paralyzed patients as well as monitoring serial changes following central neuraxial injury or illness that results in brain death. The authors have used BIS monitoring to decrease sedative agent usage and cost in intubated neuromuscularly blocked and sedated patients in the critical care setting. [61]

Neonatal Ventilation

It is clear that neonatal ventilation is not simply ventilation of small adults. The vast majority of neonatal ventilation is performed as pressure-controlled ventilation, albeit with smaller volume targets than in adults (i.e., same mL/kg body weight but smaller changes in pressure to achieve the smaller needed volumes). Note, however, that neonatal ventilation may also be performed using volume-controlled ventilation. Not all ventilators can deliver the small volumes required for this kind of ventilation, and special ventilators dedicated to neonatal ventilation have been developed (e.g., Babylog by Drager, Bird Ventilator). Many adult ventilators are equipped with a software package that allows the microprocessor that controls pressure, flow rates, and volume to perform well in this application (e.g., Drager E_4 with Neoflow, Siemens Servo 300C). Furthermore, there are hybrid modes such as pressure-support ventilation volume guarantee (PSV-VG) that combines the best of both worlds.[62] One simply sets a pressure

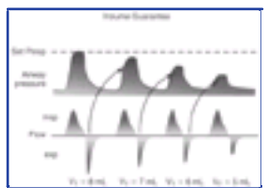

Figure 8-12 Volume guarantee ventilation. Note that the ventilator uses the prior breath to determine how much support is required to achieve the desired tidal volume and remain within the set pressure limit.

support limit as well as a desired volume for each breath. The ventilator then determines on a breath-by-breath basis how much pressure support is required to achieve the set target and remain within the pressure limit (Fig. 8-12). In this way, the mode is also self-weaning; as the patient's need for support decreases, the support, in fact, decreases (Fig. 8-13). Since the equipment varies at each institution, clinicians are urged to familiarize themselves with the available ventilator and how to use it. Guidance from a neonatal intensivist in conjunction with a respiratory therapist is ideal.

Noninvasive Ventilation

Intubation, MV, and its sequelae may be avoided in a select group of patients suffering from acute respiratory failure by using noninvasive ventilation (NIV) techniques (see chapter 2). It

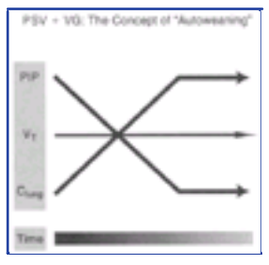

Figure 8-13 Pressure support volume guarantee ventilation. As the compliance of the lung (C lung) improves, the positive inspiratory pressure (PIP) decreases while maintaining a constant tidal volume (V_T). When the pressure support volume is reduced to an acceptable minimum level, weaning has been achieved and the patient should be evaluated for liberation from mechanical ventilation.

is important to recall that if the patient's physiology requires definitive airway control, NIV is not appropriate. This modality may be effectively used in patients who do not wish to be intubated (e.g., end-stage COPD) and in patients who need time for medical therapy to achieve its goals (e.g., congestive heart failure). In general, NIV is commonly used for temporary ventilatory and oxygenation support (i.e., <24 hr). Patients requiring longer acute management are optimally managed by tracheal intubation and MV.

For NIV to be used, three things must be true: the airway must be patent, the respiratory drive must be intact, and the patient must be cooperative (i.e., awake and alert). It is imperative that the patient understands the roles of the machine and the mask to aid with compliance and technique efficacy. It is essential to use the correct size mask to maintain a good seal for optimal results and to lessen the risk of injury from an inappropriately tight or ill-fitting mask. For nasal masks, there is a template to help the therapist select the most appropriate piece of equipment. The potential benefits of NIV over MV are a decrease in potential airway injury, nosocomial airway and tracheobronchial tree infection, and probably a shorter length of stay in a monitored bed or ICU. [63] Documented risks include barotrauma (volutrauma), pressure necrosis of the facial skin, subcutaneous tissue and musculature, and gastric dilation followed by vomiting and aspiration or hemodynamic compromise.[64] Two main types of NIV are available in the ED—CPAP and bi-level positive airway pressure (BiPAP).

Continuous Positive Airway Pressure

CPAP is a widely used type of noninvasive ventilatory support. This modality delivers a variable gas flow to achieve a constant airway pressure. It is commonly used in patients with obstructive sleep apnea to prevent upper airway collapse. [65] All forms of NIV require some type of tight-fitting mask to maintain a good seal (Fig. 8-14). The CPAP mask is a full-face mask. It must be properly fitted to the patient for both efficacy and complication avoidance (i.e., pressure necrosis). The full-face mask may be intolerable for some patients as it may induce a sense of confinement and claustrophobia. Acutely ill patients are also unable to remove the mask to eat as they are dependent on the airway pressure for oxygenation and ventilation. For the acutely ill patient in the ED, CPAP may help reduce the work of breathing while facilitating CO_2 clearance and O_2 onloading.

Bi-Level Positive Airway Pressure

BiPAP is a combination of PSV and CPAP (Fig. 8-15). The clinician can set two different pressure levels for the patient: (1) inspiratory positive airway pressure (IPAP), which is delivered during inspiration, and (2) expiratory positive airway pressure (EPAP), which is applied during exhalation. In general, IPAP exceeds EPAP. This modality is pressure limited, and flow is triggered by either the patient's inspiratory effort, or a time limit between cycles.

In the patient-triggered mode, the device senses the onset of inspiration, and the preset IPAP cycle is initiated. When the patient finishes inhalation, IPAP is cycled off, and the airway pressure is allowed to decrease to the preset EPAP level. The average starting range for IPAP is 8 to 10 cm H_2O while EPAP is 3 to 5 cm H_2O. Adjustments of these levels are

Figure 8-14 *Continuous positive airway pressure* (CPAP) mask. This is a nasal CPAP mask and has a low-pressure bladder around the rigid mask. There is a hook-and-loop closure strap system to secure the mask to the patient. Care must be taken to ensure a proper fit. The goal is to achieve a tight seal without creating undue pressure on the skin, nose, or other bony

prominences of the head, thus avoiding pressure necrosis.

generally made in 2 cm H_2O increments. Generally, increases or decreases in each are titrated to patient comfort (both IPAP and EPAP), SaO_2 (IPAP and EPAP), and the volume of gas that is moved (IPAP). A general approach is to increase IPAP until achieving reasonable V_T and pCO_2. If oxygenation remains inadequate, then EPAP is usually increased. Adjusting FIO_2 is somewhat more difficult with BiPAP than with other modes of invasive or NIV.

The BiPAP machine is connected to oxygen. It is run at the same flow rate that the patient required immediately before implementing NIV. Recall that the final FIO_2 is a blend of entrained room air and the bleed-in rate of O_2 from the pure 100% O_2 source (wall or tank). Each breath may therefore deliver a different final FIO_2 dependent on patient comfort and the respiratory effort.

When initiating BiPAP, the initial maneuvers are similar to initiating CPAP. An appropriate size full-face mask is chosen

Figure 8-15 The BiPAP machine. Each hospital may have a different BiPAP device. The key features are an O_2 connection to bleed-in O_2, a rate control, and separate controls for the level of inspiratory positive pressure (IPAP) and expiratory positive pressure (EPAP), as well as separate timing controls for IPAP and EPAP. Clinicians should familiarize themselves with the unique features of the device available at their institutions.

using the guide mentioned earlier. The mask should not place direct pressure on the bridge of the nose, the lateral ala, the inferior nasal septum, or the lip. The patient can participate in his or her care by initially holding the mask in place while the therapist adjusts the settings and the patient adjusts to the sensations and pressure (airway and mask). The mask is secured in place by using adjustable velcro straps. With the nasal mask it is important to encourage the patient to breathe with the mouth closed; in general, nasal BiPAP is quite difficult and not recommended for the acutely ill patient. It is important to check the fit of the mask for air leaks as they diminish the efficacy of the modality in proportion to the size of the leak. It is possible to obtain near-optimal settings in about 10 minutes.

Patients undergoing NIV need close monitoring and observation. [66] Patients initiated on BiPAP therapy should not be admitted to the general medical floor; step-down level care

is a minimum while ICU level care is optimal. Each hospital will have admission criteria for the care of patients on NIV that is driven by both clinician and nursing protocols designed to provide safe care of patients with potentially unstable airways and respiratory dynamics. Accordingly, these patients should be initially observed on continuous SaO_2 and hemodynamic monitoring of heart rate, blood pressure and respiratory rate. If available, continuous end-tidal CO_2 monitoring may be beneficial. An arterial blood gas should be obtained after initiating BiPAP therapy to assess CO_2 clearance and pH-pCO_2 balance. Titration of O_2 flow rate may be performed using SaO_2 monitoring instead of repeat analysis of arterial blood gases. In general, increases in IPAP will increase the V_T and lower CO_2, while increases in EPAP will increase FRC and increase O_2. BiPAP has been successfully used in the ED and in the postoperative ICU setting for short-term respiratory support in patients with a correctable underlying cause of their respiratory failure. [67]

Evaluation Adjuncts

The totality of the ventilator prescription interacts with the patient's pulmonary system in ways that may be assessed. The most straightforward is physical examination (see section on endotracheal intubation). However, the end result of the ventilator prescription is commonly evaluated using an ABG analysis. This directly measures pH, pCO_2, and pO_2 while the remainder of the values, including base excess (deficit), are calculated from an algorithm. Additional information that is frequently available as part of the ABG analysis usually includes Hgb or Hct, Na^+, K^+, Ca^{2+} (ionized), glucose, and lactate.

End-Tidal CO_2 Analysis, Capnometry, and Capnography (see Chapter 2)

Additionally, the flow pattern of exhaled gas as measured by the expired CO_2 concentration over time is evaluated by CO_2 capnometry (numeric data) or CO_2 capnography (graphic analysis). [69] Capnometry (quantitative measurement of CO_2) is a useful means of noninvasively tracking a patient's CO_2 tension. The principle behind capnometry is that the CO_2 measured at the end of a tidal exhalation ($ETCO_2$) will approximate the alveolar CO_2 and thus the blood pCO_2. If the $ETCO_2$ measurement and the arterial pCO_2 correlate, then changes in minute ventilation that are designed to produce a certain pCO_2 may be followed without the need for ABG determination. In normal lungs, the $ETCO_2$ is 1 to 5 mm Hg less than the pCO_2 as a consequence of heterogeneous alveolar expiratory dynamics (i.e., alveolar unit heterogeneity with regard to time constants for alveolar gas flow). $ETCO_2$ measurement may be accomplished by three means: infrared spectroscopy (most common), chemical analysis (common, qualitative only), and mass spectroscopy (uncommon and expensive).

Infrared capnometry passes a narrow band of infrared light through a sample of gas and determines the CO_2 concentration by analyzing the amount of light absorbed by the CO_2. Access to exhaled gas through the ventilator circuit occurs by way of either mainstream or sidestream sampling. The mainstream sensors are directly in line with the breathing circuit and pose two hazards and one disadvantage: (1) weight and rotational torque on the endotracheal tube, (2) an increase in dead space, and (3) the need to heat the sensor to avoid condensation. Sidestream analyzers continuously obtain gas

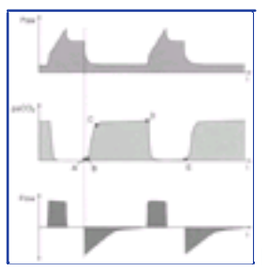

Figure 8-16 The end-tidal CO_2 ($ETCO_2$) trace. This diagram of an $ETCO_2$ capnograph represents normal exhalation flow dynamics. As exhalation progresses, the measured CO_2 rises and eventually plateaus. At the end of the plateau, immediately before the downstroke of the trace is the point at which the $ETCO_2$ value is measured and reported (i.e., point "D"). No other values are reported on the $ETCO_2$ capnometer. Thus in the absence of the capnograph, all other information is unavailable to the clinician. Abnormalities in endotracheal tube position, blockage of the airway (complete or partial), as well as correct endotracheal tube placement, are identifiable by evaluating the $ETCO_2$ tracing. *(Reproduced with permission from Drager Medical.)*

through a side port attached to the endotracheal tube, and have two disadvantages: (1) the small port is easily occluded with secretions, and (2) they are slower than mainstream analyzers. Capnometry helps ensure that the ventilator is indeed connected to the patient, and allows determination of dead space relative to tidal volume (important in patients with large anatomic dead spaces, severe lung injury, or both). However, much more information can be obtained from the graphic display of exhaled CO_2 over time (capnography).

In particular, a normal exhaled CO_2 tracing plateaus at the end of exhalation (Fig. 8-16). If there is chronic obstructive airways disease, or a significant amount of air trapping from auto-PEEP, the tracing will not plateau due to the prolonged expiratory phase required to empty the alveoli (Fig. 8-17). The presence of significant auto-PEEP may indicate increased intrathoracic pressure, impeded venous return, and thus secondary intracranial venous hypertension. Auto-PEEP may also be detected on ventilators equipped with a graphic display of flow or volume over time. However, as most ventilators in use are not so equipped, an $ETCO_2$ monitor tracing may serve as a reasonable substitute. Capnography has also been used to determine the optimal level of PEEP, [70] adequacy of cardiopulmonary resuscitation,[71] and detection of pulmonary embolus.[72] In the authors' opinion, all critically ill mechanically ventilated patients with

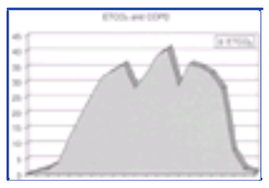

Figure 8-17 The $ETCO_2$ tracing in COPD—identification of auto-PEEP. In this diagram of an $ETCO_2$ trace in a patient with COPD who is being ventilated at a high respiratory rate, one may detect the presence of auto-PEEP based on the abnormal expiratory flow pattern. A similar suggestion could be obtained by interrogating the flow-time trace as well.

traumatic brain injury should be attached to a capnograph/capnometer device. Infrared capnography is the current standard of care in the operating room for CO_2 detection. In the near future, estimates of metabolic rate may be available on a breath-by-breath basis using a capnometry platform.

Chemical analysis of exhaled CO_2 has been effectively used for several years to confirm endotracheal placement of artificial airways. [73] The presence of CO_2 in the exhaled gas causes a color change in the monitoring device. The device is disposable, and placed between the ETT and the high-flow O_2 bag-value unit. It does not impede ventilation. In the authors' institutions, these relatively inexpensive devices are kept in all of the airway management carts, the ICUs, and the ED, and are routinely used in confirming airway placement.

Radiography.

A standard portable anteroposterior chest radiograph following the initiation of MV (and endotracheal tube placement) is the existing standard of care. It is not recommended to send a newly intubated patient requiring MV out of the ED to obtain a chest film.

RESPIRATORY THERAPY

The trained, certified respiratory therapist may be the clinician's "best friend" with regard to MV. These individuals may be invaluable in helping the clinician achieve the desired goals of MV. They are generally more familiar with individual machine performance characteristics than is the typical clinician, and they are responsible for ensuring that the device is functioning properly and with appropriate alarm limits. Multiple studies have documented that respiratory therapist-driven weaning protocols achieve a more rapid liberation from MV than those driven by clinicians. [68] To achieve these results, a weaning protocol is ideally developed in a multidisciplinary fashion, activated by clinician order, and then implemented by the therapists. The clinician is again engaged when the patient either fails to progress along the pathway, or has achieved extubation criteria. Only in rare circumstances is the clinician creating the settings on the ventilator—this is the purview of the respiratory therapist. If the clinician wants to manipulate the ventilator directly, advanced training and local hospital credentialing may be recommended.

CONCLUSIONS

MV is a complex process that requires dedication on the part of the clinician. Usually, the least difficult aspect of initiating MV is establishing an airway. The ED clinician should recall that the early period of MV may set the tone of events related to the remainder of the patient's course on the ventilator. Optimally, the ED clinician should be familiar with the spectrum of ventilatory modes available for use in the hospital; ventilator prescriptions are not ideally managed in a "one prescription fits all" fashion. Alternatives to endotracheal intubation should be considered in select patient populations. Medical adjuncts frequently enable the clinician to manage difficult ventilation issues while optimizing pulmonary dynamics. Difficult ventilatory issues (e.g., refractory hypoxemia) may be best co-managed with a dedicated intensivist in the ED if the patient cannot be rapidly transferred to the ICU.

CASE MANAGEMENT

Placing a patient on a ventilator and managing the setup and changes in ventilation requirements are formidable tasks for any clinician. The following section presents a step-by-step approach to managing two individual patients who require MV. A number of practical and logistical issues, types of ventilators, various modes of ventilation, and practical settings and subsequent changes are illustrated.

Case Scenarios

Case 1

An 18-year-old otherwise healthy 60-kg female presents with an overdose of benzodiazepines. She requires intubation for airway protection and ventilatory support. There is no evidence of aspiration or an intrinsic lung problem.

Volume-Cycled Ventilation

Devices Reviewed.

Puritan Bennet 7200, 7200a, or 7200ae or the Drager Evita 4 Pulmonary Workstation.

Target $?V_E$.

7.2 L/min. It is reasonable to assume a normal need for $?V_E$ since she has no evidence of hypoperfusion or infection, and she has not ingested any medications known to cause a metabolic acidosis that would require a higher $?V_E$ to buffer by induced hypocarbia.

Mode.

Since this patient has preserved spontaneous respirations, it is reasonable to allow her to continue to do so since she is not presenting problems with an increased work of breathing (nonlabored respirations, no accessory muscle use, no stridor, no wheezing, and no hypoxia). This means that one must couple the SIMV mode with PSV to eliminate work of breathing increases associated with the resistance of the artificial airway. Depending on the model of 7200 ventilator, the

Figure 8-18 The Puritan-Bennett 7200 Model ventilator. Note that on this particular ventilator, the controls are grouped into three distinct areas of the ventilator faceplate. To the left of the picture are display areas in green that indicate pressures (top), rate and I:E ratio (middle), and volumes (bottom). At the very top of the green section is an analog pressure meter to confirm pressures in the case of a liquid-crystal display (LCD) malfunction. The center portion (blue) houses the data entry keypad, the alarm limit-setting buttons, waveform selection, sighs, and 100% O_2 for suctioning button. Note that the "++" button is the selector to change between options that are displayed in the horizontal LCD bar above the keypad. Function 10 is the function to set the pressure support and is accessed by using the "++" key until the number 10 appears. One must then press Enter to access the function. The top of the blue section has the PEEP/CPAP knob, as well as the displays for peak flow, FIO_2 , set rate, and tidal volume. The gray section to the right indicates the alarm status as well as the alarm silence button.

Figure 8-19 Faceplate for the Drager Evita 4 Pulmonary Workstation. Note that the liquid-crystal display (LCD) screen is colored and has controls embedded in the screen to adjust the individual mode settings. The righthand controls on the front of the device control the kind of screen display, the silence button, and the standby button.

mode settings will either appear along the bottom or the left side (Fig. 8-18). Using the Drager E_4 , the mode will appear in the lower right hand side of the touch screen (Fig. 8-19).

Rate and Tidal Volume.

12 breaths per minute and 600 mL (7–10 mL/kg). These must be considered together to achieve the desired $?V_E$. This setting will guarantee the desired $?V_E$ even if the patient continues to develop respiratory depression from the benzodiazepine ingestion. It will allow her to take additional breaths using the following PSV settings if she so desires or needs to overcome an increased CO_2 production or a metabolic acidemia from an as of yet unidentified source.

Oxygen and Oxygen Adjuncts.

Start with an FIO_2 = 1.0 and PEEP = +5 cm H_2O pressure. As FIO_2 and PEEP both impact pO_2 , it is logical to consider their settings together. Since one wishes to guarantee that there is no hypoxemia to impact anaerobic metabolism, it is common practice to begin with a high FIO_2 and then reduce the FIO_2 to nontoxic levels using pulse oximetry (goal $FIO_2 < 0.60$). On the 7200 series, the FIO_2 is set using the O_2 concentration button in the central portion of the ventilator faceplate; the FIO_2 is directly entered using the numeric keypad followed by the enter key (see Fig. 8-18). On the E_4 , the FIO_2 is set by touching the faceplate numeric symbol for FIO_2 (it turns yellow), and then rotating the knob on the lower right of the ventilator faceplate to achieve the desired setting; the knob must then be pressed to signal the ventilator that you wish to accept the value (symbol reverts to green).

It is reasonable to start with a relatively low level of PEEP and evaluate the pO_2 that is achieved at the delivered FIO_2 . On the 7200, the PEEP is indicated by the PEEP/CPAP button (see Fig. 8-18). On the E_4 , the PEEP is set directly on the faceplate using the same technique as for setting the FIO_2 . The evaluation of the adequacy of the set FIO_2 and PEEP on pO_2 is easily performed by assessing the Alveolar-arterial gradient (A-a gradient; normal gradient = 50). High A-a gradients may trigger an increase in PEEP to increase FRC and pO_2 .

Pressure Support Ventilation.

Initiate PSV at 10 cm H_2O pressure, and then titrate up or down to achieve a spontaneous breath V_T approximately equal to that of the set V_T. On the 7200, the value displayed for each spontaneous breath is one breath behind what one observes in the patient. The value is displayed after depressing the tidal volume key in the grouping with ?V_E and spontaneous ?V_E (see Fig. 8-18); the V_{Tspont} will not display if one depresses the central keypad labeled tidal volume, as this key gives one the set V_T and displays its figures in the LCD bar in the middle of the faceplate (see Fig. 8-18). On the E_4, PSV is adjusted by activating the touchpad button labeled PSV. The knob is rotated to achieve the desired setting and then depressed to accept that setting (see Fig. 8-19).

Gas Delivery Waveform.

Begin with a decelerating waveform. Recall that the ramp waveform immediately begins to decrease flow after achieving the maximal inspiratory flow that one sets. This is a standard setting for the authors. The three waveforms are indicated by the three white buttons on the lower central portion of the 7200 faceplate (see Fig. 8-18); press "Enter" after selecting the waveform that is to be activated. On the E_4, the waveform may be manually selected using the setup function under "Modes," or it will be automatically selected by the E_4 using the Auto-flow feature. Auto-flow auto-matically retards flow if the maximal pressure limit is achieved, or increases flow if the patient desires more flow for a larger breath at that time. To manually set the waveform, Auto-flow must be disabled (*not recommended*).

It would not be unreasonable to start with a square waveform in this patient with normal lungs, but there is no downside to beginning with a ramp delivery pattern. The crucial information that one needs is the grouped evaluation of waveform coupled with maximal inspiratory ?Q, and the resultant P_{aw}-peak. These data are complemented by using concomitant pressure-volume curve analysis. Since the vast majority of the 7200 ventilators are not so equipped (but instead have a separate graphic display chip and display screen), the discussion assumes that the clinician is blind to the waveforms. A later discussion under Pressure Controlled Ventilation explores the use of the P-V curve and may be applied in volume ventilation as well (waveform display is standard for the E_4).

Maximal Inspiratory Flow (aka Peak Inspiratory Flow; ?Q).

Set the initial ?Q at 60 L/minute. One would set a lower ?Q if the patient had hypoxemia (?Q = 50 L/min) or a higher flow (?Q = 70 L/min) if the patient had exhalation obstruction (e.g., COPD), then evaluate the resultant P_{aw}-peak. Problems generally arise if the P_{aw}-peak is too high rather than being too low. If the P_{aw}-peak is low and the patient is well oxygenated and has an acceptable CO_2 clearance for his or her current pH, then no changes are required. If the P_{aw}-peak is too high, intervention is warranted (see discussion of P_{aw}-peak). A useful paradigm is to decide if the set V_T is simply too large (base the V_T on ideal body weight, not actual or adjusted, as the lung volume is a function of the thoracic cage size and is not influenced by the addition of adipose mass). If the V_T is appropriate, reduce the flow rate by 5 L/minute and re-evaluate; repeat if necessary. If the patient is on a square waveform, change to the decelerating setting. In general, if the ?Q is reduced to 40 L/minute and the P_{aw}-peak remains high, then one of the following conditions are true: (1) the V_T is, in fact, too large for the available lung mass, (2) there is a tube obstruction (partial), (3) the patient has a pleural space occupying disorder (pneumo-, hemo-, hemopneumo-, or hydro-thorax), (4) the patient requires a different mode, or (5) there is a ventilator dysfunction.

Writing the Orders.

The order on the chart may be best indicated as follows:
Initiate: (S)IMV at 10 ventilations/min
V_T at 600 mL
FIO_2 at 0.95
PEEP at +5 mmHg
PSV at +10 cm H_2O

Flow = 60 L/minute; Ramp waveform

Titrate PSV to achieve V_{Tspont} ~ 600 mL

Obtain ABG 20 minutes after initiating settings; notify clinician with results

Obtain STAT portable chest film to verify—endotracheal tube placement, initiation of MV; notify clinician when CXR obtained

Pressure Controlled Ventilation

Devices Reviewed.

Infrasonics Adult Star, Drager E_4 Pulmonary Workstation

Target ?V_E.

Since this is the same patient, the target remains unchanged at 7.2 L/minute. How one achieves this target is the subject of the following discussion.

Mode.

Similar to ?V_E, the mode remains the same. On the Adult Star, the mode is accessed in the second screen. Press the change screen button on the lower aspect of the display console; this will change to the second screen. Use the knob on the lower right of the console to move from one selection to another (selection will highlight). Then press the knob to access the values and rotate the knob until the desired mode appears. Press the knob again to accept. The E_4 has a mode known as PCV+. This is already set up as an SIMV-PCV-PSV mode. The settings are accessed under the Mode screen as in volume-cycled ventilation discussion, and are adjusted using the touch-rotate-press scheme reviewed earlier.

Rate.

While the rate remains the same, there is no set V_T. Instead, one must set the desired pressure change above PEEP and for the duration of that pressure change, and evaluate for the resultant tidal volume. While the goal resultant V_T may be identical to the V_T one sets in volume ventilation, one achieves that V_T in a fashion designed to limit the P_{aw}-peak and (usually) prolong the time spent in inspiration.

PC, T_i, and V_T-resultant.

In this patient with normal lungs, initially set pressure control (setting) = 20 cm H_2O pressure and T_i = 1.0 sec. The PC of 20 stems from starting with a PEEP of 5 cm H_2O pressure. Therefore, the combined P_{aw}-peak will be PC + PEEP, or 20 + 5 = 25 cm H_2O pressure. This is a safe pressure and what one might expect as the P_{aw}-peak if the patient were on a volume-cycled ventilation. Alternatively, one may use the upper pressure limit of normal, 35 cm H_2O and subtract from that the amount of initial PEEP (in this case 5 cm H_2O pressure), and then multiply the difference by ?. In this case the starting pressure would be 20 cm H_2O (35 - 5 × 2/3).

For a patient who is already on MV, take the current P_{aw}-peak, subtract the PEEP, and again multiply by ?. When setting up pressure-cycled ventilation on the Adult Star, all settings must be addressed before the mode can be activated. Thus, one will not have a mixed group of settings at any time.

The E_4 will allow one to change screens and input settings in a similar fashion but will not change the active ventilator settings until the operator accepts all of those mode settings.

One significant difference in pressure-cycled ventilation is that the pressure cannot exceed 25 cm H_2O pressure (*in this example*), and the amount of time in inspiration is longer than in volume-cycled ventilation. One must then look for the tidal volume that results from this combination. Each perturbation in resultant V_T has multiple repair strategies in PCV.

A discussion of T_i and I:E ratio is appropriate as the T_i setting directly influences the resultant I:E.

Inspiration:Exhalation (I:E) Ratio.

A normal I:E ratio is 1:3–4 for each breath cycle in a healthy individual. Assuming a rate of 10 breaths per minute, each respiratory cycle is 6 seconds. For a normal I:E ratio of 1:3 at this rate, there are 1.5 seconds spent in inspiration (T_i) and 4.5 seconds spent in exhalation (T_e). It is then imperative to evaluate the V_T -resultant and ?V_E. If these are low, the PC may be increased. If high, the PC may be decreased. T_i may be adjusted as well. Inspiration is the time for oxygen exchange. If the patient is hypoxic, the inspiratory time may be increased to increase the mean airway pressure and the time spent in inspiration in an oxygen-rich environment. For patients with severe hypoxemia, such a strategy may require inverse ratio ventilation (IRV) where the time spent in inspiration vastly exceeds the time available for exhalation. The extreme range of pressure-controlled IRV on the vast majority of ventilators is approximately 4:1. IRV is a distinctly unphysiologic and uncomfortable mode on which to breathe. Patients generally require heavy sedation and commonly in combination with neuromuscular blockade to tolerate IRV. The unique challenges of monitoring and achieving adequate sedation in patients on neuromuscular blockade have been reviewed elsewhere. [74]

Oxygen, Oxygen Adjuncts, and PSV.

FIO_2 , PEEP, and PSV remain the same as in volume-cycled ventilation settings.

Maximal Inspiratory Flow and Waveform.

In pressure-cycled ventilation, the goal is to allow the ventilator to adjust the flow setting in response to the patient's unique breath-by-breath change in compliance, resistance, and elastance. Thus, setting ?Q at its maximal value is ideal (Start: 160 L/min; E_4 : autoflow). As in volume-cycled ventilation, the ramp waveform is ideal.

Writing the Orders.

The order on the chart may be best indicated as follows:
Initiate: (S)IMV at 10 ventilations/min
PC at 20 cm H_2O
Ti at 1 second
FIO_2 at 0.95
PEEP at +5 cm H_2O
PSV at +10 cm H_2O

Flow = maximum value; Ramp waveform

Titrate PSV to achieve V_{Tspont} ~ 600 mL

Obtain ABG 20 minutes after initiating settings; notify clinician with results

Obtain STAT portable chest film to verify endotracheal tube placement, inititation of MV; notify clinician when chest film obtained

Note: There is no order to notify the clinician if the V_T -resultant is less or greater than anticipated. One should be at the bedside when ventilation is initiated and directly evaluate to adjust the parameters. Once the appropriate settings are achieved, then such an order is advisable as the clinician will want to evaluate changes in the patient's dynamics.

Waveform Analysis (See Earlier Section on Waveforms).

It is logical to evaluate not only the achieved gas delivery volume, but how the patient's lung receives the volume. This is currently best performed by using the dynamic P-V curve. Each change in ventilator prescription is best evaluated by reexamining the P-V curve profile as well as the flow-time trace. On the Adult Star, the screen must be changed twice. Then rotate the knob to reach the bottom of the screen on the left. Select graphics. This will bring up the P-V curve as well as the flow-time trace (Fig. 8-20). On the E_4 , the graphics are accessed by selecting values measured from the righthand column of white static buttons. Then touch the field labeled loops. This will bring up the P-V curve (Fig. 8-21). The green box in the upper right-hand corner of the PV-curve box will have a menu of other traces and loops—the flow-time trace is found there by selecting its label and pressing the knob.

Case 2

A 45-year-old, 70-kg male presents with high fevers, cough, and shortness of breath. The ED portable chest film demonstrates bilateral infiltrates as well as lower lobe air bronchograms. While awaiting admission to the hospital for presumed community-acquired pneumonia, the patient develops hypoxia, suffers respiratory failure, and requires urgent intubation.

Volume-Cycled Ventilation

Mode.

SIMV—As before, allow the patient to breathe spontaneously when the short acting neuromuscular blockers or heavy sedative (e.g., etomidate, fentanyl, midazolam) used to facilitate intubation wears off.

Target ?V_E .

Given the likely acidosis, this patient's ?V_E needs to exceed the lower limit of normal value. Thus, a target of 9.8 L/minute is a 40% increase above the baseline that one would expect for a slenderly built gentleman.

Rate and V_T .

An initial rate of 14 is ideal and may be coupled with a normal V_T of 700 mL. It is OK to use a normal tidal volume to start as the patient does not have a history of intrinsic pulmonary disease. One must evaluate the resultant P_{aw} -peak after starting at this setting. One may also need to reduce the tidal volume and increase the

rate if the P_{aw} -peak is high and no other mode of ventilation is available.

Alternatively, were one to follow the ARDSNet recommendations, a starting V_T of 450 to 500 mL would be ideal.[76] The required rate would be 20 breaths per minute to achieve the target ?V_E . Recall that this strategy may not clear CO_2 as efficiently as a slower rate and a larger V_T given a larger V_D/V_T ratio.

FIO_2 and O_2 Adjuncts.

Begin with a high FIO_2 (0.95 to 1.0) to ensure adequate oxygenation. Titrate as before. Initiate PEEP at a minimum of 5 cm H_2O since one will need to maintain FRC and alveolar recruitment. Starting at a higher level may result in hemodynamic compromise in a patient who likely has intravascular volume depletion. Once the patient is volume-replete, titrate to achieve the optimal PEEP as explained earlier. It is best to start with a decelerating wave form since one may assume that the infected and collapsed segments will have much longer regional time constants than their neighboring alveolar segments. Thus, a longer T_i is ideal for alveolar recruitment. Moreover, a slow flow rate will

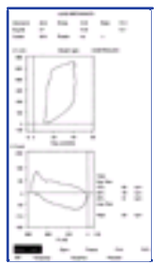

Figure 8-20 The pressure-volume curve on the Adult Star ventilator. Note that the curve is identical to the type of curve one would obtain on the E_4 with the exception of color and the ability to freeze a reference curve for comparison. Here it is accompanied by the flow-time loop, as well.

complement the decelerating waveform and further prolong the T_i . It is reasonable to start with a ?Q of 50 L/minute and evaluate SaO_2 and P_{aw} -peak.

Pressure Support.

Start with a higher PSV than the last patient since one may assume that the pulmonary compliance will be less than in a patient with normal lungs. Initiate PSV at 15 cm H_2O pressure and titrate as before to achieve similar V_T with the machine and spontaneous breaths.

Pressure-Cycled Ventilation

Neither the mode nor the target ?V_E are different. Instead of V_T , one must set PC and T_i .

Rate.

This is not as straightforward as in volume-cycled ventilation. A higher rate to clear CO_2 in volume-cycled ventilation will lead to reduced T_e in PCV if one is using a long T_i (see later). Thus a slower rate will allow for more time spent in recruitment, and more efficient CO_2 clearance than would be achieved with a higher rate. One must recall that the V_D/V_T ratio also governs the efficiency of CO_2 clearance, not just the total amount of gas that is cycled in 1 minute. Thus start with a rate of 10 breaths per minute, not 14 as in volume cycled-ventilation.

PC and T_i .

As described earlier, start with a PC level that is either related to the volume-cycled ventilation P_{aw} -peak or

Figure 8-21 Pressure-volume curve on the E_4 . This display may be brought up as a solo display, or integrated into other multi-display screens as indicated above. One accesses the menu on the lower right of the screen by activating the "values measured" button on the right of the faceplate (not the liquid-crystal display [LCD] screen). Then press Loops. The device will either display a continuous set of loops, or a reference and a single breath (yellow button illuminated on the appropriate green icons).

the maximal airway pressure that one will tolerate (35 cm H_2O pressure). In this example, use a longer T_i (start at 2.0 sec; resultant I:E at rate = 10 is I:E::1:2) given the prolonged regional time constants to manage hypoxemia, as a larger V_T will require excessive PC and lead to ventilator-induced lung injury. Progressively increase the T_i for failure to resolve hypoxemia. In this patient, one may ultimately need to employ IRV to reverse hypoxia.

Set the maximal inspiratory flow and waveform as described earlier. The subsequent evaluation is unchanged from Case 1.

Hypoxia Rescue Strategy

The authors recommend APRV as their rescue mode of choice for refractory hypoxemia. Only three ventilators at present can perform in APRV mode: the Drager Evita 4 Pulmonary Workstation, the Nellcor-Puritan Bennett 840, and the Hamilton Gallileo. The E_4 will be used as the prototype for this chapter.

Assume that your patient has refractory hypoxemia on either volume-cycled ventilation or pressure-cycled ventilation. The goal is to restore oxygenation in a rapid, safe, and efficacious fashion. Typically, these patients will have problems with airway pressures during volume-cycled ventilation, or inadequate V_T on pressure-cycled ventilation; many will be pharmacologically relaxed to tolerate pressure-cycled ventilation-IRV, or the ARDSNet protocol for lung protective ventilation. APRV represents a low-pressure, easy-to-use mode that rapidly corrects hypoxemia, and more slowly corrects hypercarbia while preserving spontaneous respiration. Indeed, the benefits of APRV are lost if the patient is chemically relaxed. [75]

Figure 8-22 Intrinsic PEEP (PEEPi). The E_4 will measure the intrinsic PEEP as well as the trapped volume responsible for that PEEPi. The function is accessed by using the "special procedure" button on the righthand side of the faceplate, activating the "start" button on the liquid-crystal display [LCD] screen, and then depressing the knob to initiate the procedure. In this example, the PEEPi is 0.4 cm H_2O pressure and represents a volume of 155 mL (0.155L) of gas.

Determine the P_{aw} -plateau on the prior ventilator settings as well as the PEEP. Then start APRV using a long time at the high CPAP level; APRV is found under the mode settings menu, and the settings are manipulated in the same fashion as using the other modes on the E_4 (touch, rotate knob, push to accept). If the prior P_{aw} -plateau was 28, then start with P_{aw} -high at 28 cm H_2O pressure. The length of time at that pressure is T_{high} ; start at approximately 6.0 seconds (<4.5 seconds is not APRV by definition). Next, set the P_{aw} -low at 0, and the release time (T_{low}) at 0.8 seconds. Keep the FIO_2 at the same settings as in the prior mode. Leave the rise time at 0.20 (default setting), and activate automatic tube compensation (compensates for intrinsic tubing resistance much like PSV). These are approximate settings that need to be adjusted based on the pressure-over-time curve, the release volumes, and the patient's response.

First, recall that the time for gas flow is quite long by comparison with cyclic ventilation. Thus, release volumes may be smaller than one would desire for a V_T on conventional ventilation. Oxygenation will be enhanced despite the small volumes due to the efficiency of exchange based on exceptional alveolar recruitment. Adjusting the pressure to which the airway pressure is allowed to drop at the end of the release phase may be done in one of two ways: setting a P_{aw} -low or adjusting the T_{low} to achieve a trace that drops to ? of the amplitude of the P_{aw} -high (see Fig. 8-6). Either method is successful. If the release volumes are <5 mL/kg body weight, the authors advocate increasing the P_{aw} -high as the initial maneuver up to a maximum of 35 cm H_2O pressure. The authors have increased pressure beyond this in an awake patient who could report discomfort with no untoward consequences, but would recommend further increases in the T_{high} before exceeding 35 cm H_2O pressure.

Next, check the intrinsic PEEP to see if the PEEPi achieved equals at least the PEEP set on the prior mode; generally, the PEEPi will be greater than the prior PEEP, mandating an evaluation of blood pressure. PEEPi is found using a special procedure button on the right of the ventilator; press start procedure to obtain the PEEPi as well as the trapped volume that has generated the intrinsic PEEP. The screen will enter freeze mode after making the measurement; remember to unfreeze it by depressing the freeze button on the right of the ventilator faceplate (Fig. 8-22).

Typically, the patient's pO_2 and SaO_2 will rise rapidly (within a few minutes of starting the mode).[19] Failure to do so has been associated with endotracheal tube malfunction, pneumothorax, and, most commonly, inadequate pulmonary blood flow associated with intravascular volume depletion or inadequate cardiac output. [19] Weaning from ARPV is quite simple once the proximate cause of the hypoxemia has been corrected. The authors simply decrease the P_{aw} -high by 2 cm H_2O pressure every 2 hours until the P_{aw} -high = 10 cm H_2O pressure. If the patient is comfortable, well saturated, on minimal pressor support, and has manageable secretions coupled with an acceptable mental status, then endotracheal tube removal is appropriate.

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Chapter 9 - Thoracentesis

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Ana Ibrado

The term *thoracentesis* is derived from the Greek *thorakos* (chest) and *kentesis* (to pierce). Classically, this refers to any maneuver whereby a sharp object introduces a conduit between the intrathoracic cavity and the atmosphere, allowing air or fluid to exit. The clinical presentation and management of pneumothorax is covered in Chapter 10 . In contrast, this current chapter concentrates on the identification and management of fluid accumulations in the chests of patients who present to the emergency department (ED).

BACKGROUND

Thoracentesis was first described by Hippocrates as a procedure to relieve disease and potentially cause demise. [1] Various approaches and instruments for piercing the chest have since been developed. In the mid-1800s, Boerhaave introduced a flexible tube designed to evacuate a hemothorax. [2] In 1852, Bowditch described thoracentesis for the first time in American medical literature, coining the term "paracentesis thoracis." [3] Shortly thereafter, Hunter began using the newly designed hypodermic needle as an instrument for thoracentesis.[2] Hewett introduced the technique for continuous aspiration in 1876. [4] The 20th century forced the discovery of antiseptics and sterile techniques to further improve outcomes from chest drainage.

Advances in science also enabled a better understanding of the structures surrounding the lungs and the mechanisms by which disease processes result in fluid accumulation in the thorax. Characterizations of these collections have changed from observation of turbidity to a host of chemical markers, histologic markers, and bacterial stains and cultures. Furthermore, identifying the complexity of these fluid collections has been accompanied by increasing use of advanced radiologic techniques from plain films to magnetic resonance imaging (MRI). Together, these advances provide the ED clinician with an ordered approach to the patient with indications for thoracentesis.

ANATOMY AND PHYSIOLOGY OF THE PLEURAL SPACE

During embryologic development, lung buds grow out of a median mass of mesenchymal tissue into future thoracic cavities. This maneuver creates two linings: the visceral pleura, which wraps the lungs; and the parietal pleura, which lines the inner surface of the thorax and diaphragm and meets the visceral pleura at the root of the lungs in the mediastinum. The space between the two linings is called the pleural space. [5]

Understanding basic anatomy is important as it underlines the similarities and differences between the two linings, which in turn determine the physiology and pathophysiology of the pleural space. Both visceral and parietal pleura are thin layers of connective tissue, each embedded with capillary beds that generate both hydrostatic and oncotic pressures. The differences in these pressures from species to species have been the subject of intense study. While the majority of pulmonology research supports the belief that fluid flows from the parietal pleura and is absorbed in the hydrostatically permissive visceral pleura, there is also evidence supporting the concept that both tissues have a net fluid flow into the pleural space, and that this fluid is removed in bulk by the lymphatic system.

A schematic of the relationship between parietal and visceral pleura, as well as the diaphragm and cardiovascular/lymphatic systems is shown in Figure 9-1 .

The visceral pleura is supplied by the bronchial arteries and empties into the pulmonary veins. In this system, the hydrostatic water pressure (H_2O) in the capillary beds, which leads to fluid movement out of capillaries, is measured at about 24 cm H_2O, while the hydrostatic pressure within the pleura generated by the negative pleural pressure is -5 cm H_2O. This generates a total capillary outflow pressure of 24 - (-5 cm), or 29 cm H_2O. The opposing pressure, that which keeps fluids in the capillaries, is the oncotic pressure, measured at about 34 cm H_2O. The small amount of fluid in the pleural space also carries enough protein to generate an oncotic pressure of 5 cm H_2O. This generates a total capillary inflow pressure (opposing force) of 34 - 5 cm, or 29 cm H_2O. Total capillary inflow pressure (29 cm H_2O) minus total capillary outflow pressure (29 cm H_2O) equals a net fluid gradient of virtually zero. [6] This well-matched pressure system within the visceral pleura supports the idea that fluid generally does not flow out of, and may even be absorbed by, the visceral pleura in the healthy state. However, the visceral pleura wraps the lungs, making it directly susceptible to

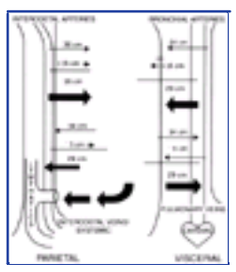

Figure 9-1 Schematic of relationships between the parietal and visceral pleura.

diseases within the lung parenchyma as well as fluid accumulation in the alveoli during pulmonary edema. Furthermore, because the pulmonary veins drain into the left ventricle, cardiac dysfunctions that result in increased left ventricular failure also affect fluid flow across the visceral pleura.

In contrast, the parietal pleura is supplied by the intercostal arteries and empties into the intercostal veins. Intercostal circulation reflects systemic pressure and naturally carries a higher hydrostatic pressure than the pulmonary vascular bed. In humans, this hydrostatic pressure is measured at about 30 cm H_2O. The parietal pleura is exposed to the same negative intrapleural pressure of -5 cm H_2O. Therefore, the net capillary outflow pressure from the parietal pleura is 30 - (-5 cm), or 35 cm H_2O. As plasma oncotic pressure is similar in both capillary beds, the opposing oncotic pressure in the parietal pleura is also around 29 cm H_2O. Therefore, total capillary inflow pressure (35 cm H_2O) minus total capillary outflow pressure (29 cm H_2O) generates a net gradient of about 6 cm H_2O from the parietal pleura into the pleural space in the healthy state. As a result, proper functioning of all these structures allows for a small amount of fluid, estimated at 0.26 mL/kg body mass, separating visceral from parietal pleura at any time. [7]

While it makes sense that this small amount of separation facilitates lung movement, obliteration of this space, as in the case of pleural symphysis, does not result in significant abnormalities in lung function. [8] [9] A more recent proposition for the role of the pleural space is to allow for pulmonary edema to escape the lung and decrease its effects on lung function. Fluid accumulated in the lung interstitium has been shown to flow quickly into the pleural space, where its effect on pulmonary function is minimal, while preserving oxygen exchange in the lung parenchyma. [10]

Pleural fluid generated over time is removed in bulk from the pleural space by the lymphatic system. The parietal pleura is embedded with large lymphatic stoma. These conduits allow particles as large as 10 MU to pass and drain the pleural space fluid in bulk through the lymphatic sinuses and into the mediastinal lymph nodes. Over the course of a day the lymphatic system maintains a pleural space outflow of 0.1 mL/kg per hour minimum, with a 30-fold capacity of 3 mL/kg per hour. [11] [12]

From this design, it is clear how various disease states can result in a pleural effusion simply by increasing the volume of fluid movement into the pleural space beyond the capacity of the lymphatic draining system or by decreasing the efficiency of the lymphatic draining system. Increasing the intravascular hydrostatic pressure or decreasing the intravascular oncotic pressure can increase fluid movement into the pleural space. Inflammatory, ischemic, or histologically destructive processes may compromise the lymphatic drainage, leading to an increase in pleural fluid.

THE ETIOLOGY OF PLEURAL EFFUSIONS

Pleural effusions are either transudates or exudates (Table 9-1). Distinguishing between transudates and exudates narrows the differential diagnosis and directs management and therapy. Generally, transudates are straw-colored and serous in appearance. Although they can be blood tinged, they have very low cellular and protein content. This is expected as transudates result from high intracapillary hydrostatic pressures and fluid

TABLE 9-1 -- Etiologies of Pleural Effusion

Transudates	
Most common	Congestive heart failure
	Cirrhosis
Less common	Nephrotic syndrome
	Peritoneal dialysis
	Pulmonary infarct (embolus)
	Superior vena cava obstruction
	Myxdema
	Meig syndrome
	Acute atelectasis
Exudates	
Most common	Bacterial pneumonia
	Malignancy
	Pulmonary infarct (embolus)
	Trauma
Less common	Other pneumonias
	Collagen vascular disease
	Pancreatitis
	Drug reactions
	Chylothorax
	Subphrenic and hepatic abscess
	Asbestosis
	Mesotheliomas
	Congestive heart failure after diuretic treatment
	Dressler syndrome
	Meig syndrome
	Esophageal rupture
	Viral pleuritis
	Uremia
	Urinothorax
Either exudates or transudates	Pulmonary embolism
	Meig syndrome

shifts to achieve equilibrium. In contrast, exudates result from either increased capillary permeability or destruction of barrier tissues, and generally contain high levels of protein and cellular components.

Transudates: Overwhelming the System

The most common cause of transudates is congestive heart failure (CHF). Left ventricular dysfunction leads to increased hydrostatic pressure in the pulmonary veins, resulting in an increased movement of fluid into the pleural space. When the increased fluid volume in the pleural space exceeds the lymphatic capacity for drainage, a pleural effusion develops. Additionally, the increased systemic hypertension associated with CHF also increases fluid flow from the parietal pleura and decreases lymphatic flow out of the thorax.[6] [13] Any process that results in compromised left ventricular output can result in a pleural effusion, including myocardial infarction, pericarditis, constrictive cardiomyopathy, and pericardial effusion from various disease processes.

Patients with cirrhosis are frequently hypoalbuminemic, leading to a chronic state of decreased plasma oncotic pressure. The imbalance between the hydrostatic and oncotic forces in the pleural space results in an effusion. [14] Alternatively, in normal situations, an intact parietal pleura over the stretchable diaphragm prevents the influx of intraperitoneal fluid into the thoracic cavity. Experiments have shown that high volumes of ascites can stretch the diaphragm enough to allow fluid to pass through preexisting

microdefects. In these cases, chest tube placement reduces both the pleural effusion and the ascites. Similarly, removal of the ascitic state also prevents the resulting effusion.[15]

The same mechanism is thought to explain acute hydrothorax, which occurs in approximately 10% of patients on continuous ambulatory peritoneal dialysis (CAPD). This occurs within the first several months after the start of CAPD. The transdiaphragmatic communications can close spontaneously on relief of pressure and volume, although recurrent episodes usually require surgical correction and chemical pleurodesis. [16]

Nephrotic syndromes also lead to protein loss and an imbalance between hydrostatic and oncotic pressures, which may result in pleural effusion. An important disease process to consider in patients presenting with nephrotic syndrome and pleural effusion, however, is pulmonary embolism (PE). [17] Patients with nephrotic syndromes have an increased incidence of thromboembolic events, which can also cause pleural effusion. A steadfast search for PE as the reason for effusion is

therefore warranted.

Superior vena caval obstruction is more common in neonates, in whom as much as 200 mL/kg per day of pleural fluid production is seen. [18] The accumulation is believed to result from obstruction of lymphatic drainage into the venous system. In adults, however, pleural effusions are not as common with superior vena caval syndromes. Therefore, in these cases, concurrent lymphatic obstruction by malignant disease should be strongly considered.

Exudates: Pathology of Tissues, Destroying the System

The main mechanism of cancer-related pleural effusions is obstruction. First, by direct metastasis to the parietal pleura, malignant tissues either block or destroy lymph stoma, thereby preventing outflow. Second, intrathoracic neoplasms, (e.g., lung cancer, metastatic breast cancer and lymphoma), involve mediastinal lymph nodes; thereby decreasing outflow. Bronchogenic carcinoma can obstruct a mainstem bronchus, causing the remaining lung to expand or, more likely, the hemithorax to collapse, thereby increasing negative pleural pressure and pulling fluid into the pleural space. Because these are mechanical entities that overwhelm the system, the protein levels in these effusions are low like transudates. Malignant effusions, however, have high lactate dehydrogenase (LDH) levels, fulfilling Light's criteria as an exudate. Other less common mechanisms involving neoplasm include: metastasis to the visceral pleura; increasing capillary permeability; and obstructing the thoracic duct, thereby creating a chylothorax. [19]

Bacterial pneumonia affects about 4 million Americans a year, 20% of whom require hospitalization. Of those hospitalized, 40% develop effusions. [20] A pleural effusion associated with bacterial pneumonia, lung abscess, or bronchiectasis is a parapneumonic effusion. [21] Empyema, on the other hand, is defined as pus in the pleural space, with or without an associated pneumonic process. [22] In the first stage of the parapneumonic effusion, called the exudative stage, fluid flows from the capillaries of the visceral pleura secondary to increased permeability rendered by the surrounding inflammation or pneumonitis. In the second, fibropurulent stage, a large accumulation of fluid containing cells, bacteria, cellular debris, and fibrin deposits on both the visceral and parietal pleura. This leads to formation of loculations. Loculations contain the empyema, preventing it from spreading, but also make it very hard to drain even with large chest tubes. In the last, organizational stage, fibroblasts grow from both pleural and form a tough peel that encases the lung and renders it functionless. In the worst cases, the empyema will drain spontaneously through the chest wall (*empyema necessitates*) or into the lung (*bronchopleural fistula*).[6] Numerous bacterial and fungal infections have been shown to result in effusions and fistulae. Fungal infections can begin in the pleural space, specifically postinstrumentation, and in the immunocompromised patient.

Nonpulmonary processes that give rise to empyemas include esophageal perforations, pneumothorax, subdiaphragmatic infections, septicemia, and manipulation or instrumentation within the thoracic cavity.

Pleural surfaces, in patients with rheumatoid arthritis with pleural effusions are inflamed and thickened, sometimes with vesicles and granules, all of which increase permeability and result in an effusion characteristically low in glucose and high in rheumatoid factor. [23] Systemic lupus erythematosus (SLE) and drug-induced lupus pleuritis, in contrast, have high glucose levels and lower LDH levels. Drugs known to have lupus-like syndromes include hydralazine, procainamide, isoniazid, phenytoin, and chlorpromazine. The following drugs reportedly cause pleuropulmonary reactions and pleural thickening and effusions rich in eosinophils: nitrofurantoin, dantrolene, methylsergide, and bromocriptine. Amiodarone also causes severe pulmonary toxicity and pleural effusions. Other disease processes that compromise the pleura include mesotheliomas, asbestosis, tuberculous pleuritis, viral infections, and fungal infections that primarily affect the pleural space (e.g., pleural aspergillosis, blastomycosis, and coccidioidomycosis). [24]

Entamoeba hystolytica trophozoites form abscesses in the liver. The abscesses can cause effusion by diaphragmatic irritation and the resulting sympathetic effusion. More dramatically, these abscesses can rupture through the diaphragm and result in a unique pleural fluid often described as "chocolate sauce" or "anchovy paste," which is rich in cytolytic enzymes, blood, and liver tissue. These are large effusions accompanied by sudden, severe pain; respiratory distress; and occasionally shock.[25] *Echinococcus granulosus* is a tapeworm that also forms pulmonary, splenic, and hepatic hydatid cysts, and presents with the same dramatic rupture as Entamoeba in about 5% of cases. When rupture occurs, however, 50% of the time it is accompanied by simultaneous rupture into the tracheobronchial tree. These patients cough up large amounts of pus and cysts. Echinococcal scolioces and hooklets are also abundant in the pleural fluid. [26]

Pancreatitis can present as either acute pancreatitis, chronic pancreatitis with pseudocyst, or pancreatic ascites. Pleural effusions due to acute pancreatitis are sympathetic effusions and usually on the left side. Effusions in pancreatic ascites, as with all other ascites, result from transfer of ascitic fluid through diaphragmatic defects and are characterized by extremely high amylase levels. [27] In contrast, pleural effusions in chronic pancreatitis with pseudocyst result from the development of sinus tracts between the pancreas and the pleura, usually through the aortic or esophageal hiatus, known as the pancreaticopleural fistula. This third mechanism leads to massive chronic pleural effusions, which reaccumulate rapidly. [28]

Yellow Nail Syndrome is the triad of deformed yellow nails, lymphedema, and pleural effusions. It is possible,

however, that each finding occurs at a different stage in the patient's life, making the presence of all three unlikely. The underlying defect is hypoplastic lymphatic vessels.[6]

Entities of Both Transudate and Exudate Nature

Of the 500,000 persons that have a PE each year, at least 30% develop a pleural effusion. [29] Therefore, when the etiology for the effusion is unclear, strongly consider a PE. PEs cause effusion by obstruction of the pulmonary vessels, leading to right-sided failure and an increased hydrostatic pressure in the parietal pleura. In this case, the fluid is transudative. Alternatively, when blood supply to pulmonary vasculature is removed, the parenchyma distal to this point undergoes ischemia and infarcts, leading to increased capillary permeability in the visceral pleura. This results in an exudative pleural effusion. Meig syndrome is the triad of ascites, pleural effusion, and benign ovarian tumor. An exudative pleural effusion is believed to result from the movement of ascitic fluid through diaphragmatic pores or defects. In addition, high systemic pressures can also generate a transudative effusion through increased fluid movement from the parietal pleura. [30]

Traumatic Injuries

Esophageal rupture can result from forceful vomiting as in the case of Boerhaave syndrome, or from instrumentation as in the case of endoscopy, Blakemore-Sengstaken tube placement, or rigid nasogastric tube placement. Hemothorax can result from sharp traumatic injuries, subclavian vein or artery cannulation, pulmonary embolism, aortic aneurysm, and supratherapeutic levels of anticoagulant. Chylothorax develops from acute disruption of the thoracic duct, usually in the setting of trauma. These are usually large-volume effusions that accumulate over an extremely short period of time, rapidly compromising both oxygenation and circulation.

DIAGNOSIS OF PLEURAL EFFUSION

Clinical Diagnosis

The three most common symptoms related to pleural effusions are chest pain, cough, and dyspnea. Parietal pleurae have nerve fibers; therefore, pleuritic chest pain is more indicative of localized irritation of the parietal pleura. Pain can often be referred to the abdomen via innervation of the intercostals. Chest pain solely from fluid collection is a dull ache. Involvement of the mediastinal pleura, because of innervation of the phrenic nerve, results in chest pain with ipsilateral shoulder pain. Lower chest pleuritic pain with shoulder pain is pathognomonic for diaphragm involvement. Significant fluid accumulation compresses the lung parenchyma, causing the opposing bronchial walls to touch, thereby stimulating a cough reflex. [6] Patients with large effusions have surprisingly acceptable arterial blood gases and evacuation of large effusions does not necessarily dramatically improve dyspnea. [6]

Because pleural effusions result from many disease processes, physical examination of the patient should involve all systems in search for other signs of a suspected disease. Lung examinations should be conducted to evaluate lung pathology and determine the extent of the effusion. While most patients will have equal hemithoraces and normal intercostal spaces, taking a few minutes to observe these is actually useful in determining action. Large effusions can result in increased hemithorax and bulging intercostal spaces on the side of the effusion. On palpation, tactile fremitus is either reduced or completely absent over the effusion, as fluid separates the lung from the thoracic wall and absorbs vibrations from the lung. Percussion over the effusion produces a characteristic dullness, which shifts when the patient changes position if the fluid is free-flowing. In general, auscultation reveals decreased to absent breath sounds depending on the size of the effusion. Pleural rubs may be appreciated if there is pleural irritation, but are often difficult to auscultate until after fluid evacuation. Palpation, percussion, and auscultation are all useful in determining the top of the effusion. However, depending on the noise level in the ED, the suspicion of complicated fluid collections, or in the patient with prior multiple thoracic instrumentations, radiologic studies can be very useful.

Radiologic Diagnosis

Understanding the appearance of effusions on plain chest x-rays requires three key concepts: (1) Effusions, when free-flowing, follow the force of gravity and accumulate in the most dependent parts of the thoracic cavity. (2) Lungs recoil proportionately when they collapse secondary to elastic recoil. This means that a collapsed lung will retain its original shape, although smaller, and float on the fluid. (3) Fluid layers evenly anterior to posterior in the upright position.

Underlying air and mass densities will affect the appearance of fluid on x-ray and must be considered when reviewing chest radiographs. [31]

On a chest x-ray, the most easily recognized abnormality that relates to a pleural effusion is the blunting of the lateral costophrenic angle on a posterior-anterior (PA) or anterior-posterior (AP) view (Fig. 9-2). At least 175 mL are needed to cause blunting, but as much as 500 mL of pleural effusion may be present without blunting.[32] [33] On a PA radiograph, pleural fluid appears like a meniscus, curving downward toward the mediastinum and higher laterally against the lateral wall in the PA view. Similarly, in the lateral view, the fluid level appears "lowest" midway through the thoracic cavity and highest at the anterior and posterior margins. In both cases, the true height of the pleural effusion coincides with the highest point on the meniscus. Pleural fluid can be encapsulated by tissue adhesions in parietal, visceral, or interlobar surfaces within the thoracic cavity. Because these adhesions anchor the fluid collection, loculated effusions are often described as D-shaped (Fig. 9-3) and are best diagnosed with either ultrasound or computed tomography (CT) (see later).

In subpulmonic effusion, the fluid remains infrapulmonary and does not cause blunting of either the lateral or posterior costophrenic angles on the upright radiograph. As much as 1000 mL of fluid may be present. The following features should raise suspicion for a subpulmonic effusion on the upright radiograph (Fig. 9-4): (1) an elevated hemidiaphragm; (2) the elevated hemidiaphragm peaks more laterally than expected, with a steep lateral slope; (3) pulmonary vessels are not clearly visible below the surface of the hemidiaphragm on lateral projection; (4) flat appearance of the

Figure 9-2 Right-sided pleural effusion. Posterior-anterior (PA) radiograph demonstrates blunting of right costophrenic angle. Both PA and lateral radiographs demonstrate the appearance of a meniscus.

posterior aspect of the hemidiaphragm on lateral projection. [34] A lateral decubitus view of this patient will show free-flowing pleural fluid parallel to the x-ray table.

Bilateral decubitus radiographs should be obtained when a pleural effusion is seen. A true simple pleural effusion will follow gravity and layer in one level below the floating lung. Unusual shapes reflect the presence of loculations, contained abscesses, and masses. The second view, a lateral decubitus view on the opposite side, draws the fluid toward the mediastinum and allows visualization of the lung parenchyma to determine the presence of infiltrates or masses (Fig. 9-5).

In the case of a massive pleural effusion, the entire hemithorax is opacified (Fig. 9-6). In such films, identification of mediastinal shift is a key to identifying the underlying process. In the absence of a diseased lung or mediastinum, large fluid accumulations should push the mediastinum contralaterally. When the mediastinum shifts toward the effusion, lungs and mainstem bronchi are likely diseased or obstructed, or both. When the mediastinum is fixed midline, it is likely to be invaded by a tumor.[34] [35] Differentiation of these disease processes is best done with CT.

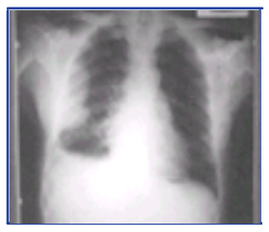

Figure 9-3 Loculated pleural effusion. This posterior-anterior radiograph demonstrates the D-shaped appearance of a right-sided loculated pleural effusion.

CT is more sensitive than plain film in detecting very small amounts of effusion and can readily assess the extent, number, and location of loculated pleural effusions. Loculated lesions can appear vague on plain film. In the distinct anatomical relationships shown on cross-sectional views in CT, free-flowing pleural fluid will form a sickle shape in the most dependent regions (Fig. 9-7) while loculated fluid collections will remain lenticular and relatively fixed in space. Additionally, CT assesses pleural thickening, irregularities, and masses that are suggestive of malignancy and other diseases which result in exudative effusions. With IV contrast dye, CT differentiates lung parenchymal disease such as lung abscess, which may be confused with empyema. [36] CT is also useful in the identification of mediastinal pathology. Finally, CT may also be useful in differentiating ascites from pleural effusion, which can be difficult to differentiate on plain radiographs. On CT, pleural effusions displace the diaphragmatic crus (displaced crus sign). They also obliterate the interface between liver and fluid (interface sign) and the bare area, a potential space behind the liver accessible only to pleural fluid (bare area sign) (Fig. 9-8).[37] [38] [39]

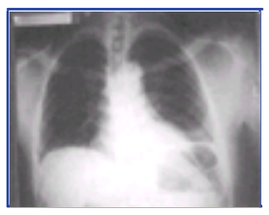

Figure 9-4 Subpulmonic effusion. This posterior-anterior radiograph demonstrates an elevated left hemidiaphragm that peaks more laterally than expected. A decubitus radiograph in the same patient demonstrates the free-flowing fluid.

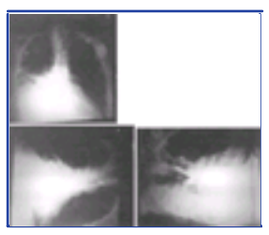

Figure 9-5 Bilateral decubitus radiographs. This patient's right-sided pleural effusion appeared on the posterior-anterior radiograph. Bilateral decubitus views demonstrate the free-flowing nature of the effusion and allow visualization of the lung parenchyma.

Ultrasound is advantageous for assessment of pleural effusions because it is easy and noninvasive. It can also be performed at the bedside. Although some details can only be seen with CT, ultrasound can identify fluid loculations, separate fluid from pleural thickening, and distinguish solid from pleural effusions especially in fluid collections that look solid in

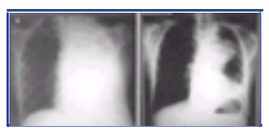

Figure 9-6 Massive pleural effusion. A repeat radiograph after thoracentesis demonstrates underlying mass.

freeze-frame. Real-time Doppler ultrasound may be more useful, as gray-scale ultrasound has a false-positive rate of 10%. [40] Ultrasound can also be used to identify both pulmonic and abdominal etiologies of the pleural effusion (Fig. 9-9).[39] [40] [41]

MRI has limited use in evaluating pleural effusions. In extreme cases where complex loculations hinder accurate

Figure 9-7 Pleural effusion. Sickle appearance of a free-flowing pleural effusion on computed tomography scan.

localization of fluid collections, the multiplanar ability of the MRI may provide some advantage. MRI is superior to CT in determining diaphragmatic injury and is safe and noninvasive.[34] [42] [43]

INDICATIONS

Thoracentesis is performed for either diagnostic or therapeutic purposes. Diagnostic thoracentesis evaluates the cause of a pleural effusion and requires removal of 50 to 100 mL of pleural fluid for laboratory studies. Most new effusions require diagnostic thoracentesis, an exception being a new pleural effusion with a clear clinical diagnosis (e.g., CHF) with no evidence for superimposed pleural space infection. The usual goal of therapeutic thoracentesis is to help relieve the dyspnea associated with a large pleural effusion, but it may also aid the radiologic work-up of a patient with a large effusion. Therapeutic thoracentesis typically requires removing a much larger volume of pleural fluid.

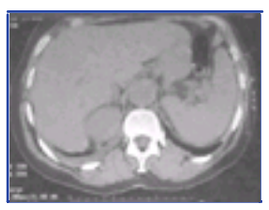

Figure 9-8 The bare area sign. A computed tomography scan demonstrates the obliteration of the potential space behind the liver with pleural fluid.

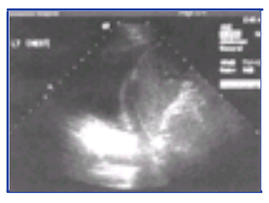

Figure 9-9 Pleural effusion. Ultrasound demonstrates the appearance of pleural fluid.

CONTRAINDICATIONS

There are no absolute contraindications to thoracentesis. It is generally recommended that patients with severe clotting abnormalities, including a platelet count <50,000 or prothrombin time or partial thromboblastin elevations of greater than twice the normal range, have platelet or factor replacement, or both, before the procedure. Patients with mild to moderate abnormalities do not require replacement before the procedure. [44] All patients with coagulation abnormalities, including those with renal failure, should be watched closely for bleeding following the procedure. Another relative contraindication is cellulitis or herpes zoster at the needle puncture site. One should attempt an alternative patient position to avoid skin puncture through these skin lesions, when possible. Extreme caution should also be exercised when performing thoracentesis on patients who are undergoing mechanical or manual ventilation.

PROCEDURE

Thoracentesis is generally an elective procedure, before which informed consent should be obtained and documented according to hospital policy. Because the procedure performed by experienced hands has a lower incidence of complications, [45] appropriate supervision by a person knowledgeable in the procedure should occur when possible. Sterile techniques should be followed through the entire procedure to avoid infection.

CHOOSING A TECHNIQUE

Several techniques for performing thoracentesis are possible (Fig. 9-10). The needle technique uses a simple 20- or 22-gauge (ga) needle. The needle catheter technique uses a plastic- or Teflon-coated catheter, which is inserted over or through a needle and subsequently left in the pleural space during fluid removal. Needle-catheters are commonly marketed and sold as preassembled thoracentesis kits. Two advantages of needle-catheter systems are: (1) a one-way valve that prevents air entry into the catheter during fluid

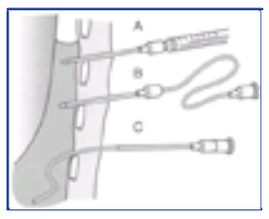

Figure 9-10 Techniques for through-the-needle catheter thoracentesis. Separate intercostal spaces depict the steps. *A,* Needle placement into pleural space. *B,* Catheter insertion through needle. *C*, Withdrawal of needle after catheter advancement.

collection and (2) an available blunt spring-loaded safety cannula that extends beyond the sharp needle tip once the pleural space is entered, protecting the lung from puncture or laceration.

Studies to determine the relative safety of needle and needle-catheter systems have had varied results. [46] [47] [48] It is generally recommended that the smallest possible needle be used. With diagnostic procedures, where only small volumes of fluid are being withdrawn, the needle technique is recommended. In therapeutic maneuvers, either technique is generally believed to be safe. The needle catheter approach avoids prolonged insertion of a needle in the pleural space while large volumes of fluid are removed.

For small or loculated effusions, suspicion for adhesions, the presence of relative contraindications, and in cases where iatrogenic pneumothorax may cause significant respiratory compromise, as with severe underlying lung disease or mechanical ventilation, needle placement under ultrasound guidance may be preferable. Ultrasound-guided needle placement for thoracentesis is associated with a significantly lower rate of complications as compared to clinically guided needle placement.[47] [48] CT-guided needle placement has also been successful in draining small pleural effusions. [49]

EQUIPMENT

Table 9-2 lists the equipment needed for thoracentesis and fluid collection. As with any procedure, appropriate preparation is essential for success.

PATIENT AND EQUIPMENT PREPARATION

A patent intravenous line should be established before initiation of the procedure in most cases. A red-top specimen tube for serum protein and LDH can be drawn at the time of IV

TABLE 9-2 -- Equipment for Thoracentesis

Basic procedure kit
Sterile towel
Sterile drape with fenestration
Basin for prep solution
Syringe(s) (10- to 20-mL)
Needles (22- and 25-ga) for infiltration
Sterile gauze pads
Povidone iodine
Local anesthetic (e.g., 1% lidocaine)—10 mL
Heparin—1 mL (see text)
Atropine—available at bedside
Syringe (50- to 60-mL) for aspiration
Sterile drainage tubing
Three-way stopcock
Needle or needle catheter (depending on technique)
Hypodermic needle (18- to 22-ga, 1 ½- to 2-in)
Over-the-needle catheter (16- to 20-ga needle)
Through-the-needle catheter (14- to 18-ga needle)
Scalpel (for needle catheter technique only)
Sterile specimen bowl or vacutainer bottle
Containers for analysis
Iced blood gas syringe
Red-top specimen tube
Purple-top specimen tube
Sterile transport media for culture or 10-mL sterile container
5 red-top specimen tubes for cytology or 10- to 50-mL plain bottle
Sterile dressing

placement. Atropine should be immediately available in the case of a vasovagal reaction during the procedure. Oxygen saturation should be monitored by pulse-oximetry and supplemental oxygen should be given as needed.

The 50- to 60-mL collection syringe should be premoistened with 1 mL of heparin (100 U/mL) to keep the pleural fluid from clotting if the procedure is for diagnostic purposes. Using a sterile technique, the skin is prepped with antiseptic in a wide area around the thoracentesis site. Sterile towels or a sterile drape should be placed around the site.

TERMINATION OF PROCEDURE

It is necessary to understand the end points of a procedure before its initiation. The most common indication for termination of thoracentesis is removal of a desired volume of fluid. For diagnostic thoracentesis the procedure is terminated upon removal of 50 to 100 mL of fluid. For therapeutic thoracentesis the procedure is terminated upon relief of patient dyspnea, or when 1000 mL of fluid have been withdrawn. Larger volumes of fluid may be removed if monitoring of the pleural pressures occurs. Pressure monitoring is not standard in the ED and in emergent situations the procedure can be done without monitoring. When feasible, pleural pressure measurements can be obtained by using a U-shaped manometer. Once 1000 mL of fluid have been removed, pleural pressures should be measured every 200 mL for the second liter and every 100 mL thereafter.[50] The procedure should be terminated once the pleural pressure exceeds -20 mm Hg.[50] [51] This, in theory, will help avoid significantly negative pleural pressures, which

have been associated with both symptomatic hypovolemia and the potentially fatal complication of reexpansion pulmonary edema. The actual benefit of this technique in reducing reexpansions problems is not known. The procedure should also be terminated if aspiration of air occurs, indicating lung puncture or laceration, unless the needle is 20-ga or smaller, making significant pneumothorax unlikely. Finally, a change in patient symptoms, including abdominal pain and worsening shortness of breath, should raise the suspicion of a patient complication and halt the procedure.

INSERTION SITE AND PATIENT POSITION

Figure 9-11 illustrates patient positioning for thoracentesis. Upright positioning is the preferred technique for draining most pleural effusions. With this technique, the patient is positioned sitting erect on the edge of the bed with extended arms resting on a bedside table. If the effusion is sufficiently large, the patient should be allowed to lean forward slightly while supported by the bedside table. The height of the effusion is then located clinically by dullness to percussion and a decrease in tactile fremitus. Never rely on the chest radiograph to determine the level of effusion as the radiographic level changes with patient positioning and respiration. Locate and mark the posterior rib at least one interspace below the top of the effusion. In all cases, the lowest level recommended is the eighth posterior intercostal space. The thoracentesis site

Figure 9-11 Positioning of patient for drainage of pleural effusion. *A,* Upright positioning. The broken line indicates midscapular line. *B,* Lateral decubitus positioning. The broken line indicates posterior axillary line. *C,* Supine patient positioning. The broken line indicates mid-axillary line.

should be at this level in the midscapular or posterior axillary line. If the patient is too ill to sit upright, use the lateral decubitus position with the side of the effusion down and the back at the edge of the bed. The needle insertion site in this position is the posterior axillary line. Alternatively, the patient may be positioned supine, with head elevated as much as possible. The needle insertion site in this position is the mid-axillary line. For both of these alternative positions, the fluid level must be determined clinically by dullness to percussion and a decrease in tactile fremitus. The needle should not be placed lower than the eighth intercostal space.

ANESTHESIA AND PLEURAL FLUID LOCALIZATION

Using a 25-ga needle attached to a syringe containing 5 to 10 mL of 1% lidocaine or equivalent anesthetic, raise a skin wheal at the upper edge of the previously selected rib in the midscapular or posterior axillary line. The upper edge of the rib is used to avoid accidental trauma to the neurovascular bundle, which runs at the inferior margin of each rib. The 25-ga needle is withdrawn and a 3.75-cm 22-ga needle is attached to the syringe of local anesthetic. This needle can both deliver anesthetic along the track of the thoracentesis catheter and serve as a small finder needle to identify the depth of penetration needed, as well as the presence of fluid at that level. Insert the 22-ga needle through the wheal and

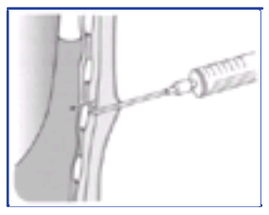

Figure 9-12 "Walking" the anesthetic needle over the superior aspect of the rib.

generously infiltrate the subcutaneous tissue and muscle down to and including the periosteum of the rib. With each 1 to 2 mm of needle advancement, the subcutaneous tissue and muscle are aspirated and then infiltrated with small alignots of the anesthetic. While the aspiration-infiltration process is continued, the needle is then "walked" above the superior edge of the rib and advanced through the intercostal space until the pleural space is entered (Fig. 9-12). Hold the needle perpendicular to the chest to avoid inadvertent trauma to the neurovascular bundle of the adjacent rib. Upon entering the pleural space, a pop may be felt. Aspirate fluid to ensure that the pleural space has been reached. Next, grasp the needle at the skin with the thumb and index finger and withdraw it. This allows for measurement of the proper depth of penetration needed during subsequent needle insertion. If no fluid is encountered, this is consistent with a "dry tap." A dry tap in the setting of a free-flowing pleural effusion indicates that either the needle is too short, or that the site chosen is too low or too high. A longer needle such as a spinal needle may be needed for a patient with significant subcutaneous tissue. If air bubbles are encountered, the lung parenchyma may have been entered, in which case the site is likely too high. If no fluid or air are encountered, the site is likely too low. If a dry tap occurs during fluid localization and the patient has no new symptoms, reevaluate the patient position and fluid level, administer local anesthetic as needed, and reattempt to aspirate fluid with the needle. If the repeat attempt is unsuccessful, obtain fluid under ultrasound guidance.

NEEDLE INSERTION TECHNIQUE

This technique uses a 3.75- to 5-cm, 20- or 22-ga needle. The needle and a 50- or 60-mL syringe are attached to a three-way stopcock. A drainage tube is attached to the stopcock. The lever of the stopcock is set to allow passage of fluid between the needle and the syringe. The depth of the pleural space as determined from the anesthetic/finder needle is now marked on the larger aspiration needle by gently grasping the needle with the index finder and thumb. The pleural space is again entered through the previous anesthetic site while gentle negative pressure is applied via the syringe. Attention to the indicated depth will prevent insertion of the needle farther than necessary, thus decreasing the chance of puncturing or lacerating the underlying lung. Fluid is first aspirated with the syringe. The stopcock lever is then turned to prevent entry of fluid back into the needle, and the fluid is expelled through the drainage tube into a sterile container or sterile vacuum bottle, where it can subsequently be transferred into appropriate specimen tubes. This process of aspirating and expelling the fluid is repeated until an adequate amount of fluid is obtained. Alternatively, fluid may be allowed to drain directly from the needle and three-way stopcock through high-pressure tubing into a vacuum bottle. The stopcock lever should be turned to prevent entry of air or fluid back into the needle when changing bottles.

Once an indication for discontinuing the procedure has been met, remove the catheter and cover the entry site with a sterile bandage.

OVER-THE-NEEDLE-CATHETER INSERTION TECHNIQUE

This technique uses an 8-French (Fr) catheter mounted on an 18-ga introducer needle. The 50- or 60-mL syringe and drainage tubing are attached to the three-way stopcock. The stopcock is adjusted to allow passage of fluid into the syringe and then this collection apparatus is temporarily set aside. The needle-catheter unit is attached to a 10- or 20-mL syringe. A small skin pierce with a scalpel at the selected insertion site is required to ease the entry of the catheter through the skin. The depth of the pleural space as determined from the anesthetic needle is now marked by gently grasping the needle-catheter unit with the index finger and thumb. The needle-catheter unit is advanced through the anesthetized area into the pleural space while constant, gentle, negative pressure is applied to the syringe. As fluid is encountered, the needle-catheter unit is angled slightly caudally. The catheter is then advanced into the pleural space while holding the needle steady. The needle is withdrawn and the exposed lumen of the catheter hub is covered with a finger to prevent the entry of air. The three-way stopcock with the attached 50- or 60-mL syringe and drainage tube is now attached to the catheter hub. Fluid is first aspirated with the syringe. The stopcock lever is then turned to prevent passage of fluid into the catheter, and the fluid is expelled through the drainage tube into a sterile container or sterile vacuum bottle, where it can subsequently be transferred into appropriate specimen tubes. This process of aspirating and expelling the fluid is repeated until an adequate amount of fluid is obtained. Alternatively, fluid may be allowed to drain directly from the needle and three-way stopcock through high-pressure tubing into a vacuum bottle. The stopcock lever should be turned to prevent entry of air or fluid back into the needle when changing bottles. If the catheter tip has multiple side ports for fluid entry, care must be taken to avoid withdrawing the catheter from the chest during fluid removal. This might expose a side port and allow air entry into the pleural space. Once an indication for discontinuing the procedure has been met, remove the catheter and cover the entry site with a sterile bandage.

THROUGH-THE-NEEDLE CATHETER TECHNIQUE

This procedure uses a 14- to 18-ga needle through which a flexible catheter is inserted into the pleural space. The catheter is withdrawn from the needle before the procedure. The catheter and drainage tubing are attached to the three-way stopcock and the stopcock is adjusted to close the catheter to the passage of air or fluid. This collection apparatus is temporarily set aside. The empty outer needle is attached to a 10- or 20-mL syringe. The depth of the pleural space as determined from the anesthetic needle is now marked by gently grasping the needle with the index finger and thumb. The needle is inserted into the pleural space through the anesthetized area while constant, gentle, negative pressure is applied via the syringe. Once fluid is encountered, the needle is held securely as the syringe is detached. The needle hub must be covered with a finger to prevent passage of air into the pleural space. The needle is then held securely and angled caudally as the catheter is inserted through the needle into the pleural space and advanced to the hilt of the needle or until resistance is encountered. The needle is withdrawn from the chest, leaving the catheter in the pleural space. The needle guard is then attached to the needle tip to prevent shearing off of the catheter. The catheter *must not be drawn back through the needle* because this may lacerate the catheter and allow its free entry into the pleural space. The catheter is then held securely to the chest wall without bending or kinking. The 50- or 60-mL syringe is now attached to the three-way stopcock. The stopcock lever is turned to allow passage of fluid into the syringe. Fluid is first aspirated into the syringe. The stopcock lever is then turned to prevent passage of fluid into the catheter, and the fluid is expelled through the drainage tube into a sterile container or sterile vacuum bottle, where it can subsequently be transferred into appropriate specimen tubes. This process of aspirating and expelling the fluid is repeated until an adequate amount of fluid is obtained. Alternatively, fluid may be allowed to drain directly from the needle and three-way stopcock through high-pressure tubing into a vacuum bottle. The stopcock lever should be turned to prevent entry of air or fluid back into the needle when changing bottles. If the catheter tip has multiple side ports for fluid entry, care must be taken to avoid withdrawing the catheter from the chest during fluid removal. This might expose a side port and allow air entry into the pleural space. Once an indication for terminating the procedure has been met, remove the catheter and cover the entry site with a sterile bandage.

PEDIATRIC PATIENTS

The indications and contraindications for performing thoracentesis are much the same in children as in adults. Positioning is also similar, but will likely require an assistant to help hold the patient and prevent patient movement. Sedation may be helpful when respiratory distress is minimal. The effusion level is again determined clinically by dullness to percussion and a decrease in tactile fremitus. The needle insertion site should not be lower than the eighth intercostal space in the posterior axillary line. Any technique as described for the adult patient may be used, but the smallest possible needle or needle-catheter is recommended.

POSTPROCEDURE RADIOGRAPH

In many centers chest radiographs are routinely obtained after thoracentesis to evaluate for procedure-related pneumothorax. This practice has been challenged and considered unnecessary as a routine procedure. The risk for pneumothorax following thoracentesis is low in patients who require a single needle pass, have no risk for adhesions, and have no new symptoms during or after thoracentesis. [52] Supporting this, studies have demonstrated that routine chest radiographs are not necessary after uncomplicated thoracentesis. [53] [54] A chest radiograph should be obtained in patients who require multiple needle passes, are at risk for adhesions, have aspiration of air, or develop any new symptoms during or after thoracentesis. Additionally, it is reasonable to obtain a postprocedure radiograph in patients who are at risk for future decompensation from expansion of a small asymptomatic pneumothorax, including those with severe underlying lung disease and who are receiving mechanical ventilation.

PLEURAL FLUID ANALYSIS

Pleural fluid analysis should occur in an organized and thoughtful pattern based on clinical suspicion for a disease process. Fluid examination in all cases begins with visual inspection. Effusions may range from clear to turbid. The presence of blood suggests trauma, malignancy, pulmonary infarct, or tuberculosis (TB). White or milky fluid suggests the presence of lipids, while purulent, malodorous fluid indicates empyema. Pleural effusion containing food particles is highly suggestive of esophageal rupture. The odor of urine suggests urinothorax. Once the general nature of the fluid has been investigated, selected testing is usually required. Table 9-3 indicates the proper collection volumes and containers to analyze for specific parameters. Table 9-4 lists common conditions and the associated pleural fluid findings.

The first step in pleural fluid evaluation in the laboratory is its categorization as exudative or transudative. In some cases it may be reasonable to hold off on further testing until this decision guides the clinician or is requested by a consultant. Given the practicality of the situation and the logistics of selective and progressive testing, some clinicians order a battery of screening tests based on the most logical etiology. As a general rule, collect samples in all appropriate containers at the time of the procedure should additional tests be required.

The pathophysiology of each category is discussed earlier in this chapter. Table 9-1 lists the common etiologies of transudates and exudates. Light et al. published criteria for separating transudates from exudates in 1972 based upon measurements of serum and pleural fluid protein and LDH. [55] The value for LDH was subsequently changed to accommodate variations in assay conditions. [56] These criteria have since become known as Light's criteria (Table 9-5). An elevated pleural protein occurs secondary to obstruction of lymphatic drainage or increased capillary permeability while elevations of LDH are believed to be due to pleural inflammation. Hence, elevations of protein or LDH are consistent with exudative effusions. Although more recent studies have failed to reproduce the exact sensitivity and specificity as described in Light's original study, modifications of Light's criteria have failed to improve overall accuracy of diagnosing exudates. [57] [58]

TABLE 9-3 -- Diagnostic Pleural Fluid Specimens

In All Cases:
6.5-mL plain (red-top) specimen tube
Lactate dehydrogenase (LDH)
Glucose
Protein
5-mL EDTA (lavender-top) specimen tube
Appearance
Color
Specific gravity
Cell counts
Differential counts
Crystals
If Exudate or Clinically Indicated:
6.5-mL plain (red-top) specimen tubes
Amylase
Triglycerides, cholesterol (lipoprotein electrophoresis)
Complement levels
Rheumatoid factor
Countercurrent immunoelectrophoresis (CIE)
Carcinoembryonic antigen (CEA)
10-mL sterile container
Gram stain
Aerobic cultures
Anaerobic cultures
Acid-fast bacilli culture and stain
Fungal culture and stain
10–50 mL
Cytology
5-mL heparinized (green-top) specimen tube
Lupus erythematosus cells
2-mL iced heparinized syringe
pH

EDTA = ethylene diamine tetra-acetic acid.

Other possible markers have not shown significant improvement in sensitivity and specificity over Light's criteria. [59] [60] [61] An exception to using Light's criteria for separating transudates from exudates may be in the setting of CHF. Effusions in CHF are due to increased capillary hydrostatic pressure and are therefore transudes. However, it has been shown that diuretic use increases the pleural fluid protein and LDH concentrations, making the fluid appear exudative by Light's criteria. [62] [63] This is hypothesized to be due to diuretic-induced shifting of fluid out of the pleural space. In the diuretictreated CHF population, therefore, measuring the serum-to-pleural effusion albumin gradient is recommended over Light's criteria. [60] [64] If the serum albumin minus the pleural fluid albumin is >1.2 g/dL, the patient likely has a transudative effusion and no further fluid analysis is necessary. Finally, with patients in whom no blood is obtained, a measured pleural fluid LDH >307 IU/L *or* a measured pleural fluid cholesterol >60 mg/dL, when used together, identifies exudates with reasonable accuracy. [59] [65]

Once a fluid is classified as transudative, it typically requires no further fluid analysis, and therapy is directed at the underlying cause of the effusion (CHF, nephrosis,

etc.).

In the presence of an undiagnosed exudative effusion, however, more extensive fluid evaluation is required. Clinical suspicion for an underlying disease process should guide additional fluid evaluation (Table 9-6), but all undiagnosed exudates, at a minimum, should have pleural fluid sent for cell count with differential, gram stain, culture, cytology, and glucose.

Exudates typically have a pleural fluid white blood cell (WBC) count of >1000 cells/mm ^{3} . Counts may reach levels >10,000 cells/mm^3 , most commonly with parapneumonic effusions.[66] The differential cell count can be useful in identifying the cause of an exudative pleural effusion. A predominance of PMNs indicates an acute process affecting the pleural surface, such as infection or pulmonary infarct. A predominance of mononuclear cells is consistent with a more chronic pleural process, including malignancy, TB, PE, and viral pleuritis. [29] If the differential lymphocyte count is >50% of the total WBC count, the effusion is considered lymphocytic. A lymphocytic pleural effusion is suspicious for TB, but may also be caused by cardiac bypass surgery, malignancy, chylothorax, and atypical infections.[67] Eosinophil counts of >10% are uncommon, but have traditionally been associated with blood or air in the pleural space.

If the fluid appears bloody, a fluid cell count and hematocrit should be obtained. In general, the presence or absence of red blood cells (RBCs) is not useful in determining the etiology of the effusion because it takes a minute amount of blood to cause a blood-tinged appearance. A grossly bloody pleural effusion or RBC count of >100,000 cells/mm^3 is suggestive of trauma, malignancy, or pulmonary infarction, [66] but a lack of RBCs does not exclude these diagnoses. Grossly bloody pleural fluid with a hematocrit of >50% of the peripheral hematocrit often requires tube thoracostomy.

The concentration of glucose in exudates is extremely variable and generally does not correlate with any specific disease process. Routine measurement of pleural fluid glucose for exudative effusion is recommended. A low glucose concentration (<60 mg/dL) narrows the differential diagnosis to empyema, ruptured esophagus, complicated parapneumonic infection, malignancy, and TB. [29] Keep in mind, however, that these diagnoses are not *excluded* by a high or normal pleural fluid glucose. Additionally, in exception to the rule described earlier, patients with active rheumatoid arthritis and rheumatoid pleural effusion will commonly have an extremely low pleural fluid glucose concentration (<20 to 30 mg/dL). [68] Comparatively, in systemic lupus erythematosus, pleural fluid glucose levels are usually normal. [69]

Evaluation for TB should occur if it is clinically suspected or if the pleural fluid is lymphocytic. Pleural fluid acid-fast bacillus staining and culture, which are infrequently positive, should be ordered. [70] Newer markers, including adenosine deaminase, polymerase chain reaction (PCR), and interferon gamma, have been found useful in the diagnosis of tuberculous pleural effusions. A pleural fluid adenosine deaminase level above 40 U/L is highly suggestive of TB. [67] [70] [71] Additionally, the presence of tuberculin sequences by PCR and elevations of gamma-interferon levels have shown increased sensitivity over pleural fluid staining and culture. [71] The availability of laboratory testing for these markers is currently limited.

Concerns about malignancy or the setting of an undiagnosed exudate are indications for cytologic evaluation. At least 50 mL of pleural fluid is required for this evaluation. Unfortunately, the initial pleural fluid cytology is often negative in malignant pleural effusions. [66] Repeated pleural samples increase the yield of malignant cells.

Pleural pH measurement provides useful information regarding pleural inflammation. Normal pleural fluid pH is approximately 7.64. Pleural fluid pH <7.3 indicates pleural inflammation. The differential diagnosis of pleural fluid acidosis includes not only empyema and complicated parapneumonic

TABLE 9-4 -- Diagnostic Features of Pleural Fluid

Pleural Fluid	Description	WBC Count	Predominant Leukocyte	Glucose	pH	Comments
Exudates						
Parapneumonic	Turbid	Elevated	P	Low	>7.3	If pH <7.2, may need thoracostomy tube; CIE may identify some aerobic bacteria
Empyema	Turbid, purulent	Elevated	P	Low	<7.3	Positive gram stain, culture; CIE may identify some aerobic bacteria
Tuberculosis	Straw color, serosanguinous	<10,000	M or P	Low	<7.4	Positive AFB stain, culture or pleural biopsy
Malignancy	Turbid, bloody	<10,000	M	Low	<7.3	Positive cytology, pleural biopsy, CEA
Pulmonary embolism or infarction	Straw color, bloody	Elevated	M or P	Serum	7.4	Hypoxia; positive lung scan
Collagen vascular disease	Turbid	Variable	M or P			Positive serum ANA, pleural complement
Rheumatoid arthritis	Green	Variable	M or P	Very low	<7.3	Elevated pleural rheumatoid factor
Systemic lupus erythematosus	Yellow	Variable	M or P	Serum	>7.3	Positive pleural LE cells
Hemothorax	Bloody	Variable	P	Serum	<7.3	Hematocrit >50% peripheral level
Chylothorax	White, cloudy	Variable	M			Elevated pleural triglycerides; chylomicrons present; positive Sudan III stain
Pancreatitis	Turbid, serosanguinous	Elevated	P	Serum	>7.3	Pleural amylase > serum
Esophageal rupture	Turbid, bloody	Elevated	P		<<7.3	Pleural amylase elevated
Transudates						
Congestive heart failure	Clear, straw color	<1000	M	Serum	7.4	Clinical features
Cirrhosis	Clear, straw color	<500	M	Serum	7.4	Clinical features
Nephrotic syndrome or hypoproteinemia	Clear, straw color	<1000	M	Serum	7.4	Clinical features
Transudate or exudate	Straw color to bloody			Low		Obstructive uropathy; pleural creatinine > serum
Urinothorax						

AFB, acid-fast bacilli; ANA, Antinuclear antibody; CEA, carcinoembryonic antigen; CIE, countercurrent immunoelectrophoresis; LE, lupus erythematosus; M, mononuclear cells (e.g., lymphocytes, macrophages, mesothelial and plasma cells); P, polymorphonuclear cells; WBC, white blood cell.

From Ross DS: Pleural effusion. In Harwood-Nuss AL, Linden CH, Luten RC, et al (eds): The Clinical Practice of Emergency Medicine. Philadelphia, JB Lippincott, 1990. Reproduced by permission.

TABLE 9-5 -- Light's Criteria

Pleural fluid is classified as an exudate if any one of the following three criteria are met:
Pleural fluid:serum protein ratio >0.5
Pleural fluid lactate dehydrogenase (LDH) >2/3 upper limit of serum reference range
Pleural fluid:serum LDH ratio >0.6

effusion, but also malignancy, TB, esophageal rupture, and collagen vascular disease. [72] Measurement of pleural pH is essential in the evaluation of suspected parapneumonic effusions since hydrogen ion concentration is a key factor in the management algorithm. The fluid must be collected anaerobically, but may be transferred from the initial 50- or 60-mL syringe into a heparinized blood gas syringe, [73] and then left at room temperature for up to 1 hour before laboratory analysis [74] without affecting the accuracy of the results. Because of these specifics regarding the collection and evaluation of pleural fluid pH, pleural fluid should routinely be transferred to a heparinized blood gas syringe and placed on ice while awaiting the decision for pH testing. The evaluation of a suspected parapneumonic effusion is described further as follows.

Pleural amylase evaluation should occur in the setting of suspected esophageal rupture or pancreatitis. [75] Elevations of pleural fluid amylase >100 U/L are seen in various cases including malignancy, post cardiac bypass surgery, esophageal rupture, pancreatitis, and ruptured ectopic pregnancy. Routine measurement of pleural fluid amylase levels rarely provides useful information.

White or milky fluid or concerns about thoracic duct injury should lead to analysis for pleural fluid triglycerides. Rarely, a longstanding benign effusion, such as one due to TB or rheumatoid disease, may have a similar gross appearance due to accumulation of cholesterol, lecithin, or globulin-rich fluid. This effusion is termed pseudochylothorax. Effusions with triglyceride concentrations of >110 mg/dL are uniformly chylous in nature. [76] The etiologies of chylous effusions include thoracic duct injury due to surgery, malignancy, trauma, and pleuritis.

Patients with suspected parapneumonic effusions warrant rapid evaluation and outcomes risk assessment based on pleural anatomy, pleural fluid bacteriology, and pleural fluid chemistry. [77] All parapneumonic effusions require at least

TABLE 9-6 -- Selective Evaluation of Exudates Based on Clinical Suspicion

Pleural Fluid Assay	Diagnosis Suspected
Amylase	Pancreatitis, esophageal rupture
Triglycerides	Chylothorax, intrathoracic TPN infusion
Glucose	Rheumatic effusion
Urea or creatinine	Urinothorax
Cytology	Malignancy
Albumin (with serum measurement)	CHF (after diuretics)
Adenosine deaminase, gamma interferon, PCR	TB (lymphocytic effusion)
Hematocrit	Hemothorax
pH	Parapneumonic effusion, empyema

CHF, congestive heart failure; PCR, polymerase chain reaction; TB, tuberculosis; TPN, total parenteral nutrition.

diagnostic thoracentesis with the goal of identifying complicated parapneumonic effusions. Treatment before the organizational stage is ideal. Patients at high risk for poor outcome require complete drainage of the pleural space. This includes patients with large or loculated effusions, pleural thickening on CT scanning (the pleural peel), aspiration of frank pus, and pleural fluid pH of <7.2.

COMPLICATIONS

The most frequently reported complication of thoracentesis is pneumothorax, which has a reported incidence of 4% to 19% in several studies. [45] [47] [52] Thoracostomy tubes were required in <50% of the post-thoracentesis pneumothoraces in each study. The mechanism for this complication is puncture of the lung or inadvertent air entry through the needle or catheter during the procedure. Pneumothorax should be suspected if there is aspiration of air during fluid removal or if the patient develops new symptoms during or after the procedure. Procedure-related factors contributing to pneumothorax include an inexperienced operator, therapeutic taps, and use of needles >20-ga. [45] [46] [48] [78] The risk of pneumothorax may be increased in patients with underlying chronic obstructive pulmonary disease. [79]

Cough precipitated during the procedure is another frequently encountered complication. Although typically considered a minor complication resulting in only patient discomfort, it may be associated with the creation of an iatrogenic pneumothorax. [78] [80] The procedure should be terminated if persistent patient coughing occurs.

Patient discomfort is reported frequently with pneumothorax. Adequate local anesthesia and judicious use of analgesics should eliminate this complication.

As with all procedures, there is potential for infection, estimated at 2%. The risk is kept low with proper attention to patient preparation and sterile technique.

Other serious complications that have been reported in <1% of procedures include: hemothorax, splenic rupture, abdominal hemorrhage, unilateral pulmonary edema, air embolism, and catheter fragment left in the pleural space. [78]

Hemothorax may be suspected by a rapid accumulation or reaccumulation of pleural fluid or by a change in patient vital signs following the procedure. Hemothorax may be due to laceration of the lung and diaphragmatic, intercostal, or internal mammary vessels. Careful attention to technique, such as avoiding the superior portion of the intercostal space, never puncturing medial to the midclavicular line, and not penetrating too deeply into the thorax during needle insertion, should be practiced. Hemothorax requires appropriate surgical consultation and drainage via a thoracostomy tube.

Puncture of the spleen or liver through the diaphragm may result in localized organ hematoma or hemoperitoneum. [80] Clinically this is suspected when the needle pass does not yield pleural fluid (dry tap) and is followed by a patient complaint of abdominal pain. If this diagnosis is suspected, appropriate resuscitation is the initial treatment, followed by a diagnostic imaging study, preferably CT scan. If the patient is hemodynamically unstable, bedside ultrasound and immediate surgical consultation should occur.

Inadvertent shearing of the plastic catheter may occur when a through-the-needle catheter technique is used. Securing the needle with a needle guard after it has been withdrawn from the chest prevents this complication. In addition,

the catheter should not be withdrawn through the needle at any time. Should a catheter be left in the chest, a method of percutaneous retrieval may be performed by radiology.

Postexpansion pulmonary edema is a rare and unexplained complication associated with rapid reexpansion of the lung. Symptoms include dyspnea, tachypnea, tachycardia, cough, and frothy sputum. [81] It is postulated that this can be avoided by monitoring the pleural pressures carefully after 1000 mL of fluid have been withdrawn and discontinuing the procedure when the pleural pressure are >20 mm Hg. [50] [51]

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Chapter 10 - Tube Thoracostomy

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John P. Mulligan

Tube thoracostomy is used to evacuate an abnormal accumulation in the pleural space. Conditions that may require treatment with a tube thoracostomy include pleural effusions, purulent fluid from an infection (empyema), or lymph fluid (chylothorax). Trauma is a common indication due to accumulations of blood or air.

The first modern methods to evacuate pleural contents were developed in the 19th century, but these techniques were not widespread until 1918, when they were used treat postinfluenza empyema. Military experience demonstrated that thoracic drainage combined with antiseptics and antibiotics reduced the mortality from thoracic trauma from 62.5% during the Civil War, to 24.6% in World War I, to 12% in World War II. [1]

PATHOPHYSIOLOGY

The pleural space is a potential space that separates the visceral and parietal pleurae with a thin layer of lubricating fluid. The parietal pleura covers the interior of the chest cavity, while the visceral pleura covers the lungs. They are both smooth, serous membranes. Normally there is a slight negative pressure in the pleural space that keeps the lung inflated. With normal inspiration, an increased negative intrathoracic pressure is developed in the chest cavity. This pressure is then transmitted to the pulmonary parenchyma, causing expansion of the lung from an influx of air into the lungs via the trachea. The addition of blood, fluid, or air in the pleural space interferes with normal inspiratory inflation, although the body can tolerate some violation of this space. Large amounts of fluid or air compromise respiratory function and induce symptoms of dyspnea, pleuritic chest pain, and anxiety. A progressive accumulation, such as occurs with a tension pneumothorax, leads to cardiovascular compromise in addition to severe respiratory dysfunction. The degree of respiratory compromise depends on the volume of pleural space occupied by the fluid or air, the baseline pulmonary status of the patient, and the integrity of the chest wall.

Pneumothorax

When the resting negative pressure that normally exists in the pleural space is lost, the lung will collapse. Air can enter the pleural space from the outside, as with a penetrating injury, or from the lung, as when a bleb ruptures. A pneumothorax is any collection of air in the pleural space, either as a result of trauma or as a spontaneous occurrence. Pneumothorax may complicate procedures such as transthoracic biopsies, thoracentesis, subclavian venous cannulation, or positive pressure ventilation. [2]

Spontaneous Pneumothorax (Fig. 10-1)

A spontaneous pneumothorax can be either "primary" or "secondary." A primary spontaneous pneumothorax results from the rupture of a subpleural bleb in a patient *without* other underlying lung disease. This is six times more common in men than women. The "typical" patient suffering a spontaneous pneumothorax is a tall, thin, 20- to 40-year-old male smoker. Symptoms can occur with rest or with activity. Common symptoms include the sudden onset of pleuritic chest pain and dyspnea. Occasionally pain is absent. Secondary spontaneous pneumothoraces are more common and occur in patients *with* underlying lung or pleural disease. The most common cause is emphysema, followed by chronic bronchitis, asthma, tuberculosis, pneumonia, bronchiectasis, atelectasis, pulmonary fibrosis, lymphoma, and a foreign body. Less common causes include pulmonary infarction, scleroderma, rupture of a hydatid cyst, and alpha $_1$ -antitrypsin deficiency. Neoplasm is a common cause in older patients. Patients with Marfan syndrome, acquired immunodeficiency syndrome, and *Pneumocystis carinii* pneumonia are also at increased risk. Morbidity and mortality increases for patients with underlying lung disease. Most patients will seek medical attention within 24 hours of onset. However, some patients can tolerate even a complete pneumothorax for surprisingly long periods of time. Certainly, many individuals will experience a small spontaneous pneumothorax, never seek medical attention, and the process will resolve spontaneously. Bilateral spontaneous pneumothoraces may occur in up to 10% of cases, but progression of a spontaneous pneumothorax to a tension pneumothorax is very rare.

Traumatic Open Pneumothorax

An open pneumothorax ("sucking chest wound") occurs when the chest wall is penetrated and the negative intrapleural pressure that keeps the lung expanded is lost because outside air enters the pleural space. With small defects a tension pneumothorax may develop because air enters, but does not exit, the pleural space with each inspiration. If the wound has a diameter larger than the trachea, air moves preferentially through the chest wall opening, rather than down the trachea, with each respiratory attempt. In this circumstance the lung will not expand and no ventilation will occur.

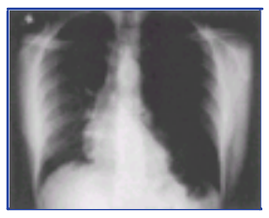

Figure 10-1 Simple pneumothorax, anteroposterior chest radiograph view. Note absence of lung marking on the affected side and deviation of the trachea contralaterally.

Traumatic Closed Pneumothorax

A closed pneumothorax may result from an injury or may occur spontaneously, but the chest wall remains intact. A pneumothorax following blunt chest trauma is usually due to a rib fracture that injures the lung and allows air into the pleural space. A closed pneumothorax can also occur without a rib fracture when an alveolus or bleb ruptures due to the abrupt increase in intrathoracic pressure against a closed glottis, such as seen with blunt chest trauma or during cardiopulmonary resuscitation. The air leak from a closed pneumothorax may be self-limited, or may progress to a tension pneumothorax.

Tension Pneumothorax

Tension pneumothorax usually results from traumatic causes, but rarely it may be from a spontaneous lung collapse (Fig. 10-2). Fractures of the trachea or bronchi, a ruptured esophagus, the presence of an occlusive dressing over an open pneumothorax, or chest compressions may result in tension pneumothorax. The risk is also increased for patients with chest trauma who are undergoing positive-pressure ventilation. Because

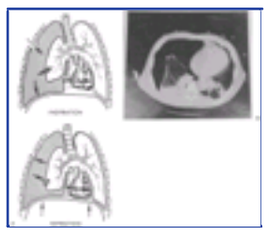

Figure 10-2 *A*, Tension pneumothorax. With inspiration air enters the pleural space from the outside or from a puncture in the lung, or both. Upon expiration, the air leaks are closed, trapping increasing amounts of air in the pleural space. Cardiac filling and eventually cardiac output are compromised. *(From Vukich DJ, Markovchick VJ: Pulmonary and chest wall injuries. In Rosen P, Barkin RM, Braen CR, et al (eds): Emergency Medicine: Concepts and Clinical Practice. St Louis, Mosby-Year Book, 1988. Reproduced by permission.)* *B*, A thoracic computed tomography (CT) scan demonstrating a tension pneumothorax. In general, whenever a tension pneumothorax is suspected, treatment should be initiated before obtaining a CT scan *(Courtesy of H.K. Liang).*

of this, any patient with a penetrating thoracic injury (even without immediate evidence of a hemo- or pneumothorax) may be a candidate for a "prophylactic" chest tube before mechanical ventilation.

A tension pneumothorax occurs when a pulmonary or bronchial injury creates a ball-valve mechanism that leads to the progressive accumulation of air in the pleural space. The negative intrathoracic pressure during inspiration draws air into the pleural space. During expiration the relative increase in intrathoracic pressure closes the tissue flap and restricts the outflow of air. As more air collects and the intrapleural pressure rises on the injured side, the mediastinum shifts toward the uninvolved side, obstructing venous return to the right atrium. Progressive hypoxia and CO_2 retention may lead to the cardiovascular collapse. In addition to the cardiac effects, a tension pneumothorax reduces functional capacity of the lung and leads to a ventilation-perfusion mismatch.

Hemothorax

Hemothoraces may be caused by injuries to the heart, lungs, great vessels, intercostal arteries or veins, mediastinal veins, chest wall vessels, or fractured ribs. Bleeding from the lung

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parenchyma is low pressure, and is usually self-limited or ceases when a chest tube is placed. Intercostal artery, pulmonary artery, and internal mammary artery bleeding can be profuse and usually requires surgical intervention.

Chylothorax

Chylothorax results from an injury to the thoracic duct from central line placement, operative injury, or chest trauma. The chyle initially collects extrapleurally, and may not begin to fill the pleural cavity for 2 to 10 days. As a result there are few initial symptoms related to a thoracic duct injury. As the fluid accumulates, patients eventually develop dyspnea and other findings similar to any pleural fluid collection. The diagnosis is made by a thoracentesis that demonstrates a milky white fluid with a high fat and lymphocyte content and 4 to 5 gm/dL of protein. The definitive treatment is either by repeated thoracentesis or tube thoracostomy combined with parenteral alimentation until the volume of chyle decreases.

Empyema/Effusions

An "empyema" is defined as infection or gross pus in the pleural space. Empyema occurs in 1% to 2% of hospitalized patients with community-acquired pneumonia. Most cases are associated with parapneumonic effusions caused by a mix of anaerobic and aerobic bacteria. The remainder of empyemas result from the complications of thoracic surgical procedures or thoracic trauma, or from esophageal perforation or subdiaphragmatic infection. *S. aureus* is the most common isolate. See chapter 9 for a discussion of effusions.

DIAGNOSIS

Symptoms

Symptoms in patients with abnormal collections of fluid or air in the pleural space may range from asymptomatic to respiratory arrest. Specific symptoms range from mild dyspnea and pleuritic chest pain for small disruptions, to severe dyspnea and pain, with a feeling of impending doom, for those with a tension pneumothorax. A cough may also be present. The severity of symptoms depends on the size of the pneumothorax, rapidity of accumulation, age of the patient, mobility of the mediastinum, and the presence of any underlying lung disease. Severely injured patients may be unable to relate symptoms, rendering the physical examination and chest radiograph as essential to diagnose a pneumo- and hemothorax. A tension pneumothorax must be considered in any patient with sudden respiratory or cardiac deterioration. Other possible causes of sudden cardiopulmonary decompensation include pericardial tamponade, massive pulmonary embolus, myocardial infarction, respiratory failure from reactive airway disease, and pneumomediastinum. A tension pneumothorax must also be considered in intubated patients who become difficult to ventilate, develop increased airway pressure, elevated central venous pressure, or elevated pulmonary artery pressure. Awake patients with tension pneumothorax rapidly develop severe dyspnea, restlessness, agitation, and a feeling of impending doom. They are usually tachycardic and tachypnic but quickly progress to hypotension.

With a spontaneous pneumothorax, 95% of patients complain of the sudden onset of sharp or pleuritic chest/shoulder pain, or both. Sixty percent of patients experience dyspnea and 12% have a mild cough. Dyspnea and anxiety are more common in older patients.

The symptoms of a hemothorax are similar to a pneumothorax, but may be accompanied by hypotension as blood accumulation in the pleural space increases. The onset of symptoms for effusions is usually much more gradual with increasing shortness of breath and dyspnea on exertion.

Physical Examination

Unstable Patients.

During the initial ("ABC") phase of resuscitation, the vital signs often point to the presence of a tension pneumothorax. This diagnosis must be considered for patients who are tachycardiac, hypotensive, and dyspneic. Once the airway is cleared, a rapid assessment of the breathing must be conducted. Unfortunately no single examination is completely reliable and so multiple examinations must be conducted rapidly. Observation of the chest wall movement and neck may reveal asymmetric chest expansion, distended neck veins, or a deviated trachea. Auscultation may demonstrate unilateral diminished or absent breath sounds. A pulsus paradoxus may be detected. There may be hyperresonance to percussion unilaterally. *Subcutaneous emphysema* that develops in the absence of an open chest wound should be assumed to have been produced by a pneumothorax (Fig. 10-3). An early sign of tension pneumothorax in an intubated patient is difficulty in maintaining ventilation or a marked increase in airway pressures.

For unstable trauma patients the diagnosis of a tension pneumothorax may be confirmed or made by needle or catheter thoracentesis. Logistics of a resuscitation may not allow time for the results of a radiograph. The diagnosis can be confirmed when there is a rapid release of air through the needle during both inspiration and expiration as the pneumothorax decompresses. The patient's vital signs should rapidly improve, as the air that was trapped in the pleural space is allowed to escape. In one prospective study, the sensitivity, specificity, and diagnostic accuracy of auscultation for hemo/pneumothorax was 84%, 97%, and 89%, respectively. A false-negative auscultation is more likely than a false-positive one. Since the specificity of auscultation is high, chest tubes may be placed before radiographic confirmation if the clinical situation warrants. [3]

Stable Patients.

For more stable patients the physical examination may indicate the presence of a pneumo- or hemothorax, but a chest radiograph is usually necessary for the definitive diagnosis. Physical findings may include tachypnea, tachycardia, unilaterally decreased breath sounds, decreased tactile fremitus, increased resonance with percussion, or subcutaneous (SQ) emphysema, but the examination may reveal no abnormalities with a small pneumothorax. A pneumothorax of 10% to 20% will unlikely be diagnosed by ausculation of the lungs, since a small pneumothorax will produce only very subtle, or undetectable, changes in breath sounds. Pleural fluid collections and hemothoraces can be difficult, if not impossible, to detect by physical examination, particularly with <500 mL of fluid in the pleural space. Breath sounds may then decrease and percussion of the bases may be dull. Lung function may not become compromised until

Figure 10-3 *A*, Following a successful cardiopulmonary resuscitation and intubation from respiratory arrest, this patient began to deteriorate. Marked subcutaneous emphysema was noted in the scrotum and abdominal wall. Curiously, little air could be palpated in the chest or neck, the usual place for air to accumulate. Assuming that the air must have come from the lungs, the scenario led to the clinical diagnosis of a tension pneumothorax. *B*, Following the placement of a chest tube, the vital signs quickly stabilized. Due to the urgency of the situation, the thoracostomy tube was performed before obtaining an x-ray, and the rush of air obtained upon entering the pleural cavity, and the normalization of vital signs, retrospectively confirmed the tension pneumothorax.

patients lose up to 40% of their blood volume into the pleural space. These patients may then present primarily in hypovolemic shock. For patients with blunt chest trauma, with a normal physical examination, including no chest tenderness, a radiograph may not be necessary. [4]

Parapneumonic empyemas often present with fever, cough, chest pain, dyspnea, and purulent sputum. The physical examination will reveal diminished breath sounds, dullness to percussion, egophony, and diminished tactile fremitus on the involved side. For postsurgical and posttraumatic empyema the patient will develop a fever, the pleural fluid drainage may be excessive and become purulent, and respiratory symptoms may worsen.

Radiography

Plain Radiographs.

A chest radiograph is essential to diagnose a pneumothorax in the stable patient. However, any patient with a possible tension pneumothorax should not be sent to radiology unless accompanied by a clinician capable of performing a tube thoracostomy or chest decompression. The best films for a hemothorax or pneumothorax are an *upright* posteroanterior (PA) and lateral chest radiograph, but for unstable patients a portable supine anteroposterior (AP) view may be the only film possible. A supine chest radiograph of a patient with a hemothorax with up to 1000 mL of blood may only demonstrate slight differences in the densities of the lung fields. On an upright chest radiograph, at least 300 to 500 mL of fluid is needed before costophrenic angle blunting can be detected (Fig. 10-4).[5] Fluid in the pleural cavity will form a miniscus, and a straight line on an upright chest radiograph is essentially an air/fluid level signifying the presence of a pneumothorax as well as a hemothorax.

It has traditionally been touted that the *expiratory* upright PA chest x-ray is the best radiograph to detect a pneumothorax. Theoretically the lung is more collapsed during expiration, accentuating the pneumothorax. However, when studied prospectively, there is no detectable benefit of the expiratory film, and certainly both projections need not be routinely taken. [6] While there is no downside to initially ordering an expiratory radiograph, the upright *inspiratory* x-ray is recommended as the initial film of choice. This projection is preferred because of its comparable sensitivity and specificity when compared to the expiratory projection, and the superior

ability of the inspiratory radiograph to detect other abnormalities such as pneumonia, cardiac, and pleural disease.

For stable patients other radiographic views can be helpful. To look for a hemothorax, the best additional views are bilateral decubitus chest radiographs. If a single decubitus film is obtained, the injured side should be up. For a small pneumothorax an upright expiratory chest radiograph can occasionally be helpful, as well as cross-table lateral and oblique views of the chest.

On a chest radiograph the partially collapsed lung of a pneumothorax appears as a visceral pleural line with no pulmonary markings beyond it. Other radiographic findings include hyperlucency of the affected hemithorax, a double diaphragm contour, increased visibility of the inferior cardiac

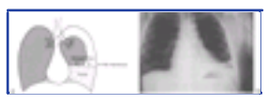

Figure 10-4 *A*, Hemopneumothorax. Note that whenever the top of the accumulated fluid is seen as a straight line as opposed to a meniscus, a pneumothorax must also be present. *B*, Following a stab wound to the chest, this radiograph demonstrated an air-fluid level (straight line) in the left hemithorax. A 10% pneumothorax is difficult to see on the poor quality film, but the air-fluid level means that a pneumothorax must be present. Otherwise, the fluid would form a meniscus tracking up the lateral chest wall. On a supine film, this subtle finding may not be appreciated.

border, better visualization of the pericardial fat at the cardiac apex, and possibly a depressed diaphragm. If *SQ air* is noted on the chest x-ray of a patient with blunt chest trauma, it can be assumed that the air came from an injured lung, and that a pneumothorax exists, or soon will.

With a tension pneumothorax the chest radiograph reveals a depressed hemidiaphragm with lung collapse on the affected side and a shift of the mediastinum and trachea to the opposite side. With a bilateral pneumothorax no mediastinal shift may be seen. Localized areas of tension pneumothorax may result from adhesions. Patients with adult respiratory distress syndrome on positive end-expiratory pressure have developed a localized subpulmonic or paracardiac tension pneumothorax despite indwelling thoracostomy tubes.

When a patient is supine a small pneumothorax may collect in various areas. The most common area for air to accumulate is anteromedial, which is seen as a radiolucent band that enhances the heart border. With subpulmonic air the visceral pleura is outlined by a radiolucent band superior and parallel to the diaphragm.

If a patient with a pneumothorax is to be observed, there are systems that measure the size of a pneumothorax to ensure it is not expanding. The simplest and most reliable way is to measure the distance from the lateral lung margin to the lateral chest wall on a chest radiograph taken during full inspiration. This method will also roughly estimate the percentage of the pneumothorax (Fig. 10-5).

Chest Computed Tomography (CT) Scan.

With increased usage of CT scanning to evaluate chest trauma, it has become obvious that standard radiographs have missed many pneumothoraces and hemothoraces in the past. Such abnormalities may be present in up to 10% of trauma patients with "normal" chest x-rays. [7] [8] [9] The clinical significance of these small, previously undetected, occult injuries is likely not great, and it has been suggested that a small pneumothorax seen onlyon CT scan may be left untreated, and simply observed, in the otherwise stable patient. Many patients with a pneumothorax seen only on CT scan may also safely undergo positive-pressure ventilation without the placement of a chest tube.[9]

CT scans are not routine for the diagnosis of a hemo- or pneumothorax, but they offer invaluable information on the etiology of such abnormalities. A CT scan is often used to evaluate patients with a spontaneous pneumothorax to assess the need for surgery. A CT scan may be useful when the diagnosis is unclear, or when looking for small amounts of pleural fluid. A chest CT scan is more accurate than a chest radiograph to estimate the size of a pneumothorax. [10] CT scans are particularly useful to determine if an empyema is loculated or draining successfully.

INDICATIONS FOR TUBE THORACOSTOMY

Pneumothorax

There is considerable controversy over the treatment of a non-tension pneumothorax. Clinicians use individual approaches because no agreed-on standard of care exists for many situations involving minor lung collapses. Many patients with a pneumothorax will require tube thoracostomy, but a significant number can be managed conservatively. Chest tube placement may be unnecessary in patients with isolated small or moderate traumatic pneumothoraces in the absence of respiratory compromise, concomitant injuries, or when positive-pressure ventilation will not be required. Otherwise healthy, reliable, and minimally symptomatic patients with no underlying lung disease and small pneumothoraces (<10% to 20%) are often treated by observation alone. Aspiration of a pneumothorax may be useful, but it has had variable success. The insertion of a small-bore chest catheter in stable but symptomatic patients who require lung expansion is often recommended. For these patients prolonged suction is rarely required, and the tube can simply be attached to a Heimlich valve or underwater seal. [11] [12] The American College of Chest Physicians has developed guidelines for the management of primary and secondary spontaneous pneumothoraces. These guideline are not always applicable to the emergency clinician and promulgate a conservative approach that may

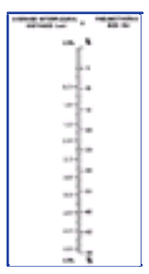

Figure 10-5 Nomogram for the prediction of pneumothorax size from an average interpleural distance (see text). The interpleural distance is measured in centimeters from the outer edge of the collapsed lung to the inner aspect of the chest wall (i.e., parietal pleura to visceral pleura). *(From Rhea JT, DeLuca SA, Greene RE: Determining the size of pneumothorax in the upright patient. Radiology 144:733, 1982. Reproduced with permission.)*

not be reasonable for all patients. The recommendation that catheter aspiration is not an acceptable method for the initial treatment of a primary pneumothorax has met with significant criticism. Nonetheless, the guidelines are helpful (Table 10-1).

Without any intervention or continuing air leak, a small pneumothorax will resolve over days to weeks. Supplemental oxygen will speed this process by increasing the rate of pleural air absorption, but this common intervention has no proven applicability in the emergency department (ED). Patients managed by observation alone should remain in the ED for 3 to 6 hours under close observation for serial examinations and a repeat chest radiograph. If the pneumothorax has not progressed, the reliable patient with access to health care can be discharged with instructions to return immediately if symptoms increase, to minimize his or her activities, and to follow up for repeat examinations and chest radiographs.

For patients with traumatic etiologies, the rapidity and type of treatment depends primarily on the stability of the patient; a hypotensive patient with a tension pneumothorax requires immediate decompression with a chest tube or needle thoracostomy, while a patient with normal vital signs anda small pneumothorax may be observed. Emergent needle thoracostomy is only a temporary solution, and once done necessitates an ipsilateral tube thoracostomy. Other factors that modify the treatment include the patient's age, the size of the pneumothorax, whether there are bilateral pneumothoraces, and whether the current episode represents a recurrence. A tube thoracostomy may not be necessary for a small traumatic pneumothorax with no associated rib fractures. Remember that a chest tube is often the safest and most expedient course, and one cannot be faulted by such expectant definitive treatment. A chest tube removes all doubt since it evacuates the intrapleural air and blood, prevents further accumulation, and allows monitoring for persistent air leaks.

Because of the risk of a tension pneumothorax, a chest tube should be considered for all patients with a penetrating chest injury if positive-pressure ventilation will be used or if they will be transported a long distance for definitive care. However, CT scans of trauma victims have demonstrated that many patients with small pneumothoraces that would have escaped detection by standard radiographs have safely undergone positive-pressure ventilation without developing a clinically evident pneumothorax. Close observation for signs of a tension pneumothorax is necessary if a chest tube is not placed and positive-pressure ventilation is used.

The treatment of a spontaneous pneumothorax is controversial. Patients can be observed, treated by aspiration, or may require a tube thoracostomy (see Table 10-1). Patients can be observed if they are asymptomatic; have no underlying pulmonary disease; and have a small (<20%), pneumothorax. Initially they should be observed in the hospital to prove they are stable, and then followed as outpatients with serial chest radiographs. Some authors recommend that a spontaneous pneumothorax be treated with catheter aspiration, followed by 6 hours of observation before discharge from the ED. However, these studies have been small, have shown inconsistent results, and have found a high recurrence rate. Other techniques include the use of pigtail catheters and small-bore thoracostomy tubes introduced over a wire.

The underlying lung pathology of a patient with a spontaneous pneumothorax is best initially evaluated by CT scan. Often this is followed by diagnostic or therapeutic visual inspection of the lung and pleural space by thoracoscopy. Extensive evaluation is not, however, usually recommended for the first episode of a small primary pneumothorax. When patients have recurrent spontaneous pneumothoraces, further evaluation (CT scan, thoracoscopy) and evaluation for surgical treatment are indicated. Patients who have had one spontaneous pneumothorax have a 30% to 50% chance of recurrence within 2 years, and after the second pneumothorax there is a 50% to 80% of a third. Surgery may be recommended for a first pneumothorax in the following situations: life-threatening tension pneumothorax, massive air leaks with incomplete reexpansion, an air leak persisting 4 days after a second tube has been placed, associated hemothorax with complications, identifiable bullous disease, and failure of easy reexpansion in patients with cystic fibrosis.

Hemothorax

Tube thoracostomy is also used to monitor the amount and rapidity of blood output, which determines the need for additional interventions, including a thoracotomy. About

TABLE 10-1 -- Guidelines of the American College of Chest Physicians for the Management of Primary and Secondary Spontaneous Pneumothorax

Primary Spontaneous Pneumothorax
Clinically Stable Patients with Small Pneumothoraces: *
Clinically stable patients with small pneumothoraces should be observed in the emergency department for 3 to 6 h and discharged home if a repeat chest radiograph excludes progression of the pneumothorax (good consensus). Patients should be provided with careful instructions for follow-up within 12 h to 2 days, depending on circumstances. A chest radiograph should be obtained at the follow-up appointment to document resolution of the pneumothorax. Patients may be admitted for observation if they live distant from emergency services or follow-up care is considered unreliable (good consensus). Simple aspiration of the pneumothorax or insertion of a chest tube is not appropriate for most patients (good consensus), unless the pneumothorax enlarges. † The presence of symptoms for >24 h does not alter the treatment recommendations.

Clinically Stable Patients with Large Pneumothoraces: *

Clinically stable patients with large pneumothoraces should undergo a procedure to reexpand the lung and should be hospitalized in most instances (very good consensus). The lung should be reexpanded by using a small-bore catheter (=14F) or placement of a 16F to 22F chest tube (good consensus). Catheters or tubes may be attached either to a Heimlich valve (good consensus) or to a water seal device (good consensus) and may be left in place until the lung expands against the chest wall and air leaks have resolved. If the lung fails to reexpand quickly, suction should be applied to a water-seal device. Alternatively, suction may be applied immediately after chest tube placement for all patients managed with a water seal system (some consensus).

Reliable patients who are unwilling to undergo hospitalization may be discharged home from the emergency department with a small-bore catheter attached to a Heimlich valve if the lung has reexpanded after the removal of pleural air (good consensus). Follow-up should be arranged within 2 days. The presence of symptoms for >24 h does not alter management recommendations.

Secondary Spontaneous Pneumothorax

Clinically Stable Patients with Small Pneumothoraces: *

Clinically stable patients with small pneumothoraces should be hospitalized (good consensus). Patients should not be managed in the emergency department with observation or simple aspiration without hospitalization (very good consensus). Hospitalized patients may be observed (good consensus) or treated with a chest tube (some consensus), depending on the extent of their symptoms and the course of their pneumothorax. Some of the panel members argued against observation alone because of a report of deaths with this approach. Patients should not be referred for thoracoscopy without prior stabilization (very good consensus). The presence of symptoms for >24 h did not alter the panel members' recommendations.

Clinically Stable Patients with Large Pneumothoraces:

Clinically stable patients with large pneumothoraces should undergo the placement of a chest tube to reexpand the lung and should be hospitalized (very good consensus). Patients should not be referred for thoracoscopy without prior stabilization with a chest tube (very good consensus). The presence of symptoms for >24 h did not alter the panel members' recommendations.

Terms	**Definition**
Spontaneous pneumothorax	No antecedent traumatic or iatrogenic cause
Primary spontaneous pneumothorax	No clinically apparent underlying lung abnormalities or underlying conditions known to promote pneumothorax (e.g., HIV disease)
Secondary spontaneous pneumothorax	Clinically apparent underlying lung disease
Pneumothorax size	Determined by distance from the lung apex to the ipsilateral thoracic cupola at the parietal surface as determined by an upright standard radiograph
Small pneumothorax	<3 cm apex-to-cupola distance
Large pneumothorax	=3 cm apex-to-cupola distance
Clinical stability	
Stable patient	All of the following present: respiratory rate, <24 breaths/min; heart rate, >60 beats/min or <120 beats/min; normal blood pressure, room air O_2 saturation, >90%; and patient can speak in whole sentences between breaths
Unstable patient	Any patient not fulfilling the definition of stable
Drainage tubes	
Small chest tube or small percutaneous catheter	=14Fr
Moderate-sized chest tube	16Fr to 22Fr
Large chest tube	24Fr to 36Fr
Simple aspiration	Insertion of a needle or cannula with removal of pleural air followed by immediate removal of the needle or cannula

From Baumann MH, Strange C, Heffner JE, et al: Management of Spontaneous Pneumothorax: An American College of Chest Physicians Delphi Consensus Statement. Chest 119:590, 2001.

*See Definition of Terms.
†This recommendation has been criticized and many authorities recommend that simple catheter aspiration is the logical first approach. (Miller AC, Harvey J: Guidelines for the management of spontaneous pneumothorax. BMJ 307:114, 1993. Miller AC, Harvey J: Pneumothorax: What's wrong with simple aspiration? Chest 120:1041, 2001.)

three-fourths of patients with a traumatic hemothorax can be managed by tube thoracostomy and volume replacement alone. For the remaining, immediate or delayed elective thoracotomy may be required. The indications for surgery following a hemothorax are somewhat controversial (Table 10-2). Early institution of blood replacement is recommended for patients with massive hemothorax (>2000 mL), since these are often associated with continuing hemorrhage. Autotransfusion of the shed blood is desirable if the technique is available (see Chapter 28).

TABLE 10-2 -- Indications for Surgery After Tube Thoracostomy Based on the Results of the Thoracostomy

Massive hemothorax, >1000–1500 mL initial drainage
Continued bleeding
>300–500 mL in first hour
>200 mL/hour for first 3 or more hours
Increasing size of hemothorax on chest film
Persistent hemothorax after 2 functioning tubes placed
Clotted hemothorax
Large air leak preventing effective ventilation
Persistent air leak after placement of second tube or inability to expand lung fully

Empyema

The treatment of patients with empyema depends on the severity of their infection and their underlying condition. Some patients with empyema can be treated with serial thoracenteses, but most will require continuous drainage with a tube thoracostomy. Thoracoscopic decortication represents definitive therapy for severe cases. Usually a diagnostic thoracentesis is done first to assess the fluid for signs of infection. Thick pus on thoracentesis, a positive Gram stain fluid glucose <60 mg/dL, pH <7.20, or elevated lactate dehydrogenase are associated with effusions requiring chest tube drainage. Once an empyema is detected, therapy should not be delayed because the fluid can become loculated within hours. The tube is left in place until the volume of the pleural drainage becomes clear yellow and is <150 mL in 24

hours.

An empyema that fails to resolve on the chest radiograph within 48 hours requires chest CT scan and a careful review of antibiotic choice. Multiloculated effusions are best managed with thoracoscopic decortication.

CONTRAINDICATIONS

For unstable injured patients with a pneumo- or hemothorax, there are no absolute contraindications to a tube thoracostomy. In critical patients the placement of a chest tube is often performed empirically, since procedures to confirm the presence of, assess the extent of, or prove the absence of pathology are prohibited by logistics of the resuscitation. In the stable patient, relative contraindications include anatomic problems such as the presence of multiple pleural adhesions, emphysematous blebs, or scarring. Coagulopathic patients should be evaluated for clotting factor replacement before any invasive procedure.

TREATMENT

Treatment of a Tension Pneumothorax During a Resuscitation

If a patient is undergoing resuscitation and is in extremis, the clinician rarely has time for formal tube thoracostomy, and needle decompression is simply not effective within seconds. Based on clinican findings or circumstances (such as a stab wound to the chest, markedly abnormal breath sounds, cyanosis, tracheal deviation, marked SQ air, and hypotension) it is suggested that the chest cavity be ventilated by immediate measures. It is assumed that the patient is intubated so one merely needs to create an exit for the air that is compressing the lung and shifting the mediastinum (Fig. 10-6). A scalpel is used to open the skin and SQ tissue by making a deep 3 to 4 cm incision over a rib in the upper chest wall at the anterior axillary line. A large *closed* Kelly clamp or scissors are used to blindly stab through the pleural via the skin incision. The instrument is guided over the top of the rib and once a pop is felt, indicating penetration of the pleura, the blades of the instrument are opened. This will allow for the immediate egress of air and the tension will be relived enough to allow the ventilated patient to normalize respiratory and cardiovascular function. When the immediate emergency is controlled, an appropriately sized chest tube is inserted through the incision. This is usually a nonsterile procedure.

Prehospital Treatment

Emergent needle thoracostomy may be used in the prehospital setting or when a patient suspected of having a pneumothorax rapidly deteriorates or presents in extremis. The needle (or catheter) may then be attached to a flutter valve (fashioned from the fingers of a surgical glove), underwater seal, or commercially available one-way (Heimlich) valve to allow for the continued drainage of the pneumothorax. The definitive treatment of a tension pneumothorax is tube thoracostomy.

For a more stable open pneumothorax, prehospital treatment involves the application of a sterile dressing over a penetrating wound to act as a one-way (flap) valve. This allows air to exit the pleural space but blocks air reentry through the wound. The patient is instructed to perform the Valsalva maneuver after deep inspiration or to cough just as the dressing is placed. Ideally, a sterile dressing of petrolatum-impregnated gauze that extends 6 to 8 cm beyond the wound in all directions is used. This underlying dressing should then be covered by gauze dressings and secured with tape on three sides only.

Equipment

Examples of instruments in a tube thoracostomy tray are listed in Table 10-3 . Since the contents of these trays vary among hospitals, emergency clinicians should familiarize themselves with the trays before an emergency. Additional important materials are listed in Table 10-4 . If possible, all equipment should be organized before beginning the procedure.

The most commonly used chest tubes are clear plastic, straight tubes with a series of holes along the distal length and open at both ends. To assess depth of insertion, a *radiopaque strip runs along the length that is commonly interrupted by the last hole*. Sizes used for adults vary from 12 to 42 French (Fr), with smaller tubes used for pneumothorax, and larger (a minimum of 36 Fr) for hemothorax and empyema (see Table 10-1 for the definition of chest tube sizes). The largest possible tube should be used to drain a hemothorax. For pediatric patients, No. 14, 16, 20, and 24 Fr tubes are adequate. Before insertion, the beveled (extrathoracic) end of the tube is cut squarely to better fit the commonly available connectors.

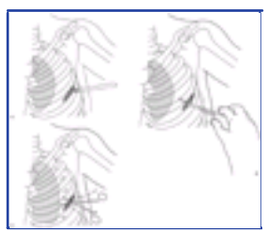

Figure 10-6 During a resuscitation involving a tension pneumothorax, there may be no time for a chest tube and needle decompression may not be effective rapidly enough. Under these circumstances the pleural cavity can be vented in seconds. This assumes that the patient is intubated. *A*, A No. 10 scalpel is used to make a deep incision in the skin and subcutaneous tissue over the fourth or fifth rib in the anterior axillary line. *B*, A long *closed* Kelly clamp (or scissors) is inserted over the top of the rib and stabbed into the pleural space. A pop is usually felt. *C*, Once the instrument is in the pleural space, it is opened wide to create a rent in the parietal pleura. Air should immediately vent. If the patient is intubated, normal cardiorespiratory function can be maintained. A chest tube is then inserted. (See also Figure 10-11 Figure 10-12 Figure 10-13 Figure 10-14 .)

PROCEDURE

Before any procedure, gown, glove, mask, and goggle precautions must be used.

Tube Location

A chest tube placed anywhere in the pleural cavity will drain blood, fluid, or air.

A chest tube that is performing its intended function, regardless of its location, can be left in place until an elective tube can replace it. However, traditional sites are usually chosen. For a pneumothorax the classic location for a thoracostomy tube was in the second intercostal space in the midclavicular line, but in practice this site is usually only used for needle decompression of a tension pneumothorax. The most common location for a chest tube is the mid- to anterior axillary line (Fig. 10-7). It is cosmetically preferable and better tolerated. Recommendations for location vary from the fourth to the eighth intercostal space, but most often the fourth or fifth intercostal space is used. This is roughly at the level of the nipple or the inferior scapular border in most patients. The entrance site should be lateral to the edge of the pectoralis major and breast tissue. *A more superior position should usually be chosen since the external landmarks can be misleading and chest tubes are more often placed too low rather than too high.* The diaphragm of a

TABLE 10-3 -- Basic Instrument Tray for Tube Thoracostomy

Sterile towels—4
Basin for preparation solution
10- to 20-mL syringe and assorted needles
No. 10 scalpel
Forceps
Large, straight (suture) scissors
Large, curved (Mayo) scissors
Large clamps (Kelly)—2
Needle holder
No. 0 or 1-0 silk on large cutting needles
Gauze pads

supine patient who is not taking a deep breath is much higher than most clinicians suspect.

There is no evidence in adults that tube location affects the ability to drain fluid collections. As the lung expands and the pleural space becomes smaller, air and fluid will follow the path of least resistance and enter a functioning drainage tube, regardless of the tube's location. Therefore, a tube anywhere in the pleural space should adequately drain a hemothorax of unclotted blood, as long as there are no loculations.

Before insertion the tube should be held beside the chest wall. The tube's tip should be near the clavicle to estimate the distance the tube should be advanced from the incision site to the apex of the lung. For a hemothorax, the tube is directed toward the diaphragm. A clamp may be placed on the tube to mark the maximum length the tube is inserted. Make sure this location is sufficient to ensure that the *last drainage hole will be within the pleural space*. This method helps to minimize subsequent repositioning of the tube. During emergency placement, it is common, yet usually clinically inconsequential, to advance the tube too far. In markedly obese patients it is common to fail to advance the tube far enough, thereby not assuring that the last hole is in the pleural space.

Patient Preparation

Patients should be started on nasal oxygen and placed on continuous pulse oximetry monitoring. For stable patients the use of parenteral analgesics or conscious sedation should be routine. For anterior axillary line insertions, the head of the bed should be elevated 30 to 60° (Fig. 10-8). This lowers the diaphragm and decreases the risk of injury to the diaphragm, spleen, or liver. The arm on the affected side is placed over the

TABLE 10-4 -- Other Materials Required for Tube Thoracostomy

Local anesthetic
Antiseptic solution
Arm restraints (padded)
Tincture of benzoin
Adhesive tape—cloth-backed
Chest tubes
No. 28 to 36 Fr for adults
No. 16, 20, 24 Fr for children
Clear, sterile plastic tubing in 6-foot lengths, ½-inch diameter
Hard plastic serrated connectors
Drainage apparatus with sterile water for water seal
Y connectors

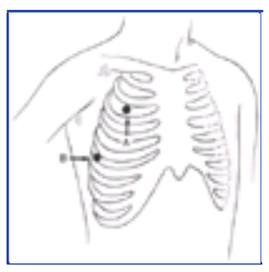

Figure 10-7 Standard sites for tube thoracostomy. *A*, The second intercostal space, midclavicular line. *B*, The fourth or fifth intercostal space, midaxillary line. The midaxillary line placement is usually preferable for all chest tubes, regardless of pathology. Always avoid the breast tissue, even in prepubescent girls. Note that placing the tube too far posteriorly will not allow the patient to lie down comfortably.

patient's head and restrained in that position. The other arm can be restrained comfortably at the patient's side. The area where the tube will be inserted should be sterilized with a povidone-iodine solution and draped with sterile towels.

Anesthesia

With the use of parenteral analgesics, conscious sedation, and generous local anesthesia, the insertion of a chest tube should

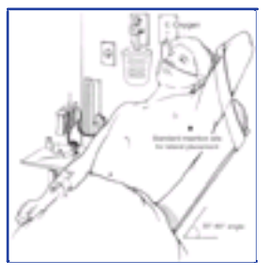

Figure 10-8 To insert a chest tube, the patient is placed semi-erect with the ipsilateral shoulder abducted and preferably restrained. Supplemental oxygen is recommended.

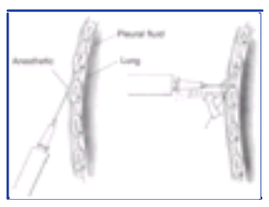

Figure 10-9 Insertion of a chest tube can be relatively painless with proper infiltration of the skin *and pleura* with local anesthetic. The liberal use of 1% lidocaine with epinephrine (maximum lidocaine dose, 5 mg/kg) is recommended. *(Redrawn from Hughes WT, Buescher ES: Pediatric Procedures, 2nd ed. Philadelphia, WB Saunders, 1980, p 234.)*

be relatively painless. Since the procedure can be extremely painful, up to 5 mg/kg of locally injected 1% lidocaine (Xylocaine) with epinephrine (1:100,000) can be used (Fig. 10-9). Note: Each mL of 1% lidocaine contains 10 mg of lidocaine. First inject the anesthetic in the SQ area with a small-bore (27-gauge [ga]) needle in the area of the skin incision. Then generously infiltrate the muscle, periosteum, and parietal pleura in the area of the tube's passage. This may require a larger-bore needle (21- or 23-ga). A common error is inadequate local anesthesia; another error is to forget that additional anesthetic may be required throughout the procedure.

In an elective situation an alternative is to use intercostal nerve blocks above and below the rib spaces of the incision and insertion. The needle and syringe may also be used to aspirate and find the pleural cavity in the area of insertion (Fig. 10-10). If air or fluid are not found, the insertion site should be changed. Once the tube is in place, anesthetic may be administered through the chest tube and instilled into the pleural space to reduce the severe irritation of the pleura. One approach for stable patients is to administer 10 mL of 0.5%

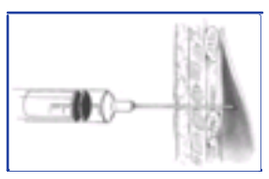

Figure 10-10 Use of the anesthetic needle to puncture the parietal pleura and establish the presence of blood or air in the pleural space. This procedure is not only diagnostic, but also may be a temporary therapeutic maneuver in a patient with tension pneumothorax. *(Redrawn from Richards V: Tube thoracostomy. J Fam Pract 6:631, 1978.)*

bupivacaine through the chest tube while the patient is lying on the contralateral side. [13] After 5 minutes without drainage of the thorax, standard gravity or vacuum drainage is reinitiated. Parenteral analgesic agents should be used as needed to control the pain associated with the initial injury and the procedure.

Insertion

The initial skin incision must be of adequate size. A common mistake is to make the skin incision too small so that it is difficult to create an adequate track for either the thoracostomy tube or an exploring finger. One method is to make the initial skin incision at an intercostal space lower than the thoracic wall entry site so that the tube may be "tunneled" up over the next rib. This is thought to provide a better seal against air leaks, although the efficacy has not been substantiated (Fig. 10-11). With a No. 10 blade, make at least a 3- to 4-cm transverse

Figure 10-11 The skin wound is made one intercostal space below the space through which the tube will pass *(A)*. By spreading tissue with scissors *(B)*, dissection is carried subcutaneously to the pleural lining. The pleura is entered with a blunt Kelly or Tonsillar clamp *(C)*. A common error in technique is to attempt to insert a large chest tube through a skin incision that is too small. *(Redrawn from Hughes WT, Buescher ES: Pediatric Procedures, 2nd ed. Philadelphia, WB Saunders, 1980, p 237.)*

incision through the skin and the SQ tissues directly over the rib located one interspace beneath the rib the tube will pass over. Using blunt dissection with a large Kelly clamp or scissors, a track is created over the rib above by pushing forward with the closed points and then spreading and pulling back with the points spread. To avoid the intercostal vessels and the nerve (located on the inferior margin of each rib) the track should pass immediately over the superior surface of the lower rib in the chosen intercostal space (Fig. 10-12). Then push through the muscle and parietal pleura with the closed points of the clamp until the pleural cavity is entered. This often takes considerable force and may require a twisting motion with the clamp. A palpable pop may be felt as the pleura is penetrated. A rush of air or fluid should occur at this point. The tips of the clamp, still within the pleural cavity, are spread widely and withdrawn to make an adequate pleural entry (Fig. 10-13). Penetrating the pleura is usually the most painful portion of chest tube placement and it is nearly impossible to completely anesthetize this portion of the procedure.

Once the pleura has been penetrated, the clinician inserts a gloved finger into the chest wall track to verify that the pleura has been entered and that no solid organs are present. The finger may be swept inside the cavity to assure that pleural adhesions are absent. The finger is left in the pleural space as a guide, and the tube is passed over, under or beside the finger into the pleural space (Fig. 10-14). This step allows the clinician to feel the tube passing into the pleural cavity, and avoids

SQ dissection with the tube. Some clinicians prefer passing the tube alone, while others recommend that the tube be held in a large curved clamp, with the tube tip protruding from the jaws (Fig. 10-15). The tube is directed superiorly

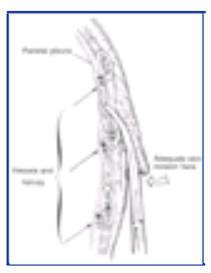

Figure 10-12 Location of the intercostal neurovascular bundle, running inferiorly and slightly medial to the rib. *(From Millikan JS, Moore EE, Steiner E: Complications of tube thoracostomy for acute trauma. Am J Surg 140:739, 1980.)*

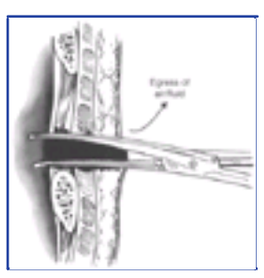

Figure 10-13 One accomplishes blunt dissection by forcing the closed points of the clamp forward, then spreading the tips and pulling back with the points spread. Note that the clamp should be spread in a direction parallel with the ribs (See Fig. 10-6); not perpendicular to the ribs as suggested in the figure. The drawing emphasizes the placement of the instrument *over* the rib. A rush of air or fluid signifies penetration into the pleural space. *(From Bricker DL: Safe, effective tube thoracostomy. ER Reports 2:49, 1981.)*

and posteriorly. The tube is then advanced superiorly, medially, and posteriorly until the marker clamp that was previously attached touches the chest wall. Alternatively, the tube may be advanced until pain is felt or resistance is met, and then pulled back 2 to 3 cm. Ensure that all the holes in the

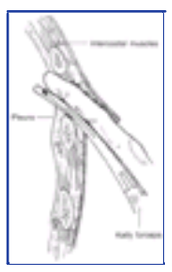

Figure 10-14 Using the finger as a guide to ensure entry into the pleural cavity, one places the tip of the tube into the pleural cavity. *(From Millikan JS, Moore EE, Steiner E: Complications of tube thoracostomy for acute trauma. Am J Surg 140:739, 1980.)*

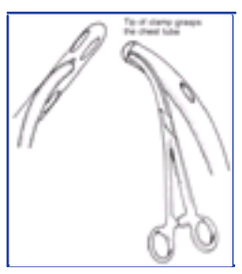

Figure 10-15 The tube is grasped with the curved clamp, with the tube tip protruding from the jaws.

tube are within the pleural space. A common error, especially in obese patients, is to fail to advance the chest tube far enough into the pleural space, leaving the drainage holes in the SQ tissue. The tube can then be rotated 360° to reduce the likelihood of kinking. Although not crucial for initial treatment, the tube can initially be directed inferiorly if only fluid is to be evacuated.

The tube is attached to the previously assembled water seal or suction setup by means of a sterile serrated connector before the clamp is released. Asking the patient to cough and thereafter observing bubbles in the water seal chamber is a good way to check system patency.

Confirmation of Tube Placement

Immediately after tube placement a finger can be passed along the tube to verify that it enters the pleural cavity. Condensation on the inside of the tube and audible air movement with respirations, the free flow of blood or fluid, and the ability of the operator to rotate the tube freely after insertion are also indicators that the tube is in the pleural space.

The definitive assessment of tube placement is the chest radiograph (Fig. 10-16). A simple pneumothorax should resolve within a few minutes of continual suction. If the radiograph continues to demonstrate a pneumothorax, consider that the tube may not be in the pleural cavity, the most proximal hole is outside the chest cavity, or a large air leak from the tracheobronchial tree may be delaying expansion.

In an emergency it is deceptively easy to advance a chest tube in a SQ plane outside of the pleural space, usually a course that follows the ribs posteriorly. Using a finger in the pleural space helps negate this action, and the error is easily detected by a radiograph.

The development of palpable SQ air is another complication of chest tube placement. It usually is limited to the insertion site and minimized with proper technique. Patients placed on positive pressure ventilation who have a continued air leak can develop significant disseminated SQ emphysema.

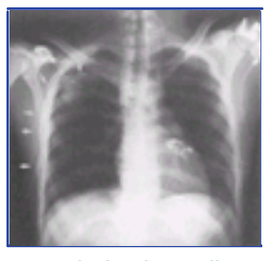

Figure 10-16 Subcutaneous (SQ) placement of a chest tube (*arrows*) can occur, as the tube can dissect through tissue planes with relative ease. If this tube had been directed posteriorly, the radiograph would erroneously "confirm" intrapleural placement despite the tube being SQ throughout its entire course.

Securing the Tube

The chest tube position should be verified on a chest radiograph before definitively securing the tube. Numerous methods to secure a tube are acceptable. One common way to secure the tube is to sew it to the skin with large 0 or 1-0 silk sutures. Nylon sutures are acceptable and more easily found, but they tend to slip on the surface of the chest tube. One common method is to use the "stay" suture. With this technique the same suture that is used to close the skin incision is also used to hold the tube (Fig. 10-17). After this suture is used to close the skin incision at the site of tube insertion, the ends are left long. Then they are wrapped and tied

repeatedly around the

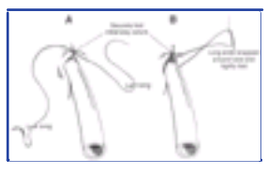

Figure 10-17 A "stay" suture is first placed next to the tube to close the skin incision. *A,* The knot is tied securely and the ends, which will subsequently be wrapped around the chest tube, are left long. *B,* The ends of the suture are wound twice about the tube tightly enough to indent the tube slightly and are tied securely.

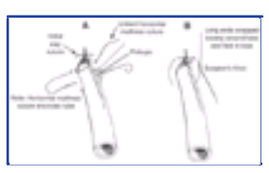

Figure 10-18 *A,* A horizontal mattress suture is placed around (above and below) the tube and is held only with a surgeon's knot. *B,* The loose ends are also wrapped around the tube and are tied loosely in a bow to identify the suture. This suture will be untied and used to close the skin incision after tube removal.

chest tube and knotted securely. The sutures must be tied tightly enough to indent the chest tube slightly to avoid slippage. If the skin incision is not closed tight around the tube, an additional stay suture is used, or a simple suture is used.

At this point it is advisable to place a suture that can both help close the skin around the tube and subsequently close the incision after the chest tube is removed. A *horizontal mattress suture* is placed approximately 1 cm across the incision on either side of the tube, essentially encircling it (Fig. 10-18). This is closed with a knot that can be easily untied since it will be opened and retied to close the incision after the tube is removed.

An occlusive dressing of petrolatum-impregnated gauze should be applied where the tube enters into the skin. This should then be covered with two or more gauze pads with a Y-shaped cut from the middle of one side to the center. These are oriented at 90° to each other (Fig. 10-19). The shaved skin and the tube may be coated with tincture of benzoin and wide (8- to 9-cm) cloth adhesive tape used to hold the tube more securely in place.

The tube can be further secured with tape or elastic adhesive dressing by taping around the tube and pressing the tape

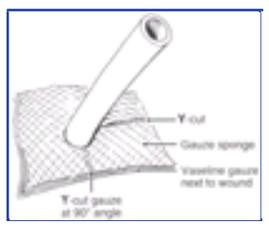

Figure 10-19 A dressing consisting of gauze sponges with a Y-cut is applied to the entry site to provide an air-tight seal. Two pieces are placed at angles.

together for 1 to 2 cm before applying the tape to the chest wall. Another method is to use two strips of tape applied with an "elephant-ear" technique at 90° to each other (Fig. 10-20). The tape is torn so that one end is split into three pieces extending halfway to the center. The two outside pieces are placed on the skin on either side of the tube site, and the center section is wrapped tightly around the tube. This is repeated with a second piece of tape placed at 90° to the first. Also securely tape the tube connections.

Drainage and Suction Systems

A basic understanding of the functions of chest tube drainage systems is necessary to prevent life-threatening complications associated with their use. Modern suction devices are much safer and easier to regulate than the old multi-bottle systems, but the older systems better demonstrate their mechanism of action. The self-contained apparatus used currently is merely a high-tech version of the old bottle system. There are two essential components to all systems: a one-way valve to allow air or fluid to drain out of the pleural space without allowing air back into the pleural space, and a suction mechanism to increase the rate of drainage. The following are general requirements for a drainage/suction system.

The simplest drainage device is a one-way valve without suction. This can be accomplished by either an underwater seal or with a flutter (Heimlich) valve attached to the end of the chest tube (Fig. 10-21). These valves allow one-way flow of air out of the chest but collapse during inspiration to prevent air from passing back into the chest. Normal respiration and coughing will create enough pressure to remove the excess air from the pleural space, and the lung will then expand. The Heimlich valve does not require suction and it may be used by outpatients. In reality, however, this theoretical advantage is rarely used by the emergency clinician.

Another simple device is the underwater seal method. In this system the intrapleural fluid or air exits under a small amount of water (the seal) and collects into a single collecting reservoir (Fig. 10-22). The intrathoracic pressure must be greater than the water pressure over the distal exit in the immersed tube to allow intrathoracic air or fluid to be expelled into the bottle. The water pressure that must be overcome to allow the expelling of air or fluid into the collecting device is the height of the water above the exit port on the tubing. Enough intrapleural pressure is easily accomplished with simple coughing unless the height of the fluid in the collecting apparatus becomes excessive. The water above the tube

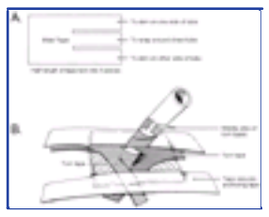

Figure 10-20 *A*, The distal half of a wide piece of tape is longitudinally split into three pieces. The two outside pieces are placed on the skin on either side of the tube, and the center strip is wrapped around the chest tube itself. *B*, This process may be repeated with a similar piece of tape placed at a 90° angle. The tape is securely anchored to the skin (benzoin is optional, but the skin must be clean and dry), and the torn tape is wrapped around the tube. Each anchoring piece is covered by another piece of tape.

acts as a seal because it is too heavy to be drawn back into the chest. This obviously requires that the collecting bottle be below the patient, usually on the floor. Inspiration cannot generate enough negative pressure to pull the collection bottle contents from the floor to the chest cavity. As the height of the collecting bottle is raised, less negative inspiratory force is required to suck the fluid in the bottle back into the chest. The problem with this system is that as the fluid collects in the reservoir, increasing intrathoracic pressure is needed to overcome the fluid pressure. To overcome this problem a two-bottle system is usually used (Fig. 10-23). The chest tube is connected to a collection reservoir that is then linked to a second bottle with 5 cm of water in the bottom. Suction can be applied to the two-bottle system by connecting a third connected chamber.

Suction is used initially for all patients with chest tubes placed for either pneumothorax or hemothorax. The suction device should have high suction flow (up to 20 L/min) and be able to keep the suction constant. A wall suction of 20 cm H_2O is normally used. Suction is useful for rapid initial expansion and drainage, but because of potential complications it should be replaced by a water seal once the expansion

Figure 10-21 A one-way Heimlich valve alone is usually sufficient to treat a pneumothorax, but should *not* be used to treat a hemothorax.

is satisfactory and there are no persistent air leaks. Suction can be added to a two-bottle system with a third bottle that is attached to an external suction (negative pressure) source (Fig. 10-24). *The amount of suction in the chest tube is dependent on the depth of water in the water seal reservoir, not by the suction from the wall valve.* When negative pressure from the suction source exceeds the depth of the water in the chamber, air enters from the top of the third tube, causing continuous bubbling. This prevents a further pressure increase in the chest tube. Since the suction applied to the end of the chest tube is dictated by the height of water in the container, and not by the dial on the wall suction, the loud bubbling heard from a chest tube collection device does not mean that

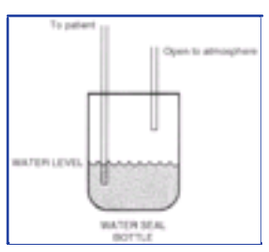

Figure 10-22 Single-bottle (water seal) collection device.

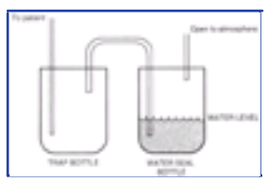

Figure 10-23 A two-bottle system. The "trap" reservoir proximal to the water seal keeps the accumulating drainage from affecting the water seal pressure.

suction is increased. The wall suction dial can be turned down until only occasional bubbling can be detected.

Current commercial systems provide "air leak chambers" to indicate the amount of an intrapleural air leak (Fig. 10-25). Bubbling in this chamber indicates the presence of an air leak, either in the drainage system or from the lung parenchyma. If an air leak is suspected from a lung injury, first confirm that the tube is not leaking, that its connections are tight, and that all holes of the chest tube are within the thorax. If the air leak is from the patient it is usually seen only with expiration or with coughing (forced expiration). Continuous air leaking, or a leak seen during inspiration, indicates a larger and possibly more significant injury. [14] Surgical intervention is indicated if an air leak persists for >72 hours or the lung is not completely reexpanded.

When the tube is functioning properly, the height of the fluid level in the drainage tube fluctuates with the respiratory cycle. Respiratory fluctuations can increase if the patient is making increased inspiratory effort due to airway obstruction or atelectasis. An absence of respiratory fluctuation or a decrease in the drainage implies that the system is blocked or that the lung is fully expanded. Thoracostomy tubes draining fluid are prone to blockage. If the tube is blocked, the chest tube or collecting tubing, or both, can be changed, "milked,"

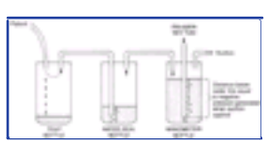

Figure 10-24 A 3-bottle system with fluid trap bottle, water seal, and adjustable vent tube. The height of the column of water in bottle 2 regulates the amount of suction applied, independent of the pressure on the suction valve. *(From Miller KS, Sahn SA: Chest tubes: Indications, technique, management and complications. Chest 91:258, 1987.)*

or "stripped" to dislodge clots. Although replacing the tube is a complicated process, the routine use of milking and stripping should be used sparingly because of the potentially high pressures generated. If the blockage is within the thorax, the tube can be *milked* by forcing air or fluid back into the chest. The tube must be clamped distally and then compressed and stripped to force the contents proximally. *Stripping* is the opposite maneuver in that the tube is clamped proximally and progressively compressed distally followed by a release to allow the tube to spring open. The sudden increase in negative pressure may extract clots and fluid from a more proximal location.

In any system the drainage reservoir must remain below the level of the chest to prevent the fluid in the collection system from reentering the chest. Simple respirations do not generate enough negative intrathoracic pressure to pull the water in the reservoir up to the height of the chest if the reservoir is kept on the floor. The length of the tubing must be sufficient to keep the reservoir below the level of the patient, but not long enough to cause it to form dependent loops of fluid or kinks. These dependent fluid loops require greater intrapleural pressure to drain. If they are high enough (15 to 25 cm H_2O), they cause a tension pneumothorax (Fig. 10-26). Similarly, as fluid accumulates in the water seal reservoir of a single bottle system, the immersed tip of the tube must be raised so that it stays 2 to 4 cm below the water surface to keep the pressure constant.

Occlusive clamping of chest tubes should only be performed with close clinician supervision. If an intrathoracic air leak is still present, clamping the tube may cause a tension pneumothorax. Patients with chest tubes in place are best transported with a Heimlich valve or water seal only, not with a clamped tube. Clamping the chest tube as a trial maneuver before removal of the tube is discouraged.

Prophylactic Antibiotics

The use of prophylactic antibiotics for patients with a chest tube placed in the ED is controversial. There is no agreed-on standard of care. Small controlled studies demonstrate a trend toward reduced empyema and pneumonia in patients treated with tube thoracostomy for penetrating trauma when cefonicid or cefazolin has been given at the time of chest tube

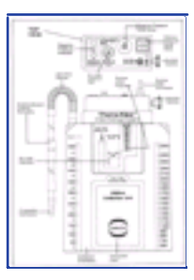

Figure 10-25 Diagram of a commercially available chest drainage system. Included is a 2500-mL fluid collection chamber, a suction control device, an air leak indicator, and a fluid sampling port. A separate attachment is available for autotransfusion. *(Courtesy of Davol, Inc, a division of CR Bard, Cranston, RI.)*

insertion and continued until removal. It would seem reasonable to administer prophylactic antibiotics to patients who receive an emergency and possibly nonsterile thoracostomy. [15] [16] The antibiotic chosen should cover *Staphylococcus aureus*.

It is less clear whether patients with blunt chest trauma or spontaneous pneumothorax who receive their thoracostomy under controlled and sterile conditions require antibiotic prophylaxis. Generally prophylactic antibiotics are not used under these circumstances.

Tube Removal

Chest tubes are generally removed when fluid loss is <200 mL/day and no air leak exists for a minimum of 24 hours. For pneumo- and hemothorax the use of preremoval radiographs is not necessarily recommended. For empyema the removal depends on the clinical and radiographic resolution of the infection.

To remove the chest tube, place the patient in a semi-upright position and remove the dressings. Prepare and drape

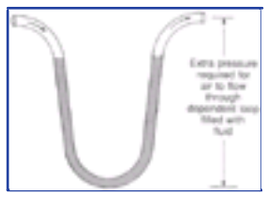

Figure 10-26 Dependent loops of fluid-filled tubing require positive intrapleural pressure greater than the vertical height of the fluid-filled loop for drainage to occur. *(From Batchelder TL, Morris KA: Critical factors in determining adequate pleural drainage in both the operated and non-operated chest. Am Surg 28:298, 1962.)*

the insertion site and follow sterile technique. If a pursestring suture was placed at the time of the insertion, then only sterile scissors are needed to cut the suture. If there is no pursestring suture, then suturing equipment will be needed to close the wound once the tube is removed. Additional equipment should be available to reinsert a chest tube if the lung collapses. A petrolatum- or antibiotic-impregnated gauze dressing should be prepared for covering the wound.

The pursestring suture that was previously placed should be loosened and readied for closing the wound. Then the skin loop of the suture holding the tube to the skin should be cut and removed from the skin. The tube should be clamped to prevent leakage of body fluids and then disconnected from the connecting tubing. The patient should inhale fully and perform a mild Valsalva maneuver. The tube is pulled out in one swift motion while the patient holds the breath. The pursestring suture is quickly tied and then covered with the occlusive dressing. The patient should be observed for 2 to 6 hours, with a chest radiograph obtained before discharge. Any increase in symptoms requires prompt reevaluation. After 48 hours, the dressing may be removed. Sutures may be removed in 7 to 10 days.

OTHER TECHNIQUES

Minicatheter Insertion

A less invasive alternative to traditional tube thoracostomy for patients with a simple pneumothorax is treatment with a minicatheter. Advantages of this technique include the ease of catheter insertion, decreased patient discomfort, less scarring, and decreased cost. After successful reexpansion of the lung, selected patients may be treated as outpatients with a Heimlich valve. Healthy patients with iatrogenic pneumothorax (e.g., following central line attempts, IV drug injection), victims of minor nonpenetrating chest trauma, and patients with spontaneous pneumothoraces are good candidates for catheter aspiration of the pneumothorax. Patients with underlying lung pathology, such as pneumonia, CHF, asthma, or emphysema, are generally not candidates for minicatheter use. The technique is somewhat less effective in patients with a spontaneous pneumothorax because of the continued air leak from ruptured blebs, but it has been 80% effective regardless of the size of the pneumothorax, and is associated with decreased pain and a shorter hospital stay.[17]

There are many protocols for using catheter aspiration as the first step in treating simple pneumothoraces. In general, patients with successful aspiration are observed in the ED for 4 to 6 hours, and if a repeat radiograph shows no reaccumulation of air, the catheter is removed. After 2 more hours of observation, another chest radiograph is obtained, and the patient is released if there is no recurrent pneumothorax. Patients with continued residual pneumothorax often receive a conventional tube thoracostomy, although selected patients can have the catherter attached to suction and admitted. Usually this catheter clogs or becomes nonfunctional in 24 to 48 hours because of the small lumen. This protocol leads to about a 60% success rate with simple aspiration. An additional 30% may respond to the use of a Heimlich valve with or without suction. About 15% of patients will eventually require a formal chest tube. Outpatient therapy consisting of a small catheter with an attached Heimlich valve may be the best strategy in select patients with a first-time spontaneous pneumothorax.

Some catheters are designed specifically for aspirating a pneumothorax. They are made of a soft, flexible, thrombosis-resistant, radiopaque material with multiple distal side ports. The pigtail catheter system is ideal for this procedure (Fig. 10-27) because its multiple holes help prevent obstruction or adherence to the pleura when under suction. A 14-ga IV catheter or an 8.5 Fr Arrow trauma catheter can also be inserted using the Seldinger (guidewire) technique. However, these catheters have only single distal holes, which can easily become obstructed or adhere.

Placement of a catheter into the pleural space is relatively simple. The patient is placed in a semi-upright position and the skin is prepared with appropriate antiseptic solution at either the fourth or fifth intercostal space at the anterior axillary line or the second or third intercostal space at the midclavicular line. A generous local infiltration of lidocaine is used for anesthesia. The exact technique depends on the type of equipment used, but most commonly it is a guidewire technique.

Guidewire (Seldinger) Technique for Catheter Aspiration

After skin preparation and sterile draping, a thin-walled 16-ga needle is advanced cephalad over the top of the rib at a 60° angle (Fig. 10-28). No attempt should be made to tunnel this tract since floppy catheters will not be easily passed unless they are advanced in a straight line. When the pleural space is identified by the aspiration of air into the syringe, the advancement of the needle is halted. A guidewire is inserted into the pleural space through the needle. Then the needle is removed while stabilizing the guidewire to prevent it from being pulled out. Now only the guidewire remains in the pleural space. A small incision is made in the skin with a No. 11 blade at the base of the wire to allow passage of the catheter through the skin. If the pigtail catheter system is used, a dilator is passed over the wire to clear a path through the soft tissues for the catheter. If a minicatheter is used, it has its own dilator/introducer. The minicatheter with an introducer is then threaded over the guidewire into the pleural space. The wire and dilator are removed, leaving the catheter in the pleural space. A three-way stopcock is attached to the catheter and air is then aspirated with a 60-mL syringe until resistance is felt. Alternatively, suction is used, since a number of aspirations may be required until all air exits. The catheter is then secured to the skin. Obtain a chest radiograph to determine if the lung is fully expanded. If residual pneumothorax is present, further aspirations can be attempted. If air cannot be aspirated, the catheter may be kinked or blocked with soft tissue. To relieve the blockage, place the patient in the full upright position and have him or her cough or take a deep breath. Alternately, the catheter can be withdrawn slightly or twisted gently.

Over-the-Needle Technique

Some commercial kits use a catheter over a long needle (Fig. 10-29). After preparation and draping, a small incision is made in the skin with a No. 11 blade. While constant suction is applied with an attached syringe, the needle and catheter combination is slowly advanced into the pleural space. When air is aspirated, the needle is no longer advanced and the catheter is then slid off the needle into the pleural space, ensuring that all side ports are within the pleural space. The catheter is then connected to a three-way stopcock, and air is aspirated with a syringe as described earlier.

Trocar Insertion

Chest tube insertion with a trocar is rarely used because of the risk of major complications to the heart, lung, and solid organs. Some clinicians use a trocar to stabilize the tube during careful guided insertion through a skin incision, but the technique of perforating the chest wall with a trocar/chest tube apparatus is discouraged.

TUBE THORACOSTOMY IN PEDIATRIC PATIENTS

Pneumothorax can occur in the neonatal population, often associated with resuscitative measures (such as mechanical ventilation) for meconium aspiration or prematurity. For the rest of the pediatric population, trauma is the most common cause.

The physical examination of the newborn with pneumothorax can vary from no abnormalities to cardiovascular collapse. Making this diagnosis in a newborn by physical examination alone is a great challenge and usually a radiograph is required because of the multiple causes of respiratory distress. Grunting respirations and tachypnea are often seen. Retractions or nasal flaring can be seen, and crepitus in the neck may be present. Cyanosis may be present or may occur only with crying or feeding. Irritability, restlessness, apneic periods, bradycardia, or tachycardia may be the only manifestation. Distention and tympany of the affected side may be found. A decrease in breath sounds is difficult to appreciate in any child because of the wide transmission of breath sounds throughout the chest and upper abdomen. With a tension pneumothorax, the cardiac impulse and the trachea may be shifted away from the affected side. The definitive diagnosis is made with radiographs taken in the AP and cross-table lateral projections. Small pneumothoraces may be seen

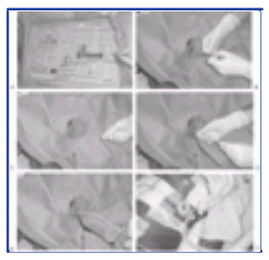

Figure 10-27 Aspiration of a pneumothorax (caused by subclavian vein catheterization) with an Arrow 14 Fr Percutaneous Cavity Drainage Catheterization Kit. This 23-cm pigtail multi-hole catheter is ideal for such purposes. Air can be aspirated from the catheter part with a syringe or the catheter can be attached to suction or a Heimlich valve. This catheter is not used for patients on a ventilator, those with continuing air leaks, or with a hemothorax. It is ideal for stable patients who have a primary pneumothorax or a collapse that can be expected to be stable if the lung is reexpanded (such as intravenous drug use induced, minor blunt trauma, secondary to central venous catheter insertion). *A*, Seldinger-type catheter kit demonstrating pigtail catheter and all necessary equipment, including local anesthesia, introducing needle and syringe, scalpel, guidewire, and dilator. *B*, After generous local anesthesia, the introducing syringe is advanced in *a straight line over the top of the fifth rib* until air is aspirated. Unless a straight track is created it will be difficult to advance the floppy catheter, and a tunneling approach cannot be used. *C*, The guidewire is advanced into the pleural space and the introducing needle is removed. *D*, Following puncture of the skin at the site of wire insertion with a scalpel, a dilator is advanced over the wire to create a track for the catheter. *E*, The pigtail catheter is advanced over the wire through the dilated tract, assuming its pigtail configuration when it is in the pleural space. A twisting motion may be needed to advance the catheter through the subcutaneous tissues. *F*, The catheter is advanced to the hilt and secured to suction. This catheter may be removed after a period of observation or the suction may be maintained for a few days. If used for a few days, the catheter will become clogged with mucus or blood, which may be cleared by injecting sterile saline through the device.

Figure 10-28 Seldinger technique for aspiration catheter insertion. *A*, Guidewire is passed through a needle over the rib into the pleural space. *B*, The needle is removed with the wire in the pleural space. *C* and *D*, A nick is made in the skin with a No. 11 blade, and the introducer and catheter are threaded into the pleural space. A twisting motion may be helpful. *E*, The guidewire and introducer are removed, leaving the catheter in the pleural space.

only on the lateral view, as the air collects at the top of the thoracic cavity.

In general, tube thoracostomy is the treatment of choice once a symptomatic pneumothorax is detected in infants. When signs of tension pneumothorax are present, immediate aspiration with a plastic catheter over-the-needle device is recommended. Small pneumothoraces (<20% of the hemithorax) in relatively asymptomatic infants (i.e., those who are without other problems and who do not require positive airway pressures) can be observed.

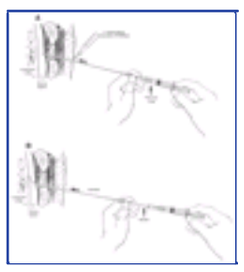

Figure 10-29 Over-the-needle catheter aspiration. *A,* A small nick is made in the skin with a No. 11 blade. The over-the-needle catheter is inserted through the nick into the pleural space. Proper placement is confirmed by the free flow of air into the attached syringe. *B,* The catheter is then threaded over the needle into the pleural space, and the needle is withdrawn.

Of all children with thoracic trauma, approximately one third will develop a pneumothorax, of which one third are isolated injuries. The high percentage of associated injuries in children with a pneumothorax also increases their mortality. [18]

The technique of tube thoracostomy in pediatric patients is essentially the same as that for adults, although an infant's size renders this procedure a formidable one. A successful placement should be viewed as technical accomplishment. The major difference lies in the size of the chest tube. No. 8 to 10 Fr catheters are used in premature infants (<3 kg), and No. 10 to 12 Fr catheters are used in newborns (3 to 5 kg). The size of the tube required increases with the patient's weight

TABLE 10-5 -- Approximate Pediatric Chest Tube Size by Weight in Kilograms

Weight (kg)	Chest Tube (Fr)
<3	8–10
3–5	10–12

6–10	12–16
11–15	17–22
16–20	22–26
21–30	26–32
>30	32–40

(Table 10-5). Various tube locations have been proposed, but recent studies have demonstrated the potential for breast deformities in adults if a midclavicular approach is used. Therefore it is recommended that in newborn infants chest tubes be inserted in the anterior axillary line through the fifth intercostal space after a skin incision 4 to 5 cm below the mammilla.[19] The other important difference is that for a

TABLE 10-6 -- Complications of Tube Thoracostomy

Infection
Pneumonia
Empyema
Local incision infection
Osteomyelitis
Necrotizing fasciitis
Bleeding
Local incision hematoma
Intercostal artery or vein laceration
Internal mammary artery laceration (with midclavicular line placement)
Pulmonary vein or artery injury
Great vessel injury (rarely)
Laceration or puncture of nerves or solid organs
Lung, liver, spleen, diaphragm, stomach, colon, long thoracic nerve, intercostal nerve, intercostal muscle
Mechanical problems
Chest tube dislodgment from chest wall
Incorrect tube position
Subcutaneous placement
Intra-abdominal placement
Air leaks
Leaks from tubing or drainage bottles
Last tube port not within pleural space
Leaks from skin site
Flow of drainage bottle contents into chest from inadvertent elevation of drainage bottles
Blocked drainage
Kinked chest tube or drainage tubes
Clots
Miscellaneous
Allergic reactions to surgical preparation or anesthesia
Pulmonary atelectasis
Persistent pneumothorax
Retained hemothorax
Clotted hemothorax or fibrothorax
Subcutaneous or mediastinal emphysema
Reexpansion pulmonary edema
Reexpansion hypotension
Recurrence of pneumothorax after chest tube removal

pneumothorax the tube should be directed anteriorly when inserted into the pleural space.

COMPLICATIONS

In general, if a chest tube is not functioning properly and the patient is deteriorating, the tube should be removed and inserted again, or another tube should be inserted.

The most common complications of chest tube insertion include infection, laceration of an intercostal vessel, laceration of the lung, and intra-abdominal or solid organ placement of the chest tube (Table 10-6). Local infection at the insertion site is common and related to the emergency nature of the procedure.

Intercostal arteries or veins may be lacerated, but can be minimized by using blunt dissection and carefully directing the tube just above the rib. The tube may adequately tamponade such bleeding, but sometimes the incision needs to be extended to ligate the bleeding vessel. If bleeding continues, a thoracic surgeon should be consulted. Injury to the lung and solid organs can be avoided by not using a trocar insertion technique and by carefully digitally exploring the pleura space before inserting the tube.

Failure of reexpansion of a pneumothorax may be due to a mechanical air leak, but it may also indicate a bronchopleural fistula, a continued parenchymal lung leak, or a bronchial injury. Tension pneumothorax can occur if a blockage in the drainage system at any point is associated with an air leak from the lung. Retained hemothorax may result from clotting or poor tube function. Reinsertion or placement of a second tube may be indicated if the first tube is not functioning properly. Often, an angled tube in the posterior diaphragmatic sulcus will promote drainage of a dependent fluid collection.

Reexpansion Pulmonary Edema.

The reexpansion of a pneumothorax or the evacuation of pleural fluid or blood has rarely been associated with unilateral pulmonary edema of the affected lung. The condition is rare, unpredictable, and may be severe. Pulmonary edema may be very symptomatic or merely a curiosity on the follow-up radiograph. Fatalities have been reported.[20] The condition may occur shortly after reexpansion or be delayed a number of hours. A common factor in these cases seems to be the prolonged period of time between pneumothorax and onset of treatment, but the exact time frame is quite variable. Usually the pneumothorax has been present at least 3 to 4 days. The proposed mechanisms include anoxic damage to the alveolar-capillary basement membrane from prolonged pulmonary collapse, loss of surfactant, or rapid fluid shifts. It has been theorized, but not substantiated, that reexpansion pulmonary edema may be ameliorated by a gradual, rather than sudden, evacuation from the pleural space. This is accomplished by removing air in small aliquots over 24 to 48 hours, rather than attaching the tube directly to suction. Treatment is supportive, occasionally requiring ventilatory support. Reintroduction of air back into the pleural space and temporary occlusion of the ipsilateral pulmonary artery have been other suggested, but unproven, interventions.

Acknowledgments

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Section III - Cardiac Procedures

Chapter 11 - Techniques for Supraventricular Tachycardias

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Bohdan M. Minczak

In controlling or converting supraventricular tachycardias (SVTs) (Table 11-1), there are three avenues of therapeutic approach the clinician may use to achieve control. One is the time-honored use of the vagal reflex; the second is the therapeutic use of pharmacological agents; and the third is the monophasic or biphasic electrical cardioversion technique (Table 11-2). Electrical cardioversion is addressed in Chapter 12 .

VAGAL MANEUVERS

Anatomy and Physiology

The physiologic effects of pressure on the carotid sinus have been known for centuries. They were first described in the medical literature in 1799 when Parry wrote a treatise about the "symptoms and causes of syncope anginosa, commonly called angina pectoris." [1] [2] He noted that pressure on the bifurcation of the carotid artery produced dizziness and slowing of the heart. Thus, the term carotid derived from the Greek *karos*, meaning heavy sleep.

The bifurcation of the common carotid artery possesses an abundant supply of sensory nerve endings located within the adventitia of the vessel wall (Fig. 11-1). These nerves have a characteristic spiral configuration, continually intertwining along their course and eventually uniting to form the carotid sinus nerve. The afferent impulses travel from the carotid sinus via Herring's nerve or carotid sinus nerve to

TABLE 11-1 -- Potential Observations with Vagal Maneuvers in the Management of Tachydysrhythmias

1. Vagal maneuvers may slow the atrial rate in ventricular tachycardia (VT) or complete heart block and may therefore demonstrate previously hidden P waves or obvious atrioventricular (AV) dissociation.
2. Abrupt changes in the heart rate without conversion result from increasing AV block with increased vagal effect.
3. Gradual slowing of the ventricular rate suggests the presence of a sinus rhythm. Vagal maneuvers rarely decrease AV conduction in the presence of a sinus mechanism.
4. The dysrhythmias most likely to convert to sinus rhythm are paroxysmal atrial tachycardia (PAT) and paroxysmal nodal tachycardia.
5. Dysrhythmias associated with AV conduction defects (PAT with block, atrial flutter, and atrial fibrillation) infrequently convert to a sinus rhythm, but the ventricular rate slows. Rarely, atrial slowing will be sufficient to allow 1:1 AV conduction, which may actually increase the ventricular rate (Fig. 11-12).

the glossopharyngeal nerve (CN IX), thence to the vasomotor center in the medullary area (the *tractus solitarius*) of the brain stem. The vasomotor center is composed of three distinct areas, each with a distinctive function. The vasomotor center is located in the reticular substance of the medulla and in the lower third of the pons. This center transmits efferent impulses downward through the spinal cord and the vagus (CN X) nerve. The efferent impulses, which originate in the medial portion of the vasomotor center, travel along the vagus nerve (CN X) to the sinus node and the atrioventricular (AV) node of the heart. The medial portion of the vasomotor center lies in immediate apposition to the dorsal motor nucleus of the vagus nerve (CN X). These medial portion vasomotor center impulses decrease heart rates. Efferent impulses originating in the lateral areas of the vasomotor center travel along the sympathetic chain to the heart and to the peripheral vasculature (see Fig. 11-1). These sympathetic impulses control either vasoconstriction or vasodilation of the vascular system. A balance between the vasoconstriction and vasodilation maintains proper vasomotor tone. [3] [4]

The afferent nerve endings in the carotid sinus are sensitive to mean arterial pressure and to the rate of change of pressure. *Pulsatile stimuli* are more effective than sustained pressures in evoking a response. Elevated blood pressure stretches the baroreceptors, leading to increased firing of the afferent nerve endings. [3] Conversely, hypotensive states result in a drop in afferent firing, as the carotid sinus baroreceptors are exquisitely sensitive to low blood pressure.

The parasympathetic and sympathetic nervous systems play independent but coordinated roles in the carotid sinus reflex. Increased firing of the carotid sinus results in reflex stimulation of vagal activity as well as in reflex inhibition of sympathetic output. The parasympathetic effect is almost immediate; it occurs within the first second and causes a decrease in heart rate. The inhibited sympathetic effect, which causes a drop in blood pressure through vasodilatation, becomes manifest only after several seconds elapse [5] , and the full blood pressure change may not take full effect until a minute has elapsed. [6] The changes in blood pressure and heart rate are independent phenomena. Epinephrine blocks the reduction in blood pressure, while a fall in heart rate is blocked by the administration of atropine.

A cerebral effect, characterized by a loss of consciousness, was once thought to be due to stimulation of the carotid

TABLE 11-2 -- Selected Vagal Maneuvers and Pharmacologic Agents

A. Vagal maneuvers
1. Carotid sinus massage
Pressure on the carotid sinus
2. Valsalva technique
Forced expiration of air against a closed glottis
3. Apneic facial exposure to cold water (*"cold water diving reflex"*)
Immersion of the face into cold water
4. Oculocardiac reflex (OCR)
The trigeminovagal reflex initiated by pressure on the eyeball
B. Pharmacological agents
1. Digoxin
2. Procainamide
3. Adenosine
4. Amiodarone
5. Calcium channel blockers
6. ß-blockers

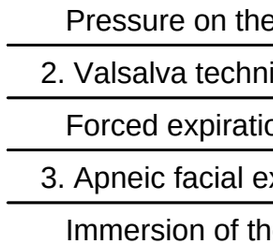

Figure 11-1 Anatomy of the carotid sinus reflex. Carotid receptors send impulses to the medulla by way of the sinus nerve of Hering and cranial nerve IX. Efferent nerves are shown on the right. *(Adapted from Scher AM: Control of arterial blood pressure. In Ruch TC, Patton HD: Physiology and Biophysics, vol 2, 20th ed. Philadelphia, WB Saunders, 1974.)*

sinus. However, it is seen only when sufficient pressure is exerted to occlude the more distal temporal artery pulsation and when contralateral carotid disease is

present. This cerebral effect is now believed to be a result of decreased bilateral cortical perfusion.

The parasympathetic branch of the carotid sinus reflex supplies the sinus node and the AV node. The effect of the parasympathetic stimulation is to slow the heart rate. The sinoatrial (SA) pacemaker is more likely to be affected than the AV node, except when digitalis has been administered. [3] [6] [7]

Indications for Vagal Maneuvers

Vagal maneuvers, and in particular carotid sinus massage (CSM), may be a useful aid to the diagnosis of syncope in the elderly. Some 14% to 45% of elderly patients referred for syncope are thought to have carotid sinus syndrome (CSS). [7] [8] [9] CSS is defined as an asystolic pause greater than 3 seconds or a reduction of systolic blood pressure >50 mm Hg in response to CSM. It shares many characteristics with sick sinus syndrome, suggesting that both are manifestations of the same disease. CSS causes cerebral hypoperfusion leading to dizziness and syncope. Analysis of patients with the syndrome indicates that it results from a baroreflex-mediated bradycardia in 29% of patients, hypotension in 37%, or both in 34%. [10] [11] Therefore, syncope, near-syncope, or a fall of unclear etiology in the elderly are important indications for diagnostic CSM. [12]

The clinician can take advantage, however, of the similarity of the vagal effects of digoxin and the vagal maneuvers. Before starting digoxin administration in a patient, the practitioner can gauge the cardioinhibitory effect that will be achieved with the drug by first performing CSM. Significant slowing or block with CSM suggests a similar sensitivity to digoxin, and a smaller loading dose should be considered.

Vagal maneuvers are also indicated in settings in which slowing conduction in the SA or AV node could provide useful information (Fig. 11-2 Fig. 11-3 Fig. 11-4 Fig. 11-5 Fig. 11-6 Fig. 11-7 Fig. 11-8 Fig. 11-9). These settings include patients with wide-complex tachycardia in whom CSM aids in the distinction between SVT and ventricular tachycardia (VT). CSM can also elucidate narrow-complex tachycardia in which the P waves are not visible, or aid in detection of suspected rate-related bundle branch block or suspected pacemaker malfunction. After CSM, a wide-complex SVT may be converted to normal sinus rhythm, P waves may be revealed after increased AV node inhibition, or ventricular complexes may narrow as the ventricular rate slows. Because CSM slows atrial and not ventricular activity, AV dissociation may be more easily seen, indicating VT (Fig. 11-2). In rapid atrial fibrillation, or atrial flutter with 2:1 block, either P waves or irregular ventricular activity with absent P waves may be revealed. Sinus tachycardia may also be more apparent once P waves are unmasked by slowing the SA node (see Fig. 11-4). Adenosine may be used for the same diagnostic purpose in these situations as well. [13] In order of decreasing frequency, the electrocardiogram (ECG) changes seen with CSM and vagal maneuvers include those shown in Table 11-3 .

Equipment and Setup

As a precaution against hypotension and life-threatening dysrhythmias, an IV line with normal saline is often started before attempting any vagal maneuver, including the use of pharmacologic agents or electrical cardioversion. The patient should be placed on a cardiac monitor and pulse oximeter.

Figure 11-2 Ventricular Tachycardia. Carotid sinus massage (CSM) slows atria but not ventricles, thus establishing the presence of atrioventricular dissociation, supporting the diagnosis of ventricular tachycardia. The QRS measures 0.16 sec. Note the atrial rate slowing from 102 to 88 beats/min while the ventricular rate is unaffected. *(From Lown B, Levine SA: Carotid sinus—Clinical value of its stimulation. Circulation 23:766, 1961. Reproduced by permission.)*

Atropine and lidocaine, as well as a transvenous or transcutaneous pacemaker and defibrillator, should be readily available at the bedside. Oxygen and appropriate delivery systems plus standard airway management equipment should be readily available. The patient should be in the supine or *slight* reverse Trendelenburg position if it can be tolerated. Occasionally SVT will convert merely by lowering the back of the bed, presumably because the supine position results in a stretching of the carotid bulb, giving maximum baroreceptor sensitivity. The supine position may also prevent syncope in the event of a significant drop in heart rate or blood pressure.

Carotid Sinus Massage

Carotid sinus massage (CSM) is a bedside vagal maneuver technique involving digital pressure on the richly innervated carotid sinus. It takes advantage of the accessible position of this baroreceptor for diagnostic and therapeutic purposes. Its main diagnostic utility is in the differential diagnosis of syncope in the assessment of tachydysrhythmias and rate-related bundle branch blocks, and in clues to latent digoxin toxicity. Its main therapeutic application is for termination of SVTs due to paroxysmal atrial tachycardia (PAT).

A diagnostic indication for CSM is evaluating digoxin toxicity. Toxicity from digoxin depends more on the response of the host than on the actual digoxin level. In cases of suspected digoxin toxicity, before the level is available, or when the digoxin level is in the "normal range," CSM may be a useful diagnostic adjunct. Significant inhibition of AV node conduction associated with ventricular ectopy (see Fig. 11-9), especially ventricular bigeminy, should lead to the suspicion of digoxin toxicity. [1]

Other therapeutic uses of CSM have been made obsolete by current medical therapy. In 1961, Lown and Levine

Figure 11-3 Paroxysmal atrial tachycardia with variable block. Carotid sinus pressure uncovers P waves hidden in the ventricular complex. The upper strip resembles atrial flutter or atrial fibrillation with ventricular ectopic beats. The lower strip shows paroxysmal atrial tachycardia with variable block at an atrial rate of 166 beats/min. *(From Lown B, Levine SA: Carotid sinus—Clinical value of its stimulation. Circulation 23:766, 1961. Reproduced by permission.)*

described the dramatic effect CSM had in the 1920s on relieving acute pulmonary edema in a group of patients with hypertension and coronary artery disease. They reported: "Relief is immediate and coincides with the onset of bradycardia. In the majority, it is associated with a drop in blood pressure. The patient is promptly able to lie flat. Fear, dyspnea, and chest oppression disappear. ..." CSM also has been reported to relieve anginal pain. The technique may be useful when the diagnosis of angina is uncertain. [14] The advantage of the CSM technique over the use of nitroglycerin is unknown. Although CSM is no longer the first approach to either pulmonary edema or angina, it remains a therapeutic or adjunct diagnostic tool in some cases, or when modern pharmacological agents are unavailable. Because adenosine may not always be readily available, and because adenosine cannot be used to assess the sensitivity of the carotid sinus, CSM remains a useful bedside tool.

Contraindications

CSM is *contraindicated* in patients likely to suffer neurologic or cardiovascular complications from the procedure. Patients with a carotid bruit should not have CSM because of the risk of carotid embolization or occlusion. A recent cerebral infarction is a theoretical relative contraindication, because marginal reduction of cerebral blood flow may produce further infarction.

The presence of diffuse, advanced coronary atherosclerosis is associated with increased sensitivity of the carotid sinus reflex. This hypersensitivity is further augmented during an anginal attack or an acute myocardial infarction. Brown and coworkers found that the degree of carotid sinus hypersensitivity was directly proportional to the severity of coronary artery disease documented by cardiac catheterization. [18] Patients with acute myocardial ischemia or with recent

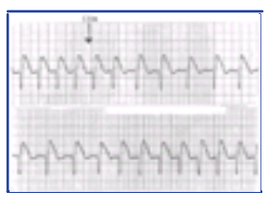

Figure 11-4 Sinus tachycardia. The sinus P wave is obscured within the descending limb of the T wave. Carotid sinus massage (CSM) transiently slows the sinus rate and exposes the P wave. The rate then increases. The strips are continuous. *(From Silverman ME: Recognition and treatment of arrhythmias. In Schwartz GR, Safar P, Stone JH, et al (eds): Principles and Practice of Emergency Medicine, vol. 2. Philadelphia, WB Saunders, 1978. Reproduced by permission.)*

myocardial infarction are already at higher risk of VT or ventricular fibrillation (VF). A CSM-induced, prolonged asystole may further predispose them to these dysrhythmias. Therefore, CSM should be avoided in these patients when pharmacologic alternatives are available.

Both digoxin and CSM act through a vagal mechanism to inhibit the AV node. Patients on digoxin may experience a greater inhibition of the AV node with longer AV block as a result. Patients with *known* digoxin toxicity should not have CSM, as AV inhibition may be profound. [19] However *cautious* CSM (as noted above) may be a useful diagnostic tool when the diagnosis is less certain.

Simultaneous bilateral CSM is absolutely contraindicated, because cerebral circulation may be severely compromised. Before attempting CSM, the clinician should first auscultate for carotid bruits on both sides of the neck. The presence of a bruit is a contraindication to massage.

Technique

The clinician should begin CSM on the patient's right carotid bulb, as some investigators have found a greater cardioinhibitory effect on this side [12] [15] [16] ; although no difference was found in one study.[10]

Keeping the patient relaxed is helpful for two reasons: A tense platysma muscle makes palpation of the carotid sinus

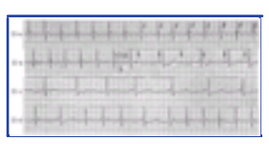

Figure 11-5 Sinus tachycardia with high-degree atrioventricular block. Arrows indicate sinus P waves. Strips II-a to II-d are continuous. The basic rhythm is sinus, but marked first-degree atrioventricular block is present. High-degree (advanced) atrioventricular block associated with transient slowing of sinus rate is produced by carotid sinus stimulation (CSS). *(From Chung EK: Electrocardiography, 2nd ed. New York, Harper & Row, 1980. Reproduced by permission.)*

difficult, and an anxious patient will be less sensitive to CSM as a result of heightened sympathetic tone.

With the head tilted backward and slightly to the opposite side, palpate the carotid artery just below the angle of the mandible at the upper level of the thyroid cartilage and anterior to the sternocleidomastoid muscle. Once the pulsation is identified, use the tips of the fingers to administer CSM for 5 seconds in a posteromedial direction, aiming toward the vertebral column. Although earlier practitioners used a longer duration of massage, a shorter period of massage minimizes the risk of complications and is adequate for diagnostic purposes in the majority of patients. [17] Pressure on the carotid sinus may be steady or undulating in intensity; the force, however, must not occlude the carotid artery. The temporal artery may be simultaneously palpated to ensure that the carotid remains patent throughout the procedure.

If unsuccessful, CSM may be repeated after 1 minute. If the procedure is still unsuccessful, the opposite carotid sinus may be massaged in a similar fashion. Simultaneous Valsalva maneuvers may also enhance carotid sinus sensitivity.

Complications

Neurologic complications of CSM are rare and are usually transient. In one review of neurologic complications in elderly

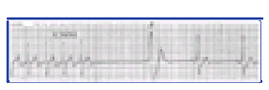

Figure 11-6 Paroxysmal atrial tachycardia. Carotid sinus massage (CSM) abolishes the dysrhythmia and results in a period of sinus suppression with a junctional (J) escape beat, followed thereafter by a sinus rhythm. Prolonged periods of asystole may produce anxiety in the clinician who is waiting for the resumption of a sinus pacemaker. *(From Silverman ME: Recognition and treatment of arrhythmias. In Schwartz GR, Safar P, Stone JH, et al (eds): Principles and Practice of Emergency Medicine, vol 2. Philadelphia, WB Saunders, 1978. Reproduced by permission.)*

patients undergoing this procedure, Munro and others found 7 complications from a total of 5000 massage episodes, for an incidence of 0.14%. [20] Reported deficits included weakness in 5 cases and visual field loss in two others. In one case, the visual field loss was permanent. Patients in this study were excluded from CSM if they had a carotid bruit, recent cerebral infarction, recent myocardial infarction, or a history of VT or VF. The duration of massage was 5 seconds. Lown and Levine described one patient with brief facial weakness during several thousand tests. [1] Carotid emboli and hypotension have both been implicated as possible causes of the neurologic deficits. Unintentional occlusion of the carotid artery, if the contralateral circulation is impaired, may also be responsible for some neurologic complications.

Cardiac complications include asystole, VT, or VF (Fig. 11-10). A normal pause of <3 seconds is part of the physiologic response to CSM; a longer pause may be diagnostic of carotid sinus syndrome. In a review of reported cases of ventricular tachydysrhythmias, 5 cases were described. [21] All 5 patients were receiving digoxin, and in several cases, VT or VF followed AV block. Digoxin is associated with more prolonged AV block resulting from CSM, perhaps leaving these patients more vulnerable.

Interpretation of Vagal Maneuvers

A pause >3 seconds, or a drop in systolic blood pressure >50 mm Hg in patients to whom CSM is administered while they are in a supine position, is diagnostic of the carotid sinus syndrome (Fig. 11-11). Patients should be supine during testing to reduce the risk of cerebral hypoperfusion. [12] [22]

Although the carotid sinus massage is one of the better known vagal maneuvers, a variety of other physical modalities are available to the clinician to affect a change in heart

Figure 11-7 Atrial flutter. Carotid sinus stimulation (CSS, *downward arrow*) produces marked slowing of the ventricular rate in atrial flutter. Note the obvious flutter waves with an atrial rate of 300 and a long period of ventricular standstill. Strips are continuous. *(From Chung EK: Electrocardiography, 2nd ed. New York, Harper & Row, 1980. Reproduced by permission.)*

rate. The anticipated rhythm response to different vagal maneuvers in the setting of different underlying rhythms is shown in Table 11-4 .

Valsalva Maneuver

In general, mean bradycardia changes are greatest for the Valsalva maneuver and the diving response. [3] [22] [23] During the Valsalva maneuver, intrathoracic pressures are increased, leading to increased arterial pressure. This increased pressure is transferred to the peripheral vascular system. Venous return to the heart is decreased, resulting in a decreased stroke volume. This is followed by increased venous pressure. All of these pressure changes lead to an initial increase in heart rate and carotid sinus pressure. As the maneuver is sustained, vagal tone is increased leading to a compensatory decrease in SA and AV conduction (the desired diagnostic/therapeutic response).

Contraindications

Patients must be able to cooperate with the clinician's commands. Remember that dyspneic or tachypneic patients may not be able to hold their breath for the period of time needed to complete the maneuver.

Technique

The patient is placed supine and a cardiac monitor is attached. Ideally, intravenous (IV) access is secured, and atropine, lidocaine, and defibrillation equipment are available. Have the patient take a deep breath and hold it. Instruct the patient to bear down and try to exhale without allowing the air to leave the lungs. The patient should try to hold this position for 10 to 20 seconds. [24] [25] An adjunct method is to have the patient take a deep breath, hold the breath and try to push the clinician's


Figure 11-8 Atrial fibrillation. Carotid sinus massage (CSM) slows the ventricular response transiently, revealing the fibrillating baseline. The ventricular rate subsequently accelerates. *(From Silverman ME: Recognition and treatment of arrhythmias. In Schwartz GR, Safar P, Stone JH, et al (eds): Principles and Practice of Emergency Medicine, vol. 2. Philadelphia, WB Saunders, 1978. Reproduced by permission.)*

hand off the patient's abdomen as the clinician gently pushes on the anterior wall of the abdomen.

Apneic Facial Exposure to Cold ("Diving Response," Diving Bradycardia) Technique.

This *technique* represents a variation on the simple valsalva maneuver. It has been found useful in children who may be unable to volitionally do a valsalva maneuver. Classically the technique consists of facial immersion, without breathing, for 15 to 30 seconds in cold water (0°C to 15°C). Alternatively ice water can be dripped into the nostril of a small child. Be sure to monitor the oxygen saturation closely when applying this technique to a small child.

The procedure is based on the classic diving reflex of bradycardia. Slowing of SVT to unmask the hidden, underlying rhythm is similar to the effects of CSM. The conversion of paroxysmal atrial tachycardia to sinus rhythm should be observed in 15 to 35 seconds. The procedure is convenient and noninvasive, and can be self-administered.[26] [27] [28] [29] [30] [31]

Oculocardiac Reflex (OCR, Trigeminovagal Reflex)

This reflex is clinically significant during strabismus surgery in children, although the manifestations of this reflex are not consistent. The oculocardiac reflex (OCR) is induced by pressure on the eyeball. Afferent pathway follows the long and short ciliary nerves to the ciliary ganglion. From there, it travels to the gasserian ganglion body along the ophthalmic division of the trigeminal nerve (CN V). The afferent pathway ends in the main trigeminal sensory nucleus in the floor of the fourth ventricle.[4] [23] [24]

Efferent impulses start at the vasomotor center and travel through the vagal nerve (CN X) and the sympathetic chain. Bradycardia results from increased parasympathetic tone.

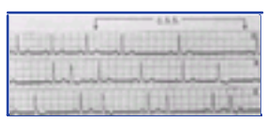
Figure 11-9 Occult premature ventricular contractions. Carotid sinus massage (CSM) reveals ventricular extrasystoles, thereby explaining the cause of palpitation in this case. *(From Lown B, Levine SA: Carotid sinus—Clinical value of its stimulation. Circulation 23:766, 1961. Reproduced by permission.)*

Decreased sympathetic tone causes vasodilation. The cardiac effect stops when eye pressure is relieved. [23] Patients should be monitored to recognize ECG changes. Atropine should be available to reverse life-threatening bradycardia.

Contraindications

Common sense dictates that the clinician should try to elicit history of the following conditions prior to attempting the oculocardiac reflex: (a) recent retinal or lens surgery; (b) glaucoma; (c) thrombotic-related eye conditions, which all seem to be obvious possible contraindications. Care should be taken when pressing on the eye globe to prevent corneal or scleral injury.

Technique

With the eyelid closed, non-rotating pressure is applied to the eyeball for 10 to 20 seconds. There is no advantage to the use of either eye. Ventricular slowing and possible decrease in blood pressure should be observed almost immediately when the pressure is applied. The cardiac effect of bradycardia will cease when pressure is removed. As with all vagal maneuvers, monitoring, IV access, atropine, lidocaine, and defibrillation should be available during the procedure.

Summary

Berk and colleagues have demonstrated in healthy volunteers that cold-water face immersion and the Valsalva maneuver can produce a greater vagal response than CSM.[15] Mehta and colleagues also found that the Valsalva maneuver was more effective than CSM for conversion of induced SVT. [16] [32] The pneumatic antishock garment has also been used to similarly increase vagal tone by stretching the carotid bulb. [33]

TABLE 11-3 -- Order of *Decreasing* Frequency of Electrocardiogram Changes with Vagal Maneuvers

1. SA slowing, occurring in approximately 75% of cases (sinus arrest occurs approximately 3% of the time)
2. Atrial conduction defects, manifested by an increase in width of the P wave on the electrocardiogram
3. Prolongation of the PR interval and higher degrees of atrioventricular block, seen in approximately 10% of cases
4. Nodal escape rhythms
5. Complete asystole, defined as sinus arrest without ventricular escape lasting >3 seconds, occurring in 4% of cases
6. Premature ventricular contractions

SELECT PHARMACOLOGIC AGENTS

Digoxin

This time-honored drug was previously a mainstay for treatment of atrial fibrillation and atrial flutter. It is the only anti-dysrhythmic with inotropic properties, but is uncommonly used by the emergency clinician because of its long delay of onset.

Digoxin is a cardiac glycoside found in a number of plants. It is commonly extracted from the leaves of the *Digitalis lanata* plant, and increases the intracellular Na+ by inhibiting Na-K-ATPase. This is the enzyme that regulates the quantity of Na+ and K+ inside the cell. Intracellular increases in the sodium ion concentration stimulate sodium/calcium exchange, leading to increased intracellular Ca+. The increased intracellular calcium can lead to increased contractility.

Digoxin has both a direct action on cardiac muscles and an indirect action on the cardiovascular system. The indirect effects are mediated by the autonomic nervous system. The results of these actions are vago-mimetic effects on the SA node and the AV node.

The consequences of these combined actions are: (1) increased force and velocity of myocardial contraction (positive inotropic effect); (2) slowing of the heart rate and AV nodal conduction (vago-mimetic effect); and (3) decrease in symptomatic nervous system effects (neurohormonal deactivating effect). [34] [35] [36] [37] [38] [39]

Indications and contraindications.

Though its use in rate control of the ventricular response in chronic atrial fibrillation is well established, it is no longer the mainstay of therapy for narrow-complex tachycardias. Newer agents have replaced digoxin in the treatment and management of narrow-complex tachycardias. Its inotropic character is still widely used in the setting of heart failure.

Figure 11-10 A run of ventricular tachycardia is seen immediately after a supraventricular dysrhythmia is terminated by cardiac sinus massage (CSM). The patient remained asymptomatic, and a normal sinus rhythm was established spontaneously within a few seconds.

Use of digoxin should be avoided in the clinical settings of sinus node disease and AV blockade. It may cause complete heart block or severe sinus bradycardia. It should not be used in the presence of accessory bypass tract rhythms (Wolf-Parkinson-White [WPW]) or Long-Ganong-Levine [LGL] syndromes) as it may cause a rapid ventricular response or VF. Patients with idiopathic hypertrophic sub-aortic stenosis (IHSS), restrictive cardiomyopathy, constrictive pericarditis, or amyloid heart disease are particularly susceptible to digtoxicity. [40]

Dosage: IV loading dose of 10 to 15 mcg/kg followed by individual parenteral dosing until desired rate is achieved. [37] [38] [41] [42] [43] [44] [45] [46] [47]

Procainamide

Another time-honored antiarrhythmic, procainamide slows conduction and decreases automaticity and excitability of atrial, ventricular, and Purkinje tissue. It also increases refractoriness in atrial and ventricular tissue. Procainamide prolongs the QT interval with little effect on Purkinje fibers or ventricular tissue. [38] [41] [46]

Indications and contraindications.

The drug is used in the rhythm and rate management of SVT, SVT with aberrancy conduction (wide-complex SVT), atrial fibrillation/flutter associated with WPW conduction, and VTs. The advantage to using IV procainamide is the ability to convert to the oral form when rate control is achieved.

The loading interval for procainamide is 20 minutes. This limits use of the drug to clinical situations when time is not a critical factor in patient care. Long-term management in the emergency department (ED) necessitates monitoring of the plasma concentrations of procainamide and its NAPA metabolite. Hypotension and conduction disturbances (torsades de pointes, heart blocks, and sinus node dysfunctions) are often signs of high plasma levels. Caution should be used in patients with histories of hypokalemia, long QT intervals, or torsades de pointes. Hematologic and rheumatologic disturbances are factors in long-term use.

Dosages: Over 25 minutes, a loading infusion of 275 mcg/kg per minute is administered. This is followed by a maintenance infusion of 20 to 60 mcg/kg per minute. [48] [49]

Adenosine

The use of vagal maneuvers has been eclipsed in recent years by the use of adenosine. The drug is an endogenous, ultrashort-acting vagal-stimulating nucleotide that occurs in all body cells. Its primarily action is to slow conduction time through the AV node.

Extracellular adenosine is rapidly cleared from the circulation by the erythrocyte and vascular endothelium system,

Figure 11-11 Hyperreactive carotid sinus reflex. Gentle pressure was applied to the carotid sinus for 3 seconds, resulting in a pause in sinus rhythm of approximately 7 seconds. This syndrome may be the cause of syncope. *(From Bigger JT Jr: Mechanisms and diagnosis of arrhythmias. In Braunwald E (ed): Heart Disease, vol 1. Philadelphia, WB Saunders, 1980. Reproduced by permission.)*

which transports adenosine intracellularly. Once inside the cell, rapid metabolism occurs via a phosphorylation or deamination cycle, producing inosine or adenosine monophosphate. Adenosine produces a short-lived pharmaceutical response because it is rapidly metabolized by the described enzymatic degradation. The half-life of adenosine is <10 seconds with the metabolites becoming incorporated into the phosphate, high-energy pool. [37] [38] [46] [50]

Indications and contraindications.

(1) Most forms of paroxysmal supraventricular tachycardia (PSVT) involve a reentry pathway involving the AV node: Adenosine is effective because it depresses the AV node and sinus node activity. Adenosine is indicated for the conversion of PSVT associated with or without accessory tract bypass conduction (WPW, LGL). (2) The other use of adenosine is in diagnostic slowing of SVT to unmask atrial fibrillation, atrial flutter, or ventricular tachycardia. The diagnostic and therapeutic effects of adenosine on tachydysrhythmias are similar to those elicited by the vagal maneuvers. Adenosine's safety is derived from its short duration of action—usually about 10 to 12 seconds.

Adenosine *should not* be used in patients with a known history of second- or third-degree atrioventricular block or sick sinus syndrome, unless there is a functioning internal pacer. Adenosine *should not* be used in patients with an underlying accessory tract bypass conduction (WPW, LGL) in the setting of atrial flutter/fibrillation. In this circumstance the heart rate may accelerate when enhanced AV node blockade permits greater use of the bypass tract. Also, if the patient has a known

hypersensitivity to the adenosine, the drug should not be used.

Dosage: The recommended initial dose is a 6-mg rapid bolus over 1 to 3 seconds. The dose should be followed by a rapid 20-mL saline flush. If there is no response within 1 to

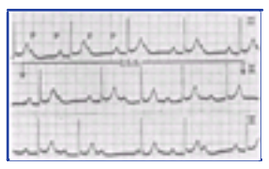

Figure 11-12 Acceleration of ventricular rate by carotid sinus stimulation (CSS). Continuous tracing. Upper strip shows 2:1 atrioventricular block: Atrial rate at approximately 102/min; ventricular rate at approximately 51/min. The second and third strips were recorded during and after CSS, when the atrial rate was reduced to 68/min; a 1:1 response occurs. *(From Lown B, Levine SA: Carotid sinus—Clinical value of its stimulation. Circulation 23:766, 1961. Reproduced by permission.)*

2 minutes, a 12-mg repeated dose should be administered in the same manner as the initial dose. This second dose should again be followed by a 20-mL saline bolus.

Side effects of adenosine are common and transient. They include flushing, dyspnea, and chest pain. Patients should be warned of a brief, but intense chest pressure. The use of 1 mg of midazolam (in the patient with an adequate blood pressure) intravenously before the adenosine is appreciated by patients treated with this agent. Important drug interactions include theophylline or related methylxanthines (caffeine and theobromine) that can block the adenosine receptor sites. Dipyridamole may block adenosine uptake and potentiate its effects. Dose adjustments or alternate therapies should be considered in these patients. [13] [37] [41] [46] [51]

Amiodarone

Amiodarone has become one of the workhorses of dysrhythmia treatment in the ED. As a Class III drug, amiodarone blocks the potassium channels, thereby prolonging repolarization and action potential duration, as well as, causing increased refractoriness of the atrial and ventricular tissues, the sinus and AV nodal tissues, and the Purkinje fibers. Amiodarone also blocks the sodium channel in depolarized tissues. At the sinus node and AV node, amiodarone slows depolarization in the SA node and slows conduction through the AV node. It blocks alpha and beta adrenergic receptors. Its calcium antagonist effect is minimal. [19] [38] [46] [52]

Indications and contraindications.

Amiodarone is used in the control of narrow-complex supraventricular and ventricular dysrhythmias. It is effective in the conversion of wide-complex tachycardias. These include atrial fibrillation and flutter with aberrancy, SVT with accessory pathway

TABLE 11-4 -- Ventricular Response to Carotid Sinus Massage (CSM) and Other Vagal Maneuvers

Type of Arrhythmias	Atrial Rate (bpm)	Response to CSM and Release
Normal sinus rhythm	60–100	Slowing with return to former rate on release
Normal sinus bradycardia	< 60	Slowing with return to former rate on release
Normal sinus tachycardia	> 100–180	Slowing with return to former rate on release, appearance of diagnostic P-waves
AV nodal re-entry	150–250	Termination or no effect
Atrial flutter	250–350	Slowing with return to former rate on release, increasing AV block: flutter persists
Atrial fibrillation	400–600	Slowing with persistence of gross irregular rate on release, increasing AV block
Atrial tachycardia w/block	150–250	Abrupt slowing with return to normal sinus on release, tachycardia often persists
AV junctional rhythm	40–100	None, +/- slowing
Reciprocal tachycardia using accessory (WPW) pathways	150–250	Abrupt slowing, termination or no effect, may unmask WPW
Nonparoxysmal AV junctional tachycardia	60–100	None, +/- slowing
Ventricular tachycardia	60–100	None, may unmask AV dissociation
Atrial idioventricular rhythm	60–100	None
Ventricular flutter	60–100	None
Ventricular fibrillation	60–100	None
First-degree AV block	60–100	Gradual slowing caused by sinus slowing, return to former rate on release
Second-degree AV block (I)	60–100	Sinus slows with increase in block, return to former rate with release
Second-degree AV block (II)	60–100	Slowing
Third-degree AV block	60–100	None
Right bundle branch block	60–100	Slowing with return to former rate with release
Left bundle branch block	60–100	Slowing with return to former rate with release
Digitalis toxicity-induced dysrhythmias	Variable	Do not attempt CSM

AV, atrioventricular; WPW, Wolf-Parkinson-White.

Adapted from Braunwald: Heart Disease. A Textbook of Cardiovascular Medicine, 6th ed. Philadelphia, WB Saunders, 2001, p 642.

conduction, and the rare adult junctional tachycardia. It is a strong second-line choice with procainamide for hemodynamically stable VT.

Its use can precipitate heart failure, hypotension, and severe bradycardia. When used with beta-blockers and calcium channel blockers, amiodarone increases the risk of hypotension and bradycardia. Torsades de pointes has been reported after the use of amiodarone in conjunction with drugs that prolong the QT interval.

Dosage: The IV dosage is 5 mg/kg (up to 150 mg over 10 minutes in adults; may repeat once if stable) given over 5 to 30 minutes, followed by a 0.4–0.8 mg/kg/hr maintenance infusion.[19] [35] [38] [46] [47] In adults the maximum daily dose is 2.2 gm/day.

Calcium Channel Blockers

Verapamil

Verapmil is a slow channel inhibitor, which exerts its rate reduction effect by controlling the calcium influx across the cell membrane of myocardial conduction and contractile cells. Verapamil prolongs the effective refractory period within the AV node and slows conduction. [3] [47]

Indications and contraindications.

Verapamil is effective in (1) converting narrow-complex PSVT to normal sinus rhythm, and (2) controlling the ventricular response in atrial fibrillation or atrial flutter, given that the atrial fibrillation or atrial flutter is not complicated by the presence of an accessory bypass tract (WPW, LGL).

Verapamil should not be used in the following settings: (1) atrial fibrillation/flutter with accessory bypass tract conduction; (2) coexistence of a sick sinus syndrome or second- or third-degree AV block unless an internal pacer is present; (3) severe left ventricular dysfunction (SBP < 90 mm Hg) or cardiogenic shock; and (4) patients with a known verapamil hypersensitivity. [13] [38] [41] [46] [47] [53]

Dosages: The initial adult dose is 2.5 to 5.0 mg IV over 2 minutes. If the initial dose is tolerated, but insufficient to affect the rhythm or rate, a repeat dose of 5 to 10 mg may be administered every 15 to 30 minutes to a maximum of 20 mg.[13] [38] [46] [47] [52] [54]

Diltiazem

Diltiazem works by inhibiting influx of Ca+ ions during cardiac muscle membrane depolarization. The therapeutic benefits in SVTs are derived from diltiazem's ability to slow AV nodal conduction and prolong AV nodal refractoriness. Its effects on AV nodal tissue are selective in that it reduces AV conduction in tissue responsible for the tachydysrhythmia but spares normal conduction tissue. [46] [47] [52] [54]

Indications and contraindications.

Its beneficial effects are (1) ventricular slowing of rapid atrial fibrillation/flutter without accessory bypass conduction, and (2) rapid conversion of narrow-complex PSVT to sinus rhythm.[38] [41] [52] [54] [55]

Diltiazem is contraindicated in the following settings: (1) sick sinus syndrome, second-degree block, and third-degree block, except in the presence of an internal pacer; (2) severe hypotension or cardiogenic shock; (3) diltiazem

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hypersensitivity; (4) use of IV beta-blockade within a few hours of need to use diltiazem; (5) atrial fibrillation or flutter with coexisting accessory bypass tract conduction (WPW, LGL); and (6) VT.

Dosage: An initial dose of 0.25 mg/kg over 2 min can be followed by a repeat dose of 0.35 mg/kg over 2 min. Maintenance infusion should be at 5 to 15 mg/hr for adults.[19] [41] [52] [54]

ß-Adrenergic Blockade

ß-blockers are very useful agents for the control of ventricular response in PSVT, atrial fibrillation or flutter, and atrial tachycardia. It is generally considered that no ß-blocker offers a distinctive advantage over another because they can be titrated to the desired effect on dysrhythmias and hypertension. However, the drugs do differ regarding their adverse reactions, speed of onset, half-life, dosage regimens, contraindications, and drug interactions.

The electrophysiological effect of ß-blockers results from the inhibition of catecholamine binding at ß-receptor sites. Their actions are on cells that are most stimulated by adrenergic actions. Typically, these sites are the sinus node, the Purkinje fibers, and ventricular tissue. [3] [38] [46] [47]

Propranolol

Propranolol is the representative drug of the ß-adrenergic blockade agents and is considered nonselective with respect to its ß-1 and ß-2 receptor antagonism. Its ß-1 effect on the heart allows for its use in controlling rapid ventricular rates. Rate slowing is mediated by (1) slowing SA node impulse formation, and (2) depression of myocardial contractility. The usual effects on the ECG are rate reduction and prolongation of the PR interval without much of an impact on QRS and QT intervals.

Because it is relatively nonselective (having effects on both a-1 and ß 1 & 2 receptors), propranolol's contraindications are somewhat extensive. [38] [47] [56] ß-blockers should be used in patients with a history of diabetes, bronchospasm, bradycardia or heart block, prior use of calcium channel blockers, hypotension, or the presence of a vasospasm condition.

Dosage: In the adult, IV propranolol is given in boluses of 0.5 to 1.0 mg, over 1 to 2 minutes, every 10–15 minutes until the desired rate is achieved, or the appearance of side effects, or a total dose of 0.2 mg/kg has been administered. [38] [39] [41] [46] [56] Children should receive a loading dose of 0.01 to 0.1 mg/kg every 10 minutes (up to 1 mg maximum single dose) with titration to effect.

Esmolol

Esmolol is a ß-1-selective (cardiac) ß-blocker with rapid action and short duration of action. At therapeutic doses, it inhibits ß-1 receptors. At higher doses, the selectivity is lost and it affects ß-2 receptors in the lung and vascular system. Esmolol is rapidly metabolized in erythrocytes and has a half-life of about 2 minutes. Its elimination half-life is approximately 9 minutes. [46] [52]

Indications and contraindications.

Esmolol is indicated for the rapid conversion of SVT and the rapid control of ventricular rate in patients with nonpreexcitation, atrial fibrillation, or flutter. It may be effective as adjunct therapy in the ventricular tachycardia of *torsades de pointes*.[41] [46] [47] [52]

Esmolol should not be used in patients with second- or third-degree heart block, or in frank heart failure. Like all ß-adrenergic blockers, care should be exercised when used in patients with bronchospastic disease and diabetes.

Dosage: This drug has a complicated dose regimen. An adult loading dose of 500 mcg/kg is given over the first minute. This is followed by a maintenance infusion of 50 mcg/kg per minute over 4 minutes. If this is not successful, a second bolus dose of 500 mcg/kg followed by a maintenance infusion of 100 mcg/kg per minute over 4 minutes is started. This bolus/maintenance dosing can be repeated up to a maximum infusion rate of 300 mcg/kg per minute for 4 minutes. [46] [47] [52] Similar dosing has been recommended for children using a 100–200 mcg/kg maintenance rate between 100 mcg/kg increases in bolus doses.

Special Consideration of Anticoagulation in Atrial Fibrillation: Evaluation and Treatment

The most common sustained tachydysrythmia that presents to the ED is atrial fibrillation (AF). Its incidence in the general population is 1% to 2%. The incidence increases with age. Approximately 1% of the population under 50 years of age has AF while 8.8% of the population older than 80 years of age has AF. [57] The connection between AF, structural heart disease, and antecedent coronary artery disease is strong. Atrial fibrillation is relatively uncommon in acute myocardial infarction (11%). Where, its added presence is associated with a 40% mortality. [58] The long recognized association between valvular heart disease and atrial fibrillation has been well documented. Rheumatic valvular disease is the classic valvular disease associated with atrial fibrillation. There are, however, other dysrhythmias associated with atrial fibrillation including WPW syndrome, atrial tachycardia, sick sinus syndrome, and AV nodal reentrant tachycardias. Long-standing medical or cardiac conditions that have strong associations with atrial fibrillation are hypertension, cardiac myxomas, diabetes, thyroid disease, left ventricular dysfunction, congestive heart failure, pulmonary edema, chronic obstructive pulmonary disease, and pulmonary embolism. [36] [58] [59]

Atrial fibrillation was initially thought to be caused by abnormal pulse formation originating in the atria. The theory was that the atria, as a result of disease, drug toxicity, or excessive endogenous hormones (i.e., catecholamines) trigger spontaneous automaticity of a sufficient number of atrial cells in multiple atrial sites. The hypothesis was that the chaotic, simultaneous firing of atrial impulses travel to the ventricles over multiple irregular routes. The transmission of these erratic low-amplitude atrial or fibrillatory "f" waves through the AV node to the ventricle creates the classic irregularly, irregular rhythm. [58] [60]

In 1959 the Russian researchers Moe and Abildskov presented their hypothesis that atrial fibrillation was a self-sustaining rhythm, independent of multiple firing focus, which was later verified in 1985.[61] At that time an animal model was constructed, showing that four to six waves or "wavelets" were needed to sustain atrial fibrillation in a multiple circulating wave of atrial reentry. These wavelets interact to maintain the optimum atrial conditions needed to maintain sustained atrial fibrillation. [58]

TABLE 11-5 -- Guidelines for Anticoagulation in Atrial Fibrillation and Cardioversion

ATRIAL FIBRILLATION
I. Duration under 48 hrs
Low risk for thromboembolism
Immediate electrical cardioversion if unstable
No anticoagulation necessary
II. Duration greater than 48 hrs or undetermined
Intermediate to high risk for thromboembolism
Immediate electrical cardioversion if unstable
Stable clinical situation
Warfarin: International Normal Ratio 2.0 to 3.0 for 3 weeks ⇒
Cardioversion, then warfarin: INR 2.0 to 3.0 for 4 weeks;
OR,
Transesophageal echo (TEE) and heparinization
Left atrial appendage clot not present ⇒
Cardioversion, then continue warfarin: INR 2.0 to 3.0 for 4 weeks
Left atrial appendage clot present ⇒
Continue warfarin: INR 2.0 to 3.0 for 4 weeks, then cardioversion

Adapted from Pelosi and Morady: Evaluation and management of atrial fibrillation. Med Clin North Am 85(2) March 2001, p 225.

Any adverse effects from this tachydysrhythmia are related to the disruption of the normal filling and ejection components of the cardiac cycle, especially if the atrial fibrillation is accompanied by a rapid ventricular rate. Classically, patients with atrial fibrillation present with feelings of palpitations, exertional fatigue, dyspnea on exertion, and lightheadedness. Patients subsequently develop fluid overload, congestive failure, frank pulmonary edema, and myocardial ischemia. [58] [62] [63]

Treatment of symptomatic rapid ventricular response to atrial fibrillation focuses upon three treatment fronts: (1) slowing the rapid ventricular response, (2) conversion to normal sinus rhythm, and (3) prevention of thromboembolism. The next section will address the issue of anticoagulation and prevention of thromboembolism. [52] [58] [63] [64]

Thromboembolism as Related to Cardioversion

Restoration of symptomatic new-onset atrial fibrillation to normal sinus rhythm can be achieved with direct cardioversion using either monophasic or biphasic defibrillators. In life-threatening presentations, patients in atrial fibrillation are immediately cardioverted if the risk of continued atrial fibrillation outweighs the risk of thromboembolism (Table 11-5).[52] [58] [63] [65]

The current guidelines for treatment of symptomatic new-onset atrial fibrillation focus on the length of time the patient has been in atrial fibrillation or flutter. If a patient has been in atrial fibrillation for less than 48 hours, cardioversion can be performed without the need for anticoagulation. Some clinicians will perform a TEE prior to cardioversion, to assure the lack of cardiac thrombi, if this time frame is questionable or unclear. Studies have shown that staying under the 48-hour limit allows cardioversion to occur with the lowest risk for thromboembolism. [52] [58] [63] [66] For patients who have been in atrial fibrillation greater than 48 hours and are not in need of urgent care, the recommendation is anticoagulation to an INR of 2.0 to 3.0 for a 3-week duration before cardioversion. [56] If this approach is not clinically acceptable, the patient should have a transesophageal echo (TEE), and heparin therapy should be initiated.

If no left atrial appendage clot is visualized on TEE, the heparinized patient should be immediately cardioverted and continued to be anticoagulated for the next 4 weeks. If a left atrial appendage clot is visualized, the patient should be anti-coagulated to an INR of 2.0 to 3.0 for 3 weeks' duration and cardioverted (see Table 11-5).[67] [68] [69] [70] [71]

A synchronized shock from a monophasic or biphasic defibrillator (see Chapter 12) should be delivered with the patient sedated. Success rates with the biphasic defibrillators have been reported to be approximately 94% to 95%. [72] [73] [74] An alternative treatment strategy with a reported success rate of 50% to 70% is the use of ibutilide in a bolus infusion or the use of amiodarone. Caution must be exercised in the use of ibutilide in patients with prolonged Q-T intervals or severe left ventricular dysfunction. Ibutilide has a 4% risk of ventricular proarrhythmia. Pretreatment with ibutilide of patients to be electrically cardioverted can increase their chances for conversion successful to nearly 100%. [37] [38] [47] [75] [76] [77] [78]

Amiodarone has the advantage of being effective for tachydysrhythmias when the mechanism is unclear with either wide-complex or narrow-complex tachycardias. Central venous access is advised if concentrations greater than 2 mg/mL are to be used. In adults, amiodarone should be given as an initial bolus of 5 mg/kg over 20 to 30 minutes, followed by a maintenance infusion of 1.0 g/24 hours for a total of 48 to 72 hours. [35] [37] [42] [46]

Other drugs with good to excellent evidence in obtaining rate control in narrow-complex atrial fibrillation include verapamil, diltiazem, procainamide, and ß-blockers. [38] [41] [46] [47] [58] These agents can be used to provide rate control until electrical cardioversion at a subsequent time.

CONCLUSIONS

The advent of ß-blockers, calcium channel blockers, adenosine, amiodarone, and other effective medications to treat tachydysrhythmias—particularly the SVTs—has diminished the therapeutic use of the vagal maneuvers. However, the vagal maneuvers still remain an important diagnostic tool. These maneuvers are especially important in unmasking the underlying rhythms of narrow-complex tachydysrhythmias and in determining the presence of the carotid sinus syndrome in patients with syncope.

The advent of medications and defibrillators, which can quickly and safely control the rate in tachydysrhythmias, has given the emergency clinician a more varied and powerful armamentarium to be used in cardioverting these life-style and even life-threatening dysrhythmias to normal sinus rhythms.

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Chapter 12 - Defibrillation and Cardioversion

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John R. Krimm

DEFIBRILLATION

Ventricular fibrillation (VF) is a potentially lethal dysrhythmia that is frequently associated with sudden cardiac death. [1] [2] [3] In addition to being associated with sudden cardiac death (SCD), independent of myocardial infarction (MI), VF often occurs as a result of myocardial ischemia, myocardial infarction, undiagnosed coronary artery disease, and electrical injuries. Use of medications such as tricyclic antidepressants, digitalis, quinidine, and other proarrhythmics that cause Q-T segment prolongation has been implicated in precipitating VF. Furthermore, chest trauma, hypothermia, profound hyperkalemia/hypokalemia, hypocalcemia, hypercalcemia, and other electrolyte disturbances can induce conditions favoring the development of VF. Although significant mortality and morbidity occurs as a result of persistent VF,[3] aggressive, early recognition and treatment in the form of defibrillation, cardiopulmonary resuscitation (CPR) and medications can provide reasonable success in reestablishing a perfusing (hopefully supraventricular) rhythm. [4] [5] Spontaneous conversion of VF to sinus rhythm, although rarely reported, is highly unlikely. [6]

The most effective treatment of VF is timely defibrillation. [2] [7] [8] [9] A significant number of victims of SCD can potentially be saved if there is access to early defibrillation (defined as defibrillation under 4 minutes of onset). [4] [10] [11] [12] In addition, the provision of CPR and, in some instances, medication may increase the success rate of resuscitation and bring about return of spontaneous circulation (ROSC). [13] However, successful defibrillation implies delivering the appropriate amount of current or energy to the fibrillating myocardium, such that the chaotic electrical activity is terminated and a supraventricular perfusing rhythm is established.[14] The delivery of current to the myocardium should be such that minimal damage is done to the existing myocytes so further dysrhythmias are not perpetuated.[15] Development of new biphasic defibrillation waveforms has set the stage for decreasing damage and may enhance successful defibrillation. [16]

Depending on the duration of myocardial ischemia and VF, administration of CPR prior to defibrillation may be the most appropriate intervention to achieve a better outcome.[17] Once defibrillation is successful, victims of VF have a tendency to refibrillate and may need pharmacological prophylaxis. [18] [19]

Lack of success for in-hospital resuscitation appears to result from delays in time to first shock from collapse. [20] [21] The development of cardiac resuscitation teams for both in-hospital and pre-hospital arenas and the development of automatic external defibrillators (AEDs) have increased the potential for the provision of rapid access to defibrillation. Therefore, an increased awareness of the need for early access to defibrillation, a familiarity with the pathophysiologic timeline of VF, and the appropriate sequence of interventions may lead to greater success in defibrillation and more survivors of SCD both in and out of hospital.

BACKGROUND

In 1775, long before the description of ventricular fibrillation and defibrillation, Peter Christian Abildgaard, a Danish physician and veterinarian, studied the effects of electric shock on chickens. He noted that the first shock rendered the chickens "lifeless" while a second countershock across the chest "revived" them. [22] In 1947, Beck and coworkers[23] successfully terminated fibrillation in a human heart by application of current directly to the myocardium. Nine years later, Zoll and colleagues[24] reported the first successful cardiac defibrillation in a clinical setting using an alternating current (AC) electrical shock applied externally to the thorax.

The research efforts of Edmark et al.[25] and Lown[26] introduced the use of direct current (DC) in defibrillation, opening the path for the development of portable defibrillators. Subsequently, defibrillation could be applied in remote, non-critical care areas of hospitals, and ambulatory clinics. With the development of modernized, protocol-driven Emergency Medical Service (EMS) systems and the advent of the mobile intensive care unit (MICU) [27] [28] [29] defibrillation was extended beyond the confines of the hospital and made available to paramedical personnel. Advances in electronics, microchip technology, and computer applications of medical algorithms led to the development of the AED, which enabled the lay public (i.e., trained "first responders" such as emergency medical technicians, police, security officers, and civilian CPR-trained "first aiders") to safely provide early access to defibrillation in appropriate areas of the community. [30]

Although the basic concepts of defibrillation have not changed, current attention is being turned toward modifying the defibrillator output waveform to a "biphasic" waveform with the intent of minimizing the amount of myocyte-damaging energy being delivered to the myocardium. (Biphasic waveforms are discussed in more detail later in the chapter.) With better, easy-to-use and readily available equipment, "time to first shock" may be decreased and this may increase the percentage of SCD survivors.

INDICATIONS AND CONTRAINDICATIONS

Immediate electrical defibrillation of the fibrillating myocardium is currently the most effective treatment to promote a successful resuscitation. [7] If a patient is found unresponsive, pulseless, and apneic, it is reasonable to assume that the underlying cardiac rhythm is VF. [7] The possibility of spontaneous conversion to an effective perfusing rhythm, although rarely reported, is highly improbable. [6] Importantly, the probability of successful defibrillation diminishes rapidly over time (Fig. 12-1).[3]

Early treatment via defibrillation is critical to survival from SCD. Access to a defibrillator with "quick-look" paddles or an AED permit immediate monitoring/assessment of the arrested patient's rhythm before electrical defibrillation. These devices can be quite helpful in the emergent situation to determine if defibrillation is indicated. Patients who are unresponsive and who have regular tachydysrhythmias as diagnosed by

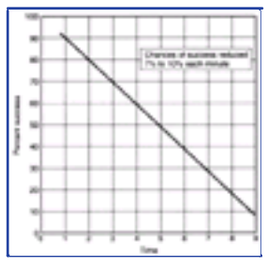

Figure 12-1 Success versus time; graph depicting relationship between survival to hospital discharge after ventricular fibrillation (VF) cardiac arrest and time interval between collapse and defibrillation. There is a 7% to 10% decrease in survival for each minute of VF duration. *(From Larsen MP, Eisenberg MS, Cummins RO, Hallstrom AP: Predicting survival from out-of-hospital cardiac arrest: A geographic model. Ann Emerg Med 22:1652, 1993. Reproduced with permission.)*

the "quick-look" method or an "unshockable rhythm" as determined by the AED are best treated with synchronized cardioversion. It is important to remember that if monitoring or an electrocardiogram (ECG) are not immediately available, an initial unsynchronized countershock "blind defibrillation" may be lifesaving. However, based on the current ubiquity of

Figure 12-2 Leads aVR, aVL, and aVF from an animal with electrically induced ventricular fibrillation (VF). From onset to 4.5 minutes, VF waves were obvious in all 6 frontal plane leads. At 5.0 and 5.5 minutes, lead aVR was a straight line. Note that aVR is the electrical sum of leads aVL and aVF. By 6 minutes, the null vector had changed, and VF was again evident in all 6 frontal plane leads. *(From Ewy GA, Dahl CF, Zimmerman M, et al: Ventricular fibrillation masquerading as ventricular standstill. Crit Care Med 9:841, 1981. Reproduced by permission.)*

"quick-look" paddles on modern manually operated defibrillators and the reliability of AEDs, "blind defibrillation" is rarely indicated. [4]

Regarding the sequence of defibrillation versus medications, current American Heart Association Guidelines [4] recommend that shocking the fibrillating myocardium takes precedence over administering medications. Review of the evidence regarding usefulness of the antiarrhythmic drugs has demonstrated that indeed the proarrhythmic effects of these drugs may decrease the possibility of a successful defibrillation. Furthermore, time delays in establishing intravenous access and endotracheal intubation may worsen electrical defibrillation outcome. [4]

In most resuscitations, CPR is initiated while the defibrillator is being readied. If the patient is unconscious, apneic, and pulseless, it is reasonable to assume that an episode of VF is occurring if cardiac monitoring is not available. In such instances, an immediate attempt at defibrillation is warranted. Although asystole and, more rarely, ventricular tachycardia (VT) may appear clinically similar to the scenario with VF, an immediate countershock is unlikely to worsen either clinical situation. This, however, should not be interpreted as an indication for shocking asystole, which does have significant adverse consequences (i.e., parasympathetic discharge that could lead to a high-level conduction block or myocyte damage, or both). [4] [14] [15] [31]

"Fine" VF can occasionally masquerade as ventricular standstill or asystole. Thus, the "quick-look" monitoring paddle electrodes should be rotated 90° from their original position or the monitor lead changed before the decision to withhold defibrillation in the victim of sudden death is made (Fig. 12-2).[32] [33] Likewise, low-voltage VF is not a contra-indication to defibrillation because it may reflect low monitor gain. Therefore, ECG gain should be maximized during the resuscitation effort of suspected VF, and a suspected problem with the monitor, lead selected, or paddle placement should be addressed before a decision to abandon defibrillation is made.

Administering more than three initial "escalating" or "stacked" countershocks before attempting interventions to ameliorate myocardial ischemia, acidemia, body temperature, or membrane stability is contraindicated because additional damage to the myocytes may occur, rendering the myocardium refractory to additional defibrillation.

CHARACTERISTICS OF VENTRICULAR FIBRILLATION

ECG Characteristics (ECG and the Electromechanical Physiology)

VF is characterized on the ECG by the presence of what appears to be a chaotic, random "squiggly line" (see Fig. 12-2). The actual display is one of rather low-amplitude baseline undulations that are variable in both magnitude and periodicity of the waveform. Care must be taken to ensure that appropriate electrode contact with the patient is maintained and that the leads are all appropriately attached so as not to interpret VF as artifact or vice versa. Although many consider VF to represent an electrically disorganized process, electrical directionality to depolarization (i.e., wavefronts) can exist [32] (see Fig. 12-2), manifesting the characteristic VF waveform or a flat line resembling asystole, depending on monitor lead orientation. It is therefore recommended that when assessing for the presence of VF versus asystole that several ECG leads be checked, differing by 90° in orientation and that the gain of the monitor be adjusted to the highest/most sensitive level. [4]

The resultant ECG tracing seen in VF is the resultant sum of voltage variations with respect to time from the discharge of multiple ectopic foci or "induced" ectopic pacemakers of the ischemic, hypoxic, electrically irritable ventricle. As the many electrical dipoles of myocyte depolarization travel through the ventricular myocardium, the randomness in the orientation of the positive leading edge of the dipoles causes the variation in the polarity of the ECG tracing. The combination of the mass of ventricular tissue undergoing VF and the sum of the dipole directions actually determines the amplitude of the undulations.

Mechanically, VF represents an uncoordinated and distinctly disorderly, ineffective contractile process. Usually the sinoatrial (SA) node depolarizes at an average rate range of 60 to 80 beats per minute. Subsequently, the atria are depolarized and the electrical depolarization front traverses the atrioventricular (AV) node. The impulse is slowed through the node, "decremental conduction," to allow for adequate ventricular filling. Upon activation of the bundle of His and the Purkinje fibers, the interventricular septum and the right and left ventricular free walls contract, decreasing ventricular volume and increasing ventricular pressure so that the blood can be propelled into the elastic aorta (i.e., the stroke volume is ejected from the ventricle). The lack of an organized ventricular systole causes a compromise in cardiac stroke volume. Subsequently, cardiac output falls, resulting in lack of adequate tissue perfusion, ischemia, and hypoxemia of the target organs. If left uncorrected, VF leads to irreversible tissue damage resulting in death.

At the tissue level, VF represents a disorganization of the orderly depolarization sequence that usually occurs in the ventricles. Normally, the refractory period of depolarized muscle prevents the development of reentrant ventricular rhythms by blocking the pathway of returning depolarization fronts. When ischemia, electrolyte disorders, cardiac drug toxicities, rapid ventricular rates, hypothermia, and certain other disorders exist, refractory periods may shorten or conduction velocities may tend to increase in certain areas of the ventricle. Wandering depolarization wavefronts can retraverse the nonrefractory areas of the ventricular myocardium, providing conditions in which a self-perpetuating, ectopic focus can develop. A combination of disorders of impulse formation (automaticity) and impulse conduction (reentry) contribute to the development of VF. [34] [35] The tendency for VF to occur is enhanced by, but is not entirely dependent on, premature ventricular impulses that occur during the "vulnerable" period of the cardiac cycle represented by early ventricular repolarization (Fig. 12-3).

Asynchronous ventricular depolarization may be confined to a small area of the ventricle if the remaining ventricle is refractory to further stimulation. Several studies have shown that a critical muscle mass is required for VF to be self-sustaining, possibly explaining why VF is so uncommon in infants undergoing resuscitation (who usually die from respiratory arrest). A large mass of muscle involved in asynchronous depolarization having a brief refractory period and a slow conduction velocity increases the tendency for the ventricles to fibrillate.

Cummins and coworkers[33] [36] have classified VF on the basis of average peak-to-trough wave amplitude (Table 12-1).

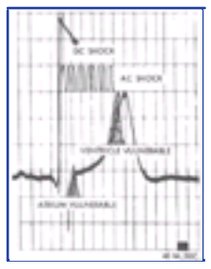

Figure 12-3 Phases of vulnerability for atrium and ventricle. Note that an alternating current shock of 0.20 seconds may end at the T wave even when synchronized with the R wave of the electrocardiogram. *(From Resnekov L: Theory and practice of electroversion in cardiac dysrhythmias. Med Clin North Am 60:325, 1976. Reproduced by permission.)*

TABLE 12-1 -- Classification of Ventricular Fibrillation (VF) Based on Mean Waveform (Peak to Trough Averaged over a 3- to 6-Second Interval) Amplitude

Terminology	Average Amplitude (mm)
Asystole	0 to <1
Fine VF	1 to <3
Medium VF	3 to <7
Coarse VF	7 to <12
Extra Coarse VF (V Flutter)	=12

Historically, it has been taught that coarse VF is easier to defibrillate than fine VF, and one of the functions of epinephrine in the setting of cardiac arrest is to coarsen fine VF to facilitate defibrillation. However, when subjected to study, this contention has been difficult to prove. Weaver and colleagues (Ann Intern Med 102:53, 1985) studied 394 patients with prehospital cardiac arrest and found that epinephrine did not augment the amplitude of VF. Although patients with coarse VF were not successfully defibrillated more often than those with fine VF, the postdefibrillation rhythm was frequently asystole (survival rate 6%) in patients with fine VF and a supraventricular rhythm (survival rate 36%) in those with coarse VF. In a canine study Jones and coworkers (J Electrocardiol 17:393, 1984) found more electrical similarities than differences in coarse and fine VF and concluded that the wave amplitude in early VF is due to electrocardiogram lead orientation rather than differences in the synchronization of electrical activity. The clinical importance of fine versus coarse VF is still unsettled. *(From Cummins RO, Stults KR, Haggar B, et al: A new rhythm library for testing automatic external defibrillators: Performance of three devices. J Am Coll Cardiol 11:597, 1988. Reprinted with permission from the American College of Cardiology.)*

They note that the amplitude of the VF waveform is associated positively with the probability of resuming a perfusing rhythm. However, the clinical importance of coarse versus fine VF in relation to ultimate survival is unclear. Signals that have an amplitude of <1 mm (when the monitor is calibrated at 10 mm/mV) should be considered to indicate asystole [33] [36] because countershock of such low-amplitude rhythms is only rarely associated with conversion to a perfusing rhythm. [37] [38] It has been reported that as VF continues in the face of ventricular ischemia/hypoxia for a time period exceeding *4 minutes*, the waveform of VF changes, decreasing in amplitude and progressing to asystole. [7] [39] This may be an indication that the myocytes are deteriorating and becoming unsalvageable with respect to VF unless some pharmacologic/mechanical resuscitation takes place first. [13]

A new approach to the analysis of VF is being explored. It is termed "AMSA," or an amplitude spectrum area. This methodology may prove to have the potential of enhancing the likelihood that an electrical countershock will restore a perfusing rhythm in a porcine model. This data, if proven useful, may allow more selective timing of shock administration and enhance the efficacy of defibrillation while decreasing myocardial damage from defibrillation. [40] Human studies of prehospital cardiac arrest ECGs suggest that the centroid frequency and peak power frequency of the ECG depicting VF may be predictive of successful countershock. [41] However, the

data to date has not led to any specific recommendations by the American Heart Association.

In summary, electrical defibrillation represents the simultaneous depolarization of sufficient ventricular tissue to render the tissue that is ahead of the VF wavefronts refractory to further electrical conduction. Following generalized depolarization, the sinus node or other pacemaker region of the heart with the highest degree of automaticity can then acquire dominance of a well-ordered depolarization-repolarization sequence. Application of the defibrillation current at various time points in the cardiac cycle may affect the outcome of the countershock. A more comprehensive discussion of this topic is contained in the cardioversion section of this chapter.

DEFIBRILLATOR CHARACTERISTICS

General standards and product evaluations for cardiac defibrillation devices are beyond the scope of this chapter. Rather, this section discusses the major components and design configurations of a prototypical defibrillator and how they relate to effective defibrillation. Only external cardiac defibrillators are discussed.

Defibrillators currently available can be subdivided into two general categories: manual and automatic. The manual defibrillator requires that the operator attach electrodes or use "quick-look" paddles to acquire the ECG. Then the operator must make the determination as to what intervention, if any, is appropriate. If there is a need to provide a countershock, the user must subsequently decide on the energy level (200J, 300J, or 360J) and the mode (synchronized for cardioversion [see section 2 of this chapter] or unsynchronized for defibrillation). Subsequently, the operator must actually depress the switches/buttons either on the defibrillation paddles or on the actual defibrillator to deliver the current (amperes) or energy (Joules) across the chest and myocardium.

The semi-AEDs require less operator manipulation. The AED is turned on, and voice or visual prompts, or both, guide the operator. The user applies self-adhesive pads with prefabricated conduction medium to the victim's chest, which both serve as ECG leads and defibrillation paddles. [42] The AED then analyzes the rhythm and advises the operator to press a "shock button," which delivers the shock to the victim of SCD, if a "shockable" rhythm (VF) or very fast VT (>180) is detected. [43] The algorithm used to analyze the acquired ECG waveform, the selected energy levels, and the actual sequence is preprogrammed by the manufacturer's medical director or can be modified by the clinician providing oversight in the use of this device. A fully automatic AED usually delivers a shock if indicated without user manipulation. These are used only in special situations. [4]

Waveforms

The first successful human defibrillation was performed using AC current. [23] Several years later DC current was shown to be more effective than AC current in accomplishing defibrillation. [44] Use of DC current also resulted in a significantly reduced incidence of postcardioversion dysrhythmias. [44] Only DC defibrillators are in clinical use today. Until recently, modern defibrillators delivered a "damped, monophasic, half-sinusoidal waveform" or a "trapezoidal truncated exponential decay" (voltage falling instantaneously) waveform. The trapezoidal waveform was modified to resemble a square waveform. The more square the waveform, the more effective it was for experimental defibrillation. [45] In a comparison of square waveforms and damped half-sinusoidal waveforms (voltage falling to zero gradually) for animal defibrillation, it was found that less peak current per kilogram was needed with the square waveforms, although the average current levels were equivalent. [46] This work was extended further, leading to the development of multiple new biphasic waveforms. These waveforms are achieved by manipulating the current (amperes), amplitude, duration, voltage, and ultimately the energy delivered to the myocardium.

In light of the current research into the utility of the various energy waveforms that modern day defibrillators

deliver, it would be useful to briefly describe/define terminology. Current output from a defibrillator is graphed with respect to time on an X-Y Cartesian plot. The form of the wave can be either "monophasic" or "biphasic." A characteristic monophasic wave is described as a rapid, positive, unidirectional increase in current flow to a predetermined peak with a return to baseline. If the return of the current to baseline is gradual, the waveform is termed a "damped" wave form (Monophasic Damped Sine-MDS waveform). These waves often resemble a sine wave (Fig. 12-4). If the return of the current level to baseline is paroxysmal/sudden, the wave is termed a "monophasic truncated exponential waveform-MTE waveform" (Fig. 12-5). If there is a rapid rise in current with respect to time with a slight plateau and then a subsequent paroxysmal/sudden reversal in current flow at a predetermined time until all of the energy is delivered with a return to baseline, this is termed a "biphasic" waveform because of the two "phases" in current flow, a positive and a negative phase. Essentially, for a biphasic waveform to occur, current travels from one pad or paddle to the other, then a reversal occurs so that current now flows from the second pad or paddle to the first. If the polarity/direction of the current flow is gradual, the wave is termed a "damped" waveform. If the current reversal is abrupt, the waveform is deemed a "truncated exponential waveform," hence the term "Biphasic Truncated Exponential Waveform—BTE waveform" (Fig. 12-6).

The highest current flow attained is termed the "peak" energy delivered. This, however, is not synonymous with the total amount of energy delivered. Energy is delivered throughout the duration or period of the wave. The current thinking is that if there is less peak energy and a smaller amount of energy delivered to the fibrillating myocytes, there will be less damage to the heart tissue. In addition, this may decrease the perpetuation of conditions favoring VF. This suggests that these waveforms will enhance defibrillation efficacy, decrease myocardial damage, and decrease post defibrillation arrhythmias. [47] [48]

The first biphasic AED approved by the Food and Drug Administration used a biphasic truncated exponential (BTE) waveform (see Fig. 12-6).[4] Additional experiments are being done to further explore various biphasic, rectilinear, first-pulse waveforms. The motive behind these modifications is to find an optimal waveform that will deliver the least amount of energy to the myocardium, thus decreasing the structural damage to the myocytes [31] while achieving successful defibrillation. [47] [49] [50] Injury to myocardial tissue has been associated with the peak current, not the amount of energy actually

Figure 12-4 Monophasic damped sine waveform (MDS) (Energy delivered versus time/msec.) *(Adapted courtesy of Cardiac Science, Inc., Irvine, CA.)*

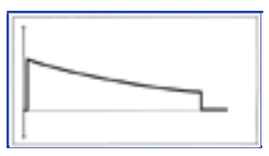

Figure 12-5 Monophasic truncated exponential waveform (MTE) (Energy delivered versus time/msec.) *(Adapted courtesy of Cardiac Science, Inc., Irvine, CA.)*

delivered to the myocytes.[51] With the biphasic defibrillators, lower energy levels (150 to 175 J) can be used without escalating the energy up to 300 J or 360 J. Experimental findings suggest that the clinical outcomes of these defibrillations are equivalent to those that used the escalating monophasic shocks. [52] There is data that also supports the claims that biphasic, low-energy defibrillations are efficient and cause less myocardial damage. [31] [53] However, the actual delivered energy is dependent on thoracic resistance/transthoracic impedance (TTI). Currently manufactured defibrillators are capable of determining TTI/resistance and can actually modify the waveform and thus the amount of energy delivered across the myocardium. A more detailed discussion of TTI is presented later in this chapter. In current clinical practice there is little clinical difference in the effectiveness of the currently available waveforms. The trend of the future will probably be to use biphasic, impedance-compensating defibrillators; however, currently, many monophasics are still in use. At this writing there is no convincing evidence to support the use of one defibrillator waveform over the other.

Stored Energy

Because the ability to defibrillate is dependent primarily on current delivered to the myocardium, [54] successful defibrillation depends on several factors—the energy stored in the battery and capacitor, the varying TTI, internal defibrillator energy

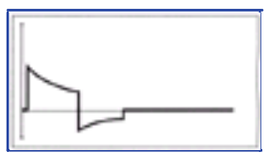

Figure 12-6 Biphasic truncated exponential waveform (BTE) (Energy delivered versus time/msec.) *(Adapted courtesy of Cardiac Science, Inc., Irvine, CA.)*

loss, and the waveform configuration. In addition, the "patient-defibrillator circuit" comprised by the patient, the electrode gel, the electrodes, lead wires, and defibrillator contribute to the outcome of a defibrillation attempt. Each defibrillator is calibrated by measuring the current delivered as a function of time across a 50-ohm impedance. It is important to note that defibrillators do not always deliver the energy indicated on the device. With a "stored" energy of 400 J (1 J = 1 watt-sec), from 155 to 410 J may be delivered. It stands to reason that the current (for a square waveform) delivered to the myocardium is related to the energy delivered by the defibrillator, the combined electrical impedance in the device and chest, and the duration of current flow:

Energy (J) = current (amperes)^2 × impedance (ohms) × duration (seconds)

Obviously, any increase in TTI will further reduce delivered current. With the development and refining of the biphasic waveform, and defibrillators that actually measure TTI/resistance and compensate for the patient defibrillator "circuit resistance," thus modifying the actual waveform of delivered energy, a more appropriate amount of current is delivered to the fibrillating myocardium. Furthermore, with better electrodes, more effective conductive material, and more efficient, smaller, lighter batteries, it is reasonable to assume that defibrillators of the future will be able to store energy for a longer time and be lighter and more portable. This may enhance compliance in the timely use of AEDs and early defibrillation.

Device Switches

Currently there are several manufacturers of electronic medical equipment. Although the equipment they manufacture may perform in a similar fashion (i.e., a defibrillator, an ECG monitor), it is vital that health care providers using the equipment be thoroughly familiar with the operation and layout of the controls in each particular unit. Furthermore, it is imperative that each piece of equipment be periodically tested to ensure optimal operation, battery charge, calibration, recording paper, and so forth.[4]

Most, if not all, currently used defibrillators have the capability of operating independently (i.e., via battery power) without a wall source of energy (i.e., AC plug to operate the monitor, event recorder, and charged capacitor and defibrillator battery). Older models may often have separate power switches for the accompanying monitor, recorder, and defibrillator, while newer units have a multi-function switch or dial. More advanced units may have pacer capability, 12-lead ECG capability, an automatic VF/VT rhythm detection option, rate alarms, sphygmomanometry, and pulse oximetry offered as options (Fig. 12-7 (Figure Not Available)). Some defibrillator monitors offer a radio-transmitter for telemetry monitoring. Currently used units have remote switches on the "quick-look" paddles to activate shock delivery. In addition, some units have energy- and event-recording switches on the paddles, giving the operator more control while in the proximity of the patient. With the discharge controls located on the paddle handles, this allows the operator the capability of positioning and holding the paddles in place while delivering the charge. Alternatively, there may be a separate control on the panel. The simultaneous activation of the control on both paddles is usually required for energy discharge.

Figure 12-7 (Figure Not Available) Hewlett Packard HP Codemaster 100/Laerdal Catalog.

The same device can be used for cardioversion as well as defibrillation (see the second section of this chapter). Before attempting defibrillation, the clinician must be completely familiar with the location and operation of the controls. This knowledge will minimize time lost fumbling with the equipment during a resuscitation of VF when time to first shock is critical to the success of the defibrillation.

Defibrillators also have a control permitting *synchronous cardioversion*. The clinician or other operator must be certain that the control is correctly set to the *asynchronous* mode to permit defibrillation; otherwise, the device in the synchronous mode would "wait" indefinitely for a repetitive series of R waves, not characteristic of VF, prior to discharging.

Energy settings may be determined by a switch or a dial setting or may be read off of a meter permitting a continuous range of settings. In each case the operator must be aware of the need to charge the device initially and to recharge after each discharge. The mechanism of charging the device may be intrinsic to the setting of the dial or meter but more commonly requires the use of a separate charge button on the device control panel or paddle handle. Full-charge accumulation usually takes from 2 to 5 seconds following activation of the charging mechanism.

Monitor controls permit alteration of lead-monitored, image size and often allow the selection of chest lead electrode versus paddle electrode monitoring. The latter is desirable when an initial "quick-look" rhythm evaluation is desired before placement of the chest lead electrodes. A hard copy paper recorder for documenting rhythms may operate in a real-time, delay, or standby mode.

Paddles or Electrode Pads

Previously, most commercial defibrillator devices were equipped with adult-sized paddle electrodes with diameters between 8 and 9 cm. Canine studies have shown that slightly larger (12.8 cm-diameter) paddles are more effective for defibrillation [55] and produce less myocardial injury. [31] Paddles that are slightly smaller (4.5 cm-diameter) produce greater damage at the same energies. The larger paddles may permit a greater amount of muscle to be depolarized while simultaneously decreasing the potentially damaging current density. If the paddles are too large with respect to the heart, the current

density may be less and defibrillation may be rendered less effective (Fig. 12-8 (Figure Not Available)). [42] Current recommendations from the Association for the Advancement of Medical Instrumentation are that a minimum electrode size of 50 cm^2 for each individual electrode be used. [56] The sum of the electrodes area should be a minimum of 150 cm^2 .[2] Although larger electrodes have been reported to have lower impedance, excessively large electrodes may hinder transmyocardial current flow.[57]

Some older defibrillators may still have a flat posterior ground shield rather than a second paddle for lateral chest wall placement. However, these are currently a rare find. The metal composition of the paddle electrode will affect TTI to the defibrillation discharge. Most modern defibrillators use stainless steel because of its durability, although copper alloys and several other metals provide a lower TTI.

Defibrillation can also be performed with self-adhesive electrode pads (8 to 12 cm in diameter) applied to the skin. Pads slightly smaller in size have also been found to be effective in short-term, acute applications. [58] The self-adhesive monitor/defibrillator pads appear to perform as well as or better than handheld paddles. [59] Stults and colleagues, in a controlled prehospital study, found that the use of self-adhesive pads shortened the time to successful defibrillation, reduced the number of countershocks, reduced the amount of rhythm artifact, and improved survival until hospital admission when compared with the use of standard hand-held paddles. [42] However, the expense of self-adhesive defibrillator pads has limited their general acceptance. If future studies support their superiority in other clinical settings, it is likely that the pads will gain wider acceptance.

Conductive Materials

Transthoracic impedance varies with the type of conductive material applied between the paddles and the chest wall. [60] The average adult human impedance is reported to range from 70 to 80 ohms (O). [53] [61] [62] The variability in impedance is contributed to body weight, chest size, chest hair, patient diaphoresis, serial shocks, paddle size, paddle contact pressure, phase of respiration, and type of conductive material used—saline-soaked pads or a specialized conductive paste. For

paddles that are 8.0 cm in diameter, the TTI is 91 ± 20 O for bare contact, 71 ± 11 O for saline-soaked gauze, and 64 ± 15 O for Redux Paste. Clearly, electrode-skin agents reduce impedance and allow more current to be delivered to the heart, but

Figure 12-8 (Figure Not Available) Current density/flow with respect to paddle placement. *(Adapted courtesy of Medtronic Physio-Control, Redmond, WA.)*

the ideal agent is the subject of some debate. Ewy and Taren recommend that Corgel, Redux Paste, American Writer, GE Gel, Electrode Jelly, or Trucon Electrode Paste be used to minimize impedance. Saline-soaked gauze pads may be used, although one must be careful not to allow the saline, coupling gels, or paste to flow into a "bridge" on the skin between the electrodes, creating a potential fire hazard. Although some form of coupling medium (e.g., paste, cream, gel, pad) should be used to reduce impedance, data conflict regarding which product is optimal. Although Redux Paste (Hewlett-Packard) has been associated with significantly lower TTI, a statistically significant increase in the success of defibrillation attempts has not been demonstrated with any specific product.

Hummel and coworkers investigated conductive materials with regard to their potential to overheat and to spark. [63] They found that the products that offer lower impedance (e.g., Redux Paste, Signagel) remained stable and did not spark after four or five defibrillation discharges, which was seen with the higher impedance products (e.g., Redux Cream, Aquasonic 100, EKG Sol, Spectra 360, and Derma-Jel). [63] With the advent of self-adhesive pads that serve as leads and paddles, conductive material will probably become more standardized.

Regarding diaphoresis and body hair, it is recommended that excessive perspiration be expediently wiped off if poor electrode contact is a problem. [4] If body hair causes poor adhesion and the possibility of electrical spark and arcing, it should be quickly removed. [64]

PROCEDURE

Victims suffering from sudden cardiac death (SCD) should be defibrillated as quickly as possible (Fig. 12-9). Current recommendations are that defibrillation be attempted up to three times prior to initiating CPR[4] [7] [65] unless additional personnel are on the scene and can perform CPR while the defibrillator device is being readied for use. Although survival from VF is highly dependent on many variables, the timeliness of defibrillation is the most important intervention determining the prognosis in cardiac arrest.[3] [9]

Upon recognition that a victim is unresponsive, apneic, and pulseless, help should be summoned immediately. However, this should not cause delays in the timely implementation of defibrillation. Saline-soaked pads or an alternate conductive medium should be applied to the chest where the "quick-look" paddles are going to be placed. Alternatively, a conductive material may be spread onto the entirety of the paddle's conductive surface prior to placement onto the victim's chest. However, care should be taken to avoid excess gel/paste on the paddles, because this could cause arcing or inadvertent harm to the operator. The paddles should then be firmly (25 lbs of downward force) applied to the chest wall and held there by the operator. This will optimize skin-electrode contact and decrease resistance. Also, this will ultimately enhance the appropriate passage of current through the heart during defibrillation. One paddle should be positioned to the right of the upper sternum below the clavicle. The second paddle is placed just to the left of the nipple in the midaxillary line and is centered in the fifth intercostal space (Fig. 12-10). Placement of both paddles close together on the anterior chest wall should be avoided. In some paddle sets, each paddle is labeled as either "sternum" or "apex" so that any rhythm detected on

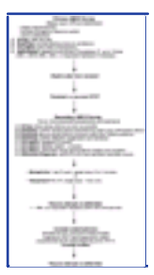

Figure 12-9 Ventricular fibrillation/pulses VT algorithm. *(From Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 102(Suppl 1):I-147, 2000.)*

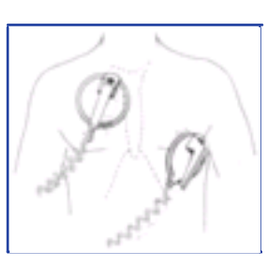

Figure 12-10 Anterolateral paddle electrode position. *(From Suratt PM, Gibson RS: Manual of Medical Procedures. St Louis, CV Mosby, 1982. Reproduced by permission.)*

the monitor can be properly aligned. This feature is irrelevant for defibrillation but is important for cardioversion (see the second section of this chapter). Anteroposterior paddle positioning is also acceptable and may deliver more current to the heart. In the patient with a normal cardiothoracic anatomy the anterior electrode can be placed to the left of the sternum over the precordium and the posterior electrode is placed just to the left of the spine directly posterior to the heart. In the tall, thin or emphysematous appearing patient the anterior paddle should be to the right of the sternum, as the heart is more retrosternal (Fig. 12-11).

With the monitor turned on and set to display the "paddle" electrodes, the rhythm is evaluated. If a flatline rhythm is detected, the operator must ensure that monitor gain is increased fully in order to rule out the presence of a "fine VF" tracing. Should the tracing remain flat during a brief pause in closed-chest cardiac massage, the paddles should be rotated 90° from the original position and the rhythm reassessed. The incidence of VF masquerading as asystole was approximately 2.5% in 1 prehospital study of patients with an initial flatline monitor rhythm. [33] Other conditions can cause a flatline rhythm during VF arrest. Cummins recommends that the rescuer also check all monitor cable connections to the patient and defibrillator, check the ECG size control, and check the power supply. [65] If VF is observed during any of these maneuvers, defibrillation should proceed without delay. Should a bradycardia or asystolic rhythm be detected, standard resuscitation measures, including basic CPR, correction of hypoxia, administration of catecholamines, correction of volume or cardiac filling deficiencies, and emergency cardiac pacing should be initiated when indicated. Although defibrillation should have no theoretical benefit for asystole, the use of countershock will result in the development of a QRS rhythm in a very small percentage of patients with flatline rhythms who receive such shocks. Presumably, such cases represent instances of fine VF simulating a flatline rhythm on the ECG.

In cases of "fine VF" in which a patient is wearing an implanted (subcutaneous) pacemaker, the pacer spikes may initially appear to be a paced but nonconducted rhythm; attention to the baseline and lack of ST changes characteristic of capture should reveal the true nature of the dysrhythmia. [66] Because injury to the pacemaker pulse generator[66A] and to the myocardium can occur by transmission of current down the pacing electrode, the clinician must be careful to situate the defibrillator paddle at least 1 inch (2.5 cm)[67] away from the pulse generator.[4]

Figure 12-11 Anteroposterior paddle electrode position. Use this placement in tall, thin individuals with retrosternal cardiac location. *(From Suratt PM, Gibson RS: Manual of Medical Procedures. St Louis, CV Mosby, 1982. Reproduced by permission.)*

In the presence of VF, the paddles should be immediately charged to a stored energy of 200 J for adults. Keep in mind that separate power switches may be needed to turn on the defibrillator and to store the charge. A button or a dial on the control panel usually sets the amount of charge. In most devices, the preset level of charge (energy) can be stored if a button on the "apex" paddle is depressed. *The clinician should always check to be certain that the defibrillator is not in the synchronous (cardioversion) mode.*

Once the paddles are charged, the clinician should instruct all personnel to " *STAND BACK*" from the patient and the stretcher to avoid stray discharge. The operator/clinician should then say aloud "I'M CLEAR, YOU'RE CLEAR, WE'RE ALL CLEAR" just prior to delivering the countershock. [4] However, there is no need for the individual who is bag-tube ventilating the patient to drop the bag and stand back *if their only contact is a rubber or plastic bag*. That individual is not touching conductive materials and will thus be protected from electrical shock. The defibrillator operator, in particular, must be sure that his or her only direct contact with the patient or stretcher is with dry paddle handles. The patient is allowed to exhale passively to minimize TTI, [68] while firm (25 lb) pressure is applied through the paddles to the thorax.[69] To minimize energy decay inside the device, the energy in the paddles is discharged through the chest as soon as possible after charging.

Simultaneous depression of both paddle discharge buttons is essential for discharge. Anticipation of

patient extremity motion subsequent to discharge of the paddles will minimize operator and patient injury.

Should no skeletal muscle contraction occur following simultaneous depression of the discharge buttons, the clinician should ensure that: firm chest wall contact has been made (some devices will not discharge without adequate contact), the device is set in the asynchronous mode, a charge has been stored, the defibrillator (not just the monitor) is turned on, and the battery is not depleted (when operating off the storage battery). If there is no muscle contraction even when these factors have been ruled out, a replacement defibrillator should be immediately brought into use.

After the first shock, the paddles should remain in place for 5 to 10 seconds to enable the clinician to check for an organized rhythm while ventilation is continued. While waiting to analyze the rhythm, the rescuer should recharge the paddles for an immediate second defibrillation (200 to 300 J for adults) should VF persist. Should VF continue after the second shock, an immediate third defibrillation (360 to 400 J for adults) should be given. Immediate recharging of the defibrillator and rapid succession in the administration of the shocks decreases chest wall impedance. If the third defibrillation is unsuccessful, closed-chest cardiac massage should be continued, hypoxia corrected, and a-agonist catecholamine (epinephrine) [70] [71] or naturally occurring antidiuretic hormone vasopressin [72] administered intravenously to elevate the diastolic pressure and to improve coronary perfusion. [4] [70] [71] [73] Following circulation of the catecholamine or vasopressin agent, an attempt at defibrillation should be repeated. Currently there are no data to suggest that "high-dose" epinephrine use is associated with superior long-term survival compared to standard doses in either children or adults. [74] Superiority of epinephrine versus vasopressin has not been satisfactorily established. [4]

Because excessive defibrillation energy may produce irreversible VF in the patient suffering toxic effects of digitalis, the lowest available energy level should be used for the initial defibrillation. If the initial energy dose is unsuccessful, the energy level can be increased cautiously for successive countershocks.

Additional therapy (see the following section) can be undertaken to enhance defibrillation. Adequate ventilation, cardiac massage, correction of electrolyte disorders, ischemia, and acidemia are intrinsic to every resuscitation. In addition, evaluation of the patient for hypothermia and rapid core rewarming when indicated should not be overlooked. When practical, electronic monitoring devices and transvenous pacemakers should be turned off or, preferably, disconnected from the patient to avoid equipment damage. Recently manufactured patient monitoring devices, however, have built-in protective filter circuitry, which makes equipment damage an unlikely occurrence.

ENHANCING DEFIBRILLATION SUCCESS

Early Defibrillation

Energy requirements for conversion of VF may increase dramatically shortly after the onset of VF. [69] The rationale for early defibrillation is that in the absence of adequate coronary perfusion, cellular metabolism continues with the depletion of energy substrates and the accumulation of toxic metabolites. Electrophysiologic changes secondary to cellular ischemia/acidemia develop rapidly and contribute to continued asynchronous transmission of VF wavefronts.

The American Heart Association Guidelines [4] support the belief that the most effective treatment for VF is early defibrillation [7] and that the probability of successful defibrillation decreases rapidly over time. [3] Clinical investigation of immediate defibrillation rather than drug therapy preceding defibrillation is limited. Martin and coworkers, in a retrospective analysis of prehospital VF resuscitation, found that survival until hospital discharge was increased when CPR followed by immediate defibrillation was used rather than CPR and drug therapy before countershock. [75] The group that received drug therapy first had a longer mean time until defibrillation (12 minutes additional), which was explained in part by the time required for IV line placement, drug administration, and drug circulation. The time lag until first shock may have confounded the results. Other studies describing treatment of prolonged VF suggest that CPR be performed for approximately 1 minute prior to administration of countershock.[17] The 2000 Guidelines suggest that administration of anti-arrhythmic medications may actually promote dysrhythmias. With the obvious controversies and confounders inherently present in these data, it is suggested that until further clinical studies are available, immediate defibrillation for VF appears to be the most appropriate course of action.

Current-Based Defibrillation

An alternative approach to defibrillation has been an attempt to use current-based (amperes) defibrillation. This approach would theoretically deliver the appropriate energy to the fibrillating myocardium by compensating for variations in thoracic impedance. Thus, patients with high impedance would not be victim to failed defibrillation secondary to insufficient energy delivery and patients with low impedance would not suffer myocardial damage due to excess current delivery. [48] [53] Based on monophasic damped sinusoidal (MDS) waveform research, it has been reported that 30 to 40 A is the optimal current necessary for defibrillation. Investigations are under way to determine values for biphasic waveform defibrillation. [76] Clinically, this is not an issue that the clinician has to adjust at the time of this writing. It may, however, modify future defibrillator design and enhance defibrillation outcome.

Transthoracic Impedance (TTI)

To accomplish successful defibrillation, sufficient current (amperes) must flow through the myocardium. Delivery of this current to the heart is dependent on the energy (joules) selected and the impedance or resistance (ohms [?]) to transmission of that energy from the paddles, through the chest wall and accompanying structures until the current traverses the heart itself. The greater the impedance or resistance, the less there is of delivered current. Reported values for human TTI using electrodes 8.0 cm in diameter range from 70 to 80 ohms with a mean of 75 ohms.[53] [61] [62] [77] We have previously discussed the factors that may affect TTI. The importance of paddle electrode composition and size, conductive materials applied paddle-to-chest-wall pressure, phase of ventilation, presence of excess chest hair, diaphoresis, and location of the paddle on the chest wall (Table 12-2) all contribute to TTI. All of these factors must be considered and dealt with to facilitate successful defibrillation.

TABLE 12-2 -- Determinants of Transthoracic Impedance*

Determinant	Effect
Interelectrode distance (chest size)	Larger distance ? higher impedance
Energy selected	Higher energy ? lower impedance
Electrode size	Larger electrode ? lower impedance
Electrode-skin couplants	Failure to use a couplant ? very high impedance
Previous shocks	Previous shock ? lower impedance, especially after first shock
Phase of respiration	Inspiration ? higher impedance
Electrode-chest contact pressure (handheld paddles)	Firm pressure ? lower impedance

*Lower impedance is preferred. Higher impedance lessens the current delivered to the heart. *(From Kerber RE: Electrical treatment of cardiac arrhythmias: Defibrillation and cardioversion. Ann Emerg Med 22:296, 1993.)*

The TTI of DC discharge also decreases with higher energy shocks, with increasing number of previous countershocks delivered (see Table 12-2)[77] [78] and with decreasing interval between the discharges. Unfortunately, each of the aforementioned maneuvers is also associated with an increased potential for myocardial injury.

When considering normal defibrillation, the chest should be relatively dry, a conductive medium must be appropriately placed onto the chest, and a good seal must be established between the chest and the paddles. The paddles should be of appropriate size. Twenty-five pounds of force should be applied to the paddles and chest. Discharge of energy should be done at the end of an expiration. Energy selected should be appropriate for the initial shock—usually 200 Joules. [4]

Nonetheless, one may be faced with the need to defibrillate a very obese patient who is unresponsive to standard paddle placement and maximum device energies. Should such a situation exist, the patient can be rolled on the side and anteroposterior defibrillation attempted. Should this prove unsuccessful, a second defibrillating device can be simultaneously charged and used to administer countershock immediately following discharge of the first defibrillator. It is interesting to note that a canine study of internal defibrillation indicated that two sequential shocks over different pathways reduced both total energy and peak voltage required to terminate VF.[79] Hence, sequential defibrillation using slightly different paddle placements may be beneficial independent of the concurrent reduction in TTI.

Energy Choice

Current International Liaison Committee on Resuscitation (ILCOR)/American Heart Association (AHA) guidelines recommend that energy for the first monophasic shock should be 200 J, and if unsuccessful, that the energy be escalated to 300 J or 360 J as needed. [4] [80]

Many investigators have referred to a so-called defibrillation energy threshold for converting VF. Davy and coworkers suggest that no unique defibrillation energy threshold exists for the in vivo heart. [81] Experimentally, they found that successful defibrillation was related to delivered energy, which follows a typical "dose-response curve." However, there is evidence for a defibrillation current threshold. [54]

Successful defibrillation is dependent on the simultaneous depolarization of a sizable mass of the myocardium resulting from the passage of current through the heart.

For a given thorax, defibrillation device, and defibrillation technique, more current is passed through the heart and, hence, more tissue is depolarized with larger energies. Once sufficient tissue has been depolarized, however, additional current is not desirable and may, in fact, produce additional tissue injury. Kerber and coworkers have suggested that the use of defibrillators that adjust defibrillation energy for the patient's TTI (measured by the device just before charging) may be one means to deliver an adequate current to the myocardium while minimizing potential harm as discussed earlier. [82] Such devices are not in routine use at present, but they may be helpful in the future for both the identification of high-impedance situations and adjustment of delivered energy. [48]

Several studies have supported the concept that when current needs cannot be predicted nor current delivery measured, a weight-adjusted dosage of energy is preferred for converting VF. Indeed, a dose based on the patient's weight has been found clinically useful for treating children suffering VF. [83] Other prospective human adult studies have questioned the importance of dose strength to conversion of VF. [84] [85] Weaver and coworkers, using monophasic waveforms for defibrillation, alternated treatment protocols to determine prospectively the merits of 175 J (200 J of stored energy) versus 320 J (400 J of stored energy) countershocks for defibrillation. [80] On test days, VF patients were shocked initially with 1 or 2 175 J discharges, and all subsequent shocks needed were 320 J. On alternate days, only 320 J shocks were given. The investigators found that 73% (n = 76) of the patients were defibrillated following the first 2 shocks in the low-energy group, whereas 81% (n = 77) of the patients were initially defibrillated in the high-energy group (difference not statistically significant). Asystole occurred in 19% of patients receiving high energy and in 12% receiving low energy. Transient or persistent heart block occurred in 25% of patients shocked with high energy versus 11% of patients shocked with low energy. Survival until hospital discharge was inversely related to the number of shocks required; no patients who required >8 shocks survived.

Weaver and associates concluded that low-energy (175 J delivered) countershocks were safe, effective, and less cardiotoxic. Obviously, many factors besides discharged energy play a role in successful defibrillation. Kerber found that defibrillation success rate is a unimodal function of transthoracic current. [86] The maximum defibrillation rate for their patients with a brief duration of VF (<1 minute) occurred at a peak transthoracic current of 38 to 41 amperes (A). Whether a similar optimum transthoracic current is found in patients with prolonged VF remains to be determined. Until energy levels based on measured TTI are shown to be predictive of defibrillation success in a variety of settings, most authorities recommend using an initial 200 J energy setting in adults. When 2 sequential defibrillations at this setting are unsuccessful, countershock at 360 J (or maximum output, if needed) is recommended (see Fig. 12-9).

Research suggests that biphasic shock waveforms can achieve successful defibrillation at energy levels <200 J and that this practice appears to be safe and effective. Escalation and nonescalation of energy has been explored. However, in clinical practice the recommendation [4] is to start with 200 J and increase energy as needed for successive shocks regardless of defibrillator used (monophasic/biphasic) at this time. It is likely that as biphasic defibrillators become

more prevalent, the recommended energy levels will be restructured.

Postdefibrillation Rhythms

Following defibrillation a number of cardiac rhythms may occur. Ultimate prognosis is linked to the rhythm that evolves during the first few minutes following defibrillation. In an evaluation of the prognostic significance of postdefibrillation rhythms in 94 patients, Hoffman and Stevenson [87] noted survival rates of 20% to 33% in patients who developed a supraventricular rhythm and 8% in those who developed an organized idioventricular rhythm; there were no survivors if defibrillation was unsuccessful or resulted in asystole or an unorganized rhythm. Survival rates may be influenced by the underlying condition or cause of the arrest or by subsequent drug therapy, but ultimate prognosis is dismal indeed if the first few attempts at defibrillation fail to restore a supraventricular rhythm.

There is the possibility that outcome of defibrillation may be related to the degree of myocardial injury incurred from defibrillation. However, data to support or refute this is being acquired. Biomedical engineers, clinicians, and researchers are working with waveforms, energy levels, and antiarrhythmic drugs to ultimately decrease myocardial injury with defibrillation. The ultimate goal is safe and effective defibrillation. The current recommendations are to terminate VF as soon as possible via defibrillation. If VF is suspected of having been present for several minutes (+/- 4 minutes), then doing a minute of CPR prior to defibrillation may be beneficial. This information is currently classified as "historical/anecdotal", although animal and preliminary clinical trials are supportive.

Drug Therapy

As previously noted, the most effective treatment modality for VF is defibrillation. [52] If after delivering three consecutive countershocks VF persists, additional interventions may be initiated to achieve termination of fibrillation. Although drug therapy is often an adjunct to defibrillation, *no medication has been proven to increase the rate of survival in cardiac arrest*.

As noted in the most recent Advanced Cardiovascular Life Support (ACLS) Algorithm and in the new AHA recommendations, [4] many previous interventions (such as drug therapy in the form of antidysrhythmics) have been deemphasized (e.g., epinephrine, lidocaine, bretylium, sodium bicarbonate). In addition, several new medications (i.e., vasopressin and amiodarone) have been introduced into the treatment regimen. Use of other medications (e.g., procainamide, magnesium sulfate) have limited application and are still being debated.

Before proceeding to administer additional shock and medications, correction of hypoxia and hypercarbia via airway management, bag-valve mask ventilation, and endotracheal intubation or airway adjuncts must be attended to immediately. Intravenous (IV) access for drug administration and blood sampling to guide further treatment should be established. Appropriate treatment of electrolyte disturbances like hyperkalemia/hypokalemia and hypomagnesemia will at best be presumptive in anticipation of laboratory results. The next most appropriate intervention should address return of blood flow to the heart and maintenance of aortic diastolic blood pressure, in an attempt to increase coronary perfusion, with the ultimate goal being to increase coronary artery perfusion pressures and decrease myocardial hypoxia/hypercarbia. If achieved, the increase in coronary blood flow during VF may remove some of the "toxic" metabolites from the fibrillating myocytes and may decrease the perpetuation of a reentrant depolarization wavefront. In addition, the drugs used should enhance cardiac function, potentiate the return of a spontaneous circulation and blood pressure, and shunt blood toward vital organs. [88] The physiological support for this proposed intervention is derived from the fact that endogenous catecholamine levels rise significantly after cardiac arrest. [89] This may be achieved by pharmacological intervention via vasopressors in the form of a-agonist catecholamines (epinephrine) or the alternative administration of the natural antidiuretic hormone (ADH; vasopressin). Amiodarone, a fairly new agent, may be considered, even before the "old classic," lidocaine. Drugs like magnesium sulfate, and procainamide may be considered. The role of sodium bicarbonate, although very limited, is also addressed in special situations. The specifics of these agents will be addressed later.

It should be noted that although adjunctive drug therapy is often used or advocated, there are no data supporting better long-term survival rates when *any drug therapy regimen is combined with defibrillation*. When evaluating any intervention, *it is paramount to focus on survival to discharge and not to be overly concerned with minor changes in initial resuscitation rates that merely provide ultimately unsalvageable patients with transient cardiac activity*.[90] Also, as a reminder, adjunctive therapy should not cause significant delay to subsequent defibrillation, if indicated.

Adrenergic Agents (Physiologic & Pharmacologic Effects; A Historical Perspective)

Drugs that stimulate a-adrenergic receptors have been advocated during cardiac arrest to increase myocardial and central nervous system blood flow during CPR. These agents may not enhance the ability to defibrillate VF, but they may enhance the development of a perfusing rhythm following defibrillation. [91] It is important to note that a agonists are potent vasopressors and that they increase systemic vascular resistance and elevate aortic diastolic pressure, resulting in increased coronary and carotid blood flow. This enhanced perfusion, and not direct ß-adrenergic stimulation, is credited with increased survival rates. [92] In fact, the ß-adrenergic properties of epinephrine have been postulated to be harmful because these pharmacologic effects increase myocardial oxygen consumption and reduce subendocardial perfusion. When isoproterenol, a potent ß agonist without a effects, was used during resuscitation, it did not favorably influence resuscitation rates. Epinephrine has been the vasopressor of choice during CPR. The ACLS algorithm recommends doses of 0.5 to 1 mg every 5 minutes. Large doses of epinephrine (0.2 mg/kg), 10 to 15 times higher than currently recommended, were touted to provide better initial resuscitation rates, but have not been shown to enhance ultimate survival rates. [74] [93]

A number of pure a agonists have been advocated to overcome the theoretical disadvantages of epinephrine. For example, Silvast noted similar resuscitation rates from prehospital cardiac arrest (e.g., from VF, asystole, and pulseless electrical activity [PEA]) when phenylephrine (1 or 2 1-mg boluses) was used in place of

standard doses of epinephrine. Methoxamine (10 to 20 mg) has also been used with success rates similar to those of epinephrine. [94] There is concern,

however, that although pure a agonists favorably augment peripheral vascular resistance, they do so at the expense of cerebral perfusion and myocardial blood flow.[95] [96] The specific role of these agents on defibrillation is unclear. Like high-dose epinephrine, the use of pure a agonists in facilitating defibrillation or enhancing long-term survival is not supported by current data. Although adherence to standard ACLS guidelines is logical until the benefits of alternative therapies are clarified, the role of adrenergic adjunctive drug therapy remains controversial. Nonetheless, epinephrine and vasopressin are included in the current VF algorithm and are discussed later. In an attempt to evaluate the usefulness of these agents we must closely review the scientific data supporting or refuting their use. The AHA in collaboration with the European Resuscitation Council and the Resuscitation Council of Latin America developed and published a list of criteria for evaluating and categorizing the information presented in the literature. A summary of these data is provided in Table 12-3 . These criteria are used to describe the usefulness of the proposed therapeutic intervention.

Epinephrine

Epinephrine hydrochloride is a catecholamine with a- and ß-adrenergic receptor-stimulating properties. Beneficial effects of epinephrine administration are attained via the a-adrenergic receptor-stimulating properties. [70] By administering epinephrine, there is an increase in myocardial and cerebral blood flow during CPR. [71] However, there are drawbacks in the use of epinephrine in that there may be an increase in myocardial workload with a concomitant decrease in endocardial perfusion, leading to the compromise of cardiac tissue. [97]

Epinephrine was initially used by surgeons in the operating room to restart an arrested heart. [98] [99] [100] They ascertained that a dose of 1 to 3 mg of epinephrine injected directly into the heart was useful in restarting the nonbeating heart. In the initial development of guidelines for cardiac resuscitation, a 1 mg intravenous (IV) dose of epinephrine was suggested. However, not all patients have a similar body weight and the concentration of the drug in the blood level was at best variable. When the dose response effects were studied regarding the administration of IV epinephrine, it was determined that epinephrine produced the most effective hemodynamic effects at a dosage of 0.045 to 0.2 mg/kg IV.[101] [102] [103] [104] This led to the recommendations of "high dose epi" in cardiac resuscitation. A subsequent outcome review regarding the efficacy of high-dose epinephrine demonstrated that although there may have been a greater rate of return of spontaneous circulation (ROSC) there was no significant increase in survival to hospital discharge. [101] [105] [106] [107] In fact, it has been reported that the use of high-dose epinephrine has led to worsening hemodynamic and neurological outcomes in many patients.[108] [109] However, a direct cause-effect relationship was not clearly demonstrated. In addition, epinephrine has been shown to be arrhythmogenic. The proposed mechanism for this arrhythmogenesis is believed to be the enhanced depletion of myocardial adenosine triphosphate (ATP) and an increase in lactate secondary to an increase in circulating epinephrine levels. [97] With ATP depletion, the sodium-potassium ATPase pump is impaired, causing an increase in the duration of the cardiac action potential due to delayed repolarization of the myocytes. This in turn leads to irregularities in various regions of the myocardium, favoring conditions for the propagation of an aberrant, reentrant depolarization. Furthermore, epinephrine has been shown to increase intrapulmonary shunting by 30%. [110] This results in significant desaturation of hemoglobin in the erythrocytes.

In light of this data, the current American Heart Association Guidelines [4] describe administration of 1 mg of epinephrine (IV) every 3 to 5 minutes as a Class Indeterminate recommendation (see Table 12-3). If this does result in a successful pharmacologic effect, higher doses of epinephrine (0.2 mg/kg IV) may be given, but are not recommended (Class IIb to III) and indeed may be harmful (see Table 12-3). The following section describes a potential alternative/addition to the use of epinephrine in VF; i.e., vasopressin.

Vasopressin

Vasopressin, also known as antidiuretic hormone (ADH), is a naturally occurring polypeptide hormone endogenously produced from within the cells of the hypothalamus. Vasopressin is stored in the posterior lobe of the pituitary gland and secreted during episodes of physiologic stress; such as dehydration, hypovolemia, hypernatremia, and hypotension. Some findings describe the release of high levels of endogenous vasopressin immediately after cardiac arrest. [111] In physiologic doses, this hormone increases the permeability of the distal convoluted tubule and collecting duct of the kidney, causing a decrease in the excretion of water by the kidney. If vasopressin is administered in a pharmacologic dosage, much higher than what would induce an antidiuretic effect, it acts as a *non-adrenergic* peripheral vasoconstrictor. It produces this effect by direct stimulation of V_1 receptors located on smooth muscle cells. Interaction of vasopressin with the V_1 receptor produces a whole compendium of results—peripheral vasoconstriction of the blood vessels in the skin, skeletal muscle, intestine, and fat, with less constriction of coronary and renal vascular beds and vasodilatation of the cerebral vasculature, resulting in a direct increase in blood pressure, pallor of the skin, intestinal cramps, nausea, bronchial constriction, an urge to defecate, and uterine contractions in women. [112] [113] Vasopressin also increases shunting of blood within the coronary and cerebral blood vessels, and promotes the local vascular endothelial release of nitric oxide, which concomitantly results in an increased flow of blood to the heart and brain. [114]

Many of the aforementioned characteristics of vasopressin are similar to the effects describing the actions of epinephrine. However, the pharmacologic actions of vasopressin have a few advantages over that of epinephrine—vasopressin is not adversely affected by acidemia, nor does vasopressin have any ß-adrenergic effects.[115] Vasopressin also has a half-life of about 10 to 20 minutes with intact circulation. This provides longer efficacy post administration and reduces the need for repeat dosing as is needed for epinephrine effects. Administration of vasopressin to victims of cardiac arrest during CPR in the field has been shown to increase both ROSC and survival to hospital admission. In addition, more of these patients survived to hospital discharge. [116] A later study of in-hospital cardiac arrest failed to yield significantly changed rates of survival of patients to discharge or change in neurological outcomes. [117] Based on the preceding information, the review committees for standardizing resuscitation [118] (the International Resuscitation Guidelines) have recommended vasopressin as a Class IIb (see Table 12-3) drug for VF or pulseless VT. A single one-time dose of 40 units IV of vasopressin may be used in lieu of the 1 mg dose of epinephrine after the first three unsuccessful shocks or after the

TABLE 12-3 -- Classes of Recommendations 2000: Classification of Therapeutic Interventions in Cardiopulmonary Resuscitation and Emergency Cardiac Care

1. Search for Evidence: Locates the Following	2. Consensus Review by Experts: Intervention Is Placed in Following Class	3. Interpretation of This Class of Recommendation When Used Clinically
Minimum evidence required for a Class I recommendation	***Class I: Excellent***	**Class I** interventions are always acceptable, proven safe, and definitely useful.
	Definitely recommended	
• Level of evidence: 1 or more RCTs	Supported by ***excellent*** evidence	
• Critical assessment: *excellent*	Proven efficacy and effectiveness	
• Results: homogeneous, consistently positive, and robust		
Minimum evidence required for a Class IIa recommendation	***Class IIa: Good to very good***	**Class IIa** interventions are acceptable, safe, and useful.
	Acceptable and useful	
• Level of evidence: higher	***Good/very good*** evidence provides support	• Considered standard of care: reasonably prudent physicians can choose
• Number of studies: multiple	Note: Contextual factors: In addition to level of evidence, these additional factors are considered in making final class of recommendation.	
• Critical assessment: *good to very good*		

• Weight of evidence/expert opinion: more strongly in favor of intervention than Class IIb		• Considered ***intervention of choice*** by majority of experts
• More long-term outcomes measured than Class IIb	Contextual factors include small magnitude of benefit; high cost; educational and training challenges; large difficulties in implementation; and impractical, unfavorable cost benefit ratios.	• Often receive AHA support in training programs, teaching materials, etc.
• Results: positive in majority of studies		*"Contextual" or "mismatch" factors may render an intervention Class IIa in one context and Class IIb in another (see Note).*
• Observed magnitude of benefit: higher than Class IIb		
Minimum evidence required for a Class IIb recommendation	***Class IIb: Fair to good***	**Class IIb** interventions are acceptable, safe, and useful.
	Acceptable and useful	
• Level of evidence: lower/intermediate	***Fair to good*** evidence provides support.	• Considered within "standard of care": reasonably prudent physicians can choose
• Number of studies: few	Note: Contextual/mismatch factors should not be used to avoid the trouble and expense of adopting new but clinically beneficial interventions.	
• Critical assessment: *fair or poor*		
• Weight of evidence/expert opinion: less in favor of usefulness/efficacy		• Considered *optional* or *alternative interventions* by majority of experts
• Outcomes measured: immediate, intermediate, or surrogate		
• Results: generally, not always, positive		
Evidence found but available studies have one or more shortcomings	***Class Indeterminate***	Note: Interventions classed
	Preliminary research stage	***Indeterminate*** can still be recommended for use, but reviewers must acknowledge that research quantity/quality fall short of supporting a final class decision.
• Promising but low level	Available evidence insufficient to support a final class decision	
• Fail to address relevant clinical outcomes	Results promising but need additional confirmation	
• Are inconsistent, noncompelling, or report contradictory results	Evidence: no harm, but no benefit	Do not use *Indeterminate* to resolve debates among experts, especially when evidence is available but experts disagree on interpretation. *Indeterminate* is limited to promising interventions.
• May be high level but report conflicting results	No recommendation until further evidence is available.	
Positive evidence completely absent *or* **Evidence strongly suggests or confirms harm**	***Class III: Unacceptable, no documented benefit, may be harmful***	Interventions are designated as Class III when evidence of benefit is completely lacking or studies suggest or confirm harm.
	Not acceptable, not useful, may be harmful	

RCT indicates randomized, controlled trial.

(From American Heart Association Guidelines 2000, Circulation 102(suppl):1, 2000. Reproduced with permission.)

administration of standard-dose epinephrine. If there is no evidence of success, 1 mg doses of epinephrine IV can be resumed every 3 to 5 minutes (Class Indeterminate) (see Table 12-3).

Lidocaine

Lidocaine has long been used to facilitate defibrillation. The rationale for use of lidocaine in VF is primarily based on anecdotal experience. Two studies of nonischemic canines have demonstrated that lidocaine increases the energy required for electrical defibrillation. [96] [119] Kerber and coworkers suggest that the elevation of the defibrillation threshold noted by others may be a function of the anesthetic used in the animal model and may not be a clinical factor. [120]

Lidocaine has complex effects on membrane responsiveness. Little change in conduction velocity occurs in normal myocardium, whereas conduction in ischemic tissue is decreased following lidocaine administration. Lidocaine increases uniformity of the action-potential duration and refractory period throughout the ventricles and can terminate ventricular reentrant rhythms. Lidocaine also raises the threshold for electrically induced VF in a canine CPR model within 5 minutes after administration, although the antifibrillatory effect is not sustained with a single bolus dose.

One retrospective study of prehospital VF arrests documented a small but statistically insignificant improvement in both defibrillation rate and survival when patients refractory to conventional therapy for VF were given lidocaine during the course of their resuscitation. Unfortunately, strict drug and therapy protocols were not followed, and variations in treatment may have masked a beneficial effect of lidocaine administration. Older clinical experience suggests that the drug may have value in aiding defibrillation. Unfortunately, this is an anecdotal generalization. Additional studies to ascertain the efficacy of lidocaine with respect to VF termination, the ability to facilitate defibrillation, survival to hospital admission, and ultimately to successful, functional discharge from the hospital have failed to demonstrate any significant benefit in administering lidocaine. [121] A study comparing survival of up to 1 hour post VF defibrillation, both with and without the use of lidocaine, found no significant differences in outcome between the 2 groups. [88] Together with the aforementioned study and a review of the data currently available by the participants of the International Guidelines Conference, the International Guidelines Committee has led participants to assign lidocaine to the Class Indeterminate (see Table 12-3).[118] Lidocaine is initially given as a bolus of 1 mg/kg to the VF sudden death victim who is refractory to conventional defibrillatory efforts. The bolus of lidocaine is suggested after the use of either epinephrine or vasopressin, or both. A second 1 mg/kg bolus can be given in 10 to 15 minutes. It is important to remember to reduce the maintenance dose (not the initial loading dose) of lidocaine in patients who have a history of hepatic or left ventricular dysfunction, or both. Currently, there is a relatively new agent available that has shown promise as an effective antiarrhythmic in VF. This drug may be an alternative to lidocaine. It is discussed in the following section.

Amiodarone

Amiodarone is a relatively new antiarrhythmic agent that has been introduced into the algorithm for the pharmacologic enhancement of VF defibrillation. Although amiodarone has been classified primarily as a class III antiarrhythmic agent-potassium channel blocker (using the Vaughn-Williams classification of antiarrhythmic drugs), it also has the properties of a class I, II, and IV antiarrhythmic drug; Class I—a sodium channel blocker, Class II—a noncompetitive alpha-beta blocker, and Class IV—a calcium channel blocker. Because of its diverse antiarrhythmic properties, amiodarone has gained acceptance as a useful drug during the management

of VF, refractory to pressors (epinephrine or vasopressin, or both), and a total of 4 defibrillation shocks.

As already mentioned, amiodarone is the only antiarrhythmic agent that manifests all 4 Vaughn Williams classes. [122] Amiodarone has been studied extensively in an attempt to determine its efficacy in enhancing termination of VF. [123] [124] [125] The data has demonstrated that amiodarone, with its lengthening of the cardiac action potential, prolongation of refractoriness of the myocytes, and deceasing cardiac oxygen consumption, is more effective than previously used agents such as lidocaine. In addition, amiodarone has important hemodynamic effects that favor its use: it increases coronary blood supply, dilates coronary arteries, causes peripheral arterial vasodilatation, improves cardiac pump performance in patients with compromised left ventricular function, and decreases systemic vascular resistance. [122] In addition, amiodarone rapidly distributes throughout adipose tissue, is highly bound to plasma proteins, and has an extensive half-life. The principle metabolite (desethylamiodarone or DEA) has properties to its parent compound, thus effectively increasing the duration of amiodarone effects in the dysrhythmic patient. This decreases the need for repeat/frequent dosing. Regarding pharmacokinetics, neither hepatic disease, renal insufficiency, age nor sex have any significant effects on the dosing of and physiologic effects of amiodarone.

Regarding side effects, the most common adverse side effect seen with IV amiodarone is hypotension (16%). This can be ameliorated by slowing the rate of infusion fluids, pressors, and chronotropic agents. Other adverse effects include: bradycardia (4.9%), liver function test abnormalities (3.4%), cardiac arrest (2.9%), VT (2.4%), congestive heart failure (2.1%), cardiogenic shock (1.3%), and AV block (0.5%). In light of these data, amiodarone is contraindicated in patients with cardiogenic shock, marked sinus bradycardia, and second- or third-degree AV block in the absence of a functioning pacemaker. However, in the situation of VF, the venue for the administration of amiodarone is modified.

Use of IV amiodarone in VF refractory to three initial countershocks has been demonstrated to improve survival to hospital admission by 29% (ARREST TRIAL). [125] In addition, the ALIVE trial (Amiodarone versus Lidocaine in Ventricular Fibrillation Evaluation) has shown amiodarone to be significantly superior to lidocaine terminating VF and increasing the number of patients who have survived to hospital admission in the prehospital setting. [50] Speculation on officially unreported data regarding survival to discharge from the ALIVE trial suggests that there may be a significant increase in survival of VF victims to hospital discharge. [126]

Currently, amiodarone is recommended after defibrillation and epinephrine or vasopressin, or both, in cardiac arrest with VF (Class IIB) (see Table 12-3). In cardiac arrest with VF, amiodarone is administered as a rapid 300 mg rapid infusion diluted in a volume of 20 to 30 mL of saline or dextrose in water. A 150-mg dose may be repeated followed by an infusion of 1 mg/min for 6 hours and then 0.5 mg/min to a maximum of 2.2 grams over a 24-hour period.

Procainamide

Procainamide hydrochloride, a sodium channel-blocking antiarrhythmic that prolongs the effective refractory period and slows conduction through the myocardial conductive tracts, is a drug that can suppress both atrial and ventricular arrhythmias. There is a dearth of studies addressing the use of procainamide in VF. However, there is one limited, retrospective study of only 20 patients that states that there may be a benefit from the use of procainamide in VF. Practical use of procainamide during VF cardiac arrest is limited because of the need to infuse procainamide slowly.

Procainamide should be avoided in torsades de pointes.

Magnesium Sulfate

Magnesium sulfate has limited use in the resuscitation of VF. Unless there is a predetermined knowledge that the victim of VF is in a low magnesium state, hypomagnesemia, or there is a manifestation of torsades de pointes, there is little indication for its use. However, a known or suspected magnesium deficiency may be treated with supplemental IV magnesium sulfate.

Physiologically, low magnesium states can interfere with the replacement of intracellular potassium. This in turn can lead to cardiac arrhythmias, symptoms of cardiac insufficiency,

and SCD. In an emergent situation, 1 to 2 grams of magnesium sulfate diluted in 100 mL of D_5W can be administered over 1 to 2 minutes. Care should be taken not to administer magnesium sulfate rapidly, as this could lead to subsequent hypotension or asystole. However, in the situation of VF/torsades thought to be due to hypomagnesemia, a more rapid administration can be carried out. [4]

Sodium Bicarbonate

The administration of sodium bicarbonate during CPR is controversial, and previous recommendations have been questioned for some time. [127] Although acidosis can reduce the responsiveness of the myocardium and vascular system to catecholamines, no clinical studies have supported the use of sodium bicarbonate for treatment of VF. Yakaitis and colleagues found that although acidosis did not alter defibrillation success in a canine model of brief VF (75 seconds), prearrest acidosis or hypoxia (in the absence of exogenous epinephrine) was rarely associated with resumption of perfusion. [128] Guerci and coworkers used a prolonged VF (20 minutes of CPR) canine model with epinephrine administration before defibrillation to assess the potential benefit of sodium bicarbonate administration. [129] These investigators failed to demonstrate an advantage from the use of sodium bicarbonate in either defibrillation success or maintenance of perfusion after defibrillation. The influence of acidosis on catecholamines appears to be most prominent at low doses and may not apply to the large amounts of epinephrine used during CPR. Current guidelines advocate the use of appropriate alveolar ventilation for the management of acidemia during cardiac arrest and in the immediate post-resuscitative period. If the patient is hyperventilated, thus removing excess carbon dioxide, an appropriate upward shift in pH will occur.

There are a number of theoretical reasons to avoid sodium bicarbonate therapy during cardiac arrest. Aside from the fact that it does not specifically facilitate defibrillation and may not be associated with a better final outcome, bicarbonate therapy will shift the oxyhemoglobin dissociation curve to inhibit oxygen release, induce hyperosmolar and hypernatremic states, produce paradoxical intracellular acidosis through an acceleration of CO_2 production, and exacerbate central venous acidosis. In addition, it may also cause a compromise in the coronary circulation.

Although sodium bicarbonate therapy should be considered for the patient with suspected or proven extreme acidosis, hyperkalemia, or both, it cannot be recommended for routine use as a standard part of the therapy for VF.

There are several exceptions to when bicarbonate may be beneficial in VF arrest; known preexisting metabolic acidosis, hyperkalemia, tricyclic overdose, or a prolonged resuscitation. Arterial blood gases with a bicarbonate and base deficit trend should be used to guide the administration of bicarbonate. 0.5 to 1 mEq/kg IV of sodium bicarbonate may be administered if the conditions noted earlier are suspected or present.

Bretylium Tosylate

Bretylium tosylate is a quaternary ammonium compound that has many complex actions on the cardiac action potential. However, it is noted for producing a hypotension and precipitating the release of catecholamines initially on injection with a subsequent postganglionic blockade. As of 1999 bretylium has been unavailable from the manufacturer. The scientific evaluation of bretylium regarding its efficacy in VF and hypothermic cardiac arrest has led to it being dropped from the current ACLS algorithms.

COMPLICATIONS

The major complications of DC defibrillation are (1) injury to skin and other soft tissue, (2) myocardial injury, and (3) cardiac dysrhythmias.

Soft Tissue Injury

When skin contact is firm and a conductive material is applied between the paddles and the chest wall, contact burns are usually minimal. Nevertheless, repeated countershocks can produce erythema of the skin resembling superficial first-degree burns. The presence of liquids (e.g., blood, IV solutions, vomitus, urine, excessive sweat) may permit the passage of current across the trunk. This electrical arcing will produce thermal burns (third-degree at times) and ineffective defibrillation. Hummel and coworkers have shown that repeated defibrillations using certain high-impedance conducting gels are associated with sparking and represent a fire hazard in an oxygen-enriched environment and an explosion hazard in the presence of nitroglycerin ointment or patches. [63] Intrathoracic injuries (extrinsic to the heart) are likely to occur but are difficult to document during the post-resuscitative period and to separate from cardiac injury (e.g., pulmonary edema). [130]

Myocardial Injury

The direct application of electrical countershock to the heart has long been known to produce epicardial and myocardial injury. Studies have demonstrated that closed-chest defibrillation is capable of producing cardiac injury. Electrical current rather than direct thermal injury produces injury. [31] [131] Multiple countershocks have been shown to produce ST segment elevation in animals and gradual cell necrosis (over days) with subsequent fibrosis. [132] The lesions are primarily subepicardial at the points of current entrance and exit. Animals receiving less than twice the defibrillation threshold value do not develop significant necrosis. The degree of cardiac injury correlates with increasing energy exposure.

Jones and coworkers, using an in vitro model, have created transient sarcolemmal microlesions during high-intensity electric field stimulation identical to that of defibrillation. [133] These lesions result in a "short-circuit" depolarization of the cells by loss of the normal sodium-potassium gradient across the sarcolemma. When the lesions are limited, the cell can recover after about 60 seconds. However, with extensive lesions, shock-induced cytosolic calcium overload can occur and result in post shock contracture.

The ability to document anatomic injury to the human heart is limited by the natural reparative process, concurrent ischemic processes producing similar microscopic changes, and the fact that several days are needed for the injuries to manifest themselves. Cardiac isoenzyme (CK MB) levels were shown to rise in patients undergoing elective cardioversion only if the cumulative delivered energy was >475 J. [134] Therefore, standard defibrillation should not generally interfere with the enzymatic diagnosis of myocardial infarction when defibrillation attempts are not excessive and given that fractionated isoenzymes are measured.

Animal studies have shown that ST segment elevation and pathologic changes are increased with more rapidly delivered discharges (1 or 3 seconds versus 15 seconds between discharges). [31] Furthermore, the cumulative energy correlates with myocardial injury for a given dosing schedule. [135]

Current research and development has led to the development of biphasic truncated energy delivery (i.e., biphasic truncated waveforms for DC defibrillators). In addition, newer model defibrillators are measuring the impedance to defibrillation current prior to actual discharge of the energy across the chest and myocardium. It is currently believed that this will lead to enhanced defibrillation success with less damage to the myocardium. This also should result in fewer adverse rhythms, e.g., persistent ventricular fibrillation, and fewer lethal arrhythmias post defibrillation, and less asystole and high-level heart blocks.

Cardiac Dysrhythmias

The rhythm that one obtains following defibrillation may be ventricular, supraventricular, or flatline (asystole). Laboratory studies have suggested a correlation between the severity of postdefibrillation dysrhythmias and the degree of myocardial damage produced. [136] Reducing the peak current delivered to the heart by changing the waveform of the discharge was associated with fewer dysrhythmias. Weaver and colleagues noted that asystole and transient heart block occurred more commonly with a higher discharge energy in prehospital VF patients. [80] Gueze and Koster also found that postdefibrillation dysrhythmias were more common following prolonged VF and higher-energy-level countershocks. [137] As stated earlier in this chapter, it is believed that the use of biphasic, impedance-compensated defibrillators will decrease the mortality and morbidity inherent to defibrillation countershocks.

Injuries to Health Care Providers

Any electrical device, including an improperly grounded or poorly insulated defibrillator, can cause injury to the device operator or to others in attendance. Other participants in a resuscitation who touch the patient or the stretcher can also serve as a ground for the defibrillator charge and can sustain electrical injury. Gibbs and coworkers estimate that the rate of paramedic injury during patient defibrillation is 1 per 1700 defibrillatory shocks. [138] They found only one paramedic who required hospital admission for therapy and monitoring of countershock ectopy. Improper use of the device for cranial countershock has been reported to produce short-term memory loss. [139] However, it must be borne in mind that such inappropriate use of these devices could easily cause much more serious injury. Fires resulting from defibrillator sparks in the presence of nitroglycerin patches or ointment, flammable gases, or an oxygen-enriched environment can also injure health care personnel as well as the patient.

SPECIAL TOPICS IN DEFIBRILLATION

Management of patients with an automatic implantable cardioverter defibrillator (AICD) is discussed in Chapter 13 .

Automatic External Defibrillators (in Hospitals)

AEDs (Fig. 12-12 (Figure Not Available)) have been developed with sophisticated digital software to take advantage of algorithms that can reliably recognize VF. [35] [65] These small, portable, battery-operated devices permit providers to defibrillate cardiac arrest patients without the need for human rhythm interpretation. [140] This advancement has taken defibrillation beyond the arena of the critical care setting such as the critical care unit (CCU), intensive care unit (ICU), emergency department (ED), or EMS vehicle and has extended the stage of defibrillation to include the lay public. The first effort to develop public access defibrillation was begun by Douglas Chamberlain in Brighton, England. AEDs were placed in/on trains, aircraft, and with families of high-risk cardiac patients. Extrapolation of the initial findings from these actions suggested that a significant increase in survival of prehospital cardiac arrest could potentially be achieved. [30] [141] [142] In light of these findings and the prior establishment of AEDs as a safe and effective resource for early defibrillation, [143] [144] an initiative to provide early, easy access to defibrillation and CPR by the lay public at various levels has been made. Early studies of these actions demonstrate an improved survival rate of victims of VF in prehospital SCD. In addition, a shorter time to defibrillation—"first shock"—and faster application of subsequent ACLS interventions [145] [146] were reported in the prehospital arena.

A group of investigators reviewed the data from in-hospital resuscitation of VF and discovered some unexpected and concerning results. Time to first shock by an in-hospital cardiac arrest resuscitation team did not occur for as long as 5 to 10 minutes. Furthermore, time to first shock was even longer in unmonitored hospital beds and diagnostic facilities. [20] These findings instigated a shift in the focus of in-hospital CPR

Figure 12-12 (Figure Not Available) SurVivaLink/Cardiac Science "Firstsave" Automatic External Defibrillator (AED). *(Courtesy of Cardiac Science, Inc., Irvine, CA.)*

training. Now the intent is to train nurses and clinical support personnel in CPR and AED technology. [147] [148] Current perception and prior research have led to the movement by various in-hospital committees and various hospital review councils to initiate placement of AEDs in various non-critical areas of hospitals. [149] [150] Also, current basic life support programs are being restructured to include AED application into the CPR training classes for general care nurses and first responders. [151] [152] Standard/typical manual defibrillators are having AED capabilities designed and built into them. This action is believed to lead to greater success rates in in-hospital resuscitations and provide the patient arriving into the ED with a better chance of survival to discharge. In fact, recommendations from the International Guidelines 2000 Conference [4] suggest that defibrillation be accomplished in 3± minutes from collapse within the confines of a hospital (Class I).

AED Application (Techniques)

Based on the design and programming of the newly devised AEDs, application and use of AEDs are quite simple. There are two major types of AEDs, fully automatic and semiautomatic. Fully automatic defibrillators are used only in special situations and are not be addressed in this chapter. The semiautomatic defibrillator is the type most commonly used in most emergency applications and is discussed in some detail here.

Various companies/vendors manufacture AEDs. However, the overall sequence of use is the same (Fig. 12-13).[42] [43]

Upon recognition of unresponsiveness in the victim, a call for help or 911, or both, if applicable, should be made. If the victim is found to be apneic and pulseless (i.e., no signs of spontaneous circulation), CPR should be initiated until an AED is brought to the scene/bedside. The AED should be placed to the left of the patient's head for the purpose of optimizing access to the patient and the AED controls. Subsequently, 4 "universal AED steps" should be completed:

1. **The AED should be turned on.** Power on is usually accomplished by depressing a switch or opening the cover of the AED. This will initiate a "self-check" of the AED. Subsequently there will be electronically synthesized voice prompts and, in many cases, visual instructions for the operator to follow (e.g., attach electrodes).
2. **Attach the electrode pads.** The pads used are self-adhesive and are placed over the skin. The correct position for pad placement is to the right of the sternum, just below the clavicle and lateral to the left nipple, about 7 cm below the axilla (Fig. 12-14). Care should be taken to avoid nitro patches or implanted defibrillators/pacemakers, or both, as they could result in poor current delivery or even explosion. The pads should be placed at least 2.5 cm (1 inch) away from these devices.[153] The pads function to monitor the rhythm and to deliver the defibrillatory shock. If the patient is very diaphoretic, a quick effort should be made to dry the chest with a dry cloth. If there is a problem with excessive chest hair, remove the pads and apply new ones. This will enhance contact, decrease TTI, and decrease potential artifact acquisition during the subsequent analysis of the rhythm. The position for placing the pads is often pictorially described on the pads or defibrillator.
3. **Analyze the rhythm.** Some AEDs are automatically programmed to analyze the rhythm once the electrodes are attached. Others will prompt the rescuer to depress an "analyze" button. To ensure that the analysis is not compromised, all contact with the patient should cease, including CPR, and no one should be in contact with the patient. All peripheral, nonessential, electronic devices should be turned off, if possible. The AED will then analyze the input waveform and provide an "advisory" if there is a shockable rhythm (i.e., VF). If a shock is "advised," the operator will proceed to the next step. If no shock is advised, CPR will be reinitiated.
4. **State "Clear the victim!" and press SHOCK.** Once a shock advisory has been given, state in a loud, definitive voice: "I'm clear, you're clear, everybody clear" and depress the shock button if it is safe to do so. No one should be in contact with the patient or any conductive material contacting the patient. The AED will then prompt the rescuer to either depress the analyze button or the AED will automatically analyze the rhythm. If another shock is advised, step 4 will be repeated until a volley of three successive shocks have been administered. These may be either escalated, with energy increasing or nonescalating, depending on the programming of the individual AED. If there is no success in achieving defibrillation, 1 minute of CPR is usually advised, followed by a repeat of steps 3 and 4 until 9 shocks have been delivered (see Fig. 12-13). In the hospital setting it is assumed that by the time algorithm for AED applications is exhausted, a resuscitation team will be at the victim's side to render further intervention if indicated.

Thump Defibrillation

The precordial thump consists of a firm, sharp, quickly delivered blow applied to the midsternum with a closed fist from a height of about 30 to 38 cm above the anterior chest wall of a pulseless patient. It is believed that such a blow is capable of generating approximately 5 J of energy. This, however, should only be considered when a defibrillator is not immediately available.

Although such a precordial thump may be of limited value in witnessed VT/VF arrests, Miller and associates noted no VF conversion in 23 prehospital VF patients. [154] Despite the fact that the rhythm was improved in 3 of 27 VT patients after a precordial thump, the rhythm deteriorated in 12 of the 27 and did not change in the other 12 patients. Caldwell and colleagues reported the results of a precordial thump in 68 cases of VT and 248 cases of VF. [155] They reported 26 favorable cardioversions, including in 5 patients in VF. Because successful internal defibrillation can occur with as little as 1 J following cardiopulmonary bypass, a vigorous precordial thump may create sufficient current flow to defibrillate the heart when the duration of arrest has been brief (i.e., witnessed cardiac arrest).

Special Situations: Transdermal Medications, Implantable Pacers/Implanted Automatic Cardioverter-Defibrillators

Care should be taken when defibrillating a patient with either a pacemaker or an implantable automatic cardioverter-defibrillator (AICD). Paddle/pad placement should be 2.5 cm (1 inch) away from the actual device to minimize the potential for perpetuation of artifact during rhythm analysis. Also, placing the paddles at an appropriate

distance from the

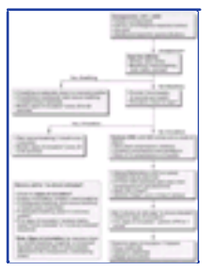

Figure 12-13 Automatic external defibrillator treatment algorithm for emergency cardiac care. *(From AHA Guidelines 2000, Circulation 102(suppl):1, 2000. Reproduced with permission.)*

ancillary device reduces the possibility of current scatter and diminution due to a potential increase in chest wall impedance. The same arguments hold true for placement of defibrillation pads and paddles away from nitroglycerine patches, nicotine patches, hormone replacement patches, and analgesic patches. There have been actual reports of injuries to the skin during defibrillation. [153]

Refractory Ventricular Fibrillation

The major determinants of successful defibrillation are the early use of countershock, adequate oxygenation, lack of serious metabolic derangements, and general health of the patient. Hargarten and colleagues note that few prehospital patients who remain in VF after the fifth shock are subsequently successfully converted to an effectively perfusing rhythm.[8] Kerber and colleagues note that patients who never defibrillated successfully despite multiple shocks had a prolonged duration of CPR preceding the first shock (21 ± 14 minutes) and systemic hypoxia and acidosis. [156] These conditions were noted to occur when initial cardiac arrest rhythm was asystole, severe bradycardia, or PEA.

A number of clinical conditions may result in the inability to convert VF initially or in the recurrence of VF following the first successful defibrillation. Patients with severe hypothermia are often refractory to initial defibrillation and

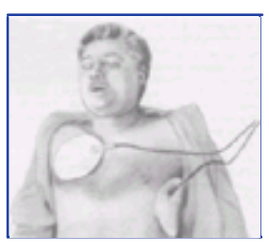

Figure 12-14 Correct automatic external defibrillator electrode placement on victim. *(From AHA Guidelines 2000, Circulation 102(suppl):1, 2000. Reproduced with permission.)*

generally require rapid core rewarming to be successfully defibrillated. Severe bradycardia will predispose to lethal escape rhythms, and emergency cardiac pacing may be required. Severe electrolyte disturbances, such as hypokalemia, hypomagnesemia, and hypocalcemia may precipitate refractory VF and be amenable only to the appropriate, rapid correction of the deficient electrolyte. Such situations may occur especially in fad dieters or abusers of diuretic medications. Uncorrected acidosis or hypoxia, such as that seen with drowning, may be the cause of persistent VF. In addition, excessive adrenergic stimulation, such as that seen with cocaine or amphetamine overdose, may require the use of a beta blocker or a mixed alpha beta blocker (e.g., labetalol) infusion before successful defibrillation. As a final note, following defibrillation, all patients should be treated with antidysrhythmic drug therapy to minimize the chance of recurrent VF.

CONCLUSION

Prompt recognition and electrical defibrillation is the preferred treatment for VF-related sudden death. Treatment should be initiated as early as possible with strict attention to selection of the proper energy dose as well as to minimization of TTI. Although repetitive high-energy shocks may be associated with myocardial injury, this factor has not been found to be a clinical problem at currently recommended energy levels. Current biphasic defibrillators may decrease myocardial injury and prove to be more successful in returning spontaneous circulation after VF.

Medications such as amiodarone, vasopressin, epinephrine, and lidocaine may have some benefit in decreasing the VF threshold and may be used at the discretion of the clinician. The clinical situation and patient history may be guides.

Pediatric Defibrillation

VF is much less common in children than in adults. In children causes are most likely sudden infant death syndrome or respiratory disease. [157] [158] [159] There is an age-related pattern in the occurrence of cardiac arrest in the pediatric population: About half of the cardiac arrests that occur in children are encountered before the age of 1 year. After 6 months of age, drowning and injuries are reported to be the major causes of death. The most common terminal rhythms reported in children under the age of 17 years are PEA, bradycardia, or asystole. [160] The etiology behind these pediatric arrhythmias is most often hypoxemia, hypotension, hypoglycemia, and acidemia. In addition, focal electrical ectopy is less likely to initiate VF in the young heart. A significant myocardial mass must be unstable and fibrillating before VF becomes established. In children (from birth to 8 years of age) with nontraumatic arrest, only 3% of the dysrhythmias are reported to be VF. In victims aged 8 to 30 years of age, the number of VF patients increases by almost 6-fold (17%). [161] Several subpopulations of pediatric patients at various ages with cardiomyopathy or myocarditis or those patients who have undergone heart surgery are at increased risk for a primary dysrhythmia.

The incidence of VF in cardiac arrest rhythms of pediatric patients is reported to be from 7% to 15%. [162] Others have reported that approximately 10% of reported pediatric cardiac arrest patients had VF. [163] In a retrospective prehospital study of pediatric patients, VF was reported to have been found in almost 20% of the cardiac arrest victims. [162] Patients who have been defibrillated from VF have been reported to have a higher survival-to-discharge rate than children who sustained asystole or PEA. [162] Therefore, there is a definite indication for early defibrillation in the pediatric population.

Procedure and Technique

The procedure for pediatric defibrillation (Fig. 12-15) is similar to the algorithm for adult defibrillation (see Fig. 12-13). However, there are a few differences that must be addressed. These differences are discussed in the following section.

Paddle/pad applications/conductive materials.

To acquire the electrical rhythm and subsequently administer an effective defibrillatory shock, appropriately sized pads/paddles must be correctly placed onto the chest. The appropriate size and placement of the paddles/pads will ensure that the appropriate current density be delivered across the myocardium to effectively defibrillate the myocytes. Furthermore, appropriate pad/paddle size (i.e., the largest surface area possible without direct electrode-to-electrode contact) will decrease TTI and enhance defibrillation. [164] To accomplish this, it is recommended that infant paddles be used in children who weigh <10 kg. However, larger paddles can be used if they do not contact each other; if contact is made between the paddles, an electrical arc or short could occur. [165] In children who weigh more than 10 kg (mean age 1 year), it is recommended that adult pads/paddles be used (8 to 10 cm in diameter). [166] Regarding enhancing skin contact and decreasing TTI, a conductive agent should be used. Never use dry paddles, as the resistance to current flow will be very large. However, refrain from using saline-soaked pads in children as they may cause arcing due to the proximity of the pads on the chest; remember that electricity will take the path of least resistance and that the current from defibrillation will travel across the chest if there is a saline bridge between the electrodes. Also, the use of ultrasound gel and alcohol pads is discouraged because of poor electrical conductivity and potentially high impedance. [62] Also, care should be taken with repeated shocks, because there have been reports of skin burns if inadequate contact and inadequate conductor gel is not used. [167]

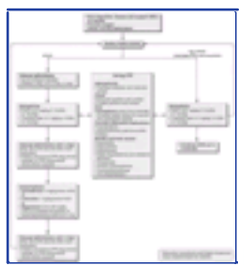

Figure 12-15 Pediatric pulseless arrest Pediatric Advanced Life Support treatment algorithm. *(From AHA Guidelines 2000, Circulation 102(suppl):1, 2000. Reproduced with permission.)*

The paddles or pads should be applied firmly to the chest—one to the right of the sternum, just below the clavicle, the other to the left of the left nipple, over the ribs (over the apex of the heart). An option when using self-adhesive pads is to place one pad just to the left of the sternum and the other over the back, approximating the position of the heart. [168]

Energy Selection and Pharmacotherapy

Currently there is no definitive evidence to support any specific energy for successful defibrillation in the pediatric population. However, available data have indicated that a dose of 2 J/kg (Joules/kilogram) may be used in the first shock attempt for children up to at least 8 years of age. [83] If this

action does not terminate VF, then the energy dose should be doubled to 4 J/kg and repeated. [169] A third shock may be given at the same energy dosage if there is still no success in defibrillation. The three shocks should be given in rapid succession, allowing time only for determination of the underlying electrical rhythm. If the first 3 initial shocks fail to terminate VF, then attention must be turned to identifying and treating hypoxia, acidemia, hypothermia, and if indicated administering pharmacological pressor agents such as epinephrine (1:10,000; 0.01 mg/kg) (intravenously or intraosseous [IO]) or (1:1000; 0.1 ml/kg tracheal tube) (see Fig. 12-15). Another defibrillatory shock (4 J/kg) should be administered 30 to 60 seconds post drug administration. CPR should be performed between interventions. If no success is achieved after 4 shocks, the agent amiodarone (5 mg/kg bolus IV/IO) (Class Indeterminate) may be administered (see Fig. 12-15). Use of amiodarone is suggested only on the basis of experience with adult victims to date. [125] Lidocaine (1 mg/kg bolus IV/IO) or high-dose epinephrine (Class IIb) may also be considered at this point if there is no success. Once the drugs have been administered, another shock at 4 J/kg should be delivered. It is also acceptable to administer three successive shocks in between drug interventions. [4]

Waveforms

Newly manufactured defibrillators and AEDs are using the biphasic waveform. As mentioned previously in this chapter, there are various forms of the wave (e.g.,

truncated, damped sinusoidal). This waveform has been described as being effective in achieving defibrillation at lower energy levels and as being less damaging to the myocardium.[52] Many new manual defibrillators and AEDs are being released for use with this new technology. However, data regarding the efficacy and use of the biphasic waveform in the pediatric population is limited as of this writing. Extrapolating from the data currently available in adult applications of biphasic defibrillation, it is the belief of these authors that biphasic waveforms will be in use in the near future in defibrillators designed for use in the pediatric population. Although use of the biphasic waveform allows for a decrease in the amount of energy needed to defibrillate, current guidelines suggest that the 2 J/kg and subsequently the 4 J/kg energy be used.[4] These energy selections may undergo modification as more high-level data is collected regarding biphasic defibrillation technology.

AEDs in Children

AEDs are now being used in adults in various areas with some success. However, pediatric AED applications are in the investigative stages. Data suggests AEDs can accurately detect VF in children of various ages. [170] [171] [172] However, current energy levels delivered by both the monophasic and biphasic defibrillators even at 150 to 200 J exceeds the 2 to 4 J/kg level. In fact, the energy actually delivered is more in the range of <10 J/kg in patients older than 8 years of age. This is based on a mean weight of 25 kg for an 8-year-old approximately 50 inches tall (128 cm). [173] Animal data suggests that this is still a safe dose for defibrillation in children. However, currently standard biphasic/monophasic AEDs are not recommended for use in children. [174] Some vendors have developed adapters to adjust energy delivery to the patient, but these AEDs are in limited distribution.

In summary, currently monophasic or biphasic AEDS are not recommended for use in children under 8 years of age or weighing <25 kg. However, there is speculation that AEDs will be modified to enable use of biphasic low-energy AEDs in the pediatric population.

CARDIOVERSION

Cardioversion is the application of direct electrical current across the chest or directly across the ventricle to normalize the conduction pattern of a rapidly beating heart. Defibrillation refers to application of electrical energy during the nonvulnerable period to restore a fibrillating ventricle to normal sinus rhythm.

The patient with a significant tachycardia may be asymptomatic or may complain of chest pain or discomfort, lightheadedness, or shortness of breath. These symptoms are the result of altered cardiovascular physiology. Rapid cardiac rhythms allow less time for ventricular filling, resulting in reduced preload and hypotension. The reduced preload as well as the increased ventricular work caused by the rapid heart rate may also result in ventricular ischemia. Pulmonary capillary wedge pressures may also rise despite shortened filling time, due to reduced ventricular compliance secondary to ventricular ischemia. Elevated pulmonary capillary wedge pressures can then lead to pulmonary edema.

Termination of rapid rhythms to alleviate or prevent these symptoms must occur quickly to prevent further deterioration. Persistently poor cardiac output due to rapid heart rate results in development of a lactic acidosis that further compromises cardiac function and makes cessation of the dysrhythmia even more difficult. Unchecked myocardial ischemia may lead to infarction with its attendant sequel. Drug therapy, rapid cardiac pacing, and cardioversion are the methods available to terminate tachydysrhythmias.

In many cases, direct-current cardioversion has specific advantages over drug therapy. The speed and simplicity of electrical cardioversion enhance its usefulness in the ED setting. Cardioversion is effective almost immediately, has few side effects, and is often more successful than drug therapy in terminating dysrhythmias. In addition, the effective dose of many antidysrhythmic medications is variable, and there is often a small margin between therapeutic and toxic dosages. Although they can often suppress an undesirable rhythm, drugs may also suppress a normal sinus mechanism or may create toxic manifestations that are more severe than the dysrhythmia being treated.

In the clinical setting of hypotension or acute cardiopulmonary collapse, cardioversion may be life saving. The key concepts in the use of this procedure include understanding the indications for its use, the equipment involved, the importance of adequate sedation, and the concerns for health-worker safety.

BACKGROUND

The first successful defibrillation of the human heart was performed in 1947 by Beck. By the 1960s, electrical energy was being used to treat dysrhythmias other than VF. AC remained in vogue until 1962, when Lown and colleagues advocated DC countershock as the method of choice for terminating atrial fibrillation. [175] The use of DC significantly decreased the incidence of VF following countershock.

A brief burst of electrical current momentarily causes depolarization of the majority of cardiac cells and allows the sinus node to resume normal pacemaker function. In reentrant

dysrhythmias, such as paroxysmal supraventricular tachycardia (SVT) and VT, cardioversion restores sinus rhythm by interrupting a self-perpetuating circuit. Cardioversion is much less effective in terminating tachycardias resulting from augmented automaticity, such as digitalis-induced dysrhythmias.

Monophasic versus Biphasic Wave Form Defibrillators

From the pioneering days of defibrillation technology in the 1950s until the start of the 20th century, traditional defibrillators generated a monophasic damped sine wave form to deliver the energy needed to convert tachydysrhythmias. Starting in the 1990s, biphasic technology was introduced for AEDs and automatic implantable cardiac defibrillators (AICD). The biphasic defibrillator is now positioned to replace the monophasic waveform defibrillator in all hospital-based defibrillators.

In contrast to the monophasic defibrillator, the biphasic wave generator delivers current in two directions. During the first phase, the biphasic current travels from one paddle to the other, similar to the monophasic wave form. However, during the second phase, the current *reverses* direction. This distinctive feature has been proven to lower the electrical threshold for successful defibrillation and cardioversion.

INDICATIONS AND CONTRAINDICATIONS

Cardioversion is indicated whenever a reentrant tachycardia causes unstable vital signs, ischemic chest pain, or otherwise significantly compromises cardiovascular or respiratory function. No specific parameters exist and the decision to perform cardioversion is best made on a case by case basis by the clinician at the bedside. It is also indicated on an elective basis when medical therapy has failed. In any situation where a prolonged rapid heartbeat can be anticipated to cause complications related to cardiac ischemia or dysfunction, early intervention with cardioversion may also be considered.

A reentrant tachydysrhythmia should be suspected when a sudden change in the heart rate occurs within a few beats. Unless the dysrhythmia is noted while the patient is being monitored, it can only be inferred from the patient's history of sudden onset of symptoms. In the unusual case of sinus node reentrant tachycardia, rapid onset and offset may be the only clue.[176] Other clues to the presence of a reentrant dysrhythmia are a history of Wolff-Parkinson-White syndrome or another known accessory pathway syndrome. Ventricular rates in excess of those predicted for age strongly suggest an accessory pathway.

Dysrhythmias due to enhanced automaticity will not be terminated by uniformly depolarizing myocardial tissue because a homogeneous depolarization state already exists. Enhanced automaticity is the cause of most cases of digitalis toxicity-induced dysrhythmia, sinus tachycardia, and, probably, multifocal atrial tachycardia. [177] Enhanced automaticity means that the threshold for phase 4 depolarization has been lowered or that the rate of ion leak during phase 4 has been accelerated. This effect on phase 4 depolarization is caused by alterations in the metabolic or chemical environment or on the cell membrane, causing pacemaker cells to fire more rapidly. Although cardioversion will not be successful in these cases, medications that suppress automaticity, including potassium and magnesium, may be useful.

In digoxin toxicity, cardioversion is not only ineffective, but it is also associated with a higher incidence of postshock VT and VF. [178] However, for a patient with a therapeutic digoxin level, the risk of cardioversion is no different from that of other patients. [179] Digoxin is still generally withheld for 24 hours prior to cardioversion as a precaution against inadvertently elevated levels. Pregnancy at any stage is not a contraindication to cardioversion. [180] [181]

Supraventricular Tachycardia with Aberrancy versus Ventricular Tachycardia

Determining the rhythm is critical for the clinician to make the appropriate clinical/pharmacologic intervention. However, at times SVT may manifest patterns on the ECG that look very similar to VT. An incorrect assessment of the ECG can prompt the clinician to implement a pharmacologic/therapeutic intervention which may result in cardiovascular collapse. Although a comprehensive discussion of SVT versus VT is beyond the scope of this chapter, some salient features to look for are presented herein to assist in the emergent decisionmaking process of the clinician.

A few caveats must be kept in the forefront when facing the task of discriminating between SVT and VT. A wide-complex tachycardia refers to a dysrhythmia where the ventricles beat at more than 100 beats/minute and the QRS duration is 0.12 seconds or more. [182] [183] The originating foci for these wide-complex tachycardias can be either supraventricular or ventricular. For a supraventricular focus to produce a wide-complex tachycardia, a preexisting or new onset intraventricular conduction block must be present, resulting in increased time of depolarization. Increased heart rate or ischemia can also precipitate the appearance of a wide-complex tachycardia when it is supraventricular in origin. If the focus of the tachycardia is below the AV node, the tachycardia is considered ventricular in origin. [182] Criteria to facilitate the process of discriminating between SVT and VT were compiled by Wellens and Brugada. The Wellens criteria use several clinical data points to help determine if the tachycardia is ventricular or supraventricular in origin. [182] The Brugada criteria extend the Wellens criteria and add a four-step decision-tree approach to the process.[185] Although the methods are not without pitfalls, a careful scrutiny of the ECG in light of the aforementioned criteria will usually lead to the appropriate diagnosis.[186]

Following are characteristics of SVT versus VT. Although the guidelines and criteria appear clear-cut, there are times when exceptions occur in the clinical setting (Table 12-4 , Table 12-5 , Table 12-6 , and Table 12-7).

Special Considerations: Wide-QRS-Complex Tachycardias

Wide-complex tachycardias (wide-complex SVT) are diagnostic challenges in clinical medicine. The criteria often used to define wide-complex SVT (WCSVT) is a tachycardia with a QRS duration of >0.12 seconds. It is important to differentiate the rhythm as one of the following: VT, SVT with aberrancy (LBBB or RBBB), or an accessory AV pathway ("pre-excitation"). The need for a proper diagnosis is obvious. Incorrect diagnosis and inappropriate treatment can be life threatening. This is especially true in misdiagnosing VT as SVT. Studies have shown that VT is often misdiagnosed, even with the

TABLE 12-4 -- Standard Criteria to Differentiate SVT from VT

Rate:	Too much overlap to make rate a useful criterion.
Regularity:	Grossly *irregular* complexes are likely to represent one of three conductions: (1) AF with aberrancy, (2) AF with conduction through an accessory pathway, or (3) irregular form of VT. Rates greater than 200 bpm with a wide-QRS-complex are highly suggestive of AF with aberrancy or AF with accessory pathway conduction.
AV Dissociation, Fusion Beats: (Table 12-6)	AV dissociation during tachycardia with a wide-complex is highly suggestive of VT.
QRS Axis:	Preservation of normal QRS axis in a wide-complex tachycardia favors a diagnosis of SVT with aberrancy. Change in axis or extreme left or right axis deviation is often seen in VT. Abrupt change in QRS morphology is frequently seen in VT but not in SVT.
QRS Duration:	QRS duration greater than 140 ms occurs more frequently in VT compared to SVT.
QRS Concordance:	Concordance in the precordial leads is rarely seen in SVT with aberrancy.
	Concordance supports a diagnosis of VT.
QRS Morphology:	*RBBB-shaped complex:* To distinguish RBBB aberration in SVT from VT, the presence of a triphasic pattern in V1 or a qRs pattern favors aberrancy. While a monophasic or biphasic QRS in V1 or an rS or QS complex in V6 favors VT.
	LBBB-shaped complex: SVT is suggested if the LBBB pattern has a small initial r wave with a steeply down sloping S wave. An initial r wave (>30 ms) and a notched, broad (>70 ms) down slope is favorable for VT. QS complex or a qR in V6 suggests VT.

AF, atrial fibrillation; AV, atrioventricular; LBBB, left bundle-branch block; QRS, QRS Interval; RBBB, right bundle-branch block; SVT, supraventricular tachycardia; VT, ventricular tachycardia.

ready availability of clinical and ECG criteria. In one study, VT was correctly diagnosed in 32% of patients presenting with wide-complex tachycardias. [184]

Etiology

Normally, ventricular depolarization is initiated when the His bundle depolarizes both ventricles simultaneously through the bundle branches and Purkinje fibers. Normal depolarization takes place within 80 to 120 ms. Prolongation of the QRS duration happens if (1) the ventricles are activated sequentially rather than simultaneously (this is the case in VT, bundle-branch blocks, or accessory pathway ventricular activation [WPW, LGL]), or (2) when His-Purkinje-myocardium

conduction is slowed from ischemia, drugs, or electrolyte disturbances.

Classification

Wide-complex tachycardias fall into three classifications based on mechanism: (1) VT, (2) SVT with aberration, and (3) pre-excitation tachycardia. VT is the most common cause

TABLE 12-5 -- Brugada Algorithm

First reported in 1991, this algorithm was designed to aid the clinician in diagnosing lethal VT from the less urgent SVT with aberrancy conduction. Taking the standard 8- to 10-point criteria used by cardiology at the time, Brugada et al. focused on four ECG criteria to aid in diagnosing VT versus SVT with aberrancy. To differentiate VT from SVT with aberrancy, the Brugada Algorithm uses the following ECG criteria. *First*, examine the ECG. Is the rhythm regular? An irregular rhythm highly suggests atrial fibrillation with aberrancy. Does the purposed dysrhythmia fit the clinical picture ascertained by the history? *Then*, ask the following questions:
• Absence of an RS complex in all precordial chest leads?
• R to S interval > 100 ms in one precordial lead? (measured from beginning of R to deepest part of S wave)
• Atrioventricular dissociation? (Table 12-4)
• Morphology criteria for VT present in precordial chest leads V1-2 and V6? (Table 12-4)
(A single "yes" response suggests VT Only when none of the VT criteria are affirmed is SVT diagnosed
Adapted from Brugada et al., 1991).
Wellens Criteria
In a landmark study published in 1978, Wellens et al. determined that the findings suggestive for a ventricular origin of tachycardia were the following * :
• QRS width over 0.14 sec
• Left axis deviation
• AV dissociation (Table 12-4)
• Certain configurational characteristics of the QRS morphology (Table 12-4)
AV, atrioventricular; ECG, electrocardiogram; QRS, QRS Interval; SVT, supraventricular tachycardia; VT, ventricular tachycardia. *Wellens et al., 1978

of wide-QRS-complex tachycardias. It is defined as 3 or more consecutive ventricular beats at a rate of 100 beats/minute. VT is further classified as nonsustained (tachycardia lasting <30 seconds) or sustained (tachycardia that lasts >30 seconds). Sustained tachycardia usually results in hypotension or syncope and requires termination intervention. SVT is a tachydysrhythmia using the normal AV conduction system for ventricular activation. This tachycardia originates in the SA or AV node. To sustain propagation, the AV node is recruited. Aberrancy refers to the existence of an aberrant or nontraditional conduction mechanism, resulting in a longer depolarization phase. SVTs with aberrancy by definition must result in wide-QRS-complex tachycardias. The two forms of aberration are fixed (i.e., a permanent bundle-branch block or functional block that is a rate-dependent bundle-branch block). The most common areas of the His-Purkinje functional block are in the left or right bundle-branches. Sudden acceleration is often the initiating cause of the SVT with aberrancy. The aberrancy is maintained by a continuous,

TABLE 12-6 -- Electrocardiogram (ECG) Evidence of Atrio-Ventricular Dissociation

1. Dissociated P waves at a slower rate than the ventricular rate
2. Presence of Fusion or Capture beats
3. Irregular changes in ST-T waves suggesting presence of dissociated P waves
4. P wave and QRS complexes at different rates on adjunct ECG recording (esophageal or nasogastric approach)
(Modified from Gupta AK, Thakur RK: Wide-QRS-Complex Tachycardias. Med Clin North Am 85:245, 2001.)

TABLE 12-7 -- Characteristics of Wide QRS Tachycardia Suggesting Ventricular Tachycardia

1. AV Dissociation, Fusion or Capture Beats
2. Positive or Negative Concordance in V1–V6
3. QRS Configuration
RBBB-shaped:
V1: Monophasic or biphasic QRS complex
V6: R:S ratio < 1
LBBB-shaped:
V1: r > 30 ms, S > 70 ms, notched S
V6: qR pattern
AV, atrioventricular; LBBB, left bundle-branch block; QRS, QRS Interval; RBBB, right bundle-branch block.
(Modified from Gupta AK, Thakur RK: Wide QRS Complex Tachycardias. Med Clin North Am 85:245, 2001).

concealed, retrograde conduction pathway, which leads back into the blocked area.

For preexcitation wide-QRS-complex tachycardias, AV conduction occurs over two circuits: (1) normal AV nodal conduction or (2) through an accessory pathway. These two pathways create a reentry circuit. Aberration appears because of the presence of intraventricular conduction block. Preexcited tachycardia is any tachycardia where the ventricles are *antegradely* activated over an accessory pathway. The most common preexcited tachycardia is atrial fibrillation with ventricular activation over an accessory pathway.

Clinical Diagnosis

The directed history on presentation may provide the most valuable clues to the diagnosis of tachydysrhythmia. The on and off occurrence of tachydysrhythmia in the past, the age of the patient, and the age of past occurrence all are important indicators of preexcitation rhythms usually found in young patients. Sudden onset of tachydysrhythmia in the older coronary-prone patient or patient with structural heart disease points more toward ventricular tachycardias. Symptoms associated with the tachydysrhythmia are important clues in diagnosis. The young patient often has few if any symptoms when experiencing the wide-complex or narrow-complex SVTs. The older patient may experience the entire range of cardiac symptoms. Tachydysrhythmia present for long periods often defines SVT. Patients with SVT often have recurrent tachycardias from their childhood or early adulthood. Attention must be paid to the medications the patient is using. Antiarrhythmic medications have a

use-dependency property. Conduction velocity is slowed as rates increase. [187]

Electrocardiographic Criteria for Differentiating Ventricular Tachycardia from Wide-Complex Supraventricular Tachycardia

The clinician should not attempt the differential diagnosis of wide-QRS-complex tachycardias without the use of the 12-lead ECG and extended rhythm strip. A common error is to attempt to determine the cause of a tachycardia based only on the rhythm strip. A comparison of past ECGs is often helpful. Examination of the ECG should focus on the following areas: rate, regularity, AV dissociation, QRS axis, QRS duration, QRS concordance, and QRS morphology (see Table 12-4).

Treatment

Therapy is dictated by the specific wide-complex tachycardia and the patient's clinical presentation. The emergency clinician's initial approach must always be led, and modified if necessary, by the patient's presentation and subsequent changes. It is recommended in all cases of wide-complex tachycardias, and narrow-complex tachycardias, which are producing hemodynamic instability, that the clinician should immediately consider use of cardioversion. Synchronized monophasic or biphasic cardioversion is the appropriate first choice of treatment for these cases.

In patients with wide-complex tachycardias who are cardiovascularly stable, the therapeutic options are more diverse. Stable, wide-complex tachycardia can always be considered VT and treated according to current VT protocols. [188] Verapamil should never be used in unknown etiology wide-complex tachycardia. A reasonable treatment protocol for stable patients may be the use of adenosine, procainamide, lidocaine, and finally cardioversion. Amiodarone is effective for most SVTs and its use in stable unknown wide-complex SVT is both appropriate and safe. [189]

EQUIPMENT AND SETUP FOR CARDIOVERSION

The critical components of preparation for cardioversion are IV access, airway management equipment, drugs for sedation, monitoring equipment, and DC delivery equipment (cardioverter).

Secure IV access is essential for delivery of sedatives, antidysrhythmics, fluids, and, possibly, paralytic agents. Although many of these drugs are not used routinely, if they are needed, timing is likely to be critical. A saline lock catheter of at least 20 ga should be inserted and secured.

A significant and preventable complication of procedures involving sedation is hypoventilation leading to hypoxia. Airway management equipment includes working suction with tonsil-tipped device attached, bag-valve-mask apparatus, oxygen, and an appropriately sized laryngoscope and endotracheal tube. A pulse oximeter is generally recommended for patients undergoing conscious sedation. Another adjunct is continuous pCO_2 monitoring. A rising pCO_2 level will be an earlier clue to hypoventilation due to sedation than pulse oximetry because the O_2 saturation may remain normal for several minutes, especially if the patient has been preoxygenated.

Sedative medications should be ready for use in labeled syringes, with a prefilled saline flush. Antidysrhythmic medications for ventricular dysrhythmias (e.g., lidocaine and amiodarone) and for unexpected bradycardia (e.g., atropine) should be readily accessible.

The cardioverter device consists of 5 components (see Fig. 12-7 (Figure Not Available)): (1) a DC depolarizer, which provides varying amounts of electrical current; (2) an oscilloscope screen for monitoring heart rate and rhythm; (3) access to a continuous ECG readout to document the patient's course and response to treatment; (4) 2 removable electrode paddles that can be applied easily to the patient's chest wall; and (5) a synchronizer, permitting discharge of energy outside the vulnerable period of the cardiac cycle. The synchronizer permits triggering of the electrical discharge by the R or S wave of the ECG (see Fig. 12-3).

Paddles must be large enough to depolarize the majority of heart fibers simultaneously; therefore, most conventional paddles have an electrode diameter of at least 4 inches. Larger paddles also limit the risk of myocardial injury by decreasing the density of current passing through the myocardium.

TECHNIQUE

If time permits, metabolic abnormalities such as hypokalemia and hypomagnesemia should be corrected before attempting cardioversion. Hypoxia should be corrected with supplemental O_2 . If a patient has a metabolic acidosis, compensatory hyperventilation after endotracheal intubation may be indicated prior to cardioversion. Respiratory acidosis should always be treated prior to the use of sedative drugs.

Sedation and Anxiolysis

Cardioversion may be extremely painful or terrifying, and *patients must be adequately sedated prior to its use*. Patients who are not adequately sedated may experience extreme anxieties and fear.[190] Several IV medications are available for sedation of patients prior to cardioversion. These include etomidate (0.15 mg/kg), midazolam (0.05–0.1 mg/kg), methohexital (1 mg/kg), propofol (0.5–0.8 mg/kg over 3–5 minutes), and thiopental (3 mg/kg). [191] [192] In addition, fentanyl (1–2 mcg/kg), a synthetic opioid analgesic, is sometimes administered 3 minutes prior to induction. The above doses are commonly used, but the agents should be titrated to effect as outlined in Chapter 34 .

Midazolam (Versed) is probably the most commonly used agent, with deep sedation occurring about 2 minutes after IV injection. Although induction with midazolam takes slightly longer than the other medications, it has the advantage that a commercial antagonist, flumazenil, is available for reversal, if necessary. Small additional doses of fentanyl (1 to 1.5 mcg/kg) may be added for more profound sedation. Fentanyl can cause respiratory depression, but it is reversed with naloxone. Methohexital has the advantage of quick onset and somewhat shorter duration than midazolam, but it has a rare association with laryngospasm. All the drugs except etomidate cause a small drop in blood pressure.

In elderly patients the pharmacodynamics and kinetics of medications used for sedation/anxiolysis are altered by coexisting illness and polypharmacy, rather than by any intrinsic effect of old age. [193] Older patients with medical conditions such as congestive heart failure, renal failure, cancer, or malnutrition will therefore experience deeper, prolonged sedation with increased respiratory depression. Drug dose should be adjusted accordingly in these patients.

Cardioverter Use

Selection of synchronized or nonsynchronized mode is the next critical step. In "synchronized" mode, the cardioverter searches for a large positive or negative deflection, which it interprets as the R or S wave. It then automatically discharges an electric current that lasts <4 msec, avoiding the "vulnerable" period (see Fig. 12-3) during repolarization when VF may be induced. When the cardioverter is set to "synchronize," a brief delay will occur after the buttons are pushed for discharge, as the machine searches for an R wave. This delay may be disconcerting to the unaware operator.

If concern exists about whether the R wave is large enough to trigger the electrical discharge, the clinician can place the lubricated paddles together and press the discharge button. Firing should occur after a brief delay. When the R- or S-wave deflection is too small to trigger firing, change the lead that the monitor is reading or move the arm leads closer to the chest.

If there is no R or S wave to sense, as in VF, then the cardioverter will not fire. *Always turn off "synchronization" if VF is noted*.

Electrode Contact

A number of substances can be used to ensure good contact between the paddle and the skin, it is necessary that they be nonflammable and have a low electrical resistance. Conductive gel or paste is most commonly used, but waxy conductive pads are also available. Generous use of conductive gel on the underside and especially along the edges of the electrode paddles is essential, both to reduce TTI and to prevent skin burns. Paste should be applied liberally but must not run onto the skin between the paddles, because the paste may divert current over the skin surface and away from the heart. Even under ideal circumstances, only 10% to 30% of the total current passes through the heart, so diversion over the skin may significantly reduce the effectiveness of an electric discharge. Saline-soaked pads are therefore generally not desirable. Pregelled adhesive electrode pads are useful if available.

Wet Gel Electrodes

The tendency for solid gel electrode pads to burn patients during cardioversion has led to the re-introduction of wet gel electrodes. The return of the wet gel electrodes was centered on conductivity and skin coupling features. On both issues, wet gel electrodes performed better than solid gel electrodes.

Electrode Position

Electrode paddles may be positioned in 2 ways on the chest wall: (1) the anterolateral (or base and apex) position, with one paddle placed in the left fourth to fifth intercostal space, midaxillary line, and the other just to the right of the sternal margin in the second to third intercostal space (see Fig. 12-10); or (2) the anteroposterior position, with one paddle placed anteriorly over the sternum and the other on the back between the scapulae (see Fig. 12-11). The anterolateral position is used for emergent cardioversion, when placement of an electrode on the patient's back may not be feasible. Paddles should be pressed firmly against the skin to avoid arcing or skin burns.

Safety is a key concern in the performance of cardioversion. Any staff member acting as a ground for the electrical discharge can be seriously injured. The operator must announce "all clear" and give staff a chance to move away from the bed before discharging the paddles. Care must be taken to clean up spills of saline or water, because they may create a conductive path to a staff person at the bedside.

Energy Requirements

The amount of energy required for cardioversion varies with the type of dysrhythmia, the degree of metabolic derangement, and the configuration and thickness of the chest wall (see Table 12-2). Obese patients may require a higher energy level for cardioversion; the anteroposterior paddle position is sometimes more effective in these patients. If patients are shocked while in the expiratory phase of their respiratory cycle, energy requirements may also be lower.

Ventricular tachycardia in a hemodynamically stable patient should be treated with lidocaine followed by procainamide, and amiodarone if necessary. If these drugs are unsuccessful, cardioversion is then used. Cardioversion with 10 to 20 J is successful in converting VT in more than 80% of cases. Cardioversion will be accomplished with 50 J in 90% of

cases, and conversion should be initially attempted at this energy level. [194] Cardioversion should be synchronized unless the T wave is large and could be misread as the R wave by the cardioverter. If initial attempts of electrical cardioversion are unsuccessful, the energy level should be doubled, and doubled again if necessary, until a perfusing rhythm is restored. Immediately following conversion of VT, antidysrhythmic medication should be given to prevent recurrence.

Patients with pulseless VT should be initially shocked with 200 J, followed by 300 J if the first shock is not successful. Reentrant SVTs generally respond to

low-energy levels. Atrial flutter, for example, usually requires <50 J for conversion. [26] Cardioversion of atrial flutter in the ED is indicated when the ventricular rate is not slowing in response to pharmacologically enhanced AV-node blockade, or if the patient is unable to tolerate the aberrant rhythm.

The majority of patients with paroxysmal atrial tachycardia (PAT) respond to adenosine. If they do not, or if urgent conversion is needed due to high ventricular rate, electric countershock should be administered in the synchronized mode at 50 J, and doubled if necessary.

In atrial fibrillation, the response to cardioversion is dependent on the duration of atrial fibrillation and the underlying cause. Cardioversion is successful in 90% of cases secondary to hyperthyroidism but in only 25% of cases secondary to severe mitral regurgitation. [195] However, 50% of cases revert within 6 months, especially those with longstanding atrial fibrillation. [196] [197]

Most patients with atrial fibrillation do not require cardioversion in the ED unless their ventricular response is excessively rapid due to a bypass tract, as in Wolff-Parkinson-White syndrome. Atrial fibrillation may also require cardioversion when sequelae of rapid ventricular contraction are present or anticipated and the ventricular rate is not responding to drug therapy aimed at slowing AV node conduction. Conversion of atrial fibrillation generally requires more energy than the reentrant SVTs (about 100 J in most cases). [198]

Pediatric Cardioversion

Pediatric cardioversion is similar to adult cardioversion. As previously described, the purpose of the procedure is to depolarize the myocytes completely at the most opportune time, during the peak of the R wave, so as not to precipitate VF, and allow a slower perfusing rhythm to resume. However, the energy levels for pediatric cardioversion are different from the adult. In the pediatric procedure the initial recommended energy dose is 0.5 to 1 J/kg, while the defibrillator is in the *synchronized mode*. If needed, a repeated cardioversion may be attempted at 2 J/kg, again while the defibrillator is in the *synchronized mode!* Remember to re-synchronize the defibrillator after each cardioversion attempt and look for the appropriate markers on the monitor to ensure that the current is delivered at the appropriate phase of the cardiac cycle (see Fig. 12-3).

COMPLICATIONS OF CARDIOVERSION

Complications of cardioversion may affect the patient, particularly the patient with a cardiac pacemaker, as well as health care personnel at the bedside. Patient complications are dose-related and may involve the airway, heart, or chest wall, or they may be psychological.

Injuries to health care personnel with cardioversion/defibrillation include mild shock and burns. There is a reported incidence of 1 injury per 1700 shocks for paramedics in the field.[138] Of the 13 injuries reported in this study, 15% were due to equipment failures, 23% were due to injuries during testing or demonstrations, and 23% were due to arcing of the electric shock from the paddle to an electrode on the patient's chest, whereas the remainder were due to direct contact with the patient or with the stretcher.

Hypoxia may result if sedation is excessive or if the airway becomes compromised. With proper preparations and precautions, airway complications can be minimized. Respirations may also be depressed by any of the anesthetic agents, and adequacy of tidal volume must be continually assessed by either direct observation or end-tidal CO_2 monitoring. An assistant should be placed in charge of monitoring the patient's airway cardiac monitor, and pulse oximetry. Routine supplemental O_2 is suggested for all patients undergoing sedation.

Chest wall burns resulting from electrical arcing are generally superficial partial-thickness burns, although deep partial-thickness burns have occurred. [199] These are preventable by adequate application of conductive gel and firm pressure on the paddles. Paddles should not be placed over medication patches or ointments, especially those containing nitroglycerin, because electrical discharge may cause ignition resulting in chest burns. [200]

Cardiac complications following cardioversion are proportionate to the energy dose delivered. In the moderate energy levels used most commonly, the hemodynamic effects are small. At higher energy levels, however, complications include dysrhythmias, hypotension, and, rarely, pulmonary edema, which may occur several hours after the countershock.[201] A transient failure of myocardial O_2 extraction due to a direct effect on cellular mitochondria has been proposed as an explanation for some of these cardiac complications. [202]

The dysrhythmias following high-dose (approximately 200 J) direct-current shocks include VT and VF, bradycardia, and AV block, in addition to transient and sustained asystole. Sustained VT or VF was reported following 7 of 99 shocks in a study of patients undergoing electrophysiologic study and requiring cardioversion for VT, VF, or atrial fibrillation. [203] These episodes occurred only in the patients with prior VT or VF. Patients with ischemia or known coronary artery disease appear to be at much higher risk for significant postshock bradycardia, with rate support pacing required after 13 of 99 shocks in the study. Asystole requiring pacing occurred only once in 99 countershocks. Therefore, the proclivity for dysrhythmias is greater in high-dose cardioversion of an ischemic heart.

Two types of VF following cardioversion have been described. The first variety occurs immediately after countershock and is easily reversed by a second, nonsynchronized shock. This type of VF results from improper synchronization, with discharge of current occurring during the vulnerable period. The second variety, which is more ominous, occurs approximately 30 seconds to a few minutes following attempted cardioversion. This dysrhythmia is characteristically preceded by the development of PAT with block or a junctional rhythm. In affected patients, it may be very difficult to convert the dysrhythmia to a sinus rhythm. This phenomenon occurs in patients who have been taking digitalis glycosides and is presumably a manifestation of digitalis toxicity. [204] [205]

In the event of VF following cardioversion, the equipment and manpower should be present for immediate defibrillation.

If postcardioversion VF occurs, switch the cardioverter to "nonsynchronized" before attempting defibrillation. Electrical discharge will not occur in the "synchronized" mode, because the machine will be searching for a nonexistent R wave.

VF is much more likely to occur if depolarization occurs on the T wave. If a patient has large T waves in the lead selected for cardioverter sensing, the electric shock may discharge during the vulnerable period of the cardiac cycle, resulting in VF. [206] Always examine the complexes on the cardioverter monitor carefully for large T waves and, if necessary, change the sensing lead. A randomly firing pacemaker can also be sensed by the cardioverter, resulting in countershock during the vulnerable period. [207]

Transient and intermittent ST-segment elevation has also been reported to occur (though rarely) after cardioversion, with myocardial injury or coronary vasospasm offered as possible explanations. [208]

An increase in serum enzyme levels (creatine kinase, lactate dehydrogenase, aspartate aminotransferase) may also occur following cardioversion, and the incidence has been reported to be between 10% and 70%. The enzyme rise is usually a consequence of skeletal muscle injury rather than myocardial damage. Cardioversion does not alter the enzyme profile of patients with myocardial infarction. [209]

CONCLUSIONS

Cardioversion is a safe and effective method of quickly terminating reentrant tachycardia. Complications related to psychological trauma, respiratory depression, and unintentional health-worker shock can be avoided with proper precautions. Adequate sedation is essential. Synchronized shock should be administered after close scrutiny of the lead used for sensing, to be sure that the R or S wave is significantly larger than the T wave. Be prepared for postshock VT or VF, and if VF occurs, switch the cardioverter to "nonsynchronized" and defibrillate. Atropine and temporary pacing equipment should be available to treat postshock bradycardia, especially in patients with myocardial ischemia or infarction.

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Chapter 13 - Assessment of Implanted Pacemaker/AICD Devices*

David W. Munter

Patients with permanent implanted pacemakers or automatic implantable cardioverter-defibrillators (AICDs) are commonly seen in the emergency department (ED). The frequency with which these patients present to the ED can be attributed to an aging population and increased usage of pacemakers and AICDs due to their technical sophistication and reliability. Fortunately the increased reliability of the devices has prevented a marked increase in patients presenting with true emergencies related to device malfunction. Complications, however, are not uncommon, with 7.4% to 15% of pacemakers failing in the first year. [1] AICD complication rates, including inadvertent shocks, occur in up to 34% of patients with the device. [2] Although the basic evaluation and treatment of patients with pacemakers or AICDs presenting with cardiac complaints is not substantially different from that of patients without the devices, a general knowledge of the range of potential problems and complications and techniques for evaluating or inactivating pacemakers or AICDs is desirable for emergency clinicians. However, these devices are complicated and most clinicians do not possess the expertise to fully evaluate them. Appropriate consultation, depending on the clinical situation, may be necessary.

BACKGROUND

The relationship between applied electricity and ventricular rhythms and arrhythmias has been understood since the late 1800s. [3] With the onset of cardiac monitoring techniques in the 1940s, the relationships between sudden death, cardiac syncope, ventricular fibrillation, and bradycardias began to be understood. [4]

The first pacemakers were developed by Zoll in the 1950s. [4] The units were transcutaneous and used primarily for Stokes-Adams disease (syncope secondary to bradycardia or asystole). These early units could only be used transiently and only in a hospital setting, as they created severe patient discomfort. [4] In 1958, the first long-term implanted pacemaker was used.[5] The indications for pacemaker use were rapidly expanded, as was the technology of the devices.

The first human defibrillation was performed intraoperatively in 1947 and the first transthoracic defibrillation in 1956. [6] Rapid defibrillation is now the standard of care for ventricular fibrillation, and its use has significantly increased survival of cardiac arrest. However, patients who survive cardiac arrest have a high risk of recurrent arrest, estimated at 22% to 60% in the first year.[7] [8]

AICDs were developed by Mirowski in the late 1960s, but they were not accepted by the medical community until the late 1970s. [3] In 1980, the first AICD was implanted in a human, followed by implantation in a series of patients [9] in whom an average 52% decrease in mortality was seen.[10] Between 1985 and 1990, more than 10,000 patients received AICDs, and the number continues to increase. [3]

PACEMAKER CHARACTERISTICS

Several hundred types of pacemakers are in use today.[1] The typical generator is a hermetically sealed device generall weighing <30 gm. Modern power sources are almost exclusively lithium-based batteries,[12] which have the advantage of a slow, steady, predictable decay rate. Half-lives of lithium batteries range from 75 to 144 months.[12] The generator is connected to sensing and pacing electrodes that are placed in various locations in the heart depending on the configuration of the pacemaker. Newer models are programmable for rate, output, sensitivity, refractory period, and modes of response, [11] and they can be reprogrammed radiotelemetrically after implantation.

Pacemakers are classified according to a standard five-letter code developed by the North American Society of Pacing and Electrophysiology/British Pacing and Electrophysiology Group (Table 13-1). The first letter designates the chamber that actually receives the pacing current, whereas the second letter denotes which chamber the pacemaker uses to sense intrinsic cardiac electrical activity. The third letter denotes the pacemaker's response to sensed intrinsic electrical activity (usually inhibited). The fourth letter refers to the pacemaker's rate modulation and programmability, whereas the fifth letter describes antitachycardiac features of the pacemaker. In normal practice, only the first three letters are used to describe the pacemaker (e.g., VVI or DDD). [13]

Pacemaker wires are embedded in plastic catheters. The terminal electrodes, which may be unipolar or bipolar, travel from the generator unit to the heart via the venous system. The typical entry point is the subclavian or cephalic vein. The terminal electrodes are placed in either the right ventricle or both the right ventricle and atrium, under fluoroscopic guidance. Proper lead placement is checked by electrograms checking sensing and pacing thresholds. [14] The typical radiographic appearance of an implanted pacemaker is seen in Figure 13-1 .

The pacemaker rate is typically programmed between 60 and 80 beats/min. A significantly different rate usually indicates malfunction. When the battery is low, the rate usually begins to drop, getting lower as the battery fades. The pulse generator output is the product of volts, milliamperes, and the stimulus duration, all of which may be preset or programmed. Sensing of intracardiac electrical activity is a combination of recognizing the characteristic waveforms of P waves or QRS complexes while also discriminating these from T waves or external interfering signals, such as muscle activity or movement. The pacing electrical stimulus is a triphasic wave consisting of an intrinsic deflection, far-field potential, and an injury current, which typically delivers a current of 0.1 to 20.0 mA for 2 msec at 15 V. [15]

Pacemakers have a reed switch, which may be closed by placing a magnet externally on the chest wall over the generator; this inactivates the sensing mechanism of the pacemaker, which then reverts to an asynchronous rate termed the *magnet rate*. Essentially the magnet turns the pacemaker into a fixed rate device. The magnet rate is usually, but not always, the same as the programmed rate.

* The views expressed in this article are those of the author and do not reflect the official policy or position of the Department of the Navy, the Department of Defense, or the U.S. Government.

TABLE 13-1 -- North American Society of Pacing and Electrophysiology/British Pacing and Electrophysiology Group Generic Pacemaker Code (NBE Code)

I	II	III	IV	V
Chamber Paced	**Chamber Sensed**	**Response to Sensing**	**Rate Modulation and Programmability**	**Antitachycardia Features**
0—None	0—None	0—None	0—None	0—None
A—Atrium	A—Atrium	I—Inhibited	I—Inhibited	P—Antitachycardiac pacing
V—Ventricle	V—Ventricle	T—Triggered	M—Multiple	
D—Dual	D—Dual	D—Dual	C—Communicating	S—Shock
			R—Rate modulation	D—Dual

AICD CHARACTERISTICS

The AICD consists of a pulse generator with sensing mechanism, battery, and attached lead system with sensing and shocking abilities. The pulse generator and battery are contained in a hermetically sealed titanium case weighing 250 to 300 g. This case is approximately 10.8 × 2 × 7.6 cm, with variations depending on the manufacturer. The battery is typically a lithium battery with a life span of approximately 100 shocks, [16] [17] although a maximum life span of 22 months was noted in one study.[7]

A variety of sensing and shocking leads are in use. Most current AICDs use two bipolar sensing electrodes that are secured in the epicardium, with two ventricular patches for shock delivery usually placed anteriorly and posteriorly over the left ventricle (Fig. 13-2 and Fig. 13-3).

A new AICD with a single transvenous lead containing both sensing and shocking components is also in use (Fig. 13-4). This model offers the advantage of obviating the need for cardiac surgery.

The electrical components of the AICD continuously monitor both rate and waveform [18] through separate sensing channels. Different models place different emphases on these channels depending on the patient's underlying disease. The rate-detection channel determines if a preset limit, individualized for each patient, is exceeded. The second channel analyzes the morphology of the waveform of intrinsic cardiac electrical activity. Essentially, variance from a baseline is measured to determine if a shockable rhythm is present. Normal sinus rhythm has periods of isoelectric activity (the baseline), but ventricular fibrillation or sinusoidal tachydysrhythmias typically lack these isoelectric segments.

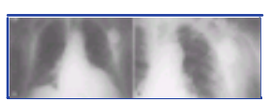

Figure 13-1 Radiographs of an implanted pacemaker showing battery and lead wires. *A*, standard view. *B*, Close-up view.

A malignant ventricular tachydysrhythmia is usually present for 5 to 15 seconds before the preset rate and morphologic criteria are met. [16] At this time, the AICD charges its capacitor and delivers a shock of 25 J. This shock is synchronized in the case of ventricular tachycardia and unsynchronized in ventricular fibrillation. The time from the initiation of the arrhythmia to delivery of the shock is from 10 to 35 seconds. The patient will clearly sense the discharge.

If arrhythmia persists after the first shock, subsequent shocks of 30 J are delivered, with each subsequent detection and shocking period lasting from 10 to 35 seconds. A total of four shocks are delivered if the rhythm is unchanged, after which the AICD does not deliver more shocks unless a subsequent change occurs in the rhythm that lasts >~35 seconds.[2]

The AICD case is implanted in a subcutaneous pocket in the abdominal wall (Fig. 13-5 and Fig. 13-6). The wires are run through a subcutaneous tunnel to the chest. A variety of surgical approaches to the heart are used. [19] The ventricular patches are sutured to the epicardium or parietal pericardium in roughly anterior and posterior positions on the left ventricle, depending on the surgical approach. The sensing electrodes may be placed in either the left or right ventricle.

In those patients in whom the new transvenous lead is used, thoracotomy is not needed—the AICD case is still in the typical abdominal position, and a submuscular patch is placed in the chest wall.

AICDs may be inactivated by a magnet, either purposely or inadvertently. They may also be interrogated via a radiotelemetry device (a procedure normally performed by a cardiologist).

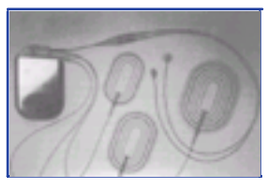

Figure 13-2 Automatic implantable cardioverter-defibrillator (AICD). *(Courtesy of Medtronics Inc., Minneapolis, MN.)*

INDICATIONS FOR IMPLANTABLE DEVICE USE

Current indications for permanent pacemaker use include complete heart block, symptomatic type II second-degree block, second-degree block with episodic ventricular arrhythmias, sick sinus syndrome, symptomatic bradycardias with syncope or presyncope, hypersensitive carotid artery syndrome, type I block with infra-His bundle block, and certain subgroups of patients with triphasic and biphasic blocks at risk of developing sudden high-degree block. [14] [20]

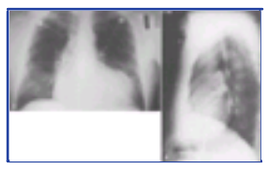

Figure 13-3 Posteroanterior *(A)* and lateral *(B)* chest radiographs demonstrating typical appearance of AICD with ventricular patches.

The use of AICDs is generally limited to patients who are at high risk of sudden cardiac death from ventricular arrhythmias. Currently accepted indications are a documented episode of hemodynamically significant, sustained ventricular tachycardia or fibrillation; ventricular arrhythmia refractory to standard antiarrhythmic therapy as demonstrated electrophysiologically; persistent electrophysiologically inducible ventricular arrhythmia despite best available drug therapy; and recurrent syncope in a patient with electrophysiologically inducible ventricular arrhythmia in whom no effective drug is tolerated or available. [3] [16] [21]

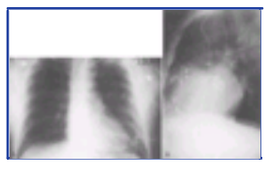

Figure 13-4 Posteroanterior *(A)* and lateral *(B)* chest radiographs demonstrating typical appearance of single-lead transvenous AICD.

Contraindications to AICD use are life expectancy of less than 6 months, New York Heart Association class IV heart failure, treatable causes of ventricular arrhythmias, or incessant or very frequent ventricular arrhythmias that result in rapid battery depletion.

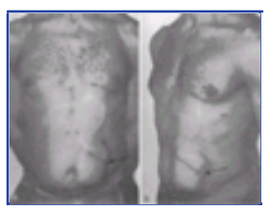

Figure 13-5 Typical external appearance of AICD implanted in the abdominal wall. *(Courtesy of Lawrence B. Stack, MD. From Munter DW, DeLacey WA: Automatic implantable cardioverter-defibrillators. Emerg Med Clin North Am 12:579, 1994. Used with permission.)*

COMPLICATIONS OF PERMANENT PACEMAKERS

Complications are commonly seen with permanent pacemakers. Failure rates in the first year postimplantation range from 7.4%

Figure 13-6 Radiographic appearance of the implanted AICD in the abdominal wall.

to 15.0%, most occurring within the first 30 days.[1] After this initial period, approximately 6% of pacemakers fail each year. [22]

Pacemaker failure can be categorized as failure to pace, failure to sense, failure to capture, inappropriate pacemaker rate, and other complications (e.g., vascular or infectious) (Table 13-2). Failure to sense is the most common problem, accounting for 32% to 57% of failure cases. [3] [22] [23]

Failure to Pace

This condition is characterized by the lack of production of pacemaker spikes despite the lack of intrinsic cardiac electrical activity or an intrinsic cardiac rate falling below the threshold for pacing. Causes of failure to pace include lead fracture or disconnection, battery depletion, component failure, and oversensing.

Lead fracture or disconnection may occur months to years after pacemaker implantation and may be due to inherent stress at the lead connection site. Blunt trauma may also cause lead fracture.[24] [25] [26]

Battery depletion is normally a *gradual process* and is usually detected on routine checkups before complete failure of the pacemaker. [23] Patients who have not had regular follow-up may present with previously undetected battery depletion.

TABLE 13-2 -- Complications of Permanent Pacemaker Use

Failure to pace (no pacemaker activity present)
Lead fracture
Lead disconnection
Battery depletion
Component failure
Oversensing
External interference
Failure to sense (constant pacemaker spikes despite ongoing intrinsic cardiac electrical activity)
Lead dislodgement
Lead fracture
Fibrosis around lead tip
Battery depletion
Pacer in asynchronous mode
External interference
Low-amplitude intracardiac signal
Failure to capture (pacemaker spikes but no subsequent cardiac activity)
Lead dislodgement including perforation
Lead fracture
Lead disconnection
Poor lead position
Fibrosis around lead tip
Battery depletion
Metabolic abnormalities
Medications
Inappropriate pacemaker rate (runaway pacemaker)
Pacemaker reentrant tachycardia
Resetting from external interference
Battery depletion
Other
Infections: pocket, wires
Lead displacement: cardiac perforation, tamponade, pericarditis, vascular perforation
Vascular complications: thrombosis, superior vena cava syndrome
Psychiatric: anxiety, panic attacks

Component failure may be due to various external influences including blunt trauma, therapeutic radiation, electrocautery, transthoracic defibrillation, diathermy, electroshock therapy for depression, magnetic resonance imaging (MRI), and extracorporeal shock-wave lithotripsy. [13] [23] [27] [28] [29] [30]

Pacemakers may oversense or misinterpret nonQRS complex electrical activity (e.g., P waves, T waves, muscular activity, or chest thumping). [23] If this activity is interpreted as a QRS complex, the ventricular spike will be inhibited. If, however, the pacemaker is dual chambered and the electrical activity is misinterpreted as a P wave, it will stimulate firing of the ventricular electrode, which can lead to overpacing. External stimuli can also be misinterpreted and lead to oversensing. This interference can include electrocautery, MRI, diathermy, transcutaneous electrical nerve stimulation (TENS), electroshock therapy, ultrasound dental scalers, static electricity, or vibration (e.g., from a tractor or helicopter). [27] [31] [32] [33] [34] [35]

Failure to Sense

This condition is characterized by the presence of constant pacemaker spikes despite ongoing intrinsic cardiac electrical activity that should inhibit the device. Failure of the pacemaker to sense cardiac electrical activity can be due to lead

dislodgement, lead fracture, normal development of fibrosis around the lead tip that occurs with time, battery depletion, external interference, or an intrinsically low-amplitude cardiac signal.

Lead dislodgement, the most common reason for failure to sense, can be due to an enlarged right ventricle, poor initial lead positioning, blunt trauma, or patient manipulation of the generator unit in the chest wall pocket (pacemaker twiddler's syndrome). [23] [36] [37] [38]

Fibrosis normally develops around the tip of the electrode, and this fibrosis can lead to abnormal sensing or higher required thresholds for pacing. [36]

Several cardiac and metabolic abnormalities cause the intrinsic cardiac electrical activity to be of lower than normal amplitude, causing undersensing of the QRS complexes.

Failure to Capture

This condition is the appropriate presence and timing of pacemaker spikes, but without resultant cardiac activity. Reasons for failure to capture include lead dislodgement (e.g., myocardial perforation), lead fracture, lead disconnection, poor lead position, lead tip fibrosis, battery depletion, and metabolic abnormalities (e.g., hyperkalemia) or medications (e.g., lidocaine, flecainide) that make the myocardium less responsive to electrical impulses. Lead dislodgement is the most common reason for failure to capture. [23]

Inappropriate Pacemaker Rate

This condition, also known as a *runaway pacemaker*, is a rare complication that is usually seen in dual-chamber pacemakers. [39] [40] In dual-chamber pacers, it is caused by an endless loop reentry tachycardia, often initiated by a retrograde P wave. [40] In older or single-chamber pacemakers, it can be due to component failure or battery depletion. [41] Component failure is rare, as almost all pacemakers have circuitry to prevent high discharge rates.

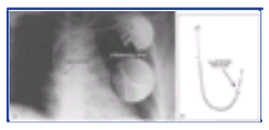

Figure 13-7 *A* and *B*, Appearance of fracture of atrial "J" retention wire. *A*, Radiographic appearance of fracture of atrial "J" retention wire. *B*, Diagram of fracture site. *(Courtesy of Telectronics Pacing Systems, Englewood, CO.)*

Other Complications

Infections including localized skin or pocket infections, more complicated infections along the route of the wires, or endocarditis occur in 1% to 15% of patients. [42] [43] [44] These infections are initially treated with broad-spectrum antibiotics against *Staphylococcus aureus*, but they often require removal of the pacemaker. [13] [44]

Lead displacement, in addition to causing failure to sense or capture, can cause injury to the myocardium including cardiac perforation with resultant tamponade or restrictive pericarditis, [36] [45] [46] and it is usually seen early after pacemaker implantation.

Fracture of the J-shaped retention wire within the lead can result in a protrusion of the wire outside the protective plastic coating. This protruding wire can then puncture the superior vena cava or right atrium, resulting in bleeding or cardiac tamponade. [47] Figure 13-7 illustrates this fracture.

Vascular complications include thrombosis or superior vena cava syndrome. Many pacemaker patients have a benign thrombosis of the upper arm or shoulder, but only about 2% have a serious thrombotic or embolic event. [44] Superior vena cava syndrome is a rare complication. [48]

Psychiatric complications include panic attacks and anxiety, which may prompt ED evaluation. [13] [44]

COMPLICATIONS OF AICDs

The most common complication is the delivery of inappropriate shocks by the device when not indicated by a tachydysrhythmia. Up to 35% of AICD patients receive inappropriate shocks.[49] Causes of inappropriate shock delivery include misinterpretation of sinus tachycardia, atrial fibrillation, muscular activity (e.g., shivering), T waves, or extraneous sources (e.g., pacemaker spikes or vibrations) as a shockable tachydysrhythmia. [50] [51] Likewise an unsustained tachydysrhythmia also may be shocked. Component failure, such

as electrode failure or migration, may result in false sensing and resultant shocking. Pacemaker magnet testing has produced inappropriate shocks resulting in ventricular fibrillation. [52]

Other reported long-term complications include interference with cardiac pacemakers, particularly after AICD discharge [53] [54] ; component failures such as patch migration or distortion,[55] lead fracture, generator case rotation or fracture, and battery depletion; constrictive pericarditis and pericardial effusions, [56] [57] [58] cardiac fibrosis, and atrial or ventricular wall perforations after repeated shocks [58] [59] ; abdominal pocket infections[60] ; thrombosis and pulmonary embolism; erosion into the lung with hemoptysis[61] ; patient trauma sustained during falls after delivery of a shock, either due to the physical "jolt" of the shock [62] or to suspected postshock bradycardia and hypotension[63] [64] ; and psychiatric disorders, such as adjustment disorder, panic attacks, or major depression. [65] [66]

The AICD may be inadvertently inactivated by any strong magnetic force including microwave ovens, industrial engines, metal detectors, magnets in speakers, refrigerator door magnets, bingo wands, and model airplane starters. [2] This inactivation is not normally noted until routine follow-up, but if the patient has a tachydysrhythmia, no shock will be delivered.

EVALUATION OF PACEMAKER PATIENTS

Pacemaker patients presenting to the ED for any problem that may be associated with the device should have an evaluation that includes investigation of potential pacemaker malfunction.

Historical Issues

Pertinent information about the pacemaker unit should be obtained. The brand and manufacturer, type (NBE code),* implantation site, programmed rate, and any changes seen during follow-up at a pacemaker clinic should be noted. The time of implantation is important as certain types of complications such as lead failure, migration, or perforation typically occur within 3 months, whereas generator or battery failure usually occur later. [36]

Some medications, such as lidocaine or flecainide, can raise myocardial thresholds to pacing, [23] and their use should be evaluated.

Patient symptoms that may be related to pacemaker failure should be ascertained. Chest pain may be due to cardiac ischemia, but pacemaker complications such as cardiac perforation, pericarditis, or infection may also be the cause.

Recent trauma, especially to the chest or back, can cause failure of the generator unit, lead fracture, or lead displacement. Recent cardioversion or defibrillation, MRI, diathermy, lithotripsy, or electroconvulsive therapy for depression can have the same effect. [24] [25] [26] [27] [28]

Symptoms of decreased cerebral perfusion, such as syncope, near-syncope, or orthostatic light-headedness, may indicate pacemaker malfunctioning that could lead to bradycardias. A runaway pacemaker can also cause these symptoms due to low cardiac output during the tachycardic phase.

Palpitations are more typically due to intrinsic cardiac activity, but pacemaker failure to sense can cause an irregular rhythm due to inappropriate generation of pacer spikes, which may be perceived by the patient.

The time and results of the last follow-up visit should be obtained if possible.

Physical Examination

The patient should be examined for potential pacemaker complications. The head and neck should be inspected for venous engorgement, which may indicate thrombosis or superior vena cava syndrome. [36]

The chest wall, and especially the pacemaker pocket, should be inspected and palpated for erythema, edema, tenderness, and location of the pacemaker generator. The initial course of the lead wires should also be palpated. If the generator unit is malrotated or in a position other than the original pacemaker pocket, this may indicate "pacemaker twiddler's syndrome" (i.e., purposeful or inadvertent manipulation by the patient), [37] [38] which can lead to lead dislodgement or disconnection.

The cardiac examination may be altered by the functioning of the pacemaker. A paradoxically split second heart sound is normal and is due to the origination of the pacemaker spike in the right ventricle. If the second heart sound is widely split, and widens more with inspiration, a left ventricular origin may be present due to cardiac perforation or lead migration. [36] A pericardial friction rub may be due to pericarditis, potentially from lead perforation. [45] A new murmur may be the result of infective endocarditis. [36]

X-Ray

A chest radiograph should be obtained and compared to old films, preferably ones taken shortly after implantation. Location of the generator and battery type should be ascertained. Lithium batteries are seen as a single radiopaque portion of the generator unit, whereas older pacemakers have four to five button batteries. Lead locations should be noted and migration should be sought. Lead presence outside the myocardial shadow is indicative of perforation. The connection site at the generator unit should be examined for disconnection. The lead wires should be traced and examined for fracture, or J retention wire fractures (see Fig. 13-7).

Electrocardiogram

The electrocardiogram (ECG) contains important information about pacemaker function and should be compared to old ECGs. An asynchronous mode pacemaker (i.e., VOO) will have regular pacer spikes followed by QRS activity. A left bundle-branch pattern is normal, as the lead is usually in the right ventricle. Absence of pacer spikes in the asynchronous mode is termed *failure to pace*. Absence of a QRS complex after a pacer spike represents failure to capture. Some pacemakers have a programmed feature called hysteresis, in which the intrinsic rate below which pacing is triggered is somewhat lower than the resultant pacing rate. Hence the pause following a spontaneous QRS complex may be longer than an R-R interval at normal pace.

Pacemakers that sense intrinsic cardiac activity and are inhibited by same (i.e., VVI) should only generate pacer spikes if the patient's intrinsic rate falls below the programmed rate. In this case, pacer spikes followed by QRS complexes, again in a left bundle-branch pattern, should be noted. Failure to pace or capture may be seen. Pacer spikes occurring despite an intrinsic rate above the programmed rate represent failure to sense.

Electrocardiograms from patients with dual chamber pacemakers (i.e., DDD) may demonstrate various rhythms. [36] These include the patient's intrinsic rhythm, atrial pacer spikes followed by a P wave and then an intrinsic QRS, or two pacer spikes sequentially followed by a P wave and then a QRS complex. Failure to pace, capture, or sense may again be noted.

The electrical axis of the pacer spike and paced QRS complex should be compared to old ECGs. A change of the pacer spike axis may signal lead migration. [36] Normally, the paced QRS complex is a left bundle-branch pattern. A right bundle-branch pattern may be due to left ventricular lead placement, but a change from old ECGs from a left to a right bundle-branch pattern may be due to migration of the lead or perforation of the ventricular septum.

Paced rates that are higher than the programmed rate can be due to pacemaker-mediated tachycardia, a reentrant tachycardia normally seen only in dual-chambered pacemakers, [39] [40] or runaway pacemaker, normally seen only in older pacemaker models and due to battery depletion or component failure. [41] External electrical sources can mimic pacemaker spikes and be misinterpreted as an inappropriate pacing rate. [36]

General Management

Patients who are found to have failure to sense or capture should undergo the basic evaluation outlined earlier; in addition, serum electrolyte assays should be performed to assess metabolic abnormalities. These patients and patients with infections, lead displacement, disconnection or fracture, or vascular complications will

*North American Society of Pacing and Electrophysiology-British Pacing and Electrophysiology Group generic pacemaker code (NBE code).

require cardiology consultation for intervention as determined by the etiology of the malfunction.

Patients who have failure to pace may undergo a magnet test in the ED to rule out battery depletion or oversensing.

Patients with pacemaker-mediated tachycardia or runaway pacemaker need prompt cardiology evaluation, and those with unstable conditions require acute intervention in the ED, as outlined in the next section.

Use of a Magnet in Pacemaker Assessment

Indications

Magnet testing of a pacemaker is used on patients with "failure to pace" to assess for battery depletion, component failure, or oversensing. It is also indicated for pacemaker-mediated tachycardia or runaway pacemaker in an attempt to terminate the rhythm.

Equipment and Setup

Typically, a ring magnet (Fig. 13-8) is used for this procedure. Different brands of magnets are also available for specific pacemakers, but in almost all cases, a standard pacemaker ring magnet will suffice. The patient should undergo a baseline ECG and should be on a cardiac monitor. Placement of a magnet on a pacemaker closes a reed switch and reverts the pacemaker to an asynchronous, or fixed rate, mode.

Technique

The location and orientation of the pacemaker generator should be ascertained by palpation. It is typically in the left or right upper chest wall (Fig. 13-9). The magnet is placed

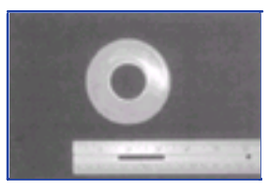

Figure 13-8 Ring magnet.

directly on the chest wall over the pacemaker generator (Fig. 13-10). A repeat ECG is obtained to compare to the baseline (Fig. 13-11 and Fig. 13-12). Some brands of pacemakers have either a several-second delay or a series of two to three rapid pacer spikes before reverting to the asynchronous mode. [36] If the patient has an underlying bradycardia associated with failure to pace that is corrected by magnet placement, the magnet should be left over the generator. Likewise, if a pacemaker-mediated tachycardia or runaway pacemaker is reverted to a normal rate or rhythm, the magnet should be left in place pending consultation with a cardiologist.

Complications

Incorrect alignment of the magnet may cause only intermittent pacing in the asynchronous mode. Some pacemakers react erratically to a non-brand-specific magnet; if this is considered an issue, attempt to obtain a brand-specific magnet.

Interpretation

In the case of failure to pace, three outcomes are possible: (1) The pacemaker will fire at its programmed rate (this is the expected outcome). In the case of failure to pace, this indicates that the pacemaker was oversensing and was inappropriately

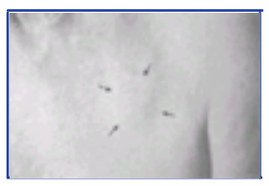

Figure 13-9 Typical location of pacemaker pocket in upper chest wall.

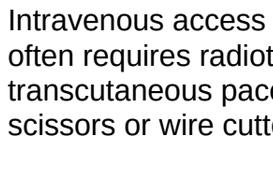

Figure 13-10 Application of ring magnet over pacemaker generator.

inhibited. The cause of the oversensing should then be investigated. (2) The pacer may not produce any spikes at all. This is indicative of component failure and requires cardiology consultation. (3) The pacemaker may produce spikes, but at a rate lower than the programmed rate, which is indicative of battery depletion; this also requires cardiology consultation.

Management of Pacemaker-Mediated Tachycardia or Runaway Pacemaker

Indications

These maneuvers are indicated for correction of an inappropriately high pacemaker rate.

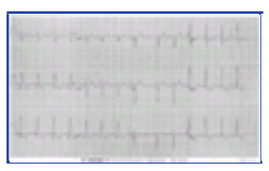

Figure 13-11 Electrocardiogram of patient with a nonfiring pacemaker. Intrinsic cardiac rate is 80 beats/min, and no pacemaker activity is seen.

Equipment and Setup

Intravenous access should be obtained and the patient should be placed on a cardiac monitor. A baseline ECG should be obtained. Because the definitive treatment often requires radiotelemetric reprogramming of the pacemaker, a cardiology consultation should be obtained. A ring magnet will be needed as well as a transcutaneous pacemaker unit with pads. If open lead disconnection is required, local anesthetic, sterile drapes, scalpel blade and handle, hemostats, and Mayo scissors or wire cutters will be needed, as well as either a transcutaneous pacemaker or portable pacemaker unit with alligator clip.

Technique

The initial procedure is to place a ring magnet over the pacer generator as described earlier. If the pacer then reverts to an asynchronous mode at the appropriate programmed rate, the magnet should be left in place.

If magnet placement has no effect, then isometric pectoral exercises should be attempted.[13] [39] This is done by having the patient place his or her hand from the same side as the pacemaker generator onto the opposite shoulder and push against the shoulder as long and as hard as possible. This creates rapid muscle activity in the pectoral muscle surrounding the pacemaker generator, which may be interpreted as ventricular activity and inhibit the pacemaker, [39] [40] terminating the reentrant tachycardia.

If this maneuver is unsuccessful and the patient's condition is stable, continue to monitor the patient and await cardiology consultation. If the patient's condition is unstable, the next maneuver is to attempt a standard chest *(precordial)* thump.[13] [39] This is performed by firmly striking the midsternum with a clenched fist from a distance of 30 to 38 cm. This procedure may be repeated once if needed. [39]

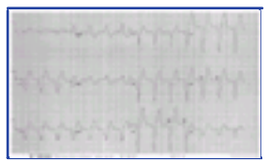

Figure 13-12 Electrocardiogram of same patient with magnet applied over pacemaker, producing a paced rhythm. Pacer spikes are evident (*arrows*) and the magnet rate is 85 beats/min. Note the left bundle-branch bundle typical of a pacer lead in the right ventricle.

If the chest thump is unsuccessful, attach transcutaneous pacemaker pads anteriorly and posteriorly, and attach the leads to the transcutaneous pacemaker generator. Pace the patient at an initial output of 2 to 5 mA and a rate of 40 beats/min. [39] This will stimulate chest wall movement that may be interpreted as ventricular activity and inhibit the pacemaker, terminating the reentrant tachycardia. Unipolar DDD pacemakers are normally inhibited at a low output. Bipolar DDD pacemakers often need higher outputs of up to 10 to 20 mA.[39] If increasing the output is unsuccessful, raise the transcutaneous pacing rate slightly to attempt to deliver chest wall stimulation outside the pacemaker's particular ventricular refractory period. [39]

If these maneuvers are unsuccessful and the patient is hemodynamically unstable due to the pacer-induced tachycardia, the solution of last resort is to cut the pacer leads from the generator unit. The generator unit should be palpated to ascertain position and orientation. Apply transcutaneous pacemaker pads and connect them to the transcutaneous pacemaker. This is a precautionary measure, as cutting the leads may lead to profound bradycardia or asystole. A portable pacemaker generator should be available. Use povidoneiodine for sterile preparation of the site over the generator and apply sterile drapes. Instill local anesthesia in the skin and subcutaneous tissue overlying the generator in the area of the lead connections. Make a skin incision with a scalpel, and expose the lead wires with blunt dissection. Alternatively, make an incision through the previous scar and remove the pacemaker unit. [41] Cut the lead wires close to the generator using Mayo scissors or wire cutters. At this point, the patient may require transcutaneous pacing. An alternate procedure is to insert a needle into the cathode (negative) lead wire (this is the only wire on unipolar models, and it is identified on bipolar units either by marking or by a white band). [41] Connect an alligator clip to the needle, and connect the alligator clip to the negative terminal of the portable pacemaker generator. Ground the positive terminal via an alligator clip to the subcutaneous tissue of the incision, and pace the patient using the portable generator. [41]

Complications

Incorrect alignment of the magnet may cause pacing only intermittently in the asynchronous mode. Some pacemakers will react erratically to a non-brand-specific magnet; if this is a problem, attempt to obtain a brand-specific magnet. There are no potential complications from isometric pectoral muscle exercise. Chest thumps can result in sternal or rib fractures, myocardial contusion, pulmonary contusion, extrasystolic complexes, or ventricular tachydysrhythmias. [39] Transcutaneous pacing to stimulate chest wall movement is normally uncomfortable for the patient and can also result in diaphragmatic or arm muscle stimulation as well. Open disconnection of the pacemaker lead wires can be complicated by unanticipated bleeding, difficulty exposing the generator unit, and subsequent infection. A more common complication is the termination of the paced rhythm, resulting in a profound bradycardia or asystole that requires ongoing transcutaneous or portable generator pacing.[41]

Interpretation

Correction of the pacer-induced tachycardia by lead disconnection only is generally indicative of component failure or

severe battery depletion, whereas termination by noninvasive procedures is more indicative of a pacemaker-induced endless loop reentrant tachycardia. [39] [41] [67]

EVALUATION OF AICD PATIENTS

Patients with an AICD who present to the ED can have noncardiac complaints unrelated to the AICD, cardiac complaints, or AICD-related problems, including AICD shocks. The evaluation should focus on potential AICD problems.

Historical Issues

Patients with AICDs have severe underlying cardiac disease, and will often present with cardiac chest pain, shortness of breath, or congestive heart failure. Potential AICD complications such as pericarditis, pericardial effusion, cardiac fibrosis, atrial or ventricular perforation, and infections of the wires or leads can all present with chest pain. Mortality in AICD patients is typically due to their underlying disease, and any complaints of chest pain must be pursued aggressively.

Generator pocket infections or wound infections will produce complaints of pain and fever.

The most common AICD-related complaint in patients presenting to the ED is that of one or more AICD shocks. Patients describe the shock as a feeling of being kicked or punched in the chest.[68] [69] The number of shocks received should be ascertained. Associated symptoms of syncope or near-syncope indicate a probable tachydysrhythmia and appropriate shock. Many patients fall down when they experience an AICD shock, and the presence of any fall-related trauma should be queried.

Physical Examination

The abdominal pocket and subcutaneous tunnel should be evaluated for signs of infection. Heart and lung sounds should be auscultated. A pericardial rub is indicative of pericardial fluid, which may be a result of pericarditis from the AICD. The patient should be examined for any signs of trauma if he or she fell.

X-Ray

A chest radiograph is generally not helpful, but one should be obtained and compared to old films to look for electrode fracture, displacement of sensing electrodes, and patch migration or distortion.

Electrocardiograms

An ECG should be obtained. Immediately after an AICD shock, the ECG often shows abnormalities such as ST-segment elevations or depressions.[59] [70] If these changes are due solely to the shock, they will resolve within 15 minutes. Otherwise, the ECG should be examined and compared to old studies for evidence of ischemia.

Use of a Magnet for AICD Inactivation

The patient who is experiencing inappropriate AICD discharges in the ED can be treated by magnet inactivation of the device similar to the approach described earlier for the pacemaker patient.

Technique

The method for inactivating an AICD device is outlined in Table 13-3 . The orientation of the device in the abdominal pocket should be determined, with the lead connections normally cephalad. A ring magnet is then placed over the corner adjacent to the lead connections (usually the upper righthand corner of the device) (Fig. 13-13). A series of beeping tones, which correspond to the sensed QRS complexes, will sound. In the absence of organized QRS activity, random beeps will sound.[68] When the magnet is left in place for 30 seconds, a continuous beep is heard. This indicates that the AICD is inactivated. The magnet should then be removed, and the AICD will remain inactivated. The AICD may be reactivated by applying the magnet for 30 seconds and removing it when the steady beep changes to intermittent beeping.

Clinical Follow-Up

The AICD patient who has component failure, such as patch migration or lead fracture or dislodgement; infection; vascular complications, such as thrombosis or perforation; or cardiac complications, such as perforation or pericarditis, requires cardiology consultation for admission.

The AICD patient who received a single shock and had prodromal symptoms indicative of low cardiac output should be evaluated for myocardial infarction, electrolyte imbalance, or drug toxicity, as well as for any sustained trauma. If findings of this evaluation are normal, the patient is usually released home after discussion with the cardiologist.[2]

The patient who received a single shock without related symptoms consistent with a ventricular tachydysrhythmia requires a more extensive evaluation including cardiac monitoring, determination of drug levels as appropriate, and electrolyte level measurement. [68] [69] [71] If the ED evaluation is normal, patients require cardiology consultation. The cardiologist normally interrogates the AICD using either phonogram or telemetry units. The decision to admit or release the patient with potential alterations of antiarrhythmic drug therapy is made based on the results of this testing.

Patients whose condition is unstable, who report more than one shock in succession or more than two single shocks in a 1-week period, or who have evidence of ischemia, electrolyte imbalance, or drug toxicity often require admission to a monitored setting for further evaluation and continued monitoring. [2]

TABLE 13-3 -- Method for Inactivation of AICD

1. Determine the orientation of the device in the abdominal pocket, radiographically or by palpation.
2. Place a ring magnet over the upper righthand corner of the device.
3. A beeping tone will sound, which corresponds with the sensing of QRS complexes.
4. Leave the magnet in place for at least 30 sec.
5. When the beeping changes to a continuous tone, the device is inactivated.
6. Remove the magnet.

From Munter DW, DeLacey WA: Automatic implantable cardioverter-defibrillators. Emerg Med Clin North Am 12:579, 1994. Used with permission.

Figure 13-13 Placement of magnet on AICD.

THE PACEMAKER/AICD PATIENT IN CARDIAC ARREST

Patients with pacemakers in cardiac arrest may require defibrillation or cardioversion, depending on their presentation. Although most pacemakers have circuitry that protects them from high current flows, various pacemaker-related problems can develop from defibrillation or cardioversion. These include damage to circuitry resulting in complete destruction, decrease in output, or runaway pacing; acute or chronic increases in the pacing threshold, which is normally temporary; undersensing lasting up to 10 days; reprogramming; resetting to a different mode, usually asynchronous; lead displacement; and myocardial thermal or electrical burns at the electrode-myocardium interface leading to ventricular fibrillation. [27]

Due to these potential complications, Barold and colleagues have suggested guidelines for defibrillation and cardioversion in pacemaker patients. [27] The first of these is use of the anterior-posterior paddle position, if possible, preferably with specific anterior and posterior paddles. When impossible, the paddles should be placed along a line perpendicular to the line between the pulse generator and the tip of the ventricular lead. For a patient with a pacemaker generator in the left upper chest wall, the appropriate paddle placement would be left lateral chest wall and right midsternal border. For a patient with a pacemaker generator in the right upper chest wall, the appropriate paddle placement would be left upper chest wall and right lower chest wall. Second, the paddles should be placed at least 10 cm from the pulse generator or lead. Third, because of potential damage to the pacemaker or leads, a transcutaneous pacemaker and pads should be readily available if needed, and standby cardiology consultation should be arranged in case emergent reprogramming is needed. Fourth, the patient must be admitted to the hospital and pacemaker functioning must be monitored carefully with repeated threshold testing.

Because of the lower current used by AICDs, the possibility of pacemaker damage from AICD discharge in patients with both is remote. [27]

The AICD patient who is in cardiopulmonary arrest may be managed in the same manner as patients without the device, with minor modifications. Cardiopulmonary resuscitation (CPR) and transthoracic defibrillation will not harm the AICD. [68] The AICD may spontaneously discharge without warning if the patient's rhythm meets the preset criteria for shocking. In the case of an AICD shock, medical personnel performing CPR may experience a mild electrical shock, [72] but to date no reports of injury to medical personnel from such a shock have been noted.

Some authors recommend leaving the AICD activated during cardiopulmonary arrest, [68] allowing it to discharge up to the maximum of four times allowed by the circuitry; but deactivation may be necessary to alleviate fears of medical personnel, to avoid AICD shock-induced arrhythmias, or if temporary cardiac pacing is needed. [2]

Defibrillation does not harm the device or circuitry, and may be performed with the usual technique, although a theoretical possibility exists that the epicardial patches can shield the myocardium from the delivered energy. For this reason, paddle placement over the apex and right sternum may be indicated for AICD patients with anterior and posterior myocardial patches, with anterior and posterior paddle placement in AICD patients with a single patch over the cardiac apex. [72]

CONCLUSION

Many patients have implanted pacemakers or AICDs. These patients have underlying cardiac disease and may present to the ED with various complaints. Complications associated with these devices are not uncommon. The evaluation of these patients must be directed toward the potential complications. Pacemaker function may be evaluated by ECG and magnet testing, but most problems will require cardiology consultation and intervention. Runaway pacemaker represents a true emergency and must be dealt with expeditiously. AICD complications can be initially evaluated in the ED, but they normally require specialized interrogation devices to analyze the problem. Patients with pacemakers or AICDs in cardiopulmonary arrest require modifications of standard paddle placement for defibrillation or cardioversion, and if an AICD is present, it may need to be deactivated with a magnet.

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Chapter 14 - Basic Electrocardiographic Techniques

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The electrocardiogram (ECG) is a graphic recording of the electrical activity of the heart. The heart itself is unique among the muscles of the body in that it possesses the ability to generate regular electrical impulses to produce rhythmic cardiac contractions. These impulses can be measured and recorded at the body surface. The standard ECG is obtained by applying electrode leads over the chest and limbs to record the electrical activity of the cardiac cycle. Developed in the early 1900s, the ECG remains the most important initial diagnostic tool for the assessment of myocardial disease, ischemia, and cardiac dysrhythmias.

Electrocardiography is performed widely throughout the health care field, including in ambulances, ambulatory clinics, emergency departments (EDs), and inpatient hospital units. Standard ECG machines are small, self-contained, and portable, allowing them to be used in virtually any setting. As a result, clinicians, nurses, and many other health care providers should be familiar with the procedure of standard 12-lead electrocardiography. Emergency clinicians should also be familiar with the alternative leads and other accessory techniques available in electrocardiography, as well as the pitfalls of lead misplacement, misconnection, and tracing artifacts.

BACKGROUND

In 1903, Dutch physiologist Willem Einthoven first published his recordings of the cardiac cycle using a new device, the string galvanometer. [1] Einthoven's instrument consisted of a thin silver-coated quartz filament stretched across a magnetic field. When an electrical current passed through the string, it caused movement from side to side. The filament was connected to electrode leads placed on the limbs to measure differences in potential caused by the electrical activity of the heart. Einthoven magnified these measurements with a projecting microscope and recorded them photographically. [2]

While others had previously recorded cardiac electrical activity, Einthoven's instrument laid the basis for modern clinical electrocardiography. His work described the standard frontal plane limb lead ECG using bipolar electrodes, and established standards for recording rate and amplitude. In addition, he described five separate electrical deflections, which he termed P, Q, R, S, and T, establishing basic ECG nomenclature. [3] Einthoven won the Nobel Prize in 1924 for his ECG recording machine, which has been called "probably the most sophisticated scientific instrument in existence when it was first invented." [4]

Thomas Lewis visited Einthoven's laboratory and recognized the potential clinical utility of the ECG machine. Lewis became the leading authority on electrocardiography in the early 1900s and was instrumental in the development and clinical application of this new technology. [2] Using the ECG machine, Lewis determined that atrial fibrillation was due to a "circus conduction" involving the auricle of the heart. He published much of his clinical work on ECGs in his landmark texts, *The Mechanism of the Heart Beat* in 1911 and *Clinical Electrocardiography* in 1913.[5] [6]

The development of smaller, portable bedside ECG recording machines after World War I led to their rapid dissemination and use in the clinical setting. In the early 1930s, Francis Wood and Charles Wolferth first reported the use of ECGs to differentiate cardiac and non-cardiac chest pain. [2] Along with Frank Wilson, their work also led to the development of the unipolar "exploring" electrode lead, which measured electrical activity anywhere in the body with a zero potential central terminal as a reference. These leads could be placed directly over the chest, forming the basis for the standard precordial leads. [7]

In 1938, the American Heart Association, in conjunction with the Cardiac Society of Great Britain, established the standard six precordial chest lead positions (V1–6).[8] These precordial leads, along with Einthoven's original bipolar limb lead system (I, II, III) and the augmented unipolar limb leads developed by Emmanual Goldberger (aVR, aVL, and aVF) in 1942, comprise the standard 12-lead ECG used today.

INDICATIONS

Numerous situations in the ED may require a 12-lead electrocardiogram—the typical ED patient undergoing electrocardiography has, in fact, approximately 3 indications for obtaining an ECG.[9] The most frequent indication for ECG performance in the ED is the presence of chest pain. Other reasons include dyspnea, syncope, diagnosis-based indications (e.g., acute coronary syndrome [ACS], suspected pulmonary embolism), and system-related indications (e.g., "rule out myocardial infarction" protocol, admission purposes, operative clearance).[9] Within a given diagnosis, the ECG may perform many functions. In the chest pain patient, for example, the ECG helps establish the diagnosis of ACS or, alternatively, some other noncoronary ailment. Moreover, it is used to select appropriate therapy, determine the response to ED-delivered treatments, establish the correct inpatient disposition location, and help predict the risk of both cardiovascular complication and death.

The initial 12-lead ECG obtained in the ED can be a helpful guide for determination of cardiovascular risk and, as such, the choice of in-hospital admission location. Brush and colleagues have classified the initial ECG into high- and low-risk groups. The low-risk electrocardiographic group had absolutely normal ECGs, nonspecific ST-T wave changes (NSSTTW), or no change when compared with a previous ECG. High-risk ECGs had significant abnormality or confounding pattern, such as pathologic Q waves, ischemic ST segment or T-wave changes, left ventricular hypertrophy, left bundle-branch block, or ventricular paced rhythm. Patients with initial ECGs classified as low-risk had a 14% incidence of acute myocardial infarction (AMI), 0.6% incidence of life-threatening complications, and 0% mortality rate. Patients with initial ECGs classified as high-risk had a 42% incidence of AMI; 14% incidence of life-threatening complications, and 10% mortality rate.[10] Another approach to risk prediction involves a simple calculation of the number of electrocardiographic leads with ST-segment deviation (elevation

or depression)—with an increasing number of leads being associated with higher risk. Along similar lines, the clinician can also predict risk with a summation of the total millivolts of ST segment deviation; once again, higher totals are associated with greater risk.[10]

BASIC EQUIPMENT

The 12-lead ECG.

Although there is variability depending upon the workplace, most ECG machines today are three-channel recorders with computer memory. Such multi-channel systems, recording electrical events in several leads concurrently, offer advantages over the antiquated single-channel recorder systems—capturing transient events on multiple leads simultaneously; banking the data in computer memory for storage, comparison, and transmission; and allowing for data presentation on a single sheet of paper.[11] The ECG tracing is printed in a standardized manner on a standardized paper by the electrocardiograph, which has default settings regarding the speed with which the paper moves through the machine, as well as the amplitude of the deflections to be made on the tracing. ECG paper is divided into a grid, with a series of horizontal and vertical lines; the thin lines are 1 mm apart, and the thick lines are separated by 5 mm. At the standard paper speed of 25 mm/sec, each vertical thin line thus represents 0.04 sec (or 40 msec), whereas the thick vertical lines correspond to 0.20 sec (or 200 msec). Recordings from each of the 12 leads are typically displayed for 2.5 sec by default setting; the leads appearing horizontally adjacent to each other are separated by a small vertical hashmark to represent lead change.

The standard ECG includes 12 leads derived from 10 electrodes placed on the patient; each is color-coded and represented by a two-character abbreviation (Table 14-1). The placement of limb leads on the left and right arms (LA and RA, respectively) and the left and right legs (LL and RL, respectively) by color can be recalled with the help of several mnemonics: "Christmas trees below the knees" (the green and red leads are placed on the lower extremities); "white on right and green to go" (the white lead is placed on the right arm and the green lead is placed on the leg that controls the gas pedal, while the red lead is correspondingly placed on the leg that is closer to the brake); and "smoke over fire" (the black left arm lead is placed over the red left leg lead, as with telemetric monitoring pads). Use of these mnemonics may help prevent right/left confusion during lead placement, as well as the

TABLE 14-1 -- Conventional Leads for the 12-Lead Electrocardiogram

Location	Notation	Color
Right arm	RA	White
Left arm	LA	Black
Left leg	LL	Red
Right leg	RL	Green
Precordial leads	V1	Brown/Red
	V2	Brown/Yellow
	V3	Brown/Green
	V4	Brown/Blue
	V5	Brown/Orange
	V6	Brown/Violet

consequences of limb lead reversal and misinterpretation of the ECG (see "Lead Misplacement and Misconnection," later in this chapter).

Standard 12 leads.

The standard 12-lead ECG depicts cardiac electrical activity from 12 points of view, or leads, which can be grouped according to planar orientation. Six leads (I, II, III, aVR, aVL, and aVF) are oriented in the frontal, or coronal, plane and derived from the four limb electrodes. The six precordial leads (V1, V2, V3, V4, V5, and V6) are oriented in the horizontal, or transverse, plane with each representing cardiac electrical activity from that perspective. Leads I, II, and III are termed limb leads; they are bipolar in that they record the potential difference between two electrodes (Fig. 14-1). The fourth electrode located on the right leg serves as an electrical ground. The positive poles of these bipolar leads lie to the left and inferiorly, approximating the major vector forces of the normal heart. This early convention was established so that the tracing would feature primarily upright complexes. In contrast, augmented leads aVR, aVL, and aVF are unipolar leads, with the positive electrodes located at the respective extremities. These augmented leads serve to fill the electrical gaps between leads I, II, and III. Lead aVR stands alone with a polarity and resultant orientation opposite the other limb and augmented leads. This is due to the fact that its positive electrode is located in the opposite direction (superior and to the right) of the major vector force of the normal heart (inferior and to the left); thus, its complexes usually appear "opposite" to most or all of those in the other leads.

Merging of the vector axes of the limb and augmented leads around a central axis yields a hexaxial system of representation of cardiac electrical activity in the frontal plane (Fig. 14-2). The six precordial leads, oriented in the horizontal plane, represent six unipolar electrodes with vector positivity oriented toward the chest surface, with the central terminal of the hexaxial system serving as a negative pole. In contrast to

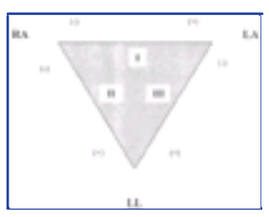

Figure 14-1 Bipolar limb leads. Leads I, II, and III are shown as a triangle, known as Einthoven's triangle. Left arm (LA), right arm (RA), and left leg (LL) placement is shown. These bipolar leads are oriented such that the positive poles lie inferiorly and to the left (given that the bottom apex of the triangle is directed toward the left leg)—as does the major electrical vector of the heart.

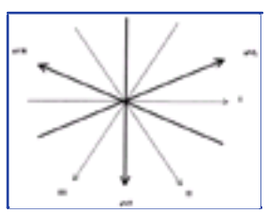

Figure 14-2 Hexaxial system of limb and augmented leads in the frontal plane. Each lead is separated by 30° in this frontal plane representation of the limb and augmented leads. Augmented leads are shown in boldface. Arrows denote positive polarity. Note that the inferior leads (II, III, aVF) logically lie at the bottom of this figure, and the lateral leads (I, aVL) lie on the left side of the figure, where the lateral aspect of the heart is located were this to be superimposed on a patient.

the frontal plane leads, the angles between each of the precordial leads in the horizontal plane are not equal; however, they can vary depending upon lead placement and body habitus.

Lead placement.

The four limb electrodes are conventionally placed on the extremities as follows: RA on the right wrist; LA on the left wrist; RL on the right ankle; and LL on the left ankle. Electrodes may be affixed more proximally on the limbs if necessary (e.g., amputation, severe injuries), ideally with a notation made on the ECG. [12] Others note that the electrodes may be placed on any part of the arms or legs, providing they are distal to the shoulders or inguinal/gluteal folds, respectively. [13]

The precordial leads should be placed as follows: V1—right sternal border, 4th intercostal space; V2—left sternal border, 4th intercostal space; V3—midway between V2 and V4; V4—left midclavicular line, 5th intercostal space; V5—left anterior axillary line, same horizontal level as V4; and V6—left midaxillary line, same horizontal level as V4 and V5. Note that V4 through V6 are placed at the same horizontal level, not all in the 5th intercostal space. If V5 and V6 are situated following the contour of the intercostal space rather than on the same horizontal level, they will be superiorly displaced as the ribs curve around the side of the thorax (Fig. 14-3).

Intercostal space number can be determined by first palpating the sternal angle (angle of Louis), which is the junction of the manubrium and body of the sternum. This transverse bony ridge is located about 5 cm caudad from the sternal notch in the adult. Immediately lateral and inferior to it is the second intercostal space; two spaces farther down lies the fourth intercostal space, where V1 and V2 should be placed. Alternatively, one can count down from the medial clavicle; beneath the clavicle lies the first rib, below which is the first intercostal space.

If the patient's anatomy or injury precludes placement of a precordial lead as described earlier, it is permissible to

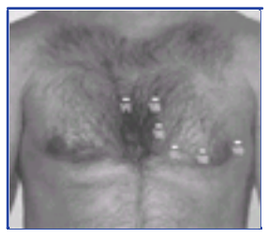

Figure 14-3 Precordial lead placement for the standard 12-lead ECG. If multiple or repeat ECG tracings are anticipated, the original lead placements should be marked on the patient's chest wall or stick-on leads should be left in place after the ECG wires are removed.

attach it within the radius of the width of one interspace of the recommended position, with appropriate notation on the tracing. If the situation demands further displacement, it is recommended that the lead be omitted with appropriate documentation on the tracing. [12]

Pediatric lead placement.

In addition to the standard 12-lead tracing, leads V4R and V3R should also be recorded. These are mirror images of their left-sided counterparts (see "Additional Leads" later in this chapter). The chest of the tiny infant may not accommodate all the precordial leads; in such cases, the following array is recommended: V3R or V4R, V1, V3, and V6. Limb lead placement is as in adults. [14]

FEATURES OF THE ELECTROCARDIOGRAM

Discussion of the interpretation of the ECG is beyond the scope of this chapter. Other features of the procedure itself, including a description of the other data found on the ECG tracing, will be detailed later.

In addition to the patient demographic data that are entered by the operator, the tracing will often feature computations regarding rate, intervals, and axes along the top of the paper. On some tracings, a computer-generated "reading" will also be displayed at the top of the tracing. These interpretations are not infallible. A sample of nine of these programs was compared with the readings of eight cardiologists; the "gold standard" in this study was the clinical diagnosis made independently of the interpretations of these tracings, based on other objective data (e.g., echocardiography, cardiac catheterization). The performance of the programs was good, with correct interpretations in a median of 91% of cases, but the cardiologists were significantly better (median 96% correct). [15] Of note, this study did not evaluate interpretations

of acute ischemia and cardiac rhythm disturbance—perhaps the most critical issues in ECG interpretation.

Adjustable features.

Somewhere on the tracing, notation of the ECG paper speed (mm/sec), the calibration (mm/mV), and the frequency response in Hertz (Hz) will be evident (in Fig. 14-4 , these appear in the lower left corner of the tracing). *Calibration*, or standardization, refers to the amplitude of the waveforms on the tracing. It is usually set at a default value of 10 mm/mV, and is graphically depicted by a plateau-shaped waveform that appears at the extreme left side of the tracing, in front of the first complex (see Fig. 14-4*A*). This calibration can be modified by the operator, or by the computer itself, as was the case in Figure 14-4*B* , where the patient appeared to have acquired voltage criteria for left ventricular hypertrophy, when in reality the tracing was unchanged from his baseline (see Fig. 14-4*A*). Increasing the calibration to 20 mm/mV is helpful when trying to decipher P wave morphology. Decreasing the calibration to 5 mm/mV is helpful in cases wherein the amplitude of the QRS complex (usually in the precordial leads) is so large that it encroaches upon those of the adjacent leads. Standardization may not be uniform throughout a given tracing. At times the

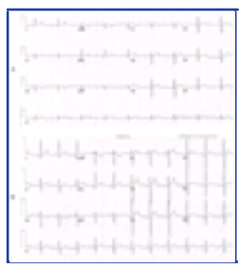

Figure 14-4 *A,* Normal 10 mm/mV calibration. Note the box-shaped mark to the left of the complexes; this is a graphic representation of the calibration for the tracing. This parameter should be routinely noted before ECG interpretation. Note the change in *B*. *B,* Abnormal 20 mm/mV calibration. The calibration in this tracing was (inexplicably and unexpectedly) changed to 20 mm/mV by the ECG, not by the operator. When compared with a baseline ECG, it appeared that the patient had developed voltage criteria for left ventricular hypertrophy as well as ST segment elevation. *A,* which was recorded minutes later with correction of calibration to the standard 10 mm/mV, was unchanged from baseline tracings.

calibration will be adjusted automatically by the electrocardiograph based upon the waveform amplitudes it perceives. For example, it is possible to have normal calibration (10 mm/mV) in the limb and augmented leads, with half-standard calibration in the precordial leads (5 mm/mV); this may occur in instances of marked left ventricular hypertrophy. In this case, the calibration pulse at the lefthand side of the paper will have a downward stairstep appearance.

Paper speed usually is set at a default of 25 mm/sec. It may be manipulated for purposes of deciphering a dysrhythmia, as described later in this chapter (see "Alteration in Amplitude and Paper Speed"). *It is important that the clinician examine all ECG tracings for standardization and speed parameters before attempting clinical interpretation.*

ADDITIONAL LEADS

Although not considered standard of care in the routine evaluation of patients in the ED, additional electrocardiographic leads have been investigated for the evaluation of the patient with possible ACS. These additional, or nontraditional, leads

include posterior leads (V7, V8 and V9), right ventricular leads (especially V4R), and procedural leads (transvenous pacemaker wire placement and pericardiocentesis). Acute posterior and right ventricular myocardial infarctions are likely to be underdiagnosed, as the standard 12-lead ECG does not assess these areas directly. The standard ECG coupled with these additional posterior leads constitutes the 15-lead ECG, the most frequently used extra-lead ECG in clinical practice.

15-Lead ECG.

In a study of all ED chest pain patients, Brady et al. reported that the 15-lead ECG provided a more accurate description of myocardial injury in those patients with AMI, yet failed to alter rates of diagnoses, use of reperfusion therapies, or change disposition locations. [16] Looking at a more select population of ED patients, Zalenski and associates investigated the use of the 15-lead ECG in chest pain patients with a moderate-to-high pretest probability of AMI, who were already identified as candidates for hospital admission. [17] In this 15-lead ECG study, the authors reported an 11.7% increase in sensitivity with no loss of specificity (i.e., no increase in false-positive findings) for the diagnosis of ST elevation AMI. They concluded that "the findings of ST elevation (STE) by use of these extra leads can strengthen the ED diagnosis of acute myocardial infarction on the initial tracing and may provide an indication for thrombolytic treatment." They further suggest that, in the diagnosis of posterior AMI, leads V8 and V9 are superior to reliance upon detecting the reciprocal ST segment depression seen in leads V1–V3.

Possible indications for 15-lead ECGs in patients with suspected acute ischemic heart disease include (1) ST segment depression in leads V1 through V3; (2) all STE inferior and lateral AMIs; or (3) isolated STE in leads V1 and V2, or both. These indications, despite their apparent clinical utility, remain unproved, and the 15-lead ECG is currently not considered standard of care for evaluation in the ED.

Posterior leads.

The posterior leads V8 and V9 are placed on the patient's back—V8 at the tip of the left scapula and V9 in an intermediate position between lead V8 and the left paraspinal muscles. An additional lead, V7, may also be placed on the posterior axillary line equidistant from lead V8 (Fig. 14-5). The degree of ST segment elevation in the posterior leads is often less pronounced compared with the STE seen in the standard 12 leads in patients with STE AMI. This diminution of posterior lead STE results from both the relatively greater distance of these leads from the posterior surface of the heart as well as presence of air and soft tissue between the epicardium and electrocardiographic leads.

Right-sided leads.

The right ventricular electrocardiographic leads are placed across the right side of the chest in a mirror image of the standard left-sided leads and are labeled V1R to V6R; alternatively, RV1 to RV6 is another commonly used nomenclature for this lead distribution (Fig. 14-6). Lead V4R (right fifth intercostal space mid-clavicular line) is the most useful lead for detecting STE associated with right ventricular infarction and may be used solely in the evaluation of possible right ventricular infarction. The STE that occurs in association with right ventricular infarction is frequently quite subtle, reflecting the relatively small muscle mass of the right ventricle; at other times, the STE is quite prominent, similar in appearance to the ST segment changes seen in the standard 12 leads.

Invasive procedural leads.

A patient may present with a severely compromising bradydysrhythmia and require a transvenous pacemaker. In such instances, the pacing wire must be

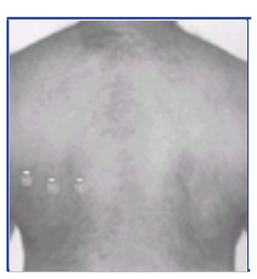

Figure 14-5 Posterior lead placement. Leads V7, V8, and V9 are placed on the same horizontal plane as V6, with V7 at the posterior axillary line, V8 at the tip of the left scapula, and V9 near the border of the left paraspinal muscles.

placed without the benefit of fluoroscopy. The wire can be advanced using electrocardiographic guidance. Such placement requires that the patient be connected to the limb leads of a grounded electrocardiographic machine and the pacing wire connected to the V lead. As the electrode enters the vena cava superior and high right atrium, the P wave and QRS complex

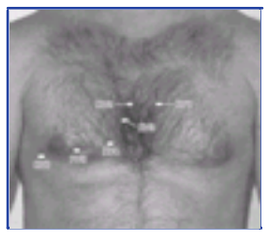

Figure 14-6 Right-sided lead placement. Right-sided leads RV1–RV6 are placed on the chest as a mirror image of the standard precordial leads.

will be negative. While traversing the atrium, the P wave and QRS complex will become positive, with the latter becoming larger as the ventricle is approached. If a balloon-tipped flotation catheter is used, the balloon should be deflated once in the right ventricle, and advanced until contact is made with the endocardium and the ventricle is captured. Ventricular wall contact is indicated by marked ST segment elevation.

In patients with suspected pericardial effusion who undergo urgent pericardiocentesis, an electrocardiographic lead may be placed on the syringe needle; this form of monitoring assists in the correct positioning of the catheter in the pericardial space. With advancement of the needle, ST segments are monitored. The sudden appearance of ST segment elevation indicates that the needle has moved too far internally (i.e., beyond the pericardial space) and has made contact with the epicardium.

ALTERNATIVE LEADS AND TECHNIQUES FOR RHYTHM ASSESSMENT

Electrocardiographic rhythm assessment depends on a clear signal of both atrial and ventricular electrical activity over a period of time. While continuous 12-lead ECG rhythm monitoring has the advantage of recording cardiac activity over multiple leads (thus maximizing atrial and ventricular monitoring), it is often impractical. Moreover, correct identification of the cardiac rhythm on ECG can be difficult depending on the clinical setting. Rapid atrial or ventricular rates, especially those above 150 beats/min, often lead to simultaneous or near-simultaneous deflections that can alter the usual waveforms or cause smaller deflections to be buried within larger ones (such as P waves buried within the QRS complex). In addition, rapid rates result in smaller, narrower waveforms that make visual recognition on the ECG challenging. Finally, assessment of atrial activity is generally more difficult due to the smaller electrical impulse and resulting ECG waveform that is generated by the atria.

A number of alternative techniques have been developed to improve rhythm assessment. These techniques include alterations in the standard 12-lead ECG, as well as the addition of non-standard leads to monitor cardiac, and particularly atrial, rhythm activity.

Alteration in amplitude and paper speed.

Most 12-lead ECG machines today allow alteration of both amplitude and paper speed from the basic 10 mm/mV and 25 mm/sec standards, respectively. Increasing the amplitude, most commonly to double the standard or 20 mm/mV, can increase the prominence of smaller deflections, such as the P wave, and improve recognition of the atrial rhythm (Fig. 14-7*A–D* Fig. 14-7*A–D*). In addition, clinicians have also used photocopy enlargements of the standard ECG to enhance smaller deflections visually.[18]

Increasing the paper speed, again most commonly to double the standard, or 50 mm/sec, has the effect of artificially slowing the rhythm. This technique is most advantageous when assessing patients with marked atrial or ventricular tachycardia. Increasing the paper speed exaggerates any existing irregularity (such as in atrial fibrillation) and can improve recognition of smaller deflections, such as P waves, in the presence of a significant tachycardia. Faster paper speeds also make it possible to measure short ECG intervals (such as P-R or R-R) more accurately (Fig. 14-8*A, B*).

Accardi et al found that overall diagnostic accuracy improved when clinicians were provided ECGs recorded at the faster 50 mm/sec paper speed, as opposed to a standard 12-lead ECG, in patients with narrow complex tachycardias. Moreover, they reported this improved rhythm assessment likely would have resulted in fewer treatment errors.[19]

Alternative leads.

Rhythm assessment often requires electrocardiographic monitoring over continuous periods of time, making the standard 12-lead ECG (requiring 10 electrodes), and even unipolar precordial V1 monitoring (requiring 5 electrodes), not feasible. A number of alternative lead systems requiring fewer electrodes have been described. Many of these systems use the limb bipolar leads (RA, LA, LL) in alternative positions over the chest. Leads I, II, or III are then recorded depending on the positions of the positive and negative electrodes.

Lewis leads.

In 1910, Thomas Lewis first described alternative positions for the RA and LL leads to enhance detection of atrial fibrillation. The RA lead was placed over the right second costochondral junction, while the LL lead was placed in the right fourth intercostal space 2.5 cm to the right of the sternum—leaving the LA and RL leads in their usual positions. Lewis[20] reported enhancement of atrial activity when the RA served as the negative electrode and LL as the positive electrode (lead II) with this new configuration. Other alternative lead placements to enhance atrial activity detection have also been described (Table 14-2 and Fig. 14-9).[21] [22] [23]

Vertical sternal "Barker" leads.

In this alternative lead system, the positive electrode is placed at the xiphoid process and the negative electrode is placed just below the suprasternal notch on the manubrium. Herzog et al[18] reported that vertical sternal leads produce a larger P wave than other systems, including the Lewis leads. In addition, the vertical sternal leads are placed over bone, which may reduce muscle activity artifacts on recordings (see Fig. 14-9).

Limb-precordium leads.

A sequential pattern of bipolar leads on the chest, termed limb-precordium leads, has been proposed in combination with the original Einthoven limb leads. In this system, standard limb leads are placed on the patient. The RA electrode is then repositioned sequentially at the fourth intercostal space just right of the sternum, fourth intercostal space just left of the sternum (low parasternal), 1st intercostal space just left of the sternum, and 1st intercostal space just right of the sternum (high parasternal). During this sequential mapping, tracings are recorded for leads I and II until atrial activity is identified. Brenes-Pereira reported that this mapping system allowed for the identification of P waves for a majority of patients when none was detected initially on a standard 12-lead ECG. [24]

MCL leads.

Modified bipolar chest leads (MCL) are the most commonly used leads for cardiac rhythm monitoring. The positive electrode is placed on the chest at a precordial position (V) concordant with the MCL desired (e.g., the V_1 position for MCL_1). The negative electrode is placed on the left shoulder. On standard ECG machines, the LA electrode is placed at V_1 , RA at the left shoulder, LL at V_6 , and RL at a remote location on the chest to serve as ground. Lead I would then reflect MCL_1 and lead II, MCL_6 . MCL_1 may be useful in distinguishing atrial activity, MCL_5 and MCL_6 more commonly in ST/T wave monitoring, and both MCL_1 and MCL_6 may be useful in evaluating wide complex tachycardias (see Fig. 14-9).[25]

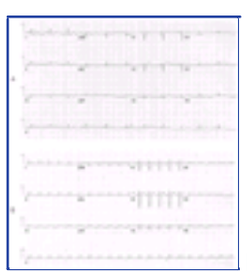

Figure 14-7a *A,* Baseline ECG of patient before development of abnormal rhythm (10 mm/mV). Note the P wave morphologies, especially in leads I, II, and V1. *B,* ECG during ectopic atrial tachycardia (10 mm/mV). Note the change in P wave morphology, especially in lead V1.

Esophageal leads.

The esophageal lead (E) was first described by Brown in the 1930s. [26] Since then, both unipolar and bipolar esophageal leads have been developed. [27] Because of its posterior location, this lead is often superior at detecting atrial deflections and recording the activity of the posterior surface of the left ventricle. The electrode, which is connected to the ECG machine by thin wires, is either swallowed, or passed through the nares, into the esophagus. Once in the esophagus, the location of

the electrode is determined either by fluoroscopy or by making a series of low-to-high esophageal recordings. The position of the electrode in the esophagus is adjusted by slowly pulling the electrode wire out the nares or mouth. For the normal adult, leads $E_{15–25}$ (electrode is located in the esophagus 15 to 25 cm from the nares) generally records atrial activity; $E_{25–35}$, activity of the AV groove; and $E_{40–50}$, activity of the LV posterior surface. The E lead should be recorded through lead channel I, simultaneously with the lead channel II and the other surface channels.

LEAD MISPLACEMENT AND MISCONNECTION

Limb lead reversals.

Whereas the limb electrodes are not often misplaced, the cables that link them to the ECG machine are at times improperly connected. This can result in "ECG changes" that are in actuality artifacts. There are a multitude of possibilities for misconnection of the limb leads; some of the most probable are summarized here. It is helpful to categorize these possibilities into those that are recognizable without comparison to an old ECG versus those that are not.

Recognizable without old ECG.

The most common of all misconnections is left and right arm lead reversal (Fig. 14-10). *The hallmark is a negative P wave and primarily negative QRS complex in lead I, creating a right or extreme axis deviation* (depending upon the QRS complex in lead aVF). Dextrocardia should also be considered with this

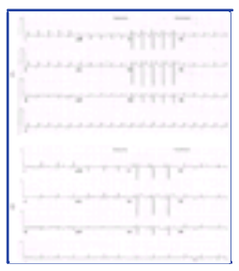

Figure 14-7b *C,* ECG during ectopic atrial tachycardia (20 mm/mV). The P waves are now easier to see in all leads. *D,* ECG after reversion to normal atrial focus (20 mm/mV). Contrast these accentuated P waves to those in *C*.

presentation; the pattern of precordial lead transition will differentiate between dextrocardia and arm lead reversal. Moreover, lead aVR is actually aVL in this circumstance, and thus may feature an upright QRS, which is highly unusual for aVR. [28] Transposition of the RA and LL cables is also easily recognized; all leads are upside down compared with the usual patterns, with the exception of aVL, which is unchanged.

Anytime the RL lead is transposed with another extremity lead, one of the limb leads will appear as virtually a straight line, and thus is easily recognized if this finding is not incorrectly ascribed to poor electrode contact or function. An exception to this rule is if the leg leads are reversed (RL ? LL), in which case the ECG is virtually identical to one with correct placement of the leads. Reversal of the leg leads is largely insignificant in that the potentials at the left and right legs are essentially the same.[29]

Recognizable with old ECG.

In addition to the reversal of the leg leads, one other limb lead reversal that is not readily recognizable without comparison to a prior tracing is transposition of the LA and LL leads. This causes reversal of lead I with II on the tracing, as well as aVL with aVF—both are difficult to discern at times without a baseline ECG for comparison. Furthermore, lead III will be upside down (although a negative QRS complex in III is not unusual), and aVR will be unchanged (Fig. 14-11*A, B*).[29]

Clues to limb lead reversal are summarized in Table 14-3 .

Precordial lead misplacement and misconnection.

Unlike the limb leads, the precordial electrodes are more prone to misplacement, especially when variations in body habitus (e.g., obesity, breast tissue, pectus excavatum, chronic lung disease) make proper lead placement more difficult. This may cause some variability in the amplitude and morphology of the complexes in the precordial leads; however, these changes are not usually grossly abnormal, and therefore can be difficult to detect. Variation often becomes evident when comparing the current tracing with an old ECG. [29] In such cases, it is useful to go to the bedside and examine where the electrodes were positioned relative to the recommended placement (see "Lead Placement" earlier in this chapter). One

Figure 14-8 *A,* ECG with tachycardia at normal paper speed (25 mm/sec). Because of the rapid rate, the actual P waves are difficult to discern, thus making rhythm determination difficult. The computerized interpretation is sinus tachycardia with first-degree AV block. *B,* ECG with tachycardia at double paper speed (50 mm/sec). With increased paper speed, atrial P wave activity is accentuated, demonstrated atrial flutter with a 2:1 AV block.

cannot ensure, however, that the baseline ECG was done with proper lead placement. When comparing the precordial leads on the current ECG with a baseline tracing, ST segment and T wave changes should be viewed in light of the relative morphologies of the associated QRS complexes. If there is a marked difference between the two tracings in the amplitude and polarity of the QRS complex in a given precordial lead, the corresponding ST/T wave changes may be due to lead

TABLE 14-2 -- Alternative Leads for Rhythm Assessment

Lead I[1] :	RA = negative electrode	LA = positive electrode
Lead II[1] :	RA = negative electrode	LL = positive electrode
Lead III[1] :	LA = negative electrode	LL = positive electrode
Alternative Lead	**Negative Electrode Position**	**Positive Electrode Position**
Lewis[2]	R 2nd costochondral junction	R 4th intercostal space, "1" right of sternum
Drury	2nd R costochondral junction	7th R costal cartilage
	Center of sternum	Inferior angle of scapula 2" right of spine
Schoenwald	3rd intercostal space along R sternal border	L leg
	3rd intercostal space along R sternal border	R arm
Lu	1st intercostal space directly above V1	Approximately 3" directly below V4
Vertical Sternal ("Barker" Leads)	Below suprasternal notch at manubrium	Xiphoid process

MCL_1	L shoulder (1 cm inferior to L mid-clavicle)	V1 (4th intercostal space R sternal border)
MCL_6	L shoulder (1 cm inferior to L mid-clavicle)	V6 (~6th rib mid-axillary line)

[1] First, set the ECG machine to record the rhythm strip using this lead. If the recoding rhythm strip is lead I, the RA wire becomes the negative electrode that is placed as noted in table, and the LA wire becomes the positive electrode that is placed as noted in the table. If lead II or lead III is the lead that is set to record the rhythm strip, the positive and negative electrodes will vary.
[2] Example: One way to record the Lewis lead: Set the ECG machine to record lead I, use the RA wire as the negative electrode, and place it in the R 2nd costochondral junction. Use the LA wire as the positive electrode and place it in the right 4th intercostal space, 1 inch right of the sternum. The Lewis lead may also be recorded on lead II and lead III, but the wires that serve as the positive and negative electrodes will vary.

placement, although cardiac ischemia cannot be completely excluded as the cause.

Misconnection of the precordial cables is usually easy to detect. The expected progression of P, QRS, and T wave morphologies across the precordium will be disrupted (Fig. 14-12*A, B*). An abrupt change in wave morphology evolution—followed by a seeming return to normalcy in the next lead—is a good clue to precordial lead misconnection.[29]

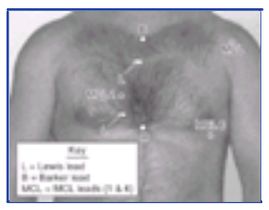

Figure 14-9 Alternative leads. The figure displays three of the more commonly used alternative lead strategies for atrial rhythm clarification (Lewis leads, vertical sternal or Barker leads, and MCL1), and ST/T wave monitoring (MCL6).

ARTIFACTS

Electrocardiographic artifacts are commonly encountered, yet not always easy to recognize. They can be attributed to either physiologic (internal) or nonphysiologic (external) sources; the former includes muscle activity, patient motion, and poor electrode contact with the skin. Tremors, hiccups, and shivering may produce frequent, narrow spikes on the tracing, simulating atrial and ventricular dysrhythmias (Fig. 14-13). [28] [30] A wandering baseline featuring wide undulations, as well as other "noise" on the ECG, can often be traced to patient movement and high skin impedance, leading to inadequate electrode contact to the skin. Minimizing skin impedance and artifacts may be achieved by (1) avoiding electrode placement over bony prominences, major muscles, or pulsating arteries; (2) clipping rather than shaving thick hair at electrode sites; and (3) cleaning and (most importantly) drying the skin surface before reapplying the electrode if the tracing features substantial artifacts.[28] [31] Nonphysiologic artifacts are most often due to 60-Hertz electrical interference, which is ascribable to various other sources of alternating current near the patient. This will manifest as a wide, indistinct isoelectric

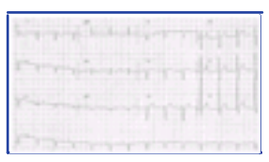

Figure 14-10 Arm lead reversal (LA RA). The most common of limb lead reversals, the clues lie in leads I and aVR. Lead I features a negative P wave, as well as a principally negative QRS complex and T wave. This could suggest dextrocardia, but the precordial leads demonstrate normal transition, which is not consistent with dextrocardia. Note also the unusual appearance of aVR in this tracing.

baseline. Other sources of nonphysiologic artifact include loose connections, broken monitor cables, and mechanical issues with the machine (e.g., broken stylus, uneven paper transport). The 60-Hz artifact due to electrical current interference can be minimized by shutting off non-essential sources of current in the vicinity, as well as straightening the lead wires so that they are parallel to the patient's body in the long axis. [28] [30] [32]

Differentiation of artifacts from true ECG abnormality is intuitively important; moreover, clinical consequences have been reported that are directly attributable to confusion of artifacts with disease. Unnecessary treatment and procedures—including cardiac catheterization, electrophysiologic testing, and even implantation of a pacemaker and an automatic defibrillator—have been reported. [33] Characteristics that may aid in differentiating artifacts from dysrhythmia include absence of hemodynamic instability during the event (or even absence of any symptoms); normal QRS complexes occurring during the dysrhythmia; instability of the baseline on the tracing during and immediately after the "dysrhythmic" event; association with body movement; and observance of "notches" amidst the complexes of the pseudodysrhythmia, which "march out" with the normal QRS complexes that precede and follow the disturbance. [34] [35]

CONCLUSION

Electrocardiography is a simple, noninvasive, and invaluable bedside test. While used principally for the diagnosis and treatment of cardiovascular disorders, it has numerous other applications as well. Knowledge of standard lead placement and the features of the 12-lead ECG are essential. Several features—including adjustment of calibration, paper speed, and the addition of multiple accessory leads—can add to the diagnostic sensitivity of the procedure. Strategic repositioning of limb and precordial leads as described will aid in the assessment of difficult atrial rhythms. The addition of other thorax leads, such as V8 and V9 (posterior wall) and V4R (right ventricle), will increase the sensitivity of the ECG for ACS involving those difficult-to-assess areas of the heart. Pitfalls include failure to recognize lead misconnection and misplacement, as well as artifacts.

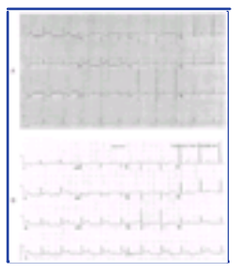

Figure 14-11 *A,* Limb lead reversal (LA ⟷ LL). A patient with a history consistent with ACS was brought to the ED after this ECG was recorded in a clinic. Leads I and aVL suggest an acute high lateral infarct but, surprisingly, there are no corresponding changes in leads V5 and V6. The deep T wave inversions in III and aVF were at first thought to be inferior ischemia or reciprocal changes (see also *B*). *B,* Correction of lead reversal (LA ⟷ LL). After the leads were reconnected, this tracing reveals an acute inferior wall MI, as well as deep T wave inversion in aVL—a harbinger of acute inferior MI. Comparing this tracing with that in *A,* note the following: lead I ⟷ lead II; lead aVL ⟷ aVF, and lead III is inverted. Thus, inferior changes become lateral, and lateral become inferior.

TABLE 14-3 -- Clues to Improper Limb Lead Connections

Reversed Leads	Old ECG Necessary for Detection?	Key Findings
LA RA	No	PQRST upside down in lead I
		Precordial leads normal (not dextrocardia)
LA LL	Yes	III is upside down
		I ⟷ II; aVL ⟷ aVF; aVR no change
LA RL	No	III is straight line
RA LL	No	PQRST upside down in all leads except aVL
RA RL	No	II is straight line
LL RL	Cannot detect change	Looks like normal lead placement
LA LL	No	I is straight line
RA RL		aVL, aVR are same polarity and amplitude *and* II is upside down III

From Surawicz B, Knilans TK: Chou's Electrocardiography in Clinical Practice, 5th ed. Philadelphia, WB Saunders, 2001.

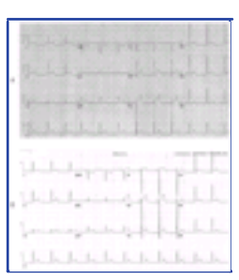

Figure 14-12 *A* and *B*, Precordial lead reversal (V2 ⟷ V3). Note the usual precordial progression of R wave growth in leads V2 and V3 is disrupted in the tracing displayed in *A; B* shows a return to a normal V3 transition zone.

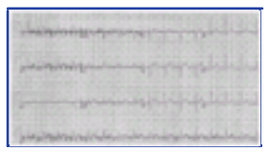

Figure 14-13 Artifact due to physiologic cause. The patient's monitor was alarming due to a perceived heart rate of >200 beats/min, and the computerized alert system called this ventricular tachycardia. The patient, who has Parkinson's disease, was without complaint. The ECG demonstrates a marked artifact, giving the appearance of atrial flutter in lead V1.

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Chapter 15 - Emergency Cardiac Pacing

Edward S. Bessman

The purpose of cardiac pacing is to restore or ensure effective cardiac depolarization. Emergency cardiac pacing may be instituted either prophylactically or therapeutically. Prophylactic indications include those situations where there is high risk of atrioventricular (AV) block, including certain toxidromes and instances of acute myocardial infarction. Therapeutic indications include symptomatic bradyarrhythmias, asystole, and overdrive pacing. Several approaches to pacing exist, including transcutaneous, transvenous, transthoracic, epicardial, endocardial, and esophageal. Transcutaneous and transvenous are the two techniques most commonly used in the emergency department (ED). Since it can be instituted quickly and noninvasively, transcutaneous pacing is the technique of choice in the ED. Transvenous pacing should be reserved for patients who require prolonged pacing or who have a very high (greater than 30%) risk of heart block. Transcutaneous pacing is generally a temporizing measure that may precede transvenous cardiac pacing.

EMERGENCY TRANSVENOUS CARDIAC PACING

The transvenous method of endocardial pacing is commonly used and is both safe and effective. In skilled hands, the semifloating transvenous catheter is successfully placed under electrocardiographic guidance in 80% of patients. [1] The technique can be performed in less than 20 minutes in 72% of patients and in less than 5 minutes in 30% of patients. However, in some instances anatomic, logistical, and hemodynamic impediments can prohibit successful pacing by even the most skilled clinician. As with other medical procedures, it should not be performed without a thorough understanding of its indications, contraindications, and complications. [2]

Background

The ability of muscle to be artificially depolarized was recognized as early as the 18th century. Initial efforts focused on the transcutaneous approach (see later in this section). Over the succeeding years, several scattered experiments were reported and in 1951 Callaghan and Bigelow first used the transvenous approach to stimulate the asystolic heart in hypothermic dogs. [3]

Furman and Robinson demonstrated the transvenous endocardial approach in humans in 1958. [4] They treated two patients with complete heart block and Stokes-Adams seizures, reconfirming that low-voltage pacing could completely control myocardial depolarization. The catheter remained in the second patient for 96 days without complication. Other early clinical studies proved that transvenous pacing is a valuable procedure in medicine. [5] [6] [7] Fluoroscopic guidance was used for placement of the pacing catheter in all of these studies.

In 1964, Vogel and colleagues demonstrated the use of a flexible catheter passed without fluoroscopic guidance for intracardiac electrocardiography. [8] One year later, this technique was used by Kimball and Killip to insert endocardial pacemakers at the bedside. [9] They noted technical difficulties including intermittent capture, difficulty passing the catheter, and catheter knotting in 20% of their patients. During the same year, Harris and associates confirmed the ease and speed with which this procedure could be accomplished. [10]

Before 1965 all intracardiac pacing was done asynchronously, which meant that the pacing catheter could cause electrical stimulation during any phase of the cardiac cycle. Asynchronous pacing frequently resulted in the pacemaker firing during the vulnerable period of an intrinsic depolarization; this occasionally caused ventricular tachycardia or fibrillation. In 1967 a *demand pacemaker generator* that sensed intrinsic depolarizations and inhibited the pacemaker for a predetermined period of time was used successfully by Zuckerman and associates in six patients. [11] Since then there has been steady progress in the design and functionality of pacemakers. Table 15-1 summarizes the four-letter code that is used to describe modern pacemakers (there is a fifth letter for combined pacemaker-cardioverter/defibrillators). The most commonly used emergency transvenous pacemaker is represented by the code VVI, meaning that the ventricle is paced, the ventricle is sensed, and when a native impulse is sensed, the pacemaker is inhibited. Dual chamber pacing (DDD or DDDR) is the preferred methodology for permanent pacing but is less commonly used emergently because of the increased complexity of the procedure (see later in this section).

Rosenberg and colleagues introduced an improved pacing catheter known as the Elecath semifloating pacing wire. [1] The Elecath was stiffer than the Flexon steel wire electrode that was in prevailing use. Rosenberg and coworkers achieved pacing in 72% of patients, with an average procedure time of 18 minutes. They also noted that 30% of their patients were paced in 5 minutes or less. [1] The technique of heart catheterization using a flow-directed balloon-tipped catheter was introduced by Swan and Ganz in 1970. [12] This concept was used successfully by Schnitzler and coworkers for the placement of a right ventricular pacemaker in 15 of 17 patients. [13]

In 1981, Lang and colleagues compared the bedside use of the flow-directed balloon-tipped catheter with insertion of a semirigid electrode catheter in 111 perfusing patients. [14] These researchers found a significantly shorter insertion time (6 minutes and 45 seconds compared with 13 minutes and 30 seconds), a lower incidence of serious arrhythmias (1.5% compared with 20.4%), and a lower incidence of catheter displacement (13.4% compared with 32%) with the balloon-tipped catheter. They concluded that the balloon-tipped catheter was the method of choice for temporary transvenous pacing (Table 15-2).

Kruger and associates retrospectively reviewed the experience of general internists with transvenous pacemaker placement under electrocardiogram (ECG) guidance. [15] They reported a 4% risk of complications and a 14% incidence of electrode malfunction, and these percentages were noted to be similar to those reported by university cardiologists. They concluded that pacemaker placement by primary care clinicians was safe and effective when done under ECG guidance without fluoroscopy.

Indications

The purpose of cardiac pacing is to stimulate effective cardiac depolarization. In most cases the specific indications for cardiac pacing are clear; however, some controversial areas

TABLE 15-1 -- Four-Letter Pacemaker Code

First Letter	Second Letter	Third Letter	Fourth Letter
Chamber-Paced	**Chamber-Sensed**	**Sensing Response**	**Programmability**
A = atrium	A = atrium	T = triggered	P = simple
V = ventricle	V = ventricle	I = inhibited	M = multiprogrammable
D = dual	D = dual	D = dual (A-triggered and V-inhibited)	R = rate adaptive
O = none	O = none	O = none	C = communicating
			O = none

remain. The decision to pace on an emergent basis requires knowledge of the presence or absence of hemodynamic compromise, the etiology of the rhythm disturbance, the status of the atrioventricular (AV) conduction system, and the type of dysrhythmia. In general, the indications can be grouped into those that cause either tachycardias or bradycardias (Table 15-3). Transcutaneous cardiac pacing has become the mainstay of temporary emergent cardiac pacing. It is often used pending placement of the transvenous catheter or as a means to determine whether potentially terminal bradyasystolic rhythms will respond to pacing.

Bradycardias

Sinus node dysfunction.

In a review of 200 initial pacemaker implants at Montefiore Hospital during 1975, 36.5% were used for sinus node dysfunction; 11.3% for sinus arrest; 20.2% for tachybrady (sick sinus) syndrome; and 5% for sinus bradycardia. [16] Patients without myocardial infarction who present with symptomatic sinus node dysfunction should be paced promptly if medical therapy fails. Escher and Furman note that pacing is indicated until the etiology of the dysrhythmia is clarified and stability is ensured. [17]

In the asymptomatic patient, a more intensive cardiac evaluation is required in order to decide whether pacing will be beneficial. This evaluation frequently includes

24-hour Holter monitoring, noting sinus node recovery times, and occasionally coronary care unit monitoring.

TABLE 15-2 -- History of Transvenous Pacing

Date	Investigator	Event
1700	Early investigators	First restimulation studies
1951	Callaghan & Bigelow	First transvenous approach in dogs
1952	Zoll	Transcutaneous cardiac stimulator
1958	Falkmann & Walkins	Implanted pacing wires after surgery
1958	Furman & Robinson	First transvenous pacer in humans
1964	Vogel et al.	Flexible electrocardiographic catheter without fluoroscopy
1965	Kimball & Killip	First bedside transvenous pacing
1966	Goetz et al.	Demand pacemaker developed
1967	Zuckerman et al.	Use of demand pacemaker clinically
1969	Rosenberg et al.	Semifloating pacing catheter
1973	Schnitzler et al.	Balloon-tipped pacers

Sinus bradycardia occurs in an average of 17% of patients with acute myocardial infarction. [18] It occurs more frequently in inferior than in anterior infarction and has a relatively good prognosis when accompanied by a hemodynamically tolerable escape rhythm. However, sinus bradycardia is not a benign rhythm in this situation; it has a mortality rate of 2% with inferior infarction and 9% with anterior infarction. [19] Several mechanisms have been suggested to explain sinus node dysfunction with infarction. Among these, ischemia of the node or its neurologic controls [19] and reflex slowing secondary to pain play dominant roles. [20] Sinus node dysfunction frequently responds to medical therapy but requires prompt pacing if this fails.

Asystolic arrest.

Transvenous pacing in the asystolic or bradyasystolic patient has little value. In one study of 13 patients who had suffered cardiac arrest, capture of the myocardium was noted in 4 patients, but there were no survivors. [21] Transvenous pacing alone may also not be effective in postcountershock pulseless bradyarrhythmias. [22] This failure of pacing has also been demonstrated with transcutaneous pacemakers, suggesting that failure of effective pacing is primarily related to the state of the myocardial tissue. [21] Pacing has no proven value in traumatic cardiac arrest. Other causes of failure to pace include catheter malposition and dislodgment of the pacing wire during closed-chest massage. [21] Cardiac pacing may be used as a "last ditch" effort in bradyasystolic

TABLE 15-3 -- Indications for Cardiac Pacing*

Bradycardias
Without myocardial infarction
Symptomatic sinus node dysfunction (sinus arrest, tachybrady [sick sinus] syndrome, sinus bradycardia)
Second- and third-degree heart block
Atrial fibrillation with symptomatic slow ventricular response
With myocardial infarction
Symptomatic sinus node dysfunction
Mobitz II second- and third-degree heart block
New left bundle-branch block (LBBB); right bundle-branch block (RBBB) with left axis deviation, bifascicular block, or alternating bundle-branch block
Prophylaxis—cardiac catheterization, after open-heart surgery, threatened bradycardia during drug trials for tachydysrhythmias
Malfunction of implanted pacemaker
Tachycardias
Supraventricular dysrhythmias
Ventricular dysrhythmias
Prophylaxis—cardiac catheterization, after open-heart surgery

*Many indications are relative and are dependent upon a variety of symptoms and parameters.

or asystolic patients but is rarely successful and is not considered standard practice. Early pacing is essential when done for this purpose if success is to be achieved [23] (see later in this section).

Atrioventricular block.

Atrioventricular block is the classic indication for pacemaker therapy. In symptomatic patients without myocardial infarction and in the asymptomatic patient with a ventricular rate below 40, pacemaker therapy is indicated. [24]

In patients with acute myocardial infarction, 15% to 19% progress to heart block: approximately 8% develop first-degree block, 5% develop second-degree block, and 6% develop third-degree block. [25] First-degree block progresses to second- or third-degree block 33% of the time, and second-degree block progresses to third-degree block about one third of the time. [26]

Atrioventricular block occurring during anterior infarction is believed to occur because of diffuse ischemia to the septum and infranodal conduction tissue. These patients tend to progress to high-degree block without warning and a pacemaker is often placed prophylactically. Some pacemakers are prophylactically paced on a temporary basis, even in the absence of hemodynamic compromise.

During inferior infarction, early septal ischemia is the exception and typically block develops sequentially from first-degree to Mobitz type I second-degree, then to third-degree atrioventricular (AV) block. These conduction abnormalities frequently result in hemodynamically tolerable escape rhythms because of sparing of the bundle branches. The hemodynamically unstable patient who is unresponsive to medical therapy should be paced promptly. Whether and when the stable patient should be paced is unclear, but placing a transcutaneous pacer is one option that can be tried before placing a transvenous pacing catheter.

One study, which reviewed the indications for temporary and permanent pacemaker insertion in 432 patients with myocardial infarction, concluded that patients with

second- or third-degree AV block should be paced. This recommendation was made due to a higher incidence of sudden death or recurrent high-degree block over the following year that was found in patients who were not continuously paced. [27]

Trauma.

Pacing is not a standard intervention; in traumatic cardiac arrest in selected cases, it may be considered. Several rhythm and conduction disturbances have been documented in the patient with nonpenetrating chest trauma. In these patients, traumatic injury to the specialized conduction system may predispose the patient to life-threatening dysrhythmias and blocks that can be treated by cardiac pacing. [28]

Hypovolemia and hypotension can cause ischemia of conduction tissue and cardiac dysfunction. [29] Marked bradyarrhythmias that persist even after vigorous volume replacement may rarely respond to cardiac pacing in patients with such trauma. [30]

Bundle-Branch Block and Ischemia

Bundle-branch block occurring in acute myocardial infarction is associated with a higher mortality rate and a greater incidence of third-degree heart block than uncomplicated infarction. Atkins and associates noted that 18% of patients with myocardial infarction had bundle-branch block. [31] Of these patients, complete heart block developed in 43% who had right bundle-branch block and left axis deviation, in 17% who had left bundle-branch block, in 19% who had left anterior hemiblock, and in 6% who had no conduction block. The investigators concluded that right bundle-branch block with left axis deviation should be paced prophylactically.

A study by Hindman and colleagues confirmed the natural history of bundle-branch block during myocardial infarction. [32] In their study, the presence or absence of first-degree AV block, the type of bundle-branch block, and the age of the block (new versus old) were used to determine the relative risk of progression to type II second-degree or third-degree block (Table 15-4).

Because of the increased risk, most should consider pacing the following conduction blocks: *new-onset* left bundle-branch block, right bundle-branch block with left axis deviation or other bifascicular block, and alternating bundle-branch block. [32] One authority recommends prophylactic pacing for all new bundle-branch blocks when myocardial infarction is evident. [33]

Whether to place a transvenous pacemaker prophylactically in patients with left bundle-branch block before insertion of a flow-directed pulmonary artery catheter (PAC) remains controversial. Some researchers strongly advocate this procedure because of the risk of transient right bundle-branch block and life-threatening complete heart block in association with the placement of a PAC. [34] One study notes that this risk is low in patients with prior left bundle-branch block but continues to recommend temporary catheter placement for all cases of *new* left bundle-branch block. [35] One solution to this problem is to place a transcutaneous pacemaker before catheterization as an emergency measure should heart block develop. In these cases, a temporary transvenous pacemaker can be placed in a semi-elective manner when needed. [36] In any event, the trend toward decreased PAC use, particularly outside of the critical care setting, makes it unlikely that this will be an issue in the ED. [37]

TABLE 15-4 -- The Influence of Different Variables on Risk of High-Degree Atrioventricular Block in Patients with Bundle-Branch Block During Myocardial Infarction

Patients	**Progressing to High-Degree AVB (%)**
Infarct location	
Anterior	25
Indeterminate	12
Inferior or posterior	20
PR interval	
>0.20 sec	25
=0.20 sec	19
Type of BBB	
LBBB	13
RBBB	14
RBBB + LAFB	27
RBBB + LPFB	29
ABBB	44
Onset of BBB	
Definitely old	13
Possibly new	25
Probably new	26
Definitely new	23

AVB, Atrioventricular block; BBB, bundle-branch block; LBBB, left bundle-branch block; RBBB, right bundle-branch block; LAFB, left anterior fascicular hemiblock; LPFB, left posterior fascicular hemiblock; ABBB, alternating bundle-branch block. *(Reprinted by permission of the American Heart Association from Hindman MC, Wagner GS, JaRo M, et al: The clinical significance of bundle-branch block complicating acute myocardial infarction. 2. Indications for temporary and permanent pacemaker insertion. Circulation 58:690, 1978.)*

Tachycardias

Hemodynamically compromising tachycardias are usually treated by medical means or electrical cardioversion (see Chapter 12). Since 1980, there has been increased interest in pacing therapy for symptomatic tachycardias. Supraventricular dysrhythmias, with the exception of atrial fibrillation, respond well to atrial pacing. By "overdrive" pacing the atria at rates 10 to 20 beats/min faster than the underlying rhythm, the atria become entrained, and when the rate is slowed, the rhythm frequently returns to normal sinus. A similar procedure is done for ventricular dysrhythmias. [38] Overdrive pacing is especially useful for recurrent prolonged Q-T interval arrhythmias such as those seen with quinidine toxicity or torsades de pointes. [39] While an attractive thought, there is no reported experience with these techniques in the ED. Transvenous pacing may also be useful in patients with digitalis-induced dysrhythmias in whom direct current (DC) cardioversion may be dangerous or in patients in whom there is further concern about myocardial depression with drugs. [40]

Cardiac Pacing in Drug-Induced Dysrhythmias

Significant dysrhythmias can occur from excessive therapeutic medication (often in combination therapy) and from overdose of cardioactive medications. Because these drugs have direct effects on the myocardial pacemaker and conduction system cells, cardiac pacing is usually of little therapeutic value. Both bradycardias and tachycardias may result. Tachycardic rhythms from amphetamines, cocaine, anticholinergics, cyclic antidepressants, theophylline, and others do not benefit from cardiac pacing. A drug-induced torsades de pointes may theoretically be overdriven by pacing, but data on this technique are lacking. Any drug that affects the central nervous system (opiates, sedative-hypnotics, clonidine, and others) may produce bradycardia. Rare causes of toxin-induced bradycardia include organophosphate

poisoning, various cholinergic drugs, ciguatera poisoning, and, rarely, plant toxins. Cardiac pacing is not used for bradycardias from these sources; rather, the underlying CNS depression is addressed.

Severe bradycardia and heart block often accompany overdose of digitalis preparations, beta adrenergic blockers, and calcium channel blockers. While intuitively attractive, cardiac pacing is rarely successful in serious toxin-induced bradycardias. [41] [42] [43] [44] In beta-blocker overdose, pacing may increase heart rate but rarely benefits blood pressure or cardiac output. Worsening of the blood pressure may be seen from loss of atrial contractions with ventricular pacing. Likewise, calcium channel blocker overdose and digitalis-induced bradycardia and heart block rarely benefit from cardiac pacing. Pharmacologic interventions, such as digoxin-specific Fab, glucagon, calcium, and inotropic medications, and vasopressors remain the mainstay in the treatment of drug-induced dysrhythmias. Given the lack of success of pacing, possible downsides, and the greater effectiveness of specific antidotes, it is not standard to routinely attempt cardiac pacing in the setting of drug overdose. However, as a last ditch effort, cardiac pacing can be supported.

Contraindications

There are no absolute contraindications to transvenous cardiac pacing; however, the severely hypothermic bradycardic patient can often be managed without pacing. Severe hypothermia will occasionally result in ventricular fibrillation when pacing is attempted. Because ventricular fibrillation under these conditions is difficult to convert, caution is advised when considering pacing the severely hypothermic and bradycardic patient. Rapid warming is often recommended first, followed by pacing if the patient's condition does not improve.

Equipment

Several items are required to insert a transvenous pacemaker adequately. Like most special procedures, a prearranged tray is convenient. The usual components required to insert a transvenous cardiac pacemaker are listed in Table 15-5 .

Pacing Generator

Many different pacing generators are available, but in general, they all have the same basic features. The on/off switch frequently will have a locking feature to prevent the generator from inadvertently being switched off. An amperage knob allows the operator to control the amount of electrical current delivered to the myocardium, usually 0.1 to 20 mA. The pacing control mode is the gain control for the sensing function of the generator. By increasing the sensitivity, one can convert the unit from a fixed rate (asynchronous mode) to a demand (synchronous mode) pacemaker. In the fixed-rate mode, the unit fires despite the underlying intrinsic rhythm; the unit does not sense any intrinsic electrical activity. In the full-demand mode, however, the pacemaker senses the underlying ventricular depolarizations, and the unit does not fire as long as the patient's ventricular rate is equal to or faster than the set rate of the pacing generator. A sensing indicator meter and rate control knob are also present. An example of a pacing generator is shown in Figure 15-1 .

Pacing Catheters and Electrodes

Several sizes and brands of pacing catheters are available. In general, most range from 3 Fr to 5 Fr in size and are approximately 100 cm in length. Along the catheter surface are lines that are marked at approximately 10-cm intervals; these can be used to estimate catheter position during insertion. Two

TABLE 15-5 -- Suggested Transvenous Cardiac Pacemaker Equipment

Pacemaker Tray
10-mL syringe
1% lidocaine
Alcohol wipes
Povidone-iodine (Betadine)
Several gauze pads
4 sterile drapes
No. 11 scalpel blade
0.9 normal saline—2 ampules
Sterile gloves
Needle holder
Two 22-ga needles
Scissors (suture)
Two 4-0 silk sutures on needles
Sterile basin
Electrical Hardware
Insulated connecting wire with alligator clamps at each end (or a male-to-male adapter)
Spare 9-V battery
Medtronic pacing unit no. 5375
3 Fr Balectrode Pacing Kit (catalog no. 11—KBE1)
12-lead electrocardiographic machine (well grounded)

Figure 15-1 Pacemaker energy source controls and connections.

basic types of pacing catheters are currently in use: (1) the flexible semifloating or floating catheter and (2) the rigid fixed-position catheter. [39]

The flexible catheters are more advantageous than the rigid catheter by virtue of the flexible catheter's ability to be inserted in low flow states, as well as in their decreased tendency to perforate the ventricle. For emergency pacing, the 4 Fr semifloating bipolar electrode with or without the balloon tip is used most frequently (Fig. 15-2). The balloon holds approximately 1.5 mL of air, and some of the injection ports have a locking lever to secure balloon expansion. Before insertion, the balloon is checked for air leakage by inflating it and immersing it in sterile water. The presence of an air leak is noted by a stream of bubbles arising at the surface of

the water. An inflated balloon helps the catheter "float" into the heart in low flow states but is obviously not advantageous in the cardiac arrest situation.

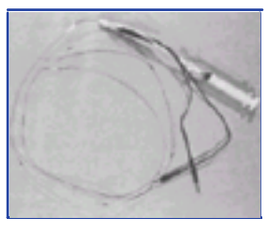

Figure 15-2 Balloon-tipped pacing catheter.

For all practical purposes, temporary transvenous pacing is accomplished with a bipolar pacing catheter. The terms *unipolar* and *bipolar* refer to the number of electrodes in contact with that portion of the heart that is to be stimulated. All pacemaker systems must have both a positive (anode) and a negative (cathode) electrode; hence, *all stimulation is bipolar*. In the typical bipolar catheter used for temporary transvenous pacing, the cathode (stimulating electrode) is at the tip of the pacing catheter. The anode is located 1 to 2 cm proximal to the tip, and the two electrodes may be separated by a balloon or an insulated wire. The distinction between the unipolar and bipolar pacing catheter is that a bipolar catheter has both electrodes in a relative close proximity on the catheter, and both may contact the endocardium. In the bipolar catheter, the electrodes are usually platinum rings that encircle the pacing catheter. When properly positioned, both electrodes will be within the right ventricle so that a field of electrical excitation is set up between the electrodes. With the bipolar catheter, the cathode does not need to be in direct contact with the endocardium for pacing to occur, although it is preferable to have direct contact.

A unipolar system is also effective but is used infrequently for temporary transvenous pacing. In a unipolar system, the cathode is at the tip of the pacing catheter, and the anode is located in one to three places: (1) in the pacing generator itself, (2) more proximal on the catheter (outside the ventricle), or (3) on the patient's chest. A bipolar catheter system may be converted to a unipolar system by simply disconnecting the positive proximal connection of the bipolar catheter from the pacing generator and running a new wire from the positive (pacing generator) terminal to the patient's chest wall. Such a conversion may be required in the unlikely event of failure of one lead of the bipolar system.

Theoretically, the field of electrical stimulation of a pacing catheter is equal to the distance between the electrodes. If the field of excitation is not close enough to the myocardium, depolarization will not occur. When a catheter is passed blindly in an emergency, it seems advantageous to ensure the best chance of capture by separating the electrodes by more than the standard 1 to 2 cm. A pacing catheter that uses this configuration (Davison pacing lead, Electro-Catheter Corporation, Rahway, NJ) is a hybrid of the standard bipolar and unipolar catheters. This catheter has the cathode at the tip, but the anode is situated 19 cm proximal to the tip. This configuration allows pacing with a very wide field of excitation. Pacing has been reported to occur with this catheter when the catheter is placed anywhere within the thoracic venous system. The catheter is a hybrid because both electrodes are present on the same catheter (bipolar), but both electrodes will not be positioned in the same cardiac chamber (unipolar).

ECG Machine

An ECG machine can be used to record the heart's inherent electrical activity during pacer insertion and to aid in localization of the catheter tip without fluoroscopy. The ECG machine must be well grounded to prevent leakage of alternating current, which can cause ventricular fibrillation. Such leakage should be suspected if interference of 50 to 60 cycles per second (Hz) is noted on the ECG.

The ECG machine should be placed in such a manner as to allow easy visibility of the rhythm during insertion. One method is to place the machine on the same side of the patient as the operator at the level of the midthorax (Fig. 15-3). Note

Figure 15-3 Position of an electrocardiogram device during femoral vein insertion of a pacemaker catheter. Operator is on right side of patient facing cephalad.

that the operator stands at the head of the patient during internal jugular or subclavian vein passage of the catheter and at the midabdomen for femoral or brachiocephalic vein insertion.

Introducer Sheath

An introducer set or sheath is required for venous access (see Chapter 22). Some pacing catheters are prepackaged with the appropriate equipment, whereas others require a separate set. The introducer set is used to enhance passage of the pacing catheter through the skin, subcutaneous tissue, and vessel wall. To allow passage of the pacing catheter, the sheath must be one size larger than the pacing catheter. A makeshift sheath can be made with an appropriate-sized intravenous (IV) catheter. For the 3 Fr balloon-tipped catheter, a 14-gauge, 1.5-to 2-inch IV catheter is suitable. The 4 Fr balloon-tipped catheter will also fit through a 14-gauge catheter or needle.

A balloon-directed pulmonary artery catheter (Paceport pacing system, American Edwards Laboratories, American

TABLE 15-6 -- Advantages and Disadvantages of Pacemaker Placement Sites

Venous Channels	Advantages	Disadvantages
Brachial	Very safe route	Often requires cutdown
	Vessel easily accessible, either by cutdown or percutaneous approach	Easily displaced and poor patient mobility
		Not reusable if cutdown technique is performed
		Catheter is more difficult to advance than with central or larger vessels
Subclavian	Direct access to right heart (especially via left subclavian)	Pneumothorax and other intrathoracic trauma are possible
	Rapid insertion time	
	Reusable	
	Good patient mobility	
Femoral	Direct access to right heart	Increased incidence of thrombophlebitis
	Rapid insertion time	Can be dislodged by leg movement and poor patient mobility
	Reusable	
		Infection
Internal jugular	Direct access to right heart (especially via right internal jugular)	Possible carotid artery puncture
		Dislodgment with movement of the head
	Rapid insertion time	Thrombophlebitis

Reusable	

Hospital Supply Corporation) has been developed. It has a separate lumen that allows the passage of a transvenous pacing catheter. [45] This catheter is 7.5 Fr and has an opening 19 cm from the catheter tip that allows passage of the 2.4 Fr pacing wire. This stainless steel wire is Teflon-coated for easy passage and has a flexible tip. Combination pulmonary artery or pacemaker catheters are also available but are not widely used in the emergency setting.

Overall, the key to success with this procedure is preparation. It is imperative that one examine all the components of the tray before starting the procedure and ensure that all wires, sheaths, dilators, and syringes fit as expected.

Procedure

Patient Preparation

Patient instruction is an extremely important aspect of any procedure. Frequently there is not enough time to give patients a detailed explanation. Nor to obtain written informed consent. Nonetheless, sufficient information should be provided so that the patient feels at ease. It is always prudent to obtain and document informed consent from the patient if possible prior to any invasive procedure, or to document that the circumstances did not allow informed consent. Patients should be assured that they will feel no discomfort after the venipuncture site has been anesthetized and that they will feel better when the catheter is in place and is functional. Continued reassurance is required during the procedure because patients are usually facing away from the operator and because their faces are often covered, they may be unsure of what is occurring. When appropriate sedation anxietolysis should be considered.

All operators should wear surgical masks, caps, gloves, and gowns to decrease the risk of infection before catheter placement. This aseptic precaution should also be explained to the patient.

Site Selection

The four venous channels that provide an easy access to the right ventricle are the brachial, subclavian, femoral, and internal jugular veins (Table 15-6). The route selected is often one of

personal or institutional preference. The right internal jugular and the left subclavian veins have the straightest anatomic pathway to the right ventricle and are generally preferred for temporary transvenous pacing. In some centers a particular site is preferred for permanent transvenous pacemaker placement, and, if possible, this site should be avoided for temporary placement.

The subclavian vein can be accessed by both an infraclavicular and a supraclavicular approach; the infraclavicular approach is most commonly reported for all temporary transvenous pacemaker insertions. This route is preferred because of its easy accessibility, close proximity to the heart, and ease in catheter maintenance and stability. The supraclavicular approach has been described in the literature for several years and has gained popularity among some clinicians. [46] [47] The *left* subclavian vein is preferred because of the less acute angle traversed when compared with the right-sided approach. However, a recent study reported safe and efficient emergency transvenous pacing via a right-sided supraclavicular approach. [48]

Some clinicians believe the internal jugular approach is as easy as and safer than subclavian catheterization. [48] Either site is acceptable. The right internal jugular vein is preferred because of the direct line to the superior vena cava. Problems with this approach include dislodgment of the pacemaker with movement of the head, carotid artery puncture, and thrombophlebitis (see Chapter 22).

During cardiopulmonary resuscitation, the use of the right internal jugular vein and the left subclavian veins for pacemaker insertion have been demonstrated to result in the highest rates of proper placement in the right ventricle. [49] The right internal jugular vein is the more direct route of the two and may be the most appropriate site. Because of the extremely low flow state during cardiopulmonary resuscitation, a larger (5 Fr), semirigid catheter may be a more appropriate choice than the 3 to 4 Fr catheters commonly used.

Femoral veins, like neck veins, are reusable and easily catheterized. Problems include easy dislodgment, infection, and increased risk of thrombophlebitis. [50] [51]

Brachial vein catheterization is easy to perform but results in a higher incidence of infection and vessel thrombosis. [52] In addition, the catheter is easily dislodged with arm motion. This approach is seldom used in the emergency setting.

Skin Preparation and Venous Access

The skin over the venipuncture site is cleaned twice with an antiseptic solution such as povidone-iodine and isopropyl alcohol. A wide area is prepared because of the tendency for guidewires and catheters to spring from the hands of the unsuspecting operator. Preparation of the skin is shown in Chapter 22 . Similarly, wide draping is carried out in the standard manner to maintain a sterile field and to allow clear visibility of the venipuncture site.

The infraclavicular approach is used in this chapter to illustrate venous access, although the mechanics are generally the same for other vascular approaches. The reader is referred to Chapter 22 for the specific techniques of venous access.

Occasionally a patient who already has a central venous line in place requires the emergent placement of a pacing catheter. An existing central venous pressure (CVP) line can be used to place the pacing catheter if the catheter lumen is large enough to accept a guidewire. The CVP line should be withdrawn 1 to 2 in. to expose an area of sterile tubing. The tubing is transected through a sterile area while being held

Figure 15-4 Insertion of the pacing catheter through the introducer sheath.

firmly at the skin level. A guidewire can then be passed through the tubing, and the tubing can be withdrawn, leaving only the wire in the vein. The guidewire and the tubing should never be released because embolization may result. An introducer unit can then be passed over the guidewire, as is done in the Seldinger technique (see Chapter 22), and the pacing catheter can be placed (Fig. 15-4).

Bedside ultrasound (US) can be useful as an aid to securing central venous access and its use in the setting of emergency transvenous pacing has been reported (see Chapter 69). [53] [54]

Pacemaker Placement

Electrocardiographic guidance.

The patient should be connected to the limb leads of an ECG machine, and the indicator should be turned to record the chest (V) lead.

With newer ECG machines, the pacemaker may be attached to any of the V leads (usually V_1 or V_5) that are displayed during rhythm monitoring. As the tracing on the ECG machine is slightly delayed with the newer devices, advancement of the catheter after initial insertion must be carefully evaluated. The pacing wire should be

inserted about 10 to 12 cm into the selected vein. The *distal* terminal of the pacing catheter (the cathode or lead marked negative, "-") must be connected to the V lead of the ECG machine by a male-to-male connector or by an insulated wire with an alligator clip on each end (Fig. 15-5). The pacing catheter is thus an exploring electrode that creates a unipolar electrode for intracardiac ECG recording. The ECG recorded from the

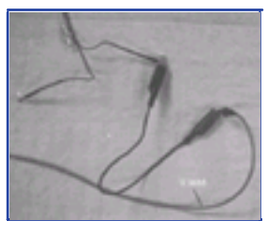

Figure 15-5 Using alligator clips to connect the *negative* lead of the pacemaker catheter to the V lead of an electrocardiographic machine.

electrode tip *localizes the position of the tip of the pacing electrode*. If a balloon-tipped catheter is used, the balloon is inflated with air *after* the catheter enters the superior vena cava.

The pacing catheter should be advanced both quickly and smoothly. The V lead should be monitored, and the P wave and QRS complex should be observed to ascertain the location of the pacing catheter tip. The use of an ECG to guide the placement of a pacing catheter is based on two concepts. First, the complex will vary in size depending on which chamber is entered. For example, when the tip of the pacing catheter is in the atrium, one will see large P waves, often larger than the corresponding QRS complex. Second, the sum of the electrical forces will be negative if the depolarization is moving away from the catheter tip and positive if the depolarization is moving toward the catheter tip. Therefore, if the catheter tip is *above* the atrium, both the P wave and the QRS complex will be negative (i.e., the electrical forces of a normally

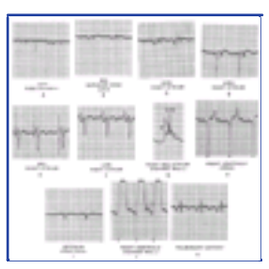

Figure 15-6 *A–K,* Intracardiac electrocardiography: Electrical signals of atrial and ventricular depolarization and repolarization from different vascular and intracardiac locations (see text). *(A–F and H–K from Bing OH, McDowell JW, Hantman J, et al: Pacemaker placement by electrocardiographic monitoring. N Engl J Med 287:651, 1972. G from Goldberger E: Treatment of Cardiac Emergencies, 3rd ed. St. Louis, CV Mosby, 1982, p 252.)*

beating heart will be moving away from the catheter tip). As the tip progresses inferiorly in the atrium, the P wave will become isoelectric (biphasic) and will eventually become positive as the wave of atrial depolarization advances toward the catheter tip. The ECG resembles an aVR lead initially when in the left subclavian vein (Fig. 15-6*A*) or midsuperior vena cava (Fig. 15-6*B*). At the high right atrium, both the P wave and QRS complex are negative; the P wave is larger than the QRS complex and is deeply inverted (Fig. 15-6*C* and *D*). As the center of the atrium is approached, the P wave becomes large and biphasic (Fig. 15-6*E*). As the catheter approaches the lower atrium (Fig. 15-6*F*), the P wave becomes smaller and upright. The QRS complex is fairly normal. When striking the right atrial wall, an injury pattern with a P-Ta segment is seen (Fig. 15-6*G*). As the electrode passes through the tricuspid valve, the P wave becomes smaller, and the QRS complex becomes larger (Fig. 15-6*H*).

Placement in the inferior vena cava may be recognized by a change in the morphology of the P wave and a decrease in the amplitude of both the P wave and the QRS complex (Fig. 15-6*I*).

Once the pacing catheter is in the desired position, the balloon is deflated by unlocking the port and removing the syringe. One should avoid drawing back on the syringe, because this may cause balloon rupture. If the operator suspects that the balloon may be ruptured, it should not be inflated. Instead, the pacing catheter should be withdrawn and the balloon checked for leaks. If a leak is found, a new pacing catheter should be used.

After successful placement of the catheter within the right ventricle, the tip should be advanced until contact is made with the endocardial wall. When this occurs, the QRS segment will show ST segment elevation (Fig. 15-6*J*). Ideally, the tip of the catheter should be lodged in the trabeculae at the apex of the right ventricle; however, pacing may be successful if the catheter is in various other positions within the ventricle or outflow tract.

If the pacer enters the pulmonary artery outflow tract, the P wave again becomes negative, and the QRS amplitude diminishes (Fig. 15-6*K*). If the catheter is in the pulmonary artery, the pacing catheter should be withdrawn into the right ventricle and readvanced. Sometimes a clockwise or counter-clockwise twist of the catheter will redirect its path in a more favorable direction. If catheter-induced ectopy develops, the catheter should be slightly withdrawn until the ectopy stops; then it should be readvanced. Occasionally an antidysrhythmic drug such as lidocaine may be needed to desensitize the myocardium. Once ventricular endocardial contact is made, the catheter is disconnected from the ECG machine. The proximal positive and negative leads are connected to their respective terminals on the pacing generator. The pacing generator is then set to a rate of 80 beats/min or 10 beats/min faster than the underlying ventricular rhythm, whichever is higher. The full-demand mode is selected, with an output of about 5 mA. The pacing generator is then turned on. If complete capture does not occur or if it is intermittent, the pacer will need to be repositioned. When proper capture occurs, the pacer is tested for optimal positioning. This is done by testing the thresholds for sensing and pacing and with chest radiographs, physical examination, and ECG.

Catheter placement without an electrocardiograph.

Occasionally it is necessary to use a transvenous pacemaker in an emergency setting when a well-grounded ECG machine is not available. Blind insertion of the transvenous pacing catheter is a safe and effective alternative to placement with ECG guidance. In this technique, the pacing catheter is placed 10 to 12 cm into the venous port and is connected to the pacing generator as noted previously. The pacing rate is selected at *twice* the intrinsic heart rate, and the output is set at an amperage that is too low to capture the ventricle, usually less than 0.2 mA. The unit is then turned on to first *sense* but *not* to pace. On entering the ventricle, the pacer will sense on every other beat. The balloon can then be deflated, the amperage can be increased to 4 to 5 mA for initiating pacing, and the pacemaker can be advanced to capture the ventricle. If this does not occur within an additional 10 cm, the pacing catheter should be withdrawn to its original position and then advanced again. As with ECG placement, proper positioning must be ensured.

In elective, nonemergent cases fluoroscopy is a valuable tool in the placement of transvenous pacemakers. Its use depends on the operator's preference, the patient's condition, and its availability. Generally transvenous pacemakers are not inserted under fluoroscopy without ECG monitoring because of the high incidence of ventricular dysrhythmias. [39]

If the cardiac output is too low to "float" a pacing catheter or if the patient is in extremis, there may not be enough time to advance a pacing catheter using the previously described techniques. Such a situation would be asystole or complete heart block with malignant ventricular escape rhythms (although one can make a case for transcutaneous pacing in such conditions). In such emergent situations, the pacing catheter is connected to the energy source, the output is turned to the maximum amperage, and the asynchronous mode is selected. The catheter is then blindly advanced in the hope that it will enter the right ventricle and that pacing will be accomplished. The pacing catheter is rotated, advanced, withdrawn, or otherwise manipulated according to the clinical response. The right internal jugular approach is the most practical access route in this situation. In such instances, there is the theoretic advantage of using the previously described Davison catheter, because one is interested in rapid capture only until the patient is stabilized.

Ultrasound guidance.

As bedside US has become more widely available in the ED, new uses have been discovered. One promising technique involves using US to assist with the placement of emergency transvenous pacing catheters.[55] [56] The advantages of US over fluoroscopy are its safety and ready availability. Further experience will be necessary to confirm its utility.

Dual Chamber Pacing

Synchronous pacing of the atria and ventricle through a *dual-chamber pacing catheter* that senses and paces both atria and ventricles (DDD mode) results in more physiologic pacing with further improvement in cardiac output. It also allows for variations in the atrial rate and improved coordination of atrial and ventricular contractions. This catheter is placed in the same manner as the ventricle-only pacing catheter (VVI mode) but requires greater skill and experience because of the need to properly place the catheter in both the atria and ventricle. When attaching the catheter to the pacing generator, it is important to ensure that the atrial catheter is connected to the atrial electrode and the ventricular catheter is connected to the ventricular electrode. There are devices that have dual-chamber pacemaking capability and can operate in a variety of modes, including DDD mode. However, it is seldom necessary to employ this modality in the ED.

Testing Threshold

The threshold is the minimum current necessary to obtain capture. Ideally, this is less than 1.0 mA, and usually it is between 0.3 and 0.7 mA. If the threshold is in this ideal range, good contact with the endocardium can be presumed.

To determine the threshold, the pacing generator should be placed in the full-demand mode at 5 mA with a rate of approximately 80 beats/min. The amperage (output) should then be reduced slowly until capture is lost. This current is the threshold. This maneuver should be carried out two or three times to ensure that this value is consistent; the amperage should then be increased to 2.5 times the threshold to ensure consistency of capture (usually between 2 and 3 mA).

If one reduces the output to below the threshold and then slowly increases it, there may be a difference in the point at which capture returns. This difference is called *hysteresis* and

represents the time interval between sensing and pacemaker firing. If the difference in capture current is greater than 20%, the pacing catheter should be repositioned, because serious dysrhythmias may result if the pacemaker fires during the vulnerable period of repolarization. [39] [57]

Testing Sensing

The sensing function should be tested in patients who have underlying rhythms. The pacemaker system is again set in full-demand mode with complete capture, and the rate is decreased until it is suppressed by the patient's intrinsic rhythm. This is done several times to ensure accuracy of the sensing function.

In bipolar systems, another method of evaluating the sensing mode is to take a unipolar ECG from each end of the bipolar lead on a chest lead at one-fourth standardization to permit observation of the entire complex. [39] The voltage of the QRS complex is multiplied by 4 and, if adequate, should be greater than the sensing threshold by greater than 1 mV (Fig. 15-7). Another method is to set the ECG machine on lead I and connect the wires from the proximal electrode to the right arm lead and the left arm lead to the distal electrode. A lead I is created, which, when the QRS voltage is multiplied by 4, should also be at least 1 mV more than the sensing threshold.

Securing and Final Assessment

After the pacemaker's position has been tested for electrical accuracy, the introducer sheath should be withdrawn (Fig. 15-8) and the catheter secured to the skin with suture (e.g., 4-0 nylon or silk). A fastening suture should be sewn to the skin and the catheter tied securely in place. The excess pacing catheter should be coiled and secured in a sterile manner along with the introducer. A large sterile dressing should be applied. Pacemaker function should again be assessed, and a chest film should be taken to ensure proper positioning. Ideal

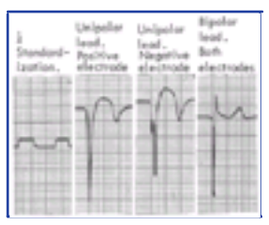

Figure 15-7 Testing unipolar sensing with a bipolar system. *(From Goldberger E: Treatment of Cardiac Emergencies, 3rd ed. St. Louis, CV Mosby, 1982.)*

positioning of the pacing catheter is at the apex of the right ventricle (Fig. 15-9).

A 12-lead ECG should be obtained after transvenous pacemaker placement. If the catheter is within the right ventricle, a left bundle-branch pattern with left axis deviation should be evident in paced beats (Fig. 15-10). If a right bundle-branch block pattern is noted, coronary sinus placement or left ventricular pacing due to septal penetration should be suspected.

With a properly functioning ventricular pacemaker, large cannon waves will be noted on inspection of the venous pulsations at the neck. This is caused by the atria contracting against a closed tricuspid valve. On auscultation of the heart,

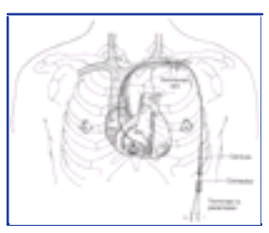

Figure 15-8 Pulling back the introducer sheath (cannula). RA, right atrium; RV, right ventricle; LV, left ventricle.

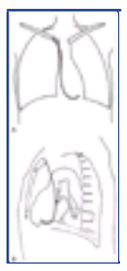

Figure 15-9 Normal pacemaker position on posteroanterior *(A)* and lateral *(B)* chest films. *(From Goldberger E: Treatment of Cardiac Emergencies, 3rd ed. St. Louis, CV Mosby, 1982.)*

a slight murmur secondary to tricuspid insufficiency from the catheter interfering with the tricuspid valve apparatus may be evident. [58] A clicking sound, heard best during expiration following each pacemaker impulse, may also be noted here and is believed to represent either intercostal or diaphragmatic muscular contractions caused by the pacemaker.[59] Note that this can also be a sign of cardiac perforation. [60] On auscultation of the second heart sound, paradoxical splitting may be noted. This represents a delay in closure of the aortic valve because of delayed left ventricular depolarization.

As in any procedure, the patient should then be assessed for improvement in his or her clinical status. An evaluation of vital signs, mentation, improvement in congestive symptoms, and urinary output must be noted. In addition, complications secondary to the procedure should be sought and treated as needed.

Complications

The complications of emergency transvenous cardiac pacing are numerous and represent a compendium of those related to central venous catheterization, those related to right-sided heart catheterization, and those unique to the pacing catheter itself (Table 15-7).

Problems Related to Central Venous Catheterization

Inadvertent arterial puncture is a well-known complication of the percutaneous approach to the venous system. [61] This problem is usually recognized quickly because of the rapid return of arterial blood. Firm compression over the puncture site will almost always result in hemostasis in 5 minutes or less.

Venous thrombosis and thrombophlebitis are also potential problems with central venous catheterization. Thrombophlebitis, which occurs early after insertion, is said to be a rare complication. Some experts believe that it can be managed without removal of the catheter or anticoagulation. [52] When thrombophlebitis occurs in long-term implanted pacemakers, removal and anticoagulation may be required. In one series, only 0.1% of permanent pacemakers were in this category, and in a small percentage of these, occult malignancies were found.[52] Complete thrombosis of the innominate vein is also a rare problem, with pulmonary embolism an even more uncommon event.[62] Femoral vein thrombosis, however, appears to be a much more common event associated with femoral vein catheterization. [50] [63] Studies using noninvasive techniques have shown a 37% incidence of femoral vein thrombosis, with 55% of these having ventilation-perfusion scan evidence of pulmonary embolism.[63] Thrombosis in the right atrium may also occur and has been treated successfully with thrombolytic agents. [64]

Pneumothorax is consistently a problem with the various approaches to the veins at the base of the neck. The decision to place a chest tube in patients with this complication depends on the size of the pneumothorax and the clinical status of the patient (see Chapter 10). In addition, laceration of the subclavian vein with hemothorax,[65] thoracic duct laceration with chylothorax, air embolism, wound infections, pneumomediastinum, hydromediastinum, [66] hemomediastinum, phrenic nerve injury,[67] fracture of the guidewire with embolization, [68] [69] and catheter or guidewire knotting[70] [71] are all potential complications. [39] [65]

Complications of Right-Sided Heart Catheterization

A common complication of the pacing catheter is dysrhythmia, with premature ventricular contractions being a common occurrence. One study noted a 1.5% incidence of serious dysrhythmias with a balloon-tipped catheter using ECG guidance, compared to a 32% incidence with the semirigid catheter using fluoroscopic guidance, suggesting that the balloon catheter was the preferred type of catheter. [14] Another study noted a 6% incidence of ventricular tachycardia during insertion. [50] The ischemic heart is more prone to dysrhythmias than the nonischemic heart. [72] The therapy for catheter-induced ectopy involves withdrawing the catheter from the ventricle. This usually stops the ectopy; however, if after repeated attempts it is found that the catheter cannot be passed without ectopy, myocardial suppressant therapy may be used to desensitize the myocardium.

Misplacement of the pacing catheter has been well studied. Passage of the catheter into the pulmonary artery can be diagnosed cardiographically by observing the return of an inverted P wave and the decrease in the voltage of the QRS complex. Misplacement in the coronary sinus may occur and should be suspected in the patient in whom a paced right bundle-branch

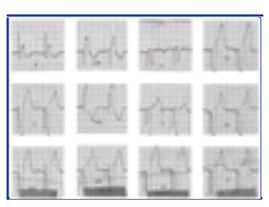

Figure 15-10 Electrocardiogram pattern of right venticular pacemaker.

pattern on the ECG is seen with right ventricular pacing (Fig. 15-11). Rarely, a right bundle-branch pattern can be seen with a normal right ventricular position; therefore, all right bundle-branch patterns do not represent coronary sinus pacing. [73] Further evidence for coronary sinus location can be obtained by viewing the lateral chest film. Normally, the catheter tip should point anteriorly toward the apex of the heart; however, with coronary sinus placement, the catheter tip is displaced posteriorly and several centimeters away from the sternum (Fig. 15-12). Other potential forms of misplacement include left ventricular pacing through an atrial septal defect or a ventricular septal defect, septal puncture, extraluminal insertion, and arterial insertions. [74]

Perforation of the ventricle is a well-described complication that can result in loss of capture, [75] hemopericardium, and tamponade.[76] [77] Reported symptoms and signs of this problem include chest pain, pericardial friction rub, and diaphragmatic or chest wall muscular pacing. [78] At least one case of a post-pericardiotomy-like syndrome and two cases of endocardial friction rub have been reported without perforation. [79] [80]

Pericardial perforation is suggested radiographically when the pacing catheter is outside or abuts the cardiac silhouette and is not in proper position within the right ventricular cavity (Fig. 15-13).[81] ECG clues include a change in the QRS and T wave axis or a failure to properly sense. In suspected cases, a two-dimensional echocardiogram usually demonstrates the catheter's extracardiac position. Uncomplicated perforation can usually be treated by simply pulling back the catheter and repositioning it in the right ventricle.

During the insertion of a temporary pacing catheter when a nonfunctioning permanent catheter is in place, there is a small risk of entanglement or knotting. This potential also exists with other central lines and Swan-Ganz catheters. Frequently these lines can be untangled under fluoroscopy using specialized catheters.

Local and systemic infections,[52] balloon rupture, pulmonary infarction, [82] phrenic nerve pacing,[83] and rupture of the chordae tendineae are also potential complications.[82]

Complications of the Pacing Electrode

The complications related to the pacing electrode can be separated into three groups: mechanical, organic, and electrical.

Mechanical failures include displacement, fracture of the catheter, and loose leads. Displacement can result in intermittent or complete loss of capture or improper sensing, malignant dysrhythmias, diaphragmatic pacing, or perforation. Displacement should be suspected with changes in amplitude, with vector changes greater

than 90 degrees, or with a change in threshold.[84] Frequently catheter fractures may be detected by a careful review of the chest film or may be suspected because of a change in the sensing threshold. As with displacement, intermittent or complete loss of capture may result.

Organic causes of pacemaker failure result in changes in the threshold or sensing function.[85] Progressive inflammation, fibrosis, and thrombosis may result in more than a doubling of the original threshold.[86] This may occur in 3 to 4 weeks and should be expected in prolonged temporary pacemakers. Physiologic and pharmacologic factors that affect the threshold have been studied. Sleeping, eating a heavy meal, lowered aldosterone concentration, potassium infusions,[87] and myxedema[88] all increase the threshold by raising the resting membrane potential. The threshold for cardiac pacing tends to decrease with exercise, sympathetic amines, glucocorticoids, and toxic levels of procainamide.[89]

TABLE 15-7 -- Complications of Transvenous Cardiac Pacing

Year	Author	No. of Patients	Catheter	Route	Result
1969	Rosenberg et al[1]	111	Flexon steelwire vs unipolar semifloating (ECG)	96 Subclavian	12 inconsistent pacing, 3 local infection, 2 pneumothorax, 1 subclavian artery puncture; 16% complication rate
				5 Basilic	
				1 External jugular	
1973	Schnitzler et al[13]	17	3 Fr bipolar semifloating balloon (ECG)	Antecubital vein	2 PVCs, stable pacing, no thrombophlebitis
1973	Weinstein et al[51]	100	6 Fr bipolar (fluoroscopy)	Femoral	2 ventricular tachycardia, 2 perforations, 2 required repositionings, 1 questionable thrombophlebitis and pulmonary embolism, 1 local infection
1973	Lumia & Rios*	142 insertions in 113 patients	Bipolar (fluoroscopy)	61 Brachial	12 ventricular tachycardia and fibrillation in 9 patients, 3 perforations in 2 patients; local hematoma, abscess, and bleeding in 30%; 16.9% complication rate
				81 Femoral	
1980	Pandian et al.†	20	5 Fr bipolar (fluoroscopy)	Femoral	25% deep venous thrombosis
1980	Nolewajka et al[63]	29	6 Fr cordis (fluoroscopy)	Femoral	34% venous thrombosis by venogram with 60% of these with pulmonary embolism by VQ scan
1981	Lang et al[14]	111	Balloon, semifloating vs semirigid	Subclavian	Serious dysrhythmia: 1.5% balloon-tipped, 20.4% semirigid
					Catheter displacement: 13.6% ± 4.4 days balloon-tipped; 32% ± 1.9 day semirigid
1982	Austin et al[50]	113 insertions in 100 patients	4–7 Fr bipolar (fluoroscopy)	Brachial	Failure to sense or pace in 37%; repositioning in 37% of brachial insertions; repositioning in 9% of femoral insertions; fever, sepsis, local infection only in femoral insertions; 20% complication rate
				Femoral	

ECG, electrocardiogram; PVC, premature ventricular contraction; VQ, ventilation-perfusion.

*Lumia FJ, Rios JC: Temporary transvenous pacemaker therapy: An analysis of complications. Chest 64:604, 1973.
†Pandian NG, Kosowsky BD, Gurewich V: Transfemoral temporary pacing and deep venous thrombosis. Am Heart J 100:847, 1980.

In some patients the atrial contribution to ventricular filling is extremely important. Transvenous ventricular pacing results in loss of the atrial kick and ultimately a decrease in left ventricular stroke volume. This phenomenon is called *postpacer syndrome* and occasionally is severe enough to preclude the use of a pacemaker.[90] A bichamber sequential pacemaker that stimulates the atria and ventricles in sequential fashion is a viable alternative for patients unable to tolerate the loss of the atrial kick.[91]

Electrical problems with pacing include pacemaker generator failure, dysrhythmias, and outside interference. Electrical interference is of occasional importance during aeromedical transport.[92] Usually converting the unit to a fixed mode will permit continued pacing. Although ventricular tachycardia and ventricular fibrillation have been reported to result from pacemakers, these dysrhythmias are rare. Because of this, patients who present with such dysrhythmias should be evaluated for a nonpacemaker etiology.[93] Direct current cardioversion and electroshock therapy are safe procedures to carry out in patients who have pacemakers as long as the current does not go directly over the subcutaneous lead or generator pack.[94]

Conclusion

Temporary transvenous pacing is a rapid, safe, and reliable method for achieving effective electrical stimulation of the heart. Symptomatic bradycardias unresponsive to pharmacologic treatment and some tachycardias are indications for its use. In acute myocardial infarction, it serves both a therapeutic and a prophylactic function.

EMERGENCY TRANSCUTANEOUS CARDIAC PACING

Transcutaneous cardiac pacing (TCP) is a rapid, minimally invasive method of treating severe bradycardias and asystole. Electrodes are applied to the skin of the anterior and posterior chest walls, and pacing is initiated with a portable pulse generator. In an emergency setting, this pacing technique is faster and easier to initiate than transvenous pacing. Pulse generators are sufficiently portable to be used in EDs, hospital wards, intensive care units, and mobile paramedic vehicles.

Background

In 1872, Duchenne de Boulogne reported a successful resuscitation of a child by attaching one electrode to a limb while a second electrode was rhythmically touched to the precordium of the thorax.[95] Successful overdrive pacing of the human heart, using a precordial electrode, was reported by VonZiemssen in 1882. [96]

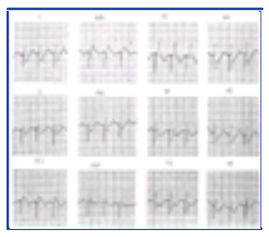

Figure 15-11 Coronary sinus pacing. Note the paced right bundle-branch block pattern.

In 1952, Zoll introduced the first practical means of TCP. Using a ground electrode attached to the skin and a subcutaneous needle electrode over the precordium, he reported the successful resuscitation of two patients in ventricular standstill. [97] One patient was paced for 5 days and subsequently was discharged from the hospital. Zoll later introduced a machine that delivered impulses lasting 2 msec through 3-cm-diameter metal paddles pressed firmly against the anterior chest wall. This device was the first commercial transcutaneous cardiac pacemaker. During the 1950s, Zoll and Leatham demonstrated the effectiveness of TCP in patients with bradycardia and asystole.[98] [99] [100] [101] Leatham used larger electrodes (4 × 6 cm) and a longer pulse duration (20 msec) to successfully pace two patients with bradydysrhythmias. [101]

Until the late 1950s, TCP was the only clinically accepted method of cardiac pacing. The original technique using bare metal electrodes had adverse effects, including local tissue burns, muscle contraction, and severe pain. [97] [101] With the development of the first implantable pacemakers from 1958 through 1960 and the improvement of transvenous electrodes during the early 1960s, TCP was rapidly discarded. [102]

Refinements in electrode size and pulse characteristics have led to the reintroduction of TCP into clinical practice. [103] [104] Increasing the pulse duration from 2 to 20 msec or longer was found to decrease the current output required for cardiac capture. [105] [106] Longer impulse durations also make the induction of ventricular fibrillation less likely. [105] Larger surface area electrodes (80 to 100 cm^2) decrease the current density at the underlying skin and therefore decrease pain and the possibility of tissue burns.[103]

Indications and Contraindications

General indications for cardiac pacing are discussed earlier. TCP is the fastest and easiest method of emergency pacing. This technique is useful for initial stabilization of the patient in the ED who requires emergency pacing while arrangements or decisions for transvenous pacemaker insertion are being made. The equipment is readily mastered, the procedure is fast, and it is minimally invasive. [104] [107] Refinements in equipment have made TCP the emergency pacing procedure of choice. TCP also is gaining widespread prehospital use in helicopter ambulance programs and inhospital use in the cardiac catheterization laboratory, operating room, intensive care unit, and on general medical floors.[108] [109] [110] The technique may be preferable to transvenous pacing in patients who have received thrombolytic agents. No central venous puncture, with the attendant risk of hemorrhage, is required. Limited experience suggests that TCP also may be useful in the treatment of refractory tachydysrhythmias by overdrive pacing.[111] [112] [113] [114] [115] Although small pediatric electrodes for TCP have been developed, experience with pediatric TCP has been limited.[116] [117]

TCP is indicated for the treatment of hemodynamically significant bradydysrhythmias that have not responded to atropine therapy. *Hemodynamically significant* implies hypotension, anginal chest pain, pulmonary edema, or evidence of decreased cerebral perfusion. This technique is *temporary* and is indicated for short intervals as a bridge until transvenous pacing can be initiated or until the underlying cause of the bradyarrhythmia (e.g., hyperkalemia, [107] drug overdose[118]) can be reversed. Although often unsuccessful, TCP may be

Figure 15-12 Coronary sinus position. *A,* Posteroanterior view. *B,* Lateral view. *(From Goldberger E: Treatment of Cardiac Emergencies, 3rd ed. St. Louis, CV Mosby, 1982.)*

attempted in the treatment of asystolic cardiac arrest. In this setting the technique is efficacious only if used early after arrest onset (generally within 10 minutes). [119] [120] TCP is not indicated for treatment of prolonged arrest victims with a final morbid rhythm of asystole. [117] [121] [122] [123]

Delay from the onset of arrest to the initiation of pacing is a major problem that limits the usefulness of TCP in prehospital care. Hedges and colleagues reported that everyday availability of pacing increased the number of patients who received pacing within 10 minutes of hemodynamic decompensation and increased long-term patient survival as well.[120] Prehospital pacing may be most useful in treatment of the patient with a hemodynamically significant bradycardia who has not yet progressed to cardiac arrest (e.g., heart block in the setting of acute myocardial infarction) or in the patient who arrests after the arrival of prehospital providers. [119] [120]

In conscious patients with hemodynamically stable bradycardias, TCP may not be necessary. It is reasonable to attach electrodes to such patients and to leave the pacemaker in standby mode against the possibility of hemodynamic deterioration while further efforts at treatment of the patient's underlying disorder are being made. This approach has been used successfully in patients with new heart block in the setting

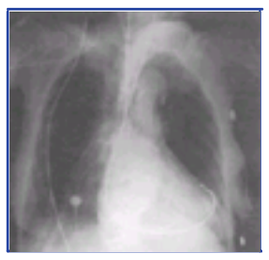

Figure 15-13 A pacing catheter that is outside or abuts the cardiac silhouette and is not properly positioned within the right ventricular cavity suggests myocardial perforation. *(From Tarver RD, Gillespie KR: The misplaced tube. Emerg Med, Feb 29, 1988, p 97.)*

of cardiac ischemia.[124] Generally when a transvenous pace-maker becomes available, transvenous pacing is preferred because of better patient tolerance.

Equipment

Few medical product lines have changed as rapidly as commercial transcutaneous pacemakers. Patent controversy, corporate acquisitions, and rapid product evolution have all contributed to this rapid change. [125] Despite this instability in the marketplace, transcutaneous pacemakers are now standard equipment in most EDs and are rapidly spreading to other inhospital and prehospital care settings. The pacemakers introduced in the early 1980s tended to be asynchronous devices with a limited selection of rate and output parameters. Units introduced more recently have demand mode pacing and more output options and are more likely to be combined with a defibrillator in a single unit. Combined defibrillator-pacers offer advantages in cost, ease, and rapidity of use when compared with stand-alone devices. An example of one combined unit is shown in Fig. 15-14 . A full-featured stand-alone pacemaker is illustrated in Fig. 15-15 .

All transcutaneous pacemakers have similar basic features. Most allow operation in either a fixed rate (asynchronous) or a demand mode. Most allow rate selection in a range from 30 to 200 beats/min. Current output is usually adjustable from 0 to 200 mA. If an electrocardiography monitor is not an integral part of the unit, *an output adapter to a separate monitor is required* to "blank" the large electrical spike from the pacemaker impulse and allow interpretation of the much smaller ECG complex. Without blanking protection, the standard ECG machine is swamped by the pacemaker spike and is uninterpretable. This could be disastrous, because the large pacing artifacts *can mask treatable ventricular fibrillation*

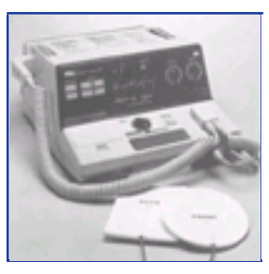

Figure 15-14 Combined defibrillator-transcutaneous pacemaker unit (Zoll-PD). The unit defibrillates through standard hand held paddles and has additional cable connections for electrocardiograph monitoring electrodes and for pacing electrodes. *(Courtesy of ZMI Corporation, Cambridge, Mass.)*

(Fig. 15-16). Pulse durations on available units vary from 20 to 40 msec and are not adjustable by the operator.

Two sets of patient electrodes are usually required for operation of the device. One set of standard ECG electrodes is used for monitoring. The much larger pacing electrodes deliver electrical impulses for pacing. One pacing electrode is placed over the mid-dorsal spine, and the other is placed over the left anterior chest. The posterior electrode serves as the ground.

Newer combined defibrillator-pacemakers can use a single set of electrodes for ECG monitoring, pacing, and defibrillation. This approach makes use of the device simpler,

Figure 15-15 "Stand alone" transcutaneous pacemaker (Zoll NTP). This unit has a built-in monitor and strip chart recorder. *(Courtesy of ZMI Corporation, Cambridge, Mass.)*

although the ECG waveform and analysis may be suboptimal. Provisions generally are made for separate ECG monitoring electrodes for use as desired by the operator.

Technique

Pad Placement

The pacing electrodes are applied as shown in Figure 15-17 and are attached to the instrument cable. The anterior electrode (cathode, or negative electrode) is placed as close as possible to the point of maximal impulse on the left anterior chest wall. This electrode adheres to the skin and has a large surface area for electrical contact. The second electrode is placed directly posterior to the anterior electrode. Failure to capture may be due to misplacement of the electrodes, and failure to pace may be rectified with a small change in anterior electrode position. ECG electrodes (if used) are placed on the chest wall or limbs, or both, as required and connected to the instrument cable. Some clinicians prophylactically apply pacing electrodes to all critically ill patients with bradycardia to facilitate immediate TCP should decompensation occur.

There is little risk of electrical injury to health care providers during TCP. Power delivered during each impulse is less than 1/1000 of that delivered during defibrillation.[126] Chest compressions (cardiopulmonary resuscitation) can be administered directly over the insulated electrodes while pacing. [127] Inadvertent contact with the active pacing surface results only in a mild shock.

Pacing Bradycardic Rhythms

To initiate TCP, the pacing electrodes are applied, and the device is activated. In the setting of bradyasystolic arrest, it is reasonable to turn the stimulating current to maximal output and then decrease the output as appropriate after capture is achieved. In a patient who has a hemodynamically compromising bradycardia but is not in cardiac arrest, the operator should slowly increase the output from the minimal setting until capture is achieved. Rate and output selections are adjustable (Fig. 15-18). Generally a heart rate of 60 to 70 beats/min will maintain an adequate blood pressure (by blood pressure cuff or arterial catheter) or the desired degree of mentation.

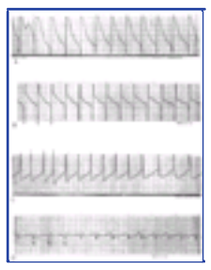

Figure 15-16 The top three rhythm strips *(A–C)* are taken from a standard wall-mounted electrocardiograph monitor. They all demonstrate large pacer spikes without capture. The underlying rhythm cannot be determined and could be treatable ventricular fibrillation. The bottom rhythm strip *(D)* demonstrates a tracing on the same patient with the external pacer monitor (special dampening). Note that the pacing spikes are much smaller, and it is easily seen that the underlying rhythm is asystole, without pacer capture. The presence of a T-wave after the QRS complex is a good indicator of ventricular capture.

Assessment of electrical capture can be made by monitoring the ECG on the filtered monitor of the pacing unit (Fig. 15-19). Additionally, bedside US may prove useful in determining ventricular capture.[128] [129] Ideally, pacing should be continued at an output level just above the threshold of initial electrical capture. One study in 16 normal male volunteers who were paced without sedation noted cardiac capture at a mean current of 54 mA (range, 42 to 60 mA).[130] Most subjects could tolerate pacing at their capture threshold; only 1 subject required discontinuation of pacing at 60 mA because of intolerable pain. Heller and associates compared subjective pain perception and capture thresholds in 10 volunteers paced with 5 different transcutaneous pacers. [131] Capture rates (40% to 80%), thresholds (66.5 to 104 mA), and subjective discomfort varied from pacemaker to pacemaker.

Failure to capture with TCP may be related to electrode placement or patient size. Patients with barrel-shaped chests and large amounts of intrathoracic air conduct electricity poorly and may prove refractory to capture. In one study, thoracotomy was found to nearly double the pacing threshold. [132] A large pericardial effusion or tamponade also will increase the output required for capture.[133] Failure to electrically capture with a transcutaneous device in these settings is an indication to consider immediate transvenous pacer placement.

Patients who are conscious or who regain consciousness during TCP will experience discomfort because of muscle contraction. [124] [130] [131] Analgesia with incremental doses of an opioid agent, sedation with a benzodiazepine compound, or both, will make this discomfort tolerable until transvenous pacing can be instituted.

Overdrive Pacing

Overdrive pacing[111] [112] [113] [114] [115] of ventricular tachycardia or paroxysmal supraventricular tachycardia is performed in patients who are stable enough to tolerate the brief delay associated with the necessary preparation for this technique. Little data exist on the efficacy or use of this procedure in the ED. The patient is sedated as explained earlier, pacing and monitoring electrode pads are placed in the standard position as detailed earlier, and brief trains (6 to 10 beats) of asynchronous pacing are applied. The pacer rate must be set approximately 20 to 60 pulses/min greater than the dysrhythmia rate. [134] Generally, an impulse rate of 200 pulses/min is used for ventricular tachycardias (rate generally 150 to 180 beats/min), and a rate of 240 to 280 pulses/min is used for paroxysmal

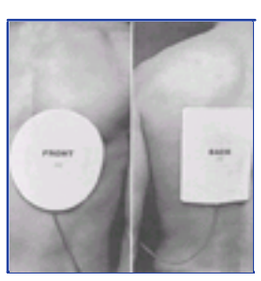

Figure 15-17 Correct placement of transcutaneous pacemaker electrodes (see text). *(Courtesy of ZMI Corporation, Cambridge, Mass.)*

supraventricular tachycardias (rate commonly 200 to 250 beats/min).

Because rhythm acceleration is possible during overdrive pacing, it is essential that full resuscitation equipment, including a defibrillator, be available at the bedside.

Complications

The major potential complication of TCP is *failure to recognize the presence of underlying treatable ventricular fibrillation*. This complication is primarily due to the size of the pacing artifact in the ECG screen, a technical problem inherent in systems without appropriate dampening circuitry.

A theoretical complication of TCP is the induction of dysrhythmias. In animal models using epicardial electrodes, the threshold current required to induce ventricular fibrillation decreases as electrical impulse duration increases. At a 10-msec impulse duration, ventricular fibrillation can be induced with currents as low as 25 mA delivered through epicardial electrodes.[105] Because TCP impulses are of even longer duration (20 msec) and of higher current (50 to 200 mA), there has been concern about possible induction of ventricular fibrillation during TCP. Studies of fibrillation thresholds using large *precordial* electrodes have shown the relationship of threshold-to-impulse duration to be the opposite of that seen with *epicardial* electrodes. With *cutaneous* precordial electrodes, the current required to induce ventricular fibrillation *increases* as pulse duration *increases*.[106] The apparent paradox may be explained by the differing nature of the electrodes. Epicardial electrodes are localized over a small area of the myocardium, whereas transcutaneous electrodes deliver a broad electrical charge to the myocardium as a whole. The implication is that longer impulse durations, although more dangerous with internal pacing, seem to decrease the chance of inducing ventricular fibrillation with

Figure 15-18 Rate and output selections.

TCP. Nonetheless, asynchronous TCP for tachydysrhythmias has been associated with rhythm acceleration and development of ventricular fibrillation. [113]

Experience with prolonged TCP in humans has not been extensive. Zoll and colleagues reported 25 humans paced for up to 108 hours with impulses of 20-msec duration.[100] Pacer-induced dysrhythmias did not occur. Leatham and associates paced 1 patient for 68 hours with impulses of 20-msec duration. [101] The patient died 2 days after pacing was discontinued. Pathologic examination revealed no evidence of pacerinduced myocardial damage. Madsen and colleagues paced 10 healthy volunteers at threshold for 30 minutes and found no enzyme or echocardiographic abnormalities. [135]

Studies of repetitive direct current countershocks in dogs have induced tissue damage with energy levels that are 1000 times greater than those required to pace the heart transcutaneously. In a canine study, 10 animals with chronic heart block that were paced for 60 minutes (20-msec duration at 80 beats/min with 8-cm-diameter cutaneous electrodes) did not develop pacer-related dysrhythmias. Serial cardiograms and cardiac enzymes revealed no evidence of ischemia or infarction. [136] Examination of the canine hearts after the dogs were sacrificed 72 hours after pacing did not reveal clinically significant myocardial damage. [137] A single primate paced for 1 hour with 20-msec impulses of 400 mA similarly had no evidence of tissue damage at autopsy and at microscopic examination after sacrifice 24 hours later.[138] Based on these studies, TCP appears unlikely to produce cardiac injury with short-term use in humans.

Soft tissue discomfort with the potential for injury may still occur with current transcutaneous pacemakers. One study with the Zoll transcutaneous pacemaker found only 2 of 30 subjects (patients and volunteers) who were paced while conscious required discontinuation of pacing owing to discomfort. [130] Most reported the

discomfort as "mild or moderate and easily tolerable." Sedation would presumably improve a conscious patient's ability to tolerate TCP.

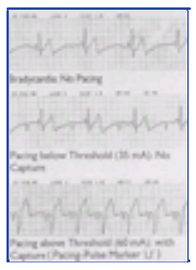

Figure 15-19 Assessing electrocardiogram capture with TCP. Note that the monitor has been adapted to accommodate the large pacing artifact so as not to obscure the underlying ventricular activity.

Nonetheless, prolonged use may still induce local cutaneous injury; Pride and McKinley reported one 7-week-old child who was paced for 45 hours without a pad change and who developed third-degree burns.[139]

Conclusion

Devices that pace the heart externally have been available for clinical use since 1952. Technologic improvements have minimized the complications associated with earlier use of the transcutaneous route and have enabled the reapplication of this relatively old pacing technique to a select subset of cardiac emergencies. The introduction of combined defibrillator-pacemakers promises to make pacing more available in prehospital and health care settings. Although the technique is still not universally available, it is rapidly becoming the standard of care for resuscitation protocols and equipment. Pacing instituted earlier in the course of bradycardiac rhythms, including the prehospital phase of care, may improve the poor survival rate currently associated with these rhythms.

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Chapter 16 - Pericardiocentesis

Richard J. Harper

Pericardiotomy under direct vision was first done in 1815, and in 1840 the first blind approach using a trocar was carried out successfully on a patient with tamponade from malignancy.[1] By the end of the 19th century, the trocar-and-cannula method of pericardiocentesis was commonly used. The subxiphoid approach was first described in 1911.

Blind, electrocardiography (ECG)-assisted pericardiocentesis has a significant morbidity rate, reportedly as high as 15% to 20%. [2] [3] For this reason ultrasound diagnosis of pericardial effusion with fluoroscopic or ultrasound guidance has become the standard for *elective* pericardiocentesis because of its lower (0.5% to 3.7%)[4] [5] [6] incidence of complications. Even if tamponade physiology is present, echocardiographic diagnosis and guidance is essential. The ECG-assisted blind pericardiocentesis technique remains the standard procedure only for truly *emergent* pericardiocentesis when a lengthy delay may be associated with obtaining and organizing ultrasound or fluoroscopic assistance. Echocardiographic diagnosis and guidance are described elsewhere (see Chapter 69).

CAUSES OF PERICARDIAL EFFUSION AND TAMPONADE

The medical literature concerning pericardiocentesis categorizes pericardial fluid collection as one of the following: acute hemopericardium (largely secondary to trauma) and pericardial effusion from other causes. This categorization is based on the fact that these two clinical entities are different in their time course, etiology, and treatment.

Acute Hemopericardium

Acute hemopericardium has several causes, including coagulopathies, cardiovascular catastrophes, and acute injury resulting from either blunt or penetrating trauma. All of these causes result in rapid accumulation of whole blood in the pericardial sac. The blood accumulates too fast for the relatively inelastic pericardial sac to stretch and accommodate the fluid. The result is cardiac tamponade produced by small fluid volumes and with an essentially normal pericardial size.

Penetrating Trauma

Traumatic tamponade due to penetrating trauma may result from obvious external injury such as knife or gunshot wounds, or it may be insidious, as seen with iatrogenic cardiac perforation during cardiac or vascular procedures.

In external penetrating trauma, tamponade is most commonly the result of a stab wound.[7] Approximately 80% to 90% of stab wounds to the heart demonstrate tamponade,[7] [8] compared with 20% of gunshot wounds. Stab wounds cause tamponade more often presumably because the pericardial rent is small enough to seal, trapping blood in the pericardial space. [7] [9] Larger pericardial wounds from gunshots generally drain into the pleural space and produce a hemothorax. [10] Cardiac tamponade is often suspected with anterior chest wounds, but it is imperative to remember that any penetrating wound of the chest, back, or upper abdomen may involve the heart.

Iatrogenic causes of cardiac tamponade are relatively uncommon but well-known complications of invasive or diagnostic procedures. Pacemaker insertion (either transthoracic or transvenous) and cardiac catheterization, including valvuloplasty and angioplasty, are two of the main causes, from the inadvertent penetration of cardiac chambers or coronary vessels.[11] [12] [13] Penetration of vascular structures is common during transthoracic pacemaker placement. [14] Tamponade is also seen as a complication after cardiac surgery, although it is usually anticipated, and mediastinal or pericardial drainage helps to control and prevent it. [11] [15] Pericardiocentesis itself can cause tamponade by lacerating myocardium or coronary vessels. [16] [17]

Cardiac tamponade may result from perforation of the right atrium or, less commonly, of the right ventricle or superior vena cava by a central venous pressure (CVP) catheter or subclavian hemodialysis catheter. [18] This event is usually not diagnosed early and is therefore often fatal. [19] Perforation may occur during placement or, more commonly, 1 to 2 days later, when the catheter erodes through tissue, particularly if a catheter made of stiff material is used or when the left internal jugular vein approach is used.[20] Tamponade from CVP line placement is seldom seen in the emergency department (ED) but must always be considered when there is sudden decompensation in a patient with a CVP line in place. Tamponade should always be considered when a patient deteriorates hemodynamically after an invasive diagnostic or therapeutic procedure involving the heart. Prevention involves proper placement of central venous catheters in the superior vena cava rather than the right atrium or ventricle.

Blunt Trauma

Blunt trauma may cause hemopericardium, most often as the result of major chest injury with associated rib and sternal fractures. Cases have been reported, however, in which tamponade occurred in blunt trauma with no obvious signs of injury to the thorax. [21] Such incidents may be more common than are clinically recognized, judging by the reports of constrictive pericarditis and pericardial defects found months to years later in trauma patients who were not originally noted to have effusion. Pericardial effusion due to blunt trauma may also be a late finding, becoming symptomatic 12 to 15 days after trauma. [22]

Severe deceleration injury may cause tamponade as a result of aortic or caval injury. [23] This appears to be an uncommon development, with two case series reporting tamponade in 3.6% (1 of 28 patients) and 2.3% (1 of 43 patients) of victims of aortic injury. [24]

Theoretically, cardiopulmonary resuscitation (CPR) can cause pericardial effusion secondary to the blunt trauma of chest compressions, broken ribs, or intracardiac injections. Early studies reported pericardial effusion in 1% to 3% of CPR survivors. [25] Echocardiographic studies showed small cardiac effusions (but not tamponade) in 12% of survivors, only 4% of whom had received intracardiac injections. [26] Thus, although case reports of tamponade exist,[27] [28] CPR and intracardiac drug injections are unlikely to cause significant effusion, much less tamponade.

Nontraumatic Hemopericardium

Nontraumatic but acute hemopericardium caused by a bleeding diathesis, aortic dissection and ventricular rupture behaves

much like traumatic tamponade because of its acute nature. These types of hemopericardium are less obvious in etiology than hemopericardium caused by external trauma.

Bleeding diathesis may cause spontaneous bleeding into the pericardial sac. The incidence of spontaneous pericardial tamponade in anticoagulated patients has been reported to range from 2.5% to 11%.[11] [29] Thrombolytic therapy has also been implicated in tamponade secondary to bleeding diathesis. Among 392 patients, only 4 (1%), all with large anterior myocardial infarctions, developed tamponade secondary to hemopericardium without ventricular rupture. [30]

A dissection of the ascending aorta may extend around the base of the aorta into the pericardial sac, causing dramatic, rapid, and usually fatal tamponade. This pathologic abnormality may be due to conditions such as syphilis, Marfan syndrome, or atherosclerosis. Infection may create pseudoaneurysms of the aorta, which also can present as tamponade.[31]

Ventricular rupture after myocardial infarction is a common source of acute hemopericardium. Although the prognosis is often grim, survival is possible with prompt recognition and definitive treatment. [32] [33]

Nonhemorrhagic Effusions

Nonhemorrhagic effusions usually accumulate slowly, allowing the pericardium to stretch and accommodate up to 2000 mL of fluid. [34] This slower accumulation, often over weeks to months, allows the circulatory system to adapt and permits more time for evaluation and treatment, even in a moderately hypotensive patient. [35] [36] In many cases of small nonhemorrhagic effusion, tamponade does not occur, and the effusion may resolve with treatment of the underlying disease or may be managed successfully by elective pericardiocentesis.

Many disease processes, ranging from the common to the rare (Table 16-1), can cause pericardial effusion. The cause of nonhemorrhagic tamponade may not be obvious on examination in the ED, and tamponade is frequently misdiagnosed as congestive heart failure or respiratory disease. Although neoplasm has generally been the most common underlying cause of nonhemorrhagic effusion, [29] [37] some reports[38] [39] have identified infectious complications of the human immunodeficiency

virus (HIV) as a common etiology of large nonhemorrhagic pericardial effusion and tamponade (Table 16-2).

HIV-related effusions have been ascribed to many opportunistic bacterial and viral infections, with mycobacterial infections being the most common. [38] [39] [40] Kaposi sarcoma and lymphoma[41] [42] have caused noninfectious pericardial effusions in HIV patients. Cancer is a prominent cause of nonhemorrhagic effusions; the pericardium is involved in 20% of patients with disseminated tumors [43] and 8% of all patients with cancer. [44] There is primary pericardial involvement in 69% of acute leukemias, in 64% of malignant melanomas, and in 24% of lymphomas; however, the incidence of actual tamponade in these malignancies is not known. Of metastases to the pericardium, 35% originate in the lung, 35% in the breast, 15% in lymphomas, and less than 3% in each of the other cancers. [44] Thus, any patient who is known to have one of these malignancies should be considered at risk for tamponade. Metastasis to the heart is usually a late finding in cancer, and other foci located elsewhere are usually evident. [45] Classic findings of tamponade, such as *pulsus paradoxus*, are frequently absent in cancer patients with tamponade, and their symptoms are usually attributed to their malignancy. [44]

TABLE 16-1 -- Causes of Pericardial Effusion

Neoplasm	Mesothelioma
	Lung
	Breast
	Melanoma
	Lymphoma
Pericarditis	Radiation (especially after Hodgkin's disease)
	Viral
	Bacterial
	Staphylococcus
	Pneumococcus
	Haemophilus
	Fungal
	Tuberculosis
	Amebiasis
	Toxoplasmosis
	Idiopathic
Connective tissue disease	Systemic lupus erythematosus
	Scleroderma
	Rheumatoid arthritis
	Acute rheumatic fever
Metabolic disorders	Myxedema
	Uremia
	Cholesterol pericarditis
	Bleeding diatheses
Cardiac disease	Acute myocardial infarction
	Dissecting aortic aneurysm
	Congestive heart failure
	Coronary aneurysm
Drugs	Hydralazine
	Phenytoin
	Anticoagulants
	Procainamide
	Minoxidil
Trauma	Blunt
	Major trauma
	Closed-chest CPR
	Penetrating
	Major penetrating trauma
	Intracardiac injections
	Transthoracic and transvenous pacing wires
	Pericardiocentesis
	Cardiac catheterization
	CVP catheter
Miscellaneous	Serum sickness
	Chylous effusion
	Löffler syndrome
	Reiter syndrome
	Behçet syndrome
	Pancreatitis
	Postpericardiotomy
	Amyloidosis
	Ascites

Data from Guberman BA, Fowler NO, Engel PJ, et al: Cardiac tamponade in medical patients. Circulation 64:633, 1981; and Pories WJ, Caudiani VA: Cardiac tamponade. Surg Clin North Am 55:573, 1975.

Radiation pericarditis, particularly after treatment for Hodgkin's disease, is a common cause of effusion. [34] Effusion occurs in approximately 5% of those patients who receive 4000 rad to the heart.

Approximately 15% to 20% of patients on dialysis for renal failure develop pericarditis, and 35% of those with pericarditis

TABLE 16-2 -- Etiology of Pericardial Effusion in Two Studies *

	Krikorian[29] (120 Patients) (%)	**Guberman[37] (56 Patients) (%)**
Neoplastic disease	—	32
Pericardial invasion	16	—
Radiation pericarditis	7.5	4
Etiology uncertain	18	—
Traumatic hemopericardium	9	—
Hemopericardium, nontraumatic	2.5	—
Rheumatic disease	12	2
Uremia/dialysis	5	9
Bacterial infection	2.5	12.5
Congestive heart failure	1.5	—
Uncertain etiology	12.5	—
Idiopathic pericarditis	13.5	14
Cardiac infarction	—	—
Iatrogenic diagnostic procedures	—	7.5
Myxedema	—	4
Aneurysm	—	4
Anticoagulation and cardiac disease	—	11
Postpericardiotomy	—	2

*_Note:_ Various complications related to human immunodeficiency virus (HIV) infections are now probably the most common causes of large nonhemorrhagic pericardial effusions. Effusions related to bacterial, viral, and mycobacterial infections and Kaposi sarcoma and lymphoma are common.

develop tamponade.[46] [47] Up to 7% of patients on chronic dialysis may have effusions, sometimes of 1 L or more. [45] Some series have reported tamponade in 34% of uremic patients who have effusions. [47] Pericardial effusion in renal failure may be managed with dialysis alone in many cases.

Thirty percent of myxedema patients may have pericardial effusions, but few have tamponade. [37] Most of the other etiologies listed in Table 16-1 are isolated case reports, and their exact incidences have not been determined.

Other Causes of Pericardial Tamponade

An interesting but rare cause of cardiac tamponade is pneumopericardium. Pneumopericardium is most commonly seen with pneumothorax and pneumomediastinum as a complication of respiratory therapy in infants, but it may also occur from similar barotrauma in adults. [48] Pneumopericardium also occurs spontaneously in asthma,[49] after blunt chest injury,[50] [51] and even after high-speed motorcycle rides.[52] Pneumopericardium is usually benign, but tension pneumopericardium has been reported as a cause of life-threatening tamponade after blunt chest trauma [51] [53] and after pericardiectomy.[54] The appearance of life-threatening pneumopericardium and tamponade has also been described immediately[55] and 6 days after penetrating chest trauma.[56]

PATHOPHYSIOLOGY OF TAMPONADE

The pericardium is a tough, leathery sac normally containing about 25 to 35 mL of serous fluid. [57] It is not rapidly elastic, although it does demonstrate stress relaxation within minutes of increased intrapericardial pressure, providing a slight ability to accommodate sudden increases in fluid. [58] As fluid accumulates, the first 80 to 120 mL is easily accommodated without significantly affecting pericardial pressure (Fig. 16-1).[59] However,

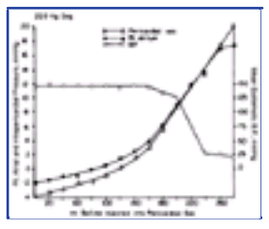

Figure 16-1 Production of cardiac tamponade by injections of saline into the pericardial sac. Although pericardial space can acutely accommodate 80 to 120 mL of fluid without a significant increase in pericardial pressure, note steep increases in pressure and drop in blood pressure at about 200 mL of saline. Once critical volumes are reached, very small increases cause significant hemodynamic compromise. *(From Fowler NO: Physiology of cardiac tamponade and pulsus paradoxus. II: Physiological, circulatory, and pharmacological responses in cardiac tamponade. Mod Concepts Cardiovasc Dis 47:116, 1978. Reproduced by permission of the American Heart Association, Inc.)*

if an additional 20 to 40 mL is rapidly accumulated, the intrapericardial pressure almost doubles, often leading to sudden decompensation. With effusions that develop over weeks to months, the pericardium lengthens circumferentially to a huge size and can accommodate liters of fluid.

Pericardial compliance, which helps determine the pressure-volume response curve (Fig. 16-2), [57] varies considerably in different individuals and various disease states. The

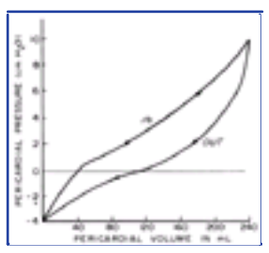

Figure 16-2 Relationship of intrapericardial pressure to volume of pericardial fluid. Note that pressure drops rapidly when a small amount of fluid is removed. *(From Pories W, Gaudiani V: Cardiac tamponade. Surg Clin North Am 55:573, 1975. Reproduced by permission.)*

pressure-volume relationship demonstrates hystersis; the withdrawal of a quantity of fluid drops the pressure more than the addition of the same amount of fluid raised the pressure.

As pericardial fluid accumulates, the increased intrapericardial pressure is transmitted across the myocardial wall and causes compression of the atria and perhaps the vena cava and pulmonary veins. This reduces right ventricular filling in diastole, producing decreased stroke volume and cardiac output. [60] Pulse pressure narrows as reflex sympathetic stimulation increases. Severe tamponade is produced with intrapericardial pressures of 15 to 20 mm Hg. [61]

As stroke volume decreases, heart rate increases to maintain cardiac output. Sympathetic discharge causes both arterial and venous vasoconstriction. [61] [62] Vasoconstriction increases venous pressure, which helps to restore the normal venousatrial and atrioventricular filling gradients. These compensatory mechanisms are often effective and may permit establishment of a new homeostasis with normal cardiac output.

With chronic effusion and in early tamponade, cardiac contractility is not affected, and myocardial perfusion is normal. [60] [63] [64] As pressure continues to increase, coronary perfusion pressure drops, so in its later stages, tamponade causes myocardial ischemia. Before hypotension occurs, left ventricular blood flow has already decreased 37%.[65] For comparable degrees of hypotension, experimental animals in hemorrhagic shock have five times greater coronary blood flow than animals in cardiac tamponade.[65] Severe experimental tamponade is followed by large increases in creatine kinase MB and microscopic evidence of cardiac injury resulting from ischemia.[66]

As intrapericardial pressure continues to rise, the heart's compensatory mechanisms fail. Myocardial ischemia and perhaps lactic acidosis from poor tissue perfusion may be the triggering events that disrupt the delicate equilibrium. [67] Atrial

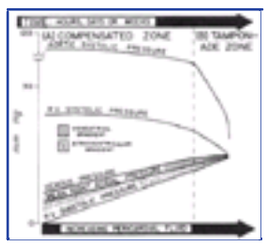

Figure 16-3 Summary of physiologic changes in tamponade. RV, right ventricle. *(From Shoemaker WC, Carey JS, Yao ST, et al: Hemodynamic monitoring for physiological evaluation, diagnosis, and therapy of acute hemopericardial tamponade from penetrating wounds. J Trauma 13:36, 1973; and Spodick D: Acute cardiac tamponade: Pathologic physiology, diagnosis, and management. Prog Cardiovasc Dis 10:65, 1967. Reproduced by permission.)*

pressure rises rapidly (Fig. 16-3). The atria and pulmonary circulation, being at much lower pressure than the systemic arterial pressure, are more vulnerable to the rising intrapericardial pressure. A "pressure plateau" occurs in which right atrial pressure, right ventricular diastolic pressure, pulmonary artery diastolic pressure, and pulmonary capillary wedge pressure are virtually identical.

This equalization of pressures leads to the echocardiographic hallmark of tamponade: right ventricular collapse. At this point hypotension is severe, bradycardia is common, and pulseless electrical activity (PEA) may occur. Unless intrapericardial pressure is immediately decreased, pulmonary blood flow ceases, and cardiac arrest follows.[67]

Total blood volume affects cardiac compensation, and it is possible to encounter a "low-pressure" cardiac tamponade. [68] The hypovolemic patient with tamponade has a decreased venous pressure, which not only decreases cardiac output, but also may obscure the diagnosis, because *distended neck veins or an elevated CVP are not present*. In a patient with a chronic pericardial effusion, the onset of hypovolemia can lower filling pressure enough to precipitate tamponade, and conversely, providing additional volume may temporarily offset increased pericardial pressure.

Ventilation and blood CO_2 levels have significant effects on cardiac tamponade. This is of particular significance, because trauma patients with tamponade may also have respiratory impairment. Pericardial pressure decreases 3 to 6 mm Hg with a hypocarbia of 24 torr and increases 2 to 4 mm Hg when the PCO_2 reaches 57 torr.[69] This degree of hypercarbia induced pericardial pressure rise can decrease cardiac output by 25%. Similarly, fluctuations in intrapleural pressure induced by intermittent positive-pressure ventilation are transmitted to the pericardial space and can reduce cardiac output another 25%. [70] The clinical implications of these findings are that patients suspected of having tamponade should normally be allowed to breathe spontaneously under careful monitoring and should not be ventilated

with positive pressure unless it is absolutely necessary, as their hemodynamic status may deteriorate precipitously.

DIAGNOSIS OF CARDIAC TAMPONADE

Patient Profile and Symptoms

Pericardial effusion is rarely diagnosed based on physical findings. In contrast, pericardial tamponade can be diagnosed based on clinical criteria, but specific clinical signs are often absent. Particularly in the setting of acute hemorrhagic tamponade, the time from the first signs of tamponade to full arrest may be brief. [71]

Classic clinical findings have been described for tamponade. However, these findings are often obvious only when the patient is unstable due to tamponade. Ideally, tamponade is diagnosed early, when the patient suffers no more than dyspnea, weakness, or perhaps right heart failure. It is common to attribute respiratory symptoms (e.g., dyspnea on exertion) to a more common condition such as heart failure or pulmonary pathology and to overlook pericardial effusion until the classic late signs (e.g., hypotension) appear. [72]

Acute pericardial tamponade may resemble tension pneumothorax, acute hemothorax, hypovolemia, pulmonary edema, or pulmonary embolism. Severe right ventricular contusion

can mimic the findings of tamponade. [73] The patient is often agitated or panic-stricken, confused, uncooperative, restless, cyanotic, diaphoretic, and acutely short of breath. In the late stages, the patient is moribund. Hypotension in the presence of severe cyanosis and distended neck veins is a helpful but late finding.

Physical Signs

The classic physical findings of tamponade were first characterized by Beck in 1935. He described two triads, one for acute and one for chronic compression. [74] The chronic compression triad consists of high CVP; ascites; and a small, quiet heart. The triad in acute compression consists of high CVP, decreased arterial pressure, and muffled heart sounds. Unfortunately, in most major trauma series, only about one third of patients demonstrate the complete acute triad, [67] [75] although almost 90% have one or more signs. [7] *The simultaneous occurrence of all three physical signs is a very late manifestation of tamponade and is usually seen most consistently shortly before cardiac arrest* (see Fig. 16-3).

Careful hemodynamic monitoring reveals earlier changes that indicate the progression of tamponade (Table 16-3). [76] In grade I tamponade, cardiac output and arterial pressure are normal, but CVP and heart rate are increased. In grade II tamponade, blood pressure is normal or slightly decreased, CVP is increased, and tachycardia persists. In grade III tamponade, the classic findings of Beck's acute triad occur. Although this sequence represents the natural history of acute tamponade, the time course varies. Some patients are stable at a given stage for hours; others proceed to cardiac arrest within minutes. [67] [76] Unfortunately, not all patients with early tamponade respond with a predictable pattern of change in vital signs. Brown and coworkers found that 6 of 18 patients with tamponade, defined through right heart catheterization, responded to tamponade with elevated systolic blood pressure. [77] After pericardiocentesis, these patients had a marked reduction in systolic blood pressure accompanied by increased cardiac output. All of these patients had previously been hypertensive.

Pulsus Paradoxus (see also Chapter 1)

Pulsus paradoxus is defined as an exaggeration of the normal inspiratory fall in blood pressure. [62] [75] A paradoxical pulse (pressure) is one of the classic physical signs of tamponade, but it is not pathognomonic. It is also caused by pulmonary emphysema, asthma, labored respirations, obesity, cardiac

TABLE 16-3 -- Shoemaker System of Grading Cardiac Tamponade

Grade	Pericardial Volume (mL)	Cardiac Index	Stroke Index	Mean Arterial Pressure	CVP	Heart Rate	Beck's Triad
I	<200	Normal or ?	Normal or ?	Normal	?	?	Venous distention, hypotension, muffled heart sounds usually not present
II	=200	?	?	Normal or ?	? (=12 cm H_2 O)	?	May or may not be present
III	>200	??	??	??	?? (up to 30–40 cm H_2 O)	?	Usually present

From Shoemaker WC, Carey SJ, Yao ST, et al: Hemodynamic monitoring for physiologic evaluation, diagnosis, and therapy of acute hemopericardial tamponade from penetrating wounds. J Trauma 13:36, 1973.

failure, constrictive pericarditis, pulmonary embolism, and cardiogenic shock. [7] [35] [67] Measuring the paradoxical pulse is difficult and time-consuming, and any frightened, hypotensive patient with labored breathing can demonstrate this finding (Fig. 16-4).

If the difference between inspiratory and expiratory systolic blood pressures is greater than 12 mm Hg, the paradoxical pulse is abnormally high. [78] Most patients with proven tamponade will demonstrate a difference of 20 to 30 mm Hg or more during the respiratory cycle. [7] [35] [67] This may not be true of patients with very narrow pulse pressures (typical of grade III tamponade); they will have a "deceptively small" paradoxical pulse of 5 to 15 mm Hg. The decreased *pulsus paradoxus* with hypotension occurs because the paradoxical pulse is a function of actual pulse pressure, and the inspiratory systolic pressure may be below the level at which diastolic sounds disappear. [62] For this reason, the ratio of the paradoxical pulse to the pulse pressure is a more reliable measure. A paradoxical pulse greater than 50% of the pulse pressure is abnormal. [62]

Pulsus paradoxus in tamponade has been correlated with the degree of impairment of cardiac output. In atraumatic patients, a 15% *pulsus paradoxus* in the face of relative hypotension was found in 97% of patients with moderate or severe tamponade and only 6% of patients with absent or mild tamponade. [78] A similar study of right ventricular diastolic collapse by echocardiography found that an abnormal *pulsus paradoxus* had a sensitivity of 79%, a specificity of 40%, a positive predictive value of 81%, and a negative predictive value of 40%. [79]

The absence of a paradoxical pulse does not rule out tamponade. Although the mean paradoxical pulse was 49 mm Hg in one series of nonhemorrhagic tamponade, [37] 23% of the patients had a paradoxical pulse of less than 20 mm Hg, and 1 patient had no measurable paradoxical pulse. An abnormal *pulsus paradoxus* has been reported to be absent in tamponade when there is an atrial septal defect, aortic insufficiency, localized collections of pericardial blood, or extreme tamponade with hypotension. [68] It may also be absent when left ventricular diastolic pressure is intrinsically elevated owing to poor left ventricular compliance. This was seen in one half of uremic patients with tamponade. [47] [80] In traumatic tamponade, *pulsus paradoxus* is deemed unreliable. [68] [80] [81] [82] In one study, only 35% of trauma patients had an abnormal paradoxical pulse when elevated CVP and decreased heart sounds were present. [82] In another study of 197 traumatic cases, only 8.6% of the diagnoses of tamponade were made by finding an abnormal *pulsus paradoxus*. [83]

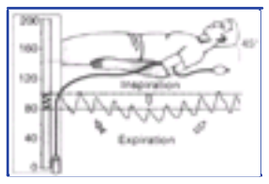

Figure 16-4 Normally systolic blood pressure drops slightly during inspiration. To measure *pulsus paradoxus*, the patient breathes normally while lying at a 45-degree angle. The blood pressure cuff is inflated well above systolic pressure and slowly deflated. When the pulse is first heard only during expiration, this is the upper value. The cuff is deflated until the pulse is heard during both inspiration and expiration, and this is the lower value. The difference in the two values is the amount of *pulsus paradoxus*. A difference of more than 12 mm Hg is abnormal.

Although the absence of a paradoxical pulse rules against severe tamponade, it does not completely rule it out. Whether time is taken to determine *pulsus paradoxus* depends on the patient's status. If the patient is moribund or rapidly deteriorating, taking time to check this parameter is obviously a poor choice of priorities.

Venous Distention

Venous distention, reflecting increased CVP, is also a late sign in cardiac tamponade (see Fig. 16-3). It may be masked by venoconstriction as a result of vasopressors (e.g., dopamine), intrinsic sympathetic discharge, or hypovolemia. [35] [67] [76] [81] Neck vein distention may be obvious clinically, but the measured CVP is more reliable than the presence of venous distention. The CVP reading should take into account positive-pressure ventilation and the effects of a Valsalva maneuver. Most patients with significant tamponade will have a CVP of greater than or equal to 12 to 14 cm H_2O.[81] *Hypovolemia changes the intrapericardial pressure-volume curve in tamponade and will lower the CVP reading at any given stage in the tamponade process.*

Animal studies have documented that right atrial pressure can be normal in tamponade when hypovolemia is present. One case of low-pressure cardiac tamponade was reported in a patient with no jugular venous distention, no paradoxical pulse, and a right atrial pressure of 8 mm Hg. [68] Thus, although the initial CVP reading is useful and diagnostic if grossly elevated (e.g., 20 to 30 cm H_2O),[54] [81] a series of CVP readings looking for an upward trend is the most sensitive diagnostic tool. [81] A rising CVP, especially when there is persistent hypotension, is extremely suggestive of tamponade in the trauma patient. In the rare case of the hypovolemic patient in whom tamponade is suspected but who demonstrates a low CVP, a fluid challenge will help clarify the situation and will also improve cardiac output at least temporarily.[68]

Ancillary Testing

Routine chest radiographs and electrocardiograms may be useful in increasing the level of suspicion for pericardial effusion and tamponade. Noninvasive diagnosis of effusion, however, must be made by computed tomography or, preferably, cardiac ultrasound. If available, bedside ultrasound is the fastest and most reliable for the emergency clinician to demonstrate a significant pericardial effusion, although it may not be diagnostic of tamponade.

Chest Radiographs

Chest radiographs are not useful in the diagnosis of acute traumatic tamponade, because *the cardiac size and shape do not change acutely*. However, the radiographs may reveal hemothorax, bullet location, or even pneumopericardium.

In the patient without trauma and with chronic effusion, a chest film often reveals an enlarged, sac-like "water bottle" cardiac shadow. Unfortunately, it is difficult to differentiate pericardial from myocardial enlargement, and radiographs cannot be used to distinguish between simple pericardial effusion and tamponade. One finding that is useful in identifying effusion on the plain chest film is the epicardial fat pad sign. The water density space between the radiolucent epicardial fat and the mediastinal fat represents the pericardial tissues and is normally less than 2 mm. An increase in this width suggests pericardial fluid or thickening (Fig. 16-5). This sign may be seen in 41% of upright lateral and 23% of frontal chest films in proven pericardial effusion. [84] The diagnostic value may be enhanced by using a supine rather than upright cross-table, lateral chest radiograph. Obtaining a supine lateral film increases the sensitivity of the epicardial fat pad sign from 31% to 51%. [85]

Electrocardiograms

Electrocardiograms may suggest, but should *not* be used to diagnose, pericardial effusion or cardiac tamponade. Most electrocardiogram changes, such as PR-segment depression, low-voltage QRS complexes, and electrical alternans, have acceptable specificity but poor sensitivity for pericardial effusion or tamponade.[35] [86] [87] Low voltage is defined as a QRS amplitude less than or equal to 5 mV in all limb leads (or a sum of the limb lead QRS amplitude less than or equal to 30 mV), and PR depression is defined as greater than or equal to 1 mV depression in at least 1 lead other than aVR. In a study correlating the electrocardiogram with echocardiographic evaluation, electrocardiogram signs had an overall sensitivity of only 1% to 17% and a specificity of 89% to 100% for pericardial effusion.[35] Others have demonstrated significantly higher sensitivity, i.e., in the range of 32 to 68% for voltage criteria. [88] PR-segment depression is the most common electrocardiogram finding in pericardial tamponade, and low voltage is most commonly associated with a moderate to large effusion. It is important to note that none of the ECG findings differentiate tamponade from effusion.

Electrical alternans is caused by pendulum motion of the heart within the pericardial sac. [89] Alternans of the QRS complex has been seen in about 22% of medical tamponade cases[72] but in only 5% of cancer patients with tamponade.[44] Electrical alternans of both the P wave and the QRS complex (total electrical alternans) is a rare finding, but when seen is thought to be pathognomonic of tamponade (Fig. 16-6).[35] [90] Like electrical alternans, low voltage may be a finding associated with tamponade, but not simple effusion.[91]

Echocardiography

Echocardiography is the best available tool for diagnosing pericardial effusion and has the further advantage of being noninvasive. [92] Echocardiography is very sensitive in the diagnosis of pericardial effusion and tamponade. [93] [94]

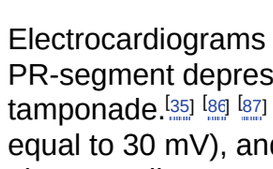
Figure 16-5 Epicardial fat pad sign. The water density space between the radiolucent epicardial fat and the mediastinal fat represents the pericardium and its contents and should be 2 mm or less. An increase suggests pericardial fluid or thickening. *A,* Left anterior-oblique chest film. *B,* Lateral chest film. In acute tamponade, the chest radiograph has very minimal diagnostic value.

The disadvantages of echocardiography are that it requires ultrasound equipment and is dependent on a skilled operator who is specifically trained in echocardiography. Even when immediately available, echocardiography may take at least 5 minutes, which may be too much time for a patient who is deteriorating rapidly. If the patient in not in full arrest and ultrasound is available, ultrasound should always be used to diagnose effusion and tamponade and to guide the procedure (see Chapter 69). Pericardial fluid is relatively easy

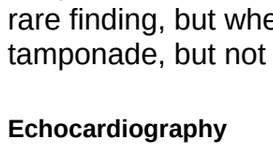
Figure 16-6 Overall, the electrocardiogram (ECG) has a low sensitivity for pericardial effusion or tamponade, but PR depression, low voltage, or electrical alternans may be seen. Lewis lead ECG showing total electrical alternans of both amplitude and configuration of P and QRS complexes. This is rarely seen but is highly suggestive of tamponade. Note that electrical alternans may not be evident in standard ECG leads. *(From Sotolongo RP, Horton JD: Total electrical alternans in pericardial tamponade. Am Heart J 101:854, 1981. Reproduced by permission.)*

to demonstrate with bedside ultrasound, but since many ill patients will demonstrate some pericardial fluid, bedside ultrasound may not be able to differentiate

incidental fluid from tamponade.

Computed Tomography

At some institutions, computed tomography (CT) is much more readily available than echocardiography. However, it requires that the patient be transported to the site of the CT equipment and patient stability must be considered. If clinically indicated, CT is effective in defining the presence and extent of pericardial effusion in the stable patient.[95] In certain circumstances, CT can provide a more definitive diagnosis than echocardiography. In one series, eight equivocal echocardiograms were evaluated by CT.[96] Two patients thought to have pericardial effusion by ultrasound were found by CT to have pleural effusions. Another patient with pericardial effusion by ultrasound was found by CT to have an epicardial lipoma. CT defined three loculated pleural effusions not seen by ultrasound. A final two patients had hemopericardium visualized by CT but not ultrasound. In circumstances where the patient is stable and ultrasound produces equivocal results or is not available, CT may provide a definitive diagnosis of pericardial effusion.

INDICATIONS FOR PERICARDIOCENTESIS

There are two indications for pericardiocentesis: (1) to diagnose the cause or presence of a pericardial effusion and (2) to relieve tamponade. The former is an elective procedure and ideally should be accomplished under ultrasound guidance. The latter may be semi-elective and performed with ultrasound guidance or emergent and performed blind with ECG assistance.

Diagnostic Pericardiocentesis

The use of pericardiocentesis for diagnosis of the etiology of nonhemorrhagic effusions is widespread, although opinions of its utility vary. [34] [97] [98] Neoplastic cells, blood, bacteria, viruses, and chyle can be sought. Measurement of pericardial fluid pH can be helpful, because inflammatory fluid is significantly more acidotic than noninflammatory fluid.[99] When a specific etiology is suspected, additional diagnostic testing may be useful (e.g., adenosine deaminase in tuberculosis, and carcinoembryonic antigen in suspected malignancy). [100]

The diagnostic accuracy of pericardiocentesis varies greatly from series to series, depending on the vigor with which a definitive etiology was sought and the prevalence of certain etiologies in the patient population under consideration. In one large series, fluid was obtained in 90% of the taps, but a specific etiologic diagnosis was obtained in only 24% of the fluid specimens. [29] Certain diagnoses are unlikely to be made from pericardial fluid. Pericardial fluid has been shown to give false-negative cytologic results in several cases of lymphoma and mesothelioma. [29] In HIV patients, effusions caused by Kaposi sarcoma and cytomegalovirus have been diagnosed by pericardial biopsy after fluid studies were non-diagnostic. [101] [102]

An alternative diagnostic tool is subxiphoid pericardiotomy. This technique, performed in the operating suite, obtains both fluid and a pericardial biopsy specimen. It is more likely to provide a definite diagnosis and has been performed safely without general anesthesia. [103] [104] In a prospective series of 57 patients, 36% obtained a definitive diagnosis; 40%, a probable diagnosis; 16%, a possible diagnosis; and 7% remained undiagnosed with subxiphoid pericardiotomy. [105] Although it is uncertain whether this technique is safer than ultrasound-guided pericardiocentesis, published reports show a low rate of complications in experienced hands. [105]

Regardless of technique, the need to sample small effusions or obtain pericardial tissue has been questioned. A prospective series found a diagnostic rate of 6% with pericardial fluid and 5% with pericardial tissue when a small persistent effusion was sampled for the specific purpose of diagnosis. In contrast, when patients from the same population had therapeutic intervention for tamponade, the yields from fluid and tissue were 54% and 22%, respectively. [98]

The use of pericardiocentesis as a diagnostic tool in traumatic tamponade is limited. When used diagnostically to determine the presence of pericardial bleeding in trauma, the procedure has a false-negative rate of between 20% and 40%. [81] [106] [107] [108] The reason for the high false-negative rate (defined as no blood aspirated) is well demonstrated by typical stab wounds of the heart.[8] [105] Ninety-six percent of the patients had blood in the pericardium, but it was clotted in 41% of the patients and partially clotted in another 24%. In only 19% was the blood completely fluid and thus capable of giving a true-positive result on pericardiocentesis.

Therapeutic Pericardiocentesis

Tamponade of Uncertain Etiology

The primary reason for performing pericardiocentesis in the ED is as part of the treatment for cardiac arrest or in peri-arrest situations. In particular, the presentation of PEA with elevated jugular venous pressure should cause immediate consideration of pericardiocentesis. In this setting, blind, ECG-guided pericardiocentesis can be life saving. However, the overwhelming majority of patients with PEA have neither significant effusion nor tamponade, and other etiologies for the PEA also should be sought. Pericardiocentesis also may be considered in other presentations of effusion with existing or incipient tamponade.

Tamponade Caused by Nonhemorrhagic Effusions

Pericardiocentesis is often, at least temporarily, therapeutic in cardiac tamponade. Most nonhemorrhagic effusions are liquid and can be drained easily through a small needle. Removal of even a small amount of fluid can immediately and dramatically improve blood pressure and cardiac output. Pericardiocentesis relieves tamponade due to nonhemorrhagic effusions in 60% to 90% of cases. [29] [37] [44] Patients in whom it fails often have purulent pericarditis or malignant invasion of the pericardium.

Pericardiocentesis without catheter placement may be much less useful for long-term management of these patients; 26% of the patients in the study by Guberman and coworkers eventually required pericardial resection. [37] In Krikorian's series, 24% of the patients were managed successfully with one pericardiocentesis, 37% needed multiple taps or an indwelling catheter, and 39% required surgical drainage. [29] Fifty-five percent of the last group had traumatic hemopericardium.

Patients with renal failure and pericardial effusion may be better managed by dialysis than pericardiocentesis. In one series, 63% of these patients were successfully managed with dialysis alone, and only 6% needed surgical treatment over the long term. [29] Tamponade is less frequent with pericarditis when it occurs within the first months of dialysis, and such patients are much more likely to be successfully managed without invasive intervention. [46] When invasive treatment is needed for dialysis patients, pericardiocentesis is probably a poor choice; 9 of 10 patients who received it had complications in one series, and it was the only invasive treatment that resulted in death.[46]

An algorithm for the urgent management of nonhemorrhagic cardiac tamponade is shown in Figure 16-7 .

Use in Hemorrhagic Tamponade

Pericardiocentesis is never the definitive treatment in hemorrhagic tamponad[110] [111]

Although aspiration of a small quantity of fluid may cause dramatic improvement, blood usually reaccumulates. [35] [90] Thus, patients with pericardial hemorrhage ultimately require thoracotomy to explore and repair the cardiac injury.

One of the greatest potential drawbacks of pericardiocentesis in traumatic tamponade is that it may delay thoracotomy. In one study of 25 trauma patients with cardiac injury,[110] all of those who were operated on within 2 hours of injury survived, regardless of age or type of wound. With greater delay, none survived. Sugg and colleagues, in a study of 459 similar patients, found a mortality rate of 43% when pericardiocentesis was the sole treatment, but only 16% when surgery was performed.[107] Most investigators agree that with early thoracotomy and little or no reliance on pericardiocentesis, the number of deaths due to stab wounds has decreased.[81] [108] [111] [112] Sugg and associates reported that 10 of 18 patients with traumatic tamponade who were managed by repeated pericardiocentesis alone died within 1 to 2 hours.[107] At autopsy, all patients had repairable wounds.

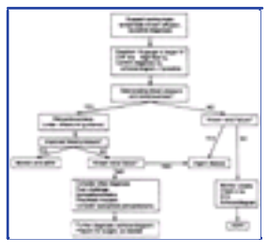

Figure 16-7 Management of *nontraumatic* cardiac tamponade. IV, intravenous line; CVP, central venous pressure; ECG, electrocardiogram.

Nonetheless, while other temporizing treatments are instituted (see discussion later in this chapter) and arrangements for definitive surgical treatment are being made, pericardiocentesis may temporarily improve the patient's hemodynamic situation (Fig. 16-8). Some clinical evidence supports the usefulness of pericardiocentesis as a temporizing measure. In a study of 174 patients with tamponade from penetrating trauma, 96 underwent operating room thoracotomy, 44 underwent ED thoracotomy, and 34 received only pericardiocentesis followed by observation. [83] Of those who underwent operating room thoracotomy, 68% were hemodynamically unstable, and preoperative pericardiocentesis decreased the mortality rate from 25% to 11%. Ninety-one percent of those who underwent ED thoractomy were unstable, and pre-thoracotomy pericardiocentesis decreased the mortality rate from 94% to 63%. For the unconscious and hypotensive or agonal patient, emergency thoracotomy is the preferred treatment (see Chapter 18).

When a trauma patient's condition is relatively stable, but a high level of suspicion for a penetrating cardiac wound is present, an alternative to thoracotomy is the subxiphoid pericardial window. [103] [113] [114] The procedure has been done under local anesthesia. Although it is possible to perform the procedure in the ED, [114] most authors believe the procedure should be reserved for the operating suite. [115] [116] [117]

CONTRAINDICATIONS

There is no absolute contraindication to pericardiocentesis. It should not be performed when better treatment modalities are immediately available (e.g., dialysis for uremic patients and immediate surgery for trauma patients). For diagnostic or non-emergent pericardiocentesis, echocardiographic or CT diagnosis is imperative. Ultrasound or fluoroscopic guidance should be used in all non-emergent situations.

EQUIPMENT FOR PERICARDIOCENTESIS

Fluoroscopic or Ultrasound Guidance

Pericardiocentesis is ideally performed in the cardiac catheter laboratory under fluoroscopic or echocardiographic guidance. In the ED, echocardiography is useful for directing pericardiocentesis. With ultrasound, the area of the heart with the greatest fluid accumulation can be accurately identified and its relationship to the body wall clarified.[118] [119] An entry site and angle of penetration can then be chosen that have the greatest likelihood of obtaining fluid while simultaneously avoiding vital structures. Ultrasonographic diagnosis and guidance is described elsewhere (see Chapter 69).

Electrocardiographic Assistance

Although the procedure can be performed with only a syringe and a spinal needle, electrocardiogram monitoring is desirable. An alligator clamp is used to connect the needle to *any* of the precordial leads (V leads) of a properly grounded electrocardiogram device (Fig. 16-9). Generally the V lead (usually V_1 or V_5), which permits a continuous display during rhythm monitoring, is used. When the alligator clamp connects the base of the pericardiocentesis needle to the V lead wire, the operator must set the machine to record the V lead as the rhythm strip.

Other Equipment

The traditional needle choice has been a 7.5- to 12.5-cm (3-to 5-inch), 18-ga spinal needle with an obturator. It is best to leave the obturator in the needle during initial passage through the skin to avoid obstruction of the needle lumen. More recently, the shorter Teflon-sheathed Intracath needle has been used. Alternatively, the clinician can use the guide

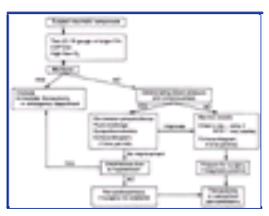

Figure 16-8 Management of *traumatic* cardiac tamponade. IV, intravenous lines; CVP, central venous pressure; ECG, electrocardiogram; R/O, rule out.

wire (Seldinger) technique, inserting a plastic catheter over a flexible guide or J wire. With this technique, an 18-ga, thin-walled needle is used for placement of the wire. The catheter (after removal of the accompanying introducer) may be left in place for prolonged drainage, if needed. [120] [121]

For drainage of blood, pus, or other viscous effusions, a large catheter such as a No. 7 to 9 Fr Cordis sheath should be inserted. [122] Alternatively, the guide wire technique can be used to insert a radiopaque, 16-ga, flexible, fenestrated, central venous catheter, which can then be connected to closed suction drainage

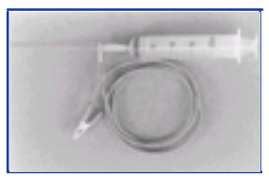

Figure 16-9 Equipment for emergent pericardiocentesis: long, 18-ga spinal needle; wire with alligator clips for connection to the electrocardiograph machine; and syringe (three-way stopcock optional). Sterile skin preparation and local anesthetic are also required.

and left in place for long periods of time. [123] Pigtail catheters with side and end holes or nephrostomy drainage catheters also can be used. [121] Multi-lumen catheter patency can be maintained by slow continuous flush with a heparinized saline solution. [121] Complete "sets" containing necessary equipment for placing a catheter using the guide wire technique are commercially available (Fig. 16-10), including sets designed for pediatric use. [124]

A three-way stopcock may be attached to the needle or catheter to allow removal of more than one filled syringe without much movement of the needle. The continuous

Figure 16-10 An example of the contents of a prepackaged pericardiocentesis set: finder needle, Seldinger wire, dilator, catheter guide, and pigtail catheter. Sterile skin preparation and local anesthetic are also required.

motion of the heart may require minor changes in needle or catheter position during the procedure. Lengthy or repeat drainage is much safer if the steel needle is withdrawn and a plastic catheter is left in place.

PROCEDURE

Temporizing Measures

While preparing for pericardiocentesis in the unstable patient or attempting to stabilize the patient while the operating suite is readied for thoracotomy or subxiphoid pericardiotomy, temporizing measures should be considered. In the patient with suspected tamponade and without jugular venous distention, the administration of a fluid bolus may improve hemodynamics.[68] In the setting of non-penetrating tamponade, a fluid challenge has been recommended [59] [125] ; animal experiments have found this to be beneficial, with or without nitroprusside for afterload reduction. [126] However, a follow-up prospective evaluation in patients with tamponade found no benefit from either fluid challenge or nitroprusside; cardiac output remained unchanged at a mean of 5.1 L/min, in contrast to 9.1 L/min after pericardiocentesis. [127] In the trauma patient with penetrating cardiac injury, fluid resuscitation may produce improvement or deterioration. Animal experiments indicate that the response depends on whether fluid infusion produces recurrent bleeding from the cardiac wound. [128] One report found that dextran solution for volume expansion produced significant hemodynamic improvement in patients with subacute ventricular free wall rupture after acute myocardial infarction. [32] In summary, judicious volume expansion may produce temporary beneficial hemodynamic results, but this is not uniformly true.

Vasopressors also have been recommended as a temporizing measure in tamponade. Dopamine, dobutamine, norepinephrine, and isoproterenol have been evaluated. Norepinephrine produced increased cardiac output in animal models of tamponade [129] [130] but failed to increase cardiac output in patients with malignant effusion.[129] Isoproterenol increased cardiac output in animal models but detrimentally affected cardiac blood flow. [129] Both dopamine and dobutamine have produced increased cardiac output and other improvements in hemodynamics in the setting of tamponade. [22] [127] Either of these agents may be helpful as a temporizing agent in tamponade, but dobutamine may be preferable on theoretical grounds because of its greater beta activity. [130]

Preparation

All necessary equipment must be checked and laid out in advance. Full resuscitation equipment must be on hand, including a defibrillator. The patient must have an IV line in place and be attached to a cardiac monitor. The non-emergent patient may require sedation, but in an emergency, pericardiocentesis is usually performed on patients who are already obtunded or unresponsive as a result of low cardiac output. Use of sedation in these patients not only is unnecessary, but also carries a high risk of hemodynamic or respiratory deterioration. Premedication of the patient with atropine may help to prevent vasovagal reactions. When possible, the presence of pericardial effusion and the optimal anatomic approach should be determined in advance by echocardiography. If surgery may be needed, preparations should already be under way to ensure prompt availability of both an operating room and a surgeon.

If the patient's clinical condition permits, the chest should be elevated at a 45° angle to bring the heart closer to the anterior chest wall. If the abdomen is distended because of gastric contents or previous positive-pressure ventilation, a nasogastric tube should be used to decompress the stomach. The entire lower xiphoid and epigastric area should be carefully prepared with 10% povidone-iodine solution and sterilely draped, if time permits.

If the patient is awake, the skin and the proposed route of the pericardial needle should be anesthetized by infiltration with 1% plain lidocaine or 0.5% bupivacaine. Note that the pericardium is very sensitive and should be anesthetized in patients who are awake. [120]

Anatomic Approach

The choice of anatomic approach in the past has been governed largely by conjecture and theory, not by actual study of patients with pericardial effusion. Traditionally the subxiphoid approach was preferred and recommended in most texts and articles as the optimal choice. However, two-dimensional echocardiography allows direct visualization in the individual patient of both the areas of maximal effusion and the location of vital structures. Studies of echocardiography-directed pericardiocentesis have found that the intercostal space near the heart apex is usually the best site for puncture, not the traditional subxiphoid approach. [119] [122] Careful cadaver studies have corroborated this finding, demonstrating greater safety with a parasternal approach in the fifth intercostal space and showing that the greatest number of injuries (usually to the right atrium) occurred with variants of the subxiphoid approach. [14] In contrast, studies of intracardiac injection using the same routes have found an increased incidence of pneumothorax when parasternal or intracostal approaches are used (see discussion of complications of pericardiocentesis). This risk may increase with underlying lung disease. Whenever time and the patient's condition permit, clinicians should rely on echocardiography to define the extent of and optimal approach to pericardial effusion. When time or circumstances prevent the use of ultrasound, the clinician should use the approach with which he or she is most familiar.

Parasternal Approach

In this approach, the needle is inserted perpendicular to the skin in the left fifth intercostal space medial to the border of cardiac dullness (Fig. 16-11). Older texts identify the puncture site as being at least 3 to 4 cm lateral to the sternal border to avoid the internal mammary artery. However, anatomic studies indicate that penetration immediately lateral to the sternum is less likely to cause this complication. [14]

Subxiphoid Approach

In the traditional subxiphoid approach, the needle is inserted between the xiphoid process and the left costal margin at a 30° to 45° angle to the skin (Fig. 16-12). Because the heart is an anterior structure, an angle greater than 45° may intercept the liver or stomach. In this approach, the needle enters the pericardium at the angle at which it becomes the diaphragmatic pericardium. Recommendations regarding needle aim vary widely, including among others the right shoulder, the

Figure 16-11 Parasternal approach for pericardiocentesis. The patient is depicted in a supine position, although a preferable position would be sitting at a 45° angle, if the patient's clinical condition permits. Note ECG monitoring via alligator clamp attached to lead.

sternal notch, and the left shoulder.[120] [125] The only anatomic study conducted demonstrated that the subxiphoid approach is likely to injure the thin-walled right atrium when one aims for the right shoulder.[14] Aiming for the left shoulder directs the needle toward either the left ventricle or the anterior wall of the right ventricle (Fig. 16-13).

Apical Approach

In the less commonly used apical approach, the needle is inserted 1 cm lateral and in the intercostal space below the apical beat, within the area of cardiac dullness; it is aimed toward the right shoulder. [120] If the apex cannot be palpated, the needle is inserted just inside the area of cardiac dullness. This area is close to the lingula and left pleural space, and pneumothorax is more frequent; a concomitant pleural effusion may be inadvertently tapped. In theory, this technique is used because the coronary vessels are small at the apex, and if a ventricle is entered, it is the thick-walled left ventricle, which is more likely to seal off any ventricular injury. Data are insufficient to say whether these theoretical advantages are clinically important. With echocardiographic guidance, the apical approach may be more commonly

used.[131]

Electrocardiogram Monitoring

After the skin has been punctured but before the pericardial needle is advanced, the needle obturator is removed, and an aspirating syringe is attached. At this time, electrocardiogram monitoring is begun. Attach a sterile electrical cord with alligator clips (see Fig. 16-9) from the pericardial needle to any precordial lead (V lead) of the electrocardiogram machine. The V lead is then recorded, as the needle becomes an "exploring electrode." The machine must be properly tested and internally grounded. Small current leaks can induce dysrhythmias.[35] The purpose of the electrocardiogram monitoring is to prevent ventricular puncture. When the needle touches the epicardium, a current-of-injury pattern, often simulating a wide complex PVC with an elevated ST segment, is noted on the electrocardiogram (Fig. 16-14). This current of injury may be local and could be missed if a lead other than a V lead is monitored or if a cardiac monitor (which has a lower

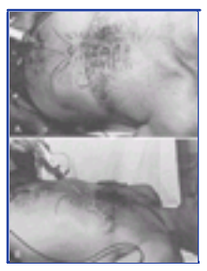

Figure 16-12 *A* and *B*, Xiphosternal approach for pericardiocentesis. The needle is aimed for the sternal notch or the left shoulder. Note the electrocardiography monitor. Although the patient is shown in a supine position, a preferable position would be sitting at a 45° angle, if the patient's condition permits. This general approach is also used for intracardiac injection of advanced cardiac life support drugs.

frequency response than the electrocardiogram machine) is used. Usually one notes ST-segment elevation on contact with the heart or pericardium in the absence of an effusion, but a premature contraction or other ventricular dysrhythmia also may be induced by direct mechanical stimulation of the ventricular epicardium by the needle. Contact with the atrium can cause atrial dysrhythmias, marked elevation of the PR segment, or atrioventricular dissociation.[16] If there is abnormal myocardial scarring secondary to infarction or other diseases or if there is malignant infiltration of myocardium, no current of injury may be generated.[17] Thus, electrocardiogram monitoring is not infallible in preventing myocardial penetration. In addition, the incessant motion of the heart makes it almost impossible to merely *touch* the epicardium.

With continuous electrocardiogram monitoring, the operator slowly advances the needle and syringe while gently aspirating. The needle will penetrate the pericardium (this is usually not palpable) at about 6 to 8 cm below the skin in adults and 5 cm or less below the skin in children.[57] The awake patient may complain of sharp chest pain as the sensitive pericardium is entered. As soon as pericardial content is aspirated, the needle should not be further advanced. If a current of injury is noted (see Fig. 16-14), the needle is touching epicardium and can easily lacerate myocardium or coronary vessels. The needle should be withdrawn a few millimeters until the current of injury disappears. At this point, the needle should be safely positioned in the pericardial space, although heart motion

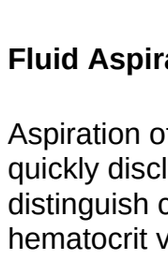

Figure 16-13 Subxyphoid approach to catheter placement into pericordial space. A short needle (16- or 18-ga) is inserted into the left xiphocostal angle perpendicular to the skin and 3 to 4 mm below the left costal margin *(A)*. After advancing the needle to the inner aspect of the rib cage, the needle's hub is depressed so that the needle points toward the patient's left shoulder. The needle is then cautiously advanced about 5 to 10 mm until fluid is reached *(B)*. The fingers may sense a distinct "give" when the needle penetrates the parietal pericardium. Successful removal of fluid confirms the needle's position. The syringe is then disconnected from the needle, and the flexible tip of the guide wire is advanced into the pericardial space *(C)*. The needle is withdrawn and replaced with a soft, multihole pigtail catheter (No. 6 to 8 Fr) using the Seldinger technique. After dilating the needle tract, the catheter is advanced over the guide wire into the pericardial space *(D)*. Once the catheter is properly positioned, aspiration of fluid should result in rapid improvement in blood pressure and cardiac output, a decrease in atrial and pericardial pressures, and a decrease in the degree of any paradoxical pulse *(E)*. Electrical alternans, if present, also decreases or disappears. *(From Spodick DH: The technique of pericardiocentesis. J Crit Illness 2:91, 1987.)*

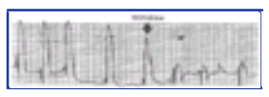

Figure 16-14 Current of injury. There is an obvious change in the electrocardiogram when the pericardiocentesis needle touches the epicardium. Following slight withdrawal *(arrow)*, the ST-segment elevation diminishes.

may quickly bring it back into contact with the myocardium. This is particularly a risk if the presence of a large effusion has not been demonstrated by ultrasound. If the scenario permits, some clinicians would now use the properly placed needle to pass a wire, then catheter, into the pericardial space, rather than attempting to drain the fluid or blood with the needle alone (see Fig. 16-13). This is a technically difficult procedure.

Fluid Aspiration and Evaluation

Aspiration of blood during pericardiocentesis raises the possibility of cardiac puncture. If fluoroscopy is available, the injection of a small amount of contrast will quickly disclose intracardiac placement. In other circumstances, the needle may need to be repositioned and the aspirate reexamined. Laboratory tests may help distinguish circulatory blood from hemorrhagic pericardial fluid. The latter should have a lower hematocrit measurement than venous blood. Substantially different hematocrit values rule out the possibility that the needle was in a cardiac chamber. Hemorrhagic pericardial fluid usually is about 0.10 pH unit lower than simultaneously obtained arterial blood.[99] Bloody pericardial fluid may clot, particularly when bleeding is brisk, so clotting of the aspirated blood does not eliminate the possibility of a pericardial source. Nonclotting blood is indicative of defibrinated pericardial blood. Practically, however, there is rarely time for such analysis.

If an indwelling catheter is to be placed, a guide wire is advanced through the needle (see Fig. 16-13). Then a dilator is passed over the wire to expand the needle tract. The guide wire should be maintained in sight and stabilized at all times. If intracardiac placement of the needle or guide wire is suspected, positioning must be verified by ultrasound or fluoroscopy or by using the techniques described earlier *before* the needle tract is dilated. Once the tract has been dilated, the pigtail catheter is placed over the guide wire. If the dilator is not used, particularly with the subxiphoid approach, the pigtail catheter tip may hang in the subcutaneous (SQ) tissue, making placement difficult.

After the catheter is placed, or if a decision is made to do a single aspiration, as much fluid as possible should be aspirated from the pericardium. The removal of even 30 to 50 mL may result in marked clinical improvement in patients with tamponade. The catheter may be placed for continuous or intermittent drainage. A chest film should be obtained after the procedure to rule out iatrogenic pneumothorax. Patients should be monitored closely for 24 hours for signs of reaccumulating fluid or iatrogenic complications from the procedure. Repeat ultrasound examination is recommended. Diagnostic evaluation of nonhemorrhagic fluid is similar to the analysis of pleural fluid (see Chapter 9).

COMPLICATIONS

The failure of pericardiocentesis to yield fluid ("dry tap") may be considered a complication, as the procedure has failed to achieve its desired result. If a dry tap is considered a complication, it is by far the most frequent one during blind pericardiocentesis. In addition, the pericardial needle can injure any organ within its reach, causing pneumothorax, myocardial or coronary vessel laceration, and hemopericardium. [112] Air embolism may be caused by air entering the heart. [132] The pericardial needle can also induce dysrhythmias from direct irritation of the epicardium or from small currents leaking from the connected electrocardiogram machine. [2]

Assessing the frequency of complication from pericardiocentesis is not straightforward. Changes in diagnosis of effusion by ultrasound or CT and guidance of the procedure by ultrasound or fluoroscopy have greatly reduced the likelihood of complication. [6] [131] [133] [134] [135] No recent data is available related to complications of blind or electrocardiogram-guided emergency pericardiocentesis. The complication rate is expected to be quite different. For example, Wong and colleagues reported that most complications occurred in patients who were found retrospectively to have no effusion. [3] The procedure is also performed frequently in moribund patients, and distinguishing between a poor outcome resulting from a poorly performed procedure as opposed to the underlying condition can be difficult.

A summation of the results of 5 recent reports on echocardiographically guided pericardiocentesis [6] [131] [133] [134] [135] includes 564 procedures with a 98% success rate. No deaths were reported. There were five incidents of pneumothorax, four reported cardiac punctures, one significant dysrhythmia, one hemothorax, one pericardial-pleural shunt and one instance of purulent pericarditis. The major complications will be discussed individually.

Cardiac Arrest and Death

Cardiac arrest and death is extremely rare in echocardiographically guided pericardiocentesis. In blind or ECG-guided pericardiocentesis, the patient is usually in full arrest and attribution of death to procedure or pre-procedure condition is nearly impossible. For example, in one series of 52 patients, the only death occurred in a patient in cardiogenic shock who had a nonproductive pericardiocentesis and who, on postmortem examination, had severe arteriosclerotic heart disease, not tamponade. [3] An additional case of cardiac arrest (successfully resuscitated) in this series was in a patient with a nonproductive pericardiocentesis; the cause of the arrest was not discussed. [3]

In a series of 352 pericardiocenteses performed under fluoroscopic guidance, only 2 deaths resulted. [4] Ultrasound or CT confirmation of effusion was used in all but 15 cases. The two deaths occurred during or after the procedure, but whether they should be attributed to the procedure is unclear. One patient with aortic rupture penetrating into the pericardial space died of cardiac arrest immediately after the puncture. The other death, in a post myocardial infarction patient with left ventricular aneurysm, was due to ventricular fibrillation that occurred about 15 minutes after the procedure.

Cardiac Chamber, Vessel, or Lung Laceration

Cardiac chamber, vessel, or lung laceration occurs more frequently during blind or ECG-guided procedures. Nonfatal cardiac puncture, pneumothorax, suppurative pericarditis, costochondritis, and pneumoperitoneum have been reported. [47] Most cardiac perforations occur in the right ventricle, but left ventricular [4] as well as atrial punctures have been reported. [16]

In Krikorian and Hancock's series, 13 of 123 patients developed hemopericardium as a result of pericardiocentesis, 1 as a result of a lacerated coronary artery. [29] One patient died from a punctured ventricle. Surgical control was necessary for four patients who developed tamponade, whereas eight patients with hemopericardium did not develop tamponade and were managed conservatively. Several cases of induced tamponade occurred in patients with platelet counts >50 × 10^9 /L.

Guberman and coworkers reported 3 right ventricular lacerations in 46 patients; 1 laceration was fatal. [37] Wong and colleagues found five right ventricular punctures, four in patients with nonproductive pericardiocentesis, but none causing any adverse sequelae. [3] In a series of dialysis patients, 9 of 10 receiving pericardiocentesis had serious complications, including 3 deaths and 2 myocardial lacerations. [46] Duvernoy and coworkers [4] reported 23 penetrations (all right ventricular except 2 in which both the right and left ventricles had been perforated), along with 4 cases of significant arterial bleeding in a series of 352 procedures.

Researchers differ in their opinions as to the adverse effects of ventricular puncture. Most ventricular punctures during the procedure occur in the lower aspect of the right ventricle. Because right ventricular pressure is lower, [57] puncture should cause less bleeding; however, the right ventricular wall is also thinner and more vulnerable to laceration. In a series of patients with ultrasound-directed pericardiocentesis, ventricular puncture still occurred in 1.5% but was without consequence due to small needle size. [122] In another study, right ventricular laceration occurred in one patient despite the use of echocardiography, producing tamponade and necessitating emergency surgery. [98] Of the 23 perforations in the series by Duvernoy and colleagues, [4] only 3 were considered "major" complications, with 2 of the patients requiring thoracotomy.

A small number of pneumothoraces and pneumopericardia have been reported in various series but have been without clinical consequence other than drainage. A single case of tension pneumothorax has been reported after pericardiocentesis, but a cause-and-effect relationship was unclear. [136]

Dysrhythmias

Serious dysrhythmias induced by pericardiocentesis are rare. Premature ventricular contractions (PVCs) occur commonly during the procedure and are benign in most cases. Most case series report no dysrhythmias. [3] [37] [47] [122] Krikorian and Hancock reported only 1 episode of ventricular tachycardia and several "hypotensive vasovagal reactions," which were associated with bradycardia and responded to atropine and fluid loading. [29] Duvernoy and colleagues [4] reported 1 case of ventricular tachycardia and 1 case of atrial fibrillation among 352 procedures. Maggiolini reported transient third-degree heart block in a single patient. [133]

Adverse Physiologic Consequences

There have been a few case reports of adverse consequences even when pericardiocentesis inflicts no injury. Most of these have to do with the fact that during pericardiocentesis, the stroke volume of the previously collapsed right ventricle increases 77% with the first 200 mL of fluid removed. [60] Generally, this increase in stroke volume is greater initially than that demonstrated by the left ventricle. This can have significant consequences for both right and left ventricular function. In three of six patients in whom large effusions were removed by pericardiocentesis, there was right ventricular dilation and overload, with abnormal septal motion and either no increase in right ventricular ejection fraction or a decrease. [137] These patients returned to normal hemodynamic status slowly.

Sudden pulmonary edema also has been reported after pericardiocentesis, presumably due to a sudden increase in venous return to the left ventricle at a time when peripheral vascular resistance is still high from compensatory cate-cholamine secretion. [138] [139] [140] Supporting evidence for this explanation is that right ventricular stroke volume increases more after relief of tamponade than the stroke volume increase of the left ventricle. [63] Circulatory collapse with persistently low arterial blood pressure has been reported in a patient who was drained of 700 mL of clear fluid at a rate of 100 mL/min. [141] These authors suggest that ketamine anesthesia may have played a role, but the relative ischemia created by tamponade, coupled with the sudden increase in left-sided preload, created a persisting imbalance. They recommend that pericardial drainage rate not exceed 50 mL/min. Given the rare occurrence of pulmonary edema or primary cardiac compromise, it is unclear that this recommendation is justified.

A case of brief profound bradycardia and rebound hypertension has also been reported after surgical relief of tamponade. [142] Such responses have not been noted in large series of patients receiving pericardiocentesis.

SUMMARY

In *nontraumatic patients,* tamponade should always be considered in the differential diagnosis of shock, especially in patients who are on anticoagulants, have had recent myocardial infarction, a history of pericardial disease, malignancy, or suspected aortic dissection or when a CVP catheter is in place. Tamponade should also be considered in the differential diagnosis when hypotension persists following closed-chest CPR or attempts at cardiac pacing. Its association with PEA should alert the clinician to the potential that tamponade exists.

In any patient with blunt or penetrating chest or upper abdominal trauma, the possibility of *traumatic tamponade* must also be considered. If clinical deterioration occurs in the ED pending operative care, temporizing pericardiocentesis should be considered if other therapy fails. When such a patient arrives with no obtainable blood pressure or in profound shock and unconscious, immediate thoracotomy and pericardiotomy are indicated after intubation. [111] [143] [144] Pericardiocentesis may cause a dangerous delay in this situation and has a low success rate.

Management of tamponade requires a sound understanding of pathophysiology; an ever-vigilant evaluation; and the knowledge of when the patient's clinical condition requires blind or ECG-guided pericardiocentesis as contrasted to the safer alternative of echocardiographically guided diagnosis and therapy.

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Chapter 17 - Artificial Perfusion During Cardiac Arrest

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The modern era of cardiopulmonary resuscitation (CPR) was introduced by Kouwenhoven and colleagues in 1960 in a classic paper that brought together the concepts of mouth-to-mouth ventilation, closed-chest compression, and external defibrillation. [1] Although CPR and advanced cardiac life support (ACLS) interventions have saved many lives, overall survival after cardiac arrest remains low. [2] [3] [4] [5] [6] [7] [8] Evolving data from both laboratory and clinical studies suggest that restoration of heart function after cardiac arrest is related to the generation of adequate coronary perfusion. [9] [10] Despite this insight, artificial perfusion is the principal weak link in the resuscitation armamentarium. Subsequently, periodic revision of the recommended standards for CPR and the development of alternative methods of CPR are revisited by international resuscitation organizations as new science becomes available. [11] This chapter updates the current understanding of the mechanism of blood flow during CPR, reviews standard and alternative techniques of CPR, and summarizes methods for improving vital organ perfusion during cardiac arrest.

BACKGROUND

The mechanism of blood flow during CPR has been the subject of debate since the 1960s. Two mechanisms for blood flow have prevailed over time: the "cardiac pump" model and the "thoracic pump" model. Controversy continues over the nature of the pump at work during external chest compression. Future studies might help resolve this controversy, but now it seems that the driving force for blood flow produced by standard external chest compression is a combination of intrathoracic pressure fluctuations and direct cardiac compression. The current concept of blood flow during CPR is based on a combination of these models and provides the theoretical basis for the newer CPR techniques.

Kouwenhoven and colleagues first proposed the traditional cardiac pump mechanism of blood flow. [1] Pressure on the chest compresses the heart between the sternum and the vertebrae, forcing blood into the arterial circulation (Fig. 17-1 (Figure Not Available)). [12] Closure of the atrioventricular valves during chest compression was thought to prevent retrograde blood flow. With the release of chest compression, the heart expands and fills with blood. This model assumes that compression of the ventricles raises intraventricular pressure, rather than intrathoracic pressure, above that of the aorta and pulmonary artery, creating a pressure gradient that generates forward blood flow. The validity of this model has been questioned almost since its introduction. Several studies of simultaneous compression and ventilation have suggested that increases in intrathoracic pressure alone can produce forward flow of blood. Weale and Rothwell-Jackson, in 1962, showed that chest compression induces almost equivalent increases in arterial and venous pressures in animals, thus challenging the cardiac pump hypothesis. [13] They hypothesized that closed-chest compression creates a generalized increase in intrathoracic pressure that is transmitted equally to the heart and intra- and extrathoracic vessels, because the atrioventricular valves remain open (Fig. 17-2 (Figure Not Available)). [12] Additional evidence for the thoracic pump model has been provided by the reported success of cough CPR. During cough CPR, perfusion is maintained by intermittent increases in intrathoracic pressure by self-induced coughing.

Although multiple studies have been published in support of each of these models, [14] it is possible that neither fully explains blood flow during CPR. An alternative explanation is that both mechanisms play a role. Maier and associates studied the effect of varying the rate, force, and duration of compressions in CPR on large dogs.[15] These investigators demonstrated that the relative contribution of the thoracic pump and direct cardiac compression models to blood flow varied with the CPR technique used. Direct cardiac compression predominated when chest compressions were delivered at higher rates (high-frequency CPR), and the thoracic pump mechanism predominated in low-momentum compression techniques, such as simultaneous compression and ventilation (SCV) CPR. In addition, Babbs and coworkers noted that the optimal technique of CPR varied with the size of the experimental animal and the size of the pad performing chest compressions. [16] Large animals in arrest were more likely to benefit from SCV CPR than smaller animals. Direct cardiac compression was proposed to play a greater role in smaller animals. Interestingly, sustained intrathoracic pressure elevations can occur during CPR in the setting of increased airway resistance. [17] The inability to adequately exhale during CPR can lead to the auto-positive end-expiratory pressure (auto-PEEP) phenomenon. The presence of auto-PEEP can adversely impact cardiac filling and cardiac output.

In summary, the mechanism of forward blood flow during closed chest compression appears to be multifactorial. Key factors include body size, chest configuration (particularly anteroposterior diameter), previous thoracic surgery, molding of the chest with continued CPR, size of hand or paddle performing chest compressions, and rate and force of chest compressions. Knowledge of the mechanisms of blood flow during CPR allows the clinician to use alternative techniques that may provide better perfusion pressures during cardiac arrest. The optimal technique for CPR may not be the same in every patient. For example, obese patients with large anteroposterior diameters may benefit more from SCV; thin patients may benefit from faster compression rates. The key to clinical implementation of these changes is being able to assess perfusion during ongoing CPR. Advances in this area are discussed later in this chapter.

INDICATIONS AND CONTRAINDICATIONS

CPR is generally indicated for all patients in cardiac arrest. One contraindication to initiating or continuing CPR is when the provider has reason to believe that resuscitation will be a futile intervention. Often, for such patients, an advance directive that outlines the patient's preferences regarding resuscitative efforts is available. At other times, the patient's condition or response to resuscitative efforts will guide subsequent therapy. Details regarding the ethics and associated laws surrounding the initiation or continuation of resuscitative efforts for specific circumstances are beyond the scope of this text.

Figure 17-1 (Figure Not Available) Cardiac pump model of cardiopulmonary resuscitation. During the relaxation phase, negative intrathoracic pressure enhances blood return to the heart. During closed-chest compression, the heart is squeezed between the sternum and the spine, a pressure gradient is developed between the ventricles and great vessels, and antegrade flow occurs because of the one-way arrangement of heart valves. RV, right ventricle; LV, left ventricle. *(From Luce JM, Cary JM, Ross BK, et al: New developments in cardiopulmonary resuscitation. JAMA 244:1366, 1980. Copyright 1980, American Medical Association.)*

A special caution is warranted regarding new implantable left ventricular assist systems that serve as bridges until cardiac transplant or as mechanisms to enhance cardiac function in nonsurgical heart failure patients. These new devices can be operated by a hand pump in the setting of battery failure. When this condition is identified by the external hydraulic line or when described by family members, these patients should be resuscitated using the hand pump or a backup electronic pump. Although these patients can be resuscitated with standard cardiac drugs or cardioversion/defibrillation as warranted, they should not receive chest compressions.

Figure 17-2 (Figure Not Available) Thoracic pump model of cardiopulmonary resuscitation. Closed-chest compression causes a generalized increase in intrathoracic pressure that squeezes all structures, including the pulmonary reservoir, which is filled during the relaxation phase. A pressure gradient is developed, and blood flows into the head, because the thick-walled carotid artery remains patent while the thin-walled jugular vein is squeezed shut, or because of a venous valve. RV, right ventricle; LV, left ventricle. *(From Luce JM, Cary JM, Ross BK, et al: New developments in cardiopulmonary resuscitation. JAMA 244:1366, 1980. Copyright 1980, American Medical Association.)*

STANDARD CPR TECHNIQUE

Guidelines for the performance of CPR have been recommended by the American Heart Association and are revised

TABLE 17-1 -- Guidelines for Cardiopulmonary Resuscitation

	Adult	Child	Infant	Neonate
Compression rate (per min)	100	100	=100	120
Compression depth	4–5 cm	1/3–1/2*	1/3–1/2*	1/3*
Compression duration	50% of cycle	50% of cycle	50% of cycle	50% of cycle
Compression mode	Both hands	Heel of one hand	Apposed thumbs†	Apposed thumbs
Compression-to-ventilation ratio	15:2 (1 or 2 rescuers)	5:1 (1 or 2 rescuers)	5:1 (1 or 2 rescuers)	5:1 (1 or 2 rescuers)

From American Heart Association: Guidelines 2000 for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 102(suppl): I1, 2000.

*Anteroposterior diameter of chest.
†May be performed with ring and middle fingers one fingerwidth below intramammary line if rescuer hands are too small.

periodically to reflect ongoing research. [11] Current guidelines are summarized in Table 17-1 .

Traditionally, external chest compression was performed two fingerbreadths above the xiphoid-sternal notch, compressing the sternum 4.0 to 5.0 cm in the normal-sized adult. A simplified method of achieving correct hand position, particularly for lay rescuers, is "in the center of the chest, right between the nipples." The pressure is released completely after each compression, and an equivalent amount of time is allotted for relaxation as for compression. The chest compression rate was previously 80 to 100 per minute. Rescuers working within the range of 80 to 100 compressions per minute will naturally drift to the lower end of the range, especially when fatigued. Therefore, the newly recommended compression rate for adult victims is 100 compressions per minute. Two ventilations are given after each 15 chest compressions in one-rescuer CPR, and 2.0 seconds are allowed for each breath in order to provide good chest expansion. For adult victims, it is now recommended that 2 rescuers also use a compression-ventilation of 15:2. The compression-ventilation ratio of 5:1 results in interruptions, which subsequently lead to a marked reduction in blood flow and blood pressure. Once the patient is endotracheally intubated, the rescuer need not stop compressions for the ventilatory pause. Rather, ventilation should be performed asynchronously at a rate of 12 to 15 per minute.

In children (1 to 8 years), compressions are performed with the heel of one hand over the lower half of the sternum, ensuring that compressions do not occur on or near the xiphoid process. The sternum is depressed approximately one third to one half of the depth of the child's chest, which corresponds to a compression depth of approximately 2.5 to 4.0 cm, at a rate of approximately 100 compressions per minute. One or two rescuers providing compressions should pause after every fifth compression and provide effective ventilation.

Chest compressions in infants (younger than 1 year old) currently are performed 1 fingerwidth below the intramammary line. The sternum is depressed with 2 to 3 fingers to a depth of approximately one-third to one-half of the infant's chest (1.25–2.5 cm). The recommended compression rate in infants is at least 100 per minute. A 5:1 compression-to-ventilation ratio is maintained for both one- and two-rescuer CPR. The preferred two-rescuer technique for performing chest compressions in the infant is the two thumb-encircling hands technique. [18] [19] Using the same location, sternal depression is performed using the two apposed thumbs while the infant's chest is encircled with the back supported by the fingers of both hands. If the rescuer's hands are too small to encircle the chest, compressions may be performed with the ring and middle fingers one fingerwidth below the intramammary line.

Previous guidelines suggested that sternal compression depth for a neonate was 1.3 to 2.0 cm. Because these recommendations were considered complex and difficult to remember, a relative gauge that is sufficient to generate a palpable pulse or about one-third of the anteroposterior depth of the chest is preferred. Use of an absolute gauge for compression depth is discouraged. Chest compressions should be coordinated with ventilation at a ratio of 5:1 with a compression rate of 120 per minute.

MECHANICAL DEVICES FOR STANDARD CPR

Mechanical resuscitators that can provide standard chest compressions and ventilations during adult CPR have been developed. Early clinical studies have demonstrated that mechanical CPR devices are comparable to standard manual CPR.[20] [21] [22] Three human studies that compared mechanical with standard CPR demonstrated improvement in the concentration of end-tidal carbon dioxide (Pet CO_2) during mechanical external chest compression. However, no survivors were reported in any study.[20] [22] [23] In addition, one animal study showed improvement in the coronary perfusion pressure and Pet CO_2 concentrations, but again, revealed no survival advantage.[24] Nonetheless, the decay in quality of chest compressions with time periods as short as 5 minutes is well recognized. [25]

The nomenclature has been changed to differentiate the various types of mechanical adjuncts to reflect automatic versus manual devices and piston-type versus belt-type devices. Piston-type chest compression devices provide an acceptable alternative to standard manual CPR in circumstances that make manual chest compressions difficult. Advantages of mechanical devices include controlled, constant chest compressions; the elimination of operator fatigue; and the freeing of personnel to perform other functions. The most commonly used device is the Thumper Cardiopulmonary Resuscitator (Michigan Instruments, Inc., Grand Rapids, MI).

The Thumper

The Thumper is a gas-powered mechanical device that is in relatively broad use. This device consists of a compressed gas-powered plunger mounted on a backboard and a time-cycled volume ventilator (Fig. 17-3).

Figure 17-3 The Thumper Cardiopulmonary Resuscitator.

The Thumper delivers chest compressions at rates consistent with American Heart Association guidelines with a compression duration that is 50% of the cycle length. Every fifth compression is followed by ventilation with an adjustable tidal volume. The Thumper can be driven by wall oxygen at 50 psi or by standard portable oxygen tanks.

Setup of the Thumper

The Thumper can be positioned from either side of the patient. Care should be taken to ensure that the base plate is positioned horizontally under the patient's posterior thorax with the patient lying near the center of the backboard. After the arm/column assembly and piston are fitted on the base plate, the piston pad position should be adjusted so that the pad lies over the lower one third of the sternum. The compressor piston is positioned after the oxygen hose has been connected; therefore, CPR is not interrupted during assembly of the Thumper. The piston column is calibrated with rings, each indicating 1.25 cm of piston excursion. Before

TABLE 17-2 -- Summary of Alternative Cardiopulmonary Resuscitation Data

Type of CPR	Mechanism	Hemodynamic Findings	Outcomes	Complications	Recommendations	Reference
IAC	Augments AP	? ROSC	? Survival*	Abdominal injury	Acceptable alt.	[35] [36] [37]
High frequency	Cardiac compression	? MAP, CO, CPP	No survival studies	= Traditional CPR	Experimental	[15] [39] [40] [41]
Vest	Thoracic pump	? CO, CPP, ROSC	? Survival†	No reported harm	Acceptable alt.	[4] [42] [43] [44] [45] [46]
AC-DC	Cardiac + thoracic	? CPP	Discrepant (see text)	Local trauma	Acceptable alt.	[7] [46] [47] [48] [49] [50] [51] [52] [53] [54] [55] [56] [57] [58] ??[59] [60] [61] [62] [63] [64] [65] [66] [67]
Chest compression only	Cardiac pump	Similar to traditional	Similar to traditional	Similar to traditional	Acceptable alt.‡	[72] [73] [74] [75] [76] [77] [78] [79] [80]
Impedance threshold	? Intrathoracic pressure	PetCO_2 , ? CPP	Under study	No add'l risk	Acceptable alt.§	[82] [83] [84]
Lifestick	Cardiac + thoracic	? ROSC	No improvement	? Sternal FX	Experimental	[85] [86] [87]

AC/DC, active compression/decompression; AP, aortic pressure; CO, cardiac output; CPP, coronary perfusion pressure; Pet CO_2 , end-tidal CO_2 ; IAC, interposed abdominal compression; MAP, mean arterial pressure; ROSC, return of spontaneous circulation; Sternal FX, sternal fracture.

*No survival benefit demonstrated in pre-hospital setting.
†Survival defined as 6 hrs postarrest. No ? in survival to discharge.
‡For lay rescuers unable or unwilling to perform mouth-to-mouth breathing.
§Only for use with active compression-decompression device.

the device is started, the piston height should be adjusted so that one ring is just visible on the piston column. With the Thumper operational, chest compressions should be adjusted to a depth of 5 to 6.25 cm or 20% to 25% of the patient's anteroposterior chest diameter, once the machine is in operation. The device should not be set to function at a predetermined chest compression force.

The ventilation hose can be connected to an endotracheal tube, an esophageal obturator, or a facemask. The Thumper uses a Patient Demand Valve (PDV) attached to the arm/column assembly and an associated tidal volume control that provides ventilation. The delivered tidal volume can be set from 400 mL to 1200 mL. The ventilation rate is preset to be synchronized with the Thumper chest compressions to provide ventilation on every fifth upstroke. The ventilator inspiratory-to-expiratory ratio is fixed at 1:2. It is pressure limited to 55 cm H_2O of airway pressure.

Use of the Thumper requires operation of 3 key switches: (1) RUN/STOP control, (2) the compression depth control, and (3) ventilation volume control. When a pause in compressions is required for other procedures, the operator can easily stop compressions by turning off the RUN/STOP control. Subclavian or jugular central lines can be placed with the Thumper in position, although it is recommended that the device be turned off during needle advancement to avoid arterial or lung injury. Defibrillation should be performed during the compression phase, when thoracic impedance is minimized.

ALTERNATIVE CPR TECHNIQUES

Because controversy continues as to the type of pump mechanism at work during chest compression, modifications of the standard CPR technique have been used in an effort to maximize coronary perfusion.[26] [27] [28] [29] [30] [31] [32] [33] [34] Proposed pumps include direct cardiac compression, intrathoracic pressure augmentation, abdominal pressure augmentation, or various combinations. The outcomes and recommendations of these alternative techniques are summarized in Table 17-2 .

Interposed Abdominal Compression CPR

The use of abdominal compressions during CPR evolved from the hypothesis that venous return might be improved and from laboratory observations that compression of the abdomen during cardiac arrest resulted in aortic pressure fluctuations similar to those with chest compressions. The technique of interposed abdominal compression (IAC)-CPR interposes abdominal compressions between chest compressions (Fig. 17-4).[35] [36] [37] CPR is performed using American Heart Association guidelines; however, during the relaxation phase, the abdomen is compressed. IAC-CPR has been studied extensively as a potential alternative to standard CPR using animal, human, and mathematical models.[38]

Four prospective randomized human clinical trials have compared IAC-CPR with standard CPR. [39] [40] [41] [42] Three of the studies involved in-hospital cardiac arrest victims, and reported improved return of spontaneous circulation (ROSC), [41] improved 24-hour survival, [41] [42] and improved survival to discharge [41] using IAC-CPR. No survival advantage using IAC-CPR has been demonstrated in the pre-hospital setting. [39]

IAC-CPR appears to be safe despite theoretical concerns over abdominal injury. Only one human case report described traumatic pancreatitis after IAC-CPR that was attributable to abdominal compression.[43] At the current time, IAC-CPR is not considered the technique of choice for external CPR because outcome benefits have been reported by only one center and the abdominal compression techniques used have not been consistent among studies. Also, use of IAC-CPR requires additional training and one additional rescuer to perform the technique. However, because of overall encouraging in-hospital results, supportive hemodynamic data, and apparent safety, the use of IAC-CPR is currently recommended as an acceptable alternative to standard CPR for

Figure 17-4 An artist's conception of basic rescuers performing interposed abdominal compression cardiopulmonary resuscitation. For clarity, both rescuers are shown on the same side of the victim. With two rescuers, the first compresses the chest and performs ventilation while the second compresses the abdomen. With three rescuers, ventilation, chest compression, and abdominal compression are performed by each individual. Ideally, the rescuer performing chest compressions is on the victim's right side and the rescuer performing abdominal compressions is on the victim's left side. *(From Voorhees WD, Niebauer MJ, Babbs CF: Improved oxygen delivery during cardiopulmonary resuscitation with interposed abdominal compressions. Ann Emerg Med 12:128, 1983. Reproduced by permission.)*

in-hospital resuscitation when sufficient personnel trained in the technique are available. [11]

High-Frequency (High-Impulse) CPR

Rapid manual CPR uses standard CPR techniques, but chest compressions are performed at a rate of 120 per minute. Animal data have shown that rates of 120 result in increased cardiac output, aortic pressure, coronary perfusion pressure, and blood flow as well as 24-hour survival. [15] [44] Swart and coworkers demonstrated that at a compression rate of 100 per minute, shorter compression duration (i.e., duty cycles less than 50% or "high-impulse" CPR) also improved resuscitation hemodynamics.[45] Kern and colleagues found that patients who had CPR with chest compression rates of 120 per minute had significantly higher levels of end-tidal CO_2 excretion (a surrogate for cardiac output) compared to patients with chest compression rates of 80 per minute. [46] Because survival studies have not been performed to clarify the effect of higher compression rates, high-impulse CPR should be considered an experimental technique and is not recommended for routine use in patients with cardiac arrest.

Vest CPR

The CPR vest was designed to take advantage of the thoracic pump mechanism of blood flow.[47] [48] [49] [50] Niemann and colleagues demonstrated hemodynamic improvement and 24-hour survival in animals receiving vest/binder CPR compared with standard CPR. [48] In contrast, Kern and coworkers found no improvement in resuscitation or 24-hour survival when vest CPR was compared with mechanical CPR.[49] Similarly, vest CPR proved to be of no benefit in one prehospital cardiac arrest study.[50] A recent study has reported improved 6-hour survival, but there was no improvement in survival to discharge. [7]

Randomized studies have not demonstrated harm with vest CPR. However, technical difficulties exist with the current device because of its size and subsequent energy requirements. The additional training and equipment requirements with CPR vests also add to the complexity of resuscitation. Despite these limitations, vest CPR is considered an acceptable alternative to standard CPR in the hospital or during ambulance transport, assuming there are an adequate number of well-trained personnel to properly perform CPR (Fig. 17-5).

Active Compression-Decompression

After publication of the initial case report in which a patient was resuscitated from cardiac arrest with the aid of a plunger, active compression-decompression (ACD)-CPR became one of the most heavily researched areas of advanced life support. [51] Standard CPR involves a forceful or active chest compression phase with elastic recoil of the chest wall during the relaxation phase (passive decompression). ACD-CPR involves both active compression and active decompression of the thorax using a device that consists of a circular suction cup with a handle and a force gauge that is applied to the midsternal chest (Fig. 17-6).[5]

At least 21 studies have evaluated the role of ACD-CPR in human cardiac arrest. [5] [51] [52] [53] [54] [55] [56] [57] [58] [59] [60] [61] [62] [63] ???[64] [65] [66] [67] [68] [69] [70] [71] Laboratory evidence has shown that ACD-CPR can decrease the venous system pressure to a greater extent than the arterial pressures during the

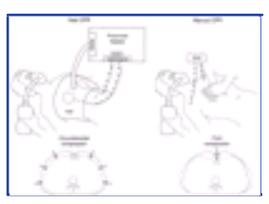

Figure 17-5 A comparison of vest cardiopulmonary resuscitation (CPR) and standard manual CPR. With vest CPR, a pneumatic system inflates and deflates a bladder surrounding the chest. The compression phase results in circumferential compression as opposed to point compression during manual CPR. The vest CPR system is equipped with defibrillator electrodes that monitor the patient's electrocardiogram as well as allow defibrillation without removal of the vest.

active decompression phase, resulting in improved venous return and increased CPR-diastolic coronary perfusion pressures. However, most human studies failed to

demonstrate long-term survival benefits. One study group demonstrated a neurologically intact survival benefit with the use of ACD-CPR. [58] However, these results should be interpreted with caution because the study investigators were unable to control for the use of ACD-CPR by basic life-support providers, and the resuscitation study team was unblinded to the technique used for external cardiac compression. Stiell and colleagues performed the most methodologically robust prospective study to date, and failed to demonstrate any significant benefits of ACD-CPR in either the basic or advanced life-support setting. [54] Despite the abundance of studies that have evaluated the role of ACD-CPR, meta-analysis of data is difficult because of inconsistent outcome definitions, inability to control for background confounders, differences in randomization schemes, and variability in prehospital response time for basic and advanced life support.

The use of ACD-CPR requires additional equipment and training. Additional concerns with ACD-CPR include difficulties with application of the technique, increased energy expenditure requirements, and mounting evidence of increased local trauma. [70] [71] However, since one center has demonstrated improvement in long-term outcomes, and most studies have not demonstrated harm, ACD-CPR has been approved as an acceptable alternative to standard CPR when rescue personnel are adequately trained to use this technique.

Chest Compression-Only CPR

Factors affecting widespread application of basic life support (BLS) techniques involve education of laypersons and acceptance of mouth-to-mouth ventilation. Current BLS techniques are difficult for laypersons to learn, retain, and correctly perform. [72] Surveys among laypersons, BLS instructors, and clinicians have reported resistance to performing mouth-to-mouth breathing for a stranger. [73] [74] [75] Correspondingly, the American Heart Association simplified the current requirements for and teaching of CPR. [11]

Data from 6 well controlled animal studies suggest that chest compression-only BLS performed by the lay public may be equally effective as standard BLS-CPR with rescue breathing using 24- to 48-hour survival as an outcome measure. [76] [77] [78] [79] [80] [81] Three important clinical trials corroborate these experimental data. Bossaert and colleagues found that when good-quality chest compression-only CPR was performed on prehospital cardiac arrest patients, there was no difference in long-term survival when compared with chest compression and ventilation CPR (15% vs. 12%). [82] Van Hoeyweghen and coworkers

Figure 17-6 The active compression-decompression (ACD) device uses a suction cup positioned at mid-chest at the level of the nipples. The device is pushed downward into the chest during the compression phase of CPR. The force of compression can be approximated by the gauge within the handle. During the decompression phase, the handle is actively pushed up and away from the chest while the suction keeps the device attached. This active withdrawal characterizes the decompression phase of CPR. The value of this adjunct is uncertain.

retrospectively found that only 10% of prehospital cardiac arrest patients who received chest compression-only CPR survived long term. This compares with a 16% survival rate for patients receiving good-quality chest compressions and ventilations, and a 7% survival rate when no bystander CPR was performed. [83] Hallstrom and colleagues reported that slightly more prehospital cardiac arrest patients survived in the chest compression-only CPR group than in the standard CPR group (14.6% vs. 10.4%). [84] Long-term survival following cardiac arrest with chest compression alone has been reported. Fifteen of the CPR-only cohorts from the Ontario Prehospital Advanced Life Support Study were discharged alive from the hospital, representing 3.6% of all study survivors. [85] However, some patients had hemodynamic compromise, rather than a true arrest, and this may have falsely inflated reported survival rates.

Continued efforts to improve and simplify BLS have led to the rationale for a "staged" approach for teaching BLS. [86] While CPR with compressions and ventilations remains the ideal method of maintaining blood flow until the arrival of emergency medical system personnel, chest compressions with an open airway at a rate of approximately 100 per minute is recommended if rescuers are unwilling or unable to perform mouth-to-mouth rescue breathing. [11]

Impedance Threshold Valve CPR

The reduction of intrathoracic pressures during ACD-CPR led to the recognition that occlusion of the airway during the chest wall decompression phase resulted in an additional decrease in negative intrathoracic pressure, and increased blood return to the chest. [65] Based on this mechanism, a small inspiratory impedance threshold valve (ITV) inserted into any respiratory circuit was developed to occlude the airway selectively during the decompression phase of CPR, without resistance to exhalation or active ventilation. [87] [88] The ITV has been shown to significantly increase vital organ flow when used with either standard CPR or ACD-CPR in a porcine model, and to decrease defibrillation energy thresholds when used together with ACD-CPR. [89] In patients undergoing ACD-CPR, the ITV cohort had significantly increased end-tidal CO_2 , systolic, diastolic, coronary perfusion pressures, and ROSC rates compared with patients treated with ACD-CPR alone. [88]

The potential benefits of the ITV are promising. However, comparative data with standard CPR and long-term survival data remain under study. Presently, the ITV is acceptable as an adjunct for use with a cardiac compression-decompression device to augment hemodynamic parameters. However, there are no data at this time to support the use of ITV with standard CPR.

Phased Chest and Abdominal Compression-Decompression (Lifestick CPR)

Lifestick CPR (Lifestick Resuscitator, Datascope Corporation, Fairfield, NJ) combines the hemodynamic advantages of IAC-CPR and ACD-CPR (Fig. 17-7). The device has adhesive chest and abdominal pads that are connected to an adjustable rigid frame with a handle at each end. The chest and abdomen are compressed in an alternating pattern by a single operator in a seesaw manner.

Tang and colleagues recently demonstrated higher resuscitation rates, 48-hour survival, and cerebral recovery with the Lifestick resuscitator compared to conventional chest compressions using a porcine model. [90] Correspondingly, using a mathematical model of adult human circulation, Babbs and colleagues demonstrated improved blood flow and systemic perfusion pressures with phased chest and abdominal compression-decompression compared to standard CPR. [91] Arntz and colleagues recently studied the feasibility, safety, and efficacy of the Lifestick compared with conventional resuscitation in a small cohort of 50 patients. [92] A

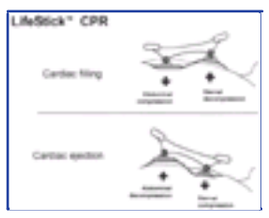

Figure 17-7 Lifestick Resuscitator used for active compression-decompression cardiopulmonary resuscitation (ACD-CPR).

significantly lower incidence of sternal fractures were found at autopsy with the Lifestick when compared to subjects receiving conventional CPR (3 vs. 9; $p < 0.05$). In addition, a higher proportion of patients with asystole or ventricular fibrillation achieved ROSC in subjects resuscitated with the Lifestick. However, no survival advantage was reported with the Lifestick over conventional CPR. [92] This method may prove to be a practical alternative to standard CPR in the future. However, large-scale clinical data are currently lacking, and the Lifestick Resuscitator remains experimental.

ASSESSMENT OF ONGOING CPR

The prognosis for resuscitation of patients in cardiac arrest depends on the no-flow interval before CPR is initiated, the time interval from collapse to defibrillation (if the rhythm is ventricular fibrillation), and the initial cardiac rhythm. Once the resuscitation efforts are initiated, there is no ideal criterion to judge the efficacy of CPR. In most animal and clinical studies, outcome (i.e., resuscitation or death) is the only criterion used. Recent studies have focused attention on assessing the effectiveness of ongoing resuscitation efforts, and identifying indices predictive of successful outcomes.

Pulses

Once considered a mainstay of CPR, the pulse check has been scrutinized for its reliability. [93] [94] Although femoral artery pulses are frequently assessed by medical personnel during CPR, accurate identification of a central pulse in a collapsed and unresponsive patient is extremely difficult. The clinician should exercise caution when making critical resuscitative decisions based on the presence or absence of a pulse. Palpating the pulse is also not an accurate means of assessing the adequacy of CPR because the pulse is more likely to represent the systolic-to-diastolic pressure gradient rather than the coronary perfusion pressure (CPP). In addition, femoral pulses perceived to be arterial may actually be venous. Blood frequently flows in a retrograde fashion to the lower half of the body during chest compression, because there are no valves in the inferior vena cava. The presence of carotid pulses may indicate forward blood flow with chest compression, but the extent of blood flow and, more importantly, tissue perfusion cannot be gauged by the presence or absence of a pulse.

Emerging data support the difficulties associated with pulse check interpretation. Flesche and colleagues studied the ability of trained medical professionals to accurately palpate central pulses in manikins as well as human models. [95] They found that fewer than 10% of medical students and trained rescuers detected an existing pulse in manikins, and it required 6 seconds to identify a pulse in healthy volunteers. Detecting pulses in collapsed and artificially ventilated patients is inherently more difficult. The carotid pulse check should under no circumstance be permitted to cause a delay in the decision to initiate CPR in an obviously lifeless person. Excessive time in locating a pulse and inaccurate determination of the presence of a pulse was reported to cause delays in chest compression or automated external defibrillator (AED) attachment in 10% of cardiac arrest patients. [11] Because of these difficulties, recent guidelines suggest that the "pulse check" should not be taught to lay rescuers.

Central Venous Oxygen Saturation

The ability to accurately predict ROSC and successful resuscitation based on surrogate markers of perfusion has been intensively studied. Mixed venous oxygen saturation obtained from pulmonary artery (S_{VO_2}) and central venous oxygen saturation (Sc_{VO_2}) have been shown to reflect the balance of systemic oxygen delivery (supply) to consumption (demand) in multiple disease states. [96] [97] [98] [99] [100] [101] The ultimate goal of artificial circulatory support is to maintain oxygen delivery as to avoid the complex cascade of events that results in cellular dysoxia and end-organ injury.

Although no single marker of perfusion is ideal, oxygen transport variables and mixed venous oxygen saturation provide the clinician with valuable physiologic information. Combined with other clinically important data (e.g., Pet_{CO_2}), mixed venous oxygen saturation can provide prognostic information as well as help guide the clinician in the post-resuscitative period. Pulmonary artery catheter placement is not necessary to analyze mixed venous blood. A number of studies have supported the use of central venous (right atrial or superior vena cava) blood for mixed venous blood (pulmonary artery) during spontaneous circulation, closed chest CPR, and circulatory failure. [102] [103] [104] [105] Emerman et al. reported no significant differences among pulmonary artery, central, and femoral venous blood gases during closed-chest CPR in animal models. [105] The close association between Sc_{VO_2} and S_{VO_2} allows placement of a central venous catheter as opposed to the more technically demanding pulmonary artery catheter.

The fundamentals of oxygen transport are beyond the scope of this text and are described in detail elsewhere. [106] Rivers and colleagues evaluated continuous central venous oxygenation saturations in 100 patients who experienced 68 episodes of cardiac arrest. [107] Central venous saturations were measured continuously by a fiberoptic catheter in the central venous location. ROSC was defined as an aortic blood pressure of at least 60 mm Hg for more than 5 minutes. Patients with a return of spontaneous circulation had a higher initial and statistically higher mean and maximal central venous oxygen saturation than those without a ROSC. No patient attained ROSC without reaching a central venous saturation of at least 30%. Furthermore, a central venous oxygen saturation of greater than 72% was 100% predictive of a ROSC.

Coronary Perfusion Pressure

As early as 1906, researchers noted the importance of achieving adequate diastolic pressure. [108] During diastole, the aortic pressure exceeds the right atrial pressure, resulting in blood flow to the coronary arteries. CPP represents the pressure gradient between aortic and right atrial pressures as measured during the relaxation phase of standard CPR. [109] [110] The resuscitation literature provides abundant evidence that CPP is the best hemodynamic predictor of ROSC. [10] [110] [111] [112] [113] [114] [115] [116] [117] A CPP of greater than 20 mm Hg in canine models and of greater than 10 mm Hg in swine is very accurate in predicting ROSC after electrical defibrillation. [115] [118] [119] The utility of monitoring CPP during human CPR has also been demonstrated. [9] Paradis and colleagues measured the CPP of 100 patients undergoing CPR. Only patients with maximal CPPs of 15 mm Hg or more had ROSC, and the fraction of

patients with ROSC was directly proportional to the CPP. Patients achieving a CPP of >25 mm Hg had a >80% likelihood of ROSC.

The traditional method of measuring CPP requires placement of a central venous line as well as an aortic arch catheter. The technical demands and time required for line placement, combined with the need for monitoring and signal amplification, make this modality somewhat limited in the everyday practice of emergency medicine. Rivers and colleagues, in an attempt to examine the validity of interchanging arterial sites, concluded that femoral artery relaxation-phase pressure was not statistically different from aortic relaxation-phase pressure. [120] Thus, substitution of a femoral artery catheter for the aortic arch line may encourage the clinical applicability of this potentially useful data.

In summary, the aortic diastolic pressure and CPP appear to be the best available criteria for assessing perfusion during CPR and correlate with survival in animal models. Although in the non-research setting, technical and time constraints associated with data retrieval may limit CPP usefulness outside of the research setting, these hemodynamic parameters appear helpful in directing resuscitative efforts.

End-tidal Carbon Dioxide Monitoring in Cardiac Arrest

The importance of Pet_{CO_2} monitoring was first recognized by Ruldolf Eisenmenger in 1929. [121] In a canine CPR model using the "biomotor," Eisenmenger very eloquently described the physiologic importance of monitoring Pet_{CO_2} and correlated subsequent values with the likelihood of "futility." Since then, researchers have made significant strides in defining the basic physiology of carbon dioxide production and removal in cardiac arrest states. [122]

Carbon dioxide is a byproduct of cellular metabolism that is eliminated primarily through the lungs. Blood transport occurs primarily as the bicarbonate ion with protein-bound transport contributing to a lesser degree. The partial pressure difference of CO_2 between the pulmonary capillaries and the alveoli accounts for the rapid diffusion of CO_2 into the alveoli. Normally, levels of alveolar carbon dioxide and therefore Pet_{CO_2} are determined by carbon dioxide production, alveolar ventilation, and pulmonary blood flow.

During low-flow states, Pet_{CO_2} levels reflect predominantly pulmonary blood flow; in cardiac arrest, the level is determined entirely by the cardiac output generated by

CPR.[123] [124] [125] [126] [127] [128] Falk and associates reported that PetCO_2 levels accurately reflected cardiac output in low-flow and no-flow states in 10 patients in an intensive care unit, thus correla ting Pet CO_2 and cardiac output.[127] Correspondingly, PetCO_2 values during CPR may offer prognostic information as to the likelihood of achieving ROSC. Sanders et al. studied the end-tidal CO_2 values in both in-hospital and prehospital cardiac arrest. [129] Resuscitated patients had higher initial Pet CO_2 levels (greater than 10 mm Hg). Over 20 minutes, Pet CO_2 levels averaged 18 mm Hg in survivors and 6 mm Hg in those who could not be resuscitated. Varon and colleagues reported similar findings; no patient in their study survived either in-hospital or prehospital cardiac arrest with a Pet CO_2 level of <14 mm Hg.[126] Levine reported that a PetCO_2 level of <10 mm Hg after 20 minutes of resuscitation in prehospital cardiac arrests with pulseless electrical activity was 100% predictive value of no ROSC.[130] Similar findings were reported by Cantineau and colleagues. [131] Thus, PetCO_2 measurement during resuscitation can be used to predict death in cardiac arrest patients with electrical activity but no pulse. Pet CO_2 has also been used to evaluate the effectiveness of chest compressions during cardiac arrest. As previously mentioned, palpation of peripheral pulses during CPR is often inaccurate. Paradis and others have demonstrated that a number of patients without palpable pulses do, in fact, have recordable blood pressures once invasive catheters are placed. [20] [132] Thus, monitoring of PetCO_2 may provide direct feedback as to the effectiveness of external chest compressions.

Measurement of exhaled CO_2 is highly accurate, whether by mainstream or side stream technology.[133] [134] [135] In mainstream sampling, a sensor is placed directly in the exhaled ventilator circuit. An infrared detector is placed in-line to afford real-time monitoring of the exhaled CO_2. Although highly accurate, error may occur if condensation accumulates on the sensor. Heated sensors have helped decrease the frequency of this problem. In a side-stream analyzer, a sample of air is aspirated to a distal sensor and analysis of the gas takes place away from the patient. This form of Pet CO_2 monitoring can be applied to non-intubated patients and is also very accurate. The disadvantages of side-stream analyzers include plugging of the sampling line from secretions and minor delays in response times. Both techniques use infrared spectrophotometry, whereby an infrared light is passed through the exhaled gas. The amount of light absorbed is proportional to the concentration of the exhaled CO_2 . Typically, most capnometers express the exhaled CO_2 as partial pressure as opposed to a percentage. The newer pH-sensitive paper exhaled CO_2 devices provide a more qualitative means of measuring exhaled CO_2 and have a role in endotracheal tube placement. Due to their semiqualitative limitations, a role in monitoring pulmonary bed perfusion is limited. However, Nakatani and colleagues found that qualitative Pet CO_2 estimates using the EASY CAP device (Nellcor, Inc., Hayward, CA) were associated with survival to hospital admission. [136] Similar results have been reported for children. [137]

When using PetCO_2 as a surrogate marker of cardiac output during arrest, one must be cautious of the pharmacologic effects of both epinephrine and bicarbonate. High doses of epinephrine given during CPR will typically result in a decrease in the Pet CO_2 level.[138] [139] The vasopressor effect of epinephrine leads to an increased afterload and a subsequent decline in cardiac output [138] ; epinephrine also decreases pulmonary blood flow and increases peripheral shunting. [140] These findings have been consistent in animal models, but not humans.[141] [142] Unlike epinephrine, bicarbonate given by bolus injection during a resuscitation results in a falsely elevated PetCO_2 . [143] Carbon dioxide production from the degradation of sodium bicarbonate is the cause of this transient but significant rise in the Pet CO_2 . When minute ventilation is held constant, the rise in Pet CO_2 is transient and lasts only a couple of minutes.

In summary, although debate continues over the exact correlation among Pet CO_2 , coronary, and cerebral perfusion pressures, [144] [145] PetCO_2 can be used as an adjunct to assess the efficacy and effectiveness of CPR. Ease of use, noninvasiveness, and the potential application in the prehospital setting make it an attractive modality. Combined with other clinically important data such as the length of down time and

duration of pulseless electrical activity, Pet CO_2 can guide the clinician in making decisions regarding the effectiveness of CPR as well as provide reassurance when to terminate resuscitative efforts. A PetCO_2 level of 10 mm Hg or less measured 20 minutes after the initiation of advanced cardiac life support accurately predicts death in normothermic cardiac arrest associated with pulseless electrical activity.

COMPLICATIONS

CPR as a clinical procedure remains a mainstay of cardiac arrest management. Although not detailed in this chapter, proper technique is essential for minimizing the risk of complications, which can include rib or sternal fractures, pneumothorax, gastric aspiration, abdominal solid organ injury, and gastric rupture. Bush and colleagues estimate that iatrogenic injuries occur in approximately 3% of children who receive CPR. [146] Because of this *low* rate of CPR-related trauma, observed injuries in children undergoing prehospital CPR may instead be the result of nonaccidental trauma. Iatrogenic injury rates with CPR in adults are several-fold more frequent. [147] [148] Although bleeding complications have been reported in patients who undergo CPR following thrombolysis, brief periods of CPR either before or after thrombolytic therapy are not contraindicated. [149] [150] [151]

SUMMARY

Regardless of the closed-chest CPR technique used, assessment of ongoing CPR efforts is desirable for rational management of patients in cardiac arrest. Hemodynamic monitoring, especially of the aortic diastolic pressure and aorta-to-right atrium perfusion gradient (CPP), currently represents the best indicator of adequate cardiac perfusion during ongoing CPR. Oxygen transport variables and mixed venous oxygen saturation provide the clinician with valuable physiologic information, and do not require the technically cumbersome placement of a pulmonary artery catheter. However, in most clinical cardiac arrest settings, it remains difficult to measure these hemodynamic parameters. Therefore, indirect indices of perfusion such as the use of Pet CO_2 may be useful in the overall evaluation of patients in cardiac arrest.

Despite considerable advances in CPR research, cardiac arrest carries a high mortality, and few survive to leave the emergency department (ED). The pervasive dilemma confronting emergency clinicians remains when to cease unsuccessful resuscitative efforts. Departmental guidelines are often based on the duration of CPR. Attempts at resuscitation often continue for prolonged periods of time and use considerable resources. Ongoing research suggests that interventions such as echocardiography and capnography may in the future yield accurate methods of predicting survival and certain death in the ED. [152]

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Chapter 18 - Resuscitative Thoracotomy

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Trauma is the leading cause of death in persons younger than 44 years old. [1] Cumulative trauma mortality has a steep curve in the initial hour after the incident, and 50% of deaths occur within the first hour. [2] Major improvements have been accomplished in the prehospital assessment, transport, and care of trauma victims. This has increased the number of patients arriving at the emergency department (ED) in various stages of shock, allowing emergency clinicians the opportunity to resuscitate patients who previously would have died at the scene. Penetrating cardiac injuries are an increasing cause of traumatic deaths in urban areas. [3] These patients, as well as others, represent a population for whom survival is occasionally possible if an aggressive approach using emergency department thoracotomy (EDT) is taken. Therefore, it is of paramount importance to recognize those patients who may respond to a resuscitative EDT. EDT is a dramatic and heroic intervention outside of the operating room and in the absence of trained cardiothoracic surgeons (Fig. 18-1). It is the rare patient who will survive this intervention, but exceedingly dismal survival rates do not negate the occasional dramatic save gleaned from EDT. EDT is more common in major university centers and Level I trauma centers, and it is not usually attempted outside this setting. Importantly, while this procedure may be in the realm of emergency medical practice, there is no standard of care that supports EDT as a procedure that must be undertaken by all emergency clinicians.

As a multidisciplinary specialist, the emergency clinician may find it necessary to perform procedures considered to be in the province of other specialties. EDT is one of these procedures and it is also one of the most invasive procedures performed in the ED. Thus there is often reluctance to perform ED and a tendency to delay its performance until a poor outcome becomes a certainty. However, the need for the emergency clinician to have an expertise in EDT is born of necessity. Few of the 6900 registered hospitals in the United States are Level I trauma centers with in-house surgical coverage, whereas nearly all hospitals provide 24-hour emergency clinician coverage. When a patient presents to the ED in extremis, an emergency clinician trained in EDT may perform this procedure without hesitation.

The focus of this chapter is threefold. The first is to discuss the indications and contraindications to the performance of an EDT. Mechanism of injury, prehospital/ED vital signs, as well as outcomes, significantly influence these decisions. The second is to discuss the pathophysiology and diagnosis of disease processes that would require such an invasive procedure. Finally, the technical aspects of performing an EDT will be discussed.

Although this chapter provides considerable detail of the rationale for and the technique of resuscitative thoracotomy, the emergency clinician should have a systematic plan *after opening the chest*. In the trauma patient receiving a resuscitative thoracotomy, the clinician seeks to relieve any cardiac tamponade; to support cardiac function (with direct cardiac compression, potential cross-clamping of the aorta to improve coronary perfusion, and internal defibrillation when indicated); and to control hemorrhage from the heart, pulmonary vessels, thoracic wall, and great vessels. Although certain circumstances may require this sequence of resuscitation to be altered, the emergency clinician should have a systemic plan prior to opening the chest. From an organizational standpoint, each institution should have guidelines for the initiation of resuscitative thoracotomy and subsequent patient care in the ED. Ideally, a plan of chest wound management after the EDT and immediate critical care should be established with the service that will provide surgical backup when members of the surgical team cannot be on site at the time of the resuscitation. With such a plan, the team approach to resuscitation allows for optimal patient care.

INDICATIONS AND CONTRAINDICATIONS

Chest Injuries (General)

The first successful thoracotomy was reported more than 100 years ago for a patient dying from a stab wound to the heart. Beall et al. [4] formally introduced EDT as a component of resuscitation of patients with penetrating chest injuries. The indications for EDT gradually expanded to include extrathoracic as well as blunt trauma. As a result, the overall survival rate became quite poor, fueling controversy over the indications for EDT. The decision to perform EDT must be made quickly

Figure 18-1 An emergency department thoracotomy is a dramatic and heroic intervention that has a very low survival rate. In the proper setting, its use can be advocated (see text). In this case of multiple gunshots, the abdomen was also opened in an attempt to cross-clamp the abdominal aorta, but no readily reversible pathology of the chest or abdomen was found.

and must be based on an understanding of which patients are likely to benefit from the procedure. The decision should be based on a realistic judgment that the patient has a chance of survival, but will not tolerate any delay in operative intervention.

The first decision point in determining which patients may benefit from an EDT is the paramedic's initial contact with the patient. [5] Important information in formulating a decision to perform an EDT includes the mechanism of injury, site of major injury and the time of injury, EMS arrival, and, when relevant, the time vital signs or cardiac electrical activity or both ceased.

An analysis of prehospital time in patients with penetrating chest trauma receiving EDT demonstrated that when times exceeded 30 minutes, no patient survived to be transferred out of the ED following EDT. For the patients with a prehospital time of less than 30 minutes, 63% were alive in the ED, although all patients subsequently died.[6]

For patients in cardiac arrest, tracheal intubation and duration of prehospital cardiopulmonary resuscitation (CPR) correlate with survival following thoracotomy. Recent observations suggest that 5 minutes of prehospital CPR approaches the limits of viability of *nonintubated* trauma patients. The value of field intubation is dramatic; the average time of CPR tolerated by intubated survivors is double that of nonintubated survivors (9.4 vs. 4.2 minutes). [7]

The type of electrical activity in conjunction with mechanism of injury is helpful in determining who may benefit from EDT. Asystole in the face of *blunt trauma* without vital signs in the field should be considered an absolute contraindication to EDT. Such patients simply do not survive, regardless of intervention. In contradistinction to many trauma centers, the Denver group[5] advocates performing EDT in those patients with blunt trauma and field vital signs if organized electrical activity is present. In the face of penetrating torso trauma, several studies have noted the presence of organized electrical activity to be associated with increased survival. [5] Those patients with a mechanism of penetrating torso injury with asystole as the presenting rhythm should still be considered as candidates for EDT. Branney et al. [5] noted 4 of 34 functional survivors who presented with this scenario. Danne et al. [8] reported a 5% survival in patients with penetrating chest trauma and asystole at ED presentation.

The mechanism of injury is of utmost importance when considering performing an EDT. In the largest EDT series to date, Branney et al. [5] reviewed 868 charts of 950 consecutive patients over 23 years. No blunt trauma patient without vital signs in the field survived after EDT. Survival following blunt trauma when field vital signs were present was 2.5%. Rhee et al.[9] examined 4620 cases of EDT from 24 studies over a 25-year period. The overall survival rate for penetrating trauma was 8.8%, while for blunt trauma it was 1.4%. Based upon these studies, the American College of Surgeons Committee on Trauma recommended *withholding EDT for patients with blunt trauma who arrest prior to ED arrival*.[10] In spite of this consensus, some centers' stance is that patients with blunt trauma with vital signs at the time of paramedic contact should still be considered for EDT. [5]

While survival remains the ultimate gauge of the effectiveness of EDT, it is appropriate to consider the quality of survival, specifically neurologic function. It is somewhat surprising that in general, survivors of EDT have good neurologic outcomes. Rhee et al. [9] reported that 280 of 303 (92.4%) patients discharged following EDT were neurologically intact. Moore et al. [11] reported the results of 146 emergency thoracotomies for mixed trauma, including 15 patients who survived resuscitation in the operating suite. Eighty percent (12 of 15) of these patients went on to become long-term survivors; 75% had full neurologic recovery. It is probably not possible to predict accurately which patients are likely to survive intact, but the Denver study demonstrated that all survivors with full neurologic recovery had respiratory efforts at the scene; in 75% of these patients, respiratory efforts were still present on arrival in the ED. The presence or absence of a palpable pulse was not an absolute prognostic indicator. Sixty-six percent of long-term survivors (11 patients with penetrating trauma and 1 with blunt trauma) had no detectable pulse on arrival in the emergency care unit.[11]

The first 24 hours following EDT will rapidly demonstrate which patients will become long-term survivors. The San Francisco experience with 168 emergency thoracotomies for mixed trauma illustrates that most patients with fatal injuries die within 24 hours. [12] Of patients surviving the first 24 hours, 80% (33 of 41) recovered and left the hospital. Full neurologic recovery occurred for 90% of these survivors. Overall, only 2.4% (4 of 168) remained severely disabled or in a persistent vegetative state. Of these 4 patients, only 1 (0.6%) lived beyond 2 months.

Cardiac Injuries—Penetrating

Those patients with vital signs following penetrating cardiac injury will present with symptoms consistent with pericardial tamponade or shock. The presentation is determined by wound size, presence of tamponade, rate of bleeding, chambers involved, and the patient's ability to compensate. Eighty percent of cardiac stab wounds result in pericardial effusion regardless of the presence of shock. [13] Tamponade may occur if the wound is less than 1 cm in size (dependent on the chamber involved) while wounds greater than 1 cm usually continue to bleed regardless of the chamber involved. Low-pressure atrial wounds usually form a thrombus before tamponade develops. The thicker-walled left ventricle may spontaneously seal stab wounds up to 1 cm in length. As little as 60 to 100 mL of blood that acutely fills the pericardium will impede diastolic filling, reduce stroke volume, reduce cardiac output, and increase catecholamine output.

Cardiac tamponade is the *decompensated* phase of cardiac function resulting from increased intrapericardial pressure (see also Chapter 16). The progression from compensated cardiac function to uncompensated tamponade can be sudden and profound. Although one may suspect tamponade based on well-described signs, the clinical diagnosis of pericardial tamponade in the unstable trauma patient is made difficult because of the combined effect of hemorrhagic and cardiogenic shock. The classic signs of Beck triad (distended neck veins, hypotension, and decreased heart sounds) have *limited diagnostic value* for acute penetrating cardiac trauma.[14] Additional signs of tamponade include tachycardia; *pulsus paradoxus*; elevated central venous pressure (CVP); persistently decreased central venous oxygen saturation, agitation and confusion (reflecting decreased cerebral perfusion); air hunger; and cold, clammy skin. Patients with a rising CVP and persistent hypotension should be presumed to have cardiac tamponade; however, there may be a poor correlation

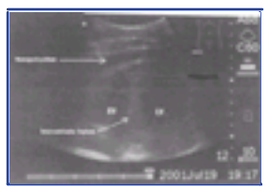

Figure 18-2 Bedside ultrasound demonstrating hemopericardium.

between CVP and the extent of tamponade, even when the blood volume has been corrected. [15]

Ultrasonography is being used with increased frequency to make the diagnosis of pericardial effusion and tamponade. Classic ultrasound findings are pericardial fluid with right atrial or right ventricular collapse during diastole. In trained hands, ultrasound is a sensitive and effective test that can be performed rapidly to assist in the evaluation of the trauma victim. The FAST examination (Focused Assessment with Sonography for Trauma) is a rapid bedside screening examination for the detection of hemopericardium and hemoperitoneum. Rozycki et al.[16] reported a sensitivity of 100% and a specificity of 97.3% for pericardial fluid using the FAST examination (see Fig. 18-2).

In addition to those factors previously discussed, survival following penetrating cardiac wounds is related to the mechanism of penetration with victims of stab wounds faring better than victims of gunshot wounds. Rhee et al.[9] noted that 16.8% of patients with stab wounds survived to hospital discharge following EDT. Branney et al.[5] reported 29% survival for stab wound victims presenting with tamponade, 15% survival in patients without tamponade, and 9% survival following absence of vital signs in the field.

In contrast, gunshot wounds are often large injuries, and are unable to seal themselves; only 20% present with tamponade. Penetrating cardiac injuries from gunshot wounds are more likely to present with profound hemodynamic compromise. Additionally, increasing popularity of larger caliber weapons has made it more difficult to resuscitate patients suffering from a gunshot wound to the chest. One study demonstrated that although 9% of patients were successfully resuscitated following a gunshot wound to the chest, all subsequently died from blast injury. [6] Of the 112 patients with penetrating gunshot cardiac wounds reviewed in the Denver study, [5] only 2% of patients survived neurologically intact.

Cardiac Injuries—Blunt

Blunt trauma to the heart ranges from minor contusion to frank cardiac rupture. Blunt cardiac rupture accounts for 5% of motor vehicle fatalities. [17] Approximately 20% of these patients will have a simultaneous aortic rupture.[6] In 1935, Bright and Beck made the observation that of 152 patients dying from cardiac rupture from blunt trauma, 30 patients lived 30 minutes or longer.[18] Fifty percent or more of patients with this injury present without vital signs to the ED. Branney et al. [5] observed a 2% survival rate for those resuscitated with EDT following blunt chest trauma. Survivors had vital signs in the field. The poor outcome associated with this type of injury may be secondary to poor cardiac function (secondary to contusion) even though mechanical causes of hemorrhage have been treated. Indications and contraindications for EDT for this mode of trauma have been discussed earlier in this chapter.

Pulmonary Injuries

Pulmonary injuries can be divided into three types: parenchymal, tracheobronchial, and large-vessel. Parenchymal and tracheobronchial injuries rarely require EDT. Most of these injuries are either rapidly fatal or can be adequately treated initially by tube thoracostomy.

Tracheobronchial injury is more common with blunt than penetrating trauma and most patients with this injury die at the scene. [19] The airway is usually maintained, even in the presence of a complete transection. The stiff tracheobronchial cartilage tends to hold the lumen open while the paratracheal and parabronchial fasciae preserve the relationship of proximal to distal bronchi. Ninety percent of tracheobronchial tears occur within 3 cm of the carina [20] and most commonly involve the main stem bronchi. Less frequently, vertical tears may occur along the membranous cartilage line of the trachea. Complete division of the trachea is extremely rare. Depending on the size and location of the injury, patients may present with massive hemoptysis, airway obstruction, and pneumomediastinum or pneumothorax, with or without tension. Massive subcutaneous emphysema and pneumomediastinum are usually seen, although up to 10% of patients with this injury initially will have no x-ray findings.[21] If hemorrhaging is profuse, or if the site of injury can be determined, the use of a bifid endotracheal tube or the unilateral intubation of a main stem bronchus will secure the airway.

Lacerations of the parenchyma unaccompanied by major vessel injuries generally respond to a tube thoracostomy. Although the associated hemothorax may be significant, re-expansion of the lung often halts or reduces bleeding. Reduction of parenchymal bleeding by negative pressure approximation of the visceral and parietal pleural surfaces is successful in 72% to 98% of cases. [22] If the initial chest tube drainage is more than 800 mL, with continued drainage at a rate of 50 mL every 10 minutes, or if there is persistent hypotension, immediate thoracotomy should be considered. Such patients are much more likely to have injuries to a major vascular structure than simple parenchymal injuries.

As with cardiac injuries, survival following EDT for pulmonary injuries is related to the mechanism of injury. Branney et al. [5] reported 17% survival following stab wounds, 3% following gunshot wounds, and 5% following blunt trauma.

Air Embolism

Presentation

Air embolism is a complication of pulmonary parenchymal injuries that requires immediate thoracotomy if there is hemodynamic instability. Until recently, the occurrence of air embolism following penetrating injuries of the lung had been

considered a rare event.[23] The preoperative and postmortem diagnosis of air embolism is difficult and it is likely that most air emboli are not detected. Air embolism is confirmed at thoracotomy by needle aspiration of a foamy air-blood admixture from the left or right ventricle or by visualization of air within the coronary arteries. Preoperative demonstration of air by aspiration from a central venous catheter or the femoral artery is rare, but it has been reported. [24]

Air embolism may appear in either the right or the left side of the circulatory system. Involvement of the right side of the circulation is referred to as *venous* or *pulmonary* air embolism. Generally, venous air is well tolerated, but death may occur when the volume of air reaches 5 to 8 mL/kg. The rate at which air moves into the circulation and the body's position are important determinants of the volume that can be tolerated. Death usually results from obstruction of the right ventricle or the pulmonary outflow tract. If the mean pulmonary arterial pressure exceeds 22 mm Hg, air may pass into the systemic circulation through a previously patent atrial foramen. Injuries of the vena cava or the right ventricle also can create portals of entry into the right circulatory system.

Air embolism involving the left side of the circulatory system is referred to as *arterial* or *systemic* air embolism. The lethal volume depends on the organs to which it is distributed. As little as 0.5 mL of air in the left anterior descending coronary artery has led to ventricular fibrillation. Two milliliters of air injected into the cerebral circulation can be fatal. Clinical manifestations of arterial air embolism are related to the involvement of the coronary or cerebral circulation. The distribution of arterial air is partly a function of body position.

Systemic air embolism following injury of the lung has only recently been described. The formation of traumatic bronchovenous fistulas creates potential entry points for air to move into the left side of the circulatory system. The only requirement is the formation of an air-blood gradient conducive to the inward movement of air. Although a lowered intravascular pressure from hemorrhage is a risk factor, the most important element in all reports of air embolism has been the use of

positive-pressure ventilation. [25]

In a review of 447 cases of major thoracic trauma, Yee et al. found adequate chart data to suggest the diagnosis of air embolism in 61 patients. [26] This incidence of 14% is remarkable in light of the small number of reported cases before 1973. [27] Blunt injury should not preclude consideration of this diagnosis, because 25% of patients with air embolism reported by Yee and coworkers had blunt trauma with associated lung injury secondary to multiple rib fractures or hilar disruption. The overall mortality was 56% (34 of 61 patients). Refractory cardiac arrest accounted for 63% of the operative deaths, with exsanguination or severe brain trauma as the cause in the remaining 37%.

The diagnosis of air embolism is easily overlooked because of the similarity of the signs and symptoms to those of hypovolemic shock. Two valuable signs that were present in 36% of patients were hemoptysis and the occurrence of cardiac arrest *after* intubation and ventilation. Additionally, postintubation circulatory and central nervous system dysfunction immediately following initiation of positive pressure ventilation and air in the retinal vessels is sufficient to make the provisional diagnosis of systemic air embolism. Focal neurological changes, seizures, and other central nervous system dysfunction in the absence of head injury are also suggestive.

Management

In those at risk for air embolism, spontaneous ventilation is preferred. A high index of suspicion with rapid control of the source of air embolism is essential. The patient should immediately be placed in the Trendelenburg (head-down) position to minimize cerebral involvement by directing the air emboli to less critical organs. Consideration may be given to isolating the injured lung if unilateral chest injury is present by selective intubation of the contralateral lung. If this step is unsuccessful, the next step should be a left anterolateral thoracotomy. The exposed thorax should be flooded with sterile saline. Peripheral bronchovenous fistulas can be identified by the bloody froth created during positive-pressure ventilation. A quick search for hilar injuries should be carried out in the patient with blunt trauma. If the source of air embolism is not readily apparent, a contralateral thoracotomy should be performed. Once the bronchovenous communication is controlled, needle aspiration of the residual air that commonly remains in the left ventricle and the aorta should be performed. If the patient is hypotensive, consideration should be given to aortic cross-clamping or occlusion. "Reflex" cross-clamping of the aorta before control of bronchovenous fistulas and removal of residual air will result in further dissemination of air to the heart and the brain.

Adjunctive Therapy

As mentioned earlier, air emboli traverse capillary beds if the blood pressure is high enough. After control of the bronchovenous fistula, a brief period of proximal aortic hypertension can be produced by cross-clamping of the descending aorta. Systemic arterial pressure should be maintained with adequate fluid resuscitation. Vasopressors such as dopamine, epinephrine, or norepinephrine may be required to increase systemic pressure in hopes to facilitate left to right passage of air bubbles. [28]

Left atrial pressure should be maintained at a high level. The ventilator inspiratory pressures should be kept as low as possible, and 100% oxygen should be used to facilitate diffusion of nitrogen from emboli. Additionally, high frequency ventilation, which allows for small volumes, has been used with success in individual patients. The most important adjunctive therapy is the use of hyperbaric oxygen.

Hyperbaric oxygen therapy is beneficial because it (1) compresses air bubbles; (2) establishes a high diffusion gradient, which greatly speeds the dissolution of the bubbles; (3) improves the oxygenation of ischemic tissues and lowers intracranial pressure; and (4) reduces the reperfusion injury that invariably follows the passage of bubbles. When it is available and logistically practical, hyperbaric oxygen therapy should be considered, *even though it may be many hours before it can be initiated*. Preferentially, treatment is begun within 6 hours of traumatic insult; however, the effectiveness of hyperbaric oxygen therapy is illustrated by cases of success and improvement even when as many as 36 hours elapsed before pressurization. [29]

Major Vascular Injuries

Mortality from major vascular injury has risen from less than 1% in 1947 to as high as 15% in recent years. [30] High-speed automobile accidents have accounted for some of this rise; however, major vessel injury is more frequent in penetrating trauma. In fact, 90% of major vascular injury is caused by

penetrating trauma, 10% by blunt trauma. Major vascular injury resulting in rapid deterioration following blunt or penetrating trauma requires the use of an EDT for diagnosis, resuscitation, and control of hemorrhage. Mavroudis et al. [31] reviewed 76 patients with thoracic vascular injury from mixed trauma that received an EDT because they were moribund or an immediate thoracotomy in the operating suite for hemodynamic instability. The three most common sites of vascular injury were the pulmonary artery (28% of cases), intercostal artery (23% of cases), and pulmonary vein (20% of cases). Aortic injuries account for only 12% of the injuries requiring immediate surgical intervention. However, even with immediate intervention, survival rates are low (14 to 29%). Air embolism was the cause of death in 18% of cases. [31] In the review by Branney et al., [5] 73 patients suffered great vessel penetrating injury with 1 survivor, while 30 patients were diagnosed with this injury from blunt trauma without any survivors.

Blunt and Penetrating Abdominal Injury

In the setting of penetrating abdominal injury, thoracotomy with cross-clamping of the thoracic aorta to control hemorrhage from the injury has been advocated. Because survival rates have been dismal for those undergoing this procedure, it is rarely performed, and its routine use is not recommended. The collective survival rate for 194 cases described in the literature is only 5%. [32] Conversely, Branney et al. [5] found that 8 of 76 (10%), of patients undergoing EDT for penetrating abdominal injury survived neurologically intact. While it is true that aortic cross-clamping does not substantially affect the rate and volume of bleeding from major venous injuries as well as from aortic collaterals, patients may benefit in other ways. The lack of blood volume following major vascular injury renders CPR ineffective. [33] Cross-clamping may reduce the volume of the vascular circuit during resuscitation and improve flow to vital organs.

Aortic cross-clamping for massive hemoperitoneum was originally conceived as a preoperative "prophylactic" procedure to prevent sudden hypotension following abdominal decompression. Applied in the operating room in this role, it is of clear benefit when systolic pressure cannot be raised above 80 mm Hg prior to laparotomy. [34] Kralovich et al. [35] studied the hemodynamic consequences of aortic occlusion in a swine model of controlled hemorrhagic arrest. Following hemorrhage and a period of apnea, animals were randomized into two groups dependent upon whether or not the thoracic aorta was occluded (blinded to the operators) for 45 minutes during resuscitation. There was no difference between groups in return of spontaneous circulation. However, the occluded aorta group experienced statistically greater impairments in left ventricular function and systemic oxygen utilization in the post resuscitation period. The animals required more volume and vasoactive therapy for hemodynamic optimization than the non-occluded animals.

Open-Chest Resuscitation for Nontraumatic Arrest

Failure to resuscitate patients from cardiac arrest is a result of (1) a delay in the onset of CPR, (2) the use of less than optimal resuscitative techniques, or (3) the intractability of the underlying disease process. Acute coronary syndromes account for nearly 500,000 deaths yearly, and approximately 350,000 of these deaths are from sudden death prior to hospital admission. At present, <10% of CPR attempts conducted outside of hospital special care units result in survival.

The first human survivor of open-chest CPR was reported in 1901. This technique of resuscitation was the method of choice throughout the first half of the 20th century. In 1960, Kouwenhoven published favorable survival rates using closed-chest CPR in humans. After further refinement by Pearson and Redding, closed-chest CPR gradually became the preferred method. Due to its ease of application, the American Heart Association recommended closed-chest CPR as the initial treatment for patients experiencing cardiac arrest.

The goal of CPR is the restoration of coronary perfusion pressure, which is the prime determinant for return of spontaneous circulation as established in animal models. Paradis et al. [36] found that a minimal coronary perfusion pressure of 15 mm Hg is required in humans to obtain return of spontaneous circulation. Although a coronary perfusion pressure of 15 mm Hg does not guarantee return of spontaneous circulation, there is 100% failure of resuscitation if this coronary perfusion

pressure is not attained.

Although there are a limited number of human studies on open-chest CPR, the hemodynamic superiority compared to closed-chest CPR is compelling. Del Guercio et al.[37] measured cardiac output during both closed- and open-chest CPR with in-hospital cardiac arrest patients. Open-chest CPR produced a mean cardiac index of 1.31 L/min per m^2 compared to 0.6 L/min per m^2 during closed-chest CPR. Boczar et al.[38] further examined 10 patients unresponsive to closed-chest CPR, and measured coronary perfusion pressure during closed-chest CPR followed by open-chest CPR. The mean coronary perfusion pressure in the closed-chest group was 7.3 mm Hg versus 32.6 mm Hg in the open-chest group. All patients obtained a coronary perfusion pressure of at least 20 mm Hg at some time during their open-chest CPR phase. This easily surpasses the minimal coronary perfusion pressure required for return of spontaneous circulation.

Although the prime objective of basic and advanced CPR is return of spontaneous circulation, maintenance of cerebral viability is also essential. Neuronal viability appears to be threatened by a cerebral perfusion pressure of less than 30 mm Hg or a cerebral blood flow of less than 15 mL/100 g per minute. The brain tolerates low flow (5 to 10 mL/100 g per minute) better than no flow or "trickle" flow (less than 5 mL/100 g per minute). Animal studies show that closed-chest CPR provides cerebral blood flow of 15% to 30% of pre-arrest values under optimal circumstances. Sterz et al.[39] used xenon-enhanced computed tomography to show that open-chest CPR generated near-baseline cerebral blood flow. An earlier study using closed-chest CPR showed global cerebral blood flow of approximately 50% of control values.

At present, the precise indication for open-chest resuscitation of nontraumatic arrests is undefined, and the procedure is not considered standard of care. However, in the setting of cardiac arrest from hypothermia, open-chest CPR may be considered. Cardiopulmonary or veno-veno bypass is the most rapid method of core rewarming, but it is rarely available on an immediate basis. Open thoracotomy with mediastinal irrigation has been used successfully for severe hypothermia with cardiac arrest. With severe hypothermia, ventricular fibrillation is resistant to chemical or electrical conversion, and

prolonged periods of closed-chest compression must be provided during the long process of rewarming.

Patients surviving neurologically intact in previously published case reports of open-chest resuscitation with direct cardiac rewarming had 30 to 180 minutes of internal massage.[40] This proves the ability of open-chest CPR to provide prolonged hemodynamic support. It should be noted that case reports also exist in which closed-chest CPR was maintained for prolonged periods of time during successful hypothermic resuscitation.[41] The rate of core rewarming with this technique can be as fast as 8°C per hour, and it preferentially rewarms the heart and lungs first. Such a rewarming sequence prevents "rewarming shock," which may occur if the extremities or abdomen rewarm first, producing metabolic and circulatory demands that exceed the capacity of the heart. Sterile saline heated in a microwave oven to 40°C can be poured slowly over the heart and into the thorax. Performing a thoracotomy for hypothermic arrest does not preclude the subsequent use of cardiac bypass. If there are no major contraindications to bypass, such as head trauma, open-chest resuscitation with mediastinal rewarming can be performed while arrangements for cardiac bypass are made.

In the setting of normothermic cardiac arrest, open-chest CPR may be considered. In spite of demonstrated hemodynamic superiority in both animal and human models of open-chest versus closed-chest CPR outcome benefit is lacking. Long-term survival is rare when the duration of closed-chest resuscitation is *continuous* for more than 30 minutes without cardiac response. The probability of long-term survival for witnessed medical arrests is approximately 86% during the first 10 minutes of resuscitation.[42] This is reduced to 30% by 16 minutes and becomes less than 1% after 30 minutes.[43] [44] This temporal survival profile is predicated on basic life support beginning within 4 minutes and advanced life support being initiated within 8 minutes of cardiac arrest.

Timing of the cardiac arrest appears to be essential in determining the outcome benefit from open-chest CPR. Most myocytes can tolerate up to 20 minutes of normothermic ischemic anoxia in vivo. In a canine study, Kern et al.[45] demonstrated a negative association between survival and duration of untreated ventricular fibrillation exceeding 20 minutes prior to open-chest CPR.

There is a paucity of human data evaluating a temporal window of effectiveness. A common theme in the literature of human open-chest CPR is that the technique was performed on the majority of patients that had no flow (unwitnessed arrest or lack of bystander CPR), and prolonged downtimes before the initiation of open-chest CPR. The clinician may consider open-chest CPR as his or her initial therapy of choice in the subgroup of patients with witnessed in-hospital cardiac arrest without significant underlying comorbidities, those with mechanical lesions, or those for whom standard CPR may be ineffective. The prehospital cardiac arrest patient who remains without a perusing rhythm after the initial defibrillation has a poor prognosis with conventional treatment. Whether open-chest CPR has a role in the management of these patients is not established and awaits randomized control trials.

EQUIPMENT

The clinician must carefully consider the instruments to be included in a resuscitation thoracotomy tray. The inclusion of too many instruments makes the tray cumbersome and delays the procedure. Nonessential instruments are best kept available in the resuscitation room in case they are needed for specific repair (e.g., Foley catheter for stellate wound tamponade).

The following items are essential for a thoracotomy tray:

- Scalpel with attached No. 20 blade
- Mayo scissors (or long Metzenbaum scissors)
- Rib spreaders
- Liebsche knife (or sternal osteotome with hammer)
- 2 tissue forceps (10 in.)
- 2 Satinsky vascular clamps
- 3 Hegar needle holders (10 in.)
- 2-0 or larger silk sutures on large-curve needle
- Teflon patches
- Suture scissors
- Aortic tamponade instrument
- Skin stapler (6 mm staples)

The following items are optional for the tray and can be supplied as needed by an assistant:

- 6 towel clips
- 4 to 6 hemostats (curved and straight)
- Metzenbaum scissors
- Right-angled clamp
- Foley catheter (20 Fr, 30-mL balloon)—sterile saline/syringe
- Chest tube (No. 30, Argyle)
- 12 lap sponges or gauze pads
- 6 towels

In addition, functioning suction and sterile suction tips, antiseptic solution, sterile gloves, a defibrillator with internal paddles, and overhead surgical lights are needed in the resuscitation room. In the unlikely event that the patient awakens during the procedure, sedative and analgesic agents should be available for sedation, amnesia, and pain control.

PROCEDURE

Preliminary Considerations

In the urban prehospital setting in which a trauma center is less than 15 minutes away, patients with penetrating thoracic injuries should receive immediate transportation with the shortest possible field time. Two studies suggest that this "scoop-and-run" approach provides a better survival rate. [46] [47]

For all trauma victims presenting to the ED with hypotension, the initial working diagnosis must be volume depletion. Other possibilities should be rapidly excluded, including tension pneumothorax, cardiac tamponade, air embolism, and neurogenic or cardiogenic shock. A useful algorithmic overview of the approach to chest trauma is shown in Figure 18-3 .

Because a large amount of blood may be lost into the chest, an autotransfusion system should be available (see Chapter 28). The use of autotransfusion has several benefits.[48] The most important advantages are (1) immediate availability of compatible, warm blood; (2) significantly higher levels of 2,3-diphosphoglycerate (DPG) than in stored blood; (3) elimination of transfusion diseases; (4) less risk of exhausting the banked supply of the patient's blood type; and (5) decreased exposure to the inflammatory mediators in stored blood.

Airway Control During Thoracotomy

Patients undergoing resuscitative thoracotomy in the ED require assisted ventilation. Airway control is best obtained

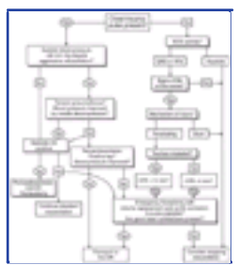

Figure 18-3 An algorithmic approach to chest trauma. QRS, organized electrical activity; VFib, ventricular fibrillation; (+)TAP, pericardial tap yielding blood; US, ultrasonography.

with standard orotracheal intubation, but exposure of the thoracic organs and surgical repairs or procedures may be hampered by frequent inflations of the left lung. Selective one-lung ventilation using a specialized double-lumen endotracheal tube is an established technique in thoracic surgery, but the availability of and experience with these devices is limited in the ED setting. The right lung often can be selectively intubated by blindly advancing a standard single-lumen endotracheal tube to a depth of 30 cm (measured at the corner of the mouth) in adult patients.[49] Although the left lung and the right upper lobe are not ventilated with the tracheal tube in this position, animal studies and preliminary data from humans suggest that selective right lung ventilation provides adequate oxygenation and ventilation for at least 60 minutes, thereby expediting the thoracotomy by minimizing the technical problems encountered by continual left lung inflation. [49]

Anesthesia and Amnesia

Comatose patients undergoing resuscitation may regain consciousness during a successful EDT, but the use of paralyzing agents may mask the return of awareness. The clinician must be cognizant of this phenomenon and administer adequate analgesia, amnestic, and muscle relaxing agents to the ventilated patient. Ketamine (2 mg/kg IV) and midazolam (0.10 to 0.2 mg/kg IV) have been recommended,[50] but ideally, agents such as etomidate with minimal effects on cardiovascular performance should be used. It would be prudent to administer anesthetic agents routinely if the paralyzed patient demonstrates perfusion during resuscitation.

Anterolateral Thoracotomy Incision

Prior to beginning the procedure, the patient should be intubated and manually ventilated. An assistant may pass a nasogastric tube, which will help differentiate the esophagus from the aorta (Fig. 18-4*A*), but the thoracotomy should not be delayed for this. If CPR is being performed, closed-chest compressions should be continued up to the point of the initial incision. Great care should be taken to avoid injury to any health care workers during performance of the incision. Universal precautions should be taken to avoid blood exposure and suction should be available to minimize contact with blood.

When the site of injury is unknown and the patient's status requires immediate intervention for possible intrathoracic injuries, a left anterolateral incision over the fifth rib with dissection into the fourth intercostal space provides the

Figure 18-4 Left anterolateral thoracotomy. *A,* Several towels or sandbags are placed under the left scapula and the arm is raised above the head. The patient should be intubated. A nasogastric tube can be inserted to facilitate differentiation of the esophagus from the aorta. *B,* The left anterolateral submammary incision is the suggested initial approach. Ideally the incision is made between the fourth and fifth ribs. Generally the incision is just inferior to the nipple (male) or along the inframammary fold (female). The incision begins on the sternum and extends to the posterior axillary line, where it should be deep enough to partially transect the *latissimus dorsi* muscle. *C, Dashes* indicate the incision site of the inframammary fold in women.

best access to the heart and great vessels. In the setting of cardiac arrest, time should not be taken to count the rib spaces. An incision just beneath the nipple in the male or along the inframammary fold in the female will approximate the fourth intercostal space (Fig. 18-4*B,C*). In patients with suspected left subclavian vessel injuries or aortic arch injuries, better exposure and control may be obtained through use of the third intercostal space.

The first sweep of the scalpel (No. 20 blade) should separate skin, subcutaneous fat, and the superficial portions of the *pectoralis* and *serratus* muscles. It is important to establish wide exposure from the outset by extending the skin incision past the posterior axillary line. To facilitate this action, quickly wedge towels or sheets under the left posterior chest and place the patient's left arm above the head (see Fig. 18-4*A*). Inadequate exposure, rib fractures, and additional delays occur when the skin incision is too limited.

Just before opening the pleura, ventilations should be stopped momentarily. This will allow the lung to collapse away from the chest wall. To enter the pleural space, a small incision is made in the intercostal muscles and one blade of the scissors is inserted. The intercostal muscles are then cut with the scissors to expose the thoracic cavity (Fig. 18-5*A*). The intercostal incision is placed just over the top of the fifth rib so as to avoid the intercostal artery. A scalpel may be used to divide the intercostal muscles but there is a greater risk of lung laceration. The use of Mayo or Metzenbaum scissors reduces this risk. Some surgeons prefer to begin the thoracotomy incision over the sternum, whereas others begin 2 cm lateral to the edge of the sternum, hoping to avoid the internal mammary artery. Should the internal

mammary artery be transected during the procedure, hemorrhage is generally minimal until after perfusion is reestablished. If bleeding actively, the internal mammary artery should be ligated or clamped.

After entering the pleural cavity and dividing the intercostals, the next step is to gain good exposure. This is accomplished by placing a chest wall retractor (rib spreader) between the ribs with the handle and ratchet bar directed downward (Fig. 18-5*B*). If the retractor is placed with the handle up, the ratchet bar will prevent extension of the incision into the right chest. Ribs may be broken during spreading, so care must be taken to avoid being cut on the sharp bone edges. If massive hemothorax is encountered, clots should be removed manually, blood suctioned, and towels used to absorb any blood that spills from the chest. When the site of injury is to the right of the heart and cannot be reached, a transsternal extension into the right chest is performed with a Liebsche knife or a sternal osteotome.

Pericardiotomy

If cardiac arrest has occurred, the question of whether to open the pericardial sac arises. If the myocardium cannot be visualized, the pericardium should be opened. However, in some cases, the myocardium can be evaluated through the intact pericardium such as a nontraumatic cardiac arrest. Nonetheless, if there is no other obvious injury in the chest and a cardiac injury is possible, the pericardium should be opened routinely,

Figure 18-5 *A*, When entering the pleural cavity, it is important to make the incision on top of the rib to avoid the intercostal vessels. Once a hole has been made into the pleural space, the incision is widened with blunt scissors by cutting the intercostal muscles. The fourth and fifth fingers of the operator's free hand are inserted into the pleural space to fend off the lung as the scissors divide the intercostal muscle. Momentary cessation of ventilation will collapse the lung. Alternatively, the right mainstem bronchus can be intubated, which permits continuous ventilation and oxygenation without inflating the left lung into the operating field. *B,* The incision must always be carried to the posterior axillary line to maximize exposure. The rib spreader should be placed with the handle laterally. Because it can be difficult to determine if tamponade has occurred using visual inspection alone, the pericardium must be opened to definitively determine if tamponade is present. Using tissue pick ups with teeth, the operator must press hard against the pericardium to engage it within the tissue pick ups. The incision is started near the diaphragm and anterior to the phrenic nerve, which is easily identified as a thick tendon-like structure. Using blunt scissors, the incision is carried to the root of the aorta.

because *it may be difficult to definitively rule out pericardial tamponade by visual inspection alone*.

If the clinician is confident that tamponade is not present, it is usually best to leave the pericardial sac closed while other life-threatening injuries are addressed. Opening the pericardium increases the risk of complications. For example, any delay in beginning cardiac compressions will add to the risk of cerebral damage. The myocardium or a coronary vessel may be injured, or the left phrenic nerve may be cut. If there has been previous pericardial disease, adhesions may be present. Attempts to separate these adhesions rapidly may result in tears of the atrial or right ventricular wall. The incidence of traumatic rupture of the atria or the right ventricle during massage is greater when the pericardium is open. With an intact pericardium, pressure is distributed over a larger area and the pericardial fluid seldom allows the compressing fingers to remain in one spot for a prolonged period.

Pericardiotomy is required if tamponade is present or suspected. This is performed in a location anterior and parallel to the left phrenic nerve. The incision begins near the diaphragm to avoid possible injury of the coronary arteries. The pericardial sac is lifted with forceps, and scissors are used to make a small hole in the sac; the scissors are then used to extend the incision in a cephalad direction along the anterior pericardium. The final incision should extend from the apex of the heart to the root of the aorta. When the pericardium is under tension, it may be difficult to grasp the pericardium with forceps. In that case, sharp, straight Mayo scissors are used to divide the pericardium by layers. If the heart is in arrest, speed is important, and sharp scissors should be used to "catch" the pericardium and to start the pericardiotomy. To do this, the point of the scissors is held almost parallel to the surface of the heart with enough pressure to create a wrinkle in the pericardium that can be punctured as the scissors are moved forward. Moderate pressure must be used to puncture the fibrous pericardium. The sudden "give" that occurs when the pericardium opens may result in a laceration of the myocardium if the point of the scissors is unnecessarily angled toward the heart. Clots of blood are removed from the pericardial sac by the sweeping motion of a gloved hand or with sterile lap sponges or gauze pads.

Direct Cardiac Compressions

Three techniques for cardiac compression have been advocated: one-handed compression, one-handed with sternal compression, and two-handed (bimanual) compression (Fig. 18-6). The *one-handed compression* method is performed with the thumb placed over the left ventricle, the opposing fingers over the right ventricle, and the apex of the heart resting in the palm of the hand. The *one-handed with sternal compression* method also is performed with the fingers flat. The fingers of the hand are held tightly together to form a flat surface over the left ventricle while compressing the heart up against the sternum. To perform the *two-handed compression* method, the left hand is cupped and placed over the right ventricle. The

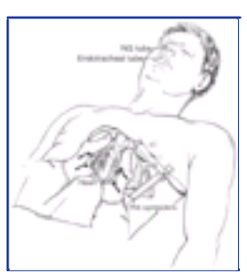

Figure 18-6 Two-handed method of cardiac massage. The ventricles are compressed toward the interventricular septum. Note how the hands flank the left anterior descending artery, which overlies the septum. Avoid using excessive finger *tip* pressure or lifting the heart, which slows ventricular filling by distorting the soft atrial caval junction.

fingers of the right hand are held tightly together to form a flat surface supporting the left ventricle. This flat surface compresses the heart against the cupped surface of the left hand. Of these three, the bimanual technique is consistently superior, and whenever possible, it is the preferred method. [51]

A difference of opinion exists regarding the optimal rate at which the heart should be compressed. Most recommend a rate of 50 to 60 compressions per minute; however, no physiologic data support such a recommendation. Johnson and Kirby studied the relationship of compression rate to cardiac output and blood pressure and found these parameters to be directly related. [52]

It is important to remember the following points while performing cardiac compression: (1) Finger *tip* pressure should be avoided at all times. Compression is performed using the entire palmar surface of the fingers. (2) Whichever technique is used, the force of compression should be perpendicular to the plane of the septum. The anterior descending coronary artery is located over the interventricular septum and is a helpful landmark to orient proper hand placement. It is clearly seen, with or without the pericardium open. (3) The fingers should be positioned so that the coronary arteries will not be occluded. (4) Venous filling of the heart is especially sensitive to changes in position. It is important to maintain a relatively normal anatomic position of the heart to *prevent* kinking of the vena cava and pulmonary veins. The heart should not be angled more than 30 degrees into the left chest. (5) It is also essential to completely relax the heart between compressions. If present, intra-arterial pressure monitoring is of tremendous value for assessing the consistency and effectiveness of compressions.

Control of Hemorrhagic Cardiac Wounds

One may partially control active bleeding from ventricular wounds by placing the finger of one hand over the wound while using the other hand to stabilize the beating heart. This maneuver buys time while the clinician prepares to repair the injury. The use of surgical staples for ventricular wound closure is an extremely rapid method for controlling hemorrhage (Fig. 18-7).[53] This technique may be particularly useful with large or multiple lacerations. Another advantage is that stapling does not expose the operator to the risk of a needle stick. Macho and coworkers reported a 93% success rate in temporarily controlling hemorrhage in 28 patients (33

lacerations) with penetrating injuries to both atria and ventricles using a standard skin stapler with wide (6 mm) staples placed at 5-mm intervals (Auto-Suture 35W, U.S. Surgical Corp., Norwalk, CT).[54] Control was rapid as 20 patients had hemorrhage control in less than 1 minute and less than 2 minutes in an additional six. The rotating long neck of the Ethicon Proximate Quantum Skin Stapler (Model PQW-35, Ethicon, Inc., Somerville, NJ) also is advantageous for obtaining proper orientation of the staples during placement. The successful use of staples has been duplicated in other centers. The staples may be left in place and reinforced or replaced on further wound exploration in the operating room.

Alternatively, the wound can be repaired by placement of several horizontal mattress sutures under the tamponading finger (Fig. 18-8). Nonabsorbable 2-0 silk sutures are customarily used. Smaller sutures should *not* be used, and nylon sutures should be avoided. Some clinicians prefer to use even larger silk sutures, such as No. 1 or 2 (note that this is *not the same as* 0 [1-0] or 0-0 [2-0] sutures). When multiple sutures are needed, they should all be in place before they are tied. This allows for a rapid and equal distribution of wound tension, which prevents tearing of the myocardium. [55] Passing the suture through Teflon pledgets also prevents the suture from cutting through the myocardium. It is especially important to use Teflon pledgets for reinforcement when the myocardium has been weakened by the blast effect of a bullet.[56] A readily available alternative to Teflon pledgets is the use of small rectangles of pericardial tissue cut from the opened pericardium.

With large wounds that cannot be palpably controlled, an incomplete horizontal mattress suture should be placed on either side of the wound (Fig. 18-9). The free ends are then crossed to stop the bleeding. The actual reparative sutures can then be accurately placed. It must be stressed that suturing the myocardium requires good technique. Excessive tension may tear the myocardium and aggravate the situation. Keys to success include the use of an appropriate-sized suture, a generous "bite" with the needle, and the application of only enough tension necessary to control bleeding.

If exsanguinating hemorrhage is not controlled by the aforementioned methods, temporary inflow occlusion can be used. Inflow occlusion may be applied intermittently for 60 to 90 seconds. During occlusion the heart shrinks, hemorrhage is controlled, and sutures can be placed in a decompressed injury. Two techniques that are useful are vascular clamping of the superior and inferior vena cava for partial inflow occlusion, [57] and the Sauerbruch grip (Fig. 18-10) for occlusion of the vena cava between the ring and the middle finger of the left hand for partial inflow occlusion. [58] The Sauerbruch grip can be performed quickly with the added advantage of cradling and stabilizing the heart during repair of wounds over either

Figure 18-7 Technique of cardiac stapling to temporarily control hemorrhage. An assistant can approximate tissues with fingertip pressure or, as illustrated, two half-horizontal sutures can be used to approximate the wound edges and reduce bleeding. A skin stapler with wide (6 mm) staples is used to place staples 5 mm apart. This technique may be used for atrial and ventricular lacerations. Following stabilization of the patient's condition, the wound is revised in the operating room.

ventricle or the left atrium. The Sauerbruch grip will only interfere with the repair of wounds involving the right atrium.

Insertion of a Foley catheter (20 Fr with a 30-mL balloon) through a wound is another technique for temporarily controlling hemorrhage. [59] Following insertion of the catheter, the balloon is inflated, the catheter is clamped to prevent air embolism, and gentle traction is applied (Fig. 18-11). Enough traction is applied to slow the bleeding to an acceptable level for visualization and repair. Attempts to achieve complete hemostasis with excessive traction may pull the catheter out and potentially enlarge the wound. The balloon will effectively occlude the wound internally. When repairing the wound, the operator must be careful with the suture needle to prevent rupturing the balloon. A purse string suture is commonly used. Temporarily pushing the balloon into the ventricular

Figure 18-8 *A*, Technique of repair. Multiple horizontal mattress sutures are placed 6 mm from the wound edge before tying. The wound is closed just enough to stop the bleeding. Teflon pledgets are used for reinforcement. Closure without pledgets incurs the risk of sutures ripping through the contracting myocardium. Similarly, the use of simple vertical sutures should be discouraged because of the risk of suture dissection through the myocardium. *B,* For repairs near a coronary artery, care is taken to pass the suture under the artery. Note that rectangles of pericardial tissue may be substituted for ready made Teflon pledgets.

lumen during needle passage is important to prevent rupture. Alternatively, the balloon can be temporarily deflated during suture placement. It is important to use normal saline when inflating the balloon. Use of air will result in air embolism if the suture needle ruptures the balloon.

Foley catheters have several advantages over other methods of controlling cardiac wounds. With the digital method,

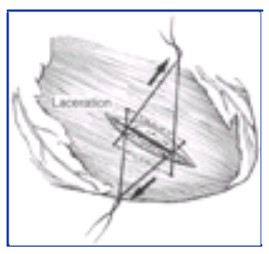

Figure 18-9 Hemorrhage control using two widely placed incomplete mattress sutures. An assistant then crosses the two "half-horizontal" sutures to bring the wound edges into apposition. By controlling the hemorrhage in this manner, the assistant's hands are outside of the operative field, fully exposing the wound edges. This facilitates a more orderly closure of the wound. Following repair of the wound, the sutures may either be removed or tied to each other.

Figure 18-10 Sauerbruch maneuver: the *method of choice* for reducing heavy bleeding from cardiac wounds. Venous inflow occlusion is achieved by using the first and second or second and third fingers as a clamp.

the fingertip will often slip if there is a strong heartbeat, the wound cannot be visualized during repair, and digital pressure significantly interferes with cardiac massage. Intermittent total venous inflow occlusion is an effective method of controlling bleeding and decompressing the heart, but such control will be at the expense of a poor cardiac output. Comparatively, the Foley catheter causes less cardiovascular interference, although inflation near the base of the ventricle may obstruct blood flow. Attempts to elevate the heart for control and repair of posterior cardiac wounds will often result in cardiac arrest by reduction of both venous and arterial flow. With posterior injuries, one cannot continuously view the wound for digital control of bleeding. Use of a Foley catheter does not require continued viewing after initial placement. If bleeding can be controlled, repairs in this location should await full volume expansion or cardiopulmonary bypass. [60] Regardless of location, the most valuable feature

Figure 18-11 Serial illustration. Gentle traction on an inflated Foley catheter will control hemorrhage and allow easy repair. The balloon is inflated with saline, and care is taken to avoid rupturing the balloon with the suture needle. This technique is particularly useful with injuries of the inferior cavoatrial junction, with posterior wounds, and during cardiac massage. Volume loading can be obtained by infusion of blood or crystalloid solutions through the lumen of the catheter. Care should be taken to avoid an air embolus through the lumen of the catheter during placement.

of Foley catheter use is the ability to control hemorrhage without interfering with cardiac compression.

Deliberate *fibrillation* (to halt myocardial contractions) should be considered as a last resort for repair of difficult wounds of the ventricle or the proximal aorta. Elective cardiac arrest is best tolerated if there is adequate blood volume and oxygenation before fibrillation. To induce fibrillation, the internal cardiac paddles are placed perpendicular ("on-edge") to the surface of the heart and are discharged at 20 J (Fig. 18-12).[57] This produces a local area of depolarization. The resulting disparity in relative refractory periods sets up a circus movement, which produces ventricular fibrillation. The heart should be massaged intermittently during repair, and the duration of fibrillation should not exceed 3 to 4 minutes.

Defibrillation is accomplished while the internal paddles are firmly pressed *tangentially* over the right and left ventricles. Following repair, the epicardium is often dry and should be moistened with saline to improve electrical conduction. An energy level of 20 J is used. If the initial attempt is unsuccessful, repeated shocks at the same setting should be used. Higher energy levels can cause myocardial necrosis. [61] Defibrillation through an intact pericardium also should begin with 20 J. If unsuccessful, the shock should be repeated once and then increased to between 40 and 60 J. Should internal paddles *not* be available, defibrillation using standard paddles placed *on the chestwall* is recommended.

Management of the wounded heart that has spontaneously arrested is controversial. Some clinicians have recommended a rapid repair of ventricular wounds while the heart is arrested. Others consider immediate cardiac massage and reversal of cardiac arrest to be more important. Immediate cardiac massage to maintain blood flow is probably the best approach. When cardiac arrest occurs, physiologic reserves have been depleted, and a delay for repair during arrest would only diminish the chance of a successful resuscitation.

Wounds of the atria are initially managed with partial occlusion clamps (Fig. 18-13). Because of the thin structure and instability of the atrial wall, digital pressure will not effectively stop bleeding. Injuries near the caval-atrial junction are not amenable to clamping. In this location a Foley catheter should be used to tamponade the wound (Fig. 18-14).[59] Care must be exercised to avoid obstruction of atrial filling with the inflated balloon. During wound closure, the catheter should

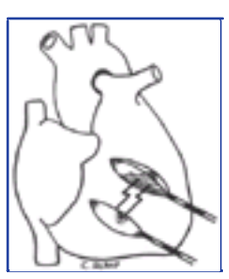

Figure 18-12 Technique for elective fibrillation: 20 J (watt-sec) is delivered through internal defibrillating paddles placed perpendicular to the epicardium. Coronary vessels should be avoided during paddle placement.

be pushed away from the ventricular wall or the balloon temporarily deflated to avoid rupture of the balloon. Skin staples also have been used for closure of atrial wounds. [54]

Wounds of the septa, valves, and coronary arteries require definitive repair in the operating suite. Hemorrhage from a coronary artery can generally be controlled with digital pressure. Ligation of a coronary artery should be avoided when possible.

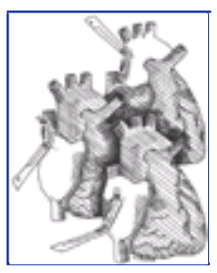

Figure 18-13 Use of a partial occluding clamp in different locations for control of bleeding and subsequent repair.

Control of Hemorrhagic Great Vessel Wounds

Wounds of the great vessels can be controlled with digital pressure or partial-occlusion clamps. Exsanguinating hemorrhage from the left subclavian artery can be prevented by cross-clamping of the intrathoracic portion of the artery. Cross-clamping of the right subclavian artery is very difficult. For injuries of this vessel, compression with laparotomy pads in the apex of the pleura from below, and the supraclavicular fossa from above (Fig. 18-15), will prevent further bleeding as the patient is stabilized and moved to the operating suite. [53]

Large or difficult vena caval injuries may be controlled with a temporary intravascular shunt to maintain venous return while providing vascular isolation of the injured segment.[62] This is a difficult and time-consuming procedure that is best done in the operating room, if the patient survives long enough.

Occasionally, fluid resuscitation can be accomplished by infusing fluid directly into the right atrium. The usual technique for placing atrial catheters using a purse string suture in the right atrium has several disadvantages. First, it is relatively time-consuming for a patient in cardiac arrest, and cardiac massage is often interrupted to allow suture placement. In addition, the suture may tear the atrial appendage, the suture holes are frequently associated with fluid leaks through the thin atrial wall, and the catheter can slide out of place. Samuelson described an innovative technique utilizing an umbilical cord clamp with a center hole (Fig. 18-16*A*). A standard umbilical cord clamp (Hollister, Inc., Libertyville, IL) is

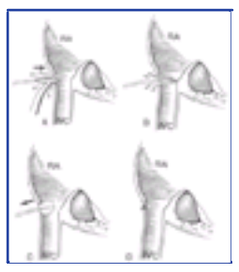

Figure 18-14 Wounds of the inferior cavoatrial junction are difficult to manage with simple vascular clamping. *A–D,* Use of a Foley catheter provides satisfactory control.

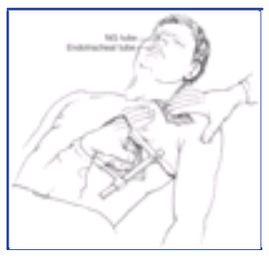

Figure 18-15 Cross-clamping for control of subclavian bleeding is difficult and time-consuming. Compression with laparotomy pads in the apical pleura from below and the supraclavicular fossa from above will control hemorrhage while the patient's condition is stabilized and the patient is transported to the operating room.

modified by drilling a hole through the center to match the size of standard IV tubing or whatever catheter is to be used. [63] The diameter of the hole should hold the catheter firmly enough to prevent slippage. These clamps, by necessity, must be prepared in advance and gas sterilized.

To place a catheter using this technique, the right atrial appendage is gently grasped between the left thumb and index finger. Scissors are used to create a small opening in the atrial appendage, and a previously flushed catheter is inserted into the right atrium. The umbilical cord clamp is placed over the catheter at the edge of the appendage and snapped shut (Fig. 18-16*B*). The cord clamp holds the catheter securely, provides good hemostasis, and does not interfere with the operative field.

Aortic Cross-Clamping

When the systolic pressure cannot be raised above 70 mm Hg, temporary occlusion of the descending thoracic aorta can maintain myocardial and cerebral perfusion (Fig. 18-17).[35] Kravolich et al. suggests that the benefit of significant improvement in coronary perfusion pressure may be overstated since both occluded and non-occluded groups attained the necessary increase in this parameter and return to spontaneous circulation. Selective clamping is necessary when the aorta has been injured with blunt trauma (Fig. 18-18). Aortic occlusion has a limited role in controlling hemorrhage below the diaphragm. When there is a tense abdomen with massive hemoperitoneum, aortic cross-clamping is clearly beneficial when applied just before laparotomy. This has been referred to as *prophylactic cross-clamping* to prevent a sudden drop in blood pressure when the abdomen is decompressed. [64] As a preoperative procedure, cross-clamping should be applied when the systolic pressure is less than 80 mm Hg in the setting of a tense abdomen.

Figure 18-16 *A*, Modified umbilical cord clamp. *B,* Modified umbilical cord clamp securing a catheter in the right atrial appendage.

The aorta lies immediately anterior to the vertebrae, actually lying on the vertebral bodies themselves. The esophagus lies anterior and slightly medial to the aorta. To expose the descending aorta, the left lung is retracted in a superomedial direction by an assistant. To achieve adequate exposure, it is sometimes necessary to divide the inferior pulmonary ligament (Fig. 18-19). The aorta can be quickly identified by advancement of the fingers of the left hand along the thoracic cage toward the vertebral column. On some occasions, the operator may choose to have an assistant simply occlude the aorta with digital pressure. Because both the aorta and esophagus are covered on their anterolateral surface by mediastinal pleura, the pleura must be opened and the aorta bluntly dissected away from the esophagus prior to clamping. To locate the aorta, use a DeBakey aortic clamp or a curved Kelly clamp for blunt dissection and spread open the pleura above and below the aorta (Fig. 18-20). The esophagus, which lies medially and slightly anteriorly, is separated from the vessel. It may be difficult to separate the esophagus from the aorta by feel in a hypotensive or cardiac arrest situation. A nasogastric tube passed from above may help identify the esophagus. When the aorta is completely isolated, the index finger of the left hand is flexed around the vessel and a vascular clamp is applied with the right hand. The brachial blood pressure should be checked immediately after the occlusion. If the

Figure 18-17 Manual cardiac massage and cross-clamping of the aorta to increase coronary and cerebral perfusion selectively.

systolic pressure is more than 120 mm Hg, the clamp should be slowly released and adjusted to maintain a systolic pressure of 120 mm Hg. [34]

Given the need for speedy intervention, the simplest and most desirable approach to aortic occlusion is to have an assistant digitally compress it or use the aortic tamponade instrument (Fig. 18-21). The application of vascular clamps risks significant complications, including injury to the vessel or adjacent structures, inadvertent dislodgment, or inadequate occlusion as a result of improper application. The aortic tamponade instrument, however, may be applied blindly to the vertebral column, permitting safe, quick, and complete aortic occlusion. [65] This technique may be most prudent when isolation of the aorta is difficult. The instrument's unique shape allows it to remain in place and to provide atraumatic occlusion with little interference in the operative field compared to digital compression. The degree of occlusion can be varied by the amount of pressure exerted by the operator.

Potential complications of aortic cross-clamping are multiple: ischemia of the spinal cord, liver, bowel, and kidneys. In addition, iatrogenic injury of the aorta and the esophagus may occur. Failure to monitor blood pressure every 60 seconds during aortic occlusion may result in cerebral hemorrhage or

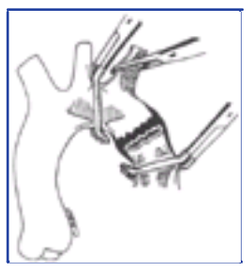

Figure 18-18 Traumatic rupture of the aorta. Three clamps are required for control. Back-bleeding will occur if fewer than three clamps are used.

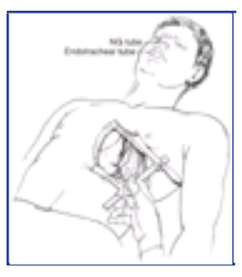

Figure 18-19 Adequate exposure of the descending aorta may require division of the inferior pulmonary ligament.

left ventricular failure if pressure elevation is excessive. Fortunately, these complications are infrequent. In a report of 12 patients surviving EDT with cross-clamping

for as long as 60 minutes, no lasting impairments of renal, myocardial, or neurologic function were noted. [66] Whenever possible, the aorta was unclamped for 30 to 60 seconds every 10 minutes to increase renal perfusion. Final release of the aorta is always performed gradually.

INTERPRETATION AND HEMODYNAMIC MONITORING

Following EDT, the systolic blood pressure after the first 30 minutes of resuscitation may be used as a decision point for further treatment. A report of EDT for blunt and penetrating trauma demonstrates the relationship between blood pressure at 30 minutes and eventual outcome. [11] Of the 146 cases reviewed, 45 patients (31%) were transferred to the operating room following initial resuscitation and aortic cross-clamping when necessary. For those patients who survived with full neurologic recovery, the average systolic blood pressure after the first 30 minutes of resuscitation was 110 mm Hg. In those who were long-term survivors but had significant brain damage, the average systolic blood pressure was 85 mm Hg. No survivals were recorded when the mean systolic blood pressure was less than 70 mm Hg. Thus, the blood pressure response to EDT appears to be predictive of survival. For patients who remain lifeless with systolic blood pressures below 70 mm Hg despite control of hemorrhage, volume replacement, and cross-clamping for 30 minutes, Moore et al. [11] recommend that heroic measures be discontinued. Transfer of these patients to the operating room for definitive repair of these mortal wounds would be futile.

Figure 18-20 *A*, Identification of the aorta: in the posterior mediastinum the aorta lies directly anterior to the vertebral bodies. The esophagus is anterior and slightly medial to the aorta. In the lower thorax, both are covered on the anterolateral surface by mediastinal pleura, which must be dissected prior to isolating the aorta for cross-clamping. *B,* Aortic cross-clamping: Using blunt dissection, one spreads the pleura above and below the aorta. The vessel should be fully mobilized and clearly separated from the esophagus before clamping. It may be difficult to differentiate the aorta from the esophagus. The aorta is the more posterior structure and is in contact with the vertebral bodies. Passage of a nasogastric tube from above will often aid the rapid identification of the esophagus.

COMPLICATIONS

A variety of significant complications occur in patients surviving EDT, but most of these are related to the primary injury rather than the thoracotomy. Proper performance of the technique should avoid injury to intra-thoracic structures. Particular attention must be paid to the position of the left phrenic nerve and coronary arteries during pericardiotomy and repair of cardiac wounds. Infection of the wound and thoracic cavity is always a concern given the fact that the procedure is performed out of the operating room, under emergent conditions, and often by clinicians relatively inexperienced in the technique. Surprisingly, serious infection is uncommon. In a combined series of 142 EDTs, there were no reports of wound infections. It should be noted that most patients received antibiotics just before or during the procedure. As a general rule, excessive attention to antiseptic skin preparation should be avoided as it may unnecessarily delay performance of the procedure.[67] Antibiotics should be administered as soon as possible.

Another potentially serious complication of EDT is injury or disease transmission to health care workers. In an emotionally charged environment in which many clinicians are attempting to perform life-saving surgery under the harshest of conditions, it is easy to suffer a needle stick or scalpel or scissor injury. Seroprevalence of human immunodeficiency virus (HIV) in U.S. EDs is estimated to range from 2% up to 6% to 9% in urban areas.[68] Tardiff et al.[68] noted an HIV-positive rate of 7.2% in their study of trauma patients presenting to their urban ED.

Occupational exposure to both hepatitis B and C is of concern to health care workers, as well. Sloan et al. [69] found a 3.1% incidence of hepatitis B in trauma patients brought to their inner-city ED. Hepatitis C is now the most common viral hepatitis seen in health care workers since the advent of the hepatitis B vaccine. Approximately 2200 health care workers per year seroconvert following occupational exposure. One urban ED reported an 18% prevalence of hepatitis C in its ED patients.[70] Additionally, patients with penetrating trauma appear to be of greater risk of any type of occult infection. Clearly, the risk of exposure to staff must be kept in mind, and universal precautions should be rigorously followed. In addition, clinicians performing this procedure must be constantly attentive to their own safety and those of their coworkers. Although EDT must be performed rapidly in order to be of value to the patient, excessive haste is not warranted if it threatens the health of members of the medical team.

CONCLUSION

The growing incidence of penetrating trauma coupled with advances in prehospital care has increased the number of patients arriving to the ED requiring advanced resuscitation. For selected patients, an EDT may be life saving, although survival rates are minimal. Resuscitative thoracotomy is

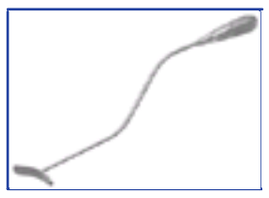

Figure 18-21 The use of a Conn aortic compressor is the method of choice for aortic occlusion because it is fast, does not interfere with the operative field, and is associated with minimal risk of injury. Alternatively, the more awkward technique of direct digital occlusion can be used. *(Courtesy of Pilling Company, Ft. Washington, PA.)*

usually attempted only in major trauma centers where surgical backup is immediately available or in smaller hospitals where a plan for post-thoracotomy care has been developed. The mechanism of injury in conjunction with the presence or absence of vital signs both at the scene and in the ED should be considered before an EDT is performed. Victims of penetrating trauma who had any vital signs at the scene are potential candidates for resuscitative thoracotomy and have a chance of survival. This procedure is most effective when it is used to prevent, rather than treat, a cardiac arrest. In contrast, the survival rates for victims of blunt trauma are negligible. With blunt trauma, resuscitative thoracotomy generally should be considered only when vital signs are still present on arrival at the emergency care unit.

Factors closely associated with patient outcome following resuscitative thoracotomy include: mechanism of injury; electrocardiographic activity; presence or absence of vital signs including neurologic function at the scene and on ED admission; and systolic blood pressure after aortic cross-clamping, if performed. The following points should be considered: (1) blunt trauma victims who lose their vital signs en route to the ED rarely survive despite thoracotomy, and they generally should not be candidates for resuscitative thoracotomy, (2) penetrating trauma victims who lose their vital signs en route to the ED may still survive and should receive an immediate thoracotomy, (3) further efforts generally should be discontinued following thoractomy when traumatic arrest patients do not exhibit cardiac activity or tamponade, (4) consider discontinuing further efforts following thoracotomy if the systolic pressure cannot be raised above 70 mm Hg after 30 minutes of maximum management.

Debate regarding *who* should perform an EDT is not necessary. It stands to reason that whoever performs this resuscitative procedure must be credentialed for the technical and critical care aspects of patient management. It is currently *not* a standard of care that all EDs have the capability of performing an EDT in the absence of surgical backup. Because aortic cross-clamping is only temporizing and should not exceed 30 minutes, rapid transfer of the thoracotomy patient to the operating room is desirable. Whenever possible, a plan of chest wound management and post-thoracotomy critical care should be established with the emergency clinician's surgical backup. With such a plan, a team approach to resuscitation and, hence, optimal patient care, is possible.

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Section IV - Vascular Techniques and Volume Support

Chapter 19 - Pediatric Vascular Access and Blood Sampling Techniques

Marie M. Lozon

The tasks of sampling blood and obtaining vascular access in an infant or child can challenge and frustrate even the most skilled emergency clinician. In some instances, venous access simply cannot be obtained within the desired time frame, making vascular access one of the most difficult clinical procedures during resuscitation and treatment of the critically ill child. The use of invasive monitoring techniques with arterial and central venous catheters is commonplace in contemporary pediatric emergency and intensive care, but simple blood sampling and venous access remains a formidable challenge in the emergency department (ED).

This chapter reviews the basic principles and techniques of blood sampling, as well as selection and placement of intravenous (IV) and intra-arterial catheters in infants and children, including those placed in the central circulation. The use of umbilical catheters in newborns is also reviewed. Although rarely required, emergency cutdown is occasionally life saving, and a section of the chapter is devoted to this technique.

PATIENT PREPARATION AND RESTRAINT

Fear and anticipation of pain associated with procedures or injections makes the hospital experience traumatic for children. Before beginning any painful procedure in a child, the procedure itself and the reasons for it should be explained to the parents. In children capable of understanding, the procedure should be explained in developmentally appropriate language before starting and prior to each successive step. The use of deceptive phrases such as "This won't hurt" should be avoided. A gentle, honest explanation that the procedure will hurt a bit and "it is okay to cry, but not to move" will provide realistic expectations for the child and set limits as well. Depending on the situation, the parents should have the option to remain with their child during the procedure. [1] If they remain in the room, their role should be solely to provide comfort to the child and not to assist in any potentially painful procedure. Distracting the child with simple conversation regarding school, friends, hobbies, pets, or television shows can also decrease the level of the child's anxiety. Despite their seeming composure, the potential for parents to faint at the site of blood or needles should always be addressed. Parental injury under such circumstances can be a source of litigation against the clinician.

The success of blood sampling or obtaining vascular access is dependent on proper positioning and restraint of the patient. In most cases, this requires the assistance of at least one other staff person. For most procedures this entails restraint of the extremity a joint above and below the intended insertion site. Although most of these procedures can be performed quickly, sometimes a significant amount of time is required to complete venipuncture or cannulation in a neonate, during which time he or she can become cold from being exposed on the examination table (especially if very ill). Overhead lights or a warming bed are very useful for this age group to prevent accidental hypothermia.

In recent years, products to provide topical anesthesia prior to needle sticks have become available. Parents are often aware of these products and may ask for them for their children. While theoretically attractive, such measures are rarely practical in the acute care setting. In an emergency situation, waiting even a few minutes for IV access may not be possible, but in the appropriate clinical setting, spending the additional time to relieve pain and anxiety in certain patients is warranted. The topical use of lidocaine-prilocaine (also known as entectic mixture of local anesthetics or EMLA) cream has a proven track record for relieving pain associated with needle puncture.[2] However, optimal analgesia requires application 1 hour prior to the procedure, which may not be practical in some clinical settings. Newer products such as ELA-max cream (4% or 5% liposomal lidocaine) (Ferndale Laboratories, Ferndale, MI), which has a faster time to skin anesthesia than EMLA (30 minutes vs. 60) may be better suited for ED use.[3] Another advantage of ELA-max is its lack of the prilocaine (the active ingredient in EMLA), which has been associated with rare cases of methemoglobinemia. Liquid lidocaine iontophoresis uses small electrodes to deliver a low level of current, which causes drug molecules to rapidly pass through tissues (Numby Stuff electrodes with a Phoresor II, Iomed, Inc., Salt Lake City, UT). [4] This technique is quite useful to relieve the pain of needle sticks, but setup time and training make it less attractive than simple cream application for ED use. Iontophoresis electrodes may cause a mild stinging and warmth, which older children tolerate well, but may frighten younger ones. [5] [6] Iontophoresis works faster than EMLA,[7] with times to skin anesthesia as rapid as 13 to 20 minutes, but has not been compared to ELA-max. It has been demonstrated that triage nurses can prospectively identify children likely to need venipuncture and apply anesthetic cream early for increased efficiency. [8] Many of the more invasive and technically difficult procedures described in this chapter are performed in emergency situations, where sedation of the critically ill child may not be appropriate. Occasionally, however, sedation may be called for to reduce the trauma to the child and make the procedure easier to perform (see Chapter 34).

BLOOD SAMPLING TECHNIQUES

Capillary Blood Sampling

Indications and Contraindications

Capillary blood sampling, or heel stick puncture, is a frequently used technique to obtain blood samples in young infants. In older children and adults, this technique is often used to obtain blood samples from the finger, toes, and ear lobe when repeated measurements, such as blood glucose, are needed. Capillary blood sampling is most often indicated in a young infant when an adequate sample of blood can be obtained by the heel stick puncture technique and when an alternative technique is not practical. It is an option for obtaining "arterialized" blood for blood gas analysis when arterial access is unavailable, as in many chronically ill neonates and young infants, or when the clinician is not comfortable obtaining a percutaneous arterial sample. Noninvasive

monitoring techniques such as pulse oximetry (see Chapter 2) have reduced the frequency with which these samples must be taken.

Sampling from an area of local inflammation or hematoma should be avoided. Also, repetitive sampling from the same site may induce inflammation and subsequent scarring and hence should be avoided. In general, heel stick sampling is not ideal for blood gas analysis (1) when the infant is hypotensive, (2) when the heel is markedly bruised, or (3) when there is evidence of peripheral vasoconstriction. Capillary blood does not always produce an accurate analysis of arterial P_{O_2} . When the capillary P_{O_2} is greater than 60 mm Hg, the arterial P_{O_2} may be considerably higher, with possibly adverse consequences to infants receiving supplemental oxygen. In this situation, the use of either transcutaneous oxygen saturation or a transcutaneous P_{O_2} monitor may allow adjustment of the inspired oxygen concentration until either an arterial P_{O_2} or a repeat capillary P_{O_2} can be obtained. [9]

Equipment and Setup

The necessary equipment for capillary blood sampling is shown in Table 19-1 . A 3-mm lancet (Becton-Dickinson, Rutherford, NJ) or automated disposable incision device (e.g., Tenderfoot, Surgicutt) should be used to perform this procedure; a scalpel blade should not be used. The use of the former devices will prevent the puncture from penetrating more than the maximum safe distance. Blood collection is performed using either heparinized capillary tubes or 1-mL Microtainer tubes with a collector attachment (Becton-Dickinson, Rutherford, NJ). If capillary tubes are used, a clay or wax sealer will be needed to close off one end.

Technique

Although capillary blood sampling may be performed by the finger stick or heel stick methods, the latter will be described. The recommended sites for heel stick puncture are the medial-most and lateral-most portions of the plantar surface of the heel (Fig. 19-1), but not on the curve of the heel. [10] [11] This avoids penetration of the calcaneus and the risk of osteochondritis. Cautiously prewarming the foot for 5 minutes using a warm towel will produce hyperemia and will enhance blood flow. The foot is immobilized in a dependent position with one hand. After the heel is cleansed with alcohol and allowed to dry, the skin is punctured with the lancet (Fig. 19-2*A*). Allowing the alcohol to dry will avoid falsely high glucose levels in the specimen. Although it is tempting and commonly done, *squeezing of the foot should be avoided*, since this may inhibit capillary filling and may actually decrease blood flow. Furthermore, squeezing may dilute the sample with serum or tissue fluid and make analysis less accurate. If blood does not flow freely, another puncture may be required.

The first small drop of blood is wiped away with gauze, and another drop is allowed to form. A heparinized capillary tube is placed in the drop of blood, and the proximal end of the inverted tube is allowed to fill by capillary action (Fig. 19-2*B*). The tube (or tubes, if several tests will be needed) is sealed at one end by sticking the end into clay or wax before the tube is sent to the laboratory (Fig. 19-2*C*). If 1-mL Microtainer tubes are used, the tube is held at an angle of 30 to 45 degrees from the surface of the puncture site. The collector end is touched to the drop of blood, and blood is allowed to drain into the tube (see Fig. 19-2*B*). Gentle tapping

TABLE 19-1 -- Equipment for Capillary Blood Sampling

Blood collection tubes (capillary tubes or Microtainers)
Warm wet towel or diaper
Alcohol pads
Lancets or automated disposable incision devices
Clay sealer (used with capillary tubes)
Sterile bandage
Nonsterile examination gloves

of the tube will facilitate flow to the bottom. For heparinized tubes, blood should be collected until the level reaches the demarcation on the tube. Over- or underfilling of the tube may result in clotting or erroneous test results, or both. Once an adequate specimen is obtained, a dry dressing is applied to the puncture site.

When a heel stick is performed for arterialized blood samples, the technique used is similar to that discussed previously for routine blood sampling, with the following differences: The infant's foot must be wrapped in a warm towel for a few minutes. The first drop of blood must be discarded and the remaining blood allowed to flow freely into a heparinized capillary tube. The tip of the tube should be placed as near the puncture site as possible to minimize exposure of the blood to environmental oxygen and the tube should be filled as completely as possible. Collection of air in the tube as well as excessive squeezing of the foot should be avoided, because this may artificially lower the P_{O_2} . When the tube is full, the free end is occluded with the gloved finger to prevent entry of air, and both ends are plugged with clay or capped with adapters.

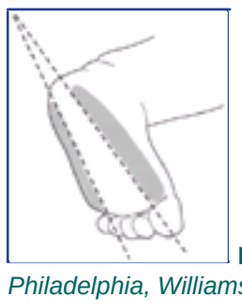

Figure 19-1 Acceptable sites for heel stick puncture illustrated by shaded areas. *(Adapted from the Textbook of Pediatric Emergency Procedures; Henretig FM, King C [eds]. Philadelphia, Williams & Wilkins, 1997.)*

Figure 19-2 *A*, The heel stick is performed on the lateral or medial aspect of the heel. *B*, The collector end of the Microtainer is touched to the drop of blood, and blood is allowed to flow down the wall of the tube to the bottom; or *C*, a heparinized capillary tube is placed in the drop of blood, and the proximal end of the inverted tube is allowed to fill by capillary action. *D*, The blood is maintained in the capillary tube using the index finger to maintain capillary tension on the end of the tube. *E*, When using a microtainer tube, the Microtainer is capped. *F*, when using a capillary tube, the ends are capped or (*G*) sealed with wax or clay. Avoid squeezing the foot, and keep the proximal end of the Microtainer below the puncture site. *(Adapted from the Textbook of Pediatric Emergency Procedures; Henretig FM, King C [eds]. Philadelphia, Williams & Wilkins, 1997.)*

Complications

When properly performed, heel sticks are associated with a low incidence of complications. Lacerations should not occur when the procedure is performed with a proper incision device. Heel sticks may cause infection (local infection, bacteremia, or osteomyelitis), scarring, and calcified nodules. [12] [13] [14] When the heel stick technique is used for the procurement of "arterialized" blood for pH, P_{CO_2}, and P_{O_2} analysis, the most important potential error is that false information (inaccurate P_{O_2}) may result in exposure of the infant to improper amounts of supplemental oxygen. Erroneous values due to hemoconcentration, hemolysis (squeezing heel too vigorously), or contamination with alcohol used for skin cleansing can also occur.

Interpretation

Numerous studies have compared the reliability of capillary blood with that of arterial blood for determination of pH, P_{CO_2}, and P_{O_2}. [15] [16] [17] [18] Although the results have been variable, most investigations have documented a reasonable correlation between arterial and capillary samples for pH and P_{CO_2} determinations (except when the patient is in shock or has an extremely high P_{CO_2}). Unfortunately, the P_{O_2} determination has not been found to be as reliable when performed on blood obtained by capillary sampling. Most studies indicate that the capillary (heel stick) P_{O_2} correlates poorly with the arterial P_{O_2}, especially if the arterial P_{O_2} is greater than 60 mm Hg. In nearly all situations, the capillary P_{O_2} is equal to or less than the arterial P_{O_2}, but in any individual case, one does not know how closely the capillary value approximates the arterial level. Therefore, *reliance solely on a capillary sample of blood for P_{O_2} measurement in an acutely sick infant may be fraught with potential risks*.

Venipuncture

Indications and Contraindications

Although many laboratory tests for the small infant may be performed on blood obtained by heel sticks, larger volumes of blood may be required, making heel sticks impractical. Hence venipuncture is the usual method used for obtaining larger quantities of blood for infants and children.

Venipuncture is also the recommended method to obtain blood for culture. The heel stick capillary tube procedure has been used in some centers for the procurement of blood from infants for cultures, [19] but there is a significant incidence of false-positive results with the technique. In the newborn infant, blood obtained immediately after sterile insertion of an umbilical arterial or venous catheter may be used for culture; even then, there is some controversy concerning the incidence of false-positive results. If venipuncture is unsuccessful, arterial blood may also be used to obtain blood cultures. When collecting blood intended for culture, the area of venipuncture should be prepared with both povidone-iodine and alcohol and allowed to dry. The blood culture collection bottle will likely state a desired volume of sample for optimal results, but 1.0 mL of blood is generally acceptable in a small infant. Iodine solutions and other detergents can irritate infant's skin; therefore, these should be washed off after blood is collected.

Femoral venipuncture for routine blood samples has long been discouraged, especially in children younger than 1 year of age, due to the risk of septic arthritis of the hip. [20] In an emergency setting or when few venous access sites exist,

TABLE 19-2 -- Equipment for Venous Blood Sampling in Infants and Children

Tourniquet (rubber band for scalp veins and tiny infants)
A 3-, 5-, or 10-mL syringe
A 21- or 23-ga butterfly needle
Evacuated blood tubes
Alcohol pads
Povidone-iodine swabs (if blood cultures are needed)
Sterile 2 × 2-in. or 4 × 4-in. gauze pads
Nonsterile examination gloves

blood for laboratory analysis may have to be obtained from such less desirable venous sites or from arterial puncture.

Equipment and Setup

The equipment required for venipuncture in an infant or child is listed in Table 19-2 . A small-gauge butterfly needle is usually preferred over a needle and syringe for obtaining blood in infants and young children, as it is easier to control once the vessel has been entered. Suction is also more controlled with the butterfly needle and syringe. The butterfly needle may also serve as an infusion line once adequate amounts of blood are obtained. The 23-gauge butterfly needle will generally suffice for venipuncture, regardless of age group. In older children and adolescents, a straight needle and syringe or the Vacutainer system (Becton-Dickinson, Rutherford, NJ) can be used much more easily than in infants. However, the negative pressure within the evacuated blood tube, once connected, may be sufficient to collapse the punctured vein. A 3- or 5-mL syringe is less likely than a 10-mL syringe to cause vein collapse in young infants.

Technique

Like adults, the usual site for venipuncture in infants and children is the antecubital fossa. However, any reasonably accessible or easily visible peripheral vein (e.g., on the scalp [for very small infants], hands or feet) may be used (Fig. 19-3). Veins on the dorsum of the hand can be used, providing they will not be needed for IV cannulation. The external jugular and femoral veins or arterial sites are rarely needed for routine samples in the stable patient.

All needed equipment should be assembled and ready for immediate use. The use of a topical anesthetic (e.g., EMLA, ELA-max) may be offered, if appropriate. Drawing blood from infants and small children is usually a two-person procedure. It should be emphasized that immobilization of the extremity is mandatory and this duty should not be relegated to a family member. The butterfly needle and the syringe can be attached either before or after skin penetration. However, if done before, the assembly should take place out of sight of the child. To minimize the number of venipuncture attempts, the optimal site for needle insertion should be chosen after a survey of the most prominent peripheral veins. If an extremity vein is to be used, a tourniquet should be applied proximal to the selected vein; in small infants, a rubber band will serve as an adequate tourniquet, but one must be certain to remove the rubber band following venipuncture.

The tourniquet should not be so tight as to impede arterial filling. The area surrounding the chosen site of skin penetration is cleansed with alcohol and allowed to dry.

Slight distal traction is applied to the skin to immobilize the vein, and the needle is inserted quickly through the skin and slowly into the vein at an angle of approximately 30 degrees, with the

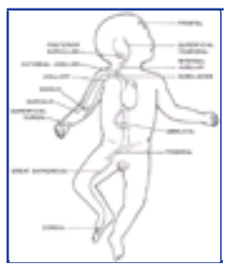

Figure 19-3 Venous access sites in the neonate and young infant. If venous access is unavailable, arterial blood may be used for most laboratory tests, including blood cultures.

bevel up (Fig. 19-4*A*). Successful vessel penetration is heralded by a flashback, or flow of blood, into the butterfly tubing. *Gentle suction is applied by slowly withdrawing the plunger of the syringe.* Alternatively, suction may be applied with a Vacutainer system in which the Vacutainer needle punctures the sealed end of the butterfly device (see Fig. 19-4*B*). If the suction is excessive, the vein will collapse, and blood flow will stop. If the required amount of blood is more than the capacity of the attached syringe, the tubing is pinched off, the filled syringe is removed, the next syringe is attached, and gentle suction is once again applied after release of the pinched tubing. After the required amount of blood is withdrawn, the needle is removed, and a sterile dressing and direct pressure are applied to the puncture site.

Although peripheral sites for venous or arterial sampling are preferable, the external jugular and femoral veins may be used in infants for the performance of a venipuncture during resuscitations or when peripheral sites are inadequate. The external jugular vein lies in a line from the angle of the jaw to the middle of the clavicle and is usually visible on the surface of the skin. The vein is more prominent when the infant is crying. An assistant is needed to restrain the infant in a supine position with the head and neck extended over the edge of the bed. Alternatively, a towel roll or pillow placed under the shoulders can be used. The head is turned approximately 40 to 70 degrees from the midline (Fig. 19-5), and the skin surrounding the area to be punctured is cleansed with alcohol. Finger pressure just above the clavicle will distend the jugular vein. Using a 21- to 25-gauge straight needle with a syringe or a 21- to 25-gauge butterfly needle attached to a syringe, the clinician punctures the skin and advances the needle slowly until the jugular vein is entered. The syringe is connected to

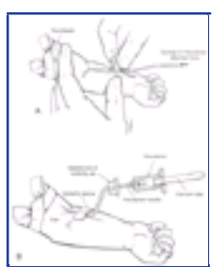

Figure 19-4 *A*, Technique for obtaining blood by antecubital venipuncture with a butterfly needle and a syringe. Once blood is obtained, the butterfly needle may serve as an infusion site. Note that this procedure often requires two persons to carry it out—one to hold the arm and insert the needle and the other to aspirate the blood. *B*, As an alternative to a syringe, a Vacutainer system may be used to apply suction. The Vacutainer needle punctures the sealed end of the butterfly set. Use of this method helps to prevent the premature clotting of blood that may occur if there is a delay in filling the collection tubes.

the needle at all times to maintain a constant negative pressure and avoid an air embolism. After the appropriate amount of blood is obtained, the needle is withdrawn, and slight pressure is applied to the vessel. The infant should be placed in an upright position after the needle is removed, and slight pressure should be continued for 3 to 5 minutes. Close observation of the puncture site should follow.

The femoral vein lies medial to the femoral artery and inferior to the inguinal ligament (Fig. 19-6 *A* (Figure Not Available)). Unlike for adults, the use of this vein for blood sampling is reserved for situations in which patients present in extremis and no other sampling sites are present. An assistant positions the hips in mild abduction and extension while the artery is palpated and its location identified by placing a mark on the abdomen just superior to the femoral triangle (see Fig. 19-6 *A* (Figure Not Available)). The femoral triangle is then prepared with alcohol; a povidone-iodine scrub is also recommended when obtaining blood cultures. The technique of needle insertion is similar to that for external jugular venipuncture (see Fig. 19-5). The clinician punctures the skin and then directs the needle or catheter toward the umbilicus at a 30- to 45-degree angle to the skin, remaining just medial to the femoral artery pulsation (Fig. 19-6 *B* (Figure Not Available)). A slight constant negative pressure is applied throughout

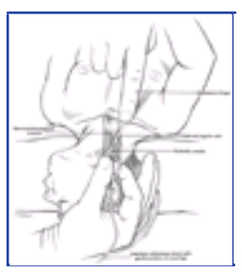

Figure 19-5 External jugular venipuncture. A syringe or a butterfly needle may be used. Venous distention is aided when an assistant's finger occludes the vein, or when the infant cries. The neck is extended, either over the side of the bed or by placing a rolled towel under the shoulders. This procedure requires two persons. Gloves should be worn.

insertion. After the needle enters the femoral vein, the desired blood samples are withdrawn, and the needle or catheter is removed (unless venous access with an IV catheter is desired). Pressure is applied to the femoral triangle for a minimum of 5 minutes, and the site is observed closely for recurrent bleeding.

Scalp veins can be very useful for venous sampling in small infants (younger than 3 months) when other options are not readily available. [21] The anatomic considerations and technique are discussed later in the section on peripheral venous cannulation. Care should be taken with venipuncture and cannulation of scalp veins near the face, as an infiltration could have negative cosmetic consequences. Although sampling or cannulating scalp vessels may be medically necessary, parents of tiny infants are often very upset by the site of their child's head being poked and every effort should be made to explain the rationale for the procedure.

Complications

Complications of venipuncture include hematoma formation, local infection, injury to structures adjacent to vessels, and phlebitis. All of these complications are uncommon. Special care should be used when puncture of the external jugular vein or femoral vein is attempted. Inadvertent deep puncture in the neck can produce injury to the carotid artery, the vagus or phrenic nerve, or the apex of the lung. In the femoral triangle, injury to the femoral artery, femoral nerve, and hip capsule may occur.[20] [22] However, such structures are unlikely to be injured when proper technique is used.

Arterial Blood Sampling

Indications and Contraindications

Arterial blood gas evaluation provides useful information that is essentially unavailable by other means (see Chapter 20) and is important for evaluation of respiratory status and acid-base equilibrium in infants or children with respiratory distress, shock, intoxication, diabetic ketoacidosis, or other metabolic derangement. Arterial blood may also be used for routine laboratory analysis if venous blood is difficult to obtain. Possible sites for arterial blood sampling include (1) radial, brachial, temporal, *dorsalis pedis*, and posterior tibial arteries; (2) umbilical arteries in the newborn infant; and (3) capillaries ("arterialized"). The radial artery is probably the

one most commonly used. The ulnar artery should not be the preferred site for arterial puncture to preserve the collateral circulation

Figure 19-6 (Figure Not Available) *A*, Anatomy of the femoral triangle. The vein is always medial to the artery. *B*, The needle insertion site is located one finger width below the inguinal ligament, just medial to the artery. Use the index and middle fingers to identify the course of the femoral artery. Contrary to the figure, keep both fingers proximal to the entry point of the needle to avert self-puncture. The needle is pointed medially toward the umbilicus at a 45° angle from the skin surface. *(From Pediatric Advanced Life Support Drug Lecture Slides, 1990. Reproduced with permission. Copyright American Heart Association.)*

to the hand, although some clinicians advocate performing punctures and catheterization of the ulnar artery. No vein or nerve is immediately adjacent to the radial artery, which minimizes the risk of obtaining venous blood or damaging a nerve. This is not the case with the brachial artery, and the risk of both complications appears to be greater when this artery is used.[23] Also, the brachial artery has little collateral circulation, causing many clinicians to avoid using it except in extreme circumstances.[24]

The temporal artery is also adjacent to a vein, and if the patient's head is in an oxygen hood, it is nearly impossible to obtain a sample in a steady state. Femoral arteries should not be used for obtaining routine blood samples from the infant or child. Transcutaneous monitoring of P_{O_2}, P_{CO_2}, and oxygen saturation may provide useful adjuncts to arterial sampling in many patients. Nonetheless, they do not replace intermittent arterial sampling, which remains necessary for the stabilization of infants and children and for verification of the accuracy of these noninvasive methods. One should avoid puncture of an artery when infection, burn, or other damage to cutaneous defenses exists in the overlying skin. The presence of adequate collateral circulation and any potential coagulation problems should also be addressed.

Equipment and Setup

The equipment required for arterial puncture in an infant or child is listed in Table 19-3 . A small-gauge butterfly needle is usually preferred over a needle and syringe for arterial puncture in infants and children. As in venipuncture, a 23-gauge butterfly needle is most often used, although in newborns, use of a 25-gauge butterfly may be beneficial. Some clinicians prefer to use a 25-gauge needle connected to a syringe, but use of a butterfly allows for better control of the needle while an assistant aspirates the syringe and may also permit a larger volume of blood to be withdrawn.

Technique

Since the radial artery (Fig. 19-7) is most frequently used to obtain intermittent arterial samples from infants and children, the technique for it will be described. Although the merit of the Allen test (see Chapter 20) has been challenged, it is prudent to first assess the adequacy of collateral circulation in the hand. Both radial and ulnar arteries are compressed. After a short time, compression of the ulnar artery is released while the radial artery remains compressed. Adequacy of collateral flow to the hand by the ulnar artery is marked by return of color to the hand. One should then heparinize a tuberculin syringe (or use a prepacked syringe with anticoagulant) if blood gases are being obtained. All heparin should be ejected from the syringe; a 23- or 25-gauge butterfly needle should then be attached to the syringe. The amount of heparin coating the barrel of the syringe is adequate to anticoagulate the sample; excess heparin may result in inaccurate P_{CO_2} determinations because of dilution of the blood sample.[25] [26]

The clinician should hold the infant's wrist and hand in the left hand (if the clinician is righthanded). The child's hand is held fully supinated with the wrist dorsiflexed. The pulsations of the radial artery should be palpable just proximal to the transverse wrist creases. A small indentation can be made in the skin with a fingernail to mark the insertion site. The area is cleansed with alcohol and allowed to dry. Topical anesthetic cream or an intradermal wheal of 1% lidocaine may be used if the clinical situation allows. The skin is penetrated at a 30° to 45° angle (Fig. 19-8), and while the plunger of the syringe is withdrawn, the needle is advanced slowly until the radial artery is punctured or until resistance is met

TABLE 19-3 -- Equipment for Arterial Blood Sampling in Infants and Children

Heparin solution for coating syringe or prepackaged heparinized syringe
A 1- or 3-mL Tuberculin syringe
A 23- or 25-ga butterfly needle
Povidone-iodine solution
Alcohol pads
Ice-filled container (bag or cup)
Nonsterile examination gloves
Sterile 2 × 2-in. or 4 × 4-in. gauze pads

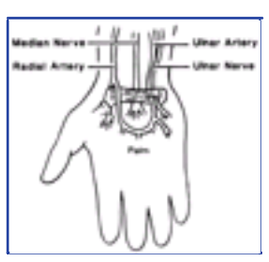

Figure 19-7 Anatomy of the volar surface of the wrist and the palm. The radial artery is preferred for sampling procedures.

(Fig. 19-9). In contrast to the procedure in adults, it is necessary in infants to provide continuous, but gentle, suction on the plunger of the syringe. One can be sure that the radial artery is punctured when blood appears in the hub of the needle. Other clinicians prefer to attach the syringe to the butterfly needle only after blood return is noted and suction is thereafter applied.

If resistance is met while pushing the needle deeper or no blood returns, the needle is slowly withdrawn to the point at which only the distal needle tip remains beneath the skin, and then the procedure is repeated after checking the location of the pulse. Slight reorientation of the needle laterally or medially may be necessary. After the desired amount of blood is obtained, the needle is removed, and light pressure is applied for 5 minutes or longer to control bleeding.

Figure 19-8 For arterial blood sampling, the needle should be inserted under the skin at a 30° to 45° angle. A butterfly needle and syringe are used if larger volumes of blood are required. The wrist is held dorsiflexed by the nondominant hand.

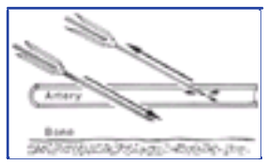

Figure 19-9 Resistance met during passage of the blood gas needle usually indicates contact with bone. The needle should be withdrawn slowly. If the needle has traversed both walls of the artery, blood will be obtained as the needle is slowly withdrawn into the arterial lumen.

Complications

The complications of radial artery puncture include infection, hematoma formation, arterial spasm, tendon injury, and nerve damage. [27] [28] With the use of proper technique, however, the complication rate is extremely low. If the infant starts to cry before blood is obtained, the P_{O_2} and P_{CO_2} may not reflect the infant's true steady state. Another potential problem is the dilutional effect of heparin on the P_{CO_2}. The heparin in the dead space of the tuberculin syringe may decrease the P_{CO_2} by 15% to 25% when 0.2 mL of blood is obtained and by approximately 10% with 0.4 mL of blood. This emphasizes the need for all heparin to be ejected from the dead space of the syringe *before the needle is applied*. The use of a syringe (e.g., Becton-Dickinson 1-mL U-100 insulin syringe) with minimal dead space or the use of syringes prepared with lyophilized heparin eliminates this problem (see Chapter 20). [29]

VASCULAR LINE PLACEMENT: VENOUS AND ARTERIAL

Intravascular lines are indicated when access to the venous or arterial circulations is necessary. An IV line may be positioned in peripheral veins (scalp, hand, forearm, foot, ankle, axilla, thigh) or central veins (superior vena cava via the internal jugular, axillary, superficial temporal, posterior auricular, or subclavian venous approach[21] [30] and the inferior vena cava via the umbilical or femoral venous approach). Likewise, intra-arterial lines may be positioned peripherally (radial, posterior tibial, dorsalis pedis, or superficial temporal arteries) or centrally (abdominal or thoracic aorta via an umbilical or femoral artery approach). Techniques to secure access to these intravascular spaces are discussed in the following sections (see also Chapter 20 Chapter 21 Chapter 22 Chapter 23). Remember to consider the use of topical anesthetic if the clinical situation warrants.

Peripheral Venous Catheterization: Percutaneous

Indications and Contraindications

In general, peripheral IV lines are indicated when the patient is unable to attain medical and nutritional goals with enteral

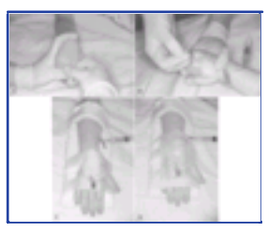

Figure 19-10 Technique for peripheral venous catheterization. *A*, The catheter is directed at a 10° to 20° angle toward the insertion site and advanced until blood return is seen in the catheter and hub. The stylet is removed, and the T-extension tubing is attached. *B*, Taping technique for butterfly and intravascular catheters using a crisscross pattern. *C*, Hand and forearm secured to an arm board. *D*, Covering with the plastic wrapper from the T-extension tubing for protection.

therapy. These lines provide maintenance fluids to support adequate hydration and serve as a route for administering medications. In the acute setting, peripheral IV lines provide a route for administering resuscitative medications and fluids as well as antibiotics.

Equipment and Setup

Materials needed for placement of a peripheral IV line in an infant are listed in Table 19-4 . The two devices commonly used for peripheral IV insertion are the butterfly needle and the plastic over-the-needle catheter. The former ranges in size from 21- to 27-ga. Butterfly needles are used primarily for infusions of short duration and are removed after completion. Examples of such use include certain chemotherapeutic agents and single-dose antibiotic administration. Due to their rigid nature, they tend to infiltrate very easily in the active child. Placement of a butterfly needle in a vein close to a flexor surface is contraindicated. For the most part, over-the-needle catheters, such as Angiocath, Medicut, or Quikcath, have become the mainstay of peripheral venous catheterization. These thin-walled, flexible catheters range in size from 14- to 24-ga. For infants, a 22- to 24-ga catheter will suffice in most cases. The selection of catheter size is dependent on the catheter's intended purpose. In general, the smallest gauge appropriate for the clinical situation should be used. Larger diameter catheters allow for rapid administration of fluids in emergency situations. The use of T-connector extension tubing connected to the catheter after insertion facilitates withdrawal of blood for specimen collection, makes flushing the catheter and maintaining patency easier (especially while taping and securing the IV), and allows for dressing changes without disturbing the IV dressing.[31]

Both commercially available and homemade devices are often used to protect the IV site from a child's attempts to remove it. An arm or leg board appropriate for the size of

TABLE 19-4 -- Equipment for Peripheral Intravenous Insertion in Infants and Children

22- or 24-ga venous catheters
Tourniquet (rubber band for infants)
IV solution and tubing
T-connector extension set
Pretorn tape (½-, 1-, and 2-in.)
Alcohol pads
Povidone-iodine swabs (for blood culture)
Arm or leg board
Non-sterile examination gloves
Sterile 2 × 2-in. or 4 × 4-in. gauze pads
Protective covering (container from IV catheter, T-extension set, or prefabricated cup)
IV fluid chamber with microdrip
A continuous infusion pump
Saline flush solution
3- or 5-mL syringes

the child should be handy to provide for stabilization of the extremity after insertion. In newborns or small infants, fashioning an arm board from two tongue depressors taped together and covered with 4 × 4-in. gauze will provide the appropriate length needed. One should have primed and ready an IV fluid chamber with microdrip and a continuous infusion pump. Fluid administration in an infant must be carefully monitored. *Macrodrip tubing and liter bottles should not be used*; inadvertent infusion of large amounts of fluids in an infant may be disastrous. An infusion pump is an ideal way of limiting fluid infusion while keeping the vein open.

Technique

A number of IV sites are available for placement of a peripheral IV needle or catheter in the infant (see Fig. 19-3). The most common sites chosen for IV insertion in infants and children are the superficial veins of the dorsum of the hand; the antecubital fossa; the dorsum of the foot; and, in newborns and small infants, the scalp.

The veins of the dorsum of the hand are the most often used. These vessels are relatively straight and lay flat on the metacarpals and therefore are stabilized without undue difficulty. If the hand is chosen, one should take into consideration the age and hand preference of the patient. Veins in the antecubital fossa (cephalic and basilic veins) are easily accessible; however, their angulation across the fossa may make advancement of the catheter difficult. These veins may not be easily visible and yet may be palpable. It is recommended to select the most distal vein that is large enough to accommodate the catheter and leave the larger, more proximal veins in case (1) initial attempts are unsuccessful or (2) prolonged IV therapy may be needed and percutaneous central venous catheter placement (e.g., peripherally inserted central catheter [PICC] line) is contemplated. Tributaries of the dorsal venous arch on the dorsum of the foot, like those on the dorsum of the hand, are relatively straight, and the extremity is easily immobilized after insertion. Because indwelling catheters in this location will prevent mobility, this site should be considered only in preambulatory patients or after attempts at other sites have been unsuccessful. The scalp veins are easy to cannulate, but their use is primarily limited to very small infants. If a peripheral vein on the hands, feet, or antecubital fossa is being used, the extremity can first be immobilized by taping it to an arm board, a padded splint, a full plastic IV fluid bag, or a sandbag. The particular site is a matter of preference, and the clinician should choose the vein that appears to be the easiest to cannulate.

With few exceptions, the same techniques used for IV insertion in adults may be used in infants and children, especially in the veins of distal extremities. If a peripheral extremity is used, a tourniquet may be placed proximal to the planned site of entry. Time permitting, nitroglycerin ointment (0.4 to 0.8 mg) is advocated by some clinicians to induce local vasodilation, thereby aiding venous cannulation. [32] Warming the extremity is a more accepted and commonly used maneuver. Some EDs use transillumination devices commonly found in neonatal intensive care units to assist in finding veins in infants. [33] [34]

If the clinical situation allows, a topical anesthetic (e.g., EMLA, ELA-max) may be appropriate. The tubing of the butterfly infusion set or the T-extension set should be flushed before venipuncture with a sterile IV solution, such as normal saline, to prevent air embolism. If a plastic catheter is used, the catheter, with stylet in place, is directed through the skin at a 10° to 20° angle (Fig. 19-10*A*). [35] The catheter with stylet is slowly advanced until blood return is noted. One then advances the catheter over the stylet into the vein. The stylet is removed, and the IV line is connected to the hub of the catheter by means of a T-extension set. After 1 mL of solution is flushed through the line, the site is inspected for signs of infiltration, such as hematoma or local swelling.

The catheter is fixed to the skin with a piece of 0.5 in. tape passed around and over the catheter hub and fixed to the skin. A second piece of tape is placed adhesive side uppermost under the catheter hub and crossed over the catheter in a V shape (Fig. 19-10*B*). The tubing of the T-extension set is looped back, and a piece of tape is placed midway over the tubing and secured to skin. This ensures against accidental dislodgment if the IV tubing is suddenly pulled. The hand and forearm are securely taped to the arm board for immobilization (Fig. 19-10*C*). The clear plastic wrapper of the extension tubing package (or perhaps a commercially available plastic dome protector or even a plastic medication cup cut in half) is then taped over the hub of the catheter as a protective covering (Fig. 19-10*D*). Occasionally the flow rate of the infusion may be positional, especially if the catheter spans a joint or abuts a venous valve. Careful repositioning or adjustment of the hand position or catheter with strategically placed sterile gauze or slight withdrawal of the catheter may be all that is required to remedy the problem.

If blood specimens or cultures are needed, one can obtain these simultaneously during IV insertion and spare the child an additional needlestick. The T-extension tubing and attached syringe should not contain any flush solution. After appropriate preparation of the insertion site and successful placement of the catheter into the vein, the T-extension tubing and syringe are connected to the hub of the catheter, and the blood is aspirated into the syringe. After the desired quantity is obtained, the syringe is removed, and the T-extension tubing is connected to the IV infusion tubing, and the infusion pump is

set at the desired rate. Remember to prepare the skin with appropriate povidone-iodine solution if cultures are needed.

If the scalp veins are used, the area surrounding the planned site of insertion can be shaved and cleansed with an iodine solution. Arteries and veins can usually be differentiated on the scalp because arteries are more tortuous than veins. [21] In addition, the flow of blood is away from the heart in arteries and toward the heart in veins. If an artery is entered during placement of the needle and fluid is infused, blanching will occur in the area. If this happens, the catheter or needle should be removed, light pressure should be maintained for 5 minutes, and the procedure should be repeated at another site. A rubber band may be used as a tourniquet around the scalp *(never the neck)* to produce venous dilation. One should always ensure that the rubber band is removed after venous cannulation. When removing this rubber band, it should be carefully slipped over the catheter or butterfly needle or cut with a pair of scissors. Although cutting the rubber band with scissors is often the easiest technique, the clinician must take care to hold both ends of the cut rubber band to avoid having the infant "snapped" by one or both ends of the rubber band. Placing a piece of tape on the rubber band before placement on the scalp will facilitate lifting the rubber band away from the scalp.

If a scalp vein butterfly infusion set is used, the wings of the butterfly are grasped between the thumb and forefinger, and the needle is introduced beneath the skin approximately 0.5 cm distal to the anticipated site of vein entrance (Fig. 19-11). The needle is advanced slowly toward the vessel until blood appears in the tubing, indicating that the vessel has been entered. The tourniquet should then be removed. The needle should be flushed with 0.5 to 2 mL of IV fluid, such as normal saline, to ensure that the needle is properly in place within the vein. If infiltration occurs, as noted by a subcutaneous (SQ) bump, the IV line should be removed and the process repeated at another site.

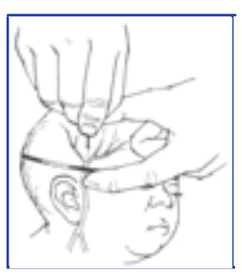

Figure 19-11 Using a rubber band as a tourniquet to distend the scalp veins, the needle is introduced approximately 0.5 cm distal to the anticipated site of the vessel puncture. Gloves should be worn.

After the wings are secured with tape, the tubing of the butterfly set should be taped in a loop on the scalp so that it is not inadvertently pulled. A wisp of cotton may be placed under the wings of the butterfly if the infusion is positional. A small cup may be taped over the wings and the needle to protect the IV line (Fig. 19-12). The catheter of the butterfly set should then be connected to the tubing from the IV system.

Complications

Complications of IV fluid therapy include infection [36] ; injection of sclerosing agents into the SQ space, with resultant necrosis and sloughing of the skin (especially in small infants)[37] ; air embolism,[38] and administration of inappropriate volumes of fluid. Because the life span of an IV needle or catheter is usually fairly short (less than 72 hours) in the small infant, the decision concerning elective removal and replacement of the IV system is not usually a problem. Of course, it is important to pay meticulous attention to sterility during insertion and maintenance of the IV system to decrease the risk of infection.

Peripheral Venous Catheterization: Venous Cutdown

Indications and Contraindications

With the development of small IV catheters and butterfly needles and the rapidity and safety of intraosseous (IO) cannulation (see Chapter 26), peripheral venous cutdowns are rarely used in infants. Even in experienced hands, a saphenous vein cutdown may take more than 10 minutes, may last no longer than percutaneous catheterization, and is associated with a higher rate of infection than other routes of vascular access. [39] [40] [41] Nonetheless, if peripheral venous, central venous, and IO cannulation cannot be performed, venous cutdowns may provide an alternative means of emergency venous access (see section on *Emergency Vascular Access* and

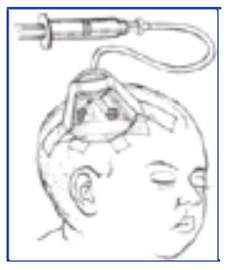

Figure 19-12 Protecting the intravenous line with a plastic medicine cup.

Chapter 23). For the purpose of illustration, the exposure and cannulation of the saphenous vein are discussed (Fig. 19-13 Fig. 19-13). The same principles apply when a cutdown is performed on an arm vein.

Equipment and Setup

Successful venous catheterization via cutdown in the small infant requires sterile instruments, an assistant, good lighting, and a selection of catheters. Silastic catheters, which can be obtained in 2, 3, and 4 Fr sizes (Dow-Corning Company), seem to remain patent longer, and can be sterilized with the instruments to make a "cutdown tray." Standard 19- to 22-gauge IV catheters (Angiocath, Deseret Medical, Inc., Sandy, UT) are also useful.

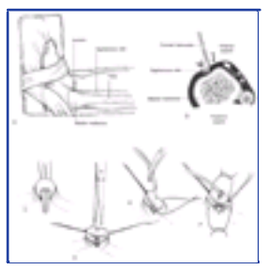

Figure 19-13a Venous cutdown (saphenous vein). *A*, Immobilization of the ankle and the site of skin incision. *B*, A curved hemostat scoops up the vein. The point of the hemostat should be kept against the bone. *C*, The vein is dissected free. *D*, With a proximal and distal tie to stabilize the vein and control bleeding, an incision is made in the upper one third of the vein. *E* and *F*, The infusion catheter is threaded into the vein lumen and advanced.

Technique

The clinician should begin with complete immobilization of the thigh, leg, ankle, and foot by taping them to a padded arm board, which in turn is attached to the table or bed where the procedure is being performed (see Fig. 19-13*A*). The area around the medial malleolus is prepared with a povidone-iodine solution and draped with sterile towels. Local anesthesia should be performed (intradermal 1% lidocaine) in an area about 1 cm proximal and 1 cm anterior to the medial malleolus. Fortunately, there are no major nerves or tendons that accompany the vein in this location (see also Chapter 23).

A tourniquet is placed in the mid-leg and a transverse skin incision is made (usually about 2 cm in length); a small mosquito hemostat is inserted into the wound, with the

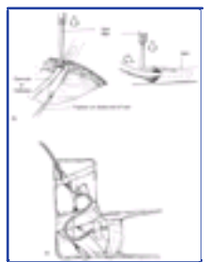

Figure 19-13b *G*, A vein lifter/dilator facilitates placement of the catheter into the vein lumen. *H*, The incision is sutured, and the catheter is secured. *(C from Suratt PM, Gibson RS: Manual of Medical Procedures. St. Louis, CV Mosby, 1982. Reproduced by permission.)*

concavity of the clamp upward. The tip of the hemostat is advanced to the bone in one corner of the wound, and all tissues lying against the bone and in the SQ region are "scooped up" with the hemostat (see Fig. 19-13*B*). This will invariably lift the vein out of the wound along with surrounding tissues. A fine forceps or a mosquito hemostat is used to separate and remove all nonvenous structures, leaving only the saphenous vein tented over the hemostat (see Fig. 19-13*C*). To avoid injury to the vein during dissection, one spreads the ends of the hemostat parallel to the direction of the vein, *never* transversely.

Two 4-0 silk sutures are passed under the vein; one silk suture is pulled distally to stabilize the vein, and the other suture is pulled proximal to the site of venipuncture. The distal suture may be tied, but if left untied, it can still be used for stabilization of the vein. Removal of the untied distal suture following vein cannulation may allow for subsequent vein recannulation following eventual catheter removal. If the distal suture is left untied, longitudinal traction on it permits hemostasis and continued exposure of the vein above the wound. Fine scissors or a scalpel blade may be used to make an oblique or V-shaped incision (venotomy) in the anterior vein wall between the sutures (see Fig. 19-13*D*).

The Silastic catheter (pre-filled with saline solution) is grasped with forceps and is advanced into the vein for a distance of 2 to 3 cm (see Fig. 19-13*E* and *F*). This is usually the most difficult and time-consuming portion of the procedure. A vein dilator or forceps may be used to hold open the incision in the vein (see Fig. 19-13*G*). Downward pull on the distal tie will give countertraction and will stabilize the vein during catheter advancement. The tourniquet is then removed. One ties the proximal suture around the vein with the catheter inside, taking care not to occlude the catheter by tying the suture too tight. If the distal suture was tied, the free ends of the suture can be tied around the catheter, providing additional stability to the catheter. If the distal suture was not tied, it is

now removed. When the distal suture is left untied, the proximal suture is still tied to secure the catheter, but the ends are left long so that the suture can be pulled out of the incision and removed to allow recannulation once the infusion catheter is removed.

Continued infusion of saline through the catheter from an attached syringe will ensure patency. The catheter is oriented into either corner of the incision, and the incision is closed with interrupted 4-0 nylon sutures. The skin suture nearest the catheter is wrapped around the catheter and tied to hold the catheter in place. Bleeding can be controlled with direct pressure. Antibiotic ointment is placed over the wound, and a sterile occlusive dressing is applied. The IV tubing is connected and taped securely to the footboard to prevent inadvertent removal of the catheter (see Fig. 19-13*H*).

Change the dressing carefully every day, using sterile technique with reapplication of antibiotic ointment. When cared for properly, catheters can remain in place for as long as 7 to 10 days. Generally, though, a line is replaced, using another site, after 3 to 4 days. Obviously, at the first sign of infiltration or infection the catheter must be removed. Unfortunately, once the vein has been used for a cutdown, it is usually rendered useless for future venous cannulation.

Mini-cutdown.

The cannulation of a small vein with a catheter or tube may be difficult and very time-consuming if one is not experienced in the technique. As an alternative, the

mini-cutdown procedure may be used. Once the vein is exposed through a skin incision and SQ dissection, it is cannulated directly with a standard IV catheter (Medicut, Angiocath) rather than nicked with a scalpel (Fig. 19-14). A silk suture or hemostat may be placed under the vein to immobilize it during puncture, but with the mini-cutdown technique, the vein is not tied off after being cannulated. The catheter will not be as secure with this modification, but the technique is useful when time is critical. The vein is not destroyed with this technique. In essence, the mini-cutdown uses the percutaneous technique of cannulation, except that venipuncture is performed through a skin incision under direct visualization (see Chapter 22 and Chapter 23).

Complications

In addition to the problems discussed previously, venous cutdowns can result in wound infections and phlebitis. Adjacent

Figure 19-14 The mini-cutdown procedure using a standard intravenous catheter over-the-needle system is technically easier than the full cutdown and may be preferred in an emergency.

structures may be injured during the incision and subsequent blunt dissection. When the mini-cutdown technique without ligatures is used, extravasation of infusate may result. Light pressure on the closed wound will generally prevent continued extravasation.

Central Venous Catheterization: Percutaneous

Percutaneous placement of central venous lines (CVLs) has become the technique of choice of many clinicians for securing central venous access in neonates and young infants (see Chapter 22).[39] This technique has largely supplanted the conventional technique of cutdown for central venous catheterization. Both percutaneous and venous cutdown catheterizations require central venous catheters, which can be purchased separately or within self-contained kits (Arrow International, Inc., Reading, PA; Gesco International, San Antonio, TX).

Indications and Contraindications

Percutaneous central venous cannulation is indicated to secure vascular access (1) when peripheral venous access is limited or impossible, (2) for emergency drug and fluid administration during cardiac arrest and shock, (3) when hyperalimentation and IV infusions are required for days to weeks, (4) when low-birth-weight neonates and young infants require central venous access, and (5) when precise hemodynamic monitoring is needed in a critically ill or injured child. Contraindications to percutaneous placement of central venous catheters include an uncorrected coagulopathy; local infections or burns at insertion sites; malformations or deformations that may distort vascular anatomy; vascular insufficiency of an extremity; obstruction or compression of the access veins by tumor, abnormal vessels, hematoma, thrombus, abscess, or malformation; or absence of access veins.[22] [43] Bacterial septicemia is a relative contraindication, and delaying placement of central venous access until cultures have been sterile for 48 hours is generally recommended.

Equipment and Setup

Percutaneous central venous catheterization in infants and children can be performed using any number of sterile over-the-needle catheters ranging in size from 22 to 16 gauge (choice depends on the age of the patient) and equipment similar to that used for percutaneous peripheral venous catheterization (see Table 19-4). If insertion of a larger indwelling catheter is desired, commercially available kits are convenient (Gesco International, Inc., San Antonio, TX; Arrow International, Inc., Reading, PA; Cook, Inc., Bloomington, IN). The catheters in these kits are typically made of a silicone elastomer, polyvinyl chloride, or polyethylene; some are available with an antimicrobial coating that helps prevent infection. Catheter length is variable, and 1- to 3-lumen catheters are available. Rapid volume replacement, as in the case of severe dehydration or acute blood loss from trauma, is best achieved by inserting a short, large-bore catheter for the initial resuscitation and stabilization. If the patient requires hemodynamic monitoring or multiple medication infusions, the catheter can be replaced later with a larger indwelling catheter using the Seldinger technique.

Other necessary equipment includes: sterile forceps and scissors; povidone-iodine solution; gauze pads; sterile drapes;

gowns; gloves; caps and masks; syringes (3 mL, 5 mL, and 10 mL); Tegaderm (Medical Products, Inc., St. Paul, MN), Op-Site (Smith and Nephew Medical, Massilon, OH), or other sterile transparent skin coverings; Luer-Lok three-way stopcocks; 0.25 to 1.0% lidocaine; flush solution (1 to 2 U heparin per mL normal saline), and IV tubing with a T-connector extension. Depending on the access vein to be used, restraint of the extremity, pelvis, or head may require a padded support, an assistant, or both.

Techniques

Percutaneous placement of central venous catheters can be accomplished using two methods that differ only in the use of a guidewire. The guidewire (Seldinger) technique is preferred when catheters are inserted into the femoral or subclavian vein. When using the basilic or cephalic vein of the forearm and antecubital space, axillary vein, or superficial temporal or posterior auricular scalp vein, many clinicians prefer to insert the catheter through an introducer needle. Details of the pediatric femoral, external and internal jugular, and subclavian and antecubital approaches follow.

Femoral catheterization.

The safety and efficacy of percutaneous femoral venous catheterization have been demonstrated. [44] Femoral venous catheterization is the central venous access route most commonly used in infants and children in emergency situations. [39] The femoral anatomy is easily learned, and the arterial pulse provides a landmark for catheter insertion. In case of inadvertent arterial puncture or venous laceration, hemostasis can be achieved by application of direct pressure. Also, femoral catheterization is less likely to interfere with emergency procedures in the region of the head, neck, and chest during medical or trauma resuscitations. In addition, the specific risks associated with subclavian and internal jugular vein catheterization (pneumothorax and carotid or subclavian artery puncture) are avoided. Risks of the procedure include thrombosis and infection; these can occur with any type of venous catheter.

Technique.

The child must be adequately restrained to permit exposure of the inguinal region. In some situations, it might be necessary to sedate the child to perform the procedure (see Chapter 34), but this should be done with caution in a child with marginal perfusion or respiratory compromise. Although data studying the use of this technique in the pediatric population are sparse, [45] [46] the use of ultrasound to locate the femoral artery may be helpful if the femoral arterial pulsation is not strong, if edema makes palpation of the artery difficult, or if the artery is difficult to locate when wearing gloves (see Chapter 69). Note that during cardiopulmonary resuscitation (CPR), palpable pulsations or Doppler tones in the femoral vein may be detected. Hence, if the vein is not found medial to the pulsations, catheterization of the pulsating vessel during CPR may be considered as a last resort when other options for vascular access or drug delivery are unavailable. Both groins are generally prepared with povidone-iodine in the event that the initial attempt is unsuccessful.

The introducer needle supplied with the kit can be used with or without a syringe to enter the femoral vein. The femoral artery is palpated with one finger, and the needle is placed in the skin just medial to the artery. One enters the skin at a 30° to 45° angle approximately 1 cm below the inguinal ligament. The general course of the needle is in a line directed toward the umbilicus. When blood return is noted, the wire is gently passed through the needle into the proximal vein. If a syringe has been attached to the introducer needle, continuous, gentle suction is applied while the needle is inserted. When the syringe is removed to insert the wire, a sterile gloved finger is often placed over the open hub of the needle to prevent air embolus or blood loss. The wire should not meet resistance when gently introduced and

the proximal end should always be visible protruding from the hub of the needle. If there is resistance to passage of wire, it should be removed to assess the needle's position within the vessel. If there is resistance to removal of the wire, the needle and the wire must be withdrawn together to prevent shearing off the end of the wire.[47] An alternative method that may be useful when placing the 4 Fr double-lumen Arrow catheter is to remove the tubing from a 21-gauge butterfly needle (Abbott Hospitals, Inc., North Chicago, IL) and use the needle to enter the vein (Fig. 19-15*A*). The butterfly needle is very easy to hold in a stable position and is also shorter than the needles supplied with the assembled kits. When blood return is obtained, the wire is passed through the butterfly needle into the proximal vein.

A small incision (1 to 2 mm) is then made along the wire's entry point at the skin to allow passage of the vein dilator (Fig. 19-15*B*) or the catheter itself. The incision is generally made with a No. 11 scalpel blade with the sharp edge of the blade pointed away from the wire. The dilator, if used, is advanced over the wire gently, then removed. The catheter, which has been flushed with saline, is then advanced over the wire into the vein; and the wire is then removed (Fig. 19-15*C*).[48] Occasionally it is useful to rotate and advance the dilator (or catheter) simultaneously as it enters the vein. Many times the dilator step is skipped in a critical situation and the catheter is placed directly after placement of the wire. Blood return is noted from the catheter ports, which are then flushed with a sterile saline solution. The catheter is subsequently secured with silk or nylon sutures (Fig. 19-15*D*). A sterile transparent skin covering placed over the exit site may be used as an impermeable dressing.

This technique is useful in children as small as 1000 g. When one is placing femoral venous catheters in children smaller than 1500 g, a smaller single-lumen catheter (3 Fr or 24-ga) should be used, because a larger catheter may occlude blood flow in the femoral vein.

External jugular venous catheterization.

The external jugular vein is superficial and easily visible. This site should be selected for catheterization only after catheterization of other, more peripheral sites has been unsuccessful. Also, the external jugular vein is undesirable as a primary catheterization site during resuscitative efforts, since manipulation of the head and neck may compromise management of the airway. In young infants, use of the Seldinger technique is difficult due to the short length of the infant's neck, as well as the low success rate of central venous catheter placement resulting from the acute angle of entry of the external jugular vein into the subclavian vein. [49]

Technique.

The external jugular vein lies in a line from the angle of the jaw to the middle of the clavicle and is usually visible on the surface of the skin. The vein is more prominent when the infant is crying. An assistant is needed to restrain the infant in a supine position with the head and neck extended over the edge of the bed. Alternatively, a towel roll or pillow placed under the shoulders can be used. The head is turned approximately 40° to 70° from the midline (see Fig. 19-5). The skin surrounding the area to be punctured is cleansed with

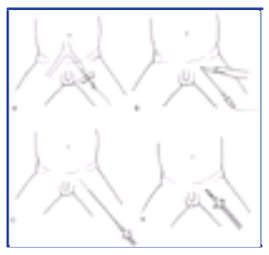

Figure 19-15 Technique for inserting a femoral venous catheter. *A*, A 21-ga butterfly catheter is used to enter the femoral vein, and the guidewire is passed through the butterfly needle into the proximal vein. Note that the tubing has been removed from a standard butterfly set. *B*, A small incision is made alongside the wire, and the dilator is advanced over the wire and into the vein. *C*, The catheter is advanced over the wire and into the vein. *D*, The wire is removed and the catheter secured. Note that many commercial kits have a self-contained 21-ga needle, making modification of a butterfly needle catheter unnecessary.

alcohol. The area is covered with a sterile drape, and 1% lidocaine may then be infiltrated into the skin. A finger may be placed just above the clavicle to distend the jugular vein.

Using an 18- to 22-ga catheter with a syringe, the catheter is aligned parallel to the vein, and the skin is punctured approximately one half to two thirds of the distance from the angle of the jaw to the clavicle. The catheter is advanced slowly until the jugular vein is entered. The syringe is connected to the catheter at all times to maintain a constant negative pressure and avoid an air embolism. After the appropriate amount of blood is obtained, the catheter is advanced and secured in place. If the Seldinger technique is used, proceed as described for femoral catheterization. The catheter should be passed far enough to reach the superior vena cavaright atrium junction. The catheter is checked for blood return, and the line is secured with sutures and a sterile occlusive dressing applied. A chest radiograph is warranted to assess the proper location of the catheter, as well as to rule out the possibility of an iatrogenically induced pneumothorax.

Internal jugular venous catheterization.

The internal jugular veins lie within the carotid sheath containing the carotid artery and vagus nerve. The lower part of the vein lies within the triangle formed by the sternal and clavicular heads of the sternocleidomastoid muscle and becomes more lateral and anterior to the artery as it joins the subclavian vein. The right internal jugular vein is preferred over the left, since the internal jugular and innominate vein and the superior vena cava form a nearly straight line into the right atrium. This lessens the chance for pneumothorax or injury to the thoracic duct (see Chapter 22). Like the external jugular, this site should be chosen for catheterization only after catheterization of other, more peripheral sites has been unsuccessful.

Technique.

Three approaches (the anterior, median or central, and posterior approaches as discussed in Chapter 22) to internal jugular catheterization are possible. The median or central approach is recommended in pediatric patients and will be described here. The child is positioned in the same fashion as that described for external jugular venous

catheterization. The medial or central approach uses the apex of the angle formed by the sternal and clavicular heads of the sternocleidomastoid muscle as the puncture site. If one could imagine a line from the mastoid process to the sternal notch, the apex of the angle formed by the two muscular heads would fall approximately along the middle third of that line. [47] The skin surrounding the area to be punctured is cleansed with alcohol. The area is covered with a sterile drape, and 1% lidocaine may then be infiltrated into the skin. An 18- to 22-ga needle with a syringe is used, and the needle is introduced at the apex of the triangle at an angle of 30 degrees downward relative to the coronal plane and directed caudad toward the ipsilateral nipple (Fig. 19-16 (Figure Not Available)). The needle is advanced slowly until the jugular vein is entered. The syringe is connected to the needle at all times to maintain a constant negative pressure and avoid an air embolism. After blood flow is obtained, the syringe is removed, and a finger is placed over the hub of the needle. A guidewire is then inserted during a positive-pressure breath or exhalation, the needle is removed, and a catheter is introduced using the Seldinger technique (see Chapter 22). The catheter should be passed far enough to reach the superior vena cava/right atrium junction. The catheter is checked for blood return, the line is secured with sutures, and a sterile occlusive dressing is applied. A chest radiograph is warranted to assess the proper location of the catheter, as well as to rule out pneumothorax.

Subclavian Venous Catheterization.

The subclavian vein is a popular route of central venous access in the adult patient but is used far less frequently in children. The technique is more difficult in the child because of the vessels' smaller size, as well as their more cephalad location under the clavicles. An infraclavicular approach to the subclavian vein has been used, but due to the high risks of pneumothorax and hemothorax, especially when performed during emergencies, this approach should be considered only if other

peripheral or central venous access sites are unobtainable. [39] [47] [50] The younger the patient, the higher the risks of these complications. Also,

Figure 19-16 (Figure Not Available) Technique for internal jugular venous catheterization (medial or central approach). The needle is inserted at the apex of the triangle formed by the sternal and clavicular heads of the sternocleidomastoid muscle. The needle is angled 30° downward relative to the coronal plane and directed toward the ipsilateral nipple. *(From Textbook of Pediatric Advanced Life Support, 1994. Reproduced with permission. Copyright American Heart Association.)*

subclavian venous access may interfere with resuscitative efforts or be unavailable due to the placement of cervical spine immobilization devices in a trauma patient.

Technique.

The technique for subclavian venous catheterization differs from that for the adult in that the approach to the vein is more lateral in children. The infraclavicular approach is described. The equipment needed is the same as that used for femoral catheterization. The patient is placed in the Trendelenburg position with the head turned away from the side to be punctured and a towel roll placed under the shoulders (Fig. 19-17 (Figure Not Available)). The right side is preferred, since the dome of the lung is more cephalad on the left side. The needle insertion site is at the distal one third of the clavicle in the depression created between the deltoid and pectoralis major muscles. The skin is prepped with antiseptic

Figure 19-17 (Figure Not Available) Technique for subclavian venous catheterization. The needle is inserted at the distal one third of the clavicle in the depression created between the deltoid and pectoralis major muscles. The needle should be parallel to the frontal plane and directed medially and slightly cephalad toward a fingertip placed in the sternal notch. The patient is shown in a 30° Trendelenburg position. *(From Textbook of Pediatric Advanced Life Support, 1994. Reproduced with permission. Copyright American Heart Association.)*

solution, the area covered with a sterile drape, and the skin infiltrated with 1% lidocaine.

The finder needle is introduced and advanced slowly while negative pressure is applied with the attached syringe. The syringe and needle should be parallel to the frontal plane and directed medially and slightly cephalad, beneath the clavicle toward the posterior aspect of the sternal end of the clavicle (i.e., with the lower end of the fingertip placed in the sternal notch). [39] The needle is advanced until blood return is obtained. The bevel of the needle is then pointed caudad to direct the guidewire to the superior vena cava. The wire is then inserted during a positive pressure breath or natural exhalation and the catheter is introduced over the wire using the Seldinger technique as previously described for femoral catheterization. As in adults, the cardiac monitor may show a rhythm disturbance if the wire is advanced too far. Auscultation of bilateral breath sounds should be performed and a chest radiograph obtained to confirm the proper positioning of the catheter in the superior vena cava, as well as to rule out procedural complications such as pneumothorax or hemothorax. The catheter is then secured in place with sutures, and a sterile, occlusive dressing is applied.

Antecubital access.

Percutaneous insertion of central catheters by way of peripheral antecubital veins is used most frequently to obtain central venous access in patients with very small caliber vessels (e.g., low-birth-weight neonates and very young infants). These PICC lines are small Silastic catheters ranging in size from 23- to 16-ga. [51] [52] These catheters are rarely inserted as an ED procedure; more often they are placed in a stable child who will require longer term fluid or nutritional therapy or an extended course of IV antibiotics. The most common sites include scalp, neck, and arm veins. The catheter is then threaded centrally. PICC lines offer several advantages over conventional peripheral IV catheters and percutaneous central venous catheters. They can remain in place for up to 3 months, sparing veins from multiple reinsertions with peripheral venous catheterizations; the long-arm catheter is simpler to insert than central venous catheters and poses no risk of producing a pneumothorax or hemothorax on insertion.

Technique.

The arm of the vessel to be cannulated is initially stabilized using a support board or the help of an assistant. The remainder of the procedure requires sterile technique. Povidone-iodine is used to cleanse the skin overlying the vessel to be cannulated, and 1% lidocaine is infiltrated at the skin site to be punctured. This skin site is punctured with an 18-ga needle to ease insertion of the introducer through the skin. The catheter to be inserted is chosen based on the size of the access vessel.

Typically a 23-ga silicone elastomer catheter with other needed accessories is used, as in a kit prepared by Gesco International, Inc. (San Antonio, TX). Advantages of this catheter include (1) a double-wing silicone adapter, which precludes the need to make homemade blunt-end adapters to fit small cannulas and simplifies the taping procedure, and (2) a breakaway introducer needle that can be peeled off the catheter, thereby precluding the need for sliding the introducer off the catheter and placing an adapter. Because the length of this catheter (33.5 cm) is longer than needed in low-birth-weight neonates and young infants, the distance from the insertion site to the superior vena cava-right atrium junction is estimated (i.e., by measuring the distance between insertion site and the right nipple [Fig. 19-18*A*]), and the catheter is cut 1 to 3 cm longer than the estimated distance to compensate for variability between the estimated and actual needed length of the catheter. The end of this catheter is then connected to a Luer-Lok stopcock and syringe and filled with flush solution; the catheter is then ready for use.

The 20-ga breakaway introducer needle (Gesco International, Inc.) is also filled with flush solution and then directed slowly through the insertion site and into the access vein. When blood return occurs, the catheter is picked up approximately 1 cm from its tip and guided into the introducer needle (Fig. 19-18*B*). The catheter is advanced in 1-cm increments until the previously estimated distance is reached (i.e., the catheter tip is at the superior vena cava-right atrium junction). The breakaway introducer needle is then withdrawn several centimeters from the insertion site before peeling the introducer off the catheter to avoid inadvertent catheter laceration. If accidental laceration occurs, blunt-end adapters should be readily available. An alternative method, the

Figure 19-18 Techniques for insertion of central catheters from peripheral veins. *A*, A tape measure is used to determine the catheter length. *B*, Placement of the catheter through the specialized breakaway butterfly-type introducer needle.

Microintroducer technique, introduces a guidewire through the catheter used to gain access to the vein. A small "nick" is made with a scalpel, as described previously for central venous catheters, and an introducer catheter is threaded over the wire to make a larger entry. Finally, the silicone PICC line is carefully threaded into the introducer catheter and advanced as described earlier. [53]

Immediately after catheter placement and withdrawal of the introducer needle, the clinician will be able to manipulate the position of the catheter until clotting starts to occur at the insertion site. After the sterile field is discontinued, the catheter should never be advanced. The function of the catheter is checked by withdrawing blood, by noting the presence of residual air bubbles within the catheter, or both. After the clinician is assured that no air bubbles remain in the line, the catheter is flushed. This catheter should be "easy" to flush; if it is not, the clinician should reposition the catheter and recheck its function. If the catheter remains difficult to flush, it should be considered clotted and should be removed. Alternatively, position may be confirmed by chest radiograph and fibrinolytic therapy used, if considered appropriate.

A transparent skin covering is placed and is removed only when the catheter is removed; it is not routinely removed as are coverings of some surgically placed central venous catheters. Stabilization sutures are not routinely placed during this procedure. Occasionally a small amount of bleeding occurs at the insertion site; this

generally stops spontaneously or with gentle pressure. With the three-way stopcock in place, central venous pressure measurement and infusion of medications, IV fluids, and hyperalimentation solutions can be performed. Many clinicians at centers that frequently perform PICC insertions use sonography or fluoroscopy to increase likelihood of successful catheter placement. [54]

Complications

The incidence of complications from central venous catheterization ranges from 10% to 50%. [52] [54] Infection and thrombosis are the major risks associated with these catheters. [43] [55] Other complications include accidental displacement, phlebitis, hemorrhage, hematoma, dysrhythmia, air embolus, vascular obstruction or perforation, right atrial perforation, and localized edema. Blood sampling from indwelling central venous lines must be performed with caution, because the risk of contamination increases each time the system is opened. Morbidity from complications can be minimized by removing catheters as soon as they are no longer needed.

Emergency Vascular Access

The first steps in managing pediatric resuscitations are to establish an adequate airway, ensure adequate ventilation, and enhance blood circulation. Maintaining or reestablishing adequate circulation often requires prompt access to the intravascular space for administration of fluids or medications, or both. The difficulty of obtaining venous access during pediatric resuscitations can be considerable. [56] [57] In the review by Rossetti and colleagues, IV access required 10 or more minutes in 24% of the cases. The average time required for a cutdown was 24 minutes. Children who were successfully resuscitated had vascular access achieved significantly sooner than those who were not resuscitated. Emergency IV access was most prolonged in children younger than 2 years of age. This last finding is important, since the majority of cardiopulmonary arrests in children occur in this younger age group.

If no IV line is available, appropriate drugs can be given via the endotracheal tube (see Chapter 27) while attempts at venous access are initiated. Resuscitation courses aimed at enhancing the emergency care of children now stress the early use of intraosseous access [39] (see Chapter 26). Now experts suggest that intraosseous or central venous access should be attempted concurrently with attempts at peripheral venous access when treating a child undergoing CPR or in extremis from shock. Intraosseous access was previously felt to be effective only in children near or under the age of 6. This limitation has since been abandoned. The femoral vein is usually the central vein of choice in emergencies, because its consistent anatomic location and large size make it the safest and easiest central vein to catheterize. The femoral vein also can be accessed with minimal interference to resuscitative efforts.

Umbilical Vein Catheterization

Indications and Contraindications

The major indication for umbilical vein catheterization is access to the vascular system for emergency resuscitation and stabilization of the newborn. The umbilical vein may also be used for exchange transfusions and short-term central venous access in newborns. The umbilical vein may remain patent for about a week after birth (sometimes longer). [58] In the neonate who presents to the ED requiring emergency access, a peripheral vein would be preferable, but attempting to cannulate one of the umbilical vessels could be life-saving. The procedure is technically easier than umbilical artery cannulation.

Equipment and Setup

The supplies and equipment for catheterization are listed in Table 19-5 . The infant is placed beneath a radiant warmer, and the extremities are restrained. Oxygen is administered as needed, and the audible beep on the cardiac monitor is turned on. The operator should wear a surgical cap and mask and a sterile gown and gloves.

Technique

Holding the umbilical stump up, the cord is scrubbed with a bactericidal solution. Pooling of liquid at the infant's side should be avoided, because this may be associated with blistering of the skin under a radiant warmer. The umbilical area

TABLE 19-5 -- Umbilical Vein and Artery Catheterization Equipment

Infusion solution (usually $D_{5–10}$ W with electrolytes. Some clinicians also add 1 unit heparin per milliliter of fluid to "prevent" clotting in the catheter)
Fluid chamber, IV tubing, infusion pump, filter (0.22 µm), short length of IV tubing, three-way stopcock
Umbilical artery catheter (3.5 to 5 Fr)
3-0 silk suture on a curved needle
Curved iris forceps without teeth
Small clamps, forceps, scissors, needle holder
Sterile drapes
Light source
10 mL of heparinized solution for flush (1 to 2 units heparin per milliliter of fluid)
Surgical cap, mask, gown, and gloves

is draped in a sterile fashion, with the infant's head left exposed for observation.

To provide hemostasis and to anchor the line after placement, a loop of umbilical tape or a purse string suture is placed at the junction of the skin and the cord (Fig. 19-19). The cord is cut with a scalpel about 1 cm from the skin, and the vessels are identified. The vein is usually located at 12 o'clock and has a thin wall and large lumen. It may continue to bleed after cutting, whereas the two arteries have thicker walls and smaller lumina, and constriction reduces bleeding after being cut. Occasionally a persistent urachus may be mistaken for the umbilical vein, but the presence of urine should identify the mistake.

The catheter (3.5 Fr [preterm infants] to 5.0 Fr [term infants]), which has been flushed with heparinized saline and attached to a three-way stopcock, is placed in the lumen of the umbilical vein and advanced gently. The catheter is advanced only 1 to 2 cm beyond the point at which good blood return is obtained. This is usually only 4 to 5 cm in a term-sized infant. If the catheter is pushed farther than this, it will do one of two things: It may enter the *ductus venosus* and then move into the inferior vena cava, or it may enter a branch of the portal vein within the liver (evidenced by resistance at 5 to 10 cm).

The *inferior vena caval site* may be a desirable location in some newborn infants in whom peripheral vascular access is limited and for whom central venous access is desired for central venous pressure monitoring or infusion of medications, high concentrations of glucose (greater than 10%), IV fluids, and hyperalimentation solutions. The catheter must be inserted approximately 10 to 12 cm in a term-sized infant to reach the inferior vena cava. The *portal vein site* can be verified by radiographs that document the placement of the catheter. Note that an umbilical venous catheter will proceed directly cephalad (without making a downward loop) until it passes through the *ductus venosus* (Fig. 19-20). Some practitioners use standardized graphs to estimate length of insertion, which are based on the shoulder-to-umbilicus length (Fig. 19-21*A*). Such graphs are useful if stored in the drawers of the warming beds used to resuscitate newborns and small infants, along with the catheters and other equipment. Shoulder-to-umbilicus length is the perpendicular line measured from the tip of the shoulder to the horizontal level of the umbilicus. Alternatively, if the graph is not available, the shoulder-to-umbilicus length multiplied by 0.6 gives an approximate insertion length that will result in the tip of the catheter above the diaphragm but below the right atrium, in

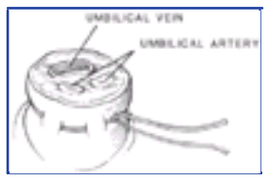

Figure 19-19 When placing an umbilical vein catheter, a pursestring suture or umbilical tape is passed around the base of the cord to provide hemostasis and to anchor the line.

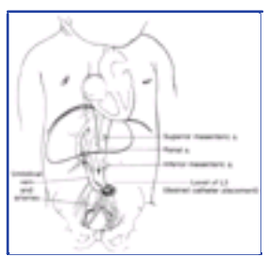
Figure 19-20 An umbilical vein catheter is directed toward the head and remains anterior until it passes through the *ductus venosus* into the inferior vena cava. *(From Ludwig S, Fleisher GR: Textbook of Pediatric Emergency Medicine. Baltimore, Williams & Wilkins, 1994.)*

the inferior vena cava.[58] There are also multiple regression formulae to estimate catheter length, based on birth weight, [59] but during a resuscitation, it is most prudent to gently pass the line only 4 to 5 cm in a term infant to avoid injecting hyperosmolar fluids into the portal vessels, which potentially could result in liver necrosis. Remember to account for the length of the umbilical stump in the calculation.

Air embolism may occur at the time of catheter removal if the infant generates sufficient negative intrathoracic pressure (as during crying) to cause air to be drawn into the patent umbilical vein. Therefore, caution must be used during catheter removal to ensure that the vein is promptly occluded (by tightening a pursestring suture or applying pressure on or just cephalad to the umbilicus).

Complications

Complications of umbilical venous catheters include hemorrhage, infection, injection of sclerosing substances into the liver (resulting in hepatic necrosis), air embolism, catheter tip embolism, and vessel perforation.[54] It is most important that one follow careful technique in insertion and maintenance of catheters to minimize such complications.

Umbilical Artery Catheterization

Indications and Contraindications

Umbilical artery catheterization is a useful procedure in the care of newborn infants who require frequent monitoring of arterial blood gases and arterial blood pressure, fluid and medication administration, and exchange transfusions. It is imperative for the clinician to remain aware of potential complications. [60] One of the two umbilical arteries may be cannulated for resuscitation purposes, but an umbilical vein is generally technically easier to cannulate and may be preferred in an emergency.

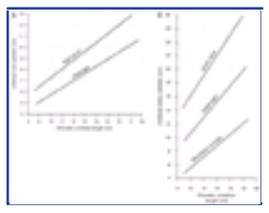
Figure 19-21 After measuring the shoulder-to-umbilicus length, a standardized graph can be used to determine the appropriate length of the umbilical venous catheter (*A*), or umbilical arterial catheter (*B*). The venous catheter should be inserted into the inferior vena cava below the level of the right atrium. The appropriate length of the arterial catheter depends on whether a "high" or "low" line is desired (see text for explanation). *(From The Johns Hopkins Hospital, Nechyba C, Gunn VL: The Harriet Lane Handbook: A Manual for Pediatric Home Officers, 16th ed. St. Louis, CV Mosby, 2002.)*

Equipment and Setup

The equipment required for umbilical artery catheterization is identical to that used for umbilical venous catheterization (see Table 19-5). Additional equipment needed for continuous arterial pressure monitoring and infusion should be readily available.

Technique

The technique of umbilical artery catheterization is similar to that described for umbilical vein catheterization in the preceding section. After the umbilical arteries have been located, the cord is grasped with a curved hemostat near the selected artery (Fig. 19-22). Using two hemostats to grasp each side of the cord and slightly evert the edges can sometimes aid in exposure of the arteries. Using the curved iris forceps without teeth, one gently dilates the artery, sometimes with repeated passes of the forceps. Umbilical artery spasm may make the procedure difficult. A 3.5 to 5 Fr catheter is attached to a three-way stopcock and flushed with sterile heparinized solution. The catheter may then be introduced into the dilated artery. A 3.5 to 4 Fr catheter is recommended for infants weighing less than 2 kg and a 5 Fr catheter for those weighing more than or equal to 2 kg.

When the catheter is being inserted, tension should be placed cephalad on the cord, and the catheter should be advanced with slow, constant pressure toward the feet (Fig. 19-23). Resistance is occasionally felt at 1 to 2 cm and should be overcome by gentle, sustained pressure. If the catheter passes 4 to 5 cm and meets resistance, this generally indicates that a "false passage" through the vessel wall has occurred. Occasionally one may bypass the perforation by attempting catheterization with the larger 5 Fr catheter.

The optimal position of the catheter tip in the descending aorta remains the subject of some debate. [59] [61] If a low (L3 to L4) position is desired, the catheter may be advanced 7 to 8 cm in a 1-kg premature infant or 12 to 13 cm in a full-term infant. Graphs are available to estimate the proper length of insertion for a high or low catheter location (see Fig. 19-21*B*). Once sterile technique is broken, the line may not be advanced. It is therefore preferable to position the catheter too high and to withdraw as necessary according to the location on a radiograph. After it has been positioned appropriately, the catheter should be tied with the previously placed suture (see Fig. 19-23) and taped to the abdominal wall (Fig. 19-24). A radiograph should be obtained and the catheter repositioned, if necessary, with the tip at the lower border of the L3 vertebra. Some clinicians prefer to place the catheter high (T6 to T9 vertebrae). There are no unequivocal data to support either preference.

Radiographs of an arterial catheter (Fig. 19-25) will show the catheter proceeding from the umbilicus down toward the pelvis, making an acute turn into the internal iliac artery, continuing toward the head into the bifurcation of the aorta, and then moving up the aorta slightly to the left of the vertebral column. [62]

Most unsuccessful umbilical artery catheterization attempts fail because the catheter perforates the arterial wall approximately 1 cm below the umbilical stump, where the umbilical artery begins curving toward the feet. In this instance, the catheter is advanced in the extraluminal space, and resistance is met at 4 to 6 cm. The following maneuvers make it possible to avoid perforating the umbilical arterial wall in most cases:

1. The catheter should be advanced slowly. When slight resistance is met at approximately 1 cm, the catheter should be advanced very gently with steady pressure. It should never be forced, because it will likely perforate the wall. A catheter or feeding tube with a molded tip

should be used. A catheter tip that has been cut with scissors is more difficult to insert and advance.
2. Because the artery curves toward the feet, the umbilical stump should be held with a curved clamp and should be pulled toward the head so that the catheter is inserted toward the feet in as straight a direction as possible.

Figure 19-22 The umbilical cord is grasped with a curved hemostat near the selected artery. The umbilical artery is then dilated with a curved iris forceps.

Complications

If the catheter becomes plugged or fails to function properly or if there is blanching or discoloration of the buttocks, the heels, or the toes, then the catheter should be removed at once. Umbilical arteries are most easily cannulated in the first few hours of life but may provide a viable vascular route as late as 5 to 7 days of age.

Complications include hemorrhage, infection, thromboembolic phenomena (especially to the kidneys, the gastrointestinal tract, and the lower extremities), aortic thrombosis, aortic aneurysm, vasospasm, air embolism, vessel perforation, peritoneal perforation, and hypertension. [54] [63] [64]

Percutaneous Arterial Catheterization

Indications and Contraindication

Despite the growing use of noninvasive devices for monitoring transcutaneous oxygen and carbon dioxide, percutaneous

Figure 19-23 The catheter is introduced into the dilated artery and advanced toward the feet. The suture placed around the base of the cord is tied to the catheter.

peripheral arterial catheterization is indicated when there is a need for frequent blood gas sampling, continuous arterial blood pressure monitoring, or both. Arteries used for peripheral catheters in infants include the radial, [65] ulnar, [66] femoral, [67] temporal, [68] and posterior tibial arteries. [69]

Percutaneous radial artery catheterization has become widely accepted and has been shown to be a safe method in infants and children. The catheter allows for preductal blood gas determinations if placed in the right radial artery. Only the procedure for radial artery catheterization is described here, but catheterization of other vessels is similar.

The following are contraindications to peripheral arterial catheterization:

- Situations in which adequate peripheral arterial samples can be obtained by percutaneous punctures
- Situations in which circulation of the extremity to be catheterized is compromised
- Situations in which occlusion of the vessel to be catheterized results in compromised perfusion of that extremity
- The presence of an ongoing bleeding diathesis
- The presence of localized infection or inflammation overlying the artery to be cannulated
- Situations in which intensive monitoring of line function is not available.

Figure 19-24 The tape is pleated above and below the catheter.

Equipment and Setup

The equipment needed for arterial catheterization is essentially the same as that required for percutaneous peripheral venous catheterization (see Table 19-4). The catheters used are usually 22- or 24-ga over-the-needle catheters, a T-piece connector, and a stopcock. One should connect the T piece and the stopcock and then fill them with normal saline solution. An infusion pump with heparinized saline (1 to 5 U/mL) should be readied. [70]

Technique

The procedure should be performed with good lighting and an adequate work area, with the infant's heart and respiratory rates monitored closely. The radial artery may be palpated proximally to the transverse wrist crease on the palmar surface of the wrist, medial to the styloid process of the radius. The artery is then compressed, and the hand and fingers are observed for color change. If blanching or

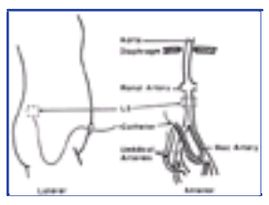

Figure 19-25 The umbilical artery catheter makes a loop downward before heading cephalad (schematic drawing of a radiograph interpretation).

cyanosis is noted (indicating poor collateral circulation), catheterization is not performed. If locating the artery by palpation is difficult, some clinicians find transillumination devices, [71] or Doppler probes helpful.

The infant or child's hand and lower forearm are secured to an arm board with the wrist dorsiflexed 45° to 60° with the aid of a roll of gauze placed underneath. Care must be taken to leave the fingers exposed to assess the peripheral circulation. The radial artery is palpated at the point of maximal impulse and can be marked with a gentle indentation by one's fingernail. The area over the radial artery is prepared with a povidone-iodine solution and washed with alcohol. Topical or local anesthetic (such as 1% lidocaine without epinephrine), or both, may be used at the planned insertion site. The catheter with stylet is inserted through the skin just proximal to the transverse wrist crease at a 10- to 20-degree angle (Fig. 19-26). The catheter with needle is advanced slowly until blood appears in the catheter hub, signifying puncture of

Figure 19-26 The catheter assembly is introduced into the radial artery through skin at a 10- to 20-degree angle. This is a smaller angle than is used for simple arterial puncture. Gloves should be worn.

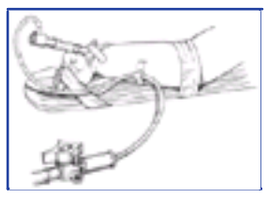

Figure 19-27 One technique of taping the arterial catheter. The arm board should be well padded and secured.

the anterior arterial wall. The catheter is slowly advanced until blood appears in the needle and then the needle angle is carefully lowered to approximately 10°. The catheter is slowly advanced over the needle into the lumen of the artery, and the needle is removed. The stopcock and T-piece connector are attached to the catheter hub. The stopcock is opened to the syringe to confirm pulsatile blood return. It is then flushed with 0.5 mL heparinized flush solution very gently to clear the catheter while the fingers and the hand are observed for evidence of blanching or cyanosis.

The puncture site is then covered with antibiotic ointment, and the catheter is fixed to the skin by a thin piece of tape placed adhesive side uppermost under the catheter hub and crossed over the catheter in a V shape. A second piece of tape is passed around and over the catheter hub and is fixed to the wrist (Fig. 19-27). A small piece of tape is used to attach the T-piece connector to the wrist area or to the splint. The fingers should be easily visible.

Only heparinized normal or half-normal saline is used for infusion. Some clinicians prefer to add 1 to 5 U of heparin per milliliter of infusion solution infused at a rate of 1 to 2 mL/hour. Medications, blood or blood products, amino acid solutions, IV fat solutions, and hypertonic solutions are not infused through the catheter.

The catheter must be removed when there is evidence of blanching or cyanosis or when it is impossible to withdraw blood from the catheter or difficult to flush the catheter.

Complications

Complications, which have been reported with every type of arterial catheter, include hemorrhage, thrombosis, spasm, infection, scars, air embolism, retrograde blood flow, transient elevation in blood pressure with rapid (less than 1 second) infusion, and nerve damage. Thrombosis or spasm may result in blanching or cyanosis of the extremity or skin.[72] There is potential for loss of digits, an entire extremity, or large areas of skin, as well as cerebral infarction with temporal artery catheters. [73] Saladino and colleagues note that complications from ED-placed arterial lines are uncommon and generally minor. [27]

Arterial Cutdown Catheterization

Indications and Contraindications

Arterial catheterization by cutdown on the posterior tibial artery, radial artery, and temporal artery may be indicated when the need exists for frequent monitoring of arterial blood gases or blood pressure and when percutaneous access is not possible. [69] Arterial cutdowns are contraindicated when (1) adequate peripheral blood gas samples can be obtained by percutaneous punctures or catheterization, (2) circulation of the extremity to be catheterized is compromised, or (3) occlusion of the vessel to be catheterized results in compromised perfusion of that extremity.

Equipment and Setup

Successful arterial cutdown catheterization in the small infant requires sterile instruments, an assistant, good lighting, and a selection of catheters. Previous clinical experience is helpful. The equipment required for performing an arterial cutdown catheterization can be found on a cutdown tray, available in most EDs. Also needed are a 22- or 24-ga over-the-needle catheter, T-extension connector tubing, stopcock, a 5- or 10-mL syringe filled with flush solution (normal saline with 1 to 5 U heparin/mL) and silk suture ties.

Technique

The anatomy (Fig. 19-28) and technique for posterior tibial arterial cutdown are described in detail (Fig. 19-29). The same technique is applicable for the radial artery. The clinician stabilizes the foot in a neutral position by taping the externally rotated lower leg to a splint. The *posterior* tibial artery is then localized by Doppler ultrasound just posterior to the medial malleolus. The operator prepares for the procedure by scrubbing and donning a gown and gloves; the foot is prepared with a povidone-iodine solution.

Following SQ injection of 1% lidocaine, a 5- to 7-mm transverse incision is made in the skin over the artery posterior to and at the midlevel of the medial malleolus (Fig. 19-29*A*). Using blunt dissection in a vertical direction (parallel to the vessels), the tissue is separated with a small, curved forceps, and the artery is identified. The artery courses with the vein just anterior and superficial to the nerve and is usually pulsatile. The artery is isolated by sliding a small, curved forceps beneath it and gently elevating the vessel (see Fig. 19-29*B*). Excessive

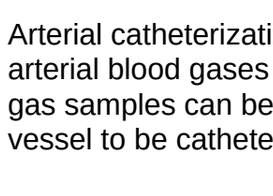

Figure 19-28 Anatomy of the posterior tibial artery and surrounding structures.

Figure 19-29 *A*, Posterior tibial artery cutdown technique. With the foot prepared and immobilized, a 5- to 7-mm incision is made in the skin posterior to and at the midline of the medial malleolus. *B*, A curved forceps and a silk suture are inserted beneath the posterior tibial artery, which courses just posterior to the medial malleolus.

manipulation of the artery can cause spasm; if this occurs, a few drops of 1% lidocaine applied locally may result in dilation. A silk tie (without a needle) is then placed beneath the artery to stabilize it during cannulation.

At a 10-degree angle, a 22-gauge Angiocath with the catheter bevel down is inserted into the artery over the surface of the forceps. When blood return is seen, the catheter is advanced over the stylet to its full length (Fig. 19-30). The needle stylet is then removed, and the catheter is connected to the T-connector tubing and a three-way stopcock that has

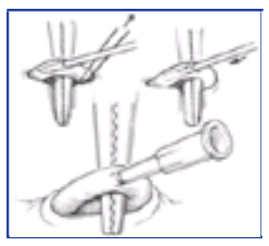

Figure 19-30 Technique of inserting the arterial catheter. A silk tie is used *only to stabilize the artery during cannulation.* It is never tied. The catheter is inserted under direct vision without making an incision in the vessel.

been prefilled with heparinized flush solution. Patency is checked by observation of blood return with pulsations; the catheter is then flushed slowly and gently. The silk suture is removed, and the skin incision is sutured. The catheter is sutured to the skin over the heel. The catheter and connector are then secured to the heel with tape (Fig. 19-31). The stopcock is then connected to the infusion line.

Complications

The complications of arterial cutdown are similar to those of percutaneous arterial catheterization. They include hemorrhage, thrombosis, or spasm resulting in loss of tissue; infections; permanent scars; and nerve damage. Complications have been reported with all types of arterial cutdown. Follow-up data and computed tomography data suggest an association

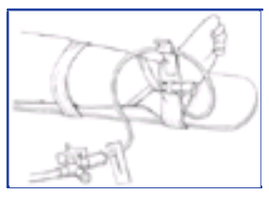

Figure 19-31 The skin incision is closed, and the catheter and connector are secured to the heel with tape.

between temporal artery catheterization by the cutdown technique and cerebral infarct that may result in hemiparesis. [74] Therefore, temporal artery catheterization should be the last choice for arterial catheterization.

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Chapter 20 - Arterial Puncture and Cannulation

Dave Milzma
Tim Janchar

Arterial puncture remains the current standard for blood sampling of acid-base balance and true arterial blood gas. Arterial blood pressure is considered second only to heart rate as the most important vital sign. The absence of arterial blood pressure defines cardiac arrest and serves as the best endpoint of resuscitation. Blood pressure can be obtained by noninvasive methods either by auscultation or automated pneumatic cuff, both of which can be unreliable at times. Both methods have known inaccuracies in determining true systolic blood pressure, but reasonably good accuracy with regard to mean pressure. [1] [2] Intra-arterial cannulation with continuous blood pressure transduction and display is currently the accepted standard for comprehensive arterial pressure monitoring. [1] [3] It is an invasive modality and requires expertise and sufficient support to prepare for accurate monitoring on a continual basis. Accurate monitoring of arterial pressure reflects the force of systemic perfusion and is the most important determinant of cardiac work.

Early scientists reported cutting down to obtain direct measurement of arterial pressure in animal models. [1] [2] J.L.M. Poiseuille first introduced the use of a mercury manometer for the measurement of blood pressure in 1828. Advances came slowly until the space age in the 1960s introduced electrical monitoring of arterial pressure with transducers and recorders rather than simple mechanics, thus permitting mathematical waveform analysis in addition to visual analysis. More recent advances have included continuous, invasive monitoring of arterial blood gas values. [4] Even such new improvements as continuous monitoring still require arterial puncture and cannulation. In the future the traditional, macroscopic invasive approach will likely give way to noninvasive molecular analysis, capable of detecting changes in arterial oxygenation and perfusion before a measurable clinical presence. It is a distinct possibility that comprehensive noninvasive monitoring will be introduced and even become the standard before the next edition of this text.

INDICATIONS AND CONTRAINDICATIONS

Despite the advent of new, noninvasive monitoring of respiratory functions and venous marker tests, there remain many patients in the emergency department (ED) whose care and resuscitation would be enhanced from arterial puncture or cannulation (Table 20-1). The indications for direct, arterial placement of a catheter for blood pressure monitoring fall into three major categories [5] [6] :

1. *Direct arterial blood sampling.* Catheter access removes the need for multiple arterial punctures and allows for either repeat arterial blood sampling or the placement of new sensors for continuous monitoring of blood gas and other chemistry values.
2. *Continuous real-time monitoring of blood pressure.* Patients with acute hypo-perfusion and those receiving vasoactive drug infusion require the superior monitoring and moment-to-moment change detection afforded by arterial catheterization.
3. *Failure or inability to use indirect blood pressure monitoring.* Some acutely ill patients such as those with severe burns or morbid obesity may need ongoing perfusion monitoring that can best be accomplished by arterial catheterization.

Although acute respiratory decompensation and metabolic emergencies are the most common reasons for arterial blood gas sampling, all lab tests are possible from arterial sampling. Blood cultures from an indwelling arterial line have sensitivity and specificity equal to those of cultures obtained from a venipuncture site. [7] [8] Patients with respiratory illness can be managed without arterial puncture due to recent advances in noninvasive pulse oximetry and continuous end-tidal carbon dioxide monitoring, but there is still a role for arterial blood sampling. The initial correlation between noninvasive values with acid-base status via arterial sampling is still imperative in critical illness to set a baseline or verify a trend. Metabolic and electrolyte monitoring often require additional arterial sampling, especially in patients with severe diabetic ketoacidosis who require frequent pH, electrolyte, and glucose measurements for accurate treatment.

Arterial systolic and diastolic pressures can be continuously and accurately observed by arterial catheterization with an electromechanical pressure transducer attached to a monitor.[6] Such capabilities are routinely used in the operating room or intensive care unit (ICU) and prove most useful when available in an ED for critically ill patients. Many interventions, such as the long-term use of vasoactive drugs (e.g., nitroprusside and dopamine), are best administered with continuous arterial pressure monitoring. The response of trauma and cardiac patients to acute resuscitative efforts also may be more easily followed by the use of arterial catheterization.

Arterial puncture is used routinely by cardiologists and radiologists for cardiac and neuro-angiography. Angiography is becoming a less common indication for arterial puncture in the ED due to advances in ultrasound, computed tomography-angiography, as well as rapid and routine magnetic resonance imaging with intravenous contrast. However, angiography is still considered for suspected cases of peripheral arterial trauma, suspected aortic injury and aneurismal or embolic disease.

Few contraindications to arterial puncture exist; none are absolute, but should always be considered on a risk-benefit evaluation. For example, post-thrombolysis, arterial cannulation should only be performed if it will provide essential data that cannot be obtained by any other method and the patient's condition requires ongoing assessments. Once a patient has received thrombolytic therapy, single arterial puncture of the radial artery is preferred. Arterial pucture considerations should be considered before administration of thrombolytic agents. Arterial puncture can be performed in patients who are anticoagulated or who have other coagulopathies, but should be undertaken with extreme care in patients with disseminated severe coagulopathies.

There are definite reports of patients with complications from bleeding requiring transfusion. Some patients, all of whom were anticoagulated at the time of puncture, even suffered compression neuropathies secondary to hematomas as a result of arterial puncture. [9] Repeated arterial sampling in these patients should be accomplished by insertion of an

TABLE 20-1 -- Arterial Puncture and Cannulation

Indications	Relative Contraindications	Strict Contraindications
Blood gas sampling	Previous surgery in the area, especially cut-down	Inadequate circulation to the extremity
Continuous pressure monitoring		Raynaud syndrome
Frequent need for blood sampling (ongoing resuscitation)	Anticoagulation	Buerger disease (thromboangiitis obliterans)
	Coagulopathy	
Inotropic support—use of continuous infusion of vasoactive agents	Skin infection at the site	Full thickness burns
	Atherosclerosis	
Major surgery involving fluid shifts/blood loss	Inadequate collateral flow	
Hypothermia-induced or severe exposure	Partial thickness burns	
Diagnostic angiography		
Therapeutic embolization		

indwelling cannula to minimize the number of puncture sites in the arterial wall and should be performed by only the most experienced clinicians.

The presence of severe arteriosclerosis, with or without diminution of flow, is only a relative contraindication to arterial puncture, especially when followed by cannulation. Often patients require continuous hemodynamic monitoring for vasopressor administration. Combined with advanced cardiovascular disease, this makes invasive monitoring more valuable than most risks.[6] An alternative site should be considered if an isolated decreased palpable pulse or bruit is felt over a selected site. Evidence of decreased or absent collateral flow in areas where flow normally exists, such as a positive Allen test (discussed later in Techniques), should also lead one to consider an alternative site. One must avoid puncture of a specific arterial site when infection, burn, or other damage to cutaneous defenses exists in the overlying skin.

EQUIPMENT

To obtain a single sample of arterial blood by the percutaneous method, a 3-mL syringe (preferred and most common) is attached to a needle. Needle size should vary based on puncture location and patient size and age. Most adults should have a 20-gauge (ga), 2.5-in. needle for a femoral sample and a 1.25-in. 22- to 23-ga for a radial artery puncture. For pediatric arterial sampling, slightly shorter length needles in the range of 22- to 24-ga should be used in the same sites as in adults.

Precoated blood gas syringes allow for longer shelf life and ready use. If necessary, a regular syringe may be prepared with 1 or 2 mL of a heparinized saline solution (1000 IU/mL) drawn into the syringe to coat the barrel and needle. The heparin must be fully ejected through the needle immediately before puncture to minimize heparin-related errors.

Even dry heparin may produce arterial blood gas (ABG) result abnormalities due to a heparin-induced dilutional effect. [10] The newest blood gas and chemistry analyzers require only 0.2 mL of whole blood for accuracy and some point of care devices can perform analysis on drops of blood. However, sample sizes aspirated in heparin-coated syringes with <1.0 mL will result in heparin error to ABG values.

Stored heparin solution has a higher PO_2 and a lower PCO_2 than blood.[11] A dilutional effect from heparin would mean that the addition of 0.4 mL of heparin solution to a 2-mL sample of blood (dilution of 20%) will lower the pCO_2 by 16%.[10] Proper technique with full ejection of excess heparin will prevent such problems. A falsely low pCO_2 is the most clinically significant change caused by excess heparin. [10] [11] Neither pO_2 or pH levels are significantly altered by the addition of heparin in most instances, although a slight increase in pO_2 and minimal decrease in pH may occur if high concentrations of heparin are used (25,000 IU/mL). [12]

The fluid-filled recording systems used with arterial cannulation have a great influence on the accuracy of pressure measurements. The frequency responses of tubing, transducers, and other components of the monitoring system influence the measurement accuracy of systolic and diastolic pressures. Failure to recognize recording system artifacts will lead to errors in pressure interpretation. [2]

Various catheter types have demonstrated similar frequency response characteristics, but some studies have found more variable effects on complication rates. There is some debate over complications due to catheter composition, such as possible increased thrombosis with Teflon catheters. [13] [14] Another contributing element leading to thrombosis is catheter diameter; the incidence of thrombosis is inversely related to the ratio of vessel lumen to catheter diameter. [15] Thus, the risk for thrombosis decreases as the catheter diameter decreases. Incidence of thrombosis also increases with increased duration of catheter placement.

There are specific recommendations relative to vessel and puncture site location. Table 20-2 lists the usual equipment for arterial cannulation, although many kits contain most needed supplies. Shorter catheters are ideal for peripheral artery cannulation, whereas a longer catheter using the Seldinger technique is preferable for the femoral artery (Fig. 20-1). Some studies suggest that thrombosis is less likely with a nontapered catheter. [16] As mentioned in the preceding paragraphs, an 18- to 20-ga catheter should be used in adults to cannulate the femoral artery. Small children and infants require a 22- to 24-ga catheter, which may need to be inserted through a femoral cutdown technique. Older pediatric patients usually require 20- to 22-ga catheters, based on the patient's size.

The tubing that connects the catheter to the pressure transducer has a significant effect on monitoring system accuracy. The higher the frequency response of the entire system, the more accurate the determination of systolic and diastolic pressure; however, artifact also becomes more of a problem. [5] [17] Stiff, low-capacitance, plastic tubing should be used for arterial catheterization and monitoring. The electronic pressure transducer connection should be placed as close as possible to the patient and zeroed appropriately, because the frequency response of a tube is inversely related to its length. [17] [18] [19]

TABLE 20-2 -- Equipment for Insertion and Maintenance of an Indwelling Arterial Cannula

Percutaneous insertion
Iodophor solution (alcohol, only if patient is allergic)
1% lidocaine (without epinephrine)
10-cm × 10-cm dressing sponges
Adhesive tape
Iodophor ointment
Arm board for brachial, radial, or ulnar cannulations
Appropriate-sized intravenous catheters
Syringes
Pressure tubing
Two 3-way stopcocks
Pressure transducer
Connecting wire
Monitor display
500–1,000 mL Bag of normal or heparinized saline
Pressure blood infuser, set up with continuous flush device
Additional equipment required for cutdown insertion technique
Scalpel blade (No. 11)
4-0 nylon or silk suture on skin needle
Needle driver

The pressure wave produced with each contraction is transmitted from the artery through the catheter and connecting tubing to a measuring device. The arterial fluid wave is received by an electromechanical transducer, which changes the mechanical pressure wave into an electrical signal that can display on the monitor. With the use of minicomputers, various numerical values and hemodynamic paramenters can be displayed or stored for future analysis. Currently, electronic transducers are most commonly used, but the technology is likely to change. The most basic system for obtaining blood pressure values uses a manometer, which has been used since antiquity as the standard reference for measuring pressure. [20]

After arterial cannulation, a mercury manometer can be connected to the arterial system by a catheter filled with

Figure 20-1 Catheters for arterial cannulation. A standard intra-catheter for arterial cannulation may be used for direct vessel cannulation. Newer devices with a modified guidewire and quick-flash identifier (Arrow International, Inc. Reading, PA) or a needle with an attachable guidewire to aid cannulation are also available.

heparinized saline.[21] [22] The catheter is arranged to have a fluid meniscus at the same level as the heart when there is no pressure input. The meniscus is below an air column in a vertical tube long enough to avoid saline contamination of the manometer at maximal pressures (Fig. 20-2). Although excellent for recording steady pressures, it cannot respond to pressure changes more than a cycle every 2 to 3 seconds. Thus it displays only a mean value (mean arterial pressure [MAP]) in millimeters of mercury (mm Hg). Current high fidelity systems record pressure cycles up to 500 cycles/sec and display the systolic and diastolic pressure, MAP, trend monitoring, and other calculated hemodynamic values. [21]

A continuous method of pressure tubing flush is required to maintain the patency of the catheter lumen during intra-arterial pressure monitoring. A three-way stopcock through which the tubing is intermittently flushed (a minimum of every 15 to 30 min) with saline is a simple, effective method. Continuous flush devices push a set amount of fluid (usually 2 to 3 mL/hour) through the line. [6] A typical monitoring system that includes this device is shown in Figure 20-3 . The pressure transducer must be mounted at the level of the patient's heart.

Intravascular transducers are also available but have many potential disadvantages and are used infrequently. Most of the numerous brands are fragile, temperature sensitive, of variable quality, and much more difficult to place into vessels than catheters. Anecdotal findings of fibrin deposition on these devices have been noted, but no reports demonstrated any increased incidence of thrombus formation. The greatest advantage of these intravascular transducers is the continuous trending of arterial gas values and the elimination of potential error induced by catheters, stopcocks, and connecting tubing. [23] [24] Despite obvious advantages, the use of these expensive, disposable devices has yet to penetrate many EDs. Usage of these devices is likely to happen only after it occurs in other hospital settings, such as the ICU.[4] So many transducer and monitor combinations exist that a discussion of their relative merits is beyond the scope of this chapter.

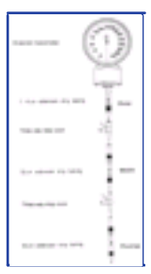

Figure 20-2 Assembly technique for anaeroid manometer system. The middle and proximal extension tubings contain heparinized saline. The middle extension tubing is arranged to form a fluid meniscus at the same level as the heart when the proximal stopcock is closed to the middle tubing (i.e., no pressure input). The distal extension tubing is filled with air and held vertically so that there is no saline contamination of the manometer at maximal pressures. Approximately 10 to 12 cm of air in the distal and middle tubings is optimal. The same system can be used with a mercury manometer in place of the anaeroid manometer. Sterility of the extension tubing and stopcocks is essential. *(From Zorab JSM: Continuous display of the arterial pressure: A simple manometric technique. Anaesthesia 24:433, 1969. Copyright © 1969 by the Association of Anaesthetists of Great Britain and Ireland. Reproduced by permission.)*

SITE SELECTION

The radial, brachial, and femoral arteries are usually punctured for blood gas sampling in adults. Pediatric sites also include foot sites and the uterine artery in newborns.

The procedure is usually accomplished at the bedside using percutaneous methods similar to central venous catheter placement to cannulate arteries. Technique depends on site selection: Radial and dorsal pedalis artery cannulation is usually placed with an over-the-needle catheter with or without a guidewire. Larger vessels such as femoral or axillary always use needle puncture of the artery followed by the catheter placed over a guidewire (Seldinger technique). In decreasing order of frequency, the following arteries are used in adults and children: radial, femoral, brachial, axillary, dorsalis pedis, and ulnar. The temporal and umbilical arteries are often cannulated in infants and neonates. Such arteries, as well as the radial artery, are very safe for these patients (see Chapter 19).[25]

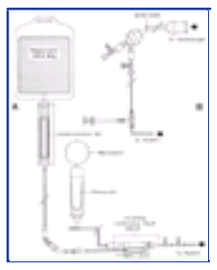

Figure 20-3 Arterial pressure monitoring systems. *A,* System for continuous flush. Heparin (2 mL of a 1:1000 unit solution) is added to a 1-L bag of normal saline, and the bag is pressurized to 300 mm Hg using a metered blood pump (not shown). The continuous flush device is set to deliver 3 mL/hour of the heparinized saline. A mechanical pressure transducer is depicted. The transducer device is a sterile, inexpensive, fully assembled monitor that can be used during patient transfer. Alternatively, the electronic transducer depicted in *B* may be used. *B,* System for manual flush. A heparinized saline flush solution can be injected manually through a syringe at the proximal or distal port. The transducer dome should be maintained at the level of the patient's heart. *(From Beal JM [ed]: Critical Care for Surgical Patients. New York, Macmillan, 1982. Reproduced by permission.)*

The potential consequence of complete blood flow loss through a vessel due to intraluminal thrombosis must be considered when choosing a site for arterial puncture. Since the most frequent complication of arterial catheterization is bleeding, the ability to control hemorrhage must also be considered. For these reasons, the radial and femoral arteries are favored due to their good collateral blood flow and ease of compression in case of hemorrhage. Patient comfort and nursing care concerns also should be considered during site selection.

The arterial pulse is palpated to ascertain the location of the vessel, and the overlying skin is prepared sterilely with an iodophor or other antiseptic solution. The patient's skin should then be anesthetized with a wheal of local anesthetic (1% lidocaine) without epinephrine placed through a small needle (25- or 27-ga). One study found no significant alterations in pCO_2 or pH from the pain or anxiety of an unanesthetized arterial puncture (Table 20-3).[26] If the patient is

TABLE 20-3 -- Parameters that Affect Interpretation of Arterial Blood Gases

Parameter	Heparin*	Air Bubble in Sample	Delayed Analysis?
pO_2	No significant change†	Elevated	Variable¶
pCO_2	Lowered‡	No significant changes§	Elevated£
pH	Unchanged‡	No significant changes§	Lowered£

*Use only 1000 IU/mL concentration. Fill dead space of needle and syringe only, and collect 3 mL of blood.
?Anaerobic storage at room temperature for 20 minutes results in no significant change.
†There are reports of slight increases in pO_2 with excessive heparin.
¶Changes unpredictable at 20 minutes, regardless of storage method.
‡The falsely lowered pCO_2 that occurs with added heparin is the most clinically significant change noted. pH may be decreased if a large volume of concentrated heparin (25,000 IU/mL) is used.
§If stored at 4°C for 20 minutes.
£Minimal changes up to 2 hours, if stored at 4°C.

cooperative (with careful discussion of the procedure before-hand), in extremis, or unresponsive to pain in the area to be punctured, this anesthetic infiltration may be omitted. If local anesthesia is to be performed, care must be taken to use a small amount of local anesthetic—a large wheal will obscure the pulse.

The arterial pulsation is then isolated with the index and middle fingers of the gloved, nondominant hand and the vessel course identified. The skin should be punctured through the anesthetic wheal, immediately distal to the palpated pulse under the index finger (Fig. 20-4). The older technique of placing the needle between the index and middle finger risks self-puncture and is no longer advised. The needle should be advanced slowly toward the pulsating vessel at an approximate 30° angle with the skin. A larger angle is required for femoral artery puncture. Once the needle enters the arterial lumen, the syringe plunger should be allowed to rise with the arterial pressure on its own in order to discriminate between chance of venous sampling. If no blood flow is obtained, the needle should be withdrawn slowly, because both walls of the vessel may have been punctured. A sample may be obtained during needle withdrawal. Redirection of the needle should occur only when the needle has been retracted to a location just deep to the dermis.

There are reports of using either hand-held Doppler or ultrasound probe to assist in vessel location for both venous and arterial puncture and cannulation. [27] Although viable in difficult cases, the clear majority of procedures should be accomplished without this extra delay or expense. However, both ultrasound and handheld Doppler have had increased usage due to improved technology and decreased costs. If used, the probe should be held over the artery proximal to the puncture site. An important indication of vessel identification is loss of audible pulsations with compression. After at least 1 to 2 mL of blood has been obtained, the needle is removed from the artery. Firm pressure is applied at the puncture site for a minimum of 5 minutes. If the patient is on anticoagulant therapy or has a coagulopathy, 10 to 15 minutes of pressure is required.

Proper handling of the sample and rapid analysis is very important.

When the needle is withdrawn, it is imperative to expel any air bubbles present in the syringe to avoid false elevation of the pO_2.[28] Removal of air is neatly and easily accomplished by tapping the inverted syringe (needle pointing to the sky) to force any air to the top; then carefully and slowly depressing the syringe plunger to push out remaining air. A gauze pad or alcohol wipe when pierced by the needle and placed at the hub will collect any expelled blood (Fig. 20-5). The needle is removed and the syringe is capped to ensure anaerobic conditions. Air in the sample will significantly increase the pO_2 (mean increase, 11 mm Hg) after 20 minutes of storage, even if kept at 4°C. The pH and pCO_2 are not significantly altered by air bubbles if the blood is stored at 4°C for 20 minutes without significant deterioration. [12] [28] If blood is stored at room temperature for longer than 20 minutes, the pCO_2 will increase and the pH will decrease, probably as a result of leukocyte metabolism. In a stored sample, the pO_2 varies to such an extent that the change is unpredictable for chemical interpretation at 30 minutes, regardless of storage method. High leukocyte or thrombocyte counts, such as those seen in leukemic patients, may shorten acceptable storage intervals. [29] [30]

In summary, arterial blood gas samples should always be kept on ice and analyzed within 15 to 20 minutes. Samples that cannot be analyzed any sooner should be considered faulted because they will not reflect real-time patient perfusion or oxygenation status.

Percutaneous Technique for Arterial Cannulation

Direct Over-the-Needle Catheter Cannulation

Placement of an angio-catheter directly into an arterial lumen in a manner similar to placement of an intravenous catheter is the most practiced and simplest method—when it works. The only routine site for this technique is the radial artery. Although other sites, namely the femoral artery, may be attempted, use of a catheter over a guidewire is strongly advised at most other sites.

The operator should always remember to take time for proper alignment of the desired site. Delays, complications, and inability to successfully cannulate an artery often occur due to failure to properly prepare the desired site and involved limb. An important preparatory step is ensuring that the target limb is secured flat and not rotated; any rotation could result in the desired artery being shifted from the expected anatomical position, making it more difficult to cannulate. For example, to adequately prepare the radial artery, the wrist and hand should be immobilized in mild, dorsiflexion with some padding for support underneath the wrist. As usual, sterile skin prep and local anesthetic injection using a 25-ga or smaller needle with sufficient infiltration to assure a painless procedure can then be performed. The subcutaneous infiltration of lidocaine or similar anesthetic may reduce vessel spasm at the time of arterial puncture.

The catheter assembly should be checked for proper movement and function. Alternatively, a 3-mL syringe with the plunger removed could be used as a blood reservoir. The catheter is advanced toward the palpated artery at a comfortable angle for the operator, generally 30 to 45° from the skin. Making a small nick with a No. 11 scalpel or a larger bore needle will eliminate the problem of catheter damage from kinking on the skin. The needle tip is often perceived to pierce the artery, but successful puncture is confirmed by identifying a "flash" of arterial blood flow into the needle hub and reservoir. As the needle-catheter assembly advances through the

Figure 20-4 Arterial puncture: The syringe-needle is held at a 30 to 45° angle and should enter with the bevel up and advanced slowly. The landmark is best located by palpating the pulse using the index and middle fingers placed immediately proximal to the needle entry site. The needle should *not* be passed between the fingers due to risk of operator self-injury. *(Picture courtesy of JMM, with permission.)*

skin toward the artery, the initial flash of arterial blood is obtained by the needle alone, which protrudes beyond the catheter. For this reason, the needle-catheter should be lowered and advanced 2 mm forward to ensure that the catheter tip has cannulated the vessel, along with the needle. Confirmation of the catheter within the vessel lumen will be continued arterial blood return. The catheter alone can now be advanced over the needle with care into the artery. If the catheter fails to thread, it has not properly entered the vessel lumen and should not be forced to advance without active blood return confirming placement.

When blood flow into the needle-catheter has ceased, it is likely to have pierced the back side of the artery wall. This double-puncture method is useful for cannulating small vessels, yet it is not recommended to the inexperienced clinician

Figure 20-5 Removal of air bubbles from the syringe. Air bubbles are finger-tapped to the top of the syringe. An alcohol swab is placed over the top of the needle. The plunger is advanced to expel air while drops of blood are collected on the alcohol swab. After removal of the bubbles, the syringe is capped and sent to the laboratory.

as a routine procedure.[5] If double puncture has occurred and blood has ceased to flow into the collection reservoir, the entire needle-catheter assembly should not be removed. Instead, simply retract the needle slightly to determine if blood flow can be re-established into the catheter. If blood flow occurs, gently advance the catheter. If not, the catheter should be slowly withdrawn until pulsatile blood flow reappears and then the catheter can be advanced into the artery. The important point is for the clinician to be aware whether the needle tip or the catheter is the leading edge within the vessel. [5]

Once the catheter is fully advanced into the vessel lumen, occlusive pressure is held transiently on the proximal artery to limit blood loss, and the needle is removed. A narrow bore, low compliance pressure tubing is then fastened to the catheter. An appropriate sterile dressing should be applied after the apparatus has been securely sutured to the wrist.

Occasionally one will encounter difficulty advancing the catheter into the lumen. The "liquid stylet" method may aid further passage of the catheter. [31] A 10-mL syringe should be filled with about 5-mL of sterile normal saline. The syringe is then attached to the catheter hub, and 1 to 2 mL of blood should be easily aspirated to confirm intraluminal position. The fluid from the syringe is then slowly injected, and the catheter is advanced behind the fluid wave. Alternatively, a more popular method is to use a guidewire that easily passes into the vessel lumen. [32] [33] This modified Seldinger technique is discussed fully in the next section.

The number of repeat attempts with additional arterial punctures increases the size of the developing hematoma and real risk of vessel wall damage, thrombosis, and even loss of arterial flow through the vessel. Despite the added trauma, there is no reported increase in complications when both walls, rather than one, are punctured from a single cannulation attempt.[34] [35] [36]

Guidewire Techniques for Arterial Cannulation

Using a "modified" Seldinger technique can often rescue a failed over-the-needle catheter, direct cannulation attempt. If the catheter has been placed in the arterial lumen with blood return, a proper sized guidewire may be gently passed through

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the catheter into the artery. The catheter should then be advanced fully into the vessel over the guidewire. The clinician is cautioned that stiffer guidewires, unlike most prepackaged ones, do not have a softer tip and the vessel wall may be damaged, even perforated with excessive force. Alternatively, catheter sets are available with a wire stylet that permits a modified Seldinger technique for catheter placement. The over-the-needle catheter follows the self-contained guidewire during cannulation. Numerous commercially available sets feature differing styles of guidewire and reservoir attachments to an over-the-needle catheter assembly. One readily available device of this type is the Arrow arterial catheterization system (Arrow International, Inc., Reading, PA) (Fig. 20-6). These kits are extremely practical for smaller vessels, especially radial, brachial, and axillary arteries and have excellent success rates at first-time placement. Although some authors have suggested that guidewire-based techniques will improve arterial cannulation success rates in some patients, [33] it appears that success is more a function of operator experience and personal preference. [37]

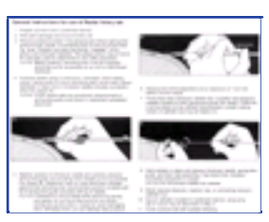

Figure 20-6 Step-by-step arterial cannulation, using the guidewire technique (Arrow arterial catheterization kit). *(Courtesy of Arrow International, Inc., Reading, PA.)*

Seldinger Technique

An alternate to placing an indwelling cannula is the Seldinger technique, [32] which is described in detail for venipuncture in Chapter 22 . Overall success rates with the Seldinger, guidewire-directed technique are superior compared to direct arterial cannulation. [37] There are a few available kits designed specifically for arterial cannulation (Fig. 20-7), but single lumen venous catheters with guidewires may be used if catheter size and length are appropriate for specific arteries (see following section for guidelines). Guidewire technique should be used initially for critical patients.

A needle is percutaneously placed into the arterial lumen, as described previously. A guidewire is then placed through the needle into the vessel lumen, and the needle is removed. A catheter is then threaded over the wire, and the wire is pulled out. Although most kits have vessel dilators, especially with larger catheter sizes, caution is advised. Only the tract should be dilated, not the artery, to avoid unnecessary blood loss and excessive arterial injury.

Figure 20-7 Femoral vessel cannulation kit. (Arrow International, Inc., Reading, PA.) A 16-ga single lumen vessel with a guidewire for Seldinger placement includes entire

procedural needs with local anesthetic, subcutaneous puncture syringe, needle, scalpel, and suture material.

Cutdown Technique for Arterial Cannulation

The cutdown technique is rarely practiced, but in certain circumstances it may be used to obtain arterial access by the most experienced personnel. Cannulation is performed after direct visualization of the vessel. A cutdown can be performed on any artery but is most commonly reserved for distal lower limb arteries and, rarely, the brachial. After a site has been selected, the overlying skin should be surgically prepared with an iodophor solution. Using sterile technique, local anesthetic solution is injected subcutaneously in a horizontal line 2 to 3 cm long and perpendicular to the artery. This step may be omitted if the patient is unconscious or otherwise anesthetic at the cutdown site.

Using a scalpel with a No. 10 or 15 blade, the skin is incised along the anesthetic wheal. Underlying tissues are spread parallel to the artery with a mosquito hemostat. The pulse is palpated repeatedly throughout the procedure to ensure proper positioning. Once the surrounding soft tissue has been removed, and after exposing the artery approximately 1 cm, the artery should be isolated by passing 2 silk sutures underneath it, using the hemostat. Strip away only enough perivascular tissue to expose the artery. Perivascular tissue will help limit bleeding at the time of catheter removal. An over-the-needle catheter device, such as that used in the percutaneous method, is then introduced through the skin just distal to the incision and advanced into the surgical site (Fig. 20-8).[31] Alternatively, a modified Seldinger guidewire setup may be used to catheterize the artery. The arterial wall is punctured with the needle tip, and the catheter is threaded into the vessel lumen. When this has been accomplished, the 2 silk sutures, which have been used only to control the vessel, are removed, and the skin incision is closed. *The artery is not tied off as the vessel would be during a venous cutdown.* Firm pressure, as used following arterial puncture, should be applied over the cutdown site. The separation of the soft tissue during the procedure may allow considerable hemorrhage into the tissue if pressure is not applied.

Catheter and Site Care

Once the catheter has been placed successfully, it should be advanced until the hub is in contact with the skin. The catheter is then secured by fastening it to the skin with suture material. Silk (2-0) or nylon (5-0) sutures provide the best anchoring. To accomplish this, a moderate bite of skin is taken with the needle, and a knot is tied in the suture leaving both tails of the suture long. Take care to avoid pinching the skin too tightly. The loose ends of the suture should then be tied around the catheter at its hub. Then, after laying two ties, a second set of knots should be placed on the back portion without occluding the lumen by constriction (Fig. 20-9).

After tying the catheter in place, a drop of antibiotic ointment is applied to the puncture site, [38] and a self-adhesive dressing is applied over the area. The catheter and its connecting tubing are further secured with sterile sponges and adhesive tape. All tubing connections must be tight and secure. If the tubing becomes disconnected inadvertently, the patient may exsanguinate rapidly.

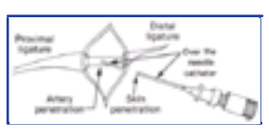

Figure 20-8 Placement of an arterial line using the cutdown technique. Note that the catheter enters the surgical wound percutaneously to minimize bacterial entry into the healing wound and permit better stabilization of the catheter. Catheter entry of the vessel is more parallel to the vessel than is illustrated. Ligatures are used only to *temporarily* isolate the artery and to control bleeding. *The artery should not be tied off.* The catheter is secured by suturing the hub to the skin (see Fig. 20-9).

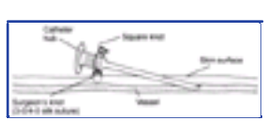

Figure 20-9 Illustration of a technique for securing a vascular catheter to adjacent skin.

Fluid-Pressurized Systems

When successful arterial cannulation has been performed, the catheter should be attached to a pressurized fluid-filled system. A three-way stopcock is commonly interposed between the patient and the transducer for blood gas sampling and to allow flushing of the system. Flushing can be periodic or continuous at a rate of 3 to 4 mL/hr through a continuous flow device (Fig. 20-10). Many institutions have begun using normal saline in place of heparinized solution to maintain patency. There is no significant difference for patency; however, heparinized flush solution in pressurized arterial lines results in greater long-term accuracy of pressure monitoring, but no real difference in catheter blockage has been reported. [39]

Literature review supports the use of normal saline solution for maintaining patency of intermittent vascular catheters. In one study, a change to normal saline solution as an alternative to heparinized saline solutions (2 mL 1:1000 heparin/L of saline) to maintain arterial line patency resulted in elimination of heparin-associated risks such as drug incompatibilities, thrombosis, local tissue damage, and hemorrhage. Additionally, decreased potential for infections, substantial money savings, and decreased nursing time make it an attractive alternative. There are, however, some studies that found the use of saline as a continuous flush for radial artery catheters are associated with an increased frequency of catheter occlusion and malfunctions compared with solutions containing heparin. [40] [41] For

Figure 20-10 Pressure transducer fluid monitoring set; a pressurized saline (heparinized solution may be substituted) connected to an intravascular line and arterial catheter, which is then fixed to a three-way stopcock. The fluid wave is directly monitored by the electormechanical transducer, which records pressure cycles and displays multiple hemodynamic values.

short-term setups as in the ED, saline is sufficient. Change to heparinized flush is an option depending on prevailing practice within the ICUs of the institution.

Procurement of a blood sample from the arterial catheter system is easily performed. A syringe is attached to the three-way stopcock, and blood is aspirated and discarded to clear the line. Studies examining the necessary discard volume of flush-blood solution have found considerable variation dependent on the volume of the system.[42] [43] Short lengths of tubing between the catheter and aspiration port minimize the discard volume. For a tubing length of 91 cm (36 in.), 4 to 5 mL should be aspirated[43] ; for a tubing length of 213 cm (84 in.), 8 mL should be aspirated. [42] A second syringe, which has been heparinized, is then attached, and 3 mL of blood are aspirated and sent for blood gas analysis. If the blood is to be used for other tests, the second syringe does not need to be heparinized. The stopcock and line should be flushed after sampling to avoid clotting.

SELECTION OF ARTERIES FOR CANNULATION

Radial and Ulnar

The artery most frequently used for prolonged cannulation is the radial artery. Widespread collateral flow exists in the wrist due to two major palmar anastomoses known as *arches* (Fig. 20-11). The superficial palmar arch lies between the aponeurosis palmaris and the tendons of the flexor digitorum sublimis. The arch is formed mainly by the terminal ulnar artery and the superficial palmar branch of the radial artery. The other major communication of these two vessels, the deep palmar arch, is formed by connections of the terminal radial artery with the deep palmar branches of the ulnar artery. [44] Some collateral flow is almost always present at the wrist, with the deep arch alone being complete in 97% of 650 hand dissections at autopsy. [45] Despite these findings, Friedman noted the absence of palpable ulnar pulses in 10 of 290 (3.4%) healthy children and young adults. [46] Interestingly,

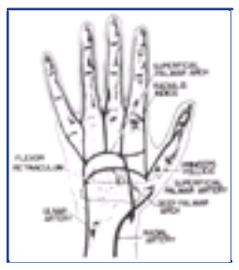

Figure 20-11 Arterial anatomy of the hand and wrist. *(From Ramanathan S, Chalon J, Turndorf H: Determining patency of palmar arches by retrograde radial pulsation. Anesthesiology 42:758, 1975. Reproduced by permission.)*

this was always a bilateral finding. Radial pulses were present in all subjects.

Before attempting radial artery cannulation, assess the adequacy of collateral flow to the hand by performing a bedside examination. This examination was originally described by E.V. Allen in 1929[47] and used to assess arterial stenosis in the hands of patients with thromboangiitis obliterans. The Allen test is performed to identify patients with increased risk for ischemic complications from radial artery catheterization. The procedure has seen many modifications [48] [49] since originally being described in a cooperative patient. The basic Allen test is performed as follows: The examiner occludes both the radial and ulnar arteries with digital pressure, and the patient is asked to tightly clench the fist repetitively to exsanguinate the hand. The hand is then opened, and the examiner releases the occlusion of the ulnar artery (Fig. 20-12). After 2 minutes, the test is repeated with release of the radial artery. Rubor should return rapidly to the hand with release of pressure from either vessel.

An abnormal (positive) Allen test, suggestive of inadequate collateralization, is defined as the continued presence of pallor 5 to 15 seconds after release of the artery.[15] [32] [49] [50] If the return of color takes >5 to 10 seconds, radial artery puncture should not be performed. Be careful to avoid overextension of the hand with wide separation of the digits, because this may compress the palmar arches between fascial planes and give a false-positive result. [51] Barber and associates[47] reported a modified Allen test that is useful in unconscious or anesthetized patients who cannot clench their fists. An Esmarch bandage is used to exsanguinate the hand, and the test is performed as previously described. Time permitting, performance of some variation of the Allen test is desirable before ulnar or radial puncture for cannulation or blood gas sampling.

The true predictive value of the Allen test is still questioned, as there are numerous reports of permanent ischemic sequelae, post-cannulation, following a normal Allen test.[49] [52] [53] Notably,

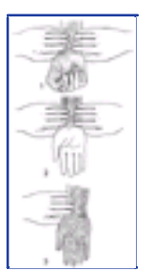

Figure 20-12 Allen test. Before puncturing the radial artery it is important to identify a competent ulnar artery. This can be done as follows: (*1*) The examiner compresses both arteries, and the patient repeatedly makes a tight fist to squeeze all the blood out of the hand. (*2*) The patient then extends the fingers, and the examiner observes the blanched hand. (*3*) Compression of the ulnar artery is released, and the examiner observes the hand filled with blood. If filling does not occur within 5 to 10 seconds, radial artery puncture should not be done. If brisk filling occurs, the test is then repeated with release of the radial artery to assess radial artery patency. If both radial and ulnar arteries demonstrate patency, the wrist may be used for arterial puncture. *(From Schwartz GR [ed]: Principles and Practice of Emergency Medicine. Philadelphia, WB Saunders, 1978, p 354. Reproduced by permission.)*

other studies have found no ischemic complications following radial artery catheterizations after abnormal Allen tests. [35] [54] Although there are no guarantees against digital ischemia following radial artery cannulation, [55] the finding of an abnormal Allen test should result in the search for an alternative site. If available, this alternative arterial site should be used and the abnormal Allen test documented for medicolegal reasons.

Once adequate collateral flow has been ascertained, arterial puncture may be performed. At the wrist, the radial artery rests on the flexor digitorum superficialis, flexor pollicis longus, and pronator quadratus, and against the radius. [45] The pulsation of the artery should be isolated on the palmar surface of the wrist. The radial artery is more superficial closer to the wrist and provides a more consistent cannulation due to fixation and less mobility. Dorsiflexing the wrist at about a 60° angle over a towel or sandbag, preferably

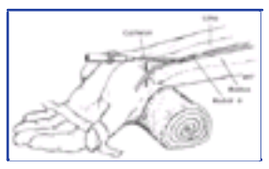

Figure 20-13 Percutaneous arterial cannulation at the wrist. The catheter unit is advanced 1 to 2 mm into the vessel lumen after blood first appears in the flash chamber. While the needle is fixed, the catheter is threaded over the needle. *(From Beal JM [ed]: Critical Care for Surgical Patients. New York, Macmillan, 1982. Reproduced by permission.)*

fixing the wrist to an arm board, will also significantly help isolate the artery. This degree of preparation should be considered standard when time for setup is allowable (Fig. 20-13). [34] [35]

Antegrade radial artery cannulation may be accomplished in infants and children when radial arteries are obstructed and retrograde blood flow is observed during a failed cut down attempt at standard retrograde arterial cannulation. [56] Additionally, displacement of perivascular interstitial fluid in neonates and bright light makes the course of the artery visible so that under direct vision, cannulation of the artery becomes as easy as venous cannulation. [57] Doppler ultrasound use on select patients with poor peripheral pulses may facilitate percutaneous radial artery cannulations and minimize the number of punctures needed for placement. [27]

The ulnar artery is seldom used because its smaller size makes it more difficult to puncture than the radial artery. At the wrist, the ulnar artery runs along the palmar margin of the flexor carpi ulnaris in the space between it and the flexor digitorum sublimes. [45] Caution is necessary because the artery runs next to the ulnar nerve as both pass into the hand just radial to the pisiform bone. The ulnar artery can also be made more accessible with dorsiflexion of the wrist.

Brachial

The brachial artery appears safe for arterial puncture, but it does not have the anatomic benefit of the collateral circulation found in the wrist. The brachial artery begins as the continuation of the axillary artery and ends at the head of the radius, where it splits into the ulnar and radial arteries. The preferred puncture site of the brachial artery is in or just proximal to the antecubital fossa. In this region the artery lies on top of the brachialis muscle and enters the *fossa* underneath the bicipital aponeurosis with the median nerve occupying the medial side of the artery (Fig. 20-14). Both the radial and axillary arteries are preferred upper extremity sites to the brachial artery. There is an increased ischemic complication risk from reduced collateral circulation as well as the necessity of maintaining the arm in extension for puncture or prolonged cannulation. Despite all of these theoretical possibilities, the safe cannulation of the brachial artery has been demonstrated by some investigators.[58] Bazaral et al. found only one minor thrombotic occurrence in more than 3000 brachial artery

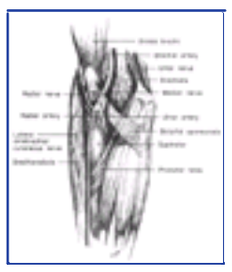

Figure 20-14 The right brachial artery and its branches. *(From Christensen JB, Telford IR: Synopsis of Gross Anatomy. New York, Harper & Row, 1966. Reproduced by permission.)*

catheterizations over 3 years in cardiac surgery patients. [59] A longer catheter (10-cm) is required for the brachial artery so that sufficient length is available to traverse the elbow joint.

Axillary

Axillary artery cannulation as described by Adler and coworkers [60] is also a safe means of monitoring arterial blood pressure for a long time. The left axillary artery is preferred in order to decrease the possibility of cerebral embolization of flush solution or thrombus. The path from the left subclavian to the left carotid artery is less direct than on the right side, whereas the vertebral arteries are equally vulnerable.

To cannulate the axillary artery, the arm is placed in 90° abduction. The axillary pulse is then palpated high in the axilla between the insertion of the pectoralis major and the deltoid muscles. The artery may then be cannulated percutaneously, and a Seldinger guidewire technique with a longer catheter (18-ga, 10-cm) is strongly recommended. This site is seldom used and is unfamiliar to many clinicians. Due to positioning and extra time for setup, this site should be avoided in the ED. There are few reported complications from catheter placement in the axillary artery. [61]

Dorsalis Pedis

The dorsalis pedis artery continues from the anterior tibial artery and runs from approximately midway between the malleoli to the posterior end of the first metatarsal space, where it forms the dorsal metatarsal and deep plantar arteries. The lateral plantar artery, a branch of the posterior tibial,

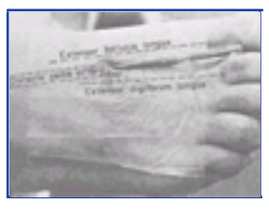

Figure 20-15 A 20-ga catheter in the dorsalis pedis artery, illustrating the relationship to surrounding tendons. The catheter is secured with Steri-Drape. Splinting is not needed. *(From Johnstone RE, Greenhow DE: Catheterization of the dorsalis pedis artery. Anesthesiology 39:655, 1973. Reproduced by permission.)*

passes obliquely across the foot to the base of the fifth metatarsal. The plantar arch is completed where the lateral plantar artery joins the deep plantar artery between the first and second metatarsals. On the dorsum of the foot, the dorsalis pedis artery lies in the subcutaneous tissue parallel to the extensor hallucis longus tendon, and between it and the extensor digitorum longus (Fig. 20-15).[62]

The artery should be cannulated in the mid-foot region. Although this vessel is amenable to cutdown, the vascular anatomy of the foot is quite variable. This is of no consequence if a pulse can be palpated, but Huber, in his dissection of 200 feet, noted the dorsalis pedis artery was absent in 12% of patients. [63] In 16% of patients, the dorsalis pedis artery provides the main blood supply to the toes. [64] Although the dorsal pedis and posterior tibial arteries form similar collateral foot circulation as in the hand, the nature of advancing vascular disease makes this a more difficult cannulation, with increased complication compared to the wrist. Nevertheless, this site has its major utility in pediatric monitoring cases. Attempts to predetermine collateral flow with a modified Allen test using the posterior tibial and dorsalis pedis arteries is not as easily performed in the foot as in the hand, nor is there good data to prove its validity. Monitoring problems also exist with this artery. The pressure wave obtained with an electronic transducer attached to the dorsalis pedis artery will be 5 to 20 mm Hg higher than that of the radial artery and, in addition, will be delayed by about one tenth of a second. [62]

Femoral

The femoral artery is the second most commonly used vessel for prolonged arterial cannulation. Based on its ease of cannulation and low record of complication, it has been called the vessel of choice for arterial access. [61] [65] [66] Along with the axillary artery, the femoral artery more closely resembles aortic pressure waveforms than those from any other peripheral site.[5] The femoral artery is the direct continuation of the iliac artery and enters the thigh after passing below the inguinal ligament. Arterial puncture must always occur distal to the ligament to prevent uncontrolled hemorrhage into the pelvis or peritoneum. [67] The artery may be easily palpable midway between the public symphysis and the anterior superior iliac spine. One can also place the thumb and fifth finger on the aforementioned distal sites and locate the artery underneath the middle knuckle. When puncturing this vessel, care must be taken to avoid the femoral nerve and vein, which create the lateral and medial borders, respectively (Fig. 20-16).

A longer, larger diameter catheter is required for accurate monitoring of the femoral artery due to the relatively greater depth at which it lies and greater vessel size. Only the Seldinger technique is recommended for this site, enabling placement of a 15- to 20-cm plastic catheter for prolonged monitoring. Use of catheter-through-the-needle or over-the-needle catheter devices should be avoided because cannulating the vessel is difficult due to its distance beneath the skin. Leakage around the catheter can occur with catheter-through-the-needle or over-the-needle catheter devices due to high arterial pressures and loose fit of the cannula in the hole in the vessel wall. Regardless of the device used, the needle should enter the skin at an angle of about 45° instead of the usual 15 to 20°.

The extremely large ratio of arterial diameter to catheter diameter is thought to beneficially reduce the incidence of thrombosis, particularly total occlusion. However, occlusions have been reported with femoral cannulation for monitoring purposes. [68] A commonly postulated disadvantage of this site is the possibility of increased bacterial contamination because of its proximity to the warm, moist groin and perineum; however, no studies confirm this hypothesis. [69] The femoral area is inconvenient for any patient who is awake and mobile, or if the patient is able to sit in a chair. If the patient is that mobile, then risk/benefit from invasive monitoring should be reconsidered. Despite theoretic difficulties, some large hospitals use femoral arterial lines almost exclusively, and the intensive care nursing staff is often

more comfortable caring for these lines than those at other sites.

Umbilical and Temporal

In the neonate, arterial access can be accomplished through the umbilical artery for a short time. After this artery closes, the temporal artery provides a safe alternative. Prian described the use of the temporal artery, noting its accessibility and the lack of clinical sequelae if it undergoes thrombosis. [70] The cutdown method should be used with a 22-ga catheter after the artery's course has been traced with an ultrasonic flow detector. Because of the increasing accuracy of ear oximeters and the use of capillary blood gases for pH determination, prolonged arterial cannulation will become less frequent during infant care. Further discussion of infant arterial cannulation is provided in Chapter 19 .

COMPLICATIONS

Long-term arterial cannulation is safe if care is taken to avoid complications. Almost all difficulties one may encounter can be avoided or their incidence markedly decreased by adhering to a few simple principles. Reported clinical sequelae of arterial puncture and cannulation range from simple hematomas to life-threatening infections and exsanguination. Other potential complications include ischemia, arteriovenous fistula, and pseudoaneurysm formation. The incidence of complications varies with the site selection, method of

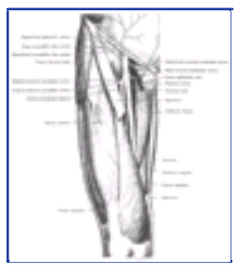

Figure 20-16 The right femoral vessels and some of their branches. The femoral nerve (not shown) lies lateral to the artery and may be deep to the artery. *(From Warwick R, Williams PL [eds]: Gray's Anatomy, 35th ed. Edinburgh, Churchill Livingstone, 1973, p 676. Reproduced by permission.)*

cannulation, and clinician's procedural skill and experience. Early detection of complications is greatly aided by enhanced vigilance and concern of the patient's clinicians and nurses. It is difficult to compare complication rates at various sites, because most published studies have primarily used the radial artery.

No studies have compared the approach and complication rates of arterial catheters in the ED compared to ICU or to OR uses. Over 24 months, 2119 ICU patients had an arterial catheter placed at admission: 52% at the radial site and 45% at the femoral site. The most common complication was vascular insufficiency (4%), followed by bleeding (2.1%) and infection (0.6%). There was no difference reported for infection rates for femoral vs radial sites. [71] There are reports of complications from arterial puncture for procedures unrelated to long-term cannulation such as arteriography or simple arterial puncture for blood sampling as routinely performed in the ED. In a study of 2400 consecutive cardiac catheterizations over a 12-month period, complications occurred in 1.6% of patients including 17 needing vascular repair and 28 needing transfusion. [72]

A commonly encountered problem is hematoma formation at the puncture site. Zorab[22] reported this complication in 50% of catheterizations. The bruising was of minimal clinical significance in Zorab's study, but leakage, when it occurs around the catheter or from the puncture site after the catheter's removal, can be dangerous. Compression neuropathy secondary to bleeding has been reported after brachial artery puncture in anticoagulated patients; in some cases, surgical decompression has been necessary.[9] The large amount of soft tissue surrounding the femoral artery makes bleeding in this area difficult to control. Large hematomas are

not uncommon after femoral artery catheterization; indeed, Soderstrom and associates [65] reported two cases of bleeding that required transfusion after femoral puncture. More commonly, hematomas are painful, slow to resolve, and prone to infection. Multiple-site punctures and inadequate pressure applied for sufficient time account for most hematomas. Furthermore, hematomas may make further procedures in the groin difficult to complete.

Thrombotic occlusion after radial arterial cannulation occurs in nearly 50% of infants and small children; however, ischemia from occlusion is rare because of collateral blood supply from the ulnar artery. [73] Insertion sites closest to the bend of the wrist increase the chances of maintaining patency. Nonpatency is 4 times more likely with insertion in sites =3 cm above the bend in the wrist. [74] Slogoff et al.[54] described 1700 cardiovascular surgical patients who underwent radial artery cannulation without any long-term, ischemic complications, despite evidence of radial artery occlusion after de-cannulation in more than 25% of patients. Serious complications after radial artery cannulation are extremely rare in the absence of contributing factors, such as preexisting vasospastic arterial disease, previous arterial injury, protracted shock, high-dose vasopressor administration, prolonged cannulation, or infection. [53] [75]

Prevention of bleeding complications may be accomplished with frequent careful inspection of the puncture site and with the use of prolonged compression after removal of the catheter or needle. Firm pressure should be maintained for =10 minutes after removal of a peripheral artery catheter and longer after femoral cannulation or if the patient is anticoagulated. Five minutes of pressure is sufficient after puncture for a blood gas sample in an individual with normal coagulation. Exsanguination, a related complication, may occur if the arterial line apparatus becomes disconnected. This is more common in the obtunded or combative patient, and restraints are often required for patients with indwelling arterial cannulas. Exsanguination should not occur if tight connections are maintained throughout the system and if frequent, careful inspections of both the circuit and the patient are made.

Meticulous attention to aseptic technique is necessary during insertion and catheter maintenance to minimize the risk of catheter-related infection. [76] [77] Serious infections rarely complicate arterial cannulation. Most simple interventions can reduce the risk for serious catheter-related infection. The strongest supportive evidence is from usage of full barrier precautions during catheter insertion, specialized nursing care, and newer generation catheters with antiseptic hubs or antimicrobial agent impregnated catheters.[76] The incidence of catheter-related infections increases with prolonged cannulation. [69] Catheters placed with sterile technique have an extremely low rate of infection up to 96 hours. Catheters changed over a guidewire every 96 hours have an infection rate of about 10% at the radial and femoral sites.[66]

Most infections begin locally at the puncture site and remain localized, although systemic sepsis has been reported. [75] Radial and femoral sites have a similar incidence of complications, but axillary cannulations seem to have a much higher incidence of infection (although no large studies of cannulation at this site exist). [70] [78] Arterial cannulas are more prone to infectious complications than other vascular catheters. Many mechanisms have been proposed for this occurrence. [77] [79] The arterial pressure monitoring system usually consists of a long column of fairly stagnant fluid and is subject to frequent manipulation. Stamm and colleagues [78] found that patients were at greater risk for systemic infection if they had an arterial line and required frequent blood gas determinations than if they had the cannula alone. The sampling stopcock is a site of frequent bacterial contamination.

The risk of infection also increases as the duration of cannulation is prolonged. Older studies recommend that catheters be changed after 4 days if continued monitoring is necessary.[78] [79] In addition, Makai and Hassemer[79] recommend changing the entire fluid-filled system, including transducer chamber-domes and continuous flow devices, every 48 hours. However, other risks for noninfectious concerns increase with more frequent catheter and site changes when based solely on length of catheterization of a site. Therefore, daily evaluation of the site is advised and catheter change should not be mandatory until 7 to 8 days, if the site remains clean.

Shinozaki and coworkers[80] demonstrated a marked reduction in equipment contamination when the continuous flush device was located just distal to the transducer, as opposed to closer to the three-way stopcock used for sampling. This setup reduces the length of the static column of fluid between the sampling stopcock and the transducer. As mentioned previously, a drop of iodophor or antibiotic ointment applied to the puncture site decreases the incidence of local wound infection. [39] This technique has drawn a great deal of criticism, however. The current standard is a clean, dry dressing, not an occlusive type. An antibiotic or silver impregnated catheter is always recommended for long-term placements.

Thrombosis of the vessel in which the cannula is placed is another frequently encountered problem. The incidence with which this occurs varies with the method used

to determine the presence of the clot. Bedford and Wollman [14] found a >40% occlusion rate when radial artery catheters were left in place for >20 hours. All of these occluded vessels eventually re-cannulized. Angiographic studies show deposition of fibrin on 100% of the catheters left in place for >1 day, although clinical evidence of ischemia secondary to occlusion with thrombus present occurs in <1% in most studies. Most reports of nonangiographic catheterizations that mention thrombosis are studies of the radial artery. Therefore, it is difficult to compare the incidence of thrombosis at other sites, although during the 176 femoral catheterizations of Soderstrom and coworkers and Ersoz and associates,[65] [81] *dorsalis pedis* pulses were decreased in only 2 patients, and no clinical signs of ischemia were noted. Larger catheter sizes, trauma during cannulation, and the presence of atherosclerosis have all been postulated to increase the incidence of thrombosis; however, conflicting studies abound. Downs and colleagues associated tapered catheters with an increased incidence of thrombosis. [16]

Arterial spasm after puncture (usually following multiple attempts) can predispose to thrombus formation and can even lead to ischemic changes without fibrin deposition. Successful reversal of spasm after intra-arterial lidocaine, reserpine, and phentolamine has been reported, but no reliable studies of efficacy in this situation have been published. [82] Thrombosis can be minimized by decreasing the duration of catheterization, by proper flushing, and by using larger arteries. Surgical embolectomy or thrombectomy is rarely required because the smaller vessels that are most likely to occlude usually have good collateral circulation. A normal (negative) result on an Allen test or a similar test suggests but does not ensure adequate collateral flow. [49] [55] The larger femoral artery,

which has poor collateralization, rarely occludes with catheterization when used for monitoring purposes.

Another complication of thrombosis is occlusion of the catheter. Time until occlusion of radial and femoral artery catheters has been compared. Radial cannulas became occluded at an average of 3.8 days, whereas femoral cannulas occluded after 7.3 days. [66] The importance of this comparison is minimal if the clinician follows infection prophylaxis guidelines and changes arterial catheters after 4 days.

A few less common complications are easily prevented. One that occurs only with the percutaneous catheter-through-the-needle method is catheter embolization. Once the catheter has been placed through the needle, it should never be pulled back, because the end of the catheter may be sheared off by the sharp needle bevel. If this occurs, surgical removal of the catheter tip is necessary.

Skin necrosis is a complication of radial artery cannulation involving an area of the volar forearm proximal to the cannula. [83] [84] Wyatt and colleagues [83] believe this is secondary to the poor blood supply of this area and state that taking the precautionary steps described previously prevents or decreases the incidence of necrosis. One feared complication of indwelling radial and brachial arterial catheters is the occurrence of a cerebrovascular accident secondary to embolization from flushing of the catheters.[16] [85] As little as 3 to 12 mL of flush solution has shown reflux to the junction of subclavian and vertebral arteries. [65] A fatality due to air embolism from a radial artery catheter has been reported and was re-created in a primate model. [86] Although these animals are much smaller (7 kg) than an adult human, as little as 2.5 mL of air introduced at a relatively low flush rate was found to embolize in a retrograde fashion to the brain. Cerebral embolization can be prevented with the use of continuous flush systems (3 mL/hour) and by ensuring the integrity of the tubing and transducer systems to prevent air entry. In addition, small volumes (<2 mL) of intermittent flush solution should be used.

Complication rates also vary according to the method of arterial cannulation. Mortensen [87] studied the three main techniques (discussed earlier in Techniques), but unfortunately, most of his arterial cannulations were for angiographic purposes. The complications associated with prolonged cannulation time are therefore underrepresented. For Mortensen's series, cutdown arteriotomy exhibited the lowest incidence of complications (7.7%), whereas the Seldinger technique had a complication incidence of 17.7%. Complications of percutaneous cannulation were 11.3%. Apparently, false passage of the guidewire, the catheter, or both were associated with increased intimal damage and complications. *It is imperative that the wire or catheter be advanced only if no resistance is met!*

In actuality, arterial puncture and cannulation are safe procedures when care is taken and basic principles are kept in mind. The operator should be skilled and should seek an atraumatic insertion. Once the monitoring system is set up, it should be manipulated as little as possible. Any handling should be performed with a flawless aseptic technique. The tubing and other fluid-filled devices should be changed every 48 hours, and catheters should be inserted into a vessel that provides a vessel-to-catheter ratio as great as possible without compromising other needs. If these principles are followed and the patient and system are carefully inspected at frequent intervals, complications of arterial puncture and cannulation can be minimized.

INTERPRETATION

An indwelling arterial cannula can provide valuable information about the hemodynamic status of a patient (through continuous pressure monitoring) and about the patient's respiratory and metabolic status (through intermittent sampling for blood gas analysis and other blood tests). The partial pressure of carbon dioxide and pH of the blood can be used to define four major groups of metabolic derangement: respiratory acidosis or alkalosis, and metabolic acidosis or alkalosis. Rarely will a disorder be strictly classified into one of these groups; however, a simple chart such as that shown in the appendix helps determine the relative effects of metabolic and respiratory influence on the blood pH. (See also the discussion in the Appendix .)

A rough estimate of the contribution of respiratory factors may be made by assuming that for every 10 torr (mm Hg) that the pCO_2 varies from 40, the pH will inversely vary 0.08 pH units from 7.4. Adequacy of blood oxygenation can be determined from the measured pO_2 of the arterial blood and from the known concentration of oxygen that the patient is inspiring. To avoid iatrogenic complications of intensive care, one must be absolutely certain that the data are from an arterial sample that has been properly analyzed before basing one's treatment decisions on the numbers obtained. Not uncommonly, one may accidentally puncture a vein when attempting to obtain an arterial blood sample. Furthermore, false readings may result if the sample is not free of air bubbles, not promptly chilled, and not analyzed within 20 to 30 minutes. Although still controversial, blood gas values that are *uncorrected* for body temperature appear more appropriate for guiding therapy in hypothermic patients.[88] [89]

An indwelling arterial catheter also provides continuous blood pressure monitoring. The trend of a patient's pressure helps one assess the effect of various therapeutic interventions. The absolute systolic and diastolic pressures measured will vary at different catheter sites, with higher peak systolic pressures measured at the periphery; the pressures will also be higher when measured in the distal lower limb. [17] [65] A wide variance between direct arterial pressure and pressure measured with a standard pneumatic cuff will always exist in some patients. Data averaged over a population group, however, compare fairly well. [17] For this reason, the cuff pressure and that displayed on the monitor should be compared regularly. A change in their relationship may be the first indication of difficulties with the direct measuring system. Auscultatory methods usually give a slightly lower value than direct measuring systems.

Waveform analysis may also provide an early indication of thrombosis in the arterial catheter. Many variables affect the waveform, including cardiac valvular disease, arteriosclerosis, and other peculiarities of an individual's cardiovascular system that may contribute to pulse wave reflections. [90] Waveforms may vary tremendously among patients, but after an adequate monitoring system has been established, a change in an individual's pressure wave is usually indicative of thrombosis or other malfunction in the monitoring system. A change in waveform may also indicate a change in the patient's cardiovascular status, such as a papillary muscle rupture. Once again, before making a therapeutic decision based on an electronically generated number, the patient should be rechecked with a pneumatic cuff; this device is less fallible than the electromechanical system.

Radial systolic arterial pressures poorly estimate the actual ascending aortic pressure, with >50% of cases reporting a difference in values by 10 to 35 mm Hg. Mean arterial pressures or even diastolic pressures were found to be highly accurate with >90% of the values being within 3 mm Hg of aortic values. [91] Longer catheters have also been successfully used from radial sites to more accurately reflect central aortic pressure for cardiac surgery patients. [92]

CONCLUSION

As intensive care knowledge and technology grow and develop, cannulation of the arterial system may decrease in frequency. Oximeters can determine the quality of blood oxygenation percutaneously and are becoming more accurate and sophisticated (see Chapter 2). Electronic sphygmomanometers are being refined for continuous indirect blood pressure monitoring. As these devices improve and noninvasive sampling methods for clinically relevant electrolytes and physiologic markers are refined, the indwelling arterial cannula may in time become considered overly invasive. At the time of publication, the current need for frequent blood sampling for chemical and hematologic analysis remains a strong indication for its use in the most critically ill patients. Overzealous blood gas analysis may lead to iatrogenic anemia in the ICU. Multiple reports document the advantages to limiting frequent blood sampling (and its associate waste). [93]

Arterial puncture and cannulation are invaluable aids to the emergency and critical care clinician. Long-term catheterization is a safe procedure when the catheter is placed, maintained, and removed with care. The radial artery is the most favored location for puncture, but as more experience is gained and reported with femoral artery catheterization, the latter may become a more frequently used site. Selection of either site is associated with a low complication rate and should be determined by the skill of the clinician and the nursing team and the relative convenience and comfort of the patient.

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Chapter 21 - Peripheral Intravenous Access

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Richard Zane

Intravenous (IV) access is a mainstay of modern medicine. IV cannulation is a procedure performed by nearly all involved in the health care profession—clinicians, nurses, clinician assistants, phlebotomists, and emergency medical technicians. In the United States, more than 25 million patients have peripheral IV catheters placed per year, allowing access for medication administration, fluids and nutrition, and blood sampling for laboratory analysis. IV access can usually be accomplished in 2 to 5 minutes, although in small children it may take considerably longer. [1] [2] [3] [4] Despite their growing number, dedicated IV teams are very costly and not always cost effective, making it paramount for clinicians to continue to master IV cannulation. [3] [5]

Bloodletting, or bleeding, has been a long-established medical practice dating back to the time of Hippocrates. The most common ancient technique for bleeding was to tie a bandage around the arm so the veins of the forearm would distend. The vein was then opened with a sharp knife and the blood collected in a bowl or basin. By the Middle Ages, this was usually done by barber-surgeons. In 1656, Sir Christopher Wren injected opium intravenously into dogs using a quill and bladder, thereby becoming the father of modern IV therapy.[6] Blood transfusions also date back to the mid-1600s. French physician Jean Denis successfully transfused lamb's blood to a 15-year-old boy in 1667. [7] [8]

Originally, 16- to 18-gauge (ga) steel needles were used for infusions. However, in the 1950s, with the introduction of the Rochester needle (a resinous catheter on the outside of a steel introducer needle), IV plastic catheters soon replaced indwelling metal needles and increased comfort and mobility. [6] [9] Today, plastic catheters are the mainstay of IV access.

INDICATIONS AND CONTRAINDICATIONS

Obtaining timely and adequate access is a major priority during a cardiac arrest and major trauma. In normal perfusion, differences in delivery times for injections centrally vs. peripherally are minimal essentially a matter of seconds. [10] Canine studies show that 90% of peripheral IV fluid reaches central circulation beneath inflated pneumatic anti-shock garments. [11] During cardiopulmonary resuscitation, medications have been shown to reach the central circulation faster with central access than with peripheral venous access. [12] However, peripheral IV cannulation is still the procedure of choice even during cardiopulmonary resuscitation (CPR) because of the usual speed, ease, and safety with which it can be accomplished. [13]

Saline locks, commonly known as heparin locks because of prior use with heparin flushes, are preferable when IV medications are needed and there are limited foreseeable fluid requirements. Saline locks cost much less than a full IV fluid and tubing assembly; therefore, saline locks have largely replaced the time-honored method of maintaining access by a slow, constant infusion. [13a] Such locks are especially helpful when prompt vascular access may be suddenly needed. Their disadvantage is that irrigating the catheter requires a separate syringe and flush. [13]

In terms of contraindications to IV placement, with the risk of extravasation of irritating or tissue-injuring solutions (colchicine, phenytoin, vasoconstrictors, and others) or suboptimal volume flow, peripheral IVs should not be placed in extremities with massive edema, burns, sclerosis, phlebitis, or thrombosis. Furthermore, extremities on the side of radical mastectomies or dialysis grafts should also be avoided, although they should be used when an urgent condition exists and other peripheral access is not possible. Veins that drain from an area of neck trauma or into an affected traumatic extremity or the side of a chest or abdominal trauma are also suboptimal. The patency and flow of the vessels and fluid or medications may not be delivered to the circulatory system. Cannulation at sites of cellulitis should be avoided as it may cause bacteremia. Also avoid extremities with shunts or fistulas to minimize shunt infections or thrombosis. Similarly, IV access in feet and ankles are suboptimal for long-term use, but may suffice in the emergency department.

Blood samples for laboratory analysis are usually drawn before IV cannulation to avoid contamination with IV fluid or medication. However, several studies have shown that accurate basic electrolytes and hematologic values can be drawn from peripheral IV lines when infusions are shut off at least 2 minutes, at least 5 mL of blood are wasted, and all tubes are filled to the top to avoid inaccurate bicarbonate readings. [14] [15] [16] By adopting this technique, one can reduce the number of peripheral needle sticks, minimizing trauma and sclerosis of the vein and improving patient satisfaction.

PERIPHERAL INTRAVENOUS CENTRAL CATHETERS

In this modern age of multiple types of IV access devices, familiarity with peripheral devices is necessary to ensure proper selection. A common option is the *PICC line*, or peripheral IV central catheter. It is a relatively recent addition to the IV access armamentarium and shares attributes of both central and peripheral venous access. A PICC line is composed of a thin tube of biocompatible material and an attachment hub that is inserted percutaneously into peripheral veins and advanced into a large central vein with radiographic confirmation of placement. PICC lines are suitable for long-term vascular access for blood sampling and infusion of hyperosmolar solutions such as those used for total parenteral nutrition. These lines should be inserted as soon as intermediate-term access is anticipated. [17]

ANATOMY

Success of cannulation depends on familiarity with the vascular anatomy of the extremities. In the upper extremity, the veins of the hands are drained by the metacarpal and dorsal veins, which connect and form the dorsal venous arch and are excellent sites for IV therapy. These veins can easily accommodate 22- and 20-ga catheters. The wrist and forearm's venous supply is the basilic vein, which courses along the ulnar portion of

the posterior forearm; it is often ignored because of its location, but can be easily accessed if the patient's forearm is flexed and the cannulator stands at the head of the patient.[18] On the radial side of the forearm, the cephalic is best known as the "intern vein." Easily accessed, this vein can accommodate 22- to 16-ga catheters. The median veins of the forearm course through the mid-forearm. Accessory cephalic veins at the top radial aspect of the forearm are easily stabilized and accessible.

The antecubital veins consist of the medial cubital, basilic, and cephalic veins and are often selected for midline catheters or blood draws. IV placement here is easy, but mobility of the arm is often subsequently restricted. The larger veins above the antecubital space, the cephalic and basilic veins, are often more difficult to see, but can be accessed if necessary without difficulty (Fig. 21-1).

The relevant lower extremity venous anatomy starts with the dorsal digital veins, which become the dorsal metatarsal veins and then form the dorsal venous arch. The arch ultimately splits into (1) the greater saphenous vein, which travels up the medial aspect of the ankle, and (2) the lesser saphenous vein, which courses laterally up the opposite side. These are the vascular structures most accessible for IV therapy.

The external jugular vein is formed below the ear and behind the angle of the mandible. It then passes downward and obliquely across the sternocleidomastoid, then under the

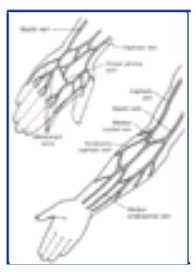

Figure 21-1 Anatomy of veins in hands and arms for intravenous cannulation. *(Adapted from Millam DA: How to insert an IV. Am J Nurs 79:1268, 1979.)*

middle of the clavicle to join the subclavian vein. This vein is often cannulated in adults and children who have venous access problems, but it usually only provides access for a short time (Fig. 21-2). It is important to note the presence of valves in the external jugular, notably about 4 cm above the clavicle, as they can significantly impede IV function.[13] Flow is commonly dependent on the position of the neck.

PREPARATION

Safety.

In the era of HIV and hepatitis, safety in placing IVs cannot be overemphasized. Universal precautions must be applied to all patients, especially in emergency care settings where risk of blood exposure is increased and infection status of patients is largely unknown. [19] One study showed that 11% of all hospital IV catheter injuries to health care workers occurred in the emergency department (ED). [20] Newer catheter devices have emerged to prevent inadvertent needle injuries. The Protectiv IV Catheter Safety System (Johnson & Johnson, Inc., Arlington, TX), Insyte Antoguard Shielded IV catheter (Becton-Dickinson, Franklin Lakes, NJ), Saf-T-Intima IV Catheter Safety System (Becton-Dickinson, Franklin Lakes, NJ), Vacutainer Brand Safety-Lok (Becton-Dickinson, Franklin Lakes, NJ), Shamrock Safety Blood Collection Set (Winfield Industries, San Diego, CA) has a protective sleeve that encases the sharp stylet as it is retracted from the catheter. The Insyte Autoguard Shielded IV Catheter's needle is instantly encased inside a tamper-resistant safety barrel by pressing the activation button. The Saf-T-Intima IV catheter, puncture-guard winged set, Vacutainer Brand Safety-Lok, and

Figure 21-2 The external jugular vein may be cannulated in the same manner as any other peripheral vein, which often negates the need for central venous catheterization. This site is especially useful to obtain blood and to infuse fluids and medication in obese adults, infants, and IV drug users. The major disadvantages are that flow is very dependent on the position of the neck (a significant problem in children and obtunded or restless adults), and valves may abut against the tip of the catheter. Air embolism from a disconnected catheter is another potential hazard with this access route.

TABLE 21-1 -- Materials Required for Peripheral IV Line Insertion

Povidone-iodine
Alcohol pads
Tourniquet
Gauze sponges
Tape
Tegaderm
Intravenous catheter
1-in tape
¼-in tape
Gloves

Shamrock safety winged needle are all types of winged safety devices that have shields which advance over the needle to prevent needle exposures. [6]

Choosing catheter gauge.

The catheter gauge will depend on the clinical scenario. The smallest, shortest catheter is a 22-ga, which is sufficient for routine maintenance fluids and routine antibiotics. A 20-ga or 18-ga is necessary for blood product administration and a 16-ga needle is suggested for resuscitating patients. [18]

Appropriate site.

Site selection will depend largely on the expected duration of IV therapy, patient's activity level, and condition of the extremities. When choosing a place to initiate IV therapy, the best starting place is the hand. Then advance cephalad as necessary. Hand veins are appropriate for 22-ga IV catheters. Cephalic, accessory, or basilic veins are ideal for larger bore IVs. Avoid veins that are not resilient and feel hard and cordlike, as they are often thrombosed. [6] Deep, percutaneous antecubital venipuncture or external jugular vein cannulation are also options in the patient with difficult veins or those that may need IV access in a hurry. [21]

In patients who have undergone radical mastectomy, avoid the arm on the same side as the surgery because circulation may be impaired, affecting flow, causing edema and other complications like thrombosis. [6] [18] Furthermore, while lower extremity veins can be useful locations for IV access, especially in children, they are often easily traumatized and may lead to deep vein thrombosis. [6]

Anesthesia for IV placement.

Prospective studies continue to demonstrate that local anesthesia, such as buffered or plain lidocaine or benzyl alcohol, significantly decrease perceived patient pain before IV cannulation. [22] [23] [24] Although somewhat time-consuming and impractical, and sometimes as painful as cannulation itself, anesthesizing at the site of cannulation should be at least considered as part of routine IV care. Similarly, in the pediatric population, 2.5 g of EMLA (eutectic mixture of local anesthetics) can also be applied to the vein to ensure local anesthesia. [6] The main disadvantage of using EMLA is that one must wait up to an hour for anethestic onset before cannulation. [25] In one study regarding the use of 4% liposomal lidocaine (ELA-Max) in children, the authors reported that a 30-minute application was as safe and effective as EMLA for reducing pain during venipuncture (see Chapter 30). [26]

IV assembly.

Collecting the proper supplies before inspecting the patient's veins and performing cannulation is imperative. Listed in Table 21-1 are the necessary materials. They are shown in Figure 21-3 .

IV fluids and lines should be prepared as well, if needed. The cap should be removed from the IV line and the tab

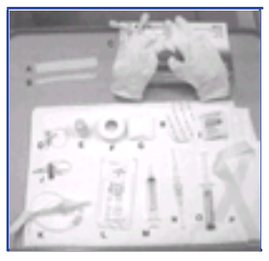

Figure 21-3 Materials for intravenous insertion. *A* and *B*, Tape for securement. *C*, Gloves as part of universal health precautions. *D*, Saline lock. *E*, Saline. *F*, Tape roll. *G*, Gauze. *H*, Tegaderm. *I*, Alcohol swabs. *J*, Butterfly needle, if needed. *K*, Intravenous tubing. *L*, Iodine swabs. *M*, Syringe for phlebotomy. *N*, Angiocatheter. *O*, Prepped saline flush. *P*, Tourniquet.

removed from the IV bag. The IV tubing should be clamped shut and the spiked end inserted into the IV bag (Fig. 21-4). The drip chamber should be pinched and filled halfway (Fig. 21-5). The clamp should then be opened slightly to flush the IV tubing (Fig. 21-6). If saline locks are being used, the locks should similarly be flushed before cannulation. This can be accomplished by attaching the lock to a saline-filled syringe and flushed (Fig. 21-7).

Inspection and positioning.

After collecting supplies and making appropriate preparations, palpation is the next crucial step in successful cannulization. Position the patient comfortably on a flat surface. Place a 1-in wide tourniquet on the patient's upper arm or forearm sufficiently tight enough to impede venous flow but not to the extent arterial flow is compromised. Start by placing the tourniquet under the arm (Fig. 21-8). Fold both ends of the tourniquet above the arm and cross the ends (Fig. 21-9). Pull the overlying end taut and tuck the middle portion below the underlying end, creating a loop (Fig. 21-10 and Fig. 21-11). After tourniquet placement, palpate with the index and middle fingers of one's nondominant hand—veins are soft, elastic, resilient, and pulseless. [18]

Cannulation.

Wash hands, put gloves on, and clean the site with iodine or alcohol, or both. Studies suggest that iodine is better as an antiseptic than alcohol in terms of fewer infections.[27] Most skin preparations require drying to optimize surface antibacterial activity (Fig. 21-12). Stabilize the vein without contaminating the prepared site. One method is to position one's thumb alongside the vein and pull down and then place the index finger more cephalad and push upward (Fig. 21-13). Take the angiocatheter between the thumb and forefinger of the dominant hand with the bevel up, angled 10 to 30° between angiocath and vein, aligned parallel to the vein. Puncture the vein (Fig. 21-14). Once a flash is seen,

Figure 21-4 Insertion of spiked end of IV tubing into IV bag.

advance the catheter several millimeters more to ensure it has entered the vein and not just the wall. Avoid advancing too far and puncturing the posterior wall; loosen the stylet and advance only the catheter (Fig. 21-15). Take the fingers anchoring the vein and occlude the vein at the tip of the catheter to prevent extravasation of blood from the angiocatheter. Remove the needle and connect the saline lock, IV

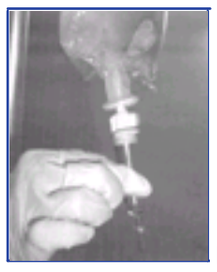

Figure 21-5 Pinching drip chamber to fill bulb halfway before infusing fluid.

Figure 21-6 Flushing IV tubing.

lining, or syringe for phlebotomy and release the tourniquet (Fig. 21-16 and Fig. 21-17). [18]

External jugular vein cannulation deserves a special note. In the patient with otherwise little peripheral access, cannulate as follows: Place patient in the Trendelenburg position to fill the external jugular. See Fig. 19-5 in the Pediatric Vascular Access chapter. Rotate the head to the opposite side.

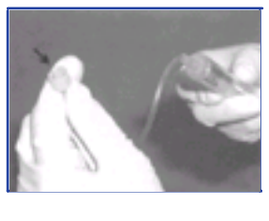

Figure 21-7 Flushing saline lock.

Figure 21-8 Application of tourniquet: Place tourniquet 3 to 4 cm proximal to insertion site.

Figure 21-9 Crossing tourniquet ends and applying tension.

Figure 21-10 Tucking middle portion of one end snugly under opposite end to make loop.

Figure 21-11 Distal portion of tucked end free for one-hand release of tourniquet.

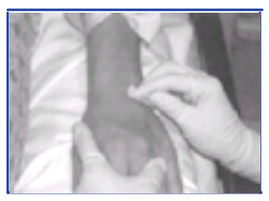

Figure 21-12 Prepping insertion site with alcohol.

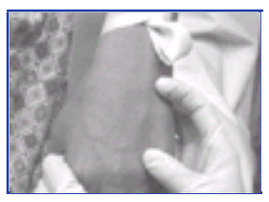

Figure 21-13 Grasping skin and pulling taut to apply traction.

Figure 21-14 Insertion of catheter.

Figure 21-15 Advancing catheter and removing needle.

Figure 21-16 Phlebotomy.

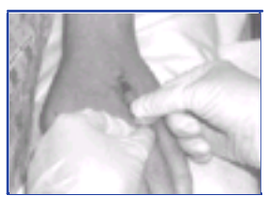

Figure 21-17 Attaching IV saline lock.

Figure 21-18 Securing IV down: placing tape under hub of catheter, sticky side up.

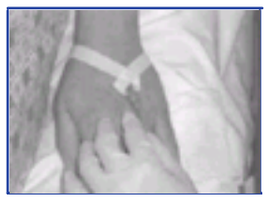

Figure 21-19 Crossing ends of tape over top of hub.

Figure 21-20 Transparent polyurethane dressing.

Prep the area as described earlier. Align the cannula in the direction of the vein with the point aimed toward the ipsilateral shoulder. Puncture midway between the angle of the jaw and midclavicular line, lightly compressing the vein with a free finger above the clavicle. Proceed as previously described for cannulation. [13]

Anchoring the device.

After the IV has been connected to the saline lock or IV tubing, anchoring the device is essential. Use a ½-in wide strip of tape, adhesive up, under the hub of the catheter and fold it over like a bow (Fig. 21-18 and Fig. 21-19). This will secure the cathether and prevent lateral movement. Clear polyurethane dressings can also be used with or instead of tape (Fig. 21-20). Saline locks can be connected to needleless hubs to prevent accidental needle injury (Fig. 21-21). Then secure the loose saline lock or IV tubing with tape to prevent accidental dislodgement (Fig. 21-22). IV tubing can be similarly connected to the angiocatheter and anchored (Fig. 21-23). Commercially available securement devices can also be used. Dressings should then be signed and dated to assure timely dressing changes. [6] Topical antibiotics

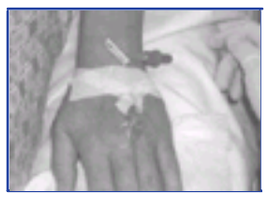

Figure 21-21 Attachment of IV saline lock to angiocatheter. This can be used for IV fluids or medications.

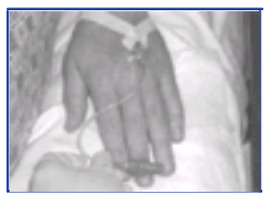

Figure 21-22 Securing the saline lock.

or iodophor ointment should be applied to the insertion site to prevent infection. [28]

Maintaining patency.

An important component of IV care is maintaining patency with frequent flushing. Until recently, heparin solutions had been used to flush catheters and maintain patency but have been shown to cause problems such as hemorrhage. Saline flushes are as effective as heparin in maintaining patency and preventing phlebitis in peripheral devices. In a meta-analysis comparing saline and heparin flushes, there was no statistical difference between the incidence of clotting, phlebitis, and duration of IV patency. With the advantage of decreased costs and avoiding complications such as bleeding and heparin-induced thrombocytopenia, heparin flushes should be replaced with saline flushes. [29] [30] [31]

Dressing.

It is not cost-effective to continually redress peripheral venous catheters at periodic intervals. Sterile gauze or transparent, semipermeable, polyurethane dressings can be used and left on until removal of the catheter without increasing infection as long as the site is regularly evaluated. [32] There is emerging evidence that the type of securement

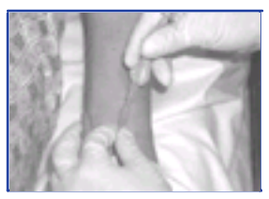

Figure 21-23 Attaching the IV tubing for infusion.

techniques (e.g., the StatLock IV, a sterile, adhesive-backed dressing and proprietary distal male luer-tip extension set) can decrease mobility and the risk of dislodgement. [33]

Adjuncts.

Often, patients have nonvisible and nonpalpable veins. Several adjuncts can increase the likelihood of successful cannulation.

Nitroglycerin ointment applied to the hands of patients with small caliber veins has been shown to increase vein diameter size two to six times the original diameter and increase the rate of successful first-attempt cannulation without complications. Once the tourniquet is applied to the wrist, 0.5 to 1 cm of 2% nitroglycerin is applied to a 2.5 cm square area, left on for 2 minutes, and then rubbed off. [34] Nitroglycerin is useful and safe in the pediatric population, as well. [35]

In the late 1980s, several small studies demonstrated the potential uses of a venous distention device—a cardboard mailing tube placed over the forearm with a sealed bulb at one end that would cause a vacuum within the tube. Ninety percent of the patients predetermined to be difficult to access were cannulated using this device. There were few reported complications, such as petechiae and discomfort. [36] [37] Another useful, common method of increasing venous distention is simply to ask the patient to open and close his or her fist. This causes increased blood flow into the arm or hand and distends the veins. Light tapping can also increase venous distention, although heavy tapping may cause the vein to spasm. Lowering the arm below the level of the heart can also increase venous distention. If these methods are inadequate, heat packs can be applied for 10 to 20 minutes to increase venous engorgement. This is particularly useful in the pediatric population. [6]

Percutaneous brachial vein cannulation.

Brachial vein cannulation is an option when attempts at peripheral IV access have failed or are contraindicated and may obviate the need for central venous access or surgical cutdown. Complications include brachial artery puncture, hematoma, and transitory paresthesias.

Figure 21-24 *A*, Anatomy of the vessels of the arm. *B*, Corresponding ultrasound image obtained with an Aloka 7.5-MHz ultrasound probe on the right arm. BA, brachial artery; BAV, basilic vein; DBV, deep brachial vein. *(From Keyes LE, et al: Ultrasound-guided brachial and basilic vein cannulation in emergency department patients with difficult intravenous access. Ann*

Emerg Med 34:711, 1999.)

To cannulate the brachial vein, the brachial artery must be palpated in the antecubital fossa. Prepare the site in the usual manner and apply a tourniquet above the antecubital space. At a point immediately medial or lateral to the pulse, an angiocatheter with an attached syringe is inserted and advanced at a 45° angle, maintaining suction on the syringe. After entering the vein, continue 2 to 3 mm more to ensure cannulation. Advance the catheter and remove the needle as usual. [21] [38]

Ultrasound-guided deep brachial vein cannulation has been reported in the literature. [39] In this study, a 7.5 MHz probe was used to identify the more laterally and superficially situated basilic vein, as well as the noncompressible brachial artery and brachial vein. After identification of the appropriate vessels using ultrasound, a second operator inserted a 1.8- to 2-in, 18- to 20-ga IV catheter successfully in a majority of patients (Fig. 21-24).

COMPLICATIONS

Though IV placement is a common procedure, it is not without complications. Phlebitis, infiltration, infection, nerve damage, air embolism, bruising, and thrombosis are the most common complications and rarely cause significant morbidity or fatality.

Phlebitis.

Phlebitis is a common complication after IV cannulation and administration of medication, especially vancomycin, potassium, and any hyperosmolar solution or cytotoxic agents.[40] [41] IV devices facilitate infection by damaging epithelial and mucosal barriers to infection and provide microorganisms direct access to the bloodstream.[42] The most common infectious complication of peripheral IV access is a self-limited cellulitis. Bacteremia and sepsis occur rarely. Phlebitis is described as the presence of a palpable cord accompanied by warmth, erythema, tenderness, and induration. Phlebitis will usually manifest as discomfort for the patient and necessitates removal of the catheter and replacement on

another extremity. The occurrence of phlebitis can be minimized by reducing trauma to the venous wall. Not placing IVs in the lower extremities or across joint sites, when possible, also decreases the incidence of IV-related phlebitis, as trauma, motion, and stagnant blood in varicose veins will be minimized. [6] In one study, phlebitis occurred in 15% of patients receiving IV infusions. Of these patients, 6.5% had local staphylococcus epidermidis colonization of the catheter. Interestingly, this study also demonstrated that an increased incidence of phlebitis was not related to duration of the IV but rather to patients with higher hemoglobin levels, for unclear reasons.[43] In another study, bacteremia developed from peripherally inserted lines 0.4% of the time. [44] With such low levels of clinically significant bacteremia, some argue routine replacement of catheters is now no longer needed. [43] The only large, prospective study investigating the incidence of phlebitis associated with peripheral IVs required replacement of the IVs every 72 hours. [32] Therefore, it is still standard practice to change IV lines every 72 hours.

The role of in-line filters to prevent phlebitis is controversial. It is thought that particulates from reconstituted medications, degradation products, precipitates, glass from vials, and other foreign debris all may play a part in postinfusion phlebitis. In-line filters may therefore play a role in preventing phlebitis, but given their cost, risk of clogging, and paucity of evidence that they improve outcomes, they have not become standard of care. [45]

Occasionally patients recently discharged from the hospital or ED will return with concern about a lump or cord at an IV site, often convinced that a piece of the catheter is still in the arm. This is a manifestation of vein injury from IV cannulation, and it may not be painful or show erythema or redness. Resolution is often quite slow (weeks) but no specific intervention is helpful or required.

Extravasation.

Usually, infiltration of a vein is a relatively minor and common complication of IV therapy. This often occurs when the catheter is dislodged from the vein during infusion. However, if the infusions are hypertonic, vasopressors, or chemotherapies, there is a significant risk of skin sloughing when infiltration and extravasation occur (Table 21-2). *Pain at the infusion site or the alarm sounding on an infusion pump device requires inspection of the infusion site for extravasation.* In extreme cases, skin slough with grafting may be required (Fig. 21-25).[6] If dopamine or norepinephrine extravasates, phentolamine may be used as an antidote to prevent ischemia to the local area. The reversal of ischemia with phentolamine is a common technique, but its ability to reverse or prevent a skin slough is not certain and not well documented. However, if infiltration of these vasopressors occurs, the editors suggest that it be routinely used. To inject phentolamine, 5 mL of a standard phentolamine solution is diluted with equal parts of saline. Using a 25- to 27-ga needle or a TB syringe, the entire area of skin blanching is injected with multiple small aliquots of the solution. Hyaluronidase was suggested in the past to ameliorate some effects of extravasation of other solutions. Although it was a common suggestion, its efficacy was not well established. Now this solution is not commercially available.

Infection.

Infection can be a costly and potentially devastating complication of IV therapy. Although rare with peripheral IVs, intravascular device-related bloodstream infections are often the least recognized cause of nosocomial infection. Peripheral IV catheters are most often associated

TABLE 21-2 -- Medications/Solutions That May Cause Tissue Injury When Extravasation Occurs in a Peripheral Vein

Aminophylline
Calcium chloride 10%
Carmustine
Chlordiazepoxide
Colchicine
Crystalline amino acids 4.25%/dextrose 10%
Crystalline amino acids 4.25%/dextrose 25%
Dactinomycin
Daunorubicin
Dextrose 10%
Dextrose 50% in water
Diazepam
Dobutamine
Dopamine
Doxorubicin
Epinephrine
Ethyl alcohol
Mechlorethamine
Metaraminol
Mithramycin
Mitomycin
Nafcillin
Nitroglycerine

Norepinephrine
Parenteral nutrition solutions
Phenytoin*
Potassium solutions
Propylene glycol
Renografin-60
Sodium bicarbonate 8.4%
Sodium thiopental
Tetracycline
Vasopressin
Vinblastine
Vincristine
Vindesine
Many medications and IV solutions will cause pain and occasionally skin slough if significant amounts extravasate into soft tissues. Therefore, any complaint of pain during infusion or signs of tissue swelling should prompt an investigation for extravasation. Most extravasations have no specific therapy, so prevention is the only option. Phentolamine, injected subcutaneously to reverse vasoconstriction, is the most common technique, but its efficacy has not been well studied.

**Use a maximum concentration of 1 mg/mL of saline or phosphenytoin solution to minimize this risk.

with *Staphylococci epidermidis*, *Staphylococcus aureus*, and *candida* infections.[46] Infectious complications can be significantly reduced by handwashing, wearing gloves, site preparation with iodine, and monitoring site for signs of infection. [6]

Nerve injury.

Another rare complication of IV cannulation is nerve injury. Any peripheral nerve is potentially vulnerable to a needle-induced injury and sequelae can range from minor motor or sensory abnormality to complete paralysis. Nerve damage may come from needle damage, intraneural microvascular damage from hematomas, or toxic effects of the agent injected.[47] The first symptoms are often pain, numbness, or paresthesia. Pain may persist for years and can be debilitating. Fortunately, most simple procedures do not result in nerve injury as nerves often roll or slide away from the needle. Like all procedures, knowledge of relevant anatomy is essential. Should a patient complain of numbness or severe pain after needle puncture, injection into that site should immediately stop. [48] [49]

Air embolism.

Air embolism is another significant, although exceedingly rare, complication of peripheral IV

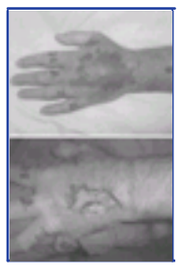

Figure 21-25 Extravasation of phenytoin. Pain at the infusion site or the alarm of an infusion pump requires inspection of the IV site for extravasation when irritating solutions are infused. In this case, an unconscious patient received a concentrated phenytoin infusion for status epilepticus, but his comatose state did not allow him to complain of pain. Over a few days, a skin slough occurred, requiring many weeks to resolve. This could have been avoided with the use of a more dilute solution (i.e., <1 mg/mL phenytoin to saline solution) or a central vein. The top panel shows the site one day after the phenytoin extravasation. The bottom panel was taken on follow up approximately a month later. *(Pictures courtesy of Dr. Mahesh Shrestha.)*

access. The symptoms are chest pain, shortness of breath, sudden vascular collapse, cyanosis, and hypotension. If air embolism is suspected, the patient must be placed in the left lateral position, ideally with the head and chest tilted downward. Such near fatal complications can be prevented by eliminating air from IV tubing before initiating therapy and not letting IVs run dry. [6] If the air bubbles are present near the top of the IV line, tap the tubing while holding it taut so they escape to the top. Similarly, curling the tubing around a pen or syringe can accomplish the same goal. If the air is near the Y connector, one can use a needle and syringe to directly remove it. If all else fails and the air is between the Y connector and the patient, the tubing will likely have to be disconnected and flushed. [50]

Other complications.

Bruising is a common complication of IV therapy. This is an inconsequential and common sequela of blood sampling or IV access, although it is often distressing to patients. Contrary to popular belief, flexing the elbow after venipuncture does not prevent bruising in the antecubital site. [51] Applying direct pressure immediately after decannulation is the most useful technique to prevent bruising.

Suppurative thrombophlebitis is another extremely rare complication of peripheral IV therapy. This is not an issue in ED patients—it most frequently occurs in patients with thermal injury, long-term cannulation with a plastic catheter, or lower extremity cannulation. [42] Local signs of inflammation or suppuration are often absent and can occur 2 to 10 days after catheter removal.[52] Treatment may include surgical excision of the entire length of involved vein and involved tributaries. As with all the infectious complications of IV therapy, it can be prevented by meticulous aseptic cannulation technique. [53]

Thrombosis and subsequent pulmonary embolism is a rare complication of peripheral IV access and is more commonly associated with centrally placed IV catheters and central lines.[6] Although rare, thrombosis and pulmonary embolism may occur in peripheral IV lines if saline locks are not flushed or fluids are allowed to run out. Should this occur, aspirate the line. If the return appears bloody, discard the syringe, gently flush the saline lock, and resume infusion. If there is no bloody aspirate, use 2 to 3 mL of saline to gently flush the line. If there is resistance, stop flushing immediately as there is a risk of an embolism developing. Recannulization at another site is then recommended. [54]

The Centers for Disease Control recommendations for IV care are:

1. Record and date the time of the catheter insertion in an obvious location near the insertion site.
2. Do not palpate the insertion site after the skin has been cleansed with antiseptic.
3. Palpate the insertion site for tenderness daily through an intact dressing.
4. Visually inspect the site if the patient reports tenderness.
5. Wash hands before and after palpating, inserting, replacing, or dressing any intravascular access site.
6. Replace dressings when they are damp, loose, or soiled.[44]

SPECIAL SITUATIONS

One of the greatest challenges in IV cannulation is establishing access in small children and infants (see Chapter 19). Some clinicians recommend a technique similar to the Seldinger technique, placing a guidewire into the angiocatheter once a flashback is obtained and then advancing the angiocatheter over the wire. [55] In establishing peripheral IV access in small children and infants, the technique is similar to that used in adults, but dorsal veins of the hands and feet are often the preferred site, while trying to reserve the antecubitals for PICC lines. In neonates, superficial scalp veins are convenient access sites as well. [17]

In children younger than 6 years who are critically ill, the intraosseous (IO) route is excellent (chapter 26) if peripheral IV access is difficult. It is possible because of the presence of noncollapsable veins that drain the medullary sinuses of the marrow. Such conduits drain into the central venous circulation via nutrient and emissary veins and allow administration of drugs, crystalloid, and blood products intraosseously with nearly immediate systemic absorption. [56] The recommended location for IO placement is the anteromedial tibia, 1 to 2 cm

below the tibial tuberosity. A 14- to 20-ga IO needle with stylus is recommended. This needle is placed at a 90° angle to the bone and advanced using a twisting motion through the skin and bone cortex, in a slightly caudal direction away from the growth plate to a depth of 1 cm. Placement is confirmed by feeling the pop of the needle and then aspirating bone marrow. IV fluids, medications, and blood can be delivered via this route. The overall complication rate for the IO route is low, although infiltration and osteomyelitis have been reported. These infusions must be delivered using a pressure pump because the viscosity of the marrow can be up to 10 mL/min. The needle must be secured well to avoid displacement. [57] [58] Recent swine experimental data and pediatric oncology data have demonstrated that lab values obtained from bone marrow aspirates may be reliable for blood gas and chemistry values, with the exception of potassium and ionized calcium and oxygenation levels. [59] [60] Laboratory porcine data have shown promise for the possible use of IO blood gas values in the setting of hypothermia. [61] While rare, there have been several case reports of compartment syndrome requiring amputation following IO infusion. To prevent such complications, IO placement of a catheter must be definitively confirmed by visualization of bone marrow, secured, and then carefully observed for early signs of compartment syndrome. [62]

IO infusions into adults are under research. Even though adults have a much less active bone marrow than young children, they still have patent sinusoidal vascularity. Of several commercially available IO kits for adults, the most promising one involves placing the IO needle device in the manubrium of the sternum. One study examined emergent bone marrow infusion into adult patients in whom IV access could not be established within a reasonable time frame. The success rate of adequate insertion was 100% and all patients' cardiovascular and general conditions improved or permitted additional IV lines to be placed. Despite a small sample size, the study offered IO injection as a rapid and safe alternative method for emergently accessing the vascular system for administration of fluids, medications, and blood products. [63] Further research is needed to address the optimal site of injection, instrument used, and insertion technique of adult bone marrow infusion.

CONCLUSION

Peripheral IV access is a necessary skill in the armamentarium of health care providers. It is quickly and easily performed and often life saving. Knowledge of anatomy and sterile technique is essential to successful cannulation and is a skill that can be mastered with minimal training.

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Chapter 22 - Central Venous Catheterization and Central Venous Pressure Monitoring

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CENTRAL VENOUS CATHETERIZATION

The use of central venous access in the emergency department (ED) and intensive care settings has increased in conjunction with new technologies and more invasive approaches to patient care. Advanced monitoring techniques, transvenous pacemakers, and parenteral nutrition all require rapid, reliable methods of central venous access. Select central venous catheterization has also gained acceptance in resuscitation and treatment of the critically ill child (see Chapter 19). Peripheral venous sites can be used for some of these procedures, but use of peripheral sites requires long catheters that must be threaded accurately into the superior vena cava (SVC) or inferior vena cava (IVC). Peripheral veins may be collapsed, thrombosed, buried in subcutaneous (SQ) tissue, or otherwise difficult to locate. Several large central veins, including the subclavian, jugular, and femoral, have predictable relationships to easily identified landmarks and can be cannulated within minutes. Consequently, cannulation of these vessels has become a common practice in a variety of clinical settings, and it is a skill required of all clinicians involved in the care of acutely ill patients.

Even in the most experienced hands, all routes and techniques described in this chapter are associated with complications and some degree of failure. Given the invasive nature of blindly catheterizing the central circulation, considering the close proximity of other organs and structure, and recognizing the less than ideal circumstances under which catheters must be placed, some complications are unavoidable. While knowledge of the anatomy is critical, and attention to technique is paramount, some complications of central venous catheterization are simply inevitable. It is desirable to limit complications, but their occurrence cannot naively be viewed as evidence of faulty technique or substandard care.

Numerous authors have reported success rates and complication rates from central vein catheterization. While data vary widely, and clinical experience portends better results, the 10% to 20% failure rate, the 5% to 10% complication rate, and the 4% malposition rate reported by Sznajder et al. [1] with percutaneous subclavian, anterior jugular, and posterior jugular catheterization are representative of the significant clinical issues surrounding these procedures.

BACKGROUND

Subclavian venipuncture was first described by Aubaniac in 1952, and its use was promptly adopted by others. [2] [3] [4] Subsequently the role of central venous pressure (CVP) monitoring in the maintenance of optimal blood volume was described and helped popularize subclavian venipuncture in the United States. [5]

Numerous reports of clinical experience with the infraclavicular (IC) subclavian technique followed. These stressed the clinical usefulness of the procedure, the ease with which it is performed, and its low complication rate. Subclavian venipuncture was described as useful in the management of hypovolemia, burns, cardiac arrest, chronic IV therapy, and septic shock. [6] [7] [8] [9] [10]

Early enthusiasm for IC subclavian venipuncture was eventually tempered by a growing awareness of serious and occasionally fatal complications. [11] These complications suggested the need for a safer method. In 1965, the supraclavicular (SC) approach was described, with advantages of a more direct approach to the subclavian vein and a potentially lower complication rate. [12] Later these two approaches were compared during the performance of cardiopulmonary resuscitation (CPR). The SC approach offered a significant decrease in catheter tip malposition and CPR interruption. [13]

An early mention of the internal jugular (IJ) approach appeared in a pediatric handbook in 1963. [14] An IJ approach in the adult (with what later became known as the *central approach*) was described in 1966. [15] Subsequently a variety of approaches were described, ultimately to be grouped into the anterior, central, and posterior approaches. [16]

All of these techniques, as well as the femoral and cephalic-basilic approaches, have inherent advantages and disadvantages, but each has a place in the practice of emergency medicine. Often the choice of a particular approach is determined solely by the individual clinician's prior experience with that approach. In general, success rates are higher and complications less frequent when these techniques are used by more experienced technicians. [17] Although every clinician has a preferred method for achieving central venous access, any clinician caring for critically ill patients should master several of these techniques.

INDICATIONS

There are several commonly encountered clinical situations in which central venous access is indicated. If necessary, any central venous approach could be used in each of these situations. However, experience suggests that certain approaches offer advantages over others in most clinical settings. Advantages and disadvantages of each technique are outlined in Table 22-1 and discussed in detail after the general indications.

Central Venous Pressure Monitoring

Although supplanted to a great extent by more sophisticated and accurate methods, particularly right heart catheterization with a balloon-tipped pulmonary artery catheter, CVP measurement may be useful in select hypovolemic patients.

Volume Loading

Subclavian venipuncture has been widely used as a vehicle for rapid volume resuscitation. Unfortunately, it is often misused in this regard. The flow rate of saline through a peripheral 5-cm, 14-ga catheter is roughly twice that through a 20-cm, 16-ga central venous catheter, with equivalent pressure heads. [18] The difference in flow is even greater for blood

TABLE 22-1 -- Advantages and Disadvantages of Central Venous Access Techniques

Technique	Advantages	Disadvantages
Basilic (peripheral) puncture	Low incidence of major complications	Greater incidence of minor complications of infection, phlebitis, and thrombosis
	Performed under direct visualization of the vein	
	Allows large quantities of fluid to be given rapidly	Hinders free movement of arms
		More difficult to place catheter in correct position for central venous pressure monitoring
Internal jugular puncture	Good external landmarks	"Blind" procedure
	Less risk of pneumothorax than with subclavian puncture	Has a slightly higher incidence of failures than subclavian approach
	Bleeding can be recognized and controlled	More difficult and inconvenient to secure
	Malposition of catheter is rare	
	Almost a straight course to the superior vena cava on the right side	
	Carotid artery easily identified	
	Useful alternative approach to cutdown in children younger than 2 years of age	
Femoral puncture	Good external landmarks	Difficult to secure in potentially ambulatory patients
	Useful alternative to other supradiaphragmatic approaches in patients with coagulopathies, superior vena caval trauma, or patients undergoing CPR	"Dirty" site
Infraclavicular subclavian approach	Good external landmarks	Higher incidence of complications, especially in hypovolemic shock
		"Blind" procedure
		Should not be attempted in children younger than 2 years of age
Supraclavicular subclavian approach	Good landmarks	"Blind" procedure
	Less risk of pneumothorax	
	Most practical method of inserting a central line in cardiorespiratory arrest	
	Malposition of catheter is uncommon	

From Knopp R, Dailey RH: Central venous cannulation and pressure monitoring. JACEP 6:358, 1977.

products because their higher viscosity further slows their passage through small-gauge catheters. [19] Consequently, the placement of peripheral large-bore catheters is the preferred method of rapid volume loading, unless time would be lost searching for a venipuncture site or unusually large volumes need to be infused.

Use of a large-bore (8 Fr) introducer catheter overcomes the impairment of flow seen with small-gauge central venous catheters. Flow rates through introducers alone can exceed those of common IV tubing.[20] In trained hands placement of an introducer device is a useful method of fluid resuscitation when very rapid flow rates are required. The benefit of increased flow rate is potentially limited by catheter kinking. [21] This problem is more common when long introducer catheters are placed via the IC route. A significant risk of introducer insertion is catheter misplacement, which may result in rapid infusion of fluid or blood into the chest cavity or mediastinum.

Emergency Venous Access

The predictable anatomic locations of the subclavian and femoral veins, and the speed with which they can be cannulated (often within 30 seconds) have prompted their use in cardiac arrest and other emergency situations. The need for a central line during CPR is controversial. Although there is not a universal standard of care, some clinicians advocate use of a central line routinely in cardiac arrest. It has been suggested that therapeutic drug levels are reached more rapidly if given centrally.[22] [23] However, the clinical importance of this observation remains theoretical. When easily obtained, central venous cannulation is preferred over peripheral venous access, because it provides a rapid and reliable route for the administration of drugs to the central circulation of the patient in cardiac arrest.

Routine Venous Access

Patients with a history of IV drug abuse, major burns, or obesity and those requiring long-term care may have inadequate peripheral IV sites. Central venous cannulation may be indicated as a means of venous access in these patients even under nonemergent conditions.

Routine Blood Drawing

The potential complications of central venous cannulation do not justify its use in routine blood sampling. Lines already in place may be used for this purpose if they

are properly cleared of IV fluid. A 20-cm, 16-ga catheter contains 0.3 mL of fluid, so at least this much must be withdrawn to avoid dilution of blood samples. Further, to avoid aspiration of crystalloid diluted blood from the peripheral vein, it is advised that the IV be turned off for 2 to 3 minutes prior to using the catheter for a blood draw. Because of the increased risk of infectious complications, air embolus, and venous backbleeding, the IV

tubing should not be repeatedly disconnected from the catheter hub. Interposition of a three-way stopcock in the IV tubing simplifies access and is an acceptable method of blood sampling in the intensive care setting, regardless of the IV site.

Infusion of Hyperalimentation and Other Concentrated Solutions

Hyperalimentation by way of the subclavian vein is safe and reliable. [24] Use of the IC technique frees the patient's extremities and neck; this procedure is therefore well suited to long-term applications. However, strict aseptic technique is necessary to minimize infectious complications. [25] Hyperosmolar or irritating solutions that have the potential to cause thrombophlebitis if given through small peripheral vessels are frequently infused by way of the subclavian vein. Examples are potassium chloride (>40 mmol/L), hyperosmolar saline, 10% dextrose infusions, chemotherapeutic agents, and acidifying solutions such as ammonium chloride. Some clinicians prefer to obtain central access because of the potential harm of extravasation of vasoactive substances (dopamine, norepinephrine), which may result in soft tissue necrosis.

Other Indications

Other indications for central venous access include placement of a pulmonary artery catheter or transvenous pacemaker, performance of cardiac catheterization and pulmonary angiography, and hemodialysis. Catheters such as the Uldall or Quinton device can be inserted within minutes, permitting use of the subclavian vein for emergency or short-term hemodialysis.[26]

Relative Indications for Different Approaches

Subclavian approaches.

Subclavian venipuncture is the most frequently used means of central venous access. The IC approach was the first popular means of central venous access and has been widely taught during residency training for more than 30 years. It is effective, useful in many clinical situations, and relatively easy to learn. Although generally safe in experienced hands, the IC approach is associated with significant complications, most notably pneumothorax. The SC approach is an important alternative to IC venipuncture. The SC approach may be preferable during CPR because it minimizes physical interference with the functions of chest compression and airway management. The IC approach requires deep penetration of a moving chest wall and frequently demands an interruption of chest compression. An SC subclavian venipuncture can be performed without cessation of CPR and involves superficial penetration of the relatively motionless neck. [13] The SC approach also avoids interference with airway management, which commonly occurs when the internal jugular vein is cannulated. [27] When a true central venous location is required, the SC approach is superior to the IC approach and long peripheral line insertion techniques because of the low incidence of catheter tip malposition with the SC approach. [28] In addition, the SC technique has been performed in the sitting position in patients with severe orthopnea. Placement of a central line with the patient in a sitting position is virtually impossible with other central venous access routes. [29] Finally, the low complication rate reported for SC subclavian venipuncture makes it a more attractive alternative, especially in the seriously ill patient. [27] [28] [29]

Internal jugular approach.

As is true of the SC subclavian approach, the IJ technique is useful for routine central venous access and for emergency venous access during CPR, since the site is removed from the area of chest compressions.[30] Comparison of IJ and subclavian cannulation has found a significantly greater incidence of proper venipuncture and catheter passage with the IC subclavian approach as compared with the posterior IJ method (98% vs 84%). [31] A 20% rate of catheter malposition was noted with each method. In 1 retrospective study, only 0.4% of 248 IJ cannulations resulted in clinically significant morbidity, compared with 4.2% of 298 subclavian insertions, even though the overall complication rate was similar. [32] Although there may be a slight difference in complications between the two routes, in the absence of specific contraindications, the clinician should use the technique with which he or she is most familiar. The IJ route is slightly more technically difficult than the subclavian route but is faster and easier than a venous cutdown. Bedside ultrasound guidance offers the potential to allow safer catheterization of the internal jugular vein (see Chapter 69).

Femoral approach.

The cannulation of the femoral vein for central venous access has become increasingly popular, especially for venous access, infusion ports, and the passage of transvenous pacemakers and pressure measurement catheters in critically ill patients. [33] Some investigators recommend femoral vein cannulation for hypotensive trauma patients with an 8.5 Fr catheter connected to genitourinary irrigation tubing immediately after 2 peripheral catheters are established. [34] Other indications for urgent femoral cannulation include emergency cardiopulmonary bypass for resuscitation purposes, charcoal hemoperfusion for severe drug overdoses, and dialysis access.[35] Advantages of the femoral site over other central venous access sites are that the femoral area is less congested with monitoring and airway equipment than the head and neck area and that the conscious patient, who is still bedridden, may turn the head and use the arms without movement of the central line. The femoral site is contraindicated in the ambulatory patient who requires central access.

Other approaches.

When other methods of central venous catheterization are not possible, central venous access may be obtained via the external jugular (EJ) vein or basilic-cephalic vein.[36] [37] Although generally accessible for peripheral IV access, the valves and tortuousity of these veins often preclude or delay placement of standard central venous catheters. Successful cannulation of the central venous circulation is generally possible by these routes only with the use of guidewires. When time is available for a careful, deliberate attempt, these methods avoid the complications of pneumothorax, carotid or subclavian artery puncture, and hidden hemorrhage associated with other methods of central venous cannulation.

The EJ approach can be used in both children and adults, but success is more common in adults. [37] [38] The EJ vein must be visible to the eye or via ultrasound for percutaneous cannulation to be successful (see Fig. 21-2). This route is used primarily as a peripheral venous action site. Central venous catheterization by the EJ route is technically more difficult than IJ cannulation, but it is successful 70% to 100% of the time in adults. [39] While use of a straight guidewire has been described, the use of a J wire is more reliable and is the preferred method. [38] The J wire is more easily advanced

because its round tip deflects off vessel walls and navigates sharp angulations in the vessel course more easily.

When rapid access to the central venous circulation is not important, the basilic-cephalic route may be considered. This route has the lowest incidence of complications since the basilic and cephalic veins are located far away from vital organs and major arteries. When the patient is upright, the basilic vein is preferred over the cephalic vein because of a higher incidence of successful central catheter passage, although the overall success rate of SVC cannulation is similar for both techniques in the supine patient. [40] Nonetheless, both veins have valves, which may impede catheter advancement. [41]

CONTRAINDICATIONS

Contraindications to the various techniques of central venous access are shown in Table 22-2 . Most listed contraindications must be considered relative, with clinical use of these techniques based on clinical conditions and available options for vascular access. Each technique is contraindicated in patients with distorted local anatomy or landmarks. Skin lesions such as local cellulitis, burns, abrasions, or severe dermatitis are relative contraindications to any access routes. Other relative contraindications include those conditions predisposing to sclerosis or thrombosis of the central veins, such as vasculitis, prior long-term cannulation, or illicit IV drug use via any of the deep venous systems. Mumatz and colleagues reviewed the placement of central venous catheters in patients with disorders of hemostasis. They found that even in patients with low platelet counts (i.e., <50 × 10^9 /L), bleeding complications are uncommon and generally easily managed, in the absence of arterial puncture.[42] The incidence of bleeding complications (other than bleeding requiring digital pressure)

TABLE 22-2 -- Relative Contraindications to Specific Central Venous Access Routes *

General
Distorted local anatomy†
Extremes of weight
Vasculitis
Prior long-term venous cannulation
Prior injection of sclerosis agents
Suspected proximal vascular injury
Previous radiation therapy
Bleeding disorders
Anticoagulation or thrombolytic therapy
Combative patients
Inexperienced, unsupervised physician
Subclavian vein
Chest wall deformities
Pneumothorax‡
Chronic obstructive pulmonary disease
Jugular vein
Intravenous drug abuse via the jugular system
Femoral vein
Need for patient mobility
Basilic-cephalic veins
Cardiac arrest§
Anticipated future use of these vessels

*Use of this technique must be based on clinical conditions and available options for vascular access.
†May use contralateral side.
‡Use of ipsilateral side.
§May use standard (short) large-bore IV catheters.

in patients with abnormal hemostasis in their study was only 3%, and no benefit could be found from correcting an abnormal international normalized ratio (INR) or platelet count prior to the procedure. This conclusion challenges prior anecdotal recommendations that patients with a coagulopathy receive blood component therapy to correct a coagulopathy prior to percutaneous central venous catheterization.

Subclavian Approach

Patients in whom subclavian access is contraindicated include those who have undergone previous surgery or trauma involving the clavicle, the first rib, or the subclavian vessels; patients who have undergone previous radiation therapy to the clavicular area; patients with significant chest wall deformities; and those with marked cachexia or obesity. However, clinicians in burn centers routinely place central catheters through burned areas. Patients with unilateral deformities not associated with pneumothorax (e.g., fractured clavicle) should be catheterized on the opposite side.

Subclavian venipuncture is not contraindicated in patients who have penetrating thoracic wounds unless the injuries are suspected or known to involve the subclavian vessels or SVC. Generally, *the vein on the same side of the chest wound should be cannulated* to avoid the possibility of bilateral pneumothoraces. When subclavian vessel injury is suspected, cannulation should occur on the opposite side. [43] With penetrating wounds that may involve the SVC, neither subclavian vessel should be cannulated, and venous access below the diaphragm should be sought. Use of the subclavian approach in patients with coagulation disorders or in those receiving heparin therapy is contraindicated. A more visible and accessible site should be chosen (preferably percutaneous cannulation of a peripheral vein), because it is impossible to apply direct pressure to a bleeding subclavian vessel. The procedure should not be performed in combative patients because of the greater possibility of pneumothorax, vessel laceration, air embolism, and septic complications. Although subclavian venipuncture has been used successfully in children younger than 2 years of age,[44] it is not generally recommended for use in small children. [45]

Internal Jugular Approach

Cervical trauma with swelling or anatomic distortion at the intended site of IJ venipuncture is the most important contraindication to the IJ approach. Neck motion is limited when the IJ line is in place, and this limitation represents a relative contraindication in conscious patients. Although bleeding disorders are relative contraindications to central venous cannulation, the IJ approach is preferred over the subclavian route, as the IJ site is compressible. In the setting of severe bleeding diatheses, the femoral approach should be considered. Carotid arterial disease (obstruction or atherosclerotic plaques) is a relative contraindication to IJ cannulation, since inadvertent puncture or manipulation of the artery may dislodge a plaque. In addition, prolonged compression of the artery to control bleeding may impair cerebral circulation if collateral blood flow is compromised. If a preceding subclavian catheterization has been unsuccessful, the ipsilateral IJ route is generally preferred for a subsequent attempt. In this manner, bilateral iatrogenic complications are avoided.

External Jugular Approach

The greatest disadvantage of the EJ approach for central venous access is the time required to successfully pass a J wire and catheter into appropriate position. [46] However, the ease and relative safety of this approach make the EJ vein an excellent site of simple IV cannulation for fluid or drug administration, especially during an emergency when peripheral veins cannot be cannulated.

Femoral Vein Approach

Contraindications to femoral cannulation include known or suspected injury to the groin, iliac vessels, or IVC. Percutaneous femoral line placement is not recommended in a patient who is in cardiac arrest or has an absent femoral pulse, unless other alternatives have been exhausted. [47] In a

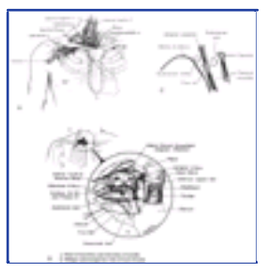

Figure 22-1 *A* and *B,* Subclavian vein and local anatomy. *C,* Subclavian area, sagittal section. *(A from Linos D, Mucha P, von Heerden J: The subclavian vein: A golden route. Mayo Clin Proc 55:316, 1980. B from Davidson JJ, Ben-Hur N, Nathan H: Subclavian venipuncture. Lancet 2:1140, 1963. C from Brahos G: Central venous catheterization: SC approach. J Trauma 17:873, 1977. Reproduced by permission.)*

canine model of cardiac arrest, palpable pulsations in the groin were found to be venous rather than arterial in origin 50% of the time. [48]

ANATOMY

Subclavian System

The subclavian vein begins as a continuation of the axillary vein at the outer edge of the first rib (Fig. 22-1). It joins the IJ vein to become the innominate vein 3 to 4 cm proximally. The subclavian has a diameter of 10 to 20 mm and is valveless. After crossing the first rib, the vein lies posterior to the medial third of the clavicle. It is only in this area that there is an intimate association between the clavicle and the subclavian vein. The costoclavicular ligament lies anterior and inferior to

the subclavian vein, and the fascia contiguous to this ligament invests the vessel. Posterior to the vein, separating it from the subclavian artery, lies the anterior scalene muscle, which has a thickness of 10 to 15 mm. The phrenic nerve passes over the anterior surface of the scalene muscle and runs immediately behind the junction of the subclavian and IJ jugular veins. The thoracic duct (on the left) and the lymphatic duct (on the right) pass over the anterior scalene muscle and enter the subclavian vein near its junction with the IJ vein. Superior and posterior to the subclavian artery lies the brachial plexus. The dome of the left lung may extend above the first rib, but the right lung rarely extends this high.

Jugular System

The anatomy of the IJ vein is relatively constant, regardless of body habitus. The vein drains the cranium, beginning as the superior jugular bulb, which is separated from the floor of the middle ear by a delicate bony plate. The IJ vein emerges deep to the posterior belly of the digastric muscle. At its origin the IJ vein courses adjacent to the spinal accessory, vagus, and hypoglossal nerves, as well as the internal carotid artery. Several

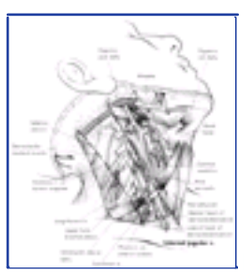

Figure 22-2 Structures in a dissection of the neck. The superficial veins and the sternocleidomastoid muscle have been removed, as have the submandibular gland and a segment of the facial vein. The cutaneous nerves have been cut down to short stumps arising from the second, third, and fourth cervical nerves. The internal jugular vein is drawn somewhat more medial in this illustration than is commonly found. *(From Hollinshead WH: Textbook of Anatomy, 3rd ed. New York, Harper & Row, 1974, p 765. Reproduced by permission.)*

tributary veins enter the IJ vein at the level of the hyoid bone. The IJ vein, the internal (and, later, the common) carotid artery, and the vagus nerve course together in the carotid sheath, with the IJ vein occupying the anterior lateral position. [49] The only structure that maintains a fixed anatomic relationship with the IJ vein is the carotid artery. The vein invariably lies lateral and slightly anterior to the carotid artery, and the course of the artery serves as a guide to venous cannulation. At the level of the thyroid cartilage, the IJ vein can be found just deep of the sternocleidomastoid muscle (Fig. 22-2).

The IJ vein emerges from under the apex of the triangle of the two heads of the sternocleidomastoid muscle and joins the subclavian vein behind the clavicle. As the vein approaches its supraclavicular junction with the subclavian vein, it assumes a more medial position in the triangle formed by the two heads of the sternocleidomastoid muscles, following the anterior border of the lateral head. In this lower cervical region, the common carotid artery assumes a deep paratracheal location. The brachial plexus is separated from the IJ vein by the scalenus anterior muscle. The phrenic nerve is anterior to the scalenus anterior muscle. Although quite deep, the stellate ganglion lies anterior to the lower brachial plexus.

Unlike the subclavian vein, the IJ vein is quite distensible. The vessel diameter is increased with performance of a Valsalva maneuver and the assumption of the head-down (Trendelenburg) tilt position. Prolonged palpation of the carotid pulse will decrease the diameter of the IJ vein. [49] Rotating the head 90 degrees toward the opposite side or extending the neck will not change the size of the IJ vessel significantly. Severe rotation of the head will bring the sterno-cleidomastoid muscle anterior or medial to the IJ vein and may make cannulation impossible without first traversing the carotid artery. The diameter of the IJ vessel is largest below the cricoid ring, where it may reach 2 to 2.5 cm.[49]

Femoral System

The femoral vein is most easily cannulated percutaneously in patients with a palpable femoral pulse. The femoral vein lies

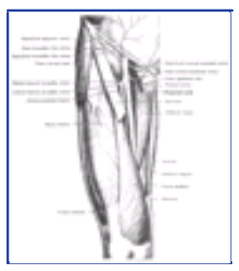

Figure 22-3 The right femoral vessels and some of their branches. The femoral nerve (not shown) lies lateral to the artery and may be deep to the artery. *(From Warwick R, Williams PL [eds]: Gray's Anatomy, 35th ed. Edinburgh, Churchill Livingstone, 1973, p 676. Reproduced by permission.)*

just medial to the artery in the femoral canal below the inguinal ligament. Beneath the femoral vessels lie the psoas muscle and the hip (Fig. 22-3). As the femoral vein progresses distally in the leg, it becomes closer, and almost posterior, to the femoral artery. When cannulating this vessel distally to the inguinal ligament, ultrasound guidance can be helpful to avoid arterial puncture.

Basilic and Cephalic System

Considerable variation is present in the venous vasculature of the upper extremities. Nonetheless, the cephalic and basilic veins can usually be located in the volar antecubital region (Fig. 22-4). The interconnecting median antecubital vein is often the most prominent, thus making it a popular site for venipuncture during blood sampling. The basilic vein merges proximally with the brachial vein to form the axillary vein,

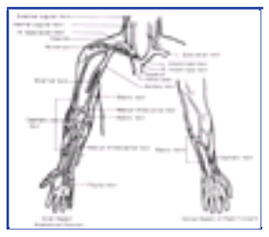

Figure 22-4 Major veins of the upper half of the body. *(From Hedges JR: Vascular access. Curr Top Emerg Med 2:1, 1981. Reproduced by permission.)*

which subsequently meets the cephalic vein to form the subclavian vein near the distal clavicle. The IJ and EJ veins join the subclavian vein to form the innominate vein bilaterally. Many venous valves exist in the peripheral vessels. Vascular anastomoses may permit aberrant advancement of a long line from the upper extremity. In particular, lines threaded up the cephalic vein may dead-end in a venous plexus or enter the EJ vein. Furthermore, lines passed through the basilic vein may easily enter the IJ vein.

TECHNIQUE AND EQUIPMENT

Some clinicians prefer to first locate the position of a central vein with a small exploratory or "finder" needle, rather than directly cannulating the vein with only the larger needle that will accommodate a guidewire or catheter. While this may be desirable in some circumstances, and can minimize trauma and complications of a larger needle, no specific standard exists. In clinical practice, this exploratory needle is seldom used in an emergency situation. The smaller introducing needles used with the Seldinger technique have largely supplanted the need for this exploratory procedure.

Seldinger and Other Techniques

The most commonly used means of central venous cannulation is the Seldinger (guidewire) technique, by which a thin-walled needle is used to introduce a guidewire into the vessel lumen. Seldinger originally described this in 1953 as a method for catheter placement in percutaneous arteriography. [50] To obtain vascular access, a small needle is used to enter the intended vessel. Once the introducing needle is positioned within the vessel lumen, a wire is threaded through the needle, and the needle is removed. The wire, now within the vessel, serves as a guide over which the selected catheter is placed. Although the Seldinger technique involves several steps, it may be performed quickly once mastered. More important, this technique broadens the application of central vein cannulation, permitting the insertion of standard infusion catheters,

TABLE 22-3 -- Materials for Central Venous Cannulation

1% lidocaine
26-ga needle
2-mL Luer-Lok syringe (for anesthetic)
10-mL non-Luer-Lok syringe (for catheter placement)
Swabs
Preparation solution
Gloves
Drapes
Catheter device
Intravenous tubing
Intravenous solution
Needle holder
4-0 silk (or nylon) sutures
Suture scissors
Antibiotic ointment
Gauze pads
Tincture of benzoin
Cloth tape

multi-lumen catheters, large-bore rapid infusion systems, introducer devices, and even peripheral cardiopulmonary bypass cannulae. Given this degree of flexibility, the use of Seldinger-type systems is advantageous, despite greater cost.

The basic materials required for central venous cannulation are listed in Table 22-3 . The catheter may be a component in a guidewire system, or of the over-the-needle variety (the other widely used method of catheter placement). To obtain central access from the basilic-cephalic system (and occasionally from the femoral vein), a through-the-needle catheter passage technique is used. This is detailed in a special section after discussion of these more commonly used approaches.

Needle

Virtually any needle or catheter can be used to introduce a guidewire into a vessel, but there are advantages to using needles specifically designed for passage of a guidewire. These needles must be large enough to accommodate the desired wire, yet as small as possible to minimize bleeding complications. The needles provided with central vein catheters or introducer devices are usually thin walled, thereby maximizing lumen size relative to overall needle diameter. If a needle that is not thin walled is used, a size that is 1 ga smaller (larger bore) than that listed in Table 22-4 should be used.

Standard needles may have a uniformly straight-bore lumen throughout their length. A wire passing into a straight needle may encounter an obstacle at the proximal end. The proximal end of a Seldinger needle incorporates a funnel-shaped taper that guides the wire directly into the needle (Fig. 22-5).

TABLE 22-4 -- Needle Sizes for Venous and Arterial Catheters*

Standard Full-Length Coil Guidewire Catheter Size (Fr)	Needle Gauge†
3	21-
4 to 4.5	20-
5 to 6.0	20- to 19-
6 to 8.5	19- to 18-

*Any sized catheter from 3.0 to 8.5 Fr may be introduced using a 22-ga needle if a solid wire (Cor-Flex, Cook Critical Care) is used.
†All needle gauges are for thin-walled needles only.

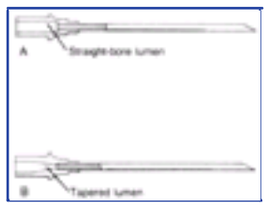

Figure 22-5 Introducing needles. *A,* Ordinary needle with a straight-bore lumen. *B,* Seldinger needle with a tapered lumen, allowing easy entry of the guidewire.

It is advisable to use a non-Luer-Lok or slip tip type, as the added twisting that is required to remove a Luer-Lok syringe from the introducer needle may dislodge a tenuously placed needle. Systems now exist that permit passage of the wire without removal of the aspirating syringe.

Guidewire

Two basic types of guidewires are used: straight or J-shaped. The straight wires are for use in vessels with a linear configuration, whereas the J wires are for use in tortuous vessels. Both wires have essentially the same internal design (Fig. 22-6*A*). The flexibility of the wire is a result of a stainless steel coil or helix that forms the bulk of the guidewire. Within the central lumen of the helix is a straight central core wire, called a mandrel, which adds rigidity to the steel coil. The mandrel is usually fixed at one end of the helix and terminates 0.5 and 3.0 cm from the other end, creating a flexible or floppy tip. Wires are also available with two flexible ends, one straight and the other J-shaped. The flexible end of the guidewire allows the wire to flex on contact with the wall of a vessel. If the contact is tangential, as in an infraclavicular approach to the subclavian vein, a straight wire is generally

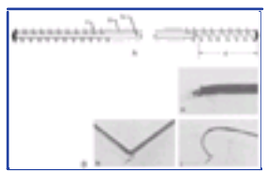
Figure 22-6 *A*, Guidewire internal structure: safety wire (a), core wire (mandrel) (b), coiled wire (c), flexible tip (d). *B*, Flexible end of a straight-spring guidewire knotted on a vessel dilator (a), bent junction of the rigid and flexible portions of a straight-spring guidewire with protrusion of the central core (arrow) (b), partially fractured tip (arrow) of a J-spring guidewire (c). *(From Schwartz AJ, Horrow JL, Jobes DR, Ellison N: Guide wires—A caution. Crit Care Med 9:348, 1981. ©1981 Williams & Wilkins, Baltimore. Reproduced by permission.)*

preferred. If the angle is more acute, as in an external jugular approach to the subclavian vein, or if the vessel is particularly tortuous or valves must be traversed, a J-shaped wire is used. The more rounded leading edge of the J wire provides a broader surface to manipulate within the vessel and decreases the risk of perforation. This is especially advantageous when attempting to thread a wire through a vessel with valves. Many guidewires also contain a straight safety wire that runs parallel to the mandrel to keep the wire from kinking or shearing.

The standard size for guidewires is from 0.025 to 0.035 in. (0.064 to 0.089 cm) in diameter, permitting introduction through an 18-ga thin-walled needle. A modification of this standard wire uses a bare mandrel with the flexible coil soldered to its end. This construction provides a wire with a diameter of only 0.018 in. (0.047 cm) but with the same rigidity as the larger wires. The manufacturer states that such a wire can be introduced through a 22-gauge thin-walled needle, yet still guide an 8.5 Fr catheter (Micropuncture Introducer Sets and Trays with Cor-Flex Wire Guides, Cook Critical Care Inc., Bloomington, IN).

It is important to emphasize that guidewires are delicate and may bend, kink, or unwind. A force of 4 to 6 lb may cause rupture of a wire. Wires should thread easily and smoothly and should never be forced. Embolization of portions of the guidewire is possible, and sharp defects in the wire may perforate vessel walls (Fig. 22-6*B*). Wires should be inspected for defects such as kinks, sharp ends, or spurs before use.

Catheters

A number of different catheter and introducer devices have been developed and the method of passage into the vessel varies accordingly. Single-, double-, and triple-lumen catheters generally are placed by sliding the catheter directly over the guidewire into the intended vessel. Larger catheters or nonlumen devices are generally introduced with a sheath-introducer system. Over-the-needle catheters are simply placed over the needle once intravascular placement is attained.

The Desilets-Hoffman type sheath introducer became available in 1965 to aid in arteriography procedures that required many catheter changes. [51] The sheath-introducer unit includes two catheters, an inner dilator and an outer sheath


Figure 22-7 Desilets-Hoffman sheath introducer. *A*, Guidewire. *B*, Sheath-introducer. *C*, Dilator. *D*, Assembled device.

(Fig. 22-7). The dilator is rigid with a narrow lumen to accommodate the guidewire. It is longer and thinner than its sheath and has a tapered end that dilates the SQ tissue and the vessel defect formed by the needle. The sheath (or introducer catheter when used as a cannula for introducing Swan-Ganz catheters, transvenous pacemakers, or other devices), has a blunt end and is simply a large diameter catheter.

Many modifications of the sheath exist, with side arms and diaphragms to aid in placement of nonlumen devices. Care must be taken in the use of side-arm sets for rapid fluid administration, because some catheters may be 8.5 Fr in diameter but may have only a 5 Fr side arm. This problem can be addressed by introducing a short segment of an 8 Fr feeding tube through the diaphragm at the catheter hub for rapid fluid administration.

Special catheters have been developed to prevent bacterial contamination and line sepsis. These catheters are impregnated either with antiseptics (silver sulfadiazine and chlorhexidine) or antibiotics (minocycline, rifampin, or cefazolin) to reduce bacterial colonization and microbe growth. Also, heparin-coated catheters are available that prevent fibronectin binding, thereby inhibiting the formation of bacterial biofilms on the catheter's surface. Whether these in vivo effects lead to decreased nosocomial infection rates and cases of line sepsis remains controversial. Few published studies demonstrate a statistically significant reduction in the infection rate when compared to traditional catheters.[52] [53] However, the overall risk of infection is generally low, thus small differences are difficult to detect. Two meta-analyses independently found that the use of antimicrobial and heparin bonded catheters reduced the risk of infection from about 5% to 3%. [52] [53] An impregnated catheter costs $25 to $40 more than a comparable standard device. This compares favorably to the estimated $6000 to $10,000 in additional medical costs associated with a case of line sepsis. Additionally, a catheter-related bloodstream infection may increase mortality by 15% to 35%. Convincing data do not exist to recommend one catheter type over another, though the silver sulfadiazine- and chlorhexidine-impregnated catheters are the most widely used and offer only modest additional expense. It is important to ensure that no known hypersensitivity to the impregnating-agent exists before insertion of these catheters.

Guidewire Placement

An introducing needle large enough to accommodate the guidewire is attached to a small syringe (Fig. 22-8). The needle and syringe are introduced together, and the selected vessel is entered with the needle tip (technique for specific veins and approaches is detailed later). Once a free return of blood is obtained, the syringe is removed, and the needle hub is stabilized to prevent needle movement and displacement of the tip from the vessel. At times detachment of the syringe from the thin-walled needle may lead to loss of the needle's intravascular position. The need to detach the syringe can be eliminated by use of the Arrow Safety Syringe. This device incorporates a hollow syringe through which the guidewire can be passed directly into the thin-walled needle without detachment. This also reduces the risk of air embolism, which can occur when the needle is open to the air. If the needle is removed from the syringe, the needle hub is capped with a thumb before passing the guidewire to minimize the potential for air embolism.

Next, the flexible end of the guidewire is threaded through the needle. The straight wire is easily introduced by threading its flexible end into the hub of the needle. Introduction of the J wire is accomplished by advancing a plastic sleeve contained in the kit to the floppy end of the wire, straightening out the J shape. This

straightened end is then introduced into the needle hub. Once the J wire has been advanced, the sleeve is removed and set aside (Fig. 22-9). It is important not to accidentally discard this sleeve as it will make insertion of the J wire very difficult.

The wire should thread smoothly into the vein without resistance. The wire should not be forced if resistance is met, but it should be removed from the needle and the syringe reattached to confirm intravascular placement. It is extremely important for the wire to slip easily from the needle during removal. If any resistance to removal of the wire is felt, the wire and the needle should be removed as a single unit to prevent shearing of the wire and resultant wire embolism. It has been recommended by some that no wire should ever be withdrawn through the introducing needle. [54] While there are no data to support this recommendation and newer wires are stronger and more resistant to shearing, it represents the

Figure 22-8 Procedure for placement of Seldinger-type guidewire catheter. *A*, The selected vessel is cannulated with a thin-walled needle, or an existing IV catheter is chosen to be changed with the wire technique. *B*, The guidewire is threaded through the vessel, with the flexible end first, into the lumen of the vessel. If a J wire is used, a sleeve will facilitate entry into the needle (see Fig. 22-9). *C*, The needle is removed so that only the wire now exits from the vessel. *D*, The skin entry site is enlarged with a No. 11 scalpel. *E*, The catheter sheath and the dilator are threaded over the wire and advanced to the skin. The wire must be visible through the back of the device. *F*, If the proximal wire is not visible, it is pulled from the skin through the catheter until it appears at the back of the catheter. *G*, The sheath and the dilator are advanced as a unit into the skin with a twisting motion. It is best to grasp the unit at the junction of sheath and dilator to prevent bunching up of the sheath. The wire (at the back of the catheter) must be held while the sheath and dilator are advanced as a unit. *H*, Once the sheath and the dilator are well within the vessel, the guidewire and the dilator are removed. Contrary to the illustration, latex gloves should be worn.

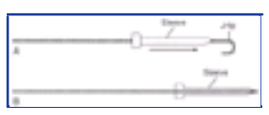

Figure 22-9 J wire. *A*, Plastic sleeve in retracted position, demonstrating the J tip. *B*, Plastic sleeve is advanced to straighten the curve to allow easy introduction into the needle hub. In an emergency, care must be taken to not misplace or throw away the sleeve. Without it, placing the J wire into the hub of the needle is very difficult.

safest course of action (Fig. 22-10). The recommendation to remove the needle and the wire as a unit is sometimes disregarded because of reluctance to abandon a potentially successful venipuncture. The clinician performing the procedure must use both caution and good judgment to determine the best course of action, but should not withdraw the guidewire against resistance. Manipulation of the wire within an introducer needle should be done only with standard coil guidewires. Solid wires (such as Cor-Flex Wire Guides from Cook Critical Care) have a small lip at the point the flexible coil is soldered to the wire. This lip can become caught on the edge of the needle tip, shearing off the coil portion of the wire. Solid wires must thread freely on the first attempt or the entire wire and needle assembly must be removed.

Occasionally, a wire must be teased into the vessel; rotating the wire or needle often helps in difficult placements. If the wire does not thread easily, another helpful maneuver is to pull back slightly on the needle itself just before advancing the wire. This helps if the opening of the needle is abutting the vessel's inner wall, blocking the wire's entry, or if the vein is compressed by introduction of the needle. Changing wire tips from a straight to a J wire or vice versa also may solve an advancement problem. If the inner lumen of a vessel is smaller than the diameter of the J, it will prevent the wire from reforming its natural shape, causing the spring in the coil to generate resistance. Any advantages of a J wire will be negated if the wire fails to regain its intended shape. In this instance, a straight tip should be introducible without problem. Alternatively, if the angle of entry of the needle and the vessel is more acute than was suspected, the straight wire may not be able to bend appropriately as it encounters the vessel's far wall. A J-tipped wire may be used and threaded in such a manner that the wire resumes its J shape away from the far wall. All of these maneuvers are performed with gentle free motions of the wire within the needle. If at any time the wire cannot be advanced freely, improper placement must be suspected and the attempt reevaluated.

If it is threading easily, the guidewire should be advanced until at least one quarter of the wire is within the vessel. The further into the vessel the wire extends, the more stable its location when the catheter is introduced. However, advancing the guidewire too far may result in ventricular ectopy secondary to endocardial irritation, myocardial puncture leading to tamponade, or entanglement in a previously placed pacemaker, internal defibrillator, or IVC filter. In both left and right IJ and IC approaches, fluoroscopic study during guidewire passage has determined the mean distance from

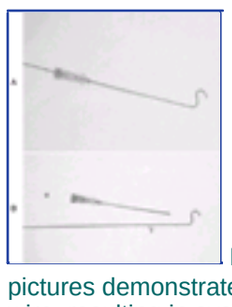

Figure 22-10 Although newer guidewires are more resistant to shearing, if a guidewire will not advance, it is best to withdraw both the needle and wire in one motion. These pictures demonstrate a permanently deformed guidewire that could not be advanced. Withdrawing the wire with the indwelling introducer needle in place within a vessel may shear off a portion of the wire, resulting in systemic embolization.

skin to SVC-atrial junction to be 18 cm. [55] This distance has been recommended as the greatest depth of guidewire insertion for these approaches. (It should be noted that 18 cm is not necessarily the appropriate final depth for the catheter being placed—see later discussion).

Cardiac monitoring is recommended for all central line insertions. Any increase in premature ventricular contractions or new ventricular dysrhythmia should be interpreted as evidence the guidewire is inserted too far, and should be remedied by withdrawing the wire until the rhythm reverts to baseline. Usually after a moment the procedure can be continued, with care taken not to re-advance the wire. Persistent ventricular dysrhythmias require standard Advanced Cardiac Life Support (ACLS) treatment and consideration of a new vascular approach.

Occasionally a wire threads easily past the tip of the needle and then suddenly will not advance farther. If the introducer needle demonstrated free blood return at the time of wire entry and the initial advancement of the wire met no resistance, the wire is most likely located properly in the vessel and can serve as a guide for the catheter. If further confirmation is needed, the needle may be removed, the wire fixed in place with a sterile hemostat, and a radiograph taken to confirm the position of the wire. [56] This confirmation may be advisable if the location of a wire is suspect and the introduction of a large-sized sheath is planned. A freely advancing wire may suddenly stop once it is well within a vessel if the vessel makes an

unsuspected bend or is being compressed or deviated by another structure, such as a rib or muscle.

Sheath Unit and Catheter Placement

Once the wire is placed into the vessel, the needle is removed in preparation for passage of the catheter (see Fig. 22-8). A small skin incision is then made at the site

of the wire. The incision should be approximately the width of the catheter to be introduced and should extend completely through the dermis. The guidewire is stabilized at the point of the skin incision and the dilator/sheath assembly is threaded over the wire to a point 1 cm from the surface of the skin. Once the dilator/sheath is advanced over the wire and before it enters the skin, the wire must protrude from the proximal end of the dilator. It is essential that the wire be grasped as the dilator/sheath is advanced to avoid farther advancement into the circulation and potential loss of the wire. If the wire is not protruding from the proximal end of the dilator, the wire should be withdrawn at the skin entry point until it protrudes a sufficient amount to be grasped. The wire must always be visible protruding from the end of the dilator at all times during dilator advancement.

The dilator/sheath assembly is threaded into the skin with a twisting motion until it is well within the vessel. When using a sheath/dilator, it is best to grasp the unit at the junction of the sheath and dilator. This prevents the thinner sheath from kinking or bending at the tip or from bunching up at the coupler end. If a rigid-walled sheath is used, the dilator need only be advanced a few centimeters into the vessel and the sheath slid off and advanced to its hub. If a thin-walled sheath is used, the introducer-sheath unit is kept intact and advanced through the skin to the hub. This adds rigidity to the sheath and prevents it from kinking before being fully seated in the vessel. When placing right-sided subclavian catheters, it is sometimes helpful to make a banana-like bend in the dilator before introduction to facilitate proper location in the SVC. [57] After the sheath is in place, the wire and dilator are removed together. The sheath hub must be covered at this point and until attachment of the infusion tubing or cap to avoid air embolism.

If a single-lumen catheter is used instead of a sheath/dilator, the catheter itself is passed over the wire to its desired depth and the wire is removed. When a soft catheter is used, a tract from the skin to the vessel must be created before the catheter can be introduced. This is accomplished by passing and then withdrawing a separate dilator over the guidewire, after the needle is removed but before the catheter is placed. After the dilator is removed the soft catheter is threaded into the position over the wire. Occasionally, it is easier to advance the soft catheter over the wire up to the skin edge and then to advance the catheter and wire together as a unit into the vessel. It is imperative that the guidewire protrudes from the catheter hub and be firmly grasped as the wire and catheter are advanced. Once the catheter is placed, the wire is gently removed with care taken to maintain the desired catheter insertion length. When removing the wire from a catheter it must slip out easily. If any resistance is met, both the wire and catheter must be removed as a single unit and the procedure reattempted. A common cause of a "stuck wire" is a small piece of adipose tissue wedged between the wire and the lumen of the catheter. This problem can be avoided by creating a deep enough skin nick and adequate dilation of the tract before insertion of the catheter.

Placement of multiple lumen catheters requires identification of the distal lumen and its corresponding hub. The distal lumen is found at the very tip of the catheter. The corresponding hub is usually labeled "distal" by the manufacturer. If there is any confusion, a small amount of sterile saline can be injected through each hub until it is observed exiting the distal lumen. Once the distal hub is identified, its cover cap is removed to allow passage of the guidewire (remember to replace this or immediately begin infusing saline upon completion of placement). The catheter is placed by threading the guidewire into the distal lumen and advancing it until it protrudes from the hub. At this point the device is placed in the same manner as a single-lumen catheter. If a soft multiple-lumen device is placed, a separate dilator is used to create a tract over the guidewire prior to placing the catheter. An alternate method of placing multiple-lumen catheters is to thread the catheter through a standard Desilets-Hoffman sheath introducer system. Any lumen in a multiple-lumen device that is not immediately used for an infusion must be flushed with a heparin solution. Saline solutions alone should not be used as flushes for central catheters.

The depth of catheter insertion is an important consideration. The SVC begins at the level of the manubriosternal junction and terminates in the right atrium, approximately 5 cm lower. For lines placed in the subclavian, jugular, basilic, and cephalic systems, the proper position of the catheter is in the SVC, not the right atrium or ventricle. Therefore, the catheter should be threaded approximately 2 cm below the manubriosternal junction. Many commonly used catheters are long enough to reach the atrium or ventricle. For example, the standard catheters marketed for subclavian venipuncture are often 20 to 30 cm long. Twenty cm from the skin insertion site is more than sufficient to reach the SVC for a subclavian catheter placed in the average adult male. Catheters 15 to 16 cm in length are recommended to avoid unintended placement to an excessive depth. The proper distance to advance the catheter can be estimated by placing the catheter parallel to the chest wall before insertion. Alternatively, formulas have been developed to determine optimal insertion length based on patient's height. One set (Table 22-5) was found to yield accurate placement in the SVC on 95% of 228 attempts. [58] Proper placement should always be confirmed by a postprocedure chest radiograph.

Many different catheters are currently manufactured. Although this leads to great flexibility in choice and cost, it often leads to confusion when a clinician is handed an unfamiliar catheter during an emergency. It is best to use one

TABLE 22-5 -- Formulas for Catheter Insertion Length Based on Patient Height (in cm) and Approach

Site	Formula	In SVC, %	In RA, %
RSC	(Hgt/10)-2 cm	96	4
LSC	(Hgt/10) +2 cm	97	2
RIJ	Hgt/10	90	10
LIJ	(Hgt/10) + 4 cm	94	5

Hgt = Patient height

SVC = Superior Vena Cava

RA = Right Atrium

RSC = Right Subclavian

LSC = Left Subclavian

RIJ = Right Internal Jugular

LIJ = Left Internal Juglar

From Czepizak C, et al: Evaluation of formulas for optimal positioning of central venous catheters. Chest 107:1662, 1995. (Reproduced by permission.)

brand routinely and to ensure that all medical personnel are thoroughly schooled in its use.

Replacement of Existing Catheters

In addition to placing new catheters, the guidewire technique can be used to change existing devices. Central veins are often cannulated in seriously ill patients who will require subsequent pulmonary artery wedge pressure monitoring, transvenous pacemaker placement, or placement of a different catheter. Any CVP catheter that is initially inserted should have a lumen large enough to accept a guidewire, facilitating conversion to a different catheter. The guidewire technique can also be used to change single-lumen central venous catheters to triple-lumen or sheath-introducer sets. Not all commercially available CVP catheters will accept a guidewire.

Replacement of an existing catheter begins with selection of a guidewire longer than either of the devices to be exchanged. Using sterile technique, the guidewire is inserted into the existing central venous catheter until a few centimeters are protruding and from the proximal end. With one hand holding the wire securely, the catheter and wire are removed as a single unit until the tip of the catheter just clears the patient's skin. The wire is grasped at the point at which it exits the skin, and only then is the other end of the wire released. The catheter is then slid off the wire, and the new device is inserted in the normal fashion. Caution must be exercised with this technique because catheter embolization can occur if a catheter is cut to allow use of a shorter guidewire for the exchange. [54] In patients without evidence of sepsis, the technique of guidewire exchange does not increase the incidence of catheter-related infections. However, in septic patients this maneuver does appear to increase infections related to central catheters. [59]

Over-the-Needle Technique

An optional method for cannulation uses percutaneous placement of an over-the-needle catheter. Over-the-needle devices (such as the Angiocath) use a tapered plastic catheter that passes through the vessel wall into the lumen using the needle tip as a guide. There are advantages to this system. The catheter does not pass

through a sharp needle, and the risk of shearing with resultant catheter embolization is thus decreased. Also, the hole made by the needle in the vessel wall is smaller than the catheter, producing a tighter seal. The internal jugular and subclavian vein via the SC approach are the most popular and appropriate targets for this approach. These devices are used primarily when rapid central venous access is required (e.g., during a cardiac arrest). The catheters are not suitable for high-volume fluid resuscitation, and they are too small for passage of a pacemaker lead. Once the clinical situation stabilizes, they may be exchanged for a larger central catheter via the Seldinger technique.

Insertion is straightforward, and should be preceded by quickly sterilizing the patient's skin (see later in this section). Longer peripheral-type catheters (such as a 16-ga 5 ¼-in. Angiocath) are required in adults. Smaller diameter devices, such as 20-ga catheters, may be easier to pass but also provide lower infusion rates. The needle is attached to a syringe, and very slowly advanced into the vein with steady negative pressure applied to the syringe. This may be difficult due to the longer length of the needle relative to the catheter. With over-the-needle catheters, the needle extends a few millimeters past the tip of the catheter. Blood return will be obtained when the tip of the needle is in the vein, while the catheter may actually be outside the lumen. If the needle is withdrawn before the catheter is advanced, the catheter tip will remain outside the vein. So, after the venous flash, the needle should be advanced a few millimeters and then held steady while the catheter is easily advanced into the vein. The catheter should then be secured and placement verified as detailed later in this chapter.

SPECIFIC VESSEL ACCESS TECHNIQUES

Strict adherence to the principles of sterile technique, including antiseptic skin preparation, sterile gloves and sterile procedural fields, is believed to reduce septic complications. Violation of these principles for the sake of speed is seldom justified. The few extra seconds required to put on gloves and to swab and drape the field will rarely make a critical difference in patient survival outside of the cardiac-arrest resuscitation scenario. It is recognized that the ideal practice of aseptic technique often cannot take place during resuscitation scenarios. For this reason, all central lines placed in an unsterile setting should be replaced at the earliest possible opportunity.

The area of the needle puncture should be widely prepared with povidone-iodine solution. In iodine-allergic patients, pHisoHex or Hibiclens are acceptable alternatives. If subclavian or IJ approaches are planned, the prepared area should include puncture sites for the IC and SC subclavian and IJ approaches. This permits the clinician to change the site following an unsuccessful attempt without repeating the preparation. In this circumstance, a standard preparation should include the ipsilateral anterior neck, the supraclavicular fossa, and the anterior chest 3 to 5 cm past the midline and the same distance above the nipple line. Preparation for femoral access includes shaving the groin or trimming groin hairs (preferred) as needed, followed by application of povidone-iodine solution to cover an area the breadth of, and extending 10 cm above and below, the inguinal ligament.

A significant decrease in the rate of catheter contamination has been observed by inserting the catheter through a previously placed iodophor-impregnated sterile film after the usual sterile preparation and draping. [60] However, there is little evidence that aggressive aseptic techniques offer additional benefit. In one prospective trial, 137 patients received standard antiseptic skin preparation and were then randomized to one of two groups. Clinicians in the aseptic group prepared as for surgery, with a 5-minute scrub and full gown, hat, mask, and gloves, whereas the "nonsterile" clinicians did not necessarily even wash their hands or use sterile gloves. No difference in rate of contamination was seen, and the contaminating organism in 87.1% of the overall cases was *Staphylococcus epidermidis*.[61] The need to perform an operating suite-style preparation in patients receiving hyperalimentation is also unproved. In a study of 63 patients with long-term subclavian catheterization for hyperalimentation, a simple iodophor spray preparation was associated with only 1 infection. [25]

Each approach to central venous cannulation is described separately in the following section. It is assumed that proper sterile procedure and any needed local anesthesia will be

provided. As in any invasive task, the procedure should be described briefly to awake patients, and each step should be restated as it is about to be performed. After the following descriptions of the common approaches to the central veins, puncture site care, placement verification, and other adjuncts to the procedure will be summarized.

Subclavian Vein, Infraclavicular Approach

Descriptions of subclavian venipuncture often unduly focus on angles and landmarks and overstate the effects of patient positioning. The most important factors governing success or failure are knowledge of anatomy and meticulous attention to the details of the procedure. [62]

Positioning.

The patient is placed in the supine position with the head in a neutral position and the arm abducted. Placing the patient in the Trendelenburg position (10 to 15 degrees) decreases the risk of air embolism. The claim that this position distends the vein is controversial. [63] Venographic studies show no change in the diameter of the subclavian vein associated with the Trendelenburg position, although these observations were limited to normovolemic patients. [64] Further, the vessel is hemmed by the semirigid costoclavicular ligament on its anteroinferior aspect and therefore will not distend in a direction that facilitates IC venipuncture. [12] Magnetic resonance imaging clearly demonstrates that the caliber of the subclavian vein is determined by its attachment to adjacent structures and is not affected by the CVP, although another study did detect mild dilation of the subclavian vein on B-mode ultrasound of normal subjects during Trendelenburg positioning. [65] [66]

Abduction of the arm has been recommended to flatten the deltoid bulge.[67] This is sometimes a helpful maneuver in muscular individuals but is not generally necessary. Abduction moves medially the point at which the subclavian vein passes beneath the clavicle. [64] Turning the head to the opposite side has no effect on the vessel size or on the relative positions of the vessel and the clavicle. [64] [68] This maneuver does change the relative positions of the subclavian and IJ vein and has been postulated to cause an increased incidence of catheter malposition in the jugular vein. [65] Placing a pillow under the back is commonly recommended to make the clavicle more prominent, but as the shoulder falls backward, the space between the clavicle and first rib narrows, making the subclavian vein less accessible. [65] Significant compression of the subclavian vessels between these bony structures occurs as the shoulders retract. [65] Based on the findings of seven cadaver dissections, Tan and colleagues recommend that the shoulders be in a neutral position with mild shoulder retraction. [69] The clinical case series of Boyd et al. also suggest that a small "bump" between the shoulder blades may enhance IC subclavian cannulation. [70]

Venipuncture site.

The right subclavian vein is usually cannulated because of the lower pleural dome on the right and because of the need to avoid the left-sided thoracic duct. The anatomically more direct route between the left subclavian vein and the SVC is a theoretic advantage of left-sided over right-sided subclavian venipuncture. However, it has not been proved that there is a higher incidence of catheter malposition when the right IC approach is used.

In the conscious patient, the point of needle entry is anesthetized with 1% lidocaine. SQ infiltration of the periosteum of the clavicle will make the procedure less painful but is not always necessary. Opinions vary as to the best point of needle entry. The junction of the middle and medial thirds of the clavicle is the standard site. There the vein lies just posterior to the clavicle and just above the first rib, which acts as a barrier to penetration of the pleura. This protective effect is lost when a more lateral location is chosen. Approaches include entry just laterally and inferiorly to the junction of the clavicle and the first rib, with the needle aiming at this junction, and entry at the site of a small tubercle in the medial aspect of the deltopectoral groove. [71] [72] In our opinion, the point of entry is less important than the direction taken by the needle after entry. Points lateral to the mid-clavicle should be avoided, because this location requires a deeper puncture and potentially increases the risk of pneumothorax.

Needle orientation.

The *needle bevel should be oriented inferomedially* for the IC approach to direct the catheter toward the innominate vein rather than toward the opposite vessel wall or up the IJ vein (Fig. 22-11). Alignment of the needle bevel with markings on the barrel of the syringe permits awareness of bevel orientation after skin puncture. Some investigators advise puncturing the skin with a No. 11 scalpel blade to avoid skin plugs in the needle. Others suggest filling the syringe with 2 to 3 mL of 1% plain lidocaine to both anesthetize the SQ tissue and flush the needle.

Before insertion of the needle, the left index finger is placed in the suprasternal notch and the thumb is positioned at the costoclavicular junction (Fig. 22-12). These serve as reference points for the direction of needle travel. The needle is aimed immediately above and posteriorly to the index finger. Vessel entry, signaled by a flashback of dark venous blood, usually occurs at a depth of 3 to 4 cm. If the needle tip is truly intraluminal, there will be free flow of blood. The return of pulsatile flow signifies arterial puncture. A single arterial puncture without laceration rarely causes serious harm. Use of this technique eliminates the need to measure angles, to "walk" the clavicle, or to concentrate excessively on maintaining the needle parallel to the chest wall. All of these techniques are based on fear of complications rather than knowledge of anatomy.[68] One should avoid any sweeping motions of the needle tip to prevent unseen injuries. In patients who are being ventilated with positive pressure, it is advisable to halt ventilation for a moment as the needle penetrates the chest wall. Interruptions should be kept to a minimum and should not exceed the

30-second standard.[73]

Unsuccessful attempts.

Cannulation of the subclavian vein may not be successful on the first attempt. It is reasonable to try again, but after three or four unsuccessful attempts, it is best to try another approach or allow a colleague to attempt the procedure. One must use a new setup each time blood is obtained, because clots and tissue will clog the needle and mislead the clinician even if subsequent procedures are performed correctly. If several attempts are made, the admitting clinician or anesthesiologist must be informed so that proper precautions are taken to identify subsequent complications. It is advisable to obtain radiographs of the chest even after unsuccessful attempts. If the initial puncture site was properly placed, then for aesthetic reasons, one should use the same needle hole for subsequent attempts (i.e., one should avoid an embarrassing pincushion appearance of the upper chest). If the subclavian route is unsuccessful on one side, it is best to attempt an IJ catheterization on the same side rather than attempt a subclavian cannulation on the opposite side. In this manner, bilateral complications are avoided.

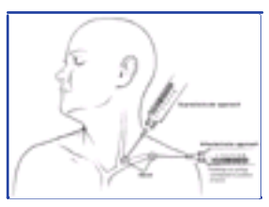

Figure 22-11 Needle bevel orientation using supraclavicular and infraclavicular venipuncture. The orientation of the needle bevel may help in positioning the catheter properly by guiding the direction of the catheter during advancement. If the bevel is aligned with the markings on the syringe, the orientation of the bevel is always certain.

Subclavian Vein, Supraclavicular Approach

Positioning.

The goal of the SC technique is to puncture the subclavian vein in its superior aspect just as it joins the IJ vein. The needle is inserted above and behind the clavicle, lateral to the sternocleidomastoid muscle. It advances in an avascular plane, away from the subclavian artery and the dome of the pleura. The right side is preferred because of the lower pleural dome, because it is the direct route to the SVC, and because the thoracic duct is on the left side. The Trendelenburg position may be helpful for distending the vein, because the

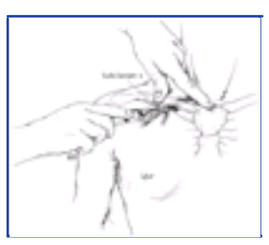

Figure 22-12 Hand position during subclavian venipuncture. Note that surgical gloves should be worn during this procedure, in contrast to the illustration. *(From Linos D, Mucha P, von Heerden J: Subclavian vein: A golden route. Mayo Clin Proc 55:318, 1980. Reproduced by permission.)*

subclavian vein is not bound by fasciae on its superior aspect. [28] The patient's head may be turned to the opposite side to help identify the landmarks. However, Jung and colleagues found that tilting the head toward the catheterization site during catheter passage reduced catheter malposition in children. [74]

Needle orientation.

After the area of the supraclavicular fossa has been prepared and draped, a point is identified 1 cm lateral to the clavicular head of the sternocleidomastoid and 1 cm posterior to the clavicle (Fig. 22-13). Alternatively, the junction of the middle and medial thirds of the clavicle has been used as the landmark for needle entry with good success in a cadaveric study.[75] In either case, the area is anesthetized with 1% lidocaine. If a 3-cm-long needle is used for anesthesia, it may also be used to locate the vessel in a relatively atraumatic manner. The subclavian vein can almost always be located with this needle because of the vein's superficial location and the absence of bony structures in the path of the needle. A 14-ga needle (or 18-ga thin-walled needle) is then advanced, following the path of the scout needle; gentle negative pressure is applied using an attached syringe.

When seeking the subclavian vein, the needle is aimed so as to bisect the clavicosternomastoid angle, with the tip pointing just caudal to the contralateral nipple. The *bevel is oriented medially* to prevent catheter trapping against the inferior vessel wall. The tip of the needle is pointed 10 degrees above the horizontal. Successful vessel puncture generally occurs at a depth of 2 to 3 cm.

Internal Jugular Approach

Positioning.

In preparation for all 3 IJ approaches, the patient is tilted 15 to 30 degrees in the Trendelenburg position and the head is turned slightly away from the side of venipuncture. The IJ vein is distensible, and tilting the patient increases the diameter of the vessel. If the patient is awake, he or she should be instructed to perform a Valsalva maneuver during

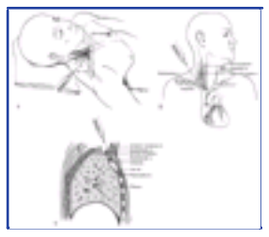

Figure 22-13 *A* and *B*, For the supraclavicular approach, the needle is inserted above and behind the clavicle, bisecting the angle made by the clavicle and the lateral border of the sternocleidomastoid muscle (clavisternomastoid angle). The point of entry is 1 cm lateral to the clavicular head of the muscle and 1 cm posterior to the clavicle. The needle traverses an avascular plane, puncturing the junction of the subclavian and internal jugular vein behind the sternoclavicular joint. The right side is preferred because of a direct route to the superior vena cava and the absence of the thoracic duct. The needle is directed 45° from the sagittal plane and 10° to 15° upward from the horizontal plane, aiming toward the contralateral nipple. Note that the vein is just posterior to the clavicle at this juncture. *C*, Sagittal view of the supraclavicular area. As the subclavian vein passes over (and somewhat anterior to) the first rib, it is separated from the subclavian artery by the anterior scalene muscle. The dome of the pleura is posterolateral to the confluence of the great veins.

vessel cannulation. In the unconscious patient, abdominal compression by an assistant can be used to help distend the vein.

Venipuncture site.

Familiarity with the anatomy of the neck is important to increase the probability of successful cannulation and to minimize complications. Most authors favor cannulation of the right side of the neck, which provides a more direct route to the SVC and avoids the thoracic duct. Although it is probably clinically insignificant, the cupola of the pleura is also slightly lower on the right side. The left IJ approach is more circuitous and, when used with a stiff Teflon catheter, may result in a major

venous puncture leading to hydrothorax, hydromediastinum, or even pericardial tamponade. [76] [77] [78] [79]

Central route.

This approach is favored by some, who believe that the incidence of cannulation of the carotid artery is decreased and the cupola of the lung is avoided with this method.[80] The triangle formed by the clavicle and the sternal and clavicular heads of the sternocleidomastoid is first palpated and identified. A local anesthetic skin wheal or a marking pen can mark the lateral border of the carotid pulse, and all subsequent needle punctures can be performed laterally to that point.

Some practitioners prefer to attempt cannulation with the catheter apparatus initially. Others use a small-gauge "locator" or scout needle to identify the vein. The smaller needle allows

one to ascertain the location of the vein and minimizes injury to deep structures by an incorrectly placed larger needle. Use of a locator needle can be time consuming in a cardiac arrest situation.

When using the scout needle technique, a 22-ga, 3-cm needle attached to a 5- to 10-mL syringe is introduced near the apex of the triangle and is directed caudally at an angle 30° to 40° to the skin. The needle should initially be directed parallel and slightly laterally to the course of the carotid artery (Fig. 22-14). If three fingers are lightly placed over the course of the carotid artery, the parallel course of the IJ vein can be estimated. The vein consistently lies just lateral to the carotid artery. Prolonged deep palpation of the carotid artery may decrease the size of the vein, and the three-finger technique should be used only long enough to identify the course of the artery.

Negative pressure should be maintained on the syringe at all times as the needle is advanced or retracted. The vein is more superficial than might be expected, and deep probing with the needle should be avoided; the vein is usually encountered at a depth of 1.0 to 1.5 cm. If the IJ vein is not entered at a depth of 3 to 5 cm, the needle should be withdrawn to just below the skin surface and directed toward the ipsilateral

Figure 22-14 Internal jugular vein approaches. *A*, Central approach. *B*, Posterior approach. *C*, Anterior approach. Note that contrary to the photographs, gloves should be worn during the procedure.

nipple underneath the medial border of the lateral (clavicular) head of the sternocleidomastoid. *During any type of central venous cannulation, a needle should always be withdrawn to the surface before being redirected to avoid lacerating or otherwise damaging important nerves, vessels, or other structures.*

The vein should be entered at 1 to 3 cm, and dark blood should be easily aspirated (bright red blood indicates carotid artery penetration and the need for needle repositioning). After locating the IJ vein, the locator needle is withdrawn and replaced with a 14-ga, 5-cm needle attached to a syringe. A drop of blood from the locator needle can be placed at the edge of the sterile field in line with the point of vessel entry, thus serving as a guide to recannulation. The larger needle is advanced through the skin along the path determined by the smaller needle until blood is aspirated. Care must be taken to cover the needle hub with a gloved thumb whenever the needle lumen is exposed to air. This practice will prevent an air embolus when the patient inspires.

The central approach has been used in children with good success [37] [81] (see Chapter 19). However, success is greater with larger infants (>10 kg) and those with higher CVPs (>10 cm H_2O). With infants and children, needle puncture

occurs at the apex of the triangle bordered by the two heads of the sternocleidomastoid muscle and the clavicle. Similar to the approach for adults, one passes a 22-ga needle attached to a 2- to 5-mL non-Luer-Lok syringe into the skin at a 45-degree angle and directs it caudally and laterally toward the ipsilateral nipple. The vessel is usually entered at a depth of 1 to 2 cm. The locator needle is then withdrawn, and a 17- to 19-ga needle is inserted into the skin until the IJ vein is penetrated.

Posterior and anterior routes.

For the *posterior approach*, the skin is entered at the lateral edge of the sternocleidomastoid muscle one third of the way from the clavicle to the mastoid process (see Fig. 22-14). The locator needle is directed caudally and medially toward the sternal notch until blood is aspirated.

To perform the *anterior approach,* the course of the carotid is identified and marked by the index and middle fingers (see Fig. 22-14).[82] The small needle should then enter the skin at the midpoint of the medial aspect of the sternocleidomastoid muscle. The needle is directed at an angle of 30 to 45 degrees to the coronal plane caudally toward the ipsilateral nipple. The proximity of the carotid artery in the anterior approach may prohibit venous cannulation without carotid puncture. [49] Doppler ultrasound can facilitate difficult IJ cannulation, a matter discussed in more detail later and in Chapter 69 .[83]

External Jugular Vein Approach

Positioning.

With the patient in the Trendelenburg position, the EJ vein is distended by instructing the patient to perform a Valsalva maneuver and then tamponading the vein just cephalad to the clavicle with a finger.

Venipuncture.

The vein is approached from the side while slight traction is placed on the vein to stabilize it. The needle is advanced at a small angle from the skin plane (about 10 degrees) until the operator feels it "pop" into the lumen of the vein. The needle or catheter should be advanced slightly after feeling the pop to ensure intraluminal placement. As discussed later, use of the EJ vein as a site for central vein access requires the use of a guidewire.

Femoral Vein Approach

Positioning and needle orientation.

The patient must be supine. The femoral pulse is identified and the scout needle (at least 4.4 cm length) is inserted at 45 degrees to the skin in a cephalic direction just medial to the femoral pulsation. Because CPR can produce *venous pulsations,* unsuccessful venous aspiration medial to the pulsations should be followed by an attempt directly over the pulsations.

Venipuncture.

During needle advancement, negative pressure is maintained within the syringe at all times while the needle is under the skin. The needle is directed posteriorly and advanced until the vein is entered, as identified by a flash of dark, nonpulsating blood. If the vessel is penetrated when the syringe is not being aspirated, the blood flash may be seen only as the needle is being withdrawn. The femoral vein lies just medial to the femoral artery at the level of the inguinal ligament. It is closer to the artery than many clinicians appreciate. As the vein progresses distally in the leg, it runs closer to, and almost behind, the femoral artery (see Fig. 22-3). This anatomical fact should be considered if the cannulating needle is introduced more than a few centimeters distal to the inguinal ligament.

Basilic and Cephalic Approach

Venipuncture.

The basilic and cephalic venous systems are entered through the large veins in the antecubital fossa (see Fig. 22-4). Tourniquet placement aids venous distention and initial venous puncture. When veins are not visible, they may be reached with a cutdown procedure, as described in Chapter 23 . The basilic vein, located on the medial aspect of the antecubital fossa, is generally larger than the radially located cephalic vein. Furthermore, the basilic vein generally provides a more direct route for passage into the axillary vein, subclavian vein, and SVC.

CATHETER PASSAGE TECHNIQUE

Once there is a venous flashback into the syringe, it is detached from the needle, and a catheter is passed either via the Seldinger technique or, alternatively, over or through the needle. Removing the syringe must be done with care to avoid dislodging the needle tip from the lumen of the vein. If the syringe is tightly attached to the needle, a hemostat may be used to grasp and secure the needle hub during removal of the syringe. Needle tip displacement may also occur if blood specimens are drawn at this time. Hence, it is best to delay blood sampling until the catheter has been advanced. The needle hub should be occluded with the thumb to avoid air embolism.

SPECIAL CONSIDERATIONS FOR THE FEMORAL AND SMALLER VESSELS

Femoral Vein Approach

Once in the femoral vein, the needle is stabilized; often, a hemostat is helpful for holding the needle during removal of the syringe. A premeasured section of a 90-cm catheter may then be inserted using a through-the-needle system. One determines the appropriate length by holding the catheter over the patient's body and estimating the distance from the skin puncture site to the right atrium. Contamination of the catheter must be avoided while this maneuver is performed. Once the catheter is placed, it is secured with sutures and dressed in the same manner as other central lines.

In situations requiring rapid volume infusion, in the absence of intra-abdominal trauma, the femoral vein may be cannulated with a sheath introduced via the guidewire technique. The introducer will allow rapid transfusion of large volumes of blood or crystalloid solution for fluid resuscitation. The femoral vessels may also be cannulated under direct visualization using a cutdown technique (see Chapter 23).

External Jugular Vein Approach

Central venous catheterization via the EJ vein is time consuming and often difficult. Use of the EJ vein for achieving

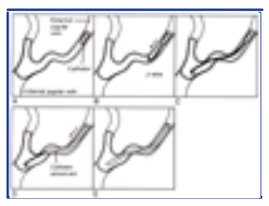

Figure 22-15 Insertion of a catheter over a wire via the external jugular vein. Successful passage may require many attempts and manipulations of the J wire to navigate turns and valves. *(From Blitt CD, Wright WA, Petty WC: Central venous catheterization via the external jugular vein, a technique employing the J-wire. JAMA 229:817, 1974. Reproduced by permission.)*

central venous access requires use of a guidewire. After cannulation of the vein and intraluminal placement of the guidewire, the guidewire is advanced into the thorax by rotating and manipulating the tip into the central venous circulation (Fig. 22-15). Guidewire advancement is the most difficult and time-consuming portion of the procedure, and this time constraint limits the usefulness of the technique in an emergency. A small-radius J-tipped wire, a distended vessel lumen, and exaggeration of patient head tilt, coupled with skin traction, may facilitate successful guidewire passage. Partially withdrawing the wire and twisting it 180° before readvancing the tip may also be helpful.

Basilic and Cephalic Approach

Passage of a catheter into the central circulation is difficult using the basilic and cephalic routes and failure is common. The cephalic vein may terminate inches above the antecubital fossa or bifurcate before entering the axillary vein, sending a branch to the EJ vein. The cephalic vein may also enter the axillary vein at right angles, defeating any attempt to pass the catheter centrally. Furthermore, both the basilic and cephalic systems contain valves that may impede catheterization. Abduction of the shoulder may help to advance the catheter if resistance near the axillary vein occurs. The incidence of failure to place the catheter in the SVC ranges from a high of 40% to a low of 2%.[40] [84] The greatest success rate (98%) reported was obtained with slow catheter advancement with the patient in a 45° to 90° upright position. [40] Flexible catheters were introduced into the basilic vein until the tip was judged to be proximal to the junction of the cephalic and basilic veins and distal to the junction of the IJ vein with the innominate vein. The wire stylet was withdrawn 18 cm, and the catheters were advanced slowly 1 cm at a time, with 2 seconds allowed between each 1-cm insertion. The natural flexibility of the Bard catheters contributed to negotiation into the SVC when the patient was upright. This time-consuming technique is contraindicated when the patient cannot tolerate an upright position.

ASSESSING LINE PLACEMENT

Once the catheter has been passed, it must be carefully secured. All tubing and connections should first be checked for tightness to prevent air embolism, fluid loss, or bleeding. The technique for securing a catheter depends on the type of equipment and the site of puncture. In general, all catheters should be secured with sutures (or skin staples[85]) and a sterile dressing placed. Most systems have some type of anchoring device to simplify securing of the catheter. Since dressings are inspected and changed periodically, it is prudent to place a simple dressing, avoiding excessive amounts of gauze and tape. An effective method for securing an IJ catheter is shown in Figure 22-16 . Care is taken to protect the skin against maceration. Transparent dressings made of polyurethane are popular and simple. They also yielded a lower rate of catheter colonization than newer hydrocolloid dressings in a prospective, randomized, controlled trial. [86]

Before the infusion of fluids, the IV fluid reservoir should be lowered below the level of the patient's right atrium and the line checked for backflow of blood. The free backflow of blood is suggestive, but not diagnostic, of intravascular placement. However, backflow may occur with a hematoma or a hemothorax if the catheter is free in the pleural space. A pulsatile blood column may be noted if the catheter has been inadvertently placed into an artery. Less pronounced pulsations may also occur if the catheter is advanced too far and reaches the right atrium or ventricle. Pulsations also may be noted with changes in intrathoracic pressure due to respirations, although these pulsations should be at a much slower rate than the arterial pulse. A final method of checking intravascular placement is to attach a syringe directly to the catheter hub and aspirate venous blood. It is also advisable to


Figure 22-16 Internal jugular line secured by looping around the ear. *(From Boulanger M, et al: Une nouvelle voie d'abord de la veine jugulaire interne. Can Anaesth Soc J 23:609, 1976. Reproduced by permission.)*

ensure that the catheter is easily flushed with a heparin solution, if no heparin sensitivity on the part of the patient exists. This carries the additional benefit of removing air from the system. Radiographs are also always indicated to verify catheter location and assess for potential complications, except for routine femoral line placements.

In an awake patient, infusing fluids via a catheter tip positioned in the internal jugular vein may produce an audible gurgling sound or flowing sound in the patient's ear.[87]

Radiographs

Following placement of lines involving puncture of the neck or thorax, the lungs should be auscultated to detect an inequality of lung sounds suggestive of a pneumo- or hemothorax. A chest film should be obtained as soon as possible, checking for hemothorax, pneumothorax, and catheter tip position. Because small amounts of fluid or air may layer out parallel to the x-ray plate with the patient in the supine position, the film should be taken in the upright or semi-upright position whenever possible. Proper catheter tip position is shown in Figure 22-17 . Misplaced catheters should be repositioned. In ill patients, a rotated or oblique projection on a chest radiograph may be obtained, and the clinician may be confused as to the proper position of the catheter (Fig. 22-18). In such cases, a repeat radiograph is necessary. A misplaced catheter tip is usually obvious on a properly positioned standard posteroanterior (PA) chest radiograph, but occasionally the injection of contrast material may be required. For example, a catheter in one of the internal thoracic veins may simply appear more lateral than expected, but because of the close proximity of these veins and the superior vena cava, malposition may not be appreciated by this subtle finding.

Postprocedure radiographs are not always warranted for routine *replacement* of catheters over guidewires. If such patients

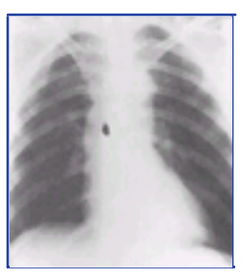
Figure 22-17 A chest film showing the proper catheter tip placement in the superior vena cava *(arrow)*. The tip should not lie within the right atrium or the right ventricle.

are stable and hemodynamically monitored, radiography may be safely deferred in the absence of apparent complications or clinical suspicion of malposition. [88] It is not standard to perform a radiograph following femoral line placement.

Redirection of Misplaced Catheters

Improper catheter tip position occurs commonly. It has been reported that only 71% of subclavian catheters are located in the SVC on the initial chest film. [122] Complications of improper positioning include hydrothorax, hemothorax, ascites, [123] chest wall abscesses,[124] embolization to the pleural space, [125] and chest pain.[126] More commonly, improper location yields inaccurate measurements of the CVP or is associated with poor flow caused by kinking. [127] An unusual complication caused by improper tip position is cerebral infarction, which can occur following inadvertent cannulation of the subclavian artery. [128]

Misdirection or inappropriate positioning of the tip of a central venous catheter is not uncommon. These events if promptly recognized and corrected represent inconsequential complications. Loop formation, lodging in small neck veins, tips directed caudally, and innominate vein position are common problems. Misplaced catheters should be repositioned as soon as logistically possible. If the catheter is being used for fluid resuscitation, the malposition may be tolerated for some time. If vasopressors or medications are infused, it is more critical to properly position the catheter tip. A number of options are available to remedy malpositioning. One strategy is to insert a 2 Fr Fogarty catheter through the lumen of the central line, advancing it 3 cm beyond the tip. The entire assembly is withdrawn until only the Fogarty catheter is in the subclavian vein. One milliliter of air is injected into the balloon, and the Fogarty catheter is advanced. It is hoped that the

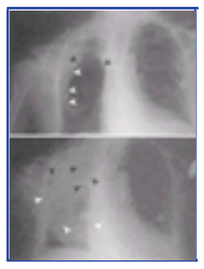

Figure 22-18 A chest radiograph should be routinely taken to assess position of a central catheter introduced via the chest or neck. *A*, In this case a poorly positioned patient produced a rotated and oblique film, and the catheter (black arrow) appeared, at first glance, to be in the correct position in the right subclavian vein. The supine position of the patient did not allow identification of an early hydrothorax (white arrow). *B*, A repeat radiograph shows the obvious intrapleural position of the catheter, and a large hydrothorax after infusion of 2 liters of saline.

blood flow will direct the assembly into the SVC. The balloon is deflated and the central line is advanced over the Fogarty catheter, which is then withdrawn. [89]

One anecdotal strategy is to withdraw the catheter until only the distal tip remains in the cannulated vessel. This measurement is best appreciated by comparing the indwelling catheter length with another unused catheter. The clinician then simply readvances the catheter, hoping that it becomes properly positioned. Other manipulations with guidewires have been suggested, but often reinsertion with another puncture is required for the misplaced catheter to be positioned properly.

ULTRASOUND-GUIDED CENTRAL VENOUS ACCESS

In maximizing the success and minimizing the complications of central venous access, there is no substitute for experience. However, some patients will present challenges to even the most seasoned clinician. Anatomic (skeletal or vascular) abnormalities, whether congenital or acquired, may be encountered and can thwart successful cannulation. When time permits, difficult cases may be simplified by using a Doppler ultrasound device to identify the location of major veins. The course of these vessels can be marked on the skin surface and used as an anatomic guide during needle placement. Alternatively, more sophisticated ultrasound imaging systems have been adopted for guiding venous cannulation.

The most commonly described imaging tool is a handheld 7.5 MHz real-time mechanical sector transducer with an attached needle guide. The device is coupled with a small video display (Fig. 22-19). To use the instrument, the nonsterile transducer is covered with acoustic coupling gel and placed inside a sterile sheath. Additional sterile gel is placed on the skin over the site being imaged, and the unit is used to determine the location, orientation, and diameter of the target vessel. When accessing the jugular or femoral systems, this is done by placing the transducer according to traditional puncture site landmarks. Imaging the subclavian vein from the IC approach is more difficult, but can be attempted by identifying the axillary vessels at their most proximal position under the distal clavicle and then following the vessels medially as they course beneath the clavicle. [90] Once the vessel is identified, the overlying skin may be marked for later venipuncture or a needle and syringe secured to the transducer for immediate cannulation. Under ultrasound guidance, the needle is advanced through the skin and SQ tissue toward the target vessel. Once the needle is in close proximity to the vessel, one will see compression of the vein. Once the vessel wall has been penetrated, the vein will refill with blood and assume its original shape. The transducer can then be detached and cannulation proceeds.

Most prospective analyses of the device have examined IJ vein cannulations [91] [92] [93] and have uniformly suggested advantages to the technique. Ultrasound-guided attempts demonstrated greater overall success, as well as an increased rate of successful first punctures. Clinically significant complications were too infrequent to definitely conclude greater patient safety with the ultrasound technique; studies specifically examining complications demonstrated less frequent hematoma formation and inadvertent arterial puncture. [90] [91] Ultrasound guidance may assume greater prominence in central venous access in the future. However, given the generally favorable success rate and low complication rate of traditional access techniques, the relative infrequency of central venous cannulation in many EDs, and the considerable cost of the device, the routine role of ultrasound imaging guidance remains uncertain.

COMPLICATIONS

The medical literature is replete with reports of the complications of large vein venipuncture. Some are minor and inconsequential, such as hematoma formation, while others are serious and life-threatening, such as hemothorax. *No clinician can expect to routinely perform these procedures and be complication-free.* Common complications for the different approaches are summarized in Table 22-6 and Table 22-7 . Key injuries categorized by organ system and by approach are discussed in the sections that follow. The FDA has released a three-volume video entitled "CVC Complications," which was sent to all hospitals where such catheters are placed. It is also commercially available from the Internet (at www.fda.gov).

Published rates vary widely and complication rates depend on one's definition. One 3-year retrospective review

Figure 22-19 *A*, Surface ultrasound-directed central vein identification. A handheld transducer allows noninvasive localization of veins, in this case the subclavian. The device shown includes a needle guide, which allows simultaneous visualization and penetration of a targeted vessel. *B,* Surface ultrasound image of the subclavian artery (left) and vein (right). *(Courtesy of Irene R. Skolnick, Dymax Corp, Pittsburgh.)*

of all central catheters placed in the ED (SC, IJ, and femoral lines) reported a mechanical complication rate of 3.5%, or 22 of 643 lines placed. [94] Complication was defined as pneumothorax, hematoma, line misplacement, hemothorax, or any issue with the CVC (excluding infection or thrombosis) that required an inpatient consultation. In general, failure and complication rates increase as the number of percutaneous punctures increase.

Pulmonary Complications

Pulmonary complications of subclavian and internal jugular venipuncture include pneumothorax, hemothorax, hydrothorax, hemomediastinum, hydromediastinum, tracheal perforation, and endotracheal cuff perforation. Pneumothorax is the most frequently reported complication, occurring in up to 6% of subclavian venipunctures. [95] Initially the importance of this complication was minimized, but reports of fatalities caused by tension pneumothorax, bilateral pneumothorax, and combined hemopneumothorax followed. [96] [97] One would expect a higher incidence of pneumothorax if the procedure were performed during CPR or positive-pressure ventilation. A small pneumothorax can quickly become a life-threatening tension pneumothorax under positive-pressure ventilation.

The treatment of a catheter-induced pneumothorax is controversial, but certainly not all patients will require a formal tube thoracostomy. [98] Some authors conclude that many stable outpatients exhibiting a pneumothorax following central venous catheter insertion can often be successfully managed with observation alone (60% in their series) or catheter (pigtail/Heimlich valve) aspiration, reserving large tube thoracostomy for refractory cases or emergent settings (Fig. 22-20). Critically ill patients, or those on mechanical ventilation will likely require invasive treatment of a catheter-induced pneumothorax.

Hemothorax may occur following subclavian vein or artery laceration, pulmonary artery puncture, or intrathoracic infusion of blood. Hydrothorax occurs as a result of infusion of IV fluid into the pleural space. Hydromediastinum is an uncommonly reported complication that is potentially fatal. [99]

Vascular/Bleeding Complications

The most common vascular complication is inadvertant artery puncture. This is usually easily recognized and controlled with simple compression. Rarely an artery is lacerated to an extent that bleeding is significant and arterial repair is necessary. In cardiac arrest, low flow, or shock states, arterial puncture may not be obvious, and arterial cannulation and the intra-arterial administration of medications has occurred. When the systolic blood pressure rises, arterial pulsations become more obvious. In critically ill patients, however, this complication may escape detection for some time. The subsequent development of ischemia or thrombosis of an artery that has been cannulated or injected with detrimental medication reflects the blind nature of this procedure in an emergency.

Air embolism is a very rare, but potentially serious complication from any central venous cannulation. Undoubtedly, minor and clinically inconsequential amounts of air enter the venous circulation during many cannulation procedures. Maintaining constant occlusion (with the operator's finger) of all needles that are located in central veins will minimize this occurrence. A 14-ga needle can transmit 100 mL of air per second with a 5-cm H_2O pressure difference across the needle. [100] Air embolism may occur if the line is open to air during catheterization or if it subsequently becomes disconnected. The recommended treatment is to place the patient in the left lateral decubitus position to relieve air bubble occlusion of the right ventricular outflow tract. [101] If this is unsuccessful, aspiration with the catheter advanced into the right ventricle has been

TABLE 22-6 -- Complications of Central Venous Access

General
Vascular
Air embolus
Adjacent artery puncture
Pericardial tamponade
Catheter embolus
Arteriovenous fistula
Mural thrombus formation
Large vein obstruction
Local hematoma
Infectious
Generalized sepsis
Local cellulitis
Osteomyelitis
Septic arthritis
Miscellaneous

Dysrhythmias	
Catheter knotting	
Catheter malposition	
Subclavian and internal jugular approaches	
Pulmonary	
Pneumothorax	
Hemothorax	
Hydrothorax	
Chylothorax	
Hemomediastinum	
Hydromediastinum	
Neck hematoma and tracheal obstruction	
Tracheal perforation	
Endotracheal cuff perforation	
Neurologic	
Phrenic nerve injury	
Brachial plexus injury	
Cerebral infarct	
Femoral approach	
Intra-abdominal	
Bowel perforation	
Bladder perforation	
Psoas abscess	

advocated.[102] Emergent cardiothoracic surgical consultation may also be warranted.

Catheter embolization resulting from shearing of a through-the-needle catheter by the needle tip is a serious and generally avoidable complication. Embolization can occur when the catheter is withdrawn through the needle or if the guard is not properly secured. Because through-the-needle catheters are less commonly need, this problem is rare. However, when present, subsequent adverse events occur frequently following embolization and include arrhythmias, venous thrombosis, endocarditis, myocardial perforation, and pulmonary embolus.[54] The mortality rate in patients who did not have these catheters removed has been reported to be as high as 60%.[103] Transvenous retrieval techniques are usually attempted and followed by surgery if they are unsuccessful. [104] Entire guidewires may also embolize to the general circulation if the tip is not always secured by the operator.

Perforation or laceration of vascular structures may cause hemothorax, hemomediastinum, and volume depletion. These are rarely serious complications, but fatalities have been reported. Surgical repair is occasionally required. [105] Arteriovenous fistula formation has also been reported. [106]

Delayed perforation of the myocardium is a rare but generally fatal complication of central venous catheterization by any route. [107] [108] The presumed mechanism is prolonged contact of the rigid catheter with the beating myocardium.[109] The catheter perforates the myocardial wall and causes tamponade either by bleeding from the involved chamber or infusion of IV fluid into the pericardium. The right atrium is involved more commonly than the right ventricle. [76] All who insert such catheters or care for such patients, or both, should be aware of this deadly complication, which results in profound deterioration with hypotension, shortness of breath, and shock. Emergent echocardiography, pericardiocentesis, and operative intervention by a chest surgeon all may be required for patient salvage. This can also occur with misplacement of the central venous line in the pericardiophrenic vein.[110] Fortunately this complication is preventable by using a postinsertion chest film to confirm catheter tip position and repositioning any catheter if the tip is within the cardiac silhouette.

Catheter knotting or kinking may occur if the catheter is forced or repositioned or if an excessively long catheter is used. [111] The most common result of kinking is poor flow of IV fluids, although rare complications as severe as SVC obstruction caused by a kinked catheter have been seen. [112]

Thrombosis and thrombophlebitis occur rarely because of the large caliber and high flow rates of the vessels involved. [68] It is important to determine that the catheter tip rests in the SVC, especially during the infusion of irritating or hypertonic solutions. [112] Thrombi may also form secondary to prolonged catheter contact against the vascular endothelium. One autopsy study found a 29% incidence of mural thrombi in the innominate vein, SVC, and right ventricle of patients who had central lines in place an average of 8 days before death.[113] However, no complications were directly attributable to these small, firmly adherent thrombi.

Thoracic duct laceration is a frequently discussed complication of left-sided subclavian venipuncture; however, it is extremely uncommon, and has been reported only as a complication of internal jugular, but not subclavian, cannulation. [95]

Although poorly studied, it has been promulgated that patients with a coagulopathy may experience significant bleeding from central catheter placement, especially if arterial puncture/laceration has occurred. Traditionally, prophylactic blood component therapy (fresh frozen plasma, platelet infusions) has been suggested in patients with a coagulopathy prior to percutaneous placement of a central venous catheter. While intuitively reasonable, this concept has no support in the literature. Mumtaz et al. have challenged this concept as unproven and unnecessary, citing a 3% bleeding rate in coagulopathic patients who experienced only minor bleeding that could be controlled with digital pressure. Although central venous access may be safely performed in patients with underlying disorders of hemostasis, without correction of the coagulopathy, caution is urged. It would be prudent to target central access in patients with coagulopathies to areas of arterial compression. [42]

Infectious Complications

Infectious complications include local cellulitis, thrombophlebitis, generalized septicemia, osteomyelitis, and septic arthritis. [95] The incidence of septic complications varies from 0% to 25%.[114] [115] The frequency with which infectious complications are seen is directly related to the attention given to

TABLE 22-7 -- Anatomic Structures That Can Be Injured by Central Venous Cannulation

Structure	Anatomic Relation to Vein	Error in Procedure	Injury
Subclavian Vein Cannulation			
Subclavian artery	Posterior and slightly superior, separated by scalenus anterior—10 to 15 mm in adults, 5 to 8 mm in children	Insertion too deep or lateral	Hemorrhage, hematoma, possible hemothorax

Brachial plexus	Posterior to and separated from the subclavian vein by the scalenus anterior and the subclavian artery (20 mm)	Same as with subclavian artery	Possible motor or sensory deficits of hand, arm, or shoulder
Parietal pleura	Contact with posteroinferior side of the subclavian vein, medial to the attachment of the anterior scalenus muscle to the first rib	Needle penetrates beneath or through both walls of the subclavian vein	Pneumothorax
Phrenic nerve	Same as with parietal pleura	Placement of needle above or behind the vein or by penetration of both its walls	Paralysis of the ipsilateral hemidiaphragm
Thoracic duct	Cross the scalenus anterior and enter the superior margin of the subclavian vein near the internal jugular junction	Same as with phrenic nerve	Soft tissue lymphedema or chylothorax on left
Internal Jugular Vein Cannulation			
Carotid artery	Passes with jugular vein in carotid sheath, consistently medial and deep to the vein	Insertion site too medial or needle course not directed at ipsilateral nipple	Hematoma, possible cerebral thromboembolism or airway obstruction
Phrenic nerve	Passes along anterior surface of scalenus anterior, behind the vein	Insertion too deep	Paralysis of the ipsilateral hemidiaphragm
Brachial plexus	Separated from the internal jugular by the scalenus anterior	Insertion too deep or too lateral	Possible motor or sensory deficits of hand, arm, or shoulder
Femoral Vein Cannulation			
Femoral artery	Lies lateral to the vein in the femoral triangle	Needle passed too laterally	Hematoma
Psoas muscle	Directly posterior to the artery and vein	Needle passed too deep	Hematoma, psoas abscess
Bowel	Proximal and deep to femoral vein	Needle passed too deep and above inguinal ligament	Enterotomy, peritonitis
Synovial capsule of hip	Deep to the psoas muscle	Needle passed too deep, particularly in small children	Arthritis

From Knopp R, Dailey RH: Central venous cannulation and pressure monitoring. JACEP 6:358, 1977.

aseptic technique in insertion and aftercare of the catheter. One study suggested a higher incidence of contamination with triple-lumen catheters [116] ; for the most part, however, an acceptably low incidence of bacteremia and sepsis (3.1%) using these devices has been encountered. [117] Femoral venous catheterization may be related to a greater risk of infection than subclavian catheterization. Merrer et al. reported overall infectious complications from femoral vs subclavian catheters to be 19.8% and 4.5% respectively. The most common organisms recovered from colonized femoral catheters, or involved with infectious complications from femoral catheters, were coagulase-negative staphylococci, Enterobacteriaceae, Enterococcus species, and *Pseudomonas aeruginosa*. [118]

Neurologic Complications

Neurologic complications are extremely rare and are presumably caused by direct trauma from the needle during venipuncture. Brachial plexus palsy and phrenic nerve injury with paralysis of the hemidiaphragm have been reported. [119] [120] Infusing medications into the internal jugular vein via a malpositioned catheter may result in a variety of neurologic complications from retrograde perfusion of intracranial vessels. [121]

Subclavian Approaches

Although both approaches to the subclavian are relatively safe (Fig. 22-21), the IC approach is more likely to be associated with complications. In a randomized prospective comparison of SC and IC venipuncture in 500 ED patients, complication rates were 2.0% and 5.1%, respectively. [129] The most significant complications have been pneumothorax and subclavian artery puncture; the highest incidence of pneumothorax is 2.4%. [29] Adherence to recommended techniques for SC subclavian venipuncture decreases the risk of these complications because the needle is directed away from the pleural dome and subclavian artery. [12] The relatively superficial location of the vein when approached from above the clavicle (1.5 to 3.5 cm) lessens the risk of puncture or laceration of deep structures.

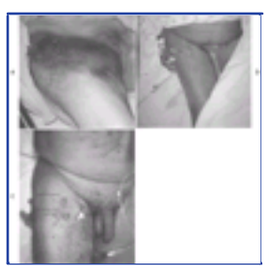

Figure 22-20 *A,* A femoral vein catheter is more prone to deep vein thrombosis and infection than a subclavian/internal jugular line, but it is a standard access route in the emergency department. Strict attention to sterile procedure and limiting use for a few days will negate most of the negatives of this approach. *A*, Significant hemorrhage can occur after puncture of the femoral artery, but this area is readily compressed. The femoral route may be the route of choice in the patient with a coagulopathy who requires a central line. The femoral vein will accept: (*B*) a triple-lumen catheter or (*C*) a large sheath introducer.

Catheter tip malposition should be expected with some frequency, as high as 27.6% in one study examining the IC technique. [130] Because of the more direct path to the SVC, the SC approach may be advantageous in this regard. For those SC series in which malposition has been reported, the overall rate is 1.1%. [13] [28] [29] The highest incidence of malposition using the SC technique, 7%, occurred during the performance of CPR. [13]

The incidence of failure to establish a functioning SC line ranges from 0% to 5%, [12] [28] [29] [131] with an overall rate of 4%. The failure rate reported for the IC technique ranges from 2.5% to 8%.[68] A recent case series of 178 SC attempts, often in patients with difficult anatomy, supports the high placement success (97.8%) and low significant complication rate (0.56%). [132]

IJ Approach

Many complications of IJ cannulation are similar to those of subclavian access. Infection, catheter malposition, thrombosis, and damage to surrounding structures are complications common to all puncture sites for central venous cannulation. The reported rates of thrombosis for internal jugular vein catheterizations range from a report of no significant thrombosis in 1 study to a high of 66% of patients exhibiting some thrombosis in a study of 33 medical intensive care unit patients. [133] No reports of significant pulmonary embolus directly attributable to an IJ catheter were found. Such wide variation in the reported incidence of complications is common, in part because of the different methods of detecting and reporting complications, variable experience with the different techniques, and the different patient populations.

The number of complications increases, especially those due to thrombosis and infection, with longer duration of catheterization and increasing severity of the patient's illness. [17] Complications also seem to be higher with the use of the left IJ vein as opposed to the right. [49] [76] [77] Reported complications thought to be due at

least in part to the use of the left-sided approach include mediastinal migration of the catheters and at least one instance of fatal pericardial tamponade.

One fairly common complication unique to the IJ approach is a hematoma in the neck. [33] With the IJ approach, pressure can be maintained easily on the area of swelling, and most hematomas will resolve spontaneously. If carotid arterial puncture is recognized and treated with compression, it rarely causes significant morbidity in the absence of marked

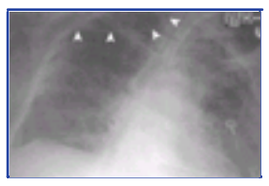

Figure 22-21 A subclavian line can easily be advanced into the neck (internal or external jugular vein). Malpositioned catheters should be discovered and replaced as soon as possible. Infusing saline into this catheter is not harmful, but hyperosmotic and vasoactive medications should not be given through this catheter for prolonged periods of time.

atherosclerotic disease, although arteriovenous fistulas have been reported after IJ puncture. [134] Several neurologic complications unique to the IJ site of venipuncture have also been reported as a result of hematomas or of direct injury. [135] [136] These complications include damage to the phrenic nerves, an iatrogenic Horner syndrome, trauma to the brachial plexus, and even an instance of the passage of a catheter into the thecal space of the spinal canal. [137] [138] [139] [140] If the carotid artery is punctured, one may again attempt IJ or subclavian cannulation on the same side after appropriate, prolonged (15 to 20 minutes) compression. The IJ vein valve is frequently damaged when cannulated, often resulting in its incompetence. The clinical significance of this, if any, is unknown. [141]

Arterial puncture is a contraindication to attempting the IJ route on the opposite side, because bilateral hemorrhage may occur with resultant airway compromise. The clinician should be prepared to rapidly intubate should this occur. Even in the face of a coagulopathy, however, the IJ approach has been found to be successful (up to 99.3% of cases) and safe (<1% complication rate). [142]

Femoral Approach

Femoral lines are generally associated with less severe complications than are IJ or subclavian approaches because of the avoidance of thoracic trauma. The most common complications of femoral venipuncture are inadvertent arterial and venous perforation. As mentioned in the Indications section, arterial puncture is more common in the patient who is in a low- or no-flow circulatory state; during cardiac arrest, the success rate for femoral catheterization may be as low as 77%, compared with 94% for subclavian vein catheterization. [143] Prolonged (>15 minutes) pressure should stop any arterial hemorrhage in a patient with normal clotting mechanisms. Prolonged arterial bleeding in patients, particularly those receiving thrombolytic or antiplatelet therapy, may warrant evaluation by a vascular surgeon or duplex imaging to evaluate for pseudoaneuyrsm formation, or both. A large study of military casualties found a 1.6% incidence of major hematomas, but these were mostly young, previously healthy patients. [144]

The peritoneum can also be violated, with possible resulting perforation of the bowel. Bowel penetration is especially likely if the patient has a femoral hernia. Injury to the bowel is usually minimal and is unlikely to require specific treatment. Nonetheless, the potential bacterial contamination of the femoral puncture site may pose a significant problem. Aspiration of air on placement of a femoral line necessitates removal of the catheter and reinsertion at another site. A case has been reported in which a patient developed clinical signs of peritonitis that were found to be due to infiltration of IV fluids into the anterior abdominal wall from a femoral catheter. [145]

A psoas abscess may result from penetration posteriorly of the underlying psoas fascia. The bladder, when distended, can also be punctured during femoral cannulation, although bladder puncture is unlikely to require therapy beyond removal of the aberrantly placed catheter. Strict aseptic technique should be maintained to prevent septic arthritis in the unlikely event that the hip capsule is punctured. This complication has been reported in infants. [146]

The femoral nerve can also be injured by an errant needle puncture. [142] [145] Complications can be minimized if the patient has a pulse and the femoral vein is approached medial to the femoral pulsations. A helpful mnemonic is NAVEL, which describes the anatomy of the region from lateral to medial: *n*erve, *a*rtery, *v*ein, *e*mpty space, and inguinal *l*igament.

A controlled trial by Merrer and colleagues identified significantly increased risks of infectious and thrombotic complications with the femoral vs the subclavian approach. [118] Deep venous thrombosis (DVT) may also occur in or near cannulated lower extremity veins, and propagate to the IVC. Several prospective studies followed cohorts of patients with femoral lines, and found iliofemoral DVT in frequencies ranging from 6.6% to 10%. [147] [148] Most were clinically silent, without leg swelling or suspicion for pulmonary embolism. The relevance of long-term complication cannot be extrapolated to the short-term value of this procedure in the ED setting. However, such DVTs definitely pose embolic risk, and may lead to fatal pulmonary emboli. [149] IJ or subclavian cannulation may result in comparable rates of upper extremity DVT. [150] The true incidence of clinical sequelae from upper extremity DVT remains unclear, and lower extremity DVT is felt to be more often responsible for large or fatal pulmonary embolism. Therefore, some authors believe that the additional risk of lower extremity DVT in patients receiving longer-term femoral catheterization (perhaps sixfold above baseline) is not justified given the existence of alternative approaches. [147] Others disagree, stating that femoral vein catheterization is a standard intervention and a valuable, generally safe technique in the ED setting.

Basilic-Cephalic Approaches

Cannulation of the central venous system through the arm veins has the lowest major complication rate of all the approaches. Superficial local infections are common (10% to 20% incidence) and rarely lead to more serious problems, including sepsis. Catheter malposition is common, and studies have shown this to occur in 10% to 40% of placements. [84] [151] Cannulation of these veins requires immobilization of the entire extremity and shoulder to prevent catheter movement

and kinking. Immobilization may be independently associated with complications.

CONCLUSION

Cannulation of the central venous circulation is a necessary skill for emergency clinicians. Safe application of the various techniques available requires detailed knowledge of anatomy and operative technique, as well as a healthy respect for potential complications. Inexperienced clinicians should not undertake these techniques without supervision, although even in experienced hands, complications should be expected. While the IC subclavian approach is most commonly encountered in clinical practice, familiarity with the SC, IJ, femoral, and basiliccephalic approaches will provide the emergency clinician with a full complement of techniques for gaining access to the central venous circulation under a variety of clinical demands.

CENTRAL VENOUS PRESSURE MEASUREMENT

Although described by Forssman in 1931, it was not until the early 1960s that measurement of CVP became commonplace as a means of assessing cardiac performance and guiding fluid therapy.[5] CVP measurements are most frequently used as a guide for determination of a patient's volume status and fluid requirements and for investigation of tamponade.[152] [153] Critical commentaries have been written by some researchers who regard CVP monitoring as ineffective, outmoded, and unreliable. [154] The astute clinician, however, can maximize the usefulness of this diagnostic tool by careful consideration of its indications and limitations. CVP is one of many variables that must be correlated in the development of an overall management plan for the care of critically ill patients.

Physiology

Simply stated, the CVP is the pressure exerted by the blood against the walls of the intrathoracic venae cava. Because the pressure in the great veins of the thorax is generally within 1 mm Hg of right atrial pressure, the CVP reflects the pressure under which blood is returned to the right atrium. [155] The pressure in the central veins has two significant hemodynamic effects. First, the pressure promotes filling of the heart during diastole, a factor that helps determine cardiac output. Second, the CVP is also the backpressure of the systemic circulation, opposing the return of blood from the peripheral blood vessels into the heart. [156] The CVP therefore affects both the ability of the heart to pump blood and the tendency for blood to flow from the peripheral veins. [27] [155] The CVP reading is determined by a complex interaction of intravascular volume, right atrial and ventricular function, venomotor tone, and intrathoracic pressure. [155]

One can measure CVP accurately by placing the tip of a pressure monitoring catheter into any of the great systemic veins of the thorax or into the right atrium. [156] Because the risks of catheter placement in the atrium include atrial perforation and cardiac dysrhythmias, any large vein within the thorax is preferred. [157] The catheter is commonly connected to an electronic pressure transducer interfaced with a monitoring system capable of calculating a mean pressure value and displaying pressure waveforms.

Indications and Contraindications for CVP Measurement

The four major indications for CVP monitoring are as follows:

1. Acute circulatory failure
2. Anticipated massive blood transfusion for fluid replacement therapy
3. Cautious fluid replacement in patients with compromised cardiovascular status
4. Suspected cardiac tamponade

The procedure is contraindicated when other resuscitation therapeutic and diagnostic interventions take priority over central venous access and CVP transducer setup and calibration.

A common misconception is that CVP consistently reflects pressures found in the left side of the heart. The measurement that best reflects left ventricular pressure changes and reserve is the left atrial pressure, or the nearly equivalent pulmonary capillary wedge pressure (PCWP). The development of the flow-directed pulmonary artery catheter has allowed repeated measurements of PCWP, thus permitting reliable estimation of the left atrial pressure.

The CVP is most helpful in patients without significant preexisting cardiopulmonary disease. Numerous studies highlight the apparent unreliability of right-sided hemodynamic monitoring in patients with underlying coronary artery or other cardiac disease or pulmonary disease. [131] [158] [159] [160] Ultimately, however, the differences noted are not a failure of CVP monitoring to reflect central hemodynamics. Rather, the disagreements noted by previous authors simply highlight the complexity of the relationship between ventricular and vascular compliance, blood volume, and filling pressures in all but very healthy patients. As when making pulmonary arterial and pulmonary arterial occlusion pressure measurements, the clinician is cautioned to be fully aware of the assumptions that such measurements make and to recognize the scenarios in which these assumptions do not hold true.

Procedure

Although CVP may be determined with a manometry column assembled at the bedside (Fig. 22-22), the most common technique in practice is measurement with an electronic transducer interfaced to a monitoring system (Fig. 22-23). Typical transducers include a nipple valve attached to a pressurized bag of saline to allow easy flushing of the system. To use these manometers, the transducer is attached to the patient's central line with a length of flexible yet fairly rigid-walled tubing filled with saline. A three-way stopcock is placed between the patient and the transducer to simplify line flushing and calibration.

All air bubbles are flushed from the system by opening the stopcock to air and flushing saline through the line. Air bubbles should not be flushed into the patient. Even tiny bubbles left in the tubing will dampen the CVP wave and potentially cause underestimation of venous pressure.

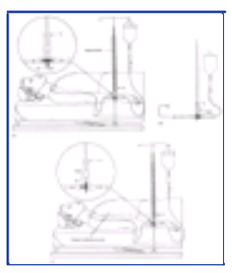

Figure 22-22 *A*, Simple manometry column used to measure CVP at the bedside. The stopcock is turned to direct the flow to the patient, bypassing the manometer. This is the position that is maintained to keep the catheter patent. The tubing is always flushed before connecting it to the patient's central venous pressure catheter. *B*, The stopcock is turned to fill the manometer to 25 cm H_2O. *C*, The stopcock is opened to the patient, and the column of water in the manometer is allowed to fall and stabilize before a reading is taken. Note that the zero mark is horizontally aligned with the tricuspid valve (midaxillary line in a supine patient).

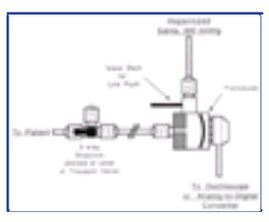

Figure 22-23 General configuration of an intravascular pressure transducer. A working understanding of these devices, particularly regarding proper setup, zeroing, and line debubbling, will maximize their effectiveness and accuracy.

After the system has been flushed, the stopcock (with the transducer still open to air) is placed at the level of the patient's tricuspid valve. The monitor detecting the transducer's signal is then "zeroed," or calibrated. The transducer is calibrated at the level of the tricuspid valve, which can be approximated on the skin surface as a

point at the midaxillary line and fourth intercostal space. [155] Finally, the stopcock is set so that the transducer is in continuity with the patient's venous catheter.

In spontaneously breathing patients, readings should be taken at the end of a normal inspiration. If the patient is receiving positive-pressure ventilation, the CVP changes during the respiratory cycle are reversed, rising with inspiration and decreasing with expiration. In these patients, readings should be taken near the end of expiration.[161] Thus, during both normal and mechanical ventilation, the lowest reading is a useful estimate of the mean CVP.

A reading may be taken after proper assembly of the equipment and after accurate placement of the tip of the catheter has been established. To ensure optimal measurement, the patient should be in the supine position. Whenever the patient is repositioned, care must be taken to ensure that the transducer has been recalibrated to reflect the new position of the patient.

Errors in CVP Measurement

A number of extrinsic factors may alter the accuracy of the CVP reading (Table 22-8).[27] In addition to the position of the patient, these include changes in intrathoracic pressure,[162] catheter tip malposition, obstruction of the catheter, and failure to calibrate or zero the line. Activities that increase intrathoracic pressure, such as coughing or straining, may cause spuriously high measurements. The patient should be relaxed at the time of the measurement and breathing normally. In mechanically ventilated patients, the CVP will be elevated to an extent directly proportional to the ventilatory pressures being delivered and inversely proportional to the mechanical

TABLE 22-8 -- Faulty Central Venous Pressure Readings

Increased intrathoracic pressure (ventilator, straining, coughing)
Reference points in error
Malposition of catheter tip
Blocking or ball-valve obstruction of catheter
Air bubbles in circuit
Readings during wrong phase of ventilation
Readings by different observers
Vasopressors (presumed)

compliance of the lung. Care should be exercised in interpreting filling pressures in this circumstance, as ventilator-induced elevations in CVP are not artifactual, but represent changes in the hemodynamic physiology of the patient. As in spontaneously breathing patients, CVP measurements are only meaningful in a relaxed, sedated, or sedated and paralyzed subject.

Another reason for faulty readings is malposition of the catheter tip. If the catheter tip has not passed far enough into the central venous system, peripheral venous spasm or venous valves may yield pressure readings that are inconsistent with the true CVP. [27]

If the catheter tip is passed into the right ventricle, a falsely high CVP is obtained. Recognition of a characteristic right ventricular pressure waveform on the patient's monitor should hopefully preclude this error. Such fluctuations may occasionally be seen in appropriately positioned CVP lines when significant tricuspid regurgitation or atrioventricular dissociation (cannon a waves) is present.

Inaccurate low venous pressure readings are seen when a valve-like obstruction at the catheter tip occurs either by clot formation or by contact against a vein wall. As mentioned earlier, wave damping due to air bubbles in the transducer or tubing also leads to faulty readings.

Using poorly zeroed lines may result in inaccurate measurements that may be interpreted as a change in the patient's status when none has actually occurred. The transducer should be zeroed to the same level for every measurement.

Some investigators mention a falsely elevated CVP in patients who are receiving vasopressors, but controlled data on this aberration are lacking. One animal study suggests that fluid can be infused into one lumen of a multilumen catheter without affecting the CVP reading at another lumen. [163]

Interpretation of the CVP measurement

Because determination of the CVP can aid the clinician in assessment of the critically ill patient, it is paramount that the clinician knows the normal values and the variables that may affect these values and can recognize the pathologic conditions that correlate with abnormal values. Although various ranges for normal have been reported,[153] [164] [165] a summary of these values is as follows:

Low: <6 cm H_2O

Normal: 6 to 12 cm H_2O

High: >12 cm H_2O

In the late stages of pregnancy (30 to 42 weeks), the CVP is physiologically elevated, and normal readings are 5 to 8 cm H_2O higher in pregnant women.

A CVP reading <6 cm H_2O is consistent with low right atrial pressure and reflects a decrease in the return of blood volume to the right heart. This may indicate that the patient requires additional fluid or blood. A low CVP reading is also obtained when vasomotor tone is decreased, as in sepsis, spinal cord injury, or other forms of sympathetic interruption.[153] [164] [165]

A CVP reading falling within a normal range is viewed in relationship to the clinical situation. A reading >12 cm H_2O indicates that the heart is not effectively circulating the volume presented to it. This situation may occur in the case of either a normovolemic patient with underlying cardiac disease such as left ventricular hypertrophy (with associated poor ventricular compliance) or a patient with a normal heart who is over-hydrated and over-transfused. A high CVP is also related to variables other than pump failure, which include pericardial tamponade, restrictive pericarditis, pulmonary stenosis, and pulmonary embolus. [164]

Changes in blood volume, vessel tone, and cardiac function may occur alone or in combination with one another; therefore, it is possible to have a normal or high CVP in the presence of normovolemia, hypovolemia, and hypervolemia. One must interpret the specific CVP values with respect to the entire clinical picture. The response of the CVP to an infusion is more important than the initial reading.

Fluid Challenge

Monitoring of the CVP may be helpful as a practical guide for fluid therapy. [152] [153] Serial CVP measurements provide a fairly reliable indication of the capability of the right heart to accept an additional fluid load. Although the PCWP is a more sensitive index of left heart fluid needs (and in some clinical situations, PCWP measurement is essential), serial measurement of CVP can provide significant information.

A fluid challenge can help assess both volume deficits and pump failure. Although a fluid challenge can be used with either PCWP monitoring or CVP monitoring, only the fluid challenge for CVP monitoring is discussed here. Slight variations in methodology of fluid challenge are reported in the literature. Generally, aliquots of 50 to 200 mL of crystalloid are sequentially administered, and measurements of CVP levels are obtained after 10 minutes. [131] [164]

The fluid challenge is generally carried out in the following manner [153] : Fluid is administered by a route other than that used for monitoring. An initial CVP reading is taken, and fluid is infused at a rate of 20 mL/min over a 10-minute period. The infused volume is allowed to equilibrate for 10 minutes, and a reading is taken. If the CVP is >5 cm of H_2O over the initial measurement, the fluid challenge is discontinued, and one assumes that the right ventricle is unable to handle an additional fluid load. Increases of between 3 and 5 cm H_2O over the initial CVP value are equivocal, and additional measurements are taken over the next 30 minutes if this reading is obtained. An increase of <2 cm H_2O over the original reading is indicative of volume depletion. The fluid challenge is repeated until measurements indicate that adequate volume expansion has occurred. The fluid challenge is discontinued as soon as hemodynamic signs of shock are reversed or signs of cardiac incompetence are evident.

Cardiac Tamponade

In cardiac tamponade, pericardial pressure rises to equal right ventricular end-diastolic pressure. The pericardial pressure encountered in pericardial tamponade characteristically produces an elevated CVP. The degree of CVP elevation is variable, and one must interpret measurements cautiously; CVP readings in the range of 16 to 18 cm H_2O are typically seen in acute tamponade, [153] [166] but elevations of up to 30 cm H_2O may be encountered. The exact CVP reading is often lower than one might intuitively expect, and it is not uncommon to encounter tamponade with a CVP of 10 to 12 cm H_2O. A normal, or even low, CVP reading may be seen if the tamponade is associated with significant hypovolemia. An excessive rise in CVP following fluid challenge may be more important than a single reading in the diagnosis of pericardial tamponade.

Excessive straining, agitation, pneumatic antishock garment inflation, positive-pressure ventilation, or tension pneumothorax may increase intrathoracic pressure, producing a high CVP reading, and may erroneously suggest the diagnosis of pericardial tamponade. Increases in vascular tone, as seen with the use of dopamine or other vasopressors, may also elevate the CVP, mimicking tamponade and complicating volume estimation.

CONCLUSION

CVP monitoring provides useful hemodynamic monitoring information in those individuals with a relatively normal cardiopulmonary system who do not otherwise warrant PCWP monitoring.

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Chapter 23 - Venous Cutdown

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Patricia Lanter

"The standard cutdown is well known to all trauma surgeons and needs no description." [1] Were this a true statement, the remainder of this chapter would be superfluous; however, clinician training in the technique of venous cutdown has been largely informal. For 5 decades, the mechanics of venous cutdown have been handed down from house officer to house officer as one of the rites of internship. There are, in fact, few detailed descriptions of the procedure in the medical literature, and the scientific data documenting its usefulness or complication rate are sparse.

An early description of venous cutdown technique was provided in 1940 by Keeley, who offered the procedure as an alternative to venipuncture in patients who were in shock or had small, thin veins. [2] In 1945 Kirkham gave the first detailed description of the saphenous vein cutdown at the ankle. [3] The procedure changed little until 1990, when Shockley developed a modified cutdown technique. In 1997 Klofas adapted the Shockley method for the distal saphenous vein and developed a model on which to teach it.

The most significant changes over the past 5 decades have involved not the technique itself, but the cannulas that are used. Keeley and Kirkham used metal needles. With the advent of plastic cannulas in the mid-1940s, the cutdown became more popular as a means of providing long-term intravenous (IV) infusion. Clinicians have used IV tubing, feeding tubes, and even nasogastric tubes as cannulas in the management of hypovolemic patients. Currently, large-bore catheters (10-ga, 8 Fr) are often inserted by cutdown in the management of hypovolemic shock. [4]

The frequency of performing venous cutdowns is impossible to estimate. The popularity of central venous cannulation by the internal jugular, subclavian, and femoral routes has markedly decreased the frequency of venesection. In the Advanced Trauma and Life Support (ATLS) provider course, venous cutdown is no longer a mandatory procedure. Rather, it is optional, to be taught at the director's discretion. [5] Nevertheless, the cutdown remains an excellent method of obtaining venous access in several emergent clinical situations. Although a cutdown is mechanically simple to perform, ease of performance does not guarantee that the procedure will be performed efficiently and without complications. Performance of a rapid, effective cutdown can be achieved only by thorough knowledge of the anatomy and the procedure and attention to its many details.

INDICATIONS

There are no absolute indications for venous cutdown, simply because several options for venous access usually exist. The indications for use of the procedure are relative, depending to a great extent on clinician experience and preference. There are several clinical situations in which the venous cutdown may be used.

Venous Access in Infants

Small children present a unique challenge to the clinician who does not perform pediatric venipuncture regularly (see Chapter 19). The challenge is greater still if the procedure must be performed rapidly in a child or infant in shock or in a child in whom few, if any, veins are visible. Other options frequently used include intraosseous infusion or cannulation of the femoral, subclavian, or internal jugular veins. In selected circumstances (e.g., *status epilepticus*), the rectal route of administration, with mucosal absorption, is an effective alternative to cutdown.

A venous cutdown may be performed when all accessible peripheral sites, including scalp veins, have been exhausted. The distal saphenous vein is large enough to cannulate in most children and has a predictable anatomic location. Therefore, venous cutdown at the ankle is commonly used for both emergent management and long-term venous access.[6] [7]

Hypovolemic Shock

Rapid percutaneous insertion of large-bore (14-ga) catheters is appropriate in most cases of hypovolemic shock. Unfortunately, peripheral vessels frequently collapse in hypovolemia or have been rendered useless by IV drug abuse or previous venous catheterization. The venous cutdown is an acceptable alternative in these instances, although percutaneous insertion of a large-bore introducer device into a central vein can usually be performed more rapidly. [8] When the cutdown can be performed quickly, it offers the advantage of direct visualization of the vessel during cannulation.

Use of the cutdown as a vehicle for the insertion of IV extension tubing and rapid transfusion was popularized during the Vietnam War. [9] The technique also has been found useful in civilian practice for the resuscitation of patients with profound hypovolemia. [10] The flow rate for saline through a standard IV extension set (3 mm inside diameter) cut to a length of 28 cm (12 in.) and inserted directly into the vein is >15% to 30% than that through a 5-cm, 14-ga catheter. The difference is greater if pressure is applied to the system. The improvement in flow rate through large-bore lines is greater for blood than for crystalloid solutions, because the viscous characteristics of blood greatly impede its passage through small-bore tubing. [10] A unit of blood can be transfused in 3 minutes using IV extension tubing inserted into the vein. Consequently, large-bore lines placed by venous cutdown are an excellent mechanism for the treatment of severe hypovolemia. High-flow infusion techniques are discussed elsewhere (see Chapter 24).

CONTRAINDICATIONS

Venous cutdown is contraindicated when less invasive alternatives exist or when excessive delay would be required for the procedure to be performed. [12] Although highly skilled operators may perform a cutdown in <60 seconds, [13] studies by Rhee and colleagues, [14] Iserson and Criss, [15] and Westfall and Price [8] have shown that, on average, the procedure requires at least 5 to 6 minutes to complete. The modified guidewire method described both by Shockley and Klofas may decrease that time by 22%. [16] [17] Percutaneous insertion of large-bore catheters is the preferred method of rapid fluid infusion unless high flow rates are required or peripheral vessels have collapsed. Another method of rapid fluid infusion

that is technically easier and faster than venesection is the percutaneous insertion of large-bore introducer devices into the subclavian, internal jugular, or femoral veins (see Chapter 22). These devices typically use 8 Fr catheters with flow rates comparable to those obtained with IV tubing. The use of the subclavian and internal jugular vessels also is preferable to the cutdown for long-term venous access.

Other contraindications are relative. In the presence of coagulation disorders, impaired healing, or compromised host-defense mechanisms, the need to perform a cutdown should be weighed carefully against the potential complications. Cutdowns should be avoided when there is an infection over the site and in extremities with injuries proximal to the cutdown site.

ANATOMY

Detailed knowledge of anatomy is imperative to the success of this procedure. Veins in both the upper and lower extremities may be used. The choice of a particular vein should be governed by its accessibility and size and by the clinician's experience and training. The anatomy of individual vessels and their relative merits as cutdown sites are described as follows.

The Greater Saphenous Vein

The greater saphenous vein is the longest vein and runs subcutaneously throughout much of its course (Fig. 23-1). It is most easily accessible at the ankle but may also be cannulated below the knee and below the femoral triangle. The greater saphenous vein begins at the ankle, where it is the continuation of the medial marginal vein of the foot. The vein crosses 1 cm anterior to the medial malleolus and continues up the anteromedial aspect of the leg. [18] At the level of the malleolus, the vein lies adjacent to the periosteum and is accompanied by the relatively insignificant saphenous nerve, which, if transected, causes sensory loss in a small area along the medial aspect of the foot. At the ankle, the vessel can be exposed with minimal blunt dissection. The vein's superficial, predictable, and isolated location has made the distal saphenous vein the classic pediatric cutdown site. [19]

The saphenous vein lies superficially on the medial aspect of the knee. A cutdown performed 1 to 4 cm below the knee and immediately posterior to the tibia has been described in the pediatric literature. [6] This site is distal enough to avoid interference with the performance of other resuscitative procedures, yet proximal enough to allow the passage of a long line into the central circulation. [20] However, it is seldom used for venous cutdown. Disadvantages of this site include kinking of the line as the knee is flexed and the risk of injury to the saphenous branch of the genicular artery and the saphenous nerve. [21]

In the thigh, the saphenous vein begins on the medial aspect of the knee and crosses anterolaterally as it ascends toward the femoral triangle. Proximally, it enters the fossa ovalis and joins the femoral vein. Three to 4 cm distal to the inguinal ligament, the saphenous vein is of large caliber (4 to 5 mm outside diameter) and is easily isolated from the surrounding fat. Also lying anteromedially in the thigh is the lateral femoral cutaneous vein, which has a smaller diameter (2 to 3 mm) and lies lateral to the greater saphenous vein. [22] [23] The accessibility and large diameter of the greater saphenous

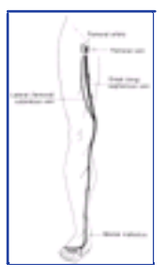

Figure 23-1 Superficial veins of the lower limb.

vein in the thigh make its use an option in the treatment of profound hypovolemia. [10]

The Basilic Vein

The basilic vein is a preferred site for venous cutdown in the upper extremity. Veins of the dorsal venous network of the hand unite to form the cephalic and basilic veins, which travel along the radial and ulnar sides of the forearm, respectively (Fig. 23-2). At the level of the mid-forearm, the basilic vein crosses anterolaterally and is consistently found 1 to 2 cm lateral to the medial epicondyle on the anterior surface of the upper arm. The medial cubital vein crosses over from the radial side of the arm to join the basilic vein just above the medial epicondyle. The basilic vein then continues proximally, occupying a superficial position between the biceps and pronator teres muscles. In this segment it lies in close association with the medial cutaneous nerve, which supplies sensation to the ulnar side of the forearm. The vein penetrates the brachial fascia in the distal third of the upper arm and then occupies a deeper position. [24]

The basilic vein is generally cannulated at the antecubital fossa 2 cm above and 2 to 3 cm lateral to the medial epicondyle. It is exposed through a transverse incision on the medial aspect of the proximal antecubital fossa. The size of this vein enables it to be located easily, even in the hypotensive or hypovolemic patient; large catheters can be passed without difficulty under most circumstances. The median

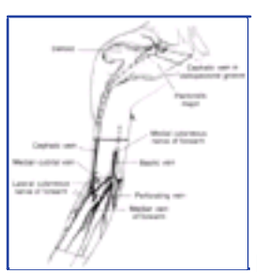

Figure 23-2 Veins of the upper limb.

cubital vein is accessible through the same incision. Superficially at this level, there are no important associated structures, but the brachial artery and the median nerve are found deep to the basilic vein.

A more proximal insertion site has been recommended by Simon and colleagues[25] to avoid the network of interconnecting veins at the level of the antecubital fossa. However, in the distal third of the upper arm, there is a closer association between the basilic vein and the medial cutaneous nerve. Transection of this nerve produces sensory loss on the ulnar side of the forearm.

The Cephalic Vein

This vessel begins on the radial aspect of the wrist and crosses anteromedially, ascending toward the antecubital fossa. In the forearm it lies in close association with the lateral cutaneous nerve, which supplies sensory innervation to the radial aspect of the forearm (see Fig. 23-2). In the antecubital fossa it lies subcutaneously, just lateral to the midline, and then ascends in the upper arm, overlying the lateral aspect of the biceps muscle. At the shoulder the cephalic vein lies in the deltopectoral groove. Just below the clavicle, it passes deep to end in the axillary vein. [24]

Venesection is easily performed on the cephalic vein because of its large diameter and superficial location. In the forearm it is important to avoid the lateral cutaneous nerve. A good location is in the antecubital fossa at the distal flexor crease. Cutdown on the cephalic vein at the wrist has also been reported, but the thin skin overlying the vein at this level usually permits simple percutaneous cannulation when the vein is available for cannulation. [26] The cephalic vein may also be entered in the deltopectoral groove. The slightly deeper position and physical interference with the performance of other procedures make this approach more difficult.

The Brachial Veins

The brachial veins are small, paired vessels lying on either side of the brachial artery. In contrast to the vessels described earlier, these are not superficial and will not

accommodate large cannulas. Their most superficial location is 1 to 2 cm above the antecubital fossa just medial to the biceps muscle. Palpation of the brachial pulse serves as a useful landmark. Because of its proximity, the brachial artery may be inadvertently cannulated in the pulseless patient. In addition, there is the risk of injury to the closely associated median nerve. Time-consuming blunt dissection is usually required because of the vessels' greater depth. For these reasons, brachial vein cutdown is *not recommended* as an emergency venous access route and should be used only in the absence of a suitable alternative. [12] This site may be acceptable when time and vessel size are not critical factors, but it is difficult to justify the deep dissection and associated risks that are involved.

The External Jugular Vein

The external jugular vein begins below the angle of the mandible and is formed by confluence of the posterior auricular and retromandibular veins. It descends posterolaterally across the surface of the sternocleidomastoid muscle and then pierces the fascia to join the subclavian vein deep to the clavicular head of this muscle. The greater auricular nerve, which supplies sensation to the external ear, travels parallel to the external jugular vein. [18] A venous cutdown may be performed on the external jugular vein at its superficial location on the sternocleidomastoid muscle. This is not recommended as a first-line venesection site for the following reasons: (1) Performance of a cutdown may cause physical interference with airway management and central venous cannulation. (2) There is risk of injury to the greater auricular nerve. (3) It is difficult to immobilize the area adequately. (4) Cervical spine immobilization frequently prohibits access to the area. (5) It is potentially a hazardous procedure in the uncooperative patient. [12]

As a general rule, cutdown on the external jugular vein should be performed only when other means of venous access are exhausted. The external jugular vein is an acceptable site for emergency *percutaneous* venous cannulation, especially in children.

EQUIPMENT

The materials required to perform a formal venous cutdown are listed in Table 23-1 . All necessary instruments should be available on a sterile tray before the procedure is begun. The standard cutdown tray is shown in Figure 23-3 . A time-consuming search for the proper instrument can be avoided if only necessary instruments are included on standardized trays. Pediatric patients also require both a warming table or radiant warmer and a padded extremity board.

Catheter choice depends on the desired function of the venous line. When central venous pressure (CVP) monitoring is needed, the catheter chosen must be long enough to reach the superior vena cava. The average distance from the antecubital fossa to the superior vena cava is 54 cm in the adult male. This distance can be approximated by aligning the catheter over the chest with the tip at the level of the manubrial-sternal

TABLE 23-1 -- Materials Required for Venous Cutdown*

Curved Kelly hemostat
Scalpel with No. 11 blade
Small mosquito hemostat
Tissue spreader
Iris scissors
Plastic venous dilator or lifter
4-0 silk suture ties
4-0 nylon suture on cutting needle
Antibiotic ointment
Gauze sponges
1-in. tape
Arm board
Intravenous catheter
Rolled gauze bandage

*See Figure 23-3 .

junction. Lumen size is relatively unimportant when the line is inserted for monitoring the CVP or to infuse drugs, but it is a critical factor in the treatment of hypovolemia. Short, large-bore catheters are preferred when fluid must be delivered rapidly. Silastic catheters, IV plastic tubing, or 5 or 8 Fr pediatric feeding tubes may be used as infusion catheters in older children and adults.

Table 23-2 Table 23-3 Table 23-4 list the flow rate of various fluids through some commonly used catheter systems. Knowledge of relative flow rates is essential if maximal benefit is to be obtained from the time spent performing the cutdown. Excellent flow rates can be achieved by threading IV tubing (sterile tubing may be cut to the appropriate

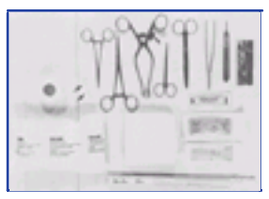

Figure 23-3 Venous cutdown tray. Note the small plastic vein dilator-lifter *(arrows)*, which is especially useful in children.

length—leaving a slight bevel on the end will facilitate cannulation of the opened vein) directly into the vein or by using a 5-cm, 10-ga IV catheter. [4] [13]

TECHNIQUE

The technique of venous cutdown is essentially the same regardless of the vessel cannulated (Fig. 23-4 Fig. 23-5 Fig. 23-6 Fig. 23-7 Fig. 23-8 Fig. 23-9 Fig. 23-10 Fig. 23-11 Fig. 23-12 Fig. 23-13). Detailed knowledge of the local anatomy is important if the procedure is to be performed rapidly without injury to associated structures. For children, adequate immobilization of the lower leg or elbow (depending upon cutdown site) on a padded board should be accomplished before beginning the procedure (see Fig. 23-13). Even in emergency situations, reasonable precautions should be taken to avoid infection. The area of the skin incision should be widely prepared with an antiseptic solution and then draped. A tourniquet placed proximal to the cutdown site helps in the visualization of the vein.

In the conscious patient, the site is infiltrated with 1% lidocaine. A skin incision is made transverse to the course of the vessel (see Fig. 23-4). A longitudinal incision decreases the risk of transecting neurovascular structures but may not provide sufficient exposure. A transverse incision involving all layers of the skin is the best approach. Subcutaneous (SQ) fat should bulge from the incision. The clinician bluntly dissects SQ tissue by spreading the tissue gently with a curved hemostat *in a direction parallel to the course of the vein with the tips downward*. Bleeding is usually minimal unless the vein is nicked. A tissue spreader or a self-retaining retractor may be used to provide a wider field. The vein is

TABLE 23-2 -- Comparative Average Flow Rates (mL/min) for Tap Water*

Catheter	Pressure, 200 mm Hg (95% CI)	Gravity (95% CI)
Central Venous Catheters		
USCI 9 Fr introducer		
Internal diameter, 0.117 in.; 5 ½ in.	566 (±16)	247 (±2)
USCI 8 Fr introducer		
Internal diameter, 0.104 in.; length, 5 ½ in.	540†	243 (±5)
Deseret Angiocath		
Gauge, 14; length, 5 ¼ in.	341 (±6)	157 (±6)
Deseret Angiocath		
Gauge, 16; length, 5 ¼ in.	195 (±4)	91 (±2)
Deseret subclavian jugular catheter		
Gauge, 16; length, 12 in.	142 (±4)	54 (±3)
Peripheral Venous Catheters		
Intravenous tubing		
Internal diameter, 0.12 in.; length, 12 in.	500 (±21)	222 (±4)
Argyle Medicut		
Gauge, 14; length, 2 in.	484 (±8)	194 (±5)
Deseret Angiocath		
Gauge, 14; length, 2 in.	405 (±2)	173 (±4)
Vicra Quick-Cath		
Gauge, 14; length, 2 ¼ in.	—	167 (±1)
Argyle Medicut		
Gauge, 16; length, 2 in.	353 (±4)	151 (±3)
Deseret Angiocath		
Gauge, 16; length, 2 in.	231 (±1)	108 (±1)
Vicra Quick-Cath		
Gauge, 16; length, 2 in.	—	108 (±1)

CI, confidence interval.

From Mateer JR, Thompson BM, Aprahamian C, Darin JC: Rapid fluid resuscitation with central venous catheters. Ann Emerg Med 12:150, 1983. Reproduced by permission.

*Mean of three trials with hydrostatic pressure head of 1 m.
†95% confidence interval not calculated because all three trials resulted in 11.1 sec for 100-mL flow.

then isolated from the adjacent tissue and mobilized for 1 to 3 cm (see Fig. 23-5).

For the standard venous cutdown technique, after the vein is mobilized, a hemostat can be used to pass proximal and distal silk ties placed under the vein for stabilization. An alternative approach is to simply use the hemostat tips (with no ligatures) in a "spread" position to elevate and stabilize the exposed vein during incision and cannulation. The hemostat is placed as in Fig. 23-7 , only without taking the time to pass ligatures.

If ligatures are passed before cannulation, the distal ligature may or may not be tied after initial placement. If the distal ligature is tied, it should not be cut because the proximal tie is useful in controlling the vein (see Fig. 23-6 and Fig. 23-7). Using a hemostat, the vessel is elevated and stretched flat. This provides good visualization and control of the vessel and limits bleeding when the vessel is incised. Alternatively, placing gentle traction on the proximal tie will control oozing around the puncture site. The vessel is incised at a 45° angle, through one third to one half of its diameter (see Fig. 23-7). A No. 11 blade (as illustrated) or a pair of iris scissors may be used to incise the vessel. Too small an incision may cause threading of the catheter into a false channel in the adventitia; conversely, the vessel may be torn completely and may retract

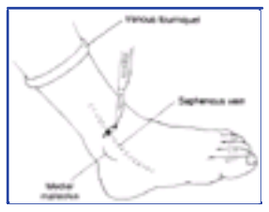

Figure 23-4 A skin incision is made perpendicular to the course of the vein.

from the field if the incision is too large. [27] A longitudinal incision is sometimes made to avoid transecting the vessel, but the lumen is more difficult to identify with this technique. *The vessel incision must enter the actual lumen of the vein*, although some bleeding will occur after the vein has merely been nicked. Incision of the vessel is unnecessary when an IV catheter with an introducing needle is used. The vessel is simply punctured, as in percutaneous venous cannulation (see Mini-cutdown later in this chapter).

Before being introduced into the vessel, the cannula is beveled at a 45° angle unless a cannula with a tapered tip is used. A short bevel is preferred, and a sharply pointed tip is to be avoided because it may pierce the posterior wall or otherwise damage the vein. The rounded tip of a feeding tube may be more difficult to introduce, but it may be advanced less traumatically. If using an IV cannula, the cannula may be introduced directly through the skin incision or through a separate stab wound. The latter method, using an Intracath device, is illustrated in Figure 23-8 and Figure 23-9 . Theoretically, the percutaneous approach reduces the risk of infection. [28] Threading the catheter into the vein is often the most difficult and time-consuming portion of the procedure.

Figure 23-5 The skin is retracted and the vein exposed by blunt dissection. *(From Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

TABLE 23-3 -- Comparative Average Flow Rates (mL/min, 200 mm Hg Pressure) for Red Blood Cells

Catheter	Diluted PRBCs, Hct 45% (95% CI)	Diluted PRBCs, Hct 45% Through Blood Warmer (95% CI)	PRBCs, Hct 65% (95% CI)
Central Venous Catheters			
USCI 9 Fr introducer			
Internal diameter, 0.117 in.; 5 ½ in.	343 (±21)	218 (±26)	124 (±2)
USCI 8 Fr introducer			
Internal diameter, 0.104 in.; length, 5 ½ in.	324 (±23)	—	—
Deseret Angiocath			
Gauge, 14; length, 5 ¼ in.	210 (±7)	171 (±9)	63 (±6)
Deseret Angiocath			
Gauge, 16; length, 5 ¼ in.	125 (±4)	—	—
Peripheral Venous Catheters			
Intravenous extension tubing			
Internal diameter, 0.12 in.; length, 12 in.	312 (±1)	—	—
Argyle Medicut			
Gauge, 14; length, 2 in.	287 (±21)	192 (±15)	96 (±6)
Deseret Angiocath			
Gauge, 14; length, 2 in.	257 (±11)	—	—
Argyle Medicut			
Gauge, 16; length, 2 in.	220 (±5)	—	—
Deseret Angiocath			
Gauge, 16; length, 2 in.	158 (±14)	—	—

CI, confidence interval; Hct, hematocrit; PRBC, packed red blood cells.

From Mateer JR, Thompson BM, Aprahamian C, Darin JC: Rapid fluid resuscitation with central venous catheters. Ann Emerg Med 12:151, 1983. Reproduced by permission.

Difficulty in threading may be encountered for several reasons. The lumen may have been incorrectly identified, or a false passage may have been created. This frequently occurs and may be difficult to recognize because the catheter can easily advance between layers of the vessel wall. Other causes of difficult threading are penetration of the posterior vessel wall, the presence of venous valves, or use of a catheter that is too large for the vessel being cannulated. Identification of the vessel lumen may be facilitated through use of a plastic venous dilator or elevator. The small, pointed tip of the device is threaded into the vessel to expose the lumen in advance of

TABLE 23-4 -- Comparative Average Flow Rates in Milliliters per Minute

Catheter	Tap Water at 200 mm Hg	Diluted PRBCs at 200 mm Hg	Tap Water Gravity	Diluted PRBCs Blood Warmer at 200 mm Hg	PRBCs, at 200 mm Hg
Central Venous Catheters					
USCI 9 Fr introducer					
Internal diameter, 0.117 in.; 5 ½ in.	566 (±16)	343 (±21)	247 (±2)	218 (±26)	124 (±2)
USCI 8 Fr introducer					
Internal diameter, 0.104 in.; length, 5 ½ in.	540*	324 (±23)	243 (±5)	—	—
Deseret Angiocath					

Gauge, 14; length, 5 ¼ in.	341 (±6)	210 (±7)	157 (±6)	171 (±9)	63 (±6)
Peripheral Venous Catheters					
Intravenous extension tubing					
Internal diameter, 0.12 in.; length, 12 in.	500 (±21)	312 (±1)	222 (±4)	—	—
Argyle Medicut					
Gauge, 14; length, 2 in.	484 (±8)	287 (±21)	194 (±5)	192 (±15)	96 (±6)
Argyle Medicut					
Gauge, 16; length, 2 in.	353 (±4)	220 (±5)	151 (±3)	—	—

PRBC, packed red blood cells.

From Mateer JR, Thompson BM, Aprahamian C, Darin JC: Rapid fluid resuscitation with central venous catheters. Ann Emerg Med 12:151, 1983. Reproduced by permission.

*95% confidence interval not calculated because all three trials resulted in 11.1 sec for 100-mL flow.

the catheter (see Fig. 23-10). A sterile 20-ga needle bent at a 90° angle may also serve as a vein elevator. A vein dilator is useful in pediatric cutdowns but is generally unnecessary in adults. The clinician can facilitate the threading of large catheters in adults by grasping the proximal surgical edge of the vessel with small forceps or a mosquito hemostat. Countertraction is applied as the catheter is advanced (see Fig. 23-11). At no time should one force a catheter that will not advance.

Once the catheter is advanced into the lumen, air is back-bled from the cannula, and the cannula is connected to

Figure 23-6 Proximal and distal ties are passed under the vein. If the vein is to be sacrificed, the distal suture is tied to prevent bleeding, and the ends are left long to help stabilize the vein during cannulation. The proximal tie is not tied at this point, but traction on it will control back-bleeding. *(From Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

IV tubing. The proximal ligature is tied around the vessel wall and the intraluminal cannula. The tourniquet is now removed, the catheter is affixed to the skin, and the incision is closed (see Fig. 23-12). An antibiotic ointment is applied where the catheter passes through the skin, and the wound is dressed. In an emergent situation, skin closure can be delayed and the wound simply wrapped with a sterile dressing (e.g., Kerlix) (see Fig. 23-13). The IV tubing should be looped under the outer layers of the dressing to minimize the risk of inadvertent removal of the cannula should the external IV line be tugged.

Mini-Cutdown

An alternative method designed to preserve the vein and bypass the time-consuming step of placing a catheter into the vessel has been described. [29] A skin incision and blunt dissection are used to locate the vessel. Once identified, the vein is punctured under direct vision with a standard percutaneous venous catheter. The needle may be introduced through a separate stab incision or through the skin incision. If an over-the-needle device (e.g., Angiocath, Medicut) is used, the

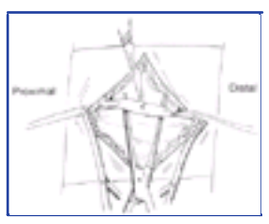

Figure 23-7 The vein is stretched flat and incised at a 45° angle. Approximately one third of the lumen must be exposed. Traction on the proximal tie will control back-bleeding. *(From Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

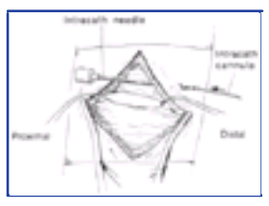

Figure 23-8 Use of the Intracath needle to produce a separate stab incision. The cannula is introduced into the wound by retrograde passage through the introducing needle. *(From Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

needle is withdrawn and discarded. With a through-the-needle device, the cannula is threaded into the vein, and the needle is withdrawn to the skin surface (Fig. 23-14). A guard is placed on the needle tip, the catheter device is fixed to the skin, and the incision is closed. This method eliminates the need for tying or cutting the vein, thereby permitting repeated catheterization. Venipuncture is easier and uses the same equipment as for percutaneous venous cannulation. The mini-cutdown is therefore used in the treatment of chronically ill patients who require long-term IV therapy or in children who have limited accessible veins. A simple skin incision may also permit direct visualization of veins in the obese patient and may facilitate standard percutaneous venipuncture.

Hansbrough and coworkers[4] described the mini-cutdown procedure with a 10-ga IV catheter (Deseret 10-ga Angiocath). The flow rates of blood and saline with this catheter are equal to the rates obtained when IV extension tubing is placed in a vein using the more time-consuming standard venous cutdown technique. This catheter allows one to infuse a unit of whole blood in 2 to 3 minutes if pressure and oversized IV tubing (e.g., urology irrigation tubing) are used.

Figure 23-9 A cannula threaded through the stab incision. The Intracath needle has been withdrawn following introduction of the cannula into the wound. *(From Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

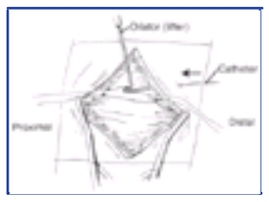

Figure 23-10 Threading the catheter with the aid of a venous dilator (lifter). This is technically the most difficult part of the procedure. The lifter is especially helpful in small veins.

MODIFIED CUTDOWN TECHNIQUE

Shockley and Butzier[16] describe a further modification whereby a guidewire, dilator, and sheath system are inserted following standard cutdown and venotomy. The guidewire, dilator, and sheath system should be set up before making the skin incision. Once the vessel has been incised, the end of the guidewire is inserted, followed by the dilator and sheath. The wire and dilator are then removed, leaving the sheath. They found that ligatures were not usually needed with this technique, and there was increased vessel salvage in the event of transection. In addition, they found this technique to save more than 2 minutes' time over the standard technique when performed by novices. Klofas[17] used a similar technique at the distal saphenous vein. In addition, he developed a model for teaching the modified technique using wood, gauze, cast padding, and tape.

Removal of catheters inserted by cutdown requires only cutting of the skin stitches holding the catheter in place, followed by withdrawal of the catheter. Backbleeding from

Figure 23-11 In larger veins, a mosquito hemostat can facilitate the placement of the cannula by opening the lumen and providing countertraction.

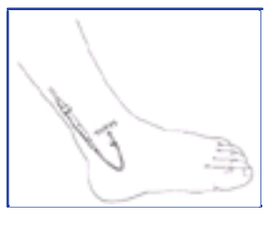

Figure 23-12 The incision is closed, and the catheter is sutured in place.

the proximal venous end is controlled by a simple pressure dressing and is generally not a significant problem.

COMPLICATIONS

The complications of venous cutdown include local hematoma and infection, sepsis, phlebitis, embolization, wound dehiscence, and injury to associated structures. An indirect but significant complication is deterioration of an unstable patient during a time-consuming cutdown attempt. Documentation of complications and their frequency has been sparse. Bogen[30] reported a 15% complication rate in 234 cases. Infection and phlebitis each occurred at a rate of 4%. Infectious complications may result from the introduction of pathogens during line placement, transcutaneous invasion along the course of the cannula, or deposition of blood-borne organisms on the catheter tip.[31] A clear correlation exists between the incidence of infectious complications and the length of time that a catheter is left in place. Moran and associates[32] found that the infection rate rose from 50% to 78% when a catheter was left in place for more than 48 hours. Druskin and Siegel, [31] studying a mixed population of patients who had undergone cutdowns and others who had catheters percutaneously inserted, found that the incidence of culture-positive catheter tips rose from 0 to 52% after 48 hours.[31] In the study by Moran and colleagues,[32] *Staphylococcus albus* was the predominant organism that was isolated, but organisms more commonly thought of as pathogenic (*S. aureus, Enterococcus* spp., and *Proteus* spp.) were isolated with

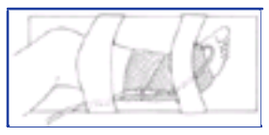

Figure 23-13 The cutdown site is securely dressed and splinted.

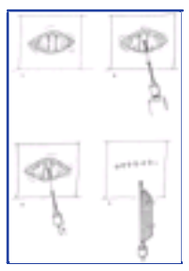

Figure 23-14 The mini-cutdown technique is an alternative to the venous cutdown method. The vein is cannulated under direct vision using standard percutaneous catheters. A separate entry site (shown) may be used, or the vein can be cannulated through the skin incision. Note that the vein is not tied off with this technique. A standard Angiocath IV set also may be used instead of the through-the-needle catheter shown here.

greater frequency from cutdowns that had been in place for long periods. Rhee and colleagues [14] reported a 1.4% infection rate (one episode of cellulitis) following 73 cutdown attempts. All catheters were removed within 24 hours.

There is some evidence that the rate of infectious complications decreases when a broad-spectrum antibiotic ointment is applied to the cutdown site. Moran and coworkers[32] found a rate of infectious complications of 18% when topical polymyxin B-neomycin-bacitracin (Neosporin) was used, compared with a 78% rate in a placebo-treated group. In this study, it was also shown that topical antibiotic use results in only a moderate decrease in the incidence of phlebitis (from 53% to 37%) but a significant decrease in the incidence of phlebitis associated with positive cultures (from 86% to 14%). This suggests that phlebitis is primarily a chemical or an irritative process rather than the result of infection. Whatever the cause, the incidence of phlebitis is clearly related to the duration of catheterization. [9] [30] [33] Early catheter removal is a key factor in the prevention of both phlebitis and the infectious complications of venous cutdown. This is especially true of lines inserted during emergency resuscitative treatment. Such lines should be removed as soon as the patient's condition stabilizes and alternative routes exist. [10] [12]

Proper attention to the details of surgical technique will limit the occurrence of minor complications, such as local hematoma, abscess, and wound dehiscence. One can avoid injury to associated structures by selecting a site in which the vein is isolated and specifically avoiding brachial vein cutdown.

CONCLUSION

The venous cutdown is a time-honored, simple surgical technique that is useful in the management of seriously ill patients. It is an excellent means of venous access in children and in markedly hypovolemic patients. Complications are potentially serious but can be controlled by good surgical technique and prompt removal of the catheter following clinical improvement.

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Chapter 24 - High-Flow Infusion Techniques

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Kenneth V. Iserson

High-flow infusion techniques date back to the Vietnam War era. During that period, surgeons placed intravenous (IV) tubing directly into veins to facilitate rapid high-volume infusions. Rapid isotonic volume resuscitation following hemorrhage has been promoted as a means to minimize shock-related organ injury and death. [1] Since the early 1990s, laboratory [2] [3] and clinical investigations [4] have raised concerns that overzealous volume resuscitation of patients with penetrating trauma before definitive control of hemorrhage may increase hemorrhage volume and mortality. However, similar laboratory studies have demonstrated that delayed volume resuscitation is associated with increased metabolic acidosis. [5] Regardless of the optimal timing for fluid resuscitation (i.e., either "as soon as possible" or "after control of hemorrhage"), high-volume techniques remain an important therapeutic intervention for patients with significant volume deficits.

A difficulty in describing high-flow techniques is due to a progressive increase in flow rates described as "high-flow." Whereas 1L/hour of isotonic fluid was once considered a rapid infusion, isotonic fluids can now be placed through peripheral IV lines into normovolemic adults with commercially available equipment at rates exceeding 800 mL/min.[6] "High-volume" infusion of crystalloids may be set arbitrarily at >500 mL/min. Massive transfusion of blood has been traditionally defined as transfusion of an amount at least equal to the recipient's blood volume within a 24-hour period. [7] However, with commercially available equipment, the in vitro flow rate of admixed erythrocytes can exceed 800 mL/min through one line.[8] In one case, a patient survived after receiving more than 12 units of blood product per hour for 30 hours.[9]

IMPORTANCE OF TECHNIQUE

Several acute traumatic and nontraumatic conditions (e.g., gastrointestinal bleeding, septic shock, anaphylaxis) require prompt restoration of circulating blood volume. Hypovolemic shock also results in an increase in intravascular capacity, requiring much more IV fluid to be infused than the volume of blood lost. [10] During resuscitation, sufficient intravascular volume (preferably with adequate oxygen-carrying capacity) to supply nutrients for cellular metabolism must be restored. Placement of one or more small bore peripheral IVs may not be adequate when truly rapid volume replacement is required. In addition, it can be difficult and time-consuming to place more than one IV line (e.g., in patients with severe shock, a history of IV drug abuse, fragile veins, massive swelling, or obesity). [11] In these settings rapid high-volume infusion techniques facilitate maximal crystalloid or blood product infusion, or both, through a minimum number of infusion sites.

ROLE OF HIGH-FLOW INFUSION

High-flow infusion may be used by all clinicians, paramedical personnel, and nurses who treat critically ill patients. Trauma patients, patients with gastrointestinal bleeding, and those in septic or anaphylactic shock are prime candidates. The techniques involved are relatively easy to learn, and the equipment is inexpensive and similar enough to that already used for standard IV fluid therapy that no major barriers should exist to using high-flow techniques when indicated.

Some techniques, especially those using the automated external pressurization devices, reduce the time needed for personnel to infuse fluid by conventional IV techniques.[12] Large peripheral IV catheters (i.e., 12- or 10-ga) are not much more difficult to place than the 14- or 16-ga catheters that are routinely recommended. In the pediatric population, a venous cutdown, whether to directly insert IV tubing or catheters, requires a significant amount of time and skill. [13] Although venous cutdown lines (see Chapter 19 and Chapter 23) can be used in conjunction with high-flow infusion systems, cutdowns are not addressed further in this chapter.

INDICATIONS AND CONTRAINDICATIONS

The primary indication for high-volume infusion is hypovolemic shock. Although the infusion may be needed only for a short period until vital signs stabilize, preparations are often made for the necessary equipment to be available "on standby" in case the patient's condition deteriorates again.

Often it is unclear initially whether the patient requires high-volume resuscitation. Despite historical evidence suggesting massive volume loss, patients may be hypotensive for other reasons or may arrive in a volume-resuscitated condition after a prolonged transport with associated volume administration. If any doubt exists regarding the need for high-volume infusion, short, rapid boluses (10 to 20 mL/kg) of fluid can be given and the patient repeatedly reassessed.

The goals of shock resuscitation are to minimize further volume loss and to maintain organ perfusion until the shock-induced pathophysiologic state can be reversed. In hemorrhagic shock, control of ongoing blood loss is essential. When the blood loss is internal, excessive volume resuscitation accompanied by elevation of blood pressure may exacerbate the blood loss preoperatively: The classic clinical example of this phenomenon is the patient with a leaking aortic aneurysm. Hence volume resuscitation must be closely monitored.

Success with rapid IV infusion techniques is often fleeting. Often the goal is to buy enough time to institute definitive operative intervention. Interpretation of the adequacy of fluid resuscitation, at least at the present time, is largely clinical. Evaluation of the entire clinical picture, including vital signs, urine output, peripheral perfusion, and mentation, is necessary to determine the success of infusion therapy. Occasionally, of course, when a thoracotomy has been performed, direct observation of the central circulation allows an accurate determination of the state of vascular filling.

With rapid infusion consisting primarily of crystalloid solutions, patients sometimes have enough blood loss to leak "water" from bleeding sites. This is essentially a visual manifestation of an extremely low hematocrit and indicates a nearly hopeless prognosis. Otherwise, hematocrit measurements are virtually useless in assessing the intravascular status of unstable patients. Some patients, especially younger ones, continue to have enough vascular tone that they appear to stabilize after an initial large bolus of fluid. These patients must be carefully observed and evaluated, because their condition can deteriorate again without warning.

Rapid high-volume infusion is relatively contraindicated in cases of neurogenic shock following trauma. Because the hypotension of neurogenic shock is not due to hypovolemia, hemodynamics do not improve with administration of large amounts of fluid. Rather, the fluid often collects extravascularly and may lead to pulmonary edema. Patients with hypotension secondary to pericardial tamponade may benefit from administration of small, rapid boluses of fluid until a pericardiocentesis or definitive repair is performed, but large volumes of fluid do not appear to be beneficial. Patients whose hypotension is not due to hypovolemia (e.g., those with pump failure or impaired venous return) can be harmed by the fluid overload that would accompany a rapid, high-volume infusion.

EQUIPMENT FOR HIGH-FLOW INFUSION

The key to performing effective rapid infusion therapy and to obtaining the proper equipment is to consider the entire infusion system, rather than individual parts. Each part of the infusion system must be able to meet the desired flow and time requirements. Based on Poiseuille's law, it is the internal diameter (ID) of the system that primarily determines flow rate. The ID affects flow rates to the fourth power, whereas all other elements—length, viscosity, pressure gradient—affect flow only to the first power. Therefore, to obtain a maximal flow rate, the goal is to optimize the ID of all parts of the system. For example, more rapid infusion can occur by increasing the diameter of a small-gauge IV catheter than by starting a second line with another small-ID catheter. [14]

Although some very large experimental catheters, exceeding 4.5 mm (14 French [Fr]) ID, have been developed and used successfully for infusion in humans, [15] [16] large, commercially available percutaneous IV catheters have a maximum ID of 2.16 mm (12-ga, Vygon Mosquito 123) or 3 mm (9 Fr). [17] Theoretically, an IV catheter for rapid fluid replacement should have its maximum ID maintained throughout its length. This means not only that the main portion of the catheter should have a maximum ID consistent with structural stability, but also that the reduction in ID from narrowing at the catheter tip should be minimal. Some catheters lose more than 36% of their ID in this taper. [17]

Poiseuille's law applies to IV tubing as well as to indwelling catheters. Most standard IV tubing for blood transfusions has an ID of 3 mm. [6] [18] Larger tubing is now available, some with an ID of 3.66 (11 Fr) to 4.0 mm. Using even the 3.66-mm tubing can increase flow rate of saline from 70% (12-ga catheter, pressurized) to 126% (8 Fr catheter, no pressure) over rates with the 3-mm tubing. [6] A comparable difference would be expected with blood products.

Even if large-diameter tubing is used, adding small ID extension tubing can negate the benefit of the other system components. If large-ID IV tubing is used, it is essential that similarly sized extension tubing also be available.

In setting up a high-flow infusion system for either crystalloid fluid or blood, all IV tubing must be of the "Y"-type (i.e., there must be at least two attachments for IV bags). This feature allows one bag to be changed while the other is infusing. At the flow rates achieved by rapid high-flow systems, not having Y-type tubing will normally cut the overall system infusion rate by at least half. Y-type tubing is also necessary to admix blood with warmed saline, as described in the following section.

External Pressure Devices

Pressurizing the fluid being infused increases the fluid flow rate dramatically. [6] [11] This can be accomplished by manually squeezing the bags or using commercial pressure devices. [10] The most practical means of adding pressure to a high-flow IV system is with external pressure devices (Fig. 24-1). The standard maximal external pressure exerted by these units is 300 mm Hg. Most centers use pneumatic pressurization with three-way stopcocks. Although easy to use, they do not maintain a constant pressure on the fluid while the bag empties. Other, more elaborate external pressure devices have been advocated, including blood pumps attached to a constant air-pressure source. [19] More recently, these pressurization devices have not only been powered by wall air, but have also been contained in a rigid metal or plastic container for safety, more rapid access, and extremely rapid pressurization and depressurization (Infuser-1 and Alton Dean Infuser, North Salt Lake City, UT). These devices have the advantage of maintaining constant pressure on the fluid bag during emptying. Using the air-pressure devices dramatically decreases the time needed for changing bags, thus enhancing the effective flow rate (Table 24-1). [12] [20]

Blood Warmers

In nearly all high-volume infusions, blood products are administered. Rapid infusion of cold blood (which is stored at 4°C) may be associated with significant complications including ventricular fibrillation and cardiac arrest. [21] [22] Hypothermia is a major, yet often unappreciated problem during the resuscitation of seriously ill patients, particularly those who require multiple blood transfusions. [23] Blood warmers heat blood either before the infusion (prewarming) or during the infusion (in-line). Most commonly available warmers are the in-line type, and most of them significantly slow the flow rates of high-flow systems. [23] [24] [25] Some blood warmers have more rapid flow rates. [26]

Prewarming the blood is cost-effective and simple, requiring less setup time for resuscitation and no special training beyond standard nursing skills. One proposed method for keeping flow rates high while warming blood to approximately body temperature is to dilute the blood with an equivalent volume of 70°C saline. [2] [3] [4] The rapid equilibration of temperatures between the two mixing fluids prevents damage to the erythrocytes. [27] [28] [29] The warmed saline-blood mixture (approximately 37°C) must then be infused rapidly to prevent cooling to room temperature. *All personnel using this technique should be cautioned that the 70°C saline must never be used for direct infusion* (Fig. 24-2).

Figure 24-1 Three types of external pressure devices. *A*, Infuser-1. *(As modified in Iserson KV, Reeter A, Woods W, Criss E: Pressurization of IV bags: A new configuration and evaluation for use. J Emerg Med 3:89, 1985.) B*, Infusable *(Biomedical Dynamics Corp., Minneapolis, MN). C*, Alton Dean infuser *(North Salt Lake City, UT)*.

Unlike commercially available systems, this system permits multiple patients and multiple IV lines in each patient to be infused with warmed blood simultaneously. The only cost is for the single warming oven and the minimal cost of the "Plasma Transfer Sets", i.e., the IV tubes used for each unit (see later).

When transfusing cellular blood products rapidly, it is essential not only to warm the fluid but also to dilute it adequately to reduce the incidence of erythrocyte hemolysis. Cellular destruction in blood flow systems is directly proportional to the magnitude of shear stress and cellular-solid surface interactions. As hematocrit and plasma protein

TABLE 24-1 -- Mean Time (Minutes to Seconds) for Two Fluid Systems

System	Pump-up/Drain (First Liter)	Take Down/Setup	Pump-up/Drain (Second Liter)	Total Time
System 1:	1:16	0:22	1:10	2:48
3.66-mm ID tubing				
Pneumatic pressure device				
12-ga (0.0839-in. maximum ID)				
IV catheter				
System 2:	4:19	1:37	3:37	9:33
3.0-mm ID tubing				
Hand-pumped pressure device				
14-ga (0.0563-in. maximum ID)				
IV catheter				

P values	<0.0005	<0.0005	<0.0005	<0.0005

ID, internal diameter

Adapted from Iserson KV, Criss E: Combined effect of catheter and tubing size on fluid flow. Am J Emerg Med 4:238, 1986; and Iserson KV, Newberg CE, Clemans SB: Non-standardization of the manufacture of intravenous catheters. J Clin Engineering 12:367, 1987.

concentration decrease with dilution, so does the risk of hemolysis. [30] [31]

PROCEDURE

Usually the most difficult part of initiating a rapid, high-volume fluid system is ensuring adequate preparation through in-service training of clinicians and nurses, stocking essential components, and setting up the components to be used. The equipment should be located conveniently close to the patient and within reach of the practitioners.

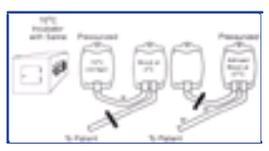

Figure 24-2 (1) During all handling of blood, standard universal precaution procedures should be followed. (2) Remove a 250-mL bag of normal (N) saline from the 70°C incubator. Check the bag for clarity, discoloration, particulate matter, obvious decreased volume, and cracking of the outer wrapper. Do not use if problems are found. (3) Check the incubator thermometer to ensure that its temperature is between 65° and 75°C. Do not use if above 75°C. (4) "Spike" the packed red blood cell (PRBC) unit from the blood bank with one end of a Fenwal plasma transfer set (No. 4C2243). Spike the other end of the transfer set into the 70°C, 250-mL N saline bag. Open the clamp, raise the saline bag above the level of the PRBC bag, and manually squeeze the saline (or use external pressure bag or other external pressure device) into the PRBC bag. (5) A towel or potholder-type kitchen glove may be used to squeeze the 70°C saline bag, if desired. (6) After all of the 70°C saline is transferred into the PRBC bag, tightly close the clamp on the transfer set tubing. (7) Do not remove the transfer set tubing spike from the PRBC bag (to prevent contamination). The now-empty saline bag may be removed from the other end of the transfer set and discarded, or simply left in place. (8) On the patient's side, spike the unused part of the PRBC bag (now admixed with 70°C saline) with one limb of the Y-type blood set already attached to the patient. The admixture temperature will be approximately 37°C. (9) Close the clamp on the other limb of the Y set, which has been infusing standard warm or room-temperature normal saline into the patient. (10) If the blood is to be rapidly infused, pressurize the external pressure bag or device. (11) Open the clamp on the PRBC admixture bag to begin infusion of 37°C PRBC unit into the patient. (12) Additional units of warm-admixed PRBC may be prepared using the same technique. Close the Y set clamp, remove the empty admixed PRBC bag, and replace it with the second unit. Open the clamp and begin infusing the second unit. (13) Infuse the blood into the patient as rapidly as necessary. (14) For each unit of warm-admixed PRBC infused, document in the patient's record that the patient received "1 unit of PRBC admixed with 250 mL of N saline at an infusion temperature of approximately 37°C." Include start/stop times of infusion and the name of the person preparing and administering the infusion. *(From Iserson KV, Knaut MA, Anhalt D: Rapid admixture blood warming: Technical advances. Crit Care Med 18:1138, 1990. © 1990, Williams & Wilkins, Baltimore.)* Following admixture, the infused blood will be between 35° and 40°C. *Cautions:* (1) Keep the blood bag hanging when adding saline. (2) If the blood is admixed, it must be infused or discarded to avoid contamination. (3) Do not mix the blood manually or shake the bag. (4) *Do not* infuse 70°C saline *directly* into patients!

Although new equipment items continue to be developed to assist in high-volume infusion, all items are limited by the same essential principle—the minimum ID of the system must be as large as possible.[15] [16] Equipment that only offers increased pressure, decreased setup and takedown time, or decreased system tubing length should be accompanied by an appropriately large system ID.

Given appropriate preparation, the next challenge is establishing IV access with a large-bore catheter. Catheter placement should proceed with the largest size catheter that can be quickly placed. Even if only a small-bore catheter can be placed initially, it can often be replaced by a larger catheter using the Seldinger technique (see Chapter 22). Furthermore, alternative sites can be sought for a larger-bore catheter as volume resuscitation begins through a smaller-bore catheter. Ideally, a 10- or 12-ga, 9 Fr or larger diameter catheter is placed in either a peripheral or, for central access, femoral vein. Central veins about the neck should be avoided, if possible, because extravasation with rapid flow rates may lead to a large hematoma and possible airway obstruction.

Once one or more catheters are in place, large-bore Y-type tubing, no smaller than 3.66 mm ID, should be attached. If an extension tube is needed, it must have at least as large an ID as the rest of the tubing. Prewarmed 1-L bags of crystalloid solutions (ideally at 37°C) should be initially hung in an external pressure device.

When erythroctye units are available, they can be diluted 1:1 with 70°C preheated 0.9 normal saline. (*Note*: Do not use lactated Ringer's solution, since the calcium causes clotting.) When performing this procedure, use a special system (e.g., Fenwal plasma transfer set, No. 4C2243, Fenwal Laboratories, Deerfield, IL) to guarantee that the hot saline will not be directly infused into the patient (Fig. 24-2).[29] [30] Many types of laboratory incubators can be used for preheating the saline. A constant temperature monitor should be available to ensure adequate heating. According to tests by the manufacturer, bacterial growth within the unopened heated saline units should not occur for at least 2 weeks if the units are left in their overwrap packaging. [28] When the 1:1 dilution is used, the admixed blood is then at approximately 37°C and ready for rapid infusion. Mistakes may be minimized by preheating only one particular-sized bag (250 mL is usually the right amount for diluting the erythrocyte unit) and premarking the bags for dilution with bright orange or yellow tape. Heated saline bags should be discarded if not used after 2 weeks to avoid chemical leaching from prolonged heating. [32]

If a rapid-infusion system is used in one part of an institution, it is essential that other interactive units (e.g., the operating room and intensive care unit) be made aware of it. In addition, any equipment that the other units use in managing critical care patients must be compatible with that used for rapid infusion in the emergency department.

COMPLICATIONS OF HIGH-FLOW INFUSION

The complications of high-volume infusion include all those related to IV access and fluid infusion. Complications specifically associated with high-volume infusion include:

1. The catheter may be misplaced, resulting in large-volume infusion into a closed space, such as the chest or pericardium. Rather than being resuscitated by the rapid fluid infusion, the patient may deteriorate as a result of, and in proportion to, the amount of fluid infused.
2. Excessive fluid may be inadvertently infused. Unfortunately, there is no totally satisfactory way to monitor intravascular volume in the acute setting, and excessive fluid resuscitation is surprisingly easy. Close attention to vital signs, pulmonary status, and urine

output is important. When available, central venous pressure monitoring or, preferably, Swan-Ganz catheter monitoring may be used, but these are generally not available in the acute resuscitation setting.
3. Problems related to massive blood transfusion may occur. The major and most immediate problems include hypothermia (with resultant dysrhythmia) and coagulopathies. These may be ameliorated with the use of warmed blood and the timely use of fresh frozen plasma (see Chapter 29).

The complication rate for high-volume fluid therapy in patients with severe hypovolemia is unknown. Candidates for rapid infusion generally have conditions associated with poor outcomes. Limited information on massive, rapid fluid boluses in otherwise healthy animals suggests that there are few problems that do not resolve once the infusion is stopped.[33] However, in the injured patient, cellular changes accompanying injury (e.g., pulmonary, neurologic) may not allow reversal of fluid-induced changes as easily. Further, concerns have been raised that over-resuscitation may exacerbate hemorrhage through various mechanisms.[2] [3] [4]

If a patient is believed to be overloaded with fluid, normal techniques to reduce the amount of fluid (e.g., stopping the infusion, using diuretics, and appropriately using cardiovascular or respiratory support) should be instituted.

CONCLUSION

Rapid infusion of IV fluids is a common occurrence in the management of acutely ill or injured patients. While enthusiasm for rapid infusion has been tempered by the lack of proven benefits on outcome in hemorrhagic shock studies, its skillful performance remains a requirement of all who treat critically ill patients. Correct performance of rapid IV infusion requires stocking the proper equipment; regularly training personnel; and, perhaps most important, understanding by all involved that flow rate is most affected by the minimum ID component in the system.

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Chapter 25 - Indwelling Vascular Devices: Emergency Access and Management

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Pino D. Colone

The use of indwelling vascular lines is becoming an increasingly important component of modern medical care. These lines provide routes for short- and long-term infusion of hyperalimentation fluids, chemotherapeutic agents, antibiotics, blood products, and anesthetic agents. [1] In addition, they provide access for such life-saving procedures as hemodialysis (HD) and plasmapheresis. The increasing complexity of medical care, coupled with an emphasis on outpatient care delivery, has resulted in an increased use of indwelling lines as well an increased likelihood that patients will present to the emergency department (ED) with one of these devices in place. It is estimated that more than 5 million central venous catheters (CVCs) and approximately 386,000 tunneled, cuffed CVCs were inserted in the United States[2] [3] during 1993. Other totally implantable devices are routinely seen in the ED. For the purpose of this chapter all implanted devices or intermediate- to long-term catheters for vascular access will be considered vascular access devices (VADs). Arteriovenous (AV) fistulas and AV grafts will also be discussed due to their similarities to the VADs. It is important that all emergency physicians are familiar with the devices in common use, the best methods to access them in an emergency, and their complications.

HISTORICAL PERSPECTIVE

A major advance, which would ultimately lead to the development of several types of indwelling catheters, was the introduction of Silastic (silicone rubber). This biocompatible material is an ideal substrate for an intravenous catheter as it is chemically inert, antithrombogenic, rigid at room temperature, and pliable at body temperature. In 1973, Broviac and colleagues used this material to develop a 90 cm × 0.22 mm indwelling right atrial catheter for total parenteral nutrition (TPN). [4] This prototype catheter was widely used for hyperalimentation and served as a template for catheters designed for other uses. In 1979, Hickman reported experience with a 0.32 mm catheter that could be used for blood products and drug therapy for recipients of bone marrow transplants. [5] Further modification resulted in the double-lumen Hickman, a fusion of the Broviac catheter [internal diameter (ID) 1.0 mm, external diameter (ED) 2.2 mm], and the Hickman (ID 1.6 mm, ED 3.2 mm). The increased diameter requires cannulation of a large vein, but it allows concomitant infusion of TPN through one port and IV drugs and blood products through the other. Another major advance was the development of totally implantable devices, first described by Fortner in 1972. [6] Since that time they have become a mainstay of treatment in oncology patients. Totally implantable devices allow less painful intravenous access, and improve quality of life by permitting unrestricted mobility.

In addition to their many uses for administration of drugs and blood products, indwelling vascular lines have been used for short- and long-term HD. Temporary access for HD via an external AV shunt was pioneered by both Quinton and colleagues [7] and Scribner and associates [8] in 1960. This original shunt was composed of a loop of tubing lying on the volar forearm connecting the radial artery to a wrist vein. Although it provided effective dialysis, it was associated with a high rate of infection, thrombosis, and restriction of patient activity. Brescia and coworkers [9] then introduced the peripheral subcutaneous (SQ) autogenous AV fistula in 1966. This Brescia-Cimino internal fistula used a side-to-side anastomosis (the current procedure of choice for long-term HD, connecting the radial artery to the cephalic vein in the nondominant hand). Several catheters have been developed for short-term dialysis. Erben and colleagues [10] described routine use of percutaneous cannulation of the subclavian vein for HD in 1969 and in 1979 Uldall and associates [11] reported development of a single-needle, subclavian, HD catheter.

INDWELLING VENOUS ACCESS DEVICES (VADS)

VADs are typically chosen based on the least invasive, smallest catheter with lowest complication risk that will last the length of therapy anticipated. [12] Length of therapy is often the major consideration when choosing a device. Long-term VADs consist of cuffed, tunneled right atrial catheters and implantable ports. Medium-term VADs include midline catheters (lasting weeks), peripherally inserted central catheter (PICC) lines (lasting months), and Silastic subclavian/jugular catheters. Short-term devices (not reviewed in this chapter) include short peripherals, SQ (butterfly), subclavian and jugular catheters, and temporary epidurals. Additional vascular access devices include those used for dialysis, as well as AV fistulas and grafts.

Cuffed, Tunneled, Right-Atrial Catheters (Broviac, Hickman, Hemocath, Leonard, Raaf)

There are several cuffed, tunneled, right-atrial (RA) catheters, each with differences tailored to specific applications. The **Broviac** (Fig. 25-1) is an all-Silastic (polymerized silicone rubber) single-lumen catheter with a 1.0 mm ID. It is 90 cm with a thin intravascular segment (55 cm). The **Hickman**, also a Silastic single-lumen catheter, has a 1.6 mm ID lumen. This allows for more frequent blood sampling without jeopardizing luminal patency. [13] A double-lumen variation of the Hickman also includes a small-bore lumen permitting drug infusion through the smaller lumen while the larger lumen is used for blood sampling and transfusions. [14]

Hemocath/Permacath (Fig. 25-2) has the largest bore of the RA catheters, 2.2 mm ID. Quinton Instrument Co. manufactures it for HD, plasmapheresis, long-term nutritional support, and pain control. Although some clinicians consider cuffed double-lumen HD catheters an alternative to AV shunts and fistulas for long-term HD, [15] the Dialysis Outcomes Quality Initiative (DOQI) panel concluded that the mean technique life span for cuffed tunneled catheters was 1 to 1.5 years. Thus, they should be reserved for bridge access only and discouraged from use as a permanent access. [16]

Figure 25-1 Broviac pediatric single-lumen catheter (4.2 Fr) with Dacron cuff.

Insertion of the RA catheters is typically done in an operating or interventional radiology suite. The device is introduced via the upper anterior abdominal wall and tunneled subcutaneously to enter the superior vena system via the cephalic, subclavian, internal or external jugular veins. The distal tip of the flexible catheter is advanced to the distal superior vena cava (SVC) or into the mid-RA area. The SQ tunnel isolates the venous puncture site from the skin and decreases the potential for bacterial contamination. The Dacron cuffs (one near the venous entrance site and one near the skin exit site) anchor the catheter and are also believed to inhibit colonization of the CVC by skin organisms. [17]

The advantages of an RA catheter include ease of insertion and use, minimal interference with patient activity, low incidence of major complications or unintended dislodgment, ease of removal, and potential repair via a kit. Disadvantages include the need for regular maintenance and the fact that some patients find it cosmetically unacceptable.

Totally Implantable Venous Access Devices (TIVADs)/Ports (Port-A-Cath, Infuse-A-Port, Mediport)

Since 1983, implanted ports have become the mainstay of treatment for long-term cancer therapy. TIVADs are tunneled RA catheters, but differ from Broviac-Hickman catheters in that they have a SQ portal with a self-sealing septum (Fig. 25-3 Fig. 25-4 Fig. 25-5) that may be accessed by needle puncture through intact skin. They require less manipulation and have lower complication rates than other Silastic catheters. [18] Cosmetically, they are superior to external tunneled catheters, require less maintenance, and afford patients greater freedom of movement. Ports are often used when prolonged access is necessary.

TIVADs may be inserted on an outpatient basis under local anesthesia using a SQ tunnel or an open cutdown. Many physicians believe that the cutdown technique offers speed (mean placement time 15 minutes), safety (negligible risk for pneumothorax), and low cost, with avoidance of early and late complications. [19] Placement is typically in the nondominant arm unless there is vein occlusion or planned radiation therapy on the contralateral side. Once inserted, the ideal position of the catheter tip is the junction between the right atrium and the SVC. As with any catheter, incorrect positioning increases the potential for complications, including deep vein thrombosis. [20]

Disadvantages of this type of device include the need for a specific access needle and special training for users of the device, the small gauge (20–22) of the access needle—which limits fluid infusion rates—and the potential for SQ extravasation. [15]

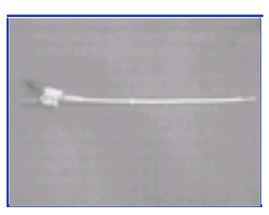

Figure 25-2 Permacath (Quinton Instrument Co.) double-lumen catheter (13.5 Fr).

Peripherally Inserted Central Catheters (PICCs) (Nontunneled, Noncuffed)

PICCs were first described in the 1970s. Their popularity grew immensely in the 1980s. Originally they were developed for the neonatal population, given their small size and minimal invasiveness. [21] Subsequently their use expanded into the adult arena and currently includes prolonged antibiotic therapy, intravenous fluids (IVFs), chemotherapy, TPN, and delivery of medications that are irritating to the peripheral vessels.

PICCs (Fig. 25-6) are made of two substances, either polyurethane (Intracath) or silicone (Intrasil), and are radiopaque, measuring 50 to 60 cm in length with an outside diameter of 2 to 7 French (Fr). The catheter may be single- or double-lumen configuration, and can be open- or close-ended or valved (e.g., Groshong). An open-ended PICC cannot prevent feedback of blood into the catheter and therefore must be flushed one or more times daily with heparinized saline. The Groshong three-way valve reduces blood backup into the catheter and therefore requires flushing as little as once a week. The most common type of PICC line in use today is the 5-Fr, double-lumen, closed-ended catheter.

The device selected should be based on the number of lumens necessary for therapy, recognizing that the potential for infection increases with lumen number. An access site is chosen based on many factors including the suitability of target vessels, the patient's body habitus, handedness, ability to manage self-care, comorbid conditions, the desired infusion rate, the number and compatibility of concurrent infusions, the infusate characteristics, and the estimated duration of therapy. Infusate that is hyperosmolar (TPN) or vesicant requires rapid dilution. As such, the tip must be in the SVC, where the estimated flow is 2000 mL/minute. PICC lines are most frequently placed in the superficial veins proximal to the antecubital fossa (usually the basilic or the cephalic). However, they may also be placed translumbar or

transhepatically when the SVC is thrombosed or occluded.[22]

PICC line advantages include usefulness in a wide variety of clinical situations, and ease of placement, use and maintenance. It is an excellent vehicle for medium-term intravenous therapy.

Midline Peripheral Catheters

Midline catheters are often confused with PICC lines. They are also placed peripherally in the superficial veins of the antecubital fossa or upper forearm. Midlines are typically 20 cm in length, with the tip terminating near the axillary vein. They are designed for short-term use; however, they last longer than a peripheral IV. Since it does not enter the central

Figure 25-3 Norport System single-lumen catheter and specially tapered Huber needle.

circulation with high flow, the delivery of medication types is limited. Differentiating between these two catheters in situ may be difficult, because the outward appearance is similar. Obtaining an x-ray film to determine tip placement will differentiate between the two types of catheters.

Hemodialysis Catheters

Vascular access has been referred to as the Achilles heel of the renal patient. Clinical practice guidelines of the National Kidney Foundation-Dialysis Outcomes Quality Initiative (NKF-DOQI) recommend early AV fistula construction and avoidance of catheters for permanent or prolonged vascular access. [23] However, in a recent study it was demonstrated that more than half of the patients began dialysis using a central catheter because a well-developed AV fistula was not available. [24] The risk of requiring three or more vascular accesses is almost double among patients who start HD using a central catheter. These patients are more likely to present to the ED for problems related to vascular access.

AV Fistula

An autogenous AV fistula is constructed by connecting an artery (usually the radial) to a nearby vein. The radial-cephalic (Brescia-Comino forearm) fistula is the most common choice for initial fistula placement. A secondary choice is the brachial-cephalic—if that is unsuitable, the proximal thigh is a consideration. Over time, the venous portion of the shunt is subjected to high pressure and flow becomes arterialized (hypertrophied and dilated), rendering it suitable for repeated vascular access. Full epithelialization of the shunt does not occur for 3 to 6 months.

An AV fistula is the preferred means of vascular access for HD, yet national data show that only 23% of patients used an AV fistula in 1997. [23] There is a general consensus that the use of the autogenous fistula for vascular access is associated with the longest period of graft patency and with relative freedom from thrombotic and infectious complications. In one study, the mean length of patency was 3.1 years for wrist fistula versus 2.6 years for forearm, with the overall mean length of patency for autogenous AV fistulas being 2.85 years. [24]

AV Graft

If a forearm Brescia-Comino fistula cannot be constructed or has failed, an AV bridge graft using a donor vein or synthetic material is a well-accepted alternative. A standard graft is 6 to 8mm in diameter and usually positioned in a U-shaped SQ tunnel in the forearm. The graft is attached by end-to-side

Figure 25-4 Port-A-Cath double-lumen catheter segment.

anastomoses to the brachial artery and antecubital vein. If no suitable antecubital vein is available, a straight bridge graft between the brachial artery and either the axillary or basilic vein is often used.

Compared with AV fistulas, AV grafts have a significantly higher incidence of thrombosis, infection, pseudoaneurysm formation, and limb loss with a significantly lower mean length of patency.[25] However, they have a low incidence of aneurysm formation and are comparatively easy to revise.

Several synthetic materials are used for grafts with polytetrafluoroethylene (PTFE) being the most common. The estimated life span of a PTFE graft is 3 to 5 years, [26] but in clinical practice is often less than 2 years. [24]

ACCESSING VADS IN THE EMERGENCY DEPARTMENT

In general, when IV access is required in patients with VADs, standard methods of peripheral access should be attempted first in order to preserve the lifespan of the VAD and avoid complications. However, VADs and even AV fistulas and shunts may be accessed in emergency situations for phlebotomy and infusion of medications and fluids. Because of the previously discussed complications of infection and catheter malfunction, dislodgment and fracture, only personnel with the requisite knowledge and skill should access VADs. When VADs are accessed, povidone-iodine and alcohol are acceptable cleansing agents.

The need to administer parenteral medications to patients lacking other means of vascular access is the most common reason to access a VAD in the ED. Assuming that proper access methods are used to prevent infection (described later), the greatest risk associated with medication administration is sludging in the catheter with resultant occlusion. Medications should always be followed by a saline flush both to clear the catheter and to ensure that the medication reaches the circulation. Medications that are known to be incompatible when mixed (e.g., calcium and bicarbonate) should not be administered concurrently, even through separate lumen of multilumen catheters. Taylor and Taylor[27] state that diazepam and phenytoin crystallize irreversibly on contact with the silicone catheter walls of long-term venous access catheters and TIVADs, thereby necessitating catheter replacement. In contrast, the manufacturers of these devices (Evermed, Pharmacia) hold that crystallization does not occur and that the silicone catheter wall absorbs these drugs without damage or the need for removal.

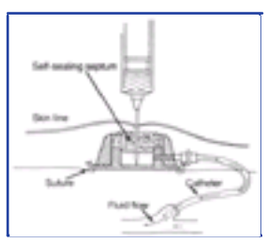

Figure 25-5 Port-A-Cath system (Deltec, Inc., St. Paul, MN). The Port-A-Cath system is accessed by inserting a Huber needle through the skin and portal septum.

Blood specimens including cultures can also be obtained in the ED assuming appropriate access technique is used. Phlebotomy requires stopping infusions, removing fluid occupying dead space in the catheter, and following the blood draw with a flush of heparinized saline.

Accessing Long-Term Venous Access Catheters

The catheter (with the exception of Groshong catheters) is first clamped to prevent air embolism. Patients usually carry their own clamps; however, a hemostat without teeth will suffice. In an emergency, sterile tape or tubing wrapped around the teeth of a hemostat also protects the catheter. The cap is removed, and a 10-mL syringe of sterile water or normal saline is attached. Three to 5 mL of solution are injected and then withdrawn to ensure patency. More pronounced aspiration might be necessary to ensure patency of Groshong catheters. This procedure is done slowly with a syringe at least 10 mL in volume to avoid damage to the cap area. [27]

Phlebotomy is accomplished by withdrawing dead space solution, reclamping, and using a separate syringe to remove the desired amount of blood. [27] Bolus medications are then injected and IV solutions infused through the catheter, which is clamped whenever unattached. A 5-mL normal saline flush should be delivered between medications. On completion, 3 to 5 mL of heparin (100 U/mL) are injected, the line is clamped, and the cap is repositioned. [27] [28] Groshong catheters should not be flushed with heparin but instead should be flushed briskly with 5 to 20 mL of saline.

Accessing Implantable VADS

The procedure for accessing totally implantable VADs is unique, because these devices are not external. Instead, a circular reservoir (cylinder) lies subcutaneously on the anterior chest wall. The cylinder is first palpated, and the overlying skin is prepared with povidone-iodine solution. A 10-mL syringe filled with sterile water or normal saline is attached to connecting tubing, which in turn is applied to a 19- to 22-gauge, 90-degree tapered (Huber) needle. The Huber needle is a specialized needle designed for use with the TIVAD to prevent damage to the portal septum. It has a 90-degree bend with a slightly curved tip, opening on the side rather than on the end. Most importantly, the Huber is a noncoring needle. This avoids damage to the Silastic septum, allowing up to 2000 punctures. The TIVAD should never be accessed with a standard 19-gauge needle unless in a code situation when the Huber is not available immediately. A clamp should be applied to the connecting tubing whenever the system is open. Air is expelled, and the Huber needle is inserted through the reservoir septum. The needle is inserted slowly and steadily through the diaphragm to the back of the reservoir, approximately 1.25 cm (or 0.5 inch) through the dome. [29] Although incomplete perforation of the septum will block flow, substantial pressure also may damage the back of the device and bend the needle tip. The clamp is removed slowly, and 5 mL of solution is injected to ensure patency. If patency is not easily demonstrated, the same measures described under Accessing Long-Term Venous Access Catheters may be used, including positioning, Valsalva maneuver, heparin flush, and low-dose thrombolytic therapy. [27] [28]

Once the solution has been injected, gentle negative pressure is applied to demonstrate the backflow of blood. The Huber needle is then stabilized by building 4 × 4-inch gauze about the needle and further reinforcing with 2.54 cm (1-inch) silk tape. Phlebotomy is performed through the extension tubing after first removing 8 to 9 mL of blood with a separate syringe. IV solutions may also be delivered through extension tubing, although the rate of flow will be limited by the Huber needle's radius. A 5-mL normal saline flush should be delivered between medications. The procedure is completed with a 3- to 5-mL heparin (1000 U/mL) flush [27] and with removal of the Huber needle.

Accessing Percutaneous Multilumen Catheters

Percutaneous multilumen central catheters are used infrequently by outpatients. They are usually placed in the subclavian vein to facilitate patient comfort. One port is available for each lumen, and after povidone-iodine preparation is completed, access is gained by either inserting a needle or syringe into the protective cap or removing the cap entirely. A 5-mL normal saline or sterile water flush and verification of backflow precede all subsequent procedures. Phlebotomy is performed through the proximal 18-gauge lumen to prevent mixture with medications being delivered through the other two ports. The proximal lumen port of the Arrow multilumen catheter is white and is the longest of the three tails. Tails attached to the more distal lumina are blue and brown. IV infusions are delivered in similar fashion, and a normal saline flush is injected between medications. The procedure is terminated with a 3- to 5-mL heparin (100 U/mL) flush.

Accessing Arteriovenous Fistulas, Shunts, and Catheters

AV fistulas, shunts, and Uldall and Manhurkar catheters are placed in patients who require hemodialysis and represent the sole access for that purpose. Consequently, routine use of

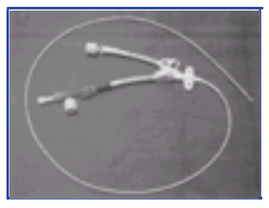

Figure 25-6 Double-lumen peripherally inserted central catheter (5.0 Fr, 18 ga).

these sites for phlebotomy and fluid administration is strongly discouraged. In fact, venipuncture in the same extremity as a patent AV fistula is not recommended, except for the veins in the dorsum of the ipsilateral hand. When standard IV access cannot be obtained under emergency circumstances, however, fistulas, shunts, and catheters may all be used to administer IV solutions and medications. If possible, fistula patency should first be ascertained by noting a bruit and palpable thrill, although these signs may not be appreciable if the patient is in extremis.

Prepare the area overlying the fistula with povidoneiodine solution and access the fistula with the smallest gauge needle appropriate to the task. [30] Puncture 1 to 2 cm from the anastomosis ends nearest the venous side, and avoid aneurysmal sites. [30] When complete, monitor the area for hemorrhage, and apply local pressure to avoid significant bleeding. AV shunts are accessed similarly by placing the smallest needle possible into the catheter, bridging arterial and venous circulations. Local pressure should be applied for 5 minutes after completing the procedure. Uldall and Manhurkar catheters are used in much the same way that multilumen central catheters are accessed. The retaining cap on each arm may be either removed or injected. Up to 5000 U of heparin are present within the two lumen, and so it is imperative that aspiration be performed before administering fluid or medications. After use, flush each catheter arm with 10 mL of normal saline and instill 1.5 mL of heparin solution (1000 U/mL) into each catheter arm.

COMPLICATIONS OF VADS

The use of VADs is now sufficiently commonplace to ensure that patients with these devices will present to the ED on a regular basis. Given a complication rate of 4% to 10%,[31] it is essential that emergency clinicians be aware of these complications and their management. Fifty-two percent of reported complications are associated with health care practitioner technique; 12% are associated with device failure; 6% are related to actions taken by the patient, as well as pathophysiologic events (such as thrombosis); and 30% have an unidentified cause. [32]

Complications of VADs include (1) infection, (2) clotting abnormalities, (3) malfunction, and (4) miscellaneous problems (Table 25-1).

Infection

Infection is the most common complication leading to CVC removal and potentially the most serious. Once the organisms

TABLE 25-1 -- Complications of Indwelling Vascular Access Devices

Infection	Coagulopathy	Malfunction	Miscellaneous
Skin/exit site	Bleeding	Occlusion	Embolism
Reservoir/pocket	Local puncture site	Medication delivery failure	Air
Tunnel	Arterial bleeding	Precipitants	Thrombus
Catheter tip/lumen	Over-heparinization	Thrombosis	Catheter
Sepsis	Heparin rebound	Failure to infuse/withdraw	Fragment
	Thrombosis	Pinch off syndrome	Arrhythmias
	DVT	Steal syndrome	Cutaneous dacron
	Fibrin sheath	Malposition	Cuff erosion
			Catheter dislodgment

DVT, deep venous thrombosis

spread beyond the confines of the device itself, the danger to the patient increases significantly, with an estimated case fatality rate of 10% to 20%. [24] With each access there is potential for local, device, or generalized infection with subsequent line failure. The definitions of CVC-related infections are not uniform, making it difficult to compare results of various investigations. However, it is generally agreed that risk factors for infection include site of insertion; hospital size; duration of catheter placement; type of catheter; and patient factors. Most studies show a lower incidence of infection for completely implanted CVCs than for external systems, presumably due to lack of direct access of cutaneous organisms with TIVADs. The rates of CVC infection tend to be highest in the first 3 months following insertion, with skin flora being most common.[31] [33] The rate of infection decreases significantly, reaching a plateau after 5 to 6 months. [33] The most common infecting organisms are listed in Table 25-2 .

Clinical findings are unreliable in the diagnosis of infection secondary to VADs. Fever, rigors, and elevated white blood cell count may be sensitive but not specific, whereas purulent drainage at the insertion site may be specific but not sensitive. Therefore, evaluation beyond physical examination alone is essential when infection is suspected, including blood cultures, possibly catheter segment culture, and transesophageal echocardiography if valvular vegetations are suspected (*Staphylococcus aureus*, persistent bacteremia or fungemia post-catheter removal, or lack of clinical improvement).

TABLE 25-2 -- Microorganisms Causing Indwelling Catheter Infection

Bacterial	
Gram-positive cocci	*Staphylococcus aureus, Staphylococcus epidermidis, Streptococcus faecalis, Streptococcus bovis,* group C streptococci, and viridans streptococcus
Gram-negative bacilli	*Pseudomonas aeruginosa, Klebsiella* sp., *Acinetobacter* sp., *Serratia* sp.
Gram-positive bacilli	*Bacillus cereus, Bacillus laterosporus, Corynebacterium* sp.
Atypical	*Mycobacterium neoaurum, Mycobacterium fortuitum, Mycobacterium chelonae*
Mycotic	*Malassezia furfur, Malassezia pachydermatis, Aspergillus fumigatus, Aspergillus flavus, Candida albicans*

Two sets of blood cultures are typically drawn, one set through the catheter itself and one from a peripheral site. Positive blood cultures for *S. aureus*, coagulase-negative staphylococci, or *Candida* species, in the appropriate patient setting and in the absence of another identifiable source of infection, should increase the suspicion for catheter-related bloodstream infection. [31] When blood obtained from a CVC yields a colony count at least 5- to 10-fold greater than that for blood obtained from a peripheral site, this suggests a catheter-related source of infection. [34] Similarly, catheter infection should be assumed if the peripherally derived blood culture are negative and the CVC-derived cultures are positive. [33] Not only is blood drawn from the catheter more likely to yield a positive culture, this may occur earlier in the course of infection. One study demonstrated 16 of 17 patients with CVC-related infections had positive blood culture results at least 2 hours earlier from the CVC than from peripheral blood cultures. [35]

Infusate-related bloodstream infection is uncommon and is defined as the isolation of the same organism from both infusate and separate percutaneous blood cultures, with no other source of infection. When this diagnosis is suggested, cultures of the infusate should be obtained in addition to catheter and peripheral cultures.[31]

Although it has been common practice to immediately remove a CVC in the setting of acute infection, the need for removal has been called into question. [35] The CVC should be removed and cultured if the patient is immunocompromised or severely ill (e.g., sepsis, shock) or if there is purulent discharge or erythema of the catheter exit site. The CVC should also be removed in a patient with sepsis and without other source of infection. If blood cultures are positive, or if the CVC is changed over a guidewire and has significant colonization, the catheter should be removed and cultured, and a new catheter should be placed at a new site. Without evidence of persistent bloodstream infection, or if the infecting organism is coagulase-negative *Staphylococcus* and there is no suspicion of local or metastatic complications, the CVC may be left in place.[31]

If a CVC is removed, it should be cultured to determine the offending organism. The most widely used laboratory technique for the clinical diagnosis of catheter-related infection is the semiquantitative method, in which the catheter segment is rolled across the surface of an agar plate and colony-forming units (CFUs)

are counted after overnight incubation. Quantitative culture of the catheter segment requires either flushing the segment with broth, or vortexing in broth, followed by serial dilutions and surface plating on blood agar. A yield of 15 CFUs or more from a catheter, by

semiquantitative culture, or a yield of 10[2] or more from a catheter by quantitative culture, with accompanying signs of local or systemic infection, is indicative of catheter-related infection.[31]

HD catheters deserve special consideration. The process of HD requires several connections to the graft, thereby increasing the risk of infection. The rate of bacteremia in HD patients attributed to the graft varies from 48% to 73%. The incidence is highest when central venous dialysis catheters are used. Native AV fistulas carry the lowest risk of infection. Unfortunately, prosthetic AV fistulas (i.e., prosthetic grafts) are more commonly used in the United States. [36] As with other CVCs, empirical antibiotic therapy should be initiated based on epidemiologic and patient factors, followed by narrowed spectrum after isolation and determination of sensitivities. The most common infecting organism is *S. aureus*. There has been an association between nasal carriage of *S. aureus* in hemodialysis patients and catheter infection. Reduction of carriage rates has resulted in a decreased incidence of bloodstream infections. [31]

Antimicrobial Therapy

Pending culture results, the initial choice of an antibiotic is empirical and depends on the clinical setting, the site of infection, type of device, host factors (e.g., immunocompromised state), severity of illness, and whether or not the device is removed. There are not compelling data to support either the choice of a specific empirical antibiotic or duration of therapy for device-related infections. [31] If *coagulase-negative* staphylococcus is the suspected organism, empirical vancomycin should be initiated, followed by semisynthetic penicillin or other appropriate antibiotics as guided by sensitivity studies. When *S. aureus* is the suspected organism, b-lactam antibiotics (e.g., penicillins, cephalosporins, carbapenems, and monobactams) should be first-line therapy. In penicillin-allergic patients or those with methicillin-resistant *S. aureus* (MRSA), vancomycin is the drug of choice. Vancomycin is typically recommended in areas of significant rates of MRSA, followed by a semisynthetic penicillin. In absence of significant rates of MRSA, penicillinase-resistant penicillins, such as nafcillin or oxacillin, should be used. Additional coverage should be initiated in immunocompromised or severely debilitated patients. This should include coverage for enteric organisms as well as *Pseudomonas aeruginosa*, with a third- or fourth-generation cephalosporin (e.g., cefoperazone, ceftazidime, cefepime) or an aminoglycoside. With infections involving gram-negative bacilli, quinolones with or without rifampin should be instituted for 14 days. If fungemia is suspected or confirmed, initiation of antifungal and catheter removal is imperative. Parenteral amphotericin B should be initiated for patients who are hemodynamically unstable or who have received prolonged fluconazole. Fluconazole should be initiated when patients are hemodynamically stable and have not had recent therapy with fluconazole, or those with a fluconazole-susceptible organism. Duration of therapy should continue for 14 days following the last positive blood culture and evidence of clinical improvement. [31] [33] [36] [37] [38]

There are variable recommendations regarding exit site/tunnel/pocket infections. [31] [33] Jones recommends aggressive local care in the early stages and use of topical antibiotic ointment for short-term treatment, adding that long-term treatment should be avoided due to the risk of *Candida* colonization.[33] Mermel and coworkers, however, recommend that in patients with complicated infections, such as tunnel infection or port abscess, catheter removal is required and antibiotics should be initiated for 7 to 10 days.[31] There are not sufficient data to make strong recommendations regarding a strict duration of antibiotic treatment; however, there are generally agreed upon ranges of therapy. [31] [33] [37] Patients who are not immunocompromised and in whom there is no evidence of complication (e.g., endocarditis, septic thrombosis/emboli, osteomyelitis) should receive antibiotics for 5 to 7 days if the catheter is removed, and 10 to 14 days if the CVC is retained. Those with persistent bacteremia or fungemia after catheter removal should be treated for 4 to 6 weeks. Those who develop complications should be treated for 6 to 8 weeks. [31] If the CVC is retained, antibiotic lock therapy (ALT) should be considered.

ALT is a promising area of research in CVC-related infections. Most infections in tunneled catheters originate in the hub and spread to the catheter lumen. This, combined with the deposition of fibrin, makes eradication of organisms difficult. ALT involves filling the catheter hub and lumen with a higher concentration of antibiotics and leaving them in place for extended periods of time. Studies have suggested that ALT alone may be as effective as parenteral antibiotics followed by ALT. The duration of ALT is most often 2 weeks.[31] Some recommend the use of prophylactic vancomycin lock solutions as a beneficial and cost-effective method of preventing infection in long-term tunneled and cuffed CVCs.[39]

Thrombolytics (e.g., streptokinase, urokinase) were initially considered promising in conjunction with antibiotic therapy for intraluminal catheter-related infections due to fibrin deposits that develop over time and provide a nidus of infection. These have not been shown to be beneficial, however. [40] [41]

Prophylactic Measures

Antibiotic prophylaxis during initial line insertion.

Prophylaxis with vancomycin or teicoplanin during central line insertion has not consistently demonstrated reduced incidence of catheter-related bloodstream infection. Based on the limited available data, the Centers for Disease Control and Prevention guidelines currently recommend against prophylactic use of vancomycin because it is an independent risk factor for the acquisition of *Vancomycin-Resistant Enterococcus (VRE)*,[39] and more recently, staphylococci with reduced susceptibility to glycopeptides. One study showed a reduced infection rate when vancomycin was used to flush solutions or TPN, [42] although this beneficial effect was not demonstrated in a subsequent study.[43] Rather than use antibiotic prophylaxis, it is recommended that efforts focus on interventions that are not likely to encourage the emergence of antimicrobial resistance, such as maximal barrier precautions.[43]

Impregnated catheters.

The use of antimicrobial or antiseptic impregnated catheters or silver impregnated collagen cuffs may be an effective intervention to reduce CVC-related bloodstream infection. Hanley and colleagues reported beneficial results with triple-lumen catheters in an intensive care setting. [44] These findings may be applicable to long-term tunneled or nontunneled catheters, as well as AV fistulas, but further studies are necessary. Nonetheless, the Hospital Infection Control Practices Advisory Committee of the CDC has made use of these devices a category II recommendation.[44]

Routine line changing.

Despite the incidence of infection and the potential complications, routine changing of CVCs is not recommended. [45] Cobb and coworkers found that

replacement of CVCs every 3 days did not prevent infection, and, in fact, doing so over a guidewire increased the risk of bloodstream infection. [45]

Coagulopathy

Bleeding may be seen with any indwelling CVC or AV fistula, but is more commonly seen with nontunneled CVCs or temporary dialysis catheters. It is often related to preexisting or transient thrombocytopenia, drug- or uremia-induced platelet dysfunction, trauma, infection, heparinization, or heparin rebound. [46] Treatment includes direct pressure for 5 to 10 minutes, supportive measures, and correction of the underlying cause. If bleeding stops, the patient should be observed for 2 hours for evidence of rebleeding and to detect graft thrombosis. If bleeding cannot be controlled by direct pressure or with thrombogenic agents (e.g., Surgicel, Oxycel), consider IV protamine. The dose of protamine is 0.01 mg per unit (U) of heparin, or 10 to 20 mg for 1000 to 2000 U of heparin. Laboratory studies should include hemoglobin, platelet count, prothrombin time/partial thromboplastin time (PT/PTT), and blood urea nitrogen. If uremic platelet dysfunction is present, consider desmopressin or cryoprecipitate.[30] If possible, consult with the patient's nephrologist or vascular surgeon, given the potential for thrombus formation with the administration of protamine and the need for close follow-up.

There are conflicting data in the literature regarding a bleeding phenomenon know as "heparin rebound." It is unclear whether this occurs after a single administration of heparin or whether it requires repeated exposures to heparin as in dialysis patients. It may account for episodes of post-dialysis bleeding that are seen in the ED.

Heparin rebound is most likely to occur in dialysis patients, who, because of being at high risk for bleeding, have heparin continuously infused into the dialyzer inlet line and protamine sulfate into the outlet line. Thus, only the blood in the extracorporeal circuit is anticoagulated. When the inactive heparin-protamine complexes are metabolized during the hours post dialysis, active heparin may be released into the patient's circulation. This rebound effect may be seen up to 10 hours post dialysis.[46] Experts explain it as an increase in thrombin activity following a discontinuation of heparin [45] [47] or as a reappearance of anticoagulant activity after adequate neutralization of heparin with protamine. [27] [48] [49] [50]

Thrombus Formation

It has been estimated that from 2% to 42% of CVCs are associated with deep venous thrombosis (DVT). [20] Numerous risk factors have been established for catheter-related DVT, including the composition, diameter and position of the CVC, elevated intraluminal pressure, turbulent blood flow, vascular calcification, endothelial injury, and increased levels of fibronectin. [20] [51] Polyurethane and silicone catheters have a lower rate of CVC-related DVT than polyethylene or Teflon-coated catheters. An external diameter of less than 2.8 mm is also associated with a lower incidence of DVT. Incorrect placement of the CVC in the SVC, as opposed to the junction of the SVC and the right atrium, results in a higher incidence of catheter-related DVT. [20]

DVTs may be asymptomatic or may not be recognized due to the underlying condition of the patient. Luciani and associates found that upper-extremity DVT was relatively common, occurring in 11.7% of their patient population, the vast majority of whom (76%) were asymptomatic. [20] They recommend routine Doppler screening for at least the first 3 months after catheter placement. Pulmonary embolism can occur with the catheter in place as well as days to weeks after its removal. [52]

Most HD access failure (80% to 85%) is related to thrombosis, with 85% to 90% of these thromboses associated with venous outflow stenosis. [16] Histologically, hyperplasia of the endothelium and fibromuscular vessel wall also occurs. Over time, fibrin deposits build up on the tip of the CVC. This may continue to the point of occlusion, preventing infusion or aspiration from the CVC. Oftentimes, early detection may be enhanced by a high index of suspicion, noting prolonged bleeding post cannula withdrawal or a change in the bruit over the device, or both. Prompt consultation with a nephrologist and vascular surgeon is indicated. Systemic heparin or local thrombolytic agents, such as urokinase (both in bolus fashion, and in continuous infusion), may be tried. The clinician should be familiar with local institutional recommendations for thrombolysis in the setting of recent CVC occlusion attributed to a fibrin plug. Atkinson and colleagues reported good response with urokinase and follow-up tissue plasminogen activator (t-PA) when urokinase failed. [53] The initial treatment was 2 to 3 mL of urokinase (5,000 U/mL). Catheters remaining occluded received 2 mL of t-PA (1 mg/mL). The t-PA was repeated once if needed.

Embolization

A serious but comparatively uncommon complication of CVC use is embolization with either air, catheter fragment, or thrombotic emboli. [54] [55] [56] Air emboli develop when the catheter lumen is left open to air or the catheter is fractured, perforated, or cut. Care must be taken to maintain a closed system by clamping the catheter appropriately to prevent the delivery of air into the venous circulation. The externalized portion of a right atrial catheter may be accidentally cut [37] or perforated during clamping, especially if an improper clamp is used. [57] Should this occur, an appropriate clamp must immediately be placed between the damaged portion and the skin (except for Groshong catheters). [27] Air embolism is suggested by an open or damaged catheter in association with tachypnea and hypotension. [58] The patient must be placed in a left lateral decubitus and Trendelenburg position (to reduce ventricular outflow obstruction by air pockets), and supportive measures including high-flow oxygen should be initiated. Attempt at catheter repair may be made if a repair kit is available and the operator is familiar with the technique (Fig. 25-7). Repair kits that contain silicone adhesive, plastic clamps, injection caps, and a 12 Fr catheter replacement segment (e.g., Evermed) are available for externalized catheters.

Embolization of catheter thrombi may occur during flushing and injection of solutions. Anderson and colleagues [59] prospectively evaluated the size and frequency of catheter thrombi in 43 patients by aspirating after a urokinase flush. Clots were noted in 40 of 43 subjects and 153 of 508 total specimens. Thrombi varied in size from small fragments to 5 cm in length. Haimov[28] reported no clinical pulmonary emboli after heparin flushes in 70 patients; however, Zureikat and associates reported 1 case of pulmonary embolus associated with Broviac catheterization in a 2-month-old. [52]

Embolization of a catheter fragment is a potentially life-threatening complication causing acute dyspnea, palpitations,

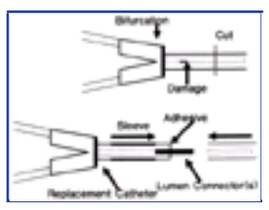

Figure 25-7 Catheter repair. A hollow lumen connector and outer sleeve are placed to splice a replacement catheter to the remaining catheter segment.

atypical chest pain, hypoxia, and atrial fibrillation. Sequelae of catheter fragment embolization include sepsis, lung abscess, dysrhythmias, vascular or cardiac perforation, and sudden death. [55] Catheter fragments are identified radiographically, and may be removed either surgically or using intravascular retrieval methods. Neumann and colleagues reported aid of transesophageal echocardiography to visualize the catheter. [60]

Catheter Displacement

Catheter displacement may occur accidentally secondary to patient movement or iatrogenically, or both. Harvey and colleagues [49] reported two cases of Hickman catheters being withdrawn accidentally by patients. Chardavoyne and coworkers [50] described a similar case in which a TIVAD (Infuse-A-Port) spontaneously withdrew from the central circulation after documented placement. Care must be taken when handling both long-term venous access catheters and TIVADs, especially in active patients.

Catheter Occlusion

Catheter occlusion or low flow may be caused by improper positioning, kinking or compression of the catheter, intraluminal thrombi, extraluminal thrombi, or fibrin deposits at the catheter tip, and by intraluminal precipitation of infusate. "Pinch off syndrome" occurs when the line (most often a PICC) is compressed between the clavicle and the first rib. [36] Maneuvers to facilitate flow include the Valsalva maneuver, the reverse Trendelenburg position, slight tension on the catheter, IV hydration, and extension of the arms above the head. [27] [61] Overzealous withdrawal on the syringe will collapse the catheter and make demonstration of patency difficult. If these measures are unsuccessful, instilling 3 to 5 mL of heparin followed several minutes later by gentle aspiration is often successful in dislodging a clot. Use of urokinase is a reasonable next step. One milliliter of urokinase (5000 U) may be injected and aspiration attempted after a 10-minute interval. A urokinase flush has been reported to successfully open lines in 95% of cases. [28] Atkinson and colleagues have advocated an initial treatment with 2 to 3 mL of urokinase (5000 U/mL). [53] If the urokinase failed to resolve the occlusion, they administered 2 mL of t-PA (1 mg/mL). The t-PA was repeated once if needed.

Catheter Fracture

Fracture of a VAD can occur either subcutaneously or in the externalized portion. [29] Subcutaneous fractures cause localized pain and swelling and require line removal. Fractures in the externalized portion may be repaired using commercially available kits (see Fig. 25-7).

Subclavian Steal

"Vascular steal syndrome" is an uncommon (1% to 3% incidence) but serious complication of AV fistulas that is difficult to predict and often leads to graft failure. It occurs because of a preferential flow through the low-resistance fistula at the expense of the distal circulation. The syndrome is manifested by pain, pallor, numbness, motor weakness, and diminished or absent pulses distal to the fistula [62] and may also manifest as intermittent claudication. [63] Symptoms may be precipitated by dialysis owing to lowered systolic blood pressure or P_{O_2} . [64] They must be differentiated from the nonspecific complaints of diabetic or uremic neuropathy as they may lead to the development of irreversible neuromuscular dysfunction and tissue necrosis. The recognition and prompt correction of hand ischemia would lead to increased salvage and utilization of functioning fistulas. There is no reliable method to predict the development of steal syndrome after construction of a fistula. [62]

Severe cases of steal syndrome require ligation or removal of the vascular access device.

AFTERCARE INSTRUCTIONS

Before release from the ED, each patient should be instructed regarding proper catheter care to prevent complications and prolong the device's lifetime. [27] Patients may bathe and swim normally; they should avoid direct pressure on the reservoir and report local bruising or bleeding immediately.

Long-term venous access catheters and multilumen catheters should be dressed in sterile fashion and observed daily for bleeding and signs of infection, which include fever, pain, redness, swelling, and purulent drainage. VADs should be gently flushed on a routine basis. [27] Heparin flushes are essential to prevent thrombosis. Tunneled (e.g., Hickman) and nontunneled (e.g., PICC) catheters require flushing twice weekly with 5 mL of heparin (10 U/mL). TIVADs require heparin flushing every 4 weeks. Generally, Groshong catheters are flushed with 5 mL of saline once weekly. Manhurkar and Uldall catheters are "flushed" during dialysis. Manhurkar catheters also are used for phoresis, in which case they are treated three times a week with normal saline and heparin, as outlined earlier. Signs of infection and any inability to flush any indwelling catheter should be reported immediately to a physician.

SUMMARY

Numerous vascular access devices are currently available for long-term patient care, including tunneled, cuffed, right-atrial

catheters, PICC lines, midline catheters, and AV fistulas and grafts. These devices potentially provide the emergency clinician with circulatory access to a large, high-risk patient population in a relatively safe and painless manner. However, many life-threatening risks, including hemorrhage, thrombosis, and sepsis are inherent in accessing these devices. A thorough knowledge of the various access devices and the potential complications will adequately equip the provider to minimize these risks, ensuring decreased patient and catheter morbidity and mortality.

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Chapter 26 - Intraosseous Infusion

Rachel Stanley

Establishing vascular access in a critically ill or injured pediatric patient can be one of the most difficult and frustrating procedures a clinician has to perform. From a procedural or logistical standpoint, it is simply not possible to rapidly establish adequate vascular access for all ill children. In a review of intravascular access in pediatric cardiac arrest, the mean (± SD) time needed to establish venous access was a disappointing 7.9 ± 4.2 minutes. [1] Although peripheral percutaneous venous access was the fastest method noted in the review (mean time, 3.0 minutes), it was successful in only 17% of cases. Success rate was highest for an intraosseous (IO) infusion (83%), followed by surgical cutdown (81%), and central venous catheterization (77%). The mean time required to obtain a functioning IO line was 4.7 minutes, followed by 8.4 minutes for a central line, and 12.7 minutes for a cutdown. Another study of emergency department (ED) pediatric arrests demonstrated failure to gain intravenous (IV) access entirely in 6% of the patients. [2] Clearly, in the real world these times will vary according to institution, clinical scenario, and experience of the operator.

Small peripheral vessels in children often collapse during shock, and the child's increased body fat makes peripheral cannulation time-consuming and at times impossible. Central venous cannulation can be equally difficult and carries the risk of pneumothorax or arterial injury. Alternative routes for drug administration, such as the endotracheal and rectal routes, may not provide rapid, reliable drug absorption during cardiac arrest. For example, epinephrine administered by the endotracheal route, although effective in a normally functioning cardiovascular system, is poorly absorbed and produces minimal physiologic response when administered during cardiac arrest. [3] [4]

In other emergency situations such as air and ground transports of severely compromised patients and mass casualties (situations from accidents, fires, or explosions), it may be difficult or impossible, even for experienced professionals, to gain IV access in children or adults. In these situations where obtaining rapid vascular access is essential, the IO route is indicated. The American Heart Association, the American Academy of Pediatrics, and the American College of Surgeons recommend vascular access via the IO route in emergency situations in children when venous access is not immediately possible. [5] [6] In addition, recent studies suggest this approach may also have a place in the management of premature infants, term neonates, and extremely ill or injured adults. [7] [8] [9] [10] [11] [12] [13] At this juncture there is no universally accepted standard of care promulgated for IO access in the general medical community, but its use is becoming more familiar to many clinicians outside the pediatric critical arena.

IO access is not, however, universally successful. Studies using the IO route in acutely ill children and adults have demonstrated that success rates are highest (85%) in children younger than 3 years old and lowest (50%) in children older than 10 years and in adults. [14] The main causes for failure were errors in landmark identification and bending of the needles. The difficulty with needle penetration has been overcome by the Bone Injection Gun (BIG) (Wais Medical, Tri-anim, Sylmar, CA), a spring-loaded device designed to penetrate adult bone, and First Access for Shock and Trauma ([FAST], Pyng Medical Corporation, Vancouver, BC, Canada), a device designed to penetrate the sternum. [15] This device is not widely used at this time but may hold promise if its benefit can be verified in the routine ED setting.

BACKGROUND

One of the earliest references describing the IO route was by Drinker and colleagues, who in 1922 examined the circulation of the sternum and suggested it as a site for transfusion.[16] The route was not used clinically until 1934 when Josefson, a Swedish clinician, administered liver concentrate into the sternum of 12 adult patients with pernicious anemia and reported that all 12 improved.[17] Subsequently, the technique became widespread in Scandinavian countries.

In 1940 the technique was introduced to American clinicians by Tocantins, who described a series of animal and clinical studies that demonstrated fluid was rapidly transported from the medullary cavity of long bones to the heart. [18] [19] He recommended using the manubrium sternum in older children and adults and the upper tibia or lower femur in children age 3 or younger. [19] [20] Over the next two decades, thousands of cases of IO infusion of blood, crystalloid substances, and drugs were reported.[21] [22] [23] The procedure was more commonly used in children because of difficulty with other forms of IV access. Nevertheless, during the 1940s IO infusion was also used extensively in adults, and a sternal puncture kit for bone marrow infusions was a common component of emergency medical supplies during World War II.[8] [24] During this time relatively few complications were reported, considering the needles were often left in place for 24 to 48 hours. Heinild and coworkers in 1947 reviewed 982 cases of IO infusion and reported only 18 failures and 5 cases of osteomyelitis. None of the cases of osteomyelitis occurred in patients who received isotonic solutions.

With the introduction of plastic catheters and improved cannulation techniques, the need for IO infusion as an alternative route for IV access diminished, and the technique was all but abandoned. It was not until the mid-1980s that the technique was reintroduced in response to the need for immediate vascular access during cardiopulmonary resuscitation (CPR).[2] [25] Since then, the technique has become widespread throughout the United States and is recognized as an accepted alternative to IV access in pediatric emergencies and, increasingly, in neonatal and adult emergencies. In addition, the safety, ease, and effectiveness of the technique have led to its use for prehospital emergency care. [14] [26] [27] [28]

In addition to the FAST and BIG devices, described earlier in this chapter, a much broader selection of needle types and sizes has made IO access easier and more successful in children and adults.[8] Today, IO devices are available in most trauma rooms and prehospital environments, and have recently been reissued to medical elements of the U.S. military.[8]

and rapidly transporting fluids or drugs to the central circulation. The bone, like most organs, is supplied by a major artery (nutrient artery). The artery pierces the cortex and divides into ascending and descending branches, which further subdivide into arterioles that pierce the endosteal surface of the stratum compactum to become capillaries. The capillaries drain into medullary venous sinusoids throughout the medullary space, which in turn drain into a central venous channel (Fig. 26-1). The medullary sinusoids accept fluid and drugs during IO infusion and serve as a route for transport to the central venous channel, which exits the bone as nutrients and emissary veins.[29] The medullary cavity functions as a rigid, non-collapsible vein, even in the presence of profound shock or cardiopulmonary arrest. [30] Radiographic studies have demonstrated that radiopaque dye spreads only a few centimeters in the medullary space before being transported to the venous system.[31] The richly vascular red marrow cavity of the long bones is gradually replaced by less vascular yellow marrow after age 5. [32]

Almost every drug and fluid commonly used in resuscitation has been reported in clinical and preclinical IO studies. Medications and fluids that have been administered through IO infusion are listed in Table 26-1 . Crystalloid infusion studies in animals have demonstrated that infusion rates of 10 to 17 mL/min may be achieved with gravity infusion and rates as high as 42 mL/min with a pressure infusion. [33] [34] [35] IO crystalloid infusion has been shown to produce a significant increase in blood pressure in a hemorrhagic shock model in rabbits. [36] In small animals (7 to 8 kg) the size of the marrow cavity is the rate-limiting factor, whereas in larger animals (12 to 15 kg), the size of the needle determines the flow. [34] Blood under pressure can be infused approximately two thirds as fast as crystalloid fluids. [34]

Comparisons of IO and IV infusion of drugs have demonstrated that the drugs reach the central circulation by both routes in similar concentrations and at the same time (Fig. 26-2). [18] [37] This holds true even during CPR, where sodium bicarbonate has been shown to provide greater buffering capacity when administered by the IO route than by the peripheral IV route. [38]

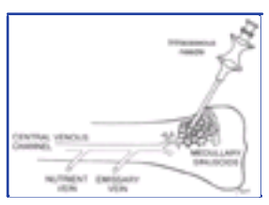

Figure 26-1 Schematic diagram illustrating the venous drainage from the marrow of a long bone with an intraosseous (IO) needle in place.

TABLE 26-1 -- Medications and Fluids that can be Administered Intraosseously

Medications
Adenosine
Antibiotics
Antitoxins
Anesthetic agents
Atracurium besylate
Atropine
Calcium chloride
Calcium gluconate
Contrast media
Dexamethasone
Diazepam
Diazoxide
Digoxin
Dobutamine
Dopamine
Ephedrine
Epinephrine
Heparin
Insulin
Levarterenol
Lidocaine
Lorazepam
Mannitol
Morphine
Naloxone
Pancuronium
Phenobarbital
Phenytoin
Propranolol
Sodium bicarbonate
Succinylcholine
Thiopental
Vecuronium

Fluids
Crystalloids
Dextrose solutions
Sodium chloride solutions
Lactated Ringer's solution
Colloids
Blood and blood products
Packed red blood cells
Plasma

Data from Getschman SJ, Dietrich AM, Franklin WH, et al: Intraosseous adenosine. As effective as peripheral or central venous administration? Arch Pediatr Adolesc Med 148:616, 1994; Sawyer RW, Bodai BI, Blaisdell FW, et al: The current status of intraosseous infusion. J Am Coll Surg 179:353, 1994.

Voelckel demonstrated that bone marrow blood flow responds to both the physiologic stress of hemorrhagic shock and vasopressors given during resuscitation after hypovolemic cardiac arrest in dogs. After successful resuscitation, bone marrow blood flow decreased following high-dose epinephrine but was maintained after high-dose vasopressin. These findings emphasize the need for pressurized IO infusion techniques during hemorrhagic shock and certain drug therapy in animal models.[39]

INDICATIONS AND CONTRAINDICATIONS

IO infusion is a means of achieving rapid temporary vascular access until a patient can be stabilized and traditional IV access obtained. It is indicated when fluid or drugs must be

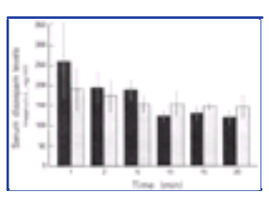

Figure 26-2 Serum diazepam levels (ng/mL, mean ± SE) graphed for the intraosseous (IO) *(shaded area)* and intravenous (IV) *(blackened area)* groups as a function of time when injected during normal perfusion. Initially the IV drug level is slightly higher, but overall the difference between the two routes of administration is not significant.

introduced into the circulation rapidly and venous access is not readily available. The primary indication is cardiac arrest in an infant or child, but there are increasing reports of successful use in preterm and term infants. In one study, 30 IO lines were placed in 20 preterm and 7 full-term neonates with a variety of illnesses (e.g., respiratory distress syndrome, perinatal asphyxia, congenital cardiac anomalies) in whom conventional venous access had failed. All survived the resuscitation with no long-term effects from IO line placement. Gestational age ranged from 32 to 41 weeks and birth weight ranged from 515 to 4050 g. [9] In 1999, Abe et al studied the speed and ease of establishing newborn emergency vascular access using turkey bones and plastic infant legs to simulate IO access. They used fresh umbilical cord to simulate umbilical venous catheterization (UVC). They demonstrated that for individuals who do not frequently perform newborn resuscitation, IO placement was easier and quicker to perform than UVC. The results of this study have yet to be confirmed in a clinical trial with live infants.

IO infusion is also indicated in adult patients in whom peripheral and central venous access has been unsuccessful. In pediatric patients it may be used as a first line of vascular access if peripheral vascular access does not appear to be readily obtainable. Other indications include shock, trauma, extensive burns, severe dehydration, status epilepticus, or any situation in which the emergency administration of fluids or drugs is necessary but not feasible by other routes. [40] IO infusion into the tibia has also been used as a site for lower extremity venography. [41]

There has also been renewed interest in the IO route by the military for use in special operations where adverse environmental conditions make obtaining IV access very difficult. Operations conducted in the dark, during harsh weather conditions, or under other highly stressful situations (e.g., nuclear, biological, or chemical attack) may make IV access impractical or impossible. The Army Institute for Research has compared several IO infusion devices including the FAST, the BIG, the SurFast Hand-Driven Threaded-Needle (Cook Critical Care, Bloomington, IL), and the Jamshidi Straight-Needle (Allegence Health Care, McGaw Park, IL). [15] Success rates for these devices were similar (94% to 97%) and all were inserted in <2 minutes. The participants rated no one device as significantly better than the others. It was concluded that each device was easy to master and could be appropriately used during Special Operations when IV access could not be accomplished. [15]

The revival of IO anesthesia has also significantly improved dental anesthetic techniques. Primary IO anesthesia is useful for short procedures where it is desirable to minimize the feeling of numbness and the ballooning of tissue. [42] IO anesthesia provides profound anesthesia of sufficient duration for most dental procedures and is particularly useful for situations that are refractory to conventional anesthetic techniques [42] [43]

In addition to serving as a route for fluid administration, the IO needle may be used for obtaining blood type, cross-match, and blood chemistry determinations from the marrow cavity. Serum electrolyte, blood urea nitrogen (BUN), creatinine, glucose, and calcium levels are very similar to those in samples obtained from an IO aspirate.[44] [45] Blood gas values obtained from the IO site also are similar to those obtained from central venous sites during steady- and low-flow states in one animal model and may be an acceptable alternative to judging central acid-base status during CPR. [46] A complete blood cell count may not be reliable because it reflects the marrow cell count rather than the cell count in the peripheral circulation. Furthermore, the aspirated blood usually clots within seconds, even if it is placed in a tube that contains heparin. Brickman and colleagues demonstrated that bone marrow aspirates obtained from an IO needle in the iliac crest could be reliably used to type and screen blood for transfusion. [47]

Relatively few contraindications to IO infusion exist. Osteoporosis and osteogenesis imperfecta are associated with a high fracture potential; therefore, the procedure should be avoided when these diagnoses are known unless absolutely necessary. A fractured bone must be avoided because as fluid is infused, it increases the intramedullary pressure and forces fluid to extravasate at the fracture site. This may slow the healing process, cause a nonunion of the bone, or lead to a compartment syndrome. A similar extravasation of fluid can occur through recent IO puncture sites placed in the same bone. Hence, recent prior use of the same bone for IO infusion represents a relative contraindication to IO line placement. Needle insertion through areas of cellulitis, infection, or burns should also be avoided.

EQUIPMENT AND SETUP

The only equipment necessary for establishing IO access is a sturdy needle with a stylet and a syringe for aspiration. Needles range in size from 13 to 20 G and are made by several companies. Standard needles for drawing blood or administering medications are *not adequate* for IO infusions; generally, they are not sturdy enough to penetrate bone. A cadaver study of IO puncture suggests that non-styletted needles (2.5-cm, 18-G phlebotomy needles and 7.6-cm, 14-G IV needles) enter the marrow space successfully only about half the time.[48] In the past, an 18-G spinal needle was commonly used for children younger than 12 to 18 months. This needle, although readily available in most EDs, often bends, is too long for rapid fluid infusion, and has a greater risk of occlusion from clotted blood. [49]

Figure 26-3 Needles used for intraosseous (IO) infusion. *Left to right,* Illinois bone marrow aspiration needle, Illinois sternal/iliac aspiration needle, Jamshidi disposable sternal/iliac aspiration needle, Cook IO needle with 45° trocar, and Sur-Fast IO needle.

Several needles are currently used in EDs for IO infusion (Fig. 26-3). Bone marrow aspiration needles such as the Rosenthal or Osgood can be used if available. They are large enough (16-ga) to be used on older children or adults and are good for fluid administration. The 16-G Illinois sternal/iliac aspiration needle (Monoject, Division of Sherwood Medical, St. Louis, MO) has an adjustable plastic sleeve to prevent the needle from penetrating too deeply or through the opposite cortex. Its disadvantage is that it has a long shaft and cumbersome handle, which make it vulnerable to dislodgment from the bone during transport or other procedures. The Cook IO needle (Cook Critical Care, Bloomington, IN) comes in 18- and 16-ga sizes and has a detachable handle that decreases the likelihood of its being dislodged. A useful feature of this

Figure 26-4 The FAST sternal intraosseous (IO) infusion device. *(Courtesy of Pyng Medical Corporation, Richmond, British Columbia.)*

Figure 26-5 The Bone Injection Gun (BIG).

needle is a line located 1 cm from its tip, which serves as a depth marker. The Sur-Fast needle (Cook Critical Care, Inc, Bloomington, IN) has a threaded shaft to permit a more secure needle placement. The Jamshidi Disposable sternal/iliac aspiration needle (Allegence Health Care, McGaw Park, IL) comes in either 15- or 18-G sizes and, like the larger Illinois sternal/iliac aspiration needle mentioned earlier, features an adjustable plastic sleeve. However, its shorter shaft length and smaller handle make it much easier to use. A 13-G needle manufactured by MedSurg Industries (Rockville, MD) is good for fluid resuscitation. However, like the Illinois sternal/iliac aspiration needle, it has a large handle that makes it cumbersome.

Several other devices approved by the FDA for IO access are gaining popularity with both military and prehospital care providers for use in adults. Although not commonly used in the ED, the FAST needle (Fig. 26-4) is designed only for sternal use. The device includes a hand-held introducer that allows insertion of a stainless steel-tipped flexible infusion tube to a predetermined depth in the sternal manubrium. The FAST needle has a bone probe that detects the anterior surface of the bone and a built-in mechanism that releases the handle from the stylet and infusion tube once the infusion tube has reached the preset depth. These features help prevent overly penetrating the sternum. [50]

The BIG (Fig. 26-5) incorporates a loaded spring to allow easy penetration of the bone. The depth of injection can be adjusted allowing for use in different locations.[15] Hubble compared IO versus saphenous vein cutdown access times and success rates in human adult cadavers using the BIG. He concluded that IV access was achieved more rapidly with a higher success rate and fewer complications using the BIG via the IO route than saphenous vein cutdown in adult cadavers.[51]

TECHNIQUE

Originally the sternum was the primary site for IO infusion. After several cases of osteomyelitis and mediastinitis in

Figure 26-6 Schematic diagram demonstrating intraosseous (IO) insertion sites. *A*, The proximal tibia. The IO needle is inserted 1 to 3 cm distal to the tibial tuberosity and over the medial aspect of the tibia. *The bevel of the needle is directed away from the joint space. B,* The distal tibia. The IO needle is inserted on the medial surface of the distal tibia at the junction of the medial malleolus and the shaft of the tibia, posterior to the greater saphenous vein. The needle is directed cephalad, away from the growth plate. *C,* The distal femur. The IO needle is inserted 2 to 3 cm above the external condyles in the midline and directed cephalad away from the growth plate.

children, the site was abandoned.[52] Other sites, such as the clavicle and humerus, have been used, but neither has gained popularity. Currently the site of choice is the proximal tibia, followed by the distal tibia and distal femur. However, introduction of the FAST needle has resulted in renewed interest in the sternum in adults. [50]

The tibia is popular because it is a large bone with a thin layer of subcutaneous tissue that allows landmarks to be readily palpated. It is the preferred site of IO insertion in infants and children younger than 6 years old, and its insertion does not interfere with airway management and cardiopulmonary resuscitation. On the proximal tibia, the broad, flat, *anteromedial surface* is used, with the tibial tuberosity serving as a landmark. The site of IO cannulation is approximately 1 to 3 cm (2 fingerwidths) below the tuberosity on the medial or flat surface of the tibia (Fig. 26-6*A*). This location is far enough from the growth plate to prevent damage but is in an area in which the bone is still soft enough to allow easy penetration of a needle. This site may be used in adults but is more difficult and requires a 13- to 16-G needle to penetrate. If a smaller-gauge needle is desired, use of the spring-loaded BIG is required.

The patient's leg should be supported by placing a small sandbag or towel roll behind the knee. The site is prepared with a povidone-iodine solution or alcohol. Local anesthesia is usually unnecessary since the majority of patients in whom IO infusion is used have altered mental status from shock or are in cardiopulmonary arrest. However, if the patient is conscious, the skin and periosteum should be anesthetized. The proximal tibia is grasped with the palm of the nondominant hand (Fig. 26-7). The fingers and thumb stabilize the proximal tibia. To avoid self-puncture, care must be taken to ensure that the hand does not extend behind the insertion site.

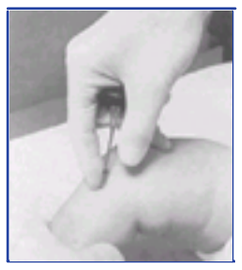

Figure 26-7 Insertion of an intraosseus (IO) needle. The fingers and thumb are wrapped around the proximal tibia to stabilize it. A towel (not shown) may be placed behind the knee for support. The needle is grasped firmly in the palm, and a rotary motion is applied with moderate pressure. The plastic sleeve can be adjusted to prevent it from being forced too deeply into the bone or through the bone. Note that the needle is directed away from the joint space.

Figure 26-8 Intraosseus (IO) needle in situ. *A*, The needle and tubing are secured with tape and the extremity is immobilized on a leg board. A long leg board may be needed with an active child. *B*, The needle is protected from accidental dislodgement by wrapping a stretch bandage around the extremity and placing a small plastic drinking cup around the needle.

The bony landmarks are palpated and the site is identified. The needle should be grasped firmly in the palm of the hand and directed either perpendicular (90°) to the long axis of the bone or slightly caudad (60°–75°) to avoid penetration or injury to the growth plate. The needle is then advanced with a *twisting or rotary motion* to cut the bone and facilitate puncture of the cortex. Considerable resistance will be encountered, but once the cortex has been penetrated, there is a sudden decrease in resistance and a crunching feeling as the needle moves through the bony trabeculae into the marrow cavity. The distance from the skin through the cortex of the bone is rarely greater than 1 cm in an infant or child, and penetration to this depth is usually adequate. A common mistake is to advance the needle through the opposite side of the bone. This can best be avoided by holding the needle in the palm of the hand with the index finger approximately 1 cm from the bevel of the needle to avoid pushing it past this mark. Another method is to use a needle with a preset plastic depth indicator on the shaft. [53] The stylet is then removed, and a 5- to 10-mL syringe is used to aspirate blood and marrow contents for confirmation of position. Many times, particularly during cardiac arrest, blood aspiration is not possible. Other signs of successful needle insertion include the needle's ability to stand upright without support and infused fluids that flow easily without evidence of swelling or extravasation.

If there is excessive resistance to fluid infusion, the needle may be pulled back a few millimeters and another attempt made to infuse fluids. If continued resistance is met or evidence of extravasation exists, the needle should be removed and an attempt made on the other extremity. If the needle appears to be infusing properly, the test syringe is disconnected, and the needle is then connected to infusion tubing. The needle and tubing should be secured with tape and the extremity immobilized on a leg board (Fig. 26-8*A*). The needle should be protected from accidental dislodgement by wrapping a stretch bandage around the extremity and placing a small plastic drinking cup or commercially available shield around the needle (see Fig. 26-8*B*). The IO needle should be removed as soon as secure IV access has been obtained. A sterile dressing is placed over the puncture site, and pressure should be applied to the dressing for 5 minutes. [54]

The distal tibia, although a preferred site in adults, may be used as well in children. [49] [55] The cortex of the bone and the overlying tissue are both thin. The site of needle insertion is the medial surface at the junction of the medial malleolus and the shaft of the tibia, posterior to the greater saphenous vein (see Fig. 26-6*B*). The needle is inserted perpendicular to the long axis of the bone or 10°–15° cephalad to avoid the growth plate. [53]

The distal portion of the femur is occasionally used as an alternate site, but because of thick overlying muscle and soft tissue, it is more difficult to palpate bony landmarks (see Fig. 26-6*C*). If chosen, the needle should be inserted 2 to 3 cm above the external femoral condyles in the midline and directed cephalad at an angle of 10°–15° from the vertical.[56]

The introduction of the FAST needle, which allows safe and effective penetration of the sternum, has resulted in renewed interest in the sternum as an IO site in adults. Currently this site is rarely used in the ED but it may be attempted in life-threatening situations. The sternum has several advantages over peripheral bones including a large, relatively flat body that can be readily located by unskilled clinicians; retention of a high proportion of red marrow; and thinner, more uniform cortical bone overlying a relatively uniform marrow space. In addition, it is less likely to be fractured in major trauma. [50]

The FAST needle is packaged with alcohol and iodine, a protective dressing that also acts to secure the needle in place, and a threaded tip remover (Fig. 26-9). After prepping the overlying skin with iodine and alcohol, an adhesive target patch is placed over the midline of the manubrium with the target zone hole approximately 1.5 cm below the sternal notch (Fig. 26-10*A*). The introducer is placed in the center of the target zone. The introducer contains a "bone cluster" of needles forming a circle, which sense the cortex of the sternum (see Fig. 26-10*B*). Pressure on the handle also releases an inner needle upon which a plastic

infusion tube with a small metal tip is loaded. The central needle advances exactly 5 mm beyond the circular cluster of needles, stopping at the cortex. The metal tip is then positioned in the cortex-medullary junction. Withdrawal of the handle leaves only the plastic infusion

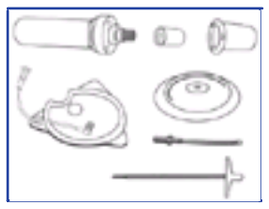

Figure 26-9 The FAST sternal intraosseous (IO) infusion device comes packaged with all necessary equipment.

Figure 26-10 *A*, After prepping the overlying skin with iodine and alcohol, an adhesive target patch is placed over the midline of the manubrium with the target zone hole approximately 1.5 cm below the sternal notch. *B*, The introducer, which contains a "bone cluster" of needles, is placed in the center of the target zone. *C*, Pressure on the handle releases an inner needle upon which a plastic infusion tube with a small metal tip is loaded. The central needle advances exactly 5 mm beyond the circular cluster of needles, stopping at the cortex. The metal tip is now positioned in the cortex-medullary junction. Withdrawal of the handle leaves only the plastic infusion tube protruding from the insertion site. A plastic dome is attached via Velcro fasteners to the target patch, securing the tube in place.

tube protruding from the insertion site. Marrow aspiration and rapid flow of fluid help verify position. A plastic dome is attached via Velcro fasteners to the target patch, securing the tube in place (see Fig. 26-10*C*). Removal of the infusion tube requires the use of an included threaded-tip remover. The tube can also be removed by direct pulling; however, the metal tip is sometimes left behind and must be extracted through a small incision. [15]

The BIG incorporates a loaded spring to facilitate penetration of the bone. Removing the safety pin from one end and screwing or unscrewing the opposite end adjusts the depth of the injection (Fig. 26-11*A* (Figure Not Available)).

Palmar force on the back of the unit, combined with pulling on flanges with the middle and ring fingers, results in firing of the gun (directions on package) (see Fig. 26-11*B* (Figure Not Available)). Aspiration of marrow, followed by flushing with the same syringe, and flow through the IV tubing help confirm placement. The slotted safety pin is slid into the needle to maintain stability and the needle is removed with small clamps that are rotated back and forth (Fig. 26-11 *C* (Figure Not Available)). The site is then dressed in a manner determined by the care provider. [15]

Figure 26-11 (Figure Not Available) The BIG is positioned with one hand to the site while the other hand is used to pull out the safety latch *(A)*. Palmar force on the back of the unit, combined with pulling on the flanges with the middle and ring fingers, results in firing of the gun *(B)*. Before firing, be sure the BIG is 90° to the surface. The BIG is then removed *(C)*, followed by the stylet *(D)*. Aspiration of marrow, followed by flushing with the same syringe, and flow through the IV tubing help confirm placement. The slotted safety pin is slid into the needle to maintain stability and the needle is removed with small clamps that are rotated back and forth *(E)*. *(Courtesy of WaisMed, Caesarea, Israel.)*

COMPLICATIONS

As with any procedure, complications may arise when attempting to use the bone for vascular access. Complications may be divided into two categories: technical difficulties and latent soft tissue or bone problems. Technical difficulties are the most common, but these decrease as familiarity with the technique increases (Fig. 26-12). The most common mistake is to place excessive pressure on the needle during insertion and force it entirely through the bone (Fig. 26-13). This may be avoided by placing the index finger against the skin to prevent the needle from going in too deeply. Also, attention to depth of the needle or use of a sheath on the needle to prevent excess penetration will decrease occurrences of this problem. Incomplete penetration of the bone may also occur, in which case blood will not be aspirated and fluid will extravasate if infused. Serum levels of medication are decreased when IO infusion is performed through bones with multiple cortical defects. [57] If several attempts are made to place a needle in the same bone, fluid may extravasate from previous puncture wounds. If extravasation occurs, the needle should be removed and pressure applied.

The needle may be blocked periodically by clots forming around the bevel or by bony spicules obstructing the flow of fluid. This complication may require that the line be flushed with 3 to 5 mL of sterile saline every 10 to 15 minutes to keep it open.

A major concern for any person receiving IO infusion is infection. This concern often leads clinicians to shy away from using the bone and to continue searching for other methods of vascular access. Although the potential for infection is real, its actual incidence is low. A literature review of more than 4000 cases from 1942 to 1977 found a 0.6% incidence of infection. [2] Although most of the affected access sites were not placed under emergency conditions, the needles were often left in 1 to 2 days, thus increasing the likelihood of infection. A survey of more than 1000 U.S. and foreign medical schools

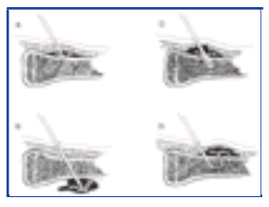

Figure 26-12 Schematic diagram of possible problems encountered with intraosseous (IO) infusion. *A*, Incomplete penetration of the bony cortex. *B*, Penetration of the posterior cortex. *C*, Fluid escaping around the needle through the puncture site. *D*, Fluid leaking through a nearby previous cortical puncture site.

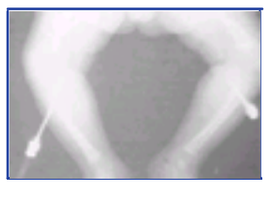

Figure 26-13 Radiograph of bilaterally misplaced IO needles with penetration through the posterior tibial cortices.

found that the incidence of infection for IO needles placed in emergency conditions was less than 3%. [58]

The most common infection is cellulitis at the puncture site, which usually responds well to antibiotics. Osteomyelitis is less common, but it also usually responds to antibiotics. Heinild and colleagues reported three cases of osteomyelitis in 25 patients who received infusions of undiluted 50% dextrose in water ($D_{50}W$). [23] Undiluted $D_{50}W$ injected into rabbit femurs produces edema and pyknotic marrow nuclei that improve within 1 month. [59] In addition to infection, inflammatory reactions of the bone may be seen. These are most common when hypertonic or sclerosing agents are used and may produce an elevation of the periosteum with a positive bone scan (Fig. 26-14). Unlike the clinical appearance of a patient with osteomyelitis due to bacteria, a child with a sterile inflammatory reaction does not look "toxic." One hypertonic sclerosing drug that may be used during cardiac arrest is sodium bicarbonate. Heinild and colleagues [23] reported 78 cases of bicarbonate infusion with no complications. Animal studies have reported a decrease in cellularity with edema and destruction of some cells, but these changes are temporary and completely resolve in a few weeks. [59] [60] [61]

Another complication that has been reported is necrosis and sloughing of the skin at the site of infusion. [62] This occurs if fluid or drugs extravasate from the puncture site into the surrounding tissues. Care should be taken when infusing drugs such as calcium chloride, epinephrine, and sodium bicarbonate to prevent dislodgement of the needle and extravasation into the tissue. It is best to infuse such drugs gently and not under pressure, because pressure frequently causes extravasation.

Injury of the growth plate and developmental abnormalities of the bone are ongoing concerns. These fears have not been supported in the literature, however. There have been no reports of growth plate damage or permanent abnormalities of the bone. One animal study specifically examined damage to the epiphysis; sodium bicarbonate was injected directly into the epiphysis, and no radiologic evidence of epiphyseal injury was found. [63] [64] By pointing the needle away from the joint space and using the previously mentioned landmarks for insertion, the danger of epiphyseal injury is remote.

Fat embolism is frequently mentioned as a potential complication. [2] [49] However, this condition is rare and has been

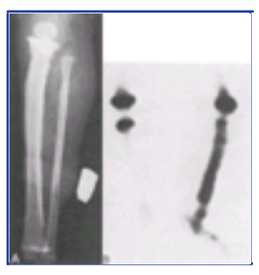

Figure 26-14 Radiograph *(A)* and bone scan *(B)* of the tibia demonstrating an inflammatory reaction 4 days after the patient received intraosseous (IO) phenytoin and phenobarbital. The periosteum is elevated along the length of the bone, mimicking osteomyelitis on the plain films and the bone scan. A diagnosis of osteomyelitis requires either clinical evidence of infectious toxicity or positive cultures (blood or periosteal aspirate).

reported only in adult patients. [65] Animal studies addressing this issue found no changes in blood gases during IO infusion and limited evidence of fat globule collection in the lungs. [66] [67] Because the marrow in infants and children is primarily hematopoietic, this potential complication is unlikely to occur. In one animal study, no significant hemolysis was detected in the setting of pressurized IO blood infusion. [67]

Compartment syndrome following IO line use during cardiac resuscitation has been reported. [68] [69] Tibial fractures have also been reported. [70] [71] Follow-up radiographs of patients who have undergone IO needle placement or attempts at such placement are indicated. Cortical defects may be seen on radiographs for up to 40 days after injection. [72]

One study evaluating the use of the FAST sternal IO device in 50 adults found no serious adverse effects. Minor complications included bruising, swelling, redness, or tenderness at the time of removal. Difficulty with use of the removal tool was reported in 12 cases and 2 devices required removal using a small incision. [50]

INTERPRETATION

The determination of whether an IO infusion is functioning may be made by assessing the ease with which fluid infuses and the clinical response of the patient. If the needle is properly placed, fluid should flow smoothly, with only occasional flushes necessary to keep it open. A clinical response to fluid or medications should be seen at approximately the same time and in the same magnitude as with IV administration.

CONCLUSION

Although not always successful, IO infusion provides a means of rapidly accessing the cardiovascular system in emergencies. The technique is not intended to replace traditional venous access; instead, it should serve as a temporizing measure for resuscitation until venous access can be obtained. It is currently used throughout the United States and Canada and has been reviewed extensively in emergency medicine and pediatric literature. The technique is also used by prehospital emergency care personnel, who have demonstrated a high success rate in the field. [14] [50] [73] The technique renders hope for those who infrequently care for critically ill or injured children, because this skill is easily mastered even if done infrequently. Recent use in adults, dental anesthesia, and in neonatal resuscitation underscore the need for emergency care providers to become familiar with the equipment and techniques.

Complications are most commonly related to technical mistakes. By carefully locating landmarks, staying away from the growth plate, and paying attention to the depth of the needle, complications can be minimized. Finally, as with any technique, practice on cadaver or animal leg bones (e.g., chicken, pig) greatly improves one's skill.

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Chapter 27 - Endotracheal Drug Administration

James H. Bryan

Endotracheal (ET) administration of selected medications is a simple, rapid, and effective method of drug delivery to the central circulation. However, this technique of drug administration should be reserved for use in situations in which a patient's condition warrants immediate pharmacologic intervention and when more conventional means of drug delivery, such as by intravenous (IV) or intraosseous (IO) access, are not readily available. Such circumstances occur infrequently, but when they do, knowledge of the appropriate drugs and dosages that can be delivered effectively by this route may prove to be life-saving. It is important, therefore, that clinicians be familiar with the concept and method of ET drug therapy.

ET drug administration dates to 1857, when Bernard demonstrated the ability of the lung to rapidly absorb an instilled solution of curare. [1] In this experiment, curare was instilled into the upper respiratory tract of dogs by way of a tracheostomy. After the dogs were tilted to an upright position, they died within 7 to 8 minutes. Bernard concluded that the alveoli must be permeable to curare. Over the following decades, other investigators expanded this work and demonstrated that solutions containing salicylates, atropine, potassium iodide, strychnine, and chloral hydrate were also absorbed rapidly from the lung and excreted in the urine after injection of their aqueous solutions into the tracheas of experimental animals. [2]

In 1915, Kline and Winternitz provided experimental data suggesting that intratracheal therapy in pulmonary disease might prove to be an effective therapeutic route.[3] Further studies showed that moderate quantities of fluid could be insufflated into the lungs without marked changes in the lung architecture. [4] The use of intrapulmonary medication in the treatment of lung disease gained further acceptance when studies demonstrated that the inhalation of epinephrine mist dramatically relieved the symptoms of asthma.[5]

In the late 1930s and 1940s, when certain chronic suppurative disorders of the lung failed to respond to parenteral antibiotics, drugs were administered by direct instillation into the lungs. During this period, several important observations were made concerning ET drug therapy: (1) penicillin delivered by the ET route demonstrated a depot effect, resulting in therapeutic blood levels that lasted twice as long as those noted with intramuscular injections [6] ; (2) various diluents mixed with penicillin affected both the rate and the degree of absorption from the lungs [7] ; and (3) higher serum drug levels were attained with direct ET drug administration than with aerosolized administration. [7]

In the 1950s investigators attempting to elucidate the mechanism responsible for adverse anesthetic reactions made two important findings. The first was that drugs delivered endotracheally were absorbed much more rapidly than those applied to the posterior pharynx. A second discovery was that the rapid absorption of drugs applied locally to the larynx and trachea resulted in blood levels significant enough to cause adverse anesthetic reactions. [8]

In 1967 Redding and coworkers studied the use of ET administration as a route of drug delivery in a canine model of cardiopulmonary arrest. [9] They administered epinephrine by the IV, intracardiac, and intratracheal routes, and then evaluated its effectiveness in the resuscitation of dogs that had undergone both respiratory and circulatory arrest secondary to hypoxia. Their study revealed that all three routes of drug administration were equally effective in restoring the circulation of dogs in hypoxia-induced cardiac arrest, again demonstrating that the ET route of drug delivery provides an effective window to the systemic circulation. They concluded that whichever of these routes is most immediately available should be used. A decade passed before further research was published concerning the use of ET drug therapy as an alternative means of systemic drug administration.

RECOMMENDATIONS REGARDING ENDOTRACHEAL DRUG DELIVERY

In the late 1970s Roberts and Greenberg and associates revived the study of ET drug delivery with a series of laboratory experiments and clinical uses of ET epinephrine.[10] [11] [12] [13] Since then, a number of important animal and human studies, as well as a number of case reports, have been published dealing with various aspects of ET drug administration. These investigations addressed: (1) appropriate drug dosing; (2) the effect of drug solution volume; (3) the effect of different diluent solutions; (4) the role of different ET drug delivery techniques; and (5) the effects of hypoxia, hypotension, shock, and cardiopulmonary arrest on the absorption, distribution, and efficacy of endotracheally administered drugs.

Although the American Heart Association (AHA) makes specific recommendations regarding the use of ET drug delivery for cardiac resuscitation (Table 27-1),[14] [15] [16] [17] much of the literature remains controversial and is at times contradictory. It is likely, therefore, that many of these issues will continue to be the subject of future investigations. Specific studies related to each of these topics are discussed as follows.

Appropriate Dose

All investigators agree that the ET dose of a medication should be at least equal to the IV dose of the same drug given for the same indication. Most studies on the subject indicate that higher doses are needed endotracheally than intravenously. For Advanced Cardiac Life Support medications in adults, the AHA recommends a dose 2 to 2.5 times the usual IV dose.[14] This recommendation is supported by the results of a study of epinephrine administered immediately following intubation in the prehospital setting,[18] as well as studies of endotracheally administered lidocaine, indicating a 3 mg/kg dose was needed to obtain therapeutic serum levels. [19] [20] Other studies, however, suggest that ET administration requires higher doses.

Studies and case reports of ET epinephrine have produced conflicting results. Some animal studies [21] and case reports [12] have shown positive effects or recovery from cardiovascular collapse, or both, when epinephrine was used in doses equal to recommended IV doses. In other animal [22] and

TABLE 27-1 -- American Heart Association Guidelines for Tracheal Drug Administration

	Adults	Pediatric	Neonatal
Medication dose	2–2.5 times recommended IV dose	Epinephrine 10 times the IV dose (class IIb); it is logical to similarly increase the dose of other resuscitation drugs compared to IV dose	Same as IV dose (specified for epinephrine and naloxone only)
Total volume to instill	10 mL	5 mL	1 mL
Diluent	Normal saline or distilled water	Normal saline	Normal saline

Adult data from American Heart Association.[14]

Pediatric data from American Heart Association.[15]

Neonatal data from American Academy of Pediatrics. [16] [17]

human[23] studies, however, epinephrine in doses of approximately 0.02 mg/kg and 0.01 mg/kg, respectively, were shown to be unreliable in producing a physiologic response. In addition, studies using both normotensive and cardiac arrest canine models have shown that epinephrine doses of 0.01 mg/kg produce serum levels approximately one-tenth that produced when the same dose is given intravenously. [11] [24] [25] These studies recommend increasing the ET epinephrine dose to 0.1 mg/kg and are the basis for the AHA recommendation to use a 10-fold increased dose when administering ET epinephrine to pediatric patients. [15] However, ET drug delivery is associated with a depot effect. That is, when larger doses of drugs are administered via the ET route, the drug is "stored" and released slowly over time, similar to a continuous IV drip. This presumably occurs due to local vasoconstriction or lymphatic storage of the drug, or both, [10] or from pooling in the lung tissue due to poor lung perfusion.[26] With epinephrine use, the depot effect produces a potential for postresuscitative arrhythmias, hypertension, and tachycardia, with resultant increased myocardial oxygen demand.

Given these data, it seems reasonable in adults to start with a dose 2 to 2.5 times the usual IV dose. If this appears ineffective, higher doses may be used on subsequent administration.

Single-Dose Volume

The need to increase the volume of the instilled medication to enhance effectiveness was recognized by Redding and associates during their early studies. [9] The goal is to use a volume that will enhance delivery and absorption of the drug while producing minimal deleterious effects on pulmonary gas exchange. The AHA recommends a total volume of 10 mL in adults, [14] 5 mL in pediatric patients,[15] and 1 mL in neonates.[16] In canine studies, Mace compared undiluted lidocaine to diluted lidocaine (volume, ~6.5 mL) and found significantly higher plasma lidocaine levels in the animals receiving diluted lidocaine. [27] There were no changes in the arterial blood gas values before and after ET drug administration. In another animal study, lidocaine diluted with normal saline to volumes up to 25 mL produced no changes in arterial blood gases or clinical condition, and no change was seen in lung gross anatomy or histology. [28] In contrast, a study comparing normal saline to distilled water revealed a decreased PaO_2 for both solutions (water producing the greatest effect), but this study used large volumes (2 mL/kg) of solution. [29]

Studies of endotracheally administered lidocaine in human subjects revealed that dilution with distilled water to a total volume of 10 mL resulted in higher plasma lidocaine levels, but also produced a decrease in PaO_2 of approximately 40 mm Hg that persisted for more than 1 hour.[30] A total volume of 5 mL yielded lower plasma levels but also produced a shorter period of hypoxemia. The authors concluded that a total volume of 5 to 10 mL (in agreement with the AHA guidelines) would produce optimal results. Volume recommendations in a clinical setting requiring multiple drug doses (e.g., prehospital arrest without peripheral venous access in a patient facing long transport) are lacking. In this setting it may be appropriate to limit the diluent volume for an individual adult administration to 5 mL.

Appropriate Diluent

Due to their availability during emergency situations, both normal saline and distilled water have historically been the diluents of choice for ET drug administration. The AHA Adult Advanced Cardiac Life Support Guidelines recommend either diluent, noting that "tracheal absorption is greater with distilled water as the diluent than with normal saline, but distilled water has a greater adverse effect on PaO_2 ".[14] Naganobu and colleagues studied epinephrine diluted to 2 mL with either normal saline or distilled water and administered through the ET tube into a distal bronchus of dogs. They found that peak serum epinephrine levels were 13 times higher when diluted with distilled water vs normal saline. In addition, the mean arterial pressure increased significantly when diluted with distilled water but it did not change significantly when diluted with normal saline. They found only minimal changes in the PaO_2 with either diluent and concluded that distilled water is a better diluent than normal saline.[31] Finally, a study of ET epinephrine diluted with normal saline vs distilled water found no difference in arterial blood gases following delivery of either diluent solution.[32] In this latter canine cardiac arrest model, survival rates for ET epinephrine in either diluent were equal to the survival rate with IV epinephrine.

The use of saline is supported by the canine study of Greenberg and associates, which reported that administration of normal saline via the ET route produced fewer detrimental effects on arterial blood gases than did distilled water. [29] The safety of endotracheally administered normal saline is further supported by a study in which

no changes in pulmonary

status (arterial blood gas, oxygen saturation, gross anatomy, or histology) were observed in dogs given lidocaine diluted with normal saline to total volumes of between 6 and 25 mL.[28] Hence, while some diluent is helpful and recommended, the optimal diluent is controversial; saline may produce less pulmonary dysfunction, but distilled water appears to deliver a greater amount of drug.

Technique for Endotracheal Drug Delivery

Multiple techniques have been used to deliver medications via the ET tube, including direct instillation into the proximal end of the ET tube, administration via a catheter that extends just beyond the distal tip of the ET tube, deep endobronchial administration using a longer catheter, administration via ET tube monitoring ports, and injection through the side of the ET tube with a needle. The AHA guidelines for adults recommend the use of a catheter that extends beyond the distal tip of the ET tube.[14] However, the pediatric guidelines advise against using a catheter or feeding tube, noting they are often cumbersome and require finding the correct size to place through the ET tube.[15] Neonatal guidelines recommend either direct instillation into the proximal end of the ET tube or use of a 5Fr feeding tube that is inserted down the ET tube.[16] Several studies have, however, indicated that use of a catheter or feeding tube may not be needed to enhance the drug's effectiveness.

Greenberg and Spivey instilled radiopaque contrast material directly into the proximal end of the ET tube and compared its distribution to contrast instilled via a catheter extending out the distal end of the tube.[33] Their study revealed that both techniques were equally effective in distributing the contrast agent to the peripheral lung fields as long as instillation was followed by five rapid manual hyperventilations. Although some studies have suggested that drug absorption with direct instillation into the ET tube is inconsistent during cardiopulmonary arrest,[20] [23] at least one case report has shown successful resuscitation using this method.[12] Using a porcine cardiopulmonary arrest model, Jasani and coworkers showed no difference in resuscitation rates or physiologic responses between epinephrine administered by direct injection into the ET tube, via a catheter extending out the distal end of the ET tube, or via a monitoring lumen built into the sidewall of the ET tube.[34] In a related study, no difference was detected in resuscitation rates or plasma epinephrine levels when epinephrine was instilled during apnea vs instillation during the ventilator inspiratory cycle.[35]

However, in studies in which subjects had normal perfusion, conflicting results have been reported. In a study using female volunteers, no difference was found in plasma lidocaine levels when the drug was administered directly into the proximal end of the ET tube vs deeper administration into the trachea or lungs.[36] However, a later report by the same group demonstrated significantly higher plasma lidocaine concentrations when the drug was administered directly into the proximal end of the ET tube.[37] In direct contrast, when administered to dogs, significantly higher plasma epinephrine levels were obtained via the deep endobronchial route vs direct ET tube instillation.[38]

Given these conflicting studies, use of a catheter to enhance deep pulmonary delivery seems reasonable. However, if a catheter is not readily available, any method of ET drug administration is reasonable.

Effects of Hypoxia, Hypotension, and Cardiopulmonary Arrest

Several authors have examined the effect of cardiopulmonary arrest on the absorption of medications administered via the ET route. Although concern existed that medication absorption might decrease in states of hypoxia or low blood flow, studies reveal the opposite to be true. In a hemorrhagic shock model, Mace demonstrated that higher plasma lidocaine levels were obtained via the ET route during shock than during nonshock states.[39] In a lamb model, when epinephrine was administered endotracheally, higher plasma epinephrine levels were achieved during hypoxia-induced low pulmonary blood flow than during baseline, normal pulmonary blood flow.[40] Finally, higher plasma lidocaine levels were initially observed when lidocaine was administered endotracheally to dogs with hypoxemia than to dogs that were not hypoxemic.[41] However, this study did not find a difference in the pharmacokinetics of lidocaine (i.e., pharmacological effect) in hypoxemic and nonhypoxemic dogs when the drug was administered via the ET route.

Despite evidence indicating that cardiopulmonary arrest does not inhibit absorption of endotracheally administered medications, other studies have indicated that ET drugs are unreliable during cardiopulmonary arrest.[20] [22] [23] [42] [43] In addition, in a neonatal ventricular fibrillation model using newborn piglets, 0.01 mg/kg ET epinephrine did not produce significant increases in serum epinephrine levels or in mean arterial pressures.[44] While high-dose epinephrine has never been recommended for neonates due to a fear of hypertension and intracranial hemorrhage, these data suggest that using the higher dosage of the recommended range (0.01 to 0.03 mg/kg) might be a reasonable alternative. More importantly, these studies serve to emphasize that ET drug administration should not be used in lieu of attempts to obtain definitive access to the systemic circulation. ET drug administration should not be performed when another more direct means of accessing the central circulation is available.

INDICATIONS

ET drug therapy is indicated whenever there is a need for emergent pharmacologic intervention and other access, either IV or IO, is not readily available. This most frequently occurs in patients with poor veins (e.g., chemotherapy or dialysis patients, intravenous drug users, elderly patients), in patients with small or obscured veins (e.g., pediatric patients, burn patients, obese patients), or during prehospital cardiovascular collapse.

Specific indications for the delivery of a drug endotracheally are the same as those for IV and IO administration. However, there are only a limited number of emergency drugs that can be given safely and effectively by the ET route (Table 27-2 and Table 27-3). Medications that have been administered endotracheally and found to be safe and effective in both experimental animal models and human studies or case reports include epinephrine, [10] [12] [45] atropine, [46] [47] [48] lidocaine, [36] [39] [41] [49] and naloxone. [50] [51]

Diazepam has also proven effective. [52] [53] However, in one animal model, it produced pneumonitis when 0.5 mg/kg was administered via the ET route. [54] Because diazepam is sparingly soluble in water, it is available only in a solution of

TABLE 27-2 -- Endotracheally Administered Drugs Shown to Be Effective Experimentally and Clinically

Atropine
Diazepam*
Epinephrine
Flumazenil†
Lidocaine
Naloxone

*See text—effective, but produced pneumonitis in one animal model. Midazolam may be preferred when rapidly active anticonvulsant must be given via the endotracheal route.
†See text—effective in one human study in elective surgery patients. Clinical application in the emergency department remains uncertain.

propylene glycol, ethanol, and benzyl alcohol. It is unknown if the reported pneumonitis was due to the direct effects of the diazepam or to that of the diluent.

In one case report, 5 mg of diazepam (i.e., approximately 0.1 mg/kg) was administered via a tracheostomy to an adult female, resulting in cessation of seizure activity within 2 minutes. [53] In this report, no change in arterial blood gas values or chest x-rays was noted over the ensuing 5 days. Whether the apparent lack of adverse pulmonary effects in this patient implies that diazepam can be safely used via the ET route is still speculative. The lower dose used (0.1 mg/kg) may have been a factor in reducing pulmonary damage. A further issue, given the low solubility of diazepam in water, is the ability to dilute stock diazepam solutions to a total volume of 10 mL (as is recommended for ET drug administration). A volume of 10 mL of propylene glycol or ethanol used as diluent may further compromise pulmonary function. Additional studies are needed to resolve these issues, but some authors and the American Heart Association have removed diazepam from their list of medications that can be given safely via the ET route. [15] [55] [56]

It is of interest to note that in one study of midazolam, no pathologic changes were seen in lung sections after drug administration. [57] In addition, midazolam is available commercially in aqueous solution and could, therefore, be diluted with normal saline or sterile water for ET administration. Given the less irritating solvent solution used for commercial midazolam, this agent seems to be the better choice when a rapidly acting anticonvulsant must be given via the ET route. Fortunately, both diazepam and midazolam are readily administered and effective when given rectally.

Experimental studies of vasopressin, [58] midazolam, [57] propranolol, [47] and metaraminol [59] in animal models suggest that these medications also may be effective when administered endotracheally, but no clinical studies have been conducted to date to verify these findings. In 6 patients undergoing elective surgery, Palmer and colleagues demonstrated that therapeutic blood levels of flumazenil were obtained

TABLE 27-3 -- Endotracheally Administered Drugs Shown to Be Effective Experimentally but Not Proven Clinically

Flumazenil
Metaraminol
Midazolam
Propranolol
Vasopressin

within a minute after ET delivery of 1 mg of drug diluted in 10 mL of saline. [60] Interestingly, aliquots of 0.1 to 0.2 mg of the drug are currently recommended during IV administration. The role of flumazenil by this route at these doses remains to be determined.

CONTRAINDICATIONS

At present, the only true contraindication to the ET delivery of an appropriate drug is the presence of another form of access to the systemic circulation through which the needed drug can be delivered rapidly and effectively. The pharmacokinetics and pharmacodynamics of ET drugs delivered during various states of cardiovascular or pulmonary compromise are to a large extent unknown, and, for this reason, more conventional routes of rapid and effective drug administration should be used when available.

No drug should be delivered by the ET route without experimental or clinical evidence to support its effectiveness and safety. A complete list of drugs that are contraindicated relative to delivery by the ET method is not available. Specific emergency medications that have been shown to be ineffective or unsafe when given via the ET route include sodium bicarbonate, isoproterenol, and bretylium. In a study using a canine model, sodium bicarbonate was shown to inactivate lung surfactant.[61] Isoproterenol, even when given in doses 10 times the IV dose, failed to produce significant changes in arterial blood pressure or heart rate. [47] Studies of bretylium also indicate low serum levels after ET administration, even when administered at doses of 20 mg/kg. [62]

EQUIPMENT

Patients in need of ET drug therapy first require management of the airway, usually in the form of tracheal intubation. Once this procedure has been performed (see Chapter 4), little other equipment is needed for the ET delivery of a drug. It should be noted that studies of endobronchial administration of medications via a Combitube (Kendall-Sheridan, Argyle, NY) or laryngeal mask airway (LMA) [Intavent International SA, Henley-on-Thames, UK] reveal subtherapeutic absorption of drugs when administered at recommended ET doses.[63] [64] [65] Kofler and colleagues determined that use of epinephrine with a Combitube required 10 times more epinephrine than that used with an ET tube to obtain the same serum concentration and hemodynamic effects. [63] The studies using a Combitube specifically evaluated effectiveness when the tube was placed in the esophagus, requiring medications to travel out the side holes to reach the trachea. Presumably, a Combitube that enters the trachea directly would function equivalently to an ET tube, but no studies have been done to support this assumption.

In the Procedure section that follows, several different techniques for ET drug administration are described. The equipment listed is required to perform any of the four different techniques described. This equipment is suggested for the ideal situation or technique, or both. *At no time should drug delivery be delayed while searching for the "perfect" piece of equipment.*

1. Manual bag-valve ventilation device capable of delivering an FiO_2 of at least 50%. In circumstances in which ET drug delivery is indicated, the patient's condition almost

always warrants supplemental oxygen. Although the technique may not result in any significant deterioration in respiratory function, it is still advisable to administer additional oxygen after drug delivery. The bag-valve ventilation device also is used to deliver several rapid insufflations immediately after drug delivery to assist delivery of the drug distally, where it may be absorbed more rapidly and effectively. [33]
2. A fine-bore catheter or special ET tube to deliver the drug at or beyond the distal end of the ET tube. The ideal catheter is at least 8 Fr in size and at least 35 cm (14 in.) in length. The most effective length or diameter of the catheter is unknown, but it is recommended that the catheter be long enough to protrude past the distal end of the ET tube. The diameter of the catheter should be large enough to allow rapid delivery of a 10-mL volume of solution. Several different types of tubes and catheters commonly available in the emergency department can be used for this purpose:

   A 16-ga central venous pressure or cutdown catheter. Since most of these catheters are only 30 cm long, the proximal end of the ET tube requires shortening if the catheter is to protrude past the distal end of the ET tube.

   An 8 or 10 Fr polyethylene pediatric feeding tube (e.g., Argyle, St. Louis). These tubes are much longer than needed and should be cut to the desired length to reduce dead space. Luer-Lok-type syringes and IV adapter locks both fit nicely onto the proximal end of the tube.

   An 8 Fr (or larger) pediatric pulmonary suction catheter without the control port. Because this catheter is designed to extend past the tip of the ET tube, it is an ideal length. However, with some brands, it is difficult to attach a syringe or IV adapter lock after the suction control port is removed.

   Alternatively, two ET tubes with built-in ports are available. They allow ET instillation of drugs without removing the bag ventilation device. Unless these special tubes are used for all patients requiring intubation, the major drawback is the obvious need to re-intubate or decide before intubation if IV access is expected to be a problem. In these cases, the patient can be intubated using one of these specialty tubes. The available ET tubes include the following:

   ET tubes designed for bronchoscopy (e.g., Hi-Lo Jet Tracheal Tube, Mallinckrodt Medical, St. Louis, MO). These tubes have two additional ports, one used for monitoring or irrigation (opaque lumen) and one used for jet ventilation (transparent lumen). They are available in adult and pediatric sizes (cuffed and uncuffed). In a porcine cardiopulmonary arrest model, successful resuscitation using this tube was comparable to resuscitation using other forms of ET drug administration.[34] The major disadvantage of this ET tube is the need to be familiar with the specific ports before use. Determining which is the irrigation port if one has never seen the tube previously could be time consuming. In addition, the port requires placement of an IV adapter lock to use prefilled syringes (in which most emergency medications are now supplied).

   ET tube with side port (ETSP) [EMT tracheal tube, Mallinckrodt Medical, St. Louis, MO]. This tube is designed specifically for ET drug administration. The instillation lumen opens into the tube at the eye, approximately 1 cm from the end of the tube. The injection port has an IV adapter lock, which makes it amenable to use with prefilled syringes. The ETSP is not available in pediatric sizes. In addition, in one study comparing the administration of lidocaine via the ETSP vs administration to the proximal end of the standard ET tube, serum lidocaine measurements never reached therapeutic levels in the ETSP group [in contrast to the ET group and IV control group]. [66]

3. An IV adapter lock. This can be placed as needed onto the proximal end of the ET tube or catheters described previously to convert them for use with prefilled syringes. This adapter is usually unnecessary if a standard syringe is used.
4. A 10- to 20-mL syringe, preferably a Luer-Lok type, big enough to deliver the desired volume of drug solution plus an additional 5 mL of air. Unfortunately (for ET drug therapy), most of the medications now prescribed for emergency situations come in prefilled syringes with built-in needles (Fig. 27-1). This type of apparatus usually does not allow one to draw up an additional volume of air to empty the catheter of solution. Also, the needle on the prefilled syringe requires an IV adapter lock in order to adapt to most catheters for injection.
5. Desired diluent solution. As previously described, the drug should be delivered in a final volume of 10 mL for adults, 5 mL for children, and 1 mL for neonates. An adequate volume of diluent, usually normal saline, must be available. Distilled water is an acceptable alternative.
6. Desired medication to be instilled (see Table 27-2).
7. An 18- or 19-ga needle for use in drawing up the drug solution or for injecting the solution, or both. Use of an 18-ga, 8.9 cm (3.5 in.) spinal needle is recommended for direct instillation of medications into the proximal end of the ET tube.
8. Alcohol wipes for cleaning vials or injection ports, or both.
9. Gloves, mask, and eye protection. After instillation, the solution often refluxes out of the ET tube, making blood and body fluid precautions of paramount importance.

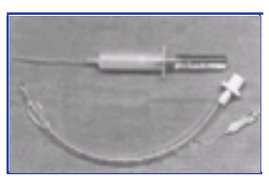

Figure 27-1 Example of a prefilled syringe, which is frequently used in endotracheal therapy.

PROCEDURE

Four procedures are described in this section, listed in order of generally accepted use, but definitive studies have not

clearly identified the best technique. Most important, the procedure of choice is the method that will deliver the medication to the patient in the timeliest fashion. As stated previously, one should not delay drug administration to find the perfect piece of equipment or diluent (normal saline or distilled water are both acceptable in most cases).

It is assumed for all procedures that vials and IV adapter locks will be cleaned with alcohol wipes before use and that universal precautions will be practiced at all times. In addition, all patients require prior intubation (see Chapter 4), and the ET tube should be secured to prevent the tube from being expelled if the patient coughs. If present, the cuff of the tube should be inflated.

Use of a Catheter

Provide ventilations with the bag-valve ventilation device with supplemental oxygen while the required drug dose is drawn up in either a 10- or a 20-mL syringe. Dilute the drug with normal saline as needed to attain a total volume of 10 mL (for adults), 5 mL (for children), 1 mL (for neonates). Draw back the plunger to add 5 mL of air to the liquid in the syringe. If the drug to be delivered is in a prefilled syringe, place an IV adapter lock on the catheter. The syringe can be attached to the catheter at this time or once the catheter has been placed within the ET tube (Fig. 27-2).

Interrupt the connection between the proximal end of the ET tube and the bag-valve ventilation device. Place the catheter within the lumen of the ET tube, ideally in such a

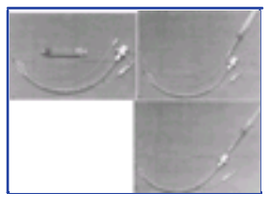

Figure 27-2 Use of a central venous catheter for endotracheal drug delivery.

manner that the distal end of the catheter extends ~1 cm beyond the distal end of the ET tube. For the catheter to reach deep enough, the proximal end of the ET tube may need to be cut to a shorter length. Hold the proximal ends of the catheter and the ET tube at all times during the procedure. If it has not already been done, attach the syringe to the catheter. If external cardiac compressions are being performed, *they should be interrupted during drug delivery*. Inject the drug solution rapidly and forcefully through the catheter into the trachea followed by the 5 mL of air needed to flush the catheter of any remaining drug solution. Promptly remove the syringe and catheter from the ET tube. If the patient makes an effort to cough, place a thumb over the opening of the ET tube to prevent expulsion of the solution. As soon as possible after drug delivery, reconnect the bag-valve ventilation device with supplemental oxygen to the ET tube. Deliver five rapid ventilations to promote distal dispersion of the drug. Resume chest compressions if necessary.

Direct Instillation into the Endotracheal Tube

When special ET tubes or catheters are not available, the drug may be instilled directly into the proximal end of the ET tube. Studies using this technique have diluted the medication with normal saline and also flushed the tube with normal saline after the drug is instilled. [34]

While the patient is being ventilated, draw up the desired drug into a syringe (or use a prefilled syringe). Because no catheter is used, there is no need to draw up an additional

volume of air into the syringe. Dilute the drug to a final volume of 10 mL (adults), 5 mL (children) or 1 mL (neonates) with normal saline or distilled water. Attach an 18- or 19-ga needle (some authors recommend using an 8.9 cm [3.5 in.] spinal needle). If using a prefilled syringe, in a second syringe, draw up an appropriate volume of normal saline such that the total instillation volume (drug plus saline) equals 10 mL (adults), 5 mL (children) or 1 mL (neonates). Attach an 18- or 19-ga needle. This will be used to flush the ET tube after drug instillation.

Interrupt the connection between the proximal end of the ET tube and the bag-valve ventilation device. Insert the needle of the first syringe (drug) into the proximal opening of the ET tube. Hold the proximal end of the needle with one hand to prevent loss of the needle into the tube. Discontinue cardiac compressions, and inject the drug solution rapidly and forcefully. If using a prefilled syringe, flush the tube immediately with the diluent in the second syringe. If the patient makes an effort to cough, place a thumb over the opening of the ET tube to prevent expulsion of the solution. Reattach the bag-valve ventilation device and deliver five rapid insufflations. Resume chest compressions if needed.

Use of Endotracheal Tubes with Irrigation/Drug-Delivery Lumens

Unless these ET tubes are used for every intubation, their usefulness in emergent ET drug delivery is dependent on the provider's recognition before intubation that IV access will be problematic. If this is recognized, intubate the patient using an ET tube with monitoring lumen (e.g., Hi-Lo Jet ET tube) or with an ETSP (e.g., EMT tracheal tube). While ventilating the patient with supplemental oxygen, draw up the required drug dose in either a 10- or 20-mL syringe with an 18- or 19-ga needle attached (or use a prefilled syringe). If necessary, dilute the drug with normal saline or distilled water to attain a total volume of 10 mL (adults), 5 mL (children) or 1 mL (neonates). Draw back the plunger to add 5 mL of air to the liquid in the barrel of the syringe. Attach the syringe or insert the needle into the IV adapter lock on the ET tube monitoring/irrigation lumen (Hi-Lo Jet tube) or on the drug-delivery lumen (EMT tube). Discontinue chest compressions and rapidly and forcefully inject the drug solution during the inspiratory phase of ventilation (i.e., when the bag is squeezed). Deliver five rapid hyperventilations with the bag-valve ventilation device and resume chest compressions if necessary.

The advantage of this method of drug delivery is that it does not require interruption of the connection between the bag-valve ventilation device and the ET tube. In addition, it offers the theoretical advantage of allowing drug delivery during the inspiratory phase of ventilation, although at least one study has shown that this is not an important factor. [35]

Injection Through the Endotracheal Tube Wall

This method of drug delivery has not yet been evaluated scientifically but has been used clinically. [67] [68] While the patient is being ventilated, draw up the desired drug into a syringe with an attached 18- or 19-ga needle (or use a prefilled syringe). Dilute the drug with normal saline or distilled water to attain a total volume of 10 mL (adults), 5 mL (children) or

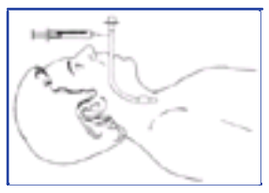

Figure 27-3 Method of drug injection through the endotracheal tube wall.

1 mL (neonates). No additional air needs to be added to the syringe. Insert the needle into the side of the ET tube proximally (Fig. 27-3). Discontinue chest compressions and rapidly and forcefully inject the drug solution into the ET tube during the inspiratory phase of ventilation (i.e., when the bag is squeezed). Deliver five rapid hyperventilations with the bag-valve ventilation device and resume chest compressions, if necessary. As with ET tubes with drug-delivery lumens, this technique requires no interruption of the connection between the bag-valve ventilation device and the ET tube. In addition, placing an IV adapter lock on the needle allows it to be left inserted in the ET tube for use with additional medications. [68]

COMPLICATIONS

Reported complications of ET drug therapy are rare, due in part to the infrequent use of this technique. In addition, because most or all patients who receive ET drug therapy are in cardiopulmonary arrest or are otherwise critically ill, it is difficult to ascertain if an adverse outcome is the result of the therapy or the patient's underlying condition.

With regard to the techniques of ET drug administration, no serious complications have been reported. A theoretical complication is the loss of a needle or catheter down the ET tube, but specifically holding the catheter or needle while instilling the drug can prevent this complication. Thus the techniques of ET drug administration seem to provide a safe method of drug delivery.

Following ET drug administration, the administered emergency drugs may produce adverse effects. Epinephrine administered during CPR has been noted to produce prolonged hypertension, tachycardia, and dysrhythmias after the return of a perfusing rhythm in both animal models [24] and human case reports.[12] It appears that this side effect is related to the depot effect, in which larger doses of drugs administered endotracheally are released slowly over time (similar to an IV drip). In addition to epinephrine, atropine and lidocaine also exhibit a depot effect when administered endotracheally. [61] No serious long-term sequelae have been reported, however, due to this effect.

A second area of potential concern with ET drug therapy is a transient decrease in arterial oxygen content during or after drug delivery. As discussed previously, if total volumes

are maintained between 5 and 10 mL in adults, the effect on pulmonary function appears minimal and likely of no clinical consequence. It should be remembered, however, that any decrease of PaO_2 in a critically ill patient, regardless of how small or how transient, may have deleterious effects. The potential for this adverse effect should always be considered when administering drugs by the ET route, and supplemental oxygen should always be administered in an effort to improve oxygenation and offset any transient drop in arterial oxygen content that might develop.

CONCLUSION

It is important that clinicians be familiar with ET drug therapy as it may be a life-saving procedure in some emergent situations. The great variability in the effectiveness of this procedure, however, mandates that it be used only when IV or IO access is not available. Little information is available on the pharmacokinetics, pharmacodynamics, and effectiveness of medications administered via the ET route during cardiopulmonary arrest. Consequently, the ideal dose, diluent, volume, and technique have yet to be determined and may be drug and situation dependent. Despite these limitations, ET drug therapy has been beneficial for some patients. If other access is not available, appropriate medications should be delivered via the ET route using the procedure that will ensure delivery to the patient in the shortest time period.

Acknowledgment

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Chapter 28 - Autotransfusion (Autologous Blood Transfusion)

Charlene Babcock Irvin

Traumatic injury affects men and women during their most productive years.[1] This fact, coupled with increasingly efficient and rapid emergency transportation systems, has resulted in growing numbers of traumatic injury victims arriving at emergency facilities in potentially salvageable condition. The ensuing urgent demand for blood often exceeds the immediately available supply of homologous banked blood.[2] Successful approaches to this problem have included earlier hemostasis (i.e., definitive surgery), use of volume expanders (crystalloid, colloid), and autotransfusion. Other approaches to treat acute hemorrhagic shock that are currently under investigation include the use of hypertonic saline[3] and manufactured blood substitutes.[4]

Autotransfusion may be defined as "collection and reinfusion of the patient's own blood for volume replacement."[5] Emergency autotransfusion most often involves collection of shed blood from a major body cavity, usually the pleural space (hemothorax) and occasionally the peritoneal space. Autotransfusion in the emergency department (ED) is generally limited to acute hemothorax with clinically significant hypovolemia. The following discussion examines the advantages and potential complications of emergency autotransfusion, patient selection, available equipment, and procedural technique for several widely used devices.

While autotransfusion is a basic technique that is theoretically possible in any ED, it is not a standard of care under all settings. Since the procedure requires familiarity with the equipment and continuing education and quality control, it may be counterproductive to institute it in a hospital that has a low trauma census or in a setting where it will be used infrequently enough that staff education issues are problematic. Autotransfusion will be most useful in trauma centers or high-trauma-volume community hospitals where the technique is used often enough to be considered routine by clinicians and nursing staff.

BACKGROUND

Reports of autotransfusion can be found in the Western medical literature as early as 1818. Blundell, [6] an English practitioner, considered the possibility of reinfusion of shed blood after witnessing a woman exsanguinate from uterine hemorrhage. His subsequent work with autotransfusion of shed blood in dogs suggested that the procedure was clinically feasible. In 1886, Duncan [7] used the technique without noticeable ill effects to reinfuse blood shed during an amputation. In 1914, the use of the technique in ectopic pregnancies was popularized by Thies [8] , and 3 years later Elmendorf[9] published a description of the first case of autotransfusion from traumatic hemothorax. Also in 1917, Lockwood [10] used the procedure for the first time in the United States during a splenectomy. In 1922, Burch [11] reviewed 164 cases of autotransfusion published over the preceding 8 years in the European medical literature, largely originating from Germany.

The discovery of ABO blood typing at the turn of the century and the institution of blood banks in the 1930s led to the almost exclusive use of homologous blood up to and following World War II. Interest in autotransfusion concomitantly declined, and only sporadic reports appeared in the literature during this period. During the 1960s and 1970s, cardiopulmonary bypass surgery generated extensive data regarding intraoperative retrieval of large quantities of blood for reinfusion. Concurrently, the Vietnam War created a new demand for readily available blood in areas remote from conventional reserves of homologous bank blood. Thus, revitalized interest, coupled with growing experience, generated the early publications of such investigators as Dyer and associates, [12] Klebanoff and colleagues[13] [14] , and Symbas and coworkers,[15] thus initiating the "new era" of autotransfusion.

ADVANTAGES

The advantages of autotransfusion over banked blood transfusion in patients who are hypovolemic from traumatic blood loss include the following:

1. Shed blood is immediately available (collection and initiation of reinfusion can be accomplished within minutes).
2. Blood compatibility is not a problem, which avoids both transfusion reactions and delays associated with crossmatching.
3. Shed blood when rapidly reinfused is normothermic, consequently reducing the complications related to the administration of room-temperature fluids. [16]
4. The risk of indirect patient-to-patient transmission of infectious diseases (e.g., hepatitis, malaria, cytomegalovirus, or human immunodeficiency virus) is eliminated. [17]
5. Levels of 2,3-diphosphoglycerate (2,3-DPG) have been found to be significantly higher in autotransfused red blood cells (RBCs) than in stored homologous cells. [18] [19]
6. There are no direct complications due to hypocalcemia or hyperkalemia. [20] [21]
7. There is less risk of inadvertent circulatory overload and adult respiratory distress syndrome (ARDS). [22]
8. Autotransfusion allows preservation of limited stores of banked blood, thereby ensuring its availability for other uses. [17]
9. Autotransfusion lowers the cost of medical care. [17] No blood drawing, typing, or crossmatching is required; thus, time, money, and personnel expenditures may be conserved. Davidson[23] reported the cost of autologous blood to be three- to eightfold cheaper per unit compared with banked blood.
10. Reinfusion of autologous blood may be acceptable to those patients whose religious convictions prohibit transfusions with homologous blood. Techniques for intraoperative or extraoperative collection of autologous blood that involve blood storage or reinfusion of shed blood are objectionable to Jehovah's Witnesses. Nonetheless, salvage when extracorporeal circulation is

uninterrupted may be acceptable to many members of that religion. [24]

PATIENT SELECTION

Indications

In general, all victims of severe trauma, whether blunt or penetrating, should be considered potential candidates for autotransfusion. More specifically, Reul and colleagues[25] have described three categories of patients for whom emergency autotransfusion is suitable. The ideal candidate is a blunt or penetrating trauma victim with a hemothorax containing 1500 mL or more. A second category is the hemorrhaging patient with an immediate need for transfusion, for whom insufficient homologous blood is available because of the urgency of the situation, a blood bank shortage, or a difficult crossmatch. Under these circumstances, Reul and coworkers used autotransfusion regardless of the type of injury or degree of contamination. A third category is the patient with massive blood loss (less than one whole-body blood volume) for whom autotransfusion can serve as a supplement to homologous replacement. O'Riordan [21] added a fourth category, the trauma patient who urgently requires blood transfusion and whose religious convictions prohibit homologous transfusion.

It is reasonable to consider use of autotransfusion in all patients who have a hemothorax and require even minimal blood replacement. When the need for homologous blood transfusion is borderline, autologous blood can be readily reinfused without the risk of complications associated with banked blood. Reasonable indications for initiating blood collection for possible autotransfusion may be summarized by these four situations:

1. Blunt or penetrating chest trauma with significant hemothorax (500 mL or more) suggested on a chest film
2. Multiple trauma with shock of uncertain etiology when emergent tube thoracostomy is performed prior to chest film
3. Emergency thoracotomy
4. Any hemothorax when there is an urgent need for blood and the patient's religious beliefs prohibit homologous transfusions

Because of the equipment required and the need for specialized care, autotransfusion is generally performed in the ED, operating room (OR), or intensive care unit (ICU) settings. Although seldom used in the out-of-hospital setting, it may be useful under certain circumstances. Barriot and colleagues reported on the emergency medical services of the Paris Fire Brigade's experience with autotransfusion from a hemothorax. They concluded that this technique may be effectively used in the out-of-hospital setting.[26] [27]

Contraindications

Reul and coworkers[25] suggested the following general contraindications to the use of emergency autotransfusion:

1. The known presence of malignant lesions in the area of traumatic blood accumulation.
2. Known renal or hepatic insufficiency (relative contraindication based on individual scenario).
3. Wounds older than 4 to 6 hours (because of the theoretical problem of bacterial overgrowth).
4. Gross contamination of pooled blood, usually as a result of injury to the gastrointestinal tract.

Huth and colleagues[28] found that major combined pancreatic-proximal intestinal injury may also constitute a contraindication owing to the potential for systemic reinfusion of large amounts of pancreatic enzymes.

Depending upon the clinical situation, particularly the urgency of the need for blood, some of these contraindications may be overruled by the lack of available (banked) blood. The National Blood Resource Education Program Expert Panel [29] has limited contraindications to active infection or gross contamination and the possibility of malignant cells in the salvaged blood. Investigation has confirmed that even when using systems that separate and wash cells, tumor cells will be resuspended and reinfused to the patient.[30]

Several investigators believe that the reinfusion of limited amounts of contaminated blood from the peritoneal cavity may be accomplished with an acceptable risk, [31] [32] [33] but the current consensus is that exsanguinating hemorrhage is the only acceptable indication for autotransfusion when there is recognized intestinal contamination. Indeed, Klebanoff[34] believes that autotransfusion has "no place" when there is extensive fecal or urinary contamination of the pooled blood. Thus, the advisability of autotransfusing possibly contaminated blood from the peritoneal cavity remains controversial (see Complications later in this chapter).

EQUIPMENT AND MATERIALS

Autotransfusion Units

While there are several commercially available systems that provide easy initiation of autotransfusion in the ED setting, items currently available in most EDs will also allow rapid infusion of autotransfused blood. Symbas [35] described a simplified collection system using standard ED materials (Fig. 28-1). After insertion of a chest tube, drainage is established into a standard chest tube bottle containing 400 mL of normal saline, maintaining a suction of 12 to 16 mm Hg. If autotransfusion is required, the collected blood in the chest bottle is reinfused in one of two ways:

1. The chest bottle may be disconnected from the pleural drainage tube and simply inverted on an intravenous (IV) stand for reinfusion through a filter into the patient. During infusion, a second sterile chest bottle with saline is connected to the chest tube for continuing collection.
2. After disconnection from the pleural drainage tube, the chest bottle may be connected to a standard blood collection bag and the salvaged blood transferred to this bag for subsequent reinfusion through a blood filter in the conventional manner. Symbas [35] reported on more than 400 patients autotransfused by this method since 1966, with no adverse effects attributable to the procedure.

Two other highly simplified techniques have been described that do not involve use of commercial autotransfusion units. Schweitzer and coworkers [36] reported successful autotransfusion in dogs by means of a chest tube connected to a Heimlich flutter valve (Bard-Parker, Rutherford, NJ). The valve is connected in turn to a 1900-mL Abbott Receptal blood collection bag. Drainage is entirely by gravity; no

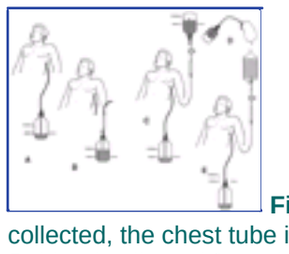

Figure 28-1 Technique of autotransfusion from traumatic hemothorax. *A*, Blood is collected into sterile blood collection bottle with 400 mL normal saline. *B*, After blood is collected, the chest tube is disconnected from the collection bottle. *C*, Blood is infused directly from the blood collection bottle while a new chest tube drainage bottle is connected to the chest tube. Or, *D*, blood collected from the chest tube is transferred into a sterile blood bag. *E*, Blood transferred into blood bag is infused into patient. *(From Symbas PN: Extraoperative autotransfusion from hemothorax. Surgery 84:722, 1978.)*

suction is applied (Fig. 28-2). Similarly, Barriot and colleagues [26] [27] described the European experience with a device called a Hemotraum that is adapted for out-of-hospital autotransfusion. Blood from the chest tube fills a 750-mL sterile bag by gravity via a 120-µm micropore filter. When full, the bag is clamped and disconnected, and the collected blood is reinfused through a 50-µm micropore filter. While this blood is being transfused, a second sterile bag is connected to the chest tube. No anticoagulant is used. Neither

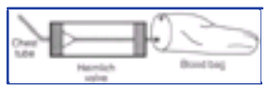

Figure 28-2 Heimlich valve connected directly to Abbott Receptal Bag. *(From Von Koch L, Defore WW, Mattox KL: A practical method of autotransfusion in the emergency center. Am J Surg 133:770, 1977.)*

of these techniques has undergone clinical trials in the United States. They may offer advantages for autotransfusion on the battlefield, in small rural hospitals, or the out-of-hospital care setting.

Although the described method for autotransfusion using these easily assembled items did not include the addition of anticoagulant, it may be appropriate to add an anticoagulant, especially when blood is rapidly accumulating from a great vessel injury (see Anticoagulation later in this chapter).

There are many types of commercially available autotransfusion systems designed for intraoperative use, but only two are practical for use in the ED. One type uses a chest tube to withdraw shed blood into an autotransfusion bag, while the other type uses a suction catheter. Systems using a chest tube differ based on the presence of an in-line thoracostomy unit incorporating a water seal. Several units offer this feature, including the Pleur-Evac, Thora-Klex, and Sahara autotransfusion systems (Genzyme Biosurgery Corporation, Falls River, MA: http://www.genzymebiosurgery.com/1-800-367-7874), the Atrium Ocean and Oasis ATS systems (Atrium Medical Corporation, Hudson NH: http://www.atriummed.com/1-800-528-7486), and the Throa-Seal and Aqua-Seal (Kendall Health Care, Mansfield, MA: www.kendallhq.com/1-800-962-9888).

The Abbott Receptal System, manufactured by Abbott Laboratories (Abbott Park, IL: http://abbott.com/1-847-938-4310), uses a chest tube and blood collection unit, but does not include a standard chest tube drainage system with a water seal. This device uses wall suction to withdraw blood through the chest tube into a blood collection bag for future reinfusion. Because there is no chest tube drainage system in series with this device, it is unable to detect air leaks. It also has no overflow protection. In other autotransfusion units in series with disposable chest tube drainage systems, excess blood spills over to the collection chamber of the chest tube drainage unit.

Von Koch and associates[37] reported their experience with the Sorenson unit (now the Abbott Receptal system) in autotransfusing an average of 1000 mL of salvaged blood in 30 trauma patients. They found that this unit could be assembled quickly and easily operated. Its use resulted in minimal air-blood interfacing (a source of hemolysis). They also described the unit as "efficacious, inexpensive, cost-effective, and safe." Davidson [23] described the step-by-step use of this system in the ED and characterized it as "simple to use, efficacious, cost-effective, and safe" for the emergency setting. Autotransfusion using this device is described in the following section.

ED autotransfusion systems that use a suction catheter instead of a chest tube include the Bloodless Surgery Station (Harvest Blood Conservation Inc., Plymouth, MA: www.harvesttech.com/1-877-842-7837) and the Autovac Intraoperative (IO)(Boehringer Laboratories, Norristown, PA: http://www.boehringerlabs.com/1-800-642-4945). The Harvest unit is freestanding and can be connected to a standard electrical outlet. This system provides its own suction while collecting blood through a special suction catheter. It is capable of suctioning 3.75 liters of blood per minute with less than 100 mm Hg suction pressure. The Autovac unit is designed to suction blood directly to a reservoir that is connected in series to wall suction (similar to the Abbott Receptal but collection of blood occurs from a suction catheter instead of a chest tube).

Blood Filters

In-line filtration is used routinely during reinfusion of blood products to reduce the danger of microembolization and resultant pulmonary insufficiency. [20] [38] [39] The relationship between the presence of microaggregates and the development of acute respiratory distress syndrome (ARDS) [39] is controversial; however, most investigators advise some form of micropore filtration. Pore size seems to be the only issue, and recommendations range from 20 to 170 µm. [40] [41] The majority of

investigators believe that a pore size of 40 µm minimizes the risk of microembolization without undue elevations in filtration pressures. [16] [25] [42]

Vacuum Suction

There is general agreement that the level of vacuum suction should be limited to minimize red cell hemolysis. [43] The exact level at which clinically significant hemolysis occurs is uncertain and there is wide variation (5 to 100 mm Hg) in the levels used by various investigators. [23] [25] [37] [40] [43] Dyer and coworkers[12] reported that levels below 100 mm Hg kept hemolysis to a minimum. Pressure of 60 mm Hg or less has been preferred by several researchers for aspiration of hemothorax or hemoperitoneum,[25] [37] [40] but in the operating room, adequate suction to maintain a bloodless surgical field may require pressure well over 100 mm Hg. [12] [31] [41] With most commercial systems, the manufacturer's recommendations specify a suction limitation of 150 mm Hg.[44] A second vacuum source of 300 mm Hg should be available for occasions when high-flow suction is required. [45]

To minimize hemolysis, reducing the air-blood interface is as important as limiting the vacuum level. Therefore, suctioning should be from pooled blood whenever possible, with the suction tip kept well below the surface of the blood. [46] [47]

Anticoagulation

Anticoagulation of the aspirate during autotransfusion has been accomplished using several methods. These include local and systemic heparin, [13] [48] [49] acid citrate dextrose (ACD),[20] [41] [50] citrate phosphate dextrose (CPD),[25] [37] and normal saline (i.e., no anticoagulant). [15] [27] Local heparinization of the tubing and reservoir may lead to the formation of platelet microaggregates on the filter and in the line [25] [51] and systemic heparinization could lead to further life-threatening hemorrhage in an already bleeding patient.[25] [34] [51] Therefore, the use of heparin as an anticoagulant during emergency autotransfusion of the trauma patient is discouraged by most investigators.[25] [34] [52]

In several early studies, ACD was used as an alternative to heparin. [20] Raines and coworkers[41] found no clinical or laboratory evidence of intravascular coagulopathy after autotransfusion using ACD, even in patients who received more than 8000 mL of autologous blood. More recent studies report the use of CPD for extracorporeal anticoagulation. CPD avoids the complications of heparinization, [25] necessitates less volume as an anticoagulant, and results in less acidosis than does ACD.[25] [46] [53] Reul and colleagues[25] found CPD to be well-tolerated, even in large amounts. CPD undergoes such rapid metabolism that anticoagulation is confined, to a large degree, to blood in the autotransfusion apparatus. Rarely, excessive use of CPD can cause citrate intoxication because of chelation of calcium and subsequent cardiac dysrhythmias. Use of insufficient or outdated CPD may result in clotting of collected blood.

Investigators have recommended a range of 25 to 70 mL of CPD per 500 mL of collected blood.[25] [37] A 1:7 ratio of CPD to blood has also been suggested, [46] [52] which is comparable to the standard 67 mL of CPD per unit of banked donor blood.[25] Klebanoff [34] stated that CPD is the safest method of anticoagulation for autotransfusion and that the use of CPD avoids the problem of clot formation on the blood filter, thus maintaining higher platelet counts in reinfused blood.

Davidson has noted that for the average chest wound, added anticoagulant may not be required. [23] Blood retrieved from pleural and abdominal cavities frequently will not clot because it is devoid of fibrinogen, [54] likely because moderate rates of bleeding allow time for defibrination by contact with serosal (pleural and peritoneal) surfaces and by mechanical agitation from respiratory and cardiac movements. Dog studies have documented pleural deposition of fibrin following experimental hemothorax, further substantiating this mechanism as the cause of hypofibrinogenemia in collected blood. [55] Others[15] [27] [56] report corroborating clinical findings and recommend simple reinfusion through a filter without any anticoagulant. However, wounds of the great vessels may

bleed at a rate that allows coagulable blood to enter the collection reservoir, clotting off the entire system. [23] [37] [52] In such an instance, an anticoagulant, specifically CPD, is appropriate.

PROCEDURE

Mattox[57] described the properties of the ideal autotransfusion device: (1) easy and quick assemblage, (2) cost-effectiveness, (3) easy operation, (4) in-line microfiltration, (5) minimization of air-fluid interfaces, and (6) simple anticoagulation technique. Several devices currently being marketed conform to these specifications, and these are discussed in detail.

Pleur-Evac Autotransfusion System

The Pleur-Evac autotransfusion device is an example of an autotransfusion system that allows for chest tube collection of blood in series with a disposable chest tube drainage system. It consists of a sterile, single-use, disposable, rectangular-shaped, flexible blood collection bag supported by a rigid metal frame. The bag and frame are designed to attach in series directly to a standard Pleur-Evac underwater seal chest tube drainage unit. On another type of Pleur-Evac, the water suction control chamber is replaced by a dry suction control with a dial that may be set to regulate suction between 0 and 40 cm H_2O, regardless of the amount of source suction applied. Inside the collection bag in line with the incoming drainage tube is a 200 µm nylon mesh filter. Attached to the collection bag are two latex tubes, one (red-tipped) for collecting shed blood from the drainage site and one (blue-tipped) for connection to the Pleur-Evac drainage unit. An injection port is provided on top of the autotransfusion system (ATS) bag for the addition of anticoagulant to collected blood.

Collection

1. The Pleur-Evac underwater seal drainage unit (Fig. 28-3 (Figure Not Available)) is first prepared in standard fashion and connected to a source of suction. Slide the metal hanger over the patient drainage tubing port on the right-hand side of the unit (Fig. 28-4), and pull it down flush with the top and side of the drainage unit.
2. Unwrap a replacement ATS bag and fit it into the wire frame provided (Fig. 28-5). Close both white clamps on the bag tubing, and place the frame and bag on the wire hanger.
3. Connect the ATS bag in series to the chest tube drainage system (see Fig. 28-3 (Figure Not Available)):
   a. Clamp the drainage tubing coming from the patient (*A*).
   b. Remove the red protective cap from the collection tubing (*B*) on the ATS replacement bag and connect it to the patient chest drainage tubing (*C*) using the red connector.
   c. Remove the blue protective cap from the suction tubing (*D*) on the ATS replacement bag and connect to the underwater seal drainage unit (*H*) using the blue connector.
4. Using a syringe and an 18-gauge (or smaller) needle, inject anticoagulant through the rubber diaphragm (*E*) on the ATS bag cap. The manufacturer of the Pleur-Evac unit does not offer recommendations regarding specifics of anticoagulation of blood collected in the unit; however, general guidelines are a 1:7 ratio of CPD to blood, or an ACD-to-blood ratio of 1:7 to 1:20.
5. Open all clamps; make sure all connections are airtight. The system is now operational.

Reinfusion

1. Removal of ATS bag from chest drain and patient chest tube:
   a. When the ATS bag is full, use the negative-pressure relief valve (located on top of the drainage unit) (see Fig. 28-3 *F* (Figure Not Available)) to reduce excessive suction in the unit, and close the white clamps on the chest drainage tubing and ATS bag.
   b. Disconnect all connections to the ATS bag.
   c. Attach the male (blue) and female (red) connectors on top of the ATS bag to each other, and remove the bag from the drainage unit by spreading and disconnecting each metal support arm.
2. ATS bag reinfusion setup and priming:
   a. Prepare a standard Y-type blood infusion line with a high-capacity 40-µm in-line micropore filter, prime the line and filter with normal saline, and connect it to a large-bore IV access (14-gauge or larger) (Fig. 28-6).
   b. Invert the bag so that the spike port (see Fig. 28-3 *G* (Figure Not Available)) points upward. Remove the protective cap and insert the free recipient arm of the Y tube infusion line into the spike port using a constant twisting motion.
   c. Return ATS bag to upright position and place on standard-height IV pole (Fig. 28-7).
3. Reinfusion using the Pleur-Evac autotransfusion bag may be assisted using a pneumatic pressure blood pump, not to exceed 150 mm Hg infusion pressure. However, if a pneumatic pump is used, all air in the bag must be removed before infusion to minimize the risk of air embolism. To accomplish this, the red and blue connectors may be disconnected temporarily, one clamp opened, and the bag slowly squeezed until all air is out of the unit. Then the clamp is closed again, and the red-to-blue connection reestablished.
4. During reinfusion, autologous blood collection may be continued with a second ATS bag, repeating steps 2 through 5 under Collection.

Atrium Ocean Series

Atrium Medical Corporation also offers an array of chest tube drainage systems with optional in-series autotransfusion bags. The Atrium 2050 is one example. This system is very similar to the previous chest drainage system with in-series autotransfusion capabilities.

Collection

1. After the Atrium chest tube drainage system has been appropriately set up, attach the blood collection bag hanger on the front face of the chest drain or adjacent bed rail.
2. Insert the Atrium ATS blood collection bag into the hanger (Fig. 28-8). Be sure both ATS clamps are firmly closed prior to connector cap removal.
3. Connect in-line ATS bag in series to chest drain:
   a. Close the patient chest tube slide clamp firmly and separate by depressing connector lock (Fig. 28-9).

   b. Remove cap from female ATS bag (*A*) connector and insert male patient chest tube connector (*B*) (Fig. 28-10).
   c. Remove second male ATS cap (*C*) and insert into female chest drain connector (*D*) (see Fig. 28-10). This will complete the connection of the ATS bag to the patient and the chest drain.
4. Open both in-line ATS bag clamps prior to opening the patient chest tube clamp. The patient tube and ATS bag should be free of dependent loops to ensure maximum drainage efficiency.
5. When required, anticoagulant should be added directly to the ATS bag through the anticoagulation injection site located on the front face of the ATS bag. After appropriate alcohol swabbing of the anticoagulant port, a 20-gauge or smaller needle can be inserted for administering anticoagulant via syringe. When anticoagulation is desired, the manufacturer recommends an ACD-to-blood ratio range of 1:7 to 1:20, or the use of CPD anticoagulation.

Figure 28-3 (Figure Not Available) The Pleur-Evac Drainage Unit with the autotransfusion bag attached. *A*, Clamp for chest tube drainage. *B*, Collection tubing on autotransfusion bag. *C*, Chest tube drainage tube. *D*, Suction tubing on autotransfusion bag. *E*, Anticoagulant injection port. *F*, Negative pressure relief valve. *G*, Spike port for reinfusion of collected blood. *H*, Suction tubing on chest tube drain to be connected to the transfusion bag or chest tube. *(Courtesy of Pleur-Evac, Genzyme Biosurgery, Cambridge, MA 02142.)*

Reinfusion

1. To remove the ATS bag from the chest drain and patient chest tube:

a. Securely close the patient chest tube clamp and both ATS bag clamps.
b. Disconnect the chest drain side first (*D*), and then disconnect the patient chest tube connector (*B*) (see Fig. 28-10).
c. Immediately place the male patient chest tube connector (*B*) into the female chest drain connector (*D*) and open the patient chest tube clamp (Fig. 28-11).
d. Reconnect the ATS bag connectors to each other (see Fig. 28-11).

2. To set up and prime the ATS bag reinfusion:
   a. Prepare a standard Y-type blood infusion line with a high-capacity 40-µm in-line filter, prime the line and filter with normal saline, and connect it to a large-bore IV access (14-gauge or larger) (see Fig. 28-6).
   b. Prime the IV blood administration set and microemboli blood filter with sterile saline.
   c. Invert the ATS bag with the spike port pointing upward and remove the tethered cap using a sterile technique.
   d. Insert the saline blood filter spike into the ATS bag using a firm twisting motion.
   e. Return the ATS bag to an upright position and place it on a standard-height IV pole.
   f. Open the filtered air vent located on top of the ATS bag first, followed by the IV clamp to complete priming. All remaining air within the IV circuit must be evacuated prior to patient connection.
3. During reinfusion, autologous blood collection may be continued with a second ATS bag, repeating steps 2 through 5 under Collection.

Figure 28-4 Attaching the metal hanger to the Pleur-Evac system, which will hold the blood collection bag. *(Courtesy of Deknatel, Inc., Fall River, MA 02720. Reproduced by permission.)*

Figure 28-5 The blood collection bag that is attached to the standard Pleur-Evac system. *(Courtesy of Deknatel, Inc., Fall River, MA 02720. Reproduced by permission.)*

Figure 28-6 Preparation for reinfusion. After air is vented from the collection bag to minimize the chance of air embolism, insert the free recipient arm *(L)* of the prepared Y-type infusion line into the stem of the collection bag. *(Courtesy of Abbott Laboratories, Abbott Park, IL 60064. Reproduced by permission.)*

OTHER THORACOSTOMY SET AUTOTRANSFUSION SYSTEMS

In addition to the units described, the companies Genzyme Biosurgery, Atrium, and Kendall make other chest tube

Figure 28-7 Reinfusion of collected blood with the Pleur-Evac system. A second collection may be obtained while this blood is infusing. *(Courtesy of Deknatel, Inc., Fall River, MA 02720. Reproduced by permission.)*

drainage systems with autotransfusion capabilities. These systems offer numerous features. Genzyme Biosurgery offers several products that are very similar to the Pleur-Evac. The Thora-Klex, for example, uses a patented one-way valve in the chest tube drainage system. This allows it to maintain function even if the unit is tipped over. The Sahara, also available through Genzyme, is 100% latex-free. The procedures

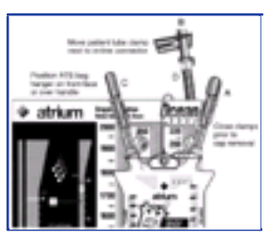

Figure 28-8 Atrium chest tube and autotransfusion system (ATS) prior to connection. *A*, Female ATS connector. *B*, Patient chest tube connector. *C*, Male ATS connector. *D*, Chest tube drain connector. *(Courtesy of Atrium Medical Corporation, Hudson, NH 03051.)*

outlined earlier are very similar for most units with in-line chest tube drainage systems.

Analysis of the minor differences among these systems is beyond the scope of this chapter. Therefore, users should obtain specific information regarding a particular system directly from the manufacturer.

Continuous Reinfusion Systems

An optional feature called *continuous reinfusion* or *autotransfusion* is available from the major manufacturers of chest tube drainage systems (Genzyme, Atrium, and Kendall). In this chest tube drainage system, a catheter on the bottom of the first collection chamber can be hooked to an IV infusion pump, permitting blood to be pumped directly back into the patient (it essentially uses the first blood collection chamber as the autotransfusion bag). This system is somewhat more hazardous as there is greater potential for air embolism and incorrect anticoagulant dosing. The procedure to initiate continuous reinfusion is as follows (Fig. 28-12):

1. Set up the chest tube drainage system and initiate blood collection from the chest tube to chest drain as recommended.
2. Inject appropriate anticoagulant into the front "anticoagulant injection site" after swabbing the area with alcohol. The appropriate dose can be estimated based on the volume of blood in the chamber at the initiation of continuous reinfusion.
3. Drape the end of the ATS access line (*B*) (Fig. 28-13) up over the top of the drain hanger or patient chest tube so that the end of the ATS access line is hanging down (Fig. 28-14).
4. Insert a microemboli blood filter (*C*) into the end of the ATS access line (*B*) (see Fig. 28-13).
5. Attach a non-vented IV set to the filter so that the IV tubing is in a "spike up" position (*D*) (see Fig. 28-13).
6. Insert IV tubing into a "self-priming" IV pump (*E*) and set the infusion pump to priming mode.

7. Open the ATS access line (*A*) by releasing the clamp (*F*) (see Fig. 28-12) and commence priming until ALL air in the ATS access line, filter, and IV tubing is fully purged. (Note, if a non "self-priming" infusion pump is used, the system MUST be manually primed prior to initiation of the infusion to avoid massive air embolism).
8. Attach the primed administration set to the patient's IV line (*G*) (see Fig. 28-14).
9. To prevent air from entering the system, keep at least 75 mL of blood in the blood collection compartment of the chest drain at all times. If this is not done, air embolism is a significant risk (see Nonhematologic Complications later in this chapter). One way to reduce the risk of air embolism is to set the IV pump dose (or volume to be infused) equal to the amount currently in the chest tube drainage compartment minus 75 mL. Using this technique, the IV pump will automatically stop when that volume has been infused and will ensure that, in the event no more blood is collected in the chest tube, the system will remain primed and the risk of air embolism will be reduced.
10. If continuous reinfusion is used, maintenance of proper proportions of anticoagulant to collected blood may become problematic.

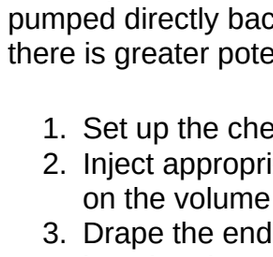

Figure 28-9 Separation of the patient chest tube and chest tube drainage system by depression of connector lock and pulling to separate. *(Courtesy of Atrium Medical Corporation, Hudson, NH 03051.)*

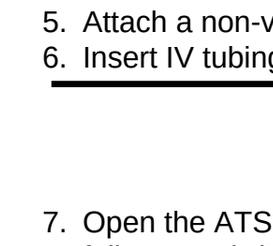

Figure 28-10 Atrium chest tube and autotransfusion system (ATS) during blood collection. *A*, Female ATS connector. *B*, Patient chest tube connector. *C*, Male ATS connector. *D*, Chest tube drain connector. *(Courtesy of Atrium Medical Corporation, Hudson, NH 03051.)*

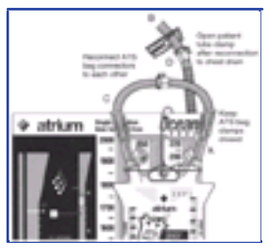

Figure 28-11 Atrium chest tube and autotransfusion system after blood collection. *A*, Female ATS connector. *B*, Patient chest tube connector. *C*, Male ATS connector. *D*, Chest tube drain connector. *(Courtesy of Atrium Medical Corporation, Hudson, NH 03051.)*

Abbott Receptal Disposable Suction Liner System

The Abbott Receptal ATS consists of a closed, rigid, nonsterile plastic canister into which a gas-autoclaved plastic bag is placed for blood collection. The canister can be mounted on an IV pole and connected to a vacuum regulator valve for control of negative suction pressure. The collection bag is placed in line with disposable collection tubing that has a separate inlet valve for admixture of anticoagulant and aspirated blood. This inlet is connected via sterile tubing to a bottle of CPD (Fig. 28-15). Unlike systems described previously, the Abbott unit does not include an in-line thoracostomy drainage device.

Collection

1. To collect autologous blood from a hemothorax, first open the included "Trauma Drainage Tubing Set" containing one 36 Fr chest tube, latex drainage tubing (see Fig. 28-15*B* and *C*), and a male-to-male connector (*D*). While tube thoracostomy is being performed in the usual manner, the burette set (*A*) is connected to the CPD bottle, and the burette is filled with 150 mL of CPD.
2. Connect the yellow-tipped (*E*) end of the latex drainage tubing (the end with the side port) to the inlet port (*F*) of

the red liner cap attached to the collection canister (see Fig. 28-15 and Fig. 28-16). Then remove the protective cap from the side port and connect the anticoagulant (CPD) administration line (Fig. 28-17). Prime the liner with 50 mL of CPD from the burette.
3. Connect the downstream suction hose (see Fig. 28-15*H*) to wall suction and turn wall suction to maximum. Be sure that the regulator on the autotransfusion stand does not exceed the preset 60 mm Hg during collection (100 mm Hg in special situations such as post-thoracotomy); otherwise, excessive hemolysis of RBCs may result.
4. When the chest tube is in place, connect the latex drainage tubing and begin collection. During collection, stay ahead of the accumulating blood volume with the CPD in 50-mL increments. Always keep the ratio no less than 1 part CPD to 10 parts blood (1:7 ratio of CPD to blood is recommended by the manufacturer); otherwise, the collected blood may clot, especially with massive ongoing hemorrhage. Do not overfill the liner bag; it will overflow, spilling blood into the regulator valve.

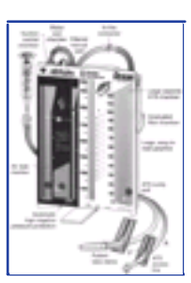

Figure 28-12 Atrium continuous autotransfusion system. *A*, Autotransfusion system (ATS) access line. *F*, ATS access line clamp. *(Courtesy of Atrium Medical Corporation, Hudson, NH 03051.)*

Reinfusion

1. Prepare a standard Y-type blood infusion line with a high-capacity 40-µm in-line filter, prime the line and filter with normal saline, and connect it to a large-bore IV access (14-gauge or larger) (see Fig. 28-6 and Fig. 28-18).
2. When the liner bag is full, temporarily clamp the chest tube, discontinue suction, and remove the yellow end of the latex drainage tubing (*E*) from the red liner lid. The liner lid tubing connector (*J*) is now removed from the canister tee (*K*) (Fig. 28-19) and connected to the inlet port (*F*) of the liner cap, thus sealing the top of the collection lid (Fig. 28-20).
3. Remove the liner assembly from the canister by pushing upward on the thumb tab (see Fig. 28-20). Lift out the

liner bag, invert the bag, and unscrew the protective cap (*N*) over the bottom stem of the liner. Now insert the free recipient arm (see Fig. 28-6*L*) of the Y-tube infusion line into the stem of the collection bag, and hang the liner bag on the IV stand by the attached tab (*M*) (see Fig. 28-18). Before infusion, briefly disconnect the liner lid tubing connector (see Fig. 28-18*J*), vent all air from the bag (to eliminate the possibility of air embolism), and then reconnect it to the inlet port.
4. Gravity flow, manual squeezing of the liner bag, or an in-line roller pump may be used to hasten reinfusion. Although some reports [22] [35] mention the use of encircling pneumatic blood pumps during reinfusion, Abbott cautions that such devices may damage the pump or rupture the liner bag.
5. During reinfusion, autologous blood collection may be continued with a second liner bag. Be sure that the new liner bag is fully extended before placing it into the canister (Fig. 28-21). If the bag is crumpled at the top of the canister, blood may be sucked directly into the regulator valve. Insert the new liner into the canister, and snap the lid in place with the thumb tab directly over the canister tee (Fig. 28-22). The unit is now ready for collection assembly as previously outlined.

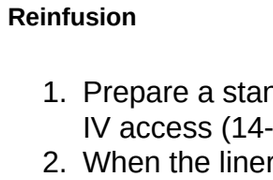

Figure 28-13 Infusion pump set up for continuous autotransfusion. *A*, Autotransfusion system (ATS) access line. *B*, End of ATS access line. *C*, Blood filter. *D*, IV tubing with "spike up" position. *E*, Self-priming IV pump. *G*, line to patient IV access. *(Courtesy of Atrium Medical Corporation, Hudson, NH 03051.)*

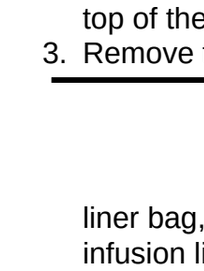

Figure 28-14 Autotransfusion connection to patient. *A*, Autotransfusion system (ATS) access line. *B*, End of ATS access line. *C*, Blood filter. *D*, IV tubing with "spike up" position. *E*, Self-priming IV pump. *G*, Patient IV access. *(Courtesy of Atrium Medical Corporation, Hudson, NH 03051.)*

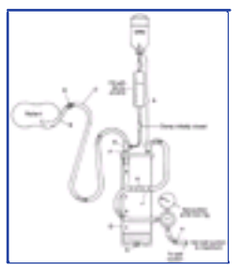

Figure 28-15 Abbott Receptal disposable collection apparatus. *A,* Anticoagulant volume control burette (fill with 50 mL of citrate phosphate dextrose [CPD] anticoagulant). *B,* Chest tube. *C,* Latex drainage tubing. *D,* Male-to-male connector. *E,* End of drainage tubing with side port. *F,* Inlet port of red liner cap attached to collection canister. *G,* Collection liner bag. *H,* Downstream suction hose (do not exceed 60 mm Hg of suction). *J,* Liner lid tubing connector. *K,* Canister tee. *N,* Liner stem with protective cap. *(Modified from Young GP, Purcell TB: Emergency autotransfusion. Ann Emerg Med 12:180, 1983. Reproduced by permission.)*

Boehringer Autovac Intraoperative System

This system is designed to be used with a suction catheter. A unique advantage of the Boehringer Autovac IO is the optional integral vacuum regulator built into the cap. This feature allows direct attachment to a wall vacuum without an external regulator. It automatically limits applied suction to a maximum of 100 mm Hg, thereby minimizing RBC hemolysis. The number of connections on the collection line has been reduced to one, and the male connector has a skirt shield, reducing the chances of inadvertent contamination through contact with nonsterile surfaces. The collection bag is prepackaged within a disposable rigid plastic canister and will hold up to 1000 mL of blood. Inside the collection chamber is a shutoff system consisting of a hydrophobic filter that, upon contact with blood or any liquid, quickly occludes, preventing overflow into any downstream component (e.g., wall suction regulators).

Collection

1. The vacuum line is attached to the connector on the canister (Fig. 28-23). Models not equipped with the optional integral regulator must be attached to a wall regulator set at no greater than 100 mm Hg vacuum.

2. Anticoagulant is injected through a port on top of the canister. Boehringer Laboratories recommends (and offers for sale) anticoagulant citrate dextrose solution A (ACD-A), which contains 0.73 g of citric acid, 2.2 g of sodium citrate, and 2.45 g of dextrose per 100 mL. Initially 40 mL is injected into the bag, and as collection proceeds, ACD-A is added to maintain a proportion of 1:5 to 1:10 ACD-A to blood.
3. During collection, the canister is periodically agitated to facilitate mixing of blood and anticoagulant.
4. When full, blood will come into contact with the hydrophobic filter at the top of the canister, automatically shutting off the system.

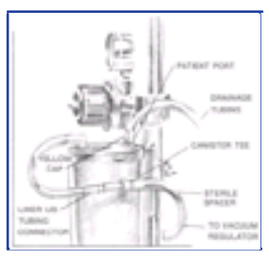

Figure 28-16 Abbott Receptal collection apparatus. Detail of canister connections before attachment of anticoagulant line. *(Courtesy of Abbott Laboratories, Abbott Park, IL 60064. Reproduced by permission.)*

Reinfusion

1. Engage both slide clamps at the top of the canister. Both collection and vacuum lines are now occluded, creating a closed system.
2. Disconnect the canister from suction and collection lines. If further collection is desired, another canister can now be attached to these lines.
3. The flexible blood bag is removed from the rigid canister by first removing the white safety tape and then popping the lid from the lower portion of the canister.
4. Keeping the bag upright, temporarily loosen the slide clamp over the vacuum connector and gently squeeze the bag to expel any residual air through the filter shutoff mechanism. Then re-engage the slide clamp.
5. The bag may now be attached to a blood administration set in the usual fashion (Fig. 28-24). The manufacturer recommends using an in-line microaggregate filter (20 to 40 µm).
6. An external pneumatic cuff around the bag may be used to increase flow rates, but cuff inflation pressures should not exceed 300 mm Hg.

HARVEST BLOODLESS SURGERY STATION

A newer device on the market for direct suctioning of blood (through a suction catheter and not a chest tube) in anticipation of autotransfusion is the Harvest Bloodless Surgery Station. While this unit also provides additional capabilities not generally useful for autotransfusion in the ED setting (like RBC washing), some features do make it potentially attractive for ED use. It requires only an electrical outlet to function as it has a built-in suction system. It is a freestanding unit that comes attached to a specifically designed stand, so it is freely mobile to different locations. It also has a specially designed suction catheter that allows rapid suction of up to 3.75 L per minute (Fig. 28-25).

Additional General Autotransfusion Information

1. Use each liner bag only once.
2. After reinfusing a total of 3500 mL, or 7 units, of autologous blood, it has been suggested that 1 unit of fresh frozen plasma be given for every 2 units (1000 mL) of autotransfused blood.[41]
3. To minimize risk from bacterial overgrowth, collected blood must not be allowed to stand for prolonged periods of time before reinfusion. Some authors [46] [52] [58] advise a limit of no more than 4 hours between collection and reinfusion. The age of collected blood should be calculated from the time of injury, and reinfusion of blood older than 4 to 8 hours should be considered hazardous. Because one is performing the procedure for significant hypovolemia in the ED, the collected blood should generally be transfused as soon as the collection bag is full.
4. If some or all of the collected blood becomes clotted in the liner bag, the blood should be discarded.
5. The blood filter used during reinfusion is changed as needed (usually after each 1000- to 2000-mL transfusion). In practice, the need for a new filter will become apparent as perfusion pressures increase and flow rates visibly slow down.

COMPLICATIONS

Complications from autotransfusion generally are clinically insignificant if the proper technique is followed and if less than 3000 mL of blood is reinfused. They may be categorized as hematologic and nonhematologic (Table 28-1).

Hematologic Complications

The most frequent hematologic consequence of autotransfusion is thrombocytopenia. Samples taken from collected autologous blood generally show very low platelet counts although not as low as found in banked blood. [56] Until patients receive greater than 3500 mL of autologous blood, in vivo platelet counts do not drop below 60 × 10^9 /L.[26] [35] [43] Above this level, trauma surgery can be satisfactorily performed. [58] Platelets collected from autotransfusion reservoirs function abnormally when tested in vitro by aggregation or serotonin uptake and release, but postinfusion samples drawn from

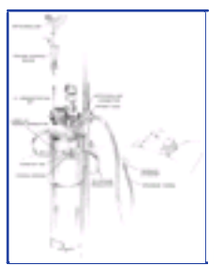

Figure 28-17 Proper attachment of anticoagulant line for Abbott Receptal device. The preferred anticoagulant is citrate phosphate dextrose (CPD) in a 1:7 volume ratio of anticoagulant to blood. *(Courtesy of Abbott Laboratories, Abbott Park, IL 60064. Reproduced by permission.)*

patients aggregate normally.[41] A canine study demonstrated significant in vivo platelet dysfunction when autotransfused volumes exceeded an amount equivalent to 1 total blood volume.[19] Thus, platelet counts should be followed and significant thrombocytopenia treated with platelet infusion.

The most common coagulation factor abnormality after autotransfusion is hypofibrinogenemia, especially when the volume of autologous blood used exceeds 4000 mL.[15] [51] [56] Because of the liver's capacity to replenish fibrinogen rapidly, the low postautotransfusion levels have not proved to be clinically significant. [25] [27] [52] Yet some investigators[25] believe that hepatic insufficiency is a relative contraindication to autotransfusion unless fibrinogen is supplemented.

Symbas and colleagues[15] [35] [59] extended their work with laboratory dogs to the clinical study of victims of traumatic hemothorax. They found no clinical evidence of coagulopathy following autotransfusion as long as the volume collected and reinfused remained equal to or less than one half the patient's

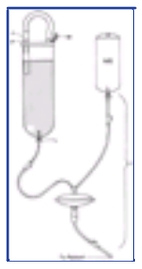

Figure 28-18 Reinfusion apparatus. Inlet port of liner cap (*F*), Y-type blood infusion line with in-line 40-µm filter (*I*), liner lid tubing connector (*J*), liner bag connection with infusion line (*L*), attached hanger tab at top of liner bag (*M*), and normal saline IV fluid for priming and maintaining patency of the system during liner bag changes (*N/S*). *(Modified from Young GP, Purcell TB: Emergency autotransfusion. Ann Emerg Med 12:180, 1983. Reproduced by permission.)*

total blood volume. In those few patients who required a larger autotransfusion volume, a proportional decrease in platelets and fibrinogen occurred, requiring subsequent correction with fresh frozen plasma and platelets. Other investigators [20] [23] have confirmed these findings and have shown that both platelet and fibrinogen levels return to normal by 48 to 72 hours, without replacement therapy. Similarly, elevations in prothrombin and partial thromboplastin times, which were encountered routinely, were not clinically significant. These coagulation abnormalities also self-corrected within 48 to 72 hours. [23] [25] [43] On the other hand, Schonberger and colleagues found that intraoperative autotransfusion of greater than 800 mL shed blood may provoke a significant derangement of hemostasis. [60]

Hemolysis occurs with autotransfusion in part because of prolonged exposure of the cells to serosal linings of the traumatized body cavities. [61] Hemolysis also results from

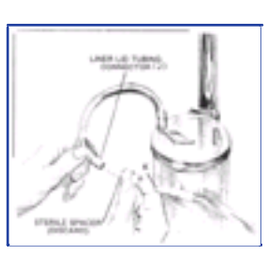

Figure 28-19 Preparation for reinfusion. Removal of liner lid tubing connector (*J*) from canister tee (*K*). Inlet port (*F*) is shown. *(Courtesy of Abbott Laboratories, Abbott Park, IL 60064. Reproduced by permission.)*

mechanical factors during collection and reinfusion, such as high vacuum pressures during aspiration, roller pump trauma, or excessive exposure to air-fluid interfaces. [25] An elevated plasma-free hemoglobin is a consistent finding in patients who have received autotransfusions. [25] [35] [51]

Most samples of blood taken from the autotransfusion reservoir have free hemoglobin levels of less than 1.0 g/L, [25] [35]

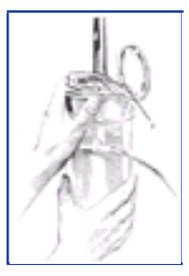

Figure 28-20 Preparation for reinfusion. Removal of liner assembly from the canister after connecting the liner lid tubing connector (*J*) to the inlet port of the liner cap. *(Courtesy of Abbott Laboratories, Abbott Park, IL 60064. Reproduced by permission.)*

Figure 28-21 Straightening the liner bag before insertion into the canister. *(Courtesy of Abbott Laboratories, Abbott Park, IL 60064. Reproduced by permission.)*

but some levels have been reported to be as high as 13.9 g/L). [51] Plasma-free hemoglobin values immediately after autotransfusion range from 0.1 to 1.0 g/L. [25] [35] [51] When the binding capacity of haptoglobin is saturated and the threshold of tubular resorption of hemoglobin is exceeded, hemoglobinuria is seen. This threshold corresponds to a plasma-free hemoglobin concentration of 1.0 g/L. [62]

In the past it was believed that elevated levels of free hemoglobin following hemolytic transfusion reactions caused renal failure by precipitation of hemoglobin in and obstruction of renal tubules. However, more recent evidence suggests that the mechanism of renal failure in this setting is independent of free hemoglobin. Rather, it is a result of an antigen-antibody-induced intravascular coagulation that, compounded by vaso-constriction and hypotension, leads to renal ischemia. [63] Indeed, it has been shown that "massive hemoglobinuria may follow the transfusion of immunologically compatible hemolyzed RBCs with minimal symptoms and a benign outcome" and that isolated free hemoglobin levels of up to 130 g/L may be tolerated without renal compromise. [64] Even though renal failure as a direct consequence of autotransfusion has not been reported, [34] transient elevations in serum creatinine do occur, [21] and in the presence of shock and systemic acidosis, acute tubular necrosis remains a potential complication. [62] Some researchers [25] believe that renal insufficiency is only a relative contraindication to autotransfusion. The clinician must judge the urgency of the need for blood and the availability of an alternate source.

Finally, the hematocrit falls in direct proportion to the quantity of blood transfused, averaging a decline of 10% to 20%. [25] [35] [32] However, nontraumatized RBC survival has been reported to be normal in all cases studied. [15] [36] [41]

In general, although coagulation problems should be anticipated, they have not proven to be clinically important when volumes of autotransfused blood remain below 2000 mL in adult patients. [52] When reinfused volumes exceed 3500 mL, [41]

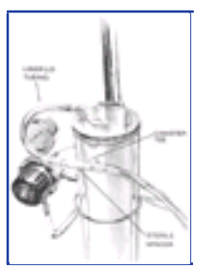

Figure 28-22 Proper placement of the liner bag lid on the canister. *(Courtesy of Abbott Laboratories, Abbott Park, IL 60064. Reproduced by permission.)*

laboratory evidence of a dilutional coagulopathy may become evident. When volumes of autotransfused blood are greater than the patient's total blood volume, animal studies suggest that there is increasing risk of a true consumptive coagulopathy. [19]

Recommendations regarding the volume of autologous blood that should trigger infusion of fresh frozen plasma or platelets range from 25% of total blood volume (about 1250 mL in a 70-kg adult) [55] to 3500 mL. [41] Others advise reliance on laboratory tests and clinical findings rather than a volume-based protocol. [52] Prudent clinical judgment dictates application of the more liberal guidelines for replacement therapy in those patients with extensive hepatic injury, intractable shock, or ongoing losses requiring immediate surgical intervention.

Nonhematologic Complications

The theoretic risk of sepsis after the administration of potentially contaminated blood always exists within the nonsterile surroundings of the typical ED resuscitation area. Experience has shown this risk to be minimal after competent autotransfusion from an isolated hemothorax, [14] [20] [34] and there is no evidence to suggest that routine prophylaxis with systemic antibiotics is beneficial in this situation.

The issue of whether to autotransfuse shed intraperitoneal blood is somewhat more complex. Recovery and reinfusion of hemoperitoneum without evidence of bowel perforation has proved to be relatively safe. Klebanoff and associates have concluded that "for contaminant-free conditions at least, as in ruptured ectopic pregnancies, ruptured

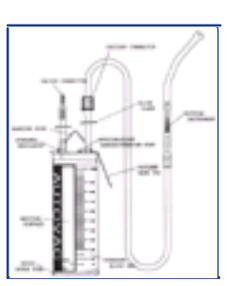

Figure 28-23 Boehringer Autovac System, collection configuration. *(Courtesy of Boehringer Laboratories, Inc., Norristown, PA. Reproduced by permission.)*

spleen and liver, traumatic hemothorax, and vascular surgery, autotransfusion can be performed readily to reduce the need for homologous blood replacement." [14]

Reinfusion of autologous blood with possible enteric contamination is still considered by most investigators to be ill-advised in all but the most desperate of circumstances, such as in the patient who will exsanguinate before homologous blood can be made available. If contaminated blood is infused, systemic antibiotics should be given. [33] [52] [65] Several small series describing collection and reinfusion of intestinally contaminated hemoperitoneum have been reported. [31] [65] [66] [67] [68] The overall mortality rate among this select group approximates 20%, not unexpectedly high considering the severity of associated injuries. Experimental studies with dogs also show that autotransfusion of hemoperitoneum contaminated by intestinal contents, urine, or bile is tolerated. [14] [32]

Another complication, microemboli secondary to platelet microaggregation or fat emboli, has largely been eliminated by the use of micropore filters. [25] [41] [43] In most instances, during reinfusion of collected blood, there is a mild increase in screen filtration pressures, indicating the formation of microemboli trapped by the filter. [43] There has been no clinical evidence of pulmonary insufficiency or unexplained elevation of the alveolar-to-arterial oxygen gradient that might be attributed to the passage of microemboli beyond the micropore filter systems. [25]

Air embolism has been reported sporadically as a complication of autotransfusion. [20] [66] [69] [70] This uncommon but often fatal complication has been associated, in all cases

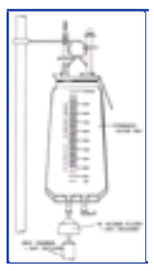

Figure 28-24 Boehringer reinfusion apparatus. *(Courtesy of Boehringer Laboratories, Inc., Norristown, PA. Reproduced by permission.)*

reviewed, with autotransfusion systems using automated roller pump units in which the aspirate reservoir was inadvertently allowed to run dry. Air embolism with gravity or with a manually assisted technique is rare.

Available data indicate that although autotransfusion is not free of complications, the risk-to-benefit ratio weighs heavily in its favor in the resuscitation of select trauma victims. Klebanoff and coworkers reviewed the evidence as of 1970 and determined that "in over 1000 documented cases of autotransfusion in the Western literature, not a single death or major complication was attributed directly to the transfusion." Klebanoff [14] and Symbas[35] reported autotransfusing more than 400 patients with traumatic hemothorax without any significant morbidity related to the procedure. Mattox and associates [20] reported autotransfusing 69 patients an average of 3.9 units, with only 1 death (from air embolism) directly attributable to the procedure.

CONCLUSION

Autotransfusion, a technique almost 200 years old, has become a subject of renewed interest in the emergency setting. The previously feared complications of hematologic or metabolic embarrassment and sepsis have not proved to be of clinical significance when appropriate patient selection and

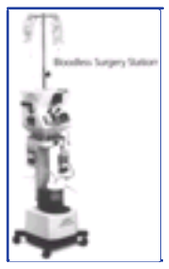

Figure 28-25 The bloodless surgery station. *(Courtesy of Harvest Blood Conservation, Inc., Plymouth MA 02360.)*

careful technique are followed. In addition, the use of autologous blood has several advantages over the transfusion of stored homologous blood in the emergency patient, including ready availability of compatible blood, homeostasis of core temperature, higher levels of 2,3-DPG in RBCs, and cost-effectiveness. Autotransfusion has been endorsed by the Council on Scientific Affairs of the American Medical

TABLE 28-1 -- Potential Complications of Autotransfusion

Hematologic
Decreased platelet count
Decreased fibrinogen level
Increased fibrin split products
Prolonged prothrombin time
Prolonged partial thromboplastin time
Red blood cell hemolysis
Elevated plasma-free hemoglobin level
Decreased hematocrit level
Nonhematologic
Bacteremia
Sepsis
Microembolism
Air embolism

Association as a procedure that has been found to be "effective, safe, and cost-effective for many trauma and surgical patients" when used by experienced health care providers. [17] Although the technique is not totally free of complications, the benefits to be gained from autotransfusing the selected trauma patient outweigh the relatively limited risks.

While no generally accepted standard of care has been promulgated mandating the routine use of autotransfusion in the ED setting, many hospitals, especially trauma centers, have instituted the technique with success. Current equipment allows autotransfusion from the chest cavity to be performed relatively easily, and without highly technical devices. However, low-volume hospitals, or those with a low trauma census may find this technique impractical or cumbersome.

Acknowledgment

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Chapter 29 - Transfusion Therapy: Blood and Blood Products

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Transfusion of blood components (red cells, white cells, platelets, whole plasma, or plasma fractions) is an everyday occurrence in the practice of emergency medicine. Technical advances have made component therapy directed at specific acute and chronic pathologic conditions practical and affordable. Thus the emergency clinician must possess knowledge of the appropriate indications for transfusion therapy, the associated complications, and the techniques of administration.

BACKGROUND

Red Cell Antigens and Antibodies

In the early 1900s the Austrian Karl Landsteiner found that an individual's serum reacted with the red cells of some but not all other individuals, thereby discovering the red cell antigen-antibody system. Red blood cell (RBC) membranes contain a series of glycoprotein moieties, or antigens, which give the cell an individual identity. Two different genetically determined antigens, type A and type B, occur on the cell surface. Any individual may have one, both, or neither of these antigens. Because the type A and type B antigens on the cell surface make the RBC susceptible to agglutination, these antigens are termed *agglutinogens*. The presence or absence of agglutinogens is the basis for the ABO blood group classification and the blood types are named accordingly as A, B, or AB. Blood type O contains neither the A nor the B agglutinogen. The relative frequencies of the different blood groups are listed in Table 29-1 .

Genes on adjacent chromosomes determine the presence or absence of agglutinogens and hence the individual's specific ABO blood group. These allelomorphic genes can be only one of the three different types—that is, A, B, or O—allowing for six possible combinations of genes (OO, OA, OB, AA, BB, and AB). There is no dominance among the three different allelomorphs; however, type O is basically functionless in that it causes such weak agglutination that it is

TABLE 29-1 -- Frequency of Blood Groups in the General Population

Blood Groups	Frequency (%)
Type	
O	47
A	41
B	9
AB	3
Rh factor	
Rh^-	15
Rh^+	85

From Guyton AC: Textbook of Medical Physiology, 6th ed. Philadelphia, WB Saunders, 1981.

normally insignificant. The different combinations of genes signify the individual's genotype, and each person is one of six different genotypes. The resultant blood groups for the various genotypes are listed in Table 29-2 .

Within the first year of life, antibodies begin to form against the standard red cell agglutinogens not present in the individual patient. These *agglutinins* are ?-globulins of the IgM and IgG types and are probably produced by exposure to agglutinogens in food, bacteria, or exogenous substances other than blood transfusions. Agglutinins reach their peak titer between 8 and 10 years of age and then gradually decline throughout the remaining years of life. In the absence of type A agglutinogens (blood types B and O), anti-A antibodies, or *agglutinins,* spontaneously develop in the plasma. Similarly, in the absence of type B agglutinogens (blood types A and O), anti-B antibodies develop. When both A and B agglutinogens are present (blood type AB), no agglutinins are formed.

The reaction between red cell antigens and the corresponding agglutinins results in red cell destruction when noncompatible blood types are mixed. As many as 300 different red cell antigens have been identified, but clinically the A and B antigens are most important given their potential to cause transfusion reactions. With the first transfusion of ABO-*incompatible* blood, severe, potentially fatal agglutination can occur. The Rh system is likewise very important because there is a high likelihood (30% to 50%) that a transfusion of Rh-positive blood to an Rh-negative patient will result in formation of Rh antibodies. These antibodies are capable of causing severe hemolysis following a second exposure to the Rh antigen. Of the 40 antigens in the Rh system, D is the most antigenic, but others can also stimulate the production of antibodies in recipients lacking the antigen (e.g., E), thus complicating future transfusions. Other antigen systems in which antibodies could potentially cause hemolytic reactions are the Kell (K and k alleles), Duffy (Fy^a and Fy^b), Kidd (Jk^a and Jk^b), and MNS (M and N; closely linked S and s) systems. Other antigen systems are rarely of clinical importance in transfusion therapy.

Crossmatching

Compatibility testing, or *crossmatching*, involves mixing the donor's RBCs and serum with the serum and RBCs of the recipient to identify the potential for a transfusion reaction. "Major" and "minor" crossmatch procedures are outlined in Table 29-3 . The end point of all crossmatches is the presence of RBC agglutination (either gross or microscopic) or hemolysis. Testing is performed immediately after mixing, after incubation at 37°C for varying times, and with and without an antiglobulin reagent to identify surface immunoglobulin or complement. Each unit of blood product, when properly crossmatched, can be administered with the expectation of safety.

TABLE 29-2 -- The Blood Groups with Their Genotypes and Constituent Agglutinogens and Agglutinins

Genotypes	Blood Groups	Agglutinogens	Agglutinins
OO	O	—	Anti-A and anti-B
OA or AA	A	A	Anti-B
OB or BB	B	B	Anti-A
AB	AB	A and B	—

From Guyton AC: Textbook of Medical Physiology, 6th ed. Philadelphia, WB Saunders, 1981.

TABLE 29-3 -- Crossmatch Procedures

	Major Crossmatch	Minor Crossmatch
Donor	Red cells	Serum
Recipient	Serum	Red cells
End point	Agglutination or hemolysis at 37°C	

Transfusion Reactions

While the most common complication of transfusion is a minor allergic reaction, the immediate life-threatening complications of transfusion are usually related to transfusion reactions. Based on 1995 data, the risks associated with transfusion are outlined in Table 29-4 . [1]

Most serious transfusion reactions occur because of patient-blood product mis-assignment related to clerical error.

When incompatible blood is given, the result may range widely from no effect to death. If the recipient does *not* have antibodies (naturally occurring or acquired) directed against the foreign RBC antigen received, there will be no immediate reaction, but antibodies to the infused blood may develop within weeks, limiting the safety of subsequent transfusions from the same donor or same antigenic type. If the recipient's serum *has* preformed antibodies directed against the donor RBCs (incompatibility in the *major* crossmatch), the recipient will begin to hemolyze the donor cells within seconds or minutes.

In most cases of major crossmatch reactions, RBCs of the *donor* blood are agglutinated and hemolyzed. It is rare for transfused blood to produce agglutination of the recipient's cells, because the plasma portion of the donor blood becomes diluted by the plasma of the recipient. This reduces the titer of the infused agglutinins to a level too low to cause significant agglutination. Because the recipient's plasma is not diluted to any significant degree, the recipient's agglutinins can react with donor cells.

The end result of antigen-antibody incompatibility is red cell hemolysis. Occasionally, this is immediate, but more often, the cells first agglutinate. They are then trapped in small vessels and become phagocytized over hours to days, releasing hemoglobin into the circulatory system.

Clinical manifestations of acute hemolysis are chills, fever, tachycardia, abdominal pain, back pain, hypotension, fainting, and an anxious "feeling of impending doom." From

TABLE 29-4 -- Estimated Risks of Transfusion per Unit in the United States (1995)

Minor allergic reactions	1:100
Bacterial infection (platelets)	1:2,500
Viral hepatitis	1:5,000
Hemolytic transfusion reaction	1:6,000
HTLV I/II infection	1:200,000
HIV infection	1:420,000
Acute lung injury	1:500,000
Anaphylactic shock	1:500,000
Fatal hemolytic reaction	1:600,000
Graft-vs-host disease	Rare
Immunosuppression	Unknown

HIV, human immunodeficiency virus; HTLV, human T-cell leukemia-lymphoma virus.

From Greenburg AG: Am J Surg 173:49, 1997.

the liberation of intracellular material associated with hemolysis, vasoactive substances may cause hypotension and shock; other substances may precipitate disseminated intravascular coagulation and high-output cardiac failure. Alternatively or additionally, anoxic acute renal failure may result. Hemolytic transfusion reactions are estimated to occur once per every 6000 blood units transfused, with a fatality rate of 1 per every 100,000 units transfused.

An incompatibility in the *minor* crossmatch usually causes no serious reaction, although the recipient's red cells can be hemolyzed if the titer of the antibody is sufficiently large. Even when major and minor crossmatch compatibility indicates the safety of a transfusion, a delayed hemolytic transfusion reaction can occur days to weeks later. Usually seen in multiply transfused patients or in multigravida women, these reactions may be unavoidable without complete RBC antigen typing, a procedure occasionally indicated for recipients of repeated transfusions. Fortunately, 90% of transfusions are now given as packed RBCs that contain a small volume of plasma, minimizing the chance for a transfusion reaction due to donor sensitization.

Additional antibodies *not* caused by sensitization from transfused RBCs include autoantibodies (both cold- and warm-reacting) and various agglutinins. Autoantibodies can be "cold," reacting with red cells *more strongly at 4°C* than at 37°C. These antibodies are common and usually harmless; however, they may be associated with disease states in higher titers (e.g., anti-I in *Mycoplasma* infections). If active at higher temperatures (up to 28 to 32°C), pathologic cold antibodies may cause hemolysis or sufficient RBC agglutination to obstruct blood flow through the small vessels of the hands and feet on exposure to cold. These would also be present and identifiable at 37°C. The primary significance of cold antibodies stems from their ability to complicate crossmatching procedures in the blood bank.

Warm antibodies, reacting *more strongly at 37°C* than at lower temperatures, may be harmless or have the potential to cause hemolytic anemia of variable severity. Characteristics of the IgG antibody itself determine its significance to the patient. Usually harmless warm autoantibodies that can occasionally cause hemolysis are seen in patients taking a-methyldopa. Harmful warm autoantibodies are encountered in approximately 80% of patients with autoimmune hemolytic anemia.

Additional problems in pretransfusion testing may occur with antibodies directed against substances that can attach themselves to the RBC surfaces and cause agglutination of "innocent bystanders." Examples are the fatty acid-dependent agglutinins; penicillin and cephalosporin antibodies; bacterial polysaccharides; and nonspecific agglutination associated with a high erythrocyte sedimentation rate, caused by high levels of the acute-phase reactants fibrinogen, a $_2$ -macroglobulin, and IgM. A delay in pretransfusion testing may occur when the blood bank has to identify various proteins on RBC surfaces to ascertain their clinical significance.

Miscellaneous Transfusion Problems

Pyogenic transfusion reactions, such as fever and chills, are common and result from the presence in the donor plasma of proteins to which the recipient is allergic. Full-blown anaphylactic reactions occasionally result.

Theoretically, citrate salts, which are the usual anticoagulants in donor blood, may combine with ionized calcium in

the plasma, producing hypocalcemia. In clinical practice, the hemodynamic consequences of citrate-induced hypocalcemia are minimal, although the Q-T interval may be prolonged on the electrocardiogram (ECG) with citrate infusion. Supplemental calcium administration is usually not necessary even during massive blood replacement as long as circulating volume is maintained, because the liver is able to remove citrate from the blood within a few minutes. Alterations in this recommendation may be necessary in the presence of severe liver disease.

Concern has been raised regarding an association between transfusions and diminished organ function or death, or both, in critically ill adult patients. [2] In contrast, Wu and colleagues, in a U.S.-based study of Medicare patients with acute myocardial infarction, found RBC transfusions beneficial in elderly patients when hematocrit values were <33%.[3] A prospective randomized trial of trigger-guided transfusion vs more liberal transfusion in adults showed a trend for greater survival when a transfusion trigger was used, especially among those less acutely ill with a transfusion trigger of 7 g/dL hemoglobin. [4] Hence, blood transfusion may have deleterious end-organ effects in certain patient populations. The use of transfusion triggers is recommended. It is believed that leukocyte-depleted packed RBC

transfusions have a better safety profile in these critical patients. For example, Jensen and colleagues found that postoperative infection in transfused patients can be reduced by using leukocyte-depleted packed RBCs.[5] Clinicians can expect transfusion triggers and recommendations regarding the use of leukocyte-depleted RBCs to evolve as additional studies are performed.

Infectious Complications of Transfusions

Although relatively uncommon, transmission of infectious diseases is the transfusion-related complication most feared by the lay public. Transmission of a wide variety of infectious diseases has been reported, but modern screening methods have sharply reduced the frequency of transmission. Viral illnesses remain the most problematic.

It is estimated that 0.1% of all transfusions may transmit virulent bacteria. However, sepsis is an uncommon occurrence because both the citrate preservative and refrigeration kill most bacteria. Concern over sepsis is responsible for the practice of completing transfusions within 4 hours and returning unused blood products to the blood bank refrigerator for future use only if they have been unrefrigerated for <30 minutes. The virulent pathogens most commonly transmitted are the gram-negative organisms.

Syphilis may theoretically be transmitted by transfusion, but both refrigeration and citrate markedly reduce the survival of *Treponema pallidum*. Therefore, only fresh blood or platelet transfusions are of concern for this specific infectious risk. Currently all U.S. blood is screened for syphilis, and to date the largest recall in the United States was prompted by incorrect testing for syphilis in 138,000 units.[6] The incubation period for syphilis transmitted by transfusion is 4 weeks to 4 months, and the initial clinical manifestation is commonly a rash.

The risk of parasitic infection via transfusion is exceedingly low (<1:1,000,000), although prospective blood product donors who have been to an endemic region within 6 months or treated with malarial prophylaxis within 3 years are not allowed to donate blood products.

Viruses are the organisms most likely to be transmitted by transfusion and the agents with the greatest potential to cause serious disease. Important agents include hepatitis virus, cytomegalovirus, Epstein-Barr virus, human immunodeficiency virus, and West Nile virus.

Most blood products have the potential to transmit hepatitis, with type C being the most common (90% of transfusion-related hepatitis). Historically, the likelihood of developing post-transfusion hepatitis ranges from 1 per 1000 to 1 per 30,000 units of blood administered. Routine testing of blood donors for hepatitis C has occurred since 1991, but initial screening tests were relatively inaccurate. Since April 1999 use of the nucleic amplification technique (NAT) to detect hepatitis C virus ribonucleic acid (HCV RNA) has been mandatory. This test has essentially eliminated false positives, and has a sensitivity of >99%.[7] The incubation period for HCV is 2 to 12 weeks following parenteral infusion.

Both cytomegalovirus and Epstein-Barr virus may cause a mononucleosis-like syndrome 2 to 6 weeks after a transfusion. Blood products with negative serologic findings for both agents should be used for seronegative recipients in high-risk groups, such as pregnant females, premature or low-birth-weight newborns, marrow or organ transplant recipients, and immunosuppressed patients.

The acquired immunodeficiency syndrome (AIDS) epidemic has affected transfusion therapy profoundly. In the United States, 3% of AIDS cases have been linked to blood products. The estimated likelihood of transmitting HIV through transfusion is 1 per 493,000 units of blood donated. One inadvertent consequence of transfusion therapy during the AIDS epidemic has been exposure of the virus to approximately 65% of hemophiliacs through their frequent use of pooled blood products. Fortunately the risk has decreased due to improved screening of donated blood. Using the U.S. blood pool as a standard, a hemophiliac patient requiring a monthly infusion of cryoprecipitate over a lifetime of treatment (estimated 60 years) has a 2% risk of developing HIV based on current screening practices and HIV prevalence in the donor pool.[8] [9] The risk of HIV transmission to hemophiliacs has been further decreased by the availability of biosynthetic coagulation factors developed using recombinant deoxyribonucleic acid (DNA) technology.

Efforts to reduce the risk of HIV transmission to the general population receiving blood products began early in the course of the epidemic and have had considerable success. Voluntary deferment of blood donation by high-risk groups was encouraged beginning in 1983, and formal screening of all blood products began in 1985. All units testing positive for the HIV antibody are routinely discarded, but the potential for HIV transmission has existed during the 6- to 8-week "window" before development of detectable antibody. This problem was addressed through improved screening techniques and the prevalence of HIV in the U.S. blood supply declined throughout the 1990s.[10] In addition to improved screening techniques, efforts are now being made to inactivate viruses in blood products before transfusion.

Directed and Autologous Donations

The system of "*directed donations*," by which friends or family members may give blood to a specific individual, has been proposed to answer the concern over HIV transmission. Some believe that the blood products derived from a relative or a friend have a lower likelihood of testing positive for an HIV infection. At this time, directed donation systems are in place

in some institutions, but the practice has not been widely supported. Limited studies have shown an increased association between direct donor units and infectious disease markers, malaria, and high-risk activities.[11] There is concern that direct donor products may be less safe because social pressures may limit self-deferment of high-risk donors and because clerical errors may increase owing to the increased complexity of this system. Finally, there is concern that the directed donation plan will disrupt the normal anonymous blood donor system, leaving fewer units available for other needy patients.

Although of limited clinical applicability in emergencies, autologous donations are commonplace in elective surgery. It has been suggested that up to 10% of the blood supply could be provided through this mechanism. Most appropriate applications at this time include elective cardiac, gynecologic, orthopedic, and vascular surgical cases. Benefits of this system include avoidance of exogenous bloodborne disease and sensitization. The individual can donate 1 unit of blood weekly, until 3 days before surgery. As blood can be stored up to 35 days, the donations usually begin 5 weeks before needed. The blood donor will require iron supplements and must maintain a hemoglobin >11 g/dL.

COLLECTION AND STORAGE OF BLOOD PRODUCTS

Table 29-5 lists some characteristics of blood and its components. Whole blood is collected from donors into 500-mL plastic bags containing 63 mL of citrate-phosphate-dextrose (CPD) with a resultant hematocrit of 35% to 40%. Immediately after collection, sophisticated techniques permit separation of the whole blood into various components and fractions. Blood components such as fresh frozen plasma

TABLE 29-5 -- Characteristics of Blood and Its Components

Component	Volume	Shelf Life	Requirements for Transfusion
Whole blood*	450 mL blood	21 days at 4°C	Crossmatched
ACD	63 mL anticoagulant and preservative 35–40% hematocrit		
CPD			
CPD-A		35 days at 4°C	
	280 mL 70% hematocrit	Same as for whole blood	Crossmatched
Packed red cells concentrate washed	250 mL 70% hematocrit	1 day at 4°C	Crossmatched
Frozen-thawed red cells†	250 mL 70% hematocrit	? years when frozen, 1 day after thawing	Crossmatched
Platelet concentrate	30 mL 10^{10} platelets	5 days at 22°C	Type-specific if possible, but not essential, not crossmatched
Fresh frozen plasma	200–250 mL	1 year at -18°C, 24 hr after thawing‡	ABO-compatible; random donor, not crossmatched
Cryoprecipitate	10–25 mL per bag 60–120 units of factor VIII	1 year at -18°C, 6 hr after thawing	ABO-compatible; random donor, not crossmatched
Factor IX or prothrombin concentrate	25 mL per vial	Check label	None required
Granulocyte† concentrate	400 mL 10^{10} leukocytes	Transfuse within 24 hr at 22°C	Specific donors for each patient, crossmatched

*ACD, acid-citrate-dextrose; CPD, citrate-phosphate-dextrose; CPD-A, citrate-phosphate-dextrose-adenine.
†Special order—few hospitals have facility inhouse.
‡Use immediately to correct deficiency of coagulation factors.

(FFP), packed RBCs, granulocytes, and platelets are prepared from a single donor, separated, and transfused as single units. Minor blood fractions including albumin, ?-globulin cryoprecipitate, and fibrinogen are often pooled from multiple donors. Within 24 hours, blood is essentially devoid of normally functioning platelets and some clotting factors, especially the labile factors V and VIII. Separation into individual components permits specialized storage and transfusion techniques designed to optimize the survival and availability of each component.

As is true of whole blood, packed RBCs can be stored up to 21 days, although newer preservatives such as ADSOL (adenine, dextrose, saline, mannitol, and water) may allow 49-day storage. Red cell viability decreases approximately 1% per day. The storage of blood contributes to a variety of other derangements or "storage lesions." Cell metabolism continues during storage, causing a mild acidosis. This acidosis is buffered effectively by the bicarbonate derived from metabolism of citrate, assuming normal hepatic function. Even in massive transfusions, acidosis is usually more the result of the disruption of normal physiologic function than the storage of blood products themselves. Levels of 2,3-diphosphoglyceric acid (2,3-DPG) decrease during storage, shifting the oxygen-hemoglobin dissociation curve to the left. This shift is of small clinical significance, because 2,3-DPG levels are usually normal in transfusion recipients within 24 hours of infusion. Potassium commonly leaks from red cells during storage because of a less efficient sodium-potassium adenosine triphosphatase (ATPase)-dependent pump. Most of the potassium is either absorbed by the remaining blood cells, excreted by the kidney, or shifted back into the cells owing to the alkalosis produced by metabolism of the citrate in the preservative. Hyperkalemia is clinically relevant only in newborns and patients with renal impairment.

USE OF BLOOD PRODUCTS

Whole Blood

Once commonly used to provide red cells, coagulation factors, and plasma proteins, whole blood transfusion has been replaced to a great extent by component therapy. Although intuitively an ideal transfusion agent, whole blood is seldom used except for autologous transfusions (e.g., autotransfusion) and for exchange transfusions. Whole blood is *not* the indicated treatment for hypovolemic shock that can be treated effectively with crystalloids (e.g., lactated Ringer's solution, 0.9% sodium chloride), colloids (e.g., plasma protein, albumin), or packed red cells; it is *not* indicated for correction of thrombocytopenia, replacement of coagulation factors, or treatment of anemia.[12] The plasma of whole blood is no more effective than 5% albumin as a volume expander. In addition to providing components or fractions that are potentially unnecessary, whole blood transfusion exposes the patient to additional risk. The incidence of transfusion reactions following transfusion with whole blood is approximately 2.5 times greater than the incidence of reactions following transfusion with packed RBCs. [13] In addition, whole blood contains antigenic leukocytes and serum proteins, which may produce allergic reactions (a risk of 1%). Because of the recognized advantages of component therapy, most blood banks currently do *not* stock significant quantities of whole blood.

Packed Red Blood Cells

Packed RBCs are prepared by centrifugation and removal of most of the plasma from citrated whole blood. One unit of packed RBCs contains the same red cell mass as 1 unit of whole blood at approximately half the volume and twice the hematocrit (70% to 80%). One unit of packed RBCs raises the hematocrit approximately 3% in an adult or increases the hemoglobin level of a 70-kg individual by 1 g/dL. In children, there is an approximate rise in hematocrit of 1% for each mL/kg of packed cells. For example, if 5 mL/kg of packed RBCs is transfused, the hematocrit will rise by approximately 5%. Actual changes depend on the state of hydration and the rate of bleeding.

Because most of the plasma has been removed, packed RBCs cause fewer transfusion and allergic reactions than whole blood. After centrifugation, red cells can be washed to further remove leukocytes, platelets, microaggregates, and plasma proteins. Washing reduces the titer of anti-A and anti-B, permitting safer transfusion of type O packed RBCs in non-O recipients. Washing does not totally eliminate the risk of hepatitis. Frozen deglycerolized RBCs are also relatively free of platelets, plasma, and white blood cells, having been washed after an indefinite period of frozen storage in glycerol. Frozen and fresh RBCs function similarly; frozen RBCs provide normal levels of 2,3-DPG. Washed or frozen preparations should be given to patients who have had febrile (nonhemolytic) reactions to previous transfusions as a result of leukocyte antibodies or IgA sensitization. Blood bank procedures require that these be prepared to order, with routine crossmatching. Considerable delay (6 hours) may occur if the transfusion service does not have the capability to wash RBCs.

Packed RBCs contain less sodium, potassium, ammonia, citrate, and antigenic protein and fewer hydrogen ions than whole blood. This may offer an advantage in patients with reduced cardiovascular, renal, or hepatic function. The rate of urticaria is still relatively high at 1% to 3% of transfusions, but the incidence of adverse reactions to packed cells is approximately one third that noted with whole blood. The benefit of increased hemoglobin must be weighed against the potential for electrolyte and acid-base imbalances following packed RBC administration. Especially in cases of massive transfusion (>10 units), there is a significant risk of metabolic and respiratory acidosis, and hypocalcemia, which can reach life-threatening levels. Although underlying illness or injury plays a major role in cause of death, the overall mortality of patients requiring massive packed RBC transfusions is approximately 70%. [14]

Transfusion of packed RBCs is indicated to provide additional oxygen-carrying capacity and expansion of volume. Packed cells are most commonly used to treat acute hemorrhage and anemia not amenable to nutritional correction. When treating acute hemorrhage, packed RBCs are usually given: (1) if the hemoglobin level falls below 10 g/dL, (2) after rapid crystalloid infusion fails to restore normal vital signs, or (3) concurrently with crystalloid infusion in the treatment of obvious life-threatening blood loss.

Criteria for transfusion of packed RBCs to patients with anemia, also known as "transfusion thresholds," vary. [15] A hemoglobin level <10 g/dL is 1 criterion commonly suggested for transfusion before surgery. The National Institutes of Health does not recommend transfusion in asymptomatic nonsurgical patients with a hemoglobin >7 g/dL (hematocrit 21%). Healthy individuals sustaining acute blood loss may have no significant physiologic impairment with hemoglobin levels as low as 6 to 8 g/dL (hematocrit, 18% to 24%). The patient with circulatory shock and a hemoglobin concentration of 8 g/dL or greater may not benefit from a transfusion if volume-resuscitation has already been undertaken. [16] Patients with severe or chronic anemia or heart disease or those who otherwise require fluid restriction can receive packed RBCs more safely than whole blood. Furthermore, to prevent circulatory overload in susceptible patients, a rapid-acting diuretic, such as furosemide, can be administered intravenously at the beginning of the transfusion.

Blood Substitutes

Concerns over infection, the limited blood supply, the availability of blood in isolated locations, storage difficulties, and the risk of transfusion reactions has fueled interest in the development of blood substitutes that are viable in the clinical setting. Blood substitutes can be categorized as synthetic emulsions and modified hemoglobin.

Synthetic emulsions are made from fluorinated hydrocarbons, a perfluorocarbon base with particle size of 0.2 microns, thereby permitting capillary flow. These emulsions can dissolve large quantities of oxygen in a linear relation to the partial pressure of oxygen. At high inspired oxygen levels in the lungs, oxygen goes into solution and the process is reversed at the comparatively low oxygen tensions found in the tissues. The emulsions are subsequently eliminated unchanged by the lungs. Research with perfluorocarbons has been directed at finding a safe short-term vehicle for oxygen delivery in the absence of available blood. It is unlikely that synthetic emulsions will be used in the long-term management of anemia or blood loss. [17]

Modified hemoglobin solutions are biosynthetic products in which the hemoglobin molecule is altered by polymerization or encapsulation, or both. This creates a product that is fully oxygen saturated at ambient FiO_2, and also mimics the sigmoidal oxygen dissociation curve. A significant advantage of modified hemoglobin solutions is the absence of antigenic characteristics of blood groups, obviating the need for compatibility testing. Because hemoglobin solutions are derived from either human or bovine sources, the potential for disease transmission exists. It is possible that recombinant DNA technology will be used in the future, substantially eliminating this risk. Modified hemoglobin has been used successfully in the clinical setting, notably in cardiac and orthopedic surgery, and also in trauma resuscitation.[18]

Fresh Frozen Plasma

FFP is prepared by separating plasma from the cellular components of single-donor whole blood, followed by rapid freezing and storage at =18°C. Freezing preserves soluble coagulation factors of the intrinsic and extrinsic clotting systems, including the labile factors V and VIII. FFP also contains fibrinogen, although not as much as cryoprecipitate. Plasma stored for 3 months retains approximately 60% of the normal factor VIII activity and the product has a shelf life of up to 1 year. Ideally, transfused plasma should be compatible with the recipient's ABO group. Rh compatibility is not considered essential. [19] Each unit of FFP has a volume of approximately 200 to 250 mL.

FFP should be given to patients with a hereditary or acquired deficiency of coagulation factors, provided that a preparation of the specific deficient factor is not available. FFP is indicated for the clotting factor deficiencies resulting from massive blood replacement. However, pathologic hemorrhage following massive

transfusions is often caused by thrombocytopenia rather than by a depletion of clotting factors. One unit of FFP per 5 to 6 units of packed cells or whole blood is a reasonable replacement formula if specific clotting tests are not rapidly available, but plasma replacement is best dictated by evaluation of prothrombin time (PT) and partial thromboplastin time (PTT). FFP is indicated for rapid reversal of serious acute bleeding from warfarin (Coumadin) anticoagulants or for prophylaxis before surgery or an invasive procedure. In an emergency situation, 5 to 10 mL/kg of FFP will effect a rapid reversal of the vitamin K-dependent factors II, VII, IX, and X. As a rough guide, 1 unit of FFP increases all coagulation factor levels by 2% to 3% in the average-sized adult. In *life-threatening* hemorrhage from warfarin excess, factor IX concentrate (Konyne 80, Proplex, Mononine) may be used, but such therapy should not be routine because of the high incidence of hepatitis and the possibility of thrombosis with these products.[20]

FFP may be valuable in patients with other clotting abnormalities, such as a congenital deficiency of factor II, V, VII, X, XI, or XIII; von Willebrand syndrome; hemophilia A (factor VIII deficiency); hemophilia B (factor IX deficiency); or hypofibrinogenemia. However, the effectiveness is limited in severe clotting abnormalities because of the large volume that is generally required. For example, FFP may be successful in the treatment of hemarthrosis or other minor bleeding tendencies in hemophilia, but specific factor replacement is preferred. FFP is also used to treat the acquired deficiency of multiple factors such as those seen in severe liver disease, disseminated intravascular coagulation, or vitamin K depletion, and for plasma exchange in thrombotic thrombocytopenic purpura or hemolytic uremic syndrome. FFP should not be used for volume expansion or to enhance wound healing.

Reactions to FFP include fever, chills, allergic responses, HIV infection, and a risk of hepatitis similar to the risk with whole blood. FFP should be infused rapidly and given immediately after thawing because of the rapid loss of labile clotting factors.

The usual starting dose is 2 bags of FFP if the PT is >1.5 times normal or the activated PTT (aPTT) is >1.5 times the top normal value. If the PT is <22 seconds or the aPTT is in the 55 to 70 second range, 1 bag of FFP may be sufficient to bring the deficit into the hemostasis range (Table 29-6). Each 5 to 6 units of platelets contain the equivalent of 1 unit of FFP, so concomitant platelet infusions may lower FFP requirements.

Cryoprecipitate

Cryoprecipitate is prepared from single-donor plasma by gradual thawing of rapidly frozen plasma. This process causes precipitation of proteins rich in fibrinogen as well as clotting factors VIII and XIII. Each 15- to 25-mL bag of cryoprecipitate contains 60 to 120 units of factor VIII, 125 to 250 mg of fibrinogen, and an unknown amount of von Willebrand factor. Cryoprecipitate is a plasma product and as such requires ABO compatibility.

Cryoprecipitate is used to correct a deficiency of coagulation factor VIII (in hemophilia A and in von Willebrand syndrome), factor XIII, or fibrinogen. It is of no value in the treatment of factor IX deficiency (hemophilia B). One bag of cryoprecipitate per 5 kg of body weight will raise the recipient's factor VIII level to approximately 50% normal. The large number of units that must be given increases the chance of exposure to bloodborne diseases—thus cryoprecipitate is not recommended for the treatment of HIV-negative hemophiliacs. Factor VIII concentrate is a better choice because of improved methods of viral inactivation and the availability of factor VIII prepared using recombinant DNA technology.

Mild deficiencies of factor VIII are considered to exist at 10% to 30% of normal activity. Severe deficiencies exist at <3% of normal activity. The goal when treating bleeding depends on the site and severity of hemorrhage, but in general, one should seek at least 50% of normal factor VIII activity. Life-threatening hemorrhage requires correction to 100% activity. The amount of cryoprecipitate required to correct coagulation defects ranges from 10 to 20 units/kg for minor bleeding, such as hemarthrosis, to 50 units/kg for bleeding control in surgery or trauma. Specific replacement should be guided by laboratory assay of factor VIII activity. The half-life of factor VIII in plasma is 8 to 12 hours.

Rarely, cryoprecipitate may be required to correct significant hypofibrinogenemia (<100 mg/dL). FFP may also be used to treat mild degrees of hypofibrinogenemia.

Factor VIII Concentrate

Human Antihemophilic Factor

Factor VIII extracted from pooled human plasma produces a concentrated stable product with a shelf life of up to 2 years. Significantly more concentrated than cryoprecipitate and

TABLE 29-6 -- Transfusion of Blood Products

Blood Product	Waiting Time to Receive in Emergency Department	Initial Amount to Transfuse	Expected Response in 70-kg Adult
Un-crossmatched Rh^+ or Rh^- RBCs	5 min	2–10 units, 10 to 20 mL/kg per hr or as needed based on clinical condition	Stabilize patient in shock
Un-crossmatched type-specific whole blood	15 min		Change in hemoglobin/hematocrit depends on hydration and rate of bleeding
Typed and screened whole blood	25 min		Approximate rise of 1 g/dL hemoglobin per unit
Crossmatched whole blood	1 ¼ hr		Each unit raises hematocrit 2–3%
Packed RBCs	1 ½ hr		In children, each mL/kg of packed cells raises hematocrit by 1%
Frozen RBCs	4–6 hr (if not prepared inhouse)		
Platelet concentrate†	5 min if available	1 unit per 10 kg, usually 6 to 10 units per transfusion in an adult	Rise of 5000 to 10,000 platelets per mm^3 per unit; 6 units usually sufficient to stop bleeding
Cryoprecipitate	20 min	1–2 bags per 10 kg (7–15 bags) 10-min push, or 20–50 units/kg	Rise of 3% in factor VIII level per bag (40–100% activity desired)
Factor IX or prothrombin concentrate	Immediately available (reconstituted powder)	10–50 units/kg	30–100% rise in factor IX activity
Fresh frozen plasma	40 min	1 bag per 7 kg (4–10 bags for adult) 10-min push,* 3–10 mL/kg, depending on clinical condition	Correction in coagulation status; 1 unit raises all coagulation factors by 2–3% in average-sized adult

†Also consider thrombocytopenia as a cause of bleeding from massive transfusion.
*Administer 1 bag per 4 to 6 units of blood transfused to replace diluted and inactivated coagulation factors.

available for home use, factor VIII concentrate was a major breakthrough in the treatment of hemophilia. Unfortunately the presence of viruses in the donor pool contributed to the high prevalence of hepatitis and HIV in hemophiliacs who used earlier versions of this product. Newer products (Alphanate, Hemofil, Humate-P, Koate-DVI, Monarc-M, Monoclate-P) are produced using one or more methods to reduce viral contamination including heat treatment, pasteurization, organic solvents and detergents, gel filtration, and immunoaffinity chromatography. These methods have markedly reduced the risk of viral transmission, especially lipid encapsulated

viruses (HIV, hepatitis B and C). To date there are no reports of transmission of these viruses with the products listed earlier.

Recombinant Antihemophilic Factor

Since the gene for factor VIII production was discovered in 1984, research into recombinant genetics has aimed to provide a safer product that theoretically will be more readily available and less expensive to produce. Two recombinant-DNA derived factor VIII preparations (Recombinate, Kogenate) were approved by the FDA in 1993. More recent introductions include Bioclate, Helixate, Helixate FS, and Kogenate FS. These genetically engineered products have hemostatic activity equivalent to plasma-derived factor VIII and minimal risk of viral contamination. Because some of these products are prepared using human albumin and other animal proteins, there is potential for viral transmission. Products such as Helixate FS and Kogenate FS are prepared without human albumin, which should eliminate viral contamination. Although costlier, they are a better choice for young and newly diagnosed patients who have not already been exposed to hepatitis or HIV. [21]

Use of Factor VIII Concentrate

Administration of 1 unit of factor VIII concentrate per kg of body weight should increase the factor VIII activity by 2% to 2.5%. Dosage should be individualized based on the severity of bleeding, the known deficiency of factor VIII activity, and the presence of factor VIII antibodies. Factor VIII levels should be increased to 20% to 40% of normal for minor bleeds (small joint), 40% to 60% for moderate bleeds (large joint, neck, oral cavity), and 60% to 100% for life-threatening bleeds (intracranial, intra-abdominal, pharyngeal).

Antibodies develop in up to 15% of factor VIII recipients. Administration of massive doses of factor VIII has proven beneficial in overwhelming the endogenous antibody response. In addition, immunoadsorbent techniques to remove the antibody have met with some success. The general use of immunosuppressives and plasmapheresis has had limited success. Activated prothrombin complex has been effective, but concern over the cost of preparation, the significant hepatitis risk, and the thrombogenicity associated with its use limits its application.

DDAVP

A synthetic analogue of pituitary vasopressin, 1-deamino-(8-D-arginine)-vasopressin (DDAVP), has been found to stimulate the endogenous production of factor VIII in a subset of mild hemophiliacs. The exact mechanism is unknown, but treatment with 0.3 mg/kg intravenously over 15 minutes has been recommended when avoidance of the inherent risks of the factor VIII concentrate is desired.

Factor VIII Inhibitor Bypassing Activity

Factor VIII inhibitor bypassing activity (FEIBA) is a product derived from pooled human plasma containing factors II, VII, IX, and X. It promotes coagulation by bypassing the need for factors VIII and IX. FEIBA is vapor heated to achieve >10 logs of reduction in all target viruses. FEIBA is used to treat bleeding episodes in hemophilic patients with antibodies to factor VIII. Adverse reactions include headache, fever, chills, flushing, nausea, vomiting, and an occasional allergic reaction. The risk of thrombotic complications exists, especially in patients with liver and heart disease, or those who are pregnant or breast-feeding. [22]

Factor IX Concentrate

Factor IX is prepared from pooled human plasma and is available as a lyophilized powder either as an isolated factor concentrate (Alphanine, Mononine) or as prothrombin complex concentrate, which also includes the liver-synthesized, vitamin K-dependent factors II, VII and X (Bebulin, Konyne, Profilnine, or Proplex). Factor IX is also available using recombinant technology.

Historically, the use of factor IX concentrate has carried a very high risk of hepatitis transmission. However improved donor screening and new methods of viral reduction have substantially reduced the risk of viral transmission. As with factor VIII concentrate, the risk of HIV and hepatitis transmission is very low using current human-derived products. Recombinant factor IX is not derived from human products and carries no risk of viral transmission.

Factor IX is indicated for the treatment of bleeding episodes in hemophilia B patients with severe deficiency of factor IX. FFP is the preferred treatment in patients with mild-moderate deficiency. Administration of 1 unit/kg body weight will increase the factor IX concentration approximately 1%. High levels of factor IX are not required to control bleeding. Levels should be increased to 15% to 25% of normal for mild-moderate bleeding, and 25% to 50% of normal for more serious bleeds or before major surgery.

Hypercoagulable states have been reported following factor IX infusions, particularly with use of prothrombin complex concentrate.

Platelet Concentrates

Platelet concentrates are prepared by rapid centrifugation of platelet-rich plasma. Platelets are obtained by single-donor apheresis or from random donor whole blood units. HLA-matched platelets may be used when patients develop HLA antibodies from repeated random donor platelet transfusions. Platelet concentrates contain most of the platelets from 1 unit of blood in 30 to 50 mL of plasma. One unit (pack) of platelets per 7 kg of body weight will raise the platelet count by 50,000/mm^3 in the absence of antibodies; therefore, 1 unit of platelet concentrate raises the platelet count by 5000 to 10,000/mm^3. The usual adult dose is 6 to 10 units of platelet concentrate, depending on the clinical condition. Assuming a zero platelet level, 6 units given to a normal-sized adult should increase the platelet count to >50,000 per mm^3. If there is no evidence of platelet consumption, this transfusion should be adequate for 3 to 5 days. In cases of severe platelet consumption, the transfusion may be required every 6 to 24 hours. Some hospital blood banks prepare platelet concentrates regularly; in some cities a central blood bank service, such as the American Red Cross, prepares platelet concentrates regularly and delivers units on an "as-needed" basis within 1 to 2 hours of the request. Platelet concentrates are viable for 5 days when kept at *room temperature* and gently agitated at intermittent periods or when kept in motion. They should not be refrigerated.

This issue of prophylactic platelet transfusion remains controversial. Spontaneous bleeding rarely occurs if the platelet count is >10,000 to 20,000/mm^3. Even in the event of surgery or trauma, excessive bleeding is uncommon in patients whose platelet count exceeds 50,000/mm^3. It is generally recommended that active hemorrhage be treated with platelet transfusion if the platelet count is <50,000/mm^3, but prophylactic transfusion may be safely withheld until the platelet count is <20,000/mm^3. Patients with idiopathic thrombocytopenic purpura (ITP) should not receive platelets prophylactically, but they may be transfused if life-threatening bleeding occurs.

Crossmatching is unnecessary for platelet transfusion, but the donor and the recipient should be ABO- and Rh-compatible. Note that platelet concentrates contain enough RBCs to sensitize an Rh-negative individual. There may be a diluting effect to the platelet count that results in thrombocytopenia with massive blood transfusions. When more than 8 to 10 units of blood are transfused, the platelet count must be routinely evaluated, and platelets must be replaced accordingly. Clinically significant platelet depletion rarely occurs if <15 units of blood (or 1.5 to 2 times blood volume) have been transfused.

Each 5 to 6 units of platelets contains 250 to 350 mL of plasma (about 1 bag of FFP), which includes coagulation factors that may reduce the requirements of FFP. Platelets may be infused rapidly (1 unit/10 minutes), using specialized platelet filters.

Granulocyte Transfusions

Granulocyte transfusions are indicated in severely neutropenic patients with suspected or proven bacterial infections unresponsive to appropriate treatment. They are rarely given in the emergency setting. White blood cell transfusions require prior arrangements with a large blood bank service that has the capabilities of collecting granulocytes from a suitable donor; the collection procedure takes 4 to 6 hours on a continuous-flow cell separator. Transfusions must be repeated frequently (every 12 hours) to provide a sufficient number of white blood cells to help the patient.

Blood Products for Jehovah's Witnesses

There are more than 1.5 million Jehovah's Witnesses in the United States. Based on the religious belief that the Bible prohibits blood or blood product transfusion (Acts 15:28–29), Jehovah's Witnesses do not accept transfusions of whole blood, packed cells, white blood cells, platelets, plasma, or autologous blood. Some may permit infusion of albumin, clotting factor solutions, or dextran or other plasma expanders and intraoperative autotransfusion. [23] Although no guidelines for administration of blood products to Jehovah's Witnesses are absolute, certain recommendations can be made. Even though a transfusion may be necessary to save a patient's life and would otherwise be considered standard care, the administration of blood or blood products, or both,

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in the face of refusal after informed consent can be legally considered as battery or a violation of a patient's right to control what is done to his or her body. In the awake and otherwise competent adult, courts have ruled that clinicians cannot be held liable if they comply with a patient's directive and withhold life-saving blood administration following specific and detailed informed consent of the consequences of such an omission of treatment. The issue becomes clouded when patients are incompetent, unconscious (most Jehovah's Witnesses carry cards informing medical personnel of their religious beliefs), or minors.

In the absence of specific directives to the contrary, it is prudent to administer blood products to patients who are unconscious, judged to be incompetent adults, or who are minors. Although case law often upholds the patient's wishes, actual damages against clinicians are difficult to document in the United States. When done under documented life-threatening circumstances, significant clinician liability would be extremely unusual. Pregnant females and significant providers for dependents have been deemed appropriate recipients of blood products against their wishes. Explicit documentation of the intent of the clinician to preserve life coupled with an accurate description of the discussion of the issue with the patient or the family and a clarification of the patient's mental capacity is mandatory. Furthermore, emergency legal assistance (such as court orders, appointment of a temporary guardian) should be sought immediately with rapid judicial resolution. Various clinical techniques to maximize oxygen delivery and minimize oxygen consumption should be used. Examples include limited blood draws, the use of erythropoietin and nutritional support, hypothermia, volume expansion, sedation, and oxygen.

ADMINISTRATION OF BLOOD COMPONENTS

When it has been decided that a patient needs a transfusion and the patient's condition is stable enough, the clinician should question the patient or the patient's relatives concerning any previous transfusion reactions and whether the patient abides by any religious prohibitions to transfusions. A tube of blood (approximately 2 mL for every unit of blood product to be crossmatched) should be drawn from the patient and put into a red-topped, nonanticoagulated tube. The tube must *not* contain a serum separator gel. The label should be signed by the individual drawing the blood sample. This identifying signature will be used in the blood bank's crossmatching procedures.

Emergency Transfusion

In an emergency or life-threatening situation, three alternatives to fully crossmatched blood exist. The preferred substitute is type-specific blood with an abbreviated crossmatch. The abbreviated crossmatch includes ABO and Rh compatibility. In addition, the recipient's serum is screened for unexpected antibodies, and an immediate "spin" crossmatch is performed at room temperature. This abbreviated crossmatch requires approximately 30 minutes. Many institutions are now using this procedure as their standard crossmatch for most patients. The safety and utility of the type-specific abbreviated crossmatch have been demonstrated repeatedly, and transfusion reactions occur only rarely. [24]

The second preference for an alternative to fully crossmatched blood is type-specific blood that is only ABO- and Rh-compatible, without screen or immediate spin crossmatch. The patient's ABO group and Rh factor can be determined within 2 minutes, and, in an emergency, typing of the blood group and the Rh factor is all that is necessary before transfusion. Type-specific blood that is not crossmatched has been given in numerous military and civilian series without serious consequences. While the type-specific blood is being transfused, the antibody screen and the crossmatch are carried out in the laboratory; the transfusion should be stopped if an incompatibility is found.

Ideally, type-specific blood should be compatible for both Rh factor and ABO group; however, Rh-positive blood may be transfused to Rh-negative patients in an extreme emergency or in times of disaster or blood shortage. Sensitization to the Rh factor is most problematic for Rh-negative women of reproductive age. [25] Any sensitized patient may experience a transfusion reaction if exposed again to Rh-incompatible blood. However, significant, subsequent transfusion reactions with Rh-incompatible blood in men sensitized to the Rh factor are very rare.

A third alternative to fully crossmatched blood is group O blood, although type-specific blood is generally preferable. [26] It is rare that a few minutes cannot safely be expended to allow the blood bank to release type-specific blood. This assumes, however, that transport of the blood specimens and other logistic issues can be resolved to make type-specific blood available within minutes. Often this is not the case. In addition, type O blood is often stored outside of the blood bank so as to be readily available for life-threatening emergencies. Thus, despite the theoretical preference for type-specific blood in emergency situations, type O is often a reasonable and practical alternative.

Group O negative *whole* blood was in the past designated the "universal donor" blood, because a recipient's naturally occurring antibodies (anti-A and anti-B) do not react with donor group O RBCs. Nonetheless, some donor serum may have a high titer of naturally occurring anti-A and anti-B antibodies capable of hemolyzing the recipient's (patient's) RBCs if large quantities of blood are transfused. True universal donor blood is low in anti-A and anti-B titer. Because group O donors are not regularly screened for unsafe levels of anti-A and anti-B titers, the use of even small amounts of group O *whole blood* that is not crossmatched is potentially dangerous. The significance of varying titers of anti-A and anti-B antibodies in the donor whole blood may be essentially eliminated if *packed cells* are used instead of whole blood. Other RBC antigens on type O RBCs may sensitize the patient or cause antibody production, complicating future crossmatching or possibly causing future hemolytic transfusion reactions.

Approximately 25% of patients receiving a transfusion of 5 or more units of type O whole blood develop hyperbilirubinemia suggestive of a minor hemolytic reaction. Large amounts of group O whole blood may cause the patient to acquire significant amounts of anti-A and anti-B antibodies that have been passively transfused; hemolysis of RBCs may then occur when the recipient's original blood group is subsequently transfused. In a resuscitation, one should continue to use group O blood if large amounts (i.e., >2 units) of *whole blood* have already been given.

One may transfuse both Rh-positive and Rh-negative group O packed cells in patients who are in critical condition.

It is a common *misconception* that patients who are Rh-negative will have an immediate transfusion reaction if given Rh-positive blood. There is no particular advantage in the Rh factor determination because preformed, naturally occurring anti-Rh antibodies do not exist. Theoretically, individuals who are Rh-negative may become sensitized either through pregnancy or by previous transfusions, resulting in a delayed hemolytic transfusion reaction if Rh-positive blood is transfused. However, this scenario is very rare and is of no great clinical significance when compared with life-threatening blood loss. Many advise the routine use of the more widely available *O Rh-positive* packed cells in all patients for whom the Rh factor has not been determined, except in females of childbearing age, for whom future Rh sensitization may be an important consideration. Once resuscitated with Rh-positive packed cells, patients may receive their own type without a problem. Because individuals with O Rh-negative blood represent only 15% of the population and the blood may be in short supply, it is reasonable to save O Rh-negative blood for Rh-negative females of childbearing potential and to use *group O Rh-positive packed cells routinely as the first choice for emergency transfusions*. In a study of emergency blood needs, Schmidt and colleagues reported 601 units of Rh-positive type O blood transfused to 193 patients, including 8 Rh-negative women, before blood type was determined. No acute hemolytic reaction occurred, and no women were sensitized. *Rh immune globulin prophylaxis is recommended only for Rh-negative women with childbearing potential receiving Rh-positive blood.*

If noncrossmatched blood is transfused, the laboratory should receive a plain (without a serum separator) red-top tube of venous blood as soon as possible to begin a formal crossmatch procedure. Whenever possible, this should be drawn before any blood is transfused. Brickman and coworkers have demonstrated that bone marrow aspirates obtained by an intraosseous needle can be used for crossmatching. [27]

Rh immune prophylaxis with human immune globulins (RhoGAM) is indicated for Rh-negative pregnant women who may be bearing Rh-positive children and may have fetomaternal transplacental hemorrhage. These events include bleeding in early pregnancy, such as spontaneous or elective abortion, ectopic pregnancy, and other potential causes of antepartum hemorrhage such as trauma. Administration of Rh immunoglobulin in *threatened abortions* is advocated by some. The product suppresses the immune response of Rh-negative women to Rh-positive RBCs, and it is effective when given up to 72 hours after exposure to fetal erythrocytes. Dosing of Rh immunoglobulin is 50 µg intramuscularly (IM) for first-trimester bleeding and 300 µg IM for later bleeding. [28] In the setting of *significant fetal-maternal transfusion* (usually only in the third trimester), doses may be increased. In such circumstances, Rh immunoglobulin is prepared in the blood bank and the correct dose is suggested on an individual basis, following confirmation of Rh status, evidence of prior sensitization, and testing for fetal erythrocytes in the mother's blood.

Transfusion Coagulopathy

Within the past 10 years it has been appreciated that pathologic hemostasis occurs following massive blood transfusions. [29] [30] [31] The exact cause of the transfusion coagulopathy is not well understood. Although such abnormalities rarely develop within the time frame of the initial resuscitation in the emergency department (ED), an understanding of the problem leads to a more intelligent approach to transfusion practices and the anticipation of potential problems. The term *massive transfusion* is loosely defined but is usually considered to be the transfusion of >10 units of blood to an adult (equivalent to 1 blood volume) within 24 hours. In patients who are given a transfusion equal to 2 blood volumes, only approximately 10% of the original elements remain. Considering the significant alteration in blood and blood products that occurs during storage, one can readily appreciate the underlying problem associated with such massive transfusions. The development of transfusion coagulopathy is multifactorial and in large part is related to tissue injury and duration of shock. [32] Abnormalities in platelets and plasma clotting factors also play a

role.

Platelets

Transfusion coagulopathy is related in part to dilution of the recipient's platelets by transfused blood, which is devoid of functioning platelets. Dilutional thrombocytopenia is a well-recognized complication of massive transfusion, and a platelet count should be obtained routinely if >5 units of blood are transfused. Generally, platelet therapy should be considered after the first 10 units of blood have been given, although the most useful parameter for estimating the need for platelet transfusions is the platelet count.

Plasma Clotting Factors

Disseminated intravascular coagulopathy plays a secondary role in post-transfusion bleeding. Factors V and VIII are labile in stored blood and absent in packed cells. Fibrinogen is relatively stable in stored blood but is absent in packed cells. A deficiency of most clotting factors, especially factors V and VIII and fibrinogen, occurs with massive transfusions. This deficiency probably occurs on a "washout" (i.e., dilutional) basis, although the dynamics are poorly understood. The replacement of these factors may be required. Specific assays for the individual factors are available, but it is more practical to measure PTT, PT, and fibrinogen levels. FFP has been used to correct clotting factor abnormalities secondary to dilution from massive transfusions, but its effectiveness has not been firmly established. Cryoprecipitate has also been used to replace factor VIII and fibrinogen, but it is rarely required, because FFP contains some fibrinogen. FFP should be infused to correct the coagulopathy as indicated by clotting studies, but as a general guide, 1 to 2 units of FFP may be given empirically for each 5 to 6 units of blood in the massively traumatized or bleeding patient. Cryoprecipitate may be required if fibrinogen levels fall below 100 mg/dL and are not adequately supplemented with FFP.

ORDERING OF BLOOD

Ordering a type and crossmatch procedure on a blood product implies that the decision has already been made to administer a transfusion. A "type and hold" or "type and screen" (no crossmatch) request alerts the blood bank to the *possibility* that a blood product will be required for the patient, so appropriate units can be acquired and kept on hand. A type and crossmatch procedure takes 45 minutes and restricts a unit of blood to a specific patient. This limits a valuable resource and should not be requested lightly. In the ED, a crossmatch procedure should be requested for a blood product only if the adult patient (1) manifests shock, (2) has *symptomatic* anemia

(usually associated with a hemoglobin <10 g/dL) in the ED, (3) has a documented loss of 1000 mL of blood, or (4) requires a blood-losing operation immediately (e.g., thoracotomy).[33] A type and hold can safely be requested for all other situations in which a blood transfusion is considered possible during the patient's care; a desirable ratio of units crossmatched to units transfused can thus be achieved. Hooker and colleagues found that the empiric trigger of pre-hospital hypotension (systolic blood pressure <100 mm Hg) was a useful discriminator for ordering early crossmatched blood. [34]

The number of units requested for a crossmatch procedure is determined by the size of the patient, response of the patient to the injury and subsequent emergency treatment, and presence of ongoing blood losses (e.g., arterial or massive gastrointestinal bleeding). In the majority of fatalities from massive hemorrhage, the patients die from hypovolemia rather than from lack of oxygen-carrying capacity. Table 29-6 provides specific guidelines for administering blood components.

RBC preparations for transfusion are not routinely tested for the presence of sickle hemoglobin. Donors with sickle trait are not excluded, and blood with sickle trait can be safely given to almost every patient, because occlusion of blood flow caused by intravascular sickling would occur only in extreme conditions of acidity, hypoxia, or hypothermia that are unlikely to be compatible with life. Nonetheless, when transfusion is being performed in infants and patients with known sickle cell anemia, the blood bank should be alerted, and a "sickle prep" should be requested for donor blood to avoid the infusion of sickle-trait blood into such patients. In rare instances, blood from a donor with a mild variant, such as hemoglobin SC disease, has caused massive intravascular sickling and death in a sick, hypoxic, acidotic infant.[35]

Blood Request Forms

The most important part of ordering blood components for a patient is proper identification of the patient and the intended unit of blood. Transfusion of an incorrect unit is a potentially fatal error. *Most transfusion mistakes are clerical errors.* Several identification systems have been established to minimize the

Figure 29-1 The Typenex blood recipient identification system. The identity of the patient and the blood sample are ensured by numbered labels on the tube and on the bracelet. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

Figure 29-2 In the blood bank, crossmatched units of blood are identified with numbered labels from the patient's blood sample. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

risk of improper transfusions (Fig. 29-1). Just before administering the blood, the nurse or clinician must check the identity of the numbered labels. In addition, the blood bank laboratory slip must identify the patient by name and number and contain the identification number of the unit of blood. Because most serious transfusion reactions are due to misidentification issues, one cannot be overcautious in adhering to these identification procedures (Fig. 29-2).

Usual procedures require a separate *blood bank request form* for each unit of RBCs or whole blood ordered. A number of units of FFP, cryoprecipitate, and platelet concentrates may be ordered on one form with proper identification (depending on individual blood bank procedures). When the blood bank indicates that the units ordered are ready, the person picking up the blood, along with the blood bank technician, must check the notation on the *blood release form (transfusion form)* to verify the identity of the patient (name, hospital number) and to ensure that the blood unit has been prepared for that patient (blood group and type, unit number). Immediately before administering the blood to the patient, the nurse or clinician must check the release form, blood unit, and patient tag for identity as well as the expiration date of the unit (Fig. 29-3).

INTRAVENOUS ADMINISTRATION

One should not open the unit of blood until and unless a free-flowing intravenous (IV) access line has been established in a large-bore vein. A 14-ga IV catheter is preferred, both to minimize hemolysis and to ensure rapid infusion of fluid for the treatment of hypovolemia or hypotension. When much blood must be given rapidly, administration by means of a high-flow infusion system is preferred. Standard central venous pressure lines are generally too small for adequate volume resuscitation in patients whose conditions are critical. Likewise, the purpose of a large-bore infusion line is defeated

Figure 29-3 Before administration of the blood unit, the numbered labels on the patient's bracelet and on the unit of blood are checked for identity. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

if blood is piggybacked with an 18- to 20-ga needle through a side port in the infusion tubing. For an elective transfusion, however, blood may be given through a smaller lumen. Combining hemodilution (250 mL saline to 1 unit packed RBCs) and pressurization can safely increase the flow rate through 20- and 22-ga catheters several-fold.[36] No significant hemolysis occurs when small-gauge (21-, 23-, 25-, and 27-ga) short needles are used for transfusion of fresh blood or packed cells in infants and children and when the maximum rate

Figure 29-4 An example of a blood administration Y set with two adapters for insertion into a unit of blood or saline; note the in-line filter. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

Figure 29-5 One upper adapter has been inserted into a bag containing normal (0.9%) saline. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

of infusion is <100 mL/hour.[37] For rapid infusion, however, the blood administration tubing is connected directly to the infusion catheter. The infusion site should be monitored for infiltration, infection, or local reactions. Antiseptic technique is essential (Fig. 29-4 Fig. 29-5 Fig. 29-6 Fig. 29-7 Fig. 29-8).

If the patient already has a suitable IV line in place, a solution of 0.9% normal saline only should be used to flush

Figure 29-6 The entry site of the unit of blood, into which the other upper adapter of the Y set should be inserted. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

Figure 29-7 Inserting the hard plastic spike of the upper adapter. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

the system before administering the blood. Other IV fluids are *not* to be used because of the risks of hemolysis or aggregation (with 5% dextrose in water) [38] or clotting (with lactated Ringer's solution). [39] No medications should be placed into the unit of blood or added to the infusion line for the same reasons.

Filters

All blood and blood products should be infused only through an appropriate filter, such as those supplied in-line in the

Figure 29-8 After the plastic tubing has been primed with saline, the blood flows through into the patient. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

Figure 29-9 Administration of blood through a microaggregate filter attached between the unit of blood and the hard plastic spike of the administration set. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

blood administration tubing sets. In the past, filtration was required merely to keep the IV line from becoming blocked by clots. Adverse consequences that result from infusing unfiltered blood products have since been recognized. Debris consisting of clots and aggregates of fibrin, white blood cells, platelets, and intertwined RBCs (ranging in size from 15 to 200 µm) accumulate progressively during storage of blood. The usual filter, made of a single layer of plastic with multiple 170-µm pores, traps larger particles yet allows rapid infusion of 2 to 3 units of blood before flow is obstructed. Purified *components* of blood plasma can be safely administered through a filter with pores as fine as 5 µm.

It has been suggested that microaggregates of debris, which could pass through a 170-µm filter, may in part contribute to the syndrome of "shock lung" seen after transfusions of many blood units in patients suffering from severe trauma and hemorrhage. Some clinicians therefore recommend the use of a microaggregate blood infusion filter with a mesh pore size of 40 µm (Fig. 29-9) when multiple units of blood are administered to a trauma victim, a patient with compromised pulmonary function, or a neonate. Microaggregate filters tend to become blocked, impeding the rate of infusion more quickly, and are not commonly required in the emergency setting. In addition, whether the infusion of microaggregates (between 40 and 170 µm in size) is in fact harmful is still an unsettled issue. [40] Standard filters should be replaced after 2 to 3 units of blood product have been administered; most microaggregate filters should be changed after each unit. It is generally agreed that a significant number of platelets is removed by microaggregate filters, and some advise against

using these filters when platelet packs are infused. Others believe that although platelets are removed with the microaggregate filters, the trapped platelets can be removed with saline flush without any significant loss. [41] Table 29-7 lists some available in-line filters.

Rate of Infusion

One unit of whole blood can be safely administered to a hypotensive patient at a rate of 20 mL/kg per hour. In the setting of hypovolemic shock and continued hemorrhage, there is no limit to the transfusion rate, Multiple units may be transfused simultaneously, even under pressure (see Chapter 24). In the stable patient, 1 unit of *whole blood* (500 mL) should be administered over approximately a 2-hour period (3 to 4 mL/kg per hour). After this time, RBCs begin to lose metabolic activity. In addition, the unit of blood, which is an excellent culture medium, is likely to become contaminated if bacteria and fungi are allowed to grow at room temperature. Packed cells should be given at approximately the same rate; plasma products may be given more rapidly. In a patient with a healthy cardiovascular system, one should administer FFP more rapidly (about 15 to 20 minutes/unit) to correct coagulation deficits, because the coagulant activity begins to deteriorate rapidly after 20 to 30 minutes of thawing. In patients with severe anemia and congestive heart failure, a rapidly acting diuretic, such as furosemide, can be given (0.5 mg/kg IV) at the onset of transfusion to obviate circulatory overload.

If a transfusion of blood must be interrupted or delayed for some reason, the remainder of the blood unit should be returned to the blood bank. More convenient refrigerators in

TABLE 29-7 -- In-Line Filters for Blood Transfusion

Filter	Pore Size	Use and Contraindications
Standard		
Fenwal STD Blood Filter	170 µm	All blood components
McGaw STD Blood Filter	170 µm	All blood components
Special Use		
Fenwal 4C2100	170 µm	Platelets, cryoprecipitate, antihemophilic factor concentrates, fresh whole blood
Microaggregate Filters		

Fenwal Microaggregate Blood Filter 4C2423 or 4C2131	20 µm	Removes most platelets and leukocytes from blood being transfused; do not use with fresh whole blood or concentrates of platelets or WBCs; primarily indicated for use with patients receiving *multiple transfusions* of stored blood, patients with *compromised pulmonary function,* and those undergoing cardiopulmonary bypass; recommended for use in most newborns
Fenwal PDF-10 4C2428		
Intersept Blood Filter (Johnson & Johnson Co., New Brunswick, NJ)	20 µm	
Alpha Micron-40 (Alpha Therapeutics Corp., Los Angeles)	40 µm	
Bentley Disposable Blood Filter PF 127 (Bentley Labs., Inc., Irvine, CA)	27 µm	
Hemonate (Gesco Labs., San Antonio, TX)	40 µm	
Swank In-Line Blood Filter IL-700 (Pioneer Filters, Inc., Beaverton, OR)	20 µm	
Pall Ultipor SQ405 (Pall Biomedical Product Corp., Glen Cove, NY 11542)	40 µm	

the ED or on the hospital unit *should not* be used to store blood products unless they are temperature-controlled or continuously monitored and alarmed.

Patients who are in hemorrhagic shock can receive blood through two large-bore catheters at different sites. Usually gravity provides a sufficient pressure gradient if the unit is raised above the patient to increase the rate of infusion when the clamps are wide open. If a pressure pump is used (Fig. 29-10 and Fig. 29-11), the infusion can be quicker.[42] A standard sphygmomanometer cuff should never be wrapped around a unit of blood to create increased infusion pressure, because the nonuniform application of pressure could burst the plastic bag containing the blood component.

One can dilute packed RBCs with *normal saline* (0.9%, without dextrose) before infusion simply by opening the clamps on the upper tubes of the Y infusion set and leaving the lower (recipient end) clamps closed. Although it is generally agreed (and recommended by the editors) that lactated Ringer's solution should never be mixed with blood because of possible clot formation, two studies have demonstrated that small amounts of lactated Ringer's solution *is* compatible with blood. King and associates found that 100 mL could be added to a unit of packed RBCs without precipitating blood clotting, [43] while Cull and coworkers suggest that up to 150 mL of lactated Ringer's solution may be safely added as a diluent to each unit of packed RBC. [44] Therefore, blood inadvertently mixed with small amounts of lactated Ringer's solution need not be routinely discarded. Dilution will allow for more rapid infusion by decreasing the blood viscosity, which is dependent on hematocrit, at the risk of increasing volume. Alternatively, the direct addition of approximately

Figure 29-10 A controlled-pressure administration device for rapid infusion of blood products. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

200 mL of normal saline to the bag of packed RBCs has been recommended to bring the hematocrit in the blood bag to approximately 45%.

Rewarming

Blood is stored at approximately 4°C to maintain cellular integrity and to prevent the overgrowth of microorganisms. Blood products usually passively warm to 10°C by the time they are administered to the recipient, unless administered under pressure. Adverse effects of hypothermia on cardiac conduction and flow rates are evident when rapid administration of a large volume of blood is performed without prewarming.

Various mechanisms have been used to warm blood to 35 to 37°C. An ideal blood warmer should allow liberal flow rates while preventing thermal hemolysis of blood cells. Commonly used devices are bath coils that allow a plastic tube to reside in a closely regulated warm water bath, as well as dry heat devices that allow blood to circulate through flat, thin bags sandwiched between aluminum blocks that contain electric heating elements. Both devices have relatively low flow rates and suboptimal thermal clearance.[45] Blood bag immersion in warm water baths is safe, but it is considered imprecise and slow. Although much interest surrounds the use of microwave heating devices, the technique is not recommended by the Association of Blood Banks because of the risk of hemolysis. Herron and colleagues advocate keeping the packed RBC temperature <50°C as they noted significant hemolysis beginning at 51 to 53°C. [46]

Rapid admixture warming is a promising alternative technique (see Chapter 24).[47] The unit of whole blood is

Figure 29-11 A rubber bladder is pumped up, and the blood unit is squeezed uniformly against a reinforced mesh. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

mixed with an equal amount of normal saline, which has been preheated to 60 to 70°C. Once mixed, the product is administered to the patient with a resultant delivery temperature of approximately 35°C. This technique combines dilution of blood product and warming into one step. Regardless of the rewarming technique used, warming refrigerated blood to body temperature decreases its viscosity two-fold to three-fold and avoids venous spasm, thus facilitating transfusion.

Monitoring

During the first 5 to 10 minutes and then every 15 minutes during a transfusion of any blood product, the patient must be carefully monitored for evidence of a transfusion reaction. Signs and symptoms are hives, chills, diarrhea, fever, pruritus, flushing, abdominal or back pain, tightness in the chest or throat, and respiratory distress. A potentially life-threatening acute hemolytic transfusion reaction in a patient who has received prior transfusions may differ clinically from a minor allergic reaction only by its effects on the patient's pulse and blood pressure. One can safely treat an allergic reaction to leukocytes or plasma proteins that cause hives, itching, fever, or chills by administering an antihistamine (but not into the blood infusion line) and stopping the transfusion.

Transfusion should be stopped immediately when the following signs are encountered: an increase in pulse rate, a decrease in blood pressure, respiratory symptoms, chest or abdominal discomfort, or a sensation of "impending doom." Normal saline should be administered to maintain blood pressure and urine output. Samples of urine and blood should be sent to the laboratory to verify the presence of free hemoglobin. The blood bank should also receive a clotted sample of

blood to reassess the presence of any immune reaction. If the blood bank concludes that the reaction is a nonhemolytic allergic response, premedication with antihistamines (diphen-hydramine or hydroxyzine) and antipyretics is indicated before the next transfusion. Alternatively, washed cells could be used.

The patient in whom a hemolytic transfusion reaction is suspected should be treated vigorously and promptly. [48] Most mortality and morbidity is secondary to hypotension and shock leading to cardiovascular instability, renal insufficiency, respiratory manifestations, or hemorrhagic complications of disseminated intravascular coagulation. The initial treatment is directed at treating the hypotension by infusion of 5% dextrose in saline or lactated Ringer's solution or vasopressors, if required. The volume and rate of infusion are determined by blood pressure response. Symptomatic treatment with acetaminophen, a warming blanket, an inhaled or subcutaneous ß-agonist agent for bronchospasm or subglottic edema, or antipruritics or antihistamines is of secondary importance.

If an acute hemolytic transfusion reaction occurs, there may be some benefit from alkalinization of the urine with IV sodium bicarbonate to prevent the precipitation of free hemoglobin. Forced diuresis with mannitol to maintain the urine output at 50 to 100 mL/hour has also been advocated. The benefit from alkalinization and diuresis in the prevention of acute renal shutdown is uncertain, although the use of these techniques is commonly advocated. After shock is controlled, an assessment of hemostasis, respiratory function, renal function, and cardiac function will help direct later therapy of the complications; disseminated intravascular coagulation may require the administration of plasma, platelets, or fibrinogen, and acute tubular necrosis may dictate careful fluid management. Hemolytic transfusion reactions have become unusual. They are rarely fatal and are usually attributable to an error in identification (such as can result from the treatment of two "John Doe" patients simultaneously).

Delayed, or "late," hemolytic transfusion reactions may occur days, or even weeks, after transfusion of RBCs. They are characterized by falling hemoglobin levels, jaundice, hemoglobinemia, and indirect hyperbilirubinemia. [49] This complication is usually self-limited and is not life-threatening. Therapy is symptomatic, but future attempts at crossmatching for transfusions may be difficult because of the presence of RBC antibodies. Individuals so affected should wear identification tags or bracelets to alert medical personnel that prior transfusion reactions have occurred.

On completion of a transfusion, an entry in the patient's record should indicate the type and volume of the transfusion and the presence or absence of any reaction. The progress note, the transfusion record sheet, or the transfusion laboratory slip can be used for this purpose and should be signed and dated by the clinician or nurse, in accordance with hospital policies. The bag in which the blood was stored might be discarded or returned to the blood bank, as individual policies dictate.

The clinician should emphasize to the patient and family how critically important any blood transfusion is to the patient's care. It could then be suggested that the family consider arranging for replacement donations of units of blood to afford future patients the luxury of an ample, available supply of blood products.

CONCLUSION

Transfusion therapy is an essential part of resuscitation from traumatic blood loss, as well as life-saving therapy for familial and acquired coagulopathies or bleeding disorders. Although transfusion of blood products always poses a potential risk to the patient, improvement in transfusion protocols, donor screening, and viral-reduction methods have greatly improved the safety. Nonetheless, recognition of potential risks, proper product selection, careful patient education and rigorous adherence to hospital policies and protocols continue to be essential. Future research will focus on development of blood substitutes and genetically derived blood products.

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Section V - Anesthetic and Analgesic Techniques

Chapter 30 - Local and Topical Anesthesia

Douglas McGee

Local anesthetic agents are important tools used in the everyday practice of emergency medicine. This chapter describes the mechanism of action, the nuances of clinical use, and adverse reactions to anesthetics commonly used in the emergency department (ED). Detailed technical guidance for the performance of topical and infiltrative local anesthesia is provided.

BACKGROUND

The first local anesthetic was cocaine, an alkaloid in the leaves of the *Erythroxylon coca* shrub from the Andes Mountains. Early Incan society used cocaine for invasive procedures, including cranial trephination. In 1884, Koller used topical cocaine in the eye and was credited with the introduction of local anesthesia into clinical practice.[1] In the same year, Zenfel used a topical solution of alcohol and cocaine to anesthetize the eardrum, and Hall introduced the drug into dentistry.[2] In 1885, Halsted demonstrated that cocaine blocked nerve transmission, laying the foundation for nerve block anesthesia.[3] The search for alternatives to cocaine led to the synthesis of the benzoic acid ester derivatives and the amide anesthetics used today. It was not until the 1960s that a detailed understanding of the physiochemical properties, mechanism of action, pharmacokinetics, and toxicity of these agents emerged.

PHARMACOLOGY AND PHYSIOLOGY

Chemical Structure and Physiochemical Properties

Most useful local anesthetic agents share a basic chemical structure:
Aromatic segment—Intermediate chain—Hydrophilic segment

Subtle variations of this basic structure determine each agent's main physiochemical properties: the negative log of dissociation constant (p K_a), the partition coefficient (a measurement of lipid solubility), and the degree of protein binding. Each of these properties determines the drug's potency, onset, and duration of action. Physiochemical properties are not the sole determinant of clinical activity; other factors influence the drug's effect.

The intermediate chain between the aromatic and hydrophilic segments is either an amino-ester or an amino-amide; these two chemical structures form the basis for the two main classifications of local anesthetics. *Ester*-type agents include procaine, chloroprocaine, cocaine, and tetracaine. The *amide*-type agents include lidocaine, mepivacaine, prilocaine, bupivacaine (Marcaine), and etidocaine. Different pathways metabolize each class. Esters are hydrolyzed by plasma pseudocholinesterase. Cocaine, an ester, is also partly metabolized via N-demethylation and nonenzymatic hydrolysis. Individuals with pseudocholinesterase deficiencies may have a greater potential for cocaine toxicity if large doses are used. Although this has not been an issue when cocaine is used clinically as an anesthetic. Amides are metabolized in the liver by enzymatic degradation. Local anesthetics are poorly soluble weak bases combined with hydrogen chloride to produce the salt of a weak acid. In solution, the salt exists both as uncharged molecules (nonionized) and as positively charged cations (ionized). The nonionized form is lipid-soluble, enabling it to diffuse through tissues and across nerve membranes. The ratio of nonionized to ionized forms depends on the pH of the medium (vial solution or tissue milieu) and on the p K_a of the specific agent. The pK_a is the pH at which 50% of the solution is in the uncharged form, and 50% is in the charged form. When the pH of the solution or tissue is less than the p K_a , more of the drug is ionized. When the pH increases, more of the drug is in the nonionized form. Because the nonionized form of drug can diffuse through tissues and nerves, manipulating the pH of the solution can alter a drug's diffusion properties.

Local anesthetics are available in single-dose vials or ampules and in multidose vials, with and without epinephrine. Most solutions have a pH greater than 5. Multidose vials contain methylparaben, an antibacterial preservative. Local anesthetics premixed with epinephrine also contain an antioxidant (sodium bisulfite or sodium metabisulfite) to prevent deactivation of the vasoconstrictor. These solutions must be adjusted to a more acidic pH, approximately 3.5 to 4.0, to maintain the stability of epinephrine and its antioxidant. These properties as they relate to the amide group are depicted in Table 30-1 .

Nerve Structure and Impulse Transmission

Functional and Structural Components of a Peripheral Nerve

The functional nerve unit includes the nerve axon and its surrounding Schwann cell sheath. The Schwann cell (Fig. 30-1) may surround several unmyelinated axons, or a single myelinated nerve fiber, forming a myelin sheath. Junctions between sheaths along the axon called *nodes of Ranvier* contain sodium channels necessary for depolarization. As myelin sheath thickness increases from autonomic to sensory to motor fibers, the nodes of Ranvier are spaced farther apart. The most important structure affecting nerve impulse transmission is the axon membrane (Fig. 30-2). The membrane is made of a double layer of phospholipids into which are embedded

TABLE 30-1 -- pH and Additives of Amide Local Anesthetics

Solution Content	pH (Range)	Preservative (Methylparaben)	Antioxidant
Plain, single dose	4.5–6.5	-	-
Plain, multidose	4.5–6.5	+	-
Commercial epinephrine, single dose	3.5–4.0	-	+
Commercial epinephrine, multidose	3.5–4.0	+	+
Prepared epinephrine, single dose	4.5–6.5	-	-

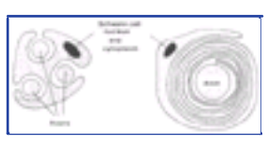

Figure 30-1 Schwann cell sheath of unmyelinated *(left)* and myelinated *(right)* nerve fibers. *(From Wildsmith JAW: Peripheral nerve and local anesthetic drugs. Br J Anaesth 58:692, 1986. Reproduced by permission.)*

protein molecules that serve as channels containing pores for the movement of ions in and out of the cell. Most pores have a filter, or gate, that controls ion-specific movement, and a sensor mechanism that opens or closes the gate. Bundles of nerve fibers (Fig. 30-3) are embedded in the *endoneurium,* which is made of collagen fibrils, and are surrounded by a cellular layer, the *perineurium*. The perineurium functions as a diffusion barrier and maintains the composition of extracellular fluid around the nerve fibers. Surrounding the entire structure is the outer layer of a peripheral nerve, the *epineurium*, composed of areolar connective tissue.

The Nerve Impulse and Transmission

At rest, the inside of a nerve fiber, or axoplasm, is negative (-70 mV) compared with the outside. This resting potential is the net result of the differences in ionic concentrations on each side of the axonal membrane and the forces that tend to maintain that difference. Specifically, there is a surplus of sodium extracellularly and of potassium intracellularly. The sodium channel is closed, preventing these ions from moving along their concentration gradient (out ? in). Although potassium can leave the cell to follow its concentration gradient (in ? out), the need to maintain electrical neutrality inside the cell prevents it from completely doing so. Potassium is in equilibrium between the concentration gradient and the electrochemical gradient, creating the negative resting potential.

The sodium channel opens when a nerve is stimulated. Sodium ions enter slowly at first until a critical threshold is reached. Sodium ions then enter the cell rapidly, along the electrochemical and concentration gradients, causing depolarization. The influx of sodium is halted when the membrane potential reaches +20 mV but potassium continues to move out of the cell, repolarizing it until the resting potential is reached. When the excitation process has been completed and the nerve cell is electrically quiet, the relative excess of sodium inside the cell and potassium outside the cell is

Figure 30-2 Axon membrane. *(From Wildsmith JAW: Peripheral nerve and local anesthetic drugs. Br J Anaesth 58:692, 1986. Reproduced by permission.)*

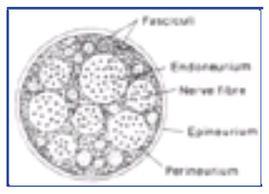

Figure 30-3 Cross section of peripheral nerve. *(From Wildsmith JAW: Peripheral nerve and local anesthetic drugs. Br J Anaesth 58:692, 1986. Reproduced by permission.)*

readjusted by the adenosine triphosphate (ATP)-dependent sodium-potassium pump.

Depolarization of a portion of the nerve causes a current to flow along the adjacent nerve fiber. This current makes the membrane potential less negative and actuates the sensor to open the next sodium channel. The action potential cycle is repeated, propagating the impulse. Nerve conduction is essentially unidirectional because the sodium channel is not only closed but inactivated as well, and delayed closure of specific potassium channels prevents the critical threshold from being reached in the segment just depolarized. An impulse spreads continuously down the axon in unmyelinated nerve fibers. In myelinated fibers, current flows from node to node causing intervening segments to depolarize at once. This saltatory conduction causes a faster rate of impulse transmission in myelinated fibers.

Mechanism of Action

How local anesthetic agents produce nerve conduction blockade depends on the active form of the agent and specific physiologic and cellular activity.

The Active Form

Anesthetic solutions contain uncharged and charged forms. The concentration of the uncharged form increases in more alkaline milieus. Only this uncharged lipid-soluble form can cross tissue and membrane barriers. Once the uncharged drug is through a barrier, the uncharged form re-equilibrates into uncharged and charged forms in a proportion dependent on the prevailing pH. Because local anesthetics are more effective in alkaline solutions, it was originally thought that the uncharged form was responsible for conduction blockade. Alkaline solutions are currently believed to be more effective because of increased penetration through tissue barriers. The cationic charged form is responsible for the actual neuronal blockade.

The Physiologic and Cellular Basis for Neuronal Blockade

The prevention of sodium influx across the nerve membrane forms the physiologic basis for conduction blockade. Local anesthetics slow sodium influx, decreasing the rate of rise and amplitude of depolarization. If sufficient anesthetic is present and the firing threshold is not reached, the action potential is not formed. With no action potential, no impulse is transmitted, and impulse conduction is blocked, resulting in local anesthesia.

The means by which anesthetic agents prevent sodium influx is still not completely understood. It is believed that the cationic charged form blocks the action potential from inside

the membrane; the agent enters the sodium channel from the axoplasmic side and binds to a receptor.[4] [5] This "specific receptor" theory is well accepted and is considered the predominant mechanism in preventing sodium influx. However, this theory cannot account for the action of benzocaine and other neutral compounds or the uncharged base forms of the common local anesthetics.

In summary (Fig. 30-4), when a local anesthetic (other than benzocaine) surrounds the perineurium, it equilibrates into its uncharged and charged forms based on the tissue pH and pK_a . The uncharged lipid-soluble form penetrates tissue, nerve sheath, and nerve membrane to gain access to the axoplasm and re-equilibrates into both forms. The charged form enters the sodium channel and decreases sodium movement into the cell thereby halting nerve transmission. The uncharged base is also involved with sodium channel blockade, but the exact nature of this mechanism is unknown.

Activity Profile during Neuronal Blockade

A local anesthetic's activity profile (onset, potency, and duration) and its ability to produce a differential blockade in mixed nerves are a function of physiochemical properties, the physiologic environment, and, to some extent, manipulation by the clinician.

Onset of Action

The pK_a of an anesthetic is the primary physiochemical factor that determines onset of action. Increased tissue penetration and shortened onset of action is found in drugs with a lower pK_a because more of the lipid-soluble uncharged form is present (Table 30-2 and Table 30-3). Although in isolated nerve fibers onset of action directly parallels p K_a , other physiochemical factors influence drug activity. For example, prilocaine and lidocaine have the same p K_a , but lidocaine's onset is faster because of its enhanced ability to penetrate through non-nervous tissue.

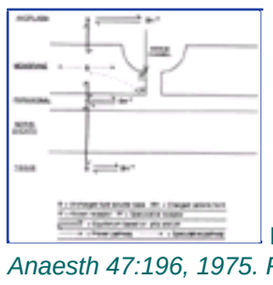
Figure 30-4 Mechanism of action of local anesthetic agents (see text for details). *(Modified from Ritchie JM: Mechanism of action of local anesthetic agents and biotoxins. Br J Anaesth 47:196, 1975. Reproduced by permission.)*

TABLE 30-2 -- Activity Profile with Primary Physiochemical Determinant

Agent	Onset: pK_a	Potency: Lipid Solubility	Duration: Protein Binding
Tetracaine	Slow	8	Long
Procaine	Slow	1	Short
Chloroprocaine	Fast	1	Short
Lidocaine	Fast	2	Moderate
Mepivacaine	Fast	2	Moderate
Prilocaine	Fast	2	Moderate
Bupivacaine	Moderate	8	Long
Etidocaine	Fast	4–6	Long

The site of administration also influences the onset of action. Onset times are prolonged as the amount of interspersed tissue or the size of the nerve sheath increases because of the greater distance that the agent must travel to reach its receptor. The pattern of onset for large nerves is determined by the structural arrangement of fibers. Peripheral (mantle) fibers are blocked before core fibers. Because mantle fibers innervate more proximal regions, nerve blockade proceeds in a

proximal-to-distal progression.

Adding sodium bicarbonate to raise the anesthetic solution's pH yields a higher concentration of the uncharged lipid-soluble form and decreases onset time. Increasing the total dose by using a higher concentration of the same volume or a greater volume of the same concentration also shortens onset time. For most procedures performed in the ED, the onset time of most agents is short enough that manipulation to achieve shorter times is unnecessary.

Potency

The lipid solubility of an anesthetic is a primary physiochemical factor determining potency. The drug's partition coefficient, not the concentration of lipid-soluble form determined by pK_a or pH, confers its lipid solubility. Because the nerve membrane is lipid, lipophilic anesthetics pass more easily into the cell and few molecules are needed for conduction blockade (see Table 30-2 and Table 30-3).

The degree of vasodilation produced by the anesthetic also affects potency since vasodilation promotes vascular absorption, reducing the amount of locally available drug. Lidocaine is more lipid-soluble than prilocaine or mepivacaine, but it produces more vasodilation. Although lidocaine is twice as potent as prilocaine or mepivacaine in vitro, it is equipotent in vivo. Although not a primary reason for its use, epinephrine, by producing vasoconstriction and making more molecules available to the nerve, increases the depth of anesthesia. Drugs more readily absorbed by fat have reduced potency. Increased concentration also increases potency. Choosing an anesthetic for its potency is usually not necessary for any given site, since the concentration of an agent may be manipulated to make most drugs equianesthetic. For example, lidocaine, being one-fourth as potent as bupivacaine, is usually used at 4 times the concentration (1% to 2% vs. 0.25% to 0.5%, respectively). For different sites and techniques, different concentrations and volumes of a given agent are needed to produce adequate blockade.

Duration

The degree of protein binding of an anesthetic primarily determines the duration of action. Agents that bind more

TABLE 30-3 -- Physiochemical Properties of Selected Local Anesthetics

Agent	Type	Site of Metabolism	pK_a	Lipid Solubility (Partition Coefficient)	Protein Binding (%)
Tetracaine	Ester	Plasma	8.5	High (4.1)	76
Procaine	Ester	Plasma	8.9	Low (0.02)	6
Chloroprocaine	Ester	Plasma	8.7	Low (0.14)	—
Lidocaine	Amide	Liver	7.9	Medium (2.9)	64
Mepivacaine	Amide	Liver	7.6	Medium (0.8)	78
Prilocaine	Amide	Liver	7.9	Medium (0.9)	55
Bupivacaine	Amide	Liver	8.1	High (27.5)	95
Etidocaine	Amide	Liver	7.7	High (141.0)	94

tightly to the protein receptor remain in the sodium channel longer (see Table 30-2 and Table 30-3). Like potency, the duration of action is reduced by the vasodilation produced by local anesthetics. Prilocaine, which is less protein-bound than lidocaine, produces a longer duration of action because of its lesser degree of vasodilation. The duration of action also varies with the mode of administration. The duration of action is shorter when agents are applied topically.

The duration of action may be prolonged by several methods. Increasing the dose, usually by increasing the concentration, prolongs duration to limits imposed by toxic effects. Although controversial, raising the pH of the anesthetic solution also has been shown to prolong duration. [6] [7] The most practical way to increase duration is to use solutions that contain epinephrine. [8] Epinephrine causes vasoconstriction, decreases systemic absorption, and allows more drug to reach the nerve. The effect of epinephrine varies according to the agent. Anesthetics that intrinsically produce more vasodilation (e.g., procaine, lidocaine, mepivacaine) benefit more from epinephrine's vasoconstrictive action. The long-acting, highly lipid-soluble agents (e.g., bupivacaine, etidocaine) are less affected because they are substantially taken up by extradural fat and released slowly. In fact, lidocaine with epinephrine may be effective as long as bupivacaine without epinephrine. Generally, most ED procedures can be accomplished quickly before anesthesia wears off regardless of which drug is selected. Choose agents with a long duration of action when the procedure is lengthy or if postoperative analgesia is desired.

TOPICAL ANESTHESIA

Local anesthetic agents may be applied topically to mucous membranes, intact skin, and lacerations. There are sufficient differences among these sites to merit a separate discussion of each one. Topical anesthesia of the eye is discussed in Chapter 64 .

Mucous Membranes

Agents and Properties

Effective anesthesia of the intact mucous membranes of the nose, mouth, throat, tracheobronchial tree, esophagus, and genitourinary tract may be provided by several anesthetics (Table 30-4). Tetracaine, lidocaine, and cocaine are the most effective commonly used agents (Table 30-5). Benzocaine (14% to 20%) is commonly used for intraoral or pharyngeal anesthesia. Prilocaine-phenylephrine (Prilophen) is another topical agent. The anesthesia produced is superficial and does not relieve pain that originates from submucosal structures.

Tetracaine solution is an effective and potent topical agent with a relatively long duration of action. It is used in concentrations from 0.25% to 1% with a recommended maximum adult dose of 50 mg. However, in an overdose, it has the disadvantage of severe cardiovascular toxicity without any preceding central nervous system stimulatory phase.

Lidocaine also is an effective topical agent that is marketed in a variety of forms (solutions, jellies, and ointments) and concentrations (2% to 10%). It is commonly employed as the 2% viscous solution prescribed for inflamed or irritated mucous membranes of the mouth and pharynx. Patient misuse by repeated self-administration can lead to serious toxicity. Topical lidocaine provides an adequate duration for most procedures, with a maximum safe dose of 250 to 300 mg.

Cocaine is an effective, but potentially toxic, topical agent applied to mucous membranes of the upper respiratory tract. Although it is an ester, hepatic metabolism occurs, as does hydrolysis by plasma pseudocholinesterase. Absorption is enhanced in the presence of inflammation. Cocaine is the only anesthetic that produces vasoconstriction at clinically useful concentrations. This major advantage is offset by its susceptibility to abuse and its toxic potential. Stimulating the central nervous system directly and blocking norepinephrine reuptake in the peripheral nervous system causes the toxic

TABLE 30-4 -- Common Local and Topical Anesthetics Used in the Emergency Department

1. Benzocaine spray will produce transient anesthesia of mucus membranes. Rarely, it can precipitate methemoglobinemia in standard doses. Anbesol is a popular over-the-counter benzocaine anesthetic for dental problems, such as teething.
2. EMLA cream (lidocaine and prilocaine) will produce anesthesia of the intact skin but it must be in place for about 60 minutes to provide significant benefit. ELA-Max is another topical lidocaine preparation with a more rapid onset of action.
3. "Magic mouthwash" contains equal parts of diphenhydramine elixir, Maalox, and 2% viscous lidocaine. Each 5 mL teaspoon contains <50 mg lidocaine. It is swished, held in the mouth for 1–2 minutes, and expectorated.
4. Viscous lidocaine (2%) may be used intraorally but repeated use may produce systemic toxicity, especially in children. Each 5 mL teaspoon contains 100 mg lidocaine. It should not be swallowed, but instead expectorated after holding it in the mouth for a few minutes. Viscous lidocaine is not useful for acute pharyngitis. Systemic narcotics are preferred if pain is severe.

TABLE 30-5 -- Practical Agents for Emergency Department Use—Mucosal Application

		Maximum Dosage*			
Agent	***Usual Concentration (%)***	***Adult (mg)***	***Pediatric (mg/kg)***	***Onset (min)***	***Duration (min)***
Tetracaine	0.5	50	0.75	3–8	30–60
Lidocaine	2–10	250–300†	3–4†	2–5	15–45
Cocaine	4	200	2–3†	2–5	30–45

*These are conservative figures; see text for explanations.
†The lower dosage should be used for a maximum safe dose when feasible.

effects. Cocaine should not be administered to patients who are sensitive to exogenous catecholamines or who are taking MAO inhibitor antidepressants. Clinical manifestations of toxicity include central nervous system excitement, seizures, and hyperthermia. Central and peripheral effects of hypertension, tachycardia, and ventricular arrhythmias may be seen. Acute myocardial infarction has been reported after topical application. [9] Cocaine is commonly used as a 4% solution with a maximum safe dose of 200 mg (2 to 3 mg/kg). Coronary vasoconstriction may occur with doses as low as 2 mg/kg applied to the nasal mucosa. Topical cocaine should be used cautiously, if at all, for patients with coronary artery disease.

Dyclonine offers advantages over other topical anesthetic agents. Dyclonine is a ketone derivative without an ester or amide linkage and may be used in patients who are allergic to the common anesthetics. Extensive experience with the topical preparation has shown it to be effective and safe. [10] Dyclonine is marketed in 0.5% and 1% solutions, with a maximum adult recommended dose of 300 mg.

Benzocaine is an ester that is marketed in its neutral form in 14% to 20% preparations (Cetacaine, Americaine, Hurricaine). Its low water solubility prevents significant penetration of the mucous membranes, reducing systemic toxicity if applied to intact mucosa. However, it is not a potent anesthetic and has a brief duration of action. It is more allergenic than other topical agents are. Benzocaine is usually dispensed in an admixture with other therapeutic ingredients and is clinically effective only at relatively high (>14%) concentrations. Benzocaine is available as a nonprescription gel and liquid (Anbesol, 6.3% to 20%). It is commonly used by dentists to produce mucosal anesthesia before intraoral nerve blocks (see Chapter 31). Adriani and Zepernick recommend this agent for lubricating catheters, airways, endotracheal tubes, and laryngoscopes and describe only one adverse reaction (methemoglobinemia) in their experience with approximately 150,000 patients. [10] Methemoglobinemia may rarely occur secondary to mucosal absorption of Anbesol used repeatedly for teething infants and after standard doses of benzocaine sprays used in endoscopic procedures. [11]

Like infiltrated anesthesia, toxic reactions to topically applied anesthetics correlate with peak blood levels achieved and not necessarily the dose administered. Systemic absorption of a topical agent is more rapid, achieving a higher level than the same dose given by infiltration (Fig. 30-5 (Figure Not Available)). *The total dose for a topical anesthetic should be considerably less than that used for infiltration at a given site.* Fractionating the total dose into three portions administered over several minutes effectively reduces peak blood levels. Inadvertent suppression of the gag reflex, combined with difficulty swallowing, may lead to aspiration, an important potential adverse reaction to topical anesthesia of the nose, mouth, and pharynx. Infections from drug solutions in multidose vials for topical anesthesia of the larynx and trachea have not been substantiated.

Technique and Precautions

A commonly used "magic mouthwash" for the topical treatment of painful gingivostomatitis in children is often prescribed by emergency clinicians and pediatricians. There is little scientific study of the preparation, and it is not available commercially, but it has been safely used for decades. It consists of equal parts of viscous lidocaine (2%), Maalox as a binder, and diphenhydramine elixir. The creamy mixture is swished around the mouth and expectorated, or painted on specific lesions with a cotton swab. Packing an area with a cotton ball soaked with this mixture may be an option. Repeated doses or swallowing of the elixir can, of course, produce systemic toxicity, so careful instruction should be given to limit use of the solution to every few hours. This combination would be theoretically less toxic than simply using topical lidocaine.

Emergency physicians often prescribe 2% viscous lidocaine (20 mg/mL) for patients with pharyngitis, stomatitis, dental pain, or other inflammatory or irritative lesions in the oropharynx. Although this is a widely used intervention, and generally safe, the common misconception that topical

Figure 30-5 (Figure Not Available) Comparison of blood levels of tetracaine obtained after rapid intravenous (IV) injection of 30 mg of tetracaine over a 30- to 60-second interval, after topical application to the pharyngeal mucous membranes, after subcutaneous infiltration, and after slow IV infusion. *(From Adriani J, Campbell D: Fatalities following topical application of local anesthetics to mucous membranes. JAMA 162:1528, 1956. Copyright 1956, American Medical Association. Reproduced by permission.)*

anesthesia is totally innocuous may result in poor patient instruction with serious consequences. Topical lidocaine is helpful for painful mouth lesions, but is of little practical value for acute pharyngitis, where systemic analgesics are a better option. Seizures and death from topical lidocaine have been described when excessive repeated doses have been administered.[12] [13] Toxic blood levels may occur because the anesthetic effect of viscous lidocaine lasts only for 30 to 60 minutes, and patients with recurrent pain may either ignore or be ignorant of the safe dosing interval of 3 hours, medicating themselves more frequently. Patients tend to increase each dose to obtain greater relief and inflammation may increase systemic absorption. In addition, painful oral lesions may last for several days.

Children are at higher risk for the rare toxicity of oral lidocaine. Compared to adults, children may exhibit increased lidocaine absorption, decreased clearance, and a longer half-life.[14] Continued medication use allows lidocaine and its major metabolites, monoethylglycinexylidide (MEGX) and glycinexylidide (GX), to accumulate. Both MEGX and GX are produced from the hepatic metabolism of lidocaine and are excreted in the urine. They possess anesthetic and antiarrhythmic activity and have central nervous system toxicity potential. Although these metabolites are less potent than lidocaine, their elimination half-lives are considerably longer. Several investigators regard MEGX and GX to be the causes of central nervous system toxicity with prolonged topical use.[13]

The length of time viscous lidocaine is retained in the mouth and whether the excess is expectorated or swallowed also affect the blood level produced. Expectorating the medication after swishing it in the mouth produces much lower blood levels than when it is swallowed. It seems logical that the most hazardous mode of administration would be to retain the solution in the mouth "until absorbed."

Clearly explain the proper way to use viscous lidocaine and inform patients *not to dose themselves ad libitum*. Note that a 2% solution contains 20 mg/mL, or 100 mg per standard teaspoon (5 mL). The recommended maximum adult dose is 300 mg (15 mL of a 2% solution) no more frequently than every 3 hours. When possible, instruct the patient to decrease the dose by using direct cotton swab applications. When gargled or swished in the mouth, limit application time to 1 to 2 minutes, and instruct the patient to expectorate excess solution. Limit use to 2 or 3 days, especially if swallowing the solution is necessary to obtain relief. Prescribe lower doses for patients at risk for decreased clearance (see Systemic Toxic Reactions later in this chapter). Doses for children are prescribed at 3 mg/kg. Because infants cannot expectorate well, do not use viscous lidocaine for minor oral irritation and teething. Recommend that no food be eaten for one hour after application because anesthesia of the oropharynx can interfere with swallowing and cause aspiration. Special note should be made of the over-the-counter availability of benzocaine, commonly used for toothaches and teething. A gel or liquid (Anbesol) is available in 6.3% to 20% formulations. When used repeatedly in the oral cavity on irritated tissue, systemic toxicity, including methemoglobinemia, may occur.

Lidocaine 4% solution can be atomized using a standard nebulizer device and inhaled by the patient before nasogastric tube insertion. This method effectively anesthetizes the nasopharyngeal and oropharyngeal tissues, easing the pain of tube insertion.[15]

Intact Skin

Agents and Uses

The stratum corneum provides a cutaneous barrier that prevents the commonly marketed aqueous solutions (acid salts) from producing anesthesia but saturated solutions of the bases of local anesthetics are effective on intact skin. When applied topically to *abraded* skin, most anesthesia agents result in peak blood levels similar to those resulting from infiltration in 6 to 10 minutes.

Lidocaine cream.

Lubens and coworkers have used 30% lidocaine cream, saturated on a gauze pad adherent to an elastic patch, for a myriad of procedures. Despite its effectiveness, safety, and painless application, the practicality of its use in an emergency setting is limited. Lubens reports an impressive list of uses, including minor operative procedures (e.g., excision of lesions, incision and drainage of abscesses), lumbar puncture, venipuncture, and allergy testing.[16]

EMLA cream, ELA-Max, and tetracaine base patch.

Various topical anesthetics have been suggested to decrease the pain of venipuncture or injections, and to provide topical anesthesia to painful skin abrasions and lesions. Most have little practical application in the ED, either because of inadequate efficacy or a delayed onset of action. These agents have been extensively studied and are safe, but they are not practical in many ED settings due to the slow onset of action. However, their activity profiles make them more applicable to emergency medicine than 30% lidocaine cream. Tetracaine base is available as a solution, gel, and as a patch preparation. It is effective in crossing the lipid-rich barrier of the stratum corneum because it is highly lipophilic. EMLA, which stands for *eutectic mixture of local anesthetics*, was approved in the United States in 1992. It contains 2.5% lidocaine and 2.5% prilocaine in a unique oil-and-water emulsion yielding 5% EMLA. The mild lipophilic and hydrophilic properties of the component drugs are greatly increased when mixed together, allowing absorption through intact skin. ELA-Max and ELA-Max5 are topical lidocaine anesthetic creams with more rapid onset of action that EMLA cream. ELA-Max is a 4% concentration while the 5% ELA-Max is marketed as an anorectal cream that may benefit painful rectal procedures. Neither product has prilocaine, found in EMLA cream, and neither has FDA approval for pain relief prior to painful injections or IV insertion, but both have such potential.[17]

Tetracaine base seems to offer the advantage of being able to obtain effective anesthesia with a shorter application time and a longer duration.[18] For both preparations, onset, depth of anesthesia, duration, and blood levels vary directly with application time, use on thinner or inflamed skin, or larger dose.[19] Both preparations exhibit a reservoir effect.[20] The drug is deposited in the stratum corneum and continues to diffuse along its concentration gradient, even after it is removed from the skin.

Tetracaine base and EMLA can be useful in the ED for providing anesthesia for many procedures: venous cannulation, venipuncture, or any needle insertion including preinfiltration anesthesia and lumbar puncture; a variety of minor surgical procedures; and anesthetizing the tympanic membrane and external auditory canal. EMLA has been used effectively for ulcer debridement.[21] It requires about 60 minutes post application for EMLA cream to provide optimal topical analgesia to the intact skin for such procedures as venipuncture. Therefore early cutaneous placement of these agents (e.g.,

over common IV sites while the patient is being triaged) is important for practical ED use.

Ethyl chloride and fluori-methane sprays.

These topical agents are often used for limited skin incisions (e.g., small abscess drainage). Many patients ask for these preparations, and there is likely a placebo effect associated with their use. These agents evaporate quickly from the skin, cooling it to the point of freezing. Anesthesia is effective and immediate, but drawbacks include its short duration (only up to 1 minute), potential pain on thawing, and possible lowered resistance to infection and delayed healing. Highly volatile ethyl chloride spray is flammable.

Technique

Lidocaine cream.

This 30% cream is saturated on a gauze pad adherent to an elastic patch and placed over the area to be injected or incised during the procedure. [16] The high concentration of anesthetic and an occlusive patch are needed to achieve effective skin penetration. The duration of action varies with the application time. A 45-minute application time is needed for most procedures. To achieve a topical anesthetic duration of 30 minutes, a 2-hour application is necessary.

Tetracaine base patch and EMLA cream.

Because both agents demonstrate a reservoir effect, the anesthesia may increase, or begin, many minutes after drug removal. A precise description of application times and duration is not possible. Tetracaine base requires a minimum of 20 to 30 minutes of application time to produce several hours of anesthesia. EMLA requires an application time of 1 to 2 hours for a reported duration of 30 minutes to several hours. Occlusive dressings seem to increase EMLA penetration whenever the cream is used. Patches are more convenient and cause no loss of effectiveness.

EMLA dosing is based on the amount of cream applied, not on the amount of anesthetic. Each gram of EMLA cream contains 25 mg of lidocaine and 25 mg of prilocaine. Dosages are given in *grams of cream*, not milligrams of anesthetic. In general, apply EMLA as a thick layer to intact skin under an occlusive dressing for about 1 hour before a procedure. A thick layer approximates 1 to 2 g applied per 10 cm^2. For minor procedures such as needle insertions, apply 2.5 g of EMLA over 20 to 25 cm^2 for at least 1 hour. For more painful procedures, apply about 2 g of cream per 10 cm^2 for at least 2 hours. The maximum application area (MAA) determines the appropriate total dose applied. Base the MAA on the patient's weight as follows: up to 10 kg, MAA = 100 cm^2; 10 to 20 kg, MAA = 600 cm^2; more than 20 kg, MAA = 2000 cm^2.

Ethyl chloride and fluori-methane sprays.

Invert the bottle 25 cm from the skin and spray a stream along the proposed incision until the area turns white and hard. Make the incision or local anesthetic injection immediately, *or during the actual spraying of the agent*, because the effect is fleeting. Some clinicians use these vapocoolant sprays to decrease the pain of an injection of a more traditional local anesthetic such as lidocaine.

Iontophoresis.

Anesthetic agents may be drawn without needles into the skin by electrical current applied through electrodes using a process called iontophoresis. Lidocaine with epinephrine administered with iontophoresis provides adequate anesthesia prior to venipuncture in pediatric patients and is superior than EMLA in providing cutaneous anesthesia. [22] [23] [24] Iontophoresis is not widely used in emergency medicine but may be another alternative to applied anesthetic agents.

Adverse Events

General adverse reactions to anesthetic agents are discussed in the Complications section later in this chapter.

Tetracaine base is quite safe with low blood concentration after proper use. In one study, approximately 25% of the patients developed cutaneous erythema at the site of application. [20] This vasodilatory effect may actually be an advantage when starting IV lines or performing venipuncture. EMLA is also quite safe. Although there is a high rate of local skin reactions, these are mild and transient, disappearing 1 to 2 hours after cream removal. Despite the reported successful use of EMLA cream on skin ulcers, Powell and colleagues described an increase in bacterial growth, infection, and inflammation when used in experimental wounds. [21] [25] Methemoglobinemia resulting from the metabolites of prilocaine may occur with EMLA. [19] The risk of clinically significant methemoglobinemia seems remote when EMLA is used properly. It is contraindicated in any infant younger than 3 months of age and in those infants between 3 and 12 months of age who are currently taking methemoglobinemia-inducing drugs (nitrites, sulfonamides, antimalarials, phenobarbital, and acetaminophen). The risk of adverse effects is increased in patients with anemia, respiratory or cardiovascular disease, and glucose-6-phosphate dehydrogenase (G6PD) or methemoglobin reductase deficiencies. Prolonged inhalation of ethyl chloride spray may produce general anesthesia, coma, or cardiorespiratory arrest. Ethyl chloride is also flammable, precluding its use with electrocautery.

Lacerations

In 1980, Pryor and colleagues reported their experience with a topical anesthetic solution (tetracaine-adrenaline-cocaine [TAC]) for wound repair. [26] The original formula, used in most subsequent studies, consists of a solution of 0.5% tetracaine, 1:2000 epinephrine (adrenaline), and 11.8% cocaine. Traditionally, anesthesia is produced by firmly applying a solution-saturated gauze pad or cotton ball directly to the laceration for 10 minutes. The resulting loss of cutaneous sensation is centered about the area of application. Gel formulations of TAC and alternative mixtures of agents promise to improve the ease and safety of topical anesthetic solutions for wound repair in the ED.

Indications and Contraindications

The use of TAC (and related topical wound anesthetics) is generally restricted to young children with wounds less than 5 cm in length in whom the delay for anesthetic application is acceptable and proper application can be assured. TAC is generally not used in structures without collateral blood supply (e.g., the digits, the tip of the nose, the pinna of the ear, the penis) because of the vasoconstrictor agents (epinephrine and cocaine) in TAC. When some wound preparation is desired before anesthesia, remove large debris and clotted blood to allow for the appropriate application of TAC and then finish wound preparation once the wound is properly anesthetized.

The advantages of TAC include painless application, no distortion of wound margins, good hemostasis, and good patient and parental acceptance in the pediatric age group. TAC appears to be less effective on the trunk and extremities than on the face and scalp and less effective than lidocaine infiltration in these areas. The 10- to 20-minute onset time

may be unacceptable in a busy ED. [27] The cocaine complicates its use because of cost and federal regulations that require storage in a locked cabinet and maintenance of separate written records of its use.

Two other often mentioned drawbacks may be more theoretical than real. Vasoconstrictor-induced higher infection rates (see Complications later in this chapter) have not been clearly demonstrated. The argument that the necessary 10-minute application period is time-consuming and takes valuable nursing time is partially offset by using the child's caretaker or adhesive paper tape alone to hold the solution in place. It is not necessary for anyone to "hold" the medicine in place when gel is used.

Agents and Effectiveness

Three clinical trials directly compared TAC with infiltrated lidocaine. Without specifying wound location, Pryor's group found equal anesthetic effect. [26] Complete

anesthesia produced by TAC ranged from 82% to 86%, compared with 83% to 92% for subcutaneous lidocaine. The remaining patients obtained partial anesthesia. Hegenbarth and Anderson demonstrated results similar to Pryor. [28] Hegenbarth found TAC to be equal to lidocaine only on the face and scalp, and inferior to it at other locations. Other investigators using different study designs found TAC effectiveness to be equal to or slightly less than seen in earlier studies. [29] [30] [31] More recent studies confirm excellent rates of effectiveness, especially on the face and scalp. [32] [33] TAC is more effective than its component drugs alone. On the face and scalp, TAC was found to be superior to tetracaine alone, although on nonfacial areas, both produced equally poor results. [29] TAC was found more effective than cocaine alone and more effective than a tetracaine-epinephrine solution in the same dosage ratio. [31] [34]

In 1990, Bonadio and Wagner showed that an epinephrine-cocaine solution (epinephrine 1:2000, cocaine 11.8%) was equal to TAC in effectiveness. [32] Bonadio and Wagner also found that half-strength TAC (tetracaine 0.25%, epinephrine 1:4000, cocaine 5.9%) achieved excellent results for dermal lacerations of the face, lip, and scalp.[30] Smith and Barry compared three strengths of TAC and found equal effectiveness among them, recommending the lowest strength cocaine formulation (tetracaine 1%, epinephrine 1:4000, cocaine 4%). [35] Ernst and colleagues found similar effectiveness to TAC using a slightly different lidocaine-epinephrine-tetracaine solution (lidocaine 4%, epinephrine 1:2000, tetracaine 1%). [36]

TAC has also been compared with EMLA placed in a wound for 60 minutes prior to wound repair. In a study of 32 wounds, supplemental anesthesia was required less often with EMLA.[37] The non-cocaine-containing formulations are generally considered less toxic and have advantages in terms of reduced cost and avoidance of controlled substance precautions during storage. Schilling found that a lidocaine-epinephrine-tetracaine (LET) solution (lidocaine 4%, epinephrine 1:1000, tetracaine 0.5%) was as effective as TAC.[38] LET gel preparations are at least as effective as LET solutions. [39] Singer and Stark determined that EMLA or LET gel placed in a wound on presentation and prior to infiltration with lidocaine reduced the pain of infiltration, allowing for essentially painless injection of lidocaine. [40]

Application and Dosage

Because the topical mixtures noted earlier (especially TAC) are not innocuous anesthetics, pay close attention to the technique of application and recommended maximum dose. There is no uniformly accepted application technique, component composition, or component concentrations.

Generally, apply topical solutions such as TAC to the wound in a gravity-dependent position, carefully filling the wound cavity. After 3 minutes, place a single 2-cm × 2-cm gauze pad or cotton ball saturated with TAC to the wound. Tape or hold the pad firmly for 15 to 20 minutes. The person holding the gauze should wear latex examination gloves to minimize the risk of cutaneous absorption of the solution. The average dose of TAC solution is 2 mL.

Hegenbarth and colleagues estimated the maximum safe dose of full-strength TAC to be 0.09 mL/kg based on the known maximal safe dose of infiltrative tetracaine and mucosal application of cocaine and an estimate of solution absorption onto the applicator. [28] The key to safety is to avoid TAC on mucosal surfaces or in areas in which sniffing or swallowing may accidentally occur. Prilophen has been compared with infiltrated lidocaine for the repair of intraoral lacerations. Although the lidocaine was slightly better, little difference was demonstrated in clinical anesthesia and pain. [41] Prilophen (prilocaine-phenylephrine) may be an acceptable alternative to TAC when lacerations are repaired in close proximity to mucous membranes. Topical mucosal anesthesia is discussed elsewhere in this chapter.

One can prepare an epinephrine-cocaine gel by adding 0.15 g of methylcellulose to 1.5 mL of epinephrine-cocaine solution. Stir the mixture thoroughly for a minute or two until a gel consistency is obtained. After sterile preparation, place the wound in a gravity-dependent position apply the gel by a cotton-tipped swab to coat the entire wound cavity and margins. Allow the wound to stand for 15 to 20 minutes and thoroughly wash the wound cavity to remove the gel. In Bonadio and Wagner's study, the average dose used was 0.35 mL of gel containing only 40 mg of cocaine. [33]

Adverse Events

Mucosal application may rarely lead to significant systemic toxicity; fatalities have been reported following application. Even after nonmucosal TAC use, cocaine levels appear in the blood (tetracaine does not) and cocaine metabolites appear in the urine in the majority of patients. [42] [43] Gel formulations of TAC tend to stay in the wound and reduce the risk of solution runoff onto mucosal surfaces. There is no need to use a gauze pad to apply the medication in gel formulation or to hold it in place. Since the entire applied dose will stay in the wound, a lower dose can be used. Gel also provides a more uniform application to tissues, improving anesthetic effect. Ischemia caused by vasoconstrictors in tissues containing end arteries may occur. Avoid TAC on the digits, the tip of the nose, the penis, and the pinna. Use TAC with caution on patients with coronary artery disease, uncontrolled hypertension, seizures, and peripheral vascular disease. Patients with decreased plasma cholinesterase levels are at increased theoretical risk for systemic toxic effects, but this potential risk is of little clinical concern.

INFILTRATION ANESTHESIA

The injection of an anesthetic agent directly into tissue prior to surgical manipulation is known as infiltration anesthesia. Field block anesthesia is also considered a form of infiltration

TABLE 30-6 -- Maximum Allowable Volume (Adults)

Agent	Concentration (%)	Maximum* Safe Dose (mg)	Maximum Volume (mL)
Lidocaine	0.5	300	60
	1	300	30
Bupivacaine†	0.25	175	70
Lidocaine-epinephrine	0.5	500	100
	1	500	50
Bupivacaine-epinephrine	0.25	225	90

*These are conservative figures; see text for explanation.
†Some physicians recommend 400 mg as the maximum safe dose for bupivacaine.

anesthesia, particularly because the agents, concentrations, and recommended maximum dosages are the same. A field block is created when a field of anesthesia is injected around the operative site. The injection is made proximal to or surrounding the area to be manipulated.

Indications and Contraindications

Infiltration anesthesia is indicated when good operative conditions can be obtained by using this technique. It may be used for the majority of minor surgical procedures such as excision of skin lesions, incision of abscesses, and suturing of wounds. Infiltration anesthesia is considered quicker and safer than nerve block and general anesthesia. Local infiltration can provide hemostasis, both by direct distention of tissue and by the concurrent use of epinephrine.

The major disadvantage of local infiltration compared with nerve blocks is that a relatively large dose of drug is needed to anesthetize a relatively small area. For extensive wounds, the amount of anesthetic required may risk systemic toxicity. The maximum allowable volume can be increased by adding epinephrine, by using a lower concentration of anesthetic agent, or by doing both (Table 30-6). When large volumes are anticipated and a nerve block is anatomically feasible, the nerve block is preferred. Infiltration is avoided for large procedures in small children and in apprehensive patients, especially those with prior adverse reactions (whether vasovagal or otherwise). Local infiltration distorts the tissues that will be incised or repaired, making it undesirable in areas requiring precise anatomic alignment (e.g., some lip repairs).

TABLE 30-7 -- Practical Agents for Emergency Department Use—Local Infiltration

		Maximum Dose*,†			
Agent	***Concentration (%)***	***Adult (mg)***	***Pediatric (mg/kg)***	***Onset (min)***	***Duration‡***
Procaine	0.5–1.0	500§ (600)	7 (9)	2–5	15–45 min
Lidocaine	0.5–1.0	300 (500)	4.5 (7)?	2–5	1–2 hr
Bupivacaine	0.25	175 (225)	2 (3)¶	2–5	4–8 hr

*These are conservative figures; see text for explanation.
†Higher maximum dose for solutions containing epinephrine is in parentheses.
‡These values are for the agent alone; they can be extended considerably with the addition of epinephrine.
§Some authorities recommend up to 1000 mg or 14 mg/kg for procaine.
?Some authorities recommend up to 7 mg/kg for plain lidocaine in children older than 1 year.
¶Because of lack of clinical trial experience, drug companies do not recommend the use of bupivacaine in children under age 12, but bupivicaine is commonly used in children without problems.

Choice of Agent

Local anesthetic agents most frequently used for infiltration are 0.5% to 1% lidocaine, 0.5 to 1% procaine, and 0.25% bupivacaine (Table 30-7). Higher concentrations are of *no* additional benefit. Lidocaine is most commonly used because of its excellent activity profile, low allergenicity and toxicity, user familiarity, and ready availability. Procaine is useful for patients who are allergic to amide anesthetics. Some clinicians prefer bupivacaine because of its prolonged duration. Bupivacaine may be preferred when postoperative analgesia is desired, for prolonged procedures, or even for short procedures that may be interrupted in a busy ED.

A comparison of equianesthetic doses of lidocaine and bupivacaine for infiltration anesthesia (Table 30-8) reveals the duration of action to be the major difference between the two agents. For the majority of ED procedures, it is not necessary to extend the duration of anesthesia beyond 1 hour. Plain lidocaine would seem to be a logical anesthesia choice. Patients experience a moderate amount of pain after laceration repair when the lidocaine wears off in about 1 hour. [44] Bupivacaine reduces the pain following laceration repair for at least 6 hours. This benefit of a prolonged duration of anesthesia must be weighed against the hazards of injury to an unprotected limb or the annoyance of prolonged numbness to patients who have had simple surgical procedures.

A prolonged duration of anesthesia can be achieved by adding epinephrine, sodium bicarbonate, or both to lidocaine. Epinephrine provides excellent wound hemostasis and slows systemic absorption. This latter property decreases the peak blood level, decreasing the potential for a toxic reaction and allows a greater volume of agent to be used for extensive lacerations. The major disadvantage of epinephrine is the theoretical, but generally clinically inconsequential, damage to host defenses (Table 30-9). Added bicarbonate decreases the pain of administration, but such an addition is usually made at the bedside. Bupivacaine, if used with due caution, is safe and easy to use. The deciding factors are many, but some logical choices are as follows:

- For a wound with excessive bleeding: lidocaine with epinephrine and sodium bicarbonate
- For an apprehensive patient: lidocaine with sodium bicarbonate
- For anticipated prolonged postprocedure pain: bupivacaine

TABLE 30-8 -- Comparison of 1% Lidocaine (L) and 0.25% Bupivacaine (B)—Infiltration Anesthesia

	Lidocaine	Bupivacaine	Advantage
Onset	2–5 min	2–5 min	Equal
Effectiveness (equianesthetic dose)	Excellent	Excellent	Equal
Duration	1–2 hr	4–6 hr	B
Infection potential	No	No	Equal
Administration pain	Less	More	L
Maximum volume* —plain lidocaine	Less	More	B
Maximum volume—epinephrine	Less	More	B
Toxic potential	Less cardiotoxic; equal CNS	More cardiotoxic; equal CNS	L

CNS, central nervous system.

*See Table 30-6 for volume and concentration comparison.

Equipment

The pain of injection is reduced by use of small-gauge needles. Ideally, a 30-gauge needle is used if injection is made through the skin. If the injection is made through the cut edges of the wound, a 25- to 27-gauge needle suffices. A small-gauge needle slows the rate of injection and reduces the rate of tissue distention. A 10-mL syringe is recommended both for its ease of handling and for the relatively slow rate of injection it allows.

Technique

Once an agent has been chosen, proper administration technique minimizes pain, prevents bacterial spread, and avoids intravascular injection. Buffering, warming, and careful infiltration reduce the pain of injection.

Buffering

Lowering the pH of an anesthetic by adding epinephrine increases pain, whereas raising the pH by adding sodium bicarbonate decreases pain dramatically. It is probable that pH is not the sole factor in producing pain, since the pain produced by various agents does not correlate strictly with the pH. Sodium bicarbonate probably works by increasing the ratio of nonionized to ionized molecules, which either renders the pain receptors less sensitive or causes a more rapid diffusion of solution into the nerve and a shorter time to anesthetic onset.

To alkalinize lidocaine, add 1 mL of sodium bicarbonate (8.4%, 1 mmol/mL) to every 10 mL of anesthetic solution. As the pH of the solution is raised, the anesthetic becomes unstable and has a decreased shelf life. It was initially recommended that buffered lidocaine be prepared just prior to use to

TABLE 30-9 -- Epinephrine Use

Advantages	Disadvantages
1. Prolongs duration	1. Impairs host defenses—increases infection*
2. Provides hemostasis	2. Delays wound healing*
3. Slows absorption:	3. Do not use for:
Decreases agent toxic potential	Areas supplied by end arteries
Allows increased dose	Patients "sensitive" to catecholamines
4. Increases level of blockade	4. Toxicity—catecholamine reaction†

*Based on laboratory studies and of unknown clinical importance.
†For example, in patients taking MAO inhibitors.

avoid precipitation and degradation, but buffered lidocaine retains its effectiveness for 1 week and refrigeration may further increase its shelf life. [45] [46] Bicarbonate may be combined with plain lidocaine for both infiltrative anesthesia [47] [48] and digital nerve blocks.[49] In one volunteer study, sodium bicarbonate was effectively combined with lidocaine and epinephrine. [46]

Sodium bicarbonate can be added to bupivacaine, but the solution tends to precipitate as the pH rises. Precipitation varies directly with the concentration of bupivacaine and the time since mixture.[50] Cheney showed that 0.05 mL of 8.4% sodium bicarbonate (measured in a tuberculin syringe) could be mixed with 10 mL of 0.5% bupivacaine without precipitation.[51] Since the goal of using bupivacaine is to prolong the duration of anesthesia, and this effect can also be accomplished somewhat by using buffered lidocaine (plain or with epinephrine), it seems impractical to risk precipitation by "buffering" bupivacaine.

Warming

Warming an anesthetic to body temperature (37°C to 42°C) reduces the pain of infiltration. [52] [53] Bartfield found that lidocaine warmed to 38.9°C was more painful than room temperature buffered lidocaine during intradermal injection.[54] Brogan, using lidocaine warmed to 37°C, found the warmed lidocaine and room temperature buffered lidocaine to be equivalent during wound infiltration. [55] Neither study found a synergistic effect with combined warming and buffering. Martin found warmed (37°C) lidocaine to be no less painful than buffered lidocaine. [56] Anesthetic solutions can be warmed in a baby food warmer with thermostatic temperature control or in an IV solution warmer. Warming is not believed to adversely affect shelf life of the local anesthetic.

Injection

Place the injection in the subdermal tissues to minimize needle puncture pain and the tissue distention that occurs with intradermal placement. Placing the needle "up to the hub" and injecting while withdrawing along the just-created subdermal tunnel also minimizes tissue distention. After an initial injection, instead of totally withdrawing the needle from the tissue, redirect it along another path to lessen the number of punctures. Slowly injecting the smallest volume necessary reduces pain.

Because the patient barely feels a needle placed subcutaneously and skin puncture is often quite painful, *make all wound injections through the wound edge and not through the skin* (Fig. 30-6).[57] Infection spread beyond the wound margin has not been demonstrated clinically with this technique. Some clinicians may choose to

inject the anesthetic through intact skin in a grossly contaminated wound. Bierman described a technique of patient distraction by applying light pressure to alternate sides of the wound with one's fingers and repeated ambiguous questioning about feeling the light pressure rather than the ongoing wound injection. [58] School-age children can be asked to count backward or say the "ABCs" to distract them during injection.

Preventing a systemic toxic reaction is best accomplished by avoiding an intravascular injection. However, for infiltration anesthesia with small-gauge needles, aspiration is usually unnecessary unless the injection is deeper than the subcutaneous (SQ) area or the area to be injected contains many large vessels.

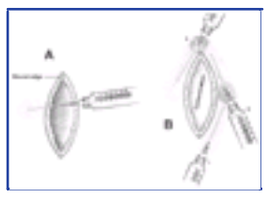

Figure 30-6 *A,* Except in the setting of gross contamination, wounds should be anesthetized by inserting the needle through the cut edges, not through the intact skin. Patients often will not feel a 25-gauge or smaller needle passed into the subcutaneous tissue when it is advanced slowly through the cut edge. However, pain generally occurs with tissue distention by the anesthetic, and hence, injection should be slow and deliberate. *B,* If a wound is grossly contaminated, the anesthetic may be introduced through the intact skin. The operator should limit the number of needle sticks. The needle is first introduced at a point in line with the wound and beyond the wound edge (*1*), and while the anesthetic is slowly injected, the needle is advanced to include 1 entire side of the wound (if possible) to a point well past the opposite end of the wound. The other side may be anesthetized by passing the needle through the area already infiltrated by the first injection (*3*), making the skin puncture painless. A 3.8-cm (1.5-in.) 27-gauge needle is a good choice. If the needle is not long enough to encompass the entire wound, the skin is painlessly punctured at a midway point that has already been anesthetized (*2*).

SPECIAL CONSIDERATIONS

Hematoma Block

Hematoma block has been used for many years to provide anesthesia for reduction of fractures, particularly of the distal forearm and hand. Its popularity has waned somewhat because of the fear of introducing infection at the fracture site and its limited efficacy. Although several studies show the hematoma block to be safe, the anesthesia it provides is not as good as that provided by the Bier block (see Chapter 33). Nevertheless, there are several reasons to consider this technique. [59] [60] The procedure is simple and quick to perform and does not require additional personnel. There is no need to wait for an anesthesiologist or for the patient to digest a meal. A lower dose of anesthetic agent is required compared with that required for the Bier block (see Chapter 33). A hematoma block is useful when the Bier block and general anesthesia are contraindicated.

Prepare the skin over the fracture site with Betadine and insert the needle into the hematoma (confirmed by aspirating blood). Slowly inject from 5 to 15 mL of plain 1% lidocaine or 5 to 10 mL of plain 2% lidocaine (larger fractures require larger volumes of local anesthetic) into the fracture cavity and around the adjacent periosteum. Adequate anesthesia occurs in about 5 minutes and may last for several hours. Do not perform this procedure through dirty skin, in open fractures, or in small children.

Intra-articular Anesthesia

The history and physical examination of an acutely traumatized joint, such as the knee often underestimates the severity of an injury. Instillation of 5 mL of 1% lidocaine after joint aspiration may help relieve pain and facilitate an examination, but its use is not routinely recommended. [61] Spasm and apprehension are often not relieved by local anesthesia, and the information gained usually does not influence the ED treatment plan. Intra-articular anesthesia of the knee has no effect on gait pattern or joint proprioception. [62] Post-procedure weight bearing may be allowed without fear of producing or increasing injury if otherwise indicated. Intra-articular anesthesia may enhance elbow use following aspiration of a hemarthrosis associated with a radial head fracture. [63] The technique of administration is analogous to arthrocentesis (see Chapter 54).

Intrapleural Anesthesia

Indications

Intrapleural anesthesia introduces local anesthetic into the pleural space (i.e., between the parietal and visceral pleura) through an epidural catheter. The anesthetic can be introduced through a previously placed chest tube. The technique can provide relief for several conditions, primarily post-thoracotomy pain; post-cholecystectomy pain; and, most importantly for emergency clinicians, posttraumatic chest pain (i.e., rib fractures, pneumothorax, hemothorax). This procedure is useful not only for pain relief, but also facilitates turning, coughing, and deep breathing. Although not unanimous, most studies show that intrapleural anesthesia is effective in providing analgesia. [64] [65] Concern has been raised that intrapleural anesthesia may create a level of anesthesia below the umbilicus and make posttraumatic abdominal examinations unreliable. Until this issue is clarified, it seems prudent to rule out intra-abdominal injury before intrapleural anesthesia is used. [66]

Technique

If a chest tube is in place, it is preferable to inject anesthesia into the pleural space through the chest tube. Theoretically, the tube should be clamped for 10 to 15 minutes to allow the anesthetic to diffuse. When the tube cannot be taken off suction, or if no tube is present, the local anesthetic is injected percutaneously. [67] With the patient in the lateral position (with the affected side up), place a 16-gauge Tuohy needle 8 to 10 cm from the posterior midline in the 8th intercostal space. The needle is angled at 30° to 40° to the skin, aimed medially, bevel up, and directed just above the rib. After perforating the posterior intercostal membrane (felt as a distinct resistance), remove the stylet and attach a well-wetted, air-filled glass syringe to the Tuohy needle. Advance the needle until it enters the pleural space, denoted by the plunger being drawn down the syringe due to the negative pressure created during inspiration. Remove the syringe and introduce an epidural catheter 5 to 6 cm into the pleural space. Remove the Tuohy needle, obtain a chest radiograph to confirm proper position, and secure the catheter.

The most commonly used anesthetic and dose is 20 mL of 0.5% bupivacaine. A repeat dose every 8 hours has been shown safe. [68] The solution presumably diffuses from the pleural space "back" through the parietal pleura and the intercostal muscle to reach the intercostal spaces, where it blocks the intercostal nerves. The level of anesthesia can extend from T2 to T12 and involve skin, chest, abdominal wall, and potentially the viscera if the visceral afferent fibers are blocked at the sympathetic chain in the paravertebral gutter.

Although not yet a consistently proven or a completely standardized technique, intrapleural anesthesia offers promise for patients and is a potentially valuable procedure for the emergency clinician.

COMPLICATIONS

Local Anesthetic Effect on Wounds

Wound Healing

Local anesthetics produce cytotoxic effects on cell structure and function in a dose- and time-related manner. These effects, at doses well below those used clinically, involve fibroblasts more than nervous tissue. Collagen synthesis is inhibited by lidocaine and bupivacaine. [69] Morris and Tracey found that lidocaine in increasing concentrations progressively reduced the tensile strength of wounds. [70] Epinephrine added to 1% and 2% concentrations of lidocaine further reduced tensile strength, but when epinephrine was added to distilled water or to 0.5% lidocaine, it had little effect. Several conclusions may be clinically relevant. Although it may delay anesthetic onset, lidocaine 0.5% solution, without epinephrine if possible, may be best for wound strength.

Eriksson and colleagues found that lidocaine reduces the inflammatory response in wounds by decreasing the number of white cells and their metabolic activity. [71] While an inflammatory response may be beneficial in a contaminated wound, it can be detrimental in a sterile wound because of the tissue toxicity that is created by the release of superoxide anions, lysosomal enzymes, thromboxanes, leukotrienes, and inter-leukins. The clinical relevance of this is unknown. None of the concerns mentioned earlier should prohibit the use of standard anesthetics or epinephrine when their use is otherwise indicated.

Wound Infection

Although not generally appreciated, it has long been known that local anesthetics possess antimicrobial activity in vitro. Lidocaine and procaine at 2% concentrations inhibit the culture growth of most gram-negative organisms, with only *Pseudomonas aeruginosa* being particularly resistant. [72] [73] Gram-positive isolates are also significantly affected by lidocaine and, to a lesser extent, by procaine. Administering anesthetics before obtaining culture material, including injecting a joint prior to arthrocentesis, may give false-negative culture results and is avoided if possible. This effect may also be significant when anesthetic ointments have been applied prior to culture. Furthermore, it has been shown that adding sodium bicarbonate to lidocaine greatly enhances its inhibitory effect on bacteria. [74] Although local anesthetics can interfere with culture testing, several studies show that, by themselves, local anesthetics do not appear to alter the incidence of wound infection. [75] [76]

Epinephrine appears to exert a deleterious effect on host defenses, at least in animal models. Studies with infiltrated and topically applied epinephrine solutions in contaminated animal wounds show an increased potential for infection. [75] [76]

Epinephrine-induced vasoconstriction may contribute to tissue hypoxia, retarding the killing of *Staphylococcus aureus* by leukocytes, and reducing leukocyte migration into the tissue. [77] Most clinical studies using topical anesthesia with vasoconstrictor properties (e.g., tetracaine, epinephrine, and cocaine mixtures) do not demonstrate significantly increased infection rates. [26] [28] [34] [78] The concerns mentioned earlier should not prohibit the use of epinephrine for wound preparation when its use is otherwise appropriate.

Local Injuries

Injuries may result from direct application of an anesthetic agent to a nerve or from passage of a needle through soft tissue structures. Factors implicated in transient or persistent neuropathy include acidic solutions, additives, the agent itself, needle trauma, compression from hematomas, and inadvertent injection of neurolytic agents. Born described a series of 49 wrist and metacarpal blocks using bupivacaine in which 8 patients developed a significant neuropathy. [79] He postulated that damage occurred from the trapping of the drug in a confined space and recommended that whenever bupivacaine is used in this situation, it be used in low concentration and volume. Infection, hematomas, and broken needles are other local problems that can be averted by using proper technique. Erroneous needle placement also can produce complications such as pneumothorax during brachial plexus or intercostal block.

It is commonly stated that epinephrine-containing solutions injected into tissues containing end arteries can cause profound ischemia and gangrene. Given the short duration of epinephrine combined with the vasodilation that accompanies many local anesthetics (e.g., lidocaine), this cause and effect is difficult to accept and hard to substantiate. Despite admonition in most textbooks, the concept remains largely theoretical. Areas of special concern include the digits, penis, tip of the nose, or earlobe. This concern is promulgated but is certainly overrated when dilute concentrations of epinephrine-containing solutions are used (1:200,000). Some authors state that small quantities of such solutions are usually tolerated on the nose and pinna but advise against their use in the fingertips. [80] Because bupivacaine can be used when prolonged anesthesia is required, and because tourniquets can be used in the digits, there is little need for vasoconstrictors in the digits, although some clinicians use a dilute epinephrine solution there, as well. The dogmatic admonition against the use of epinephrine containing anesthetics in the digits is not supported by scientific data in the context of ED laceration repair. Denkler performed an extensive review of the literature back to 1880 and identified only 48 cases of digital gangrene after digital block. Only 21 cases involved the use of epinephrine, clearly raising the question of causation. Other variables including hot soaks, tourniquets, and infection made it impossible to clearly link gangrene to the epinephrine alone. [81] Other authors have demonstrated the safe use of epinephrine containing anesthetics in digital blocks, suggesting the risk of tourniquets could be minimized if epinephrine was used instead. [82] [83] Despite a lack of evidence against its use, most clinicians tend to avoid the use of epinephrine containing anesthetics when performing digital anesthesia.

The use of phentolamine (Regitine), which produces postsynaptic a-adrenergic blockade, is recommended for vasoconstrictor-induced tissue ischemia. This medication is usually given by local infiltration in a dose of 0.5 to 5.0 mg diluted 1:1 with saline. If local infiltration is ineffective or limited by tension within a tissue compartment, or if the area of vasoconstriction is large, phentolamine may be given by the intra-arterial route. [84]

Systemic Toxic Reactions

Although they occur in only 0.1% to 0.4% of local anesthetic administrations, systemic toxic reactions are the most frequent serious adverse reactions encountered (Table 30-10). [85] After administration of a local anesthetic, some of the drug reaches its intended target, and some is absorbed quickly into the systemic circulation. Peak blood levels are generally produced within 30 minutes. Many vagal reactions, nonspecific anxiety reactions, and sensitivity to preservatives have been attributed to "allergies" or systemic toxicity to local anesthetics. Patients may also demonstrate systemic reactions to hidden allergens that may mimic a systemic reaction, such as anaphylactic reactions to the latex in surgical gloves.

High Blood Levels

Systemic toxic reactions result from high blood levels of local anesthetic. Several factors are important in producing high blood levels, including site and mode of administration, rate, dose and concentration, addition of epinephrine, specific drug, clearance, maximum safe dosage, and inadvertent intravascular injection.

Site and mode of administration.

Comparing the routes of administration for a given dose, the intravascular route produces the highest levels, followed by topical mucosal application, then infiltration (see Fig. 30-5 (Figure Not Available)). The more vascular the site, the more systemic absorption that occurs and the higher the level obtained. The following blocks are arranged in decreasing order of systemic absorption: intercostal, caudal, epidural, brachial plexus, and subcutaneous. It follows that the site of administration is an important variable in determining the safe dose of an anesthetic. For example, lidocaine 400 mg may produce a nontoxic blood level with abdominal wall subcutaneous infiltration, but when used for an intercostal nerve block, a toxic level would likely result from this dose.

Rate.

A more rapid IV injection will produce a higher blood level than a slower injection. A single topical application leads to a higher level than a dose that is fractionated over time.

Dose and concentration.

The larger the total dose, the higher the peak blood level. It is uncertain whether increasing the concentration while maintaining the total dose by decreasing the volume affects the serum level.

Addition of epinephrine.

Epinephrine produces vasoconstriction and reduces systemic absorption, thereby resulting in lower peak blood levels. Occasionally, the apprehension, tachycardia, or palpitations induced by epinephrine can be incorrectly interpreted by both clinician and patient as an "allergic" reaction.

Specific drug.

The more potent agents are more toxic on a milligram-to-milligram basis. Because anesthetics are used in equipotent doses (e.g., 1 mg bupivacaine versus 4 mg lidocaine), they are approximately equitoxic. Blood levels achieved by a particular agent depend on the agent's absorption, distribution, and clearance from the circulation. Agents with high lipid solubility and lower protein binding

TABLE 30-10 -- Differentiating Systemic Adverse Reactions

Findings	Toxic Reactions	Allergy	Vasovagal	Excess Catecholamines, Anxiety[1] (Endogenous), Vasoconstrictor (Exogenous)
Relatively specific signs and symptoms	Metallic taste	Acute rhinitis	Syncope*[2]	Headache
	Tongue numbness	Pruritus*		Hypertension*
	Drowsiness	Dermatitis		Palpitations
	Nystagmus	Urticaria*		Apprehension*[3]
	Slurred speech	Facial swelling		
	Seizures*	Laryngospasm		
	Coma	Bronchospasm*		
	Respiratory arrest*			
Overlapping signs and symptoms	Paresthesia	Lightheadedness	Lightheadedness	Paresthesia*
	Lightheadedness	Tachycardia*	Tinnitus	Lightheadedness*
	Tinnitus	Hypotension*	Tachypnea	Tremor
	Tremor	Cardiac arrest	Tachycardia (early)	Tachypnea*
	Tachypnea	Nausea and vomiting		Tachycardia*
	Tachycardia (early)		Bradycardia*	Nausea and vomiting
	Bradycardia*	Dyspnea	Hypotension*	Dyspnea
	Hypotension*		Diaphoresis	Diaphoresis
	Cardiac arrest			

1. Anxiety reaction, including hyperventilation syndrome.
*Denotes common and significant reactions:
2. Vasovagal syncope occurs with patient upright; any loss of consciousness in the recumbent position implies a severe toxic or anaphylactic reaction.
3. Although apprehension is classically associated with anxiety and vasoconstrictor reactions, milder toxic and allergic reactions may cause patient apprehension.

(etidocaine > bupivacaine > lidocaine > mepivacaine) tend to become sequestered in tissue and have a slower absorption and lower blood levels. Agents with a greater volume of distribution or a faster clearance (etidocaine > lidocaine > mepivacaine > bupivacaine) also produce lower blood levels. Together these effects produce margins of safety for each anesthetic, with etidocaine having the greatest safety margin, followed by bupivacaine, which is equal to or better than lidocaine.

Esters are difficult to measure in the blood because of their rapid hydrolysis by pseudocholinesterase. As a group, toxicity is inversely proportional to the rate of hydrolysis. Tetracaine is slowly hydrolyzed and is most toxic. Chloroprocaine is quickly hydrolyzed and is least toxic. Procaine falls between the two.

Clearance.

The liver metabolizes amides where the clearance rate is a function of hepatic blood flow and extraction capacity of the liver. Decreased hepatic blood flow, produced by norepinephrine, propranolol, or general anesthesia, slows clearance and potentially raises drug blood levels. Decreased drug extraction, associated with congestive heart failure, cirrhosis, or hypothermia, may produce a higher blood level. Hypovolemia, which decreases hepatic flow, does not raise blood levels because it causes an offsetting decrease in absorption.

Decreased clearance of esters and an increased risk for toxicity occurs in patients with either low levels of pseudocholinesterase or an atypical form of pseudocholinesterase. Low levels occur in various disease states, including severe liver disease and renal failure, and in pregnancy. Atypical pseudocholinesterase is an inherited trait, and its presence reduces the hydrolysis rate of procaine to a greater extent than low levels do.

There are significant differences between pediatric and adult drug distribution and metabolism. Neonates exhibit both reduced levels of pseudocholinesterase and reduced hepatic metabolism, increasing the risk of toxicity. In older children, the effects of increased hepatic metabolism and a relatively larger volume of distribution increase their tolerance for higher doses.

Maximum safe dosage.

The *maximum safe dose* for a drug may be defined as the dose that produces a blood level of the drug just below the toxic level (Table 30-11). One maximum dose for an anesthetic agent appropriate for all patients and all conditions cannot be stated. A maximum safe dose cannot be based solely on the weight of a patient. In an adult, peak blood levels do not correlate well with weight, because the volume of the drug distribution is relatively constant. [86] [87] As an approximation, Arthur and McNicol have recommended maximum dosages for children based on weight.[88] Plain lidocaine may be used in doses of up to 4.5 mg/kg and the addition of epinephrine allows for a maximum dose of 7 mg/kg. Bupivacaine is not recommended for children under the age of 12 years; although it is commonly used without adverse consequences. Furthermore, the dose should be modified according to the site and mode of administration.

Maximum safe doses as stated in package inserts should be used only as guidelines because most of them are derived from animal experiments and are based on absorption data only. Levels vary with administration site, use of a vasoconstrictor, and to some extent, the health of the patient. Levels can often be exceeded safely when the drug is accurately administered. Drugs may be toxic even within the "safe range" when inadvertently injected intravenously.

Inadvertent intravascular injection.

Most toxic reactions are caused by inadvertent intravenous (IV) injections of anesthetics whose doses were calculated for their intended extravascular sites. For example, lidocaine 300 mg is a safe infiltrated dose that would likely cause toxicity if directly injected into the bloodstream.

Anesthetics that are injected intravascularly must pass through the lungs before they reach other organs. Lung tissue sequesters a significant amount of drug, lowering the arterial blood concentration. Anesthetics that bypass the lungs, in cases of inadvertent injection into the carotid or vertebral arteries or in patients with intracardiac right-to-left shunts, can produce central nervous system toxicity at low doses.

TABLE 30-11 -- Calculation of Anesthetic Doses

Anesthetic solutions are marketed with drug concentration expressed as percentages (e.g., bupivacaine 0.25%, lidocaine 1%). To ascertain the strength of a solution in milligrams per milliliter, consider the following:	
A 1% solution is prepared by dissolving 1 g of drug in 100 mL of solution.	
Therefore, 1 g/100 mL = 1000 mg/100 mL = 10 mg/mL.	
To calculate the strength from the percentage quickly, simply move the decimal point 1 place to the right. Examples:	
0.25% = 2.5 mg/mL	(e.g., bupivacaine)
0.5% = 5 mg/mL	(e.g., tetracaine)
1% = 10 mg/mL	(e.g., lidocaine)
2% = 20 mg/mL	(e.g., viscous lidocaine)
4% = 40 mg/mL	(e.g., cocaine)
5% = 50 mg/mL	(e.g., lidocaine ointment)
20% = 200 mg/mL	(e.g., benzocaine)
When combined in an anesthetic solution, epinephrine is usually in a 1:100,000 or 1:200,000 dilution.	
1 mL of 1:1000 epinephrine = 1 mg.	
0.1 mL of 1:1000 epinephrine in 10 mL anesthetic solution = 1:100,000 dilution = 0.010 mg/mL.	
0.1 mL of 1:1000 epinephrine in 20 mL anesthetic solution = 1:200,000 dilution = 0.005 mg/mL.	
Some examples detailing *epinephrine* content:	
	1:100,000 / **1:200,000**
5 mL	0.050 mg / 0.025 mg
10 mL	0.100 mg / 0.050 mg
20 mL	0.200 mg / 0.100 mg
Therefore, 50 mL of 1% lidocaine with epinephrine 1:200,000 contains 500 mg lidocaine and 0.25 mg epinephrine.	

Intra-arterial injections in subcutaneous end-arteries about the head or neck are capable of retrograde flow into the cerebral circulation, if the injection pressure exceeds the arterial pressure. Because the blood volume in the brain is only about 30 mL at any given moment, even 1 mg of lidocaine injected into the carotid artery can theoretically produce toxic concentrations. Patients with low cardiac output or hypovolemia and preferential cerebral blood flow may suffer enhanced central nervous system toxicity.

Host Factors

Four factors tend to lower the body's systemic tolerance to local anesthetic agents: hypoxia, acid-base status, protein binding, and concomitant drug use.

Hypoxia.

It was initially thought that local anesthetic overdose produced central nervous system stimulation and subsequent intracellular hypoxia, which then became the key precipitant to all toxic manifestations of the drug. It is now known that hypoxia may enhance anesthetic toxicity, but it is not the primary factor.

Acid-base status.

Although studies of metabolic alkalosis have produced conflicting results, acidosis, particularly respiratory acidosis, can increase toxicity. The elevated CO_2 produced by respiratory acidosis crosses the blood-brain barrier, where it may act directly on the receptor and indirectly by lowering intracellular pH. The lower pH causes more drug to ionize, furthering the block in the sodium channel and increasing the potential for toxicity.

Protein binding.

Unbound drug concentration relates more closely to toxic effects than does total drug concentration (bound plus unbound) as measured in the blood. The amount of a-acid glycoprotein (AAG), the major plasma protein responsible for binding local anesthetics, is considerably decreased in neonates compared with adults. Arthur and McNicol imply that low AAG levels in neonates are responsible for an increased toxic potential. [88] Tucker lists several disease states that alter AAG levels and protein binding but questions whether they lead to changes in free drug concentration in vivo. [89]

Concomitant drugs.

For years, barbiturates were used to prevent and treat local anesthesia-induced seizures. Barbiturates were found to worsen anesthetic-induced apnea and cardiovascular depression. Central nervous system depressants are used with caution when concern exists for local anesthetic toxicity. Central nervous system stimulants have been shown to increase anesthetic-induced excitability and are avoided. Mixtures of local anesthetics have an additive effect on toxicity. If two drugs are used at half strength, they produce the same degree of toxicity as if each were used alone at normal strength.

Recognition of Central Nervous System Toxicity

The earliest manifestation of systemic toxicity is central nervous system stimulation resulting from blockade of inhibitory synapses. Central nervous system depression follows and is produced by direct depression of the medulla, although hypoxia may play a role. Signs and symptoms are dose-related. Potential signs and symptoms of central nervous system toxicity, in progressing order, are numbness of the tongue, light-headedness, tinnitus, visual disturbances, muscle twitching, convulsions, coma, and apnea. Drowsiness, commonly seen at lower doses with lidocaine, is not associated with bupivacaine or etidocaine. Tetracaine may produce apnea or cardiovascular toxicity without central nervous system manifestations.

Recognition of Cardiovascular Toxicity

Moderate blood concentrations of local anesthetics produce slight increases in cardiac output, heart rate, and arterial pressure because of the effects of direct peripheral vasodilation and central nervous system stimulation. At concentrations generally well above central nervous system toxicity levels, local anesthetics cause direct myocardial depression, hypotension, and bradycardia, perhaps leading to cardiovascular collapse. These agents also slow electrical conduction leading to reentry phenomenon and various supraventricular and potentially lethal ventricular dysrhythmias, especially with bupivacaine and etidocaine.

Prevention of Toxicity

Knowledge of factors contributing to toxicity guides preventive measures. Avoid esters in patients with an atypical form or a quantitative deficiency of pseudocholinesterase. Use amides with caution for patients with severe liver disease or congestive heart failure. Pay attention to maximum safe dosages, based on site, technique, epinephrine use, and patient status. Add epinephrine when possible to decrease the drug absorption rate at vascular sites. Reduce drug concentration with saline dilution to increase the volume for administration when a large area must be infiltrated. Frequently aspirate in areas of high vascularity, even though a negative aspiration may not prevent IV administration. [90] Slow infiltration is advised for safety and is also associated with less pain.

Treatment of Systemic Toxicity

No local anesthesia is given without the ability to recognize and treat a toxic reaction, including having all necessary equipment and drugs readily available and being knowledgeable in their use. Despite taking all possible precautions, toxic reactions still occur, and close obseration of the patient allows early detection and treatment.

Providing proper oxygenation and ventilation at the earliest sign of a reaction is the cornerstone of treatment. Encourage patients who are alert to moderately hyperventilate to lower the PCO_2 and raise the seizure threshold. Intubation with high-flow oxygen and hyperventilation is performed for patients who cannot adequately ventilate. Initiate IV access and monitor vital signs and cardiac rhythm closely.

Seizures are generally self-limited but are treated if they persist or prevent adequate ventilation. Because respiratory depression secondary to toxicity may follow, low-dose diazepam, 2 to 5 mg, or an ultrashort-acting barbiturate (thiopental or sodium methohexital) is preferred. Intubate the patient to ensure an effective airway and prevent further lactic acidosis if seizures persist. If toxicity is caused by an ester, especially if there is an associated pseudocholinesterase problem, succinylcholine will compete with the anesthetic for the pseudocholinesterase and may increase the toxicity of both compounds.

Treat hypotension and bradycardia with fluids, leg elevation, a- and ß-agonists (epinephrine, ephedrine, or dopamine), or atropine as the need dictates. Although lidocaine (with diazepam pretreatment) has been shown to be effective for bupivacaine-induced ventricular dysrhythmias, there is strong theoretical and experimental evidence that bretylium is more effective. [91] [92] However, until bretylium becomes available again, amiodarone is a reasonable alternative. High doses of atropine and epinephrine can be successful in correcting pulseless idioventricular rhythm. Cardiopulmonary resuscitation is instituted when necessary.

Allergic Reactions

Allergenic Agents

True allergic reactions are rare, accounting for only 1% to 2% of all adverse reactions, but they are important to recognize because of their serious potential. Ester solutions (procaine, tetracaine), which produce the metabolite para-aminobenzoic acid (PABA), account for the great majority of these reactions. Amide solutions (lidocaine, bupivacaine) are rarely involved and it is usually the preservative methylparaben (MPB), which is structurally similar to PABA, that is responsible. Although pure esters and pure amides do not cross-react, amides may appear to do so if multidose vials containing MPB are used. Patients may manifest an allergic response on first contact to a local anesthetic because of previous sensitization to these agents. MPB is found in creams, ointments, and various cosmetics, and PABA is an ingredient in many sunscreen preparations. Patients who are latex-sensitive may manifest an allergic reaction incorrectly attributed to the local anesthetic.

Although cell-mediated delayed reactions manifesting as dermatitis may occur, it is immediate hypersensitivity that most concerns the emergency clinician. A spectrum of signs and symptoms may occur, from rhinitis and mild urticaria to bronchospasm, upper airway edema, or anaphylactic shock. Onset may be immediate, occurring even during administration of the agent. Treat anaphylaxis in the usual manner.

The more frequent problem facing emergency clinicians is the patient who claims to have a past history of local anesthetic allergy. Most patients assume that any adverse reaction to a local anesthetic procedure is an allergy. Because allergy is rarely the cause, a careful history and a review of prior records, if available, are crucial in evaluating these patients. Uncover the actual cause of the past reaction and the specific agent involved. Ask about the exact signs and symptoms, technique of administration, amount of drug used, and how the patient was treated. If an allergic reaction cannot be ruled out and the drug previously used is known, use an agent from the other class (whether amide or ester). Lidocaine from a dental cartridge does not contain MPB, and if this were the allergenic source, then an ester agent could be used. However, if lidocaine from a multidose vial is implicated, do not use an ester, because MPB may cross-react with PABA. In this case, it may be safer to use an amide without MPB or to choose an alternative (see later). In most cases the allergen is an ester, and the patient can safely be given an amide without MPB. Single-dose ampules of 1% lidocaine without MPB, readily obtainable from a resuscitation cart, can be used for this purpose.

Uncertainty often exists regarding the specific agent involved, and the clinician must choose an alternative approach to local anesthesia. If the wounds are extensive and the risk is acceptable, sedation or general anesthesia may be used. Conversely, if minimal pain is expected and the procedure is short (e.g., one or two sutures or staples in the scalp), no anesthesia may be required. Other alternatives include parenteral narcotics, benzodiazepines, nitrous oxide inhalation, or a combination of these (see Chapter 34). These methods may be useful, but the degree of anesthesia produced is often not sufficient. Antihistamines injected into a wound have been successfully used for many years and represent a good alternative. Local anesthetic efficacy is found in varying degrees in all antihistamines.

Diphenhydramine and Benzyl Alcohol

Several studies demonstrated that 1% diphenhydramine (Benadryl) is as effective as 1% lidocaine for infiltrative anesthesia. [93] [94] [95] As long as diphenhydramine is not used at concentrations greater than 1%, potential problems of skin necrosis or significant sedation are rare. *Dilute the standard 5% parenteral form to 1% concentration for SQ injection* (1 mL drug to 4 mL saline). The duration of action for diphenhydramine is shorter than that for lidocaine but appears to be adequate for most procedures. The injection pain of diphenhydramine exceeds that of lidocaine but can be diminished by reducing the concentration to 0.5%. At this lower concentration, the effectiveness of this agent on facial wounds is lost. [96] The addition of epinephrine to 0.5% diphenhydramine results in a more painful solution with a

shorter duration of action than a standard buffered lidocaine with epinephrine solution. [97] There are scattered reports of benzyl alcohol (0.9%) with epinephrine (1:100,000) being used as an effective local anesthetic. This appears to be useful but is of short duration. [98] [99]

Skin Testing

Skin testing and progressive SQ challenge doses deserve special mention because they appear to be logical and well-studied approaches. However, intradermal skin testing with local anesthetics is controversial. False-positive results are

frequently produced by local histamine release in response to needle trauma, tissue distention, or preservatives in the solution. [100] In addition, a high incidence of false-negative results can occur. It is questionable whether these low-molecular-weight drugs or their allergenic metabolites are ever capable of eliciting positive responses.[101] Other disadvantages of skin testing include its time-consuming nature and its potential hazard when even minute traces of an allergen may precipitate a serious reaction. SQ challenge testing in graduating doses has been advocated and may well eliminate many false responses, but it does not eliminate the problems of time and hazard. Swanson, recognizing that allergy to pure lidocaine is extremely rare, recommends 0.1 mL as a single intradermal skin test. [102] Although his approach eliminates the time disadvantage, the intradermal placement can still produce false responses. It would seem more reasonable to give this test dose subcutaneously while exercising due caution in the unlikely even that a patient exhibits a serious reaction.

Summary of Anesthetic "Allergy" Management

Generally speaking, the optimal approach to the patient with a presumed anesthetic allergy is to determine the specific anesthetic agent associated with a presumed allergic reaction and then use a preservative-free agent from the other class (see earlier discussion). If the agent is unknown, use an antihistamine or give 0.1 mL of preservative-free lidocaine as an SQ test dose, proceeding with the full dose if no reaction occurs within 30 minutes. Given the studies mentioned earlier, the prudent choice would seem to be diphenhydramine (Benadryl).

Catecholamine Reactions

Anxiety and vasoconstrictor (epinephrine) reactions are discussed together because each produces similar manifestations caused by elevated catecholamine levels. These relatively common disorders are difficult to distinguish from each other and are generally not serious.

Excess catecholamine levels produce tachycardia; palpitations; hypertension; apprehension; tremulousness; diaphoresis; tachypnea; pallor; and, on occasion, anginal chest pain. Thus, catecholamine excess may resemble the central nervous system stimulation phase of local anesthetic toxicity.

Catecholamine reactions are usually not caused solely by exogenous epinephrine, because if it is used in its optimal concentration (1:200,000), the maximum safe dose (0.25 mg) is rarely exceeded. However, many patients produce significant endogenous catecholamines due to anxiety about the anesthetic approach or upcoming procedure. In this case, even the addition of small amounts of epinephrine could trigger a catecholamine reaction. Therefore, patient preparation includes proper explanation and reassurance to decrease anxiety. Exercise caution for patients who have hyperthyroidism, hypertension, or atherosclerotic cardiovascular disease, although these conditions do not contraindicate the judicious use of epinephrine-containing anesthetics. Do not give epinephrine-containing anesthetics to patients on monoamine oxidase inhibitors (MAOIs).

Treatment of the catecholamine reaction includes stopping further drug administration; observing the patient closely; and administering a- or ß-antagonists or benzodiazepine agents, if necessary, to combat severe reactions.

Vasovagal Reactions

Vasovagal reactions are common, especially in dental procedures (reported incidence, 2% to 3%), during which the patient is generally in an upright position. The patient initially experiences anxiety when a triggering event, commonly the sight or sensation of needle insertion, causes a loss of sympathetic tone and an increase in vagal tone. The resultant hypotension and bradycardia may lead to syncope. Addressing the patient's anxiety and administering the injections with the patient recumbent are useful preventive measures. Cardiac monitoring may help identify the onset of vagally induced bradycardia when suggested by past history. Lay the patient supine and elevate the legs. Rarely, atropine is required. Should a patient lose consciousness while in a recumbent position, consider diagnoses other than vasovagal syncope, although significant bradycardia, and even complete heart block, may accompany a vagal reaction in the supine patient.

SUMMARY

Emergency medicine cannot be practiced without the use of local anesthetic agents. Their effectiveness when applied topically or by infiltration makes them extremely adaptable to many clinical circumstances. A working knowledge of commonly employed agents is necessary to ensure the safe administration of these medications. Direct specific effort at maximizing the drugs' anesthetic effects while minimizing the pain of administration and risk of toxicity.

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Chapter 31 - Regional Anesthesia of the Head and Neck

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Kevin P. Kilgore

The use of intraoral and extraoral regional anesthesia is both simple and convenient. [4] [7] Nerve blocks are used to attain anesthesia in areas of broad distribution in the face with a minimal amount of anesthetic and resultant tissue distortion. Local anesthetic blocks are effective for closing facial lacerations, especially those of the lips, the forehead, and the midface, where the swelling caused by local infiltration is undesirable. Local anesthetic blocks are also effective for the relief of pain, for anesthesia in debridement, and for diagnostic purposes.

Patients with dental pain who do not get relief with a regional dental block most likely do not have pain of dental origin. Regional blocks are also therapeutic for both surgical procedure anesthesia and pain control for dental emergencies such as toothaches and dry sockets (see Chapter 66). In cases in which the patient is thought to be seeking drugs and one wishes to avoid narcotics, a dental anesthetic block is frequently the treatment of choice.

Topical anesthetic solutions such as tetracaine-Adrenalincocaine (TAC) solution are useful in small lacerations of the scalp and face because of the vascularity of these areas. TAC is not to be used on or near mucous membranes or the eye. Detailed information on the use of TAC appears in Chapter 30 . More extensive discussions of the general complications of local anesthetics and of regional anesthesia are provided in Chapter 30 and Chapter 33 , respectively. Ophthalmologic anesthesia is discussed in Chapter 64 . Blocks about the ears and nasal anesthesia are discussed in Chapter 65 .

The procedures and techniques described here generally carry a low morbidity. The supraperiosteal and mental nerve infiltrations can generally be learned through reading and experimentation; more sophisticated blocks (e.g., inferior alveolar block) are best learned under the instruction of an experienced clinician, a dentist, or an oral and maxillofacial surgeon.

ANATOMY OF THE FIFTH CRANIAL (TRIGEMINAL) NERVE

The fifth cranial nerve, the trigeminal nerve, is the sensory nerve to the face (Fig. 31-1 *A* (Figure Not Available)) and is the largest of the cranial nerves. It takes its origin from the midbrain and enlarges into the gasserian, or semilunar, ganglion. One gasserian ganglion supplies each side of the face. The gasserian ganglion is a flat, crescent-shaped structure approximately 10 mm long and 20 mm wide that divides into three branches: the ophthalmic, maxillary, and mandibular nerves (Fig. 31-1*B* (Figure Not Available)).

Ophthalmic Nerve

The first division, the *ophthalmic nerve* (V_1), is the smallest branch in the gasserian ganglion. It leaves the cranium through the superior orbital fissure and has 5 cutaneous branches. These branches are as follows:

1. The medial and lateral branches of the supraorbital nerve, which emerge on the face through the supraorbital notch. These two sensory nerves pierce the frontalis muscle and extend to the lambdoid suture on the back of the skull.
2. The supratrochlear nerve, which is sensory to the medial aspect of the forehead just above the glabella.
3. The infratrochlear nerve.
4. The lacrimal nerve.
5. The external nasal nerve.

In addition to being sensory to the forehead, branches of the ophthalmic nerve are sensory to the cornea, the upper eyelid, structures in the orbit, and the frontal sinuses.

Maxillary Nerve

The second division, the *maxillary nerve* (V_2), is sensory to the maxilla and associated structures, such as the teeth, the periosteum and the mucous membranes of the maxillary sinus and the nasal cavity, the soft and hard palate, the lower eyelids, the upper lip, and the side of the nose. The second division exits the cranium from the foramen rotundum and ultimately enters the face through the infraorbital canal; it terminates as the infraorbital nerve. The infraorbital nerve gives sensory branches to the lower eyelids, the side of the nose, and the upper lip.

The anatomy of the maxillary nerve is rather complicated because of its numerous branches. The first branch comprises two short sphenopalatine nerves to the pterygopalatine ganglion, also called the *Meckel ganglion* or the *sphenopalatine ganglion*. The next two branches of clinical importance are the nasopalatine and the greater (anterior) palatine nerves. The nasopalatine nerve arises from the pterygopalatine ganglion, courses down along the nasal septum, and is transmitted through the anterior portion of the hard palate by way of the anterior palatine canal. This canal is located in the midline approximately 10 mm palatally to the maxillary central teeth and immediately behind the incisors. The nasopalatine nerve is sensory to the most anterior portion of the hard palate and the adjacent gum margins of the upper incisors. This nerve is rarely blocked in clinical practice, except in dental operations (Fig. 31-2 (Figure Not Available)).

The anterior, or great, palatine nerve arises from the pterygopalatine ganglion and passes down through the posterior palatine foramen. The posterior palatine foramen is located 10 mm palatally to the third molar and the bicuspid teeth and intermingles with the nasopalatine nerve opposite the cuspid tooth. The greater palatine nerve is sensory to most of the hard palate, as well as the palatal aspect of the gingiva. It is rarely blocked in the emergency department (ED) (see Fig. 31-2 (Figure Not Available)).

The next branch consists of the posterior superior alveolar (PSA) nerve, which courses down the posterior surface of the maxilla for approximately 20 mm, at which point it enters one or several small posterosuperior dental foramina. This nerve supplies all the roots of the third and second molar teeth and two roots of the first molar tooth. A third branch consists of the middle superior alveolar (MSA) nerve, which branches off about midway within the infraorbital canal and then courses downward in the outer wall of the maxillary sinus. This nerve supplies the maxillary first and second bicuspid teeth and the mesiobuccal root of the first molar. The last branch consists of the anterior superior alveolar (ASA) nerve, which branches off into the infraorbital canal approximately 5 mm behind the infraorbital foramen, just before the terminal

Figure 31-1 (Figure Not Available) *A,* Cutaneous distribution of the fifth cranial nerve (the trigeminal nerve). Note that the supraorbital, infraorbital, and mental foramina are all in line just medial to the pupil when the person looks straight ahead. *B,* Branches of the trigeminal nerve. *(Adapted from Eriksson E: Illustrated Handbook in Local Anesthesia. Philadelphia, WB Saunders, 1980.)*

branches of the infraorbital nerve emerge. This nerve descends in the anterior wall of the maxilla to supply the maxillary central, lateral, and cuspid teeth; the labial mucous membrane; the periosteum; and the alveoli on one side of the median line. There is intercommunication among the ASA, MSA, and PSA nerves.

Mandibular Nerve

The third division, the *mandibular nerve* (V_3), is the largest branch of the trigeminal nerve. It exits from the cranium through the foramen ovale and divides into three principal branches:

1. The long buccal nerve branches off just outside the foramen ovale. It passes between the two heads of the external pterygoid muscle and crosses in front of the ramus to enter the cheek through the buccinator muscle, buccally to the maxillary third molar. The buccal nerve supplies sensory branches to the buccal mucous membrane and the mucoperiosteum over the maxillary and mandibular teeth. The cutaneous branch is the sensory nerve to the cheek.
2. The lingual nerve courses forward toward the midline. It runs downward superficially to the internal pterygoid muscle to pass lingually to the apex of the mandibular third molar. It enters the base of the tongue at this point through the floor of the mouth and supplies the anterior two thirds of the tongue, the lingual mucous membrane, and the mucoperiosteum.
3. The largest of the V_3 branches is the inferior alveolar nerve. It is sensory to all of the lower teeth, although the central and lateral incisors and the buccal aspect of the molar teeth may receive additional sensory innervation. The nerve descends, covered by the external pterygoid muscle, and passes between the ramus of the mandible and the sphenomandibular ligament to enter the mandibular canal. It is accompanied by the inferior alveolar artery and vein and proceeds along the mandibular canal, innervating the teeth. At the mental foramen, the nerve bifurcates into an incisive branch, which continues forward to supply the anterior teeth. It gives off a side branch, the mental nerve, which exits from the mental foramen to supply the skin. The mental foramen is located approximately between the apices of the lower first and second bicuspids, or premolar teeth. This is a useful site at which to perform a nerve block, because the mental nerve is sensory to the integument of the chin and the skin and the mucous membrane of the lower lip.

Figure 31-2 (Figure Not Available) The anterior one third of the palate, from canine to canine, is anesthetized by a local injection near the anterior palatine canal. There may be some overlapping branches of the anterior palatine nerve. Anesthesia of the posterior two thirds of the palate is obtained by a local injection in the area of the posterior palatine foramen. Topical anesthesia should precede local infiltration. *Note:* Do not enter the foramen itself, because the anesthetic may reach the middle palatine nerve and produce anesthesia of the soft palate, resulting in gagging. Foraminal injection may also produce pressure necrosis and permanent anesthesia. *(Adapted from Eriksson E: Illustrated Handbook in Local Anesthesia. Philadelphia, WB Saunders, 1980.)*

EQUIPMENT FOR DENTAL AND CRANIAL NERVE BLOCKS

One may easily give extraoral injections with standard injection equipment. A 3-mL Luer-Lok syringe with a 1 ½ inch 25- to 27-ga needle is readily found in the ED. Intraoral local anesthesia is also conveniently administered with a Monoject aspirating dental syringe, which uses Carpule cartridges of anesthetic and disposable needles (Fig. 31-3*A*). A needle no smaller than 27 G is recommended for deep block techniques owing to the inability to perform aspiration with smaller needles. Generally, a long needle is used for block techniques, and a short needle is used for infiltrations.

The needle is screwed to the hub of the Monoject syringe, which is really an adapter that in turn is screwed into the barrel of the syringe. The adapter may be removed for cleaning. When removing the disposable needle, one must take care not to remove and discard the adapter as well; this would render the syringe functionless (Fig. 31-4*A, B*).

One pulls back the end of the syringe on its spring, allowing room for the Carpule cartridge of anesthetic to be inserted (Fig. 31-5*A*). The metal end of the Carpule cartridge is inserted, which engages the needle. The handle of the syringe is then released and tapped to engage a barb into the rubber stopper of the cartridge (Fig. 31-5*B*). One may then perform simple aspiration by retracting the handle, pulling on the rubber stopper within the Carpule (Fig. 31-5*C*).

To discard a Carpule cartridge, one should leave the needle in place on the syringe. The handle of the syringe is then withdrawn rapidly, disengaging the barb. If the needle has been removed, great care must be taken, because the negative pressure created in the Carpule cartridge upon withdrawal of the barb may cause shattering. Other adjuncts that are helpful in the administration of intraoral anesthesia include topical local anesthetic agents such as gels or sprays. It should be noted that dental syringes are not mandatory for intraoral local anesthesia but do make the procedure simpler, particularly aspiration. Reusable glass and disposable plastic aspirating syringes that do not use Carpule cartridges are also available.

The anesthetic agent most frequently used is 2% lidocaine with a vasoconstrictor, such as 1:100,000 or 1:50,000 epinephrine. Many other anesthetic agents, such as mepivacaine (Carbocaine) and Cetacaine (a combination of benzocaine, tetracaine, butamben, and benzalkonium), with or without vasoconstrictor agents, are also available. Bupivacaine (Marcaine) with or without epinephrine is a longer acting anesthetic that is often ideal for the procedures performed in the ED. Bupivacaine with epinephrine is theoretically the best choice in the ED because of its longer duration of action. When this agent is not available in Carpule cartridge form, a Carpule cartridge containing a different anesthetic may be emptied and bupivacaine drawn up in the evacuated cartridge. Because of the rich vascularity of the oral cavity, vasoconstrictors are important in sustaining the duration of anesthesia and should be used wherever possible in the absence of medical contraindications. Buffering with bicarbonate is not recommended for oral anesthesia.

GENERAL RECOMMENDATIONS

Needles no smaller than 27-ga should be used for block techniques, since a higher gauge makes aspiration difficult, possibly resulting in inadvertent intravascular injection. An intravascular injection is not problematic except that the nerve block will not be effective. Intravascular epinephrine, although used

Figure 31-3 *A,* Local anesthesia—basic setup for intraoral application using an aspirating dental syringe. An alternative for a dental syringe is a 3-mL Luer-Lok syringe with a 1 ½ inch 25- or 27-ga needle. *B* and *C,* Intraoral dental blocks may be minimally painless if a topical anesthetic numbs the mucosa. Topical anesthesia is applied prior to injection, illustrated here for an inferior alveolar nerve block. After drying the mucosa with gauze, the pterygomandibular triangle is painted with topical anesthetic. Benzocaine is liberally sprayed into two cotton swabs and held in place by closing the teeth and lips for a few minutes after painting the area of needle penetration. The mucosa is dried with gauze before applying the topical anesthetic.

in small doses, may produce systemic symptoms (anxiety, tachycardia) but the amount of local anesthetic is inconsequential.

When an intraoral block procedure is performed, the needle should not be inserted to its full length at the hub. Should inadvertent breakage occur in such a situation, needle retrieval may be difficult. Furthermore, the direction of a needle should not be changed while the needle is deep in the tissue. One should always aspirate before injection, and inject slowly to minimize pain. A warmed anesthetic solution is also more comfortable for the patient. In addition, topical anesthetics can be placed on mucous membranes before all dental

Figure 31-4 *A,* Proper technique for removal of the disposable hypodermic needle from the dental aspirating syringe. *B,* Incorrect technique involves removal of the carpule adapter, rendering the syringe unable to accept new Carpules.

blocks to make needle puncture painless. This adjunct is suggested whenever possible (Fig. 31-3*B, C*).

An important caveat for intraoral local anesthesia is that the injection should not be made into or through an infected area. This is especially important in inferior alveolar nerve blocks, in which tracking of an infection can be serious and difficult to treat. Trismus with inadequate oral access or direct extension of infection to parapharyngeal spaces can result. Therefore, local anesthesia should be only superficial before incision and drainage, unless a block can be performed far proximal to the site of infections.

Figure 31-5 Loading the dental aspirating syringe. *A,* The end of the syringe is pulled back. When released, the Carpule cartridge engages the needle. *B,* The barb is snapped into place. *C,* Once engaged, the syringe allows aspiration.

TECHNIQUE

Topical Anesthesia

Most patients fear dental blocks greatly, and the anxiety and pain may be lessened considerably with the use of topical anesthetics that are applied to the mucous membranes before injection (see Fig. 31-3*B, C*). The area to be injected is first thoroughly dried with gauze. Copious saliva will wash away the anesthetic prematurely. A cotton-tipped applicator is generously coated with 20% benzocaine (Hurricaine, Beutlich, Inc, Niles, IL) or 5% to 10% lidocaine, and the area of injection is painted. The patient may hold the cotton swab in place. Anesthesia results in 2 to 3 minutes. Note that rather concentrated topical anesthetics must be used, and poor results are obtained with weaker preparations such as 2% viscous lidocaine. Cocaine (4%) is another acceptable topical anesthetic.

Supraperiosteal Infiltrations

The most common technique for intraoral local anesthesia of individual teeth is the supraperiosteal infiltration injection. This technique may supply complete relief of a toothache and is a useful ED procedure that can provide nonnarcotic analgesia in the middle of the night. The area to be anesthetized is selected and dried with gauze. A topical anesthetic, such as 20% benzocaine or 5% lidocaine ointment, is applied as described. The patient is asked to close the jaw slightly to relax the facial musculature. The mucous membrane of the area is grasped with a piece of gauze; the gauze is pulled out and downward in the maxilla and out and upward in the mandible to extend the mucosa fully and to delineate the mucobuccal fold. The mucobuccal fold is then punctured with the bevel of the needle facing the bone. The area is aspirated, and approximately 1 to 2 mL of local anesthetic are deposited at the apex (area of the root tip) of the involved tooth (Fig. 31-6*A, B*). It is helpful to place a finger against the *outer* aspect of the lip overlying the injection site and apply firm and steady pressure against the lip as the local anesthetic is *slowly* injected into the supraperiosteal site.

The purpose of the injection is to deposit the anesthetic near the bone that supports the tooth. Because the anesthetic must penetrate the cortex of bone to reach the nerve of the individual tooth, the injection may fail if the solution is deposited too far from the periosteum, if the needle is passed too far above the roots of the teeth, or if the bone in the area is unusually thick or dense. If anesthesia is unsuccessful, one may also inject the palatal side. It may take 5 to 10 minutes to achieve full anesthesia with this technique, and the procedure may not be as effective for the posterior molars. Infiltration of the area around the maxillary canine and the first premolars will anesthetize the MSA and ASA nerves; lacerations of the upper lip can be treated by bilateral injection in the canine fossa areas.

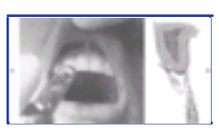

Figure 31-6 Topical anesthetic is applied to the dried mucosa before injection. *A,* Supraperiosteal injection technique above the incisors for anesthesia of the upper lip or individual teeth. *B,* Diagrammatic representation of supraperiosteal injection. The aim is to deposit the anesthetic right next to the periosteum at the level of the apex (area of the root tip) of the tooth. The palatal side of the tooth may also be injected.

Posterior Superior Alveolar Nerve Block (PSA)

Anatomy

The PSA block is used to anesthetize the maxillary molar teeth. On occasion the maxillary first molar may not be completely anesthetized by this technique alone, and may require an additional block (discussed subsequently). The landmarks for this technique are the posterior-lateral portion of the maxillary tuberosity and the second molar (see Fig. 31-2 (Figure Not Available)).

Intraoral Approach

A topical anesthetic on a cotton-tipped swab is applied for 60 seconds prior to introducing the needle for the nerve block. [4] [5] With the patient's mouth half-open and the jaw swung toward the operator, the cheek is retracted laterally. The puncture is made in the mucosal reflection just distal to the distal buccal root of the upper second molar (Fig. 31-7*A, B*). The needle is directed toward the maxillary tuberosity

Figure 31-7 Posterior superior alveolar nerve block.

(i.e., upward, backward, and inward) and then along the curvature of the maxillary tuberosity to a depth of approximately 2 to 2.5 cm. On reaching this depth, the needle is aspirated and 2 to 3 mL of anesthetic solution is injected.

Complications

Complications includes puncture of the pterygoid plexus and hematoma formation should the syringe not be aspirated before injection. Also, if the needle were advanced too far posteriorly, a Division II block of cranial nerve V will result.

Middle Superior Alveolar Nerve Block (MSA)

Anatomy

The MSA block is used to anesthetize the mesiobuccal root of the maxillary first molar in order to achieve complete anesthesia of this tooth. The landmark for this procedure is the junction between the second premolar and first molar.

Figure 31-8 Middle superior alveolar nerve block.

Intraoral Approach

A topical anesthetic on a cotton-tipped swab is applied for 60 seconds prior to introducing the needle for the nerve block. [4] [5] The cheek is retracted laterally and the

puncture is made in the mucosal reflection adjacent to the mesiobuccal root area of the first molar (the space between the second premolar and the first molar) directing the needle at a 45-degree angle (Fig. 31-8*A, B*). When the correct location has been determined and aspiration has been performed, 2 to 3 mL of anesthetic solution is injected. Massage of the tissue for 10 to 15 seconds after the injection will hasten the onset of the anesthesia.

Anterior Superior Alveolar Nerve Block (ASA)

Anatomy

The landmark for this technique is the apex of the canine tooth.

Figure 31-9 Anterior superior alveolar nerve block.

Intraoral Approach

A topical anesthetic on a cotton-tipped swab is applied for 60 seconds prior to introducing the needle for the nerve block. [4] [5] The patient is asked to close his jaw slightly to relax the upper lip. The lip is retracted anteriorly and the puncture is made in the mucosal reflection at the apex of the canine tooth directing the needle at a 45-degree angle (Fig. 31-9*A, B*). When the correct location has been determined and aspiration has been performed, 2 mL of anesthetic solution is injected. Massage of the tissue for 10 to 15 seconds after the injection will hasten the onset of the anesthesia.

Infraorbital Nerve Block

Anatomy

The infraorbital nerve block injection can be used to anesthetize the midface (Fig. 31-10). A solution of local anesthetic deposited adjacent to the infraorbital foramen anesthetizes not

Figure 31-10 Area of anesthesia of a unilateral infraorbital nerve block. Anesthesia includes the lower eyelid and the upper lip.

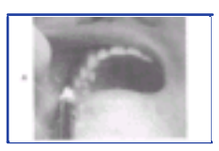

Figure 31-11 *A,* Intraoral approach for infraorbital nerve block. *B,* The position of the infraorbital foramen *(arrow)* on the inferior portion of the infraorbital ridge. During infiltration, the needle should be near but not within the foramen. *C,* Incorrect infraorbital injection technique may result in needle entry into the orbit. *(Adapted from Eriksson E: Illustrated Handbook in Local Anesthesia. Philadelphia, WB Saunders, 1980.)*

only the middle and superior alveolar nerves, but also the main trunk of the infraorbital nerve that innervates the skin of the upper lip, the skin of the nose, and the lower eyelid. The nasal mucosa is not anesthetized by this technique.

The infraorbital foramen is difficult to palpate extraorally and almost impossible to feel in the presence of facial swelling. It is found on the inferior border of the infraorbital ridge on a vertical (sagittal) line with the pupil when the patient stares straight ahead. Although one volunteer study found similar patient pain scale scores and overall preference in subjects receiving both intraoral and extraoral approaches, the intraoral approach seemed to provide nearly twice the duration of anesthesia. [6]

Intraoral Approach

A topical anesthetic on a cotton-tipped swab is applied for 60 seconds prior to introducing the needle for the nerve block [4] [5] . When performing the intraoral approach, one keeps the palpating finger in place over the inferior border on the infraorbital rim. The cheek is retracted, as in the supraperiosteal injection, and puncture is made in the mucosa opposite the upper second bicuspid (premolar tooth) approximately 0.5 cm from the buccal surface (Fig. 31-11*A, B*). The needle

should be directed parallel with the long axis of the second bicuspid until it is palpated near the foramen, a depth of approximately 2.5 cm. If the entry is too acute initially, one will encounter the malar eminence before approaching the infraorbital foramen. In addition, if the needle is extended too far posteriorly and superiorly, the orbit may be entered (Fig. 31-11*C*). Therefore, the procedure should be halted if the clinician is unsure of the location of the needle or if patient cooperation is unsatisfactory.

When proper needle location has been determined and aspiration has been performed, 2 to 3 mL of solution are injected *adjacent to, but not within, the foramen*. A finger should be held firmly on the inferior orbital rim to avoid ballooning of the lower eyelid with anesthetic solution. If one is not certain of the exact location of the infraorbital foramen, one may obtain anesthesia by performing a field block. For the latter technique, 5 mL of the anesthetic solution are infiltrated in a fanlike distribution in the upper buccal fold. This technique is not as precise as a discrete nerve block but usually produces the same effect. Massage of the tissue for 10 to 15 seconds after the injection will hasten the onset of the anesthesia.

Extraoral Approach

The infraorbital foramen may also be approached from an extraoral route (Fig. 31-12). The extraoral approach, of course, requires external preparation of the skin. In the extraoral approach, similar landmarks are used to locate the infraorbital foramen. The needle can be felt to pass through the skin, the subcutaneous (SQ) tissue, and the quadratus labii superioris muscle. After injection, the infiltrated tissue, usually visibly swollen, should be firmly massaged for 10 to 15 seconds.

Care must be taken not to anesthetize the facial artery and vein, because these may lie on either side of the needle. Vasoconstrictors should be avoided with this technique to avoid vasoconstriction of the facial artery. If severe blanching of the face occurs, warm compresses should be applied to the face immediately. Local phentolamine also may be administered (see Chapter 30).

Inferior Alveolar Nerve Block

In the setting of extreme dental pain, the emergency clinician may find the use of the inferior alveolar nerve block and the lingual nerve block useful. This injection is

somewhat more difficult than the other techniques described, and the emergency clinician is advised to view demonstrations of this procedure before attempting it. The inferior alveolar nerve block provides anesthesia to all of the teeth on that side of the mandible and desensitizes the lower lip and the chin via block of the mental nerve. This technique is primarily useful for anesthetizing patients who have sustained severe dentoalveolar trauma; those with complaints of postextraction pain, dry socket, or pulpitis (toothache); or those with periapical abscess.

Anatomy

The anatomy of the region should first be reviewed (Fig. 31-13*A*). The patient can be seated either in a dental chair or upright with the occiput firmly against the back of the stretcher, so that when the mouth is opened, the body of the mandible is parallel to the floor. Despite the use of topical anesthesia, the clinician should be ready for an unexpected quick jerk of the head when the anxious patient first feels the

Figure 31-12 *A,* The extraoral approach to the infraorbital nerve. This procedure is more difficult than the intraoral approach, especially when attempting to obtain anesthesia of the upper lip. *B,* During injection, a finger placed above the needle and below the eyelid will limit swelling of the lower lid. Continual pressure with a finger during injection limits edema of the lower eyelid from the anesthetic solution.

needle. The clinician stands on the side *opposite* the one being injected.

The technique first involves palpation of the retro-molar fossa with the index finger or thumb. With this maneuver, the greatest depth of the anterior border of the ramus of the mandible (the coronoid notch) may be identified (Fig. 31-13*B*). With the thumb in the mouth and the index finger placed externally behind the ramus, the tissues are retracted toward the buccal (cheek) side, and the pterygomandibular triangle is visualized (Fig. 31-13*C*). This technique also moves the operator's finger safely away from the tip of the needle.

Approach

The mucosa over the area to be injected may be coated with a topical anesthetic as described previously. When topical anesthesia has been obtained, the syringe should be held parallel to the occlusal surfaces of the teeth and angled so that the barrel of the syringe lies between the first and second premolars on the opposite side of the mandible (see Fig. 31-13*C*). Failing to appreciate this required angle is the most common reason for failure with this nerve block. If a large-barrel syringe is used, the corner of the mouth may hamper

Figure 31-13 Inferior alveolar nerve block. *A,* Transverse illustration of the anatomy of the pterygomandibular triangle and direction of needle *before injection*. *B,* Anatomy of the inferior alveolar nerve block (intraoral approach) *(Adapted from Eriksson E: Illustrated Handbook in Local Anesthesia. Philadelphia, WB Saunders, 1980. Reproduced with permission)*. The greatest depth of the anterior border of the ramus of the mandible, the coronoid notch, is identified with the left index finger or left thumb. *C,* The ramus is grasped between an intraorally placed thumb (positioned on the coronoid notch) and extraorally positioned index finger. The pterygomandibular triangle may then be well visualized. Note the angle of the syringe during injection, with the syringe overlying the first and second premolars on the opposite side of the mandible. Failing to appreciate this orientation is one common cause of failure. The operator should be able to feel the needle contacting the bony surface of the inner mandible. Also note that the injection is 1 cm *above* the level of the teeth. *D,* Directing the needle too far posteriorly during the inferior alveolar nerve block technique will result in entry into the area of the parotid gland. Anesthesia of the seventh nerve may result. This occurs because from an improper entry orientation and is corrected by noting *C*. *E,* To compensate for difficulty in obtaining the correct approach with a straight needle, a 25-ga 1 ½-inch needle is bent 30° with the needle guard.

efforts to obtain the proper angle. The angle is facilitated by carefully bending the 25-ga needle about 30° (Fig. 31-13*E*). Puncture is made in the triangle, at a point that is *1 cm above the occlusal surface of the molars*. If the needle enters too low (e.g., at the level of the teeth), the anesthetic will be deposited over the bony canal and prominence (lingula) that house the mandibular nerve, and not over the nerve itself.

The needle should be felt to pass through the ligaments and the muscles covering the internal surface of the mandible. One should stop when the needle has reached bone, which signifies contact with the posterior wall of the mandibular sulcus; bone *must* be felt with the needle. Failure to do so generally results from directing the needle toward the parotid gland (too far posteriorly) rather than toward the inner aspect of the mandible. The needle should then be withdrawn slightly and aspirated, and approximately 1 to 2 mL of solution should be deposited. Three to 4 mL may be required if needle positioning is suboptimal.

In children, the angulation is not parallel to the occlusal surfaces of the teeth; instead the barrel of the syringe must be held slightly higher, because the mandibular foramen is lower. One may anesthetize the lingual nerve by placing several drops of anesthetic solution while withdrawing the syringe. The anterior two thirds of the tongue can thus be anesthetized. In actual practice, the lingual nerve is consistently blocked with this procedure owing to the close proximity of both nerves. Shortly following a successful injection, the patient will report tingling in the lower lip, however, it usually requires 3 to 5 minutes to achieve complete anesthesia.

Complications

Complications include inadvertent administration of anesthetic posteriorly in the region of the parotid gland, which will anesthetize the facial nerves (Fig. 31-13*D*). This is an annoying but relatively benign complication that will cause temporary facial paralysis (similar to a Bell's palsy) that affects the orbicularis oculi muscle and results in inability to close the eyelid. Should this occur, the eye must be protected until the local anesthetic has worn off (approximately 2 to 3 hours), and the patient must be reassured. Anesthesia with bupivacaine (Marcaine) presents a more significant problem if this

Figure 31-14 Mental infiltration technique. *A,* The correct supraperiosteal approach is an *infiltration* technique. *B,* Actual introduction of the needle into the mental foramen may produce neurovascular damage, and therefore infiltration only is recommended. *(Adapted from Eriksson E: Illustrated Handbook in Local Anesthesia. Philadelphia, WB Saunders, 1980.)*

complication occurs, because bupivacaine anesthesia lasts from 10 to 18 hours in some patients.

Mental Nerve Block

The mental nerve is blocked by the infiltration of local anesthetic about the nerve as it exits its bony foramen (Fig. 31-14*A, B*). Introduction of the needle in the mental nerve foramen is to be avoided, as the needle or injection of liquid into the foramen can produce neurovascular damage. Infiltration about the foramen will

provide for anesthesia of the lower lip. Lacerations of the midline of the lips require administration of anesthetic about the mental nerve on each side of the face; this practice anesthetizes crossover fibers. Generally a 1.3-cm (½ in), 25- or 27-ga needle on a 3-mL syringe is used.

Anatomy

The mental nerve is a continuation of the inferior alveolar nerve, which innervates the mucosa and the skin of the lower lip of the ipsilateral side of the mandible, with limited crossover of midline fibers. The nerve emerges from the mental foramen below the second premolar.

Approaches

Like the infraorbital nerve, the mental nerve may be blocked using an intraoral or an extraoral approach. Syverud and colleagues found that volunteers who received intraoral topical anesthetic followed by an intraoral injection considered the technique to be less painful than the extraoral approach. [9] Before using either approach, the mental foramen should be identified by palpation about 1 cm inferior and anterior to the second premolar. It is generally best to locate the foramen using a gloved finger placed into the labial area over the mandible. Generally the foramen will be just medial to the pupil (while staring straight ahead) along a sagittal plane.

When using the *extraoral* approach, the overlying skin is prepped and anesthetized with an anesthetic skin wheal. The mental nerve is approached through the overlying skin using a needle trajectory perpendicular or at a 45° angle to the foramen. As noted above, the local anesthetic is infiltrated about the nerve approximately 3 to 5 mm *outside* of the foramen. After

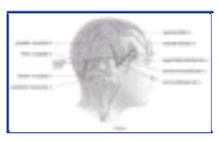

Figure 31-15 The sources of sensory nerve supply to the scalp.

placement of 1 to 2 mL of local anesthetic about the nerve, the needle is withdrawn and the area over the foramen massaged for 10 to 15 seconds to hasten anesthetic onset.

When using the *intraoral* approach, it is best to use topical anesthesia prior to infiltration. The lower labial fold adjacent to the first or second premolar is topically anesthetized. The mental foramen is again approached at about a 45° angle, and the area adjacent to the foramen is infiltrated with 1 to 2 mL of local anesthetic and the area massaged as above.

Scalp Block

Scalp blocks provide surgical anesthesia for the repair of scalp lacerations, drainage of superficial scalp abscesses, and exploration of scalp wounds.

Anatomy

As shown in Figure 31-15 and Figure 31-16 (Figure Not Available) , the scalp receives its nerve supply from branches of the trigeminal nerve (fifth cranial nerve) and the cervical plexus. The forehead is supplied by the supraorbital and supratrochlear nerves. Both nerves are branches of the ophthalmic division of the trigeminal nerve. The

Figure 31-16 (Figure Not Available) Topographic anatomy of the scalp taken above a line drawn from the upper border of the external ear to the occiput and the eyebrows. *(Adapted from Eriksson E: Illustrated Handbook in Local Anesthesia. Philadelphia, WB Saunders, 1980.)*

temporal region receives its nerve supply from the zygomaticotemporal (a V_2 branch nerve), temporomandibular, and auriculotemporal nerves (V_3 branch nerves).

The posterior aspect of the scalp is innervated by the greater auricular and the greater, lesser, and least (third) occipital nerves. The nerves that supply the posterior aspect of the scalp originate from the cervical plexus. All the nerves become superficial above a line drawn from the upper border of the external ear to the occiput and the eyebrows and converge toward the vertex of the scalp (see Fig. 31-15).

Topographically, the nerves and vessels of the scalp are located in the SQ tissue above the epicranial aponeurosis. From this level they divide into small branches that extend to the deeper layers (epicranium and periosteum) (see Fig. 31-16 (Figure Not Available)). [8]

Approaches

A scalp block can be accomplished by individually blocking each nerve that supplies the scalp, but this approach is time consuming, difficult, and cumbersome. Because the nerves on the scalp are superficially located, the scalp block can easily be performed by injecting local anesthetic agents into the SQ tissue circumferentially around the area to be blocked. Injection of local anesthetic to the deeper levels is necessary only if bone is to be removed. Note that injection of local anesthetic agents only in the deeper layers without SQ infiltration results in an unsuccessful block and a greater amount of bleeding during surgical intervention. [2]

In preparation for the block, a band of hair may be clipped (some clinicians prefer to shave the head, but this procedure is of unproven benefit). A band 1 cm wide and 3 cm away from the wound can be circumferentially clipped. Local anesthetics are injected in the clipped area.

The skin is prepared using an antiseptic solution, and a skin wheal is raised at any point along the clipped skin using a 1.3-cm (½ in), 25-ga needle. A 7.6-cm (3 in), 22-ga needle is inserted through the skin wheal into the SQ tissue and advanced along the scalp circumferentially following the previously clipped area. An injection of 0.5% to 1% lidocaine or 0.125% to

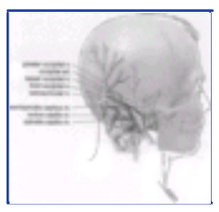

Figure 31-17 Anatomic relationship of the greater and lesser occipital nerves and the adjacent structures at the posterior aspect of the head. Note the proximity of the occipital artery, which is immediately lateral to the greater occipital nerve.

0.25% bupivacaine with epinephrine (1:200,000) is used. Epinephrine should be added to the local anesthetic agent to provide vasoconstriction and to prevent excessive blood loss and local anesthetic absorption. The total dose of the local anesthetic agents should not exceed the recommended dose for the particular agent (see Chapter 30). It may be useful to inject some local anesthetic solution into the temporalis muscle to prevent contraction of the muscle during the primary procedure.

Colley and Heavner demonstrated that when bupivacaine is used, the peak plasma local anesthetic concentrations occur within 10 to 15 minutes after injection. [3]

Thus, the first 10- to 15-minute period after the injection is the most critical period for the occurrence of local anesthetic toxicity. Colley and

Figure 31-18 *A,* The greater occipital nerve is blocked on a line 3 cm lateral to the external occipital protuberance and the base of the occipital bone. *B,* The lesser occipital nerve is blocked by injection of 2 to 3 mL of anesthetic solution along the posterior border of the mastoid process of the temporal bone.

Heavner also found that despite the scalp's high vascularity, the absorption of local anesthetics from the scalp is not excessive. Using the upper limit of the recommended dose of bupivacaine without epinephrine (175 mg), they found that peak plasma bupivacaine concentrations were 0.8 µg/mL with a 0.125% solution and 1.2 µg/mL with a 0.25% solution. Considering that the toxic plasma threshold for bupivacaine is 4 µg/mL, these concentrations suggest that a scalp block using bupivacaine has a wide margin of safety, even without the use of epinephrine. When epinephrine is used with bupivacaine, its effect on absorption becomes more pronounced with concentrations of 0.125% than with those of 0.25%. This is probably because at low concentrations (0.125%), bupivacaine has a vasoconstrictor property.[1]

Greater and Lesser Occipital Nerve Block

This relatively simple block may be useful in the ED for treating occipital neuralgia and tension headaches. For occipital neuritis, a long-acting corticosteroid, such as methylprednisolone (20 to 40 mg) may be combined with the local anesthetic (see Chapter 53).

Anatomy

The posterior aspect of the head is supplied by the posterior rami of the cervical nerves. Two important branches of these nerves are the greater and lesser occipital nerves. The greater occipital nerve becomes superficial on each side at the inferior border of the obliquus capitis inferior muscle and runs superiorly toward the vertex over this muscle. The nerve is located medial to the occipital artery. The lesser occipital nerve is located approximately 2.5 to 3.5 cm lateral and 1 to 2 cm caudal to the greater occipital nerve (Fig. 31-17).[5]

Approach

It is not usually necessary to shave or clip the scalp prior to performing greater and lesser occipital nerve blocks. The greater occipital nerve can best be blocked at the nuchal line, which is in the middle of the external occipital protuberance and the mastoid process. The nuchal line is located between the insertion sites of the trapezius muscle and the semispinalis muscles. At this site, the greater occipital nerve is just medial to the occipital artery.

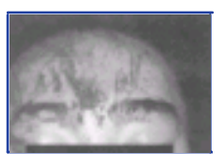

Figure 31-19 This patient had multiple small pieces of glass embedded in the forehead from a windshield injury. Removal was accomplished painlessly with bilateral supraorbital and supratrochlear nerve blocks.

The occipital artery is first palpated, and a 3.8-cm, 23- to 25-ga needle connected to a syringe that contains 5 mL of local anesthetic is inserted through the skin (Fig. 31-18*A*). After obtaining paresthesia at the vertex, 5 mL of local anesthetic solution is injected. The lesser occipital nerve is blocked by a fanlike injection of a local anesthetic solution 2.5 to 3.5 cm lateral and 1 cm caudal to the point described for the greater occipital nerve (Fig. 31-18*B*).[5]

This procedure is not usually associated with any complications; however, intra-arterial injections should be avoided by careful aspiration.

Ophthalmic (V_1) Nerve Block

The lateral and medial branches of the supraorbital, supratrochlear, and infratrochlear nerves may be blocked by

Figure 31-20 (Figure Not Available) *A,* Site for local injection of the lateral and medial branches of the supraorbital nerve. *B,* It should be noted that a finger may be placed just below the margin of the superior orbital rim to avoid swelling of the eyelid. *(Adapted from Eriksson E: Illustrated Handbook in Local Anesthesia. Philadelphia, WB Saunders, 1980.)*

percutaneous local injection at the point where they emerge from the superior aspect of the orbit. Anesthesia of the forehead and the scalp is achieved as far posteriorly as the lambdoid suture. Although anesthesia is easily obtained for suturing lacerations of the forehead and the scalp, the nerve block may also be used for debridement or topical treatment of burns or abrasions and for delicate lacerations of the upper eyelid. Such anesthesia is ideal for removing small pieces of glass that are embedded in the forehead from a windshield injury (Fig. 31-19).

Anatomy

The subtle supraorbital notch, which is in line with the pupil (when the patient is staring straight ahead), may be palpated along the superior orbital rim. This landmark is the site of injection for blockage of the supraorbital nerves. The supratrochlear nerve is found 0.5 to 1.0 cm medial to the notch. The infratrochlear nerve is not usually blocked but is found in the most medial aspect of the superior orbital rim. If the anesthetic is placed on the forehead proper, this block may not produce complete anesthesia of the skin of the upper eyelid if the sensory branches to the eyelid are given off before the supraorbital nerve transverses the forehead.

Approach

With the patient in the supine position, a skin wheal is raised. Paresthesias in the form of an electric shock sensation over the forehead are sought; these ensure a successful nerve block. One to 3 mL of anesthetic are placed in the area of the supraorbital notch. A finger or a roll of gauze should be held firmly under the orbital rim to avoid ballooning of anesthetic into the upper eyelid (Fig. 31-20 *A, B* (Figure Not Available)).

If paresthesias cannot be elicited or if the nerve block is unsuccessful, a line of anesthetic solution placed along the orbital rim from the lateral to the medial aspect will ensure block of all of the branches of the ophthalmic nerve (Fig. 31-21).

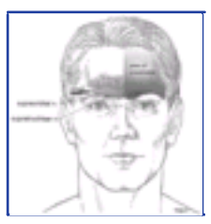

Figure 31-21 A field block of the forehead will anesthetize both the lateral and medial branches of the supraorbital nerve as well as the supratrochlear nerves. The resultant area of anesthesia is represented by the shaded area of the left forehead.

Complications

Hematoma formation or swelling of the eyelid may occur but requires only local pressure. Occasionally, ecchymosis of the periorbital region will appear the next day, and the patient should be warned of this possibility.

Although this block is infrequently used, it is easily performed and is not associated with serious side effects. Its use should be considered when anesthesia of the forehead or the anterior scalp is desired.

CONCLUSION

Nerve blocks about the head and neck are relatively painless when done carefully and slowly following topical mucosal anesthesia (for intraoral approaches) or local skin anesthesia (for extraoral blocks and approaches). Patients who appear anxious may benefit from sedation prior to attempting these blocks (see Chapter 34). These blocks should not be attempted in the uncooperative patient.

Acknowledgments

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Chapter 32 - Nerve Blocks of the Thorax and Extremities

John J. Kelly
Mark Spektor

The use of nerve blocks is an integral part of the practice of emergency medicine. This chapter provides technical guidance for the performance of commonly used nerve blocks.

While virtually every peripheral nerve can be blocked at some point along its course from the spine to the periphery, digital nerve blocks of the fingers and toes are more commonly used than proximal blocks. The reasons for this are unclear but likely include a lack of experience on the part of the clinician, the time required for a block to take effect, and occasional failure to obtain adequate anesthesia. However, with proper training and experience, nerve blocks of the thorax and proximal extremities can be very useful tools for emergency clinicians. Potential applications include femoral blocks for femur fractures, ankle blocks for treatment of foot injuries and infections, intercostal blocks for rib fractures, and wrist blocks for injuries to the palm.

The preparation, technique, choice of anesthetic, precautions, and complications are similar for all nerve blocks and are described in general in the following sections. The clinician is encouraged to use the same basic techniques and precautions for all nerve blocks. Specific precautions unique to a particular nerve block are included with the description of that block; obvious precautions, such as aspiration before injection when the needle is in close proximity to a vascular structure, are not restated to avoid redundancy.

GENERAL CONCEPTS

Indications

For most of the lacerations and injuries seen in the emergency department (ED), local infiltrative anesthesia is adequate and more efficient than using a nerve block (see Chapter 30). Local infiltration is quick, reliable, and effective compared with many of the nerve blocks, which tend to require a more extensive setup and have a less reliable and longer onset. Furthermore, those patients who require extensive repair and anesthesia of the entire extremity are often referred to a specialist, who may prefer to examine an unanesthetized limb.

In general, a nerve block is indicated when it will provide advantages over other techniques. Scenarios in which this requirement is met include the following:

- When distortion from local infiltration hampers closure (e.g., facial wounds) or may compromise blood flow (e.g., fingertip)
- When anesthesia is required over a large area and multiple injections would be painful, or the large amount of anesthetic needed for local infiltration exceeds the recommended dose
- When a nerve block is the most efficacious form of treatment, as in an intercostal block for treating a rib fracture in a patient with chronic obstructive pulmonary disease
- When local infiltration of the wound would be more painful than a regional nerve block, such as in the plantar surface of the foot or the palm of the hand
- When the block is performed in order to decrease pain during finger or toe dislocation reduction
- When extensive limb surgery or manipulation is required (e.g., extensive tendon repair) and other options are not available

Preparation

A brief history, including drug allergies, medications, and systemic illnesses should be taken from the patient. Specific history about allergies to local anesthetics may be prudent to elicit. Peripheral vascular, heart, and liver disease may increase the risk of severe complications; therefore, information about the existence of these diseases should also be sought.

Instructions

The procedure should be explained to the patient, including the pain of the needle insertion, paresthesias that may be felt, and possible complications that may occur. *The possible need for additional anesthetic or alternate procedures if the initial nerve block fails should also be discussed beforehand.* The patient should understand that the additional administration of anesthetic is part of the normal procedure rather than an attempt to correct an incomplete nerve block. It is not standard to obtain written informed consent for the nerve blocks performed in the ED.

Equipment

The degree of equipment preparation depends on the extent of the procedure. For a simple digital block, a 10-mL syringe, an 18-ga needle for drawing the solution from the vial, and a 3.75-cm, 25- or 27-ga needle for the nerve block will suffice. Note that the needle sizes given in the text are general recommendations but for the majority of blocks, a 25-ga needle is ideal. For more elaborate blocks, the necessary equipment is listed in Table 32-1 . Use of an extension tubing set between the needle and a stopcock syringe assembly facilitates independent needle placement and syringe manipulation. In addition, standard resuscitation equipment for advanced cardiac life support should be readily available any time local anesthetic agents are given.

Choice of Anesthetic

The factors influencing the choice of anesthetic agent for nerve block are similar to those for local infiltration (see Chapter 30). In general, most nerve blocks are done for the repair of painful traumatic injuries that are likely to cause pain long after the repair is complete. In such cases, anesthetics with the longest duration of action should be selected to maximize the patient's analgesia. For most of the blocks described in this chapter, 0.25% bupivacaine is suggested as the anesthetic of choice, but equal volumes of 1% lidocaine with epinephrine can be substituted. The use of epinephrine on end-organ areas is generally discouraged (e.g., toes, fingers) although the theoretical risk is largely unsubstantiated in clinical practice (see chapter 30). Higher concentrations of lidocaine (up to 2%) or bupivacaine (0.5%) are commonly used for large nerves. Ropivacaine is a relatively new amide anesthetic with a rapid onset and long duration of action (several hours). It has been reported to have fewer cardiotoxic and central nervous system effects than bupivacaine. [1] [2] Care

TABLE 32-1 -- Equipment Needed for Proximal Nerve Block Trays

6 gauze sponges
4 towels
1 antiseptic-solution receptacle
1 receptacle for saline flush solution
1 anesthetic-solution receptacle (30-mL capacity)
1 10-mL syringe for local anesthetic injection
1 30-mL syringe for nerve block injection
1 three-way stopcock
1 IV extension tubing set
1 18-ga needle for withdrawing anesthetic from the vial
1 each 3.75-cm 23-, 25-, and 27-ga needles for nerve blocks

must always be taken to *avoid exceeding* the recommended dosages of anesthetic.

Buffering the anesthetic will lessen the pain of infiltration. Buffered lidocaine has been shown to be less painful and equally as efficacious as plain lidocaine in digital blocks.[3] One part bicarbonate to 10 parts lidocaine is usually used. Heating the anesthetic from 37°C to 41°C also reduces the pain of digital blocks. [4] Slower rates of injection have also been found to significantly reduce discomfort. [5]

Positioning of the Patient

Ideally, nerve blocks should be performed with the patient in the supine position. This enhances patient comfort and prevents the unexpected vasovagal syncope that may occur when the patient is in an upright position. Vasovagal syncope may be induced from the pain of the injection or "needle phobia." For this reason, the clinician may draw the anesthetic from the vial, taking care that the patient does not see the needle. In addition, common distraction techniques have shown to help in

reducing anxiety and discomfort.[6]

Preparation of the Area to Be Blocked

To limit the incidence of infection, the field should be prepared in an aseptic fashion before needle puncture. This can be accomplished by swabbing the area with a povidone-iodine solution or alcohol and then using a standard aseptic injection technique. The antiseptic solution should be allowed to dry fully to achieve maximal antibacterial effect. Sterile drapes and gloves are not routinely required but may be considered in addition to aseptic skin preparation for the initiation of blocks that (1) are close to large joints, vessels, and nerves; (2) are located in inherently contaminated areas of the body (e.g., groin, perineum); or (3) require simultaneous palpation of the underlying structures while injecting.

Choosing the Nerves to Block

Successful anesthesia requires appropriate knowledge of anatomy. Most areas to be anesthetized have overlapping sensory innervation. Therefore, most cases require two or more nerves to be blocked. In addition, the cutaneous distribution of the various peripheral nerves differs slightly from patient to patient. A liberal margin of error should be used when determining which nerves supply the desired area of anesthesia.

Locating the Nerve

When locating a nerve to be blocked, it is best to approach it from a site with easily identifiable anatomic landmarks. The best sites are those with good structural landmarks (e.g., prominent bones or tendons) immediately next to the nerve. For example, the digital nerves are reliably found at the 2, 4, 8, and 10 o'clock positions around and just superficial to the proximal phalanx, and the median nerve lies between the palpable palmaris longus and flexor carpi radialis tendons at the proximal crease of the wrist. Nerves that course adjacent to easily palpable arteries such as in the axilla and groin are also easy to locate and are good sites for performing nerve blocks. Nerves that do not have adjacent structural or vascular landmarks are much more difficult to block.

Blocking nerves with good structural or vascular landmarks is straightforward: The landmarks are palpated, the course of the nerve in relation to those landmarks is visualized in the mind's eye, and the needle is inserted in close proximity to the nerve.

Blocking those nerves with poor landmarks, such as the radial nerve at the elbow, requires skill through practice, some degree of luck, or a nerve stimulator if such nerves are to be blocked consistently.

Nerve Stimulator

Although not standard or easy to find in any ED, a nerve stimulator is inexpensive and relatively simple to use. With minimal practice, the needle can be placed easily and reliably within a few millimeters of the nerve. The device is simple, consisting of a battery-operated nerve stimulator that delivers current in the range of 0.1 to 0.5 mA, a disposable Teflon-coated needle, and an electrocardiogram pad. One wire electrode from the nerve stimulator is attached to the electrocardiogram pad, which is placed on the limb approximately 10 to 15 cm from the injection site. The other wire electrode is attached to the Teflon-coated needle at its hub. Once inserted into the tissue, the needle tip is electrically active. When in close proximity to the nerve, electric current stimulates the nerve, producing twitching of the muscles supplied by that nerve. For example, when the ulnar nerve is stimulated, the small and ring fingers flex. Radial nerve stimulation would cause extension of the fingers or wrist, depending upon how distal the stimulation takes place. Anesthetic solution can then be injected into the area at the stimulation site.

Paresthesia

When a nerve stimulator is not available, another useful technique to ensure that the needle tip is in close proximity to the nerve is to elicit a paresthesia. By touching and mechanically stimulating the nerve with movement of the needle tip, a tingling sensation or jolt known as a *paresthesia* is felt along the distribution of the nerve. In practice, the jolt of a true paresthesia is often difficult to distinguish from the "ouch" of a pain-sensitive structure. When blocking proximal nerves of the elbow or axilla, the paresthesia travels far enough away from the injection site that it can be distinguished from locally induced pain. Paresthesias at the level of the hand and wrist are much less reliably distinguished from pain. In both cases, the paresthesia is a subjective feeling that requires intelligent and cooperative patients who understand what they are expected to feel and who remain relaxed and attentive so that they are able to distinguish an "ouch" from a jolt. All too often, the patient in pain is willing to tell the clinician what the clinician wants to hear: The sensation that just made the patient jump was a "paresthesia." Before the procedure, a simple

explanation of what the patient should or may feel will facilitate cooperation.

It should also be pointed out that while generally reliable in showing that the needle is close to its target, some authors feel that eliciting paresthesia may theoretically increase the rate of complications due to mechanical trauma or intraneural injection.[7] [8] [9]

Injecting the Anesthetic

One strives to ensure that the anesthetic agent is not inadvertently injected into the vessels or nerve bundle. In practice, such a misplaced intravascular injection is of minimal consequence; however, small amounts of epinephrine may cause systemic symptoms, such as tachycardia or anxiety. Nerve bundle injection has the potential to cause nonspecific nerve injury. Intra-arterial injection, theoretically, is more dangerous than intravenous injection. Before injection, the syringe is aspirated to check for blood. If no blood is aspirated, the anesthetic is injected while the extremity is observed for blanching, which suggests intravascular injection. If blanching occurs, the needle should be repositioned before further injection. The onset and duration of anesthesia are greatly influenced by the proximity of the injected anesthetic to the nerve. Onset is within a few minutes if the anesthetic is in immediate proximity to the nerve. Onset takes longer or may not occur if the anesthetic must diffuse more than 2 to 3 mm, which underscores the importance of locating the nerve before injection.

More anesthetic is required if it must diffuse a large distance to the nerve. A range of suggested volumes of anesthetic is given with each nerve block description. For blocks in which a definite paresthesia is elicited or a nerve stimulator is used, the minimal recommended amount of anesthetic suffices. For many of the blocks of the smaller nerves, paresthesias are not easily elicited, and the anesthetic is placed in the general vicinity of the nerve. For these blocks, or when doubt exists about proximity of the needle to the nerve, larger amounts of anesthetic are recommended. This point cannot be emphasized strongly enough. *The difference between a successful and unsuccessful block may be merely an additional 2 mL of anesthetic.* When in doubt, err on the high side of the recommended dosage. For large nerve blocks, many clinicians opt for 2% lidocaine, rather than the 1% solution that is adequate for most ED nerve blocks.

For most blocks, the onset of anesthesia occurs in 2 to 15 minutes, depending on the distance the anesthetic must diffuse to the nerve, and the type of anesthetic used. One should wait 30 minutes before deciding that the block was unsuccessful.

Complications and Precautions

Complications may result from poor peripheral nerve block technique. General precautions include measures to minimize nerve injury, intravascular injection, and systemic toxicity.

Complications are rare in clinical practice. No actual statistics exist on the complication rate from nerve blocks performed by emergency clinicians. Generally, infrequently performed blocks, blocks that require high doses of anesthetic, and blocks close to major vascular structures are more likely to have complications. The technical ability of the clinician largely determines the outcome.

Nerve Injury

Nerve injury is rare but can occur secondary to (1) chemical irritation from the anesthetic, (2) direct trauma from the needle, or (3) ischemia due to intraneural

injection. Overall, the incidence of serious neuronal injury is rare, occurring in 1.9 per 10,000 blocks. [10] Given that placement of a nerve block is a blind procedure, complications do not necessarily represent an error in technique.

Neuritis, an inflammation of the nerve, is the most common nerve injury.[8] [9] The patient may complain of pain and varying degrees of nerve dysfunction, including paresthesia or motor or sensory deficit. Most cases are transient and resolve completely. Supportive care and close follow-up are the mainstays of treatment. Concentrated anesthetics can produce a chemical irritation of the nerve. Emergency clinicians should not exceed recommended doses and concentrations of anesthetic (Table 32-2). In general, however, lidocaine 1% or 2% or bupivacaine 0.25% or 0.5% are safe for nerve blocks performed by the emergency clinician.

Direct nerve damage can be minimized by proper needle style, positioning, and manipulation. A short beveled needle should be used and maneuvered so that the bevel is parallel to the longitudinal fibers. Sharp pain or paresthesia indicates

TABLE 32-2 -- Recommended Volumes of Anesthetic for Various Nerve Blocks

Nerve	Volume (mL)
Axillary	40–50*
Elbow	
Ulnar	5–10*
Radial	5–15*
Median	5–15*
Wrist	
Ulnar	5–15*
Radial	5–15*
Median	3–5*
Hip	
Femoral	10–30*
3-in-1	30–50*
Knee	
Tibial	5–15*
Peroneal	5–10*
Saphenous	5–10*
Ankle	
Posterior tibial	5–10*
Deep peroneal	3–5*
Saphenous, sural, and superficial peroneal	4–10*
Intercostal	5–15*
Hand	
Metacarpal and web space	2–4†
Finger	1–2†
Foot	
Metatarsal	10–15†
Web space	3–5†
Toe	2–5†

Note: For most nerve blocks performed in the emergency department, 1% lidocaine or 0.25% bupivacaine is adequate. It is also acceptable to use 2% lidocaine or 0.5% bupivacaine for larger nerves (femoral, wrist, and ankle blocks). If the stronger concentrations are used, the volume in the table should be halved.

* Anesthetic: 1% lidocaine or 0.25% bupivacaine (both *with* epinephrine).
† Anesthetic: 1% lidocaine or 0.25% bupivacaine (both *without* epinephrine).

that the needle is close to or in the nerve. Excessive needle movement should be avoided when the needle tip is contacting a nerve. If a 25-ga needle is used, physical damage to a nerve should be minimal, even when directly touched by the needle tip. A 27-ga needle is theoretically attractive but this size limits aspiration testing and may break off in tissue.

Intraneural injection may rarely cause nerve ischemia and injury. Elicitation of a paresthesia or severe pain suggests that the needle has made contact with the nerve. *When a paresthesia is elicited, the needle must be withdrawn 1 to 2 mm before the anesthetic is injected.* If the paresthesia occurs during injection, the needle must be repositioned. Most neurons are surrounded by a strong perineural sheath through which the nutrient arteries run lengthwise. Injection directly into a nerve sheath may increase the pressure within the nerve and compress the nutrient artery. Impaired blood flow results in nerve ischemia and subsequent paralysis. Intraneural injection is often heralded by severe pain, which worsens with further injection and may radiate along the course of innervation. The operator may notice difficulty depressing the syringe plunger. If the needle tip is in proper position, slow injection of the anesthetic should be minimally painful, and the anesthetic should go in without resistance.

Intravascular Injection

Intravascular injection may rarely result in both systemic and limb toxicity. Inadvertent intravascular injection produces high blood levels of the anesthetic, with resultant toxicity, as discussed earlier. Particular care must be taken when administering large amounts of anesthetic in close proximity to large blood vessels.

Intra-arterial injection of anesthetic with epinephrine may cause peripheral vasospasm that further compromises injured tissue. Intravascular anesthetic is not toxic to the limb itself, although it may produce transient blanching of the skin by displacing blood from the vascular tree. Epinephrine, however, can cause a prolonged vasospasm and subsequent ischemia if it is injected into an artery. This is especially worrisome when anesthetizing areas with little collateral circulation, such as toes, fingers, penis, and tip of the nose. Severe epinephrine-induced tissue blanching or vasospasm may be reversed with local or intravascular injection of phentolamine.

Although vasospasm associated with epinephrine in anesthetic solutions used for nerve blocks is rare, experience in related clinical situations can help guide therapy. Roberts and Krisanda used a total of 5 mg of phentolamine infused intra-arterially to reverse arm ischemia following 3 mg of epinephrine inadvertently administered into the brachial artery during cardiac resuscitation. [11] Digital ischemia from inadvertent epinephrine autoinjection has been treated by both proximal "digital block"

with 2 mg of phentolamine[12] and by local infiltration at the ischemic site with 1.5 mg of phentolamine. [13]

The route of phentolamine administration may be guided by the clinical situation. Phentolamine must reach the site of vasospasm. Arterial injection has the advantage of delivering the medication directly to the spasmed arteries. Local infiltration may be effective for ischemia of a single toe or finger. For larger areas of involvement or in instances where local infiltration is ineffective, intra-arterial injection should be used. A dose of 1.5 to 5 mg appears to be effective in most cases, [11] [12] [13] although a total of 10 mg may be used for local infiltration. Phentolamine 5 mg can be mixed with 5 to 10 mL of either normal saline or lidocaine. The small volume of the distal pulp space may limit the infiltration dose volume to 0.5 to 1.5 mL in the fingertip. Larger volumes and dosages can be used in proximal infiltrations. For intra-arterial infusion at the radial artery in the wrist or the dorsalis pedis at the ankle, dosages of 1.5 to 5 mg of phentolamine are suitable. Slow infusion or graded dosages of 1 mg may provide enough phentolamine to reverse ischemia without excessive systemic effects such as hypotension.

Hematoma

Hematoma formation may result from arterial puncture, particularly during blocks where a major blood vessel is being used as a landmark to locate the nerve (e.g., axillary or femoral artery). Direct pressure for 5 to 10 minutes usually controls further bleeding. Use of small-gauge needles (e.g., 25- to 27-ga) minimizes bleeding from the punctured artery.

Infection

Infection is rare and can be minimized by following aseptic technique and using the lowest possible concentration of epinephrine. Injection should be made through non-infected skin that has been antiseptically prepared. Injection through a site of infection may spread the infection to adjacent tissues, fascial planes, and joints. This is particularly a concern in the hand and foot. See Chapter 30 for details.

Systemic Toxicity

The incidence of systemic toxicity to local anesthetics has significantly diminished in the past 30 years. Interestingly, peripheral nerve blocks have been reported to have the highest incidence of systemic toxicity.[10] Allergic reactions account for only 1% of untoward reactions.[14] See Chapter 30 for details.

Limb Injury

Injury to the anesthetized limb can result if the patient is permitted to use the limb or is advised to use heat or cold application or to perform wound care before the anesthesia has worn off. With major nerve blocks, the patient should not be released home until sensation and function have returned. With minor blocks, the patient may be sent home but should be properly cautioned. Care must be taken to avoid ischemia-producing compression dressings (e.g., elastic bandages), because the anesthetized area may not sense impending problems.

SPECIFIC NERVE BLOCKS

Intercostal Nerve Block

Blocking the intercostal nerves produces anesthesia over an area of their cutaneous distribution (Fig. 32-1), providing considerable pain relief for patients with rib contusion or rib fractures. Rib fractures are typically quite painful, causing the patient to splint respirations to avoid excessive movement of the injured site. The resulting hypoventilation, atelectasis, and poor expectoration may cause hypoxia or lead to pneumonia. This is particularly true in patients with preexisting pulmonary disease and minimal respiratory reserve, in whom further impairment of function may cause significant respiratory compromise.

Theoretically, anesthetizing injured ribs eases pain and facilitates deep breathing and coughing. Unfortunately, there are no controlled studies comparing intercostal blocks with oral analgesics in patients with the kinds of rib fractures that are commonly managed on an outpatient basis. However,

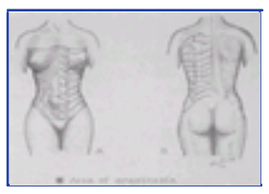

Figure 32-1 Area of anesthesia and cutaneous distribution of the intercostal nerve. *(From Moore DC: Regional Block: A Handbook for Use in the Clinical Practice of Medicine and Surgery, 4th ed. Springfield, IL, Charles C Thomas, 1971, p 159. Reproduced with permission.)*

studies do suggest that intercostal blocks may be superior to analgesics in patients who have undergone thoracotomies. [15] [16] [17] In these studies, those receiving intercostal nerve blocks had better results on pulmonary function tests, greater oxygenation, and earlier ambulation and discharge than those receiving opioid analgesics.

There are several arguments against the routine use of intercostal nerve blocks in the ED. First, rib fractures are often tolerated well in young patients, who usually require minimal oral analgesics. Second, these blocks have a relatively short duration of action. The typical duration of action of a long-acting anesthetic with epinephrine is 8 to 12 hours. However, it should be noted that patients often receive partial analgesia for up to 3 days, a period of time that cannot be attributed to the direct action of the anesthetic on the nerve. Perhaps the anesthesia reduces muscle spasm and the associated cycle of pain.

Finally, a perceived high incidence of pneumothorax and unsuccessful blocks deter many clinicians from performing intercostal nerve blocks in the ED. The true incidence of pneumothorax following intercostal nerve blocks is very low and not significant enough to prohibit the procedure. Moore reported that in more than 10,000 individual rib blocks performed, the incidence of pneumothorax was <0.1%. [18] However, Shanti and associates reported that the incidence of pneumothorax was 1.4% for each individual intercostal nerve blocked. [19] If more than one nerve requires blockade, the incidence of pneumothorax may be greater. The suggested approach to discussing intercostal blocks is to give patients the facts with regard to duration of analgesia and possible complications and then allow them to decide on the method for themselves. Often they prefer oral analgesics initially but may return for further relief of pain, at which time they are more amenable to the nerve block.

Anatomy

Each thoracic nerve exits the spine through the intervertebral foramen, which lies midway between adjacent ribs. It immediately gives off the posterior cutaneous branch, which supplies the skin and muscles of the paraspinal area. The intercostal nerve then continues around the chest wall and gives off the lateral cutaneous branches at the midaxillary line. These branches are the sensory supply to the anterior and posterior lateral chest wall.

The intercostal nerve runs with the vein and artery in the subcostal groove (Fig. 32-2). Posteriorly, the nerve is separated from the pleura and the lungs by the thin intercostal fascia. Particular care must be taken to avoid puncture of the thin fascia and underlying lung when blocking the nerve in the posterior aspect of the back. Fortunately, most rib fractures occur in the anterior or lateral portion of the ribs and can be blocked in the posterior axillary line, where the internal intercostal muscle lies between the nerve and the lung's pleura and provides a buffer for minor errors in needle placement.

Technique

For adequate analgesia of most rib fractures, the lateral cutaneous branch needs to be anesthetized. Blocks are usually performed between the posterior axillary and midaxillary line at a point proximal to the origin of this branch. Explanation of the procedure, its benefits, and its risks, including potential pneumothorax, systemic toxicity, and ineffective block, should be done before proceeding.

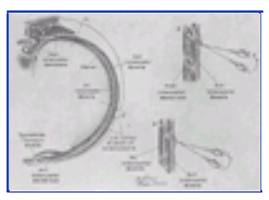

Figure 32-2 Cross section of the chest wall, demonstrating branches of the intercostal nerve, as well as sections of the chest wall at points between which the intercostal block is usually performed: at the posterior angle of the ribs (*A*) and at the midaxillary line (*B*). A block situated at the posterior axillary line is common (see text). *(From Moore DC: Regional Block: A Handbook for Use in the Clinical Practice of Medicine and Surgery, 4th ed. Springfield, IL, Charles C Thomas, 1971, p 146. Reproduced with permission.)*

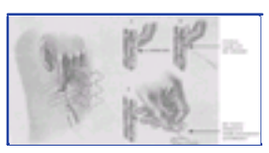

Figure 32-3 Method of retracting skin and the proper needle insertion site for intercostal block. See text for details. *(From Moore DC: Regional Block: A Handbook for Use in the Clinical Practice of Medicine and Surgery, 4th ed. Springfield, IL, Charles C Thomas, 1971, p 153. Reproduced with permission.)*

A 10-mL syringe with a 3.75-cm, 25-ga needle is used. The rib is palpated, and the area is prepared in the usual aseptic manner. The index finger of the non-dominant hand is used to retract the skin at the lower edge of the rib cephalad and up over the rib (Fig. 32-3). With the syringe in the opposite hand, the skin is punctured at the tip of the finger that is retracting the skin. The syringe is held at an 80° angle with the needle pointing cephalad. The hand holding the syringe rests on the chest wall for stability. In this position, the depth of needle penetration is well controlled. The needle is slowly advanced until it comes to rest on the lower

border of the rib.

At this point, the skin retraction is released. The skin returning to its natural position moves the needle shaft perpendicular to the chest wall and the needle tip to the inferior margin of the rib. The syringe is shifted from the dominant hand to the index and thumb of the non-dominant hand. The middle finger of the same hand rests against the shaft of the needle and, by exerting gentle pressure on the shaft, walks the needle off the lower edge of the rib. Again, the palm of the hand is planted firmly on the chest wall to ensure control of the needle. With the help of the dominant hand, the needle is slowly advanced 3 mm. The needle is aspirated, and then 2 to 5 mL of anesthetic are injected while the needle is carefully moved in and out 1 mm, which ensures that the compartment containing the nerve between the internal and external intercostal muscles is penetrated. This may also serve to minimize intravascular injections. The procedure is repeated ideally on the *two ribs above and below* to ensure that the overlapping innervation from adjacent nerves is blocked.

Although the procedure just discussed seems extensive, it takes 1 to 2 minutes to perform once the operator is familiar with the technique, and 3 to 5 intercostals can be blocked in 10 minutes total time.

Precautions

The needle must be initially placed at the lower edge of the rib. If it contacts the rib above this point, it cannot be walked off the lower edge of the rib at the proper angle. If it is inserted too low, over the intercostal space, it may be advanced too deep through the pleura and into the lung before the operator realizes the misplacement. Before inserting the needle, the depth of the bone should be estimated. If the bone is not encountered by this depth, needle position should be reevaluated. Even after the needle has been properly walked off the edge of the rib, care must be taken to avoid puncture of the pleura and lung. The depth of the intercostal groove in which the nerve runs is 0.6 cm posteriorly, diminishing to 0.4 cm anteriorly.

Because the incidence of pneumothorax is low, a chest radiograph is not routinely required after this procedure. The asymptomatic patient is observed for 15 to 30 minutes and instructed to return if problems arise. If the patient has symptoms of pneumothorax (e.g., cough, a change in the nature of the pleuritic pain, or shortness of breath), he or she should have a chest film taken before discharge.

If the clinician inadvertently causes a pneumothorax, treatment depends on the size. Many pneumothoraces are small. Those <20% may be observed for 6 hours. [20] During this time the patient may be administered high concentration oxygen to help decrease the size of the pneumothorax. If the pneumothorax does not grow in size, the patient may be released home with arrangements for follow-up. Needle or catheter aspiration of larger pneumothoraces may be all that is needed. A chest tube is necessary if this method fails.

Nerve Blocks of the Upper Extremity

The upper extremity is supplied by the brachial plexus. Its branches—primarily the median, radial, ulnar, and musculocutaneous nerves—can be blocked at the axilla, elbow, wrist, hand, or fingers. Nerve blocks at the axilla and elbow are seldom used in the ED. Nerve blocks of the wrist are performed occasionally before painful procedures or for repair of injuries to the hand. Metacarpal and digital blocks are used frequently to treat fractures, lacerations, and infections of the fingers.

Nerve Blocks at the Elbow

The median, ulnar, and radial nerves can be blocked at the elbow, providing anesthesia to the distal forearm and hand (Fig. 32-4). For most injuries extensive enough to require nerve block at the elbow, all three nerves must be blocked for successful anesthesia because of the variable and overlapping innervation of the forearm. Furthermore, injuries to the proximal and middle forearm may require additional circumferential subcutaneous (SQ) field blocks of the lateral, medial, and posterior cutaneous nerves.

Ulnar nerve: Anatomy and technique.

The ulnar nerve can be palpated in the ulnar groove on the posteromedial aspect of the elbow between the olecranon and medial condyle of the humerus (Fig. 32-5 and Fig. 32-6). This nerve supplies the innervation to the small finger and ulnar half of the ring finger and the ulnar aspect of the hand (see Fig. 32-4).

With the elbow flexed, the nerve is palpated in the groove. A 3.75-cm, 25-ga needle is inserted 1 to 2 cm *proximal to the groove* and directed toward the groove parallel to the course of the nerve. The needle tip comes to rest close to the proximal end of the groove. Care is taken to avoid blocking the nerve in the groove, where it is prone to damage. For similar reasons, paresthesia may be elicited but is not vigorously sought. Although an elbow ulnar nerve block is common, many physicians prefer to block the ulnar nerve at the wrist to limit the risk of injury. Once the needle tip is properly positioned, 5 to 10 mL of anesthetic are deposited. If a nerve stimulator is used, flexion of the small and ring fingers signals proximity to the nerve.

Radial nerve: Anatomy and technique.

The radial nerve and sensory branch of the musculocutaneous nerve run together in the sulcus between the biceps and the brachioradialis muscles on the anterolateral aspect of the elbow (Fig. 32-7). The block produces anesthesia to the lateral dorsum of the hand and the lateral aspect of the forearm (see Fig. 32-4).

The sulcus in which the nerve runs is palpated between the sharp border of the biceps muscle and the medial border of the brachioradialis muscle in the *antecubital fossa* just proximal to the skin crease of the elbow. Palpation is greatly facilitated by having the patient, with the elbow flexed at 90°, contract and relax these muscles isometrically so that their borders are better defined. The skin is punctured with a 3.75-cm, 25-ga needle halfway between the muscles, or 1 cm lateral to the biceps tendon, at a point 1 cm proximal to the antecubital crease. After proper needle insertion, 5 to 15 mL of anesthetic should be injected at this depth. Because of poor landmarks and the depth of the radial nerve at the elbow, the nerve stimulator greatly facilitates the search for the nerve, which, when stimulated, produces extension of the fingers and wrist.

Median nerve: Anatomy and technique.

The median nerve runs medial to the brachial artery in the anteromedial aspect of the elbow (Fig. 32-8 and Fig. 32-9). The nerve block

Figure 32-4 Cutaneous nerve supply of the right upper limb. *(From Bridenbaugh LD: The upper extremity: Somatic blockade. In Cousins M, Bridenbaugh PO [eds]: Neural Blockade in Clinical Anesthesia and Management of Pain, 2nd ed. Philadelphia, JB Lippincott, 1988, p 412. Reproduced with permission.)*

anesthetizes the index, middle, and radial portion of the ring fingers and the palmar aspect of the thumb and lateral palm (see Fig. 32-4).

The brachial artery is palpated in the flexed arm at the elbow just proximal to the antecubital crease and medial to the prominent biceps tendon. Once the anatomy is defined and marked in the flexed arm, the arm is extended to 30°. A 3.75-cm, 25-ga needle is inserted slightly medial to the artery and perpendicular to the skin to the depth of the artery, about 2 to 3 cm, and 5 to 15 mL of anesthetic are injected. Again, the nerve stimulator facilitates the process and produces flexion of the wrist and

index finger. Most commonly, median nerve blocks occur at the wrist.

Nerve Blocks at the Wrist

The median, ulnar, and radial nerves may be blocked at the wrist, providing anesthesia to the hand. Most extensive injuries and procedures for which a wrist nerve block could be used can also be managed using local infiltration or a digital block. Compared with direct infiltration, wrist block anesthesia can have a slow and unreliable onset and can require more time to take effect if all three nerves are to be blocked. There are several circumstances, however, in which wrist nerve blocks are more advantageous than other types of blocks or anesthesia.

Diffuse lesions that can be difficult to anesthetize with local infiltration can easily be anesthetized with a wrist block. Deep abrasions with embedded debris, commonly the result of "road burn" from bike and motorcycle crashes, can be cleaned and debrided painlessly after a nerve block at the wrist. Hydrofluoric acid burns, which require treatment with numerous SQ injections of calcium gluconate, are better handled after a wrist nerve block. Wrist blocks are also advantageous in the severely swollen and contused hand, where small amounts of anesthetic injected locally may increase the tissue pressure and produce further pain. Deep lacerations of the palm are very painful to anesthetize with local infiltration and will also benefit from a wrist block. Additionally burns of the hand lend themselves to nerve blocks prior to debridement.

Compared with nerves in the axilla and elbow, the nerves in the wrist are more easily located anatomically and can be blocked more reliably. All three nerves lie in the volar aspect of the wrist near easily palpated tendons. A nerve stimulator is not necessary but may be useful in locating the nerves, particularly when one is learning how to perform these blocks.

The anatomy and technique for blocking each nerve follow. Note that the median nerve lies in the midline and deep to the fascia, and the ulnar and radial nerves lie on their respective sides and have branches that wrap around dorsally. Blocking all three nerves at the wrist requires a block that, when viewed end-on, roughly resembles a horseshoe straddling a horseshoe stake (see Fig. 32-5).

Median nerve: Anatomy and technique.

In the wrist, the median nerve lies just below the palmaris longus tendon or slightly radial to it between the palmaris longus and the flexor carpi radialis tendons (see Fig. 32-5 and Fig. 32-6). Both tendons are easily palpated, but the palmaris longus may be absent in up to 20% of patients, in which case the nerve is found about 1 cm in the ulnar direction from the flexor carpi radialis tendon. The nerve lies deep to the fascia of the flexor retinaculum, but at a depth of 1 cm or less from the skin. The superficial position of the median nerve at the wrist is emphasized, since a major cause of failure of this block is too deep instillation of the anesthetic.

The palmaris longus tendon is located by having the patient oppose the thumb and small finger with the wrist flexed against resistance. The site of the nerve block is

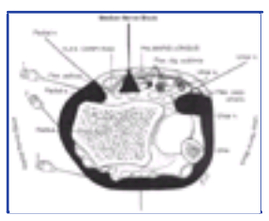

Figure 32-5 Cross section of the wrist looking cephalad. The arrow points to the (covered) median nerve. Shaded triangle depicts the area infiltrated with anesthetic. Note the relatively superficial position of the median nerve, just radial to the palmaris longus. *(Redrawn from Adriani J: Labat's Regional Anesthesia: Techniques and Clinical Applications. 3rd ed. Philadelphia, WB Saunders, 1967, p 234. Reproduced with permission.)*

selected on the radial border of the palmaris longus tendon just proximal to the proximal wrist crease. A 3.75-cm, 25-ga needle is inserted perpendicularly and advanced slowly until a slight "pop" is felt as the needle penetrates the retinaculum and a paresthesia is produced. If no paresthesia ensues, it may be elicited in a more ulnar direction under the *palmaris longus* tendon. If a paresthesia is still not elicited, 3 to 5 mL of anesthetic are deposited in the proximity of the nerve at a depth of 1 cm under the tendon. Although the nerve is surprisingly close to the skin, it is better to err slightly on the deep side of the retinaculum and continue depositing anesthetic

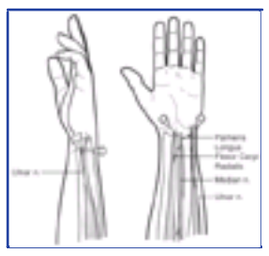

Figure 32-6 Landmarks and anatomy of the median and ulnar nerve block. The median nerve is more superficial than often expected. It lies to the radial side of the palmaris tendon.

as the needle is withdrawn, because the retinaculum is an effective barrier to a successful nerve block from a superficially injected anesthetic.

Radial nerve: Anatomy and technique.

The radial nerve follows the radial artery into the wrist but gives off branches proximal to the wrist. These branches wrap around the wrist and fan out to supply the dorsal radial aspect of the hand (Fig. 32-10).

Nerve block requires an injection in close proximity to the artery and a field block that extends around the dorsal aspect of the wrist. A 3.75-cm, 25-ga needle is inserted immediately lateral to the palpable artery at the level of the proximal palmar crease. At the depth of the artery 2 to 5 mL of anesthetic are injected. Another 5 to 6 mL are distributed in an SQ field block from the initial point of injection to the dorsal midline. The needle must be withdrawn and repositioned to complete the block. The discomfort of numerous needle sticks is decreased if the needle is repositioned to a site that has been anesthetized previously.

Ulnar nerve: Anatomy and technique.

The ulnar nerve follows the ulnar artery into the wrist, where they both lie deep to the *flexor carpi ulnaris* tendon (see Fig. 32-5 and Fig. 32-6). The *flexor carpi ulnaris* tendon is easily palpated just proximal to the prominent pisiform bone by having the patient flex the wrist against resistance. At the level of the proximal palmar crease, the artery and the nerve lie just off the radial border of the *flexor carpi ulnaris* tendon; however, the nerve lies between the tendon and the artery and deep to the artery, making it difficult to approach the nerve from the volar aspect of the wrist without involving the artery.

Nerve block of the ulnar nerve can be carried out by two different approaches: lateral and volar (see Fig. 32-6); the lateral approach may be easier because of the reason stated previously. For the lateral approach, a 3.75-cm, 25-ga needle is inserted on the ulnar aspect of the wrist at the proximal palmar crease and is directed horizontally under the *flexor carpi ulnaris* for a distance of 1.0 to 1.5 cm. At this point 3 to 5 mL

Figure 32-7 Ulnar nerve block at the elbow. *A,* Positioning patient for ulnar nerve block at the elbow. The patient is supine, the elbow flexed, and the medial epicondyle and ulnar groove are exposed. *B,* The ulnar nerve passes between the olecranon (*) and medial epicondyle (•) of the humerus. Avoid injecting large amounts of anesthesia directly into the ulnar groove. Instead, place anesthetic proximal to the groove. Alternatively, the ulnar nerve may be blocked at the wrist.

of anesthetic are deposited. The needle is directed toward the ulnar bone at a point deep to the *flexor carpi ulnaris* tendon, and the anesthetic can be inserted as the needle is withdrawn.

Like the radial nerve, cutaneous nerves branch off the ulnar nerve, wrap around the wrist, and supply the dorsum of the hand. These are blocked with a 5- to 6-mL SQ band of anesthetic from the lateral border of the *flexor carpi ulnaris*

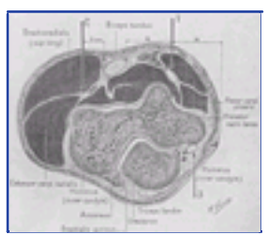

Figure 32-8 Cross section of the elbow, demonstrating (1) the median nerve, (2) the radial nerve, and (3) the ulnar nerve. *(From Adriani J: Labat's Regional Anesthesia: Techniques and Clinical Applications. 3rd ed. Philadelphia, WB Saunders, 1967, p 234. Reproduced with permission.)*

tendon to the dorsal midline (see Fig. 32-6). Another advantage of the lateral approach is that the dorsal branches can be blocked from the same injection site.

Nerve Blocks of the Digits

The digital nerve block is one of the most useful and most used blocks in the ED. Indications for choosing it include repair of finger lacerations and amputations, reduction of fractures and dislocations, drainage of infections, removal of fingernails, and relief of pain (e.g., from a fracture or burn). The digital block is superior to local infiltration in most circumstances. Wound infiltration may be a problem in the finger that has tight skin and can accept only a limited volume of anesthetic. Administration of anesthetic into this restricted space increases the tissue pressure, impairing capillary blood flow and causing pain. *Fibrous septa* in the fingertip also restrict the space available for the injected substance and even limit the spread of small amounts of anesthetic.

Anatomy.

Each finger is supplied by two sets of nerves. These nerves, the dorsal and palmar digital nerves, run alongside the phalanx at the 2 and 10 o'clock positions and the 4 and 8 o'clock positions, respectively (Fig. 32-11*A–D* Fig. 32-11*A–D*).

The principal nerves supplying the finger are the palmar digital nerves, also called the *common digital nerves*. These nerves originate from the deep volar branches of the ulnar and median nerves, where they branch in the wrist. The palmar digital nerves follow the artery along the volar lateral aspects of the bone, one on each side, and supply sensation to the volar skin and interphalangeal joints of all five digits. *In the middle three fingers, these nerves also supply the dorsal distal aspect of the finger, including the fingertip and nailbed.* While many clinicians routinely block both sets of digital nerves, only the volar (palmar) branches must be blocked to obtain adequate anesthesia of the middle three fingers distal to the distal interphalangeal joint.

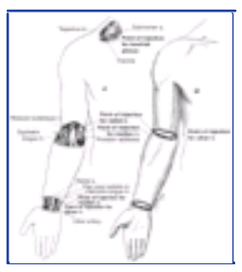

Figure 32-9 Anatomy for nerve blocks at the elbow and wrist. Black circles on skin indicate site of puncture. *(From Adriani J: Labat's Regional Anesthesia: Techniques and Clinical Applications, 3rd ed. Philadelphia, WB Saunders, 1967, p 226. Reproduced with permission.)*

The dorsal digital nerves originate from the radial and ulnar nerves, which wrap around to the dorsum of the hand. They supply the nailbeds of the thumb and small finger and the dorsal aspect of all five digits up to the distal interphalangeal joints. Unlike the middle three fingers, which require blocking of only the two volar (palmar) digital nerves, all four nerves are usually blocked in the thumb and fifth finger, especially to obtain anesthesia of the fingertip and nailbed (Fig. 32-12*A* and *B*).

Technique.

The digital nerves can be blocked anywhere in their course, including sites in the finger, in the web space between the fingers, and between the metacarpals in the hand. There are a variety of approaches to the nerves, including the dorsal and palmar approaches and the web space approach. Each has its merits. The technique is similar at each level.

The dorsal approach has the advantage of thinner, less pain-sensitive skin compared with volar approaches. The hand can be held firmly and flat on the table, preventing withdrawal. The disadvantage is that two injections are needed from this approach to block both volar digital nerves.

The dorsal approach can be used in the dorsum of the hand at the metacarpals, just proximal to the finger webs at the proximal end of the proximal phalanx, or distal to the web. Clinical situations may dictate which site to use; however,

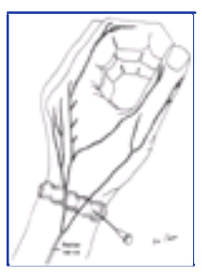

Figure 32-10 Distribution, anatomy, and blockade of the radial nerve at the wrist.

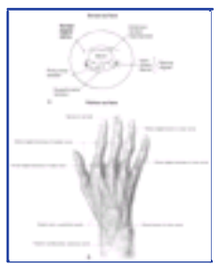

Figure 32-11a *A,* Schematic cross section of the phalanx, demonstrating the relationship of the nerves to the bone. If the anatomy is examined (*B* and *C*), it is obvious that in order to perform a digital block of the thumb and fifth finger, all four digital nerves (two volar, two palmar) must be blocked. Since almost the entire second, third, and fourth fingers are supplied by only the palmar branch (note that each palmar nerve curves around to the dorsum from the palmar surface), only the palmar nerves must be blocked to obtain anesthesia for all but the skin of the proximal dorsal digit. The exception is the ulnar side of the fourth finger, which receives some of its distal dorsal innervation from the dorsal nerve. *D,* On the palmar aspect, the digital nerves are almost adjacent to the flexor tendon, a position that is more palmar than is usually appreciated. *(From Grant's Atlas of Anatomy, 9th ed. Baltimore, Williams & Wilkins, 1978. Reproduced with permission.)*

given equal circumstances, the preferred site is just proximal to the finger web. Here the nerve's location is more consistent than in the hand, and there is more soft tissue space to accommodate the volume of injected substance than there is in the distal finger. Digital block at the web is more efficacious in onset and requires less time to anesthesia than metacarpal block done proximal to the metacarpophalangeal joint. [21]

Digital block requires only aseptic injection technique after the skin is prepared with alcohol or povidone-iodine. Sterile gloves and drapes are not necessary, although examination gloves are recommended.

The clinician must first decide whether two or four digital nerves require blocking (see earlier discussion). Anesthesia is deposited at the positions of the appropriate digital nerves (2, 4, 8, and 10 o'clock in relationship to the bone), using a 3.75-cm, 25- or 27-ga needle (see Fig. 32-11*A*). The block is performed from the dorsal surface, where the skin is thinner, easier to penetrate, and less sensitive than that of the volar surface. The needle insertion site is at the web space, just distal to the knuckle at the lateral edge of the bone (Fig. 32-12*C*). Once the needle tip is subdermal, it usually contacts the bone. A wheal of 0.5 to 1 mL of anesthetic

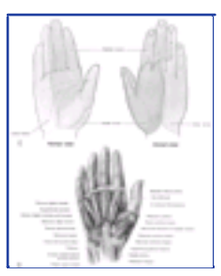

Figure 32-11b

without epinephrine is injected at this level. This serves to block the dorsal digital nerve and provide anesthesia at the injection site. The needle is then passed lateral to the bone and toward the palmar surface until the palmar skin starts to tent slightly. The needle is withdrawn 1 mm and aspirated to check for an inadvertent IV position, and 0.5 to 1.5 mL of anesthetic are injected. This procedure is repeated on the opposite side of the finger. The result is a circumferential band of anesthesia at the base of the finger. *Firm massage of the injected area* for 15 to 30 seconds enhances diffusion of the anesthetic through the tissue to the nerves.

A variation of the dorsal approach is performed as follows: After injecting one side of the finger, the needle is redirected (without removing it) across the top of the digit to anesthetize the skin on the opposite side (Fig. 32-12*D*). The needle is then withdrawn and inserted at the site that was anesthetized, and the block is continued as described earlier. The presumed advantage of this method is that it minimizes the pain of the second skin puncture. This procedure requires that the needle be placed across the dorsal aspect of the finger, allowing the possible disadvantage of extensor tendon puncture and trauma.

The *palmar* and *web space* approaches can be used most successfully for the middle three fingers where only a single puncture is required to block both volar nerves. This technique takes advantage of the anatomic fact that only the volar digital nerves must be blocked to obtain anesthesia of the total finger (except the proximal dorsal surface). If the thumb or fifth finger must be anesthetized, the dorsal branches must also be blocked to obtain anesthesia of the fingertip and fingernail area.

The palmar approach requires an injection in the palm, which is slightly more painful than an injection in the dorsal skin. The needle is inserted directly over the center of the metacarpal head, and anesthetic is slowly injected while the needle is advanced to the bone. At this point the needle is withdrawn 3 to 4 mm and angled slightly to the left and right of center to block both digital nerves without withdrawing the

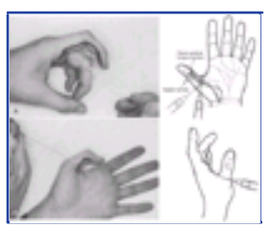

Figure 32-12a *A,* Each finger has four digital nerves: two palmar and two dorsal nerves. The palmar nerves travel in a line connecting the top of the skin creases made by flexing the proximal interphalangeal and distal interphalangeal joints *(black line)*. The nerves are more palmar than often appreciated, and are almost adjacent to the flexor tendon, so injecting the true lateral portion of the finger may miss the nerve. If the anesthesia needle is inserted at the tip of the skin creases (see probe) the nerve will be blocked. *B,* When performing a digital nerve block on the thumb, all four digital nerves must be blocked. As in the other fingers, the palmar nerve travels in a line made by connecting the top of the skin creases produced by flexing the digit *(black line)*. The anesthetizing needle (see probe) must be placed almost adjacent to the flexor tendon to block the nerve. *C,* Sites of digital nerve blocks at the base of the finger. *D,* Technique of anesthetizing the second injection site. *(From Adriani J: Labat's Regional Anesthesia: Techniques and Clinical Applications, 3rd ed. Philadelphia, WB Saunders, 1967, p 445. Reproduced with permission.)*

needle (Fig. 32-13 and Fig. 32-14). To be successful, a palpable soft tissue fullness should be appreciated. The technique requires 4 to 5 mL of anesthetic.

A variation of this is particularly useful in fingertip injuries in toddlers. The finger is pinched side-to-side just distal to the proximal finger crease. This tents the skin at the finger crease, which is injected subcutaneously with 0.5 to 1 mL of anesthetic. This single injection diffuses to the volar nerves and provides anesthesia to many fingertip injuries common in toddlers. With the web space approach, the patient's hand is held by the clinician in such a way that the clinician's thumb and index finger are over the dorsal and volar metacarpal head, respectively. The clinician's third finger is used to separate the patient's fingers to expose the web space, while the fourth and fifth fingers support the finger being anesthetized (Fig. 32-15). The needle is inserted into the web space, 1 mL of anesthetic is injected, and the needle is slowly advanced until it is next to the lateral volar surface of the metacarpal head. Anesthetic is injected, and the needle is advanced slowly past the midline of the metacarpal head to the opposite digital nerve (Fig. 32-16). The operator's index finger is used to palpate a fullness as the anesthetic is injected. By redirecting the needle to the adjacent finger without withdrawing it, both fingers may be blocked with a single puncture.

Although epinephrine-containing solutions are used routinely in digital blocks of the toes by podiatrists, without morbidity, when performing a digital block, the authors advise use of anesthetics that do *not contain epinephrine*. The injection should go in smoothly, without resistance of the syringe plunger. Although the finger is forgiving of transient pressure from excessive anesthetic, if the injection site becomes excessively tense digital perfusion may be compromised. This concern is heightened when epinephrine-containing solutions are used. If epinephrine-containing solutions are inadvertently used for a digital block in otherwise healthy individuals without peripheral vascular disease, it is unlikely that serious ischemic injury will occur. Significant vasoconstriction generally lasts less than 60 minutes, within the time interval for which an ischemic tourniquet can safely be used in the same area. [22] However, if the entire digit remains blanched for more than 15 minutes, it is prudent to reverse the a-agonism of epinephrine with phentolamine. Using a pulse oximeter on the affected finger may help quantitate the degree of ischemia.

Figure 32-12b

Figure 32-13 Palmar approach to the metacarpal head. After needle puncture in the midline, the needle is directed slightly to the right and left, and anesthetic is deposited along the course of both volar digital nerves. The needle is not withdrawn until both nerves are blocked. This technique blocks only the palmar nerves.

Onset of anesthesia occurs in 1 to 15 minutes and lasts for 20 minutes to 6 hours, depending on the anesthetic agent used.

Alternative techniques

Jet injection technique.

Jet injection for digital nerve block can be used effectively and is less painful than standard needle techniques. [23] The technique described by Ellis uses 0.15 mL of 1% lidocaine delivered by a jet injector at 2600 psi. Three injections are given to the lateral aspect of the proximal phalanx: the first, midway between the volar and dorsal surfaces; the second, dorsal to this; the third, volar. A combined total of 0.45 mL is administered to each side of the phalanx at the 2, 3, and 4 o'clock positions and the 8, 9, and 10 o'clock positions in relationship to the bone.

The potential disadvantages of jet injection include lacerations that may occur with tangential injection. Holding the injector perpendicular to the skin avoids this problem. Thick skin associated with older age, manual labor, and male gender may require larger volumes of anesthetic.

The advantages of this technique are less pain of injection and avoidance of "needle phobia," particularly in children.

Transthecal digital block technique.

The transthecal block is performed by a single injection into the flexor tendon sheath, which produces rapid and complete finger anesthesia. It was first described by Chiu in 1990 after noting rapid finger anesthesia following injection treatment of a trigger finger. [24]

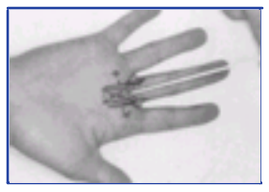

Figure 32-14 The palmar digital nerves (*N*) are adjacent to the head of the metacarpal (*MC*) in the palm. For a metacarpal head block the anesthetizing needle punctures the palm in the middle of the MC (see probe), and anesthetic is deposited both laterally and medially, blocking both nerves with a single puncture. Injection in this area is usually more painful than injecting in the web space, but it is almost foolproof for obtaining anesthesia.

Cadaver studies suggest that injected fluid diffuses out of the tendon sheath and around the phalanx and all four digital nerves.

The flexor tendon is palpated in the palm proximal to the metacarpal phalangeal joint. A 25-ga needle is attached to a 3-mL syringe and introduced at a 45° angle as it is advanced to the sheath/tendon (Fig. 32-17). Slight pressure is applied to the syringe plunger. If the sheath has been entered, the anesthetic should flow freely. If it does not, it is presumed that the tendon has been entered, and the syringe is withdrawn slowly

Figure 32-15 The palmer web space metacarpal head block allows two of the three inner fingers to be anesthetized with a single needle puncture. The clinician's index finger palpates the patient's metacarpal head on the patient's palm while the injured finger is supported and the web space is exposed. The index finger can feel the tissue distention by the anesthetic, but care must be taken to avoid passing the needle through the skin and puncturing the operator's finger. About 3 to 5 mL of anesthetic is deposited. To anesthetize the adjacent finger the needle is partly withdrawn and redirected to the other metacarpal head. Bending the needle to 30° allows easier access to the proper position without the syringe getting in the way.

Figure 32-16 Nerve block of the third digit. After injection of anesthetic into the web space skin, the needle is advanced to the digital nerve, where it passes just lateral to the volar metacarpal head. Anesthetic is injected, and the needle is advanced to the opposite digital nerve. This procedure requires about 3 to 5 mL of anesthetic. If the index finger also must be blocked, the needle is redirected without withdrawal from the skin. Thus, both fingers are blocked with a single needle puncture.

while constantly applying slight pressure. A total of 2 mL of anesthetic solution is injected. Smaller volumes are used in children. After the needle is removed, pressure is applied over the tendon proximally to facilitate distal spread. Average onset of anesthesia is 3 minutes. [25]

The advantage of this technique is the single injection. However, Hill and colleagues found the technique to be "clinically equal" to traditional digital blocks. [26] Other authors have stated that traditional digital block was easier to administer and produced less pain during and after injection. [27] Theoretically, the technique may increase the risk of injury to the tendon.

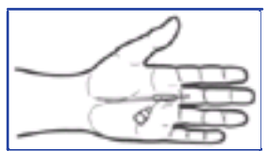
Figure 32-17 Transthecal block. The flexor tendon sheath is entered volarly just proximal to the metacarpophalangeal joint. With the use of a 25-ga needle on a 3-mL syringe the fluid should flow easily. Inject 2 mL of anesthetic and apply proximal tendon sheath pressure.

Complications and precautions.

The small size of the digital arteries and nerves makes intravascular or intraneural injection less likely. Inadvertent intravascular injection may cause digit ischemia from vasospasm or displacement of blood out of the capillary bed by the anesthetic. Blanching of the finger as the anesthetic is injected suggests intravascular injection. If this is observed, the injection should be discontinued. Usually the ischemia is transient and self-resolving, and serious complications are rare. Massage or topical application of nitroglycerin paste may be attempted if ischemia persists. Although the incidence of vasospasm and resultant ischemia is rare and primarily occurs in patients with underlying vascular disease, [28] an anesthetic agent without epinephrine is commonly recommended. As noted earlier, if one mistakenly chooses an epinephrine-containing solution and vasospasm develops, the ischemia should be relieved with local infiltration of phentolamine.

Commonly the digital nerve is lacerated or damaged by the initial injury to the finger. Careful evaluation using 2-point discrimination should be performed to determine the extent of nerve injury before nerve block. Even if nerve injury is questionable, it should be documented in the chart, and the *patient should be advised of the injury before the nerve block*. Careful evaluation and patient education should prevent misconceptions as to the cause of the nerve injury. Although most isolated digital nerve injuries are not debilitating, they heal slowly and can be annoying to the patient. Digital nerve injury proximal to the distal interphalangeal joint may be repaired surgically. Nerve repair may be immediate when specialty consultation is available or delayed following initial simple closure.

Nerve Blocks of the Lower Extremity

Metatarsal and digital blocks in the foot are used frequently to treat ingrown toenails, fractures, and lacerations of the forefoot and toes. However, similar to the upper extremities, *proximal* lower extremity blocks are often overlooked as an anesthetic choice. This is unfortunate, since treatment of many conditions will benefit from these blocks. For example, femoral nerve blocks can provide excellent analgesia for femoral shaft fractures, and nerve blocks at the ankle are an excellent method to obtain anesthesia for the treatment of foot lacerations and to perform otherwise painful procedures on the foot. The lower extremity is supplied by five nerves, the branches of which can be blocked at the hip, knee, ankle, foot, or toes.

Nerve Blocks of the Hip

Nerve blocks of the femoral, obturator, lateral femoral cutaneous, posterior cutaneous, and sciatic nerves at the hip provide anesthesia and paralysis to the skin and muscles of the leg, respectively (Fig. 32-18). These nerves are not contained in a single fascial sheath (as in the upper extremity); therefore local anesthetic injections of both the sciatic and femoral nerves are required for complete proximal lower extremity nerve blocks. Posteriorly, the posterior cutaneous and sciatic nerves can be blocked together with a single injection. Anteriorly, the femoral, obturator, and lateral femoral cutaneous nerves can be blocked by three separate injections or by a single injection using the "3-in-1" technique described by Winnie and colleagues. [29] In this technique a large amount of anesthetic is injected into the femoral nerve sheath and permitted to track into the pelvis to the point at which the anterior nerves run in a common sheath. [29]

Femoral nerve blocks alone may be the analgesia of choice for femoral shaft fractures. Studies supporting the femoral block in the treatment of femoral shaft fractures date back to 1940 and continue to appear. [30] [31] [32] In a study by McGlone and colleagues, patients preferred femoral block to opioid analgesia; however, this means of analgesia has yet to be adopted as routine treatment for femoral shaft fractures. [31] The procedure requires only femoral nerve blockade and does not rely on the performance of a successful 3-in-1 block.

Anatomy.

Anteriorly, the femoral, obturator, and lateral femoral cutaneous nerves arise from the lumbar plexus and travel a short distance in a common nerve sheath (Fig. 32-19). In the pelvis, the lateral femoral cutaneous and obturator nerves exit the sheath. The lateral femoral cutaneous nerve supplies the sensory innervation to the lateral thigh. The obturator nerve supplies the sensory innervation to the anterior medial thigh and the motor supply to the adductor muscles. The femoral nerve continues as the saphenous nerve, which supplies the sensory innervation to the medial calf and ankle.

Posteriorly, the sciatic and posterior cutaneous nerves arise from the lumbar and sacral plexuses and exit the pelvis together in the sciatic notch. The posterior cutaneous nerve supplies sensory innervation to the posterior thigh and buttocks. The sciatic nerve does not supply the thigh but at the knee divides into the tibial and peroneal nerves and supplies much of the calf and foot. The tibial nerve supplies the muscles of the calf and sensation to the plantar surface of the foot. The peroneal nerve supplies sensation to the lateral aspect of the calf and dorsal and plantar aspects of the foot. Both the tibial and peroneal nerves give off branches that form the sural nerve, which supplies sensation to the lateral aspect of the foot (Fig. 32-20*A* and *B*).

Technique for femoral nerve block.

With the patient in the supine position, the skin is prepared in an aseptic fashion. The femoral artery is palpated 1 to 2 cm below the inguinal ligament (see Fig. 32-19), and a wheal of anesthesia is raised immediately lateral to the artery. With the nondominant hand placed firmly on the artery, a 1.25-cm, 22-ga needle, attached to an extension tube setup and a 20-mL syringe, is inserted adjacent to the artery at a 90° angle to the skin and underlying vessels. The needle is advanced until a paresthesia is elicited or the needle pulsates laterally, indicating a position immediately adjacent to the artery. If a paresthesia is felt or the needle is assumed to be in the immediate vicinity of the nerve, 10 to 20 mL of anesthetic are injected. If a paresthesia is not elicited, 10 to 20 mL of anesthetic can be injected in a fan-like pattern lateral to the artery in a blind attempt to anesthetize the femoral nerve. *Given the proximity of this nerve to the femoral artery and vein, aspiration before injection is essential to reduce the risk of intravascular injection.* The onset of anesthesia should occur in 15 to 30 minutes and should last for 3 to 8 hours.

Technique for the 3-in-1 block (also called the inguinal perivascular block).

The 3-in-1 technique differs from the femoral nerve block in that it requires a larger amount of anesthetic solution and compression distal to the injection site. In the thigh, the femoral nerve is sandwiched between the *quadratus lumborum* and *iliopsoas* muscles, whose fasciae are continuous with a nerve sheath that contains all three nerves higher in the pelvis (see Fig. 32-19). A large amount of anesthetic properly placed next to the femoral nerve tracks

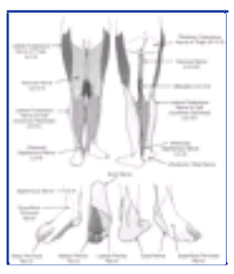
Figure 32-18 Cutaneous distribution of the nerves to the lower extremity. *(From Bridenbaugh PO: The lower extremity: Somatic blockade. In Cousins M, Bridenbaugh PO [eds]: Neural Blockade in Clinical Anesthesia and Management of Pain. 2nd ed. Philadelphia, JB Lippincott, 1988, p 425. Reproduced with permission.)*

back along the sheath and anesthetizes all three nerves (see Fig. 32-20*A*).

The 3-in-1 block requires precise injection of the anesthesia into the nerve sheath. In the 3-in-1 block, once the needle is in the correct position, the fingers palpating the artery are removed and placed gently but firmly distal to the needle while 20 to 30 mL of anesthetic are injected (see Fig. 32-20*B*). Distal pressure should be maintained for 5 minutes while the anesthetic diffuses proximally. It usually takes about 30 minutes to achieve full sensory effect of the 3-in-1 block. [33]

A nerve stimulator facilitates the search for the nerve and placement of the needle tip in the fascial sheath. A thick insulating fascia covers the nerve. When the needle punctures this sheath, the nerve is stimulated at currents of 0.5 mA or less.

Techniques for sciatic nerve block.

Labat's classic technique is performed with the patient in the lateral (Sims) position with the extremity to be blocked up, flexed, and resting on the lower dependent extremity. A line is constructed from the greater trochanter to the posterior superior iliac spine. The line is bisected and a perpendicular line is drawn from the midpoint over the gluteal muscle. A second line is drawn between the greater trochanter and sacral hiatus, and the line intersects the previous line marking the point of needle insertion. Using a 10-cm needle, 30 mL of local anesthetic are injected after the nerve is located. [34] It may take 30 minutes for complete anesthesia (Fig. 32-21).

Precautions.

The femoral nerve block and sciatic nerve block require that large amounts of anesthetic be injected close to large nerves and vessels. Standard precautions should be used to avoid intravascular and intraneural injection, and recommended anesthetic dosages should be observed. Hematoma formation is a potential complication—especially in the gluteal muscle with sciatic block.

Nerve Blocks of the Ankle

Nerve block of the five nerves of the ankle—the deep peroneal (anterior tibial), posterior tibial, saphenous, superficial peroneal (musculocutaneous), and sural nerves—provides anesthesia to the foot. Depending on the desired area of anesthesia, one or more of the five nerves are blocked. These blocks can be used in operative procedures and repair of injuries to the foot. They are particularly useful in providing anesthesia to

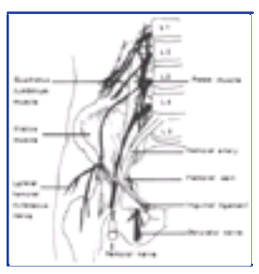

Figure 32-19 Anatomy of the femoral, obturator, and lateral femoral cutaneous nerves and the location of the injection site for the femoral and 3-in-1 nerve blocks. *(Adapted from Winnie AP, Ramamurthy S, Durrani Z: The inguinal paravascular technique of lumbar plexus anesthesia: The "3 in 1 block." Anesth Analg 52:989, 1973. Reproduced with permission.)*

the sole of the foot for laceration repair and foreign body removal.

A nerve block of the foot is better tolerated by the patient than local infiltration in all but the most minor procedures. The skin of the sole is thicker and more tightly bound to the underlying fascia by connective tissue septa than is skin in other parts of the body. Puncturing this skin can be difficult and is always quite painful. The fibrous septa can limit the amount and spread of anesthetic. If large amounts of anesthesia are injected, the volume of injected substance quickly exceeds the space available, possibly leading to painful distention of the tissue and circulatory compromise of the microvasculature. Local infiltrative anesthesia is adequate for treating minor injuries in which only small amounts of anesthetic are needed. For treatment of larger injuries, including incision and drainage, extensive wound care, and foreign body removal, the ankle block is better tolerated.

Anatomy.

The foot is supplied by the five nerve branches of the principal nerve trunks (see Fig. 32-18). Three nerves are located anteriorly and supply the dorsal aspect of the foot. Two nerves are located posteriorly and supply the volar aspect.

The anteriorly located nerves are the superficial peroneal, deep peroneal, and saphenous nerves. The superficial peroneal nerve (also called the *dorsal cutaneous* or *musculocutaneous nerve*) actually consists of multiple branches that supply a large portion of the dorsal aspect of the foot (Fig. 32-22). These are located superficially between the *lateral malleolus* and *extensor hallucis longus* tendon, which is easily palpated by having the patient dorsiflex the big toe. The deep peroneal nerve (also called the *anterior tibial nerve*) supplies the web space between the big and second toes. In the ankle, it lies under the *extensor hallucis longus* tendon. The saphenous nerve runs superficially with the saphenous vein between the *medial malleolus* and *tibialis anterior* tendon, which is

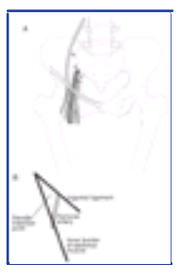

Figure 32-20 *A, B,* 3-in-1 block. See text for details.

prominent when the patient dorsiflexes the foot. The saphenous nerve supplies the medial aspect of the foot near the arch.

The posteriorly located nerves are the posterior tibial and sural nerves. The sural nerve runs subcutaneously between the *lateral malleolus* and the Achilles tendon and supplies the lateral border, both volar and dorsal, of the foot (see Fig. 32-22). The posterior tibial nerve runs with the posterior

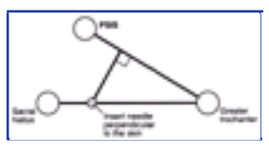

Figure 32-21 Sciatic nerve block (posterior approach). A line is drawn from the greater trochanter to the posterior-superior iliac spine (PSIS). The line is bisected and the needle will be inserted perpendicular to the line at a depth determined by a second line between the greater trochanter and the sacral hiatus.


Figure 32-22 *A* and *B,* Anatomy and innervation of the left calf and foot. See Figure 32-18 for areas for innervation. *(From Shurman DH: Ankle-block anesthesia for foot surgery. Anesthesiology 44:348, 1976. Reproduced with permission.)*

tibial artery, which can be palpated between the *medial malleolus* and the Achilles tendon. It lies slightly deep and posterior to the artery.

The posterior tibial nerve is one of the major nerve branches to the foot. After passing through the ankle, it branches into the medial and lateral plantar nerves, which supply sensation to most of the volar aspects of the foot and toes and supply motor innervation to the intrinsic muscles of the foot.

Technique.

Complete nerve block of the foot requires blocking three SQ nerves and two deeper nerves (Fig. 32-23). Once familiar with the anatomy, the experienced clinician can anesthetize all 5 nerves quickly by placing SQ band blocks around 75% of the ankle circumference and 1 deep injection next to the palpable posterior tibial artery and the other under the extensor tendon of the big toe.

The five nerves of the foot are commonly blocked in combinations of two or more. Small procedures clearly within the distribution of one nerve may require only a single nerve block; however, overlap of the nerve's sensory distribution frequently necessitates blocking a number of nerves for adequate anesthesia. Nerve block of the sural and posterior tibial nerves together anesthetizes the bottom of the foot and is the most useful combination.

Posterior tibial.

The posterior tibial nerve is blocked in the medial aspect of the ankle between the *medial malleolus* and the Achilles tendon. The injection site is determined by palpating the tibial artery just posterior to the *medial malleolus*. A point 0.5 to 1.0 cm superior to this is marked. If the artery is not palpable, a site 1 cm above the *medial malleolus* and just anterior to the Achilles tendon is used (see Fig 32-23 and Fig 32-24).

A 3.75-cm, 25-ga needle is directed at a 45° angle to the mediolateral plane (the needle is almost perpendicular to the skin), just posterior to the artery. At the estimated depth of the artery, approximately 0.5 to 1.0 cm deep, the needle is wiggled slightly in an effort to produce a paresthesia. If the paresthesia is elicited, 3 to 5 mL of anesthetic are injected after careful aspiration to check for inadvertent intravascular placement of the needle tip. If no paresthesia is produced, the needle is advanced inward, again at a 45° angle, until it hits the posterior aspect of the tibia. The needle is then withdrawn slightly, about 1 mm, and 5 to 7 mL of anesthetic are injected while the needle is withdrawn another 1 cm. A rise in the temperature of the foot, due to vasodilation from loss of sympathetic tone, may herald a successful block.

Sural nerve.

The sural nerve is blocked on the lateral aspect of the ankle between the Achilles tendon and the *lateral malleolus* (see Fig. 32-23 and Fig. 32-24). It lies superficially and is blocked at a level about 1 cm above the *lateral malleolus*. A band of anesthesia is injected subcutaneously between the Achilles tendon and the *lateral malleolus* using 3 to 5 mL of anesthetic.

Superficial peroneal nerves.

The superficial peroneal nerves are blocked on the anterior aspect of the ankle between the *extensor hallucis longus* tendon and the *lateral malleolus*. They lie superficially and are blocked using 4 to 10 mL of anesthetic placed subcutaneously in a band between these landmarks (see Fig. 32-23 and Fig. 32-24).

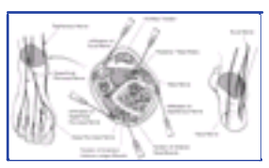
Figure 32-23 Anatomy and injection sites for nerve blocks at the ankle. *(From Bridenbaugh PO: The lower extremity: Somatic blockade. In Cousins M, Bridenbaugh PO [eds]: Neural Blockade in Clinical Anesthesia and Management of Pain, 2nd ed. Philadelphia, JB Lippincott, 1988, p 435. Reproduced with permission.)*

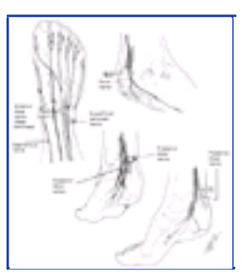
Figure 32-24 Anatomy and injection sites for nerve blocks at the ankle (lateral views). *(Adapted from Locke RK, Locke SE: Nerve blocks of the foot. JACEP 4:698, 1976.)*

Deep peroneal nerve.

The deep peroneal nerve is blocked anteriorly beneath the *extensor hallucis tendon* (see Fig. 32-23 and Fig. 32-24). It is blocked at a level 1 cm above the base of the *medial malleolus* and between the *extensor hallucis longus* and anterior tibial tendons. The tendons are palpated by having the patient dorsiflex the big toe and foot, respectively. After an SQ wheal is placed, the needle is directed about 30° laterally and under the *extensor hallucis* tendon until it strikes the tibia (at a depth of <1 cm). The needle is withdrawn 1 mm, and 1 mL of anesthetic is injected.

Saphenous nerve.

The saphenous nerve is blocked anteriorly between the *medial malleolus* and the anterior tibial tendon. It lies superficially and is blocked with 3 to 5 mL of anesthetic injected subcutaneously between these landmarks.

Nerve Blocks of the Metatarsals and Toes

Like nerve blocks in the hand and fingers, the nerve blocks in the foot and toes are commonly used in the ED. Indications for using these blocks include repair of lacerations, drainage of infections, removal of toenails, manipulation of fractures and dislocations, and otherwise painful procedures requiring anesthesia to the forefoot and toes.

Digital nerve blocks in the foot and toes are superior to local infiltration anesthesia in all but the most minor procedures. In the toes, the limited SQ space does not accommodate enough injected material for adequate infiltrative anesthesia. Furthermore, the fibrous *septa*, which attach the volar skin to the underlying fascia and bone, limit the spread and volume of injected substances. On the plantar surface, even small amounts of local infiltrate can cause painful distention and local ischemia of the tissues.

Anatomy.

Each toe is supplied by two dorsal and two volar nerves. These nerves are branches of the major nerves of

the ankle. The dorsal digital nerves are the terminal branches of the deep and superficial peroneal nerves. The volar nerves are branches of the posterior tibial and sural nerves.

The location of the nerves in relation to the bones varies with the site of the foot. In the toes, the nerves lie at the 2, 4, 8, and 10 o'clock positions in close relationship to the bone. In the proximal foot, the nerves run with the tendons and are not in close relationship to the bones (Fig. 32-25).

Technique.

The digital nerves can be blocked at the metatarsals, interdigital web spaces, or toes. The bones of the foot can be palpated easily from the dorsum and are used as the landmarks for estimating the location of the nerves. Proximally, the nerves' relationship to the bones is less consistent, making definitive needle placement and successful block less reliable. In the toes, the position of the nerves is more consistent; however, there is minimal SQ tissue space available for the injected solution. At the web space, the nerves are located in close relationship to the bone, and there is ample space for injecting the anesthesia; hence, for most procedures, the web space is the preferred site for the digital nerve block.

The technique for toe and metatarsal blocks is similar. All four nerves supplying each toe are usually blocked because of their sensory overlap. The blocks are performed from the dorsal surface, where the skin is thinner and less sensitive than that on the plantar aspect. A total of 5 mL of anesthetic is deposited in a fan-like pattern in the space between the metatarsal bones (see Fig. 32-25). A 1-mL skin wheal is placed dorsally between the metatarsal bones. The needle is then advanced until the volar skin tents slightly, and 2 mL are injected as the needle is withdrawn. Without removing it, the needle is redirected in a different volar direction, and the procedure is repeated. A total of 5 mL is used in each

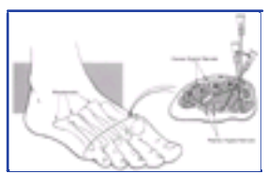

Figure 32-25 Anatomy and technique for digital nerve block at the metatarsals. *(From Bridenbaugh PO: The lower extremity: Somatic blockade. In Cousins M, Bridenbaugh PO [eds]: Neural Blockade in Clinical Anesthesia and Management of Pain, 2nd ed. Philadelphia, JB Lippincott, 1988, p 437. Reproduced with permission.)*

metatarsal space. Again, because of sensory overlap, two or more spaces need to be anesthetized for each toe to be blocked.

For the web space block, a site on the dorsum just proximal to the base of the toe is selected. Using a 10-mL syringe, a 3.75-cm, 27-ga needle is inserted at the lateral edge of the bone (Fig. 32-26). A wheal is placed subcutaneously between the skin and bone, using 0.5 to 1.0 mL of anesthetic. This serves to block the dorsal nerve and minimize pain at the needle insertion site. The needle is then advanced just lateral to the bone and toward the sole until the needle tents the volar skin slightly. The needle is withdrawn 1 mm, and 0.5 to 1.0 mL is injected. As the needle is withdrawn, another 0.5 mL is injected to ensure a successful block. The procedure is repeated on the opposite side of the toe. In this manner, two columns of anesthesia are placed on each side of the toe in the area through which the four digital nerves run. A total of 2 to 4 mL of anesthetic is used. For blocks done in the toe itself, the procedure is the same, but smaller amounts of anesthetic (i.e., <2 mL) are used because of the limited SQ space and fear of vascular compression. Alternative techniques using a single injection site, as described for the finger, can be performed (Fig. 32-27).

Complications and precautions.

The precautions that apply to the hand and fingers apply to the foot and toes. Ischemic complications can be avoided by paying attention to skin changes during the injection. Blanching heralds possible intravascular injection or vascular compression. If the skin blanches, halt the procedure and reevaluate the position of the needle and the amount and content of the injected solution. The total volume of anesthesia should not exceed the recommended amount. Although podiatrists commonly use anesthetics containing small concentrations of epinephrine

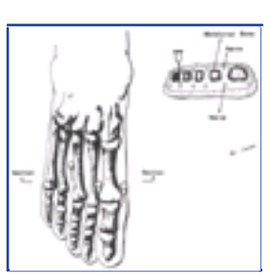

Figure 32-26 Technique of digital nerve blocks in the interdigital web spaces of the foot.

Figure 32-27 Alternative techniques of digital nerve blocks in the toes. *(From Locke RK, Locke SE: Nerve blocks of the foot. JACEP 4:698, 1976. Reproduced with permission.)*

for digital blocks in patients without underlying vascular disease, [16] this practice is generally not recommended because of concern about vasospasm and ischemia (see Complications and Precautions subsection and Chapter 30).

Note any neural or vascular injuries before the injection. The close proximity of these structures to the skin and bones means that they are frequently injured. Deficits, even if questionable, should be documented in the records and brought to the attention of the patient before the nerve block.

CONCLUSION

Regional nerve blocks of the thorax and extremities are valuable adjuncts to the clinician's armamentarium. They may be used for wound anesthesia before exploration, irrigation, debridement, and repair. They may also be used to reduce the pain of certain procedures (e.g., arthrocentesis, fracture reduction, or dislocation manipulation) and may permit better thoracic mechanics (e.g., intercostal nerve block). When these procedures are performed, be mindful of the cautions outlined here and in Chapter 30 .

Acknowledgment

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Chapter 33 - Intravenous Regional Anesthesia*

James R. Roberts

The clinical use of intravenous (IV) regional anesthesia has been well established [1] [2] [3] as a safe, quick, and effective alternative to general anesthesia in selected cases requiring surgical manipulation of the upper and lower extremities. Although often relegated to the operating room, the procedure is readily applicable to outpatient use. In the emergency department (ED) the technique provides quick and complete anesthesia, along with muscle relaxation and a bloodless operating field. The procedure is free from the troublesome side effects associated with other regional blocks, such as the axillary block. The procedure is easily mastered and has a very low failure rate, and consistently good results can be expected.

The first practical use of analgesia associated with IV injection of a local anesthetic agent was described by August Gustav Bier in 1908. [4] Colbern[5] has since proposed the eponym *Bier block*. Although the procedure has been in existence for many years, the need for special equipment and a safe anesthetic agent limited its use. However, the Bier block has now gained wide acceptance as a safe and effective procedure, and several papers extol its virtues. [6] [7] [8] [9] Although complications do exist, no reported fatalities directly attributable to the use of the Bier block have been reported. In this chapter, the techniques and complications are discussed according to their application in the ED.

INDICATIONS AND CONTRAINDICATIONS

Indications for IV regional anesthesia include any procedure of the arm or leg that requires operating anesthesia, muscle relaxation, or a bloodless field. I have used the procedure for the reduction of fractures and dislocations, repair of major lacerations, removal of foreign bodies, debridement of burns, and drainage of infection. The procedure may be carried out on any patient of any age who is able to cooperate with the clinician.

The only absolute contraindications are an allergy to the anesthetic agent and uncontrolled hypertension. Relative contraindications include an uncooperative patient and the recent ingestion of a large meal, both of which may only delay the procedure rather than contraindicate it.

EQUIPMENT

The equipment required for IV regional anesthesia consists of the following:

- 1% lidocaine (Xylocaine), *without epinephrine,* to be diluted to a 0.5% solution (*note:* 1 mL of 1% lidocaine equals 10 mg)
- Sterile saline solution as a diluent
- 50-mL syringe/18-ga needle
- Pneumatic tourniquet (single or double cuff) (*note:* Do *not* use a standard blood pressure cuff)
- IV catheters (18- or 20-ga) or a 21-ga butterfly needle
- Elastic bandage/Webril padding
- 500 mL D_5 W (5% dextrose in water) and IV extension tubing

PROCEDURE

The procedure should be explained in advance to the patient. If the patient is extremely apprehensive, premedication with midazolam (Versed), diazepam (Valium), or an opioid (e.g., morphine, fentanyl) may be helpful but need not be routinely used. The only painful portions of the procedure are the establishment of the infusion catheter and the exsanguination procedure. The procedure should not be done on patients who are intoxicated or obtunded or on those with a previous reaction to a local anesthetic.

The patient need not be free of oral intake for a specific period of time before the procedure, but it is prudent to delay the procedure if the patient has just eaten a large meal. As a precaution, a large-bore catheter and IV line of D_5W are established in the unaffected extremity. Resuscitation equipment, including anticonvulsant drugs and oxygen, should be readily available.

While the patient is being prepared, the lidocaine solution is readied but withheld until the injured extremity is exsanguinated and the cuff is in place and reinflated as discussed below. The standard dose of lidocaine is 3 mg/kg injected as a 0.5% solution (1% lidocaine is mixed with equal parts of sterile saline in a 50-mL syringe). Farrell and associates (1985) have described a procedure termed the *minidose Bier block* using 1.5 mg/kg of lidocaine and reported a 95% success rate. This lower dose may decrease the incidence of central nervous system side effects and is more desirable in the ED setting. (Additional lidocaine may be infused if the initial dose is inadequate.) Lidocaine with epinephrine should *not* be used. Plain lidocaine is also available as a 0.5% solution, and as such, it can be used directly to avoid the necessity of diluting the stronger solution.

A pneumatic tourniquet with cotton padding (to prevent ecchymosis) under the cuff is applied proximal to the pathology (Fig. 33-1). *It is strongly advised that one not use a regular blood pressure cuff*, because these often leak or rupture and are not designed to withstand high pressures for any length of time. A specially designed portable double-cuff pneumatic system, such as that marketed by OEC Zimmer Corporation, is ideal (Fig. 33-2).

The anesthetic is premixed in the syringe. The tourniquet is inflated, and a 20-ga plastic catheter or a metal butterfly needle is placed in the superficial vein, as close to the pathologic site as possible, and is securely taped in place (Fig. 33-3). It is usually desirable to use a vein on the dorsum of the hand, but importantly the injection site should be at least 10 cm distal to the tourniquet to avoid injection of anesthetic proximal to the tourniquet. The hub remains on the catheter to avoid backbleeding or the syringe is attached to the butterfly tubing. This catheter will be the route of injection of the anesthetic agent.

The tourniquet is deflated, and the extremity is exsanguinated so that when the anesthetic agent is injected, it will fill the vascular system. Exsanguination may be accomplished

* This chapter modified with permission from Roberts JR: Intravenous regional anesthesia. JACEP 6:261, 1977.

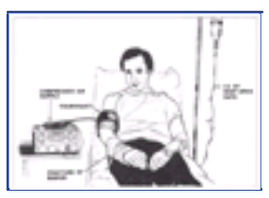

Figure 33-1 Preparation for induction of anesthesia in a patient with a fracture of the right radius. Note precautionary IV line and deflated tourniquet in place. The procedure has been explained, and preoperative sedation or analgesia has been given if required. *(From Roberts JR: Intravenous regional anesthesia. JACEP 6:263, 1977.)*

by either of two methods. Simple elevation of the extremity for a few minutes may be adequate, but wrapping the extremity in a distal-to-proximal direction with an elastic bandage, being careful not to dislodge the infusion needle, enhances the exsanguination (Fig. 33-4). Wrapping may be painful; this step can be eliminated if it causes too much anxiety to the patient. If the wrapping procedure is not done, the extremity should be elevated for at least 3 minutes. During the wrapping procedure, care must be taken not to dislodge or infiltrate the infusion catheter.

With the extremity still elevated, the tourniquet is inflated to 250 mm Hg, the arm is then placed by the patient's side, and the elastic exsanguination bandage is removed. In a child, the tourniquet is inflated to 50 mm Hg above systolic pressure.

The 0.5% lidocaine solution is then slowly injected into the infusion catheter at the calculated dose. Note that the solution is placed in the arm in which circulation is blocked,

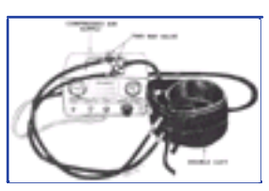

Figure 33-2 Double-cuff apparatus with two-way valves allows longer tourniquet time without pain. A standard blood pressure cuff should never be used. *(From Roberts JR: Intravenous regional anesthesia. JACEP 6:263, 1977.)*

not in the precautionary keep-open IV line on the unaffected side. At this point, blotchy areas of erythema may appear on the skin. This is not an adverse reaction to the anesthetic agent, but merely the result of residual blood being displaced from the vascular compartment, and it heralds success of the procedure.

In 3 to 5 minutes, the patient will experience paresthesia or warmth, beginning in the fingertips and traveling proximally, with final anesthesia occurring at the elbow. Complete anesthesia ensues in 10 to 20 minutes, followed by muscle relaxation. Note that adequate analgesia may exist even though the patient can still sense touch and position and has some motor function. If the "minidose" technique (1.5 mg/kg of lidocaine) does not provide adequate anesthesia, an additional 0.5 to 1 mg/kg may be infused at this time. Additional lidocaine was required in 7% of cases in one series using the minidose regimen. [10] The clinician should be patient, however,

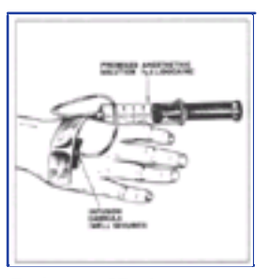

Figure 33-3 Infusion cannula securely taped in the dorsum of hand. A butterfly needle is shown here, but a plastic catheter with the hub attached may also be used. *(From Roberts JR: Intravenous regional anesthesia. JACEP 6:263, 1977.)*

and wait a full 15 minutes before infusing additional lidocaine. Alternatively, if analgesia is slow or inadequate an extra 10 to 20 mL of *saline* solution may be injected

to supplement the total volume of solution to enhance the effect. *Do not exceed a 3 mg/kg total dose of lidocaine.* The infusing needle is then withdrawn, and the puncture site is tightly taped to prevent extravasation of the anesthetic agent. The surgical procedure or manipulation is performed, including postreduction x-ray films and casting or bandaging (Fig. 33-5).

Anesthesia from a fingertip-to-elbow direction seems to occur irrespective of the site of anesthetic infusion, but selecting an injection site near the site of pathology will provide more rapid anesthesia at a lower dosage.

On completion of the procedure, deflation of the tourniquet is *cycled* to prevent a bolus effect of any lidocaine that may remain in the intravascular compartment. The cuff is deflated for 5 seconds and reinflated for 1 to 2 minutes. *This action is repeated three or four times.*

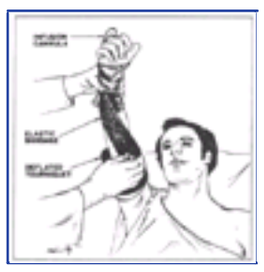

Figure 33-4 Exsanguination by elevation and elastic bandage. The tourniquet has yet to be inflated at this point. Care must be taken not to dislodge the infusion cannula. *(From Roberts JR: Intravenous regional anesthesia. JACEP 6:263, 1977.)*

Figure 33-5 A cast is applied under anesthesia. Because the tourniquet is portable, postreduction radiographs may be obtained without losing anesthesia. *(From Roberts JR: Intravenous regional anesthesia. JACEP 6:263, 1977.)*

If the tourniquet has been in place for less than 30 minutes, it is dangerous to deflate it, because adequate tissue fixation of the lidocaine probably has not occurred. This may result in a higher peak plasma lidocaine level, with increased side effects. If the surgical procedure is completed rapidly and the 3 mg/kg limit of lidocaine has been infused, the tourniquet should remain inflated until a full 30 minutes has elapsed, and only then should it be deflated using the cycling technique. It is reasonable to use a 20-minute cutoff if the minidose technique is used, because this dose is equal to a commonly administered IV bolus.

Sensation returns quickly when the tourniquet is removed, and in 5 to 10 minutes, the extremity returns to its preanesthetic level of sensation and function. After 20 minutes of observation, the patient is released (Table 33-1).

If the procedure takes longer than 20 or 30 minutes, many patients complain of pain from the tourniquet, because the tourniquet is not inflated over an anesthetized area. The use of a double-cuff tourniquet alleviates the problem of pain under the cuff.

In the double-cuff system, there are two separate tourniquets placed side by side on the extremity. One is termed the *proximal cuff*, and the other is called the *distal cuff*. The proximal cuff is inflated at the beginning of the procedure, and anesthesia is obtained under the deflated distal cuff. When the patient begins to feel pain under the proximal cuff, the distal cuff is first inflated over an already anesthetized area, and the pain-producing proximal cuff is then deflated. One must be certain to inflate the distal cuff before the proximal cuff is released; otherwise, the anesthetic will rapidly diffuse into the general circulation.

MECHANISM OF ACTION

Some of the anesthesia is undoubtedly related to the ischemia produced by the tourniquet, but most of the anesthesia is secondary to the anesthetic agent itself. Although the exact mechanism by which anesthesia is produced is unknown, the

TABLE 33-1 -- IV Regional Anesthesia: Step-by-Step Procedure

Begin IV line in uninvolved extremity.
Draw up 0.5% lidocaine (1.5 to 3 mg/kg).
Place padded tourniquet, and inflate upper cuff.
Insert small plastic IV cannula near pathologic lesion and secure.
Deflate tourniquet.
Elevate and exsanguinate extremity.
Inflate tourniquet (250 mm Hg), and remove exsanguination device. Inflate the proximal cuff only, if a double-cuff system is used.
Infuse anesthetic solution.
Remove infusion needle, and tape site.
Perform procedure.
If pain is produced by the application of the tourniquet, first inflate the distal cuff, then deflate the proximal cuff.
After the procedure has been carried out, deflate the cuff for a few seconds, then reinflate it for 1 minute. Repeat 3 times. Do not deflate the cuff if total tourniquet time is less than 30 minutes.
Observe for possible reactions.

From Roberts JR: Intravenous regional anesthesia. JACEP 6:263, 1977. Reproduced with permission.

site of action of the anesthetic may be at sensory nerve endings, neuromuscular junctions, or major nerve trunks. [11] Contrast studies have demonstrated that the anesthetic agent does not diffuse throughout the entire arm, yet anesthesia of the entire limb is obtained. For example, when the anesthetic agent is injected into the elbow and kept in that region with both distal and proximal tourniquets, anesthesia of the entire arm develops. [12] Evidence indicates that the local anesthetic does not simply diffuse from the venous system into the tissue but travels via vascular channels directly inside the nerve. Regardless of where the anesthetic is infused, the fingertips are the first area to experience anesthesia, suggesting that the core of the nerve is in contact with the anesthetic agent initially. Following release of the tourniquet, a considerable amount of the drug still remains in the injected limb for at least 1 hour. [13] This would suggest that at least a portion of the anesthetic leaves the vascular compartment and becomes tissue fixed.

PROCEDURAL POINTS

Anesthetic Agent

Using 0.5% plain lidocaine at a dose of 1.5 to 3 mg/kg is preferred. Other agents have been used without demonstrable advantage and are not recommended. [14] Bupivacaine (Marcaine, Sensorcaine) is *contraindicated* because of the potential for serious cardiovascular and neurologic complications. [15] [16]

Dunbar and Mazze[8] showed that patients with IV regional anesthesia actually had significantly lower plasma lidocaine concentrations than patients with axillary block or lumbar epidural anesthesia for similar procedures. Peak plasma concentrations are reached 2 to 3 minutes after deflation of the tourniquet, and side effects are minimal if the deflation is cycled following the surgical procedure. The plasma half-life of lidocaine is approximately 60 seconds (see the excellent detailed discussion of pharmacokinetics by Covino), [17] but the drug demonstrates a theoretical 3-compartment model similar to a direct IV infusion once the tourniquet is released. [18] Peak blood levels are related to the duration of vascular occlusion and to the concentration of the anesthetic. [17] [18]

Post-release peak plasma lidocaine levels decrease as the time of vascular occlusion (tourniquet time) increases. If the tourniquet is inflated for at least 30 minutes and the deflation-reinflation technique is used when the procedure is finished, plasma concentration of lidocaine should be approximately 2 to 4 µg/mL, below the 5 to 10 µg/mL level at which serious reactions occur. [8] Tucker and Boas[18] demonstrated a peak plasma lidocaine level of 10.3 µg/mL after a 10-minute period of vascular occlusion, compared with 2.3 µg/mL if the tourniquet was inflated for 45 minutes.

More dilute solutions of lidocaine are associated with lower peak lidocaine levels. When equal doses of lidocaine are used, the peak arterial plasma levels are 40% lower when the 0.5% solution is used than when the 1% solution is used. [18] For example, after 10 minutes of vascular occlusion, the peak plasma concentration of lidocaine has been demonstrated to reach 10.3 µg/mL with the 1% solution, compared with only 5.6 µg/mL when the drug was given under similar circumstances as a 0.5% concentration. [18]

Exsanguination

Exsanguination of the extremity before injection of the anesthetic agent is considered essential for success by many clinicians. Others do not believe that it is a critical factor. Exsanguination by simple elevation of the extremity should be done in all cases, but in certain cases one should consider avoiding the painful wrapping of the extremity with an elastic or Esmarch bandage. (Note that applying an Esmarch wrap over a fracture site is usually quite painful.) A pneumatic splint, such as the type used for prehospital immobilization, is also a reasonable alternative to painful wrapping. The process of exsanguination is believed to allow for better vascular diffusion of the anesthetic.

Site of Injection

Anesthesia is usually obtained no matter where the local anesthetic is injected, but some evidence indicates that the procedure is more successful when the anesthetic is injected distally. Sorbie and Chacho [3] note the following failure rates associated with specific sites of anesthetic injection: antecubital fossa, 23%; middle of forearm or leg, 18%; hand, wrist, or foot, 4%. For most cases, a vein in the dorsum of the hand or foot is most often used. If local pathology precludes the use of the hand, the midforearm or antecubital fossa of the elbow are acceptable, albeit less desirable, alternatives as long as the infusion catheter is well below the tourniquet to avoid systemic injection.

Although most of the literature stresses the use of this technique on the upper extremity, it may also be used successfully in the leg. It cannot, however, be used for procedures above the knee. Tourniquet pain appears to be a limiting factor when the procedure is used on the leg. One must be certain to avoid damage to the peroneal nerve by using the tourniquet in the midcalf area only.

COMPLICATIONS

Although IV regional anesthesia is both safe and simple, one should not be lulled into complacency, because complications do occur and are usually related to equipment failure or mistakes in the technique.

Anesthetic Agent

Serious complications seldom occur if proper attention is paid to technique. Reactions to lidocaine are rare and are usually systemic reactions from high blood levels.[8] [16] [19] High levels may result from miscalculation of dosages, from too rapid release of the tourniquet before the anesthetic has become tissue fixed ("bolus effect") or, rarely, from advancement of the infusion catheter proximal to the tourniquet, resulting in direct IV infusion. [20] To emphasize the safety of this procedure it should be noted that the dose of lidocaine used in the "minidose" technique is similar to an IV bolus routinely given to patients with significant cardiovascular disease, in the presence of ventricular dysrhythmias.

Generally, the central nervous system effects of lidocaine are minor, resulting in mild reactions such as dizziness, lethargy, headache, or blurred vision. This should not occur in more than 2% to 3% of patients and requires no treatment. [8] Convulsions may occur but are extremely rare.

The most common complication relating to the anesthetic agent is rapid systemic vascular infusion, which occurs when a blood pressure cuff explodes or slowly leaks, resulting in both loss of anesthesia and high blood levels. [21] Similar complications may occur if the cuff is deflated before 30 minutes after the induction of anesthesia. Both complications are the result of a bolus effect of the anesthetic, resembling an IV injection.

Van Neikerk and Tonkin[19] reported 3 seizures in a series of 1400 patients. Seizures are generally not recurrent and are treated with oxygen and anticonvulsant drugs. Transient cardiovascular reactions, such as bradycardia and hypotension, are possible with large doses of lidocaine. Vasovagal reactions do occur. If resuscitation equipment is available and a precautionary IV line is started in the opposite arm, there should not be any serious sequelae.

One case of cardiac arrest for 15 seconds following the use of 200 mg of lidocaine has been reported, but the actual clinical scenario may have been a vasovagal reaction rather than a true cardiac arrest. [22]

Additional Complications

Thrombophlebitis can occur following IV administration of anesthetics, and the formation of insignificant amounts of methemoglobin with the use of prilocaine hydrochloride (Citanest) has been reported. [23] Methemoglobinemia also can theoretically occur with lidocaine but has not been reported.

A particularly bothersome problem has been the infiltration of the infusion catheter during exsanguination, resulting in tissue extravasation of the anesthetic agent. Also, there has been some leakage of anesthetic after the infusion needle has been removed. Both problems may result in poor anesthesia but may be minimized if a small, well-secured plastic infusion needle is used instead of a metal scalp vein ("butterfly") needle and if the puncture site is tightly taped following with-drawal of the catheter.

This procedure cannot be used in manipulations or operations in which the pulse must be monitored as a guide to reduction (e.g., supracondylar fractures of the humerus), because the tourniquet occludes arterial flow. The use of the Bier block in patients with sickle cell disease is not well documented. It should be used with caution until the ischemic effect of the tourniquet on the red blood cells of such patients has been clarified. In all patients the tourniquet time should not exceed 90 minutes. Ischemia for less than that amount of time is not associated with serious sequelae.

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Chapter 34 - Procedural Sedation and Analgesia

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Baruch Krauss

Procedural sedation and analgesia (PSA) is the use of analgesic, dissociative, and sedative agents to relieve the pain and anxiety associated with diagnostic and therapeutic procedures performed in various settings. PSA is an integral element of emergency medicine residency and pediatric emergency medicine fellowship curricula, and graduates of these programs are skilled in the practice of PSA. Emergency clinicians are skilled in resuscitation, vascular access, and advanced airway management, permitting them to effectively recognize and manage the potential complications associated with PSA.

The most common clinical errors associated with PSA in a recent study of *all practitioners* were delayed recognition of respiratory depression and respiratory arrest, inadequate monitoring, and inadequate resuscitation [1] [2] —mistakes that are unlikely for emergency clinicians. The safety of PSA techniques by emergency clinicians has been well documented in numerous series in both children [3] [4] [5] [6] and adults.[3] [7] Successful and safe application of PSA requires careful patient selection, customization of therapy to the specific needs of the patient, and careful patient monitoring for adverse events. Emergency clinicians must ensure that all patients receive pain relief and sedation commensurate with their individual needs during any procedure.

TERMINOLOGY

The progression from minimal sedation to general anesthesia represents a nonlinear continuum that does not lend itself to division into arbitrary stages. Low doses of opioids or benzodiazepines induce mild analgesia or sedation, respectively, with little danger of adverse events. If, however, clinicians continue administering additional medication beyond this initial level, progressively altered consciousness ensues with a proportionately increased risk of respiratory and airway complications. If further medications are administered, the patient will advance along this continuum until protective airway reflexes are lost and general anesthesia is ultimately reached. This sedation continuum is not drug-specific, in that varying states from mild sedation to general anesthesia can be achieved with virtually all non-dissociative PSA agents (e.g., opioids, benzodiazepines, barbiturates, etomidate, propofol).

In 1985, the American Academy of Pediatrics (AAP) and National Institutes of Health (NIH) issued guidelines for the management and monitoring of children receiving sedation for diagnostic and therapeutic procedures in response to the growing use of opioids and sedative/hypnotic agents in the outpatient setting and a number of sedation-related deaths.[8] [9] In these documents, three levels of sedation were defined (conscious sedation, deep sedation, general anesthesia) to create a common language for describing drug-induced alterations in consciousness (Table 34-1). A key development in

TABLE 34-1 -- Procedural Sedation and Analgesia Terminology and Definitions

GENERAL
• **Analgesia*** **:** Relief of pain without intentional production of an altered mental state such as sedation. An altered mental state may be a secondary effect of medications administered for this purpose.
• **Anxiolysis*** **:** A state of decreased apprehension concerning a particular situation in which there is no change in a patient's level of awareness.
• **Procedural Sedation and Analgesia (PSA)**[3] **:** A technique of administering sedatives, analgesics, and/or dissociative agents to induce a state that allows the patient to tolerate unpleasant procedures while maintaining cardiorespiratory function. PSA is intended to result in a depressed level of consciousness but one that allows the patient to maintain airway control independently and continuously. Specifically, the drugs, doses, and techniques used are not likely to produce a loss of protective airway reflexes.
CURRENT SEDATION STATE TERMINOLOGY
• **Minimal Sedation (Anxiolysis)**[13] **:** A drug-induced state during which patients respond normally to verbal commands. Although cognitive function and coordination may be impaired, ventilatory and cardiovascular functions are unaffected.
• **Moderate Sedation (Formerly Conscious Sedation)**[13] **:** A drug-induced depression of consciousness during which patients respond purposefully to verbal commands, either alone or accompanied by light tactile stimulation. Reflex withdrawal from a painful stimulus is not considered a purposeful response. No interventions are required to maintain a patent airway, and spontaneous ventilation is adequate. Cardiovascular function is usually maintained.
• **Dissociative Sedation**[96] **:** A trance-like cataleptic state induced by the dissociative agent ketamine characterized by profound analgesia and amnesia, with retention of protective airway reflexes, spontaneous respirations, and cardiopulmonary stability.
• **Deep Sedation**[13] **:** A drug-induced depression of consciousness during which patients cannot be easily aroused but respond purposefully following repeated or painful stimulation. The ability to independently maintain ventilatory function may be impaired. Patients may require assistance in maintaining a patent airway and spontaneous ventilation may be inadequate. Cardiovascular function is usually maintained.
• **General Anesthesia**[13] **:** A drug-induced loss of consciousness during which patients are not arousable, even by painful stimulation. The ability to independently maintain ventilatory function is often impaired. Patients often require assistance in maintaining a patent airway, and positive pressure ventilation may be required because of depressed spontaneous ventilation or drug-induced depression of neuromuscular function. Cardiovascular function may be impaired.
ORIGINAL AAP/NIH TERMINOLOGY[8] [9]
• **Conscious Sedation:** A medically controlled state of depressed consciousness that 1) allows protective reflexes to be maintained; 2) retains the patient's ability to maintain a patent airway independently and continuously; and 3) permits appropriate response by the patient to physical stimulation or verbal command (e.g., "open your eyes").
• **Deep Sedation:** A medically controlled state of depressed consciousness or unconsciousness from which the patient is not easily aroused. It may be accompanied by a partial or complete loss of protective reflexes, and includes the inability to maintain a patent airway independently and respond purposefully to physical stimulation or verbal command.
• **General Anesthesia:** A medically controlled state of unconsciousness accompanied by a loss of protective reflexes, including the inability to maintain a patent airway independently and respond purposefully to physical stimulation or verbal command.

*From Sacchetti A, Schafermeyer R, Gerardi M, et al: Pediatric analgesia and sedation. Ann Emerg Med 23:237, 1994.

the field of PSA has been the revision of the original terminology and the adoption of clearer descriptions of varying types and degrees of sedation (see Table 34-1). Although historically popular, the widely misinterpreted and misused term *conscious sedation* has fallen into disfavor, [10] labeled as confusing,[11] "imprecise,"[12] and an "oxymoron"[10] [11] and has been replaced with the term moderate sedation.[13]

Despite improvements in PSA terminology, the system is imperfect and there is still not an objective way to assess sedation depth. Levels of responsiveness remain at best crude surrogate markers of respiratory drive and retention of protective airway reflexes. This is especially true for all levels of sedation in young children (infants and toddlers) who do not understand or are unreliable in following verbal commands. Although respiratory depression and respiratory arrest can be quickly detected using standard interactive and mechanical monitoring, there is no safe and practical way to assess the status of protective airway reflexes. There is currently insufficient data to determine whether deep sedation is associated with impairment of protective reflexes, or whether such danger is only encountered when "pushing" deep sedation to a point at which it approaches or reaches general anesthesia.

PSA GUIDELINES

Prior to the promulgation of PSA guidelines by specialty societies and governmental agencies, clinicians simply administered sedatives in varied clinical settings and applied individual judgment as to the need for specific monitoring devices and supporting personnel. Since 1985, at least 12 sets of PSA guidelines have been published, each crafted for the unique and differing settings in which PSA is practiced. Naturally, not all are in agreement. [5] The intent of each of these guidelines is to better standardize the manner in which PSA is performed in order to enhance patient safety. Those most pertinent to emergency clinicians are from the American College of Emergency Physicians,[3] the American Academy of Pediatrics,[14] and the American Society of Anesthesiologists.[12] [15]

In the early 1990s the Joint Commission on Accreditation of Healthcare Organizations (JCAHO), an independent, not-for-profit organization that evaluates and accredits hospitals in the United States, took a special interest in PSA, with the central theme that the standard of sedation care provided should be comparable throughout a given hospital. Thus, patients sedated in the ED should not receive a significantly different level of attention or monitoring than those sedated for a similar-level procedure in the operating room or in the endoscopy suite. To ensure this, the JCAHO requires specific PSA protocols that apply consistently throughout each institution. These hospital-wide sedation policies will vary from site to site based upon the specific needs and expertise available within each institution. In 2001, JCAHO released new standards for pain management, sedation, and anesthesia care.

At each hospital accreditation survey, the JCAHO will see if practitioners practice PSA consistently with their hospital-wide sedation policy, and whether they provide sufficient documentation of such compliance. Clinicians must be familiar with their hospital's sedation policies, and should work with their medical staff to ensure that such policies are suitably detailed, but yet reasonable and realistic. Unduly restrictive policies do a disservice to patients by discouraging appropriate use of analgesia and anxiolysis. Most hospitals pattern their sedation policies after JCAHO standards and definitions. It is important to note that the unique ketamine dissociative state does not fit into the existing JCAHO definitions of sedation and anesthesia. [16] A ready solution is assigning a distinct definition for "dissociative sedation" (see Table 34-1).

JCAHO[13] requires that PSA practitioners who are permitted to administer deep sedation must be qualified to rescue patients from general anesthesia. Emergency clinicians will typically perform all levels of sedation except general anesthesia. Moderate sedation suffices for the majority of procedures in adults and cooperative children, although it will not be adequate for extremely painful procedures (e.g., hip reduction, cardioversion). Deep sedation can facilitate these, but at greater risk of cardiorespiratory depression than moderate sedation. Moderate sedation is frequently insufficient for effective anxiolysis and immobilization in younger, frightened children, with deep or dissociative sedation typically appropriate alternatives.

EVALUATION PRIOR TO PSA

The practice of PSA has three essential components performed in sequence: the initial pre-sedation evaluation, sedation during the procedure, and post-procedure recovery and patient discharge from the emergency department (ED). In all but the most emergent situations, a directed history and physical examination should precede PSA. If this evaluation suggests additional risk, the advisability of sedation should be reconsidered. High-risk cases may be better managed in the more controlled environment of an operating room.

Presedation assessments are a JCAHO requirement, and most hospitals have developed specific forms to facilitate consistent documentation of the involved items. Except in emergency situations, the risks, benefits, and limitations of any PSA should be discussed with the patient (or their parent or guardian) in advance and verbal agreement obtained. Formal written informed consent is not required as a standard of care (unless a local institutional requirement) though documentation, as discussed earlier, is essential.

General.

Clinicians should assess the type and severity of any underlying medical problems. This is usually best documented by the standard ED medical record, history and physical exam, and nursing notes. Another tool used for this purpose is the American Society of Anesthesiologists' (ASA) physical status classification that is used for pre-operative risk stratification (Table 34-2 (Table Not Available)). Current medications and allergies should be verified. It is advisable to inquire regarding prior adverse experiences with PSA or anesthesia.

Airway.

The airway should be inspected to determine if there are abnormalities that might impair airway management (e.g., severe obesity, short neck, small mandible, large tongue, trismus).

Cardiovascular.

Cardiac auscultation should be performed to assess for rhythm disturbance or other abnormality. In patients with known cardiovascular disease, their degree of reserve should be evaluated, as most PSA agents can cause vasodilatation and hypotension.

Respiratory.

Lung auscultation should be performed to assess for active pulmonary disease, especially obstructive lung disease and active upper respiratory infections that may predispose the patient to airway reactivity.

Gastrointestinal.

As pulmonary aspiration of gastric contents is a dreaded complication of vomiting when protective

TABLE 34-2 -- American Society of Anesthesiologists' (ASA) Physical Status Classification

(Not Available)

From Krauss B, Green SM: Sedation and analgesia for procedures in children. N Engl J Med 342:938, 2000.

airway reflexes are impaired, clinicians should assess the time and nature of last oral intake. For elective procedures, the ASA recommends an age-stratified fasting requirement of 2 to 3 hours for clear liquids and 4 to 8 hours for solids and non-clear liquids. [17] Despite this, they acknowledge that "the literature provides insufficient data to test the hypothesis that preprocedure fasting results in a decreased incidence of adverse outcomes" in PSA. [12] [15] In non-fasting situations for non-elective or emergent procedures, where the ASA guidelines are virtually impossible to achieve, the potential for pulmonary aspiration must be balanced with the timing of the procedure and the required depth of sedation. This is consistent with the ASA guidelines, "in urgent, emergent, or other situations when gastric emptying is impaired, the potential for pulmonary aspiration of gastric contents must be considered in determining the timing of the intervention and the degree of sedation/analgesia" [12] [15] and ACEP PSA guidelines, "recent food intake is not a contraindication for administering PSA, but should be considered in choosing the depth and target level of sedation."[3]

The concept of preprocedure fasting is logistically difficult or impossible for emergency clinicians, who have no control over patients' oral intake prior to ED presentation. In actual practice, emergency clinicians routinely perform safe PSA on patients noncompliant with the ASA elective procedure fasting guidelines. [16] Procedures can sometimes be delayed for a number of hours; however, this must be balanced with prolongation of pain and anxiety for the patient, inconvenience for the patient and family, and expenditure of room space and other finite ED resources. Additionally, many ED procedures require urgent if not immediate attention (e.g., debridement and repair of animal bite wounds, acute burn management, arthrocentesis for suspected septic arthritis, reductions of joint dislocations, lumbar puncture in the uncooperative septic patient, hernia reduction, eye irrigation for ocular trauma or chemical burns, cardioversion in the hemodynamically unstable patient). Although uncommon, there may be occasions where non-fasting patients requiring urgent procedures with a substantial depth of sedation may be more safely managed in the operating room with endotracheal intubation to protect the airway.

Selecting agents less likely to produce vomiting, such as opting for fentanyl over morphine/meperidine, may decrease the aspiration potential. Concomitant antiemetic administration is an unproven adjunct, but a common consideration.

Hepatic/Renal.

The implications of delayed metabolism or excretion of PSA agents in infants less than 6 months of age, in the elderly, and in the presence of hepatic or renal abnormality should be carefully evaluated.

PERSONNEL AND INTERACTIVE MONITORING

The most important element of PSA monitoring is close and continuous patient observation by an individual capable of recognizing sedation complications. This person must be able to continuously observe the patient's face, mouth, and chest wall motion, and equipment or sterile drapes must not interfere with such visualization. This careful observation will allow prompt detection of adverse events such as respiratory depression, apnea, partial airway obstruction, emesis, and hypersalivation.

PSA personnel should understand the pharmacology of analgesic and sedative agents and their respective reversal agents, and be proficient at maintaining airway patency and assisting ventilation if needed. *PSA requires a minimum of two experienced individuals*, most frequently one clinician and one nurse or respiratory therapist. The clinician typically oversees drug administration and performs the procedure, while the nurse or respiratory therapist continuously monitors the patient for potential complications and documents medications administered, response to sedation, and periodic vital signs. The nurse or respiratory therapist may assist with minor, interruptible tasks; however, their ability to remain focused on the patient's cardiopulmonary status must not be impaired. An individual with advanced life-support skills should be immediately available, a requisite easy to fulfill in the ED setting.

During deep sedation, the individual dedicated to patient monitoring should be experienced with this depth of sedation and have no other responsibilities that would interfere with the advanced level of monitoring and documentation appropriate for this sedation level. [14] Individual hospital-wide sedation policies may have additional requirements for how and when deep sedation is administered, based on their specific needs and available expertise.

For situations in which sedation is initiated by the intramuscular, oral, nasal, inhalational, or rectal routes it is not mandatory to have IV access, although this may be preferred based upon anticipated depth of sedation, comorbidity, or for the convenience of additional drug titration. When sedation is performed without IV access, an individual skilled in initiating such access should be immediately available—again a requisite easy to fulfill in the ED setting.

EQUIPMENT AND MECHANICAL MONITORING

The routine use of mechanical monitoring has greatly enhanced the safety of PSA. With current technology, oxygenation (via pulse oximetry), ventilation (via capnography), and hemodynamics (via blood pressure and electrocardiogram [ECG]) can all be monitored non-invasively in spontaneously breathing patients. PSA mechanical monitoring should include continuous pulse oximetry with an audible signal. Pulse oximetry measures the percent of hemoglobin that is bound to oxygen and is not a substitute for monitoring ventilation, as there is a variable lag time between the onset of hypoventilation or apnea and a change in oxygen saturation of hemoglobin molecules.

Capnography provides a continuous, breath-by-breath measure of respiratory rate (RR) and CO_2 exchange and provides the earliest detection of respiratory failure. [18] [19] [20] [21] [22] [23] [24] [25] [26] Hypoventilation and respiratory depression result in a progressive decrease in respiratory rate, increase in the end-tidal CO_2 ($EtCO_2$), and a change in the shape of the CO_2 waveform. Apnea and laryngospasm result in an almost instantaneous loss of the CO_2 waveform. Early detection of respiratory compromise is especially important in infants and toddlers who have smaller functional residual capacity and greater oxygen consumption relative to older children and adults. [27] [28] In the ED setting capnography has been shown to identify respiratory depression undetectable by pulse oximetry. [21] Capnography is currently not considered a standard of care, but this technology should be considered, as it can alert practitioners to respiratory depression and apnea before hypoxemia develops, especially if the patient is on supplemental oxygen.

Although continuous ECG monitoring cannot be considered mandatory nor standard of care in the absence of cardiovascular disease, such monitoring is simple, inexpensive, and readily available.

A monitoring modality that is currently undergoing investigation for use in the ED is the Bispectral Index (BIS), which uses processed electroencephalogram (EEG) signal to measure the effect of sedative/hypnotics on the brain. This technology has been validated as a tool to monitor depth of sedation in the operating room; however, its predictive value for the remainder of the sedation continuum remains unclear. [29] [30] [31] In a 12-patient pilot study of ED BIS, a significant correlation between the BIS and a behavioral assessment of Alertness/Sedation (OAA/S) was found. [32] BIS may prove a valuable PSA monitoring modality if future studies demonstrate a high degree of discriminatory power in predicting sedation depth over the entire sedation continuum (anxiolysis and mild sedation to general anesthesia).

The sedation area should include all necessary age-appropriate equipment for airway management and resuscitation, including oxygen, a bag-valve mask, suction, and drug reversal agents. A defibrillator should be available for subjects with significant cardiovascular disease.

The need for supplemental oxygen during PSA and its benefits have not been studied. Although this intervention will decrease the incidence and severity of hypoxemia due to airway complications, it will also delay the detection of apnea with pulse oximetry. [12] [15] If oxygen is administered and capnography is not available, continual visual inspection of chest-wall motion and air movement is especially important. Supplemental oxygen cannot be considered a standard of care at this time and remains an option best left to the clinician's preference.

Vital signs should be periodically measured at individualized intervals, in most cases including measurements at baseline, after drug administration, on completion of the procedure, during early recovery, and at completion of recovery. During deep sedation it is advisable to assess vital signs every 5 minutes. Patients are at highest risk of complications 5 to 10 minutes following IV medications and during the immediate post-procedure period when external stimuli are discontinued.

DISCHARGE CRITERIA

All patients receiving PSA should be monitored until they are no longer at risk for cardiorespiratory depression (Table 34-3). To be discharged they should be alert and oriented (or returned to age-appropriate baseline), and vital signs should be stable. Many hospitals have chosen to use standardized recovery-scoring systems similar to those used in their surgical post-anesthesia recovery areas (Table 34-4). All patients should leave the hospital with a reliable adult who will observe them after discharge for post-procedural complications. It is desirable to document the name of the individual on the hospital record. Written instructions should be given regarding appropriate diet, medications, and level of activity (Table 34-5 and Table 34-6). Even though patients may appear awake and able to comprehend instructions, they may not remember details once they leave the ED. To be eligible for safe discharge, children are not required to demonstrate that they can tolerate a PO challenge (most PSA agents are emetogenic and forcing fluids post-sedation can lead to emesis before and/or after discharge), nor are they required to walk unaided. The AAP guidelines only require that "the patient can talk (if age-appropriate)" and "the patient can sit up unaided (if age-appropriate)." [14]

GENERAL PRINCIPLES

Therapeutic mistakes resulting in inadequate analgesia and sedation include using the wrong agent, the wrong dose, the wrong route and frequency of administration, and poor use of

TABLE 34-3 -- Post-Sedation Complications

Complication	Etiology
Delayed awakening	Prolonged drug action
	Hypoxemia, hypercarbia, hypovolemia
Agitation	Pain, hypoxemia, hypercarbia, full bladder
	Paradoxical reactions
	Emergence reactions
Nausea and vomiting	Sedative agents
	Premature oral fluids
Cardiorespiratory events	
Tachycardia	Pain, hypovolemia, impaired ventilation
Bradycardia	Vagal stimulation, opioids, hypoxia
Hypoxia	Laryngospasm, airway obstruction, oversedation

From Krauss B, Brustowicz R (eds): Pediatric Procedural Sedation and Analgesia. Philadelphia, Lippincott Williams & Wilkins, 1999, p 145

TABLE 34-4 -- Sample Recovery Scoring Systems

Steward Recovery Score	
Consciousness	
Awake	2
Responding to stimuli	1
Not responding	0
Airway	
Coughing on command or crying	2
Maintaining good airway	1
Airway requires maintenance	0
Movement	
Moving limbs purposefully	2
Nonpurposeful movements	1
Not moving	0
Modified Aldrete Score	
Vital Signs	
Stable	1
Unstable	0
Respirations	
Normal	2
Shallow respirations/tachypnea	1
Apnea	0
Level of Consciousness	
Alert, oriented/returned to pre-procedural level	2
Arousable, giddy, agitated	1
Unresponsive	0
Oxygen Saturation	
95–100% or pre-procedural level	2
90–94%	1
< 90%	0
Color	
Pink/pre-procedural color	2
Pale/dusky	1
Cyanotic	0
Activity	
Moves on command/pre-procedural level	2
Moves extremities/uncoordinated walking	1

No spontaneous movement	0
Sedation Score	*Action*
>8	Consider discharge if no score = 0
7–8	Vital signs q 20 min
4–6	Vital signs q 10 min
0–3	Vital signs q 5 min—consider further evaluation if prolonged

From Krauss B, Brustowicz R (eds): Pediatric Procedural Sedation and Analgesia. Philadelphia, Lippincott Williams & Wilkins, 1999, p 157.

adjunctive agents. With proper training and technique, adequate PSA can be provided under almost any circumstance. Understanding titration principles is critical to providing safe and effective PSA. Clinicians must have a thorough knowledge of the pharmacokinetics, dosing, administration, and potential complications of the PSA agents that they use. Onset time from injection to initial observed effect must be appreciated, especially when using drugs in combination, to avoid stacking of drug doses resulting in oversedation.

The correct agent (or combination of agents) and the route and timing of administration depend on the following factors: How long will the procedure last? Will it be seconds

TABLE 34-5 -- Sample Adult Disposition Instructions after PSA

1. Do not drive or operate heavy machinery for 12 hours.
2. Eat a light diet for the next 12 hours.
3. Take only your prescribed medications as needed, including any pain medication you were discharged with. Avoid alcohol.
4. Do not make any important decisions or sign important documents for 12 hours. You may be forgetful due to medications that were administered.
5. If you experience any difficulty breathing or persistent nausea and vomiting, return to the emergency department.
6. You should have a responsible person with you for the rest of the day and during the night.

(e.g., simple relocation of a dislocated joint, incision and drainage of a small abscess, cardioversion), minutes (e.g., complex fracture manipulation for reduction, breaking up loculations in a large abscess and then packing it), or prolonged (e.g., complex facial laceration repair)? How likely is it that the procedure will need to be repeated (e.g., fracture reduction)? Can topical, local, or regional anesthesia be used as an adjunct? Does the patient only require sedation for a non-invasive diagnostic imaging study?

Prior to drug administration, every effort should be made to minimize a patient's anxiety and distress, particularly in children. The emotional state of a patient on induction strongly correlates with the degree of distress on emergence and in the immediate days following the procedure. [33] [34] [35] [36] Emergency clinicians must avoid being pressured by consultants to cut corners or rush PSA. Incorporating into the presedation preparation a discussion with the consultant about the sedation plan and the length of time required to safely prepare and sedate the patient can avoid the risks associated with a hurried sedation.

Pediatric emergency clinicians should know the adult doses of the sedation agents they are administering. Understanding that the initial dose of midazolam for PSA in a 100 kg patient on a mg/kg basis is far less than the 0.1 mg/kg used in a child is essential to avoid unexpected mishaps in drug dosing.

TABLE 34-6 -- Sample Pediatric Disposition Instructions after PSA

Your child has been given medicine for sedation and/or pain control. These medicines may cause your child to be sleepy and less aware of his or her surroundings, making it easier for accidents to happen while walking or crawling. Because of these side effects, your child should be watched closely for the next few hours. We suggest the following:
1. No eating or drinking for the next 2 hours. Infants may resume half normal feedings when they are hungry.
2. No playing for 12 hours that requires normal coordination, such as bike riding or jungle gym activities.
3. No playing without an adult to watch and supervise for the next 12 hours.
4. No baths, showers, cooking, or use of potentially dangerous electrical appliances unless supervised by an adult for the next 12 hours.
If you notice anything unusual about your child, call us for advice or return to the emergency department for reevaluation.

ROUTES OF ADMINISTRATION

For non-dissociative agents, IV titration to patient response is the best method of obtaining rapid and safe analgesia and/or sedation. It is important to wait the appropriate time for the medications to produce the intended effect before adding more doses. When using opioids, doses administered in 2 to 3 minute increments, observing for side effects such as miosis, somnolence, decreased responsiveness to verbal stimuli, minimally impaired speech, and diminished pain on questioning, are appropriate initial end points. For sedative/hypnotics similar end points such as ptosis (rather than miosis), somnolence, slurred speech, and gaze alteration should be sought. Repeated doses may be given in a titrated fashion based on the patient's response during the procedure.

Oral, transmucosal (i.e., nasal, rectal), and intramuscular (IM) routes are more convenient means of administration as IV access is not necessary. However, they are much less reliable for timely dose titration to a desired response. The efficacy of intranasal drug administration in particular is operator-dependent (Table 34-7). The main advantage of these other routes is for pediatric patients in whom IV access may be problematic or for procedures that may require only minimal sedation in conjunction with the use of local anesthetics. These routes are also advantageous for simple sedation for diagnostic imaging.

With the exception of ketamine, agents administered IM have erratic absorption and a variable onset of action. As such, prolonged preprocedural and postprocedural observation may be necessary. When required because of limitations in obtaining IV access, the IM route offers little advantage over oral or transmucosal administration.

Another PSA route is via inhalation using nitrous oxide. This gas can either be delivered by a demand flow system using a hand-held mask, or delivered to young children by a continuous flow system under close clinician supervision using a nose mask.

Because individual needs may vary widely, application of arbitrary ceiling doses of analgesic and sedative regimens is unwarranted. The true ceiling dose of an agent is that which provides adequate pain relief or sedation without major cardiopulmonary side effects such as respiratory depression, apnea, bradycardia, hypotension, or allergic reactions.

TABLE 34-7 -- Technique for Intranasal Drug Administration

Proper administration is crucial for maximal nasal mucosal absorption.
The child must be supine with the head held in the neutral position, looking straight up.
The solution is placed in a 1 mL tuberculin syringe and instilled slowly, drop by drop, into the child's nostrils, alternating nostrils as one proceeds.
If the child receives a drop and immediately swallows, the solution bypassed the nasal mucosa and will be absorbed orally.
Likewise, if the child coughs and gags after a drop, the solution has bypassed the nasal mucosa and irritated the glottic area.
Time each instillation with the child's swallowing pattern, ideally instilling the solution immediately after the child swallows.
Crying is helpful in creating back pressure to keep the solution out of the posterior pharynx.
Most, if not all, children will begin to cry after the first instillation.

From Krauss B, Brustowicz R (eds): Pediatric Procedural Sedation and Analgesia. Philadelphia, Lippincott Williams & Wilkins, 1999, p 228.

There are two absolute PSA contraindications: severe clinical instability requiring immediate attention, and refusal by a competent patient. Relative contraindications include hemodynamic or respiratory compromise, altered sensorium, or inability to monitor side effects. However, even in many of these circumstances, appropriate agents can be given to provide analgesia and sedation while minimizing the chances for further deterioration. Although safely sedating patients at the extremes of age is challenging and requires additional care, *age is not a contraindication to PSA*.

DRUG SELECTION STRATEGIES

The majority of ED procedures in older children and adults can be performed without systemic sedation and analgesia. Skilled practitioners can frequently combine a calm, reassuring bedside manner with distraction techniques or careful local or regional anesthesia, or both. [37] [38] Many procedures, however, cannot be technically or humanely performed without PSA. These situations can be divided into three categories.

Insufficient analgesia.

Despite a cooperative patient, for some procedures it is impossible to achieve effective pain control with local or regional anesthesia. Examples of procedures requiring systemic PSA include fracture reductions, dislocation reductions, large loculated abscess incision and drainage, wounds that require scrubbing such as "road rash," cardioversion, bone marrow aspiration/biopsy, and extensive burn debridement.

Insufficient anxiolysis.

Despite effective local or regional anesthesia, some patients will be so frightened that procedures cannot be technically or humanely performed without PSA. Young children requiring laceration repair are frequently terrified, and older children and adults may be highly anxious in anticipation of laceration repairs in sensitive and/or personal regions (e.g., face, genitalia, perineum).

Insufficient immobilization.

Despite effective local or regional anesthesia and anxiolysis, PSA may be indicated to prevent excessive motion during procedures that require substantial immobilization (e.g., repair of complex facial lacerations). Immobilization is most commonly an issue with young children and the mentally challenged.

General considerations.

Clinicians must therefore customize their drug selection (i.e., anxiolysis, analgesia, immobilization) based upon the unique needs of the patient and their individual level of experience with specific agents (Table 34-8 (Table Not Available)). A risk-benefit analysis should be performed before every sedation (Table 34-9). The benefits of reducing anxiety and controlling pain should be carefully weighed against the risks of respiratory depression and airway compromise. Factors influencing the extent of pharmacological management are listed in Table 34-10 . Some general drug selection strategies are discussed below and shown in Table 34-8 (Table Not Available) .

Minor procedures in cooperative adults and older children.

These procedures can usually be managed with topical, local, or regional anesthesia. Systemic PSA is typically unnecessary, although mild anxiolysis (e.g., nitrous oxide, oral midazolam) can make these patients more comfortable.

Larger procedures of longer duration in cooperative adults and older children.

Supplementation of topical, local, or regional anesthesia with either nitrous oxide or IV midazolam and fentanyl permits customization of sedation depth and pain relief to the specific needs of each patient.

TABLE 34-8 -- Procedural Sedation and Analgesia Indications and Sedation Strategies

(Not Available)

From Krauss B, Green SM: Sedation and analgesia for procedures in children. N Engl J Med 342:938, 2000. Adapted with permission.

TABLE 34-9 -- Risk-Benefit Analysis for Procedural Sedation and Analgesia

• Why is PSA needed in the first place? Is the procedure very painful, frightening, or requiring extreme cooperation?
• Are the risks of PSA appropriate for the procedure involved?
• If a child, do the parents or guardian consent to the use of PSA?
• How long will the procedure take? If it is a short procedure, is it worth the added risk and expense to the patient? If it is a longer procedure, is there an appropriate agent that can be titrated to allow adequate PSA throughout the entire length of the procedure?
• Are there significant side effects that limit a particular drug's usefulness?
• Are there enough nurses and support personnel present to safely allow the use of PSA?
• What is the recovery period for a given agent? Are there enough treatment areas and staff in the ED to allow adequate observation during recovery?
• When did the patient last eat? Is a delay in waiting for a sufficient fasting time worth the time lost in performing the procedure?

From Krauss B, Brustowicz R (eds): Pediatric Procedural Sedation and Analgesia. Philadelphia, Lippincott Williams & Wilkins, 1999, p 294.

Procedures in uncooperative adults or the mentally challenged.

Essentially all procedures in uncooperative adult-sized patients are difficult without systemic PSA. Depending upon operator experience, IV midazolam/fentanyl or IM/IV ketamine/midazolam may be used in these situations. Midazolam and fentanyl can be titrated intravenously to a relatively deep level of sedation, although, as discussed previously, the risk of adverse effects increases with sedation depth. Ketamine (typically with co-administered midazolam when used in adults) can also provide the profound analgesia and immobilization necessary to perform painful procedures; however, in adults there is a risk of unpleasant hallucinatory recovery reactions. Ketamine should be used with extreme caution in older adults, as its sympathomimetic properties may aggravate underlying coronary artery disease or hypertension. Occasionally, procedures in extremely uncooperative adults or the mentally challenged are better managed in the operating room with general anesthesia.

Minor procedures in uncooperative older children and in young children.

Minor procedures (e.g., small lacerations, IV cannulation, venipuncture, superficial foreign body removal) in uncooperative children can frequently be managed

TABLE 34-10 -- Factors Influencing the Extent of Pediatric Pharmacological Management

AGE
Selected drugs and routes of administration have age limitations and are not recommended above or below a certain age (e.g., demand flow nitrous oxide in children <5 years old, nasal and rectal routes of administration in children >6 years old).
TIME OF DAY
A toddler presenting at naptime or at 9 p.m. who is tired and sleepy will usually require a smaller dosing and possibly a lower level of procedural sedation and analgesia (PSA) than required at 9 a.m. Young children presenting with facial lacerations at night, after their normal bedtime, may only require topical anesthesia and a quiet room for 20–30 minutes to achieve a painless laceration repair while the child sleeps.
FASTING STATUS
Young children can be extremely difficult and uncooperative when hungry and/or tired. In anticipation of PSA many children are kept NPO from the time they are triaged in the ED. This can further increase hunger and irritability, especially if the child waits 1–2 hours to be seen by a physician.
STAFFING AND EQUIPMENT AVAILABILITY
Staffing availability can affect the use and timing of sedation and is especially important in busy EDs with multiple sedations occurring concurrently and in smaller units that are set up for only one sedation at a time.
LOCATION OF THE INJURY
Injuries located in areas of cosmetic concern (especially on the face) or near sensory organs (e.g., ears, eyes, mouth, nose) will often require a high degree of agitation control and a concomitant level of PSA.
PREVIOUS MEDICATIONS
An accurate history of prior medication administration is important in situations where a child is referred from another facility, as this can affect the type and timing of PSA agents that can be given. In particular, a child may have received opioids or sedative/hypnotics prior to transfer and may still be sedated on arrival, necessitating an adjustment in the PSA regimen.
LEVEL OF ANXIETY
The level of anxiety of both the child and accompanying adult(s) must be accurately assessed. Children manifest anxiety in many different ways, and emergency clinicians must be facile at recognizing the varying expressions of anxiety, especially in young children. A child with a facial laceration quietly sitting on the stretcher during the initial examination will not necessarily be a calm and cooperative patient during laceration repair (infants and toddlers). The nursing assessment at triage of the state of the child and accompanying adult(s) can be very helpful in some cases in determining the need for PSA. The child who was frightened and uncooperative in triage may be calm and compliant during a procedure. Unfortunately, the reverse is also true. When confronted by an extremely anxious child, ED personnel should ascertain what the parents have told the child about the upcoming procedure. Many parents, in the hope of lessening their child's anxiety, will tell the child that he or she will get a "shot" or a "needle" and that the procedure will "only hurt for a minute." This type of parental preparation, especially in young children who do not have the cognitive abilities to mediate their anxiety, often results in a significant increase in the child's anxiety and a decrease in their ability to cooperate, especially if the child has had a previous negative experience with a procedure in the ED. It is also important to assess the parent's level of anxiety, as this will determine the degree to which they can assist during the procedure. An extremely anxious parent or a parent who must take care of other siblings during the procedure will find it difficult to assist in distracting the child or otherwise helping him or her cope with the procedure.
PREVIOUS EXPERIENCE
Childrens' previous experience in hospitals can greatly affect their response to the current situation. Direct experience is not the only way to create anxious, frightened, and uncooperative patients, though. Images from television, stories from peers, or prior witness of a sibling being forcibly restrained for a laceration repair can leave a powerful and lasting impression. This type of influence should be especially suspect in children whose anxiety seems out-of-proportion in the present situation. Eliciting from the parents a history of a previous difficult experience in the ED can be a decisive factor in determining the degree of sedation required. Children who have had a recent unpleasant laceration repair, and who now present with a new laceration, may well require PSA as opposed to simple anxiolysis (either pharmacologic or nonpharmacologic) had there been no previous trauma.
CHILD'S BEHAVIOR AT ROUTINE PRIMARY CARE VISITS
Inquiring into how a child behaves during routine primary care visits can yield important information on how the children reacts to stressful situations, how cooperative he or she will be with the anticipated procedure, and whether pharmacologic management is needed. Children who cry but hold still when vaccinated may be more compliant than children who are described by their parents as being "afraid of doctors" or "wild" during visits to the primary care physician.

by skilled practitioners using a combination of non-pharmacological techniques (e.g., distraction, guided imagery, hypnosis, comforting, breathing techniques) in conjunction with topical anesthesia, careful local anesthesia, and, when necessary, brief forcible immobilization (by personnel or by a restraining device). In other cases, supplementing non-pharmacological techniques with topical or local anesthesia and anxiolysis with oral midazolam may be sufficient to permit successful wound repair. Although oral administration is most popular and least invasive, the nasal or rectal routes can also be used depending upon operator experience and preference.

Major procedures in uncooperative children.

Major painful procedures (e.g., fracture reduction, large loculated abscess incision and drainage, arthrocentesis of a major joint)

require systemic PSA. Options include IV fentanyl/midazolam or IM/IV ketamine. Ketamine may be the best option in such children, as dissociative sedation can consistently provide the immobilization and analgesia while maintaining protective airway reflexes and upper airway muscular tone.

PHARMACOPEIA

There is no universally correct or preferred medication or drug regimen. Many options are acceptable and successful. The best choice is an agent whose pharmacologic properties are familiar to the operator, is used frequently by the operator, is easily titrable, and has a short duration of action or is readily reversible. All drugs should be given in adequate doses, and underdosing narcotics or sedatives provides no useful purpose. Dosing recommendations for PSA drugs are shown in Table 34-11 (Table Not Available) , with specialized protocols for midazolam/fentanyl and ketamine shown in Table 34-12 and Table 34-13 , respectively. Individual agents are discussed in the following section.

Sedative-Hypnotic Agents

Chloral Hydrate

Pharmacology.

Chloral hydrate is a pure sedative hypnotic agent without analgesic properties. When administered orally, the average time to peak sedation is approximately 30 minutes, with a recovery time of an additional 1 to 2 hours. [39] [40] Residual motor imbalance and agitation may persist for several hours beyond this. [41] Rectal administration is erratically absorbed and therefore not recommended.

Adult use.

The use of chloral hydrate is limited to diagnostic imaging studies in children. It has no current uses in adults.

Pediatric use.

Chloral hydrate is widely used as a sedative to facilitate non-painful *outpatient* diagnostic procedures such as EEGs [40] and CT or MRI scanning. [42] [43] [44] [45] [46] Pentobarbital IV appears to be more effective for the latter indication than chloral hydrate, [47] although many centers prefer chloral hydrate in younger children (e.g., younger than 18 months) simply to avoid the need for IV access. [43] [44] [47] This drug does not readily lend itself to frequent use in the ED.

Adverse effects.

Despite a wide margin of safety, chloral hydrate can cause airway obstruction and respiratory depression, especially at higher doses (75–100 mg/kg). [1] [40] [43] [45] [46] The incidence was 0.6% in one large series. [40] There is no known dosage threshold of chloral hydrate below which this potential complication can be consistently avoided, [1] [45] and, accordingly, standard interactive and mechanical monitoring precautions apply to chloral hydrate as they do to other PSA agents.

Because it is a halogenated hydrocarbon, overdoses of chloral hydrate can be arrhythmogenic. Beta blockers may be effective in terminating ventricular arrhythmias. Despite reports of potential carcinogenicity, the AAP has judged that the evidence is currently insufficient to avoid single doses of chloral hydrate for this reason alone. [48]

Midazolam

Pharmacology.

Benzodiazepines are a group of highly lipophilic agents that possess anxiolytic, amnestic, sedative, hypnotic, muscle relaxant, and anticonvulsant properties. They lack direct analgesic properties, and thus are commonly co-administered with opioids. Caution must be exercised when using benzodiazepines and opioids together, since the risks of hypoxia and apnea are significantly greater than when either is used alone. [49]

Midazolam is by far the most common benzodiazepine used for PSA, and is preferred over the longer-acting lorazepam and diazepam. The time to peak effect for midazolam is approximately 2 to 3 minutes when given IV. Unlike diazepam, midazolam and lorazepam are water- soluble, making parenteral administration less painful and mucosal absorption faster. Midazolam is readily reversed with flumazenil.

Adult use.

Midazolam can be effectively used for moderate and deep sedation through careful IV titration to effect, typically together with fentanyl (see Table 34-12).

Pediatric use.

The advantage of midazolam over other benzodiazepines for pediatric PSA is its short duration of action, reversibility, and availability in multiple routes of administration. Midazolam may be used for the same indications and manner as in adults. Some children require larger doses than would be typical for adults on a mg/kg basis, [50] and paradoxical responses (e.g., hyperexcitability) are not uncommon. [41] [51] [52]

To avoid the need for IV access in frightened children, midazolam has been alternatively administered via the IM, [53] oral, [51] [54] [55] [56] [57] [58] intranasal, [54] [59] [60] [61] and rectal [62] [63] routes. However, the inability to effectively titrate using these routes dictates that a reliable depth of sedation cannot be predictably or regularly achieved. Thus, these non-intravenous routes are primarily reserved for pure anxiolysis and/or minimally painful procedures. Respiratory depression can also occur via these routes. [58]

Adverse effects.

When administered by skilled practitioners using standard precautions (see Table 34-12), the safety profile for midazolam is excellent. [5] [6] [64] However, when administering benzodiazepines, one must maintain continuous vigilance for respiratory depression. [1] [41] [49] [64] [65] Such respiratory depression is dose-dependent and greatly enhanced in the presence of ethanol or other depressive drugs, especially opioids. These effects are exaggerated in the elderly. Deaths from undetected apnea have occurred, [49] underscoring the critical role for continuous interactive and mechanical monitoring.

Benzodiazepines induce minimal cardiovascular depression. Although hypotension can occur, it is rare when the agents are carefully titrated. One reason midazolam is ideal for painful procedures is its significant amnesic effect. Even though patients appear to feel pain during the procedure, it is often not remembered.

Pentobarbital

Pharmacology.

Pentobarbital is a barbiturate capable of profound sedation, hypnosis, amnesia, and anticonvulsant activity in a dose-dependent fashion. It has no inherent analgesic properties. When carefully titrated IV, sedation is evident within 5 minutes with a duration of approximately 30 to 40 minutes. [66]

Adult use.

Pentobarbital has no advantage over midazolam for adult PSA and is rarely used for this purpose.

Pediatric use.

Pentobarbital is the IV sedative of choice in many centers for diagnostic imaging in children. [44] [47] [66] [67] [68] [69] It is regarded as superior to midazolam[47] [66] [69] or chloral hydrate[47] for this indication. Pentobarbital, like midazolam, is available in multiple routes of administration.

Adverse effects.

Like other barbiturates, pentobarbital can lead to respiratory depression and hypotension, as it is a negative inotrope. [44] [47] [66] [69]

Ultrashort-Acting Sedatives

The current ultrashort-acting sedatives used or being considered for use in ED PSA are thiopental, methohexital, propofol, and etomidate. Their use is rapidly expanding in the ED setting, but their exact role is still evolving. All can rapidly produce potent sedation, and all exhibit rapid awakening (<5 minutes) following drug discontinuation. As described later, rectal thiopental and methohexital have established safety and efficacy for pediatric neuroimaging.

Some controversy surrounds the IV administration of these agents for ED PSA.[70] Proponents cite their extremely rapid onset and recovery as enormous advantages over other sedatives. Critics cite the level of continuous vigilance required to achieve a desired effect while simultaneously avoiding significant cardiopulmonary depression, as these agents can exhibit rapid swings in levels of consciousness. An additional dedicated clinician (separate from the individual performing the procedure) is required to oversee medication administration; however, critics wonder whether oversedation and impairment of protective airway reflexes can be reliably avoided in spite of this precaution. Currently available series do not sufficiently quantify depth of sedation, and it is unclear whether some subjects experienced periods compatible with general anesthesia. Although such profound obtundation creates superlative procedural conditions, it also raises important issues regarding aspiration risk and compliance with JCAHO and hospital PSA standards. [70]

Thiopental and methohexital.

Because of their lipid solubility, barbiturates are rapidly absorbed rectally. When given by this route in children, thiopental and methohexital can reliably produce anxiolysis and sedation suitable for CT or MRI scanning. [71] [72] [73] [74] [75] [76] Respiratory depression is unusual when using typical doses (see Table 34-11 (Table Not Available)) but can occur. [71] [72] [74] [75] [76] Standard interactive and mechanical monitoring are warranted as with any PSA.

When given IV, both thiopental and methohexital produce sedation within 1 minute. Clinical recovery is rapid (approximately 15 minutes) and reflects the rapid redistribution of these agents from the central nervous system (CNS) to the periphery. These barbiturates have well-established use as sedatives for rapid-sequence intubation. IV methohexital has been administered for brief PSA in both adults [77] and children,[78] [79] a technique labeled "brief unconscious sedation." [78] Procedural conditions were excellent, although in one report apnea occurred in 10% of the patients. [77] The depth of sedation achieved in these small series is not well-described, but appears to be at or past levels consistent with deep sedation. Barbiturates can frequently cause hypotension at typical IV doses, so their use should be avoided whenever possible in patients with volume depletion or cardiovascular compromise.

Propofol.

Propofol has revolutionized the practice of anesthesiology. It is often used in the ICU setting for seizure control and for benzodiazepine-resistant agitation. Carefully titrated by bolus or by using an infusion pump, this drug can induce deep sedation or general anesthesia within 1 minute. Recovery following discontinuation averages 5 to 15 minutes, even after prolonged administration. Propofol exhibits inherent antiemetic and perhaps euphoric properties, and patient satisfaction is typically high. The adverse effects of this drug are potent respiratory and cardiovascular depression. Sudden apnea and hypotension (by direct negative inotropy as well as arterial and venodilatation) are well-known, as are rapid swings in consciousness. [67] [70] [79A] [79B] [79C] [79D] [79E]

Thus far the published experience using propofol for ED PSA is limited. As expected, existing reports cite rapid recoveries and a high level of efficacy. [67] [79A] [79B] [79D] [79E] The depth of sedation achieved in these reports is again not well-described, but appears to be frequently at or past levels consistent with deep sedation.

If it is ultimately shown that emergency clinicians can consistently administer this drug in a manner that avoids oversedation, respiratory depression, and hypotension, propofol could potentially become the IV PSA sedative agent of choice in patients of all ages.

Etomidate.

Etomidate produces sedation, anxiolysis, and amnesia equal to that of barbiturates, but with significantly fewer adverse respiratory or hemodynamic effects. Its onset of action and recovery are similar to those of thiopental and methohexital. In many centers etomidate is the induction agent of choice for rapid-sequence intubation in both adults and children, especially for patients with head trauma or marginal blood pressure. It is now commonly used for PSA. Some clinicians combine etomidate with narcotics, such as fentanyl, but the use of this combination has not been well-studied. If narcotics are used as adjuncts to etomidate, an appropriate reduction in dosing to account for the synergistic effect is required. The primary adverse effects of etomidate are respiratory depression, myoclonus, nausea, and vomiting. [80] [81] [82] Myoclonus is benign but can be disconcerting. It consists of transient jerking or twitching movements that can be mistaken for a seizure. Transient adrenal suppression occurs with etomidate, but appears to lack clinical significance for single doses. [83]

As with the other ultrashort- acting agents, preliminary reports describe rapid recovery and a high level of efficacy using IV etomidate for PSA in both adults and children.[80] [81] [82] Similarly, the depth of sedation achieved in these reports is not well-described, but appears to be frequently at or past levels consistent with deep sedation.

Analgesic Agents

Fentanyl

Fentanyl is the most common opioid used for PSA due to its rapid onset, brief duration of action, rapid reversibility by Narcan, and lack of histamine release. [7] Morphine and meperidine are instead preferred for pure pain control owing to their longer duration of action. ED patients frequently receive morphine or meperidine initially for acute analgesia, and then later are administered shorter-acting agents to facilitate a needed procedure. Although longer-acting opioids can be readily used for analgesia during PSA, they will be associated with longer recovery times and a higher incidence of histamine-related effects (e.g., nausea/vomiting, hypotension, pruritus). Fentanyl lacks these effects and therefore is preferred unless simply unavailable.

Pharmacology.

Fentanyl is 75 to 125 times more potent than morphine and has no intrinsic anxiolytic or amnestic properties. A single dose given IV has rapid onset (<30 seconds), with a peak at 2 to 3 minutes and brief clinical

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TABLE 34-11 -- PSA Drug Dosing Recommendations

(Not Available)

From Krauss B, Green SM: Sedation and analgesia for procedures in children. N Engl J Med 342:938, 2000. Adapted with permission.

TABLE 34-12 -- Procedure for Moderate to Deep Sedation with Intravenous Midazolam and Fentanyl

Caveats
• Do not consider this procedure if you lack experience with the drugs or do not have the time to perform procedural sedation and analgesia (PSA) properly. Do not attempt this procedure if the pulse oximeter, suction, oxygen, or bag-mask are not working, the IV is not secured, or the room is too small or not set up for PSA.
• This is a two-person procedure, one to monitor the patient and one to perform the procedure.
• Individual response to the drugs is variable and dependent upon the patient's underlying physiologic state and the presence of concomitant drugs/medication.
• Maximum drug effect occurs 2–3 minutes following administration. Proceed slowly and patiently, allowing the medication to take full effect before giving the next dose.
• Have naloxone and flumazenil immediately available for oversedation and/or respiratory depression.
• If the patient seems overly sedated, begin the procedure. The pain of the procedure often stimulates respiration and lessens sedation.
Contraindications—Absolute (Risks Essentially Always Outweigh Benefits)
Active hemodynamic instability
Active respiratory distress or hypoxemia
Contraindications—Relative (Risks May Outweigh Benefits)
Respiratory depression or altered level of consciousness
Anticipated difficulty if ventilatory assistance should become necessary (e.g., facial deformity or trauma, small mandible, large tongue, trismus)
Protocol
• Establish IV access.
• Connect appropriate monitoring equipment to the patient. Routine supplemented oxygen is commonly used but not always mandated.
• Pulse, respiratory rate, blood pressure, and level of consciousness should all be recorded initially, and periodically throughout the procedure depending on the depth of sedation.
• Suction equipment, oxygen, a bag-valve-mask, and reversal agents should be immediately available. An age-appropriate resuscitation cart with oral and nasal airways, endotracheal tubes, and a functioning laryngoscope must be nearby.
• The order of the drugs is one of personal preference. The ratio of analgesia to sedation is determined by the nature of the procedure. Some procedures require primary analgesia and secondary anxiolysis/sedation (e.g., abscess incision and drainage, bone marrow aspiration, arthrocentesis, burn debridement, central catheter placement). In this case, administer fentanyl first. Others require primary anxiolysis/sedation with secondary analgesia (e.g., lumbar puncture, simple foreign body removal); administer midazolam first.
• Administer local anesthesia if indicated after PSA initiated (this often serves to help gauge effectiveness of systemic analgesia).
• Perform the procedure. Additional doses of fentanyl or midazolam may be required if further pain or anxiety are noted based on the response and length of the procedure.
• If hypoxemia, oversedation, or slowed respirations are seen during or after procedure, the patient should be first stimulated while oxygen is applied and the airway repositioned. If the patient's response is insufficient, assist ventilations with a bag-valve-mask. Reversal agents should be considered if there is not a prompt response to assisted ventilation.
• Continue close observation until the patient is awake and alert, and release the patient with a friend, parent, or relative only after a sufficient discharge score has been attained.

duration (20 to 40 minutes). This increase in potency and onset of action is in part related to its greater lipid solubility, which facilitates its passage across the blood-brain barrier. The effects of fentanyl can be rapidly and completely reversed with opioid antagonists (e.g., naloxone, nalmefene). When respiratory depression occurs with fentanyl/midazolam PSA, it is usually due primarily to fentanyl.

Adult use.

Because of its pharmacokinetics, IV fentanyl is an ideal agent when analgesia is required for painful procedures; it can be easily and rapidly titrated. [7] As anxiolysis and sedation do not occur at low doses of fentanyl (1–2 mcg/kg), the concurrent administration of a pure sedative, most commonly midazolam, is advisable, especially in children (see Table 34-12).

Pediatric use.

The combination of fentanyl and midazolam remains one of the most popular PSA sedation regimens in children, with a strong safety and efficacy profile when both drugs are carefully titrated to effect. [6] [64] [84] [85] Any necessary level of mild to deep sedation can be achieved using these agents.

Fentanyl is also available in an oral transmucosal preparation. Although this novel and noninvasive delivery route obviates the need for IV access, titration is difficult and efficacy is variable. [86] Furthermore, the incidence of emesis is high (31% to 45%), [86] [87] and this formulation has never become popular for PSA.

Adverse effects.

Like all opioids, fentanyl can cause respiratory depression. [6] [7] [64] [84] [85] When used for PSA, standard interactive and mechanical monitoring are required. As the opioid effect is most pronounced on the CNS respiratory centers, apnea can precede loss of consciousness. If apnea should occur, verbal or tactile stimulation should be attempted prior to administration of opioid antagonists. As discussed earlier, caution must be exercised when using benzodiazepines and opioids together, since

the risks of hypoxia and apnea are significantly greater than when either is used alone. [5] [49]

In the absence of significant ethanol intoxication, hypovolemia, or concomitant drug ingestion, hypotension is rare, even with very large doses of fentanyl (doses of 50 mcg/kg are common in adult and pediatric cardiac surgery). Because of its safe hemodynamic profile, fentanyl is an ideal analgesic agent for use in critically ill or injured patients. Additionally, nausea and vomiting are rare in distinction to analgesia with morphine or meperidine. A commonly observed reaction to fentanyl is facial pruritis and patients frequently scratch their noses. [7]

A rare side effect of fentanyl with potential for respiratory compromise is chest wall rigidity. This complication is related to higher doses (>5 mcg/kg as a bolus dose) than those used for PSA, and has not been reported in any ED series. [6] [7] [84] [85] If it should occur, chest wall rigidity usually can be reversed with opioid antagonists and/or positive-pressure ventilation. Equipment for urgent pharmacological paralysis should be available if reversal and positive-pressure ventilation are unsuccessful.

Diamorphine

Diamorphine is a promising nasal opioid that is currently available in Great Britain but not the United States. Diamorphine has a similar onset and duration of action to morphine; however, its higher water solubility permits potent doses to be delivered in the small (0.1 mL) volumes necessary

for comfortable intranasal administration. In two studies of children and teenagers with fractures, intranasal diamorphine 0.1 mg/kg provided a similar level of analgesia with faster onset than IM morphine 0.2 mg/kg. Intranasal spray administration was better tolerated than the injection, and there were no adverse events. [88] [89] Diamorphine may prove to be a useful initial analgesic for children and teenagers with acute pain, although, practically, an IV must be established to permit titration to full pain relief and PSA for any needed procedures (e.g., fracture reduction). The role for diamorphine in adults remains to be determined.

Other Short-Acting Opioids

Sufentanil, alfentanil, and remifentanil are other short-acting opioids that have a potential role in PSA. However, currently there is insufficient published experience to warrant their routine use. Although intranasal sufentanil 0.75 mcg/kg appeared promising in one small pediatric trial, [90] in another, doses of 1.5 mcg/kg resulted in oxygen desaturation in 8 of 10 children studied. [59] This low toxic-therapeutic ratio and inability to titrate would appear to limit the utility of intranasal sufentanil. In the one published report of IV remifentanil with midazolam for PSA, there was an unacceptably high incidence of hypoxemia. [91] Currently there does not appear to be a clinically important advantage to these drugs compared to fentanyl.

KETAMINE

Pharmacology.

Ketamine produces a unique state of cortical dissociation that permits painful procedures to be performed more consistently and effectively than with other PSA agents. This state of "dissociative sedation" is characterized by profound analgesia, sedation, amnesia, and immobilization (Fig. 34-1), and can be rapidly and reliably produced with IV or IM administration. Ketamine has been widely used worldwide since its introduction in 1970 and has demonstrated a remarkable safety profile in a variety of settings. [4] [6] [92] [93] [94] In 1999, the JCAHO confirmed that ED ketamine administration is fully compliant with its standards when administered according to protocol. [95] Clinicians administering ketamine must be especially knowledgeable about the unique actions of this drug and the numerous contraindications to its use (see Table 34-11 (Table Not Available)).

Ketamine differs from all other PSA agents in several important ways. First, it uniquely preserves cardiopulmonary stability. Upper-airway muscular tone and protective airway reflexes are maintained. Spontaneous respiration is preserved, although when administered IV, ketamine must be given slowly (over 1 to 2 minutes) to prevent respiratory depression. Second, it differs from other agents in that it lacks the characteristic dose-response continuum to progressive titration. At doses below a certain threshold, ketamine produces analgesia and sedation. However, once a critical dosage threshold (approximately 1 to 1.5 mg/kg IV or 3 to 4 mg/kg IM) is achieved, the characteristic dissociative state abruptly appears. This dissociation has no observable levels of depth, and thus the only value of ketamine "titration" is to maintain the presence of the state over time. Finally, the dissociative state is not consistent with formal definitions of moderate sedation, deep sedation, or general anesthesia (see Table 34-1), and therefore must be considered from a different perspective than agents that exhibit the classical sedation continuum. [13] [96]

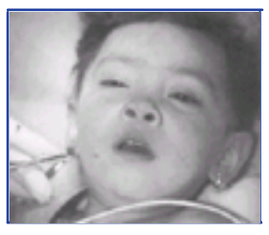

Figure 34-1 A child undergoing repair of a laceration while dissociated with ketamine. The blank stare is typical.

Ketamine is most effective and reliable when given IV or IM. Ketamine has a one arm-brain circulation time when given IV with onset of dissociation noted within 1 minute and effective procedural conditions lasting for about 10 to 15 minutes. When given IM, the same effect is achieved within 5 minutes, with effective procedural conditions for about 15 to 30 minutes. The typical duration from dosing until dischargeable recovery is 50 to 110 minutes when given IV, and 60 to 140 minutes when given IM. [92] [97]

Like the benzodiazepines, ketamine undergoes substantial first pass hepatic metabolism. As a result, oral and rectal administration results in less predictable effectiveness and requires substantially higher doses. Clinical onset and recovery are substantially longer than when given parenterally, and thus these routes are rarely used in the ED. [61] [62] [98]

Ketamine can induce salivation, and is routinely coadministered with an anticholinergic. Atropine is most commonly chosen in emergency medicine due to its ready familiarity to clinicians and nurses, although glycopyrrolate is an equally acceptable but not superior alternative (see Table 34-13).

Adult use.

Ketamine is widely and successfully used in adults throughout the developing world for both minor and major surgery, particularly in areas lacking resources for inhalational anesthesia. [92] [93] [99] [100] Hallucinatory so-called "emergence reactions" have been reported in up to 30% of adults receiving ketamine (although rare in children), and can be fascinating and pleasurable, or alternatively unpleasant and nightmarish. [92] Concurrent benzodiazepines are believed to blunt but not entirely eliminate such reactions in adults, [92] [93] [99] [100] and apprehension regarding such unpleasant recoveries has limited the popularity of ketamine administration in the developed world for adults.

One study reported success administering dissociative doses (2 mg/kg IV) of ketamine with concurrent midazolam (0.07 mg/kg IV) to 77 ED adults to facilitate painful procedures (e.g., abscess incision and drainage, fracture reduction). There were no moderate or severe emergence reactions, and only five patients experienced mild reactions. [101] Furthermore,

TABLE 34-13 -- Procedure for Dissociative Sedation with Ketamine

Caveats
Do not consider if you are not experienced with ketamine or if you do not have time to perform such sedation properly. Do not attempt the procedure if the pulse oximeter, suction, oxygen, or bag-mask are not working, or the room is too small or not set up for PSA.
This is a two-person procedure, one to monitor the patient and one to perform the procedure. Both must be knowledgeable regarding the unique characteristics of ketamine.
Ketamine is best suited for (1) short, painful procedures, especially those requiring immobilization (e.g., complex facial laceration, burn debridement, fracture reduction, abscess incision and drainage, central line placement, colonoscopy, tube thoracostomy); or (2) examinations judged likely to produce excessive emotional disturbance (e.g., pediatric sexual assault examination).
Contraindications—Absolute (Risks Essentially Always Outweigh Benefits)
Age <3 months (suggestive evidence of higher risk of airway complications)
History of airway instability, tracheal surgery, or tracheal stenosis (presumed but insufficient evidence of higher risk of airway complications)
Known or suspected psychosis, even if currently stable or controlled with medications (supportive evidence of exacerbatory potential)
Contraindications—Relative (Risks May Outweigh Benefits)
Age 3 to 12 months (higher risk of airway complications)
Procedures involving stimulation of the posterior pharynx (moderately higher risk of laryngospasm)
Active pulmonary infection or disease, including upper respiratory infection or asthma (moderately higher risk of laryngospasm)
Known or suspected cardiovascular disease including angina, heart failure, or hypertension (exacerbation due to sympathomimetic properties of ketamine). Avoid ketamine in patients with ED blood pressures 140/90 or above. Avoid ketamine in older adults with risk factors for coronary artery disease.
Head injury associated with loss of consciousness, altered mental status, or emesis (elevated intracranial pressure with ketamine)
Central nervous system masses, abnormalities, or hydrocephalus (elevated intracranial pressure with ketamine)

Glaucoma or acute globe injury (elevated intraocular pressure with ketamine)
Porphyria, thyroid disorder, or thyroid medication (enhanced sympathomimetic effect)
Protocol
IV access is unnecessary in children receiving IM ketamine. IV access is desirable in adults to permit prompt treatment of unpleasant recovery reactions should they occur.
Connect appropriate monitoring equipment to the patient.
Pulse, respiratory rate, blood pressure, and level of consciousness should all be recorded initially, and periodically throughout the procedure depending on the depth of sedation.
Suction equipment, oxygen, a bag-valve-mask, and reversal agents should be immediately available. An age-appropriate resuscitation cart with oral and nasal airways, endotracheal tubes, and a functioning laryngoscope must be nearby.
Educate accompanying family regarding the unique characteristics of the dissociative state, especially if they will be present during the procedure and/or recovery.
Adults and children of verbal age should be encouraged to "plan" specific, pleasant dream topics in advance of sedation (believed to decrease unpleasant recovery reactions).
Co-administer atropine 0.01 mg/kg (minimum 0.1 mg, maximum 0.5 mg) either IV just prior to ketamine, or draw up and have atropine by the bedside. For IM injection, mix with ketamine in the same syringe (hypersalivation suppression). Glycopyrrolate is an acceptable alternative at equipotent doses.
Benzodiazepine co-administration is unnecessary in children to blunt emergence reactions; however, such drugs should be readily available to treat rare unpleasant recovery reactions should they occur. Midazolam may also be used to attain pre-procedural anxiolysis prior to ketamine administration, especially in frightened toddlers and early school age children. Benzodiazepine prophylaxis should be considered in adults due to their higher baseline risk of unpleasant reactions and to enhance cardiovascular stability. Midazolam 2 to 4 mg slowly IV is an example of such pretreatment.
Ketamine is not administered until the clinician is ready to begin the procedure, as onset of dissociation typically occurs within 5 minutes.
Ketamine is administered as a single IM injection or IV loading dose, and there is no benefit from routine attempts to titrate to effect.
When administered IM, give 4–5 mg/kg with atropine mixed in the same syringe. Repeat ketamine dose (full or half dose IM without additional atropine) if sedation is inadequate after 5–10 minutes (unusual) or if additional doses are later required.
When administered IV, give a loading dose of 1.5 mg/kg IV over 1–2 minutes. 100 mg is a typical adult dose. IV administration more rapidly than over 1–2 minutes produces high CNS levels and has been associated with respiratory depression. Additional incremental doses of ketamine may be given (0.5 mg/kg) if initial sedation is inadequate, or if repeated doses are necessary to accomplish a longer procedure. Repeat doses of atropine are generally unnecessary.
Adjunctive physical immobilization may be occasionally needed to control random motion.
Adjunctive local anesthetic may be needed for incomplete analgesia, although this is unusual.

Route of Administration	**IM**	**IV**
Advantages	No IV access necessary	Ease of repeat dosing
		Slightly faster recovery
Peak concentrations and clinical onset	5 min	1 min
Typical duration of effective dissociation	15–30 min	10–15 min
Typical time from dose to discharge	60–140 min	50–110 min

Whenever possible, minimize lighting, noise, and physical contact during recovery until wakefulness is well-established. Advise family to not stimulate the patient prematurely.

there appears to be no reason to avoid standard dissociative doses when ketamine is administered to adults. In a 1996 study using subdissociative doses (0.2 mg/kg IV) for the goal of bronchodilation in the treatment of acute asthma, three of six adults suffered dysphoric reactions. [102]

Ketamine presents potential risks to patients with coronary artery disease, as it is sympathomimetic and produces mild to moderate increases in blood pressure, heart rate, and myocardial oxygen consumption. The actual risk remains unclear due to limited experience in adults with known coronary artery disease. [99]

Given the available data, it would appear appropriate for emergency clinicians to carefully transpose their experience with ketamine into selected adult situations. Careful patient selection can help minimize potential adverse events (see Table 34-13).[99] [101]

Pediatric use.

Ketamine is an ideal agent to facilitate short, painful procedures in children. The safety and efficacy of ketamine for this indication has been widely documented. [4] [5] [6] [92] [97] The IM route is simple and effective. Venous access is unnecessary, and atropine can be concurrently administered in the same syringe. [4] IV administration is attractive because a lower cumulative dose can be used, and recovery is faster than with the IM route. The primary caution is that with this route, ketamine must be administered slowly (each dose over 1 to 2 minutes) or respiratory depression and transient apnea can occur. [97]

Unpleasant recovery reactions are uncommon in children and teenagers, and are typically mild when they do occur. [103] [104] There is no evidence of any benefit from the prophylactic administration of concurrent benzodiazepines in children, [103] [104] and their role should be confined to treating unpleasant reactions if they should occur.

Adverse effects.

In the largest published ED series (1022 patients) the following adverse airway events were noted: airway malalignment (0.7%), transient laryngospasm (0.4%), and transient apnea or respiratory depression (0.3%). All were quickly identified and treated, and there were no sequelae. [4]

Vomiting was noted in 6.7% from the same series, and in most cases it occurred well into recovery.[4] The incidence was age-related, occurring in 12.1% of children aged 5 years or older, and 3.5% in those younger than 5 years. [94] There was no evidence of aspiration[4] ; indeed, in 30 years of regular use there have been no documented reports of clinically significant ketamine-associated aspiration in patients without established contraindications. Delayed vomiting may occur after discharge, and patients should be advised of this possibility. Because of its unique preservation of protective airway reflexes, ketamine may be preferred over other agents for urgent or emergent procedures when fasting is not assured. [4] [5] [92]

Mild agitation during recovery (whimpering or crying) was noted in 17.6% of children from the same series, with more pronounced agitation in 1.6%. The incidence was age-related, with agitation occurring in 12.1% of children aged 5 or older, and 22.5% in those younger than 5. [94] Only 2 of 1022 children had reactions that treating clinicians judged severe enough to require treatment, and both children responded promptly to small doses of midazolam. [4] Another study quantified the degree of recovery agitation using a 0 to 100 mm visual analog scale; the median rating of recovery agitation was a 5, likely below the threshold of clinical importance.[103]

NITROUS OXIDE

Pharmacology.

Inhaled nitrous oxide provides anxiolysis and mild analgesia. It is commonly dispensed at concentrations between 30 and 50% with oxygen composing the remainder of the mixture. Nitrous oxide quickly diffuses across biologic membranes and accordingly has a rapid onset of action (30 to 60 seconds). Maximum effect occurs after about 5 minutes, and the clinical effect wears off quickly upon discontinuation. At typical PSA concentrations there is preservation of hemodynamic status, spontaneous respirations, and protective airway reflexes. [105] [106] [107] [108] Nitrous oxide is widely used in dentistry at higher concentrations. [105]

Nitrous oxide has an excellent safety profile; however, as a sole agent it cannot reliably produce adequate procedural conditions. [105] [106] [107] [108] Given its relatively weak analgesic properties, in many cases nitrous oxide needs to be supplemented with an IV opioid and/or local anesthesia.

Adult and cooperative child use.

The safest method of nitrous oxide administration is via a self-administered demand-valve mask (Fig. 34-2). [106] [107] [108] Patients must generate a negative pressure of 3 to 5 cm H_2 O within the handheld mask or mouthpiece to activate the flow of gas. They can thus self-titrate themselves by inhaling at will through the mask. Naturally, this will only be effective when the patient is cooperative. This technique provides a built-in fail-safe in that if patients become somnolent, the masks will fall from their faces and gas delivery will cease.

Nitrous oxide can be used as an adjunctive anxiolytic during mildly painful procedures, or during local or regional anesthesia administration for other procedures. It may also be administered during difficult pelvic examinations, during attempts at difficult IV access, etc.

A double-tank system is commonly used to deliver the nitrous oxide and O_2 mixture. The system relies on a mixing valve preset to deliver a fixed ratio and will only deliver gas when O_2 is flowing. The double-tank system contains a fail-safe device that automatically stops the flow of nitrous oxide when the O_2 supply is depleted.

Uncooperative child use.

The primary limitation of self-administration is that it is ineffective in uncooperative patients, including most frightened young children. Continuous-flow nitrous oxide has been used in this population using a mask strapped over the nose, or over the nose and mouth (Fig. 34-3 (Figure Not Available)). [110] [111] [112] Nitrous oxide can effectively produce moderate or deep sedation when administered in this manner; however, this technique necessitates an additional clinician dedicated to continuous gas titration to avoid oversedation. Additionally, for unclear reasons the continuous-flow technique is associated with a higher rate of emesis (10%) [110] [111] [112] than self-administration (0 to 4%), [105] [106] [107] [108] posing a potential hazard when a mask is strapped over the child's mouth. Finally, the requirement of a mask hinders procedures involving the perioral and nasal areas of the face. [111]

Adverse effects and precautions.

A number of generally minor adverse effects may be seen, including nausea, dizziness, voice change, euphoria, and laughter. [105] [106] [107] [108] Nitrous oxide should be avoided in closed-space disease such as bowel obstruction, middle ear disease, pneumothorax, or pneumocephaly. Because of its property of high diffusibility, it has potential to increase the size of the closed space. This should be unlikely for short-term use in typical PSA concentrations.

Figure 34-2 *A*, A demand-flow nitrous oxide/oxygen system. *B*, Example of a face mask for a nitrous oxide system. The mask must be held in contact with the face by the patient.

A scavenging system must be in place to collect exhaled nitrous oxide, and care must be taken to ensure compliance with occupational safety regulations. Care should also be taken to avoid nitrous oxide exposure in pregnant ED staff members as nitrous oxide is a known teratogen and mutagen.

Figure 34-3 (Figure Not Available) Nitrous oxide/oxygen continuous-flow system. *(From Luhmann JD, Kennedy RM, Jaffe DM, McAllister JD: Continuous-flow delivery of nitrous oxide and oxygen: A safe and cost-effective technique for inhalation analgesia and sedation of pediatric patients. Pediatr Emerg Care 15:388, 1999.)*

Although the potential for abuse by ED staff exists, such abuse should be rare if simple steps are taken. As with other agents, a strict protocol of accountability should be in place. A simple locking device can be added to the cylinders of gas. In addition, the delivery valve or mouthpiece may be locked in the same location as controlled substances.

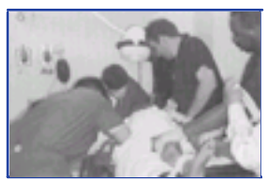

Figure 34-4 The downward spiral of fatal cocaine-induced delirium starts long before the chronic cocaine user is brought to the hospital, but physical restraints applied by police, security guards, and medical personnel only aggravate the situation. When it takes leather restraints and a bevy of personnel to subdue a delirious, violent, and totally out-of-control patient, it's time for aggressive and effective parenteral sedation and evaluation for and rapid correction of hyperthermia and acidosis. In the scenario shown here, it is impossible to even begin to adequately evaluate or treat this patient. The sagacious clinician knows, however, that this individual is an acute medical emergency.

OTHER PSA AGENTS

Historically a popular PSA cocktail was the intramuscular combination meperidine, promethazine, and chlorpromazine ("DPT," "Dem compound"). However, this regimen cannot be titrated, is frequently ineffective, and is associated with prolonged recovery times. [113] [114] Its use can no longer be recommended.

ANTAGONISTS

Reversal agents should not be routinely administered following administration of opioids or benzodiazepines for PSA, but rather should be reserved for rare situations of oversedation or respiratory depression. When administered, caution should be taken to avoid resedation after discharge by continuing to monitor patients until the effects of the PSA agents (which may last longer than the antagonist) wear off.

Naloxone.

Naloxone is an antagonist that competitively displaces opioids from opiate receptors. It rapidly reverses the analgesic and respiratory depressant effects of opioids. It may be administered IV, IM, SC, or even sublingually if needed, [115] and dosing has been standardized for infants and children. [116] Naloxone will not induce systemic opioid withdrawal symptoms in a patient without preexisting physiologic dependence. However, some patients will experience nausea with opioid reversal, and those patients with persistent pain following their procedure will be quite uncomfortable. Rapid reversal also may lead to return of anxiety and sympathetic stimulation. *If the situation permits, careful titration of small amounts of naloxone may permit partial rather than complete reversal.* The only absolute contraindication to the use of naloxone is administration to a neonate born to an opioid-dependent mother due to the risk of precipitating life-threatening opioid withdrawal.

Nalmefene.

Nalmefene is a long-acting opioid antagonist with a duration of action significantly longer than naloxone. [117] Nalmefene may be given IV, IM, or SC, although IV is the preferred route.[117] [118] Intravenously, it can be titrated in incremental doses of 0.25 mcg/kg every 2 to 5 minutes until the desired effect is attained. Although either naloxone or nalmefene will reverse analgesia due to opioids, naloxone is the preferred agent. For ED PSA, short-acting opioids such as fentanyl are commonly used and administration of a reversal agent with as prolonged duration of action as nalmefene does not confer any additional benefit. Furthermore, nalmefene would interfere with post-procedure opioid pain control.

Flumazenil.

Flumazenil is a benzodiazepine antagonist that can promptly reverse benzodiazepine-induced sedation and respiratory depression. [1] [13] [119] [120] In the setting of PSA, flumazenil is a safe and effective method of reversing oversedation caused by benzodiazepines. It is not routinely used to reverse PSA because of the potential for resedation, and many clinicians prefer to allow patients to recover on their own. Flumazenil has not been shown to substantially decrease the time of observation in the ED required of a patient undergoing PSA. Flumazenil lowers the seizure threshold and may rarely lead to life-threatening seizures. It should be avoided in settings of known benzodiazepine dependence, seizure disorder, cyclic antidepressant overdose, and elevated intracranial pressure. [121] It should also be given cautiously to patients who are on medications known to lower the seizure threshold (cyclosporine, tricyclic antidepressants, propoxyphene, theophylline, isoniazid, lithium). [121] These issues, however, are generally not involved in PSA in the ED. It has not been shown that simply taking therapeutic doses of these medications contraindicates flumazenil, and flumazenil-induced seizures are generally only associated with drug overdose. Rapid reversal also may lead to return of anxiety and sympathetic stimulation. If the situation permits, careful titration of small amounts of flumazenil will reduce the risk of adverse effects and may permit partial rather than complete reversal.

CHEMICAL RESTRAINT OF THE PSYCHOTIC OR AGITATED PATIENT

Not infrequently patients present to the ED with combative behavior due to psychosis, dementia, withdrawal syndromes,

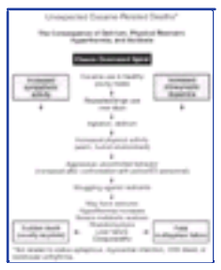

Figure 34-5 The postulated downward spiral of agitated delirium in a cocaine-intoxicated patient. An adverse outcome may theoretically be ameliorated with the aggressive use of chemical restraint.

or alcohol/drug intoxication. The latter occurs commonly in the setting of minor or major trauma and especially with stimulants such as cocaine or phencyclidine. Combative individuals can present a significant threat to themselves and their families, to other ED patients and their families, and to the ED staff.

Figure 34-6 When an indwelling venous catheter cannot be obtained in the violent patient, the clinician may mainline the appropriate medications. By enlisting the help of security personnel, the arm or foot is immobilized by brute force, and medication is given by bolus injection into a large anticubital (*A*) or saphenous vein (*B*). Alternatively, two security guards lie across the upper thighs and lower stomach, and the clinician injects directly into the femoral vein (being careful to avoid an intra-arterial injection).

While a show of force, bargaining, psychological manipulation, enlisting family members, etc., may aid the clinician in controlling disruptive individuals, some patients, particularly those with drug-induced delirium, simply cannot comply with such measures (Fig. 34-4). Security personnel may be reluctant to intervene because of the fear of contracting communicable diseases from biting and bleeding patients. Basic vital signs, tests, or other interventions cannot proceed under such conditions. This scenario taxes the clinician's self-control, patience, negotiating skills, and clinical judgment on a regular basis. *However, it is imperative that wildly agitated, violent, combative, or uncontrollable patients not be physically restrained for prolonged periods without some type of adjunctive pharmacologic intervention.* The restrained yet still struggling patient quickly becomes hyperthermic and acidotic, and may experience rhabdomyolysis or other forms of musculoskeletal trauma from the restraints. In some causes sudden death due to cardiovascular collapse or multisystem failure may ensue, even in young and previously healthy individuals. [122] [123] The prehospital hobble position (hands and feet bound behind the back and placed prone) has been particularly emphasized as an undesirable method of restraint.[124] The classic downward spiral in cocaine-related delirium in the setting of physical restraint without chemical sedation is noted in Figure 34-5 .

Rapid, safe, and effective pharmacological methods (i.e., rapid tranquilization—[RT]) are available for use in these situations to gain control of patients who are agitated, potentially assaultive, destructive, or overtly violent. It is critical to accurately assess the etiology of the combative behavior and not to mistake hypoglycemia, head trauma, pain, or hypoxia for psychosis. However, in many cases, the etiology of the agitation, violence, or uncontrolled behavior simply may not be known prior to intervention being required. Two classes of drugs are currently used for rapid tranquilization: benzodiazepines and neuroleptics.

Under most circumstances, the IV use of such medication is preferred, although venous access may be problematic. When an indwelling intravenous port is not available, direct IV injection (mainlining) may be successful. One method is to restrain the groin, anticubital area, or leg by brute force,

TABLE 34-14 -- Drug Dosing Recommendations for Rapid Tranquilization

Drug	Clinical Effects	Indications	Adult Dose[2]	Pediatric Dose	Onset (Min)	Duration (Min)	Comments
NEUROLEPTICS							
Droperidol (*Inapsine*)	CNS tranquilization, anti-emetic, anti-psychotic	Rapid tranquilization, chemical restraint	2.5–10 mg (start dose at 2.5 mg in elderly patients and patients receiving concomitant CNS depressants)	0.05–0.07 mg/kg IM/IV	IV: 3–10	60–120	May cause EPS. FDA black box warning regarding QT-related dysrhythmias. (see text). Hypotension.
					IM: 3–10		
Haloperidol (*Haldol*)	CNS tranquilization, anti-psychotic	Rapid tranquilization, chemical restraint	5–10 mg IM/IV (start at 2 mg in the elderly and patients receiving concomitant CNS depressants	5–12 years old: 0.1 mg/kg IM/IV >12 years old: Use adult dosing	IV: 5–10	60–120+	May cause EPS.
					IM: 10–20		
SEDATIVE/HYPNOTICS							
Lorazepam* (*Ativan*)	Sedation, motion control, anxiolysis	Anxiolytic and sedative adjunct to neuroleptic	1–4 mg IM/IV	0.05–0.1 mg/kg IM/IV (not to to exceed adult dosing)	IV: 2–3	IV: 45–60	Monitor for respiratory depression
					IM: 10–20	IM: 60–120	
TREATMENT OF EXTRAPYRAMIDAL SYMPTOMS							
Benztropine (*Cogentin*)	Reversal of EPS	EPS (dystonia, akathisia)	2 mg PO/IM	0.5–2 mg PO/IM	PO: 15–60	PO/IM: 24 hours	Anticholinergic effects (e.g., dry mouth, urinary retention, tachycardia).
					IM: 10		

Diphenhydramine (*Benadryl*)	Reversal of EPS	EPS (dystonia, akathisia)	25–50 mg PO/IM/IV	1 mg/kg PO/IM/IV	PO: 30–60	PO/IM/IV: 6–8 hours	Anticholinergic effects (e.g., dry mouth, urinary retention, tachycardia).
					IM: 20–30		
					IV: 5–10		

*There is no maximum dose of benzodiazepines. Massive doses may be required in severely agitated patients. EPS = extrapyramidal symptoms.

with the direct IV injection of the sedating medication (Fig. 34-6). When the patient has been controlled, indwelling venous access can be accomplished. In the less stressful situation, the IM route may be acceptable. The use of IM ketamine to control a dangerous and violent patient without venous access has been described, but the role of ketamine in this situation is presently unclear. [125]

There is no specific maximum dose for drugs used for chemical restraint.

Occasionally massive doses may be required. If benzodiazepine or neuroleptics, or both, are not effective, muscular paralysis and mechanical ventilation may be necessary to gain control of the patient.

Benzodiazepines

Historically, the first drugs used as adjuncts to phenothiazines for RT were barbiturates, allowing decreased dosing of chlorpromazine, thereby minimizing the incidence of hypotension. Benzodiazepines have now replaced barbiturates as adjuncts to neuroleptics in RT. The benzodiazepine of choice for RT in both adults and children is lorazepam (Ativan, adult dosing: 1–4 mg IM/IV; pediatric dosing: 0.05–0.1 mg/kg IM/IV) because of its bioavailability and rapid IM absorption, intermediate duration, and low side-effect profile (Table 34-14). It is commonly given with a neuroleptic (e.g., haloperidol) to achieve rapid anxiolysis and anti-psychosis. [126] This combination has been shown to have a more rapid onset and fewer extrapyramidal symptoms (EPS) than with haloperidol alone, and is well-tolerated with no respiratory depression or hypotension. [127]

Neuroleptics

The first anti-psychotic agents used for rapid tranquilization were the low-potency phenothiazines (e.g., chlorpromazine [Thorazine], thioridazine [Mellaril]). These agents are highly sedating and manifest anticholinergic effects, alpha adrenergic blockade (leading to orthostatic hypotension), a lowered seizure threshold, and cardiac toxicity in overdose. During the 1990s, these agents have been replaced by the butyrophenones haloperidol (Haldol) and droperidol (Inapsine) (see Table 34-14).

Haloperidol (adult dosing: 5–10 mg IM/IV [start at 2 mg in the elderly and patients receiving concomitant CNS depressants]; pediatric dosing—5–12 years old: 0.1 mg/kg IM/IV, >12 years old: use adult dosing) and droperidol (adult dosing: 2.5–10 mg [reduce dose for elderly patients and patients receiving concomitant CNS depressants]; pediatric dosing: 0.05–0.07 mg/kg IM/IV) are high-potency agents that, compared to the phenothiazines, are relatively free of anti-cholinergic effects, alpha-blocking properties, and cardiac toxicity, but cause more EPS. [128] The overall incidence of EPS (e.g., dystonic reactions, akathisia) with butyrophenones is low, occurring in less than 10% of RT patients within the first 24 hours, and such reactions can be effectively treated with IM or IV diphenhydramine (Benadryl, adult dosing: 25–50 mg PO/IM/IV; pediatric dosing: 1 mg/kg PO/IM/IV and/or benztropine; Cogentin, adult dosing: 2 mg PO/IM; pediatric dosing [>3 years old]: 0.5–2 mg PO/IM).[128]

Acute dystonia in particular is a non-dose-dependent, idiosyncratic reaction. Both neuroleptics have minimal effect on respiratory drive and airway reflexes, and their safety and efficacy in the ED have been well-documented.[129] [130] [131] Droperidol has a faster onset time IM, shorter duration, and is more sedating than haloperidol.[129] [132] Neuroleptics will also exacerbate pre-existing Parkinsonism, and should be avoided in these patients. A much-feared idiosyncratic reaction, occurring in 1% of patients on neuroleptics, is neuroleptic malignant syndrome (NMS), characterized by potentially severe autonomic instability (hyperthermia, hypertension, and rigidity). NMS can rarely occur in the setting of RT with patients on chronic antipsychotic medications. Haloperidol has been reported to lower the seizure threshold in animals, but this remains a theoretical concern clinically as there are no reported cases in humans despite its use in post-ictal and alcoholic patients. Although droperidol has been used extensively in the ED over the last 10 years and clinicians experienced in its use consider it safe and effective, its use has decreased since the FDA issued "black box warning" in December 2001 regarding QT-related dysrhythmias. [133] Patients receiving droperidol must be carefully screened for a history of QT-related dysrhythmias and whenever possible receive a baseline ECG or rhythm strip to ensure a normal QT interval. However, there is little rational behind the "black box warning" to justify a change in ED practice with this agent. [134]

Alternative Agents

Ketamine has been effectively used as an alternative tranquilization agent [125] ; however, it may exacerbate intracranial hypertension in patients with head trauma or CNS infections.[92] A reserve option for situations of grave danger is paralysis with succinylcholine IV or IM (1–1.5 mg/kg IV; 4 mg/kg IM), followed by rapid airway control.[135]

Acknowledgment

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Section VI - Soft Tissue Procedures

Chapter 35 - Principles of Wound Management

Richard L. Lammers

Acute traumatic wound management is one of the most common procedures in the practice of emergency medicine. There are many areas of controversy in the medical literature, numerous personal preferences, and a plethora of protocols and individual approaches. Few agreed upon standards of care exist for many aspects of wound care.[1] Also, there are many myths and misconceptions surrounding the vagaries of wound preparation and wound care. The purpose of this chapter is to give the clinician a general approach to wound care and to suggest reasonable techniques; however, lack of data to support or refute many of the described clinical issues that confront clinicians daily renders much of the following discussion intuitively practical but somewhat theoretical.

Wound care involves much more than closure of divided skin. The primary goal of wound care is not the technical repair of the wound; it is providing optimal conditions for the natural reparative processes of the wound to proceed. Primary wound healing is not an inevitable process. For centuries, victims of wounds commonly experienced inflammation, infection, and extreme scarring; in fact, these processes were considered part of normal wound repair. Only in the late 19th century did surgeons first realize that sepsis could be separated from healing. [2]

The cornerstones of wound care are cleaning, debridement, closure, and protection. The primary technical objectives in wound care are as follows:

1. Preserving viable tissue and removing nonviable tissue
2. Restoring tissue continuity and function
3. Optimizing conditions for the development of wound strength
4. Preventing excessive or prolonged inflammation
5. Avoiding infection and other impediments to healing
6. Minimizing scar formation

When presenting to emergency departments (EDs) with acute wounds, patients report that their top priorities in the management of their wounds include prevention of infection, return to normal function, good cosmetic outcome, and minimal pain during repair. [3] [4] This chapter reviews current strategies for achieving all of these goals.

BACKGROUND

Wound Healing

Emergency clinicians should have a basic understanding of the process of wound healing. Highlights of this complex phenomenon as they relate to clinical decision making are presented.

Wounds extending beneath the epithelium heal by forming scar tissue. Inflammation, epithelialization, fibroplasia, contraction, and scar maturation constitute the stages of this nonspecific repair process. [2] [5] [6] Inflammation is a beneficial response that serves to remove bacteria, foreign debris, and devitalized tissue—a biologic debridement. Polymorphonuclear and mononuclear leukocytes concentrate at the site of injury and phagocytose dead and dying tissue, foreign material, and bacteria in the wound. [7]

As white blood cells die, their intracellular contents are released into the wound. In excessive amounts, they form the purulence characteristic of infected wounds. Some exudate is expected even in the absence of bacterial invasion; however, infection with accumulation of pus interferes with epithelialization and fibroplasia and impairs wound healing. Wounds contaminated with significant numbers of bacteria or foreign material may undergo a prolonged or persistent inflammatory response and may not heal. Granuloma formation surrounding retained sutures is an example of chronic inflammation. [8]

As white blood cells remove debris within the wound, epithelial cells at the surface of the wound begin to migrate across the tissue defect. In most sutured wounds, the surface of the wound develops an epithelial covering impermeable to water within 24 to 48 hours. Eschar and surface debris impair this process. The epithelium thickens and grows downward into the wound and along the course of skin sutures. Although there is some "adhesiveness" to the wound edges during the first few days, this is lost because of fibrinolysis.

By the fourth or fifth day, newly transformed fibroblasts in the wound begin synthesizing collagen and protein polysaccharides, initiating the stage of scar formation known as *fibroplasia*. Collagen is the predominant component of scar tissue. Wound strength is a balance between the lysis of old collagen and the synthesis of new collagen "welding" the wound edges together. The amount of scar tissue is influenced by physical forces (e.g., the stresses imposed by movement) acting across the wound. In contrast, a wound that heals by secondary intention closes by contraction. *Contraction* is the movement of skin edges toward the center of the defect, primarily in the direction of underlying muscle.

Significant gains in tensile strength do not begin until approximately the fifth day following the injury. Strength increases rapidly for 6 to 17 days, more slowly for an additional 10 to 14 days, and almost imperceptibly for as long as 2 years (Fig. 35-1). The strength of scar tissue never quite reaches that of unwounded skin. Although the process of collagen formation is essentially completed within 21 to 28 days, the scar widens for another month, and collagen continues to remodel and strengthen the wound for up to 1 year. [2] [8]

Decisions regarding the optimal time for suture removal and the need for continued support of the wound with tape are influenced by (1) wound tensile strength, (2) the period of scar widening, and (3) the cosmetically unacceptable effect of epithelialization along suture tracks. Scars are quite red and noticeable at 3 to 8 weeks following closure. However, the appearance of a scar should not be judged before the scar is well into its remodeling phase. The cosmetic appearance of wounds 6 to 9 months after injury cannot be predicted at the time of suture removal. [9] Therefore, any scar revision should be postponed until 6 to 12 months after injury.

Zitelli states, "The most important factor in predicting the cosmetic result is wound location. In general, wounds on concave surfaces heal with better cosmetic results than wounds on convex surfaces. Besides location, other factors such as *skin color, wound size*, and *wound depth* are helpful in predicting the cosmetic results of wounds healing by secondary intention." [10]

Small, superficial wounds in lax, light-colored skin, especially in areas in which the skin is thin, result in less noticeable scars. Wounds on convex surfaces look better after

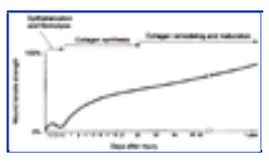

Figure 35-1 Graphic representation of the various phases of wound healing. Note that the tensile strength of scar tissue never reaches that of unwounded skin. Displayed values of tensile strength are approximate and demonstrate the general concept of wound healing.

primary closure than following secondary healing. Static and dynamic forces, and the propensity toward keloid formation, may influence the long-term cosmetic appearance of wounds more than the surgical skills of the clinician who repaired the wound. [9] Repigmentation occurs over 3 to 5 years, even in large wounds that heal by secondary intention. [10]

INITIAL EVALUATION

The approach to the management of a particular wound depends on information gathered during history taking and on the results of physical examination. The decision on whether to close a wound immediately or after a period of observation is based on various factors that affect the risk of infection. Some wounds may appear benign but conceal extensive and devastating underlying tissue damage. The discovery that an extremity wound was produced by a roller or wringer device, a high-pressure injection gun, high-voltage electricity, heavy and prolonged compressive forces, or the bite of a human or a potentially rabid animal radically alters the overall management of the affected patient. The American College of Emergency Physicians' "Clinical Policy for the Initial Approach to Patients Presenting with Penetrating Extremity Trauma" provides a useful approach to the evaluation of all wounds. [11]

History

In the initial evaluation of a wound, the clinician should identify all of the extrinsic and intrinsic factors that jeopardize healing and promote infection. These include the mechanism of injury, the time of injury, the environment in which the wound occurred, and the patient's immune status. Although the clinician would like to believe that all patients are forthright and honest when offering historical data, such is not always the case. A common example of this is the workman who sustains a human bite to the hand during a bar fight on Saturday who steadfastly claims that the injury occurred on the job Monday morning to gain workman's compensation benefits.

Wound Age: The "Golden Period"

In general, the likelihood of wound infection increases with the time that elapses before definitive wound care. [12] [13] [14] A delay in *wound cleaning* is the most important variable, and may allow bacteria contaminating the wound to proliferate. A delay in treatment of a contaminated wound for as little as 3 hours can result in infection. [15] [16] However, there is evidence suggesting that wounds in highly vascular regions such as the face and scalp can be closed without increased risk as long as 24 hours after injury.[17] Some investigators have been unable to establish any significant relationship between time of suturing and subsequent infection rates. [18] Contrary to popular belief, the "golden period"—the maximum time after injury that a wound may be safely closed without significant risk of infection— *is not a fixed number of hours*.[19] Likely this period is longer than commonly believed (Fig. 35-2).

Many factors affect infection risk, and closure decisions should not be based solely on temporal considerations. Peacock points out that "a clean razor slice of highly vascular skin of the face might be closed safely 48 hours after injury, whereas a stable-floor-nail penetration of the foot of an elderly person might not be closed safely 1 minute after injury."[6] Berk and colleagues concluded that there is little change in wound infection rates in most areas of the body for up to 19 hours after various traumatic injuries, and infection rates of simple wounds involving the head are essentially unaffected by the interval between injury and repair. [17] Hence, all data accumulated in the initial evaluation, both historical and physical, must be considered when making the decision to close a wound in a particular patient. In addition, the techniques of wound care in and of themselves may extend the golden period; a skillful clinician can often convert a dangerously contaminated wound into a clean wound that can be safely closed.[6]

Other Historical Factors

Other factors that affect wound healing or the risk of infection include the patient's age and state of health. Patient age appears to be an important factor in host resistance to infection; those individuals at the extremes of age—young children and the elderly—are at greatest risk. [20] [21] Infection rates are reported to be higher in patients with medical illnesses (e.g., diabetes mellitus, immunologic deficiencies, malnutrition, anemia, uremia, congestive heart failure, cirrhosis, malignancy, alcoholism, arteriosclerosis, arteritis, collagen vascular disease, chronic granulomatous disease, smoking or chronic hypoxia, liver failure), in obese patients, and in patients taking steroids or immunosuppressive drugs or those receiving

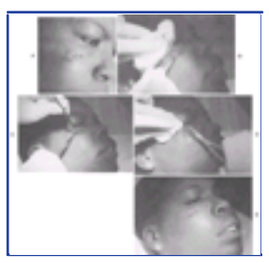

Figure 35-2 This laceration illustrates that there is no specific time frame (the so-called golden period) during which a laceration must be closed, or else relegated to an unsightly scar or a revision months later. *A*, This woman was punched in the face, suffered a laceration of the cheek, and presented to the ED 36 hours later. The wound was not infected, had contracted, and was beginning to heal by granulation. Under local anesthesia the wound was opened, irrigated, minimally debrided, and the skin edges were trimmed. *B*, Using a No. 15 blade, a 1-mm skin edge was incised. *C*, The trimmed edges were then cut by scissors. *D*, The wound was undermined to relieve tension on the skin. *E*, The wound was closed with 6-0 interrupted sutures that were removed in 5 days. No antibiotics were used and only a small linear scar resulted.

radiation therapy. Shock, remote trauma, distant infection, bacteremia, retained foreign bodies, denervation, and peripheral vascular disease also increase wound infection rates and slow the healing process. [7] [21] [22] [23]

Additional information pertinent to decision making in wound management includes:

Present medications (specifically, anticoagulants and immunosuppressive drugs)

Allergies (especially to local anesthetics, antiseptics, analgesics, antibiotics, and tape)

Tetanus immunization status

Potential exposure to rabies (in bite wounds and mucosal exposures)

Potential for foreign bodies embedded in the wound, especially when the mechanism of injury is unknown or was associated with breaking glass or vegetative matter[24]

Previous injuries and deformities (especially in extremity and facial injuries)

Associated injuries (underlying fracture, joint penetration)

Other factors (availability for follow-up, patient understanding of wound care or compliance)

Physical Examination

All wounds should be examined for amount of tissue destruction, degree of contamination, and damage to underlying structures. A common error in wound management is to assume that a traumatic wound is already contaminated and then, during the examination, to contaminate it further. Despite the fact that all traumatic wounds are contaminated to some degree, these injuries should be examined using aseptic technique. It is prudent for the examiner to wear clean or sterile

gloves and avoid droplet contamination from the mouth by maintaining distance or, preferably, by wearing a mask. [25] It has never been proven, however, that the use of sterile gloves (as opposed to clean ones) has any influence on infection rates for common lacerations, and many clinicians do not routinely

use sterile gloves for all wound care. Wounds should be examined under good lighting and after bleeding is controlled. It is appropriate, and often necessary, to obtain a bloodless field with the use of tourniquets. Distal perfusion and motor/sensory function should be assessed and documented during the evaluation of extremity wounds, and before the use of anesthetics.

Mechanism of Injury and Classification of Wounds

The magnitude and direction of the injuring force and the volume of tissue on which the force is dissipated determine the type of wound sustained. Three types of mechanical forces produce soft tissue injury: shear, tension, and compression forces. The resulting disruption or loss of tissue determines the configuration of the wound. Wounds may be classified into six categories:

1. *Abrasions.* Wounds caused by forces applied in opposite directions, resulting in the loss of epidermis and possibly dermis (e.g., skin grinding against road surface).
2. *Lacerations.* Wounds caused by shear forces that produce a tear in tissues. Tensile and compressive forces also cause separation of tissue. Little energy is required to produce a wound by shear forces (e.g., a knife cut). Consequently, little tissue damage occurs at the wound edge, the margins are sharp, and the wound appears "tidy." The energy required to disrupt tissue by tensile or compressive forces (e.g., forehead hitting a dashboard) is considerably greater than that required for tissue disruption by shear forces, because the energy is distributed over a larger volume. These lacerations have jagged, contused, "untidy" edges; consequently, they have a higher risk of infection. [21]
3. *Crush wounds.* Wounds caused by the impact of an object against tissue, particularly over a bony surface, which compresses the tissue. These wounds may contain contused or partially devitalized tissue.
4. *Puncture wounds.* Wounds with a small opening and whose depth cannot be entirely visualized. Puncture wounds are caused by a combination of forces.
5. *Avulsions.* Wounds in which a portion of tissue is completely separated from its base and is either lost or left with a narrow base of attachment (a flap). [26] Shear and tensile forces cause avulsions.
6. *Combination wounds.* Wounds with a combination of configurations. For example, stellate lacerations caused by compression of soft tissue against underlying bone create wounds with elements of crush and tissue separation; missile wounds involve a combination of shear, tensile, and compressive forces that puncture, crush, and sometimes avulse tissue. [25]

Contaminants (bacteria and foreign material).

Numerous factors affect the risk of wound infection, but the primary determinants of infection are the amount of bacteria and dead tissue remaining in the wound. [27] Also of importance is the ability of the patient's immune system to respond to bacterial invasion and the presence of local tissue ischemia or hypoxia. [28]

Essentially all traumatic wounds are contaminated with bacteria to some extent. The number of bacteria remaining in the wound at the time of closure is directly related to the risk of infection. A critical number of bacteria must be present in a wound before a soft tissue infection develops. In experimental wounds produced by shear forces, an inoculum of $=10^6$ aerobic bacteria per gram of tissue inevitably produces wound infection in time. When the mechanism of injury involves a compressive force, the infective dose of bacteria is $=10^4$ bacteria per gram of tissue. If bacterial counts after injury (or after wound management) are below this level, the wound has a very low probability of becoming infected. [12] [15]

Surgical operations are categorized on the basis of the relative levels of bacterial contamination of the wounds. Most traumatic wounds fall into one of two categories:

1. *Contaminated wounds.* Traumatic wounds <12 hours old (the most common type of wound seen in EDs).
2. *Dirty wounds.* Wounds heavily contaminated with pathogenic organisms, those with significant numbers of bacteria associated with large amounts of devitalized tissue, or traumatic wounds older than 12 hours. [29]

Infection rates in series of contaminated wounds of all types range from 1.1% to 21%; rates in series of dirty wounds range from 7% to 38%. [20] [22] [29] [30] [31] [32] [33] [34] The nature and amount of foreign material contaminating the wound often determine the type and quantity of bacteria implanted. In general, visible contamination of a wound increases infection risk. [21] The presence of undetected reactive foreign bodies in sutured wounds almost guarantees an infection. Although bullet or glass fragments by themselves rarely produce wound infection, these foreign bodies may carry particles of clothing, gun wadding, or soil into the wound. Minute amounts of organic or vegetative matter, feces, or saliva carry highly infective doses of bacteria. The bacterial inoculum from human bites often contains 100 million or more organisms per milliliter of saliva. [35]

Inorganic particulate matter, such as sand or road surface grease, usually introduces few bacteria into a wound and has little chemical reactivity; these contaminants are relatively innocuous. However, soil containing a large proportion of clay particles readily promotes infection. Presumably because of their marked chemical reactivity, clay particles damage local tissue defenses. [36] Soils with a high organic content, such as those in swamps, bogs, and marshes, also have a high infection potential. [37]

Most wounds encountered in the practice of emergency medicine have low initial bacterial counts. If wound cleaning and removal of devitalized tissue are instituted before bacteria within the wound enter their accelerated growth phase (3 to 12 hours following the injury) and if one uses aseptic technique in examining and managing these wounds, bacterial counts will remain below the critical number needed to initiate infection. [12] [15]

Wound Location

The anatomic location of the wound has considerable importance in the risk of infection. Bacterial densities on the skin surface range from a few thousand to millions per square centimeter. [37] Areas with endogenous microflora in numbers sufficient to infect a wound ($>10^5$ bacteria/cm^2) include the hairy scalp, the forehead, the axilla, the perineum, the foreskin of the penis, the vagina, the mouth, intertriginous areas, and the nails. In other regions, skin bacteria are sparse (10^2 to 10^3 bacteria/cm^2) and are not a source of infection. [25] Wounds in regions of high vascularity, such as the scalp and the face, more easily resist bacterial incursions. The high vascularity of the scalp probably accounts for extremely low infection rates with scalp injuries, despite the large numbers of endogenous

microflora. Distal extremity wounds, in contrast, are more at risk for the development of wound infections than are injuries of most other parts of the body. [20] Wounds in ischemic tissue are notoriously susceptible to infection. [38]

Devitalized Tissue

Identifying devitalized tissue is an important part of the examination of a wound. Tissue damage lowers the resistance of the wound to infection. Devitalized or necrotic tissue enhances the possibility of infection in a wound by providing a culture medium in which bacteria proliferate, by inhibiting leukocyte phagocytosis, and by creating an anaerobic environment suitable for certain bacterial species. [25] [27]

Underlying Structures

Identification of injury to underlying structures such as nerves, vessels, tendons, joints, bones, or ducts may lead the emergency clinician to forgo wound closure and consult a surgical specialist. Procedures such as joint space irrigation, reduction and debridement of compound fractures, neurorrhaphy, vascular anastomosis, and

flexor tendon repair are best accomplished in the controlled setting of the operating room, in which optimal lighting, proper instruments, and assistance are available.[25]

CLEANING

The wound should be cleaned as soon as possible after evaluation. Although most wounds are contaminated initially with less than an infective dose of bacteria, given time and the appropriate wound environment, bacterial counts may reach infective levels. The goals of wound cleaning and debridement are the same: (1) to remove bacteria and reduce their numbers below the level associated with infection, and (2) to remove particulate matter and tissue debris that would lengthen the inflammatory stage of healing or allow the growth of bacteria beyond the critical threshold. [23]

Patient Preparation

Before examining, cleaning, exploring, or repairing wounds, medical procedures should be explained to patients to allay fears and encourage patient cooperation. In general, all wound care should be performed *with the patient in a supine position,* since fainting is a common occurrence once wound preparation has commenced (Fig. 35-3). Even the most hardy or brave patient may faint at the sight of a needle, scalpel, or blood. Patient falls are a serious source of comorbidity and litigation. Likewise, relatives and friends should be allowed to stay with the patient only after their propensity for fainting has been assessed and they have been properly cautioned. The wise clinician will insist that any significant others who remain in the room to support the patient sit during the procedure and report any perceived dizziness or nausea. In general it is not suggested that parents or friends actively participate in the wound care procedures.

Wound Handling

Anyone cleaning, irrigating, or suturing wounds should wear protective eyewear and a mask, as virtually any patient may be seropositive for the human immunodeficiency virus (HIV). Although mucosal exposure to blood or tissue products that

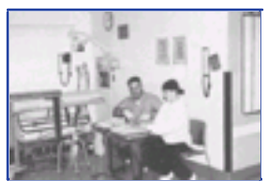

Figure 35-3 What's wrong with this picture? The patient is sitting up during laceration repair. Shortly after this picture was taken the patient fainted and nearly fell off the chair. Even the bravest patients should be *supine* for surgical procedures.

are contaminated by HIV is considered a relatively low risk for subsequent infection, universal precautions are currently recommended.

Thorough cleansing of bacteria, soil, and other contaminants from a wound cannot be accomplished without the patient's cooperation. Scrubbing most open wounds is painful, and the patient's natural response is withdrawal. Therefore, *local or regional anesthesia often must precede the examination and cleaning of a wound*. Approaches to wound anesthesia are discussed in detail in Chapter 30 Chapter 31 Chapter 32 Chapter 33 .

Despite adequate anesthesia, the patient may be unable to cooperate because of apprehension. The clinician should explain the wound cleansing procedure and assure the patient that everything possible will be done to minimize pain. Reassurance may not alleviate the fears of young children, and both sedation and physical restraining devices must be used. Approaches to sedation using parenteral sedative-hypnotics and narcotic agents and the use of inhaled nitrous oxide are discussed in Chapter 34 .

The two primary methods of wound cleaning are mechanical scrubbing and irrigation. Soaking a wound in a saline or antiseptic solution before the clinician arrives is of little value and is not recommended as a routine practice. Indeed, soaking a wound in saline may actually increase bacterial counts. [39] The following section discusses methods of scrubbing and irrigation.

Mechanical Scrubbing

Initially, a wide area of skin surface *surrounding the wound* should be scrubbed with an antiseptic solution to remove contaminants that in the course of wound management might be carried into the wound by instruments, suture material, dressings, or the clinician's gloved hand. Minimal aseptic technique requires the use of gloves during the cleaning procedure. It is important to remove all nonabsorbable particulate matter; any such material left in the dermis may become impregnated in the healed tissue and result in a disfiguring "tattoo" effect. [8] However, scrubbing the internal surface of a wound is controversial. Although scrubbing a wound with an antiseptic-soaked sponge does remove foreign particulates, bacteria, and tissue debris, an abrasive sponge may inflict more damage on tissue and provoke more inflammation.[40] [41]

Some clinicians reserve mechanical scrubbing for "dirty" wounds contaminated with significant amounts of foreign material. If irrigation alone is ineffective in removing contaminants from a wound, the wound should be scrubbed. Because the amount of damage inflicted on tissues by scrubbing correlates to the porosity of the sponge, a fine-pore sponge (e.g., Optipore sponge [90 pores per linear inch]) should be used to minimize tissue abrasion. [40] [42] Detergents have an advantage over saline because they minimize friction between the sponge and tissue, thereby limiting tissue damage during scrubbing. Detergents also dissolve particles, helping to dislodge them from the wound surface. Unfortunately, many of the available detergents are toxic to tissues. [40] [43]

Antiseptics During Cleaning

For many years, antiseptic solutions have been used for their antimicrobial properties in and around wounds (Table 35-1). Studies of antiseptics in wounds demonstrate that there is a delicate balance between killing bacteria and injuring tissue. [44] Intact skin can withstand strong microbicidal agents, whereas leukocytes and the exposed cells of skin and soft tissue can be damaged by these agents. [23]

Many antiseptic solutions have been used for cleaning wounds. Povidone-iodine (Betadine) is widely available as a 10% stock solution. The undiluted solution is best kept out of the wound proper. Although studies on the efficacy and safety of povidone-iodine solution have shown variable results, [29] [39] [45] [46] [47] [48] it appears that *dilute* povidone-iodine solution in concentrations of <1% is both safe and effective for use in contaminated traumatic wounds. The precise concentration that provides the most benefit is unknown. In contrast, povidone-iodine surgical scrub (Betadine scrub) and hexachlorophene (pHisoHex) both contain anionic detergents that are harmful to tissues and increase infection rates when used in wounds. [25] [41] [42] Aqueous iodine is irritating and corrosive to tissue and should not be used in any wound.[45]

In vitro studies have demonstrated the toxicity of chlorhexidine gluconate-alcohol (Hibiclens) to the cellular components of blood. [48] Some clinicians use hydrogen peroxide for its effervescent effect in cleaning wounds. Peroxide has been used for many years without a proven effect on most wounds and its actual role as a detriment to wound healing is probably minimal. However, because peroxide is hemolytic, it is best to use it only to clean surrounding skin encrusted with blood and coagulum or to soak off adherent blood-soaked dressings. Peroxide should not be used on granulation tissue, because oxygen bubbles lift newly formed epithelium off the wound surface.[49] Hexachlorophene was used as an antiseptic for a time but was found to be neurotoxic and teratogenic through skin absorption. Its narrow antimicrobial spectrum makes it no more effective than ordinary soap and water. [50] Quaternary ammonium compounds are less toxic to tissue but have a limited antimicrobial spectrum; gram-positive organisms are more susceptible to these solutions than are gram-negative bacteria. Benzalkonium chloride (Zephiran) is inactivated by soaps, detergents, blood, and other organic matter. Furthermore, *Pseudomonas* can proliferate in stored solutions. Consequently, use of benzalkonium

chloride has fallen into disfavor. [42]

Nonantiseptic nonionic surfactants are attractive alternatives to these toxic cleansing agents. In contrast to antiseptic solutions, these preparations cause no tissue or cellular damage, leukocyte inhibition, or impairment in wound healing. The solutions also cause no corneal injury, conjunctival irritation, or pain on contact with the wound. [48] [51] Poloxamer 188 (Pluronic F-68, Shur-Clens, and Pharma Clens) is nontoxic, even when administered intravenously, and nonallergenic. [43] This pluronic polyol has no antibacterial activity, but scrubbing experimental wounds with poloxamer has reduced infection rates, proving its ability to cleanse a wound effectively and atraumatically. [40]

A survey of wound practice by Howell and Chisholm suggests that a wide range of solutions and techniques are in clinical use in the United States. [1] However, it is important to distinguish between skin antiseptics and irrigating solutions. As a general rule, commercially available antiseptics should be used *only to clean intact skin,* and one should avoid introduction of significant amounts of these products into open wounds. It is recommended that most open wounds be irrigated with copious amounts of saline or a dilute (1%) povidone-iodine solution. Pluronic polyols may be considered when the wound is near mucous membranes and dilute povidone-iodine may be particularly irritating.

Irrigation

Properly performed irrigation is effective in removing particulate matter, bacteria, and devitalized tissue that is loosely adherent to the edges of the wound and trapped within its depths. Rodeheaver and colleagues studied the effect of irrigating experimental wounds contaminated with 20 mg of soil. Irrigating wounds with 400 mL of fluid at 1 psi removed 48.6% of the soil, whereas increasing the pressure to 15 psi removed 84.8%, reducing the infection rate from 100% to 7%. [52] *The effectiveness of irrigation is determined primarily by the hydraulic pressure at which the irrigation fluid is delivered.* [53] [54] There is preliminary evidence suggesting that low-pressure, high volume irrigation of wounds may be as effective in reducing bacterial counts in wounds as syringe irrigation. [55] [56] Port devices spiked into plastic intravenous bags that were squeezed by hand deliver a stream of fluid at 2 psi. The flow rate of irrigation fluid delivered through IV tubing with a blood pressure cuff inflated around a collapsible plastic IV bag provides <10 psi of irrigation pressure. Other studies have shown that bulb syringes or gravity flow irrigation devices deliver fluid at low pressures and are ineffective in ridding wounds of small particulate matter or in lowering wound bacterial counts. [53]

The pressure that can be delivered with a syringe varies with the force exerted on the plunger of the syringe and with the internal diameter of the attached needle. A simple irrigation assembly consisting of a 19-ga plastic catheter or needle attached to a 35-mL syringe produces 25 to 40 psi when the barrel of the syringe is pushed with both hands. [54] This high-pressure irrigation system removes significant numbers of bacteria and a substantial amount of particulate matter from the wound surface (Fig. 35-4). Commercial irrigation systems with a ring-handled syringe and a one-way valve that connects into a standard IV solution are available (Fig. 35-5) (Canyons Wound Irrigation System, Canyons International, Inc., Salt Lake City, UT; Travenol pressure irrigation set, code number 2D2113; Irrijet, Ackrad Laboratories, Garwood, NJ). These devices make irrigation of wounds easier and more efficient.

TABLE 35-1 -- Summary of Agents Used for Wound Care

Agent	Biologic Activity	Tissue Toxicity	Systemic Toxicity*	Potential Uses	Comments
Povidone-iodine surgical scrub (Betadine 7.5%)	Virucidal; strongly bactericidal against gram-positive and gram-negative organisms	Detergent can be toxic to wound tissues	Painful to open wounds; other reactions extremely rare	Hand cleanser	Iodine allergy possible; systemic absorption of iodine from burns, open wounds; not routinely used in open wounds
Povidone-iodine solution (Betadine 10%)	Same as povidone-iodine scrub; virucidal, bactericidal	Minimally toxic to wound tissues at full strength; 1% solution has no significant tissue toxicity	Extremely rare	Wound periphery cleanser; diluted to 1% for wound irrigation	Probably the safest and most effective product currently available; iodine is active agent, povidone is carrier molecule; iodine allergy possible; systemic absorption of iodine from burns, open wounds; dilute 10:1 (saline:Betadine) if used to irrigate wounds
Chlorhexidine gluconate (Hibiclens)	Strongly bactericidal against gram-positive organisms, less strong against gram-negative bacteria	Ionic detergent can be toxic to tissue/cellular components; eye and inner ear toxicity	Extremely rare	Hand cleanser	Generally avoid use in open wounds; not for use in eye/ear
Polaxamer 188 (Shur-Clens; Pluronic F-68)	No antibacterial or antiviral activity	None known; does not inhibit wound healing	None known	Wound cleanser (particularly useful on face)	Nonionic detergent used for cleansing properties; nontoxic even with IV use; will not damage eye/cornea; lack of antibacterial properties limits use
Hexachlorophene (pHisoHex)	Bacteriostatic against gram-positive bacteria, poor activity against gram-negative bacteria	Detergent can be toxic to wound tissues	Possibly teratogenic with repeated use	Alternative hand cleanser; not used on open wounds	Systemic absorption causes neurotoxicity
Hydrogen peroxide	Very weak antibacterial agent	Toxic to tissue/red cells	Extremely rare	Wound cleanser adjunct; very weak antiseptic properties	Breaks down to water and oxygen; foaming activity useful to remove debris/coagulated blood

*Based largely on in vitro studies/animal data.

Figure 35-4 An easy method of high-pressure irrigation. IV tubing with an in-line, one-way valve is attached to a bottle of sterile 0.9% saline solution. The other end is connected to a stopcock. Saline solution is aspirated into the syringe. Maximal force is exerted on the plunger of the syringe, delivering the solution in a fine stream through an attached 19-ga needle held close to the wound. A splash shield may be used instead of a needle (see Fig. 35-6).

Pulsatile jet irrigation of wounds creates pressures of 50 to 70 psi but does not appear to drive significant amounts of bacteria or surface contaminants into the soft tissues of the wound. [57] [58] Although jet irrigation can damage tissue defenses and spread fluid laterally into loose areolar tissue, [59] it is more effective in cleaning wounds, less traumatic to tissues, and less likely to produce edema than conventional scrubbing with a brush. However, this technique should generally be reserved

for use in heavily contaminated wounds in which syringe irrigation proves to be ineffective. The jet irrigation system used by many investigators was a WaterPik unit (Teledyne Aquatic Corp., http://professional.waterpik.com/int.cfm) with a sterile tip nozzle held approximately 4 cm from the wound. [42] [58] [59]

In an uncontrolled study, Hollander et al. found comparable infection rates and cosmetic outcomes in facial and scalp wounds repaired with and without irrigation. [60] Although irrigation may not be required for low risk, uncontaminated facial and scalp wounds, randomized, prospective trials are needed to answer this question.

Minimum recommended volumes of irrigation fluid vary, but for average-sized wounds, 100 to 300 mL should be used. [42] Greater volumes may be required for larger or heavily contaminated wounds. Irrigation should continue until all visible, loose particulate matter has been removed. Approximately 20% of open, undated bottles of "sterile" saline and water are contaminated; only solutions in bottles

Figure 35-5 A spring-loaded irrigation device with ring handles speeds the somewhat tedious process of wound irrigation. *(Courtesy of Canyons International, Inc., Salt Lake City, UT.)*

opened within the past 24 hours should be used to irrigate wounds. [61] [62] A potential complication of wound irrigation is that infectious material can be splashed into the face of the clinician, even when the tip of the irrigation device is held below the wound surface. A plastic cup device that fits on the end of a syringe (ZeroWet Splashield, Zerowet Inc., Palos Verdes Peninsula, CA) can be used to contain the splatter (Fig. 35-6). The wound should be positioned to allow continuous drainage of fluid during irrigation by any method. [63]

Antibiotic Solutions for Irrigation

Antibiotic solutions have been instilled directly into wounds or used as irrigation solutions. Halasz reviewed the studies of several investigators who analyzed the technique of irrigating wounds with antibiotic solutions, including ampicillin, a neomycin-bacitracin-polymyxin combination; tetracycline; penicillin; kanamycin; and cephalothin. He concluded that "organisms in the wound can be exposed to adequate concentrations of antibiotics, and that the concentration of these drugs in the wound remains in the bactericidal range for long periods of time, far exceeding that obtainable by systemic administration." [64] The use of antibiotics in irrigation solutions in lieu of antiseptic solutions avoids the tissue destruction of the antiseptics but theoretically risks topical sensitization of the patient to the antibiotic and the development of toxic tissue levels of the antibiotic. To date, there have been no reports of these complications. Within 3 hours of injury, a proteinaceous coagulum forms within the wound, surrounding the bacteria and probably preventing their contact with topical or systemic antibiotics. Therefore, the wound should be scrubbed before irrigation with an antibiotic solution. Although a decrease in wound infection has been demonstrated in some studies, [64] [66] [67] others have found no substantial benefit to irrigation with antibiotics. [68] The indications for antibiotic solutions in cleaning wounds have not been defined, and this practice is not considered standard.

Recommendations for Cleaning the Wound

The prerequisites of any wound-cleaning technique are a calm or sedated patient, satisfactory anesthesia, and a thorough

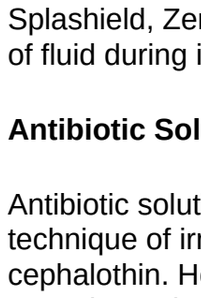

Figure 35-6 *A*, ZeroWet Splashield attached to end of a syringe. This device is used in lieu of a needle. The shield is held near the skin, and the tip of the syringe directs the irrigating solution. *B*, This protective shield allows forceful irrigation without splatter of infectious fluids. *Note that the clinician is holding open the laceration with forceps to allow irrigation of the deep structures. The margins of this small laceration were also extended to allow for better irrigation.*

scrub of the skin surface adjacent to the wound. The importance of ridding the wound of major contaminants and infective doses of bacteria is unquestionable. Two strategies to accomplish the goals of wound cleaning are apparent from this discussion. The contaminated or "dirty" wound can be irrigated or both scrubbed and irrigated with a 1% povidoneiodine solution (Betadine preparation, *not* Betadine scrub). This should be followed by flushing with a 0.9% saline solution. As an alternative, the wound can be scrubbed with pluronic polyols and irrigated with an antibiotic-normal saline solution. *Only pluronic polyols or saline should be used near the eyes.* All scrubbing should be performed with a soft, fine-pore sponge, and high-pressure techniques should be used for all irrigation. The use of hydrogen peroxide on open wounds is discouraged. Gentle scrubbing with poloxamer and normal saline high-pressure irrigation both appear to be satisfactory methods for cleaning minimally contaminated wounds.

Patients frequently irrigate their wounds with tap water before presentation. Some clinicians routinely irrigate wounds, especially extremity wounds, with tap water instead of sterile saline. Some investigators have demonstrated that tap water produced an infection rate comparable to or lower than saline (5.4% vs 10.3%). [69] [70] Even though bacteria were cultured from the faucets in one study, these organisms did not cause any wound infections. The advantage of using tap water is the large volume of irrigant that can be quickly applied to an open wound. Since no studies suggest that tap water is in any way detrimental to wound healing, the editors support the use of tap water for irrigation of wounds in the ED. Interestingly, once irrigating solutions of saline have been opened, bacterial contamination occurs quite rapidly, further supporting the use of tap water. Kaczmarek et al. demonstrated up to a 21% bacterial contamination rate in partially used opened saline bottles collected throughout the hospital. [62]

Preparation for Wound Closure

Before debridement or wound closure, the wound must be prepared and draped. Body hair should generally be left intact, since wounds that are shaved demonstrate higher infection rates. [22] [71] [72] [73] For wounds in hair-bearing areas, hair should be removed by clipping only if the hair interferes with the procedure. [72] However, it is common practice to shave the scalp in preparation for suturing, and this has not been demonstrated to be detrimental to wound healing. Stubborn hairs that repeatedly invade the wound during suturing can be coated with petrolatum jelly or water-soluble ointments to keep them out of the field. Eyebrows should not be shaved, because critical landmarks needed for exact approximation would be lost. Although shaved eyebrows will grow back eventually, shaving produces an undesirable cosmetic effect.

The *skin surface adjacent to the wound* (not the wound itself) can be disinfected with a standard 10% povidoneiodine or chlorhexidine gluconate (Hibiclens) solution. The solution is painted widely on the skin surrounding the wound but should not cover the interior of the wound itself. After hand washing, the clinician and any assistants involved in the procedure must wear gloves. Visible talc or other powders on the gloves should be rinsed before touching the patient's wound. [37] As stated earlier, the use of nonsterile gloves has not been demonstrated to increase infection rates.

Face masks are recommended and are mandatory for any clinician with a bacterial upper respiratory infection. Because droplets of saliva may leak even from around the edges of a face mask, talking in proximity to the wound must be avoided. [74]

A single fenestrated drape or multiple folded drapes are placed over the wound site. For hand wounds, a sterile glove may be applied to the patient to provide a sterile field in lieu of a fenestrated drape (Fig. 35-7). The area to be sutured can be exposed by cutting the glove, and the extremity can be placed on a sterile towel. This technique provides a clean field without the need to continually adjust the drape or to operate through a small opening. If the wound has not yet been anesthetized, anesthesia can be provided at this time (see Chapter 30 Chapter 31 Chapter 32 Chapter 33 for details).

The entire depth and the full extent of every wound should be explored in an attempt to locate hidden foreign bodies, particulate matter, bone fragments, and any injuries to underlying structures that may require repair. *The clinician should avoid the temptation to initially explore wounds with a finger in search of a foreign body or to assess wound characteristics.* Embedded glass, metal fragments, or sharp pieces of bone may cut the clinician, exposing him or her to

Figure 35-7 A sterile glove on the hand with the finger cut out, instead of an annoying drape, and a finger tourniquet to provide a bloodless field make examination and suturing of a wound easier.

contamination with the patient's blood (Fig. 35-8). Exploration with a metal probe and the liberal use of radiographs, combined with direct vision and good lighting, is a much safer approach. Lacerations through thick subcutaneous (SQ) adipose tissue are treacherous, because large amounts of particulate matter can be totally obscured in deeper folds of tissue. Unless a careful search is undertaken, these contaminants may be left in the depths of a sutured wound, and infection usually follows. Some clinicians are reluctant to extend lacerations to properly clean or explore them; however, opening the wound to permit adequate visualization may be needed for successful wound preparation.

Debridement

Debridement of foreign material and devitalized tissue is of undisputed importance in the management of the contaminated wound. With this technique, the clinician can remove

Figure 35-8 *Do not* probe a wound with a finger looking for foreign bodies! If the clinician's skin is broken, the patient's blood may introduce HIV, hepatitis, viruses, and other infectious agents. Betters ways to find a foreign body are obtaining radiographs and exploring wounds with metal probes. Extending the wound edges and performing the examination under a bloodless field and with good lighting will find most foreign bodies.

tissue impregnated with foreign matter, bacteria, and devitalized tissue that otherwise impairs the ability of the wound to resist infection and prolongs the period of inflammation. Debridement also creates a tidy, sharp wound edge that is easier to repair and results in a more cosmetically acceptable scar.

If the wound already is clean and the edges are viable, sharp debridement may not improve the outcome. Irregular wounds have greater surface areas than do linear lacerations. Because skin tension is distributed over a greater length, the scar width is usually less in jagged wounds than if the wound is converted to an elliptical defect with tidy edges. If the edges are devitalized or contaminated, the wound edges must be debrided. To avoid a wide scar in this situation, the wound can be undermined.

Excision

If significant contamination occurs in areas in which there is a laxity of tissues and if no important structures, such as tendons or nerves, lie within the wound, the entire wound may be excised.[6] This technique is the most effective type of debridement, because it converts the contaminated traumatic wound into a clean surgical wound (Fig. 35-9). Complete excision of grossly contaminated wounds such as animal bites allows primary closure of such wounds with no greater risk of infection than in relatively uncontaminated lacerations. [42] Wounds of the trunk, the gluteal region, or the thigh are amenable to this

Figure 35-9 *A–C,* Complete excision of a wound. Grossly contaminated wounds may be excised and sutured primarily.

technique. If necessary, the clinician can judge the adequacy of the excision by coloring the wound surface with a vital dye. The clinician then creates a new wound by excising all dyed tissue.[27] Most traumatic wounds can be excised with an elliptical excision. A lenticular configuration should be marked superficially around the wound with the blade of a No. 15 scalpel by cutting only the epidermis.

If a puncture wound is being excised, the axis of the excision should parallel a wrinkle, a skin line, or a line of dependency or facial expression, and the long axis should be three to four times as great as the short axis. The clinician may plan this type of excision by premarking the skin with a surgical marking pen. Tension should be placed on the surrounding skin with a finger or a skin hook. With the clinician's hand steadied on the table or on the patient, the No. 15 blade is used to cut through the skin at right angles or at slightly oblique angles to the skin surface. If complete excision of the entire depth of the wound is not necessary, the tissue scissors may be used to cut the edge of the wound, following the path premarked in the epidermis by the scalpel blade. If a complete excision is desired, the incision on each wound edge should be carried past the deepest part of the wound (see Fig. 35-9).

Excision should be planned carefully; excessive removal of tissue can create a defect that is too large to close. In hair-bearing areas of the face, particularly through the eyebrows, the incision should be angled parallel to the angle of hair follicles to avoid linear alopecia (Fig. 35-10). The wedge of excised tissue should be removed carefully, without contaminating the fresh wound surface.

Puncture wounds of the feet may contain foreign bodies that are difficult to remove. Contaminated punctures or those likely to contain foreign bodies (e.g., nail punctures through shoes) can be excised by removing a small cone of surrounding tissue with a scalpel or with a 4-mm disposable punch biopsy and the wound irrigated a second time. [77] [78]

Selective Debridement

Complete excision is impossible for most wounds because of insufficient skin elasticity, and selective debridement must be

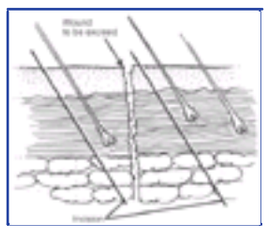

Figure 35-10 Excision through an eyebrow. Use an angled incision to remove tissue in the eyebrow, thus avoiding further injury of hair follicles. *(From Dushoff IM: A stitch in time. Emerg Med 5:2, 1973. Reproduced by permission.)*

used.[6] [27] Simple excision of a wound of the palm or the dorsum of the nose will make approximation of the resulting surgical wound edges difficult. Stellate wounds and wounds with an irregular, meandering course have greater surface areas and less skin tension per unit length than do linear lacerations. In some cases excision of an entire wound would result in the loss of too much tissue (i.e., produce a gaping defect and excessive tension on the wound edges when closed). This problem can be avoided with selective debridement and approximation of the irregular wound edges. This technique involves sharp debridement of devitalized or heavily contaminated tissue in the wound piece by piece and eventual matching of one edge of the wound with the other. Selective debridement is time consuming but preserves more surrounding tissue.

Identifying devitalized tissue in a wound remains a challenging problem. Tissue with a narrow pedicle or base, especially distally based, narrow flaps on extremities, is unlikely to survive and should be excised. Sometimes a sharp line of demarcation distinguishes devitalized skin and viable skin, but in most wounds there is usually only a subtle bluish discoloration. The comparison of capillary refill in wounded tissue with that in adjacent skin is probably the most practical test for tissue viability available to the emergency clinician. [79] If circulation is adequate, viable tissue becomes hyperemic following the release of a proximal tourniquet.

In heavily contaminated wounds, especially those with abundant adipose tissue, all exposed fat and all fat impregnated with particulate matter should be removed. The SQ adipose tissue attached to large flaps or to avulsed viable skin should be debrided before reapproximation of the wound; removal of this fatty layer allows better perfusion of the flap or the graft.

Contaminated bone fragments, nerves, and tendons are almost never removed. Every effort should be made to clean these structures and return them to their place of origin, because they may be functional later. [80]

Instruments usually required for debridement include two fine single- or double-pronged skin hooks, a scalpel with a No. 15 blade, tissue scissors, hemostats, and a small tissue forceps. The jagged wound edges are stabilized with skin hooks or forceps, and the scalpel or scissors are used to cut away devitalized tissue from one end of the wound to the other. Fascia and tendons perform important functions despite potential loss of viability. If they can be cleaned adequately, these tissues should not be debrided. They may be left in wounds as free grafts and covered by viable flaps of tissue. [81]

Following debridement or excision, the wound should be irrigated again to remove any remaining tissue debris.

Control of Hemorrhage

Wound exploration or cleaning sometimes induces bleeding. Hemostasis is essential at any stage of wound care. Not only does persistent bleeding obscure the wound and hamper wound exploration and closure, but also hematoma formation in a sutured wound separates wound edges, impairs healing, and risks dehiscence or infection. If bleeding is not a problem before wound debridement, it frequently becomes a complication once the wound edges are excised.

Several practical methods of achieving hemostasis are available to the emergency clinician. Sustained direct pressure with gloved fingers, gauze sponges, or packing material, combined with elevation, is usually effective in immediately

controlling a single bleeding site or a small number of sites until cut ends of vessels constrict and coagulation occurs. In a patient with multiple injuries and several urgent problems, hemorrhage can be controlled temporarily with a compression dressing. Several absorptive sponges are applied directly over the bleeding site, and these are secured in place with an elastic bandage (e.g., Ace wrap) or elastic adhesive tape (Elastoplast). Pressure is provided by the elasticity of the bandage. The bleeding part should be elevated. Wound care can then be deferred while the clinician attends to more pressing matters.

Although simply crushing and twisting the end of a small vessel with a hemostat avoids the introduction of suture material into the wound, this method provides unreliable hemostasis. Ligation of the vessel with fine absorbable suture material is preferred. Bleeding ends of vessels are clamped with fine-point hemostats, providing immediate hemostasis. Because nerves often course with these vessels, all clamping should be done under direct visualization. The tip of the hemostat should project beyond the vessel to hold a loop of a ligature in place (Fig. 35-11). While an assistant lifts the handle of the hemostat, a synthetic absorbable 5-0 or 6-0 suture is passed around the hemostat from one hand to the other (Fig. 35-12). The first knot is tied beyond the tip of the hemostat. Once the suture is securely anchored on the vessel, the hemostat is released. [76] [82]

In practice, the emergency clinician seldom has an assistant available to ligate vessels by this method. MacDonald describes a technique that enables a single operator to maintain tension on the ligature while removing the hemostatic clamp. [83] A needle holder is used to grasp one tail of the ligature; the other end is held by the third, fourth, and fifth fingers of the left hand. As the clamp held in the right hand is removed from the vessel, the needle holder is moved away from the left hand by extending the thumb and the index finger, maintaining tension on the ligature. The right hand can discard the clamp, grasp the needle holder, and complete the tie (Fig. 35-13).[83] Three knots are sufficient to hold the ligature in place. The ends of the suture should be cut close to the knot to minimize the amount of suture material that is left in the wound.

Clinicians can make the mistake of spending excessive time attempting to tie off small bleeding vessels while the

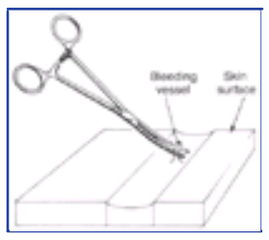

Figure 35-11 When one attempts to tie off a bleeding vessel, the tip of the hemostat should project beyond the clamped vessel.

Figure 35-12 *A,* The handles of the hemostat are raised by an assistant as a ligature is passed under them. *B,* The ligature thread stretched between the index fingertips is carried under the projecting tips of the hemostat. *(Modified from Kirk RM: Basic Surgical Techniques. Edinburgh, Churchill Livingstone, 1978, p 50. Reproduced by permission.)*

patient slowly exsanguinates. In highly vascular areas, such as the scalp, it is sometimes best to suture the laceration following wound exploration and irrigation, despite active bleeding; the pressure exerted by the closure will usually stop the bleeding. If bleeding is too brisk to permit adequate wound evaluation and irrigation, hemorrhage can often be controlled by clamping and everting the galea or dermis of each wound edge using hemostats. Lemos and Clark propose the use of scalp clips as an alternative. [82]

Cut vessels that retract into the wall of the wound may frustrate attempts at clamping and ligation. Bleeding should be controlled first by downward compression on the tissue. A suture is passed through the tissue twice, using a figure-of-eight or horizontal mattress stitch, and then tied. The double thread will constrict the tissue containing the cut vessel (Fig. 35-14). The disadvantage of this method is that the tissue constricted by the ligature may necrose and leave devitalized tissue in the wound.

Vessels with diameters greater than 2 mm should be ligated. Those smaller than 2 mm that bleed despite direct

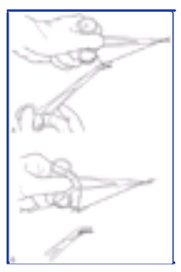

Figure 35-13 *A,* Ligature tension is maintained with one hand by grasping one tail of the suture with a clamp and keeping its base between the thumb and the forefinger. *B,* As the pedicle clamp is removed with the other hand, the ligature is tightened by extending the flexed thumb and index finger to the desired tension. Then the pedicle clamp is discarded from the right hand, and further knots are applied in the usual fashion. *(Modified from MacDonald RT: Maintenance of ligature tension by a single operator with simultaneous removal of a hemostatic clamp. Am J Surg 143:770, 1982. Reproduced by permission.)*

pressure can be controlled by pinpoint electrocautery. A dry field is required for an effective electrical current to pass through the tissues; if sponging does not dry the field, a suction-tipped catheter should be used. Trauma is minimized by using fine-tipped electrodes to touch the vessel or by touching the active electrode of the electrocautery unit to a small hemostat or fine-tipped forceps gripping the vessel. [2] The power of the unit should be kept to the minimum level required for vessel thrombosis. Bipolar coagulation (such as that provided by the Bovie unit) is preferred over monopolar coagulation because it produces approximately one third less necrosis of surrounding tissue. [42] If the amount of tissue cauterized is kept to a minimum, wound healing is no more compromised by this technique than by ligation. Cauterization of medium- and small-sized vessels can quickly provide hemostasis. Self-contained, sterilizable, battery-powered coagulation units also are available. Vessels are cauterized by

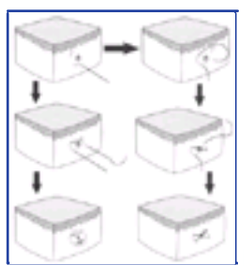

Figure 35-14 Ligation of a retracted, bleeding vessel. *A,* Horizontal mattress technique. *B,* Figure-of-eight technique.

the direct application of a heated wired filament. Although these units may damage more surrounding tissue than electrocautery units, they are compact and simple and thus well suited for use in the emergency center (Fig. 35-15).

Epinephrine is an excellent vasoconstrictor. Topical epinephrine (1:100,000) on a moistened sponge can be applied to a wound to reduce the bleeding from small vessels. Combined with local anesthetics, concentrations of 1:100,000 and 1:200,000 prolong the effect of the anesthetic and provide some hemostasis in highly vascular areas. Because vasoconstrictors theoretically may increase the risk of wound infection, they should be restricted to situations in which widespread small vessel and capillary hemorrhage in a wound is not controlled by direct pressure or cauterization. Hemostasis of a

Figure 35-15 Battery-powered cautery.

specific vessel may be obtained by directly injecting the soft tissues around the base of the bleeder with a small amount of lidocaine/epinephrine solution, even though the wound has been previously anesthetized. The combination of pressure and vasoconstriction may halt bleeding long enough for the vessel to be ligated or cauterized, or allow the wound to be closed and a compression dressing applied.

Fibrin foam, gelatin foam, and microcrystalline collagen may be used as hemostatic agents. Their utility is limited in that vigorous bleeding will wash the agent away from the bleeding site. Their greatest value may be in packing small cavities from which there is a constant oozing of blood. [76] In the vast majority of simple wounds with persistent but minor capillary bleeding, apposition of the wound edges with sutures, followed by a compression dressing, provides adequate hemostasis.

Tourniquets

If bleeding from an extremity wound is refractory to direct pressure, electrocauterization, or ligation, or if the patient presents with exsanguinating hemorrhage from the wound, a tourniquet can be used to control the bleeding temporarily. Tourniquets also are helpful in examining extremity lacerations by providing a bloodless field. However, they can cause injury in three ways:

1. They can produce ischemia in an extremity.
2. They can compress and damage underlying blood vessels and nerves.
3. They can jeopardize the survival of marginally viable tissue.

Although problems rarely develop from tourniquets used in routine wound care, potential problems can be minimized if (1) there is a limit placed on the total amount of time that an extremity is confined by a tourniquet, and (2) excessive tourniquet pressures are avoided. It is also imperative that all tourniquets be removed before releasing the patient. A tourniquet may be overlooked if it is covered by a bulky dressing.

A single-cuff tourniquet (sphygmomanometer cuff) placed around an arm or a leg effectively stops distal venous or arterial bleeding without crushing underlying structures. The length of time that a tourniquet may remain in place is limited by the development of pain underneath and distal to the tourniquet. This occurs within 30 to 45 minutes in a conscious patient, well within the limits of safety. [18]

Before application of the tourniquet, the injured extremity should be elevated and then manually exsanguinated to prevent bothersome venous bleeding. An elastic bandage (e.g., Ace wrap or Esmarch) may be wrapped circumferentially around the extremity, starting distally and moving in a proximal direction. A cuff that is 20% wider than the diameter of the limb is placed around the arm proximal to the wound and inflated to 250 to 300 mm Hg, and the tubing is clamped; the bandage is then removed and the extremity lowered. [84] Some experts recommend a cuff pressure 70 mm Hg higher than the patient's systolic blood pressure. [85] Because tourniquets

impair circulation and may produce neurapraxia, their use in the ED should be limited to a maximum of 1 hour.

Tourniquets on digits have a greater potential for complications. The maximum tourniquet time that is safe for a finger may easily be exceeded inadvertently. Also, finger tourniquets can exert excessive pressures over a small surface area at the base of the finger and injure digital nerves or cause pressure necrosis of digital vessels. For this reason, simple rubber bands should *not* be used as tourniquets. Tourniquet pressures up to 250 to 300 mm Hg are safe in digits, but pressures of only 150 mm Hg are needed for hemostasis.

A 0.5-inch Penrose drain placed around the base of a finger and stretched to no more than two thirds of its circumference provides safe and effective hemostasis. Some clinicians wrap the entire finger with a Penrose drain in the fashion of a miniature Esmarch bandage to exsanguinate the digit. The wrap is unraveled from distal to proximal, leaving two or more turns around the proximal part of the finger to serve as a tourniquet. This technique generates excessive pressures, ranging from a minimum of 300 mm Hg to >800 mm Hg.[86] A finger can be exsanguinated with a Penrose drain, but a separate drain should be used as a tourniquet (Fig. 35-16). A few millimeters of difference in total stretch makes a large difference in the pressure applied by this type of tourniquet. [86] Alternatively, a finger can be exsanguinated with a moistened piece of gauze opened to its fullest length, folded in half, and rolled tightly around the elevated finger from tip to base. A Penrose drain is stretched around the base of the finger and secured with a hemostat, and the gauze is removed.

A latex rubber surgical glove placed over a patient's hand also can serve as a finger tourniquet. The tip of the glove covering the injured digit is removed, and the latex rubber is then rolled proximally along the patient's finger to form a constricting band at the base (Fig. 35-17). Another advantage of this technique is that contamination of the wound during closure is less likely. Rolled surgical gloves produce pressures ranging from 113 to 363 mm Hg, depending on the thickness, the amount of glove finger removed, the number of rolls, and the size of the glove in relation to the size of the patient's hand. [86] Pressure under a Penrose drain ranges between 100 and 650 mm Hg, but it can be more easily controlled.[87] Commercial ring-shaped exsanguinating digit tourniquets are available (Tourni-cot [Mar-Med Company]) (Fig. 35-18). There is a danger of forgetting to remove such a small tourniquet and of accidentally incorporating it in the dressing.

These techniques provide bloodless fields in which to examine, clean, and close extremity wounds. The maximum tourniquet time on a finger should not exceed 20 to 30 minutes.[86] [87] Debridement of questionably devitalized tissue in a wound is best accomplished *without* a tourniquet or pharmacologic vasoconstriction, because bleeding from tissues is often an indication of their viability. [80]

CLOSURE

The various techniques of wound closure are presented in Chapter 36 . The remainder of this chapter addresses issues related to wound management (e.g., secondary closure, wound dressings, antibiotic use, aftercare instructions, and suture removal).

Open vs Closed Wound Management

Wounds that heal spontaneously (i.e., by secondary intention) undergo much more inflammation, fibroplasia, and contraction than those whose edges are reapproximated by wound closure techniques.[8] [87A] During wound healing, contraction covers the defect, yet it may have undesirable consequences—notably, deformity (contracture) or loss of function. Left to itself, the healing process may be unable to close a

Figure 35-16 Use of Penrose drain for exsanguination *(A)* of a wounded digit. A second Penrose drain is applied *(B)* as a finger tourniquet, and the first drain is removed *(C)*. During actual patient care, the clinician would use sterile technique including gloves.

defect completely in areas in which surrounding skin is immobile, such as on the scalp or in the pretibial area. [8] Exposed tendons, bone, nerves, or vessels may desiccate in an open wound. If the patient is careless with an otherwise adequate dressing that covers an open wound, the wound may be further contaminated. [88] The advantages of surgical closure of wounds are apparent: This procedure minimizes inflammation, fibroplasia, contracture, scar width, and contamination.

On the other hand, risks are incurred when wounds are closed. Closure of contaminated wounds increases the

Figure 35-17 Use of a sterile glove to provide a clean field and serve as a finger tourniquet. The distal end of the glove is clipped *(A),* and the glove finger is rolled proximally over the digit *(B, C)*. During actual patient care, the clinician would use sterile technique including gloves.

probability of wound infection, with impaired healing, dehiscence, and sepsis as possible complications. After cleaning and debridement, wounds left unsutured appear to have a higher resistance to infection than do closed wounds.

Sutures in themselves are detrimental to healing and increase the risk of infection. [12] [88] [89] Each suture inflicts an intradermal incision, damaging surface epithelium, dermis, SQ fat, blood vessels, small nerves, lymphatics, and epithelial appendages such as hair follicles, sweat glands, and ducts. These appendages, once divided and separated by a stitch,

Figure 35-18 As in Figure 35-7 , a sterile glove has been used to provide a sterile field for the thumb. In addition, a commercial rubber ring tourniquet device has been applied to enhance hemostasis. The accompanying tag has been left exposed to remind the operator of the tourniquet device.

usually undergo inflammation and resorption. [90] [91] Each suture is another piece of foreign material that provokes inflammation. [8] When a suture is removed, bacteria that have settled on the exposed portion of the suture are pulled into the suture track and deposited there. [90] Raised pretibial flap lacerations in elderly patients often necrose when sutured but survive and heal well by secondary intention if taped back into position. [92]

If the wound is judged to be clean or is rendered clean by scrubbing, irrigation, and debridement, it may be closed. If the wound remains contaminated despite the best of efforts, it must be left open to heal by secondary intention. If the status of the wound is uncertain, the clinician must estimate the risk of infection. Another option available is delayed primary closure.

Delayed Primary or Secondary Closure

There is a common misconception that all wounds must either be sutured within a few hours or left open and relegated to slow healing and an unsightly scar. If there is a substantial risk that closure of a particular wound might result in infection, the decision to close or to leave the wound open can be postponed (Fig. 35-19). The condition of the wound after 3 to 5 days will then determine the best strategy (Fig. 35-20). Although cleaning and debridement should be accomplished as rapidly as possible, there is no urgency in closing a wound. Edlich and coworkers point out that "the fundamental basis for delayed primary closure is that the healing open wound gradually gains sufficient resistance to infection to permit an uncomplicated closure." [25] Despite its effectiveness, delayed primary closure is a technique that is unappreciated and likely underused by most clinicians.

Open wound management is usually an outpatient procedure. The technique consists of the usual careful cleaning and meticulous debridement, followed by packing of the wound with sterile, saline-moistened, fine-mesh gauze. The packed wound is covered by a thick, absorbent, sterile dressing. Depending on the specifics of the wound and the ability of the patient to perform his or her own wound care, the packing may be changed daily at home or in the ED, or the wound

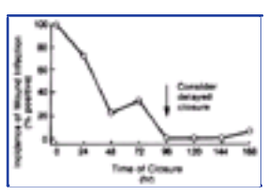

Figure 35-19 Incidence of wound infection over time when delayed closure is performed. Delayed closure is best accomplished on the fourth or fifth day to minimize the risk of infection. *(From Edlich RF, Thacker JG, Rodeheaver GT, et al: A Manual for Wound Closure. St. Paul, MN, 3M Medical Surgical Products, 1979. Reproduced by permission. © 1979 by Minnesota Mining and Manufacturing Company.)*

may be left undisturbed for several days. Sterile saline-soaked packing is standard, and there is no need to impregnate wounds with antiseptics. Prophylactic antibiotics are occasionally prescribed, but their use is neither mandatory nor of proven benefit. On the fourth postoperative day, the wound is reevaluated for closure. If no evidence of infection is present, the wound margins can be approximated (delayed primary closure), or the wound can be excised and then sutured (secondary

closure) with minimal risk of infection. Because the wound is closed before the proliferative phase of healing, there is no delay in final healing, and the results are indistinguishable from those of primary healing.

Certain wounds should almost always be managed open or by delayed closure. These include wounds that are already infected and those heavily contaminated by soil, organic matter, or feces. Also included in this category are wounds associated with extensive tissue damage (e.g., high-velocity missile injuries, explosion injuries of the hand, or complex crush injuries) and most bite wounds. Lacerations to the bottom of the feet, such as those occurring when the patient steps on an unknown object while wading in a stream or running through a field, are ideal candidates for delayed closure. Human bite wounds should probably never be sutured. Clinicians disagree as to which animal bite wounds may be closed initially. Most would suture cosmetically deforming injuries, including facial bites, and bite wounds that can be completely excised.[42] [93] Others would suture nonextremity dog bites.[94] In severe soft tissue injuries, delayed closure allows time for nonviable tissue to demarcate from uninjured tissue. Debridement can then be accomplished with maximal preservation of tissue. [88]

PROTECTION

Dressings

At the conclusion of wound repair, dried blood on the skin surface should be wiped away gently with moistened gauze, and the wound should be covered with a non-adherent dressing.

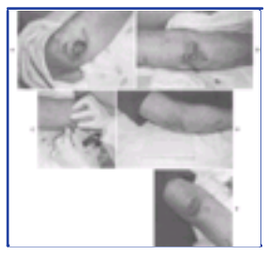

Figure 35-20 *A*, This dirty contused wound, now 18 hours old, is an ideal candidate for a delayed primary closure. *B*, At presentation the wound is anesthetized, scrubbed, irrigated, and debrided. *C*, The wound is packed with sterile gauze and covered by a dry dressing. No antibiotics were prescribed. *D*, Four days later the packing is removed, and the wound is minimally debrided. *E*, Interrupted sutures are placed as though this is a fresh, clean wound. At suture removal 10 days later, only a linear scar was evident.

Depending on the specifics of the wound and the type of repair, a dressing can consist of a simple dry gauze pad or a complex multilayer dressing. Some wounds, such as sutured scalp lacerations, do not routinely require any dressing. Although various specialized (and expensive) dressings are available, there are little data to support their use over readily available, properly applied gauze dressings.

Function of Dressings

Dressings serve various functions. They protect the wound from contamination and trauma, absorb secretions from the wound, immobilize the wound and the surrounding area, exert downward pressure on the wound, and improve the patient's comfort.[19] [95] [96] Occlusive dressings on burns or abrasions prevent painful exposure of the wound to the air and dehydration of the wound surface.[97] Sutured wounds are susceptible to infection from surface contamination during the first 2 days after wound repair. Dressings effectively protect the wound from contamination during this vulnerable period.

One of the primary functions of a gauze dressing is to absorb the serosanguineous drainage that exudes from all wounds. Absorbent dressings also reduce the development of stitch abscesses to some extent. Surface sutures produce small indentations at their points of entrance; tiny blood clots and debris overlie these indentations, allowing bacterial growth at the site. Small "stitch abscesses" can develop; these are initially undetectable but are nevertheless destructive to epithelium. Stitch abscesses rarely infect the entire wound but can slightly increase the width of the scar and produce noticeable, punctate suture marks.[19]

The most common type of dressing is constructed in three layers: a nonadherent contact layer, an absorbent layer, and an outer wrap (Fig. 35-21).[96] Ideally, this dressing provides nonadherence without maceration.

Contact Layer: Dry, Semiocclusive, and Occlusive Dressings

Petrolatum gauze (e.g., Adaptic, Xeroform, Betadine, Aquaflo) can be applied next to the wound surface to prevent the wound

Figure 35-21 A common three-layer dressing, consisting of antibiotic ointment, Adaptic, and gauze.

from sticking to the dry gauze in the absorbent layer and to protect the regenerating epithelium (Table 35-2). (Nonadherent material should always be used to cover skin grafts.)

Coarse weaves of gauze, usually available in the form of multilayered pads, absorb blood and exudate, but the dressing will adhere if the interstices of the fabric are relatively large. Capillaries, fibrin, and granulation tissue will penetrate and become enmeshed in the material. If the proteinaceous exudate from the wound dries by evaporation, the scab usually clings to the dressing.[98] [99] Some clinicians use this effect to "debride" the wound when the gauze is removed. However, it also destroys healing tissue, particularly the new epithelium. Debridement of the wound with wet-to-dry dressings is quick, but debridement with surgical instruments is more controlled and less traumatic.

Adherence to the wound can be avoided if the dressing is nonabsorbent, occlusive, or finely woven. Some clinicians use fine mesh gauze (41 to 47 warp threads per square inch) rather than petrolatum gauze on abrasions, especially on those wounds that are heavily contaminated, because removal of this type of dressing debrides only the small tufts of granulation tissue that become fixed in the mesh pores, leaving a clean, even surface. Once a healthy, granulating surface is present and re-epithelialization is proceeding, nonporous dressings can be used.[99] Fine mesh gauze also is used next to exposed tissue in wounds being considered for delayed primary closure; a protective and absorptive bulky dressing is placed on top of the wound.

Wounds covered with permeable dressings such as plain gauze tend to dry out. Drying of the wound surface damages a shallow layer of exposed dermis, which impedes epidermal resurfacing of abrasions, burns, and incisions.[96] Wound desiccation results in further epidermal necrosis, crust formation, and increased inflammation.[99] [100] If the wound is kept moist by covering it with an occlusive film soon after wound management and if the film is left in place for at least 48 hours, the epidermis will migrate over the surface of the dermis up to 100% faster than when a dry scab is allowed to form.[101] [102] [103] In one study, the occluded half of a surgical incision produced

TABLE 35-2 -- Advantages of Occlusive Dressings

1. More rapid healing
2. Less pain from air exposure
3. Better cosmetic results
4. Fewer dressing changes

5. Better protection from bacteria

Data from Eaglstein WH: Effect of occlusive dressings on wound healing. Clin Dermatol 2:107, 1984.

a more linear, less pigmented scar.[104] Protection of wounds that are healing by secondary intention with occlusive or semiocclusive dressings has several advantages,[10] including more rapid healing, less pain from air exposure, better cosmetic result, few dressing changes, and protection from bacteria.

This occlusive effect is achieved with various polyurethane-derived membranes, such as Epilock (Derma-Lock Medical Corporation), Op-Site (Smith and Nephew, Ltd.), Tegaderm (3M), Bioclusive (Johnson & Johnson), and Primaderm (ACCO, Inc.); those with soluble collagen or gelatin backing, such as DuoDerm (Convatec) and Biobrane (Woodroof Laboratories); and products with hydrogels, such as Vigilon. [99]

One fear of using occlusive dressings is that microorganisms will proliferate in the moist environment beneath the occlusive film and increase wound infection rates. [96] [105] Occlusive dressings such as DuoDerm actually serve more as a barrier to external pathogenic bacteria [106] ; although *surface* bacteria under occlusive dressings can multiply,[107] chronic wounds, usually contaminated with large numbers of bacteria, are routinely treated with occlusive dressings successfully. [108]

A paint-on collodion dressing over a wound closed with a buried subcuticular stitch provides considerably greater resistance to infection than wounds closed by the same technique but with no dressing. The use of collodion obviates the need for a gauze dressing, frequent dressing changes, and uncomfortable dressings in areas such as the groin, the axilla, and the neck.[109] However, the collodion does not allow drainage of the wound and so is rarely used.

Another concern is that occlusive dressings will macerate underlying skin. Optimal wound appearance under a dressing is a moist red surface with capillary and epithelial growth. Collagen sponge dressings provide this appearance (if they are not accidentally dislodged), whereas both DuoDerm and Op-Site adhere to the wound site, macerate it, and produce a thick eschar that may be difficult to remove. However, underneath the eschar the surface is epithelialized. [110] Wounds covered with certain occlusive dressings or with silver sulfadiazine (Silvadene [Marion Laboratories]) applications appear to be blanketed with pus; this exudate actually represents the beneficial proliferation of macrophages and polymorphonuclear leukocytes. [103] [111]

Adhesive-backed dressings (e.g., DuoDerm and Op-Site) have a tendency to adhere to and remove new epidermis, and they do not allow exudate to drain out the edges of the dressing. Between dressing changes, the wound should be coated with petrolatum or an antibiotic ointment before these products are applied. [10] Epilock has the advantage of thermally insulating the wound by virtue of its thickness, but unlike Tegaderm and Op-Site, it is opaque and does not allow inspection of the underlying wound surface.[111] Because Epilock allows drainage of exudate, it is better tolerated by patients if the overlying gauze bandage is changed daily.

Other nonadherent-type dressings include Adaptic, Xeroform, Betadine, Dermicel, and the nonabsorbent Telfa. Petrolatum gauze does not appear to enhance epidermal healing.[112]

Absorbent Layer

In dressing wounds with considerable drainage, sufficient gauze should be used to cover the wound and to absorb all of the drainage. Dressings on such wounds can be changed daily, which is frequent enough to avoid bacterial overgrowth beneath the dressing. [8] [99] Once a dressing becomes moist, pathogens can pass through it to the underlying wound. [96] Any dressing should be changed whenever it becomes soiled, wet, or saturated with drainage. Fluid accumulating under an occlusive dressing should be aspirated or the dressing changed every 1 to 2 days during the first week or until the exudate no longer accumulates. [113] A dressing that is used to absorb exudate or debride the wound must be changed more frequently than one designed solely to occlude.

Outer Layer

Dressings and bandages can serve as surface splints (as can surgical tape) by reducing mechanical stresses on the wound during the early phases of healing. Even when subcuticular stitches have been placed, these "external splints" are useful in relieving tension across the wound. They are most needed between the 7th and 42nd days, the time of collagen synthesis and remodeling. [19]

Compressive dressings may be helpful in preventing hematoma formation and eliminating dead space within a wound. They are particularly useful in wounds that have been undermined extensively and in facial wounds, in which SQ capillary bleeding and swelling can exert tension on fine skin sutures and jeopardize skin closure. Pressure dressings should be used to immobilize skin grafts. Surgical tape can serve as a pressure dressing in areas such as fingertips on which bandages cannot be easily applied. However, a pressure dressing should not be used as a substitute for good hemostasis. [8]

Pressure dressings should be applied to all ear lacerations to prevent hematoma formation and subsequent deformation and destruction of cartilage. The ear should be enveloped in the dressing so that pressure from the outer bandage is distributed evenly across the irregular surface of the pinna. Moistened cotton is packed into the concavities of the pinna until the cotton is level with the most lateral aspect of the helical rim. Square pieces of gauze cut to fit the curvature of the ear are placed *behind* (medial to) the pinna. Several more gauze squares are placed on the lateral surface of the ear; the packing is then secured in place with a circumferential head bandage. The bandage must not encompass the opposite ear because it would just as easily cause pressure necrosis of that ear if left unprotected. Application of a pressure dressing for the ear is discussed further in Chapter 65 .

Traumatic wounds are bandaged to compress or immobilize the wound or to secure and protect the underlying dressing. Most bandaging is performed on extremities, on which dressings are difficult to secure with tape alone. Rolls of cotton (Kerlix; Kling stretch gauze) are well suited for this purpose. The bandage is wound around the extremity, advancing proximally with circular, overlapping turns. Care should be taken to avoid allowing wrinkles in the bandage, which will later create pressure points, or making loose turns that shorten the effective life of the dressing. When joint surfaces are crossed, the cotton is anchored distally with several turns, unrolled obliquely across the joint several times in a figure-of-eight pattern, and anchored proximally by two complete turns. This process is repeated until the bandage is securely in place. The ends of the bandage are fastened to the skin by strips of adhesive tape.

Bandages over the forearm and the lower extremities are particularly prone to slippage because of the constant motion of these parts and because of the marked changes in extremity diameter over a short distance. The roll of bandage can be rotated 180° after each circular turn, producing a reverse spiral and reducing the bandage's mobility (Fig. 35-22). A "tube" of elastic cotton netting (e.g., Surgifix, Tubex, Surgitube, HygiNet) pulled over the bandage or unrolled from a metal applicator frame effectively stabilizes the entire dressing in these areas (Fig. 35-23). Another useful technique consists of placing strips of tape on opposite sides of the extremity, leaving the ends free. The bandage is wrapped around the dressing, covering the portions of the tape that are attached to the skin. The free ends of tape are then incorporated in the bandage (Fig. 35-24).[114]

Certain chemically treated wide-mesh weaves have the properties of cling and stretch, holding snugly in place but expanding if edema develops. [98] An elastic cotton roll (Kerlix) allows the bandage to conform to body contours, provides some mobility to bandaged joints, and permits the wound to swell without the circumferential bandage constricting the extremity. The inelastic Kling bandage better immobilizes the part. Rigid immobilization with plaster splints or braces is needed to protect wounds in mobile areas, such as around large joints.

Most scalp wounds do well when left uncovered. If a dressing is necessary, it must be held in place by a bandage. There are many techniques for bandaging heads. One method[115] is shown in Figure 35-25 . Methods of bandaging wounds in other locations of the body are described in detail in other texts. [79] [95] Dressings should be changed when they become externally contaminated, saturated with exudate, or when inspection and wound cleaning is required.

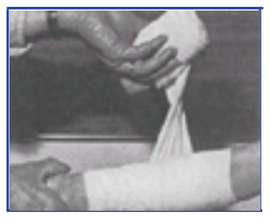

Figure 35-22 Snugness of the bandage is increased by 180° rotation of the bandage roll after each circular turn to create a reverse spiral. *(From Norton LW: Trauma. In Hill GJ II: Outpatient Surgery. Philadelphia, WB Saunders, 1980. Reproduced by permission.)*

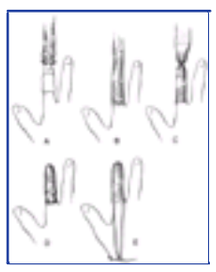

Figure 35-23 Finger dressing. *A,* The inner layer is nonadherent gauze or whatever is required for soft tissue care. The middle layer is 2 × 2 in. gauze sponges wrapped circumferentially and held in place with a small strip of tape. *B,* Begin No. 2 tube gauze at the base of the finger. It is useful to hold this end with one finger while the tube gauze applicator is pulled toward the fingertip. A twisting motion firms the wrap about the digit; generally about 90° is necessary. Excessive stretch or twisting can compromise circulation. *C,* When the fingertip is reached, make a 360° twist. *D,* Pass the applicator toward the finger base with an additional 90° twist. Repeat once more; thus, three layers are in place. *E,* Cut enough gauze to reach the base of the finger, and tape it there. As an alternative, pull the final layer beyond the tip, leaving it long enough to reach to and around the wrist (about three times the finger length). Split this gauze into two strands; bring them dorsally to the wrist, knot, and loosely wrap around the wrist. *(Redrawn from Kaplan EN, Hentz VR: Emergency Management of Skin and Soft Tissue Wounds: An Illustrated Guide. Boston, Little, Brown, 1984, p 86. Reproduced by permission.)*

Dressings vary in their absorbency, adhesiveness, occlusiveness, opacity, and insulating properties. Further research may identify types of dressings best suited for different phases of the healing wound. Currently, a two- or three-layer dressing is used for most traumatic wounds; the choice of material for the contact layer is determined by the characteristics of the individual wound. [116]

Splinting and Elevation

Although splints are readily applied to orthopedic or soft tissue injuries, immobilization of wounds and sutured lacerations is often neglected, despite the fact that these techniques

Figure 35-24 *A,* When a Kling or Kerlix wrap must be applied to an area such as the forearm, start by putting a strip of tape on opposite sides of the arm, leaving the ends free. *B,* Wrap the bandage around the arm, covering the portions of the tape that are attached to the skin. *C,* After completing one layer of wrapping, tuck the free ends of the tape down so that the nonadhesive side faces the first layer of wrapping and the sticky side faces out. Place another layer of wrapping around the arm. *D,* After completing a second layer of wrapping, the dressing will not slip because it is adhered to itself as well as to the skin. *(From Lazo J: Non-slip dressing technique. Res Staff Physician 22:103, 1976. Reproduced by permission.)*

may enhance healing and provide patient comfort. Immobilization of an injured extremity promotes healing by protecting the closure and by limiting the spread of contamination and infection along lymphatic channels. Wounds overlying joints are subjected to repeated stretching and movement, which delays healing, widens the scar, and could

Figure 35-25 *A–E,* Technique for bandaging the head. A strip of bandage (the "tie strip") 3 inches wide and 3 feet long is placed over the head in the frontal plane (*A)*. While the patient maintains downward tension on the first strip of bandage, the clinician places a full-length gauze bandage at the forehead level in a horizontal plane, winding the bandage around the head (*B)*. (The "Kling"-type bandage is preferred.) The main bandage is stabilized with several turns, passing near the patient's ear, then wrapped around one side of the tie in a full turn (*C)*. The main bandage is then taken across the front of the head, wrapped full-turn around the other side of the tie bandage (*D)*. The main bandage is wrapped around the head, from front to back, overlapping with each pass. The dressing is secured in place by tying the ends of the tie strip under the chin (*E)*. This dressing can be removed easily by untying the chin straps and gently pulling both ends of the tie strip upward (*F)*.

possibly disrupt the sutures. [25] Splints are almost always required for lacerations that overlie joints and are frequently necessary for protection of wounds involving fingers, hands, wrists, the volar aspects of forearms, the extensor surfaces of elbows, the posterior aspects of legs, the plantar surfaces of feet, and the extremities when skin grafts have been applied. Splinting is often underused by the emergency clinician in the treatment of lacerations.

A plaster or aluminum splint may be incorporated into a bandage to reduce the mobility of the part. Splinting techniques for extremities are explained more fully in Chapter 51 and Chapter 52 .

Elevation of injured extremities is important in all but trivial injuries. Elevation limits edema formation, an expected sequela of trauma and inflammation, and allows more rapid healing. [25] Elevation also reduces throbbing pain. Patients given this information are often more motivated to elevate the extremity as instructed. Slings can be used to elevate wounds involving the forearm or the hand.

Ointments

The safety and efficacy of topical antibiotic preparations used on wound surfaces are unproven and still debated. Many clinicians routinely suggest the use of antibiotic ointments over sutured wounds, while others opt for a simple dry dressing. No universal standard exists. Since a major benefit from the routine use of topicals has never been substantiated, one would intuit that there is no compelling reason to use them. However, *the application of a topical preparation involves that patient in the ongoing care of the wound, and forces the patient to evaluate the healing process on a regular basis*. These reasons alone make the use of topicals reasonable interventions.

Some investigators warn of skin sensitization by preparations containing neomycin [91] and others, of the emergence of resistant strains of bacteria with any topical antibiotic. [117] Other studies have shown that use of a triple-antibiotic preparation containing neomycin, bacitracin, and polymyxin provides a broad spectrum of

protection against infection in *abrasions* without systemic absorption and toxicity or the emergence of resistant strains of bacteria. Unless this topical antibiotic ointment is used repeatedly or on inflamed skin, there is a relatively low risk of allergic sensitization (Fig. 35-26).[118] There is evidence that the active agents in Neosporin ointment and Silvadene cream, as well as their inert bases and vehicles, improve wound healing. [97] [119] [120] In a prospective, randomized, double-blind study, Dire and colleagues found that bacitracin and Neosporin ointments reduced the infection rate over that seen with plain petrolatum ointment. [121] Mupirocin (Bactroban, GlaxoSmithKline, London), which is a topical antibiotic in a water-soluble base, is an alternative.

Ointments can be used to reduce the formation of a crust that covers and separates the edges of the wound. Lacerations surrounded by abraded skin are especially predisposed to coagulum formation. In such cases the patient can be instructed to cleanse the wound frequently and to follow the cleansing with an application of ointment during the first few days.[25] Ointments also prevent the dressing from adhering to the wound.[6] Some researchers recommend using bacitracin applied in a thin coating, not for protection against infection, but for prevention of these mechanical problems.

Note that the stronger topical corticosteroids have detrimental effects on healing. Application of 0.1% triamcinolone acetonide in an ointment retards healing in wounds by as much as 60%, whereas hydrocortisone probably does not interfere with epithelialization. [102] [112] Some clinicians believe that single and low doses of oral corticosteroids probably have no effect on wound healing but that repeated, large doses of steroids (=40 mg of prednisone per day) inhibit healing, particularly if used before the injury or during the first 3 days of the healing phase. [122] [123] There is some evidence that topical vitamin A may reverse some of the anti-inflammatory and immunosuppressive effects of corticosteroids. [124] The exact value of ointments in the treatment of lacerations has yet to be determined. However, their routine use after wound cleaning does encourage patient inspection of the wound.

Ointments should not be used on wounds closed with tissue adhesive because the ointment will dissolve the adhesive.

Wound Cultures

Cultures taken at the time of wound preparation and closure in the ED serve no useful purpose and are not recommended. Results of such cultures cannot logically guide future antibiotic selection and often only confuse the picture. It is not standard to routinely culture all infected wounds presenting after closure, unless extenuating circumstances exist. Often such cultures reveal multiple organisms, and do not reflect either the principal infecting organism or the antimicrobials that are required to cure a wound infection.

Systemic Antibiotics

Most traumatic soft tissue injuries sustain a low level of bacterial contamination. [88] The standard wound infection rate in unselected ED wounds is 2% to 5%. In a number of clinical studies of traumatic wounds, prophylactic antibiotics administered orally [20] [125] [126] and intramuscularly [13] [126A] [30] [127] in various regimens did not reduce the incidence of infection.

In experimental models of contaminated incisions, antibiotics have no therapeutic value >3 hours after the injury. [128] [129] When the wound is contaminated with >10 [9] bacteria per

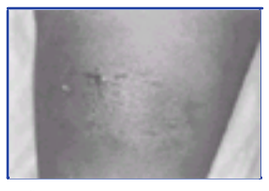

Figure 35-26 This patient used a neomycin-containing ointment on a minor wound, and developed redness, swelling, pruritis, and skin changes. The patient thought it was an infection but it was a contact dermatitis from the neomycin. Plain bacitracin ointment will not do this.

gram of tissue (such as wounds in contact with pus or feces), infection will develop despite antibiotic treatment. [42] Most clinical investigations of antibiotic use in emergency patients have omitted heavily contaminated wounds in their series. Studies of antibiotic prophylaxis for animal bite wounds have produced variable results, and no large study providing stratification of the many prognostic factors has been done. [130]

The use of antibiotics varies widely among clinicians, and because of limited scientific data, there is no clear practice standard. [131] In most soft tissue wounds where the level of bacterial contamination after cleaning and debridement is low, antibiotics have not been proven beneficial. Antibiotics may have marginal benefit when the level of contamination is overwhelming or if the amount of questionably viable tissue left in the wound is considerable (e.g., with crush wounds). Antibiotics should be considered for extremity bite wounds, puncture-type bite wounds in any location, intraoral lacerations that are sutured, orocutaneous lip wounds, wounds that cannot be cleaned or debrided satisfactorily, and highly contaminated wounds (e.g., those contaminated with soil, organic matter, purulence, feces, saliva, or vaginal secretions). They also should be considered for wounds involving tendons, bones, or joints; for wounds requiring extensive debridement in the operating room; for wounds in lymphedematous tissue; for distal extremity wounds when treatment is delayed for 12 to 24 hours; for patients with orthopedic prostheses; and for patients at risk of developing infective endocarditis. [25] If systemic antibiotics are considered necessary, they should be given intravenously or intramuscularly in the earliest stages of wound management.

The choice of antibiotic, particularly for bite wound prophylaxis, is as controversial as the indications for usage. Many species of bacteria cause bite wound infections, making complete coverage impossible.[31] [132] [133] Some antibiotic regimens recommended for bite wounds include dicloxacillin or cephalexin for high-risk dog bite wounds, dicloxacillin or cephalexin plus penicillin for human or cat bite wounds, and amoxicillin-clavulanic acid or cefuroxime for any domestic animal bite. The duration of antibiotic prophylaxis also is in question. It is common practice to provide antibiotics for 72 hours, although data from surgical studies indicate that antibiotics administered beyond the first postoperative day provide no additional protection. [134] Short courses of antibiotics do not seem to increase the incidence of resistant strains of organisms. [135] In all cases, the use of antibiotics should remain subordinate to careful cleaning and debridement. If the infection risk is high enough to warrant antibiotics, secondary closure should be considered. See additional comments on animal bites at the end of this chapter.

There are *no data to support the routine use of prophylactic antibiotics for the majority of wounds encountered in the ED*. [136] Antibiotics should not be used as a substitute for proper wound preparation or a measure to overcome factors suggesting delayed wound closure. The downsides of antibiotic use include needless expense; potential side effects (e.g., rash, anaphylaxis, diarrhea, vomiting); and the development of resistant bacteria, both in the wound and in general. If antibiotics are used, they should be given as soon as possible after wounding and continued for only 2 to 3 days in the absence of a developing infection.

Immunoprophylaxis

Although tetanus is rare, it still occurs in the United States (about 50 cases per year) and is a preventable disease. Therefore, any wound should be assessed for its potential to cause tetanus, and prophylaxis should be considered in the ED. Gergen and colleagues demonstrated that about 70% of Americans older than 6 years of age had protective levels of tetanus antibodies. [137] Levels declined as age increased, and elderly women had the lowest levels of protection. Hispanics (and likely other immigrants) were most likely to have inadequate immunity. Hence, efforts at preventing tetanus should be especially addressed in immigrants and the elderly.

Recommendations for tetanus prophylaxis have evolved since the 1980s. The guidelines published by the Public Health Services Advisory Committee on Immunization Practices, Centers for Disease Control and Prevention, differ slightly from those of the American College of Surgeons in the use of tetanus immune globulin. [138] [139]

Many cases of tetanus develop despite prior immunization; tetanus can result from chronic skin lesions and apparently minor or clean wounds. [140] In 10% to 20% of cases, no precedent wound can be identified. Patients' recall of past immunizations is imperfect, and immunity may rarely be inadequate after a complete series of tetanus toxoid.[141] Furthermore, there is no precise consensus on the definition of a "tetanus-prone wound," yet treatment decisions are based on the differentiation between clean and contaminated wounds. Some investigators warn of overtreatment [142] [143] and others maintain that the risk of therapy is minimal compared with the danger of tetanus.[144] [145] After comparing those risks and benefits, most clinicians would agree that a certain amount of overtreatment is acceptable. However, tetanus

boosters given more frequently than advised increase the incidence of adverse reactions to subsequent injections.

While any break in the skin can be classified as "tetanus prone" traditional definitions of tetanus-prone wounds include injuries >6 hours old; wounds contaminated by feces, saliva, purulent exudate, or soil; wounds with retained foreign bodies or containing devitalized or avascular tissue; established wound infections; penetrating abdominal wounds involving bowel; deep puncture wounds; and wounds caused by crush, burns, or frostbite (Fig. 35-27). When patients are questioned about their tetanus immunization status, they should be asked if they completed the primary immunization series, and if not, how many doses have been given.

Patients who have not completed a full primary series of injections may require both tetanus toxoid and passive immunization with tetanus immune globulin. Tetanus immune globulin will decrease, but will not totally eliminate, the subsequent development of clinical tetanus. The preferred preparation for active tetanus immunization in patients 7 years of age and older is 0.5 mL of tetanus toxoid (plus the lower, adult dose of diphtheria toxoid); the dose of tetanus immune globulin is 250 to 500 units given intramuscularly.[137] Mild local reactions consisting of erythema and induration are common after tetanus toxoid injections. Compared to the rate of reactions to tetanus toxoid (about 20%), reactions are about twice as common if diphtheria immunization is coupled with tetanus immunization. Some patients with high antibody levels develop a hypersensitivity reaction of tenderness, erythema, and swelling, or serum sickness. Generalized urticarial reactions and peripheral neuropathy have also been reported.

Figure 35-27 Tetanus immunization guidelines.

A significant percentage of elderly patients fail to develop protective antitoxin antibody titers after 14 days when given tetanus toxoid boosters.

Because protective levels of tetanus antibodies tend to parallel levels of antibodies to diphtheria, it has been recommended that both immunizations be given simultaneously. Both tetanus and diphtheria immunization have been implicated as a cause of adverse reactions. Tetanus and diphtheria toxoid are products of human antisera, and serious reactions are rare.[146] The most common reaction is a painful, indurated, tender eruption at the injection site, occasionally accompanied by a fever and mild systemic symptoms. This is a hypersensitivity reaction (Arthus-type reaction), not an infection or immunoglobulin E (IgE)-mediated allergy. As such, this reaction does not require drainage or antibiotics, nor does it represent an absolute contraindication to further immunizations. Local reactions are more common in patients who have been given multiple immunizations, so it is unwise to give excessive immunizations "just to be safe." In tetanus-prone injuries, "hyperreactors" can be given tetanus immune globulin. A minor febrile illness, such as an upper respiratory infection, is not a reason to delay immunization. The only absolute contraindication to tetanus toxoid is a history of anaphylaxis or a neurologic event. In such cases, tetanus immune globulin can be given safely. Pregnancy is not a contraindication to either toxoid or immune globulin, although some suggest that the toxoid be used with caution during the first trimester. Given the excellent amnestic response to the toxoid, it is likely that the primary immunization series, coupled with intermittent boosters, conveys immunity for most of one's life.

When a wound results from the bite or scratch of a wild or domestic animal, prophylaxis against rabies also must be considered (Table 35-3 and Table 35-4). Further discussion of the prevention of rabies is provided elsewhere.[147] [148]

PATIENT INSTRUCTIONS

Successful wound healing is partly dependent on the care given to the wound once the patient leaves the emergency center. Patient satisfaction depends not only on the cosmetic result, but also on the expectation of that result. [10] Therefore, the patient should receive thorough and clear instructions.

The patient should be informed that no matter how skillful the repair, any wound of significance produces a scar. Most scars deepen in color and become more prominent before they mature and fade. The final appearance of the scar cannot be judged before 6 to 12 months after the repair. [9] [91]

Patients may experience dysesthesias in or around a scar, particularly about the midface. Gentle rubbing or pressing on the skin may relieve the symptoms. If wounds extending to SQ levels lacerate cutaneous nerves, patients may be bothered by hypoesthesia distal to the wound. Dysesthesia and anesthesia usually resolve in 6 months to 1 year.[10]

Because the wound edges are rapidly sealed by coagulum and bridged by epithelial cells within 48 hours, the wound is essentially impermeable to bacteria after 2 days.[19] [149] The patient should be instructed to protect the wound by keeping the dressing clean and dry for 24 to 48 hours. In this initial period the dressing should be changed only if it becomes externally soiled or soaked by exudate from the wound. If possible, the injured part should be kept elevated.

After 48 hours, the patient may remove the dressing in uncomplicated wounds and check for evidence of infection: redness, warmth, increasing pain, swelling, purulent drainage, or the "red streaks" of lymphangitis. Not all patients are able to identify these signs of infection; it is prudent to have patients with complicated or infection-prone wounds examined in 2 days by a clinician or nurse. [150] Interestingly, patients may be more likely to fail to recognize a bona fide infection than to overdiagnose an infection when it is absent. [149] [150]

TABLE 35-3 -- Rabies Postexposure Prophylaxis Guide—July 1984 [147]

The following recommendations are only a guide. In applying them, take into account the animal species involved, the circumstances of the bite or other exposure, the vaccination status of the animal, and presence of rabies in the region. Local or state public health officials should be consulted if questions arise about the need for rabies prophylaxis.

Animal Species	Condition of Animal at Time of Attack	Treatment of Exposed Person*
Domestic		
Dog and cat	Healthy and available for 10 days of observation	None, unless animal develops rabies†
	Rabid or suspected rabid	HRIG‡ and HDCV
	Unknown (escaped)	Consult public health officials. If treatment is indicated, give HRIG‡ and HDCV
Wild		
Skunk, bat, fox, coyote, raccoon, bobcat, and other carnivores	Regard as rabid unless proven negative by laboratory tests§	HRIG‡ and HDCV
Other		
Livestock, rodents, and lagomorphs (rabbits and hares)	Consider individually. Local and state public health officials should be consulted on questions about the need for rabies prophylaxis. Bites of squirrels, hamsters, guinea pigs, gerbils, chipmunks, rats, mice, other rodents, rabbits, and hares almost never call for antirabies prophylaxis.	

From Leads from the Mortality and Morbidity Weekly Report. JAMA 252:883, 1984.

**All bites and wounds should immediately be thoroughly cleansed with soap and water.* If antirabies treatment is indicated, both human rabies immune globulin (HRIG) and human diploid cell rabies vaccine (HDCV) should be given as soon as possible, *regardless* of the interval from exposure. Local reactions to vaccines are common and do not contraindicate continuing treatment. Discontinue vaccine if fluorescent antibody tests of the animal are negative.
†During the usual holding period of 10 days, begin treatment with HRIG and HDCV at first sign of rabies in a dog or cat that has bitten someone. The symptomatic animal should be killed immediately and tested.
‡If HRIG is not available, use antirabies serum, equine (ARS). Do not use more than the recommended dosage.
§The animal should be killed and tested as soon as possible. Holding for observation is not recommended.

Patients may fail to appreciate the presence of an infection in >? of cases. Therefore, in high-risk circumstances, a scheduled, rather than an as-needed, wound check should be advised. Patients should be informed that sutures themselves do not cause pain. A painful wound is often a sign of infection or suture reaction, and pain should prompt a wound check. If there is no sign of infection after 48 hours, the patient can care for the wound until it is time for removal of the sutures.

A daily gentle washing with mild soap and water to remove dried blood and exudate is probably beneficial, especially on areas such as the face or the scalp. [147] [150] Although

TABLE 35-4 -- Rabies Postexposure Prophylaxis Schedule, United States

Vaccination Status	Treatment	Regimen*
Not previously vaccinated	Local wound cleansing	All postexposure treatment should begin with immediate thorough cleansing of all wounds with soap and water
	HRIG	20 IU/kg of body weight; if anatomically feasible, up to half the dose should be infiltrated around wounds and rest administered IM in gluteal area; HRIG should not be administered in same syringe or into same anatomic site as vaccine; because HRIG may partially suppress active production of antibody, no more than recommended dose should be given
	Vaccine	HDCV or RVA, 1 mL, IM (**deltoid area**), one each on days 0, 3, 7, 14, and 28
Previously vaccinated‡	Local wound cleansing	All postexposure treatment should begin with immediate thorough cleansing of all wounds with soap and water
	HRIG	HRIG should not be administered
	Vaccine	HDCV or RVA, 1 mL, IM (deltoid area†), one each on days 0 and 3

HDCV, human diploid cell rabies vaccine; HRIG, human rabies immune globulin; RVA, rabies vaccine, adsorbed.

From the Recommendations of the Immunization Practices Advisory Committee, MMWR 40(RR-3):1, 1991.

*These regimens are applicable for all age groups, including children.
‡Any person with a history of preexposure vaccination with HDCV or RVA, prior postexposure prophylaxis with HDCV or RVA, or previous vaccination with any other type of rabies vaccine and a

documented history of antibody response to the prior vaccination.
†The deltoid area is the only acceptable site of vaccination for adults and older children. For younger children, the outer aspect of the thigh may be used. Vaccine should never be administered in the gluteal area.

patients may bathe with sutures in place, prolonged immersion in water should be avoided. Undiluted hydrogen peroxide may theoretically destroy granulation tissue and newly formed epithelium, and it should not be repeatedly used as a cleaning agent on the healing wound itself. [49] Generally, a wound should be protected with a dressing during the first week, and the dressing should be changed daily. If the wound is unlikely to be contaminated or traumatized, it may be left uncovered.

Many clinicians wrongly admonish patients against getting sutured wounds wet, and prohibit bathing, prompting some patients to keep the original dressing in place for inordinate

amounts of time. Although widely prohibited, there is no evidence that swimming in uncontaminated water with sutures in place delays healing or promotes infection. There is no evidence that gently cleaning a sutured wound even within a few hours of closure adversely impacts infection or healing rates. [149] It is generally recommended that uncovered scalp wounds can be washed after 1 to 2 days, but many clinicians advise showering the same day. Vigorous scrubbing of wounds should be discouraged. The skin surrounding a wound should be gently cleaned in the ED after wound closure to minimize subsequent itching caused by dried blood.

Some wounds heal with wide, unattractive scars despite ideal management and closure. Wounds more likely to have significant scars are those that cross perpendicular to joints, wrinkle lines, or lines of minimum tension (Kraissel lines); that retract >5 mm; and that are over convexities or in certain anatomic locations (e.g., anterior upper chest, back, shoulders) where hypertrophic scars are common. A wound crossing a concave surface may result in a bowstring deformity; one crossing a convexity may leave a scar depression. To avoid these complications, a Z-plasty procedure can be done at the time of initial wound management, or the scar can be revised later. The patient should be told to expect suboptimal outcomes in these situations. [37]

If an injured extremity or finger is protected by a splint, it should be left undisturbed until the sutures are removed. Patients with intraoral lacerations should be instructed to use warm salt water mouth rinses at least three times a day.

Patients may ask about the efficacy of various creams and lotions (e.g., vitamin E, aloe vera, cocoa butter) in limiting scar formation. At this time there are no data to evaluate the use of these substances. Patients should be told to avoid aspirin, as it has been shown to decrease the development of tensile strength and increase the likelihood of hematoma formation. [151]

SECONDARY WOUND CARE

Reexamination

Patients with simple sutured wounds may be released with appropriate instructions for home care and told to return for suture removal at an appropriate time. High-risk wounds should be examined in 2 to 3 days for signs of infection. All wounds should be inspected if the patient experiences increasing discomfort or develops a fever, or believes that the wound is infected. [95] Bite wounds and other infection-prone wounds should be inspected in 2 days. Wounds being considered for delayed primary closure are evaluated in 4 to 5 days. [87A]

Wounds in which extensive dissection of SQ tissue has been performed may develop an intense inflammation similar in appearance to a low-grade, localized cellulitis. It is rarely necessary to open these wounds. The removal of one or two stitches may relieve some of the tension caused by mild swelling. With daily cleansing using water and a mild soap and with application of warm compresses, this type of wound reaction should subside within 24 to 48 hours. [95]

A wound that has become infected should be evaluated for the presence of a retained foreign body.

Also, in most sutured wounds that become infected, the sutures must be removed to allow drainage. If a wound exhibits a minor infection, a few sutures, or all of them, may be removed, but grossly infected wounds should be packed open to allow for further drainage. The presence of sutures in a contaminated wound considerably limits the activity of various antibiotics. [152] Infection around a suture can lead to the formation of a stitch mark. [75] Infected wounds should be treated with daily cleansing, warm compresses, and antibiotics. Wounds that have been opened should be left to heal by secondary intention, which involves wound contraction, granulation tissue formation, and epithelialization.

Suture Removal

Sutures are usually removed by medical personnel, but reliable patients can be given the appropriate materials and instructions to remove simple interrupted sutures themselves. Because wounds do not heal at a standard rate, no strict guidelines can be set for time of suture removal. The optimal time for suture removal varies with the location of the wound, rate of wound healing, and amount of tension on the wound. Certain areas of the body such as the back of the hand heal slowly, whereas facial or scalp wounds heal rapidly. Speed of wound healing is affected by systemic factors such as malnutrition, neoplasia, or immunosuppression. At the time that suture removal is being considered, one or two sutures may be cut to determine whether the skin edges are sufficiently adherent to allow removal of all the sutures. [6] Removing sutures too early invites wound dehiscence and widening of the scar, whereas leaving sutures in longer than necessary may result in epithelial tracts, infection, and unsightly scarring. [153]

Small stitch abscesses are common in wounds in which sutures remain more than 7 to 10 days. Localized stitch abscesses generally resolve following removal of the sutures and application of warm compresses. There is usually no need for antibiotic therapy with localized stitch abscesses.

Percutaneous sutures stimulate an inflammatory reaction along the suture track. Factors that determine the severity of stitch marks include the length of time skin stitches are left in place, skin tension, the relationship of the suture to the wound edge, the region of the body, infection, and tendency for keloid formation. [75] [154] The skin of the eyelids, palms, and soles and the mucous membranes seldom show stitch marks. In contrast, oily skin and the skin of the back, the sternal area, the upper arms, the lower extremities, the dorsum of the nose, and the forehead are likely to develop the permanent imprints of suture material on the skin surface. [75]

If sutures are removed within 7 days, generally no discernible needle puncture or stitch mark will persist. [154] However, at 6 days, the wound is held together by a small amount of fibrin and cells and has minimal strength (see Fig. 35-1).[90] The tensile strength of most wounds at this time is adequate to hold the wound edges together, but only if there are no appreciable dynamic or static skin forces pulling the wound apart. [6] Minimal trauma to an unsupported wound at this point could cause dehiscence. The clinician should decide on the proper time of suture removal after weighing these various factors. If early suture removal is necessary, wound repair can be maintained with strips of surgical skin tape. The key to wound tensile strength after suture removal is an adequate deep tissue layer closure.

There are some general guidelines for suture removal. Sutures on the face should be removed on the fifth day following the injury, or alternate sutures should be removed

on the third day and the remainder on the fifth day. On the extremities and the anterior aspect of the trunk, sutures should be left in place for approximately 7 days to prevent wound disruption. Sutures on the scalp, back, feet, and hands and over the joints must remain in place for 10 to 14 days, even though permanent stitch marks may result.[75] Some clinicians recommend the removal of sutures in eyelid lacerations in 48 to 72 hours to avoid epithelialization along the suture tract, with subsequent cyst formation.[155]

Removing sutures is relatively simple and the removal technique is not known to affect infection rates or scarring. The wound should be cleansed, and any remaining crust overlying the wound surface or surrounding the sutures should be removed. The skin is wiped with an alcohol swab. Each stitch is cut with a scissors or the tip of a No. 11 scalpel blade at a point close to the skin surface on one side. The suture is grasped on the opposite side with forceps and is pulled across the wound (Fig. 35-28). The amount of exposed suture dragged through the suture tract is thereby minimized. It is difficult to remove sutures with very short ends. At the time of suture placement, the length of the suture ends should generally equal the distance between sutures to permit easy grasping of the suture during subsequent removal yet avoiding entanglement during the knotting of adjacent sutures.

Once the skin sutures are removed, the width of the scar increases gradually over the next 3 to 5 weeks unless it is supported. Support is provided by previously placed SQ stitches that brought the skin edges into apposition, by a previously placed subcuticular stitch, or by the application of skin tape (Fig. 35-29). A nonabsorbable subcuticular suture can be left in place for 2 to 3 weeks to provide continued support for the wound. Although complications such as closed epithelial sinuses, cysts, or internal tracts can occur from prolonged use of this stitch, they are unusual and can be avoided by the placement of a buried subcuticular stitch using an absorbable suture.[19]

If a subcuticular stitch with reliefs has been used, the suture is cut at the midpoint of the relief. Half of the suture is removed at the original point of entry into the skin and the other half through the original exit point (Fig. 35-30).[156] If a nonabsorbable subcuticular suture cannot be removed or a portion of it ruptures during removal, the protruding end should be grasped with a hemostat, pulled taut, and cut with scissors as close to the skin as possible so that the end of the suture retracts under the skin.

If time and effort have been invested in a cosmetic closure of the face, the repair should be protected with skin tape after the skin sutures have been removed. Wound contraction and scar widening continue for 42 days after the injury. [90] Because the desired result is a scar of minimal width, the tape

Figure 35-28 Technique for suture removal. Pull should be toward the wound line *(A)* rather than away from it *(B),* which causes the wound to tear apart. *(Modified from Stuzin J, Engrav LH, Buehler PK: Emergency treatment of facial lacerations. Postgrad Med 71:81, 1982.)*

should be used for 5 weeks following suture removal. With exposure to sunlight, scars in their first 4 months redden to a greater extent than surrounding skin. In

exposed cosmetic areas and when prolonged exposure to the sun is anticipated, this should be prevented with the use of a sunscreen containing para-amino benzoic acid (PABA).

COMPLICATIONS

Infection is probably the most common cause of dehiscence. If the patient is careless or unlucky, reinjury can reopen a wound despite the protection of a thick dressing. If the suture size is too small, the stitch may break. A stitch that is too fine or tied too tightly may cut through friable tissue and pull out. Knots that have not been tied carefully may unravel. The suture material may be extruded or absorbed too rapidly. Finally, if a stitch is removed too early (i.e., before tissues regain adequate tensile strength), the wound loses needed support and falls open. If the wound edges show signs of separating at the time of suture removal, alternate stitches can be left in place and the entire length of the wound supported by strips of adhesive tape.

There are several reasons why wounds fail to heal; some are related to decisions made at the time of wound closure, and others are consequences of later events. Some of the impediments to healing include ischemia or necrosis of tissue, hematoma formation, prolonged inflammation caused by foreign material, excessive tension on skin edges, and immunocompromising systemic factors. In attempting to repair wounds, clinicians sometimes inadvertently retard the healing process (e.g., with premature closure of contaminated wounds). With the development of new methods and solutions for cleansing wounds and the discovery of the optimal concentrations of solutions currently in use, tissue-toxic antiseptic solutions can be abandoned. Better suture materials are replacing the reactive sutures that often served as foreign bodies rather than tissue supports. Improved materials used for dressing wounds enhance wound healing.

A primary cause of delayed healing is wound infection. Wound cleaning and debridement, atraumatic and aseptic handling of tissues, and the use of protective dressings minimize this complication. Inversion of the edges of a wound during closure produces a more noticeable scar, whereas skillful technique can convert a jagged, contaminated wound into a fine, inapparent scar. However, the patient's actions also affect wound healing. Delay in seeking treatment for an injury may significantly affect the ultimate outcome of the wound. Furthermore, in the first few days following an injury, the

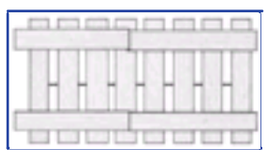

Figure 35-29 Support of the wound with surgical tape.

patient must take responsibility for protecting the wound from contamination, further trauma, and swelling.

The final appearance of a scar is determined by several factors. Infection, tissue necrosis, and keloid formation widen a scar. Wounds located in sebaceous skin or oriented 90° to dynamic or static skin tension lines result in wide scars.

Miscellaneous Aspects of Wound Care

Many parameters and scenarios are involved in the preparation for repair and definitive treatment of wounds. Many specific questions are discussed in other sections of this text but a few specific caveats are included below for completeness.

Figure 35-30 At the time of suture removal, the suture is cut at the midpoint of the relief *(A)*. The proximal portion is removed at the point of original entry into the skin *(B)*, and the distal portion is removed through the original exit point *(C)*. *(From Grimes DW, Garner RW: "Reliefs" in intracuticular sutures. Surg Rounds 1:48, 1978. Reproduced by permission.)*

Digital nerves.

The key to the best possible outcome of a digital nerve injury is making, or suspecting, the diagnosis at the time of initial presentation. Usually injury to a digital nerve is obvious, but numerous factors will hinder the timely diagnosis, and the presence or extent of nerve injury may not be immediately discernible in the ED. Numbness in the area of digital innervation, concomitant injury to a digital artery (flash/pulsating bleeding), or an electric shock sensation when exploring a laceration should alert the clinician to a possible digital nerve injury. When the presence of a digital nerve injury is in question, a follow-up visit usually confirms or eliminates this injury, so it is not critical that all decisions be made at the first visit. With regard to wound preparation issues, lacerations to the hand and fingers should be approached with caution. Debridement should be minimal, and wound preparation should be gentle yet meticulous. Digital nerves that are transected distal to the metacarpophalangeal joint may be candidates for surgical repair. It is not known exactly how far distal in the finger can the nerve be lacerated for a repair to be successful, and proper referral is therefore essential. Often injuries proximal to the distal inter-phalangeal joint are not repaired, but many other factors will influence operative decisions. Repair of a digital nerve will frequently result in return of good sensory function (but it takes months) and repair can prevent painful neuromas from developing. Most hand surgeons will not repair digital nerves at the time of initial presentation. Instead, they advise wound cleaning, skin closure, splinting, and outpatient follow-up in 24 to 36 hours, followed by delayed nerve repair.

Animal bites.

The use of prophylactic antibiotics is discussed in a previous section of this chapter. However, many aspects of the treatment of animal bites are controversial and no universal standards exist. Most bites are caused by dogs or cats, with most being sustained from family pets. Numerous organisms will accompany a bite from a dog or cat. If a bite appears clinically grossly infected within the first 24 hours, the offending organism is usually the gram-negative rod *Pasteurella multocida*. If the infection appears later than 24 hours, a host of bacteria, but predominantly *Staphylococcus aureus* and *Streptococcus viridans*, are the culprits. *Pasteurella* infections are common in cat bites. Cat bites, probably because they are puncture wounds that can not be completely cleaned, frequently become infected. The incidence of infection following dog bite lacerations is not significantly greater than lacerations in general. Many clinicians have advocated the primary closure of large dog bite lacerations. Markedly contused lacerations are good candidates for delayed primary closure. Wound cultures taken at the time of an animal bite are worthless. The use of prophylactic antibiotics for animal bites is controversial and various approaches are advocated (see earlier discussion). The best way to approach bite wounds is to adhere to the general principles and details of wound care as outlined in earlier discussions. No specific intervention has been demonstrated to be superior for the preparation of bite wounds. Care should be taken to search for underlying fractures or tooth fragments in deep animal bites.

Gunshot wounds.

A certain subset of gunshot wounds may be definitively handled in the ED, with outpatient follow-up. The landmark studies by Ordog et al. [157] [158] document a very low infection rate in gunshot wounds treated on an outpatient basis. In a retrospective study of nearly 17,000 subjects, the vast majority of patients did well, with only a 1.8% infection rate. Standard wound care was given and

prophylactic antibiotics were withheld, and satisfactory results were obtained even when the missile was left in place, and minor fractures were present (Fig. 35-31). Since most gunshot wounds are puncture wounds, minimal deep wound cleaning is possible.

Puncture wounds.

The approach to specific puncture wounds is discussed in other sections, but a few caveats are repeated here. As a general rule, it is impossible to completely clean a puncture wound. In fact, it may be counterproductive to attempt to do so in some areas of the body. Superficial soft tissue wounds having entrance and exit wounds in proximity may be debrided by passage of a sterile gauze back and forth through the wound tract. Coring out a puncture wound is usually overly aggressive initial treatment, but it may be an option if gross contamination or infection is present. In selected punctures, it may be possible to incise the skin and cutaneous tissue over the tract, converting a puncture into a linear laceration. If a through-and-through stream cannot be established, attempting to irrigate the tract of a puncture

Figure 35-31 Minor gunshot wounds may be treated as outpatients, even when bullet fragments remain and there are *minor* fractures. *A* and *B*, This through-and-through injury transversed the hypothenar eminence. No bullet remained and no bones were involved. *C*, Usually it is impossible to irrigate a puncture wound, but in this case note the saline at the exit site. *D*, After the entrance wound is debrided of the powder burn, a hemostat is passed through the wound. *E*, The instrument grasps gauze packing and pulls it into the wound. The gauze was pulled back and forth to debride the wound tract, and then a clean gauze was left in place. No antibiotics were given, the pack was removed at wound check in 24 hours, and the patient did well. Many gunshot wounds cannot be irrigated to this extent but the treatment principles are similar.

wound by inserting a needle into the depths of the wound and forcibly injecting irrigating solution has the potential to disseminate contamination and increase soft tissue swelling, and is discouraged. If gross contamination remains in a puncture wound, it is unlikely that antibiotics will prevent or totally treat an infection. This leaves the clinician with the reality that many puncture wounds usually do quite well with minimal intervention, while others do quite poorly because of their inaccessibility to wound cleaning techniques. Simply stated, it may be impossible to predict the outcome of most puncture wounds on the first encounter. The key to success with any puncture wound is to acknowledge the issues discussed earlier, relay them to the patient, and provide the necessary follow-up.

CONCLUSION

The objective of traumatic wound management is the restoration of tissue continuity and strength in the least possible

time, with maximal preservation of tissues and minimal scar formation, deformity, or loss of function.

It is important that clinicians follow the basic principles of wound care when cleaning, debriding, closing, and protecting wounds and continue to refine their management of wounds as further improvements in techniques and materials become available.

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Chapter 36 - Methods of Wound Closure

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Once the decision to close a wound has been made, the clinician must select the closure technique best suited for the location and configuration of the wound. Available techniques include hair typing (in the scalp) and use of tape, tissue adhesive, metal staples, and sutures. All traumatic wounds should be cleaned, and wounds containing devitalized tissue should be debrided before closure (see Chapter 35).

HAIR TYING OF SCALP WOUNDS

Scalp wounds that gape have traditionally been closed with suture material or skin staples. One "folk method" of scalp wound closure that has received limited discussion and study is the technique of tying together "roped" strands of hair from opposite sides of the wound. [1] [2]

The advantages of this technique are that no surgical instruments are required; no foreign material is placed in the wound; and it is relatively painless, because a local anesthetic injection is not needed. This technique may be of particular value in wilderness settings when wound repair equipment is unavailable and the wound is relatively clean. In certain superficial scalp wounds in children, this technique offers a particularly humane method of wound closure.

Indications and Contraindications

Closure by hair tying can be performed on small scalp wounds (i.e., 1 to 2 cm in length). Davies suggests the following six criteria for consideration of this technique [2] :

1. The patient's hair must be of adequate length to form "roped" strands that can be tied across the wound.
2. The wound edges should not be contused.
3. There should be no gross wound contamination.
4. There must be good wound hemostasis.
5. The galea (occipitofrontalis aponeurosis) must not be divided.
6. There must be no underlying skull fracture.

When these conditions cannot be met, the technique should not be undertaken. If local anesthesia must be used to permit evaluation of the deep structures of the wound, it may be best to simply repair the wound with sutures or staples.

Procedure

When possible, the area surrounding the wound should be cleansed with mild disinfectant, avoiding contact with the unanesthetized wound. The wound should be irrigated with normal saline. The wound should be gently explored using a gloved hand or cotton-tipped applicator to verify that the galea is not compromised and that no foreign material remains in the wound.

Hair on each side of the laceration is then twisted to form "ropes" of hair (Fig. 36-1*A*). These "roped" strands are tied across the wound in a surgical knot, with several additional throws (Fig. 36-1*B*) to tightly appose the skin edges. Davies recommends spraying a plastic sealant on the knot to avoid loosening it. [2]

Postclosure wound care is similar to that for routine scalp closure. The patient may gently shampoo the hair, but *vigorous* hair massage or combing in the area should be avoided. The knot is allowed to grow away from the wound edge and can be cut free in 2 to 4 weeks.

Complications

In 1 series of 25 children under 8 years of age whose scalp wounds were closed by hair tying, 48-hour follow-up showed no evidence of wound infection and 2 cases of mild (2 to 4 mm) wound separation.[2] The investigators noted that some children complained of the sensation that their hair was "being pulled" during wound closure, but all cooperated without restraints or anesthesia. The most common complaint noted at follow-up was that the hair-tie knot was untidy. There is little control over apposition of wound edges with this technique.

Conclusion

Closure of scalp wounds by hair tying offers an alternative for closure of small, superficial scalp wounds in children and for clean scalp wound repair in wilderness settings.

WOUND TAPE

The use of surgical tape strips to close simple wounds has become routine in recent years. Tape strips can be applied by health care personnel in many settings, including emergency departments (EDs), operating rooms, clinics, and first-aid stations. Advantages include ease of application, reduced need for local anesthesia, more evenly distributed wound tension, no residual suture marks, minimal skin reaction, no need for suture removal, superiority for some grafts and flaps, and suitability for use under plaster casts. One main advantage of wound tapes is their greater resistance to wound infection compared with standard sutures and wound staples.[3] [4] [5] [6]

Background and Tape Comparisons

Tape closure of wounds has been reported since 1600 BC.[7] It was not until the late 1950s, however, with the introduction of woven tapes and nonsensitizing adhesive, that tapes gained widespread acceptance in the United States.[8] Since then, there have been rapid advances in the manufacture of tapes with increased strength, improved adhesiveness, and presterilized packaging.

Currently there are several brands of tapes with differing porosity, flexibility, strength, and configuration. Steri-Strips (3M Corporation, St. Paul, MN) are microporous tapes with ribbed backing. They are porous to air and water, and the ribbed backing provides extra strength. Cover-Strips (Beiersdorf, South Norwalk, CT) are woven in texture and have a high degree of porosity. They allow not only air and water, but also wound exudates to pass through the tape. Shur-Strip (Deknatel, Inc., Floral Park, NY) is a nonwoven microporous tape. Clearon (Ethicon, Inc., Somerville, NJ) is a synthetic plastic tape whose backing contains longitudinal parallel serrations to

Figure 36-1 *A,* Hair on each side of a laceration is twisted to form "ropes" of hair. *B,* The "roped" strands are tied across the wound in a surgical knot along with additional throws to oppose the skin edges.

permit gas and fluid permeability. An iodoform-impregnated Steri-Strip (3M Corporation) is intended to further retard infection without sensitization to iodine.[3] Other tape products include Curi-Strip (Kendall, Boston), Nichi-Strip (Nichiban Co., Ltd, Tokyo), Cicagraf (Smith & Nephew, London), and Suture Strip (Genetic Laboratories, St. Paul, MN).

Scientific studies of wound closure tapes have been limited, and because of different investigators' choices of products and methods, it is not always easy to compare results. Koehn showed that the Steri-Strip tapes maintained adhesiveness about 50% longer than Clearon tape.[9] Rodeheaver and coworkers compared Shur-Strip, Steri-Strip, and Clearon tape in terms of breaking strength, elongation, shear adhesion, and air porosity.[10] The tapes were tested in both dry and wet conditions. The Steri-Strip tape was found to have about twice the breaking strength of the other two tapes in both dry and wet conditions; there was minimal loss of strength in all tapes when wetted. The Shur-Strip tapes showed approximately two to three times the elongation of the other tapes at the breaking point, whether dry or wet. Shear adhesion (amount of force required to dislodge the tape when a load is applied in the place of contact (angle = 0°) was slightly better for the Shur-Strip tape than for the Steri-Strip tape and approximately 50% better than for the Clearon tape. Of these three wound tapes, the investigators considered Shur-Strips to be superior for wound closure.

One comprehensive study of wound tapes compared Curi-Strip, Steri-Strip, Nichi-Strip, Cicagraf, Suture Strip, and Suture Strip Plus.[11] All tapes were 12 mm wide except for Nichi-Strip, which was 15 mm. Each tape was compared for breaking strength, elongation under stress, air porosity, and adhesiveness. Curi-Strip, Cicagraf, and Steri-Strip exhibited equivalent dry breaking strengths. However, when wet (a condition that can occur in the clinical setting), Cicagraf outperformed all tapes. All of the tested tapes had similar elongation-under-stress profiles with the exception of Suture Strip Plus. This tape did not resist elongation under low or high forces. Excessive elongation may allow wound dehiscence. Nichi-Strip was the most porous to air, and Cicagraf was almost vapor impermeable. Nichi-Strip and Curi-Strip had the best adherence to untreated skin. When the skin was treated with tincture of benzoin, however, Steri-Strip dramatically outperformed all other products. When all of the study parameters were considered, Nichi-Strip, Curi-Strip, and Steri-Strip achieved the highest overall performance rankings.

Indications

The primary indication for tape closure is a superficial straight laceration under little tension. If necessary, tension can be reduced by undermining or placing deep closures. Areas particularly suited for tape closure are the forehead, chin, malar eminence, thorax, and nonjoint areas of the extremities. Tape also may be preferred for wounds in anxious children when suture placement is not essential. In young children who are likely to remove tapes, tape closures must be protected with an overlying bandage. However, adhesive bandages (e.g., Band-Aids) should be avoided (see later).

In experimental wounds inoculated with *Staphylococcus aureus*, tape-closed wounds resisted infection better than wounds closed with nylon sutures.[6] Therefore, tape closures may be considered on wounds with potential for infection, although infection rates are generally comparable to those of sutured wounds. Tape closures work well under plaster casts when superficial suture removal would be delayed. Tape closures effectively hold flaps and grafts in place, particularly over fingers, the flat areas of the extremities, and the trunk (Fig. 36-2).[3] [4] Wounds on the pretibial area are difficult to close. This area is particularly problematic in the elderly because of tissue atrophy. One report found that wound tapes outperformed suture closure of the pretibial area with regard to time to healing and complications.[12] Tape closures can be applied to wounds following early suture removal to maintain wound edge approximation while reducing the chance of permanent suture mark scarring. Finally, because of the minimal skin tension created by tapes, they can be used on skin that has been compromised by vascular insufficiency or altered by prolonged use of steroids.

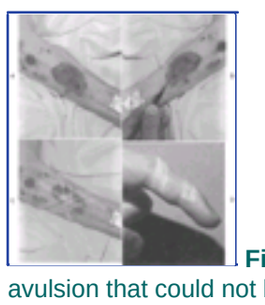

Figure 36-2 *A,* A skin avulsion is an ideal wound to close with closure tapes. An elderly woman who was on steroids had extremely thin skin and suffered an anterior tibial skin avulsion that could not be replaced with sutures. *B,* The skin edges are uncurled, stretched, and anatomically replaced. *C,* The wound should heal when closure tapes keep the skin in place. A compression dressing, such as an elastic bandage or a Dome paste (Unna) boot dressing, can be applied to minimize flap movement and decrease fluid buildup under the flap. *D,* Tape should be placed in a semicircular or spiral pattern on digits to avoid constriction.

Contraindications

There are disadvantages to tape closures. Tape does not work well on wounds under significant tension or on wounds that are irregular, on concave surfaces, or in

areas of marked tissue laxity. In many cases tape does not provide satisfactory wound edge apposition without concurrent underlying deep closures. Tape does not stick well to naturally moist areas, such as in the axilla, the palms of the hands, the soles of the feet, and the perineum. Tape also has difficulty adhering to wounds that will have copious exudates. Tape strips are also at risk for premature removal by young children.

Tape closures are contraindicated in wounds that are irregular or under tension and in those that cannot be appropriately dried of blood or secretions. They are of little value on lax and intertriginous skin and in the scalp and other areas with high concentration of hair follicles.

Tapes should never be placed circumferentially around digits because they have insufficient ability to stretch or lengthen. If placed circumferentially, the natural wound edema of an injured digit can make the tape act like a constricting band, which can lead to ischemia and possible necrosis of the digit. Semicircular or spiral placement techniques should be used if digits are to be taped (Fig. 36-2*C*).

Equipment

For a simple tape closure, the required equipment includes forceps and tape of the proper size. Most taping can be done in the ED with ¼-inch × 3-inch strips. In wounds larger than 4 cm, however, ½-inch-wide strips might be desirable. Most companies manufacture strips up to 1 inch wide and up to 4 inches long.

Procedure

Application of the tape must be preceded by proper wound preparation, irrigation, debridement, and hemostasis. Fine hair may be cut short or shaved, and the area of the tape application *is thoroughly dried to ensure proper adhesion*. Attempting to apply tapes to a wet area or over a wound that is slowly oozing blood will usually result in failure of the tapes to stick to the skin. On fingers, tapes can be applied to a wound that is kept dry by a tourniquet temporarily placed at the base of the finger.

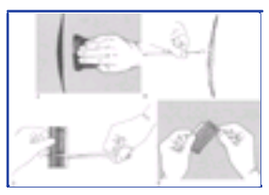

Figure 36-3a Proper technique for application of tapes for skin closure. *A,* After wound preparation (and placement of deep closures, if needed), dry the skin thoroughly at least 2 inches around the wound. Failure to dry the skin and failure to obtain perfect hemostasis are common causes of failure of tapes to stick to the skin. *B,* If desired, apply a thin coating of tincture of benzoin around the wound to enhance tape adhesiveness. Benzoin should not enter the eye, and it causes pain if it seeps into an open wound. *C,* Cut the tapes to the desired length before removing the backing. *D,* The tapes are attached to a card with perforated tabs on both ends. Gently peel the end tab from the tapes.

Tincture of benzoin and Mastisol are liquid adhesives that can be applied initially to increase tape adhesion. [6] The clinician should use sterile technique at all times. Wound tapes do not adhere unduly to surgical gloves. All tapes come in presterilized packages and can be opened directly onto the operating field.

The technique of applying tapes is shown in Figure 36-3*A–J* Figure 36-3*A–J* . After the wound has been dried and a liquid adhesive has been applied and has dried, the tapes, with backing attached, are cut to the desired length. Tapes should be long enough to allow for approximately 2 to 3 cm of overlap on each side of the wound. After the tape is cut to length, the end tab is removed. The tape is gently removed from the backing with forceps by pulling straight back. Do not pull to the side because the tape will curl and be difficult to apply to the wound. One half of the tape is securely placed at the midportion of the wound. The opposite wound edge is gently but firmly apposed to its counterpart. The second half of the tape is then applied. The wound edges should be as close together as possible and at equal height to prevent the development of a linear, pitted scar. Additional tapes are applied by bisecting the remainder of the wound. A sufficient number of tape strips should be placed so that the wound is completely apposed without totally occluding the wound edges. Finally, additional cross tapes are placed to add support and prevent blistering caused by unsupported tape ends. [5]

Taped wounds are left open, without occlusive dressings. Adhesive bandages (e.g., Band-Aids) and other dressings promote excessive moisture, which can lead to premature tape separation from the wound. The bandage also may adhere to the closure tapes, causing separation of the closure tape from the skin at the time of the removal. Tapes may remain in place for approximately 2 weeks, longer in some cases. The duration of application is a decision that varies with the requirements of each wound. The patient can be allowed to clean the taped laceration gently with a moist, soft cloth after 24 to 48 hours. However, if excessive wetting or mechanical force is used, premature separation may result. Patients may be instructed to gently trim curled edges of the closure tape with fine scissors to avoid premature removal of the tape.

Complications

Complications are uncommon with tape closure. The infection rate is approximately 5% in clean wounds closed with tape. [5] This compares favorably with rates for other standard

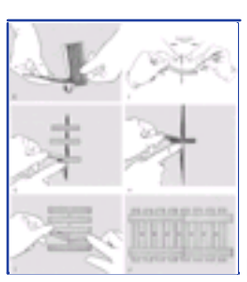

Figure 36-3b *E,* Use forceps to peel the tape off the card backing. Pull directly backward, not to the side. *F,* Place one half of the first tape at the midportion of the wound; secure firmly in place. *G,* Gently but firmly oppose the opposite side of the wound, using the free hand or forceps. If an assistant is not available, the operator can approximate the wound edges. *H,* The tape should be applied by bisecting the wound until the wound is closed satisfactorily. *I,* Wound margins are completely opposed without totally occluding the wound. *J,* Additional supporting tapes are placed approximately 2.5 cm from the wound and parallel to the wound direction. Taping in this manner prevents the skin blistering that may occur at tape ends.

closures. Premature tape separation occurs in approximately 3% of cases. [10] Other complications include (1) skin blistering, which occurs if the tape is not properly anchored with the cross-stay strip or the tape is stretched excessively across the wound; and (2) wound hematoma, which results if hemostasis is inadequate.

When tincture of benzoin is used, it should be applied carefully to the surrounding uninjured skin. If spillage occurs into the wound, the wound is at higher risk for infection.[13] Benzoin vapors cause pain when applied near an open wound that has not been anesthetized. Benzoin can also injure the mucous membranes of the eye.

Summary

Most investigators believe that the results of proper tape closure are as successful as those of suture closure. [5] [7] However, some investigators believe that tape closure leads to inferior cosmetic results. [14] In the aggregate, modern tape products and techniques serve a valuable role in minor wound

management of selected patients in the ED. Generally, closure tapes are underused, and many wounds that are currently sutured in cosmetically unimportant areas could be adequately closed with tapes. As a general guide, tapes should be considered in those cases where sutures are not clearly required, but the wound is too wide for a simple dressing.

TISSUE ADHESIVE

Adhesive tape can only be used on superficial wounds on relatively flat, hairless body surfaces. The tissue adhesive *N*-2-octylcyanoacrylate (Dermabond, Ethicon Inc.) is a bonding agent that can be used on superficial wounds, even in hair-bearing areas. Tissue adhesive (also called *tissue glue*) polymerizes when it comes in contact with water. This substance is biodegradable but remains in the wound until well after healing. [15]

Procedure

Tissue adhesive can be used to approximate wounds not requiring deep-layer closure. In preparation for closure, the wound should be anesthetized and cleaned, and when necessary, debrided. Bleeding must be controlled.

As the wound edges are held together with forceps or fingers, a small, cylindrical plastic container is squeezed to expel droplets of tissue glue through a cotton applicator tip at the end of the container. The glue is applied in at least 3 to 4 thin layers along the length of the wound's surface (Fig. 36-4). Alternatively, one can place the glue in strips perpendicular to the laceration (analogous to placement of closure tapes). The purple color of the solution facilitates placement of the droplets. The wound edges should be supported with edges approximated for at least 1 minute while the glue dries. The closure can be reinforced and protected with a bandage such as Elastoplast.

The primary advantage of tissue glue is the speed of closure. Wounds can be closed in as little as one sixth of the time required for repair with sutures. Application is rapid and painless. Wounds closed with tissue glue have less tensile strength than sutured wounds in the first 4 days [16] [17] but 1 week after closure the tensile strength and overall degree of inflammation in wounds closed with tissue glue were equivalent to those closed with sutures. [15] [18] Cosmetic results are similar to those obtained with suture repair. [17] [19] [20] [21] [22] [23] [24] [25]

Tissue glue serves as its own wound dressing and has an antimicrobial effect against gram-positive organisms. [26] [27] The material sloughs off in 7 to 10 days, thereby saving the patient from a clinician visit. Ointments or occlusive bandages should not be placed on wounds closed with tissue glue.

Complications

Percutaneous sutures provide a more secure immediate closure than tissue glue. [15] Although tissue glue is classified as nontoxic and does not cause a significant foreign body reaction, it should not be placed within the wound cavity. [17] [18] If the wound edges cannot be held together without considerable tension, tissue glue should not be used.[25] Tissue glue should not be used near the eyes, over or near joints, on moist or mucosal surfaces, or on wounds under significant static or dynamic skin tension. After polymerizing, tissue glue can

Figure 36-4 Tissue adhesive, 2-octyl cyanoacrylate in a commercially available application dispenser.

fracture with excessive or repetitive movement. Although gentle rinsing is permitted, if the adhesive is washed or soaked, it will peel off in a few days, before the wound is healed.[17] If hemostasis is inadequate or an excessive amount of glue is applied too quickly, the patient can experience a burning sensation from the heat of polymerization.

One risk involving the use of tissue glue is its ease of use. Clinicians may fail to adequately clean wounds before closure with tissue glue. [27A] Tissue glue should not be used to close infected wounds. If the clinician's gloved fingers contact the tissue glue during application, the glove may adhere to the patient's skin. Tissue glue can be removed with antibiotic ointment, petrolatum jelly, or more rapidly with acetone. [25]

Summary

Tissue glue has been approved for use in the United States since 1998. Dermabond is packaged in sterile, single-use ampules. It is best suited for superficial wounds that are under little tension and that do not require prolonged support. Cosmetic results are generally equivalent to sutured wounds when used properly. 276

WOUND STAPLES

Background

Wound stapling devices date back to the early part of this century. Several Russian, Hungarian, and Japanese investigators pioneered various instruments, but it was not until the early 1960s that significant interest in the use of these devices developed in the United States. [28] [29] Since then there has been a steady improvement in technology, including the introduction of automatic and disposable devices, precocking mechanisms, and optimal staple configurations.

Automatic stapling devices have become commonplace for closure of surgical incisions and are finding acceptance among clinicians for closure of traumatic wounds. Clinical studies of patients with stapled surgical incisions have consistently revealed that there is no significant difference between stapling and suturing when infection rates, healing outcome,

and patient acceptance are compared.[30] [31] [32] [33] [34] Four important studies have demonstrated that selected traumatic wounds in both adult and pediatric patients can be closed successfully with staples in the ED setting. [35] [36] [37] Wound stapling and nylon suture closure of skin compared favorably in wound tensile strength, complication rates, patient tolerance, efficiency of closure, scar width, color, general appearance, suture or staple marks, infection rates, and cost. However, in one study more patients in the staple group reported discomfort with removal. [37] In animal models, staples cause less wound inflammation, preserve wound defense mechanisms, and offer more resistance to infection in contaminated wounds.[38] [39] [40] [41]

The most significant advantage of wound stapling over suturing is speed of closure. On average, stapling is three to four times faster than suturing traumatic wounds.[35] [37] [42] The time for actual staple application is =30 seconds for a laceration 3 to 5 cm in length. [43] [44] [45] Cost has been cited as a disadvantage of staple closure, particularly when large, multistaple (25 to 35) surgical units are the only product available. [43] However, with the introduction of smaller devices more appropriate for the average laceration, the cost of stapling devices has been reduced significantly. [43] When clinician time and cost of instruments are considered, the cost difference is minimal[35] or favors stapling.[46]

Indications and Contraindications

Currently the indications for stapling are limited to relatively linear lacerations with straight, sharp edges located on an extremity, the trunk, or the scalp. Staples may be especially useful for superficial scalp lacerations in the agitated or intoxicated patient. Because of their superficial placement in the adult scalp (usually above the galea), staples are *not* ideal for *deep* scalp lacerations. Staples may not provide the same hemostasis that is possible with deep sutures. Also, they should not be placed in scalp wounds if computed tomography head scans are to be performed because staples produce scan artifacts. Similarly, staples should not be used if the patient is expected to undergo magnetic resonance imaging, because the powerful magnetic fields may avulse the staples from the skin surface. As they are currently configured and manufactured, staples *should not be used on the face, neck, hands, or feet*.

Equipment

Standard wound care should precede wound closure (see Chapter 35). In many cases, when debridement and dermal (deep) closures are unnecessary, only tissue forceps are needed to assist in everting wounds.

Many stapling devices are commercially available. The most versatile and least expensive stapler is the Precise (3M Corporation). Different units that hold between 5 and 25 staples can be purchased. The 10-staple unit will suffice for most lacerations. Other devices include the Proximate 11 (Ethicon, Inc.), Cricket (US Surgical, Irvine, CA), and Appose (Davis & Geck, Columbus, OH). These staplers have a minimum of 15 staples and are 3 to 5 times more expensive than the Precise stapler.

Procedure

The wound is prepared in the manner described in Chapter 35 . Whenever necessary, deep, absorbable sutures are used to close deep fascia and to reduce tension in the superficial fascia and dermal layers. Before stapling, the wound edges should be everted, preferably by a second operator. The assistant precedes the operator along the wound and everts the wound edges with forceps or pinches the skin with the thumb and forefinger. This technique allows the staple to be precisely placed. Once the edges are held in eversion, the staple points are gently placed across the wound (Fig. 36-5). By squeezing the stapler handle or trigger, the staple is advanced automatically into the wound and bent to the proper configuration (Fig. 36-6 and Fig. 36-7). One must take care not to press too hard on the skin surface in order to prevent placing the staple too deeply and causing ischemia within the staple loop. When properly placed, the *crossbar of the staple is elevated a few millimeters above the skin surface* (Fig. 36-8). Enough staples should be placed to provide proper apposition of the edges of the wound along its entire length.

After the wound is stapled, an antibiotic ointment may be applied to minimize dressing adherence, and a sterile dressing is applied. If necessary, the patient can remove the dressing and gently clean the wound in 24 to 48 hours.

Removal of staples requires a special instrument that is made available by each manufacturer of stapling devices. The lower jaw of the staple remover is placed under the crossbar (Fig. 36-9). One brings down the upper jaw by squeezing the handle (Fig. 36-10). This action compresses the crossbar, thereby releasing the staple points for easy removal. If the patient is referred for office removal of staples, it may be advisable to provide the patient with the staple removal device on ED release because many clinicians do not routinely stock the instrument. The interval between staple application and removal is the same as that for standard suture placement and removal.

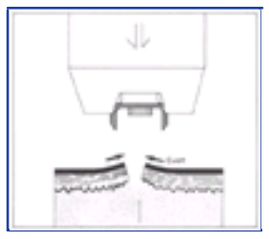

Figure 36-5 The skin edges must be approximated and everted by hand or with forceps before they are secured with staples. Failure to evert the wound edges is a common error that may cause an unacceptable result. *(Adapted with permission from Edlich RF: A Manual for Wound Closure. St. Paul, MN, 3M Medical-Surgical Products, 1979. Reproduced by permission.)*

Figure 36-6 By squeezing the stapler handle, a plunger advances one staple into the wound margins. *(From Edlich RF: A Manual for Wound Closure. St. Paul, MN, 3M*

Medical-Surgical Products, 1979. Reproduced by permission.)

Complications

Complications can occur with staple-closed wounds, although the incidence is low and equivalent to that for sutured wounds. In 2 studies of traumatic wounds closed with staples, the infection rates were reported to be 0% and 5%.[37] [43] Staple acceptance and comfort have been reported to be equal to those of sutures, but in one study, removal of staples was somewhat more uncomfortable than removal of sutures.[37] Wound dehiscence has been reported, but the incidence is not considered significant. [37] A common error is failure to *evert* the skin edges before stapling (Fig. 36-11*A* and *B*). Eversion avoids the natural tendency of the device to invert the closure. Eversion may be accomplished with forceps or by pinching the skin with the thumb and index finger, a procedure that requires some practice. Staples do cause marks in the skin similar to sutures. In patients who tend to scar more easily, the resulting scar from the staples may be more pronounced than that produced by sutures, especially if the staples are left in place for prolonged periods.

Figure 36-7 An anvil automatically bends the staple to the proper configuration. *(From Edlich RF: A Manual for Wound Closure. St. Paul, MN, 3M Medical-Surgical Products, 1979. Reproduced by permission.)*

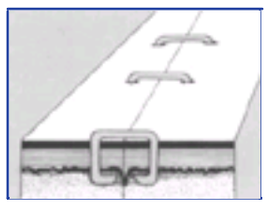

Figure 36-8 Care should be taken to ensure that a space remains between the skin and the crossbar of the staple. Excessive pressure created by placing the staple too deep causes wound edge ischemia, as well as pain on removal. Note that the staple bar is 2 to 3 mm above the skin line. *(From Edlich RF: A Manual for Wound Closure. St. Paul, MN, 3M Medical-Surgical Products, 1979. Reproduced by permission.)*

Conclusion

Overall results are favorable when staples are used for surgical incisions and traumatic lacerations of the scalp, trunk, and extremities. Wound stapling does not differ significantly from suturing in infection rates, wound healing, and patient acceptance. Stapling is clearly superior in reducing time to closure. With the introduction of new devices, the cost of wound stapling is comparable to that of suturing. Because of the increased availability and versatility of stapling instruments, they are being used more frequently in ED wound management.

SUTURES

In most situations, suturing is the closure method of choice. Currently in the United States, most traumatic wounds are closed with sutures.

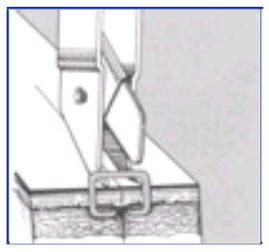

Figure 36-9 The lower jaw of the staple remover is placed under the crossbar of the staple. *(From Edlich RF: A Manual for Wound Closure. St. Paul, MN, 3M Medical-Surgical Products, 1979. Reproduced by permission.)*

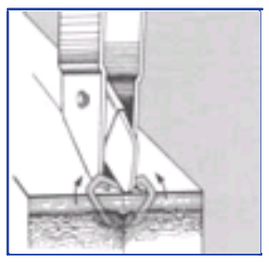

Figure 36-10 By squeezing the handle gently, the upper jaw compresses the staple and allows it to exit the skin. *(Adapted from Edlich RF: A Manual for Wound Closure. St. Paul, MN, 3M Medical-Surgical Products, 1979. Reproduced by permission.)*

Equipment

Instruments

In addition to the instruments used for debridement, a needle holder and suture scissors are required for suturing. The size of the needle holder should match the size of the needle selected for suturing—that is, the needle holder should be large enough to hold the needle securely as it is passed through tissue, yet not so large that the needle is crushed or bent by the instrument. The mechanical performance of disposable needle holders distributed by different surgical instrument companies varies considerably.[47] Instruments used to debride a grossly contaminated wound should be discarded

Figure 36-11 A very poor result occurred when staples (*some marked with arrows*) were used to close this deep scalp laceration (*A*). The wound edges were not everted (in fact, the skin overlapped significantly), and poor hemostasis was obtained because the galea was not closed by the superficial staples. Three days later during a wound check, the staples were removed, and the laceration was closed with 3-0 interrupted nylon sutures (*B*). The clinician should attempt to obtain a cosmetic closure on all scalp lacerations, because as patients lose their hair, a previously hidden, unsightly scar emerges. In general, staples should not be used to close full-thickness scalp lacerations, especially wounds that are actively bleeding.

and fresh instruments obtained for the closure of the wound. Instruments covered with coagulated blood can be cleansed with hydrogen peroxide, rinsed with sterile saline or water, and then used for suturing.

Suture Materials

A wide variety of suture materials are available. For most wounds that require closure of more than one layer of tissue, the clinician must choose sutures from two general categories: an absorbable suture for the subcutaneous (SQ) layer and a nonabsorbable suture for skin closure.

Sutures can be described in terms of four characteristics:

1. Composition (i.e., chemical and physical properties)
2. Handling characteristics and mechanical performance
3. Absorption and reactivity
4. Size and retention of tensile strength

Composition.

Sutures are made from natural fibers (cotton, silk), from sheep submucosa or beef serosa (plain gut, chromic gut), or from synthetic materials such as nylon (Dermalon, Ethilon, Nurulon, Surgilon), Dacron (Ethiflex, Mersilene), polyester (Ti-Cron), polyethylene (Ethibond), polypropylene (Prolene, Surgilene), polyglycolic acid (Dexon), and polyglactin (Vicryl, coated Vicryl). Stainless steel sutures are rarely, if ever, useful in wound closure in the ED setting because of handling difficulty and fragmentation. Some sutures are made of a single filament (monofilament); others consist of multiple fibers braided together (Table 36-1).[48]

Handling and performance.

Desirable handling characteristics in a suture include smooth passage through tissues, ease in knot tying, and stability of the knot once tied (Table 36-2). Smooth sutures pull through tissues easily, but knots slip more readily. Conversely, sutures with a high coefficient of friction have better knot-holding capacity but are difficult to slide through tissues. Smooth sutures will loosen after the first throw of a knot is made, and a second throw is needed to secure the first in place. However, the clinician may

TABLE 36-1 -- Examples of Suture Materials

Absorbable Sutures	Nonabsorbable Sutures
Monofilament	
Plain gut	Dermalon (nylon)
Chromic gut	Ethilon (nylon)

PDS (polydioxanone)	Prolene (polypropylene)
Maxon (polyglyconate)	Silk
	Steel
	Surgilene (polypropylene)
	Tevdek (Teflon coated)
Multifilament	
Dexon (polyglycolic acid)	Ethibond (polyethylene)
Coated Vicryl (polyglactin)	Mersilene (braided polyester)
	Nurulon (nylon)
	Surgilon (nylon)
	TiCron (polyester)

want to tighten a knot further after the first throw is made. This is difficult with rougher types of sutures.

Multifilament sutures have the best handling characteristics of all sutures, whereas steel sutures have the worst. In terms of performance and handling, significant improvements have been made in the newer absorbable sutures. Gut sutures have many shortcomings, including relatively low and variable strength, a tendency to fray when handled, and stiffness despite being packaged in a softening fluid. [49] [50] Multifilament synthetic absorbable sutures are soft and easy to tie and have few problems with knot slippage. Polyglactin 910 (coated Vicryl) sutures have an absorbable lubricant coating. The "frictional drag" of these coated sutures as they are pulled through tissues is less than that of uncoated multifilament materials, and the resetting of knots following the initial throw is much easier. This characteristic allows retightening of a ligature without knotting or breakage and with smooth, even adjustment of suture line tension in running subcuticular stitches. [51] Synthetic monofilament sutures have the trouble-some property of "memory"—a tendency of the filament to spring back to its original shape, which causes the knot to slip and unravel. Some nonabsorbable monofilament sutures are coated with polytetrafluorethylene (Teflon) or silicone to reduce their friction. This coating improves the handling characteristics of these monofilaments but results in poorer knot security. [50]

TABLE 36-2 -- Characteristics of Suture Materials

Suture Material Handling	Knot Security	Tensile Strength	Tissue Reactivity	Duration of Suture Integrity (days)	Tie Ability
Absorbable					
Surgical gut	poor	fair	greatest	5–7	poor
Chromic gut	fair	fair	greatest	10–14	poor
Coated Vicryl	good	good	minimal	30	best
Dexon	best	good	minimal	30	best
PDS	fair	best	least	45–60	good
Maxon	fair	best	least	45–60	good
Nonabsorbable					
Ethilon	good	good	minimal		good
Prolene	least	best	least		fair
Silk	best	least	greatest		best

Modified with permission from Hollander J, Singer A: Laceration management. Ann Emerg Med 34:361, 1999.

Three square knots will secure a stitch made with silk or other braided, nonabsorbable materials, and four knots are sufficient for synthetic, absorbable and nonabsorbable monofilament sutures. [52] Five knots are needed for the Teflon-coated synthetic Tevdek. [53] With the use of coated synthetic suture materials, attention to basic principles of knot tying is even more important. An excessive number of throws in a knot weakens the suture at the knot. If the clinician uses square knots (or a surgeon's knot on the initial throw, followed by square knots) that lie down flat and are tied securely, knots will rarely unravel. [54]

Absorption and reactivity.

Sutures that are rapidly degraded in tissues are termed *absorbable*; those that maintain their tensile strength for >60 days are considered *nonabsorbable* (see Table 36-1). Plain gut may be digested by white blood cell lysozymes in 10 to 40 days; chromic gut will last 15 to 60 days. Remnants of both types of sutures, however, have been seen in wounds more than 2 years after their placement. [49] [52] [55] The Ethicon catgut is rapidly absorbed within 10 to 14 days but with less inflammation than that caused by chromic catgut. [56] Vicryl is absorbed from the wound site within 60 to 90 days [49] [52] and Dexon, within 120 to 210 days. [57] [58] When placed in the oral cavity, plain gut disappears after 3 to 5 days, chromic gut after 7 to 10 days, and polyglycolic acid after 16 to 20 days. [59] In contrast, SQ silk may not be completely absorbed for as long as 2 years. [52] The rate of absorption of synthetic absorbable sutures is independent of suture size. [57]

Sutures may lose strength and function before they are completely absorbed in tissues. Braided synthetic absorbable sutures lose nearly all of their strength after about 21 days. In contrast, monofilament absorbable sutures (modified polyglycolic acid [Maxon, Davis & Geck] and polydioxanone [PDS, Ethicon]) retain 60% of their strength after 28 days. [60] [61] Gut sutures treated with chromium salts (chromic gut) have a prolonged tensile strength; however, all gut sutures retain tensile strength erratically. [49] [52] Of the absorbable types of sutures, a wet and knotted polyglycolic acid suture is stronger than a plain or chromic gut suture subjected to the same conditions. [50] [62]

Polypropylene remains unchanged in tissue for longer than 2 years after implantation. [63] In comparison testing, Hermann found that sutures made of natural fibers such as silk, cotton, and gut were the weakest; sutures made of Dacron, nylon, polyethylene, and polypropylene were intermediate in tensile strength; and metallic sutures were the

strongest. [50] Kaplan and Hentz used the comparison of suture strength vs wound strength as a measure of the usefulness of a suture. They stated that catgut is stronger than the soft tissue of a wound for no more than 7 days; chromic catgut, Dexon, and Vicryl are stronger for 10 to 21 days; and nylon, wire, and silk are stronger for 20 to 30 days. [64]

All sutures placed within tissue will damage host defenses and provoke inflammation. Even the least reactive suture impairs the ability of the wound to resist infection. [63] The magnitude of the reaction provoked by a suture is related to the quantity of suture material (diameter × total length) placed in the tissue and to the chemical composition of the suture. Among absorbable sutures, polyglycolic acid and polyglactin sutures are least reactive, followed by chromic gut. Nonabsorbable polypropylene is less reactive than nylon or Dacron. [50] [65] [66] Significant tissue reaction is associated with catgut, silk, and cotton sutures; highly reactive materials should be avoided in contaminated wounds. Adams found absorbable polyglycolic acid sutures to be less reactive than those of nonabsorbable silk. [67]

The chemical composition of sutures is an important determinant of early infection. The infection rate in experimental wounds when polyglycolic acid sutures are used

is less than the rate when gut sutures are used. It is surprising that plain gut sutures elicit infection less often in contaminated wounds than chromic gut sutures. [63] Lubricant coatings on sutures do not alter suture reactivity, absorption characteristics, breaking strength, or the risk of infection. [51] [63] Multifilament sutures provoke more inflammation and are more likely to produce infection than monofilament sutures if left in place for prolonged periods. [68] [69] Monofilament sutures elicit less tissue reaction than do multifilament sutures, and multifilament materials tend to wick up fluid by capillary action. Bacteria that adhere to and colonize sutures can envelop themselves in a glycocalix that protects them from host defenses,[70] or they can "hide" in the interstices of a multifilament suture and, as a result, be inaccessible to leukocytes.[68] Polydioxanone (PDS) provides the advantages of a monofilament suture in an absorbable form, making it a good choice as a subcuticular stitch. Polypropylene sutures have a low coefficient of friction, and subcuticular stitches with this material are easy to pull out. [71]

Size and strength.

Size of suture material (thread diameter) is a measure of the tensile strength of the suture; threads of greater diameter are stronger. The strength of the suture is proportional to the square of the diameter of the thread. Therefore, a 4-0 size suture of any type is larger and stronger than a 6-0 suture. The correct suture size for approximation of a layer of tissue depends on the tensile strength of that tissue. The tensile strength of the suture material should be only slightly greater than that of the tissue, because the magnitude of damage to local tissue defenses is proportional to the amount of suture material placed in the wound. [52] [72]

Synthetic absorbable sutures have made the older, natural suture materials obsolete. Polyglycolic acid (Dexon) and polyglactin 910 (coated Vicryl) have improved handling characteristics, knot security, and tensile strength. Their absorption rates are predictable, and tissue reactivity is minimal. [73] [74] The distinct advantages of synthetic nonabsorbable sutures over silk sutures are their greater tensile strength, low coefficient of friction, and minimal tissue reactivity. [63] [73] They are extensible, elongating without breaking as the edges of the wound swell in the early postoperative period. [72] [73] In contrast with silk sutures, synthetics can be easily and painlessly removed once the wound has healed. The monofilament synthetic suture Novofil has elasticity that allows a stitch to enlarge with wound edema and to return to its original length once the edema subsides. Stiffer materials lacerate the encircled tissue as the wound swells. [75]

The suture materials most useful to emergency clinicians for wound closure are Dexon or coated Vicryl for SQ layers and synthetic nonabsorbable sutures (e.g., nylon or polypropylene) for skin closure. Fascia can be sutured with either absorbable or nonabsorbable materials. In most situations, 3-0 or 4-0 sutures are used in the repair of fascia, 4-0 or 5-0 absorbable sutures in SQ closure, and 4-0 or 5-0 nonabsorbable sutures in skin closure. Lips, eyelids, and the skin layer of facial wounds are repaired with 6-0 sutures, whereas 3-0 or 4-0 sutures are used when the skin edges are subjected to considerable dynamic stresses (e.g., wounds overlying joint surfaces) or static stresses (e.g., scalp).

Needles

The eyeless, or "swaged," needle is used for wound closure in most emergency centers (Fig. 36-12). The traditional closed-eye needle requires additional handling to enable one to thread the needle with the suture, and its increased width causes more damage when passing through tissue than does a swaged needle.

Selection of the appropriate needle size and curvature are based on the dimensions of the wound and the characteristics of the tissues to be sutured. The needle should be large enough to pass through tissue to the desired depth and then to exit the tissue or the skin surface far enough that the needle holder can be repositioned on the distal end of the needle at a safe distance from the needle point (Fig. 36-13). While it is inviting to use the fingers to grasp the needle tip to pull the needle through the skin, this practice is an invitation for a needle stick. The clinician should either reposition the needle holder or use forceps to disengage the needle from the laceration.

In wound repair, needles must penetrate tough, fibrous tissues—skin, SQ tissue, and fascia—yet should slice through these tissues with minimal resistance or trauma and without bending. The type of needle best suited for closure of SQ tissue is a *conventional cutting needle* in a three-eighths or one-half circle (Fig. 36-14). The use of double curvature needles (coated Vicryl with PS-4-C cutting needles, Ethicon) may enhance the clinician's ability to maneuver the needle in narrow, deep wounds. For percutaneous closure, a conventional cutting-edge needle may

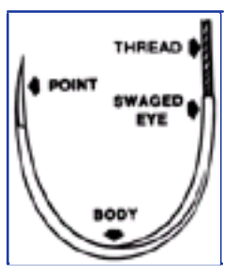

Figure 36-12 The eyeless, or "swaged," needle. *(From Suture Use Manual: Use and Handling of Sutures and Needles. Somerville, NJ, Ethicon, Inc., 1977, p 29. Reproduced by permission.)*


Figure 36-13 The needle should be large enough to pass through tissue and should exit far enough to enable the needle holder to be repositioned on the end of the needle at a safe distance from the point.

permit more precise needle placement and require less penetration force (Fig. 36-15).[76] [77]

Suturing Techniques

Skin Preparation

Before closing the wound, the skin surrounding it is prepared with a povidone-iodine solution and covered with sterile drapes. Some surgeons do not drape the face but prefer to leave facial structures and landmarks adjacent to the wound uncovered and within view. A clear plastic drape (Steri-Drape, 3M Corporation) can be used to provide a sterile field and a limited view of the area surrounding the wound. If no drapes are used on the face, the skin surrounding the wound should be widely cleansed and prepared. Wrapping the hair in a sheet prevents stray hair from falling into the operating field (Fig. 36-16). Some EDs keep a supply of oversized scrub hats to use as an alternative to wrapping.

Closure Principles

Three principles apply to the suturing of lacerations in any location: (1) minimize trauma to tissues, (2) relieve tension exerted on the wound edges by undermining and layered wound closure, and (3) accurately realign landmarks and skin edges by layered closure and precise suture placement.

Minimizing tissue trauma.

The importance of careful handling of tissue has been emphasized since the early days of surgery. Skin and SQ tissue that has been stretched, twisted, or crushed by an instrument or strangled by a suture that is tied too tightly may undergo necrosis, and increased scarring and infection may result. When the edges of a wound must be manipulated, the SQ tissues should be lifted gently with a toothed forceps or skin hook, avoiding the skin surface.

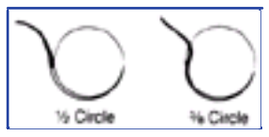

Figure 36-14 One-half and three-eighths circle needles, used for most traumatic wound closures.

When choosing suture sizes, the clinician should select the smallest size that will hold the tissues in place. Skin stitches should incorporate no more tissue than is needed to coapt the wound edges with little or no tension. Knots should be tied securely enough to approximate the wound edges but without blanching or indenting the skin surface.[78]

Relieving tension.

Many forces can produce tension on the suture line of a reapproximated wound. Static skin forces that stretch the skin over bones cause the edges of a fresh wound to gape and also continuously pull on the edges of the wound once it has been closed. Traumatic loss of tissue or wide excision of a wound may have the same effect. The best cosmetic result occurs when the long axis of a wound happens to be parallel to the direction of maximal skin tension; this alignment brings the edges of the wound together.[75]

Muscles pulling at right angles to the axis of the wound impose dynamic stresses. Swelling following an injury creates additional tension within the circle of each suture.[78] Skin suture marks result not only from tying sutures too tightly, but also from failing to eliminate underlying forces distorting the wound. Tension can be reduced during wound closure in two ways: undermining of the wound edges and layered closure.


Figure 36-15 Types of needles. *A,* The conventional cutting needle has two opposing cutting edges, with a third edge on the inside curvature of the needle. The conventional cutting needle changes in cross section from a triangular cutting tip to a flattened body. *B,* The reverse cutting needle is used to cut through tough, difficult-to-penetrate tissues, such as fascia and skin. It has two opposing cutting edges, with the third cutting edge on the outer curvature of the needle. The reverse cutting needle is made with the triangular shape extending from the point to the swage area, with only the edges near the tip being sharpened. *(From Suture Use Manual: Use and Handling of Sutures and Needles. Somerville, NJ, Ethicon, Inc., 1977, p 31. Reproduced by permission.)*


Figure 36-16 *A–D,* Technique for wrapping the scalp to keep stray hair from falling into the operating field. A scrub hat is an acceptable alternative.

Undermining.

The force required to reapproximate the wound edges correlates with the subsequent width of the scar.[79] Wounds subject to significant static tension require the undermining of at least one tissue plane on both sides of the wound to achieve a tension-free closure. Undermining involves the creation of a flap of tissue freed from its base at a distance from the wound edge approximately equal to the width of the gap that the laceration presents at its widest point (Fig. 36-17). The depth of the incision can be modified, depending on the orientation of the laceration to skin tension lines and the laxity of skin in the area. A No. 15 scalpel blade held parallel to the skin surface is used to incise the adipose layer or the dermal layer of the wound. The clinician also can accomplish this technique by spreading scissors in the appropriate tissue plane. Undermining allows the skin edges to be lifted and brought together with gentle traction.[80] Because undermining may harm the underlying blood supply, this technique should be reserved for relatively uncontaminated wounds.[76] Other potential complications of this procedure include injury to cutaneous nerves and creation of a hematoma under the flap.[64]

Layered closure.

The structure of skin and soft tissue varies with the location on the body (Fig. 36-18*A–D* Fig. 36-18*A–D*). Most wounds handled in an ED require approximation of no more than three layers: fascia (and associated muscle), SQ tissue, and skin surface (papillary layer of dermis and epidermis).[81]

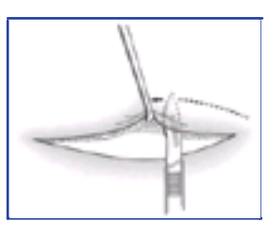
Figure 36-17 The technique of undermining. The scalpel is used to find an appropriate site; a natural plane often exists at the epidermis-dermis junction. Undermining relieves tension on the wound and renders a better cosmetic result. This technique is simple to master, but sometimes overlooked.

Closure of individual layers obliterates "dead space" within the wound that would otherwise fill with blood or exudate. The presence of dead space enhances the development of infection; however, it is not necessary to close the adipose layer of soft tissue with a separate stitch. A "fat stitch" is not necessary, because little support is provided by closure of the adipose layer, and the additional suture material that is required may enhance the possibility of infection.[6] [82]

Separate approximation of muscle and SQ layers hastens the healing and return of function to the muscle. However, *one should suture fascia, not muscle*. Muscle tissue itself is too friable to hold a suture. Layered closure is particularly important in the management of facial wounds; this technique prevents scarring of muscle to the SQ tissue and consequent deformation of the surface of the wound with contraction of the muscle. If a deep, gaping wound is closed without approximation of underlying SQ tissue, a disfiguring depression may develop at the site of the wound. Finally, layered closure provides support to the wound and considerably reduces tension at the skin surface.

There are exceptions to the general rule of multilayered closure. Scalp wounds are generally closed in a single layer. For lacerations penetrating the dermis in fingers, hands, toes, and feet, the amount of SQ tissue is too small to warrant layered closure; in fact, SQ stitches may leave tender nodules in these sensitive locations. In the sebaceous skin of the nasal tip, SQ sutures should be avoided, because they provoke inflammation and increase the risk of infection. However, deep sutures do not increase the risk of infection in minimally contaminated wounds.[83] Layered closure is not recommended in wounds without tension, those with poor vascularity, and those with moderate infection potential. With single-layer closure, the surface stitch should be placed more deeply.[64]

Suture Placement

Before suturing, the clinician should ensure adequate exposure and illumination of the wound. The clinician should assume a comfortable standing or sitting position, with the patient placed at an appropriate height. The best position for

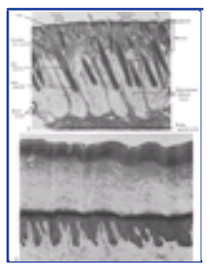

Figure 36-18a Variation in the structure of skin. *A,* Section of the skin of the scalp, ×15. *B,* Skin of the human fingertip, illustrating a very thick stratum corneum. Hematoxylin and eosin, ×65.

the clinician is at one end of the long axis of the wound. Knot construction using the instrument tie technique is described in other references. [84]

SQ layer closure.

Once fascial structures have been reapproximated, the SQ layer is sutured. Although histologically the fatty and fibrous SQ tissue (hypodermis) is an extension of (and is continuous with) the reticular layer of the dermis, [85] suturing of these layers is traditionally referred to as an "SQ closure." One approach is to close this layer in segments, placing the first stitch in the middle of the wound and bisecting each subsequent segment until the closure of the layer has been completed. [48] This technique is useful in the closure of wounds that are long or sinuous and is particularly effective in wounds with one elliptic and one linear side. The needle is grasped by the needle holder close to the suture end. Greater speed in suturing is possible if the fingers are placed

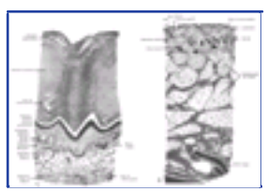

Figure 36-18b *C,* Section of human sole perpendicular to the free surface, ×100. *D,* Section through human thigh perpendicular to the surface of the skin. Blood vessels are injected and appear black. Low magnification. *(A Courtesy of H Mizoguchi. C and D after AA Maximow. From Bloom W, Fawcett DW: A Textbook of Histology, 10th ed. Philadelphia, WB Saunders, 1975. Reproduced by permission.)*

on the midshaft of the needle holder rather than in the rings of the instrument (Fig. 36-19).

The suture enters the SQ layer at the bottom of the wound (Fig. 36-20*A*) or, if the wound has been undermined, at the base of the flap (Fig. 36-20*B*), and exits in the dermis. Once the suture has been placed on one side of the wound, it can be pulled across the wound to the opposite side (or the wound edges pushed together) to determine the matching point on the opposite side. It is at this matching point along the opposite side of the wound that the needle is inserted. The needle should enter the dermis at the same depth as it exited from the opposite side, pass through the tissue, and exit at the bottom of the wound (or the base of the flap). The edges of the wound can be closely apposed by pulling the two tails of the suture in the same direction along the axis of the wound (Fig. 36-21). Some clinicians place their SQ suture obliquely rather than vertically to facilitate knot tying. When the knot in this SQ stitch is tied, it will remain inverted, or "buried," at the bottom of the wound. Burying the knot of the SQ stitch avoids a painful, palpable nodule beneath the epidermis and keeps the bulk of this foreign material away from the skin surface. The techniques of tying knots by hand and by instrument are well described and illustrated in wound care texts. [86] [87] Once the knot has been secured, the tails of the suture should be pulled taut for cutting. The scissors are held with the index finger on the junction of the two blades. The blade of the scissors is slid down the tail of the suture until the knot is reached. With the cutting edge of the blade tilted away from the knot, the tails are cut. This technique prevents the scissors from cutting the knot itself and leaves a tail of 3 mm, which protects the knot from unraveling (Fig. 36-22). [88] The entire SQ layer is sutured in this manner.

Figure 36-19 The thenar grip technique of handling the needle holder. The index finger is placed on the side of the needle holder, where it guides the placement of the needle. Neither the index nor the middle finger is placed in the ringlet hole. An alternate method (the thumb-ring finger grip) is shown in Figure 36-27 .

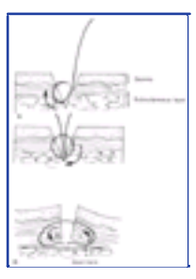

Figure 36-20 *A* and *B,* Inverted subcutaneous stitches.

After the SQ layer has been closed, the distance between the skin edges indicates the approximate width of the scar in its final form. If this width is acceptable, percutaneous sutures can be inserted. [89] Despite undermining and placement of a sufficient number of SQ sutures, on rare occasions a large gap between the wound edges may persist. In such cases a horizontal dermal stitch may be used to bridge this gap (Fig. 36-23).

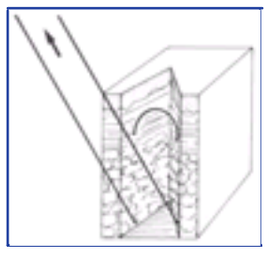

Figure 36-21 The two tails of the subcutaneous suture are pulled in the same direction, tightly opposing the edges of the wound.

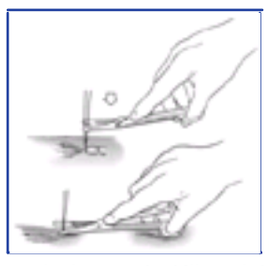

Figure 36-22 Cutting the tails of the subcutaneous suture. Note that the cutting blade is tilted away from the knot to avoid cutting it. *(Modified from Anderson CB: Basic surgical techniques. In Klippel AP, Anderson CB (eds): Manual of Outpatient and Emergency Surgical Techniques. Boston, Little, Brown, 1979. Reproduced by permission.)*

Skin closure.

The epidermis and the superficial layer of dermis are sutured with nonabsorbable synthetic sutures. The choice of suture size, the number of sutures used, and the depth of suture placement depend on the amount of skin tension remaining after SQ closure. If the edges of the wound are apposed following closure of deeper layers, small 5-0 or 6-0 sutures can be used simply to match the epithelium of each side. If the wound edges remain retracted or if SQ stitches were not used, a larger size suture may be required. Skin closure may be accomplished with sutures placed in segments (Fig. 36-24) or from end to end. Either technique is acceptable.

Unless the wound edges are uneven, sutures should be placed in a mirror-image fashion such that *the depth and width are the same on both sides of the wound*.[52] In general, the distance between each suture should be approximately equal to the distance from the exit of the stitch to the wound edge. [48] [86] Grabb suggests that "the number of sutures used in closing any wound will vary with the case, location of the repair, and degree of accuracy required by the clinician and patient. In an area such as the face, sutures would probably be placed between 1 and 3 mm apart and 1 to 2 mm from the wound edge."[65]

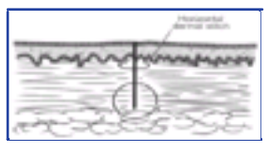

Figure 36-23 Horizontal dermal stitch. (A vertical suture also closes the deep tissue.)

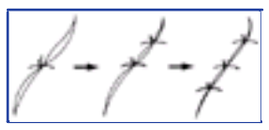

Figure 36-24 Closure of the surface of the wound in segments.

Sutures act as foreign bodies in a wound, and any stitch may damage a blood vessel or strangulate tissue. Therefore, the clinician should strive to use the smallest size and the least number of sutures that will adequately close the wound (Fig. 36-25). [63] Wounds with greater tension should have skin stitches placed closer to each other and closer to the wound edge; layered closure is important in such wounds. If sutures are tied too tightly around wound edges or if individual stitches are under excessive tension, blood supply to the wound may be impeded, increasing the chance of infection, and suture marks may form even after 24 hours. [52] [86A]

When suturing the skin, right-handed operators should pass the needle from the right side of the wound to the left. The needle should enter the skin at an oblique angle to produce an everting, bottle-shaped stitch that is *deeper than it is wide* (Fig. 36-26). If the skin stitch is intended to produce some eversion of the wound edges, the stitch must include a sufficient amount of SQ tissue. However, encompassing too much tissue with a small needle is a common error. Forcefully pushing or twisting the needle in an effort to bring the point out of the tissue may bend or break the body of the needle. Using a needle of improper size will defeat the best suturing technique. The needle should be driven through tissue by flexing the wrist and supinating the forearm; the course taken by the needle should result in a curve identical to the curvature of the needle itself (Fig. 36-27). The angle of exit for the needle should be the same as its angle of entrance so that an identical volume of tissue is contained within the stitch on each side of the wound.

Once the needle exits the skin on the opposite side of the wound, it is regrasped by the needle holder and is advanced through the tissue; care should be taken to avoid crushing the

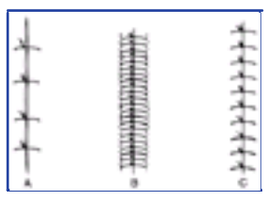

Figure 36-25 *A,* Too few stitches used. Note gaping between sutures. *B,* Too many stitches used. *C,* Correct number of stitches used for a wound under an average amount of tension.

point of the needle with the instrument. Forceps are designed for handling tissue and thus should not be used to grasp the needle. The forceps can stabilize the needle by holding the needle within the tissue through which the needle has just passed. An assistant can keep excess thread clear of the area being sutured, or the excess can be looped around the clinician's fingers. If the point of the needle becomes dulled before all of the attached thread has been used, the suture should be discarded.

If these techniques are applied to most wounds, the edges of the wound will be matched precisely in all three dimensions.

Eversion techniques.

If the edges of a wound invert or if one edge rolls under the opposite side, a poorly formed, deep, noticeable scar will result. Excessive eversion that exposes the dermis of both sides also will result in a larger scar than if the skin edges are perfectly apposed, but inversion produces a more visible scar than does eversion. Because most scars undergo some flattening with contraction, optimal results are achieved when the epidermis is slightly everted without excessive suture tension (Fig. 36-28). Wounds over mobile surfaces, such as the extensor surfaces of joints, should be everted; in time, the scar will be flattened by the dynamic forces acting in the area.

Numerous techniques can be used to avoid inversion of the edges of the wound. If the clinician angles the needle away from the laceration, percutaneous stitches can be placed so that their depth is greater than their width. [80] Converse described this method as follows: "The needle penetrates the skin close to the incision line, diverging from the edge of the wound in order to encircle a larger amount of tissue in the lower depths of the skin than at the periphery." [90] The edge of the wound can be lifted and everted with a skin hook or fine-tooth forceps before insertion of the needle on each side (Fig. 36-29). Eversion can also be obtained simply by slight retraction of the wound with the thumb (Fig. 36-30). This technique puts the operator at risk for a needle stick; eversion may be done more safely by applying slight pressure on the wound edge with a closed forceps. Each of these methods also serves to steady the skin against the force of the needle. [86] [90] Vertical mattress sutures are particularly effective in everting the wound edges and can be used exclusively or alternated with simple interrupted sutures (Fig. 36-31).[90] In wounds that have been undermined, an SQ stitch placed at the base of the flap on each side can in itself evert the wound (Fig. 36-32 (Figure Not Available)).

Interrupted stitch.

The simple interrupted stitch is the most frequently used technique in the closure of skin. It consists of separate loops of suture individually tied. Although the tying and cutting of each stitch are time consuming, the advantage of this method is that if one stitch in the closure fails, the remaining stitches continue to hold the wound together (Fig. 36-33).

Continuous stitch.

In a continuous, or "running," stitch, the loops are the exposed portions of a helical coil tied at each end of the wound. A continuous suture line can be placed more rapidly than a series of interrupted stitches. The continuous stitch has the additional advantages of strength (with tension being evenly distributed along its entire length), fewer knots (which are the weak points of stitches), and more effective hemostasis. This stitch will accommodate mild wound swelling. The continuous technique is useful as an epithelial or "surface" stitch in cosmetic closures; however, if the underlying SQ layer is not stabilized in a separate closure, the continuous surface stitch tends to invert the wound edges.

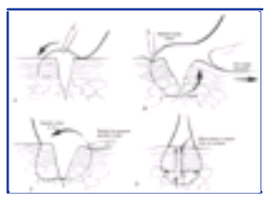

Figure 36-26 The simple suture. *A,* Hold the needle pointing down by excessively pronating the wrist so that the needle tip initially moves farther from the laceration as the needle penetrates deeper into the skin. Thus, there is more dermis in the depth of the wound than at the surface. Drive the needle tip downward and away from the cut edge, into the fat. *B,* Advance the needle into the laceration. The needle tip can be advanced directly into the opposite side. This can be achieved by rolling the needle holder as the needle enters the opposite side at the same level, and the arc pathway of the needle is controlled by retracting the skin edge. This causes more dermis to be incorporated into the depths than at the surface. As an alternative, if a small needle is used in thick skin or the distance across the wound is great, the needle can be removed from the first side, remounted on the needle holder, and advanced to the opposite side. *C,* Advance the needle upward toward the surface so that it exits at the same distance from the wound edge as on the contralateral side of the wound. Grasp the needle behind the tip and roll it out in the arc of the needle. *D,* The final position, with more tissue in the depth than the surface. The distance from each suture exit to the laceration is one half the depth of the dermis. *(Redrawn from Kaplan EN, Hentz VR: Emergency Management of Skin and Soft Tissue Wounds: An Illustrated Guide. Boston, Little, Brown, 1984, p 86. Reproduced by permission.)*

The continuous suture technique has other disadvantages. This technique cannot be used to close wounds overlying joints. If a loop breaks at one point, the entire stitch may unravel. Likewise, if infection develops and the incision must be opened at one point, cutting a single loop may allow the entire wound to fall open. There is also the theoretical problem of impeded blood supply to the wound edges, particularly if the suture is interlocked. [52] Speer found that wounds closed with an interrupted stitch had 30% to 50% greater tensile strength, less edema and induration, and less impairment in the microcirculation at the wound margin than did wounds closed with a continuous stitch.[91] The simple continuous stitch has a tendency to produce suture marks if used in large wound closures and if left in place for more than 5 days.[78] However, if all tension on the wound can be removed by SQ sutures, stitch marks are seldom a problem.

Among the variations of the continuous technique, the simple continuous stitch is the most useful to emergency clinicians (Fig. 36-34). An interrupted stitch is placed at one end of the wound, and only the free tail of the suture is cut. As suturing proceeds, the stitch encircles tissue in a spiral pattern. After each passage of the needle, the loop is tightened slightly, and the thread is held taut in the clinician's nondominant hand. The needle should travel perpendicularly across the wound on each pass. The last loop is placed just beyond the end of the wound, and the suture is tied, with the last loop used as a "tail" in the process of tying the knot (Fig. 36-35). A locking loop may be used in continuous suturing to prevent slippage of loops as the suturing proceeds (Fig. 36-36). The interlocking technique allows the use of the continuous stitch along an irregular laceration. [80]

A continuous stitch is an effective method for closing relatively clean wounds that are under little or no tension and are on flat, immobile skin surfaces in patients who have no medical conditions that would impair healing.

Figure 36-27 Motion of the needle holder. *(From Anderson CB: Basic surgical techniques. In Klippel AP, Anderson CB (eds): Manual of Outpatient and Emergency Surgical Techniques. Boston, Little, Brown, 1979. Reproduced by permission.)*

Continuous subcuticular stitch.

Nonabsorbable sutures used in percutaneous skin closure outlast their usefulness and must be removed. On occasion, wounds require an extended period of support, longer than that provided by surface stitches. Some patients with wounds that require skin closure are unlikely or unwilling to return for suture removal. Some sutured wounds are covered by plaster casts. On occasion, the patient (child or adult) is likely to be as frightened and uncooperative for suture removal as for suture placement. The continuous subcuticular (or "dermal") suture technique is ideal for these situations; the wound can be closed with an absorbable subcuticular stitch, obviating the need for later suture removal. In patients prone to keloid formation, the subcuticular technique can be used in lieu of percutaneous stitches, and disfiguring stitch marks can thereby be avoided. (Because children's skin is under greater tension than that of adults, percutaneous sutures are more likely to produce stitch marks in children.) Because stitch marks are avoided, a nonabsorbable subcuticular suture can be left in place for a longer period than a percutaneous suture.[90]

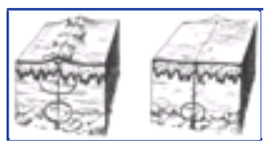

Figure 36-28 Skin edges that are everted will gradually flatten to produce a level wound surface. *(From Grabb WC: Basic technique of plastic surgery. In Grabb WC, Smith JW: Plastic Surgery: A Concise Guide to Clinical Practice. Boston, Little, Brown, 1979. Reproduced by permission.)*

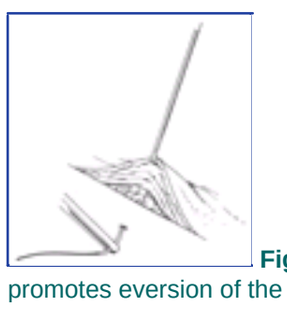

Figure 36-29 The use of a skin hook to evert the wound edge. This technique allows the operator to see the needle path, ensuring that the proper depth has been reached, and promotes eversion of the skin edges.

Although this technique is commonly used in cosmetic closures, some researchers believe that closure of the subcuticular layer alone does not alter the scar width. [92] This technique does not allow for perfect approximation of the vertical heights of the two edges of a wound [93] and in cosmetic closures it is often followed by a percutaneous stitch. Although theoretically the large amount of suture material left in the wound might increase the risk of infection, some investigators report a lower infection rate with the subcuticular technique. [92] [94] Buried, absorbable subcuticular stitches do not appear to provoke more inflammation than percutaneous running stitches with monofilament nylon.[83]

The subcuticular stitch requires a 4-0 or 5-0 suture made of either absorbable material or nonabsorbable synthetic monofilament. An absorbable suture can be "buried" within

Figure 36-30 Eversion can often be obtained by slight thumb pressure. Care should be taken to avoid a needle stick, a common complication of this technique. *(From Converse JM: Introduction to plastic surgery. In Converse JM: Reconstructive Plastic Surgery: Principles and Procedures in Correction, Reconstruction, and Transplantation, vol 1, 2nd ed. Philadelphia, WB Saunders, 1977. Reproduced by permission.)*

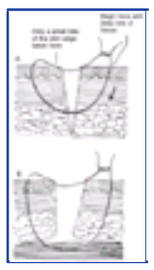

Figure 36-31 The vertical mattress suture is the best technique for producing skin edge eversion. *A,* Usual type of mattress suture for approximating and everting wound edges. *B,* "Tacking" type of vertical mattress suture, extending into deep fascia to obliterate dead space under wound. Note that only a small bite of skin is included on the inner suture. *(Modified from Converse JM: Introduction to plastic surgery. In Converse JM: Reconstructive Plastic Surgery: Principles and Procedures in Correction, Reconstruction, and Transplantation, vol 1, 2nd ed. Philadelphia, WB Saunders, 1977. Reproduced by permission.)*

Figure 36-32 (Figure Not Available) Deep dermis suturing technique. The suture enters the base of the flap, is brought up into the dermis, and exits just proximal to the wound edge along the base of the flap to be tied and cut. *(From Stuzin J, Engrav LH, Buehler PK: Emergency treatment of facial lacerations. Postgrad Med 71:81, 1982. Reproduced by permission.)*

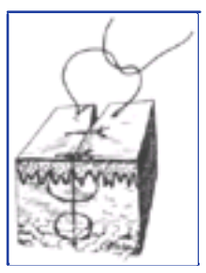

Figure 36-33 Simple interrupted stitch. Additional throws in a partially tied knot are not shown. *(From Grabb WC: Basic techniques of plastic surgery. In Grabb WC, Smith JW (eds): Plastic Surgery: A Concise Guide to Clinical Practice. Boston, Little, Brown, 1979. Reproduced by permission.)*

the wound, whereas a nonabsorbable suture is used for a "pullout" stitch. The absorbable synthetic monofilament suture polydioxanone (PDS, Ethicon) is designed for subcuticular closure. It passes through tissues as easily as nonabsorbable monofilament sutures and is absorbed if left in the wound.

Before the subcuticular stitch is placed, the SQ layer should be approximated with interrupted sutures to minimize tension on the wound. The pullout subcuticular stitch is started at the skin surface approximately 1 to 2 cm away from 1 end of the wound. The needle enters and exits the dermis at the apices of the wound (Fig. 36-37). Bites through tissue are taken in a horizontal direction, with the needle penetrating the dermis 1 to 2 mm from the skin surface. These intradermal bites should be small, of equal proportion, and at the same level on each side of the wound. [74] [90] Accidental interlocking of the stitch should be avoided. Each successive bite should be placed 1 to 2 mm *behind* the exit point on the opposite side of the wound so that when the wound is closed, the entrance and exit points on either side are not directly apposed (see

Figure 36-34 Simple continuous stitch. *(From Grabb WC: Basic techniques of plastic surgery. In Grabb WC, Smith JW (eds): Plastic Surgery: A Concise Guide to Clinical Practice. Boston, Little, Brown, 1979. Reproduced by permission.)*

Figure 36-35 Completing the simple continuous stitch. A series of square knots is tied, with the loop as one of the ties.

Fig. 36-37). Small bites should be taken to avoid puckering of the skin surface. Some clinicians prefer to place a fine (6-0) running skin suture in addition to the subcuticular suture for meticulous skin approximation. The skin suture is removed in 3 to 4 days to avoid suture marks.

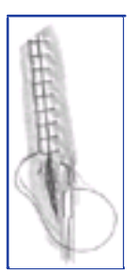

Figure 36-36 Continuous interlocking stitch. *(Modified from Suture Use Manual: Use and Handling of Sutures and Needles. Somerville, NJ, Ethicon, Inc, 1977. Reproduced by permission.)*

Figure 36-37 *A,* Pullout subcuticular stitch. The suture is introduced into the skin in line with the incision, approximately 1 to 2 cm away. *(From Grimes DW, Garner RW: "Reliefs" in intracuticular sutures. Surgical Rounds 1:46, 1978. Reproduced by permission.) B,* By backtracking each stitch slightly, one can produce a straight scar. *(From Grabb WC: Basic techniques*

of plastic surgery. In Grabb WC, Smith JW (eds): Plastic Surgery: A Concise Guide to Clinical Practice. Boston, Little, Brown, 1979. Reproduced by permission.)

If the subcuticular stitch is used on lengthy lacerations, it is difficult to remove the suture. The placement of "reliefs" consisting of periodic loops through the skin during the length of the stitch facilitates later removal (Fig. 36-38). Reliefs should be placed every 4 to 5 cm. The suture is crossed to the opposite side, and the needle is passed from SQ tissue to the skin surface. The suture is carried over the surface for approximately 2 cm before reentering the skin and SQ tissue. The subcuticular stitch is then continued at approximately the point at which the next bite would have been placed had the relief not been used.

At the completion of the stitch, the needle is placed through the apex to exit the skin 1 to 2 cm away from the end of the wound. One should tighten the stitch by pulling each end taut. If reliefs have been used, one can take up any slack in the stitch by pulling on the reliefs. The clinician can secure the two ends of the stitch by taping them to the skin surface with wound closure tape, by placing a cluster of knots on each tail close to the skin surface, or by tying the two ends of the suture to each other over a dressing. Laxity of the subcuticular stitch is often noted with a decrease in tissue swelling 48 hours after wound closure. Some clinicians tighten the stitch when they reexamine the wound after 48 hours.

Subcuticular closure using absorbable sutures that do not penetrate the skin is possible. The closure is begun with a dermal or SQ suture placed at one end of the wound and secured with a knot. After placement of the continuous subcuticular stitch from apex to apex, the suture is pulled taut, and a knot is tied using a tail and a loop of suture (Fig. 36-39). The final knot can be buried by inserting the needle into deeper tissue; the needle exits several millimeters from the wound edge. If one pulls on the needle end, the knot disappears into the wound. [73] The obvious advantage of this technique is that there are no suture marks in the skin. Another method that avoids penetrating the skin is the interrupted subcuticular

Figure 36-38 In constructing the relief to facilitate suture removal, the suture is crossed to the opposite side, going into the subcuticular area beneath the skin for approximately 2 cm before exiting *(A)*. The suture is then carried over the epidermis for approximately 2 cm *(B)* and then back under the dermis again *(C)*. Reentry is made into the wound area *(D)* at approximately the same location where the next "bite" would have been placed had the relief not been used. *(From Grimes DW, Garner RW: "Reliefs" in intracuticular sutures. Surgical Rounds 1:47, 1978. Reproduced by permission.)*

stitch (Fig. 36-40).[90] Wounds with strong static skin tension may benefit from a few interrupted dermal stitches placed horizontal to the skin surface instead of a continuous subcuticular stitch.

Nonabsorbable sutures can be left in place for 2 to 3 weeks, thus providing a longer period of support than percutaneous sutures, without the problem of stitch marks.[78] If skin sutures are used in conjunction with the subcuticular stitch, they are removed in 3 to 4 days. A subcuticular closure in itself is stronger than a tape closure. If the subcuticular technique is used exclusively to approximate the skin surface, it is advisable to apply skin tape to correct surface unevenness and to provide a more accurate apposition of the epidermis.

Mattress stitch.

The various types of mattress stitches are all interrupted stitches. The *vertical mattress stitch* is an effective method of everting skin edges (Fig. 36-41 and see Fig. 36-31). The vertical mattress stitch may be used to take a deep bite of skin in lieu of a layered closure in areas where excessive tension does not result. If the superficial loop is placed first, the tails can be pulled upward while the deep loop is placed, ensuring wound eversion in less time than with the traditional technique. [95] Unfortunately, this stitch causes more ischemia and necrosis inside its loop than either simple or continuous stitches. [96] The *horizontal mattress stitch* approximates skin edges closely while providing some degree of eversion (Fig. 36-42). [78] The horizontal mattress suture may be ideal for areas where eversion is desirable but there is little SQ tissue. The *half-buried horizontal mattress stitch,* also called a *mattress stitch with a dermal component*, combines an interrupted skin stitch with a buried intradermal stitch (Fig. 36-43). It is effective in joining the edges of a skin flap to the edges of the "recipient site"; the dermal component is placed through the dermis of the flap.[90] The half-buried horizontal mattress stitch is also useful at the scalp-forehead junction when there is tension on the wound edges. This technique halves the number of suture marks in the skin and avoids necrosis of the edge of a skin flap.

The half-buried horizontal mattress stitch is particularly useful in suturing the easily damaged apex of a V-shaped flap (Fig. 36-44). In the execution of the "corner stitch," the suture needle penetrates the skin at a point beyond the apex of the wound and exits through the dermis. The corner of the flap is elevated, and the suture is passed through the dermis of the flap. The needle is then placed in the dermis of the base of the wound and returned to the surface of the skin. All dermal bites should be placed at the same level. The suture is tied with sufficient tension to pull the flap snugly into the corner without blanching the flap. [78] [97] If the tip of a large flap with questionable viability may be further jeopardized by postoperative swelling, a cotton stent can be placed underneath the knot of the corner stitch. The cotton absorbs the tension produced by swelling.

Figure-of-eight stitch.

The figure-of-eight stitch is useful in wounds with friable tissue, on the eyelids where the skin is too thin for buried sutures, or in areas in which buried sutures are undesirable (Fig. 36-45).[98] This stitch reduces the amount of tension placed on the tissue by the suture, allowing the stitch to hold in place when a simple stitch would tear through the tissue. One disadvantage of this technique is that more suture material is left in the wound. A vertical variation of the figure-of-eight stitch is sometimes used to approximate close, parallel lacerations (Fig. 36-46).[99] Another technique involves a vertical mattress stitch. The central "island" of tissue is secured by passing the superficial portion of the stitch through the island at the subcuticular level (Fig. 36-47).[100] If the viability of the central island is questionable and the surrounding tissue is loose, it can be excised.

Correction of dog-ears.

When wound edges are not precisely aligned horizontally, there will be excess tissue on one or both ends. This small flap of excess skin that bunches up at the end of a sutured wound is commonly called a *dog-ear*. This effect also occurs when one side of the wound is more elliptical than the opposite side or when an excision of a wound is not sufficiently elliptical—that is, when it is either too straight or too nearly circular. [48] [90]

If a dog-ear is present, it can be eliminated on one side of the wound in the following manner: The flap of excess skin is elevated with a skin hook, and an incision is carried at an oblique angle from the apex of the wound toward the side with the excess skin. The flap is then undermined and laid flat. The resulting triangle of skin is trimmed, and the closure is completed (Fig. 36-48*A*).[89] [97] An alternative method consists of carrying the incision directly from the apex, in line with the wound. The flap of excess tissue is pulled over the incision while skin hooks are used to retract the extended apex of the wound. Excess tissue is excised, and the remainder of the wound is sutured. [90] If dog-ears are present on *both* sides of one end of the wound, the bulge of excess tissue can be excised in an elliptical fashion, and the wound can be closed (Fig. 36-48*B*).[97]

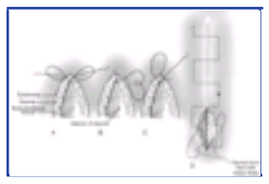

Figure 36-39 Subcuticular closure without epidermal penetration. *A,* The initial knot is secured in the dermal or subcutaneous tissue. *B,* The short strand is cut, and the needle is inserted into the dermis at the apex of the wound. *C,* The needle in the dermis, close to the corner of the wound and exiting the wound at the same horizontal level. *D,* After the subcuticular stitch has been completed, a knot is tied with the tail and the loop of the suture. *(Modified from Stillman RM: Wound closure: Choosing optimal materials and methods. ER Reports 2:43, 1981.)*

V-Y advancement flap.

If a corner stitch produces excessive tension on the tip of the flap, a V-Y closure can be used to approximate the edges without undue tension. An incision carried away from the apex of the wound converts it from a V to a Y configuration (Fig. 36-49). The newly formed wound edges are undermined, and the repair is completed. A half-buried mattress stitch is placed at the fork of the Y. [97]

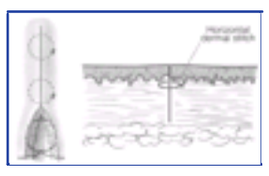

Figure 36-40 Interrupted subcuticular stitch (also called a *horizontal dermal stitch*). Absorbable sutures are used. A deep vertical suture is also shown.

Stellate lacerations.

The repair of a stellate laceration is a challenging problem. Usually a result of compression and shear forces, these injuries contain large amounts of partially devitalized tissue. The surrounding soft tissue is often swollen and contused. Much of this contused tissue cannot be

Figure 36-41 Vertical mattress stitch. The key to a tight closure is to place the inner sutures very close to the suture line (wound edge). *(From Grabb WC: Basic techniques of plastic surgery. In Grabb WC, Smith JW (eds): Plastic Surgery: A Concise Guide to Clinical Practice. Boston, Little, Brown, 1979. Reproduced by permission.)* See also Fig. 36-31 .

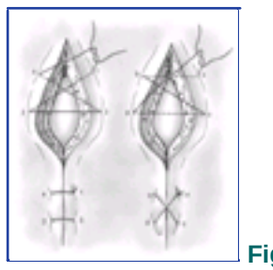

Figure 36-42 *A*, Horizontal mattress stitch. *B,* The dorsum of the hand, foot, or finger is an ideal place for a horizontal mattress suture to evert the wound edges. The relatively thin skin in these areas precludes the use of vertical mattress sutures. *(A from Grabb WC: Basic techniques of plastic surgery. Grabb WC, Smith JW (eds): Plastic Surgery: A Concise Guide to Clinical Practice. Boston, Little, Brown, 1979. Reproduced by permission.)*

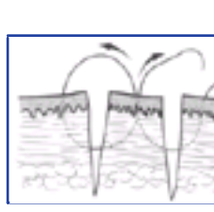

Figure 36-43 Half-buried horizontal mattress stitch. *(From Grabb WC: Basic techniques of plastic surgery. In Grabb WC, Smith JW: Plastic Surgery: A Concise Guide to Clinical Practice. Boston, Little, Brown, 1979. Reproduced by permission.)*

Figure 36-44 *A* and *B,* Approximation of a corner flap with a half-buried horizontal mattress stitch. Because of its applicability to this closure, the stitch is often called a *corner stitch*.

Figure 36-45 Figure-of-eight stitch-two methods. *(Modified from Dushoff IM: About face. Emerg Med 6:11:1974. Reproduced by permission.)*

Figure 36-46 Vertical figure-of-eight suture technique. This can be used to close parallel lacerations. *(From Mitchell GC: Repair of parallel lacerations [letter]. Ann Emerg Med 16:924, 1987.)*

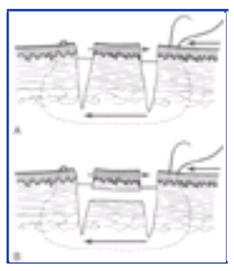

Figure 36-47 Techniques for closure of parallel lacerations. *A,* Central tissue island with intact base. *B,* Central tissue island shaved from base. *(Redrawn from Samo DG: A technique for parallel lacerations. Ann Emerg Med 17:297, 1988.)*

Figure 36-48 *A,* Correction of a dog-ear. *B,* Excision of bilateral dog-ears. *(A from Dushoff IM: A stitch in time. Emerg Med 5:1, 1973. Reproduced by permission.)*

Figure 36-49 *A* and *B,* V-Y advancement flap. *(From Rosen P, Sternbach G: Atlas of Emergency Medicine. Baltimore, Williams & Wilkins, 1979, p 132. Reproduced by permission.)*

debrided without creating a large tissue defect. Sometimes tissue is lost, yet the amount is not apparent until key sutures are placed. In repairing what often resembles a jigsaw puzzle, the clinician can remove small flaps of necrotic tissue with an iris scissors; large, viable flaps can be repositioned in their beds and carefully secured with half-buried mattress stitches. If interrupted stitches are used to approximate a thin flap, small bites should be taken in the flap and larger, deeper bites in the base of the wound. A modification of the corner stitch can be used to approximate multiple flaps to a base (Fig. 36-50). The V-Y advancement flap technique is also useful. Thin flaps of tissue in a stellate laceration with beveled

Figure 36-50 View from *above* stellate laceration, showing closure with half-buried mattress stitches.

edges are often most easily repositioned and stabilized with a firm dressing. [78]

Closure of stellate lacerations cannot always be accomplished immediately, especially if there is considerable soft tissue swelling. It may be best in some instances to consider delayed closure or revision of the scar at a later date. In complicated lacerations, inexact tissue approximation may be all that is possible initially.

Repair of Special Structures

Facial Wounds (General Features)

The ideal result in the repair of a facial laceration is an extremely narrow, flat, and unapparent scar. In addition to basic wound management, a few additional techniques can be used to achieve this result. One factor that contributes to wide scars is necrosis of partially devitalized wound edges. However, skin with apparently marginal circulation may survive because of the excellent vascularity of the face. SQ fat, which in other locations may be debrided thoroughly, should be preserved if possible in facial wounds to prevent eventual sinking of the scar and to preserve normal facial contours. Therefore, debridement of most facial wounds should be conservative.[81] Facial and forehead lacerations that follow natural skin creases or lines will heal with a less noticeable scar than those that are oblique or perpendicular to the natural wrinkles of the skin (Fig. 36-51).

Converse pointed out that "precise approximation of skin edges without undue tension ensures primary healing with minimal scarring." [90] A layered closure is essential in the cosmetic repair of many facial wounds. Approximation of the dermis with an SQ stitch or a combination of SQ and subcuticular stitches should bring the epithelial edges together or within 1 to 2 mm of apposition—close enough that the use of additional sutures seems almost unnecessary. [89] If an SQ stitch is the only stitch used to close the deeper layers, it should pass

Figure 36-51 Lacerations following natural skin lines (shown here) heal with a less noticeable scar than those that are oblique or perpendicular to natural lines (or wrinkles).

through the dermal-epidermal junction or within 1 to 2 mm of the skin surface without causing a dimpling effect. The clinician must tie this stitch snugly, pulling the two ends of the suture in the same direction (see Fig. 36-21). Should the first SQ stitch placed at the midpoint of a wound perfectly appose the skin edges, one can "protect" that stitch from disruption during further suturing by immediately placing a percutaneous stitch in the same location. If there is a slight gap in the wound edges after SQ closure, the skin can be partially approximated with a few guide stitches. The first is placed at the midpoint of the wound, and subsequent stitches bisect the intervening spaces. Guide stitches allow the definitive epithelial sutures to be placed with little tension on each individual stitch, and they protect the SQ stitches from disruption. Once the definitive stitches have been placed, the guide stitches, if slack, can be removed. Because a needle damages tissue with each passage through the skin, guide stitches should be used only when necessary.

The epithelial stitch should never be used to relieve the wound of tension; it serves only to match the epidermal surfaces precisely along the length of the wound. If there is significant separation of the wound edges after closure of the SQ layer, a 5-0 or 6-0 subcuticular suture can be used to eliminate the tension produced by this separation and to provide prolonged stability. Once the skin edges are apposed, the epithelial stitch can be used to correct discrepancies in vertical alignment. A 6-0 synthetic nonabsorbable suture is an excellent material for this stitch. A continuous stitch is preferable because it can be placed quickly, but interrupted stitches are acceptable. In a straight laceration, better apposition is achieved if the wound is stretched lengthwise by finger traction or by the use of skin hooks. When the needle is placed on one side of the wound, if that side is higher than the opposite side, a shallow bite is taken. The needle is used to depress the wound edge to the proper height, after which the needle "follows through" to the other side, pinning the two sides together. If the first side entered is lower, the needle is elevated when entering the second side to match the epithelial edges.

Grabb pointed out that "the closer the needle lies to the skin edge, the greater will be its effect in controlling the ultimate position of that edge." [68] Epithelial stitches should be spaced no more than 2 to 3 mm apart and should encompass no more than 2 to 4 mm of tissue. [80] If widely spaced, the sutures will leave marks. [89] Once skin closure is complete, final adjustments in the tension on any continuous suture line are made before the end of the stitch is tied. If any level discrepancies persist, interrupted sutures or tape can be used to flatten these few irregularities.

Surgical tape is useful as a secondary support, protecting the epithelial stitch from stresses produced by normal skin movements (Fig. 36-52). Facial wounds have a tendency to swell and place excessive stretch on an epithelial stitch. This can be minimized by applying a pressure dressing and cold compresses to the wound following closure. Surgical tape can serve to a limited extent as a pressure dressing.

Forehead

Although the forehead is actually a part of the scalp, lacerations in this region are treated as facial wounds. Vertical lacerations across the forehead are oriented 90° to skin tension lines, and the resulting scars are more noticeable than those

Figure 36-52 Wound closure tape can be used to provide additional support while sutures are in place and after they are removed. This may be especially useful in cosmetic areas, such as the face.

from horizontal lacerations. Midline vertical forehead lacerations may result in cosmetically acceptable scars with standard closure techniques; uncentered lacerations may benefit from S-plasty or Z-plasty techniques during the initial repair or during later revision of the scar.

Superficial lacerations may be closed with skin stitches alone, but deep forehead lacerations must be closed in layers. The periosteum should be approximated before the closure of more superficial layers. If skin is directly exposed to bone,

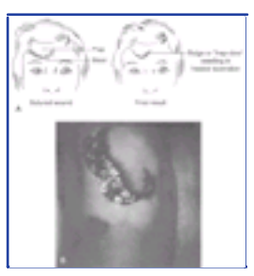

Figure 36-53 *A*, Elevation of a forehead flap. The "trap-door effect" is a natural healing process of elliptical or round lacerations. Patients should be advised of this phenomenon. *B,* This flap-type laceration of the knee will heal with a puffed-up center (trap door), even under the best of circumstances. *(A from Grabb WC, Kleinert HE: Technics in Surgery: Facial and Hand Injuries. Somerville, NJ, Ethicon, Inc., 1980. Reproduced by permission.)*

adhesions may develop that in time may limit the movement of skin during facial expressions. The frontalis muscle fascia and adjacent fibrous tissue should be approximated as a distinct layer; if left unsutured, the retracted ends of this muscle will bulge beneath the skin. If the gap in a muscle belly is later filled with scar tissue, movement of the muscle pulls on the entire scar and makes it more apparent. [81]

A U-shaped flap laceration with a superiorly oriented base poses a difficult problem. Immediate vascular congestion and later scar contraction within the flap produce the "trap-door effect," with the flap becoming prominently elevated (Fig. 36-53). This effect can be minimized by approximation of the bulk of SQ tissue of the flap to a deeper level on the base side of the wound; the skin surfaces of the two sides are apposed at the same level (Fig. 36-54*A*). A firm compression dressing helps eliminate "dead space" and hematoma formation within the wound. Despite these efforts, secondary revision is sometimes necessary. [78] Often, swelling of the flap resolves over a 6- to 12-month period. Because flap elevation can be quite disconcerting, the clinician should forewarn the patient and family about a possible trap-door effect.

When a forehead laceration borders the scalp and the thick scalp tissue must be sutured to thinner forehead skin, a horizontal or vertical mattress stitch with an intradermal component can be used (Fig. 36-54*B*).[90]

Eyebrow and Eyelid Lacerations

Jagged lacerations through eyebrows should be managed with little, if any, debridement of untidy but viable edges. The hair shafts of the eyebrow grow at an oblique angle, and vertical excision may produce a linear alopecia in the eyebrow,

Figure 36-54 *A,* Repair of a U-shaped flap laceration with a superiorly oriented base to minimize the trap door effect. *A,* Excision of edges. *B,* undermining. *C,* Approximation of SQ tissue on the flap to SQ tissue at a deeper level on the base; *D,* skin closure. *B* and *C,* When a laceration in the thin skin of the forehead borders the thicker skin of the scalp, a horizontal mattress suture with an intradermal component can enhance healing by bringing tissues to the same plane. These figures show eversion of thinner skin to obtain adequate approximation with thicker scalp tissue. *(B from Converse JM: Introduction to plastic surgery. In Converse JM (ed): Reconstructive Plastic Surgery: Principles and Procedures in Correction, Reconstruction, and Transplantation, vol 1, 2nd ed. Philadelphia, WB Saunders, 1977. Reproduced by permission.)*

whereas with simple closure, the scar remains hidden within the hair. If partial excision is unavoidable, the scalpel blade should be angled in a direction parallel to the axis of the hair shaft to minimize damage to hair follicles.

Points on each side of the lacerated eyebrow should be aligned precisely; a single percutaneous stitch on each margin of the eyebrow should precede SQ closure. The edges of the eyebrow serve as landmarks for reapproximation; therefore, the eyebrow must not be shaved, as these landmarks will be lost. Shaved eyebrows grow back slowly and sometimes incompletely, and shaving them often results in more deformity than the injury itself. Care must be taken not to invert hair-bearing skin into the wound. [93]

The thin, flexible skin of the upper eyelid is relatively easy to suture. A soft 6-0 suture (or smaller) is recommended for closure of simple lacerations. Traumatized eyelids are susceptible to massive swelling; compression dressings and cool compresses can be used to minimize this problem.

It is essential that the emergency clinician recognize complicated eyelid lacerations that require the expertise of an ophthalmologist. Lacerations that traverse the lid margin require exact realignment to avoid entropion or ectropion (Fig. 36-55*A*). Injuries penetrating the tarsal plate frequently cause damage to the globe. A deep horizontal laceration through the upper lid that divides the thin levator palpebrae muscle or its tendinous attachment to the tarsal plate produces ptosis. If this muscle

cannot be identified and repaired by the emergency clinician, a consultant should repair the injury primarily. A laceration through the portion of the upper or lower lid *medial* to the punctum frequently damages the lacrimal duct or the medial canthal ligament and requires specialized techniques for repair (Fig. 36-55*B*). If adipose tissue is seen within any periorbital laceration, one must assume that the orbital septum has been penetrated and that retrobulbar fat is herniating through the wound (Fig. 36-55*C*). The repair of lid avulsions, extensive lid lacerations with loss of tissue, and any of the other complex types of lid lacerations mentioned earlier should be left to ophthalmologists.

Ear Lacerations

The primary goals in the management of lacerations of the pinna are expedient coverage of exposed cartilage and minimization of wound hematoma (Fig. 36-55*D*). Cartilage is an avascular tissue, and when ear cartilage is denuded of its protective, nutrient-providing skin, progressive erosive chondritis ensues. The initial step in the repair of an ear injury involves trimming away jagged or devitalized cartilage and skin. If the skin cannot be stretched to cover the defect, additional cartilage along the wound margin can be removed. Depending on the location, as much as 5 mm of cartilage can be removed without significant deformity. Cartilage should be approximated with 4-0 or 5-0 absorbable sutures initially placed at folds or ridges in the pinna representing major landmarks. Sutures tear through cartilage; therefore, the anterior and posterior perichondrium should be included in the stitch. No more tension should be applied than is needed to touch the edges together.

In through-and-through ear lacerations, the posterior skin surface should be approximated next, using 5-0 nonabsorbable synthetic sutures. Once closure of the posterior surface is completed, the convoluted anterior surface of the ear can be approximated with 5-0 or 6-0 nonabsorbable synthetic sutures, with landmarks joined point by point. On the free rim, the skin should be everted if later notching is to be avoided. Care should be taken to cover all exposed cartilage. In heavily contaminated wounds of the ear (e.g., bite wounds) that already show evidence of inflammation, the necrotic tissue should be debrided, the cartilage covered by a loose approximation of skin, and the patient placed on antibiotics. [78] [101] After a lacerated ear has been sutured, it should be enclosed in a compression dressing (see Fig. 65-29).

Lacerations of the Nose

In the repair of lacerations of the nose, reapproximation of the wound edges is difficult because the skin is inflexible, and even deeply placed stitches will slice through the epidermis and pull out. When the wound edges cannot be coapted easily,

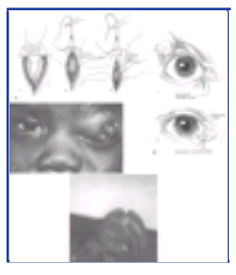

Figure 36-55 *A,* Repair of a simple lid laceration. The first sutures are placed at the lid margin so that the lid can be extended by traction with a hemostat. Interrupted absorbable sutures are used to close the tarsus (1), followed by separate closure of the muscle layer with absorbable sutures (2), and finally by closure of the skin with interrupted 8-0 black silk or synthetic sutures (3). Such a repair should not be performed by the novice. *B*, A method of identifying and repairing the canaliculus. This repair is best left to the ophthalmologist, but recognizing the potential for a canaliculus injury is the task at hand in the emergency department. *C,* Deep laceration of the left upper lid with herniation of orbital fat. For fat to prolapse, the orbital septum (and potentially the globe itself) must have been perforated. This is a wound requiring operating room exploration and repair. *D*, Lacerations of the ear require a special repair aimed at covering cartilage and preventing hematoma formation. With this through-and-through laceration of the margin of the pinna, the cartilage is trimmed just enough to allow the skin to be approximated to cover all exposed cartilage. The repair is easiest if the posterior pinna is sutured first.

6-0 absorbable sutures can be placed in the fibrofatty junction in an SQ stitch before skin closure. Because it is difficult to approximate gaping wounds in this location, debridement must be kept to a minimum. Nasal cartilage is frequently involved in wounds of the nose, but it is seldom necessary to suture the cartilage itself.

The free rim of the nostril must be aligned precisely to avoid unsightly notching. Many clinicians recommend early removal of stitches to avoid stitch marks, yet the oily nature of skin in this area makes it difficult to keep the wound closed with tape. A subcuticular stitch is recommended if the wound is gaping before closure, as this will provide support for a prolonged period. [102]

Lip and Intraoral Lacerations

Lip lacerations are cosmetically deforming injuries, but if the clinician follows a few guidelines, these lacerations usually heal satisfactorily.

The contamination of all intraoral and lip wounds is considerable; they must be thoroughly irrigated. Regional nerve blocks are preferred to local injection, because the latter method distends tissue, distorts the anatomy of the lip, and obscures the vermilion border. Losses of <25% of the lip permit primary closure with little deformity; losses of >25% require a reconstructive procedure. Extensive lacerations directly through the commissure of the mouth also require surgical consultation in most cases.[101] Deep scars in the vermilion of the upper lip may produce a redundancy of tissue that requires later revision. [101]

Large through-and-through lacerations of the lip should be closed in three layers. With a multilayer closure, the muscle layer is approximated with a 4-0 or 5-0 absorbable suture securely anchored in the fibrous tissue located anterior and posterior to the muscle. The vermilion-cutaneous junction of the lip is a critical landmark that, if divided, must be repositioned with precision; a 1-mm "step-off" is apparent and cosmetically unacceptable. The vermilion border should be approximated with a 5-0 or 6-0 nonabsorbable stay suture before any further closure to ensure proper alignment throughout the remainder of the repair (Fig. 36-56). The

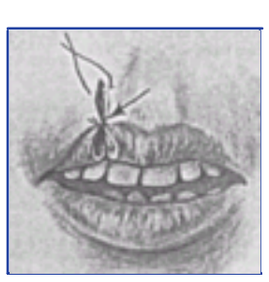

Figure 36-56 *A,* In the repair of lip lacerations, the first stitch should be placed at the vermilion-cutaneous border to obtain proper alignment. *(A from Grabb WC, Kleinert HE: Technics in Surgery: Facial and Hand Injuries. Somerville, NJ, Ethicon, Inc., 1980. Reproduced by permission.)*

vermilion surface of the lip and the buccal mucosa are then closed with interrupted stitches using an absorbable 4-0 or 5-0 suture. Finally, the skin is closed with 6-0 nonabsorbable sutures.[103]

Small puncture-type lacerations heal well only if the skin is closed and the small intraoral laceration is left open. Such injuries are common from a punch in the face when the victim's tooth lacerates the lip. In general, small lacerations of the oral mucosa heal well without sutures. If a mucosal laceration creates a flap of tissue that falls between the occlusal surfaces of the teeth or if a laceration is extensive enough to trap food particles (e.g., 2 to 3 cm or greater in length), it should be closed. Small flaps may be excised. Closure is easily accomplished with 4-0 Dexon or Vicryl using a simple interrupted suturing technique. These materials are soft and less abrasive than gut sutures, which become hard and traumatize adjacent mucosa. Similarly, nylon sutures whose sharp ends are annoying and painful should be avoided inside the mouth. Muscle and mucosal layers should be closed separately. Sutures in the oral cavity easily become untied by the constant motion of the tongue. Each suture should be tied with at least four square knots. These sutures need not be removed; they either loosen and fall out within 1 week or are rapidly

absorbed.[81] [103] [104]

All lacerations that penetrate the oral mucosa should be evaluated for the presence of a tooth fragment. A retained tooth fragment should be searched for in the depths of the wound if a tooth is missing or chipped. The search should be intensified if the patient returns with an infection of a sutured wound. Probing the wound with forceps may identify fragments not seen directly in the wound. In the setting of marked facial swelling, a radiograph of the soft tissue may help identify an embedded tooth fragment.

Tongue Lacerations

There is some controversy regarding when to suture tongue lacerations. Simple, linear lacerations, especially those in the central portion of the tongue, heal quickly with minimal risk of infection. Most tongue lacerations that occur from falls or seizures do not require sutures. Most tongue lacerations in children heal well without sutures. Snyder suggests that only those lacerations that involve the edge or pass completely through the tongue, flap lacerations, and lacerations that continue to bleed excessively need to be sutured (Fig. 36-57). All lacerations bisecting the tongue require repair. [102] Small flaps on the edge of the tongue may be excised, but large flaps should be sutured. When dilute peroxide mouth rinses and a soft diet are used for a few days, healing is rapid. Persistent bleeding from minor lacerations brings most patients to the hospital, and closure may be necessary to prevent further bleeding.

The repair of a tongue laceration in any patient is somewhat difficult, but in an uncooperative child, the procedure may prove impossible under anything other than general anesthesia. A Denhardt-Dingman side mouth gag aids in keeping the patient's mouth open. A localized area of the tongue may be anesthetized topically by covering the area with 4% lidocaine-soaked gauze for 5 minutes; the maximum safe dose of local anesthesia should be determined and exposure to greater doses avoided. Large lacerations require infiltration anesthesia (1% lidocaine with buffered epinephrine) or a lingual nerve block. If the tip of the tongue has been anesthetized, a towel clip or suture can be used to

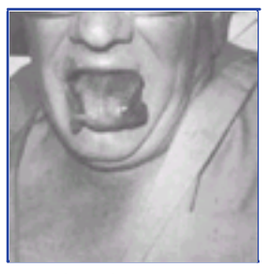

Figure 36-57 Through-and-through injuries and lacerations of the tongue margins require sutures to achieve anatomic healing. Dexon, Vicryl, or silk sutures are ideal for suturing the tongue surface. Bleeding is usually controlled with direct pressure and local infiltration of lidocaine with epinephrine. For through-and-through lacerations, the muscle layer should be closed separately (with absorbable sutures) to prevent hematoma formation. In general, buried sutures are better tolerated by the patient.

maintain protrusion of the tongue. Further anesthesia and subsequent wound cleansing and closure are possible while an assistant applies gentle traction to the tongue.

Size 4-0 absorbable sutures should be used to close all 3 layers—inferior mucosa, muscle, and superior mucosa—in a single stitch, or the stitch should include one half of the thickness of the tongue, with sutures placed on the superior and inferior surfaces as well as on the edge of the tongue. [102] Sutures on the tongue frequently become untied. This problem can be avoided if the stitches are buried. Do not use nylon sutures in the tongue, because the sharp edges are quite uncomfortable. [81] Closure of the lingual muscle layer is usually sufficient to control bleeding and return motor function to the lacerated tongue. Mucosal healing is rapid, and closure of the muscle layer with only a deep absorbable suture may be sufficient.

Scalp

The scalp extends from the supraorbital ridges anteriorly to the external occipital protuberances posteriorly and blends with temporalis fascia laterally. There are five anatomic layers of the scalp: skin, superficial fascia, galea aponeurotica, subaponeurotic areolar connective tissue, and periosteum (see Fig. 36-18*A*). Surgically, the scalp may be divided into three distinct layers. The outer layer consists of the skin, superficial fascia, and galea (the aponeurosis of the frontalis and occipitalis muscles), which are firmly adherent and surgically are considered as one layer. The integrity of the outer layer is maintained by inelastic, tough, fibrous septa, which keep wounds from gaping open unless all three portions have been traversed. Wounds that gape open signify a laceration extending beneath the galea layer. The galea itself is loosely adherent to the periosteum by means of the slack areolar tissue of the subaponeurotic layer. The periosteum covers the skull. The periosteum is often mistakenly identified as the galea, and vain attempts are made to suture the flimsy periosteum in the hope of "closing the galea" (Fig. 36-58). [105]

Several unique problems are associated with wounds of the scalp. The presence of a rich vascular network in the superficial fascia results in profuse bleeding from scalp wounds. Severed scalp vessels tend to remain patent, because the fibrous SQ fascia hinders the normal retraction of blood vessels that have been cut, allowing persistent or massive hemorrhage in simple lacerations. The subgaleal layer of loose connective tissue contains "emissary veins" that drain through diploic vessels of the skull into the venous sinuses of the cranial hemispheres. In scalp wounds that penetrate this layer, bacteria may be carried by these vessels to the meninges and the intracranial sinuses. Thus, a scalp wound infection can result in osteomyelitis, meningitis, or brain abscess. [102] Careful approximation of galeal lacerations not only ensures control of bleeding, but also protects against the spread of infection.

Shear-type injuries can cause extensive separation of the superficial layers from the galeal layer (Fig. 36-59). Debris and other contaminants can be deposited several centimeters from the visible laceration. Careful exploration and cleaning of scalp wounds are important.

Because the scalp is vulnerable to blunt trauma and because its superficial fascial layer is inelastic and firmly adherent to the skin, stellate lacerations are common in this region. Stellate lacerations not only pose additional technical problems in closure, but also have a greater propensity for infection. Multiple scalp wounds that are hidden by a mat of hair are easily overlooked.

When scalp wounds are debrided, obviously devitalized tissue should be removed, but debridement should be conservative, because closure of large defects is difficult on the scalp. When facing profuse bleeding, especially from extensive lacerations, the clinician should instruct an assistant to maintain compression around the wound during the closure rather than try to tie off bleeding vessels. Unless the vessels are large or few, ligation of individual scalp vessels seldom provides effective hemostasis, and considerable blood loss can occur during the attempt. Bleeding from scalp lacerations is best controlled by expeditious suturing. [97] A simple procedure that often provides hemostasis of scalp wounds is placing a *wide*, tight rubber band or Penrose drain around the scalp, from forehead to occiput (Fig. 36-60*A*). Sterile rubber bands may be kept on the suture cart for this purpose. The clinician also may control bleeding temporarily in some cases by grasping the galea and the dermis with a hemostat and everting the instrument over the skin edge. The disadvantage of this technique is that tissue grasped by the hemostat may be crushed and devitalized[97] and if the SQ tissue also is everted for a prolonged period, necrosis can occur.

If an assistant is not available to apply direct pressure, local anesthetics containing epinephrine are sometimes effective in controlling the persistent bleeding from small vessels in scalp wounds. If bleeding from the edge of the scalp wound is vigorous, and definitive repair must be postponed while the patient is resuscitated, Raney scalp clips can be applied quickly to the edge of the scalp wound to control the hemorrhage. The applicator is loaded by inserting the tip of the instrument into the back of the clip and then locking the handles. The clip is slid onto the bleeding wound edge and released from the applicator. When the wound is repaired at a later time, the clip is removed by reversing the procedure. The plastic clips are radiolucent and do not interfere with plain radiography or computed tomography scanning (Fig. 36-60*B*). [106] [107]

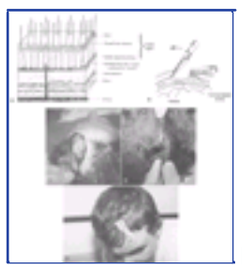

Figure 36-58 *A*, Anatomy of the scalp. Note that the skin, superficial fascia, and galea are adherent and constitute the outer layer. Blood vessels in the fascia are the major source of the blood loss noted in scalp lacerations. *B,* To temporarily control bleeding from vessels in the fascia, the galea can be everted to compress the fascia. *C,* The galea has been transected in wounds that gape open like this one, and to achieve hemostasis and obtain the best closure, the galea should be sutured. This is most easily accomplished with the use of a long needle, forceps, and 3-0 sutures that incorporate the skin, SQ tissue, and galea in a single bite *(D)*. In this figure, the needle is passing through the galea from the underside, having traversed all three layers on the other side of the laceration. If this technique is used, individual buried sutures in the galea are not required, and hemostasis is ensured. At the base of this wound is the periosteum, a tissue-like covering of the skull. In *C*, the galea is actually adherent to the avulsed flap; the anesthetic needle is touching the underside of the galea. A simple laceration that does not gape open *(E)* means the galea is intact. It can be easily closed with superficial sutures or staples.

Figure 36-59 Large partial scalp avulsion.

Before wound closure, the underlying skull should be visually examined and palpated in an attempt to detect fractures. More small skull fractures are detected with the clinician's eyes and gloved finger than with radiographs. A common error is to mistake a rent in the galea or the periosteum for a fracture during palpation inside the wound. Direct visualization of the area should resolve the issue. In wounds that expose bone but do not penetrate the skull, prolonged exposure may leave a nidus of dead bone that may develop osteomyelitis. Exposed bone that is visibly necrosed should be removed with rongeurs until active bleeding appears. [97] Hair surrounding the scalp wound usually must be clipped far enough from the wound edge so that suturing can proceed without entangling the hair in knots or embedding hair within the wound. If hairs along the wound

Figure 36-60 *A,* To achieve hemostasis of a scalp laceration, a wide, tight, sterilized rubber band or Penrose drain may be placed around the forehead and occiput. This compresses the arterial supply to the scalp. *B,* Alternatively the wound margins can be temporarily clamped to control hemorrhage. Raney scalp clips and accompanying instrument for application to scalp wound edges are shown.

edges become embedded in the wound, they will stimulate excessive granulation tissue and delay healing. [108] Vaseline or tape may be placed on stubborn hairs that persistently fall into the wound. Although clipping scalp hair is not popular with some patients, failure to expose an area adequately is a common cause of improper cleaning and closure of scalp wounds.

Unlike most wounds involving multiple layers of tissue, scalp wounds can be closed with a single layer of sutures that *incorporate skin, SQ fascia, and the galea* (Fig. 36-58*D*). The periosteum need not be sutured. To minimize the chance of infection, SQ deep sutures generally are avoided. The galea is firmly attached to the underside of the SQ fascia and is rarely identified as a distinct layer in the depths of a wound. In superficial wounds, skin and SQ tissue should be approximated with simple interrupted or vertical mattress stitches using a nonabsorbable 3-0 nylon or polypropylene suture on a large needle. Smaller suture material tends to break while firm knots are being tied and should not be used. The ends of the tied scalp sutures should be left at least 2 cm long to facilitate subsequent suture removal. The use of blue nylon, as opposed to black, may make suture removal easier. If the galea is also torn, it should be included in the skin stitch. [109] Some investigators recommend a separate closure of the galea with an absorbable 3-0 or 4-0 suture, using an inverted stitch that "buries" the knot beneath the galea. [97] Separate closure of the galea introduces additional suture material into the wound, but in extremely large wounds provides a more secure approximation of the galea than obtained with large needle single layer closure.

With microvascular techniques, large sections of skin avulsed from the scalp can be reimplanted. The emergency clinician should use the same techniques in salvaging avulsed scalp as are used for amputated extremities [109] (see Chapter 48 for further discussion).

Figure 36-61 Anatomy of the fingernail. The fingernail rests on the nailbed, also termed the *matrix*. The distal nail covers the sterile matrix; the proximal nail arises from and covers the germinal matrix. The tissue adherent to the proximal dorsal nail is the eponychium (also termed the *cuticle*), and the potential space between the nail and the eponychium is the nail fold.

There may be no absolute time interval between injury and closure that automatically precludes primary suturing of scalp lacerations. Because of the extensive collateral blood supply of the scalp, most lacerations in this area heal without problems. Nonetheless, wound care must be thorough to avoid the devastating complication of scalp infection.

Sutured scalp lacerations need not be bandaged, and patients can rinse their hair in 24 hours. If bleeding is persistent, an elastic bandage can be used as a compression dressing. Gauze sponges are placed over the laceration to provide direct local pressure beneath the elastic bandage.

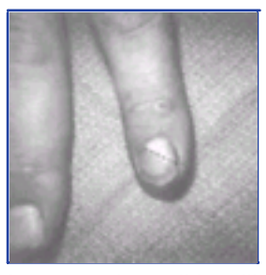

Figure 36-62 This subungual hematoma occupies about two thirds of the nail and should be drained by simple nail trephination. The injury does not require nailbed repair, because the nail is still firmly attached to the matrix. Even though there must be a nail matrix laceration (the source of the bleeding), the cosmetic result will be excellent. The presence of an underlying digital tuft fracture does not change management (see also description of nail trephination in Chapter 38).

Nail Lacerations

Injuries to the nail and nailbed (also called the *nail matrix*) are common problems in emergency medicine, yet controversy exists over proper management (Fig. 36-61). Sixty percent of patients with subungual hematomas that are greater than one half the size of the nailbed and with associated fractures of the distal phalanx have a nailbed laceration. [110] [111]

In the case of a simple subungual hematoma (even in the presence of a tuft fracture) in which the nail is firmly adherent and the disruption of the surrounding tissue is minimal, the nail *need not be routinely removed to search for nailbed lacerations* (Fig. 36-62).[112] Despite the presence of a nailbed laceration, a good result can be

expected as long as the tissue is held in anatomic approximation by the intact fingernail. Nail trephination is discussed in Chapter 38 . If the nail is partly avulsed (especially at the base) or loose, or if there are deep lacerations that involve the nailbed, the nail should be lifted to assess and potentially repair the nailbed (Fig. 36-63). When the integrity of the fingernail is disrupted a rippled nail may develop (Fig. 36-34). Anatomic repair of the nailbed theoretically should minimize subsequent nail deformity.

If the nailbed is exposed and has been extensively lacerated or partially avulsed, it may be necessary to refer the patient to a hand surgeon who can raise a flap of tissue extending from the proximal nail fold, explore the wound for foreign bodies, and clean under the nailbed. A simple nailbed laceration should be approximated with 6-0 or 7-0 absorbable sutures (to obviate the need for suture removal), generally using loupe magnification and a finger tourniquet to maintain a bloodless field (Fig. 36-65). The exposed nailbed should be protected by reapplying the avulsed nail (best choice) or by applying a nonadherent dressing or Silastic sheet for approximately 3 weeks. Reinsertion of the nail may occasionally result in infection, so cleaning the nail is recommended. After cleaning, the avulsed nail may be sutured in place or secured with wound closure tape.

The replaced nail serves three purposes: (1) it acts as a splint or mold to maintain the normal anatomy of the nailbed, (2) it covers a sensitive area and facilitates dressing changes,

Figure 36-63 This fingernail was avulsed at the base *(A)*, a common result of having a door slam on the digit. Since the nail is mobile *(B)* and there is subungual bleeding, the nail can be removed and the nailbed inspected. Any large laceration should be meticulously repaired. Absorbable sutures size 6-0 or smaller should be used. After repair, the nail is replaced under the eponychium (cuticle). See Figure 36-68 for a simple technique for removal of the fingernail.

and (3) it maintains the fold for new nail growth. Splinting should be maintained for 2 to 3 weeks. If longitudinal scar bands are formed between the proximal nail fold and the matrix, a permanently split or deformed nail may result.

A nail that is partially avulsed distally can be used as a temporary splint or "dressing" that protects and maintains the integrity of the underlying nailbed. When the base of the nail is avulsed from the germinal matrix, some authors advocate trimming the proximal portion of the traumatized nail so that it can be placed more easily in the nail fold.[113] If the *germinal matrix* of the nail is avulsed intact, the nail should be reimplanted using a 5-0 or 6-0 absorbable suture in a mattress stitch (Fig. 36-66).[81] [114] If the root is not replaced, the space between the proximal nail fold and the nailbed is obliterated within a few days. [115] [116] If an open fracture exists, the matrix must not be allowed to remain trapped in the fracture line.[117] A replaced nail may grow normally, acting as a free graft,

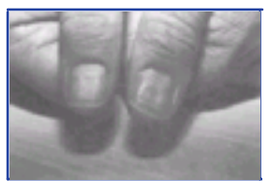

Figure 36-64 This nail is permanently deformed with ridges. Although crush injury to the nailbed is likely responsible for this deformity, nailbed repair is believed to minimize the resultant deformity.

but often it is dislodged by a new nail. Nails grow at a rate of 0.1 mm/day, and it requires approximately 6 months for a new nail to reach to the fingertip.

If part of the nailbed has been lost, the patient should be referred to a surgical consultant for a matrix graft. [81] [113] [118] Conservative therapy that allows large portions of an avulsed nailbed to granulate is inadvisable, although this is quite acceptable therapy for a fingertip avulsion that does not involve the nailbed. If the exposed nailbed is left open to granulate, it will heal with scar tissue and could produce a distorted and sensitive digit.

Wounds should be rechecked in 3 to 5 days following repair. At that time the nail fold may be repacked if nonadherent material was used, and the wound is assessed for infection. The use of absorbable suture for nailbed repair makes suture removal unnecessary. Tape or sutures are removed from any replaced nail in 2 weeks, and the old nail is allowed to fall off as the new nail grows. The value of antibiotics is unproven. All patients with nail injuries should be advised of a possible cosmetic defect in the new nail.

When repairing distal digit lacerations involving a nail, the clinician should first approximate the onychial fold (Fig. 36-67). A sturdy needle attached to a 4-0 thread is recommended for suturing lacerated nails. Needles seem to penetrate nails with the least difficulty when they enter at 90°. The point of the needle carves a rigid path through the nail. Unless the entire length of the needle is allowed to follow this path as it passes through the nail, the needle is likely to bend or break. Alternatively, an electrical cautery instrument or a heated paper clip can be used to perforate the nail, thus permitting easy passage of the needle. The method for atraumatically removing a nail is demonstrated in Fig. 36-68 .

Drains in Sutured Wounds

Drains do not prevent infection; they primarily keep wounds open to encourage drainage of purulence or blood that may

Figure 36-65 A laceration involving the nailbed, germinal matrix, and skinfold must be carefully approximated. First the nail is completely removed. (see Fig. 36-68). Fine, absorbable sutures are used to repair the nailbed under a bloodless field provided by a finger tourniquet. The avulsed nail (trimmed at the base) or a gauze pack is gently placed between the matrix and eponychium for 2 to 3 weeks to prevent scar formation *(A)*. If the original nail is replaced (the best option), it may be sutured or taped in place *(B)*. A large hole in the nail will allow drainage. The old nail is gradually pushed out by a new one. If the nail matrix is replaced quickly and atraumatically, the nail may act as a free graft and grow normally. Note: Only absorbable sutures are used to repair the nailbed.

otherwise collect in the wound. When no infection exists and drains are used in soft tissue wounds "prophylactically," they are more harmful than beneficial. Edlich and coworkers state that "drains act as retrograde conduits through which skin contaminants gain entrance into the wound. Furthermore, the presence of a drain impairs the resistance of the tissue to infection."[72] Magee and colleagues found that drains placed in experimental wounds contaminated with subinfective doses of bacteria greatly enhanced the rate of infection, whether the drain was placed entirely within the wound or was brought out through the wound. [119] Drains behave as foreign bodies, provoking rather than preventing infection. If the wound is considered at high risk for infection, instead of suturing the

Figure 36-66 Avulsion of the nail, leaving the matrix intact, requires only a nonadherent dressing to separate the skinfold from the nailbed. If the germinal matrix is avulsed, as shown in this figure, it should be replaced to its original position under the eponychium with 6-0 plain absorbable sutures. *(From Grabb WC, Kleinert HE: Technics in Surgery: Facial and Hand Injuries. Somerville, NJ, Ethicon, Inc., 1980. Reproduced by permission.)*

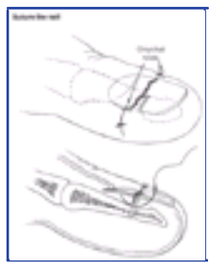

Figure 36-67 Repair of a distal finger laceration involving the nail and the onychial fold. In this case the nail is still adherent to the nail matrix and acts as a natural splint. If the nail is loose or completely transected, it is prudent to remove the entire nail and then carefully suture the nailbed under direct vision. *(From Dushoff IM: Handling the hand. Emerg Med 1976, p 111. Reproduced by permission.)*

Figure 36-68 To remove a fingernail or toenail atraumatically, the blades of iris scissors are held parallel to the nailbed to avoid lacerating the matrix. A digital block is usually performed to make the procedure painless. The closed blades are slowly advanced in the plane between the nail and the nailbed *(A)* and then gently spread *(B)* to loosen the nail. The scissors are advanced and spread in stages until the base of the nail is reached and the entire nail is loose. The nail is grasped with a hemostat and pulled from the base *(C)*, exposing the nail matrix *(D)*. The nail can be replaced, if desired, once the nailbed laceration has been repaired.

wound with a drain in place (in anticipation of disaster), the clinician should leave the wound open and consider delayed primary closure later when the risk of infection is minimal. Furthermore, drains should not serve as substitutes for other methods of achieving hemostasis in traumatic wounds.

SUMMARY

Various techniques are available for reapproximating wound edges. Stapling is fast, but this technique does not allow meticulous control of wound edges, as may be necessary for a cosmetically appealing repair. Tape and tissue adhesive are the quickest and least painful methods of wound closure. Both eliminate the risk of self-injury with suture needles. These techniques can be used only on small superficial wounds or after approximation of the SQ layer.

The traditional and most commonly used method of closure is suturing. Stitches provide the most secure closure initially, but placement of sutures is time consuming and technically more difficult than other methods. All suture materials provoke inflammation and increase the risk of infection. Suture repair is the most appropriate method for wounds with complex configurations, those that extend into SQ tissue, and those in mobile areas.

At the conclusion of any wound repair, dried blood on the skin surface should be wiped away gently with moistened gauze, and the wound should be covered with a dressing. Dressing techniques, wound care instructions, and suture removal skills are discussed in Chapter 35 .

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Chapter 37 - Foreign Body Removal

Daniel B. Stone
Theodore K. Koutouzis

Soft tissue foreign bodies (FBs) are a common occurrence in emergency departments (EDs). FB identification and removal is rewarding to both the patient and clinician. *However, it is not possible, or considered standard of care, that all soft tissues or wound FBs be identified or removed on the initial encounter.* Although the history or physical examination may suggest the presence of an FB, and a reasonable attempt should be made to find or remove it, some foreign material simply defies suspicion, identification, or removal on the first clinician examination. Missed FBs are, however, among the leading causes of malpractice claims made against emergency clinicians, usually because of simple errors in documentation or communication. Often litigation arises merely because the clinician does not pay attention to details of the mechanism of injury or nuances of the examination, or fails to inform the patient that not all foreign material is immediately accessible to diagnosis or removal. Many times known, suspected, or identified FBs can be removed at a later date without a significant increase in morbidity if the patient is properly informed and prepared. This chapter will provide guidelines for the identification, evaluation, and removal of a variety of FBs.

GUIDELINES FOR APPROACHING FOREIGN BODIES

A thorough history and physical examination must initially be performed. It is important to determine the exact mechanism of injury and to determine if the specific characteristics of the foreign material are known. For instance, did the patient step on a rusty nail or piece of broken glass? Was an FB initially present but removed before arrival by the patient? Under many circumstances, a simple direct question to the patient, asking if he or she suspects the presence of a retained FB, will initiate the proper clinical scenario.

The history, physical examination, and localization techniques available will determine the best time and place for FB removal. Some material, such as wood, should be removed immediately when accessible. Retained wood will invariably lead to inflammation and infection. Other material, such as glass or plastic, may be removed on an elective basis, whereas innocuous metallic FBs may often be permanently left embedded in soft tissue. If localization is certain and if removal can be produced under local anesthesia within a manageable period of time (1 hour is usually the upper limit of operative time using local anesthesia), and without unacceptable worsening of the injury, an attempt at removal is generally indicated on the initial visit (given clinician and support staff availability). *Before the procedure, it is prudent to inform the patient that the FB may not be located in the time allotted and that subsequent referral or additional procedures may be necessary.*

With deeply embedded, small, and inert materials (such as a BB) that are not located near any vital structures, the time, effort, and trauma involved with removal may be excessive compared with the possible adverse effects of the foreign material remaining in place. An ill-conceived extended search for an elusive but otherwise harmless FB often results in frustration for the clinician and discomfort and dissatisfaction for the patient.

When reviewing the decision of when and how to remove the FB, the possibility of the FB migrating to involve vital structures, while quite remote, should be discussed with the patient. Cases of reported missile embolization in the vascular system are influenced by missile caliber, impact velocity, physical wound characteristics, point of vessel entrance, body position and movement, and velocity of blood flow. [1] Retained bullets usually remain in soft tissues, but rarely make their way into the vascular system. This usually occurs at the time of injury. Schurr and colleagues [2] reported a paradoxical bullet embolization from the left external iliac vein to the left iliac artery via a patent *foramen ovale*. When clinicians first examined the patient, a bullet was noted on the chest radiograph, and an isolated chest wound was suspected. However, the bullet had apparently entered the chest, traversed the abdomen to the iliac vein, and then embolized back to the chest and arterial system.

All clinical decisions require an evaluation for the possibility of infection. Some FBs may produce an inflammatory reaction or infection in a few days and other objects may not cause such problems for weeks or months, often flaring up for no apparent reason. FBs such as wood will always produce inflammation eventually, while others, such as bullets, rarely do. Some inert FBs may carry dirt particles, pieces of clothing, or other sources of bacterial contamination. Expeditious removal may be necessary, even though the FB itself is relatively small and unlikely to cause a reaction. In old injuries, a thorough history of the type of foreign material and method of introduction is warranted. However, a hasty or extensive exploration for the foreign material that may or may not still exist is not recommended. The initial history should also include any unusual medical problems that would preclude use of adequate local anesthesia, such as allergy to local anesthetics, bleeding diathesis, and medical problems (including diabetes mellitus, vascular disease, uremia, or a compromised immune status) that might lead to unusual or more difficult wound management. Finally, a cooperative and willing patient is essential. *Attempting to remove an FB in an intoxicated, drugged, mentally retarded, or overtly uncooperative patient is obviously self-defeating.*

It is not uncommon to serendipitously encounter soft tissue FBs, even though their presence was not suggested by history. Anderson and associates reported that clinicians who initially treated a series of hand injuries did not suspect FBs in 75 of 200 consecutive cases. [3] A patient who experiences a sharp, sudden pain in the foot while walking barefoot across a carpet may have a sewing needle or toothpick embedded, rather than a "sprained foot" (Fig. 37-1). *An abscess or cellulitis that recurs or wounds that do not heal as expected should always be investigated for retained FBs.* [4] [5] Finally, it should be determined if metallic or other FBs that are captured on radiograph are extrinsic to the patient (located in clothes or on the table) or actually embedded within soft tissue (Fig. 37-2).

If a FB is left in place, remember to inform the patient as to why it was not removed. If the patient is referred for delayed removal, this should also be carefully explained and documented. Regardless of whether the FB is removed, all wounds should be cleaned appropriately and tetanus prophylaxis updated if indicated (see Chapter 36).

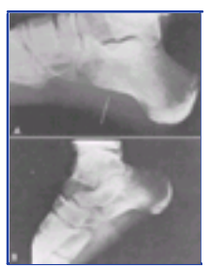

Figure 37-1 A common foreign body (FB) of the foot is a splinter, toothpick, pin, or needle that is impaled while walking barefoot on a carpet. This sewing needle was obvious, but some FBs may be mistaken for a heel spur, contusion, or tendinitis. Preoperative *(A)* and postoperative *(B)* radiographs demonstrate complete removal.

Rarely do retained lead FBs, such as bullets or shotgun pellets, leach out lead into the general circulation and produce systemic lead poisoning (Fig. 37-3). If this process occurs, it may take years to develop and can cause vague or nondescript symptoms (e.g., fatigue, arthralgia, headache, or abdominal pain) many years after the initial injury. Elevated blood lead levels are more likely to occur if bodily fluids such as joint, pleural, peritoneal, or cerebrospinal fluids bathe the lead. Bullets retained in muscle or other soft tissues are not likely to produce any sequelae related to their lead content. However, Farrell and coworkers reported unsuspected elevated lead levels in patients with retained lead fragments who presented to the ED with a variety of complaints. [6] Lead levels of up to 50 µg/dL were reported. Levels >45 µg/dL are generally considered an indication for chelation therapy. The relation between the retained lead and presenting symptoms was unclear, but this report verifies the observations of others that retained lead FBs in selected areas can significantly elevate blood lead levels and may produce symptomatic plumbism.

Finally, the patient should be clearly informed that there is no absolute guarantee that *all* foreign material has been identified or extracted, regardless if some or any FB was removed during initial exploration. The prudent clinician always leaves open the option that an occult FB may still remain in any wound and informs the patient of signs and symptoms of problems related to any retained material. Some centers routinely add this caveat on all discharge instructions for patients treated

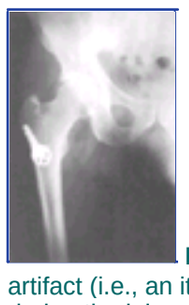

Figure 37-2 This patient fell, landed on a metal pipe, and suffered a laceration to the thigh. A radiograph was taken to rule out a fracture, and the key was seen but thought to be an artifact (i.e., an item left on the backboard). During the examination the key was found embedded in the wound. It had been in the patient's pants pocket and was forced into the wound by the pipe during the injury.

for lacerations or soft tissue defects. Patients should be assured that additional steps may be undertaken should the presence of foreign material be subsequently suspected.

IMAGING TECHNIQUES

A variety of imaging techniques are available to emergency clinicians to help detect and localize FBs. Many emergency clinicians mistakenly believe that, in the absence of adipose tissue, if the base of the wound can be clearly visualized and explored, an FB can always be ruled out. While this is commonly true, Avner and Baker detected glass by routine radiographs in 11 of 160 wounds (6.9%) that were inspected and believed by the clinician to be free of glass. [7] Whenever there is an index of suspicion for a retained FB as a result of the history, mechanism of injury, patient complaint, or examination, attempts should be made to visualize it. Modalities available include plain radiographs, fluoroscopy, computed tomography (CT), magnetic resonance imaging (MRI), and ultrasound (US) (Fig. 37-4). Fluoroscopy is not a standard technique in the ED but is helpful for localizing FBs that are

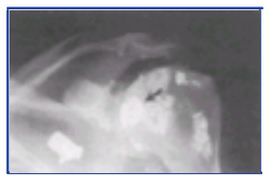

Figure 37-3 Most lead foreign bodies are well tolerated, but if a bullet is bathed in synovial, pleural, peritoneal, or cerebrospinal fluid, the lead may leach out over time and produce a significant elevation in blood lead levels. Symptoms are often vague, and the relation between the retained lead and the patient's clinical scenario may be difficult to sort out. *(From Schwartz DT, Goldfrank LR: Toxicologic imaging. In Goldfrank LR, Flomenbaum NE, Lewin NA, et al [eds]: Goldfrank's Toxicologic Emergencies. 5th ed. Norwalk, CT, Appleton & Lange, 1994, p 116. Reproduced with permission.)*

visualized on routine plain films. Yet even fluoroscopy under magnification may not identify plastic or wooden FBs. [8]

Plain Radiography

Plain radiographs are readily available, easily interpreted, and cost significantly less than CT, US, or MRI. [9] The ability

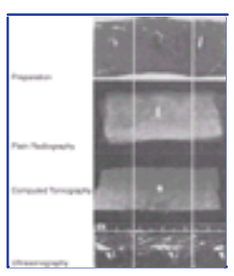

Figure 37-4 Comparison of plain radiography, computed tomography, and ultrasonography in imaging wooden, glass, and plastic foreign bodies (FBs) in an in vitro preparation. Computer manipulation of a digitized radiograph may aid in FB assessment. *(From Ginsburg MJ, Ellis GL, Flom LL: Detection of soft-tissue FBs by plain radiography, xerography, computed tomography, and ultrasonography. Ann Emerg Med 19:701, 1990. Reproduced with permission.).*

of plain films to detect FBs in soft tissues depends on the object's composition (relative density), configuration, size, and orientation. Multiple views should always be obtained when attempting to visualize an FB since many clearly radiopaque objects are obscured by superimposed bone on one view, but are quite obvious when viewed from another angle. However, certain FBs that are radiolucent may still not be visualized with this approach. Metallic objects, such as pins, bullets, and BBs, are readily visualized. Aluminum, which has traditionally been deemed radiolucent, can occasionally be visualized on plain films if the object is projected away from underlying bone. Ellis demonstrated that pure aluminum fragments as small as 0.5 mm × 0.5 mm × 1 mm could be identified in a chicken wing model simulating a human hand or foot. Ellis cautioned that other aluminum FBs, such as pull tabs from cans, may not be visualized in other parts of the body such as the esophagus or stomach.[10]

It is a common misconception that glass must contain lead to be visualized on a plain radiograph. Almost all types of glass objects in soft tissue (bottles, windshield glass, light bulbs, microscope cover slips, laboratory capillary tubes) can be detected by plain radiographs, unless they are obscured by bone (Fig. 37-5).[9] [11] Very small glass fragments (<1 mm) may be more difficult to detect by this technique, but the absence of an FB on multiple projections is strong, although not absolute, evidence that glass is not contained in a wound. [12] Other nonmetallic objects readily visualized include teeth, bone, pencil graphite, asphalt, and gravel. [9] [13]

Detection of FBs on plain films can be enhanced by requesting that the technician use an underpenetrated soft tissue technique. [14] Digitized radiographs may be manipulated to enhance identification of a suspected FB. Plain films may provide indirect evidence of the presence of an FB if one sees trapped or surrounding air, a radiolucent filling defect, or secondary bony changes such as periosteal elevation, osteolytic or osteoblastic changes, or pseudotumors of bone. [14] Vegetative material, such as thorns, wood, splinters, and cactus spines, are not readily visualized by plain radiographs.

Figure 37-5 Almost all glass is visible on a plain radiograph. Small size is the major limiting factor, not the lead content. If glass is superimposed over bone, it may be obscured, so multiple projections are required. Samples of glass are superimposed on the abdomen and exposed to x-rays. *(Reproduced with permission from Zatzkin HR: The Roentgen Diagnosis of Trauma. Copyright © 1965 by Year Book Medical Publishers, Chicago. Reproduced with permission.)*

These materials absorb body fluids as they sit in situ and become isodense with the surrounding tissues. Because of their varying chemical composition and density, plastics may or may not be visible on plain films. [15]

Computed Tomography

CT is readily available to most emergency clinicians. Since CT depends on x-ray absorption, it generally visualizes the same materials that are detected on plain films.[13] CT, however, can detect subtle differences in soft tissue densities, and may detect FBs not readily visible on plain films. [9] Depending on the degree of hydration, wood FBs may be detected using CT. Because it produces a better three-dimensional image of tissue than plain films, CT can visualize objects embedded in or behind bone.

Magnetic Resonance Imaging

Although MRI is expensive and not readily available to emergency clinicians, it may be superior to CT in detecting small, *nonmetallic*, radiolucent FBs, such as wood,

particularly in the orbit.[16] FBs, which have little or no free water, appear as a signal void on MRI.[9] Plastic is more easily visualized with MRI than with CT. MRI cannot be used for metallic objects and gravel, which contain various ferrometallic particles that produce signal artifacts on MRI.[9] Metallic objects, in addition to producing a high degree of artifact on MRI, have a theoretical risk of shifting within the magnetic field and causing structural damage to adjacent structures. This is of little importance in superficial extremity wounds, but it is particularly important when evaluating FBs in the eye, brain, or deep structures of the neck, face, or extremities.

Ultrasound

US offers an alternative way to localize radiolucent FBs. In fact, US has become the modality of choice for difficult-to-find FBs. Like fluoroscopy, it can be used to guide the tip of a hemostat to a nonvisualized object while not exposing the operator or patient to ionizing radiation.[14] US is both highly sensitive and highly specific in localizing FBs of all types.[17] US is especially useful in detecting nonradiopaque FBs such as wood or thorns. A wooden object is usually seen as a bright hyperechoic focus when the scan plane parallels the long axis of the FB or is at right angles to its long axis.[17] US may have more difficulty in visualizing small FBs in the hand or foot, where there are many echogenic structures.[18] While extremely useful for larger FBs in extremities, US may be difficult to use in some sites such as the web space of the hand. A high-resolution, high-frequency transducer provides the greatest anatomic detail. Advantages include low cost, real-time examination, and the ability to define the object in three dimensions. As with all US studies, accuracy is operator dependent. Also, the presence of air in surrounding tissue may result in false-positive results.[18] In addition, US may give a false-positive reading due to calcific tissue or small sesamoid bones. Overall, the sensitivity and specificity of high-resolution US in detecting radiolucent FBs are about 90%.

General Imaging Approach

A reasonable initial approach for localizing nonvisualized FBs in the ED is to obtain multiple-projection plain radiographs with a soft tissue technique. This technique will visualize the majority of FBs, especially metal and glass. US may be considered for objects known to be radiolucent, such as wood or thorns. Both US and fluoroscopy may be used to guide FB extraction. CT or MRI probably should not be routinely ordered from the ED during initial wound evaluation unless one suspects a particularly dangerous FB. *For suspected intraorbital or intracranial FB, CT is recommended.* CT or MRI is also warranted when a previously negatively explored wound exhibits recurrent infection, poor healing, or persistent pain.

FOREIGN BODY REACTION

Many soft tissue FBs must be removed because of either infection or FB reaction. A purulent bacterial infection *may* develop in the presence of any FB, but not in all cases. Karpman and coworkers found a 15% infection rate (*Staphylococcus aureus* and Enterobacteriaceae) in a series of 25 patients treated for cactus thorn injuries of the extremities. [19] Certain thorns (black thorns, rose thorns), redwood and Northwest cedar splinters, toothpicks, hair, and stingray or sea urchin spines are noted for their ability to initiate chronic FB reactions. Sea urchin spines and other marine FBs are covered with slime, calcareous material, and other debris that

commonly initiate an FB granuloma. The inflammatory reaction seen with cactus thorns may be an allergic reaction to fungus found on the cactus plant. Many FB reactions are thought to be due to the inflammatory response to organic material or they may represent infection from bacteria introduced at wounding. Clinically evident reactions may be delayed for weeks or even years following injury (Fig. 37-6). The chronic infection or inflammatory reaction may not be accompanied by the production of pus, but it may be quite painful or result in loss of function. FBs may also be associated with the formation of a chronic pseudotumor, development of a sinus tract, or evidence of osteomyelitis-like lesions of bone and soft tissue. [4] Organic material has also been noted to induce chronic tenosynovitis, chronic monarticular synovitis, and chronic bursitis.

Rapidly traveling projectiles with considerable inherent heat (e.g., bullets) are less likely to cause infection but are more apt to cause damage during passage through tissue. As noted earlier, the composition and location of an FB, as well as patient medical status and vocational/avocational activities, greatly affect decision making related to FB removal. One must judiciously evaluate and manage each FB injury individually.

FOREIGN BODY SCENARIOS

Certain mechanisms of injury, such as punching or kicking out a window or stepping on an unknown object while walking in a field or stream, are highly suggestive of a retained FB (Fig. 37-7). Although not all lacerations caused by glass require a radiograph, imaging should be used generously under such circumstances.

Most lacerations from metal objects do not contain foreign material, but if the patient states that considerable force was applied during injury and the instrument is not available for inspection, radiographic imaging may be warranted. Occasionally, as a knife encounters the bone, the tip of the

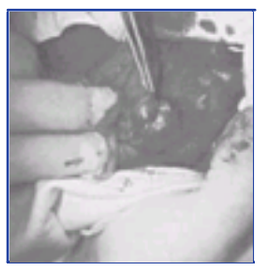

Figure 37-6 This foreign body (FB) granuloma developed after the FB was stable for 6 months in this laborer's hand. There was no gross infection, but the pain was quite bothersome. The FB and reactive tissue were excised under local anesthesia.

knife's blade may break off. Radiographs of animal or human bites should be used to assess for associated fractures. These films should be observed carefully for embedded teeth. Since the patient's perception of an FB correlates with the presence of an FB, *it is suggested that the clinician ask the patient if he or she thinks a wound may harbor foreign material*. In fact, Steele and coworkers found the positive predictive value of patient perception was 31% and negative predictive value was 89%.[20]

Many, but not all, retained FBs will produce pain when the patient moves the injured area or when the wound edges are palpated or depressed. It is prudent to carefully palpate the periphery of all wounds to elicit such tenderness. Superficial FBs may be palpated through the skin, but surprisingly large FBs may be found in seemingly minor wounds, without much external evidence. While puncture wounds are more likely to contain an FB than wide, gaping lacerations, the external characteristics of the wound do not yield firm evidence as to the presence or absence of an FB.

GUIDELINES FOR FOREIGN BODY REMOVAL

Following the initial history, examination, and *preoperative and preanesthetic documentation of the neurovascular status of the patient*, a decision must be made as to the time and place of removal. If the decision is made to have the FB removed in the ED, a 30-minute time limit is a reasonable self-imposed limit for the provider when attempting the removal of an FB. More difficult procedures should be referred. Many FBs appear superficial on radiographs, suggesting that removal will be quite easy. However, surprisingly large or presumed superficial FBs often prove quite elusive.

Localization

Superficial FBs, such as splinters, bullets, or embedded glass, may be palpated if they are near the skin surface. Deeper FBs must be localized by other techniques. A metal probe may identify the FB by feel or sound. Since glass is difficult to identify by sight in soft tissue, touching it with metal causes a characteristic grating sound. Because of the increasing incidence of human immunodeficiency virus (HIV), *probing a wound with a gloved finger to locate or identify an FB is strongly discouraged* (Fig. 37-8), since sharp objects can easily penetrate a gloved finger, exposing the clinician to bloodborne diseases such as HIV and hepatitis.

Radiographs are the best method for estimating the general location, depth, and structure of radiopaque FBs. If one strategically attaches a marker (needle or paper clip) to the skin surface at the wound entrance before taking a radiograph, the FB will be seen in relation to the entrance wound. This also helps to identify the path that leads to the FB and the relative distance from the surface to the FB. [21] Needles at two angles may also be used to aid localization (Fig. 37-9). A special technique using measurements on anteroposterior and lateral radiographs and a blind dissection method have been advocated to remove needles from the foot. [22] High-resolution US, CT, or MRI should be considered when the FB is suspected of being difficult to visualize on a conventional radiograph.

While rarely available, fluoroscopic image-intensifying equipment may be used to follow a wound's entrance, localize

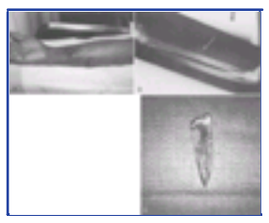

Figure 37-7 This intoxicated patient punched out a window and sustained seemingly minor puncture wounds *(A)*. He did not believe that glass was in the wound, there was little pain, and no FB could be palpated externally. A radiograph *(B)* revealed a large shard of glass deeply embedded in the wound *(C)*.

Figure 37-8 What's wrong with this picture? Although historically suggested as a useful technique to find FBs, probing the depths of a wound with a gloved finger may result in a puncture wound in the operator. The practice is strongly discouraged because of the prevalence of hepatitis and human immunodeficiency virus infections.

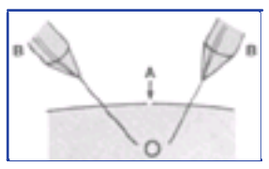

Figure 37-9 When a small entrance wound *(A)* is noted but the foreign body (FB) is not seen, noninvasive localization is preferable to blind probing. Metal markers taped to the skin or needles inserted close to the FB under local anesthesia *(B)* and radiographed at different angles provide a guide to FB localization and extraction. *(Reproduced from Hospital Medicine, © January 1981, with permission of Cahner's Publishing Co.)*

the material, grasp the FB, and remove it without making a larger incision. A potential disadvantage of this procedure is the increased amount of radiation that may be required. Ariyan has described a technique in which two needles are placed in the soft tissue from opposite directions, pointing toward the FB. [23] The extremity is rotated while the clinician watches the image under the image intensifier to obtain a three-dimensional effect. An incision is placed perpendicular to the plane of the needles, and the object is removed. US can also be used to remove an FB under direct visualization, and is particularly useful for radiolucent FBs.

Some authors have suggested injecting the entrance wound with methylene blue to outline the tract of the FB. [24] The blue line of injected dye is followed into the deeper tissues. This technique is of limited value, because the tract of the FB often closes tightly and does not allow passage of the methylene blue.

Equipment

A standard suture tray with a scalpel is usually adequate equipment for removal of most simple FBs. Tissue retractors, special pickups, and loupes may be added if needed. Since good light is essential, many clinicians use a headlamp. And, sitting on a stool makes the procedure easier on the clinician's legs and back.

Local soft tissue injection with buffered bupivacaine or lidocaine (with epinephrine for other than digital blocks) is the recommended anesthesia for removal of most soft tissue FBs. Regional intravenous anesthesia or selected nerve blocks may also be useful. *The judicious use of sedation (parenteral, rectal, or oral) is strongly advised if the clinician senses undue apprehension or anxiety in the patient.* This may be especially helpful in children. If the patient is totally uncooperative, exploration should be postponed for a more appropriate time (e.g., under regional or general anesthesia in the operating room).

To successfully remove an FB in the soft tissue of an extremity, an arterial tourniquet will provide a bloodless field. A blood pressure cuff or portable self-contained pneumatic cuff inflated above arterial pressure may be used on the upper arm, forearm, leg, or thigh. To limit bothersome backbleeding, the extremity needs to be elevated and wrapped with an elastic bandage to exsanguinate the extremity before inflating the tourniquet. In the digits, a Penrose drain or specialized tourniquet may be used as a tourniquet at the base of the finger or toes. A sterile glove may also be used as a finger tourniquet. The fingertip of the glove on the involved finger is cut, and the glove is rolled down to the base of the finger. Most patients can tolerate an ischemic tourniquet for 15 to 30 minutes, and it is safe to stop circulation to an extremity for this time period.

Operative Technique

The specific technique for removal of an FB is tailored to each clinical situation. In general, FBs should be removed only under direct vision. *Blind grasping into a wound with a hemostat in an effort to remove an FB should be avoided.* This technique is especially dangerous in the hand, foot, neck, or face, where vital structures

may be easily damaged.

In most cases one should *enlarge the entrance wound with an adequate skin incision*. Attempting to remove an FB through a puncture wound or an inadequate skin incision is a common

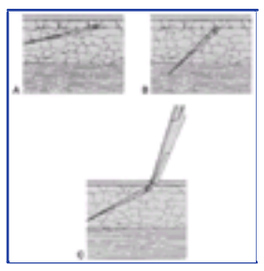

Figure 37-10 A sewing needle completely embedded below the surface *(A* and *B)* is easily located by a radiograph. Following local anesthesia, a small incision over the superficial end permits removal with a hemostatic forceps *(C)*. The hemostat is introduced through an adequate incision, spread to open the tissue, and used to "feel" the foreign body as the hemostat is advanced. *(Reproduced from Hospital Medicine, © January 1981, with permission of Cahner's Publishing Co.)*

error that is both frustrating and self-defeating. Following a proper skin incision, the wound is explored by carefully spreading the soft tissue with a hemostat (Fig. 37-10). Occasionally, the FB can often be felt with an instrument before it can be seen. In an extremity that has been made ischemic by a tourniquet, the tract of the FB may be followed, although the tract frequently cannot be identified in muscle or fat.

If the FB is difficult to visualize (such as with fiberglass or plastic FBs) or if it is located in the superficial soft tissue, excision of a small block of tissue, rather than removal of the FB alone, may be necessary. If the FB has contaminated the surrounding soft tissue, a block excision may also be required. It must be noted that excision of a block of tissue is done only under direct vision and after nerves, tendons, and vessels have been identified and excluded from the excision.

If an FB such as a thorn or needle enters the skin perpendicularly, a linear incision may pass to one side of the FB, and it will be difficult to determine where the FB lies in relation to the incision (Fig. 37-11*A* and *B*). The search must then be extended into the walls of the incision rather than through the skin. [25] In such cases it is advisable to excise a small ellipse of skin and undermine the skin for 0.5 to 1.0 cm in all directions (Fig. 37-11*C*). The tissue is then compressed from the sides in hopes that the FB will extrude and can be grasped with a hemostat.

Following FB removal, the wound is irrigated under pressure with saline. If a small incision has been made in a noncosmetic area (such as the bottom of the foot), the incision is left open and bandaged. The area may be soaked in hot water for a few days, and a return visit is necessary only if signs of infection develop. If a large incision has been created, the skin may be primarily sutured. In cases in which gross contamination has occurred, the wound should not be closed on the initial visit. The wound may be packed open and the

Figure 37-11 If a linear skin incision is used to locate a mobile foreign body (FB) that is perpendicular to the skin in the subcutaneous fat *(A),* the FB may be displaced *(B).* A modified elliptical incision is made *(C),* and the skin edges are undermined, displacing the FB into the middle of the wound. Pressure with the thumbs may be applied to the skin to force the FB into view. *(From Rees CE: The removal of FBs: A modified incision. JAMA 113:35, 1939. Copyright 1939, American Medical Association. Reproduced with permission.)*

skin may be subsequently sutured in 3 to 5 days, if free of signs of inflammation (delayed primary closure). Although the use of postoperative antibiotics is not standard, tetanus prophylaxis should be given. Antibiotics may be indicated for immunocompromised patients, but there are no data to support the routine use of antibiotics for wounds that have been thoroughly cleaned and in which all foreign material has been removed. Prophylaxis may be considered if there was excessive time between injury and removal, obvious contamination, or when the ability to adequately clean a wound is suspect. However, under these circumstances it is more prudent to opt for an open wound and a delayed closure. If prophylactic antibiotics are prescribed, 5 days of a penicillinase- resistant penicillin or first-generation cephalosporin should suffice. Under certain circumstances (e.g., stepping on a nail or a marine FB), alternative antibiotics may be indicated.

Puncture wounds to the bottom of the feet, especially through rubber-soled shoes, have been associated with *Pseudomonas* infections. Saltwater marine FBs may be contaminated with *Vibrios* species, usually sensitive to tetracyclines, aminoglycosides, or third-generation cephalosporins. Even if all foreign material has been removed, stings from marine animals may initiate a prolonged local irritation that simulates cellulitis. *However, the presence of a wound infection on a subsequent visit strongly raises concern for an occult retained FB rather than the simple conclusion that the wound is merely infected by bacteria introduced during the initial insult.*

Traumatic Tattooing

Ground-in foreign material or tattooing of the skin is a difficult problem because permanent disfigurement may occur. These injuries occur most often from falls on blacktop surfaces or asphalt, or falls from bicycles or motorcycles on a variety of surfaces. Many cases may be managed with adequate local anesthesia and meticulous debridement with a sponge, scrub brush, or toothbrush. If all foreign material cannot be removed with these methods, careful consideration should be given to a secondary excision of the tattooed area and primary closure with subsequent plastic surgery to repair the defect. However, it is usually impossible to completely remove traumatic tattooing in the ED. Referral for more extensive surgical treatment following local wound care is quite acceptable. Dermabrasion may be an acceptable delayed treatment when tattooing is superficial. [26] An alternative, although not currently available in EDs, is to refer patients to a dermatologist for laser removal of traumatic tattoos. Certain lasers have proved to be an excellent alternative. [27] The process is more specific in removing embedded material without harming surrounding tissue. The type of material will determine the number of treatments that are required. Not all tattoos can be completely removed. [28]

Foreign Bodies in Fatty Tissue

FBs located in fatty tissues may be removed by making an elliptical incision surrounding the entrance wound; grasping the incised skin loosely with an Allis forceps; undercutting the incision until the FB is contacted; and removing the FB, skin, and entrance tract in one block (Fig. 37-12). In most instances, a small portion of subcutaneous (SQ) fat should be removed along with the FB to minimize infection. FBs in fat are very mobile, and probing may displace them even further. FBs that are embedded in fat and are perpendicular to the skin may also be removed, as shown in Figure 37-11 .

Puncture Wounds to the Sole of the Foot

FBs may be introduced with puncture wounds to the sole of the foot (Fig. 37-13). A common scenario is stepping on a nail while wearing tennis shoes and socks. If the patient reports that the nail was removed intact, the possibility still remains that small fragments or fibers from the shoe or sock were introduced into the wound. When present in the wound, this material will invariably serve as a nucleus for infection. Therefore, these wounds should always be evaluated for the presence of foreign material, and some may require exploration. Proper evaluation requires adequate lighting, with the patient placed prone on a stretcher. A magnifying device may be helpful. Most retained material is usually radiolucent and routine radiographs are only required if bony injury is suspected. If a deep FB is suspected, CT, MRI, or US may be indicated.

Figure 37-12 Foreign bodies (FBs) in deep fat *(A)* may be approached by a small elliptical incision around the entry point *(B)*. The incision is then laterally undercut and grasped (without pulling) with an Allis clamp *(C)*. The ellipse is then further undercut until contact with the FB is made *(D)*. The FB may be grasped and removed along with the entry tract and the soiled local fat *(E)*. *(Reproduced from Hospital Medicine, © January 1981, with permission of Cahner's Publishing Co.)*

Initially, one visualizes the punctured area searching for obvious threads or pieces of rubber by merely elevating the small flap of calloused epidermis that commonly covers the entrance wound. Superficial and readily seen FBs, including small pieces of rust or debris, are often present. When foreign material is found, it should be removed by excising a small piece of tissue. A large laceration under the skin flap also necessitates local wound exploration. Although local anesthesia is often sufficient, regional nerve blocks for the sole should be considered if extensive exploration or debridement is anticipated (see Chapter 32). The surface of the site is irrigated, left open, and appropriately bandaged. Generally this is all that is required under these circumstances, but patients should be instructed to elevate the extremity for 24 to 48 hours so as to reduce swelling and help prevent hematoma formation.

If an infection is already present, the incidence of a retained FB is high. The puncture site may be cored out with a 2- to 4-mm-diameter incision carried up to 2 cm deep, superficially irrigated, and left open. It is best to avoid advancing an irrigating catheter into the deep recesses of the tract, as this serves no useful purpose and may disseminate infection.

Routinely removing a plug of tissue or coring a noninfected wound with no visible foreign material is quite controversial. [29] [30] This empiric procedure has been advocated by some, but it increases patient discomfort and disability, and has never been shown to reduce the incidence of serious sequelae. It is best reserved for patients with early inflammation or other findings on wound inspection that suggest a contaminated wound or a retained FB. It is likely over-treatment in the majority of cases.

A first-generation cephalosporin or a penicillinase-resistant penicillin is a reasonable antibiotic choice for obviously infected puncture wounds. These infections will be evident within a few days following the puncture. Noninfected puncture wounds have not been shown to benefit from antibiotics and their use is discouraged, except in high-risk patients. The vast majority of patients who step on a nail will have transient discomfort and will not develop complications. Although rare, the most dreaded complication of a deep puncture wound of the foot is *Pseudomonas* osteomyelitis (an organism that may live in the rubber soles of tennis shoes), but there is no evidence that routine use of prophylactic antibiotics will prevent this complication. In fact, routine antibiotics can theoretically select out resistant organisms. Lavery and coworkers demonstrated that *Pseudomonas* was the most common cause of osteomyelitis among nondiabetic patients, while polymicrobial infections and *Staphyloccoccus aureus* were more common in diabetic bone and soft tissue infections.[31] Diabetic patients are also three times more likely to develop osteomyelitis after a puncture wound compared to non-diabetics. Therefore, diabetic patients should be considered differently with respect to puncture wounds of the foot. *The key to a successful outcome of any puncture of the foot is to realize that pain, drainage, or swelling that persist for more than a few days after stepping on a nail, or that develop after a period of clinical quiescence, may represent the beginning of a serious but indolent infection or signal a retained FB.* Early in the course of a deep *Pseudomonas* infection, or osteomyelitis, the physical examination may be quite normal except for persistent aching or pain when walking. Fever is typically absent. A complete blood cell (CBC) count and sedimentation rate are usually normal. Plain films will be of no value. In this case, a bone scan or MRI may be used to evaluate for a deep FB or osteomyelitis.

Subungual Foreign Bodies

Special attention is required for deeply embedded subungual FBs. [32] Some cases may require removing a small portion of the nail with double-pointed heavy scissors and grasping the foreign material with splinter forceps (Fig. 37-14). Occasionally, complete removal of the nail may be required. A digital block should be used for any manipulation of the nail or nailbed.

An interesting technique has been suggested in which a sterile hypodermic needle, bent at its tip, is slid under the nail, hooking the FB, thereby allowing its withdrawal. Alternatively, a 19-ga hypodermic needle can be slid under

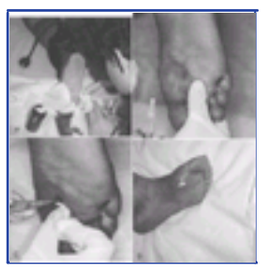

Figure 37-13 The vast majority of patients who step on a nail have no long-term consequences, but a few experience soft tissue infections or serious and debilitating osteomyelitis. The depths of a fresh puncture wound are not routinely explored, but key to avoid missing a superficial foreign body (FB) is to examine the patient under good lighting, supine, and with an arterial tourniquet if necessary (*A*). If the patient was wearing socks or tennis shoes, a small FB, like this piece of rubber, may be introduced into the wound. It may only be evident when the edges of the skin are trimmed and the wound is explored (*B*). Coring out the puncture wound tract is controversial and unproven, but it is one way to remove foreign material. In this case a No. 11 blade is inserted to the hilt and a 2 to 3 mm core of tissue is removed with the FB (*C*). This tract may be left open or a small packing applied. If a patient returns with an infection within a few days after a seemingly simple puncture wound (*D*), it is most likely a soft tissue infection, but persistent pain demands a search for osteomyelitis or a deep FB.

the nail to surround a small splinter. The needle tip is then brought against the underside of the nail to secure the splinter. The needle and splinter are then removed as a unit.[33]

A technique that can be performed by dermatologists and plastic surgeons is CO_2 laser vaporization. Starting a few millimeters from the nail edge, the nail is vaporized above the FB, allowing exposure. The FB can then be grabbed with forceps and removed. The advantages include decreased nail bed injury, less nail removal, faster recovery, and no bleeding. [34] Wooden splinters are commonly embedded under the fingernail. *Such FBs must be completely removed, because subsequent infection is certain.* If complete removal cannot be achieved with the techniques described earlier, the entire nail must be removed. This will allow all fragments to be visualized and removed. The proximity of the distal phalanx to the subungual area is a constant concern for the development of osteomyelitis.

Fishhooks

There are several methods of removing a fishhook. The preferred method depends on the location, depth of penetration, and conditions under which removal is to take place.[35] [36] [37] Initially, it should be noted whether the fishhook is single, multiple, or trebled. In addition, note the number and location of barbs. Any remaining exposed hooks should be removed or covered before removal in order to prevent subsequent injury. As with most injuries, vascular and neurologic status should be documented before and after removal.

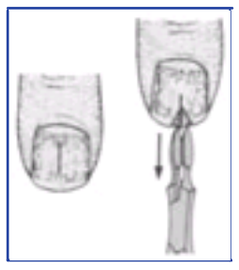

Figure 37-14 For an FB deep in the nailbed, take as small a wedge of nail as will allow access to the proximal end of the splinter, then extract the FB with splinter forceps. All wood particles should be removed. A digital nerve block is usually necessary. *(Reproduced from Hospital Medicine, © January 1981, with permission of Cahner's Publishing Co.)*

Traditionally, four removal techniques have been described: advance and cut technique, string-yank technique, needle cover technique, and retrograde technique. [38]

Advance and cut technique.

The advance and cut technique for removal requires advancement of the fishhook and cutting proximal to the barb. This method is particularly well suited for superficially embedded fishhooks. Generally, local anesthesia (1% lidocaine) is infiltrated into the tissue overlying the barb; the barb is forced through the anesthetized skin and clipped off, while the rest of the hook is removed retrograde, along the direction of entry (Fig. 37-15). The advantage to this technique is an almost always successful outcome.

String-Yank technique.

In the field or stream, removal of a fishhook may be accomplished without local anesthetic using a string-yank technique. This technique may be used in the ED, as well. Some clinicians prefer to use local 1% lidocaine to facilitate removal. This "stream" technique (Fig. 37-16) uses a looped string or fishing line passed around the belly of the hook at the point at which it enters the skin. Approximately 1 foot of string should be wrapped around the dominant hand to give strong traction, the shank of the hook should be held parallel to and in approximation with the skin by the index finger of the opposite hand, and the thumb and middle finger of the opposite hand should stabilize and depress the barb, which helps the index finger to disengage the barb from the SQ tissue. When the barb has been disengaged, a *sharp* pull (i.e., a quick tug with a snapping motion) with the dominant hand removes the hook. Care should be taken to keep bystanders out of the expected path of the hook

Figure 37-15 Method of removing an embedded fishhook when anesthesia is available and when the point of the fishhook is close to the skin. *A,* Obtain local anesthesia overlying the point of the hook. *B,* Force the point through the anesthetized skin. *C,* Clip off the barb. *D,* Remove the rest of the hook by reversing the direction of entry. *(Reproduced from Hospital Medicine, © July 1980, with permission of Cahner's Publishing Co.)*

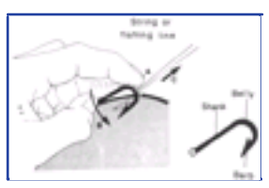

Figure 37-16 Method of removing an embedded fishhook when anesthesia is unavailable or when the barb of the fishhook lies too deep to force it out through a second wound without causing significant additional damage. Loop a piece of string (or thick suture material) around the belly of the hook and hold it down against the skin with the index finger of the left hand *(A).* Depress the shaft of the hook against the skin with the middle finger and thumb while applying light downward pressure with the index finger of the left hand to disengage the barb from the subcutaneous tissue *(B),* and pull *sharply* on the ends of the string with the right hand *(C)* to remove the hook through its entry wound. Bystanders should be out of the expected path of the hook. *(Reproduced from Hospital Medicine, © July 1980, with permission of Cahner's Publishing Co.)*

as the hook often flies out of the patient. A commercial fishhook extractor device that is based on this method but grasps the hook during removal is available (Minto Research and Development Inc., Redding, CA).

Needle cover technique.

Alternatively, an 18-ga needle may be used to cover the barb (Fig. 37-17). After adequate local anesthetic has been administered, the needle should be passed through the entrance wound of the hook parallel to the shank of the hook to sheath the barb and allow the hook to be backed out while the barb is covered. An alternative to this procedure is to insert a No. 11 blade parallel to the shank of the hook down to the barb, and using the point of the blade, free the SQ tissue that is engaged on the barb, cover the barb with the point of the No. 11 blade, and back the hook out, with the blade protecting the barb.

Retrograde technique.

This is the simplest but least successful method. By applying downward pressure to the shank of the hook, one may disengage the barb, allowing for successful removal. If resistance is observed, the procedure should be abandoned and another technique attempted. If the

Figure 37-17 Method of removing an embedded fishhook using anesthesia when the hook is large and not too deep in the skin. After anesthetizing the area with 1% lidocaine *(A),* insert a short-bevel 18-ga needle through the entry wound of the hook and attempt to sheathe the barb of the hook within the needle *(B).* If this is done correctly, the hook and needle may then be backed out together *(C). (Reproduced from Hospital Medicine, © July 1980, with permission of Cahner's Publishing Co.)*

barb does not already protrude from the skin, the retrograde technique may cause less tissue trauma. As with all wounds, tetanus prophylaxis should be administered if indicated; however, prophylactic antibiotics are not necessary.

Wooden Splinters

Because of the potential for inflammation, *pieces of wood must be completely removed from soft tissue*. By simply grasping the end of a superficial, protruding splinter, it may be adequately removed, but care should be taken not to leave small pieces of material in the wound. Some splinters cannot be visualized at the point of entry but can be easily and readily palpated beneath the skin. When a wood FB is SQ, it is advisable to cut down on the long axis of the FB to remove it via a skin incision, rather than pulling it out through the entrance wound (Fig. 37-18 Fig. 37-18). Although an incision may seem extensive and creates a laceration where only a puncture wound existed, opening the tract allows for thorough cleaning and mandatory removal of small pieces of the splinter that may otherwise remain. If the incision is linear, it may be sutured.

Particular mention should be made of certain wood splinters that are pliable and reactive, such as California redwood and northwest cedar. Any wood that is easily fragmented requires meticulous care to ensure removal of all material. Wood is generally not visible on a standard radiograph unless it is covered with lead paint. US is the technique of choice to evaluate for a wood FB, but CT or MRI may also be helpful. [39] Occasionally, the fastest method of removing small wooden splinters is complete excision of the entrance tract and the FB en bloc, followed by a linear closure.

Pencil Lead

Good judgment must be used in removing graphite from pencils when lodged in the skin. Embedded graphite is radiopaque. Because graphite invariably leaves a pigmented tattooing in the soft tissue, it is preferable to excise the material en bloc when pencil lead is found in a cosmetic area. The graphite specks cannot be irrigated or scrubbed off, and tattooing results if they are not removed. Furthermore, pencil-lead FB may resemble malignant melanoma over time and US has been

shown to be useful in differentiating between pencil-lead FB and melanoma. [40]

Metallic Fragments

High-velocity fragments (e.g., bullets, BBs, or other metallic particles caused by metal striking metal) are easy to visualize radiographically and relatively simple to remove if embedded in areas that are anatomically accessible. Before removal, the area in which the fragments are embedded should be assessed to determine which structures are involved and which structures might be encountered during removal. It is preferable to defer the removal of deeply embedded metallic FBs unless symptoms of infection develop.

The treatment of high-velocity FBs and their associated wounds (as seen with modern military or sporting ammunition) is beyond the capacity of this discussion. Retained nonexplosive metallic fragments are inert and rarely cause infection. They usually become encysted after a period of time. Lead toxicity from bullet fragments is rare, but lead fragments that are in contact with synovium are reason for concern and immediate removal (see previous discussion). In one study, Farrell and colleagues found that patients with retained lead FBs had statistically significant elevated blood lead levels. [41] The study did not differentiate the location of the FB and whether patients were, in fact, symptomatic from plumbism. Nevertheless, it may be prudent for the patient's primary care provider to monitor blood lead levels in patients with known lead FBs to prevent the development of future lead toxicity. The value of routine prophylactic antibiotics for metallic FBs left in the soft tissue has not been proven.

A sterile magnet may be used to facilitate removal of small metallic fragments. [42] The magnet is introduced into the entry site using a small scalpel and hemostat to extend and open the wound as needed. When the magnet comes in contact with the metal, a click is heard and the FB is removed while attached to the magnet. If resistance to extraction occurs, exploration can be done with the FB attached to the magnet.

Taser darts used for immobilization of violent individuals are like fishhooks and must be removed under local anesthesia after the attached wires have been cut. Backing the dart out, as with a fishhook, is not possible due to the dart's unique construction. Cutting down on the dart under local anesthesia is the preferred method of removal. [43] [44]

Marine Foreign Bodies

While most marine FBs, such as shell fragments, may be treated like other FBs, a number of marine animals carry toxins and may leave FBs that require special consideration.

Catfish.

Several species of catfish in North America contain toxic venom, and their sting can embed a FB. Envenomation occurs when the fish becomes agitated. Its dorsal and pectoral spines become locked in the extended position and penetrate the hand or foot. The venom is then

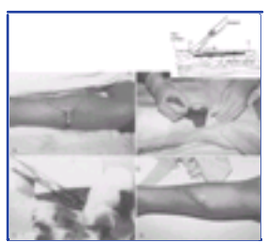

Figure 37-18a The course and depth of penetration of this large wood splinter of the leg *(A)* is uncertain, but it is axiomatic that all pieces of wood must be removed to prevent infection. To ensure complete FB removal, an incision is made over the entire course of the splinter *(B)* (*see inset*). All pieces of wood are carefully removed under direct vision *(C),* and the laceration is sutured primarily *(D)*. While it may be tempting to simply pull out the splinter and irrigate the puncture tract, such actions often lead to retained particles and complications.

secreted from an epidermal gland at the base of the spine. [45] The pain is usually ephemeral and since no specific antitoxin exists, treatment consists of local care and analgesics. The affected part should be immersed in hot water (approximately 43.3°C [110°F]) for at least 30 minutes if pain is severe. This is believed to provide symptomatic relief by decreasing vascular and muscle spasm. [46] Local injection of the wound with alkalized bupivacaine provides local analgesic and may also neutralize the toxin. [45] The wound should be inspected and any remaining spine removed. A radiograph may be taken to confirm the absence of FBs, but catfish spines and cartilage may be radiolucent. The wound should be thoroughly cleaned and irrigated. Tetanus prophylaxis should be updated. Some authors recommend empirical antibiotic therapy to cover gram-negative bacilli (e.g., *Aeromonas hydrophila*), but infection is quite rare, and routine antibiotic prophylaxis is not standard. [45]

Coelenterates.

Coelenterates including the Portuguese man-of-war, true jellyfish, fire coralbox jellyfish, and sea anemones inject several different toxins that are responsible for many marine envenomations. Reactions may be local or systemic, and pain may be severe, often described as shock-like, itching, burning, or throbbing. Substances such as tetramine, histamine, and 5-hydroxytryptamine are thought to be responsible for the localized reaction. Proteinaceous substances are implicated in the systemic response. Systemic reactions usually consist of fever, chills, and muscle spasm, but severe reactions may result in neurologic sequelae ranging from malaise and headache to paralysis and coma. Cardiopulmonary manifestations including dysrhythmias, hypotension, syncope,

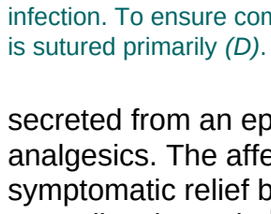

Figure 37-18b Here is another example of longitudinal incision of the overlying skin followed by withdrawal of a wood foreign body en mass to avoid retained fragments (*E*, *F*, *G*, and *H*.)

bronchospasm, laryngeal edema, and cardiorespiratory failure have been reported. [47] [48] Coelenterates deposit their venom-containing organelles, called nematocysts, onto the victim's skin (Fig. 37-19). The tentacles of coelenterates contain thousands of nematocysts and even minimal contact causes tentacles to adhere to skin. Nematocysts are then released from the tentacles onto the skin and discharge venom. Decontamination and removal of unfired nematocysts will decrease pain and systemic reactions.

Vinegar (5% acetic acid) is the initial decontaminating agent of choice and will inactivate unfired nematocysts of most species of jellyfish, Portuguese man-of-war, and sea anemones. [47] [49] This should be applied continuously for 30 minutes, or until the pain is gone. [48] Fresh water should not be applied to the area, for the osmotic shock will activate any unfired remaining nematocysts, thus discharging toxin into the dermis and increasing pain and toxicity. Other suggested, but unproven remedies, include meat tenderizer, ammonia, baking soda, urine, olive oil, sugar, and papaya latex.

After the wound has been decontaminated, any remaining tentacle fragments should be removed. Large fragments may be removed with forceps. Individual nematocysts are very small (<1 mm in length) and are not easily visible. If pain persists, it should be assumed that organelles still remain and further cleansing is required. To remove remaining nematocysts, the skin should be scraped with a hard edge, such as a credit card held perpendicular to the skin. Applying shaving cream and shaving the area gently may also remove the remaining fragments. [48] Note: Nematocysts should not be removed by rubbing the skin with sand, as this

increases venom discharge. In addition, the application of warm or cold packs to the area has not been shown to reduce pain. [49]

After decontamination, topical anesthetics or steroids may be used but prophylactic antibiotics are not necessary. Routine wound care should be followed. Allergic and systemic reactions should be treated appropriately. Antivenin is available only for box jellyfish envenomation, but its use remains incompletely understood and controversial.

Coral.

Fire corals are another type of coelenterate that produce toxicity by the presence of stinging nematocysts. After contact, a burning and intense pruritis may occur along with a series of skin eruptions. Within minutes of contact, urticaria-like lesions may appear and blister formation may result within hours. Eventually, the lesions will become lichenoid and complete resolution may not occur for 15 weeks following contact. Ultimately, hyper-pigmented areas where initial contact occurred will result. Immediate care with oral antihistamines and topical steroids tends to reduce,

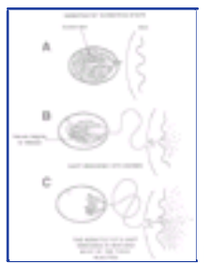

Figure 37-19 Magnified view of venom-containing nematocyst in its resting or "cocked" state *(A)*. After contact with the skin, a dart-like tail is extended and penetrates the dermis *(B),* and venom is injected *(C)*. Further stimulation of the attached nematocyst can expel more venom (see decontamination/removal technique in text).

but not prevent, symptoms. Other corals are sharp and irritating if touched. Coral is comprised of a calcium carbonate core and thousands of small marine animals. "Coral cuts" occur in divers and snorkelers exploring coral reefs. These organisms can produce deep lacerations, with local reactions such as pruritus, erythema, and urticaria occurring at the site of injury. Delayed healing may occur with secondary development of cellulitis or ulceration, perhaps as a result of contamination of the wound with bacteria or microparticles of coral. [47] Treatment consists of saline irrigation. Hydrogen peroxide may be used to remove small coral particles from the wound. [47] The wound should not be closed; wet-to-dry dressings may be used.

Sponges.

Sponges produce both an irritant and a contact dermatitis. The irritant dermatitis occurs as a result of sponge spicules embedded in the victim's skin. These spicules may be removed with adhesive tape (applied to the skin and then peeled back) and the area should then be bathed with vinegar. [47] The contact dermatitis, which is believed to be caused by a toxin, produces erythema, pruritus, and vesicles similar to poison oak. [47] Treatment is initial immersion in vinegar followed by local steroid creams.

Sea urchins and starfish.

Sea urchins and starfish are free-living echinoderms covered with venomous spines, sharp and brittle, and venom-secreting pincers located near the mouth. If sea urchins or starfish are handled or inadvertently stepped on, these spines may become embedded in the patient and a severe local reaction may result from venom contained in the spines. Systemic symptoms including muscle weakness, paralysis of lips, tongue, face, hypotension, abdominal pain, and respiratory distress may occur. Local pain usually responds quite well to immersion in hot water (43.3°C to 46.1°C [110°F to 115°F]) for 30 to 90 minutes. Retained spines may become infected or cause delayed (up to 1 to 2 months) FB granulomas. This reaction is not adequately understood but may produce an intense and persistent inflammatory reaction. Spines that penetrate joints may induce a synovitis. Echinoderm spines may discharge a purple dye that may be mistaken for a retained spine. [48] Spines are usually visible on radiographs and should be removed if possible, although they are brittle and can break off in the skin. If located in a joint or near a nerve, surgical extraction may be necessary using an operative microscope. [47] Otherwise, if removal is difficult, the spine may be left in place until it resorbs or a local reaction takes place. The wound may then be opened and drained and allowed to close by secondary intention.

Stingrays.

Stingray envenomation usually occurs by accidentally stepping on the creature that is resting on the bottom in shallow water, covered by sand (Fig. 37-20). This causes the stingray to lash out its whip-like caudal appendage, or tail, which contains 1 to 4 venom-containing serrated spines. Portions of the spine may become buried in the victim's skin. Each spine is covered with a sheath containing venom glands, and in addition to immediate toxin-induced pain, pieces of the spine or sheath may remain embedded in the wound. These fragments, while often difficult to locate, do not dissolve and must be removed. Persistent pain and inflammation, even weeks to months after the attack, mandate consideration of a retained FB, but a persistent and difficult-to-treat irritative process can occur in the absence of a retained spine or sheath. Immediate local and systemic reactions occur as a result of injection of a complex toxin. Systemic reactions may be severe and can include muscle cramps, vomiting, seizures, hypotension, arrhythmias, and (rarely) death.

Treatment consists of irrigation with saline followed by hot water immersion at 43.3°C to 46.1°C (110°F to 115°F) for 30 minutes to 1 hour in order to inactivate the heat-labile toxin. Local digital blocks without vasoconstrictors provide effective analgesia for hand wounds. All wounds should be explored and debrided, all remnants of the spine and integumentary sheath removed. [47] Wounds should heal by secondary intention. The venom can cause significant local tissue necrosis, and surgical debridement may be required.

Antibiotic therapy.

Prophylactic antibiotic therapy for marine injuries is common, although there are no convincing data to support or refute the practice. Unlike other soft tissue infections, marine injuries become infected with unusual gram-negative organisms, particularly *Vibrio* species. Although there are few studies evaluating the effects of specific antibiotics, it is recommended that quinolones, trimethoprim-sulfamethoxazole, tetracyclines, third-generation cephalosporins, or aminoglycosides be used in lieu of penicillin, ampicillin, erythromycin, or first-generation cephalosporins. [48] It is always difficult to differentiate chronic inflammation caused by toxins and foreign material from true infection, and often surgical exploration is required in persistent cases. Tetanus prophylaxis should be routine.

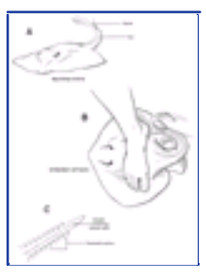

Figure 37-20 *A,* Stingray resting on the bottom of the ocean, usually covered by a layer of sand. An unsuspecting victim steps on the stingray, and the whiplike tail impales the foot (even through a heavy boot) with one or more spines *(B).* The spine has backward-facing barbs covered by a sheath with venom-containing cells (*C*), causing a toxic envenomation and the potential for multiple FBs.

Cactus Spines

The sizes of cactus spines fluctuate considerably. The difficulty of removal is generally inversely proportional to the FB size. [50] Larger embedded cactus spines are managed like wood splinter and sea urchin spine FBs. More advanced imaging techniques (US, CT, or MRI) may be required for localization of deeply embedded spines.

Deeply embedded cactus spines generally produce granulomatous reactions, and infections are rare. [19] Hence, efforts to remove deeply embedded spines should be made after carefully weighing the benefit and potential harm related to a deep exploration, especially in a sensitive location. [50] Using forceps, superficially embedded, medium- to large-sized cactus spines are best removed by direct axial traction of each spine. Smaller spines (glochids) may be difficult and tedious to remove individually. Adherent facial mask gel application and removal of spines en masse with the mask are recommended (Fig. 37-21). Depilatory wax melted in a microwave oven and applied warm, commercial facial gels, and household glue (Elmer's Glue-All [Borden Inc., Columbus, OH])

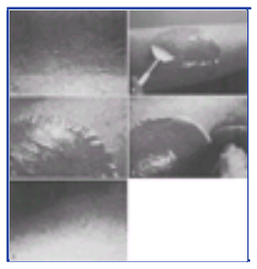

Figure 37-21 *A,* A thousand or more glochids affixed to the skin by contact with a single pad of polka dot cactus. *B,* The *professional* facial gel is spread with a fan brush. *C,* The gel is spread thin at the edges. *D,* The gel rollup is started by picking at the edge with the fingernails. *E,* When the *professional* gel is peeled off, all of the very small spines come with it. *(From Lindsey D, Lindsey WE: Cactus spine injuries. Am J Emerg Med 6:362, 1988. Reproduced by permission.)*

have all been recommended for this purpose. [50] [51] [52] [53] Over-the-counter "home use" facial mask gels are not adherent enough to be effective without multiple applications (up to eight or more).

Body Piercing and Removal

The art of body piercing predates most history books. Over the last decade, an enormous increase in the practice of body piercing has occurred. With these piercings have also come the complications associated with the practice. For centuries, the most common place was the ears. Today, the lips, tongue, eyebrow, nose, navel, nipples, and genital areas have become sites of body piercing. To date, there are a limited number of studies on post-piercing infections in areas other than the ears. Three major types of jewelry are used: (1) barbell studs, which are straight bars with a ball threaded onto both ends; (2) labret studs, which are straight bars with a ball threaded on one end and a disc permanently fixed on the other end (more commonly used on the lips); and (3) a captive bead ring, which consists of a bead with small dimples on opposite sides, held "captive" by tension from both sides of an incompetent ring. The "bead ring" is a variation of this: One bead is permanently fixed to one end, and an opening is made by removing the free end of the ring. [54]

The most common reason for removal is infection. Other symptoms such as bleeding, edema, allergic reaction, and keloid formation may prompt removal. Occasionally, tongue piercings must be removed to permit intubation. In order to remove the barbell and labret type studs, one may hold the bar with forceps and unscrew the bead on the other end. To remove the captive bead ring, hold the ring on both sides of the captive bead to release the tension on the bead. This will dislodge the bead from the ring, which is holding it in place. If the jewelry is near the mouth or nose, care must be taken to prevent aspiration of the bead. The microbiology of infections related to body piercing has not yet been determined. However, organisms such as *Staphylococcus epidermidis* and *S. aureus*, along with *Pseudomonas aeruginosa*, have been commonly implicated pathogens. Other infectious complications from body piercing such as septic arthritis, endocarditis, hepatitis B and C, and HIV have been reported. [55] [56]

Most commonly, however, local wound infections predominate and can be managed with warm compresses, antibacterial soap, and topical antibacterial ointment once the FB is removed.

Postoperative Foreign Bodies

FBs in the form of non-absorbed suture material are frequently encountered in the postoperative period. Drainage, localized pain, tenderness, and inflammatory reaction along the suture line are characteristic of a retained FB (suture abscess). In this instance, probing the wound with a sterilized needle bent in the shape of a crochet hook is frequently successful. Hooking the suture material through the sinus tract and removing it allows the wound to heal over the tract.

Ring Removal

Frequently, a ring must be removed to prevent laceration of tissue or vascular compromise. The use of thorough lubrication (a water-soluble lubricant [e.g., K-Y jelly]) and a circular motion with traction on the ring are usually sufficient. However, the string-wrap method or physically cutting off the ring may be necessary. Preferably, all rings should be removed before edema is extensive enough to cause pain or vascular compromise.

String-wrap method.

An occasional patient can remain calm during this procedure, but if swelling is significant or the digit has been traumatized, anesthesia is necessary (Fig. 37-22 Fig. 37-22). A proximal digital or metacarpal block provides sufficient anesthesia and helps to minimize tissue distention at the ring site. Before ring removal, a wide Penrose drain is wrapped circumferentially in a distal-to-proximal direction to reduce soft tissue swelling, and the wrap should remain in place for a few minutes to reach the maximum effect. Some non-anesthetized patients panic during the procedure because of increasing pain due to compression and unwinding. [57]

A 20- to 25-inch piece of string, umbilical tape, or thick silk suture is first passed between the ring and finger. Shorter lengths are discouraged, as one may need to repeat the wrapping procedure midway. If there is marked soft tissue swelling, the tip of a hemostat may be passed under the ring to grasp the string and pull it through. The distal string is wrapped clockwise around the swollen finger (proximal to distal) to include the proximal interphalangeal (PIP) joint and the entire swollen finger. The wrapping starts next to the ring. The wrap should be snug enough to compress the swollen tissue. Successive loops of wrap are placed next to each other to keep any swollen tissue from bulging between the strands. When the wrapping is complete, the proximal end of the string is carefully unwound in the same clockwise direction, forcing the ring over that portion of the finger that has been compressed by the wrap. The PIP joint is the area that is most difficult to maneuver and causes the most pain to the patient.

Occasionally, the finger must be rewrapped if it was not carefully done initially. It is not uncommon to produce abrasions or other trauma to the skin during this procedure. If the finger with the ring is lacerated or there are underlying fractures, it is prudent to cut off the ring instead of attempting this technique.

Another alternative uses an entire skein of 2-0 surgical silk ties instead of a single piece of string. The procedure is the same. The ties are passed underneath the ring with a hemostat. The short ends of the ties are clamped with a hemostat on the hand side of the ring. The long end of the skein is wrapped around the finger and then unwound from the short end, as described earlier. This method may be less traumatic to the finger than a single piece of string. [58]

Certain rings are made of extremely hard materials such as tungsten carbide or ceramic. In these cases, cracking the material with standard vice grip pliers can break

the ring. Place the pliers on the ring and adjust the jaws to fit tightly, then remove and readjust, increasing the tension with each subsequent adjustment. Continue until the material cracks and falls apart. Some rings may be lined with a metal band. A standard ring cutter can then be used to remove the band.

Ring cutter.

A ring cutter should be used when there is excessive swelling or other methods fail. Manual and power devices are available. The ring cutter has a small hook that fits under the ring and serves as a guide for a saw-toothed wheel that cuts the metal. The cut ends of the ring are spread using large hemostats (e.g., Kelly clamps), and the ring is removed. If the tension is too great to spread the ring, another cut 180° apart from the original ring cut can be performed. This will allow the ring to fall off in two pieces. A jeweler can subsequently repair cut rings.

Tick Removal

The early removal of ticks is recommended, since the hard tick of the ixodid family is likely to transmit disease. Rocky Mountain spotted fever, Lyme disease, tularemia, and ascending paralysis have been identified as tick-borne diseases. It is important to note that the rate of disease transmission before 48 hours of attachment is exceedingly small.[59] Tick removal of Ixodid ticks is difficult, because the mouthparts become cemented within 5 to 30 minutes of contact with the host's skin (Fig. 37-23*A*). Removal will become more difficult the longer the tick is attached. Inadequate or partial removal of the entire tick may cause infection or chronic granuloma infection. Traditional and folk methods of forcing the tick to disengage (e.g., the use of petroleum jelly, fingernail polish, a hot match, or alcohol) are not advised. Removal

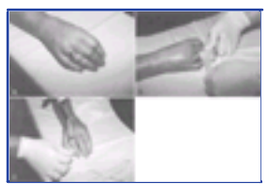

Figure 37-22a String removal technique for a tight ring. Note the absence of a laceration or underlying fracture *(A)*. A digital or metacarpal block is suggested. Edema is lessened by compression of the finger with a Penrose drain *(B)*. A long piece of umbilical tape is placed under the ring with the winding portion left long *(C)*.

by mechanical means is recommended.[60] Nonmechanical means of tick removal can cause the tick to regurgitate, increasing the possibility of infectious transmission.

Straight or curved forceps or tweezers is the recommended method of removal. If these instruments are not available, a gloved hand will suffice. Grasp the tick as close to the patient's skin surface as possible and gently apply steady axial traction (Fig. 37-23*B*). Take care not to squeeze or crush the tick body as this may expel infective agents. Do not twist or jerk the tick in order to prevent the mouthparts from breaking off during extraction. If mouthparts are left behind after removal of the body, they may be removed with tweezers. If one is still unable to remove the mouthparts, excision under local anesthesia will be needed to prevent local infection.

Many patients have great anxiety over subsequent tick-borne diseases following tick removal. Recent work has demonstrated that single-dose doxycycline (200 mg) may prevent the development of Lyme disease.[59] However, it is still not reasonable to treat all patients. When patients are in areas where the incidence of Lyme disease is high, or when a partially engorged deer tick in the nymphal stage is discovered on them, they are more likely to benefit from prophylaxis. Regardless of whether prophylaxis is given, patients should be instructed on the symptoms and signs of Lyme disease and encouraged to return or seek medical evaluation.

Zipper Injuries

The skin of the penis may become painfully entangled in a zipper mechanism. Unzipping the zipper frequently lacerates the skin and increases the amount of tissue caught in the mechanism. Although the clinician may anesthetize the skin and excise the entrapped tissue, a less invasive method may be useful.

Cutting the median bar between the faceplates of the zipper mechanism remains the most common method. The interlocking teeth of the zipper then fall apart when the median bar (diamond or bridge) of the zipper is cut in half (Fig. 37-24 (Figure Not Available)) and the skin is subsequently freed. A bone cutter or wire clippers and a moderate amount of force may be required to break the bar. The addition of mineral oil followed by traction has been demonstrated with some success. Patients with penile lacerations warrant urologic follow-up to assess for urethral injury.

Hair-Thread Tourniquet

Hair or thread fibers adherent to infant clothing occasionally become tightly wrapped around a child's digits or genitals (Fig. 37-25).[61] If these are left in place, amputation may eventually occur. The offending fibers may be difficult to visualize, and the child is often brought for evaluation only after signs of distal ischemia appear. Occasionally, the fiber can be grasped with toothless forceps or a small hemostat and unwrapped. More commonly, fibers cannot be identified because they are deeply embedded in swollen tissue.

Figure 37-22b Begin winding either clockwise (*inset*) or counterclockwise (*photograph*) to compress the skin *(D) (see details in inset)*. The ring is removed by unwrapping the tape proximally and in the same direction it was wrapped *(E* and *F)*. The most difficult area to negotiate is the proximal interphalangeal joint. Note abrasion of skin after removal.

A No. 11 blade can be used to cut the constricting bands under a regional nerve block. [62] It may be difficult to identify individual hairs that are deeply embedded in a swollen digit and even more difficult to assess the success of the intervention. Often, multiple hairs are involved. Because the bands may be quite deep, the incision should avoid known neurovascular tracts. Barton and colleagues recommend a dorsal, rather than lateral, incision on the digits. [62] If the soft tissue of the distal digit has been rotated following a circumferential dermal laceration from the tourniquet, the distal tissue can be realigned with the proximal tissue and two dorsolateral sutures placed or tissue adhesive glue applied to maintain the digit in alignment.

Generally, conservative wound care is sufficient once the band has been removed. Application of an antibiotic ointment may enhance healing and allow easier removal of serous drainage from the circumferential laceration. Clinical reassessment in 24 hours will indicate whether any constricting bands remain.

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Figure 37-23 Ticks should be removed as soon as possible to minimize the transmission of tick-borne pathogens and to limit their fixation to the skin by a secreted cement compound. This engorged tick has been attached for about a day *(A)* and has burrowed under the skin. Most home remedies are worthless. A recommended approach is to grasp the tick with forceps near its head *(B)* where it enters the skin (avoid the soft body) and gently pull it out. Some advise twisting the head counterclockwise, but this has not objectively been found to be more effective. If pieces of the tick remain, they should be dug out.

Figure 37-24 (Figure Not Available) When loose skin is caught in the teeth of a zipper (*A*) one can release it quickly and without risk to the patient by cutting the diamond that holds the slider together with a bone cutter or a pair of wire clippers. *B, C,* Alternatively, the zipper teeth can be separated by cutting the cloth between the teeth, either above or below the zipper head. The head is then moved forward or backward. Local anesthesia may be injected in to the incarcerated skin if this procedure is painful. *(From Emergency Medicine, October 15, 1982, p. 215. Used by permission.)*

Figure 37-25 This child has multiple hairs compromising the circulation to two toes. The hairs are deeply embedded in the skin creases and cannot be visualized. The only way to ensure removal of the constriction is to cut the depth of the folds with a scalpel blade (using a dorsal incision to avoid the neurovascular bundle) and attempt to extricate individual fibers. Return of circulation should be obvious by temperature and color change in the affected digit(s), before it is assumed that all of the fibers have been cut.

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Chapter 38 - Incision and Drainage

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Incision and drainage in the emergency department (ED) is most commonly done for soft tissue abscesses. Similar techniques are used for foreign body excision (see Chapter 37) or drainage of hematomas or seromas. The latter topic is discussed at the end of this chapter.

Much of this chapter deals with cutaneous abscesses, which account for 1% to 2% of ED visits. In contrast to most bacterial diseases, which are usually described in terms of their etiologic agent, cutaneous abscesses are best described in terms of their location. There has been little systematic investigation into the bacteriology of simple cutaneous abscesses, and there have been few new recommendations for improved management over the years. The probable reason for this lack of scientific rigor is the predictable and striking clinical recovery once a mature abscess is incised and drained. The exact reasons for this amelioration of local and constitutional symptoms are unknown; however, it is clear that the exact bacteriology of cutaneous abscesses, while academically interesting, is essentially inconsequential to the final outcome in most cases.[3]

ABSCESS ETIOLOGY AND PATHOGENESIS

Localized pyogenic infections may develop in any region of the body and usually are initiated by a breakdown in the normal epidermal defense mechanisms, with subsequent tissue invasion by normal resident flora. Thus, an abscess is likely to be caused by the flora that are indigenous to that area. An exception is direct inoculation of extraneous organisms, such as infections that follow mammalian bites.

Staphylococcal strains, which are normally found on the skin, produce rapid necrosis, early suppuration, and localized infections with large amounts of creamy yellow pus. This is the presentation of a typical abscess. Group A ß-hemolytic streptococcal infections, on the other hand, tend to spread through tissues, causing a more generalized infection characterized by erythema and edema, a serous exudate, and little or no necrosis. This is the presentation of a typical cellulitis. Anaerobic bacteria proliferate in the oral and perineal regions, produce necrosis with profuse brownish, foul-smelling pus [3] , and may cause both abscess and cellulitis.

Normal skin is extremely resistant to bacterial invasion, and few organisms are capable of penetrating the intact epidermis. In the normal host with intact skin, the topical application of even very high concentrations of pathogenic bacteria does not result in infection. The requirements for infection include a high concentration of pathogenic organisms, such as occurs in the hair follicles and their adnexa; occlusion, which prevents desquamation and normal drainage, thus creating a moist environment; adequate nutrients; and trauma to the corneal layer, which allows organisms to penetrate. [4] Trauma may be the result of abrasions, hematoma, injection of chemical irritants, incision, or occlusive dressings that cause maceration of the skin. Foreign bodies can also potentiate these infections and decrease the number of bacteria necessary for infection. An example of this is the ubiquitous suture abscess, which frequently develops in otherwise clean wounds closed with suture material. In recent years, body piercing has become a widespread fashion trend in western industrialized countries. Unfortunately, this body art is not without complication. The invasive application of ornaments through cutaneous and mucosal surfaces allows the penetration of various pathogens into subcutaneous (SQ) tissue.[5]

When favorable factors are present, normal flora of cutaneous areas can then colonize and infect the skin. The body area involved depends primarily on host factors. In persons performing manual labor, the arms and the hands are infected most frequently. In women, the axilla and submammary regions are frequently infected because of minor trauma from shaving and garments and because of the abundance of bacteria in these areas. IV drug users may develop infections anywhere on the body, although the upper extremities are most commonly affected.[6] [7] Deep soft tissue abscesses have also been described following attempts at access to the deep venous structures in addicts who have exhausted all peripheral venous access sites. [8] In addition, areas with compromised blood supply will be more prone to infection, because normal host cell-mediated immunity is not as available. [4] Septic emboli from endocarditis may cause abscess formation by bacteremic migration of infected material into SQ tissue.

Infections in the soft tissue often begin as a cellulitis. Some organisms cause necrosis, liquefaction, and accumulation of leukocytes and debris, followed by loculation and walling off of pus, all of which result in the formation of one or more abscesses. There may be involvement of the lymph tissues, producing lymphangitis and subsequent bacteremia. As the process progresses, the area of liquefaction increases until it "points" and eventually ruptures into the area of least resistance. This may be toward the skin or the mucous membrane, into surrounding tissue, or into a body cavity. If the abscess is particularly deep-seated, spontaneous drainage may occur, with persistence of a fistulous tract and the formation of a chronic draining sinus. This development, or the recurrence of an abscess that has been previously drained, should always suggest the possibility of osteomyelitis, a retained foreign body (FB), or the presence of unusual organisms such as *Mycobacterium* or *Actinomyces*.[3]

Bacteriology of Cutaneous Abscesses

Meislin and coworkers[1] cultured abscesses in 135 patients, and their report typifies the bacteriology and natural history of cutaneous abscesses. Their patients received simple incision and drainage, and all subjects were followed as outpatients. Both aerobic and anaerobic cultures were taken. Most (96%) cultures were positive for bacteria (Table 38-1).

In this series predominantly mixed aerobic bacteria were isolated in abscesses of the trunk, axilla, extremities, and the hand. In pure cultures, *Staphylococcus aureus* was found in 72% of cases. One third of the cultures from the perianal region contained only anaerobes. Mixed cultures of both aerobic and anaerobic bacteria were obtained from all sites of the body, but there was a 67% incidence of such mixed cultures from the perirectal area. Commonly isolated anaerobes included various *Bacteroides* spp, peptococci, peptostreptococci, *Clostridium* spp, *Lactobacillus* spp, and *Fusobacterium* spp.

TABLE 38-1 -- Characterization of 135 Outpatient Abscesses*

			Type of Bacterial Growth (Percent from Each Area)				Bacterial Species per Abscess*	
Anatomic areas	***Abscesses***	***Percent of total cultures***	***No growth***	***Aerobes only***	***Anaerobes only***	***Aerobes and anaerobes***	***Aerobes***	***Anaerobes***
	No.						*average no.*	
Head and neck	25	19	4	28	20	48	1	2
Trunk	11	8	0	45	18	36	1	2
Axilla	22	16	0	55	5	41	1	1
Extremity	16	12	19	44	13	25	1	1
Hand	8	6	25	63	0	13	2	0
Inguinal	7	5	0	29	57	14	0	3
Vulvovaginal	13	10	0	15	46	38	1	3
Buttock	12	9	0	33	33	33	1	3
Perirectal	21	16	0	0	33	67	1	5

From Meislin HW, Lerner SA, Graves MH, et al: Cutaneous abscesses: Anaerobic and aerobic bacteriology and outpatient management. Ann Intern Med 87:146, 1977. Reproduced with permission.

*Cultures with no growth were excluded.

Bacteria from abscesses in areas remote from the rectum were generally aerobic strains and were primarily indigenous microflora of the skin. *S. aureus* was the most prevalent aerobic organism; it was isolated in 24% of all abscesses.

Gram-negative aerobes were isolated infrequently from cutaneous abscesses. *Escherichia coli, Neisseria gonorrhoeae,* and *Pseudomonas* spp were rarely found. The most commonly isolated gram-negative organism was *Proteus mirabilis*. This organism was found almost exclusively in the axilla, and its presence may be related to

the use of underarm deodorants.[9]

Brook and Finegold[10] studied the bacteriology of cutaneous abscesses in children. Their results closely correlate with those of Meislin and associates.[1] Brook and Finegold found aerobes (staphylococci and group A ß-hemolytic streptococci) to be the most common isolates from abscesses of the head, neck, extremities, and trunk, with anaerobes predominating in abscesses of the buttocks and perirectal sites. Mixed aerobic and anaerobic flora were found in the perirectal area, head, fingers, and nailbed area. This study found an unexpectedly high incidence of anaerobes in nonperineal abscesses. Anaerobes were found primarily either in areas adjacent to mucosal membranes, where these organisms tend to thrive (e.g., the mouth), or in areas that are easily contaminated (e.g., by sucking fingers, which causes nailbed and finger infections or bite injuries).

Parenteral drug users develop somewhat atypical abscesses. The injection of a cocaine/heroin mixture ("speedball") may predispose users to abscesses by inducing soft-tissue ischemia.[11] Webb and Thadepalli[12] found anaerobes to be a major pathogen, regardless of anatomic location. After comparing the bacteriology of abscesses in intravenous (IV) drug abuser and non-drug-users, Summanen and colleagues[13] reported isolates of oral origin in 67% and 25%, respectively. Some injection drug users lick their needles prior to injection for "lubrication", perhaps accounting for the presence of unusual oral pathogens as *Eikenella corrodens*. Schnall and associates[14] found that skin abscesses at the site of injection contain predominantly staphylococcal and streptococcal species. Four of the 20 staphylococcal isolates were oxacillin-resistant. Anaerobes and gramnegative bacilli accounted for 24% of the isolates. Because IV drug use is associated with immunodeficiency syndromes, unusual isolates such as *Candida albicans*[15] and acid-fast bacilli[16] [17] have been obtained.

The polymicrobial nature of abscesses does not lend itself to a strict scientific interpretation of culture results, but if an unexpected or atypical organism is found in an abscess culture, the clinician should consider an underlying process not readily apparent from the history or physical examination. Most typical is finding tuberculosis or fungal isolates in immunocompromised patients (e.g., those with diabetes or acquired immunodeficiency syndrome [AIDS]). Finding *E. coli* suggests an enteric fistula or even self-inoculation of feces in some patients with a psychiatric illness such as Munchausen syndrome. Recurrent abscesses without an obvious underlying cause could indicate clandestine drug use ("skin popping"). What appears to be a typical recurrent abscess may be a manifestation of an underlying septic joint or, rarely, metastatic or primary cancer (Fig. 38-1).

Special Considerations

Parenteral drug users, insulin-dependent diabetics, hemodialysis patients, cancer patients, transplant recipients, and individuals with acute leukemias have an increased frequency of abscess formation compared with the general population. Local symptoms may not be the primary complaint, and the patient may present only with an exacerbation of the underlying disease process or an unexplained fever. These abscesses tend to have exotic or uncommon bacteriologic or fungal causes and typically respond poorly to therapy.[12] [14] [15] [16] The diabetic patient with diabetes-induced ketoacidosis should be evaluated extensively for an infectious process; a rectal examination should be included with the physical examination to rule out a perirectal abscess. This also holds true for other patients with abnormal cell-mediated immunity. The increased frequency of abscess formation in these patients and in the parenteral drug user is multifactorial. There may be intrinsic immune deficiencies in all these patients; they have an increased incidence of *Staphylococcus* carriage, and they have frequent needle punctures, which allow access by pathogenic bacteria.[18]

It is important to note that a substantial percentage of abscesses in parenteral drug users are sterile and are the result of the injection of necrotizing chemical irritants. Drug users frequently use veins of the neck and the femoral areas, producing abscesses and other infectious complications at these sites.[19] Any abscess near a vein of the antecubital fossa or dorsum of the hand should alert the clinician to possible IV drug use; however, substance users may also inject directly

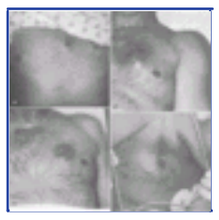

Figure 38-1 An abscess that appears in an atypical place or recurs after initial treatment is successful should raise the possibility of rare or underlying conditions. *A*, This IV drug user had an "abscess" of the chest wall drained in various EDs several times over a 2-month period, and it seemed to initially respond to drainage and antibiotics. He still manifested an area of cellulitis, minor fluctuance, and continued drainage near the center of the chest. This is an atypical place for a simple cutaneous abscess. Magnetic resonance imaging demonstrated an abscess of the sternolclavicular joint that was draining to the skin, simulating a recurrent cutaneous abscess. He required extensive surgical debridement and prolonged antibiotics. The etiologic organism was never ascertained, but pseudomonas is often present. *B*, This patient has a large "abscess" of the lateral chest wall that initially drained unusual gelatinous material, not frank pus. *C*, At follow-up 3 days later the abscess was much improved. The contents of the abscess had been sent for pathologic analysis since it had an unusual consistency, and it demonstrated a highly undifferentiated malignancy. The fluid was sterile. Normally, analyzing or culturing the contents of an abscess will not yield helpful information, but in this case the unusual consistency of the collection prompted further analysis. *D*, This patient had a sternotomy for bypass surgery a few months ago. She had been sporadically treated for a minor wound infection but then presented with a draining fluctuant mass at the inferior border of the sternum. This is the external manifestation of an extensive sternal osteomyelitis.

into the skin ("skin popping"), causing cutaneous abscesses away from veins (Fig. 38-2).

The presence of a FB may serve as a nidus for abscess formation. A history of a possible FB at the site of an abscess should be sought. Because IV drug users frequently break needles off in skin toughened by multiple injections, the clinician should maintain a high index of suspicion for retained needle fragments. If an abscess is recurrent or if the patient is a known or suspected IV drug user, radiographs or other techniques should be considered to search for foreign bodies, an underlying septic joint, or osteomyelitis.[20]

MANIFESTATIONS OF ABSCESS FORMATION

The diagnosis of cutaneous abscess formation is usually straightforward. The presence of a fluctuant mass in an area of induration, erythema, and tenderness is clinical evidence that an abscess exists. An abscess may appear initially as a definite tender soft tissue mass, but in some cases the presence of a distinct abscess may not be readily evident. If the abscess is quite deep, as is true of many perirectal, pilonidal, or breast abscesses, the clinician may be misled by the presence of only

Figure 38-2 This patient presented with a large abscess of the deltoid area (arrow) and could offer no explanation for it *(A)*. This is a typical scenario for a drug user who injects directly into the skin, and the characteristic circular skin lesion from "skin popping" found on the legs *(B)* confirmed the clinical suspicion. Even though a drug screen was positive for opioids, the patient still denied drug use and attributed the leg lesions to frequent trauma on the job.

a firm, tender, indurated area without a definite mass. To avoid misdiagnosis of an early abscess, one may aspirate the suspicious infected area with a needle and syringe to confirm the presence of pus.[21] This approach also may help identify a mycotic aneurysm or an inflamed lymph node simulating an abscess. A specific entity that is commonly mistaken for a discrete abscess is the sublingual cellulitis of Ludwig angina (see Chapter 66). Parenteral injection of illicit drugs can produce signs of simple cutaneous abscesses that unpredictably advance to extensive necrotizing soft-tissue infections. The emergency clinician must maintain a high index of suspicion to avoid missing this potentially serious condition.[22] Cellulitis and abscess formation may lead to bacteremia and sepsis, especially in the immunocompromised patient, and the presenting complaints may draw attention away from the primary focus of infection.[12]

The pain of an abscess often brings the patient to the hospital prior to spontaneous rupture. Often, however, the patient will present with a draining abscess that appears to have undergone spontaneous rupture and is manifesting a self cure. Also, the patient may have punctured the abscess in an attempt to drain it. In most cases a formal incision, drainage, and packing procedure will be helpful in eliminating the process, even though copious drainage will not be encountered. While no formal drainage may be required following the spontaneous rupture of simple cutaneous abscess, such conditions as a perirectal abscess, Bartholin gland abscess, or breast abscess often will be best served by further drainage and packing.

LABORATORY FINDINGS

Laboratory tests offer no specific guidelines for therapy of a cutaneous abscess and are not generally indicated.

An exception would be a blood or urine glucose determination to assess diabetes in patients with appropriate clinical scenarios. An abscess may produce leukocytosis, depending on the severity and duration of the abscess process; however, the majority of patients with an uncomplicated cutaneous abscess will have a normal complete blood cell (CBC) count and will not experience fever, chills, or malaise. *The presence or absence of leukocytosis has virtually no diagnostic or therapeutic implications.* In the absence of extenuating circumstances, it is not standard to send blood cultures on patients with typical cutaneous abscesses. Bacteremia may occasionally manifest a peripheral abscess from septic emboli, but this usually produces clinical characteristics that are dissimilar to cutaneous abscesses. The cutaneous abscess itself rarely produces the bacteremia.

Gram stain is not indicated in the care of uncomplicated simple abscesses.

Patients who appear "toxic" or immunocompromised and those patients who require prophylactic antibiotics (see Prophylactic Antibiotics later in this chapter) may rarely benefit from Gram stain and cultures. Gram stain results have been shown to correlate well with subsequent culture results, and in compromised hosts the test can be used to direct antibiotic choice. [21] [23] Anaerobic infections should be suspected when multiple organisms are noted on Gram

stain, when a foul odor is associated with the pus, when free air is noted on radiographs of the soft tissue, or when no growth is reported on cultures. [12]

In uncomplicated abscesses, *routine culture of pus is unnecessary* because of the expected prompt response to surgical therapy and the polymicrobial nature of abscess formation. However, in complicated cases; recurrent, unusual, or atypical abscesses; or in immunosuppressed patients, additional information may be gleaned if the abscess contents are sent for culture. The information obtained may later be useful if there is poor response to the initial surgical drainage, secondary spread of the infection, or the occurrence of bacteremia. [3] If one takes a culture, it is best to aspirate the pus with a needle and syringe *before* incision and drainage. Material should be cultured for aerobic and anaerobic bacteria. The finding of a "sterile" culture in an abscess that has been cultured with a standard cotton swab *after* incision is frequently the result of improper anaerobic culture techniques. As a side note, there is a general misconception that foul-smelling pus is a result of *E. coli*. This foul odor is actually caused by the presence of anaerobes; the pus of *E. coli* is odorless.

Unusual material from an abscess may prompt additional laboratory investigation. Occasionally a malignancy may mimic a cutaneous abscess (see Fig. 38-1*B, C*).

INDICATIONS AND CONTRAINDICATIONS

Surgical incision and drainage is the *definitive treatment* of a soft tissue abscess.[21] Antibiotics alone are *ineffective* in the setting of a localized cutaneous collection of pus. The drainage of a suppurative focus results in a marked improvement in symptoms and rapid resolution of the infection in uncomplicated cases. In the initial stages, only induration and inflammation of an area later destined to produce an abscess may be found. Premature incision before localization of pus will not be curative and theoretically may be deleterious, because extension of the infectious process and, rarely, bacteremia from manipulation can result. In some cases the application of heat to an area of inflammation may ease pain, speed resolution of the cellulitis, and facilitate the localization and accumulation of pus. It must be stressed that nonsurgical methods are not a substitute for surgical drainage and should not be continued for more than 24 to 36 hours before the patient is reevaluated. Diagnostic needle aspiration is recommended if one is unsure of pus localization.

ED bedside ultrasound can play a key role in the management of soft-tissue infections. It is an easily accessible image modality that can be used after plain radiographs have been obtained. Ultrasound offers a safe, real-time, convenient technique guiding needle aspiration of any suspicious fluid collection. [24] [25] ED ultrasound similarly can guide incisions in the setting of deep soft tissue infections (see Chapter 69).

ANCILLARY ANTIBIOTIC THERAPY

The use of antibiotics remains unproven for both prophylaxis and treatment of cutaneous abscesses. As an overview, there are no data that definitively demonstrate the need for antibiotic therapy in conjunction with incision and drainage of uncomplicated cutaneous abscesses in healthy, immunocompetent patients without valvular heart disease. The common practice of prescribing antibiotics if there is "significant surrounding cellulitis" is a vague concept that is not easily quantified, and is not supported by prospective data. The specific value of concomitant antibiotics in the immunocompromised patient, while intuitively attractive and commonly practiced, is also unproven. If antibiotics are prescribed, they should likely be given for 2 to 3 days only, and their continuation reassessed at the first follow-up visit. Since the bacteriology of abscess is complicated and multifactorial, no single specific antibiotic can be prospectively supported. Most clinicians use antibiotics that are generally effective for "soft tissue infections," but this concept is vague and frustrating to the clinician. While antibiotics are usually not harmful, the editors do not recommend the routine use of antibiotics for simple cutaneous abscesses unless there are extenuating circumstances. Unfortunately those circumstances are not clearly defined by scientific literature and remain in the realm of clinical judgment.

Patients with risk factors for endocarditis comprise a third group of patients. Because of the concern of inducing a bacteremia by manipulation of infected tissue, parenteral antibiotics are commonly given *prior to the incision-and-drainage procedure* in patients at risk from such bacteremia. Transient bacteremia has been documented (incidence range, 7% to 50%) following manipulation of noninfected tissue (brushing teeth, sigmoidoscopy, Foley catheter, rectal examination) and following simple incision and drainage of abscesses. While common, the clinical importance of transient bacteremia is uncertain. In a report by Fine and colleagues, 6 of 10 patients with cutaneous abscesses were noted to have positive blood cultures immediately following incision and drainage, whereas all cultures were negative before the procedure. [26] Blick and associates[27] evaluated the use of prophylactic antibiotics in abscesses managed in the operating room under general anesthesia. Three of 19 patients developed bacteremia after the procedure. A similar group treated with parenteral antibiotics before the procedure had only one patient with a positive blood culture. That culture yielded an organism resistant to the prophylactic antibiotic used.

Prophylactic Antibiotics

Prophylaxis for Endocarditis

The precise risk for endocarditis following incision and drainage of cutaneous abscess is unknown, and it is impossible to predict which patients will develop this infection or which particular procedure will be responsible. However, since bacteremia clearly occurs with manipulation of infected tissue, it is generally agreed that those patients at risk for cardiac complications related to transient bacteremia should be treated with appropriate antibiotics within the hour preceding the procedure. The transient bacteremia secondary to abscess drainage is of no proven concern in otherwise healthy, immunocompetent individuals without valvular heart disease. Bobrow and associates[28] demonstrated that incision and drainage of cutaneous abscesses in afebrile patients is unlikely to result in transient bacteremia.

No adequate controlled trials of antibiotic regimens appear in the literature, and data predicting the incidence of endocarditis in patients with preexisting cardiac disorders are lacking, thereby making the decision regarding antibiotic prophylaxis quite arbitrary. Although no definitive standards exist, the Committee on Rheumatic Fever and

Infective Endocarditis of the American Heart Association (AHA) has recommended that prophylactic antibiotics be given prior to incision and drainage of infected tissue in patients who have *cardiac lesions that place them at high risk for developing endocarditis*.[29] Table 38-2 (Table Not Available) lists conditions that may warrant antibiotic coverage. Durack also recommends prophylactic antibiotics for a variety of procedures known to have a significant incidence of bacteremia, including incision and drainage of cutaneous abscesses. [30] Interestingly, the antibiotic regimen suggested by the AHA (Table 38-3) does not include antibiotics effective against many organisms commonly found in some cutaneous abscesses, specifically *S. aureus*, but is mainly directed against *Streptococcus viridans* and enterococcus. These latter organisms have a predilection for valvular infection. The AHA recommends the same prophylaxis for incision of soft tissue infection that is recommended for genitourinary procedures, but with additional consideration of therapy "directed against the most likely pathogen." [31]

Two clinical situations deserve special note. Because of the frequent incidence of pre-existing valvular damage in the patient who uses IV drugs, prophylactic antibiotics may be indicated prior to the incision and drainage of abscesses in these patients. Clearly, any patient with a documented history of endocarditis must receive prophylactic antibiotics prior to the incision-and-drainage procedure. Because cutaneous abscesses may result from active endocarditis and prophylactic antibiotics may obscure subsequent attempts at identifying an etiologic organism, two or three blood cultures (aerobic and anaerobic) should be obtained from those at risk for endocarditis prior to antibiotic therapy. Patients with the diagnosis of mitral valve prolapse have traditionally been included for treatment with prophylactic antibiotics. The indication for this is unclear. The risk of an allergic reaction may outweigh the benefits of treatment in this group, [32] and clinical judgment is required. Kaye[33] suggests prophylaxis only for patients who have a holosystolic murmur secondary to mitral valve prolapse.

TABLE 38-2 -- Estimated Risk of Infective Endocarditis Associated with Bacteremia in the Presence of Preexisting Cardiac Disorders

(Not Available)

From Durack DT: Prevention of infective endocarditis. N Engl J Med 332:38, 1995.

Prophylaxis for Bacteremia in Other Conditions

Immunocompromised patients have not been adequately studied, but this is a subgroup that intuitively may benefit from prophylactic antibiotics. In contrast to patients with endocarditis risks, immunocompromised patients are at risk for developing septicemia secondary to a brief bacteremia. Since IV drug users have a high incidence of human immunodeficiency virus (HIV)-related disease,[34] [35] [36] the treating clinician must anticipate various degrees of immunodeficiency in these patients. Clinical judgment must determine the use of antibiotics in these situations.

No specific guidelines have been offered for the antibiotic regimen used prior to incision and drainage of infected cutaneous tissue in patients at risk for conditions *other than endocarditis*. Choice of antibiotics is guided by the organism anticipated to cause the bacteremia. Although the location of the abscess will give some clue to the organism involved, most abscesses contain multiple strains of bacteria. Not all bacteria are potent pathogens, so their mere presence does not predict their role in subsequent morbidity. Because *Staphylococcus* continues to be a significant organism in this setting, a broad-spectrum anti-staphylococcal drug is indicated. [6] Prophylaxis should consist of a single IV dose given a half-hour prior to incision and drainage. A first-generation cephalosporin or penicillinase-resistant penicillin is a good initial choice. Vancomycin may also be considered. Others may prefer cefazolin (Ancef, Kefzol), 1 g, IV, given a half-hour before surgery. This regimen covers staphylococcal and streptococcal species, many gram-negative organisms, and many anaerobes.

Although not widely used in the United States, parenteral antibiotics have been used to "sterilize" the abscess cavity following curettage in Great Britain. The concentration of clindamycin in the abscess cavity has been shown to be equivalent to the concentration of antibiotics found in the blood. [27] Some British articles reported success with "primary closure under antibiotic coverage," [37] [38] [39] although other British reports

TABLE 38-3 -- Prevention of Bacterial Endocarditis: Regimens for Genitourinary/Gastrointestinal Procedures [*]

Drug	Dosing Regimen[†]
Standard Regimen	
Ampicillin, gentamicin, and amoxicillin	IV or IM administration of ampicillin, 2 g, plus gentamicin, 1.5 mg/kg (not to exceed 80 mg), 30 min before procedure; followed by amoxicillin, 1.5 g orally 6 hr after initial dose; alternatively, the parenteral regimen may be repeated once 8 hr after initial dose
Ampicillin/Amoxicillin/Penicillin—Allergic Patient Regimen	
Vancomycin and gentamicin	IV administration of vancomycin, 1 g, over 1 hr plus IV or IM administration of gentamicin, 1.5 mg/kg (not to exceed 80 mg), 1 hr before procedure; may be repeated once 8 hr after initial dose
Alternative Low-Risk Patient Regimen	
Amoxicillin	3 g orally 1 hr before procedure; then 1.5 g 6 hr after initial dose

From Dajani AS, et al: Prevention of bacterial endocarditis. Circulation 83:1174, 1991.

*In the case of abscess drainage, include antibiotics directed against most likely pathogen if not included in this regimen.
†Initial pediatric doses are as follows: ampicillin, 50 mg/kg; amoxicillin, 50 mg/kg; gentamicin, 2 mg/kg; and vancomycin, 20 mg/kg. Follow-up dose should be one half the initial dose. Total pediatric dose should not exceed total adult dose.

continue to recommend the traditional procedure.[40] [41] One limitation to the abscess curettage technique is the need for general anesthesia, because the performance of curettage is extremely painful.

Therapeutic Antibiotics

In contrast to prophylaxis prior to surgery, the routine use of oral antibiotics following incision and drainage of simple cutaneous abscesses in otherwise healthy patients with no immunocompromise appears to have no value, and their empiric use cannot be scientifically supported. Llera and Levy [2] performed a randomized double-blind study to compare outcomes of patients treated with a first-generation cephalosporin following drainage of cutaneous abscesses in the ED. They found no significant difference in clinical outcome between the two groups and concluded that antibiotics are unnecessary in individuals with normal host defenses. This confirmed previous less well controlled studies.[27] [39] [40] It should be noted that high-risk patients were often excluded from these studies. The immunocompromised patient has not been adequately studied in this situation and is therefore often given antibiotics empirically, but this practice, while common, has not been supported by rigorous prospective studies.

Patients with cutaneous abscesses often have concomitant disease processes that may warrant the consideration of parenteral or oral antibiotics. Most abscesses have a defined surrounding area of induration, but this may not necessarily qualify as "significant surrounding cellulitis." While cellulitis or lymphangitis often accompany abscesses, and therapeutic antibiotics may have value under these circumstances, this concept is difficult to quantify and not well addressed nor supported in the literature. Cellulitis and lymphangitis usually subside following the draining of the abscess itself. Meislin [42] noted that pathogen identification in cases of cellulitis without abscess can be difficult, and empiric antibiotics may be helpful. IV drug users who present with an abscess and fever require parenteral antibiotic therapy after blood cultures have been drawn until bacterial endocarditis can be ruled out.[43] Obviously, patients who are clinically septic require immediate IV antibiotics as well as aggressive surgical drainage of pus. By administering IV ampicillin/sulbactam (2 g/1 g) every 6 hours, Talan and colleagues [44] achieved 100% eradication of pathogens from major abscesses in hospitalized IV drug users and non-drug users.

As a general guideline, therapeutic antibiotics should be considered for immunocompromised patients (e.g., patients with AIDS or diabetes, patients receiving chemotherapy or steroids, transplant recipients, and alcoholic patients) and to the immunocompetent patient with "significant" cellulitis, lymphangitis, or systemic symptoms, such as chills or fever. Although it has not been studied, it makes sense to also give *antibiotics prophylactically, before surgery, to all patients who will obviously be given therapeutic antibiotics*. As with prophylactic antibiotics, a first-generation cephalosporin or semisynthetic penicillin is a reasonable therapeutic choice unless the specific abscess site dictates alternative therapy. The ideal duration of therapeutic antibiotics is unknown. As a general guideline, immunocompromised patients should receive antibiotics for 5 to 7 days and immunocompetent patients, for 3 to 5 days after the procedure, depending on the severity of the condition and clinical response at follow-up.

Facial abscesses should be handled carefully and checked frequently. Any abscess above the upper lip and below the brow may drain into the cavernous sinus, and thus manipulation may predispose to septic thrombophlebitis of this system. Treatment with antistaphylococcal antibiotics and warm soaks following incision and drainage has been recommended pending resolution of the process. Areas *not* in this zone of the face can be treated in a manner similar to that used for other cutaneous abscesses.

INCISION-AND-DRAINAGE PROCEDURE

Procedure Setting

Definitive incision and drainage of soft tissue abscesses are performed in either the ED or the operating room (OR). When abscesses are drained in the ED, some centers prefer to use a special area to avoid contamination of general treatment rooms.

The choice of the locale for the procedure depends on a number of important factors. The location of the abscess may dictate management in the OR. Large abscesses or abscesses located deep in the soft tissues require a procedure involving a great degree of patient cooperation, which may only be possible under general or regional anesthesia. Proximity to major neurovascular structures, such as in the axillae or antecubital fossa, may necessitate specific management. Infections of the hand (with the exception of distal finger infections) have traditionally been managed in the OR because of the many important structures involved and the propensity for limb-threatening complications.

Lack of adequate anesthesia is the most common limiting factor in ED incision and drainage. If the clinician believes the abscess cannot be fully incised and drained because of inadequate anesthesia, the patient should be taken to the OR for management under general anesthesia. In addition to limiting proper drainage, it is inhumane and unethical to subject a patient to extreme pain when alternatives are available.

Equipment and Anesthesia

A standard suture tray provides adequate instruments if a scalpel and packing material are added. Although sterility is impossible during the procedure, one should avoid contamination of surrounding tissue. Some clinicians prefer to use an obligatory skin scrub with an antiseptic solution, but the value of this step is dubious.

It is often quite difficult to obtain local anesthesia by direct infiltration because of the poor function of local anesthetic agents in the low pH of infected tissue. Furthermore, the distention of sensitive structures by a local injection is quite painful and hence poorly tolerated by most patients. Skin anesthesia is usually possible, but total anesthesia of the abscess cavity itself generally cannot be achieved. If a regional block can be performed (see Chapter 31 Chapter 32 Chapter 33), this type of anesthesia is preferred. Alternatively, a field block may be used. It should be noted that infected tissue is very vascular, and local anesthetics are quickly absorbed. Strict adherence to maximum safe doses of the local anesthetic is required.

The skin over the dome of an abscess is often quite thin, making skin anesthesia difficult. If a 25-ga needle is carefully used, one can often inject the dome of the abscess subcutaneously. The anesthetic solution spreads over the dome through the SQ layers into the surrounding skin and provides excellent skin anesthesia. If the needle is in the proper plane (best accomplished by holding the syringe parallel, rather than perpendicular, to the skin), the surrounding skin blanches symmetrically during infiltration without having to reposition the needle (Fig. 38-3*A*). In the extremely anxious or uncomfortable patient, the judicious use of preoperative sedation (see Chapter 34) with IV opioids and sedatives or nitrous oxide makes the procedure easier for both patient and clinician. If adequate anesthesia cannot be obtained and pain limits the procedure, the patient should be treated under general anesthesia.

Some clinicians recommend the use of topical ethyl chloride or Fluori-Methane spray for the initial skin incision, but while this is an attractive concept to patients, the pain relief offered by these agents is variable and fleeting. Ethyl chloride is also highly flammable. These vapocoolant sprays may be useful to provide momentary anesthesia for local anesthetic injection or for the initial skin incision if the injection or incision is made immediately after blanching of the skin. In general, however, these agents are a poor choice for a stand-alone anesthetic agent for all but the smallest of superficial abscesses (e.g., purulent folliculitis).

Incision

One should make all incisions conform with skin creases or natural folds to minimize visible scar formation (Fig. 38-4). Extreme care should be taken in such areas as the groin, the posterior knee, the antecubital fossa, and the neck, so that vascular and neural structures are not damaged.

A No. 11 or 15 scalpel blade is used to nick the skin over the fluctuant area, and then a simple linear incision is carried the *total length of the abscess cavity* (see Fig. 38-3*B*). This will afford more complete drainage and will facilitate subsequent breakup of loculations. Attempting to drain an abscess with an inadequate incision is counterproductive and makes packing changes more difficult. A cruciate or "X" incision, or an elliptical skin *excision* is to be avoided in the routine treatment of cutaneous abscess. The tips of the flaps of a cruciate incision may necrose, resulting in an unsightly scar (Fig. 38-5). A timid "stab" incision may produce pus but is generally not adequate for proper drainage. *The scalpel is used only to make the skin incision and is not used deep in the abscess cavity*.

Exceptions to this rule regarding aggressive incision are abscesses in cosmetic areas, in areas under significant skin tension (e.g., extensor surfaces), or in areas with extensive scar tissue (e.g., sites of multiple prior drainage procedures). In these special circumstances, a stab incision or simple aspiration alone may be initially attempted, with the goal of limiting tissue injury and resultant scar formation. Use of this less aggressive approach requires that the patient be counseled that multiple decompressions (e.g., via needle aspiration) or delayed aggressive incision and drainage may be required. The abscess will need reassessment in 24 to 48 hours to determine the need for additional intervention.

Wound Dissection

Following a standard incision, the operator should probe the depth of an abscess to assess the extent of the abscess and ensure proper drainage by breaking open loculations (see Fig. 38-3*D*). An ideal instrument for this procedure is a hemostat wrapped in gauze (or a cotton swab for small abscesses) that is placed into the abscess and swirled around to all sides of the cavity (see Fig. 38-3*E*). Traditionally the operator's gloved finger has been suggested as an ideal way to assess the depth of the abscess cavity and to break up loculations, but *this is a potentially dangerous practice that should be avoided* unless it is certain that the abscess contains no sharp foreign body. Of particular concern is the abscess caused by skin popping or IV drug use. Such patients have a high incidence of hepatitis and HIV infection, and occasionally these abscesses harbor broken-off needle fragments (Fig. 38-6). One is often surprised at the depth or extent of an abscess during probing. Sharp curettage of the abscess cavity is usually not required and may produce bacteremia. [27] Although tissue probing is usually the most painful aspect of the technique and total local anesthesia is difficult to obtain, this portion of the procedure should not be abbreviated. If the procedure is limited because

Figure 38-3 *A,* To anesthetize an abscess, hold the syringe parallel to the skin and insert a 25-ga needle into the middle of the dome of the abscess, so the needle is just slightly under the skin. The anesthetic will spread over the entire abscess without moving the needle position. *B*, Note the blanching of the skin that spreads to cover the entire abscess without repositioning the needle. *C,* The skin over the abscess is incised the full length of the abscess. A small stab incision does not allow for proper drainage. *D,* An instrument is inserted into the abscess cavity to spread the skin, drain the pus, and break up loculations. *E*, One way to break up loculations is to wrap a hemostat with gauze and, *F*, swirl it around the margins of the wound. *Do not use a gloved finger to*

break up loculations in an abscess that may contain a sharp or jagged foreign body.

of pain, the use of appropriate analgesia/anesthesia is mandated. Failing to adequately pack the abscess on the first visit makes follow-up packing changes more problematic.

Wound Irrigation

Following the breaking up of loculations, some clinicians advocate copious irrigation of the abscess cavity with normal saline to ensure adequate removal of debris from the wound cavity. Although it may seem intuitively to be a helpful step, irrigation of the abscess cavity has not been experimentally demonstrated to significantly augment healing or affect outcome. Hyperemic tissue may bleed profusely, but bleeding usually stops in a few minutes if packing is used. Abscesses of the extremities can be drained during the use of a tourniquet to provide a bloodless field.

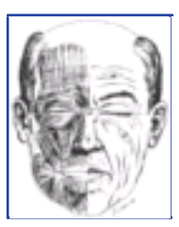

Figure 38-4 The relation of the elective lines of tension in the face to the underlying mimetic musculature. Only in the lower eyelid are these lines not perpendicular to the muscles. The left side of the drawing shows the use of this principle when common facial lesions are excised or a facial abscess is drained. *(From Schwartz SI, Lillehei RC: Principles of Surgery, 2nd ed. New York, McGraw-Hill, 1974. Reproduced with permission.)*

Packing and Dressing

After irrigation, a loose packing of gauze or other material is placed gently into the abscess cavity to prevent the wound margins from closing and to afford continued drainage of any exudative material that may otherwise be trapped. The packing material should make contact with the cavity wall so that upon removal, gentle debridement of necrotic tissue will spontaneously occur. A common error is to attempt to pack an abscess too tightly with excessive packing material. In essence, the pack merely keeps the incision open, and its main purpose is not to absorb all drainage—a dressing accomplishes this goal. Care must be exercised to ensure that the packing does not exert significant pressure against the exposed tissue and lead to further tissue necrosis. Some prefer to use plain gauze, some use gauze soaked in povidone-iodine, and some use gauze impregnated with iodine (iodoform). For large abscess cavities, gauze pads (without cotton backing) are ideal packing. *If gauze pads are used, the number of pads placed in the wound should be counted and charted—ideally, the corner of each pad should exit from the wound.* The clinician must ensure that all gauze pads will be removed when the packing is changed or discontinued. More commonly, thin (0.6 to 1.2 cm) packing strip gauze, either plain or iodoform, is used. The iodoform gauze may sting the patient for a few minutes after it is inserted. Packing, especially iodine-containing packing, will be radiopaque on a plain radiograph. If a FB is considered, the x-ray should be

Figure 38-5 A simple linear incision is preferred over an "X" or crosshatched incision. In this case, a cutaneous post-surgical scalp abscess was drained by an "X" incision and the tips of the flap necrosed, leaving a slowly healing full thickness wound.

obtained prior to packing. The value of antibiotic-impregnated gauze is uncertain.

An absorbent gauze dressing should be placed over the packed abscess, or, if an extremity is involved, a lightly wrapped circumferential dressing should be used. Generous amounts of dry gauze are used over the packing to soak up any drainage or blood. The affected part should be splinted if possible, and elevation should be routine. The dressing/splint should not be disturbed until the first follow-up visit. Drainage relieves most of the pain of an abscess, but postoperative analgesics may be required.

Following treatment, packing is often changed periodically. Most patients require a repeat visit to the clinician for packing change, but if the original packing is to be removed and not replaced (e.g., as with paronychia, hair follicle abscess), select patients may remove the packing and perform their own wound care totally at home.

FOLLOW-UP CARE

Reevaluation of a drained abscess should occur in 1 to 3 days, depending on a number of parameters. [21] [27] [40] Most lesions are reevaluated 48 hours following the procedure, with the first packing change occurring at this time. Some wounds warrant closer monitoring. Diabetic patients or other patients with impaired healing capacity, mental impairment, or physical disabilities may require a home care nurse or admission for more frequent wound care/packing changes. Wounds that are at high risk for complications, such as those about the face or hands or those with significant cellulitis, require close follow-up, ideally by the same examiner. The patient should be encouraged to play an active role in wound care. During the first follow-up visit, the compliant and able patient should be taught to perform packing or dressing changes, or both. If this is anatomically impossible, a friend or family member can be instructed in the technique.

Figure 38-6 *A,* This intravenous drug user presented with an abscess *(arrow)* of the antecubital fossa. Following incision, the clinician attempted to break up the loculations with his finger. When a radiograph was obtained, three needle fragments (arrows) were found embedded in the wound *(B).* The patient was HIV-positive and claimed no knowledge of the presence of the needles. Instead of using a finger, loculations should be broken up with an instrument.

The technique of packing change is usually one of personal preference. It should be emphasized that patients often fear a repeat visit and expect significant pain with subsequent wound care, especially if the initial incision drainage was difficult. Therefore, the specifics of packing change should be addressed prior to release home after the initial drainage procedure. Some clinicians suggest an oral opioid be taken 30 to 40 minutes before the next visit or use local anesthesia or parenteral analgesia if significant pain is anticipated. Removal of packing material is often painful, but if the packing is moistened with saline prior to removal, it may be less traumatic. If the original incision was of the proper length, loculations were adequately removed, and packing was adequate, the subsequent packing changing will be considerably easier. Once the packing is removed, the wound is inspected for residual necrotic tissue. The cavity may be irrigated with saline prior to replacing the pack if there is significant exudate, but this is often not required because the packing absorbs most debris.

The frequency of packing or dressing changes is also clinically guided. Some wounds require multiple packing changes, while other wounds require only the initial packing. In all facial abscesses, the packing should be removed after only 24 hours, at which time warm soaks should be started. Wounds large enough to require packing should be repacked at least every 48 hours (occasionally daily for the first few visits) until healing continues in a deep to superficial direction. Large wounds that are allowed to close superficially will create an unsterile dead space that will potentiate the formation of recurrent abscesses. After the first few days (and in the motivated and compliant individual), an alternative to packing is to have the patient clean the base of the abscess three times a day with cotton swabs soaked in peroxide (Fig. 38-7). This promotes drainage, produces gentle debridement, and keeps the incision open. Following cleaning, the abscess can be irrigated with tap water and a dry dressing applied.

In general, once healthy granulation tissue has developed throughout the wound and a well-established drainage tract is present, the packing may be discontinued. The patient should then be instructed to begin warm soaks of the wound. [21] Gentle hydrostatic debridement may be performed by the patient in the shower at home. With this procedure the patient holds the skin incision open and directs the shower or faucet spray into the abscess cavity. Wet-to-dry normal saline dressing changes should then follow until healing is completed. When all signs of infection (e.g., erythema, drainage, pain, and induration) have resolved and healthy granulation tissue is present, the patient may be discharged from medical care.

Complicated wounds that require prolonged care are best followed by a single clinician and should not be routinely referred back to the ED for prolonged wound care. Large perirectal or pilonidal abscesses are some conditions meeting that definition. These patients should receive early referral to their primary care clinician or specialist. Wound care centers or physical therapy departments are ideal outpatient follow-up mechanisms. Wounds in cosmetically important areas may require revision once healing is complete. Patients should be informed of this possibility early on in their care.

In select cases in which extensive or prolonged drainage occurs or in patients who are unable to return for proper follow-up care, a catheter system of drainage may be preferred. [45] [46] Following incision, a balloon-tipped or flared-tip catheter is placed into the abscess cavity, and pus is allowed to drain continuously through the catheter lumen. This technique has been most successful in pilonidal and Bartholin gland abscesses, but the technique is applicable to any abscess *not* on the face.

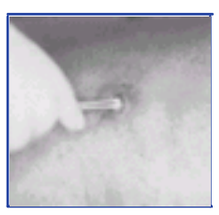

Figure 38-7 Packing change is usually performed by a health care professional. In the compliant and motivated patient, home care may replace frequent visits. Cotton-tipped applicators dipped in peroxide are used to swirl around the cavity to remove debris and the cavity is held open for tap water irrigation.

SPECIFIC ABSCESS THERAPY

Staphylococcal Diseases

The *Staphylococcus* bacterium is a ubiquitous pathogen that frequently colonizes the nose, skin, perineum, and gut. The umbilicus of neonates is also commonly colonized. It grows on the skin and thrives particularly well in hair follicles, causing boils (furuncles), wound infections, and occasionally carbuncles. The pathogenesis of staphylococcal disease is a complex host-bacteria interaction. *S. aureus* invades the skin by way of the hair follicles or an open wound and produces local tissue destruction followed by hyperemia of vessels. Subsequently,

Figure 38-8 A carbuncle is a complicated abscess on the nape of the neck. It is very common in diabetics *(A)*. Because of many crypts and loculations, simple incision and drainage are often not readily curative. This patient had multiple incisions yet was not cured *(B)*. Antibiotics may augment healing of this abscess, but wide surgical excision may be required.

an exudative reaction occurs, during which polymorphonuclear cells invade. The process then extends along the path of least resistance. The abscess may "point" or form sinus tracts. The process can disseminate by invasion of vessels and thus can infect other organs. Most cases of staphylococcal osteomyelitis, meningitis, and endocarditis occur by this mechanism.[47] [48]

Folliculitis represents a small abscess occurring at the root of a hair. Local measures, including warm compresses and antibacterial soaps and ointments, are the usual treatment, but systemic antibiotics may be required if multiple sites are involved or the patient is a chronic staphylococcal carrier. *Furuncles*, or boils, are acute circumscribed abscesses of the skin and SQ tissue that most commonly occur on the face, the neck, the buttocks, the thigh, the perineum, the breast, or the axilla. *Carbuncles* are aggregates of interconnected furuncles that frequently occur on the back of the neck (Fig. 38-8). In this area the skin is thick, and extension therefore occurs laterally rather than toward the skin surface. Carbuncles may attain large size and can cause systemic symptoms and complications. They are found in increased frequency in diabetics, and all patients with a carbuncle should be evaluated for this underlying disease. Treatment should consist of surgical drainage and administration of systemic antibiotics. Large carbuncles may be impossible to drain adequately in the ED. Carbuncles usually consist of many loculated pockets of pus, and simple incision and drainage are often not curative. Occasionally, wide excision and skin grafting are required.

Most cases of recurrent staphylococcal skin infections are caused by autoinfection from existing skin lesions or nasal reservoirs. Prevention is directed at eliminating the organism. This is accomplished by application of bacitracin to the nares and by good hygiene, including frequent cleansing with antibacterial soap. If these measures are unsuccessful, then systemic oral antistaphylococcal treatment is instituted for 2 to 3 weeks. Detection and treatment of infection in family members may be necessary.[47] [48]

S. aureus produces the ubiquitous suture abscess. A *suture abscess* is often misdiagnosed as a wound infection,

but in fact, it is a local nidus of inflammation or infection, or both, caused and potentiated by suture material. Such an abscess usually appears after sutures have been present for at least 3 to 5 days, with single or multiple discrete areas of redness and tenderness noted at the site of suture penetration of the skin. Simply removing the suture (a drop of pus may be expressed) and providing warm compresses and topical antibiotic ointment is usually all that is required. Wide opening of the wound and systemic antibiotics are seldom required. When the suture is buried, a small incision should be followed by probing of the wound with a small hook or bent needle (see Chapter 36) to snare the suture for its removal.

Hidradenitis Suppurativa

Hidradenitis suppurativa (Greek *hidros* = sweat, *aden* = gland) is a chronic, relapsing, inflammatory disease process affecting the apocrine gland that primarily involves the axilla, the inguinal region, or both. [49] Its prevalence is 0.3% to 4% in industrialized countries; most affected individuals are young women. [50] The condition results from occlusion of the apocrine ducts by keratinous debris, which leads to ductal dilation, inflammation, and rupture into the SQ area. Secondary bacterial infection ensues, leading to abscess formation and scarring. This chronic recurring process leads to draining fistulous tracts, which involve large areas and are not amenable to simple incision-and-drainage procedures (Fig. 38-9). [51]

Genetic factors may play some role in hidradenitis suppurativa. Family history is often significant in these patients. Fitzsimmons has proposed a single dominant gene transmission.[52] Individuals of African descent appear to have an increased incidence compared with Caucasians. Although certain groups appear to be predisposed to this condition, the precipitating factor for this process is unclear. Because apocrine glands become active during puberty, it is rare to find hidradenitis suppurativa in the pediatric population. [49] B-mode ultrasound images reveal larger hair follicles in affected individuals than in controls. [53]

Women are affected more frequently than men, for uncertain reasons. Shaving and depilation have frequently been

Figure 38-9 Hidradenitis suppurativa of the groin or axilla is a complicated series of abscesses that may not be amenable to simple incision and drainage. In the case of involvement with the groin, extensive surgery was required to excise recurrent infection.

suggested as causes of this discrepancy; however, this theory was not supported in a study that compared the frequency of these behaviors in patients with hidradenitis suppurativa and a group of controls. [54] Obesity is associated with an increased incidence of the disease. [55] Excessive dermal folds provide dark, wet, and warm areas, which are ideal for the proliferation of the bacteria that are needed for this infectious process. Antiperspirants and deodorants may decrease wetness and bacterial overgrowth, but they have been known to produce inflammatory responses, which could exacerbate the disease process.

The bacteriology of acute abscess formation in hidradenitis suppurativa reflects organisms seen in other soft tissue abscesses. *Staphylococcus* is the most commonly isolated organism,[56] with *E. coli* and ß-hemolytic *Streptococcus* being other important pathogens. In the perineal region, enteric flora are often found. Many of these abscesses have multiple isolates, and anaerobic bacteria are frequently found.

Hidradenitis suppurativa begins as a single inflammatory event involving an apocrine gland, which progresses to frank suppuration and at this stage is no different from a simple furuncle. The clinical entity is distinguishable only in its chronic scarring phase. By then the lesion exhibits multiple foci coupled with areas of induration and inflammation that are in various stages of healing. Progression of the process reveals coalesced areas of firm, raised violaceous dermis. The lesion is usually markedly tender. This disease classically involves the axilla and perineal or inguinal region, although multiple sites are often involved.

Initial outpatient management usually involves intervention in an acute suppurative lesion. Any fluctuant area requires drainage as described in the section on general abscess management. In cases of extensive cellulitis, a broad-spectrum, antistaphylococcal antibiotic should be used. Unfortunately, hidradenitis suppurativa is often not readily cured with localized incision and drainage. The chronic nature of the disease produces multiple areas of inflammation and SQ fistulous tracts that induce routine recurrences. The patient must be informed of this rather unfavorable prognosis and should be referred to a dermatologist or surgeon for long-term care.

Milder forms of the disease are initially treated with conservative measures. Many different approaches have been tried, with numerous case reports and case series noted in the literature. Unfortunately, few controlled studies have been performed. Patients are often counseled to lose weight, refrain from shaving, stop using deodorants, and improve personal hygiene. The benefits of these efforts are unknown. Oral antistaphylococcal antibiotics are most commonly used, with varying results.[57] There have been reports of success with topical clindamycin, [58] [59] isotretinoin,[60] [61] and laser therapy,[62] but these treatments have not been studied in a controlled setting and require further investigation. Dermal infection results from breakdown of the normal host defense mechanism, which occurs with irritation, traumatic injury, or inflammation, coupled with the availability of concentrated opportunistic bacteria. Therefore, the clinician must institute therapies that will decrease bacterial availability without causing further injury to the affected dermis.

Advanced stages of the disease are routinely managed with wide or local excision and primary or delayed closure (Fig. 38-9).[63] [64] [65] Skin grafting may be warranted.[66] Despite such radical approaches, recurrences do occur. [67] Patients must be counseled about the likelihood of recurrence before the procedure.

Breast Abscess

Postpartum mastitis occurs in 1% to 3% of nursing mothers within the first 2 to 6 weeks after delivery. The infection is usually precipitated by milk stasis following weaning or missed feedings. The cause is usually bacterial invasion through a cracked or abraded nipple by *S. aureus* or streptococci originating from the nursing child. Manifestations are redness, heat, pain, fever, and chills. Treatment consists of antistaphylococcal antibiotics, continued breast emptying with a breast pump, and application of heat. It is important to encourage continued breast emptying to promote drainage. Nursing can be continued with the noninfected breast, although passage of the antibiotics through the breast milk may result in some infant diarrhea. Cellulitis may progress to frank abscess formation. These patients may be quite ill and may appear toxic. Abscess formation complicating postpartum mastitis usually necessitates OR management, because the area is extremely tender, and adequate local anesthesia is difficult to obtain. Strict adherence to nipple hygiene to avoid cracks or inflammation is helpful in prophylaxis.

Surprisingly, most breast abscesses occur in women who are not in the puerperium, and have been termed *nonpuerperal breast abscesses*.[68] Scholefield and coworkers [69] reviewed 72 breast abscesses over a 10-year period and noted that only 8.5% of the patients were in the puerperium. These lesions have been classified anatomically.

Peripherally located abscesses are most commonly caused by *Staphylococcus* and respond well to traditional conservative incision and drainage. [69] Superficial abscesses in the SQ tissue may be drained under local anesthesia by means of an incision that radiates from the nipple (Fig. 38-10*A*).

Periareolar abscesses exhibit a more troublesome profile. The microflora often include multiple organisms, and anaerobic bacteria are important isolated pathogens.[68] [69] [70] [71] These infections may be the result of occluded and inflamed mammary ducts. Chronic disease may lead to ductal ectasia that provides a nidus for infection. The deeper and more extensive intramammary abscess appears as a generally swollen, tender breast (Fig. 38-10*B*). Fluctuance is not always obvious, since the abscess is located in the mammary tissue itself. Maier and colleagues reported that more than half of their series of 96 patients had nipple inversion due to chronic scarring.[72] These intramammary infections are complex and require incision and drainage under general anesthesia. Dixon has reported successful treatment with repeated aspiration in both lactating [73] and nonlactating patients. [74] This approach is probably best left to surgeons able to see such patients on a regular scheduled basis to track the progress of abscess resolution.

A *retromammary abscess* lies in the undersurface of the breast between the breast and the chest wall (Fig. 38-10*C*). Fluctuance may be difficult to appreciate because of the depth of the infection. Drainage under general anesthesia is required.

Recurrent abscesses are a common, troublesome complication, occurring in up to 38% of primary periareolar abscesses treated with standard incision, drainage, and antibiotics.[75] These cases require total excision of the involved area and necessitate the care of a general surgeon and further intraoperative management. It may be difficult to diagnose a breast abscess in the early stages, when cellulitis predominates. In equivocal cases antibiotics may be curative, but when pus is

Figure 38-10 *A,* A superficial breast abscess may be drained with a linear incision that radiates from the nipple. *B* and *C,* Diagrams of intramammary abscess *(B)* and retromammary abscess *(C)*. Both require drainage under general anesthesia. The abscess itself may not be fully appreciated if it is deep-seated, and the mistaken diagnosis of cellulitis may be made. *(Redrawn from Wolcott MW: Ferguson's Surgery of the Ambulatory Patient, 5th ed. Philadelphia, JB Lippincott, 1974. Reprinted with permission.)*

present, incision and drainage must be performed. Bedside ultrasound can be helpful for real-time visualization and guidance during aspiration. [76]

An active breast abscess may be the initial presentation of squamous cell carcinoma. In these cases, aspirate from the abscess should be sent for both culture and cytology. If drainage is performed, a biopsy of the abscess cavity is imperative. [77] [78] Breast abscesses may also be a complication of breast implants.[79] In general, most breast abscesses require early consultation with a surgeon.

A breast abscess in a man is an unusual occurrence. Malignancy or underlying bone or joint infection are considerations (see Fig. 38-1).

Bartholin Gland Abscess

The Bartholin glands (vestibular glands) are secretory organs located at the 5 and 7 o'clock positions on each side of the vestibule of the vagina. Asymptomatic cysts frequently occur from duct blockage and retention of secretions. Chronic low-grade inflammation from gonococcal infections has been implicated as an etiologic factor in cyst formation, but occasionally frank abscess formation results. Such patients present with swollen and tender labia and a fluctuant, grape-sized mass that may be palpated between the thumb and the index finger. *Neisseria gonorrhoeae* organisms are infrequently cultured from the abscess cavity, and various anaerobes, especially *Bacteroides* species and other colonic bacteria, are usually found. It is reasonable to take cervical and anal cultures for gonorrhea from women with Bartholin gland abscesses because of the association of these infections with venereal disease, [80] but one need not routinely treat patients for gonorrhea.

Word[81] described an effective treatment of Bartholin gland abscess with a single-barreled, sealed-stopper, balloon-tipped catheter that serves as initial and long-term therapy and may obviate the need for marsupialization (Fig. 38-11*A* and *B*). The editors recommend this technique as the standard therapy of a Bartholin gland abscess. In his original description, Word reported only 2 recurrences in 72 lesions, both of which were successfully treated with a second catheter; no patient required marsupialization. The procedure involves fistulization of the duct cavity by a catheter, which acts as a FB. While not a traditional incision-and-drainage procedure, the technique permits continued drainage of the Bartholin gland. Theoretically this catheter may be used for most abscess drainage procedures, but its use for other conditions is not widespread.

Following a small incision into the mucosa, the scalpel or a hemostat is used to puncture the abscess cavity proper (Fig. 38-12 Fig. 38-12). This may be the most difficult portion of the procedure and it can be difficult to enter this rather deep abscess cavity. Failure to obtain frank pus, or appreciate the "pop" of entering the abscess, often prognosticates failure of the procedure. It is helpful to stabilize the abscess with the thumb and forefinger, hold the hemostat in place, and *skewer the abscess onto the hemostat* to ensure entrance into the abscess. Trying to push the hemostat into the immobilized abscess is technically more difficult (see Fig. 38-12*E*). Care is required to make a stab incision only large enough to accommodate the catheter and small enough to prohibit the inflated balloon from being extruded. Once the abscess has been entered (signaled by a palpable pop or the free flow of pus), the deflated balloon is placed in the abscess cavity. Using a 25-ga

needle to minimize the hole in the stopper, the balloon is then filled with 2 to 4 mL of water (not air). Persistent pain indicates that too much fluid has been used. Most cases are cured with drainage for a few days, and the Word catheter often falls out in within a week. However, the device is ideally left in place for 2 to 4 weeks to allow for fistula formation, so follow-up is required. If the catheter falls out prematurely, it should be quickly replaced to fulfill the times needed for fistulization. This is an interesting technique that even allows for sexual intercourse with the catheter in place. Some clinicians will not reinsert the catheter if healing has progressed significantly, and it is the first drainage procedure.

Although the Word catheter is likely the best initial treatment of a Bartholin gland abscess, even if the catheter falls

Figure 38-11 Use of the Word catheter for outpatient drainage of a Bartholin gland abscess. This is a fistulization procedure rather than a standard incision and drainage. A stab incision is made on the mucosal surface *(A)*. A catheter is inserted into the cyst cavity *(B)* and filled with 3 to 4 mL of water *(C)*. *D,* Inflatable bulb-tipped catheter. *Left,* Uninflated. *Right,* Inflated with 4 mL water. *(A through C from Word B: Office treatment of cyst and abscess of Bartholin gland. JAMA 190:777, 1964. D from Word B: Office treatment of cyst and abscess of Bartholin gland duct. South Med J 61:514, 1968. Reproduced with permission.)*

out in a few days, another acceptable therapy is standard incision and drainage. The abscess is packed for 24 to 48 hours, and sitz baths are started after the first revisit. Broad-spectrum antibiotics are helpful if there is significant cellulitis or actual abscess formation has not yet occurred, but these agents are not required following routine incision and drainage.

It is preferable to make the drainage incision on the mucosal surface rather than on the skin surface. The incision is made over the medial surface of the introitus on a line parallel to the posterior margin of the hymenal ring. The abscess

Figure 38-12a *A,* An alternative to formal incision, drainage, and packing for the treatment of this Bartholin abscess is to place a Word catheter. *B*, The abscess is stabilized with the thumb and index finger, and a local anesthetic is injected into the mucosal (not skin) surface. *C*, A stab incision is made with a scalpel. *D*, The abscess is punctured with a hemostat. Deep abscesses may be difficult to puncture.

cavity is slightly deeper than most cutaneous abscesses, and one must be certain to enter the actual abscess cavity to achieve complete drainage. This is most easily accomplished if one inserts a hemostat through the mucosal incision and spreads the tips of the instrument in the deeper soft tissue. If the abscess recurs, more definitive therapy in the form of marsupialization or complete excision of the gland may be required, but these procedures are not performed initially. Because recurrence is common with simple incision and drainage, some authorities suggest definitive surgery routinely following the first infection, whereas others prefer to wait until a recurrence, or multiple recurrences, are documented.

Particular care must be taken when treating pregnant women with Bartholin gland abscess. The development of sepsis in a pregnant woman after marsupialization of an abscess has been reported,[82] so this subset of patients should be considered at high risk for complications and managed accordingly.

Pilonidal Abscess

Pilonidal sinuses are common malformations that occur in the sacrococcygeal area. The etiology of the sinus formation is unclear, but the malformation may occur during embryogenesis. Pilonidal cyst formation is thought to be secondary to blockage of a pilonidal sinus. The result of pilonidal sinus obstruction is repeated soft tissue infection, followed by drainage and partial resolution with eventual reaccumulation. The blockage is most commonly the result of hairs in the region, and the lesion may in part be a FB (hair) granuloma. Although pilonidal sinuses are present from birth, they usually are not manifested clinically until adolescence or the early adult years, and pilonidal abscess formation most commonly affects young (often white) adults. The sinuses and cysts are lined with stratified squamous epithelium and may contain wads of hair and debris when excised. When cultured, pilonidal abscesses generally yield mixed fecal flora with a preponderance of anaerobes. [83] Poor hygiene and repeated

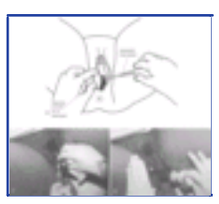

Figure 38-12b *E*, It is technically easier to enter the Bartholin gland abscess cavity if the hemostat is held steady and the abscess, held with the thumb and index finger, is skewered onto the hemostat. Attempting to puncture the deep immobilized abscess by stabbing with the hemostat may be more difficult. A palpable pop when entering the abscess or drainage of frank pus is expected. *F*, The catheter is placed to its hilt into the abscess cavity, and the balloon is filled with saline. Use a 25-ga needle to fill balloon. *G*, The catheter is left in place for 2 to 4 weeks to form a fistula.

trauma (so-called Jeep bottom in World War II) may precipitate acute infection.

The patient with a pilonidal abscess will seek care for back pain and local tenderness. On physical examination the area is indurated, but frank abscess formation may not be appreciated in this deep abscess. One will usually see barely perceptible dimples or tiny openings at the rostral end of the gluteal crease (Fig. 38-13). A hair or a slight discharge may be noticed at the opening. One may find a more caudal cyst or abscess, possibly with a palpable sinus tract connecting the two. The sinus and cyst may be chronically draining, or they may become infected as the size increases and blockage occurs. [84]

Treatment of the acutely infected cyst is the same as previously discussed for any fluctuant abscess; all hair and pus should be removed, and the lesion should be packed (Fig. 38-14). Antibiotic therapy is not usually required. The abscess cavity may become quite large, necessitating a rather lengthy incision to ensure complete drainage. It may take many weeks for the initial incision to heal. The area may be repacked at 2- to 4-day intervals as an outpatient procedure, although some prefer to discontinue packing after the first week. Because simple incision and drainage are often not curative, secondary removal of both the cyst and the sinus should be planned after the inflammatory process has resolved. The elective surgical procedure should be complete and should involve all of the possible arborizations of the sinus.

Recurrence is occasionally prevented by simple incision and drainage, especially if the incision is wide, and adequate drainage is obtained. More commonly, recurrence can be expected unless excision of the sinus tract is performed. Small abscesses may be incised and drained as an outpatient procedure performed under local anesthesia, but the disease process is often extensive, and general anesthesia may be required to complete drainage. One is often surprised by the extent of the

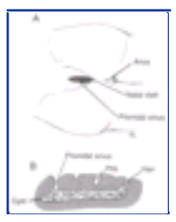

Figure 38-13 Pilonidal sinus. *A*, Sinuses occur in the midline some 5 cm above the anus in the natal cleft. *B*, Longitudinal section showing sinuses and pits. *(From Hill GJ II: Outpatient Surgery, 3rd ed. Philadelphia, WB Saunders, 1988. Reproduced with permission.)*

cyst cavity and the volume of pus that is encountered when the area is probed during initial incision; because of the degree of these abscesses, only localized infection lends itself to outpatient therapy. A method of catheter drainage for pilonidal abscesses has been described [45] [46] in which a flared-end de Pezzer catheter is used for extended periods in the abscess cavity. The catheter allows the patient more freedom from local care and provides continual drainage (Fig. 38-15).

Perirectal Abscesses

Perirectal infections can range from minor irritations to fatal illnesses. Successful management depends on early recognition of the disease process and adequate surgical therapy. Because of the morbidity and mortality associated with inadequate treatment of these conditions, patients with all but the most localized abscesses should be promptly admitted to the hospital for evaluation and treatment under general or spinal anesthesia (Fig. 38-16).

It is important to understand the anatomy of the anal canal and the rectum in order to appreciate the pathophysiology of these abscesses and their treatment (Fig. 38-17). The mucosa of the anal canal is loosely attached to the muscle wall. At the dentate line, where columnar epithelium gives way to squamous epithelium, there are vertical folds of tissue, called the rectal *columns of Morgagni*, which are connected at their lower ends by small semilunar folds called *anal valves*. Under these valves are invaginations termed *anal crypts*. Within these crypts are collections of ducts from anal glands. These glands are believed to be responsible for the genesis of most, if not all, perirectal abscesses. These glands often pass through the internal sphincter but do not penetrate the external sphincter.

The muscular anatomy divides the perirectal area into compartments that may house an abscess, depending on the direction of spread of the foci of the infection (Fig. 38-18). [84] [85] The circular fibers of the intestinal coat thicken at the rectum-anus junction to become the internal anal sphincter. The muscle fibers of the levator ani fuse with those of the outer longitudinal fibers of the intestinal coat as it passes through the pelvic floor. These conjoined fibers are connected by fibrous tissue to the external sphincter system, which consists of three circular muscle groups.

Pathophysiology

As described previously, the anal glands are mucus-secreting structures that terminate in the area between the internal and external sphincters. It is believed that most perirectal infections begin in the *intersphincteric* space secondary to blockage and subsequent infection of the anal glands. Normal host defense mechanisms then break down, followed by invasion and overgrowth by bowel flora. [86]

If the infection spreads across the external sphincter laterally, an *ischiorectal abscess* is formed. If the infection dissects rostrally, it may continue between the internal and external sphincters, causing a *high intramuscular abscess*. The infection may also dissect through the external sphincter over the levator ani to form a *pelvirectal abscess*.[85]

When infection of an anal crypt extends by way of the perianal lymphatics and continues between the mucous membrane and the anal muscles, a *perianal abscess* forms at the anal orifice. The perianal abscess is the most common variety of perirectal infection. The abscess lies immediately beneath the skin in the perianal region at the lowermost part of the anal canal. It is separated from the ischiorectal space by a fascial septum that extends from the external sphincter and is continuous with the SQ tissue of the buttocks. The infection may be small and localized or very large, with a wall of necrotic tissue and a surrounding zone of cellulites. [47] Perianal abscesses may be associated with a fistula in ano. The fistula in ano is an inflammatory tract with an external opening in the skin of the perianal area and an internal opening in the mucosa of the anal canal. The *fistula in ano* is usually formed after partial resolution of a perianal abscess, and its presence is suggested by recurrence of these abscesses with intermittent drainage. The external opening of the fissure is usually a red elevated piece of granulation tissue that may have purulent or serosanguineous drainage on compression. Many times the tract may be palpated as a cord. Patients with anal fistulas should be referred for definitive surgical excision.[85]

Ischiorectal abscesses are fairly common. They are bounded superiorly by the levator ani, inferiorly by the fascia over the perianal space, medially by the anal sphincter muscles, and laterally by the obturator internus muscle. These abscesses may commonly be bilateral, and if so, the two cavities communicate by way of a deep postanal space to form a "horseshoe" abscess.[47]

Intersphincteric abscesses are less common. They are bounded by the internal and external sphincters and may extend rostrally into the rectum, thereby separating the circular and longitudinal muscle layers.

The *pelvirectal*, or *supralevator, abscess* lies above the levator ani muscle in proximity to the rectal wall and remains extraperitoneal. The etiology of this abscess is controversial. Kovalcik and colleagues[86] suggest that supralevator abscesses

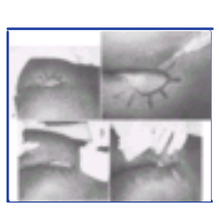

Figure 38-14 *A*, A pilonidal abscess can be quite extensive (*induration outlined by marker*) but it may be difficult to feel fluctuance because of the depth of the infection. *B*, Local anesthesia and conscious sedation make the initial ED incision and drainage successful. *C*, A deep incision yields copious pus, and occasionally other debris, such as hair. *D*, The abscess cavity is packed open. *Not shown*: When infection and inflammation have subsided, definitive treatment may include wide excision of the sinus and its lateral tracks.

are primarily an extension of an intra-abdominal process, such as diverticulitis or pelvic inflammatory disease. Read and Abcarian [87] evaluated 404 patients with perirectal abscesses in a prospective study and found that of the 36 supralevator abscesses, none was caused by an intra-abdominal or pelvic pathologic condition. They determined that supralevator abscesses were most commonly associated with ischiorectal abscesses and suggested that these conditions may be an extension of ischiorectal abscesses through the floor of the levator ani. Nonetheless, they found rare isolated pelvirectal abscesses without intra-abdominal, pelvic, ischiorectal, or perianal infection.

Causes of perirectal abscesses other than the so-called cryptoglandular process have been documented but are fairly rare. It is believed that hemorrhoids, anorectal surgery, episiotomies, or local trauma may cause abscess formation by altering local anatomy and thus destroying natural tissue barriers to infections. [86] [87] [88] Perirectal abscesses may serve as a portal of entry for organisms responsible for necrotic soft-tissue infections such as Fournier's gangrene. [89]

Epidemiology

Anorectal abscesses occur most commonly in healthy adults and are more frequent in males (a greater than 2:1 ratio). [86] [87] These abscesses commonly appear during the fourth decade of life. Possible predisposing medical conditions are diabetes mellitus, inflammatory bowel disease, and other immunocompromised states. Many patients (30%) have a history of previous perirectal abscess, and 75% of anorectal abscesses occur in the same location as the prior abscesses. [86] Of perirectal

abscesses, usually more than 45% are perianal, 20% are ischiorectal, 12% are intersphincteric, and 7% are pelvirectal. [87]

Physical and Laboratory Findings

The diagnosis of a perianal abscess is generally not difficult. The throbbing pain in the perianal region is acute and is aggravated by sitting, coughing, sneezing, and straining. There is swelling, induration, and tenderness, and a small area of cellulitis is present in proximity to the anus. Rectal examination of the patient with a perianal abscess reveals that most

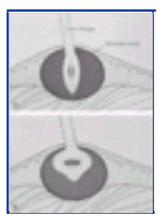

Figure 38-15 A method of prolonged drainage of a pilonidal abscess with a flared-end de Pezzer catheter. Following a stab incision, a stretched catheter (probe inside lumen) is inserted into the abscess cavity *(A).* When the probe is removed, the head of the catheter expands and remains in the abscess cavity *(B).* Drainage is continuous through the lumen of the catheter. *(From Phillip RS: A simplified method for the incision and drainage of abscesses. Am J Surg 135:721, 1978. Reproduced with permission.)*

of the tenderness and induration is below the level of the anal ring. Deeper abscesses may be difficult to localize. A CT scan is may be required to provide definitive diagnosis.

Patients with *ischiorectal abscesses* present with fever, chills, and malaise, but at first there is less pain than with the perianal abscess. Initially on physical examination, one will see an asymmetry of the perianal tissue, and later erythema and induration become apparent. Digital examination reveals a large, tense, tender swelling along the anal canal that extends above the anorectal ring. If both ischiorectal spaces are involved, the findings are bilateral.

Patients with *intersphincteric abscesses* usually present with dull, aching pain in the rectum rather than in the perianal region. No external aberrations of the perianal tissues are noted, but tenderness may be present. On digital examination one frequently palpates a soft, tender, sausage-shaped mass above the anorectal ring; if the mass has already ruptured, the patient may give a history of passage of purulent material during defecation. [85] [86] [87]

Diagnosis of *pelvirectal abscesses* may be very difficult. Usually fever, chills, and malaise are present, but because the abscess is so deep-seated, few or no signs or symptoms are present in the perianal region. Rectal or vaginal examination may reveal a tender swelling that is adherent to the rectal mucosa above the anorectal ring.

Laboratory findings usually do not aid in the diagnosis. Kovalcik and coworkers [86] found that less than 50% of their patients had a white blood cell count greater than 10.0×10^9 /L. Cultures of perirectal abscesses usually show mixed infections involving anaerobic bacteria, most commonly *Bacteroides fragilis* and gram-negative enteric bacilli.

Treatment

Successful management of perirectal abscesses depends on adequate surgical drainage. Complications from these infections may necessitate multiple surgical procedures, prolong hospital stay, and result in sepsis and death. Bevans and associates [88] retrospectively studied the charts of 184 patients who were surgically treated over a 10-year period. These patients were evaluated primarily to identify the factors that contributed to morbidity and mortality. Initial drainage was performed under local anesthesia in 38% of the patients and under spinal or general anesthesia in 62%. The authors identified three key factors in excessive morbidity and mortality: (1) a delay in diagnosis and treatment, (2) inadequate initial examination or treatment, and (3) associated systemic disease. It was their belief that the only way to examine effectively and drain adequately all but superficial well localized perirectal abscesses was under spinal or general anesthesia. This assessment was supported by evidence of an increased incidence of recurrence and of sepsis and death in patients treated with local anesthesia. Drainage of deep abscesses under local anesthesia generally does not allow drainage of all hidden loculations. In addition, local anesthesia is not adequate for treatment of associated pathologic conditions.

Small, well-defined perianal abscesses are the only perirectal infections that lend themselves to outpatient therapy. The result of incision and drainage is almost immediate relief of pain and rapid resolution of infection. Indications for inpatient drainage are failure to obtain adequate anesthesia, systemic toxicity, extension of the abscess beyond a localized area, or recurrence of a perianal abscess. Recurrence may be caused by the presence of a fistula in ano.

A perianal abscess is drained through a single linear incision over the most fluctuant portion of the abscess in a manner previously described for other cutaneous abscesses. It is extremely painful to probe a perianal abscess and to break up loculations, and liberal analgesia is advised. The patient may begin sitz baths at home 24 hours following surgery. Packing is replaced at 48-hour intervals until the infection has cleared and granulation tissue has appeared. This usually occurs within 4 to 6 days. Antibiotics are generally not required. All other perirectal abscesses require hospitalization for definitive therapy.

Use of de Pezzer catheters in anorectal abscesses has been described as an alternative to traditional incision and packing. Kyle and Isbister reported a series of 91 patients treated in this manner. They found equivalent rates of subsequent fistula surgery, less need for general anesthesia, and a shorter postoperative hospital stay when compared with patients treated with traditional incision and packing. [90] Beck and colleagues reported successful use of catheter drainage in 55 patients with ischiorectal abscess. [91] Due to the complexity of ischiorectal abscesses, this technique is probably best left to the surgeon providing ongoing care.

Figure 38-16 *A*, If a perirectal abscess spontaneously ruptures and drains, a formal incision, drainage, and packing should still be performed. *B*, A deep, poorly localized perirectal abscess of this size simply cannot be adequately drained in the ED. Arrows outline the area of induration. This patient requires extensive drainage under general anesthesia. A computed tomography scan may further evaluate the location of this abscess.

Perirectal abscesses are currently recognized as a fairly common cause of fever in the granulocytopenic patient. These abscesses have a different bacteriologic profile: *Pseudomonas aeruginosa* organisms are isolated most frequently. These patients present later because pain develops later in the course, and fever may be the first manifestation. Therefore, any patients who are granulocytopenic with vague anorectal complaints, especially those with fever, should be examined carefully for perirectal abscesses. Any abscess that is found should be drained immediately under appropriate anesthesia, and extensive IV antibiotic coverage should be initiated.

Patients who present with a spontaneously ruptured perirectal abscess may appear to have experienced a self cure, but under most circumstances they should undergo formal incision, drainage, and packing. Cases should be individualized.

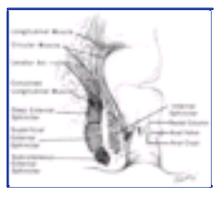

Figure 38-17 Schematic coronal section of the anal canal and the rectum. *(From Schwartz SI, Lillehei RC: Principles of Surgery, 2nd ed. New York, McGraw-Hill, 1974. Reproduced*

with permission.)

Infected Sebaceous Cyst

A common entity that appears as a cutaneous abscess is the infected sebaceous cyst. Sebaceous cysts may occur throughout the body and result from obstruction of sebaceous gland ducts. The cyst becomes filled with a thick, cheesy, sebaceous material, and the contents frequently become infected. Sebaceous cysts may be quite large and may persist for many years before they become infected. When infected, they clinically appear as tender, fluctuant SQ masses, often with overlying erythema.

The initial treatment of an infected sebaceous cyst is simple incision and drainage. The thick sebaceous material must be expressed, since it is too thick to drain spontaneously (Fig. 38-19*A*). An important difference exists between infected sebaceous cysts and other abscesses. *A sebaceous cyst has a definite pearly white capsule that must be excised to prevent recurrence* (Fig. 38-19*B* and *C*). Traditionally, in the presence of significant inflammation, it is preferable to drain the infection initially and remove the shiny capsule on the first follow-up visit or a later visit, when

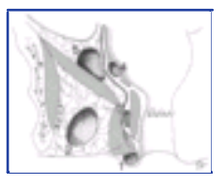

Figure 38-18 Classification of perirectal abscesses: 1, Perianal. 2, Ischiorectal. 3, Intersphincteric. 4, High intramuscular. 5, Pelvirectal. *(From Hill GJ II: Outpatient Surgery, 3rd ed. Philadelphia, WB Saunders, 1988. Reproduced by permission.)*

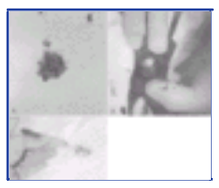

Figure 38-19 The thick, cheesy sebaceous material of a sebaceous cyst must be expressed after incision *(A)*. The shiny capsule of this infected sebaceous cyst is easily visible *(B)* and was removed in its entirety on the initial visit *(C)*.

it may be more easily identified. Alternatively, the entire cyst can be removed at the time of initial incision. At the time of capsule removal, the edges are grasped with clamps or hemostats, and the entire capsule is removed by sharp dissection with a scalpel or scissors. Following excision of the capsule, the area is treated in the same manner as a healing abscess cavity. Simple drainage without excision of the capsule often leads to recurrence.

Kitamura and associates reported a randomized study of 71 patients treated with either traditional incision and drainage or primary resection of the cyst, followed by irrigation and wound closure. In this study, the patients treated with primary resection had faster healing, fewer days of pain, and less scarring. [92]

Paronychia

A paronychia is an infection localized to the area around the nail root (Fig. 38-20). Paronychias are common infections probably caused by frequent trauma to the delicate skin around the fingernail and the cuticle. When a minor infection begins, the nail itself may act like a FB. Usually the infectious process is limited to the area above the nail base and underneath the eponychium (cuticle), but occasionally it may spread to include tissue under the nail as well, forming a subungual abscess. Lymphadenitis and lymphadenopathy are usually not seen. Generally, a paronychia is a mixed bacterial infection. *Staphylococcus* is commonly cultured from these lesions; however, anaerobes and numerous gram-negative organisms may be isolated. [93] Paronychias in children are often caused by anaerobes, and it is believed that this is the result of finger sucking and nail biting. Occasionally, a group A ß-hemolytic infection will develop in a paronychia if a child with a streptococcal pharyngitis puts his or her fingers in the mouth. [94]

A paronychia appears as a swelling and tenderness of the soft tissue along the base or the side of a fingernail (Fig. 38-21). Pain, often around a hangnail, usually prompts a visit to the ED. The infection begins as a cellulitis and may form a frank abscess. If the nailbed is mobile, the infectious process has extended under the nail, and a more extensive drainage procedure should be performed. If soft tissue swelling is present without fluctuance, remission may be obtained from frequent hot soaks (6 to 8 times a day) and a short course of oral antibiotics (3 to 4 days). [93] Incision will be of no value at this early cellulitic phase. If a significant cellulitis is present, a broad-spectrum antistaphylococcal antibiotic (cephalosporin or semisynthetic penicillin) may be tried. However, plain penicillin or erythromycin is often sufficient for limited inflammation. The digit should be splinted and elevated. [95] [96] One should never rely solely on antibiotic therapy once frank pus has formed.

Technique

When a definite abscess has formed, drainage is usually quickly curative. A number of invasive operative approaches

Figure 38-20 Paronychia. *1*, The site of the abscess at the side of the nail. *2*, The infection has extended around the base of the nail. It has raised the eponychium but has not penetrated under the nail. *3*, End stage of paronychia with a subeponychial and subungual abscess. *(From Wolcott MW: Ferguson's Surgery of the Ambulatory Patient, 5th ed. Philadelphia, JB Lippincott, 1974. Reproduced with permission.)*

have been suggested, but actual skin incision or removal of the nail is rarely required, and *neither procedure should be the initial form of treatment*. One can invariably obtain adequate drainage by simply lifting the skin edge off the nail to allow the pus to drain. This is usually curative, because a paronychia is not a cutaneous abscess per se, but rather a collection of pus

Figure 38-21 A paronychia may occur with obvious pus localization between the eponychium (cuticle) and the nail. *A*, Actual pus may be seen or, *B*, the area is swollen and tender. *C*, A paronychia can also occur in the toe. This is not a true cutaneous abscess but rather a collection of pus in a potential space. Actual skin incision is not required if eponychial elevation results in adequate drainage.

in the potential space between the cuticle and proximal fingernail. Drainage may be accomplished without anesthesia in select patients but frequently requires a digital nerve block. After softening the eponychium by soaking, a No. 11 blade, scissors, or an 18-ga needle is advanced *parallel to the nail* and under the eponychium at the site of maximal swelling (Fig. 38-22).[96] [97] Pus rapidly escapes, with immediate relief of pain. A tourniquet placed at the base of the finger may limit bleeding and aid the clinician in determining the exact extent of the infection during the drainage procedure.

If more than a tiny pocket of pus is present, one should fan the knife tip or needle or spread the scissors under the eponychium, keeping the instrument parallel to the

plane of the fingernail. When a large amount of pus is drained, a small piece of packing gauze is slipped under the eponychium for 24 hours to provide continual drainage. Cultures are generally not indicated. Antibiotics are frequently prescribed, although they are not essential if drainage is complete or if the surrounding area of cellulitis is minimal. An alternative to systemic antibiotics is to keep the operative site bathed in antibiotic ointment. After anesthesia has worn off, the patient may be started on frequent soaks in warm tap water at home. In most cases the patient may easily remove the packing. At 24 to 36 hours, the finger is soaked in hot water and the gauze pulled out; a repeat visit to a clinician is not required if healing is progressing. Once the packing is removed, the area is covered with a dry, absorbent dressing. An antibiotic ointment may be applied to the site for a few days. The benefit of antibiotic ointments in reducing infection is unproved, but instructing the patient concerning the detailed use of the ointment may prompt soaking. In addition, the ointment helps to keep the bandage from sticking.

If the infection has produced purulence beneath the nail (subungual abscess), a portion of the nail must be removed or the nail trephined to ensure complete drainage. As an alternative to nail removal, a hole may be placed in the proximal nail with a hot paper clip. A large opening or multiple holes are required to ensure continued drainage. Most commonly the proximal portion of the nail is involved. This may be treated by

Figure 38-22 An initial treatment method for a well-localized paronychia. Following a digital nerve block, the eponychium (cuticle) is elevated at the area of greatest fluctuance, and no actual skin incision is made. A tourniquet can be used to limit bleeding and facilitate drainage. *A* and *B*, A sharp instrument (scissors or a No. 11 blade) is held parallel to the nail and advanced until pus is drained. *C* and *D*, A hemostat or scissors is placed into the base and all margins of the pus collection and spread in a fan-like fashion to open up the abscess. The entire pocket is opened to break up loculations and create a cavity. *E*, A small packing is placed for 2 days. At recheck a new pack may be placed or the cavity left open to heal. *Actual incision of tissue or removal of the nail is reserved for complicated or resistant infections and is not first-line therapy.*

bluntly elevating the eponychium to expose the proximal edge of the nail. The proximal one third of the nail is then elevated from the nailbed and resected with a scissors. The distal two thirds of the nail is left in place to act as a physiologic dressing and to decrease postoperative pain (Fig. 38-23). If purulence is found below the lateral edge of the nail, the affected part may be gently elevated and excised longitudinally. [98] Care must be exercised during this procedure to avoid damage to the nail matrix. A wick of gauze should be placed beneath the eponychium for 48 hours to ensure continued drainage.

Most paronychia resolve in a few days, and one to two postoperative visits should be scheduled to evaluate healing and reinforce home care. For compliant patients with a small paronychia, home care alone may suffice after the initial drainage. A well-known but very rare complication of even a properly drained paronychia is osteomyelitis of the distal phalanx. Clinical infection lasting longer than a few weeks should prompt evaluation for this complication.

Patients occasionally present to the ED complaining of a chronic, indolent infection of the paronychia. These seldom

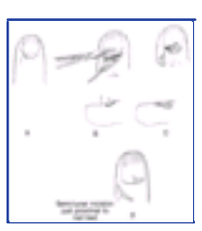

Figure 38-23 *A–C*, Aggressive treatment of recurrent paronychia or subungual abscess includes removal of a portion of the proximal nail and incision of the eponychium. *D,* Some physicians prefer to use a semilunar incision proximal to the eponychium rather than directly incising and potentially injuring the cuticle permanently. *These aggressive therapies are seldom required.*

respond to ED intervention. Frank purulence is seldom present, and conservative treatments are often unsatisfactory. Many etiologies have been described for this frustrating condition, including fungal, bacterial, viral, and psoriatic conditions. Patients with chronic paronychia unresponsive to therapy should be screened for malignancy.[99] Treatment

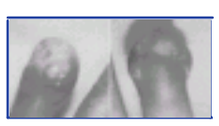

Figure 38-24 A well-developed felon. In this advanced case, the patient had little pain at the time of presentation, and the distal phalanx was almost completely resorbed owing to the extensive pressure and inflammation. This infection is extensive and warrants consultation with a hand specialist.

modalities are varied, and controlled studies evaluating the various techniques are lacking. Meticulous hand care, oral and topical antimicrobial medications, and occasionally aggressive surgical intervention have been suggested. [96] [100] These patients should be referred to a dermatologist or hand surgeon because of the prolonged treatment required.

Herpetic Whitlow

Herpetic whitlow is an infection of the distal phalanx caused by the herpes simplex virus. Digital inoculation occurs through a discontinuity of the skin. [98] Health care providers and patients with other herpes infections are most commonly infected. [100] [101] [102] [103] [104] The entity is recognized by the presence of herpetic vesicles, a burning or pruritic sensation, absence of frank pus, slow response to treatment, and a tendency to recur. In questionable cases, viral cultures can be obtained. Herpetic lesions are generally quite painful but are self-limited and resolve in 2 to 3 weeks. Surgical intervention is contraindicated, as this may potentiate a secondary bacterial infection and delay healing. [105] [106] [107] [108] Treatment is symptomatic, consisting of splinting, elevation, and analgesia as needed. Antibiotics effective against herpes infections (acyclovir, famciclovir, and others) probably shorten the course of the disease if given early. After accidental needlestick in health care workers, oral famciclovir may help prevent herpetic whitlow. [109] Consideration must be given to preventing spread of the infection to other individuals. Although an occlusive dressing may lessen the chance for viral transmission, any health care provider with this entity should refrain from patient contact until all lesions have crusted over and viral shedding has stopped. [101] [102] [103]

Felon

A felon is an infection of the pulp of the distal finger (Fig. 38-24). The usual cause is trauma with secondary invasion by bacteria. A felon may develop in the presence of an FB, such

as a thorn or a splinter, but often a precipitating trauma cannot be identified. An important anatomic characteristic of this area is that there are many fibrous septa extending from the volar skin of the fat pad to the periosteum of the phalanx; these subdivide and compartmentalize the pulp area. When an infection occurs in the pulp, these same structures make it a closed space infection. The septa limit swelling, delay pointing of the abscess, and inhibit drainage after incomplete surgical decompression. Pressure may increase in the closed space, initiating an ischemic process that compounds the infection. The infection can progress readily to osteomyelitis of the distal phalanx. Although the septa may facilitate an infection in the pulp, they provide a barrier that protects the joint space and the tendon sheath by limiting the proximal spread of infection.

The offending organisms are usually *Staphylococcus* or *Streptococcus,* although mixed infections and gram-negative infection may occur. A felon is one of the few soft tissue infections in which a culture may be helpful, since osteomyelitis and prolonged infection may occur. An initial culture may aid in the subsequent choice of antibiotics for complicated infections.

The patient developing a felon will describe gradual onset of pain and tenderness of the fingertip. In a few days the pain may be constant and throbbing and gradually becomes severe. In the initial stages, physical examination may be quite unimpressive, because the fibrous septa limit swelling in the closed pulp space. As the infection progresses, swelling and redness may become obvious. Occasionally one may elicit point tenderness, but frequently the entire pulp space is extremely tender. The patient characteristically arrives with the hand elevated over the head because pain is so intense in the dependent position. Cessation of pain indicates extensive necrosis and nerve degeneration.

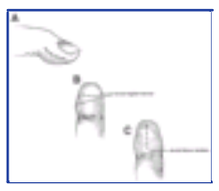

Figure 38-25 The preferred initial incision for draining a felon is one made directly into the area of most fluctuance. More aggressive incisions should be reserved for complicated cases, since they have a greater morbidity and require more complicated wound care. The unilateral longitudinal approach is a good first choice *(A)*. Fat pad incisions are acceptable but may be associated with a painful scar in an area that is often traumatized. The transverse fat pad incision should not injure digital nerves *(B),* and the longitudinal fat pad incision should avoid the flexor tendon *(C)*.

Proper treatment of a well-developed felon consists of early and complete incision and drainage. [98] Antibiotics alone are not curative once suppuration has occurred. Delaying surgery may result in permanent disability and deformity. Most surgeons routinely administer broad-spectrum antibiotics to patients for 5 to 7 days following surgical incision.

Technique

Complicated or advanced felons are best handled in the OR by a hand specialist. Minor felons lend themselves to ED management but followup is required and these infections heal slowly. The surgery of a minor felon can usually be performed as an outpatient procedure using a digital nerve block. A long-acting solution (bupivacaine) will prolong anesthesia. A tourniquet (1.25 cm Penrose drain) should be used to allow digital incision in a bloodless field.

Surgical drainage must be carefully performed to avoid injury to digital nerves, vessels, and flexor tendon mechanisms. Most commonly, a felon can be successfully managed with a limited procedure, but many surgical options have been advocated, none of which has been proven superior for all circumstances. [98] The preferred initial treatment is a simple longitudinal incision made over the area of greatest fluctuance, [110] which may occur laterally or along the volar surface (Fig. 38-25). A potential drawback to an incision in the middle of the fat pad is the production of a scar in a very sensitive and commonly traumatized area. The incision must not extend to the distal interphalangeal crease because of the danger of injuring the flexor tendon mechanism. The SQ tissue is bluntly dissected using a hemostat to provide adequate drainage. A gauze pack may be placed in the wound for 24 to 48 hours to ensure continued drainage.

Figure 38-26 Hockey-stick incision for the drainage of a felon. *Note:* An incision on the ulnar side of the index, middle, and ring fingers is appropriate. The little finger is best incised on the radial side. The site of the incision on the thumb is also preferably on the radial side, but this may depend on the occupation of the patient. *(From Chase RA: Atlas of Hand Surgery. Philadelphia, WB Saunders, 1973. Reproduced with permission.)*

Recurrent or more severe infections may require a more aggressive approach by a hand specialist. The following traditional incisions have a greater propensity for complications such as sloughing of tissue and postoperative fat pad anesthesia or instability, [110] although they may provide for more complete drainage.

The hockey-stick incision is a well-accepted drainage procedure (Fig. 38-26). This incision is advantageous if the infection points to one side of the finger. The incision begins in the midline of the tip of the fat pad, just under the distal edge of the fingernail. It is extended to the lateral tip of the finger and proximally along the side of the distal phalanx (at the junction of the volar and dorsal skin markings) to 3 to 5 mm distal to the distal interphalangeal joint. The tip of the knife blade is inserted just under the bone to a depth corresponding to the opposite edge of the distal phalanx—slightly more than halfway across the volar surface of the finger. A hemostat is inserted into the incision and is spread in the plane of the fingernail (perpendicular to the septa) to break open remaining septa and loculations. Necrotic tissue or any foreign matter is excised under direct vision, and the wound is irrigated. A small gauze pack is placed in the incision. Because the incision may produce partial numbness of the fingertip by associated digital nerve injury, the incision should *not* be made on the radial aspect of the index finger or the ulnar aspect of the thumb or little finger.

An acceptable alternative to the hockey-stick, or median, incision is the through-and-through incision (Fig. 38-27). This is basically a hockey-stick-type incision (without the curved distal portion of the "hockey stick") that is carried through to the opposite side of the finger. A hemostat is used to break up loculations, and a rubber drain (Penrose) is placed through the incision for continual drainage. The through-and-through incision is probably the easiest procedure for most felons.

The fishmouth, or horseshoe, incision is basically two hockey-stick incisions that meet at the tip of the finger. A gauze pack is placed between the flaps and should be removed in a few days. This is a rather radical procedure but allows complete visualization and debridement of necrotic tissue (Fig. 38-28). Some clinicians advise against this incision, because it is extensive and may take a long time to heal. In addition, it produces a sizable scar and an unstable finger pulp. The fishmouth incision may be used if more conservative incisions are not successful, but it is not recommended for use initially.

No matter which incision is made, it must not be carried proximal to the closed pulp space because of the danger of entrance into the tendon sheath or the joint capsule. The patient should be rechecked in 2 to 3 days. A snug dressing, splinting and elevation, and adequate opioid analgesics are prerequisites for a successful outcome and a happy patient.

On the first postoperative visit, a digital block may again be performed and any packing removed. The incision is irrigated copiously with saline, and any additional necrotic tissue is removed. At this time, the drain may be replaced for 24 to 48 hours if there is continued drainage, but usually it can be removed and a dressing reapplied. Soaking may be advised. At the first revisit, the sensitivities of the bacterial cultures are checked, and a decision to continue or change antibiotics is

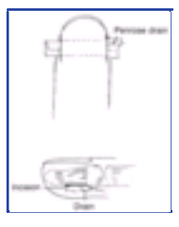

Figure 38-27 Through-and-through incision for a felon. A Penrose drain is placed for a few days to promote the withdrawal of fluid. This is an alternative to the hockey-stick incision.

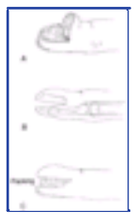

Figure 38-28 *A–C,* Fishmouth incision. This is a rather radical incision that is best reserved for resistant cases. Its advantage is that it allows for complete drainage and visualization of the infection in complicated cases. This incision takes longer to heal than others and may leave a large and sensitive scar. The fingertip may also be left unstable as a result of the lysis of all septa. The incision is seldom used on an outpatient basis for these reasons.

made. Most felons are empirically treated with antibiotics for at least 5 days. A broad-spectrum cephalosporin is a reasonable choice, pending cultures (if done).

A few additional points should be emphasized at this time. Frank pus may be encountered during incision, but usually only a few drops are expressed. One more often drains a combination of necrotic tissue and interstitial fluid. A careful search for a FB should be made even if the history is not known. Some clinicians advocate radiographic evaluation for retained FBs and a baseline evaluation of the bone for subsequent evaluation of osteomyelitis at the initial visit. Other clinicians will reserve radiographs for wounds not showing significant improvement in 5 to 7 days. Evidence of osteomyelitis, however, may not be found radiographically for several weeks after the appearance of the lesion. More radical incision and drainage may be required in persistent infections. Following adequate drainage, osteomyelitis may respond surprisingly well to outpatient antibiotic therapy with almost complete regeneration of bone if incision and drainage have been adequate. Persistent cases may require IV antibiotics.

Resistant fingertip infections are not uncommon.

Difficult or persistent cases require evaluation and care by a hand surgeon. In these cases, early consultation is advisable to avert catastrophic complications such as loss of function or amputation.

SEROMA AND HEMATOMA DRAINAGE

Although most incision-and-drainage procedures are performed for decompression of purulent collections, drainage of sterile hematomas or seromas may be required in the ED. In general, the same principles used for formal drainage of pus in the soft tissues apply to drainage of a sterile fluid collection, and hence one can directly apply the principles of this chapter to the drainage of sterile fluids. In addition, when a sterile fluid collection is drained, the operator has the option of primarily closing the incision site following wound irrigation (see Chapter 35 and Chapter 36 for wound management techniques). Drainage of a soft tissue hematoma is generally best postponed several days following an initial injury to permit hemostasis and to minimize the risk of hematoma reaccumulation following drainage. The procedure is generally reserved for those soft tissue hematomas that are large and painful (secondary to tissue distention) and are expected to either resolve slowly or result in soft tissue deformity if not drained. Seromas and hematomas rarely become infected if the overlying skin remains intact. It is best to avoid needle aspiration since this procedure rarely drains the collection completely, and it has the potential to introduce infection into a good culture medium in a closed space. If a hematoma becomes infected, it should be treated as a cutaneous abscess.

Although tempting to drain a small seemingly fluctuant noninfected hematoma that has persisted for many days, *a conservative nonoperative approach is usually best*. A persistent mass after trauma is usually concerning to patients, so a thorough explanation should be forthcoming. Most hematomas will resolve, albeit slowly (weeks), and incision often is disappointing in its yield (unless the hematoma is large and superficial) and leaves a scar. Drainage of a subungual hematoma represents a special case of hematoma drainage.

Subungual Hematoma

Subungual hematoma is an injury that is frequently seen in the ED. Any digit may be affected. The hematoma often results from hitting the fingertip with a hammer or slamming it in a door. The main concern of the patient is relief of the terrible throbbing pain that accompanies the condition as the pressure of the hematoma increases. Pain relief can be accomplished quickly with nail trephination. Trephination may be performed with a large paper clip that has been heated until it is red hot. The instrument is applied to burn a hole at the base of the nail (Fig. 38-29). Blood rapidly exits, and the blackened nail regains its normal color (Fig. 38-30). The blood usually remains fluid for 24 to 36 hours and is easily expressed with slight pressure. Care should be taken to make multiple holes or a single hole that is large enough to allow continued drainage. An oversized paper clip is the simplest apparatus. Although a portable hot-wire electrocautery unit is available and is frequently recommended, it is difficult to obtain an adequate drainage hole without adapting the instrument and its use. One can modify the electrocautery device to burn a larger hole by "fattening" the end of the wire loop and rotating the device slowly as the nail is penetrated or by removing a small rectangle of nail with the cautery device. In addition to being convenient, the cautery device is desirable because the wire stays hotter longer, thus enhancing nail penetration. In the stoic patient, no anesthesia may be necessary, but a digital block affords painless trephination, and its routine use is suggested with the anxious patient.

The majority of subungual hematomas are painful but minor injuries. Complicated cases involve fractures of the distal phalanx. When the fingertip is unstable or the mechanism

Figure 38-29 *A*, This patient slammed his finger in a car door, sustaining an acute flexion of the distal phalynx and a crush injury. It appears to be a simple subungual hematoma, but note the blood in the paronychial space. There is a communication between the nail bed laceration and the eponychium. *B*, All of the blood did not drain when the nail was trephinated. *C*, Blood accumulated in this area because the base of the fingernail has been avulsed, and lies between the eponychial fold and the skin of the cuticle. This is appreciated when the skin is debrided. The closed nature of the injury causes the confusion. With the nail removed, the nail bed laceration can be appreciated and repaired. *D*, The old trephined nail can now be replaced to its original position to keep open the eponychial space, or that space is packed with gauze for a few weeks to discourage scar formation and a subsequent nail deformity.

of injury suggests a significant distal phalanx fracture, a radiograph should be obtained. Radiographs help differentiate tendinous and bony mallet-type injuries. [111] If a significant fracture is present, the digit should be splinted. A distal phalangeal fracture with a subungual hematoma is technically an open (compound) fracture. Such injuries usually heal without problems, although osteomyelitis of the tuft is a theoretical complication. The value of routine antibiotic prophylaxis in such cases is unproved, and their use is not standard in minor cases but may be of value in significant crush injuries. The presence of an underlying fracture does not contraindicate nail trephination for fear of changing closed fracture into an open one.

It is difficult to predict the fate of the fingernail following drainage of a subungual hematoma. Some patients with subungual hematomas will lose the nail, but if the nail root or nailbed is not significantly disrupted and the nail remains implanted, a normal-appearing nail is the usual final result. Treatment of subungual hematomas by simple trephining gives an acceptable result in most patients; poor outcome cannot be predicted at presentation. [112] If the nailbed is significantly lacerated or the edges of the nail are unstable, the nail may be removed and the nailbed repaired (see further discussion in Chapter 36). Patients should be informed of possible future cosmetic problems.

One condition that may be mistaken for a simple subungual hematoma is the closed avulsion of the base of the fingernail that occurs in conjunction with a subungual hematoma from a nailbed laceration. A common mechanism is slamming a finger in a car door, with sudden flexion of the distal phalynx in conjunction with a crush injury of the nail bed. The nail itself is usually stable, so generally no repair of the nailbed appears required. However, if the subungual hematoma extends past the confines of the nail bed, such as in the paronychial space (under the skin of the cuticle), there must be a communication between the nailbed and this space. This essentially produces a paronychial hematoma, where blood occupies the space that pus occupies in an infectious paronychia (see Fig. 38-29). When this condition is present, the avulsed proximal portion of the fingernail overlies the nail fold of the cuticle, but this is only evident once the overlying skin is opened. An open reduction (replacement) of the nail must be performed. The replaced nail often grows normally but the final result may be a lost or deformed nail. Repair of the nailbed laceration is optional at this juncture, but may not be required if the nail is stable. Once the injury is anatomically

Figure 38-30 *A,* Subungual hematoma with a blackened nailbed. *B,* Following trephination, blood flows freely from the puncture site. An adequately sized drainage hole may be placed in the nail with a heated paper clip. Small holes, which tend to clog and inhibit drainage, should not be made. The hole should be large enough to allow continued drainage of blood. Note that in this example, the blood only accumulates under the nail, not in any paronychial areas.

correct, splinting and soaking follows that of a simple subungual hematoma. These injuries rarely become infected and there are no firm indications for antibiotics. Clinical judgment is suggested on individual cases.

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Chapter 39 - Burn Care Procedures

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Each year in the United States some 2 million people suffer a burn-related injury. Typical victims are children younger than 5 years of age or young adults who are exposed to fire or hot or corrosive substances. [1] Fortunately, 95% of these burns are classified as minor and are amenable to outpatient management, with most patients completing their treatment course within 2 weeks.

The classification of burns is based on three criteria [2] : depth of skin injury, percentage of body surface area involved, and source of injury (thermal, chemical, electrical, or radiation). The *seriousness* of a burn injury is determined by the characteristics and temperature of the burning agent, the duration of exposure, the location of injury, the presence of associated injuries, and the age and general health of the victim (Table 39-1).

The American Burn Association defines *minor* burns as uncomplicated partial-thickness burns of <5% of the total body surface area (TBSA) in children (<10 years old) or the elderly (>50 years old) or <10% TBSA in adults, or full-thickness burns <2% TBSA. [3] *Moderate or major* burns include injuries that involve a greater TBSA, as well as burns to areas of specialized function, such as the face, hands, feet, or perineum. More serious burns also include those due to a high-voltage electrical injury or those with associated inhalation injuries or other major trauma.

The TBSA burned may be estimated in a number of ways. It is more common to overestimate the size of a burn than to underestimate it. In adults, the "rule of nines" is a useful rule of thumb, but the formula is only a guide and *must be modified for children* who have proportionately larger heads and smaller legs (Fig. 39-1). The Lund and Browder charts are another (more precise) guide to estimating the percentage of TBSA burned (Fig. 39-2). For smaller or multiple burns, one can rapidly estimate the TBSA burned by using the area of the patient's palm as approximately 1.25% TBSA.

Throughout the course of history, clinicians have experimented with burn therapies to relieve pain and promote healing. Many treatment regimens and useless home remedies have been successful, largely due to the fact that minor burns generally do well with a modicum of intervention, and common sense wound care. While little has changed in the care of minor ambulatory burns over the past 20 years, the treatment of major burns, the development of sophisticated burn centers, increased knowledge of burn wound physiology, and the prevention of infection have significantly altered the care of seriously burned patients.

WOUND EVALUATION

Emergency clinicians should be aware that the depth of a burn wound cannot always be determined accurately on clinical grounds alone at the time of presentation and that burn injury is a dynamic process that may change over time, particularly during the 24 to 48 hours after the burning process has been arrested. It is common, for example, for a seemingly minor or superficial burn to appear deeper on the second or third return visit (Fig. 39-3). This phenomenon is not a continuation of the burning process but is considered to be a pathophysiologic event related to tissue edema, dermal ischemia, or desiccation. [4]

First-degree burns involve the epidermis only. The skin is reddened but is intact and not blistered. This injury ranges from mildly irritating or even pruritic to exquisitely painful. Minor edema may be noted. Causes include ultraviolet light (as in sunburn) and brief thermal "flash" burns. First-degree burns frequently blister within 24 to 36 hours, so the patient should be instructed appropriately. Often the skin begins to flake or peel within 5 to 10 days, but healing eventually occurs with no scarring.

Second-degree burns involve the entire epidermis and extend into the dermis to include sweat glands and hair follicles. *Superficial partial-thickness burns* involve only the papillary dermis. These burns are pink, moist, and extremely painful. Blisters may be present or the skin may slough. The burn blanches with pressure, and mild to moderate edema is common. Hair follicles are often noted to be intact. This is the most common depth of minor burn seen in the emergency department (ED). The usual causes are scalds, contact with hot objects, or exposure to chemicals. Barring infection or repeated trauma, these burns heal completely without scarring in about 2 weeks. Areas of first- and second-degree burns may be sensitive to subsequent sunburn, windburn, and skin irritation for months after the original injury appears healed.

Deep partial-thickness burns extend into the reticular dermis and appear as mottled white or pink. There is obvious edema and sloughing of the skin, and any blisters are usually ruptured. Blanching is absent. These burns are generally not painful initially, but pressure can be perceived. Within a few days, however, these burns can become exquisitely painful. This type of burn can easily be converted to a full-thickness injury by further trauma or infection. Partial-thickness burns heal by re-epithelialization from dermal appendages, including hair follicles, sebaceous glands, and sweat glands.

In *full-thickness burns,* coagulation necrosis extends into the subcutaneous (SQ) tissues. These burns may appear in a variety of colors but are usually dry, pearly white, or charred. They are initially painless, with a leathery texture. Marked edema and decreased elasticity may necessitate escharotomy when circulation is compromised. Exposure of the skin to - temperatures in excess of 77°C for more than 3 to 4 seconds generally causes a full-thickness injury. Although initially painless, in a few days these deep burns can become painful. Chemical burns often produce full-thickness injuries affecting a small or scattered surface area. Flame burns produce full-thickness injuries in less than 2 seconds if the temperature of the flame exceeds 500°C. Generally, any burn wound that is not re-epithelialized or does not possess dense epidermal budding by 14 days post-burn should be considered a full-thickness injury. [4]

Fourth-degree burns extend deeply into SQ tissue, muscle, fascia, or bone. These burns are characteristically caused by contact with molten metal, flame, or high-voltage electricity.

HISTOPATHOLOGY OF BURNS

One thermal wound theory describes 3 zones of injury in burns [5] :

1. *Zone of coagulation:* dead, avascular tissue that must be debrided.

2. *Zone of stasis:* injured tissue in which blood flow is impaired. Desiccation, infection, or mechanical trauma may lead to cell death.
3. *Zone of hyperemia:* minimally injured, inflamed tissue that forms the border of the wound. The hyperemia usually resolves within 7 to 10 days but may be mistaken for cellulitis.

TABLE 39-1 -- Characteristics of Burns, by Depth*

Classification of Burn	Etiology	Appearance	Sensation	Time to Complete Healing	Scarring
FIRST DEGREE					
• Superficial epidermal layers	• Sunburn, other UV exposure	• Dry, red	• Present	• 3–7 days	• No
	• Short flash flame burns	• Blanches with pressure	• May be quite painful		
SECOND DEGREE					
• Varying depth, blisters, or bullae formation					
• Dermal appendages spared (i.e., sweat glands, hair follicles)					
• Includes entire epidermis and some portion of the dermis					
Superficial partial thickness	• Water scald	• Blisters, peeling skin	• Painful	• 7–21 days	• Unusual if no infection and proper follow-up
	• Longer flash burn	• Blanches with pressure Skin red/moist under blisters	• Exposure to air and temperature is painful		• Pigment change may be seen
					• Burned area may be sensitive to frostbite, windburn, sunburn for many months
					• Itching may be problematic for weeks after healing
Deep partial thickness	• Flame	• Variable color	• Pressure only	• >21 days	• Severe; risk of contracture
	• Water immersion	• Wet or waxy dry, does not blanch			
	• Oil, grease, hot foods (e.g., soup)	• Blisters easily removed, skin peeling off			
THIRD DEGREE					
• Loss of all skin elements; thrombosis and coagulation of vessels	• Flame, steam, oil grease	• Leathery appearance, white or charred dry, inelastic; blanching with pressure	• Deep pressure only	• Never heals	• Very severe, high risk of contracture
	• Immersion, scald			• Requires grafting	
	• Caustic chemical, high voltage				
		• May be present under blisters			

**Modified after: Clayton and Solem, Post Grad Med 97:151, 1995 and Morgan, Bledsoe and Barker, Am Fam Phys 62:2015, 2000.*

Histologically, full-thickness burns are characterized by confluent vascular thrombosis involving arterioles, venules, and capillaries. Edema due to loss of microvascular integrity results not only from the effects of direct thermal injury, but also from the release of vasoactive mediators. The increase in vascular permeability is linked to complement activation and histamine release. Histamine increases the catalytic activity of the enzyme xanthine oxidase, with resultant production of hydrogen peroxide and hydroxyl radicals. These by-products increase the damage to dermal vascular endothelial cells and result in progressive vascular permeability. [6]

The cellular debris and denatured proteins of the eschar provide a substrate for the proliferation of microorganisms. The devitalized tissue (eschar) sloughs spontaneously, usually as a result of the proteolytic effect of bacterial enzymes. The greater the degree of wound bacteriostasis, the greater the delay in sloughing.

Partial-thickness burns result in incomplete vascular thrombosis, usually limited to the upper dermis. The dermal circulation is gradually restored, usually over several days, resulting in a significant interval of relative ischemia. The eschar in deep partial-thickness burns is thinner than in a full-thickness burn and sloughs as a result of re-epithelialization rather than bacterial proteolysis.

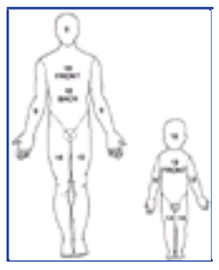

Figure 39-1 The "rule of nines" for estimating percentage of area burned. (As a rough guide, the area covered by the individual's palm is approximately 1.25% the total body surface area [TBSA].) The rule of nines is a rough estimate of the TBSA burned. Note that adults and children are different. This formula frequently *overestimates* the extent of a burn in clinical practice. See Figure 39-2 for a more accurate method of determining the TBSA burned for children.

OUTPATIENT VS INPATIENT CARE

One of the first steps in minor burn care is to select patients for whom outpatient care is appropriate (Table 39-2). *Generally there are no "unnecessary" initial admissions for patients with burn injuries.* Candidates for outpatient treatment are generally adults and children who meet the minor burn criteria detailed earlier. Clinical judgment is always the most reasonable way to decide on where the burn patient would be best cared for, and there is considerable latitude in this decision. Persons who have deep burns of the hands, face, feet, neck, or perineum; burns resulting from abuse or attempted suicide; burns involving other significant trauma or inhalation injuries; or electrical burns should generally be managed as *inpatients*.

Poor candidates for outpatient care of even minor burns include those who have concomitant medical problems such as diabetes mellitus, peripheral vascular disease, congestive heart failure, and end-stage renal disease; patients who are using steroids or other immunosuppressive agents; patients who are very young or very old; those who are mentally retarded; alcoholics; the homeless; those who are malnourished; and any individual with a suspect or unacceptable home support system. Inpatient treatment should be considered under these circumstances even though the burn might be considered "minor" by TBSA formulas. Pain control, the ability to obtain follow-up, the capacity to understand home care, and the overall social situation must influence the final decision. [7]

PROCEDURE

Initial Care of the Minor Burn Victim

Prompt cooling of the burned part is an almost instinctive response and is one of the oldest recorded burn treatments, having been recommended by Galen (129 to 199 A.D.) and Rhazes (852 to 923 A.D.).[4] Room-temperature tap water irrigation, immersion, or compresses (20 to 25°C) are optimal in obtaining pain relief and providing some measure of protection for burned tissues without the problems of hypothermia that iced solutions can cause. [8] [9] First-aid telephone advice from the emergency clinician includes immediately immersing the wound in room-temperature tap water. While immediate cold water immersion may limit the extent of a burn, and provide significant pain relief, *packing the wound in ice must be avoided*.

Emergency Medical Service (EMS) personnel should have a clear understanding of proper management of the minor burn victim. The benefits and details of early cooling should be understood and implemented. The victim should first be removed from danger. All involved clothing and jewelry, along with any gross debris, should be removed from the burned area. Chemical burns to the skin or eyes require prolonged tap water irrigation. The burn should be otherwise covered with a moist, sterile dressing—nonmentholated shaving cream makes an excellent temporary covering for out-of-hospital use if a dressing is not available. [10] Common home remedies such as butter, grease, or petrolatum usually do not adversely influence subsequent care, but they are best avoided. [11]

In the ED, the burned area should be immediately immersed in room-temperature water or covered with gauze pads soaked in room-temperature water or saline (Fig. 39-4). The gauze must be kept cool and moist to provide continued pain relief; the patient will quickly let the clinician know when additional cooling is required. Burns should be cooled or immersed until supplemental analgesia is effective and dressings are ready to be applied. Many clinicians use sterile saline for cooling, but it has no proven benefit over tap water, even when the skin is broken. It is acceptable to add ice chips to water or saline to lower the temperature. However, immersion of burned tissue in ice or ice water should be avoided, because ice immersion increases pain and risks frostbite injury or systemic hypothermia.

The potential benefits of burn cooling are listed in Table 39-3 . With the exception of pain relief and removal of debris, the benefits of burn cooling are experienced only if the burn is cooled promptly, within the first 3 minutes after injury. [12]

Prompt burn cooling arrests cell death processes by inhibiting the release of toxic substances by the dying cells. If cooling is not performed, continued cell death results as oxygen free radicals are released, which can cause a "chain reaction" of cell membrane injury, arachidonic acid release, and increased local ischemia. [13]

The threshold temperature for cutaneous pain sensation is approximately 43°C. The prompt alteration in capillary permeability and resultant edema induced by heat have been attributed to histamine release from stimulated or damaged mast cells. The tissue threshold for this phenomenon has been estimated at 52°C. Cooling produces prompt and complete, but reversible, inhibition of histamine release. Tissue edema also results from damage to the membrane sodium pump,

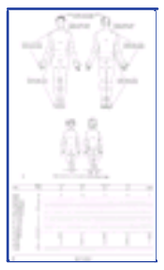

Figure 39-2 *A,* The Lund and Browder charts are somewhat more accurate than the rule of nines in estimating the TBSA burned. *B,* The proportion of TBSA of individual areas, according to age. Compared with adults, children have larger heads and smaller legs. Other areas are relatively equivalent throughout life. The rule of nines is not accurate in determining the percentage of TBSA burned in children.

Figure 39-3 It may be difficult to accurately assess the depth or severity of a burn on the first visit. *A*, This is a full-thickness burn that will not heal without a skin graft. *B*, This blistered hot water burn is likely second degree, but full-thickness burns can develop under blisters.

with resultant influx of sodium into tissue and loss of protein into the interstitial spaces. Unfortunately, this contribution to tissue edema is less responsive to local cooling.[1]

Minor burns are considered tetanus-prone, and tetanus toxoid should be administered if the patient is unsure of his or her tetanus immunization status or when it has been more than 10 years since the last immunization. Nonimmunized patients should receive human tetanus immune globulin, 250 units intramuscularly, along with tetanus toxoid, and a booster injection of toxoid in about 3 weeks.

Definitive Care of the Minor Burn

Few areas in medicine are fraught with as much mysticism, personal bias, and unscientific dogma as the care of the minor burn wound. Many clinicians are rigidly committed to a specific ritual or approach merely because "it is the way it's done" in a specific institution or because the practitioner has had success with a particular therapy in the past. In reality, the plethora of successful regimens attests to the fact that almost any noninjurious approach results in a favorable outcome. Many misconceptions probably arise because the issues associated with major thermal injury are often erroneously extrapolated to the minor burn wound. Most minor outpatient burns do very well, regardless of therapy, and it is difficult to do anything wrong if common sense is evoked.

Minor burns are *not* associated with immunosuppression, hypermetabolism, or increased susceptibility to infection. [14] Many complications seen in minor burn care result from

TABLE 39-2 -- American Burn Association's Grading System for Burn Severity and Disposition of Patients

	Type of Burn		
	Minor	***Moderate***	***Major***
Criteria	<10% TBSA burn in adult	10%–20% TBSA burn in adult	>20% TBSA burn in adult
	<5% TBSA burn in young or old	5%–10% TBSA burn in young or old	>10% TBSA burn in young or old
	<2% full-thickness burn	2%–5% full-thickness burn	>5% full-thickness burn
		High-voltage injury	High-voltage burn

		Suspected inhalation injury	
		Circumferential burn	Known inhalation injury
		Concomitant medical problem predisposing the patient to infection (e.g., diabetes, sickle cell disease)	Any significant burn to face, eyes, ears, hands, feet, genitalia, or joints
			Significant associated injuries (e.g., fracture, other major trauma)
Disposition	Outpatient management	Hospital admission	Referral to burn center

Burn = partial-thickness or full-thickness burn, unless specified; TBSA = total percentage of body surface area affected by the injury; young = patient younger than 10 years of age; adult = patient 10 to 50 years of age; old = patient older than 50 years of age.

Adapted with permission from: Hospital and prehospital resources for optimal care of patients with burn injury: Guidelines for development and operation of burn centers. American Burn Association. J Burn Care Rehabil 11:98, 1990, with additional information from Hartford CE: Care of outpatient burns. In Herndon DN (ed): Total Burn Care. Philadelphia, Saunders, 1996, p 71.

overtreatment of the injury rather than undertreatment. Examples include too-vigorous dressing changes that may peel off newly formed skin and secondary infections or pseudomembrane formation that results from topical or systemic antibiotic use.

Burn Dressings

Open burn care.

Burns are cared for using two general methods: open or closed. In the *open* method, a burn wound dressing is not used. The area is left open to the air and is washed two to three times per day, followed by application of a topical agent. This is the preferred method for managing burns of the face and neck and is an excellent way to manage minor hand burns, because it allows continuous inspection and range-of-motion exercises. The open method is impractical in young active persons, in children, or in other individuals in whom wound contamination is likely. Although many burns may be treated with this method, many patients prefer a dressing over a wound for cosmetic reasons.

Simple closed dressing.

The *closed* burn treatment method involves a dressing, of which there are various types. This is the method of choice for managing most minor burns treated in the ED. Wound preparation and basic bandaging should include the following steps (Fig. 39-5):

1. The hair in the burn itself or around the wound should *not* be shaved. The burn may be washed gently with a clean cloth or gauze pads and a mild nonalcohol-based soap or detergent (e.g., Ivory, Dove, Hibiclens) and then flushed with normal saline. However, the benefit of this

seemingly rational intervention has never been proven, and this step should be minimal. There is no need to vigorously wash a minor wound with strong antiseptic preparations (such as Betadine and others). [15]
2. Obviously sloughed skin should be debrided. This may be accomplished with scissors and forceps, but an expeditious and effective (and often painless) method is to use a dry 10-cm × 10-cm gauze pad (Fig. 39-6) to quickly debride loose skin. Meticulous and time-consuming instrument debridement is often quite stressful to the patient. Analgesia should be provided for any painful debridement. Some clinicians prefer to debride a wound on a subsequent follow-up visit when the wound has matured and reached its full extent, thereby hoping to subject the patient to this procedure only once.
3. In the absence of infection, intact blisters are often left alone at the first visit (see subsequent discussion on blisters). Ruptured blisters are usually debrided as soon as they are recognized (Table 39-4). All sloughed skin and blisters are debrided if infection is present.
4. A fine mesh gauze or a commercial nonadherent gauze such as Adaptic or Aquaphor is applied to the dry burn wound.
5. The burn is covered with loose gauze fluffs. If fingers and toes are included in the dressing, *the web spaces are padded and the digits are individually wrapped and separated with strips of gauze*. Failure to individually wrap fingers and toes may result in further injury (see Figure 39-5*D*).
6. The entire dressing is wrapped snugly (but not tightly) with an absorbent, slightly elastic material such as Kerlix.
7. Antibiotic creams or ointments may be used as an option with this dressing. The topical antibiotic may be applied to the burned skin directly or impregnated into the gauze after step 3.

Figure 39-4 To cool a burn that cannot be easily immersed in water, cover the area with unfolded gauze pads that have been soaked in room-temperature saline. Continue to frequently soak the gauze with cool saline or tap water drawn up in a syringe. Adding a few ice chips to the liquid is helpful, but do not cover the burn with ice. Towels are generally too bulky for this procedure.

TABLE 39-3 -- Advantage of Prompt Burn Cooling

Reduction or cessation of pain
Elimination of local hyperthermia
Inhibition of postburn tissue destruction
Decreased edema
Reduced metabolism and toxin production

Burn dressings should enhance healing. Much is made of specific dressings and dressing materials, but no single approach has proven superior efficacy. *The most important characteristic of a dressing is that it is capable of controlling fluid balance.* To accelerate healing, a burn dressing should be designed to keep the wound surface moist but avoid pooling of fluids. [16] The best material for this purpose is a generous amount of *simple dry gauze* applied over a nonadherent dressing or topical preparation. The outer dressing layer should be porous to permit the evaporation of water from the absorbent dressing material. Some clinicians prefer to eschew a nonadherent portion of the dressing so that subsequent dressing removal aids in minor debridement.

Biologic dressings.

Biologic dressings are natural tissues, including skin, that consist of collagen sheets containing elastin and lipid. They are not routinely used in emergency care of minor wounds. Benefits of biologic dressings include a reduction in surface bacterial colonization, diminished fluid and heat loss, prevention of further wound contamination, and prevention of damage to newly developed granulation tissue. Examples of biologic dressings include cadaveric human skin and commercially available porcine xenograft or collagen sheets.

Synthetic dressings.

Synthetic dressings are manufactured in various forms. Film-type dressings have a homogeneous structure and are usually polymers. Because these dressings are

nonpermeable, there have been problems with retention of wound exudate. Some second-generation dressings have been developed to address these problems. These products include *Tegaderm*, *Vigilon*, *DuoDerm*, *Biobrane*, *Op-Site Omniderm*, *Sildimac, and others*.[17] These preparations have theoretical benefits under certain circumstances, but none has proven superior performance over simple gauze dressings for minor outpatient burns. These products are most often used by burn centers and have little applicability for minor burns discharged from the ED.

Application of biologic and synthetic dressings.

All of these dressings are best used on fresh *partial-thickness burns*; placement over a contaminated wound often results in nonadherence and infection. Biologic and synthetic dressings are readily available. These dressings provide immediate relief of pain and are the only dressings that may actually promote faster wound healing.[2] In addition, these dressings have the potential for a reduced number of dressing changes.

Biologic dressings, such as porcine xenograft, are applied as follows: The dressing is reconstituted as instructed by the manufacturer and is carefully "fit" to the wound. All fluid and air under the graft are pressed out. The dressing is secured with edge tapes and covered with coarse mesh gauze, and then the dressing is wrapped with absorbent gauze and a semi-elastic wrap. With healing, the biologic dressing dries, curls, and separates from the healed edge underneath. The loose edges should be trimmed frequently, any blisters debrided, and fluid accumulations drained.

Synthetic dressings are applied after standard wound cleansing. Blisters must be debrided before application of the

Figure 39-5 Outpatient burn dressing of the hand. Persons with serious hand burns should be admitted to the hospital, but persons with minor burns can be treated in the outpatient setting. Following application of an antibiotic ointment or a dry, nonadherent dressing, the fingers are separated with fluffs in the web spaces *(A),* and the entire hand is enclosed in a position of function *(B)* (here with the help of a roll of Kerlix). If the wrist is involved, a removable plaster splint may be applied over the dressing *(C)*. *D,* The result of a minor burn to the hand when the fingers were not wrapped individually. Initially there were only a few blisters, but this patient now has second-degree skin loss due to an improper burn dressing that caused maceration of normal skin. Not only were the fingers *incorrectly* wrapped together in one gauze wrap, but the first wound check was *incorrectly* scheduled in 6 days, too long for the first wound check in a hand burn.

synthetic dressings. The thicker membranes are cut to size and applied to the burn surface. The edge of the membrane should extend 2 to 3 cm beyond the burn onto the surrounding unburned tissue. Membranes such as DuoDerm can be wrapped about burned digits to permit continued function with limited motion during healing. [18]

Should fluid collect between the debrided burn and the dressing, one attempt at aspiration should be performed. If the fluid reaccumulates beneath the DuoDerm or Biobrane membranes after aspiration, the nonadherent portion of the fabric should be removed. For the first 5 to 8 days, the dressing should be checked at least every other day for adherence. After epithelialization is complete, the dressing begins to separate spontaneously and may be peeled away. [13]

If infection develops under a synthetic or biologic dressing, the dressing should be removed, the wound cultured, and treatment with topical (and possibly oral) antimicrobials instituted. Xenografts should not be used on superficial partial-thickness injuries, because the xenograft tissue may become incorporated into the healing wound in as many as 35% of patients so treated.[19]

Occasionally, application of a permanent dressing—or skin autograft—is appropriately performed in the outpatient setting. Appropriate patient selection is crucial; patients should be healthy and well motivated, with small (<2% TBSA), noncosmetic deep partial-thickness or full-thickness burns. These burns may initially be treated as outlined previously, with the use of a topical antimicrobial agent recommended. The wound should be reevaluated for excision and grafting within 4 days.

Specific Clinical Issues in Minor Burn Care

Analgesia

Pain is a much-feared feature of any burn injury. Pain relief by the appropriate and judicious use of narcotic analgesics is of paramount importance in the initial care of all burn patients. Analgesia should be provided *before* extensive examination or debridement is performed. Inadequate analgesia is probably the most common ED error in the treatment of burn injuries. This error is most common in children. Parenteral narcotic analgesics have been erroneously relegated to pain control in only major burns, but it is suggested that intramuscular (IM) or intravenous (IV) narcotics be generously administered in the initial treatment of even minor painful burns.

Even with minor burns, patients often present with significant pain. Reassurance is important, but adequate analgesia is critical for gaining the patient's cooperation and ensuring comfort. Cooling may dramatically relieve pain temporarily, but parenteral opioids (meperidine, 1 to 2 mg/kg, or morphine, 0.1 to 0.2 mg/kg) are usually required, especially if painful procedures such as debridement and dressing changes are planned. We prefer to use IV opioids (occasionally supplemented with a short-acting benzodiazepine such as midazolam) for all painful procedures. For complicated debridement or dressing changes, adequate analgesia and sedation (see Chapter 34) is strongly advocated.

IM medications following the procedure are helpful for prolonged analgesia. Regional or nerve block anesthesia is an excellent alternative when practical, and when feasible, nitrous

Figure 39-6 It is difficult to do anything wrong with minor burn blisters, and many regimens are acceptable. Eventually, however, blisters will have to be debrided. An expeditious and relatively painless way to debride a burn is to use a dry gauze pad to grasp the dead skin *(A)* and peel it off *(B)*. Meticulous instrument debridement is often time consuming and stressful to the patient. Be aware that pain occurs when air comes in contact with the debrided skin and prophylactic analgesia should be provided.

oxide analgesia may be used. Oral opioids are inappropriate for initial treatment of significant pain but can be used for continued outpatient analgesia. Local anesthetics may be injected in small quantities when appropriate, such as for the debridement of a deep ulcer or other small burn. Topical analgesics have no role in burn care. A properly designed dressing will do much toward preventing further discomfort after release home; however, home burn care and dressing changes may be quite painful. For this reason an adequate supply of an oral opioid analgesic should be provided, and responsibility should be encouraged in analgesic use.

Edema

Minor burns lead to immediate inflammation mediated by the release of histamine and bradykinins, causing localized derangements in vascular permeability, with resultant burn wound edema. This edema is harmful in several ways. First, the increase in interstitial fluid increases the diffusion distance of oxygen from the capillaries to the cells, increasing hypoxia in an already ischemic wound. Second, the edema may produce untoward hemodynamic effects by a purely mechanical

mechanism: compression of vessels in muscular

TABLE 39-4 -- General Approach to Blisters in Minor Burns*

If treated <48 hours after the burn:
1. Leave all intact blisters alone.
2. If blisters have ruptured, treat them as dead skin and debride them completely.
3. Needle aspiration is generally not advised but may be used to decompress large burn blisters that appear ready to burst.
On follow-up, or >48 to 72 hours after the burn:
1. Debride large (>5 cm in diameter) intact blisters and all blisters that have ruptured. Large, firm blisters of the palms and soles may be left intact longer. Do not aspirate blisters.
2. Do not debride small or spotty blisters until they break, or until 5 to 7 days after the burn.
Five to 7 days after the burn:
1. Debride all blisters completely
Note: Intact blisters provide significant pain relief. Be prepared for an exacerbation of pain immediately after debridement.
Prophylactic analgesia is recommended.

*All blisters and burned skin are debrided in the presence of infection.

compartments. Third, edema has been associated with the inactivation of streptococcicidal skin fatty acids, thus predisposing the patient to burn cellulites. [20]

The successful management of burn edema hinges on *immobilization* and *elevation*. Most patients are unfamiliar with the medical definition of elevation and are not aware of or convinced of its value. Patient education in this regard is critical; however, certain burns (e.g., burns in dependent body areas) are prone to edema, despite everyone's best intentions. It is for this reason that lower extremity burns in general, and foot burns in particular, are prone to problems. Major burns of the hand should be elevated while the patient is still in the ED. This is most readily accomplished by hanging the injured hand from an IV pole, with stockinette used to support the bandaged hand (Fig. 39-7).

Use of Topical Preparations/Antimicrobials

Minor burns result in insignificant impairment of normal host immunologic defenses, and burn wound infection is usually not a significant problem. Topical antimicrobials are often used; however, some believe these agents may actually impair wound healing. [21] Although the procedure is of unproven value, many clinicians routinely use antibiotic creams or ointments on even the most minor of burns. Most patients expect some type of topical concoction, so a discussion of their use, or nonuse, is prudent.

Topical antimicrobials were designed for the prevention and care of burn wound sepsis or wound infection, primarily in hospitalized patients with major burns, and there is no convincing evidence that their use alters the course of first-degree burns and superficial partial-thickness injuries. As noted, the burn dressing is the key factor in minimizing complications in all burns. Nonetheless, topical antimicrobials are often soothing to minor burns, and their daily use prompts the patient to look at the wound, assess healing, perform prescribed dressing changes, or otherwise become personally involved in his or her care. Keep in mind that if a topical antimicrobial is used, its effectiveness is decreased in the presence of proteinaceous exudate, necessitating regular dressing changes if the antimicrobial benefit of topical therapy is to be realized. In reality, once-daily dressing changes are most practical and are commonly prescribed, and there are no data to indicate that this regimen is inferior to more frequent dressing changes.

Figure 39-7 Elevation of a burned hand should begin in the ED. After a properly applied hand dressing is applied, the arm is suspended from an IV pole with stockinette.

All full-thickness burns should receive topical antimicrobial therapy, because the eschar and burn exudate are potentially good bacterial culture media, and deep escharotic or subescharotic infections may not be easily detected until further damage is done. All deep partial-thickness injuries likewise benefit from the application of a topical antimicrobial. In deep partial-thickness injuries, re-epithelialization occurs from a few remaining deep epidermal appendages whose protection is important. Clinical studies and culture results support the hypothesis that surface destruction of dermal islands by bacterial enzymes and catabolic processes has the potential to convert a deep partial-thickness injury to a full-thickness injury. [22]

Initial topical therapy is prophylactic. [23] A burn wound infection that develops despite this therapy mandates a change to a different agent. Topical therapy, if chosen, should cover the usual bacteria responsible for burn wound infections (see later discussion of minor burn infections). Although topical agents are an important part of a burn treatment program, they are not substitutes for good local wound care or a careful program of management. Their successful use may prevent the conversion of deep thermal burns to deeper injury and allow better wound healing for earlier (and more successful) skin grafting.

Criteria for choosing a specific topical agent include in vitro and clinical efficacy, toxicity (absorption), superinfection rate, ease and flexibility of use, cost, patient acceptance, and side effects. Note that there are no firm scientific data that convincingly support the use of any specific topical antimicrobial in minor outpatient burns.

Specific Topical Agents

Silver sulfadiazine (Silvadene).

This poorly soluble compound is synthesized by reacting silver nitrate with sodium sulfadiazine. It is the most commonly used topical agent for outpatients, and it is well tolerated by most patients. It has virtually no systemic effects and moderate eschar penetration, and it is painless on application. Although Silvadene is commonly used, many burn specialists prefer plain bacitracin ointment as the topical of choice because of its cost, equal efficacy, and good patient acceptance.

Silver sulfadiazine is available as a "micronized" mixture with a water-soluble white cream base in a 1% concentration that provides 30 milliequivalent (mEq)/L of elemental silver. It does not stain clothes, is nonirritating to mucous membranes, and washes off easily with water. It may be used on the face, but such use may be cosmetically undesirable for open treatment. Its broad gram-positive and gram-negative antimicrobial spectrum includes ß-hemolytic streptococci, *Staphylococcus aureus* and *Staphylococcus epidermidis*, *Pseudomonas* spp., *Proteus* spp., *Klebsiella* spp., Enterobacteriaceae spp., *Escherichia coli*, *Candida albicans*, and possibly *Herpesvirus hominis*.

Silver sulfadiazine often interacts with wound exudate to form a pseudomembrane over partial-thickness injuries. The pseudomembrane is often difficult and painful to remove. Except for term pregnancy and in newborns (i.e., due to possible induction of kernicterus), there are no absolute contraindications to the use of silver

sulfadiazine. Allergy and irritation are unusual, although there is a potential cross-sensitivity between silver sulfadiazine and other sulfonamides.

Other topical preparations.

Mafenide acetate (Sulfamylon), gentamicin, chlorhexidine, povidone-iodine, and silver nitrate are products that have been replaced with newer topicals, but they are mentioned for historical interest. These products are not used in modern burn therapy, although they are generally acceptable alternatives.

Broad-spectrum antibiotic ointments.

Many nonprescription topical antimicrobials are used for minor burn therapy. Included are bacitracin zinc ointment, polymyxin B-bacitracin (Polysporin), triple-antibiotic ointments such as polymyxin B-neomycin-bacitracin (Neosporin), and nitrofurazone (Furacin). These are all soothing, cosmetically acceptable for open treatment (such as on the face), and are effective antiseptics under burn dressings. Some researchers caution against agents containing neomycin because of a potential for sensitization (Fig. 39-8). The editors suggest plain bacitracin ointment as the routine topical agent, although Silvadene is a very acceptable alternative.

Aloe vera cream.

Aloe vera cream is commercially available in a greater than or equal to 50% concentration with a preservative. It exhibits antibacterial activity against at least four common burn wound pathogens: *Pseudomonas aeruginosa*, *Enterobacter aerogenes*, *S. aureus*, and *Klebsiella pneumoniae*. Heck and colleagues compared a commercial aloe vera cream with silver sulfadiazine in 18 patients with minor burns. [24] Healing times were found to be similar, and there was no increase in wound colonization in the aloe vera group as compared with the patients treated with silver sulfadiazine. Other authors have promulgated the use of aloe gel preparations for minor burns. [25] Aloe vera cream is an acceptable inexpensive option for open or dressed outpatient care of minor burns.

Honey.

Honey has long been advocated as an inexpensive and effective topical for minor outpatient burns. The

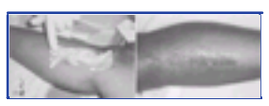

Figure 39-8 *A*, The most popular topical burn preparation is Silvadene cream. While commonly used on minor burns, it likely has little beneficial effect on healing, and minor burns rarely become infected. Nonetheless, Silvadene is a standard intervention that at least causes the patient to look at the burn and become involved in dressing changes. *B*, Many burn specialists suggest inexpensive topical antibiotic ointments (such as bacitracin and neosporin) for all outpatient burns. They are commonly used on face and neck burns. Bacitracin is preferred since a contact dermatitis, such as noted in this abrasion, can occur from the neomycin portion of some topicals.

physicochemical properties of honey (osmotic effect, pH) give this product antibacterial and anti-inflammatory properties that support its use. It may be superior to Silvadene with regard to minor burn wound healing. Honey is not widely used, but it has been promulgated as a safe, effective, and inexpensive dressing for the management of outpatient burn wounds. [26] [27] [28]

Corticosteroids.

High-potency topical steroid preparations have no beneficial effects on the rate of healing, or limitation of scarring, of thermal burns. Although likely not harmful, their use is not supported. [29]

FOLLOW-UP CARE OF MINOR BURNS

The specifics of outpatient follow-up of minor burns are controversial and often based on clinician preference and personal bias rather than on firm scientific data. Follow-up should be individualized for each patient and should be based on the reliability of the patient, the extent of the injury, the frequency and complexity of the dressing changes, and the amount of discomfort anticipated during a dressing change. Often fast-track sections of the ED are used for burn checks. The physical therapy departments of most hospitals have excellent facilities to follow outpatient burns with periodic clinician oversight.

If a topical antibiotic agent is used, the dressing should be changed daily with removal and reapplication of the topical preparation. The wound should be rechecked by a clinician after 2 to 3 days and periodically thereafter, depending on compliance, healing, and other social issues. If a dry dressing is opted for, follow-up every 3 to 5 days is usually adequate. The purpose of any burn dressing changes or home care regimen is defeated if the patient cannot afford the material or is not instructed in the specifics of burn care. Many EDs supply burn dressing material on patient release. (A complete pack includes antibiotic ointment/cream, gauze pads [fluffs], an absorbent gauze roll, a sterile tongue blade to apply cream, and tape.) Providing limited supplies of the items necessary for dressing changes may enhance compliance to follow-up if the patient has to return for additional supplies. Writing a prescription and merely stating that the dressing should be changed daily is often futile.

Daily home care can be performed by the patient with help from a family member or visiting nurse (Table 39-5). The dressing may be removed each day and gently washed with a clean cloth or a gauze pad, tap water, and a bland soap. Sterile saline and expensive prescription soaps are not required. A tub or shower is an ideal place to gently wash off burn cream. The affected area may be put through a gentle range of motion during dressing changes. After the burn is cleaned, it is inspected by the patient. The patient is instructed to return if signs or symptoms of infection, significant blistering, or skin slough develop. Following complete removal of the old cream, a new layer is applied with a sterile tongue blade and covered with absorbent gauze.

If the undermost fine mesh gauze of a dry dressing is dry and the coagulum is sealed to the gauze, the patient should simply reapply the overlying gauze dressing. If the wound is macerated, the fine mesh gauze should be removed and the wound cleaned and redressed. The patient is instructed *not to remove a dry adherent fine mesh gauze from the underlying crust*. When epithelialization is complete, the crust will separate, and the gauze can be removed at that time. Dryness in healing skin may be treated with mild emollients such as Nivea (Beiersdorf, Inc., Norwalk, CT) or Vaseline Intensive Care lotion (Chesebrough Ponds, Inc., Greenwich, CT). Natural skin lubrication mechanisms usually return by 6 to 8 weeks. [14] Excessive sun exposure should be avoided during wound maturation, as this may lead to hyperpigmentation. When the patient is outdoors, a commercially available sun block should be used. Exposure of the recently healed burned area to an otherwise minor trauma (chemicals, heat, sun) may result in an exaggerated skin response. Pruritus is common, and may be treated with oral antihistamines or a topical moisturizing cream.

Deep partial-thickness burns, along with small third-degree burns, may be initially managed in the outpatient setting with proper follow-up. Topical antimicrobials are recommended.

TABLE 39-5 -- How to Change a Burn Dressing at Home: Patient Instructions

1. Take pain medicine ½ hour before dressing change if you find dressing changes to be painful.
2. If the burn is on the hand, foot, or other area that is difficult to reach, have someone help you.
3. Have all material available. Gloves may be worn.
4. Remove the dressing and rinse off all burn cream or ointment with tap water, under a shower, or in the bathtub. The area can be gently washed with mild soap and a clean cloth or gauze pads.
5. Look at the burn and assess the healing, blistering, and amount of swelling. Note any signs of infection.
6. Gently exercise the area through range of motion.
7. Apply the burn ointment with a sterile tongue blade.
8. Cover the cream with fluffed-up gauze.
9. Wrap the area in bulky gauze.
10. Repeat this dressing change daily.

Outpatient Physical Therapy for Burn Care

When the hospital's outpatient physical therapy department is equipped to treat minor burns, it is prudent to consider this option. Many centers make available daily or periodic burn treatment, consisting of dressing changes, whirlpool debridement, and range-of-motion exercises. When patients are unable to handle their own burns at home, this can be an invaluable adjunct. An additional advantage is that medically trained personnel evaluate the burn daily, thereby decreasing clinician visits and enabling identification of problems prior to the development of serious complications. Generally all that is required from the clinician is to write a prescription for "burn care and dressing changes" and set up the appointment. The physical therapy department, or wound care center, can receive clinician input as needed during subsequent visits.

Burn Healing

Follow-up care will in part be guided by expectations of burn healing and observed healing. The following discussion is intended to serve as a general guide. However, burn healing is different from that of other wounds. [2] The timing is often variable, but it is proportional to burn depth. The inflammatory phase lasts 3 to 7 days (at times longer) and, if the burn is severe enough, is accompanied by the release of histamine and bradykinins, along with complement degradation. This degradation of complement may lead to immunologic, coagulation, and metabolic aberrations.

Within 1 to 3 weeks, neovascularization of the burn occurs, accompanied by fibroblast migration. Macrophages begin to replace the tissue neutrophils. Collagen production begins, but the molecules are often laid down in random fashion, leading to a scar. Re-epithelialization follows, but the presence of necrotic tissue and eschar impedes all aspects of wound healing. The amount of scar tissue produced is directly related to healing time. Burns requiring fewer than 16 days to heal generally do not scar excessively. [2]

Healing in superficial partial-thickness burns occurs within 10 to 14 days. After healing, the new epithelial layer tends to dry easily and crack. Using bland, lanolin-containing creams for 4 to 8 weeks following healing alleviates this problem. Deep partial-thickness burns heal by re-epithelialization from the wound edge and from residual dermal elements. Healing is slow and often unsatisfactory, frequently taking longer than 3 weeks, producing an unstable epithelium that is prone to hypertrophic scarring and contractures. This is a particular problem in burns that extend across joints. Burns that take longer than 2 to 3 weeks to heal are prone to infection; hence, topical antimicrobials should be used. Because these burns often heal in complicated fashion, they should be considered for referral to expedite early excision, grafting, and physical therapy.

Full-thickness burns can heal only by contraction and epithelialization at the wound edge. Burns larger than 2 to 3 cm must be excised and grafted.

Cosmetic and functional recovery follows complete epithelialization of a partial-thickness injury or successful skin grafting of a full-thickness burn. The ultimate goal is to prevent scar thickening; achieve and maintain optimal range of motion; and prevent secondary environmental damage to the skin, particularly from sun exposure. [15] Nonscented skin lotions may be used after epithelialization to keep the burn scar soft. Compression dressings are especially helpful in preventing scar thickening. Repeated evaluations are important, because burn contractures can occur up to 12 months after the injury. Nighttime splinting is useful in maintaining full extension of joints.

SPECIAL MINOR BURN CARE CIRCUMSTANCES

Blisters

The management of blisters in minor burns is controversial. *In reality, there is little one can do wrong when it comes to a clinical approach to blisters in minor burns.* Management arguments are generally theoretic or emotional; the ultimate outcome of a minor burn is rarely determined by how one deals with blisters. Intact blisters do offer a physiologic dressing that rarely becomes infected; however, most large blisters spontaneously rupture after 3 to 5 days and eventually require debridement. When the integrity of the blister is breached, the fluid becomes a potential culture medium. Clinical choices include debridement, aspiration, or simply leaving the blister intact.

Some studies suggest that intact burn blisters may allow for reversal of capillary stasis and less tissue necrosis. [2] Madden and colleagues have shown that burn exudate (as contained within intact blisters) is beneficial for the stimulation of epidermal cell proliferation. [30]

Swain and colleagues demonstrated that the density of wound colonization with microorganisms was much lower in minor burns with blisters left intact. [31] They also found that 37% of patients with aspirated blisters experienced a reduction in pain versus none of those whose blisters were unroofed. Other investigators believe that undressed wounds with debrided blisters have additional necrosis secondary to desiccation, which can convert a partial-thickness burn to a full-thickness injury. [3] Finally, intact blisters clearly provide some pain relief, as evidenced by a sudden increase in pain immediately following debridement. Increased pain should be anticipated and analgesia offered as appropriate when debridement is necessary. We suggest the guidelines in Table 39-4 as a general approach to burn blisters.

Minor Burn Infections

Prophylactic systemic antibiotics are not warranted in the routine treatment of outpatient burns.

It may be difficult to separate the erythema of the injury or healing process from cellulitis, but minor burns rarely become infected, with infection rates well under 5%. [32] There are bacteria on the skin at all times—normal skin usually harbors nonvirulent pathogens such as *S. epidermidis* and diphtheroids. Therefore, all burns are *contaminated* but not necessarily *infected*. Thermal trauma results in a coagulative necrosis. Burn wounds therefore contain a variable amount of necrotic tissue, which, if infected, acts much as an undrained abscess, preventing access of antibiotics and host defensive factors.

The microbial flora of outpatient burns varies with time after the burn. Shortly after injury, the burn becomes colonized with gram-positive bacteria such as *S. aureus* and *S. epidermidis*. After this period of time there is a gradual shift toward inclusion of gram-negative organisms, 80% of which originate from the patient's own gastrointestinal tract. [4] Common organisms seen on days 1 to 3 include *S. epidermidis*, ß-hemolytic streptococci, *Bacillus subtilis*, *S. aureus*, enterococci, *Mima polymorpha*, *Enterobacter* spp., *Acinetobacter* spp., and *C. albicans*. One week after the burn, these organisms may be seen along with *E. coli*, *P. aeruginosa*, *Serratia marcescens*, *K. pneumoniae,* and *Proteus vulgaris*.

Anaerobic colonization of burn wounds is rare unless there is excessive devitalized tissue, as occurs in a high-voltage electrical injury. [33] For this reason, routine anaerobic cultures are generally unnecessary in an assessment of infective organisms that produce minor infections.

The vast majority of superficial burns that have been treated properly do not get infected. Infection rates are well below 5%. [32] However, it is sometimes difficult to differentiate wound infection from the normal healing process, as both involve pain, edema, and erythema. A healing burn may produce a leukocytosis and a mild fever in the absence of infection, especially in children. Early (days 1 to 5) burn infections are generally caused by gram-positive cocci, especially ß-hemolytic streptococci. Streptococcal cellulitis is characterized by marked, spreading erythema extending outward from the wound margins. Despite the plethora of organisms and the presence of some gram-negative pathogens noted in superficial burn cultures, first-line treatment in the normal host is oral penicillin, 1 to 2 g/day. Alternatives include erythromycin, cephalosporins, and dicloxacillin.

Effective topical treatment at the time of initial burn care and subsequent dressing changes is meant to delay bacterial colonization, maintain the wound bacterial density at low levels, and produce a less diverse wound flora. Because outpatient management of burns should be attempted only when the risk of infection is minimal, the use of systemic antibiotics is unnecessary for minor burns, even in the setting of delayed treatment, diabetes, and steroid use. [34] Unnecessary antibiotic use may select out resistant organisms. Antibiotics in the management of minor burns have been recommended for patients undergoing an autograft procedure. [35] There are no data on the use of antibiotics as prophylaxis for patients with burns in the setting of valvular heart disease, although their use seems logical.

In minor burn care, wound cultures are not required or recommended. It is useless, for example, to culture blister fluid in the patient who presents for emergency care immediately after a thermal injury. Cultures are necessary only when overt infection develops, especially when this occurs while a topical or systemic antibiotic is being used. Cultures may also be of benefit when the infected wound is old, when hygiene is poor, or when there are old abrasions nearby. [36] Swab surface cultures are generally eschewed. Although they may adequately reflect wound flora, falsely sterile cultures are relatively frequent. These cultures do not reflect deep burn flora and give no quantitative information.

Sterile wound biopsy for culture is most satisfactory for the assessment of intraescharotic, subescharotic, or invasive infections and allows for quantification of bacterial flora. If a wound culture is taken, it should be obtained from the deepest or worst-appearing area of the burn.

Surface bacterial densities greater than 10^5 /cm^2 or tissue bacterial densities greater than 10^5 /g correlate with invasive infection. Surface colonization may be treated with an alternative topical agent, but truly invasive infection warrants the administration of systemic antibiotics. Generally, the infectious process resolves in 24 to 48 hours.

Foot Burns

Despite their relatively small surface area, foot burns tend to heal poorly, usually due to excessive edema; therefore, they are formally categorized as major burns. Foot burns are the most common burn category to fail outpatient therapy and subsequently require admission and inpatient care (Fig. 39-9). Zachary and coworkers reported on a series of 104 patients with foot burns. [37] No patient admitted on the day of injury developed burn cellulitis; in contrast, 27% of delayed admission patients had cellulitis. Their study also noted a higher incidence of hypertrophic scarring and need for skin grafting in the delayed admission group. Overall, fewer days of hospitalization were required for the initially admitted group.

Specific problems in the care of foot burns include pain, wound drainage, difficulty in changing dressings without help, inability of even motivated patients to comply with requirements for elevation, and prolonged convalescence. Hospital admission allows for splinting, intensive local burn care, physical therapy, and bed rest with elevation, which minimizes edema. For these reasons, initial admission for all but the most minor of foot burns is advised.

Hand Burns

Because of their functional importance, hand burns can be a devastating injury, despite involvement of a relatively small TBSA. Hand function is critical, regardless of whether the patient is dealing with loss of use during healing, later limitation by scar contractures, a long-term appearance change, or loss due to amputation. [38]

As with other burns, the depth and extent of the burn determine the severity of the injury. The entire surface of one hand represents only 2.5% TBSA, yet even small

burns can cause a disproportionate functional loss. Deep partial- or full-thickness hand burns, even if quite small, often warrant referral for early excision and grafting in order to limit scarring and maintain function. The skin on the dorsum of the hand is thinner than that on the palm and is more susceptible to burn injury, but must remain flexible to allow for finger motion. Any exposed tendon or bone, such as may be seen with an electrical burn, constitutes a true fourth-degree injury, which

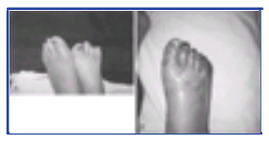

Figure 39-9 Burns of the feet are specialized burns that require a careful evaluation and an individualized treatment plan, even if the burn surface area is relatively small. It is difficult for most patients to provide ideal burn care at home when the feet are involved. *A*, It is tempting to initially treat this seemingly minor superficial second-degree foot burn in an outpatient setting, but the patient's compliance and social situation must be ideal for a successful outcome. Hospitalization until home health care can be established is prudent. *B*, An example of a foot burn that is a potential disaster, in this case due to a late presentation in a diabetic.

requires either flap closure or amputation in order to heal the wound.

Many of the issues complicating outpatient management of foot burns are relevant to the care of hand burns. After initial burn cooling, the wound should be gently cleansed with mild soap. Any loose skin or ruptured blisters should be gently debrided, rinsed, patted dry, and covered with a topical antimicrobial agent and a nonadherent, bulky gauze dressing. *The fingers should be carefully separated and bandaged individually.* Small, intact blisters that do not interfere with hand function should be left intact to serve as a biologic dressing. Elevation of the hand is very important in the first few days after a burn injury in order to minimize edema. Deep partial-or full-thickness burns to the dorsum of the hand should be splinted after bandaging to avoid the development of contractures or a boutonniere deformity.

Hospital admission should be considered for all hand burns, particularly full-thickness injuries and circumferential burns involving the digits (Fig. 39-10). If outpatient treatment is attempted, the patient must be given comprehensive instructions and should have the resources available to perform daily dressing changes and range-of-motion exercises of the fingers and wrist during these dressing changes. An initial follow-up visit should be arranged in 48 to 72 hours, but the patient should be encouraged to return if there is development of a burn cellulitis, worsening pain, fever, or lymphangitis. Ideally, the patient should be seen twice in the first week after injury and once a week after that until the burn is healed.

Facial Burns

Facial burns commonly result from unexpected ignition flash burns (e.g., from a stove, oven, or charcoal grill) or from car radiator accidents (Fig. 39-11).[39] [40] Facial burns from these sources usually do well, but often result in singeing of facial hair, significant edema, and pain. However, facial burns from these etiologies may rarely produce airway problems and require skin grafting. Concurrent globe or corneal injury is quite rare due to protective blinking reflexes. If the eye is burned, it is usually in the setting of a life-threatening concomitant burn injury. [41] Burns to the eyelids can cause significant scarring. Fluorescein staining and slit-lamp examination should be used to confirm the diagnosis of suspected corneal injury. The treatment of a corneal injury involves irrigation, topical ophthalmic antibiotic ointment, and consideration of eye patching versus protective soft contact lens (see Chapter 64). Referral to an ophthalmologist is usually prudent. Facial burns are otherwise treated in the usual fashion, and with an open (no dressing) technique. Patients are instructed to wash the face two to three times a day with a mild soap and then apply a thin layer of antibiotic ointment,

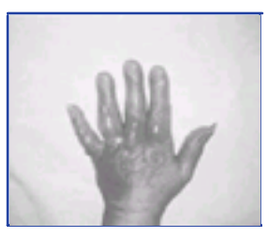

Figure 39-10 This badly burned hand requires referral to a burn center and should not be handled as an outpatient.

Figure 39-11 *A*, Flash burns to the face from lighting a gas stove. These burns are painful and may cause edema, but usually they do well. Note the singed facial hair. The eyes are usually protected by rapid reflex blinking and carbon monoxide poisoning and pulmonary burns are not an issue. Most can be handled in the outpatient setting with bacitracin ointment and no dressing. Pain control may be problematic unless opioids are prescribed. *B*, Facial and neck burns when a radiator cap was removed and the victim was sprayed with steam and hot antifreeze.

such as bacitracin zinc. There are no compelling reasons to avoid Silvadene on the face, but by tradition bacitracin ointment has become the preferred topical. Car radiator burns result from the combination of a hot liquid and steam burn. Antifreeze does not produce a caustic injury, nor is it systemically absorbed. Neck burns are treated similarly.

All patients presenting with head or neck burns should be carefully evaluated for a concomitant inhalation injury. Such patients may present with direct evidence of injury, such as oral burns, blisters, soot, or hyperemia, a history of being in an enclosed space, or with indirect evidence, such as dyspnea, wheezing, arterial hypoxemia, or an elevated carboxyhemoglobin level. The definitive diagnostic test for inhalation injury is fiberoptic bronchoscopy. [42] Flash ignition burns to the face do not pose a problem with carbon monoxide poisoning and inhalation injuries are generally not a consideration Inpatient care should be considered for all patients with significant facial burns. Outpatient pain control may be difficult in facial burns, the degree of edema may be difficult to predict, and home care can be problematic. There are no universally agreed-upon standards for admission versus outpatient treatment of facial burns, but a liberal admission policy is suggested.

Corneal contact burns, as from accidental contact with a curling iron, often present rather dramatically, with opacified, "heaped-up" corneal epithelium. Despite their appearance, the end result is usually excellent. Treatment is the same as for a corneal abrasion. [43]

Abuse of Children and Elderly Individuals

Recognition of the possibility of deliberate abuse by burning in the pediatric and geriatric populations is essential. In addition, children younger than 2 years old have a thinner dermis and a less well-developed immune system than do adults. Elderly patients (older than 65 years) likewise tolerate burns poorly. These two populations are the most prone to abuse, often by family members (Fig. 39-12). For these reasons, both groups of patients often require inpatient care. [9]

The majority of abused children are 18 to 36 months old, and for unknown reasons the majority are male. [21] Immersion burns are a common type of abuse. These are characterized by circumferential, sharply demarcated burns of the hands, feet, buttocks, and perineum. Cigarette burns and burns from hot objects such as irons should be obvious. Contact burns on "nonexploring" parts of the child also warrant suspicion. A delay in seeking treatment may be a tip-off that a burn results from abuse.

Burns in Pregnancy

There is little information in the literature concerning the special problems of the pregnant burn victim. Ying-bei and Ying-jie reported on 24 pregnant burn patients representing a wide range of burn severity. [44] Complications of the burn injuries included abortion and premature labor, although all patients in this series with burns covering less than 20% TBSA did well and delivered living full-term babies.

As the resistance of pregnant women to infection is lower than that of nonpregnant women, control of burn wound infection is paramount. Gestational age appears to have no direct bearing on prognosis. Silver sulfadiazine cream should be avoided near term because of the potential for kernicterus.

SPECIFIC BURNING AGENTS

Hot Tar Burns

Asphalts are products of the residues of coal tar commonly used in roofing and road repair. These products are kept heated to approximately 450°F. When spilled onto the skin, the tar cools rapidly, but the retained heat is sufficient to produce a partial-thickness burn. Fortunately, full-thickness burns are unusual. Cooled tar is nonirritating and does not promote infection. When cooled tar is physically removed, the adherent skin is usually avulsed (Fig. 39-13). Careless removal of the tar may inflict further damage on burned tissues. Agents such as alcohol, acetone, kerosene, or gasoline have been used to remove the tar, but these are flammable and may cause additional skin damage or toxic response secondary to absorption.

There is no great need to meticulously remove all tar at the first visit.

Obviously devitalized skin can be debrided, but adherent tar should be emulsified or dissolved rather than

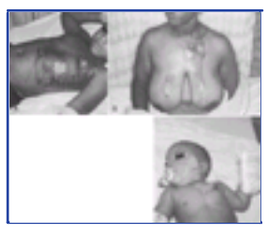

Figure 39-12 Burns can be a manifestation of child abuse, spouse abuse, or abuse of the elderly. *A*, Abuse burns from contact with a hot metal grate, from a child allegedly falling. *B*, This burn was the result of spouse abuse, caused by throwing hot soup during an argument. The delayed presentation to the hospital was a clue. *C*, Burns of the face and neck are common when a toddler pulls hot liquid from a stove. This case was never proven to be child abuse, but burns in young children often are due to abuse, especially if they are in atypical places. Although the body surface area of this burn is relatively small, the patient's age and the burn's location, coupled with the possibility of child abuse, require that this child be hospitalized.

manually removed (Fig. 39-14). Polyoxyethylene sorbitan (Tween 80 or polysorbate 80) is the water-soluble, nontoxic emulsifying agent found in neosporin and several other topical antibiotic creams. Note that the cream formulations, not the ointments, contain the most useful tar dissolvers. The creams contain a complex mixture of ethers, esters, and sorbitol anhydrides that possess excellent hydrophilic and lyophilic characteristics when used as nonionic, surface-active emulsifying agents. With persistence, most tar may be removed (emulsified) on the initial visit.

Another household product (De-Solv-It multi-use solvent) also appears logical for topical ED use. [45] The De-Solv-It product has a surface-active moiety that wets the chemical's surface and emulsifies tar and asphalt. Since the latter product is itself a petroleum-based solvent, it should be applied only briefly, and the operator should wear gloves and protective eyewear during application. It should be used only for external exposure to tar or asphalt.

Many clinicians prefer instead to emulsify the majority of tar on an outpatient basis. A generous layer of polysorbate-based ointment can be applied under a bulky absorbent gauze dressing. The patient is then released home, and the residual is easily washed off after 24 to 36 hours (Fig. 39-15). A number of dressing changes may be required. Once the residual tar is removed, the wound is treated like any other burn.

Shur-Clens, a nontoxic, nonionic detergent, also works well for tar burn wound cleansing, as do mineral oil; petrolatum; and Medisol (Orange-Sol, Inc, Chandler, AZ), a petroleum-citrus product. Butter-soaked gauze has been suggested as an emulsifier of tar.

Chemical Burns

Chemical burns usually occur in the workplace, and the offending substance is usually well-known. More than 25,000 chemicals currently in use are capable of burning the skin or mucous membranes. Commonly used chemical agents capable of producing skin burns are shown in Table 39-6 .

Injury is caused by a chemical reaction, rather than a thermal burn. [46] Reactions are classified as oxidizing, reducing, corrosive, desiccant, or vesicant or as protoplasmic poisoning. The injury to skin continues until the chemical agent is physically removed or exhausts its inherent destructive capacity. The degree of injury is based on chemical strength, concentration, and quantity; duration of contact; location of contact; extent of tissue penetration; and mechanism of action.

Immediate flushing with water is recommended for all chemical burns, with the exception of those caused by alkali metals. Flushing serves to cleanse the wound of unreacted surface chemical, dilute the chemical already in contact with tissue, and restore lost tissue water. Leonard and colleagues clearly demonstrated that patients receiving immediate copious water irrigation for chemical burns showed less

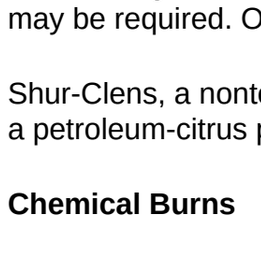

Figure 39-13 There is no compelling reason to remove all tar on the first visit. Physical removal of cooled tar usually results in avulsion of the underlying skin. Skin that is obviously loose should be debrided, but adherent tar is best liquefied with an emulsifying agent. Neomycin cream, not ointment, is a suggested emulsifier, but others are acceptable (see text). Final removal may be delayed for several days to permit loosening of the tar. Frequent dressing changes using an emulsifying agent can be performed by the patient, removing the tar over a few days.

full-thickness burn injury and a greater than or equal to 50% reduction in time of hospital stay. [47]

Acid and Alkali Burns

Alkalis cause saponification and liquefactive necrosis of body fats. Alkaline burns are *penetrating* and cause much tissue destruction. With acid burns, tissue coagulation produces a thick eschar that limits the penetration of the agent. Desiccant acids, such as sulfuric acid, create an exothermic reaction with tissue water and can cause both chemical and thermal injury. With extensive immersion injuries, acids may be systemically absorbed, leading to systemic acidosis and coagulation abnormalities.

Chemical burns may be excruciatingly painful for long periods of time. Discomfort can be out of proportion to what one might expect from the depth or extent of the burn.

The emergency care team should remove all potentially contaminated clothing. Any dry (anhydrous) chemical should be brushed off the patient's skin. The involved skin should be irrigated with large amounts of water under low pressure. Any remaining particulate matter should be carefully debrided during irrigation.

Strong alkali burns may require irrigation for 1 to 2 hours before the tissue pH returns to normal. Some recommend that *after extensive irrigation*, if the burn continues to feel "slippery" or tissue pH has not returned to normal, chemical neutralization may be helpful. [48] [49] Given that any heat of neutralization will be carried away with the irrigation solution, [50] prompt irrigation with a dilute acid (e.g., vinegar; 2% acetic acid) may hasten neutralization and patient comfort.

Wet Cement Burns

The major constituent of Portland cement, an alkaline substance, is calcium oxide (64%), combined with oxides of

Figure 39-14 Tar stuck to the face *(A)* can be emulsified with various agents and a lot of patience and persistence *(B)*. Fortunately, tar burns are usually not full-thickness burns.

silicon, aluminum, magnesium, sulfur, iron, and potassium. There is considerable variability in the calcium oxide content of different grades of cement, with concrete having less and fine-textured masonry cement having more. [47] The addition of water exothermically converts the calcium oxide to calcium hydroxide, a strongly corrosive alkali with a pH of 11 to 13. As the cement hardens, the calcium hydroxide reacts with ambient carbon dioxide and becomes inactive.

Both the heat and the $Ca(OH)_2$ produced in this exothermic reaction can result in significant burns. Because of its low solubility and consequent low ionic strength, a long exposure to calcium hydroxide is required to produce injury. This usually occurs when a worker spills concrete into his or her boots or kneels in it for a prolonged period. The burn wound and the resultant protein denaturation of tissues produce a thick, tenacious, ulcerated eschar. Concrete burns are insidious and progressive. What may appear initially as a patchy, superficial burn may in several days become a full-thickness injury requiring excision and skin grafting. [51] The pain of these burns is often severe and more intense than the appearance of the wound might suggest (Fig. 39-16). Interestingly, many workers are not warned of the dangers of prolonged contact with cement, and because initial contact with cement is

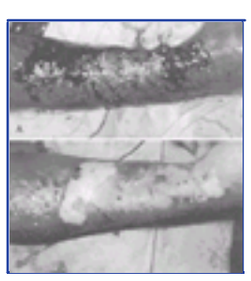

Figure 39-15 There is no need to remove all the tar on the first visit *(A)*. This extremity was covered with an emulsifying agent and with gauze, and the residual tar was washed off easily 36 hours later *(B)*.

usually painless, exposure may not be realized until the damage is done.

Treatment is as follows: Any loose particulate cement or lime is brushed off, contaminated clothing is removed, the wound is copiously irrigated with tap water (the pH of the effluent is tested and irrigation continued if the effluent is still alkaline). Compresses of dilute acetic acid (vinegar) may be applied to neutralize the remaining alkali and provide pain relief after irrigation, and antibiotic ointment is applied to the eschar during the early postburn period.

Sutilains ointment (Travase, Flint Pharmaceuticals, Deerfield, IL) is often recommended because it contains proteolytic enzymes and helps speed eschar separation, but any common topical burn preparation is acceptable. The depth of burns from wet cement can be difficult to assess in

TABLE 39-6 -- Commonly Used Acids and Alkalis

Acids	Alkalis
Picric	Sodium hydroxide
Tungstic	Ammonium hydroxide
Sulfosalicylic	Lithium hydroxide
Tannic	Barium hydroxide
Trichloroacetic	Calcium hydroxide
Cresylic	Sodium hypochlorite
Acetic	
Formic	
Sulfuric	
Hydrochloric	
Hydrofluoric	
Chromic	

the first several days. If it becomes apparent that the burns are full-thickness burns, early excision and skin grafting are recommended.

Cement burns should be differentiated from cement dermatitis, which is far more common. The latter is a contact sensitivity reaction, probably due to the chromates present in cement. The contact dermatitis can initially be treated as a superficial partial-thickness burn.

Air Bag Keratitis/Thermal Burns

Safety legislation has mandated increased use of air bags to protect automobile occupants in the event of collision. Burns from air bags can be thermal, friction, or chemical. The automobile air bag is a rubberized nylon bag that inflates on spark ignition of sodium azide, yielding nitrogen gas, ash, and a small amount of sodium hydroxide. Within seconds the superheated air is vented, and this can produce a thermal burn if it contacts an extremity, face, or upper torso. [52] [53] If the air bag ruptures, the alkaline contents of the bag are dispersed as a fine, black powder that usually causes no problems unless the eyes are exposed. Patients present with clinical evidence of a chemical keratoconjunctivitis, including photophobia, tearing, redness, and decreased visual acuity. The tear pH is usually elevated, and there may be a small amount of particulate material in the fornices. [54]

The severity of an ocular alkaline burn is related to the duration of exposure and the concentration and pH of the chemical. For this reason, prompt, copious irrigation of the eyes with frequent assessment of tear pH is essential to prevent or minimize the injury (see Chapter 64). A rising pH suggests that trapped particulate matter is releasing additional chemical. Corneal edema and conjunctival blanching are signs of serious injury and necessitate immediate ophthalmologic consultation.

Hydrocarbon Burns

Hydrocarbons are capable of causing severe contact injuries by virtue of their irritant, fat-dissolving, and dehydrating properties. Cutaneous absorption may cause even more dangerous systemic effects. Gasoline, the usual agent involved, is a complex mixture of C_4 to C_{11} alkane hydrocarbons and benzene; the hydrocarbons appear to be the major toxic agent. Lead poisoning caused either by absorption through intact skin or burns from "leaded" gasoline exposure have been previously reported but are currently quite rare, as unleaded gasoline has virtually replaced the leaded version for most purposes. [55]

Depth of injury is related to the duration of exposure and concentration of the chemical agent. Gasoline immersion injuries resemble scald burns and are usually partial thickness.[56] Occasionally, gasoline-injured skin exhibits a pinkish brown discoloration, possibly related to dye additives. A common source of exposure is a comatose patient from a motor vehicle crash who had been lying in a pool of gasoline.

The lungs are the usual site of systemic absorption and are often the only major route of excretion. The resultant high pulmonary concentrations may lead to pulmonary hemorrhages, atelectasis, and adult respiratory distress syndrome (ARDS). Treatment of hydrocarbon burns includes the following: removal of contaminated clothing, prolonged irrigation or soaking of the contaminated skin, early debridement in significant burns caused by lead-containing gasolines (to reduce systemic lead absorption), and use of topical antibiotic ointments.

Figure 39-16 Alkali burns from wet cement develop insidiously, are extremely painful, and are frequently full-thickness injuries. They are most common on the feet when cement leaks over the top of the boots *(A)* or from kneeling in wet cement while working *(B)*. The alkali can penetrate clothing.

Phenol Injury

Phenol is a highly reactive aromatic acid alcohol that acts as a corrosive. Carbolic acid, an earlier term for phenol, was noted to have antiseptic properties and was used as such by Joseph Lister in performing the first antiseptic surgery. Hexylresorcinol, a phenol derivative, is in current use as a bactericidal agent. Phenols, in strong concentrations, cause considerable eschar formation, but skin absorption also occurs and can cause systemic effects such as central nervous system depression, hypotension, hemolysis, pulmonary edema, and death. Interestingly, phenol acts differently from other acids in that it penetrates deeper when in a dilute solution than when in a more concentrated form.[46] Therefore, irrigation with water is suboptimal for phenol burns, but because water commonly is readily available, it is frequently used for irrigation.

Full-strength polyethylene glycol (PG 300 or 400) is more effective than water alone in removing phenolic compounds and should be obtained and used after water irrigation has begun. Polyethylene glycol is nontoxic and nonirritating and may be used anywhere on the body. When immediately available, polyethylene glycol can be used to remove the surface chemical before water irrigation (and chemical dilution) is begun.

Hydrofluoric Acid Injury

Hydrofluoric acid (HFA) is one of the strongest inorganic acids known; it has been widely used since its ability to dissolve silica was discovered in the late 17th century.[57] Currently, HFA is used in masonry restoration, glass etching, and semiconductor manufacturing; for control of fermentation in breweries; and in the production of plastics and fluorocarbons. It is also used as a catalyst in petroleum alkylating units. It is available in industry as a liquid in varying concentrations up to 70%. It is also readily sold in home improvement and hardware stores. Significant concentrations of HFA are present in many home rust-removal products, and in aluminum brighteners, automobile wheel cleaners, and heavy-duty cleaners in concentrations of less than 10%. Despite its ability to cause serious burns, unregulated and poorly labeled HFA products are recklessly used on a regular basis in the home and in small businesses. The public and many clinicians are generally unaware of the potential problems with this acid (Fig. 39-17).

Although HFA is quite corrosive, the hydrogen ion plays a relatively insignificant role in the pathophysiology of the burn injury. The accompanying fluoride ion is a protoplasmic poison that causes *liquefaction necrosis and is notorious for its ability to penetrate tissues and cause delayed pain and deep tissue injury*. This acid can penetrate through fingernails and cause nailbed injury. With home products, the unwary user does not realize that the substance is caustic until the skin (usually the hands and fingers) is exposed for a few minutes to hours, at which time the burning begins and becomes progressively worse. At this point the damage is done, and the absorbed HFA cannot be washed off. With higher-strength industrial products, symptoms are almost immediate.

The initial corrosive burn is due to free hydrogen ions; secondary chemical burning is due to the tissue penetration of fluoride ions. Fluoride is capable of binding cellular calcium, resulting in cell death and liquefaction necrosis. The ionic shifts that result, particularly shifts of potassium, are believed to be responsible for the severe pain associated with HFA burns.

In high concentrations, the fluoride ions may penetrate to the bone and produce demineralization. Skin exposure to concentrated HFA involving as little as 2.5% TBSA can lead to systemic hypocalcemia and death from intractable cardiac arrhythmias; it has been calculated that exposure to 7 mL of anhydrous HFA (HFA gas) is capable of binding *all* of the free calcium in a 70-kg adult.[58] [59] If the hands are exposed, the acid characteristically penetrates the fingernails and injures the nailbed and cuticle area. As with most caustics, *the pain is generally out of proportion to the evident external physical injury*. HFA burns produce variable areas of blanching and erythema, but rarely are blisters or skin sloughing seen initially. Skin necrosis and cutaneous hemorrhage may be noted in a few days.

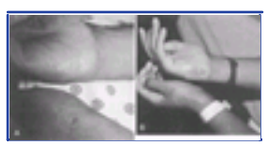

Figure 39-17 *A*, Initially this very painful hydrofluoric acid burn of the thenar and hypothenar emminence appeared minimal. *B*, Despite infiltration with calcium gluconate, a deep burn developed 3 days later.

Immediate treatment should begin with copious irrigation with water. Another approach is to wash the area with a solution of iced magnesium sulfate (Epsom salts) or a 1:500 solution of a quaternary ammonium compound such as benzalkonium chloride (Zephiran) or benzethonium chloride (Hyamine 1622). Magnesium and calcium salts form an insoluble complex with fluoride ions, preventing further tissue diffusion. While frequently recommended, topical preparations are often ineffective in limiting injury or controlling pain.

If there is no or only minimal visible evidence of skin injury and minimal pain, the burn may be dressed with topical calcium gluconate paste. This is not commercially available in the United States but is easily compounded in the pharmacy by mixing 3.5 to 7 g of pulverized calcium gluconate with 5 oz of a water-soluble lubricant such as K–Y Jelly. This will form a thick paste with a calcium gluconate concentration of 2.5% to 5.0%. Some have suggested dimethyl sulfoxide (DMSO) as a vehicle to aid in skin penetration of the calcium. Plastic wrap (e.g., Saran wrap) is used over a standard dry burn dressing to cover the calcium paste on the limbs; a vinyl or rubber glove is used over the paste when used on the hands. The wound should be completely redressed and the paste reapplied every 6 hours for the first 24 hours.

As with most topical treatments of HFA burns, calcium gluconate is only minimally effective in relieving pain, and its value is likely overestimated in the literature.

A digital or regional nerve block with long-acting bupivacaine is an excellent way to provide prolonged pain relief if the hands are involved, but this does nothing to ameliorate the injury. In most cases oral opioids are required. If bullae or vesicles have formed, these should be debrided to decrease the amount of fluoride present, and the wound should then be treated as any partial-thickness burn. Burns with HFA of less than 10% strength will heal spontaneously, usually without significant tissue loss, but pain and sensitivity of the fingertips may persist for 7 to 10 days. In addition, the fingernails may become loose.

The presence of significant skin injury or intense pain implies penetration of the skin by fluoride ions. This scenario is particularly common with exposure to HFA solutions in concentrations of greater than or equal to 20%, but tissue injury can occur with prolonged exposure to less concentrated products.

Initial treatment of a more concentrated exposure begins as described earlier and includes immediate debridement of necrotic tissue to remove as much fluoride ion as possible. Following this, a 10% solution of calcium gluconate *(note: avoid calcium chloride for tissue injections)* is injected intradermally and subcuticularly with a 30-ga needle about the exposed area, using about 0.5 mL/cm^2 of burn. Pain relief should be almost immediate if this therapy is adequate. Since the degree of pain is a measure of the effectiveness of treatment, the use of anesthetics, especially by local infiltration, may be deleted if the burn is on the arm or leg. HFA can penetrate fingernails without damaging them. Soft tissue can be injected without prior anesthesia, but if the fingertips or nailbeds are involved, they may be injected after a digital nerve block has been performed (Fig. 39-18). Before anesthesia and prior to injecting calcium, the patient can outline the affected areas with a pen to ensure accurate injection of the antidote (see Fig. 39-18*B*). Although some investigators recommend that the fingernails be removed routinely, we strongly advise *against* this unless the nails are very loose or there is obvious necrosis of the nailbed. Fingers are best injected with a 25- or 27-ga needle (a tuberculin syringe works well). [60] Nails frequently become loose in a few days, but often they return to normal and do not require removal, particularly when lower concentration nonindustrial products are involved.

Although calcium gluconate infiltration is somewhat effective, the technique has certain limitations. Injections are painful, and the calcium gluconate solution itself causes a burning sensation. Because of the volume restrictions, not enough calcium may be delivered to bind all the free fluoride ions present. For example, 0.5 mL of 10% calcium gluconate contains 4.2 mg (0.235 mEq) of elemental calcium, which will neutralize only 0.025 mL of 20% HFA.

Several authorities have advocated intra-arterial calcium infusions in the treatment of serious HFA burns of the extremities. [58] [61] Although very effective, this technique is not recommended for burns secondary to dilute HFA (i.e., concentrations <10%), because morbidity is usually quite mild. When using this technique, 10 mL of 10% calcium gluconate are diluted in 50 mL of a 5% dextrose-and-water solution. The dilute solution is given by a slow infusion into an arterial catheter. It is unclear which artery best delivers the calcium to injured tissues. If only the radial three digits are

Figure 39-18 Hydrofluoric acid burns to the fingertips are extremely painful, despite minimal clinical findings. In this case the pain was excruciating, but the patient manifested only hyperemia and minor ecchymosis. *A,* Note that the acid has penetrated the intact fingernail, producing a significant injury to the nailbed. *B,* Prior to performing digital block anesthesia in order to painlessly infiltrate the fingertips with calcium gluconate, the patient outlines the painful areas with a felt-tip marker to ensure accurate placement of the antidote. *C,* In the treatment of hydrofluoric acid burns, topical therapy is often ineffective. Calcium gluconate may be injected subcutaneously with a 25- to 27-ga needle into the nailbed via the fat pad under a digital nerve block. *Fingernails should not be removed routinely* if burns are mild, such as those seen with household products containing <10% concentration of the acid. Intra-arterial calcium infusions are often quite successful in relieving pain and limiting necrosis.

involved, probably only the radial artery need be cannulated. Otherwise, a percutaneous catheter is inserted into the brachial artery. However, some investigators have advocated the use of the radial artery in all cases, and since the arterial supply of the hand is interconnected, this may be a reasonable recommendation. [62] The radial artery is usually more easily cannulated than is the brachial artery. When the arterial access has been accomplished, *the solution is slowly infused over 4 hours*. At this point, the catheter is left in place, and the patient is observed. If pain returns at any time over the next 4 hours, the infusion is repeated. If the patient is pain free over the 4-hour observation period, the burn is dressed, and the patient is released home. This technique may be initiated in the ED but many clinicians are reluctant to cannulate an artery and infuse calcium in the ED. Such patients require hospitalization, or burn center referral, for further evaluation and observation.

Advantages of the intra-arterial method are elimination of the need for painful SQ injections and avoidance of the volume limitations of the SQ route while providing substantially more calcium to neutralize the fluoride. Both the chloride and gluconate salts of calcium have been used intra-arterially. A volume of 10 mL of 10% calcium gluconate contains 4.7 mEq of calcium, whereas the same volume of 10% calcium chloride contains 14 mEq of calcium. Thus, intra-arterial calcium chloride may provide nearly three times as much neutralizing calcium equivalent as the gluconate salt; however, because calcium chloride can irritate tissues, it is not recommended for SQ use. Disadvantages of intra-arterial calcium therapy include the possibility of local arterial spasm (which can be treated with vasodilators such as phentolamine or removal of the catheter), local arterial injury or thrombus, and the long duration of treatment required.

Infusing calcium into the general venous circulation is of no benefit for HFA burns. Some authors have advocated the use of regional intravenous calcium gluconate, similar to the method used with the Bier block for regional anesthesia. [63] Case reports have noted variable success, but this technique has neither been well studied nor rigorously compared to other options. This method would only be useful for upper extremity burns. To perform regional calcium therapy, an IV catheter is placed in the dorsum of the hand on the involved extremity. The arm is partially exsanguinated by elevation or wrapping with an elastic bandage, or both. A Bier block tourniquet, or a heavy duty blood pressure cuff, is applied proximal to the burn and inflated to 20 to 30 mm Hg above systolic pressure to stop blood flow to and from the arm. Slow deflation of a regular blood pressure cuff may thwart success of the procedure, and the use of a specialized tourniquet is recommended. Then 10 mL of 10% calcium gluconate, diluted with 30 to 40 mL of saline, is infused into the venous catheter, and the solution is kept in the arm by the tourniquet for 20 to 30 minutes. Some patients cannot tolerate arm ischemia for this period, limiting the effectiveness of this procedure. Theoretically the calcium diffuses out of the venous system and into the injured tissues. After 20 to 30 minutes, the cuff is deflated and normal circulation to the extremity is achieved. It may require 10 to 20 minutes post tourniquet deflation before the patient experiences pain relief. This procedure is safe but its efficacy is variable.

HFA burns to the eye are potentially devastating injuries that deserve special mention. Ophthalmologist referral is mandatory. Ocular exposure to liquid or gaseous HFA will result in severe pain, tearing, conjunctival inflammation, and corneal opacification or erosion. Complications include decreased visual acuity, globe perforation, uveitis, glaucoma, conjunctival scarring, lid deformities, and keratitis sicca. Optimal therapy for ocular HFA burns, other than initial irrigation, is unknown. Irrigation may be performed with water, isotonic saline, or magnesium chloride. [64] We advise copious saline irrigation. Topical antibiotics and cycloplegics, along with light pressure patching, are also recommended. The use of topical steroids has been advocated by some in order to lessen corneal fibroblast formation, but other attempted therapies such as subconjunctival injections of calcium gluconate and ocular irrigation with quaternary ammonium compounds have been associated with additional injury. [65]

Chromic Acid Injury

Chromium compounds are used extensively in industry, mainly in metallic electroplating. Chromic acid is commonly used in concentrated solutions containing up to 25% sulfuric acid. It causes sufficient skin damage to allow absorption of the toxic chromium ion if intensive irrigation is not undertaken immediately. Heated (60 to 80°C) chromic acid makes the problem of chromium absorption much worse.

Dichromate salts containing hexavalent chromium are the most readily absorbed and the most toxic because they can cross cell membranes. The mortality rate from these burns is very high if the burn exceeds 10% TBSA. Chromium absorption leads to diarrhea, gastrointestinal bleeding, hemolysis, hepatic and renal damage, coma, encephalopathy, seizures, and disseminated intravascular coagulation.

Treatment includes immediate excision of the burned tissues to lessen the total body dichromate burden. Wounds should be washed with a 1% sodium phosphate or sulfate solution and dressed with bandages soaked in 5% sodium thiosulfate solution. These actions reduce the hexavalent chromium ion to the less well absorbed trivalent form.[66]

Chelation therapy with ethylenediaminetetraacetic acid (EDTA) should be instituted, and IV sodium thiosulfate and ascorbic acid given. Hemodialysis, peritoneal dialysis, or exchange transfusion may be indicated.

Phosphorus Burns

White phosphorus is a translucent, waxy substance that ignites spontaneously on contact with air. For this reason, it is usually stored under water. It is used primarily in fireworks, insecticides and rodenticides, and military weapons.

Phosphorus causes both thermal burns from the flaming pieces and acid burns, which result from the oxidation of phosphorus to phosphoric acid. The burns classically emit a white vapor with a characteristic garlic odor. [67]

These burns are treated first with immersion in water, followed by debridement of any gross debris. The wound is then washed with a 1% copper sulfate solution, which reacts with the residual phosphorus to form copper phosphate; the latter appears as black granules and allows for easy debridement. Following debridement, the residual copper is removed by a thorough water rinse, and the wound is dressed and treated as any other burn.

Elemental Alkali Metal Burns

The commonly encountered alkali metals (sodium, lithium, and potassium) are highly reactive with water and with water vapor in air, producing their respective hydroxide with liberation of hydrogen gas. Therefore, water should never be used for extinguishing or debridement of the metal. A class D fire extinguisher or plain sand may be used for smothering the fire, followed by application of mineral oil or cooking oil to isolate the metal from water and allow safe debridement. The burn is then treated as an alkali burn.

Magnesium burns in a less intense fashion but otherwise acts as do other alkali metals. These burns may be particularly injurious, however, because if all of the metallic debris is not removed, the small ulcers that form will slowly enlarge until they become quite extensive.

EMERGENCY ESCHAROTOMY

Full-thickness burns result in an eschar that is inelastic and may become restrictive. During fluid resuscitation and as a direct result of transcapillary extravasation of fluid from thermal injury, intracellular and interstitial edema progresses. As the soft tissues become edematous and pressure rises under the unyielding eschar, first venous and then lymphatic, capillary, and ultimately arterial flow to the underlying and distal unburned tissue may be compromised. Full-thickness and extensive partial-thickness circumferential extremity burns are most likely to impede peripheral blood flow. Circumferential chest burns may restrict chest wall movement, impairing ventilation, and circumferential neck burns may result in tracheal obstruction. In such cases immediate escharotomy may be indicated.

On occasion, because of high-volume fluid resuscitation, noncircumferential and even unburned limbs may develop progressive tissue edema and ischemia requiring surgical decompression to prevent complications of nerve or muscle damage. Early elevation of the limb and active range-of-motion exercises every 15 minutes may minimize tissue edema, but once signs and symptoms of vascular impairment are present, the clinician must act quickly to prevent tissue hypoxia and cellular death. This pathophysiology may manifest itself within a time frame that mandates that the emergency clinician must intervene. Frequent reassessment of capillary refill, Doppler signals, pulsoximetry, and sensation to light touch are important to detect developing ischemia. The reluctance of non-burn specialists to perform an adequate escharotomy is illustrated by the report of Brown and colleagues, [68] who found that 44% of pediatric burn cases were inadequately decompressed prior to arrival at a referral burn unit.

Indications

The indications for escharotomy are based on clinical examination, compartment pressure, or both. A high index of suspicion and a low threshold for intervention are essential for a successful outcome. Skin temperature and palpation of pulses are unreliable and imprecise indicators of adequacy of circulation because of peripheral vasoconstriction and local edema. The patient with circulatory embarrassment significant enough to warrant escharotomy may complain of deep aching pain, progressive loss of sensation, or paresthesias, but these

parameters are difficult to quantitate in the severely burned, sedated, or mechanically ventilated patient. In the awake and cooperative patient, alteration in vibratory sensory testing with a 256-cps tuning fork may be a reliable indicator of increased compartment pressure. [69] However, gross motor activity and peripheral pulses may remain intact despite severe underlying muscle ischemia. In the series by Brown and colleagues, [68] peripheral pulses were present in 74% of the limbs that required decompression. Serial assessment of capillary refill, arterial flow (with the use of Doppler technique), arterial oxygen (with peripheral oximetry), [70] [71] and compartmental pressures (using the technique described in Chapter 55) are sensitive indicators to monitor. Muscle compartments with pressures in excess of 30 mm Hg should be decompressed. Measurements should be taken before and after escharotomy to ensure adequate decompression.

In the patient with absent distal arterial flow (as determined by Doppler ultrasonic flowmeter) but an otherwise adequate blood pressure, immediate escharotomy is indicated. Bardakjian and colleagues suggest that an oxygen saturation below 95% in the distal extremity as demonstrated by pulse oximetry (in the absence of systemic hypoxia) also is a reliable indicator of the need for emergency escharotomy. [71]

Technique of Escharotomy

Because full-thickness burns are insensible to pain and involve coagulation of superficial vessels, some SQ nerve function may exist and local anesthesia with 1% lidocaine with epinephrine may be necessary. Sedation of the awake patient is highly recommended. A properly executed escharotomy releases the eschar to the depth of the SQ fat only. This results in minimal bleeding, which can be controlled by local pressure or electrocautery. These incisions, although limb- or life-saving, are potential sources of infection for the burn patient and should be treated as part of the burn wound. The wounds should be loosely packed with sterile gauze impregnated with an appropriate topical antimicrobial such as silver sulfadiazene cream. Fasciotomy, which involves a deeper incision, may be needed for thermal or electrical burns.

Limbs

Under sterile conditions the lateral and medial aspects of the involved extremity are incised with a scalpel or electrocautery 1 cm proximal to the burned area, extending to 1 cm distal to the involved area of constricting burn (Fig. 39-19). The incision is carried through the full thickness of skin only and results in immediate separation of the constricting eschar to expose SQ fat. Because joints are areas of tight skin adherence and potential vascular impingement, incisions should cross these structures (Fig. 39-20). Care must be taken to avoid vital structures, such as the ulnar nerve at the elbow, the radial nerve at the wrist, the superficial peroneal nerve near the fibular head, and the posterior tibial artery at the ankle. The incision should extend to the great toe medially and the little toe laterally in circumferential burns of the feet, and to the thenar and hypothenar aspects of the hands (see Fig. 39-19 and Fig. 39-20). Improvement in color, sensation, Doppler flow signal strength, and oximetry values indicate adequate release.

Chest

Full-thickness circumferential chest or upper abdominal burns may impair respiration. For release of this eschar, the incision should extend from the clavicle to the costal margin

Figure 39-19 The circulatory embarrassment caused by edema beneath the encircling full-thickness burns of the legs of this patient was relieved by escharotomy incisions placed in the midmedial line of each limb. The restriction of the ventilatory excursion of the chest wall caused by the encircling full-thickness truncal burns was relieved by escharotomies placed in both anterior axillary lines and by a costal margin escharotomy. Compression of the abdominal contents and restriction of diaphragmatic excursion by the constricting deep abdominal wall burns were relieved by placement of escharotomy incisions in the lateral abdominal wall bilaterally. *(From Davis JH, Drucker WR, Foster RS, et al: Clinical Surgery. St. Louis, CV Mosby, 1987.)*

in the anterior axillary line bilaterally, avoiding breast tissue in females, and may be joined by transverse incisions, resulting in a chevron-shaped escharotomy.

Neck

Neck escharotomy should be performed laterally and posteriorly to avoid the carotid and jugular vessels.

Penis

Penile escharotomy is performed midlaterally to avoid the dorsal vein.

Complications

Complications of escharotomy include bleeding, infection, and damage to underlying structures. Complications of inadequate decompression include muscle necrosis, nerve injury (such as

Figure 39-20 Diagram showing preferred sites for escharotomy incisions. *Dotted lines* indicate the escharotomy sites. *Bold lines* indicate areas where caution is required since vascular structures and nerves may be damaged by escharotomy incisions. *(From Davis JH, Drucker WR, Foster RS, et al: Clinical Surgery. St. Louis, CV Mosby, 1987.)*

foot drop), and even amputation of the limb. Systemic complications of inadequate decompression include myoglobinuria and renal failure, hyperkalemia, and metabolic acidosis.

CONCLUSION

Patients with circumferential or nearly circumferential burns should be evaluated for deep tissue ischemia. Emergency clinicians should not hesitate to perform an escharotomy prior to transfer of the patient to the burn center if there is evidence of reduced perfusion.

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Section VII - Gastrointestinal Procedures

Chapter 40 - Esophageal Foreign Bodies

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Patients with foreign bodies (FBs) lodged in the esophagus commonly present to the emergency department (ED) for evaluation and treatment. Objects may be accidentally or purposefully swallowed. Patients may present with minor annoying irritation, life-threatening airway obstruction, or other significant complications. The emergency clinician faced with such situations needs a clear understanding of the presentation, evaluation, and treatment of these patients.

GENERAL FEATURES

Anatomy

The esophagus is a muscular tube, from 20 to 25 cm in length. There are three anatomic areas of narrowing in which FBs are most commonly entrapped (Fig. 40-1): the upper esophageal sphincter, which consists of the cricopharyngeus muscle; the crossover of the aortic arch in the mid-esophagus; and the lower esophageal sphincter (LES). The LES is the narrowest point of the esophagus and of the entire gastrointestinal tract.

Epidemiology

Patients with retained esophageal FBs generally fall into one of the following categories: pediatric patients, psychiatric patients, prisoners, and adults who are either edentulous or have underlying esophageal pathology.

Children account for 75 to 85% of esophageal FBs seen in the ED, with the peak incidence at ages 18 to 48 months. [1] [2] [3] [4] [5] [6] [7] [8] [9] The

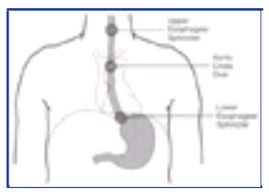

Figure 40-1 Blunt esophageal foreign bodies are most commonly lodged at one of three anatomical areas of narrowing: the cricopharyngeus muscle; the level of the aortic crossover; and the lower esophageal sphincter.

incidence is equal in boys and girls. Children frequently place objects in their mouths, and unintentional swallowing is common. As a result, children most commonly ingest coins, buttons, marbles, beads, screws, or pins. [1] [2] [4] [9] [10] [11]

Prisoners and psychiatric patients ingest a wide variety of objects, some of which may be quite unusual: spoons, razor blades, pins, nails, or practically any other object.[10] [12]

Adults who have dentures or underlying esophageal anatomic or motor abnormalities may accidentally ingest food boluses, chicken or fish bones, glass, toothpicks, fruit pits, or pills while in the act of eating. [11] Patients with an anatomic abnormality of the esophagus or a motor disturbance are more prone to entrapment of foreign objects.[10] [12] Anatomic abnormalities include strictures, webs, rings, diverticuli, and malignancies. Motor disturbances include achalasia, scleroderma, and esophageal spasm.

Complications

Impacted FBs of the esophagus must be removed or dislodged.

The time frame under which this mandate must be carried out varies widely, and depends on many circumstances. In general, however, the esophagus does not tolerate FBs well, or for prolonged periods of time, being sensitive to pressure, edema, necrosis, infection, and eventually perforation. FBs can transit the esophagus in a matter of seconds or minutes, but retained objects may become less symptomatic after time. However, the clinician must resist the urge to allow foreign objects to "pass by themselves" or "dissolve"—once they become stuck in the mucosa, they rarely do.

A wide array of complications can arise from retained esophageal FBs (Table 40-1). These include benign mucosal abrasions, lacerations, vocal cord paralysis, [13] esophageal stricture,[14] and necrosis from corrosive agents such as button batteries. Esophageal perforation [15] [16] [17] [18] [19] [20] can lead to life-threatening conditions such as retropharyngeal abscess,[21] mediastinitis, pericarditis, pericardial tamponade,[22] pneumothorax, pneumomediastinum, tracheoesophageal fistula, and vascular injuries including the subclavian vein and the aorta. [10] [23] [24] Complications are more common when FBs are entrapped for >24 hours[2] [25] and when they are sharp.[26] An estimated 1500 deaths occur annually from esophageal FBs, primarily from complications of esophageal perforation. [9]

Presentation

An esophageal foreign body impaction usually presents acutely, particularly in adults who will have a clear history of ingestion. Children commonly remember an ingestion also, but some will have a vague presentation or history. As many as one-third of children with proven esophageal FBs are asymptomatic on presentation [24] [27] [28] [29] ; therefore, a high index of suspicion is indicated, especially in a child who was seen with an object in his or her mouth that disappeared. This is especially true if there was transient coughing or gagging, even though actual ingestion was not witnessed. Poor feeding, irritability, fever, stridor, cough, and aspiration can all be caused by an underlying esophageal foreign body in a child, usually young infants. [12] [30] [31]

Dysphagia is a common presenting complaint in esophageal FBs. Drooling is suggestive of a high-grade obstruction, and the complete inability to handle oral secretions is a sign of complete obstruction.

The esophagus is well-innervated proximally, and patients typically can accurately localize foreign objects in

TABLE 40-1 -- Complications of Esophageal Foreign Bodies

Airway compromise due to tracheal compression
Aspiration pneumonia
Esophageal necrosis
Esophageal perforation
Esophageal stricture
Failure to thrive
Mediastinitis
Mucosal abrasion
Paraesophageal abscess
Pericarditis/pericardial tamponade
Pneumothorax

Pneumomediastinum
Retropharyngeal abscess
Tracheoesophageal fistula
Vascular injury including aortic perforation
Vocal cord paralysis

the oropharynx or upper one-third of the esophagus. However, scratches or abrasions of the esophagus can create a foreign body sensation. Upper esophageal foreign objects often cause gagging or vomiting. In rare cases, an upper esophageal foreign body can impinge upon the trachea, especially in children, creating stridor or frank respiratory distress. The lower two-thirds of the esophagus is not as well-innervated, and FBs in this location typically cause vague symptoms of discomfort, fullness, or non-localizing pain.

The location of retained esophageal FBs is age-related (Table 40-2). Children more typically have entrapped objects in the upper esophagus at the level of the cricopharyngeus muscle, while adults more commonly have entrapments at the lower esophageal sphincter. [31] [32] [33] [34] [35] With regard to esophageal coins in children, one report noted the frequency of impacted coins at various levels of the esophagus as: proximal esophagus 64%, middle third 8%, and distal third 26%. [35]

Evaluation

The most useful aspect of the evaluation is the history. The time of the ingestion, size and shape of the ingested object, and any current symptoms should be ascertained. The physical examination is frequently normal in patients with esophageal FBs, unless they present with a complete obstruction. In this case they will be drooling and unable to handle oral secretions. Even though asymptomatic on presentation, transient coughing or gagging should raise the index of suspicion for an esophageal FB. An examination of the oropharynx, neck, respiratory system, cardiac system, and abdomen are essential in the evaluation of possible complications.

After attending to life-threatening conditions such as airway compromise, the goal of the ED evaluation is to localize the foreign body to determine what, if any, interventions need to be undertaken to remove it or assist its transit into the stomach. Once a foreign body passes into the stomach, it

TABLE 40-2 -- Level of Entrapment of Esophageal Foreign Bodies

Level	Pediatric	Adult
Cricopharyngeus muscle	74%	24%
Aortic crossover	14%	8%
Lower esophageal sphincter	12%	68%

has a greater than 90% likelihood of passing through the entire gastrointestinal tract without any further problems. [26] Even large, irregular, and seemingly dangerous FBs will often target the entire gastrointestinal tract with relative ease.

RADIOLOGY OF ESOPHAGEAL FOREIGN BODIES

Background

Radiographic imaging of a patient with a suspected esophageal foreign body is common, and is particularly useful for the detection of radiopaque foreign objects. Traditionally, the inability to quickly identify the object by physical examination encouraged the use of plain radiography in attempts to verify and localize the retained foreign body. Plain radiography limitations require that other diagnostic approaches be considered as well.

Indications

Interactive, verbal patients can provide valuable information about the ingested culprit and can typically localize the retained body with reliable accuracy. [36] In these cases, the diagnostic workup should be tailored to the localization of symptoms and the ingested material. However, non-verbal patients including preschool children and those who are senile or debilitated warrant a low-threshold for screening radiography in cases with a suspicious history. Examples include a child seen with an object in the mouth that "disappeared" or a patient with symptomatology suggestive of an esophageal foreign body such as drooling, gagging, or unexplained respiratory symptoms.

Plain Radiographs

Plain radiographs reliably verify and localize radiopaque FBs such as glass and metal and are indicated as the main method of radiologic evaluation for these objects. As previously mentioned, FBs are most frequently entrapped at one of three locations in the esophagus: the cricopharyngeus muscle (Fig. 40-2), the aortic cross-over (Fig. 40-3), and the lower esophageal sphincter (Fig. 40-4).

Unfortunately, many ingested FBs are non-opaque including non-bony foods, plastic, wood, and aluminum such as the pull-tab from beverage cans. A magnetic metal detector has been reported to help localize radiolucent aluminum pull-tabs. [37] [38] Calcification of fish and chicken bones is often incomplete, making them radiolucent on plain films. The degree of bony calcification varies with fish species and varies between different samples of the same species, thus preventing useful guidelines.[39] [40] [41] [42] For these reasons, plain films provide little substantive evidence in the majority of cases of fish or chicken bone dysphagia. They detect only 25 to 55% of endoscopically proven bones and carry a high rate of false-negative and false-positive interpretations. [36] [42] [43] [44] [45] [46] Because of the lack of diagnostic value for detecting bones, many clinicians do not routinely order plain radiographs, opting for a computed tomography (CT) scan if radiographic evaluation is required.

When considered, a complete oropharyngeal radiograph series includes the nasopharynx to the lower cervical vertebra in both lateral and anteroposterior views. Optimum quality radiographs are mandatory. Patients should be positioned

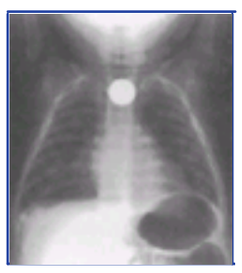

Figure 40-2 PA radiograph of an esophageal foreign body (coin) lodged at the level of the cricopharyngeus muscle. This is the most common area of the esophagus to harbor a coin in children.

upright with the neck extended and the shoulders held low. Soft tissue technique enhances the discrimination of weak radiopaque FBs. Phonation of "eeeee" during radiography prevents motion artifact from swallowing, distends the hypopharynx, and enhances soft tissue landmarks.

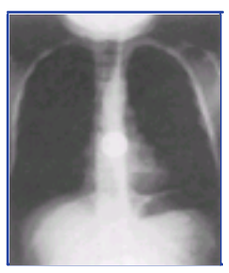

Figure 40-3 Posteroanterior radiograph of an esophageal foreign body (coin) lodged at the level of the aortic cross-over.

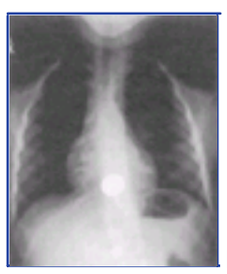

Figure 40-4 A posteroanterior radiograph of an esophageal foreign body (coin) lodged at the level of the lower esophageal sphincter (LES).

Plain radiography of the neck is limited by the radiographic properties of ingested materials and the complicated anatomy of the upper aerodigestive tract. The tongue base, palatine and lingual tonsils, vallecula, and pyriform recesses are common regions of entrapment for small, sharp objects and deserve careful interpretive attention (Fig. 40-5). Superimposition of the mandible contributes to suboptimal resolution of this region on lateral neck films. [47] Calcified

Figure 40-5 A lateral neck radiograph showing a chicken bone lodged in the pharynx with associated soft tissue swelling. The arrow points to the bone. Plain radiographs have poor diagnostic accuracy for detecting bones in the esophagus and they are often eschewed in favor of a CT scan if radiographic evaluation is deemed necessary.

airway cartilages often masquerade as FBs and contribute to false-positive rates as high as 25%. [36] [41] [43] [45] [48] [49] [50] Normal ossification of airway cartilages begins in

the third decade and progresses with age.[51] The typical curvilinear contour and well-defined margins of bony FB fragments may help distinguish them from normal laryngeal calcifications. Orientation of bony FBs is variable. The C6 vertebra approximates the level of the cricopharyngeus, a common site of FB impaction. Increased prevertebral soft tissue width, air within the cervical esophagus, and soft tissue emphysema are rare indirect findings that may help identify radiolucent objects.[42] [52]

Posteroanterior and lateral views of the chest evaluate the remainder of the esophagus. Both projections are indicated to identify multiple objects and those FBs visible in only one plane. Esophageal FBs typically lie in the vertical plane and are differentiated from airway bodies or calcifications by their location posterior to the tracheal air column on lateral radiographs. As a rule, flat objects such as coins perch in the coronal plane in the esophagus and in the sagittal orientation in the trachea. Intra-esophageal air and air-fluid levels represent indirect evidence of esophageal obstruction and may aid in verification of radiopaque FBs. Soft tissue swelling, extraluminal air, and aspiration pnuemonitis can occasionally help identify complicated impactions radiographically. Xeroradiography provides no additive benefit over plain films.[53]

In children, a "mouth-to-anus" film is frequently obtained, allowing visualization of the entire esophagus as well as the abdomen in case the foreign body has passed into the stomach or beyond. Swallowed coins or other FBs have become lodged in the nasopharynx, usually after gagging or vomiting, and could be missed if this area is not included on the radiograph. In adults, if neck or chest films are negative, abdominal films are sometimes obtained for reassurance of the presence of the foreign body in the stomach.

Contrast Esophagrams

Background

The contrast esophagram is another somewhat controversial method to evaluate for esophageal foreign body but may be considered when plain radiographs are negative. In clinical practice, however, they are seldom used since CT scanning has become widely available. This technique uses swallowed contrast to help identify the presence and location of an impacted radiolucent foreign body, the degree of obstruction, underlying anatomical abnormalities, and the presence of perforation. A variation of this technique is to have the patient swallow contrast-soaked cotton pledgets. This technique uses smaller contrast loads and may identify impacted FBs by impeding progression of the cotton or by tagging sharp irregular objects with radiopaque cotton as the bolus passes. Theoretically this variation might interfere less with follow-up endoscopy due to attenuated contrast loads. Unfortunately, liquid contrast ingestion yields overall results no better than plain film radiography. Two series document 19% to 26% false-positive and 40% to 55% false-negative interpretations using this technique, and the authors legitimately question the utility of this test. [43] [54] More importantly, contrast may interfere with the detection and extraction of FBs at endoscopy and may increase the risk of aspiration. [42] [55] [56] Routine, serial contrast esophagrams following negative plain radiography for patients with known or suspected FBs are unnecessary for diagnostic purposes in most cases. Selective use in complicated cases is reasonable but may be substituted by CT or endoscopy in many institutions.

Procedure

Esophagrams couple voluntary ingestion of enteric contrast and plain radiography. Immediately after ingestion, erect and horizontal radiographs are performed at right-angle projections (posteroanterior [PA] and lateral or right and left anterior oblique). In addition to anatomic abnormalities, radiolucent FBs may be identified by contrast delineation or filling defects within the contrast column (Fig. 40-6).

The initial choice of contrast agent is debated and should be individualized depending on the threat of aspiration and perforation. Water-soluble Gastrografin is indicated first in most cases of suspected perforation as it causes less mediastinal inflammation when extravasated; however, it can cause a severe chemical pneumonitis if aspirated and is relatively contraindicated in patients with a complete esophageal obstruction. [10] Patients without evidence of complete esophageal obstruction are instructed to swallow progressively larger aliquots of contrast agent up to approximately 50 mL. If these films are normal, the procedure is repeated with half-strength and then full-strength barium to delineate small esophageal injuries. Note that water-soluble contrast causes more pulmonary reaction than barium when inadvertently aspirated and should be used in small aliquots if aspiration or complete esophageal obstruction is a concern. Contrast esophagrams coupled with fluoroscopy are seldom used in acute esophageal FB impactions, although slowed progression or abnormal peristalsis may suggest a retained FB or anatomic abnormality. Barium interferes with endoscopy and should not be used when endoscopy is anticipated.

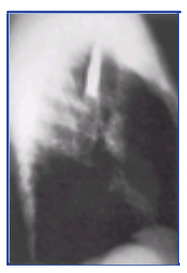

Figure 40-6 A barium swallow demonstrating a complete esophageal obstruction in the proximal to mid-esophagus.

Computed Tomography

Non-contrast CT of the neck and mediastinum is an easy, rapid, and non-invasive means of detecting (and ruling out) upper gastrointestinal FBs (Fig. 40-7).[41] [42] [44] First reported in 1983, CT has garnered increasing support in the clinical setting of suspected FB entrapment. [57] [58] [59] One series of 45 patients revealed 100% sensitivity with one false-positive CT interpretation yielding 93% specificity and 96% positive predictive value, [44] while another yielded equally impressive results in patient and cadaver models, respectively. [41] [42] CT further excels at localization and characterization of the impacted FB and identification of associated complications such as perforation.[42] [57] [60] [61] [62]

The exact role of CT for symptomatic stable patients with cervical aero-digestive FB sensation remains to be delineated. It clearly provides improved diagnostic utility for fish bone FB compared to plain radiography with and without barium. [41] [42] [44] Application to every patient with acute bone dysphagia is probably unwarranted. Use in cases with high clinical suspicion of a retained FB has the potential to reduce the number of unnecessary endoscopies. [44] Cost analysis data for this practice have not yet been published. Optimum non-contrast CT scans of the neck and mediastinum use axial 2- to 2.5-mm cuts with bone and soft-tissue windows.

Conclusions

Diagnostic radiography for esophageal FB requires case individualization. Plain radiographs clearly assist the clinician in two situations: (1) screening of children, senile adults, and nonverbal patients with a history or symptoms suspicious for purposeful or inadvertent FB ingestion, and (2) localization of known radiopaque ingestants to clarify the necessity and means of FB extraction. Conversely, attempts to verify radiolucent bodies, including bones, are often misleading. Contrast esophagrams may be used in special situations but

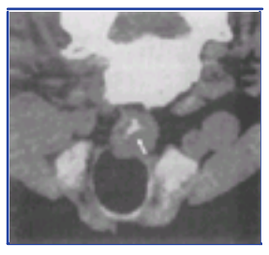

Figure 40-7 Computed tomography demonstrating an esophageal foreign body.

have largely been replaced by CT and direct endoscopy. The utility of CT to exclude fish bone FB is promising but requires discriminatory application to achieve cost efficiency.

VISUALIZATION OF ESOPHAGEAL/PHARYNGEAL FOREIGN BODIES

Direct Pharyngoscopy

Background

Direct visualization of the oropharynx is simply a physical examination using a light source and aided by a tongue blade. This examination is limited to visualizing only the upper oropharynx, tonsils, tonsillar pillars, and in some cases, the tip of the epiglottis. In many cases, a foreign body such as a fish bone can be visualized and then removed with forceps.

Indications

Direct visualization is indicated in most patients who have a foreign body sensation in the oropharynx or upper neck. The only contraindication is a patient with potential airway obstruction.

Procedure

The supplies needed for a direct visualization are a light source and a tongue depressor. Although penlights are traditionally used for this purpose, a fiberoptic headlight or a head mirror reflecting a bright light source are superior for a thorough examination. When using a headlamp, the lamp should be positioned on the forehead between the eyes, with the beam focused at a distance of approximately 2 feet. This can be tested by focusing the beam on the examiner's thumbs held together at a comfortable working distance. A head mirror is positioned with the central hole over the examiner's dominant eye. Normally the light source is positioned behind one shoulder of the patient and reflected by the head mirror into the oropharynx.

For the best direct visualization, the patient is placed in a sitting position, with the examiner standing. The entire visible oropharynx is examined using a tongue depressor to carefully depress the base of the tongue for better visualization of the pharyngeal wall, tonsils, and tonsillar pillars. Having the patient apply gentle but firm traction to their own tongue can aid exposure. The patient should generally assume a "sniffing" position with the neck slightly flexed and the head extended. Using a preprocedural topical anesthetic on the pharyngeal mucosa will minimize discomfort and gagging when using the tongue blade. In some patients, especially children, the tip of the epiglottis can be seen. If a foreign body is visible, forceps can be used to grasp and remove the object.

Indirect Laryngoscopy

Background

Indirect laryngoscopy is an examination of the middle and lower oropharynx using a mirror. This technique allows evaluation of the epiglottis, vallecula, arytenoids, arytenoid folds, and the vocal cords. Indirect visualization requires experience with the procedure and a cooperative patient.

Indications

Indirect visualization is indicated in patients who have a FB sensation in the oropharynx or upper neck, with the exception of those with airway compromise.

Procedure

Indirect visualization requires a light source (head lamp or head mirror), a gauze pad to hold the patient's tongue, a laryngeal mirror, and frequently, a topical anesthetic agent. Place the patient in a sitting position leaning slightly forward as discussed for direct pharyngoscopy. Standing in front of the patient, grasp the tongue with the gauze pad and pull lightly while having the patient open his or her mouth widely. Warm the laryngeal mirror either in warm water or a mirror warmer, check the temperature of the mirror with the back of the hand, and insert the mirror without touching the tongue (see Chapter 65 , Fig. 65-2). The mirror can elevate the uvula and soft palate slightly, but should avoid the pharyngeal wall. If the patient cannot tolerate this maneuver without gagging, instill some topical anesthetic and reattempt. Evaluate the base of the tongue, the epiglottis, vallecula, arytenoids, arytenoid folds, and the vocal cords. Use forceps to attempt to remove a visible FB. Failure to extract a visible FB with forceps is an indication for otorhinolaryngology referral for removal.

Nasopharyngoscopy

Background

Careful visualization of the oropharynx and hypopharynx is vital in the evaluation of patients presenting with complaints of FB sensation of the neck. Although a significant proportion of complete evaluations will not discover any abnormalities, the combination of direct, indirect, and flexible endoscopic nasopharyngoscopy is simple, fast, and curative in those with an identifiable FB. [36] [47] [63] One series of 168 oral, pharyngeal, and esophageal retained fish bones demonstrated the efficacy of thorough examination. Clinicians removed 73% of the bones via direct or indirect laryngoscopy. [47] Flexible nasopharyngoscopy with topical analgesia was successful in an additional 15% of cases, thereby reducing by half the need for rigid esophagoscopy and general anesthesia in this series of patients.

Indications and Contraindications

Indications for this procedure span the scope of clinical situations necessitating thorough visual examination of the pharynx and proximal esophagus, including patients with foreign body sensation and acute pill and postprandial food dysphagia.

Croup is quoted as the sole absolute contraindication to flexible nasopharyngoscopy. [64] The risk-to-benefit ratio of endoscopy should be considered in patients with coagulopathy or severe bleeding diathesis, although the risk of initiating significant hemorrhage is low. Medication allergy precludes the use of some topical agents.

Equipment

All equipment should be assembled and checked before initiation of endoscopy (Table 40-3). Nasopharyngoscopy is best accomplished via a 3- to 6-mm external diameter flexible fiberoptic nasopharyngoscope (see Chapter 65 , Fig. 65-4). Scope side ports enable suction, anesthetic injection, and FB extraction via forceps, but are not mandatory. Traditional scopes require an external light source while some newer generation fiberoptic scopes include battery-powered, self-contained light sources. Inexpensive adapters enable a laryngoscope handle to double as an endoscopy light source. The endoscope eyepiece should be adjusted to accommodate the operator's visual acuity. Yankauer suction and angled McGill forceps or a Kelly clamp should be available to retrieve

TABLE 40-3 -- Equipment for Nasopharyngoscopy

Flexible fiberoptic nasopharyngoscope
External light source (necessary for scopes without self-contained light source)

Suction and foreign body forceps (for applicable scope with channels)
McGill forceps or Kelly clamp
Topical anesthetic and decongestant
Water-soluble surgical lubricant

identified FBs orally. Topical cocaine, phenylephrine (Neo-synephrine), oxymetazoline (Afrin) and liquid or viscous lidocaine may be used for anesthesia and vasoconstriction, but are not a necessity.

Procedure

Nasopharyngoscopy is generally well-tolerated, but patients should have a clear understanding of the examination procedure prior to initiation. Discussion of the procedure with the patient should include potential complications and alternatives.

Topical anesthesia is recommended for patient comfort but is not mandatory. [36] [47] Examine the nares and choose the one with the least anatomical resistance for endoscopy. Cotton balls or pledgets soaked with 10% cocaine or a combination of 0.05% oxymetazoline (Afrin) and 2% lidocaine (Xylocaine) provide nasal mucosal anesthesia and vasoconstriction within minutes. Viscous 2% to 4% lidocaine applied by a cotton-tipped applicator is an alternative anesthetic. Atomized 2% lidocaine (Xylocaine) or topical benzocaine spray (20% Hurricane spray, Cetacaine) can be used to further anesthetize the hypopharynx. Patients may complain of discomfort or globus sensation following anesthesia and may require explanation and reassurance. Patients only rarely need an anti-sialogogue, such as glycopyrrolate. Others may need mild sedation with a short-acting benzodiazepine.

The patient is seated erect in a chair with posterior headrest support to prevent movement during examination. The examiner stands upright in front of the patient, holding the body of the scope in the non-dominant hand (see Chapter 65 , Fig. 65-5). Scope maneuvers are controlled solely with the hands. The dominant hand controls the depth of scope insertion at the naris and directs the scope along the horizontal plane at the floor of the nose. This hand should rest lightly on the patient's face for comfort and to prevent abrupt changes in scope position in case of movement during examination. Flexion in the sagittal plane is achieved with the thumb lever on the scope handle. Rotation clockwise and counterclockwise is performed via simple rotation of the wrist, not the entire upper body. It is important to maintain mild tension on the two ends of the scope for wrist action to be translated to motion at the tip of the scope. Warming the tip of the scope in water and applying silicone drops helps prevent scope fogging. Manipulation against the patient's mucosa will clear the scope during endoscopy. Water-soluble lubricant aids in passing the scope but should not be applied to the distal 2 cm of the scope.

Effective examination of the oropharynx requires some practice and reference to normal anatomy. Although many patients can localize the FB, systematic examination in a caudal direction is vital. [36] Direct the patient to focus on slow, steady mouth breathing to attenuate discomfort and gagging during the procedure. Insert the scope horizontally along

the floor of the nose and pass it below the inferior turbinate. Upon reaching the soft palate, deflect the scope tip inferiorly (via directing the thumb lever toward the eyepiece) and advance it into the oropharynx. Examine the laterally located palatine tonsils and palatopharyngeal arches, along with the proximal tongue and lingual tonsil. Advance the scope caudally to allow further visualization of the tongue base, epiglottis, and the interposed spaces, known as the epiglottic vallecula. Voluntary or manual protrusion of the tongue helps reveal this anatomy. Scan posteriorly to reveal the vocal cords, aryepiglottic folds, and pyriform recesses. Patients can gently exhale against a closed mouth and nose to distend the lower pharyngeal structures and improve visualization of these areas. Avoid contact with the epiglottis and vocal cords. Explore all pharyngeal walls fully and compare the structures bilaterally, even when patients localize symptoms to one side. Signs of trauma, including bleeding, mucosal hyperemia, and edema require close investigation to differentiate retained FBs from mucosal irritation.

If a FB is visualized (Fig. 40-8), the method of extraction depends on the location and size of the retained object. Small FBs may be extracted transnasally via forceps passed through the scope side port. Large or irregular bodies may be withdrawn orally with angled forceps. [47] Alternatively, endoscopy may be used primarily for FB confirmation. Extraction can then be performed via handheld forceps under endoscopy guidance, subsequent direct laryngoscopy, or under general anesthesia. Careful attention to prevent FB dislodgment into the airway is critical with any extraction attempt.

Complications

Complications of nasopharyngoscopy are rare and typically mild. Reflex tearing, sneezing, and coughing are the most common and are self-limited. Some patients experience a residual FB sensation for several hours after manipulation. Mild bleeding, including epistaxis, is common secondary to mucosal abrasion. Extraction of an embedded FB may result in bleeding but is also generally self-limited. Vasovagal syncope has been reported in up to 3% of patients undergoing this procedure. [64] Inadvertent dislodgement of the FB farther into the esophagus or into the airway may occur with manipulation or attempted extraction.

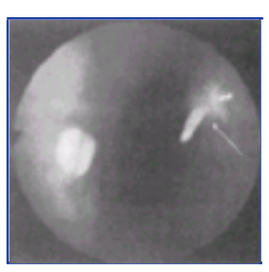

Figure 40-8 Esophageal foreign body seen on nasopharyngoscopy.

Esophagoscopy

Esophagoscopy is the definitive diagnostic and therapeutic procedure for impacted esophageal FBs. [9] [33] Although esophagoscopy is not a procedure performed by the emergency clinician, its proper role in the ED evaluation of FBs must be understood. With esophagoscopy the clinician can document the presence and location of the FB along with any underlying lesion. The clinician can then remove the object and reevaluate the esophagus after FB removal to rule out perforation or underlying pathology. Esophagoscopy may be necessary even if a radiologic contrast study does not reveal complete obstruction, because x-ray studies are not always conclusive. [8] [65] Esophagoscopy may be necessary to rule out predisposing pathology or resultant perforation, even when symptoms presumed to be due to an esophageal FB have resolved.

Esophagoscopy is the preferred method for removal of sharp or pointed objects such as bones, open safety pins, and razors. In the case of sharp objects prone to causing esophageal perforation, IV antibiotics should be administered before the procedure. Esophagoscopy is also indicated for a FB retained for more than 24 to 48 hours both to remove it and to examine for esophageal wall erosion or perforation. Esophagoscopy is the only appropriate removal technique for multiple or large esophageal FBs. This technique is also indicated in the patient with a FB proven to have passed into the stomach and who has persistent symptoms possibly caused by esophageal wall injury.

The flexible endoscope is more commonly used than the rigid esophagoscope. Flexible endoscopic procedures can usually be performed without general anesthesia, even in most children. [66] The success rate of flexible endoscopy in patients with retained esophageal FBs exceeds 96%. [33] [67]

Traditionally, esophagoscopy is more expensive than other maneuvers such as Foley catheter removal or esophageal bougienage (described later), [3] [7] [68] largely due to charges for the surgical suite, but it has a higher success rate than the other two techniques. Recently, the ED removal of esophageal FBs in children by experienced **endoscopists, while the child is under** ketamine sedation administered by the emergency clinician, has been reviewed. [69] In select cases, this approach can shorten the interval to procedural completion and expense.

ESOPHAGEAL PHARMACOLOGIC MANEUVERS

Background

Because the lower esophageal sphincter (LES) is the narrowest portion of the entire gastrointestinal tract, most FBs that reach the stomach eventually move through the gastrointestinal tract without further problems. As a large number of entrapped esophageal FBs are lodged at the LES, especially in adults, several therapeutic maneuvers have been developed to assist transit into the stomach including pharmacologic relaxation of the LES. In theory, agents that promote smooth muscle relaxation should improve mobility through the LES. Although many clinicians use pharmacologic adjuncts for all esophageal FBs, objects lodged at the LES will probably benefit most from such interventions. Nonspecific pain relief, anxiolysis, and spontaneous passage may account for the success of many pharmacologic manipulations of esophageal FBs.

Several pharmacologic agents have been shown to be unsuccessful for removing or resolving esophageal FB impactions, including diazepam, meperidine, and atropine.[11] These agents, alone or in combination, have success rates below 10%, which is no better than observation alone. [2] Glucagon, nitroglycerin, nifedipine, and gas-forming agents (Table 40-4) are described later and are the most effective pharmacologic agents for treatment of distal esophageal food impactions.

Indications and Contraindications

The indication for pharmacologic relaxation of the LES is the presence of a smooth or blunt FB such as a coin or food bolus. Angulated, abrasive, or sharp FBs should not be treated with pharmacologic modalities but instead should be removed by esophagoscopy. *Analgesics and sedatives are routinely indicated if pain is present or if the patient is excessively anxious.*

Glucagon

Pharmacology

Glucagon has been a prototype for the spasmolytic agents. [70] [71] [72] Its use has been advocated for enhancing the passage of esophageal FBs since 1977, and there have been several isolated reports of dramatic relief. [70] [73] [74] [75] Glucagon theoretically relaxes esophageal smooth muscle and decreases the LES resting pressure. One study of normal subjects found that glucagon significantly lowers the mean LES resting pressure, but causes no significant difference in the mean amplitude of contraction in the distal esophagus. [76] Glucagon has no effect on the upper third of the esophagus, a common site of coin impaction in children, where striated muscle is present and some voluntary control is operative. It only minimally affects the middle third of the esophagus, although the successful use of glucagon to dislodge food in the middle third of the esophagus has been reported. [70] [73] Peristalsis is not affected by glucagon. Glucagon was successful in approximately 30 to 69% of lower esophageal obstructions in several studies [70] [72] [73] [74] [75] ; however, in two studies glucagon was not significantly different from treatment with a placebo. [76A] [77] Its use, however, is still advocated by some authorities and has little downside. Glucagon may cause vomiting, and this action may be responsible for some of the drug's success. [78]

TABLE 40-4 -- Recommended Pharmacologic Therapies for Esophageal Foreign Bodies

Class and Agents	Site of Action	Dose and Route	Adverse Effects
Spasmolytics			
Glucagon	LES	1–2 mg IV*	Nausea, vomiting, hyperglycemia, hypersensitivity
Nitroglycerin	Body and LES	0.4–0.8 mg SL†	Hypotension, tachycardia, or bradycardia
Nifedipine	LES	10 mg SL‡	Hypotension, tachycardia (*use with caution*)
Gass-forming agents			
Tartaric acid	Distal and proximal	15 mL tartaric acid	Vomiting, increased intraesophageal pressure
		(18–20 g/100 mL)§	
		15 mL sodium bicarbonate	
Sodium bicarbonate		(10 g/100 mL)§	
Carbonated beverage	Distal and proximal	100 mL PO	Vomiting, increased intraesophageal pressure

IV, intravenously; LES, lower esophageal sphincter; PO, per os; SL, sublingually.

* May be repeated once or used in conjunction with nitroglycerin.
† 1–2 inches of nitroglycerin paste applied under an occlusive dressing may be an alternative.
‡ A capsule is punctured, chewed, held in the mouth for 3 minutes, and then swallowed. *Do not use if the patient has cardiovascular disease, is hypotensive, or has also been recently given nitroglycerin.*
§ Alternatively, dissolve 2–3 g tartaric acid and 2–3 g sodium bicarbonate in 30 mL water.

Indications and Contraindications

Glucagon is most useful for smooth FBs or food impactions at the LES that are suspected because of the patient's complaint of pain or "something stuck" in the lower chest or epigastrium. The clinical diagnosis is usually straightforward, especially if there is a complete esophageal obstruction and the patient is unable to tolerate oral secretions. Nevertheless, some clinicians recommend that the FB be localized first with radiographs (with or without contrast) to establish that the impaction is indeed there. The radiographs can then serve as the baseline study for comparison following glucagon administration. However, with a classic history and physical examination, most investigators agree that an initial contrast study can be omitted. Glucagon is not effective in upper and middle esophageal obstructions, and it is not widely recommended for use in children. Also, glucagon is usually not effective in patients with fixed fibrotic strictures or rings at the gastroesophageal (GE) junction.[71] Glucagon is contraindicated if the patient has an insulinoma, a pheochromocytoma, Zollinger-Ellison syndrome, a hypersensitivity to glucagon, or a sharp esophageal FB.

Administration of Glucagon

Some reports recommend a small test dose to check for hypersensitivity to glucagon. In practice, this is rarely done. The therapeutic dose is 0.25 to 2 mg administered intravenously over 1 to 2 minutes in the sitting patient, although one study found that in normal subjects, 1 mg provides no significant additive benefit over 0.5 mg glucagon.[76] The patient is given water orally within 1 minute after the injection of glucagon to stimulate normal esophageal peristalsis; this helps push the food through the relaxed LES into the stomach. Glucagon has a rapid onset and short duration of action. The gastrointestinal smooth muscle relaxes within 45 seconds, and the duration of action is about 25 minutes. If there are no results within 10 to 20 minutes, a second administration of 0.25 to 2 mg may be tried. Success

rates are higher when combining glucagon with gas-forming agents or even carbonated beverages. [79] [80] It is recommended that a small volume of

some oral fluid be routinely given to enhance the activity of glucagon.

Complications

Glucagon is associated with a few minor side effects. If administered too rapidly, it causes nausea and vomiting. Therefore, the adult patient must be alert and mobile enough to avoid aspiration. Occasionally vomiting dislodges the impacted food bolus. Theoretically, there is a risk of rupture of the obstructed esophagus during induced emesis, so slow injection is preferred to minimize this side effect.

The administration of glucagon is also associated with dizziness. Mild elevation of blood glucose levels is also common but is not of clinical concern and blood glucose levels do not need to be monitored. No fatalities have been reported. Although theoretically glucagon can stimulate catecholamine release with a pheochromocytoma and induce hypoglycemia from reflex insulin release with insulinoma, these endocrine tumors are rare. Nonetheless, precipitation of either profound catecholamine or insulin reactions with glucagon use should direct a workup for these underlying tumors.

Further Evaluation and Therapy

If the patient experiences symptomatic relief following glucagon administration, a post-procedure radiograph or contrast study may be obtained to confirm passage of a radiopaque object, but this is not mandatory. Adult patients with successful passage into the stomach may also be discharged home, but careful follow-up should be obtained to rule out coexistent esophageal pathology. This is because a significant number of patients (65% to 80%) will have underlying esophageal disorders. [9] If glucagon fails to produce symptomatic relief or resolve radiograph findings, its use does not preclude other methods from being used.

Nitroglycerin and Nifedipine

Pharmacology

Both sublingual nitroglycerin and nifedipine have been used in a manner similar to that of glucagon to relieve LES tone and allow the passage of a distal esophageal FB.[81] [82] [83] Although these two agents have been used less than glucagon for the treatment of esophageal FBs, both agents are useful for the relief of chest pain associated with esophageal smooth muscle spasm[57] and may be administered concurrently with glucagon. Manometric and radiographic studies after the administration of nitroglycerin reveal abolition of repetitive high-pressure wave contractions characteristic of esophageal spasm. Nifedipine, on the other hand, significantly reduces LES pressure without changing contraction amplitudes in the body of the esophagus. Thus, nitroglycerin may relieve partial or complete obstruction of the middle or lower esophagus secondary either to intrinsic esophageal disease or to simple FB impaction, and nifedipine, like glucagon, is most likely to succeed when the bolus is lodged at the GE junction.

Indications and Contraindications

Similar to the clinical indications for the use of glucagon, any patient presenting with an impacted smooth esophageal FB, especially a food bolus, may be a candidate for nitroglycerin and/or nifedipine. Also, similar to the mode of action of glucagon, neither of these agents is expected to relax a fixed fibrotic stricture or ring at the GE junction.[71] Nevertheless, because both agents have a relatively benign side effect profile, if the patient has no contraindication to their use, they may be tried with or without previous documentation of the distal esophageal obstruction by contrast study. Contraindications to their use include a history of allergic reactions, a sharp esophageal FB, hypovolemia, and hypotension.

Use and Complications

Doses of 1 to 2 (0.4 mg) sublingual nitroglycerin tablets, 1 to 2 inches of nitroglycerin paste, or 10 mg of nifedipine have been reported. [81] [82] [83] Remember that some patients with esophageal FBs may present with some degree of dehydration due to the inability to swallow liquids or their own saliva. These patients may be prone to hypotension from the vasodilation associated with the use of either agent. Ideally, rehydration should precede therapy with these agents. Sublingual niphedipine has been rarely implicated in cerebral or coronary insufficiency in patients with cardiovascular disease, so caution is warranted.

Further Evaluation and Therapy

As with the use of glucagon, if nitrate therapy fails to produce symptomatic relief or resolve radiographic findings, its use does not preclude trying another method. If a patient experiences symptomatic relief, a post-procedure radiograph may be obtained to confirm passage of a radiopaque object, but this is not mandatory. The adult patient may be discharged home, but careful follow-up should be obtained to rule out coexistent esophageal pathology, as a significant number of patients will have underlying esophageal disorders.

Gas-Forming Agents

Pharmacology

The use of gas-forming agents for the treatment of distal esophageal food impactions, especially meat boluses, was first described in 1983. [84] The combination of tartaric acid solution followed immediately by a solution of sodium bicarbonate or even carbonated beverages was reported. In theory, the use of this acid-base mixture or of a carbonated beverage (e.g., Coca-Cola) may produce sufficient carbon dioxide to distend the esophagus, relax the LES, and push impacted food through the GE junction into the stomach. [85] [86]

Indications and Contraindications

Gas-forming agents are indicated for the relief of smooth distal esophageal FB impactions, with or without prior FB confirmation by a radiographic study. They are often given to patients with food impaction or retained coins. Although gas-forming agents are more likely to succeed with distal esophageal impactions, they have also been successful in relieving obstructions in the proximal esophagus. Concurrent administration of spasmolytic agents may improve the effectiveness of gas-forming agents.[80]

Use and Complications

A solution of 15 mL of tartaric acid (18.7 g/100 mL), followed by 15 mL of a sodium bicarbonate solution (10 g/100 mL), or 1.5 to 3 g of tartaric acid and 2 to 3 g of sodium bicarbonate dissolved in 15 mL of water can be used. [84] [85] Carbonated beverages (100 mL) have also been successful in the transit of FBs into the stomach. [84] [86] Many patients with esophageal FB impactions have been noted to retch after receiving gas-forming agents, which theoretically puts

patients at risk for esophageal trauma. The only reported complication with the use of gas-forming agents for this indication was a mucosal tear of the esophagus (requiring surgical exploration to rule out mediastinitis) in a 66-year-old patient with an 18-hour-old esophageal impaction. [85] For this reason, gas-forming agents should not be given to patients with impactions of more than 6 hours duration or to patients with chest pain that might be indicative of an esophageal injury.

Further Evaluation and Therapy

As with the use of glucagon, nitroglycerin, or nifedipine, even if administration of the gas-forming agent is successful, as judged by relief of symptoms, follow-up evaluation is necessary to determine the underlying esophageal abnormality that potentially led to the FB impaction.

Papain

Papain is not recommended for treatment of an esophageal FB. It is a proteolytic enzyme that has been touted for dissolving meat impactions. [87] Papain is available commercially in a variety of meat tenderizers. This therapy has never been tested in a clinical trial. In vitro studies suggest that the commercial preparation may have no intrinsic proteolytic activity. [88] Although it is harmless when in brief contact with the normal esophagus, if it is left too long in the obstructed esophagus, papain may begin to dissolve the esophageal mucosa underlying a foreign body. [88] [89] This is likely to occur when the esophageal wall is ischemic owing to FB impaction and resultant wall pressure, when esophageal injury results from small bony spicules in the FB, or when an underlying lesion is responsible for the obstruction. The subsequent rupture and leakage of the proteolytic enzymes result in a self-perpetuating mediastinitis. Patients with esophageal FBs are at increased risk for aspiration, and pulmonary aspiration of papain results in acute hemorrhagic pulmonary edema. In general, papain is not currently recommended because of the unacceptable complication risk and the availability of safer, more effective interventions.

FOLEY CATHETER REMOVAL OF ESOPHAGEAL FOREIGN BODIES

Introduction

Foley catheter removal of esophageal FBs was first described in 1966 [90] and in the emergency medicine literature in 1981. [91] The technique is essentially unchanged since the first reports, and is now used by emergency clinicians, radiologists, otolaryngologists, and general surgeons. [92] [93] [94] [95] [96] The classic patient for this technique is a small child who is brought to the hospital shortly after swallowing a coin that is documented by radiograph, but the procedure may be used for a wide variety of smooth FBs in all ages of patients. Success rates for Foley catheter removal of FBs have been cited from 85% to 100%, with complication rates of 0 to 2%. [7] [67] [97] [98] [99] [100] Many of the reported complications were due to the nasal insertion of the catheter, and complication rates are lower when the catheter is inserted orally and at centers that perform the procedure frequently. Foley catheter extraction costs significantly less than endoscopy. [7] [101] Fluoroscopic assistance may be preferable, but it is not essential. Whether the procedure is performed in the ED or the radiology department, equipment and personnel capable of emergency pediatric airway management must be present.

Indications and Contraindications

Recently ingested smooth, blunt objects that are radiographically opaque are most suitable for balloon catheter extraction. Recently ingested FBs carry little likelihood of causing pressure necrosis, perforation, or other significant injury; however, 24 to 48 hours duration of impaction should be the upper limit for consideration of this technique. [7] [26] [102]

Coins are particularily amenable to Foley manipulation, but food boluses and button batteries have also been extracted successfully. [93] Radiographically opaque objects are most easily located by plain radiographs. Radiolucent objects can be manipulated, but uncertainties about location mandate contrast esophagrams.

Contraindications to catheter removal of esophageal FBs include total esophageal obstruction, as manifested by an air-fluid level on plain radiograph or contrast esophagram, or when patients are unable to handle oral secretions. The presence of a total obstruction prevents passage of the catheter tip distal to the FB. Esophageal perforation, as recognized by the typical symptoms and signs, requires immediate surgical consultation and precludes blind esophageal manipulation, as does airway distress. The presence of multiple esophageal FBs also precludes Foley catheter use. Sharp, irregularly shaped FBs should not be removed with this technique because esophageal perforation or laceration can result, and the balloon may burst during the procedure. Finally, lack of expertise or equipment to handle an airway problem arising during the procedure is a contraindication.

Equipment

The necessary equipment is basic and present in most EDs (Table 40-5). Although never reported, airway obstruction during the procedure is the most feared potential complication. Thus, the proper equipment and personnel capable of managing airway obstruction must also be present, including suction devices. Forceps (bayonet and Magill) of various sizes should be available to extract the FB from the pharynx. Foley catheters ranging in size from No. 8 Fr with 3-mL balloons to No. 26 Fr with 30-mL balloons have been used. In settings in which both children and adults are treated, sizes ranging from 10 Fr to 16 Fr with 5- to 10-mL balloons should suffice. Child restraint devices (e.g., papoose board), topical anesthetics, or moderate sedation may be used.

TABLE 40-5 -- Equipment for Foley Catheter Extraction of Esophageal Foreign Bodies

Standard resuscitation equipment for advanced airway management of children and adults
Foley catheters (10 to 16 Fr)
Topical anesthetics*
Parenteral sedatives*
Child restraint device*
Fluoroscope*

*Optional equipment.

Procedure

Every patient should be appropriately coached concerning the procedure. Young children should be restrained. Moderate sedation and nasopharyngeal topical anesthesia may be used; however, this may increase the risk of aspiration due to decreased airway protective reflexes. The patient is then placed in a head-down Trendelenburg, lateral decubitus, supine, or prone position. The procedure is often done under fluoroscopic guidance, although this is not mandatory if the FB has been localized on plain radiographs.

Assuming the procedure is being performed on a young child, a 12 Fr to 16 Fr Foley catheter is used. After checking for symmetric balloon inflation, the balloon is inflated and the catheter is inserted orally (Fig. 40-9). When using fluoroscopy, the catheter is visually passed distal to the FB. Intermittent inflation with 1 to 2 mL of contrast may be needed to verify the catheter tip location. If performed without fluoroscopy, the distance from nose or mouth to FB is estimated, and the catheter is inserted accordingly. On occasion, the operator feels the catheter tip passing the object. The balloon is slowly filled with 3 to 5 mL of saline or contrast agent (if fluoroscopy is used). Balloon inflation should be stopped if the patient complains of increased pain and the catheter should be repositioned before an attempt at reinflation. Fluid is preferable to air, as it is less compressible. Overdistention of the balloon is undesirable, and in children no more than 5 mL of fluid should be used.

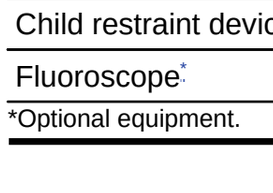

Figure 40-9 Technique of Foley catheter extraction. In children, this procedure is best done with the patient restrained on a papoose board, or consciously sedated, with the head lowered. Some operators place the patient in a prone position or roll the patient to the prone position after catheter insertion to enhance oral expulsion of the foreign body. *A,* A catheter is inserted orally into the esophagus distal to the coin. A bite block may be used to assist oral passage. *B,* The balloon is inflated. Gentle traction moves the coin proximally through the esophagus. *C,* The coin is moved steadily past the glottis. *D,* The coin is present in the mouth to be expectorated or grasped. *(Adapted from McSwain N: Esophageal foreign body. Emerg Med 21:85, 1989.)*

The catheter with its balloon inflated beyond the FB is then withdrawn with steady, gentle traction. Contact with the object can be sensed as the friction of withdrawal increases. Significant impedance to traction requires termination of the attempt. If the catheter slides past the object without dislodging it, the balloon can be deflated and repositioned. An additional 2 to 3 mL can be used to enlarge the balloon and another attempt made. Often when the object reaches the hypopharynx, the balloon and gravity act in concert to fully externalize the FB. The patient can be instructed to spit it out or the operator can grasp the object with the fingers, forceps, or a

clamp.

A follow-up radiograph may be necessary to exclude the possibility of multiple objects. If fluoroscopy is not used and no FB is retrieved, another radiograph should be obtained, because 10% to 20% of the time, the FB will pass distally into the stomach. [103]

Multiple attempts should not be required if catheter size, placement, and balloon inflation are correct. Failed attempts are best followed by a change in one of the aforementioned parameters or repeat x-ray studies to confirm the continued presence of the esophageal FB before esophagoscopy.

Complications

Complication rates of 0% to 2% have been reported. [7] [67] [97] [98] [99] [100] Many complications (nosebleeds or displacement of the foreign object into the nose) have been related to the nasal

insertion of the catheter. Complication rates are lower when the catheter is inserted orally and generally lower at centers that perform the procedure frequently. No deaths have been reported. One esophageal rupture has occurred. [100] Laryngospasm and aspiration are rare complications. Failure to either remove the object or displace it into the stomach occurs in approximately 2 to 10% of carefully selected patients, [98] [99] [100] but success rates are lower in adults or patients with underlying esophageal disorders. [100]

Disposition

Children who have a FB successfully removed by the Foley catheter need no further follow-up if they remain asymptomatic. If the FB was moved into the stomach, clinical follow-up should be adequate to verify movement of gastric FBs through the alimentary tract. Discharge instructions should include warnings about potential symptoms of gastrointestinal obstruction, perforation, and hemorrhage. Parents of children who swallow coins can be instructed to watch for coins in the stool. Adults with esophageal FBs that have been removed successfully must be referred for evaluation of possible esophageal pathology. Should a FB remain lodged in the esophagus, immediate referral for endoscopy is necessary.

ESOPHAGEAL BOUGIENAGE

Background

Displacement of esophageal FBs into the stomach can be done using naso- or orogastric tubes or esophageal bougienage. Esophageal bougienage is a technique for dislodging impacted esophageal coins by blind mechanical advancement of the coin into the stomach, a procedure first described in 1965. [104] The technique has a greater than 95% success rate with essentially no reported complications. [7] [68] [105] [106] Rates as successful as endoscopy have been reported. [102] Furthermore, bougienage is unrivaled in overall cost-effectiveness, approximating 10% of the cost of endoscopic removal. [7] [68] [102] [107]

There have traditionally been warnings against forceful advancement of esophageal FBs, but growing evidence verifies the efficacy and safety of blind esophageal bougienage as first line therapy for coin ingestions in properly selected patients. Although early articles suggested that esophageal bougienage should be performed exclusively by pediatric surgeons, the technique is easily mastered and used by emergency clinicians and junior surgical residents. [68] [106]

Indications and Contraindications

Strict patient selection is paramount for successful and uncomplicated bougienage. The criteria have changed little since initially proposed and define a group in whom a round, smooth object can be forcibly passed into the stomach with little risk. [105] [108] While many swallowed objects meet this description, only coins hold clear supportive evidence in the literature. Selection criteria are the following: a single, smooth FB, lodged <24 hours, in a patient with no respiratory distress or history of esophageal disease including prior FB or surgery. The procedure is contraindicated in patients who do not satisfy all criteria. It is important to ascertain time period of esophageal impaction to avoid the procedure when there may be underlying esophageal injury. For this reason, some advocate requirements for clearly witnessed ingestions <24 hours before presentation. [106] Plain radiographs are indicated to verify coin location and the absence of multiple esophageal bodies. Pre-procedure esophagrams are not required.

Equipment

The necessary equipment should be assembled prior to initiation of the procedure (Table 40-6). Airway equipment and drugs, topical anesthetic, suction, tongue depressor blades, bougie dilator set, water-soluble lubricant, an emesis basin, and an assistant are needed. Hurst-type esophageal dilators refer to weighted, flexible rubber bougies with a blunt, rounded tip. Nasogastric tubes and taper-tipped dilators should not be substituted. Bougie dilator size is selected based on age: 1 to 2 years, size 28 Fr; 2 to 3 years, 32 Fr; 3 to 4 years, 36 Fr; 4 to 5 years, 38 Fr; older than 5 years, 40 Fr. [106]

Procedure and Technique

Although not painful, bougienage may be frightening for patients and their parents. Both should have a clear understanding of the procedure, including the possible complications, prior to initiation. Small children may require physical restraint with bed sheets or papoose. All patients require guidance and reassurance throughout the procedure.

Topical anesthesia may be achieved with gargled 2% to 4% viscous lidocaine (Xylocaine), atomized 2% lidocaine, or topical benzocaine (20% Hurricane spray, Cetacaine). Sedation is generally not needed. Anxiolysis may be of use in some cases but must be weighed against the potential for aspiration from uncontrolled secretions or induced vomiting.

Blind esophageal bougienage is relatively straightforward and resembles placement of an orogastric tube. Patients may be positioned prone or seated upright for the procedure. The distance from nose to midepigastrium approximates the length to reach the stomach and should be noted prior to passage. The patient is asked to flex the head slightly forward and protrude the tongue. A tongue blade may be used to displace the tongue in uncooperative children. The well-lubricated, appropriately sized bougie is passed posteriorly along the roof of the mouth following the natural curve of the soft palate caudally to the hypopharynx. The patient will momentarily gag as the bougie meets resistance at the level

TABLE 40-6 -- Equipment for Bougienage Manipulation of Esophageal Foreign Bodies

Tongue depressor	
Topical anesthetic	
Emesis basin	
Yankauer suction tip and tubing connected to continuous wall suction	
Water-soluble surgical lubricant	
Hurst-type blunt-tipped bougie dilators (age-appropriate size)	
Age (years)	**Dilator Size**
1–2	28 Fr
2–3	32 Fr
3–4	36 Fr
4–5	38 Fr
>5	40 Fr

From Emslander HC, Bonadio W, Klatzo M: Ann Emerg Med 27:726, 1996.

of the cricopharyngeus muscle. Encourage the patient to swallow and gently pass the dilator through the cricopharyngeus muscle. Asking the patient to phonate helps exclude accidental laryngeal intubation; marked hoarseness or inability to phonate indicates airway obstruction and incorrect placement.

Once past the cricopharyngeus muscle, head extension enables the bougie to pass distally to the stomach with little resistance. The bougie is withdrawn after a single pass. The procedure should be terminated immediately for pain or resistance to advancement. A post-procedure radiograph of the chest and upper abdomen documents coin location and should be scrutinized for evidence of complications. Routine post-procedure esophagrams are not indicated unless a complication is clinically suspected. Barring a suspicion of complications, asymptomatic patients are released to home with appropriate precautions including the need to return for signs of respiratory compromise, chest or abdominal pain, dysphagia, hematemesis, persistent vomiting, or other concerns. Follow-up abdominal radiographs may be performed to document passage of the coin if it is not identified in the feces within one week. For adult patients, follow-up is mandated due to the 65% to 80% chance of underlying esophageal disorders. [9]

Complications

Gagging and self-limited non-bloody vomiting are not uncommon after the procedure and may reveal the dislodged coin. Patients may experience a residual FB

sensation for several hours. Pulmonary aspiration and inadvertent passage into the airway are potential complications. Likewise, traumatic pharyngeal and esophageal injury, ranging from mild self-limited bleeding to frank esophageal perforation with concomitant infection, are possible but rare.

SPECIAL SITUATIONS

Childhood Coin Ingestions

Coins are among the most commonly ingested objects in preschool-aged children. In most cases the ingestion is quickly realized by a caretaker, and in the majority of cases, the coins pass uneventfully.[109] Rarely, an esophageal coin can be clandestine for many weeks or months, producing a variety of vague respiratory or gastrointestinal symptoms. Most coins pass from the esophagus to the stomach with only transient symptoms. The child may be in pain for a few minutes as the coin migrates, but on arrival in the ED, the child is often asymptomatic. Coins that remain in the esophagus are likely to, but do not always, produce continued symptoms (e.g., drooling, pain, dysphagia, refusal to eat or drink). Rarely, esophageal coins can produce airway distress by external compression of the trachea, simulating an asthmatic attack. Coins below the diaphragm are asymptomatic and the presence of pain or symptoms requires further evaluation. Coins in the trachea produce immediate and obvious respiratory distress.

The evaluation and management of children with ingested coins is evolving.[110] The first clinical decision is whether or not to obtain a radiograph. Although some authors recommend that asymptomatic children not be radiographed,[27] it is important to remember that up to 44% of children with esophageal coins may be asymptomatic. Therefore, it is prudent to perform a film on all children with a suggestive history.[29] In most cases a single film that includes the pharynx, esophagus, and stomach will suffice to prove or exclude an ingested coin. Another advantage of obtaining radiographs is to rule out multiple FB ingestions (Fig. 40-10), which are not uncommon in children.[111] Only a single PA chest film is needed to prove the presence of a coin. If the flat surface of the coin is seen (see Fig. 40-2 , Fig. 40-3 , Fig. 40-4), this orientation assures an esophageal position. If the edge of the coin is seen, this orientation suggests that it transversed the vocal cords, but a coin in the airway is not subtle. It is advisable to routinely obtain a lateral radiograph to determine if multiple coins are stacked on top of each other (Fig. 40-10A).

Another technique to localize swallowed coins in children is to use a metal detector.[37] [112] [113] Although reported sensitivity and specificity are good for determining esophageal versus non-esophageal location, the device cannot rule out multiple ingestions, which are a contraindication to Foley catheter or bougienage removal.

Once a coin's presence has been documented, a decision concerning removal must be made. Coins in the upper and middle third of the esophagus are unlikely to pass spontaneously.[35] Coins in the distal esophagus are more likely to pass spontaneously within a few hours.[28] [35]

The decision on managing these patients depends on various factors: clinician comfort and experience with removal techniques, local protocols and procedures developed by the medical staff of each institution, and comfort level of the caretakers with various therapeutic options.

One suggested protocol (Fig. 40-11) involves radiologically localizing the coin, and if the child is *symptomatic*, immediately removing the coin. If the patient is *asymptomatic*, the coin may be removed immediately or the patient may be observed either as an inpatient or at home. If the child is asymptomatic, one common practice is to allow the child to drink a carbonated beverage and eat a small amount of soft food in the ED, wait about 1 hour, and perform another radiograph. If sent home, with an asymptomatic retained FB, the patient is allowed to eat or drink but should be rechecked in 12 to 24 hours, with the knowledge that up to 60% of asymptomatic coin FBs will pass into the stomach spontaneously.[28] [35]

The techniques of esophageal bougienage and Foley catheter removal have been described earlier. Both are options for single coins in the esophagus present for <24 to 48 hours. Another option for coins at the LES is pharmacologic relaxation of the sphincter to aid passage into the stomach. The most common method to remove esophageal coins in use today is esophagoscopy.

About half of ingested coins are in the stomach at the time of first investigation, and such patients can be safely released home to allow for almost certain spontaneous passage with a normal diet. Spontaneous passage of a coin from the stomach to the anus usually requires 5 to 7 days. There is no need for routine cathartic. Parents should be advised to check the stool for the coin and return for repeat radiographs if the coin is not found in 1 to 2 weeks. Most coins are passed unknowingly by the patient. Any abdominal discomfort or distention warrants reevaluation in the ED. If a follow-up radiograph demonstrates a persistent coin in the intestines for more than 3 to 4 weeks, an obstructive lesion may be present, and surgical referral is warranted.

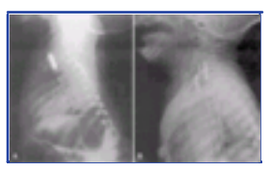

Figure 40-10 *A*, Lateral radiograph of the child shown in Figure 40-2 . This lateral view shows four coins at the same location. Multiple swallowed coins are common in children. It is important to obtain both posteroanterior and lateral films to ascertain the exact number and location of swallowed coins. *B*, A single coin was seen on the PA chest film, but this lateral film suggests three coins. However, they do not seem to be stacked directly on top of each other. This digital radiograph was accidentally exposed three times, and actually only one coin was swallowed.

Figure 40-11 Flow diagram outlining an approach to the management of swallowed coins.

Fish or Chicken Bones in the Throat

Patients who complain of a "bone" in their throat usually present to the ED within several hours of the onset of symptoms and usually have tried a home remedy, such as swallowing a piece of bread. These patients are typically able to pinpoint the location of their discomfort and present with an FB sensation. Patients who are markedly symptomatic, vomiting, or unable to swallow require definitive therapy. Those with minor complaints may be safely evaluated over a few days, often as outpatients.

In cooperative patients, a careful examination of the oropharynx, with either direct or indirect laryngoscopy or both, should be made. If the bone is seen, removal with forceps should be performed (Fig. 40-12).

If the patient feels pain in the upper throat, special attention is directed to the tonsils, since bones often lodge in this area. Strands of saliva may mimic a bone, and small bones may be difficult to see. More commonly, the area of complaint is below the oropharynx. In these patients, indirect laryngoscopy or nasopharyngoscopy should be the first step, once again removing the bone if one is seen.

Most patients presenting with an oropharyngeal FB will not have an easily identified or visualized object on examination. These patients present a diagnostic dilemma

for several reasons. Only 17% to 25% of patients complaining of a FB sensation after eating chicken or fish have an endoscopically proven bone present, and only 29% to 50% of endoscopically proved bones are seen on plain films. [57] [114] [115] The symptoms in those patients with an FB sensation but no FB on endoscopy are believed to be due to esophageal abrasions.

For these reasons, a two-tiered approach to managing these patients is proposed. [6] [9] [26] [63] The patient receives a physical examination and appropriate bone removal if one is seen. Be certain to carefully examine the tonsils, posterior pharynx, and base of the tongue, common places for bones to lodge. If no bone is seen on physical examination, plain films of the neck are obtained. Positive films are an indication for endoscopic removal of the bone. If these films reveal no FB, and the patient is stable, he or she is discharged home with follow-up within 24 hours. Patients with oropharyngeal abrasions will usually be asymptomatic at that time. If still symptomatic on follow-up, endoscopy is advocated.

This approach may well be modified at individual institutions due to increasing evidence of CT's ability to identify bones

Figure 40-12 Many fish bones become impaled in the soft tissues of the upper digestive tract. This woman felt a bone catch in her throat while eating fish. As is often the case, she was able to consistently localize the FB to the submandibular area (*A*). With only a tongue blade, local anesthetic spray, and good lighting, a fish bone was found embedded in the tonsil and was easily removed with forceps (*B*). Strands of saliva can mimic a fish bone, so be careful when probing and grasping.

in the throat, an accuracy rate exceeding 90%. [41] [44] [57] [114] A thorough physical examination including direct and indirect laryngoscopy is still indicated in these patients, as is nasopharyngoscopy in appropriately equipped facilities. These examinations may obviate the need for CT. If the initial examination is negative, reasonable alternatives include following the management approach outlined earlier or obtaining CT of the neck with appropriate referral for removal if a bone is identified.

A small bone lodged in the esophagus for a few days is annoying and painful, but it is generally not an emergency. However, impacted bones can cause serious sequelae, often weeks later, and continued complaints cannot be ignored. Importantly, *a lodged bone will not dissolve and rarely passes spontaneously once lodged in the mucosa*. Referral, and possible endoscopy, are necessary if complaints persist for more than 2 to 3 days, even if the examination and CT scan are negative.

Sharp Objects in the Esophagus

Sharp objects cause the majority of complications seen in patients with esophageal FBs. These objects include tacks, pins (Fig. 40-13), open paperclips, bobby pins, toothpicks, and razor blades. They will usually not pass spontaneously and should be removed. The only appropriate removal technique is under direct visualization with endoscopy.

Attempts at radiographic localization are appropriate for metallic or radiopaque FBs. If the object is in the stomach, most will transit the remainder of the gastrointestinal tract if less than 6 cm in length or 2 cm in diameter. If larger than this, consultation with a gastroenterologist is appropriate. If radiographs show the FB in the esophagus, endoscopic removal is indicated and attempts to remove such objects in the ED are not indicated. Complication rates for endoscopic removal of sharp FBs ranges from 0% to 3%.[8] [10] [32]

Non Radiopaque Objects in the Esophagus

Objects such as toothpicks, aluminum tabs from beverage cans, and food boluses cannot be visualized on plain radiographs, and will not normally pass spontaneously. Toothpicks cause a higher percentage of complications than any other type of esophageal FB. Localization of the FB may be accomplished by esophagram with contrast material, although the

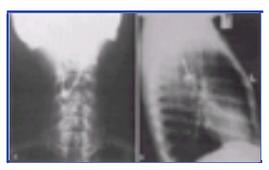

Figure 40-13 *A*, Posteroanterior radiograph of an open safety pin lodged in the upper esophagus. Sharp foreign bodies in the esophagus are best removed with endoscopic visualization. *B*, This 10-year-old child came to the ED with severe chest pain. No history of a foreign body was given. Even when the radiograph demonstrated this metallic object in the esophagus, it remained a mystery how it got there. Objects such as this are removed under anesthesia with an endoscope, and no ED intervention, except for pain relief, is indicated.

yield is low with toothpicks. As with fishbones, toothpicks often lodge in the tonsils or posterior pharynx and can be seen on direct vision.

Food boluses may be amenable to pharmacologic relaxation of the LES. The mainstay of therapy for other non radiopaque FBs in the esophagus is consultation for endoscopic evaluation and removal.

Button Battery Ingestion

Button batteries lodged in the esophagus should be considered an emergency because of the potential for serious morbidity and mortality. [15] [17] [116] These batteries range in size from 7 to 25 mm and are radiopaque (Fig. 40-14). Batteries appear as round densities, similar to an impacted coin, but some demonstrate a "double contour" configuration (Fig. 40-15). It is important to distinguish between a coin and a button battery,

Figure 40-14 Button batteries. These batteries have a wide range of sizes and can mimic coins on radiographs.

because button batteries require immediate removal. Batteries consist of two metal plates joined by a plastic seal. Internally they contain an electrolyte solution (usually concentrated sodium or potassium hydroxide) and a heavy metal, such as mercuric oxide, silver oxide, zinc, or lithium.

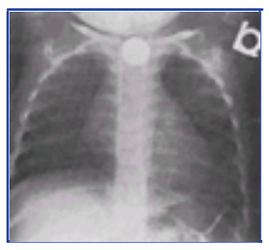

Figure 40-15 Posteroanterior radiograph of a button battery lodged in the esophagus, which can easily be confused with a coin. Button batteries can cause esophageal wall necrosis within 2 hours and must be removed expeditiously. *(From Kost KM, Shapior RS: Button battery ingestion: A case report and review of the literature. J Otolaryngol 16:4, 1987.)*

If ingested, these batteries often lodge in the esophagus. The mechanisms of injury include electrolyte leakage, injury from electrical current, heavy metal toxicity, and

pressure necrosis. Of particular concern is the development of a corrosive esophagitis or perforation as a result of caustic injury and prolonged mucosal pressure. Although essentially harmless in the stomach and intestines, batteries lodged in the esophagus should be considered an emergency situation, because even new batteries demonstrate corrosion, leakage, and mucosal necrosis within a few hours of contact with the esophagus. [16] [17]

Esophageal impaction mandates immediate removal. Options include Foley catheter removal, esophageal bougienage, or esophagoscopy. Esophagoscopy allows for direct esophageal evaluation and a more controlled extraction. In addition, the "invasive" nature of batteries may lead to rapid edema, making the catheter technique more difficult.

Once in the stomach, button batteries do not require removal. They may be followed radiographically to demonstrate passage, with little risk of gastrointestinal injury or heavy metal poisoning, even if the battery opens. [117] [118]

CONCLUSION

Patients with esophageal FBs are frequently seen in the ED. There are several important considerations in evaluating these patients (Table 40-7).

Sharp FBs in the esophagus must be removed by endoscopy. Button batteries in the esophagus must be removed expeditiously. Fish and chicken bones are problematic for a variety of reasons, and an individualized approach is appropriate. Coins are the most common ingested FB in children, and can also be managed on an individual basis.

Appropriate esophageal FBs may be removed by the Foley catheter technique, or passage into the stomach aided by esophageal bougienage or pharmacologic agents. All esophageal FBs may be removed by esophagoscopy.

TABLE 40-7 -- Important Considerations in Management of Esophageal Foreign Bodies

The esophagus does not tolerate foreign bodies (FBs) for prolonged periods of time. Do not expect impacted FBs to eventually pass by themselves. Fishbones will not dissolve over time.
1. As many as one third of pediatric patients with esophageal FBs are asymptomatic.
2. Consider esophageal FBs in the differential of the infant who is irritable, has recurrent pulmonary infections, or failure to thrive.
3. Multiple esophageal FBs are common in children and psychiatric patients.
4. Button batteries in the esophagus can cause necrosis within 2 hours and should be expeditiously removed.
5. A high percentage of adults with food boluses lodging in the esophagus have underlying esophageal disorders and require follow-up for evaluation.
6. Sharp FBs in the esophagus account for the preponderance of complications. These objects, including toothpicks, are best evaluated and treated with endoscopy.
7. Plain radiographs have little diagnostic value except for detecting metallic objects. A computed tomography scan is the best way to evaluate for bones and radiolucent objects.

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Chapter 41 - Nasogastric and Feeding Tube Placement

Leonard E. Samuels

A wide variety of patients require nasogastric (NG) intubation, including those with obstructed, preoperative, or potentially hemorrhaging upper gastrointestinal systems. Although the initial placement of feeding tubes is rarely the province of emergency clinicians, patients with feeding tube complications, and those requiring replacement of tubes, frequently present to the emergency department (ED). Therefore, the emergency clinician must be familiar with the different types of enteral tubes, as well as with their care, cleansing, and means of replacement.

PROPERTIES OF NASOGASTRIC AND FEEDING TUBES

Enteral tubes are made of various materials. Polypropylene is the most common material used for Levin and Salem sump NG tubes (Fig. 41-1), but it is too rigid for long-term use for a feeding tube. Although they are less likely to kink than others, polypropylene tubes are more capable of creating a false passage during placement. Latex (rubber) tubes are moderately firm, require greater lubrication for passage, are relatively thick-walled, and induce a greater foreign body reaction than tubes of other commonly used materials. Latex, especially in latex balloons, deteriorates more rapidly than other materials. [1] Foley catheters are primarily latex, although totally silicone Foley catheters are available for those with latex allergies. Silicone tubes are thin-walled, quite pliable, and nonreactive. However, the walls of silicone tubes are less strong and may rupture if fluid is introduced into a kinked tube. [2] Polyurethane tubes are quite nonreactive and relatively durable. Rigidity varies from manufacturer to manufacturer, depending on tube thickness. A stylet may aid in the passage of polyurethane and silicone tubes, but its use increases their rigidity and capability of tissue dissection, especially in tubes that have a small distal end bulb. [3] Some feeding tubes have weights, usually made of tungsten, which are nontoxic if released into the gastrointestinal tract.

NASOGASTRIC TUBE PLACEMENT

Indications and Contraindications

The simplest NG tube is the Levin tube, which has a single lumen and multiple distal "eyes." The advantage of the Levin tube is its relatively large internal diameter in proportion to its external diameter. The theoretical disadvantage is that a Levin tube should not be left hooked up to suction after the initial contents of the stomach have been drained, because the suction will cause the stomach to invaginate into the eyes of the tube, blocking future tube function and potentially causing injury to the stomach lining. Levin tubes are useful for diagnostic aspiration of stomach contents or for simple instillation of therapeutic agents. Tubes with lumens larger than 16 Fr are preferred for lavage procedures if large particulates are anticipated.

In practice, the Levin tube is no more prone to bleeding complications than the Salem sump tube, provided that the *intermittent* suction setting for the Levin tube is =40 mm Hg.[4] The Salem sump tube is preferred to the Levin tube for more chronic use as a drainage device because of the presence of a separate (blue-colored) channel that vents the distal main lumen to the atmosphere (Fig. 41-2). When extended gastric drainage is desired, Salem sump tubes should be placed on intermittent suction, even though the vent generally will prevent excessive vacuum at the tube tip. Note that potentially both intermittent suction and wall unit vacuum can exceed the venting capacity of the second lumen; for intermittent suction, the vacuum setting should be =120 mm Hg. [4]

The major indication for NG intubation is to aspirate the stomach contents. Aspiration may be useful in the management of upper gastrointestinal bleeding, particularly to differentiate upper from lower gastrointestinal bleeding.

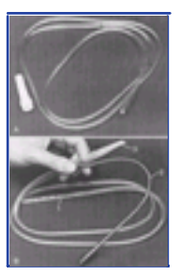

Figure 41-1 Standard nasogastric tubes. *A,* Standard Levin tube. This tube has a single lumen. It is sufficient for instilling material into the stomach or for diagnostic stomach aspiration. *B,* Salem sump tube. This tube contains a second lumen that allows venting during continuous suction. *1,* Gastric end with suction eyes. *2,* Pigtail extension (blue) of the air vent lumen. *3,* Connector for attachment of suction lumen to vacuum line.

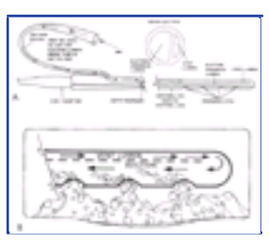

Figure 41-2 Diagram of the Salem sump tube. *A,* General design. *B,* Diagram of double-lumen principle for suction. *(Courtesy of the Argyle Division of Sherwood Medical, St. Louis, MO.)*

Except when frankly bloody fluid is obtained, the sensitivity and specificity of aspiration to detect upper intestinal bleeding are not good. [5] [6] [7] Use of Hemoccult or guaiac cards to detect bleeding in gastric aspirates is unreliable, because false-positive tests are obtained frequently. [5] Although variceal rupture has occurred during insertion of instruments into the esophagus, several studies suggest that NG tube passage is generally safe, even in the presence of esophageal varices. [8] [9] NG suction is indicated in cases where vomiting is likely to be recurrent or dangerous, such as with a paralytic ileus or intestinal obstruction, or to relieve acute gastric dilation. The trauma patient may need an NG tube as part of the evaluation for gastrointestinal injury or to decompress the stomach before surgery or peritoneal lavage. A radiopaque NG tube may help delineate transdiaphragmatic hernia of the stomach after trauma.

NG tubes are contraindicated in a few groups of patients with special predispositions to injury from tube placement. Patients with facial fractures who have a cribriform plate injury may suffer intracranial penetration with a blindly placed nasal tube. [10] A severe coagulopathy may be a relative contraindication for nasal passage of an NG tube. For patients with a coagulopathy or significant facial or head trauma, an NG tube passed through the mouth may be a better alternative (Fig. 41-3). Patients who have esophageal strictures or a history of alkali ingestion, especially recent alkali ingestion, may suffer esophageal perforation. Comatose patients may vomit during or after NG tube placement; their airways should be protected before placing the NG tube. Gagging will decrease venous return and increase cervical and intracranial venous pressure, which may be clinically significant in selected trauma patients with cervical or intracranial bleeding and in the presence of significantly elevated intracranial pressure. Indwelling NG tubes predispose patients to pulmonary aspiration because of tube-induced hypersalivation, depressed cough reflex, or mechanical or physiological impairment of the glottis. [11] Aspiration is also quite common with nasoenteral feedings in debilitated patients.

Despite their traditional use, *NG tubes are not routinely required in patients with mild to moderate pancreatitis*, [12] [13] and NG tubes may actually prolong hyperamylasemia and pain. Extended irrigation of the stomach with water in a patient with upper gastrointestinal hemorrhage can lower serum potassium levels, [14] and animal studies suggest that cold water lavage can cause rather than control bleeding. [15] [16] No study has shown irrigation to be effective in the control of bleeding,[6] [17] and vigorous lavage with cold water may lower the body temperature. However, a NG tube may be used to

Figure 41-3 An NG tube may enter the cranium or facial soft tissues in patients with severe head or facial trauma. Those with a coagulopathy may experience significant bleeding from nasal or pharyngeal trauma during passage of an NG tube. In such cases, a standard NG tube inserted through the mouth may be a better alternative.

instill air into the stomach for documentation of a suspected gastric perforation by enhancing visualization of free air under the diaphragm on an upright chest film.

Equipment

Passage of standard NG tubes or feeding tubes can be messy and may be accompanied by coughing, retching, sneezing, bleeding, and spilled water or stomach fluid. Both patient and clinician should be gowned; cleanup may be reduced if the bib area is covered with a towel and a supply of tissue or washcloths is available. The clinician requires gloves; the appropriate NG or feeding tube; a penlight or other light source; a tongue blade; an emesis basin; a glass of water with drinking straw (or an extra syringe if the patient is uncooperative); water-soluble lubricant; a stethoscope; a safety pin; a rubber band; topical anesthetic jelly, spray, or ointment; and vasoconstrictor nasal spray or liquid. For standard NG tube placement, a piston or bulb syringe (with a catheter slip-tip) should be available. NG feeding

tubes should have a compatible 50- or 60-mL syringe (some are Luer compatible and others are slip-tip compatible).

Tape torn in 4-in strips or a commercial NG tube holder (e.g., Suction Tube Attachment Device, Hollister, Libertyville, IL) should be handy for securing the tube after placement. Cotton-tipped applicators and tincture of benzoin may be helpful if the skin is greasy. Depending on the indication, other equipment may be needed (e.g., saline for irrigation, Magill forceps in an uncooperative or anesthetized patient). Make sure

Figure 41-4 Estimation of tube insertion depth. Before inserting the nasogastric tube, the clinician should estimate the length of tubing that will be required to ensure intragastric positioning without excess coiling. Holding the tube against the patient's body, measure the distance from the tip of the xiphoid to the earlobe *(A)*. Add the distance from the earlobe to the tip of the nose *(B)*. Then add another 15 cm *(C)*. Note the total distance using markers on the tube, or attach a piece of tape to the tube.

the feeding tube is designed for duodenal passage if that is desired—such tubes are usually longer than regular feeding tubes.

An antireflux valve (Keith Antireflux Valve, Sherwood Medical Industries, St. Louis) is a one-way valve used on the vent port of the Salem sump tubes. One study found the device to be cost-effective by reducing the need for gown and linen changes during inpatient NG tube use. [18] Placement of this device in the ED should be considered if extended use is anticipated. A topical anesthetic and a vasoconstrictor are also common adjuncts to tube passage.

Procedure

Explain the procedure to the patient. If the patient is alert, the head of the bed should be raised so that the patient is in the high Fowler position or upright. A towel is placed over the patient's chest to protect the gown, and an emesis basin should be available on the patient's lap. [19] The tube (typically a 16 or 18 Fr sump) should be positioned so that insertion distance can be estimated, and this distance should be marked with tape or noted using the markers printed on the proximal tube. A simple method is to extend the tube from the xiphoid to the earlobe and then from there to the tip of the nose and *then add 15 cm* (6 in) (Fig. 41-4).[20] Although other techniques of estimating appropriate NG tube insertion distance using formulas may be more accurate than the nose-ear-xiphoid method, the formulas are difficult to memorize and calculate. [21] [22] It is a common error to fail to estimate the

proper length of the tube before passage. The nares should be checked for obstruction and the tube passed down the more patent naris. Patency can be assessed by direct visualization or by having the patient sniff while first one and then the other nostril is occluded.

The pain and gagging associated with tube placement can be ameliorated significantly with the use of vasoconstrictors and topical anesthetics. The editors advocate the use of such adjuncts whenever the time and clinical situation permit. When logistically possible, topical vasoconstrictors, such as phenylephrine (Neo-Synephrine 0.5%), or oxymetazoline (Afrin 0.05%) should be sprayed into the nasal passages, usually both sides initially since one side may prove to be problematic, at least 3 to 5 minutes before the procedure. The nares, nasopharynx and oropharynx, should all be anesthetized at least 5 minutes before the procedure. Many clinicians neglect to anesthetize the pharynx and target only the nose, but gagging is reduced if the pharynx is included. Numerous topical anesthetics are available. Combinations of tetracaine, butyl aminobenzoate, and benzocaine (Cetacaine); nebulized or atomized (spray cans/bottles) lidocaine (4%); and lidocaine gels (2%) are most commonly used. Lidocaine preparations of 4% are most useful. Nebulized 4% lidocaine (2.5 mL containing 100 mg), delivered by face mask with the equipment used to administer bronchodilators to asthmatics, has been found to be superior to lidocaine spray as an anesthetic to reduce gagging and vomiting and to increase the chance of successful passage (Fig. 41-5).[23] [23A] After topical vasoconstrictor and anesthetic are administered, the tube is lubricated with viscous lidocaine or lidocaine jelly.[24] Lubrication of the nares can be facilitated using a syringe (without needle) filled with 5 mL of lubricant (Fig. 41-6). Simply putting anesthetic jelly on the tube before insertion, without prior nebulized/atomized anesthetic, will not provide satisfactory anesthesia. The mucosa should be anesthetized *at least 5 minutes before tube passage* if possible.

Topical anesthetics are generally quite safe, but attention must be paid to the total dose of administered anesthetic to

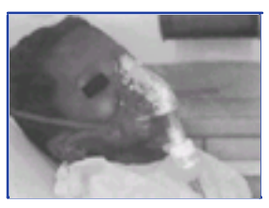

Figure 41-5 If time and situation permit, administration of nebulized lidocaine (2.5 mL of 4% solution) via a face mask and standard nebulizer used to deliver asthma medications, can decrease pain and gagging without increasing aberrant placement. The patient is instructed to breathe through the mouth and nose to anesthetize the nose and pharynx.

Figure 41-6 Whenever possible, some form of topical anesthesia should be used before passing a nasogastric tube. The method pictured can be used in addition to atomized anesthetic, and will be more effective than treating only the nasal opening. Fill a syringe barrel with 5 mL of 2% viscous lidocaine. Without using a needle, squirt the solution along the floor of the nose and allow it to drip into the nasopharynx and be swallowed. This method works best with the patient supine. Wait 5 minutes for the anesthetic to take effect.

avoid toxicity.[25] Note that each milliliter of a 4% lidocaine solution contains 40 mg of lidocaine that is absorbed systemically. Topical benzocaine used in endoscopy procedures may rarely cause methemoglobinemia. [26]

The tube is inserted into the naris along the floor of the nose and not toward the nasal bridge (Fig. 41-7) (i.e., directed posteriorly and not cephalad). Mild resistance may occur in the posterior nasopharynx, but only gentle pressure should be required to overcome this resistance. Bleeding or dissection into retropharyngeal tissue may occur if force is used, and it is better to try the other nostril or an oral tube if significant resistance is encountered. Once the tube passes into the oropharynx, *a pause will help the patient regain composure and enhance the chances for cooperation with the rest of the procedure* .

Have the patient, if he or she is alert and cooperative, sip water from a straw and swallow while the tube is advanced into and down the esophagus. This often helps ease passage of the tube. Once the tube is in the nasopharynx, flexing the neck also tends to direct the tube into the esophagus rather than the trachea. Excessive choking or gagging or any coughing, change in voice, or the appearance of condensation on the inner aspect of the tube should alert the clinician to the possibility of tracheal tube position, prompting withdrawal of the tube into the oropharynx. The tube may be inspected through the mouth to detect coiling or respiratory passage. A tube position lateral to the midline suggests correct position in the esophagus. [27] Once the tube is in the esophagus, *it should be advanced rapidly* to the previously determined depth. Slow passage of the tube prolongs discomfort and may precipitate more gagging. [19]

Confirmation of Tube Placement

Before the NG tube is secured, nonradiographic confirmation of its successful placement should be obtained. All confirmation methods have some possibility of error, so it is wise, when in doubt, to use more than one method. Radiographic evaluation is the most definitive way to confirm the position of an NG tube, but it is not standard to routinely obtain x-ray confirmation.

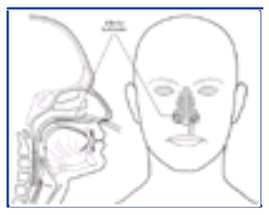

Figure 41-7 Tube location in the nostril. The nasogastric tube is directed along the *floor* of the nose, not toward the nasal bridge. The tube often slides through the tunnel beneath the inferior turbinate.

Insufflation of air into the NG tube is simple and quick. The requirement of increased pressure to instill air or the absence of borborygmi with instillation should raise concern about tube malposition or kinking. If the patient immediately burps on air instillation, an esophageal tube position should be suspected. A tube in the lungs or trachea is immediately discernible in an awake patient. Unfortunately, unrecognized passage of nasal tubes into the lungs occurs, but only in comatose, struggling, or demented patients, and insufflation is often insufficient to detect this type of malpositioning. [3] [29] [30] [31] [32] [33] [34] [35] The insufflation test also is unreliable in detecting whether the tube has advanced past the stomach into the small bowel.[32] In such patients, radiographic verification may be prudent.

Aspiration of stomach contents, especially if pH tested, is more reliable, and can be performed if positioning is in question. If the pH is less than 4, the nasoenteral tube has an approximately 95% chance of having its tip in the stomach. Furthermore, nonrespiratory placement is almost guaranteed. [28]

Aspirated fluid can occasionally be obtained from the lung or pleural space, but the pH should be 6.0 or higher. [28] [36] Approximately 2% of patients have an alkaline stomach pH.[37] Causes include duodenal reflux, antacids, H_2 blockers, or recent instillation of formula or medications. [37] [38]

Ask the patient, if he or she is awake and cooperative, to talk. If the patient cannot speak, respiratory placement is likely. Note that with small-bore tubes, patients may still be able to speak despite tracheal placement. [38]

Once correct tube position is tentatively confirmed, the tube should be secured. If the patient requires abdominal or chest radiographs for other diagnostic purposes, it is best to place the NG tube *before* obtaining the films. An NG tube deviated to the right may occasionally be seen in patients with traumatic rupture of the aorta, but this is not a reliable indicator.

Securing the Tube

The NG tube is generally secured to the patient with tape attached to both tube and nose. A butterfly bandage (or tape on each side of the nose) that then coils around the NG tube is a typical approach. The tube should be clean and possibly prepped with tincture of benzoin, as should the nose. If a tape should let go or require repositioning, both the tape and the tincture of benzoin must be replaced. It is wise to also secure the tube to the patient's gown, so that a tug on the tube will encounter this resistance before pulling on the material securing the tube to the patient's nose. A rubber band tied around the tube with a slip knot (Fig. 41-8*A, B*) and pinned to the gown near the patient's shoulder is effective. It is critical to ensure that the tube is not secured in such a way that it presses on the medial or lateral nostril. Necrosis or bleeding can result if a tube is not secured correctly.

When a Salem sump is used, the blue pigtail must be kept *above* the level of the fluid in the patient's stomach, or stomach contents may leak back through the vent lumen. If a patient needs to ambulate with a sump tube in place, the blue pigtail can be fitted into the plastic connector at the end of the suction lumen, creating a closed loop that should not leak. For a Levin tube, a syringe can be attached to the lumen and taped to the patient's gown. Alternatively, a commercial antireflux valve may be attached.

Placement Issues

NG tube placement in the unconscious patient can be attempted using the same technique as in the conscious patient, omitting those steps requiring patient cooperation. If the patient is intubated, the balloon of the endotracheal tube should be deflated briefly to allow passage of the NG tube. The NG tube is easily misplaced into the pulmonary tree in the unconscious patient, but quite unlikely if the patient is intubated. This complication may be missed during the procedure, because gag and cough reflexes may be suppressed and the patient cannot talk. In addition, the absence of swallowing may prevent successful passage of the tube. Several techniques may be used to successfully pass a NG tube in a difficult unconscious patient.

If initial standard attempts fail, the NG tube may be placed initially through a naris into the oropharynx. The tip of the tube is then visualized with a laryngoscope, grasped with Magill forceps, and pulled out of the mouth. An endotracheal

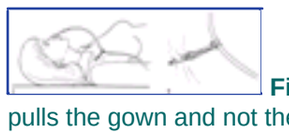

Figure 41-8 Attachment of tube to gown. *A,* Attach the nasogastric tube to the patient's gown using a rubber band and a safety pin as shown, so that the first tug on the tube pulls the gown and not the tape holding the tube in the patient's nose. *B,* Close-up view of attachment site.

tube with an internal diameter that is slightly larger than the external diameter of the NG tube is selected and is slit along its lesser curvature from its proximal end to a point 3 cm from its distal end. The slit endotracheal tube (generally 8 mm internal diameter [ID]) is then passed through the mouth into the esophagus. [39] Alternatively, a 7-mm ID slit endotracheal tube may be passed directly through the nose into the esophagus. [40] Passage into the esophagus is facilitated by the stiffness of the larger endotracheal tube and does not require active swallowing. The tip of the NG tube is then threaded into the endotracheal tube and advanced into the stomach (Fig. 41-9). The slit endotracheal tube is then removed from

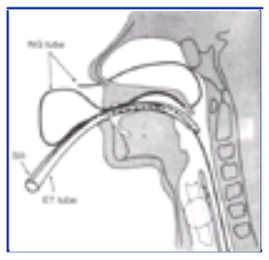

Figure 41-9 Diagrammatic representation of the separation of the nasogastric tube from the guiding endotracheal (ET) tube through the slit in the guiding ET tube. The nasogastric tube has first been passed through the nose and is pulled out through the mouth. The tip of the tube is then threaded into the guiding ET tube to ensure passage down the esophagus. The guiding ET tube is removed from the esophagus before separation from the nasogastric tube. Note the prior placement of another endotracheal tube in the trachea (partially shown) to avert passage of the guiding ET tube into the trachea. *(From Sprague DH, Carter SR: An alternate method for nasogastric tube insertion. Anesthesiology 53:436, 1980.)*

the esophagus. When the distal part of the endotracheal tube is visible, the unslit 3-cm distal part is slit with scissors. The endotracheal tube is removed, and the NG tube remains in place.[41] Any slack tubing is then advanced with the forceps or pulled back nasally, depending on the final depth required for the NG tube. The technique can also be performed by passing the slit endotracheal tube nasally, which also saves the trouble of orally advancing or nasally retracting any slack tubing.[40] [41]

In particularly passive, sedated, unconscious or toothless patients, guiding the NG tube with the fingers in the pharynx is occasionally successful (Fig. 41-10).[42] Displacing the larynx forward by manually gripping and lifting the thyroid cartilage has aided tube insertion, [43] as has simple jaw elevation. A soft nasopharyngeal airway, well lubricated, is at times easier to pass nasally than the NG tube, and then the lubricated NG tube can be passed through it. In addition, it affords some

protection to the nasal mucosa if multiple attempts to pass the NG tube are necessary, or if it is particularly important to minimize bleeding or trauma.

Cooling a NG tube increases its rigidity, and coiling it can increase the tube curvature, both of which may help pass the tube. Note that a cooled tube is more likely to dissect into tissue.

Ultimately, if all other methods fail, a flexible fiberoptic bronchoscope or esophagoscope can be placed under direct vision into and through the esophagus. [44] A guidewire is threaded into the stomach. The NG tube can be placed over the guidewire into the stomach; the guidewire is then removed. [45]

Complications

Complications of standard NG tube placement are similar to problems noted with NG feeding tube placement. The complications related to tube misplacement are discussed in that section. In addition, the clinician placing the NG tube in the patient with neck injuries should be cautious of potentiating cervical spine injuries with excessive motion during passage (especially in association with coughing and gagging in the awake patient). Furthermore, passage of a NG tube in the awake patient with a penetrating neck wound may exacerbate hemorrhage, should coughing or gagging result. Particularly serious forms of tube misplacement are

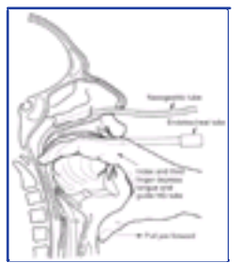

Figure 41-10 The passage of an nasogastric tube through the nose of an intubated patient. An endotracheal tube is in the tracheal via the mouth. The second and third fingers are placed into the posterior pharynx. Fingers depress the tongue. The nasogastric tube is guided down the esophagus by passing it through the second and third fingers that are in the posterior pharynx. The thumb is under the jaw and pulls the jaw forward.

pulmonary placement (Fig. 41-11) and intracranial placement (Fig. 41-12).

A tension gastrothorax can develop in patients with intrathoraracic stomach. The tension gastrothorax can occupy much of the left hemithorax, displacing the heart and lungs and causing a clinical syndrome identical to tension pneumothorax. While successful passage of a NG tube will relieve a tension gastrothorax, the high pressures of a tension gastrothorax often develop because torsion of the stomach in the chest prevents egress of air; that torsion may prevent ingress of the therapeutic NG tube. The condition is rare enough that further emergent therapy is based on case reports rather than substantial series. Relief of the tension gastrothorax has been accomplished with transthoracic puncture of the stomach with a 16-G catheter over needle. The catheter over needle was inserted in the second intercostal space in the mid-clavicular line, and then the needle was removed. The catheter was left in place attached to IV tubing with the distal end under a water seal.[46] A single 16-G puncture of the stomach is unlikely to leak and cause pleuritis; such punctures have long been used in percutaneous endoscopic gastrostomy tube placement. Inserting a chest tube into the stomach is not advisable because gastric fluid may leak into the pleural space. Once the tension on the stomach is relieved, it may be possible to pass the NG tube to prevent reoccurrence of the problem. The stomach, no longer tense and wedged in the chest, can twist to allow the tube to pass. Surgical correction of the condition permitting intrathoracic herniation of the stomach is the definitive treatment to prevent reoccurrence of tension gastrothorax.

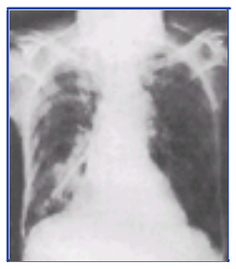

Figure 41-11 Levin tube inadvertently placed in the right main stem bronchus; an alveolar infiltrate consistent with early pneumonia is also shown. *(From Johnson JC: Letter to the editor: Back to basics for morbidity-free nasogastric intubation. JACEP 8:289, 1979.)*

NG tubes, when in place for prolonged periods, are a common cause of innocuous gastric bleeding and gastric erosions.

NASOENTERIC FEEDING TUBES

Indications and Contraindications

The most common indication for feeding tube replacement in the ED is unintentional removal of a pre-existing feeding tube. In one prospective study, 38% of tubes were removed unintentionally. Although some of these tubes had fallen out or been coughed out, more than half had been pulled out by the patient. [47] Tube rupture, deterioration, or clogging may also necessitate replacement. Management of the clogged or nonirrigating feeding tube is discussed in the section on clogged feeding tubes.

Enteric feeding tube replacement is contraindicated in the presence of vomiting, intestinal obstruction, severe ileus, upper gastrointestinal bleeding, distal enteric anastomoses, and conditions in which bowel rest is desired. [48] Occasionally, the patient whose feeding tube is displaced does not require nasoenteric feeding tube replacement because oral intake is adequate for nutritional needs. Patients suffering erosions or discomfort may be candidates for percutaneous endoscopic gastrostomy (PEG) placement.

Choice of Feeding Tube Site

Three major classes of enteral feeding tubes are in common use; classification is according to site of insertion. Tubes can enter through the nares, a cervical ostomy, or an abdominal ostomy. The mode of nutritional support is determined by the

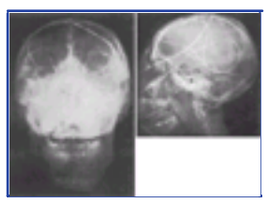

Figure 41-12 Anteroposterior and lateral skull radiographs demonstrating intracranial insertion of a nasogastric tube in a patient with multiple skull fractures. *(From Johnson JC: Letter to the editor: Back to basics for morbidity-free nasogastric intubation. JACEP 8:289, 1979.)*

patient's physiologic requirements and degree of debilitation, the disease process, the anticipated duration of inadequate oral intake, and the facilities and equipment available to initiate and maintain delivery of nutritional support. [49]

Tube feeding may be either a temporary or a permanent means of nutritional support and may have supplemental or complete responsibility for meeting the patient's nutritional needs. Enteral nutrition is less expensive and easier to use than total parenteral nutrition [50] and probably safer.[46] [51] Enteral nutrition is indicated when oral intake is less than two thirds of the patient's requirement, despite a functioning gastrointestinal system. Nasoenteric feedings are appropriate when fewer than 4 weeks of feeding are required,[48] although in some cases a pharyngostomy may be preferred because it is less irritating and cosmetically easier to conceal. Cervical or abdominal ostomies are recommended when more than 4 weeks of feeding are necessary.[48] Oral ingestion may continue with gastrostomy tubes and may be possible with NG and cervical ostomy tubes with a gauge of 12 or lower.

Enteral tubes are often categorized by the position of the tip of the tube. Tubes may terminate in the stomach (gastric tube, PEG tube) or the small intestine (jejunostomy tube, J tube). To confuse the issue, some tubes enter the stomach and terminate in the stomach (G tube) or in the proximal small bowel (J tube), whereas some tubes enter the GI tract directly through the small bowel wall (J tube). Gastric tubes are almost entirely PEG tubes. They are placed endoscopically with local anesthesia and without a surgical incision. Jejunostomy tubes are surgically placed under general anesthesia, require a surgical incision, and result in a surgical scar at the insertion site. Gastric feeding results in better digestion than intestinal feeding. J tubes are less likely to result in reflux and aspiration. Normally about 20% of gastric antral contents pass into the duodenum, with 80% refluxing back into the body of the stomach for further mixing. [22] If the feeding tube is placed in the antrum of the stomach or in the small bowel, enteral feeding solution passing into the small bowel may not be tolerated, resulting in diarrhea and paradoxical decreased nutrition.[21] [22]

The most common rationale for small intestinal feeding is to reduce regurgitation and aspiration. Disagreement exists concerning the clinical significance and frequency of regurgitation-induced aspiration. [47] [50] [51] [52] [53] [54]

Procedure

Nasoenteric feeding tube replacement requires greater time and effort if the patient is uncooperative or has a physically obstructing lesion. Nasoenteric feeding tube migration into the duodenal bulb generally requires patient positioning in the right decubitus position for about an hour after successful intragastric passage. [55]

The clinician should explain the procedure to the patient before tube passage. The patient's assistance with esophageal passage can be enlisted in two ways. First, the patient can assist with swallowing the tube. Second, the patient can vocalize when requested to confirm that the tube is not passing down the trachea. Many patients find it helpful to develop a signal to indicate to the clinician when they need a temporary reprieve and rest. It is generally advisable to restrain the hands of demented, impaired, or otherwise uncooperative patients.

The nares should be prepared before passage with generous application of a lubricant and local anesthetic. A local vasoconstrictor will dilate the nasal passages slightly and help prevent bleeding. Viscous lidocaine or water-soluble jellies (e.g., Surgilube) are excellent lubricants. A small amount of lidocaine with epinephrine can be squirted from a syringe into the nares a few minutes before lubrication. Application of lidocaine with epinephrine, application of the lubricant, or both, can also be accomplished with soaked cotton-tipped applicators. Alternatively, viscous lidocaine can be introduced into the nose with a gloved finger. The clinician should be prepared and garbed for the inevitable sneeze that this will provoke. Patients with a hyperactive gag reflex should gargle with viscous lidocaine or with benzocaine (Cetacaine) or lidocaine (Xylocaine) spray before the placement attempt.

The feeding tube stylet should be lubricated and inserted into the feeding tube before the insertion of the feeding tube into the nares. Tube stylets can be lubricated with water-soluble jelly. Dobbhoff, EntriFlex (Biosearch), and several other tubes have a preapplied lubricant that must be activated with a 5-mL flush of water. Frying pan lubricant sprays (e.g., Pam) also may work well on the stylet. Other types of lubricants may have greater potential for pulmonary complications or may damage the substance of the tubes. *The stylet should never protrude beyond the end of the feeding tube* , because these stiff, small-diameter wires have the capacity to scratch the esophagus and can encourage the creation of a false passage. The stylet may lock into position on the tube at the proximal end and should be properly secured.

An upright patient position is more comfortable for the clinician during NG passage. The distal end of the feeding tube should be moistened or lubricated and then passed down the more patent of the two nasal passages. Most persons' nostrils are fairly symmetric, but, in some cases, growths or old trauma may narrow the passage. The tube should be inserted into the nostril and then directed toward the ear. A common mistake of the inexperienced practitioner is to continue directing the feeding tube in a cranial direction. If the first nostril is impassable, the opposite side may permit tube passage. In particularly difficult cases, it may be desirable to first

pass a lubricated nasal airway (trumpet) and then pass the feeding tube through it, reducing nasal and nasopharynx stimulation.

The tube's passage into the nasopharynx can usually be detected by a lessening of resistance. One can then proceed with esophageal passage. Topical anesthesia of the pharynx may be required to ameliorate any excessive gagging that may occur at this point. Cough should warn of incipient respiratory placement of the tube. If the patient can vocalize, the tube has not yet passed through the vocal cords. The patient should bend the head forward if possible. This encourages the tube to pass into the esophagus and aids airway closure. If the patient is gagging or choking, it may be because the tube is beginning to coil in the oropharynx. Look in the mouth with a penlight to see if this is occurring. If the tube is coiled on entering the airway, temporarily pulling the tube back should relieve the problem. When pulling back, some resistance is again felt as the thicker end bulb of the tube begins to enter the posterior nasal passages; at that point, the tube has been pulled back far enough for a fresh try at esophageal intubation. The cooperative patient should swallow ice chips or take small sips of water with a straw to facilitate tube passage down the esophagus. In some cases it may be impossible to avoid coiling when the tube is advanced, unless the patient is swallowing. When the patient is uncooperative, the introduction of 5 to 15 mL of water into the mouth or into the proximal end of the feeding tube with a syringe may induce swallowing, facilitating tube passage. Although the patient may not swallow for several minutes, waiting for the swallow may mean the difference between a coiled or pulmonary tube placement and successful passage.

Another technique for passing soft feeding tubes involves attaching the distal end to a Salem sump tube by means of a gelatin cap (one half of a size-0 gelatin capsule, as used for a large medication capsule). Lubricate the joined tubes, avoiding lubrication of the soluble capsule. The Salem sump-soft tube complex is passed nasogastrically. Once the tubes are positioned in the stomach, both tubes are irrigated several times with 10 to 20 mL of water. After about 5 minutes the gelatin capsule should have dissolved, allowing independent removal of the Salem sump tube (Fig. 41-13). [2]

Placement Confirmation

Auscultatory confirmation of tube placement can be misleading. [3] [34] [35] The proper technique for auscultation is to inject 20 mL of air into the tube rapidly while listening with a stethoscope in the left upper abdomen. Air insufflation should occur without resistance and without delay in borborygmi. If the sound is muffled, faint, or delayed, the clinician may reinject another 20 mL of air and listen over the lower lung. If the sound is clearer, the tube may be in the lung. The injected air should be aspirated from the stomach after placement is confirmed so that the patient is less likely to burp or regurgitate.

Proper placement of NG feeding tubes should be confirmed with a radiograph. Tubes should be secured with tape before taking the radiograph. Tincture of benzoin applied to the tube and the patient makes the tape stick better. Commercial tube fixation devices may also be used. The position before and after the radiograph is taken is more likely to be the same if the tube is secured before filming.

Gordon suggests that radiographs are not necessary to check NG feeding tube placement if the following criteria are met in order. [2] The tube must be passed beyond the 50-cm mark in a normal-sized adult; palpation and visual inspection

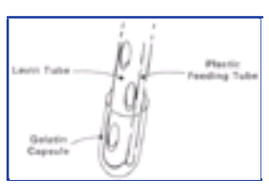

Figure 41-13 To facilitate the passage of a soft or very pliable plastic feeding tube, the tip of the feeding tube is joined to the tip of a stiffer nasogastric tube (such as a Salem sump) with the aid of a gelatin capsule. The capsule dissolves in the stomach in a few minutes, and the companion nasogastric tube separates and is withdrawn, leaving the feeding tube in place.

confirm that the tube is not coiled in the mouth or oropharynx; air insufflation occurs without resistance and without delay in epigastric borborygmi; 10 mL of water advances through the tube without difficulty; and some water can be retrieved with aspiration. Do *not* inject water into the tube if any of the preceding criteria suggest that placement may be in the lung or upper esophagus. A risk of delivering water into the airway exists. [48]

Aspirated pleural or pulmonary fluid contents can be mistaken for return of intragastric fluids. [3] In addition, radiographic confirmation of tube placement may be misleading. In viewing the radiograph, it is particularly important to study the area around the carina. An esophageal tube shows at most a mild change in course, whereas a tracheally placed tube usually deviates significantly as it travels into the right or left main stem bronchus. The end of an NG tube may appear to be in the stomach yet be in the left lung behind and below the top of the diaphragm. [56] When a stylet has been used for passage, the stylet should be left in the feeding tube for the radiograph, because the tube's course is not always visible without it. The stylets of most tubes are designed to allow insufflation and aspiration while in place. Even when stomach entry is certain, the intestinal location may be misleading on radiograph: A nasoenteric tube may lie completely to the left of midline and yet have its tip in the duodenum, or it may have a position overlying the right abdomen yet not have entered the duodenum. A contrast study is necessary to ascertain duodenal position when pulmonary placement has been ruled out. [55] [57]

The radiograph should also be examined for the presence of mediastinal air and a pneumothorax, which may suggest pulmonary or esophageal puncture. An esophageal puncture should be evaluated with endoscopy and may require surgery, depending on the size of the rent.

The end bulb of most nasoduodenal tubes will pass into the duodenum after patient positioning in the right decubitus position for an hour. Some researchers recommend pretreatment with metoclopramide to enhance gastric emptying. [50] [58] [59] One investigator found that metoclopramide enhances duodenal passage of nasogastrically placed feeding tubes in diabetic, but not in nondiabetic, patients. [55] Gastric antral motility in diabetics is often impaired; metoclopramide helps restore normal synchronized activity in these patients but has little effect on emptying in subjects who have normal antral function. The usual dose of metoclopramide is 10 mg administered IV. Also, 3 mg/kg of erythromycin lactobionate given IV over 1 hour works similarly and may be effective even if metoclopramide fails. [60] Endoscopy or fluoroscopy may be necessary if positioning and metoclopramide are not successful. [61]

Complications

Pulmonary intubation is an uncommon but well-known and potentially fatal complication of nasal feeding tube insertion (see Fig. 41-11). Coughing and respiratory distress are the most common symptoms of respiratory passage of an NG tube, but there may be relatively few symptoms in a demented or comatose patient. [62] Decreased mentation and an absent cough reflex are predisposing factors for unrecognized nasopulmonary intubation with NG tubes. [3] A small end bulb (e.g., 2.7 mm diameter) can slip past a tracheal high-volume, low-pressure cuff and pass easily to the lung periphery. [3] [62] [63]

To prevent nasopulmonary feeding tube intubation, one may choose to use the wire stylet only for initial tube passage. This will prevent pulmonary parenchymal penetration but not tracheobronchial intubation. It also will make passage of tubes positioned in the esophagus more difficult. [3] Esophageal entry of the feeding tube can be checked with the use of a laryngoscope. The metal stylet can be left in the tube during esophageal tube passage when an esophageal position has been confirmed by laryngoscopy. [64] A stylet should never be reinserted into a tube already in the patient. The stylet may puncture the tube at a kink or exit the side holes and puncture the esophagus. [65]

A pneumothorax may result when an NG tube dissects into or is withdrawn from the pulmonary parenchyma. [33] Bloody aspirate from a tube should heighten awareness of tissue damage.

A clogged or nonfunctional NG tube may prove difficult to remove. Fluoroscopy may allow careful insertion of a guidewire or stylet into an in situ tube to facilitate removal. Fluoroscopy also may identify the mechanical problem interfering with the removal. Bent-double segments are probably the most common; knots are uncommon but do occur. Excessive force should not be used in the removal of a NG tube, because serious injury to the patient may result.

Premature removal of the NG tube is the most frequent complication of feeding tube use. To help prevent removal by the uncooperative patient, the NG tube may be secured to a loop anchor passed in the same naris. The anchor works by aversive stimulation of the soft palate and nose with distraction of the NG tube, rather than by mechanical stabilization of the tube.

Sax and Bower recommend a technique for creating a separate NG tube anchor.[66] A soft weighted nasoenteric tube is cut approximately 12 in from the top. A heavy (2–0) silk suture is passed through the tube to exit the side hole. The guidewire is inserted with care, as it must not protrude from the cut end. The patient should be sedated if uncooperative. The tube is inserted through the anesthetized naris into the nasopharynx, grasped with Magill forceps, and pulled to exit from the mouth (Fig. 41-14*A*). The excess tube is trimmed without cutting the silk suture. A closed loop is made by tying the silk suture in front of the nose. The loop must be slack enough that it does not apply continuous pressure to the nose or palate at rest. The nasal feeding tube is passed through the same nostril and secured to the loop (Fig. 41-14*B*). This anchor is simpler to construct and more comfortable than anchors passed through the opposite nostril. [66]

Complications of properly placed nasoenteric tubes include nasopharyngeal erosions, esophageal reflux, tracheoesophageal fistulas, gagging, rupture of esophageal varices, and otitis media.[48] One survey of nasogastrically fed patients found that the most distressing features of NG feeding tube use were deprivation of tasting, drinking, and chewing of food; soreness of the nose; rhinitis; esophagitis; mouth breathing; and the sight of other patients who were eating. [67]

Checking feeding tolerance is difficult with small-gauge feeding tubes. Aspiration of tubes to check for residual is not recommended with tubes of 9 Fr or smaller. Aspiration is likely to clog the tubes, because they collapse under pressure and because relatively small particles can occlude the tube. For the same reasons the residual is likely to be inaccurate.[51]

Figure 41-14 Placement of a nasogastric tube anchor to secure a companion nasogastric or feeding tube in an uncooperative patient who repeatedly pulls out the feeding tube. *A*, Forceps grasp the tube in the pharynx and pull it out through the mouth. This will serve as an anchor tube. *B*, The ends of the short anchor tube are tied together to form a loop, and the companion nasogastric or feeding tube is tied to the anchor loop.

Patient Instructions

Patient instructions should include a proscription against aspiration in small tubes. To maintain catheter patency, small tubes should be flushed with 20 to 30 mL of tap water at least two to three times daily and after administration of medication. [51] [57] Water is a more effective irrigant than cranberry juice. [68] Medications should be in liquid form or be completely dissolved, or they may clog the tube. Methods of dealing with a clogged tube are discussed subsequently.

The tube should be anchored to the nose and face in such a way that it is not in contact with the skin at the nasal opening. This reduces tube discomfort and prevents necrosis of the alae, nares, and distal septum. Patients who exhibit a tendency to pull on their tubes need adequate restraints. Patients receiving tube feedings should have their heads elevated to at least 30° above the horizontal. [51] [69]

PHARYNGOSTOMY AND ESOPHAGOSTOMY FEEDING TUBES

Indications

Cervical pharyngostomy and cervical esophagostomy both have been developed relatively recently. Cervical esophagostomy was first described by Klopp in 1951, [70] and cervical pharyngostomy was described in 1967 by Shumrick.[71] Cervical esophagostomies are generally performed at the time of cervical or maxillofacial operations. Malignant growths of the proximal esophagus, head, or neck are the primary indications for esophagostomy. Cervical esophagostomies may eventually evolve a permanent sinus, allowing the feeding tube to be removed between meals.

As with an esophagostomy, a pharyngostomy may be either a simple ostomy or a permanent tract formed by suturing pharyngeal mucosa to the skin.[71] [72] [73] The more common version is a simple opening that closes very rapidly (within a few hours) if it is not stented with a tube (Fig. 41-15). Pharyngostomy is a simple procedure and does not require general anesthesia. It is useful when NG feeding tubes are contraindicated or when prolonged tube feeding is anticipated. [49] Patients with traumatic or congenital anomalies of the maxillofacial region who are undergoing an operation and patients with impaired swallowing from neuromuscular disorders are potential candidates for cervical pharyngostomy. [73] A pharyngostomy is also indicated to bypass obstructing lesions for feeding, to assist healing after head or neck surgery for malignancy, and to feed the unconscious patient. A pharyngostomy can also be used for gastric decompression if it is required for more than 3 days.[72] Cervical pharyngostomy tubes produce only mild discomfort. Patients do not usually complain about pharyngostomy tubes, as they do about a

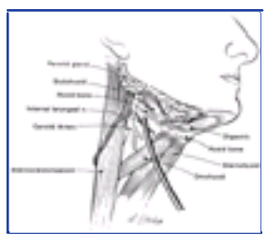

Figure 41-15 Pharyngostomy feeding tube.

NG tube. The additional comfort and ease of productive coughing are especially important in critically ill and elderly patients.

Technique of Tube Passage

The feeding tubes commonly used for esophagostomy and pharyngostomy feeding are the same as those used for NG feeding. Polyurethane feeding tubes are the most frequently used. The large (>3 g) bolus weights on some feeding tubes may be inconvenient to pass through the ostomy. Tubes that are approximately 90 cm (3 feet) long are appropriate for gastric feeding; tubes that are longer—108 to 112 cm (43 or 45 in)—are used for duodenal feeding.

The feeding tube replacement technique is the same for pharyngostomy and esophagostomy. The outside of the tube tip can be lubricated with a small amount of water-soluble lubricant jelly. Mineral oil, which irritates the airways if aspirated, should never be used. The tip of the tube is inserted into the ostomy and directed caudally to ensure that it enters the esophagus and does not pass upward into the nasopharynx or mouth (Fig. 41-16). The patient may be able to assist by attempting to swallow. The length of feeding tube required varies depending on the position of the ostomy and is several centimeters longer than the distance from ostomy to xiphoid. For duodenal feeding, the tube should be advanced about 20 cm beyond the distance from ostomy to xyphoid.

If the feeding tube persistently exits the mouth during attempts at passage instead of passing down the esophagus, the following two techniques may prove useful. After insertion of the feeding tube a short distance into the ostomy, a flashlight is used to visualize the tube in the pharynx. The feeding tube is grasped slightly proximal to the end bulb using Magill forceps, and the end bulb is directed toward the esophagus in the posterior inferior pharynx. Once the tube is properly directed, it may be possible to advance the

Figure 41-16 Proper path for an esophagostomy or a pharyngostomy tube.

remainder of the tube through the external ostomy. Sometimes it is necessary to use the forceps to advance the entire length of the feeding tube. An alternative method is to allow the feeding tube placed through the ostomy to exit the mouth for the entire distance that must be passed down the esophagus. The end bulb of the tube is then directed into the posterior pharynx, and the patient is directed to swallow as for an orogastric tube. Toward the end of tube passage, it may be necessary to use a Magill forceps or to pull back the tube slightly at the ostomy to eliminate a short loop of extra tubing in the oropharynx.

Tube replacement is more difficult in the first week after the creation of a pharyngostomy or an esophagostomy. A tract forms after the first week and helps prevent tissue dissection by the tube. The angle of a well-formed tract also encourages appropriate esophageal passage. A well-formed tract closes more slowly than a new ostomy, although in some people even a long-term ostomy may begin sealing within a few hours. If an ostomy is too narrow for the replacement tube, the ostomy should be stented with a narrower tube and the patient's surgeon contacted.

Complications

Complications of pharyngostomy and esophagostomy include local soft tissue irritation, accidental extubation because of excess length of the external tube, pulmonary aspiration from vomiting, arterial erosion with exsanguination, and esophagitis or stricture of the esophagus from reflux. Accidental pulmonary intubation is less common with cervical ostomy tubes than with NG tubes, at least partially because patients with cervical ostomies are more likely to be alert and have functioning cough reflexes. Auscultation and aspiration are still advisable techniques to check tube placement. Radiographic evaluation may also be necessary and is essential to confirm duodenal feeding.

GASTROSTOMY, GASTROENTEROSTOMY, DUODENOSTOMY, AND JEJUNOSTOMY TUBES

The mid-19th century clinician Sedillot described the first functioning gastrostomy, which formed as a complication of a war wound. The gastrostomies performed by Sedillot on two patients resulted in peritonitis and death. [73] [74] The jejunostomy procedure was first performed by Surmay in 1879. It was not until the 1890s that further innovations in surgical technique allowed the gastrostomy to be popularized. The Witzel serosal-lined gastric tunnel technique (Fig. 41-17) and the Stamm procedure of concentric pursestring sutures placed around the gastrostomy tube were developed in the 1890s. These two techniques prevent significant intraperitoneal gastric fluid leakage, a complication that had frequently resulted in the deaths of gastrostomy patients. Both Witzel and Stamm gastrostomies tend to close rapidly without a stenting gastrostomy tube. In the early 1900s, the tubular (Depage-Janeway) gastrostomy was developed. The Depage-Janeway gastrostomy results in the creation of a permanent mucocutaneous ostomy. Since the turn of the century, more than 30 different operative techniques have been described for tube gastrostomy.[49]

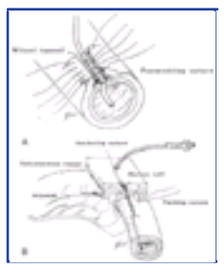

Figure 41-17 *A,* Formation of the Witzel tunnel. *B,* Final catheter placement. *(From Wiedeman JE, Smith VC: Use of the Hickman catheter for jejunal feedings in children. Surg Gynecol Obstet 162:69, 1986.)*

Operative Indications and Contraindications

Neurologic diseases constitute the most frequent indication for a gastrostomy tube. [52] Facial fractures, oropharyngeal trauma, and tracheal and laryngeal injuries may be indications for placement of a temporary feeding gastrostomy. Rare indications for gastrostomy include enhancement of nutrition by continuous feeding in severely debilitated patients who still are capable of oral intake, provision of a route for bile replacement in patients with an external biliary fistula, and the need for long-term gastric decompression. Indications for gastrostomy tube placement in children include neurologic diseases, facial reconstructive surgery for congenital deformities, and maxillofacial trauma. Young children who require long-term administration of unpalatable medications or dietary components may also require a gastrostomy. Tube duodenostomies are created almost exclusively for duodenal decompression after partial gastrectomy with Billroth II anastomoses. [73] Permanent jejunostomies are rarely used. Tube jejunostomy is indicated when the proximal bowel has a fistula or is obstructed, when recovery of small bowel motility is anticipated long before recovery of gastric motility, and after a gastrectomy.[49] [73]

Contraindications for gastrostomy feeding include severe gastroesophageal reflux, upper gastrointestinal fistulas, repeated aspiration of gastric contents, and intestinal or gastric outlet obstruction.[49] Jejunal feeding is contraindicated if the highly osmolar feeding solutions required for jejunal feeding are poorly tolerated and cause copious diarrhea.

Indications and Contraindications for Tube Replacement

The nursing home patient with a nonfunctioning or dis-placed feeding tube represents a common ED presentation. The clinician cannot always determine the location of the original feeding tube by simply looking at the patient who arrives in the ED for tube replacement. Nevertheless, the emergency clinician should attempt to ensure that the terminal end of a replaced tube is in the same viscus as the original. External inspection may or may not reveal where a feeding tube should terminate. A de Pezzer (mushroom) or Foley gastrostomy tube is designed only for intragastric termination. Some tubes have two lumina, one terminating in the stomach for decompression and the other in the small bowel for feeding. These can be confused with tubes that have two entrances to one lumen (one for continuous feeding and the other for medications) and tubes that have a second lumen leading to an inflatable balloon.

The clinician has a few options when faced with the task of replacing a feeding tube. Unfortunately, old records or nursing home personnel rarely give specific information that is helpful to the emergency clinician. If the tube is blocked, yet still present, a contrast study may demonstrate the final position of the tube. If only a stoma exists, one may request that the nursing home describe or send the prior tube to the ED. If a blocked tube is removed, it can be replaced with a similar device. If no surgical scar is seen at the stoma site, the tube is almost certainly a G tube, or a G tube that terminated in the jejunum. Placing a new tube followed by a contrast study usually settles the issue and allows the clinician to make decisions on balloon inflation, or possibly substitute a new device. When in doubt there is no downside of passing a Foley catheter without balloon inflation, taping it to the skin, and referring the patient to a consultant or the original referring clinician. It is unwise to avoid placement of some type of tube since the stoma will quickly close and may necessitate a more complicated procedure later. The only real concern of placing a gastric tube into the jejunum is that the balloon will produce intestinal obstruction if it is fully inflated.

If the tube is nonfunctioning yet still in place, the clinician must make a judgment as to the risk versus benefit of removal and replacement versus an attempt at unclogging the tube (see subsequent section on unclogging). *The major concern is that a new tube may be misplaced (i.e., into the peritoneal cavity)*. If it appears that a skin incision was used to place the tube, it is unlikely that the patient has an easily removable tube. If the patient has signs of a complication (e.g., infection, ileus, intestinal obstruction), surgical consultation is warranted.

Ease and safety of transabdominal feeding tube replacement depend on the surgical procedure performed and the length of time since placement of the feeding tube. For a simple gastrostomy, the insertion site of the tube through the gastrointestinal wall is sealed by either annular or plication sutures. The gastrointestinal wall is approximated to the peritoneum around the site of penetration to provide a further leakage barrier. The tube is then secured outside the abdominal wall. A Witzel tunnel is a serosal tunnel created when the feeding tube is placed alongside the viscus for a distance after exiting the viscus, and the bowel is pulled up over it along this distance and secured with sutures (see Fig. 41-17).[75] [76] In one type of Hickman catheter jejunostomy, the tube passes

through a Dacron cuff and a Witzel tunnel. [76] Reinsertion of a Hickman catheter through a tortuous, rough Witzel tunnel is unlikely to be simple. [77] A percutaneous gastrostomy may have been placed without any attempt to affix the stomach to the abdominal wall.

Nonoperative tube replacement techniques are safe only through an established tract between the skin and the bowel. Catheter replacement should not be attempted in the immediate postoperative period. A simple gastrostomy takes about a week to form a tract.[72] A Witzel tunnel may take up to 3 weeks after the operation to mature sufficiently for safe nonoperative tube replacement. A nonfunctional tube can still serve as the stent for the gastrostomy tract and should not be removed if it cannot be promptly and safely replaced.

Equipment

Gastrostomy tubes come in an unusually varied selection of styles and materials. Rubber, silicone, and polyurethane tubes are all in common use. Many gastrostomy tubes are designed with a flange or a crossbar (bumper) to anchor them in the stomach and prevent migration into the small bowel. A Foley catheter may be used as a replacement tube but it is more temporary, difficult to secure to the skin, and the latex balloon may be weakened by stomach acid. When possible, a dedicated feeding

tube should be used instead of a Foley catheter (Fig. 41-18).

Equipment for feeding tube insertion includes gloves, stethoscope, feeding tube, external bolster, lubricant, basin, and a syringe that fits the tube. Tincture of benzoin, tape, and absorbent dressing material may be used to dress the wound, although many are better left undressed. [78] Some feeding tubes require special plugs or connectors. Others need to be pinched with a clamp when not in use to prevent leakage. Some tubes are placed with the aid of accompanying guidewires or stents. For still others, it is necessary to use a clamp or hemostat, endotracheal tube stylet, urinary or uterine sound, laryngeal dilator (no. 14), guidewire, or other appropriate rod or support as an aid to tube passage (Fig. 41-19*A, B*).

A stylet to assist introduction of de Pezzer catheters can be fashioned by cutting half the stylet from a 9 Fr pediatric chest tube inserter. The tip can be filed smooth. The device will be 10 to 12 cm long and can be inserted alongside the de Pezzer catheter and into its tip to distend and flatten the mushroom (Fig. 41-20).

Transabdominal Feeding Tube Removal

A feeding tube may have to be removed because it is irreversibly clogged, leaking, or broken; persistently developing kinks; too large or too small; causing a hypersensitivity reaction; associated with an abscess; or not the appropriate length for feeding into the desired viscus. Before a new transabdominal feeding tube is inserted, the old tube must be removed. Most, but not all, tubes can be removed without endoscopy. It is imperative to know whether the tube in place is safe to remove before attempting to remove it. Standard de Pezzer or mushroom catheters *that have been modified with bolsters or rings at the time of endoscopic or surgical insertion* may no longer be safe to remove with traction. Tubes are occasionally secured with sutures or rigid internal bumpers or stays. It is rare, however, to encounter a tube that cannot be removed with traction. Recently placed feeding tubes may need to be left in until a tract has formed (1 to 2 weeks depending on the procedure) even if the tube is nonfunctional. *The externally visible tube does not always reveal the internal stabilization* (see Fig. 41-18).

A simple Foley catheter gastrostomy is easiest to remove. Once the Foley balloon is deflated, the tube should slide right out. If the Foley balloon cannot be deflated, cutting the tube may allow the balloon to deflate. The catheter must not be cut so close to the abdomen that it will be impossible to maintain a grip on it for a traction removal if the balloon still does not deflate. The balloon also may be punctured to cause it to deflate. To puncture a Foley balloon, traction is applied to the catheter to draw the balloon up against the ostomy. Using the taut feeding tube as a guide, a 20- or 21-ga needle is passed along the tube to puncture the balloon. It may be necessary to try again on the other side of the catheter, because the balloon may be asymmetrically inflated, and contact with the needle may be established on one side and not the other. The clinician should be careful not to track away from the ostomy into the patient's abdominal wall or to cause separate punctures of the stomach. The balloon is allowed a minute to deflate before another attempt is made at traction removal. Large nondeflating balloons should probably be punctured, whereas small balloons may be removed with traction.

Traction is an acceptable removal technique for feeding tubes that are secured by a small mushroom. A towel is placed over the orifice, and the clinician applies counterpressure with the flat part of the hand against the abdominal wall as the tube is placed under tension (Fig. 41-21). This causes the tube and end mushroom to narrow, and the tube should come out easily. The inner crossbar, if present, may remain in the stomach when the rest of the feeding tube complex is removed by traction. Obstruction from the crossbar, which will pass in the stool, has yet to be reported for adults. In small children, obstruction is a possibility, and the crossbar should be removed by endoscopy. [77] [79]

A local anesthetic may be useful in selected cases of feeding tube removal, especially when the tube is in some way secured subcutaneously—for example, by a Dacron cuff. [76] It may be difficult to remove a catheter accidentally caught by a fascial suture during operative closure. [80]

Removal of gastrostomy tubes with moderate to large mushrooms may be easier if the mushroom is distended with a sound or stylet. The length of the gastrostomy tube should be known so that the sound may be inserted to the correct depth. Firm resistance should be noted at that point. Firm resistance at deeper depths represents pressure on the viscus wall and can result in viscus puncture. [59] The premeasured stylets that come with feeding devices are useful instruments for assisting in removal. This is particularly true of gastrostomy "buttons," whose ends resemble de Pezzer catheters. Because buttons come in a variety of lengths, it is important to have the proper stylet. Following elective permanent removal of a gastrostomy tube, a pressure dressing should aid in closure of the fistula. [59]

If it is not possible to pull the inner bolster or mushroom out through the ostomy, it may be acceptable to cut the tube at the skin, push the remaining short stump into the stomach, and rely on later rectal passage. Although obstruction or impaction is infrequent, it can occur, and this alternative has the potential to be problematic with children or patients who have had previous impaction, potential for bowel obstruction,

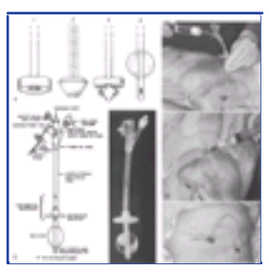

Figure 41-18 *A,* Various types of gastrostomy tubes. *1,* Polyurethane catheter with collapsible foam flange (CORPAK MedSystems of Kentec Medical Inc, Irvine, CA). *2,* Silicone catheter (American Endoscopy [Bard Interventional Products, Billerica, MA]). *3,* Latex catheter with a movable external bolster and an internal mushroom or de Pezzer-type flange on the end (American Endoscopy [Bard Interventional Products, Billerica, MA]). *4,* Balloon (Foley) catheter (Wilson-Cook Co., Winston-Salem, NC). *B,* A user-friendly gastrostomy tube is supplied by CORPAK MedSystems (Wheeling, IL). The tube is packaged with lubricant, a prefilled syringe for inflating the balloon, and an extension set. The color-coded inflation valve indicates tube size (12–24 Fr). The silicone tube uses a retention balloon and a movable bolster, similar in design to a Foley catheter. Note that the retention bolster is designed to prevent inward migration of the tube and is not to be an anchoring device sutured to the skin. *C,* Placement of the CORPACK feeding tube and inflation of the balloon. Note that the bolster on the external tube is advanced to the skin to secure the tube. *D,* A standard Foley catheter may serve as a more specialized feeding tube replacement. The latex balloon may deflate or be weakened by gastric acid; so, this catheter is not ideal for long-term use. In addition, unless the Foley catheter is secured to the skin, it may migrate and the balloon may cause a pseudo obstruction.

or stool-passing problems. Rigid or large internal mushrooms and bolsters, the very kind that cause the most difficulty with percutaneous removal, also are more likely to cause difficulty with rectal passage. In no case should a device be released into the gut with a long length of tubing attached. Remember that double-part tubes may have an additional length of tubing for duodenal or jejunal feeding that extends far past the inner bolster. Korula and Harma [81] reported the successful intestinal elimination of 63 of 64 gastrostomy tubes that were cut at the skin entrance and advanced into the stomach through the stoma. These cases included tubes with internal bumpers, and success occurred regardless of the nature of the patient's underlying medical disorder, age, or method of original tube placement. However, no patient had suspected obstruction or potential for obstruction (e.g., no prior radiotherapy, inflammatory bowel disease). The one lodged tube required endoscopic removal from the pylorus. In most cases, tube passage was documented by sequential radiographs, with a mean interval to passage of 24 days (range, 4 to 181 days).

Some clinicians and surgeons may strongly condemn cutting off the tube at the skin, even when the risks posed by the procedure are very low. It is always advisable to contact

Figure 41-19 *A,* An endotracheal tube stylet used to distend the flange of the de Pezzer catheter. *B,* A lubricated wooden cotton-tipped swab can also be used as a makeshift stylet.

the patient's private clinician before cutting the tube. In some cases endoscopic retrieval of the tube remnant will be preferred to allowing rectal passage, and the tube should not be cut until just before or during endoscopy to ensure that migration does not occur before endoscopy.

Transabdominal Feeding Tube Replacement

Dislodged tubes should be replaced as soon as possible. If a similar sized tube will not easily pass, it is preferable to replace

Figure 41-20 A modified pediatric chest tube inserter used to distend the flange of the de Pezzer catheter. The sharpened end of the inserter (trocar) has been rounded.

a dislodged tube with a smaller or temporary tube to maintain the patency of the tract, rather than wait for an ideal situation. The tract may close or narrow in a matter of hours, and it is very difficult to easily replace tubes that have been out for more than a day. Some clinicians will first gently explore the tract with a blunt probe, such as a cotton swab, to ascertain the patency and course of the tract (Fig. 41-22*A*). Gently dilating the ostium and carefully probing and dilating the tract with a hemostat may facilitate passage (Fig. 41-22*B*). It is emphasized that a false tract may be created with vigorous exploration or manipulation. Ideally the new tube slips into position with minimal force, but a number of attempts may be required to negotiate tissue planes and manipulate the tube into the proper position. A relaxed patient aids in placement. Injecting a local anesthetic into the opening of the tract may ameliorate pain. Systemic sedation/analgesia is seldom required but should be considered in the combative or extremely uncooperative patient. *The clinician should not use excessive force to pass a tube since misplacement may result.* It is not uncommon to precipitate some bleeding, and although this may be distressing to the patient or family, it is not a significant clinical issue.

A Foley catheter is a simple gastrostomy tube to replace. After the tract opening and distal Foley catheter are lubricated, the Foley balloon's integrity is checked by inflation. The catheter is then inserted into the tract. Good placement can be recognized by easy passage, prompt borborygmi with 20 mL of air insufflation, and rapid return of stomach juices with aspiration. The balloon is then inflated with saline (30-mL balloons are best), and gentle traction is applied to draw the balloon against the stomach wall. *Always inflate the Foley balloon with saline*, because balloons inflated with air deflate more easily. Tube replacement is usually successful if the tube has not been dislodged for more than 4 to 6 hours. If passage is impossible, a radiologist may be consulted to advance the tube over a guidewire that has been passed through the tract using fluoroscopy.

Figure 41-21 Gentle, firm traction *(A),* using the flat part of the opposite hand for countertraction, may remove most percutaneous endoscopic gastrostomy tubes, even those with internal mushroom bumpers *(B).*

An external bolster may be threaded onto the catheter. The external bolster is a ring or bar of material threaded onto a tube that creates a large bulge on the tube and prevents inappropriate ingress of the tube into the ostomy. The anchor must adhere strongly to the tube so that mild stress on the tube does not cause the bolster to migrate up the tube. The bolster can be salvaged from the old tube or constructed in a number of ways. An anchor may be made from the end nipple of a de Pezzer catheter. The ring from a 24 Fr catheter, taken off at its junction with the end nipple, fits snugly over a 22 Fr Foley catheter when the balloon is distended slightly. The nipple can be pushed forward to an anchoring position near the stoma. The nipple can be fixed in this position by fully distending the Foley balloon and applying a circle of adhesive tape just adjacent to the nipple on the stem on the

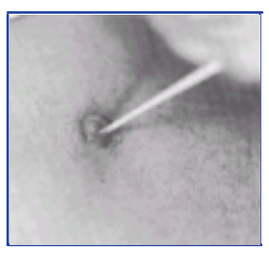

Figure 41-22 The tract of a feeding tube may close or become narrowed within a few hours after removal. When replacing a tube that has been removed, gentle probing of the tract of the previously placed feeding tube gives the clinician an idea of the patency and direction of the tract. In this case, a sterile cotton swab is gently advanced, being careful not to produce a false tract.

side away from the body.[82] Adhesive tape sticks better if the lubricant is removed and the stem is prepared with tincture of benzoin.

An external anchor may be made from a segment of tube from a large rubber catheter (Fig. 41-23). A segment approximately 3 cm in length is cut to form the bolster. Two diamond-shaped openings can be formed on both sides of the segment by bending the segment and clipping it with scissors on either side of the bend. The diameter of the holes should be slightly smaller than the catheter. A hemostat or a Kelly clamp can be inserted through both holes to grasp the external end of the gastrostomy tube, which can be bent in half—with some difficulty—to narrow its diameter (Fig. 41-24). The hemostat can then pull the tube through the bolster, which can be threaded down the tube and anchored with tape as described previously (Fig. 41-25). The outer crossbar should be located 1 cm away from the skin. [83] Contact between the crossbar and the skin promotes moisture entrapment and maceration. Too much tension on the gastrostomy tube can result in necrosis of the gastric wall where it abuts the inner mushroom or balloon. Proper placement of the external bolster helps avoid this complication (Fig. 41-26).

Many clinicians prefer mushroom or de Pezzer gastrostomy tubes, which are more difficult to replace than Foley catheters. The advantage of these catheters over Foley catheters is that the mushroom nipple keeps its shape more reliably than the Foley balloon, which tends to deflate. [67] Foley catheters also have a greater tendency to migrate internally and block the pylorus. [49] A Kelly clamp or other stylet can be placed through a side hole into the tip of a gastrostomy mushroom and used to elongate the end for easy passage through the gastrostomy (see Fig. 41-19 and Fig. 41-20). Lubrication of the mushroom may make it more difficult to maintain the stylet's position in the mushroom. Some stylets are suitable for passage down the catheter lumen to elongate the end. Tubes should never be forced through a stoma for replacement, because this can cause separation of the viscus from the external stoma and lead to viscus leak or tube misplacement. [59]

The replacement tube provided in the ED does not have to be—and in a few cases should not be—the same type placed at surgery. The tube must be compatible with the feeding system, terminate in the same viscus, and fit through the ostomy. When a Witzel tunnel jejunostomy is created, the catheter most frequently used is a Broviac catheter.

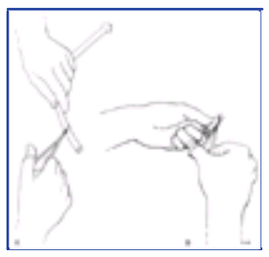

Figure 41-23 *A,* A 3-cm segment of thick latex tubing is cut from the proximal segment of a catheter. This segment is used to make an external bolster for a feeding tube and anchors the feeding tube, preventing unwanted ingress of the tube into the patient. *B,* A 3-cm segment of latex tubing is bent in half and cut to create a hole on each side of the segment.

An appropriate replacement is a lubricated polyurethane tube shortened to a total tube length of 30 to 40 cm. Although the polyurethane tube is strong enough to be used for tube replacement through the Witzel tunnel without a guidewire, Broviac (silicone) catheters are too pliant to be coaxed

Figure 41-24 A hemostat is inserted through the holes in the completed bolster and grasps the feeding tube. The end of the feeding tube has been folded to reduce its external diameter.

through the resistive tunnel.[76] Jejunal feeding tubes are generally advanced 20 to 30 cm into the jejunum.

Jejunal feeding tubes may be placed through or alongside a decompressing gastrostomy. Original placement of the jejunal feeding tube is endoscopic. Replacement of these tubes also

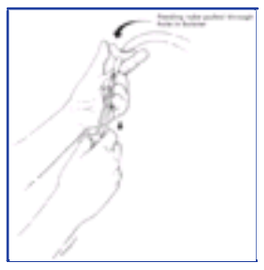

Figure 41-25 The feeding tube is pulled through the bolster. The bolster is advanced to 1 cm above the skin of the external abdomen.

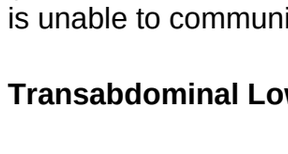

Figure 41-26 A Foley catheter can always be used as a temporary feeding tube replacement *(A)*. The thin-walled Foley balloon usually only remains inflated for a month or so, and a specialized feeding tube, such as the CORPAK (see Fig. 41-18) is preferred for long-term use. A bolster can be made to prevent inward migration of the tube. In this case the previously removed Foley catheter was used to make the bolster for the new one *(B)*.

generally requires endoscopic assistance.[84] [85] [86] Fluoroscopic techniques can be used to help guide these tubes; however, these techniques are out of the realm of emergency practice.[48] Occasionally, feeding tubes are placed in the jejunum because of gastric ileus.[49] [87] If gastric ileus is no longer present, a gastrostomy tube may suffice. The rationale for jejunal feeding, risk of aspiration, and acceptability of gastric feeding to the primary clinician should be established before changing from a jejunal feeding tube to a feeding gastrostomy tube. Techniques discussed in the nasoenteric feeding section of this chapter (metoclopramide and right decubitus position) may in selected cases coax gastrostomy-placed feeding tubes into the small bowel. Gastric decompression tubes are either clamped or put at continuous drainage.[85]

Verification of tube placement can be made radiographically using a fluoroscope or with a small amount of contrast material passed into the tube (Fig. 41-27 and Fig. 41-28). In the latter scenario, a catheter-tip syringe is used to introduce water-soluble contrast solution (e.g., diatrizoate meglumine-diatrizoate sodium [Gastrografin]) into the tube. *Barium is contraindicated.* Generally 20 to 30 mL of water-soluble solution is adequate for documenting the intraluminal tube position. A supine abdominal film should be taken within 1 to 2 minutes of dye instillation to optimize gut visualization. Since the film must be quickly obtained, it is easiest to perform the injection in the radiology suite, followed by an x-ray. *If the contrast material does not flow freely into the tube, the procedure should be terminated immediately and the position of the tube questioned.* With proper positioning, contrast will outline the gut containing the tube (e.g., stomach with gastrostomy tube). An irregular or rounded blotch with wispy edges or streamers suggests peritoneal leakage. In questionable cases, dye injection can be performed under fluoroscopy.

It should be noted that there is no universally agreed upon standard of care with regard to performing a confirmatory contrast study for all easily replaced feeding tubes. Some clinicians verify position routinely with a contrast radiograph while others use the clinical criteria outlined earlier. The editors advocate a cautious and conservative approach. The routine use of post placement contrast radiography to confirm proper placement should be considered when the tube tract is immature (i.e., <3 months duration). Passage of the replacement catheter has been difficult, aspiration of intestinal contents is not possible post tube placement, or the patient is unable to communicate symptoms that might occur with tube misplacement.

Transabdominal Low-Profile Feeding Tube Replacement

Low-profile feeding tubes are almost flush with the skin, held in place with a preformed plastic external bolster. Especially popular in children and active adults who require intermittent feedings, they are only slightly more difficult to remove and replace than Foley catheters. The tubes come in two main types, those with an internal balloon and those with an internal flange.

Tubes with a balloon can be deflated with a syringe for removal. Seat the syringe well and do not let fluid flow back into

Figure 41-27 Contrast study demonstrating correct placement of a feeding tube. Note the outline of the gastric rugae and the characteristic mucosal folds of the small intestine.

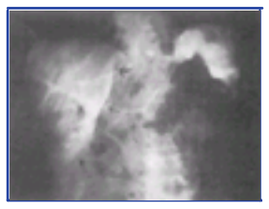

Figure 41-28 Although this feeding tube seemed to be easily replaced in an uncommunicative nursing home patient, gastric contents could not be aspirated; therefore, a contrast study was performed. Note the free flow of contrast material throughout the abdomen, especially outlining the liver *(arrows)*. This film indicates placement of the feeding tube into the peritoneum. Placing food through this tube could be disastrous.

the balloon once it is withdrawn into the syringe, or the diameter of the balloon will not be at a minimum as it is withdrawn through the stoma. If no fluid is obtained at all, the balloon may already be deflated, and a trial of gentle removal may be warranted before attempting balloon puncture techniques. If the stem appears to get stuck as you are withdrawing it through the stoma, a second attempt to withdraw fluid may obtain a few more drops and free it up. In children especially, make sure the head is resting and not flexed. A flexed neck encourages tight abdominal muscles which can squeeze the stoma around the stem. If the balloon will not deflate at all, see the instructions above for balloon puncturing techniques.

Tubes with a flange should be removed, ideally, with the obturator that comes with the tube. While this obturator is usually unavailable, many patients with this kind of tube will have a replacement of the same size and type and the obturator from the new tube can be used to remove the old one. The obturator should be well seated to distend the flange and minimize its diameter during removal. An obturator from another type or length tube may also work, but the length must be as long as or longer than the original and the seating becomes a matter of continuous pressure. A blunted pediatric chest tube inserter, a sound, or an endotracheal tube stylet may also work. Many low-profile feeding tubes are made of silicone, which is softer and more fragile than the polyurethane of the de Pezzar catheters, and a lesser degree of pressure may be appropriate.

A bolster regulates the depth of Foley catheters and most other gastrostomy tubes, but the depth of each low-profile tube is fixed. A tube of appropriate depth and diameter needs to be selected for the ostomy. All low-profile gastrostomy tube companies make a stoma measuring device, some packaged with every tube and some as a separate item. The stoma measuring device is inserted through the stoma and has a retaining hook or balloon on the end and markers on the portion protruding from the abdomen. By counting the numbers of markers, the clinician determines whether a long, medium, or short tube is appropriate. When a tube of a certain type and depth had been functioning well until it cracked, clogged, or was lost, it may be reasonable to replace it with another tube of the same type and depth without any measurements.

Once the appropriate tube is selected, the stoma should be lubricated and the tube can be passed through it. Tubes with a flange must first be distended with the accompanying obturator. Fill the balloon of balloon-stabilized tubes with saline (not air) at the end of the procedure.

Complications

If the ostomy is not a mucocutaneous type, it will close rapidly without a stenting tube. Often the stoma begins to contract within hours of feeding tube removal. The clinician may be presented with a very narrow ostomy and a tract that is difficult to identify or thread. A sound or blunt stylet can be passed down the tract more easily than a tube. This procedure can identify the opening and direction of the tract for easier tube passage. When a guidewire passes easily down a narrow tract but the needed feeding tube does not, it may be possible to dilate the tract with dilators or a dilation catheter. [88] Viscus puncture, viscus abdominal wall separation, and false tract creation with subsequent tube misplacement are risks of dilation procedures. Such procedures should generally be left to the surgeon. If tube replacement will be delayed, maintain the narrow tract with the largest available easily placed stent, usually a Foley catheter. Always secure the stent against internal migration.

The position of the gastrostomy tube should be checked by air insufflation and aspiration of gastric fluid, as is done with nasoenteric tubes. It is wise to document the results of this testing in the medical record. Air should enter the stomach without resistance and should produce immediate borborygmi. Gastric fluid should return with aspiration. It may be necessary to insert a small volume of water to get good return. Water pooling in the soft tissue may be aspirated back through a misplaced catheter. Good tube placement is indicated when more fluid returns with aspiration than was originally placed into the catheter. If replacement of the gastrostomy or jejunal tube required the overcoming of any resistive force or if either the air or the aspiration test yields uncertain results, a radiographic study with contrast should be performed. Peritoneal infusion of feeding solution can be fatal.

Complications of gastrostomy include wound infections around the catheter, performance of an unnecessary laparotomy for suspected leakage, gastrocolic fistula, pneumatosis cystoides intestinalis, bowel obstruction, peritonitis, and hemorrhage. [79] Jejunostomies can cause most of these complications, as well as other types of fistulas and small bowel obstruction from adhesions or volvulus around the jejunostomy site. [59] [73] The most common complications of gastrostomy and gastroenterostomy are local skin erosions from leakage, wound infections, hemorrhage, and tube dislodgment. [51] Peritonitis and aspiration are the most critical complications of gastrostomy feedings. [51] [73] Jejunostomies are less prone to stomal leakage and cause less nausea, vomiting, bloating, and aspiration than do gastrostomies. [49] [53]

Dislodgment of gastrostomy and jejunostomy tubes is most common in the 2 weeks after ostomy creation. [89] Extrusion of the gastrostomy tube is usually caused by excessive tension applied to the tube. Only gentle contact of the

gastric and abdominal walls is desirable. Uncooperative patients should be restrained, and mittens are often particularly helpful. Sutures and large mushrooms or balloons do not prevent purposeful removal of the gastrostomy tube by the uncooperative patient.

A small amount of drainage is to be expected at the tube entry site. Local leaks of gastric juices may macerate and irritate the skin, predispose to local infections and abscesses, and encourage the development of small granulomas. [49] [75] Granulomas are particularly common in children. They can be treated with silver nitrate at the time of dressing changes. Any dressing used around the entry site of an enteral nutrition tube should absorb fluid and not encourage persistent moisture. [78] An unusually large stoma may promote a leak. Although insertion of a larger tube or firmer traction on the tube may be transiently effective, these measures often result in further stomal enlargement. Rigid gastrostomies promote leakage by widening the stoma as they pivot. Insertion of a soft, pliant feeding tube through the widened stoma is often easy and allows later contraction of the stoma. [1] If these techniques are ineffective, temporary removal of the feeding tube may allow the stoma to shrink. Large amounts of drainage around the stoma site may occur with high residual volumes. [84] The residual should be checked and feedings withheld until residuals are less than 100 mL. Feeding residual should be checked every 4 hours when a patient is on continuous drip feeding. [87]

Pneumoperitoneum after percutaneous gastrostomy is neither unusual nor dangerous. Benign pneumoperitoneum may be present as long as 5 weeks after percutaneous endoscopic gastrostomy. [79] [84] Pneumatosis cystoides intestinalis can occur through the defect in the bowel wall created for the enterostomy tube. Although often clinically insignificant, its occurrence suggests air under pressure in the small bowel. NG suction and diet change generally permit resolution of the problem. Catheter or feeding tube removal is usually not required. [80]

Clinically significant pulmonary aspiration can occur with gastrostomy feeding. Methods of checking for silent pulmonary aspiration include checking tracheal aspirates with a glucose oxidant reagent strip or placing methylene blue in the formula and monitoring tracheal aspirate for pigmentation. [48] [54] [90]

A Foley balloon accidentally inflated in the small bowel or esophagus can lead to perforation or obstruction. [91] Careful inflation of the balloon soon after it has entered the stomach prevents viscus perforation. A gastrostomy tube may migrate in the stomach and obstruct the gastric outlet. This complication manifests itself clinically with vomiting and high residuals of feeding solution. Volvulus and jaundice may also occur as a result of balloon migration. This problem can be alleviated by gently pulling back the tube. If the balloon of a Foley catheter has migrated into the small bowel, deflation of the balloon before pulling it back further reduces the risk of intussusception. An outer crossbar will prevent distal migration. [77] [84]

Gastrocolic fistula usually manifests itself as copious diarrhea. Once it is confirmed, treatment consists of removal of the gastrostomy tube. Later creation of a gastrostomy in a different location may be possible. The patient may require hospital admission for nutritional support and monitoring of fluid and electrolyte status.

An external bolster that is snugged down too tightly may result in a short stoma and embedding of the internal bolster into the abdominal wall. An abscess may result.

Overly tight external bolsters should be loosened. The correct position is 1 cm from the external abdomen.

Dacron cuffs can serve as the nidus for an abdominal wall abscess. Generally the cuff cannot be removed independently of the tube. A replacement tube without the cuff can be inserted, if one is careful not to dissect the tube into the wall of the abscess. Extensive abscesses may require incision and drainage.

CLOGGED FEEDING TUBES

Clogging is a problem common to all feeding tubes. Although it may only be a temporary solution, it is prudent to attempt to unclog a tube before it is replaced, especially if the tube has a complex placement or the clinician is unsure of how the tube is secured internally. However, whenever feasible, clogged tubes should be replaced with a new tube. Large gastrostomy tubes are the least likely to clog. Gastrostomy tubes at least 28 Fr in size can tolerate home-blenderized foods and viscous feeding solutions. Isosmotic feeding solutions are tolerated by fairly narrow tubes and cost one sixth of what elemental feedings cost. Isosmotic feedings will clog needle catheters.[50] When tube lumina are 14 Fr or smaller, all pills and the contents of all capsules should be dissolved in water to prevent tube obstruction. [78]

Acid precipitation of feeding formulas is an important factor in the occlusion of gastrostomy and NG feeding tubes. Sodium and calcium caseinate and soy protein molecules are most soluble at a pH of 6.3 to 6.6 and least soluble at a pH of approximately 4.7. [92] They are insoluble in nonpolar organic solvents. A study of 14 feeding solutions showed Pulmocare, Ensure Plus, and Osmolite to be the most clog-prone on exposure to acidic solutions; Citrotein had the least tendency to clog. [93]

Kinking is a frequent cause of tube blockage during the immediate post-reinsertion period. Withdrawing the tube a few centimeters usually relieves the kink and obstruction. A persistently recurring kink requires tube removal and insertion of a fresh tube.

Accumulated feeding solution or medication precipitates are very difficult to clean or remove. Milking a pliant tube backward may remove some of the cheesy precipitates. Guidewires or stylets may clear the proximal portion of a clogged tube lumen but are unsafe to use in subcutaneous (SQ) areas of the lumen because they can puncture the tube and injure the patient or create a tube leak.

Fogarty arterial embolectomy catheters can be used to unclog jejunostomy [94] and gastrostomy tubes. The soft tip of the Fogarty catheter is inserted into the feeding tube and advanced while the insertion distance is monitored to avoid penetrating farther than the length of the feeding tube. The allowable length of insertion should be premeasured. A No. 4 embolectomy catheter is suitable for a 10 or 12 Fr tube, whereas a No. 5 catheter should be used in 14 Fr feeding tubes. When the catheter meets an obstruction, the balloon can be inflated, which usually opens the obstruction sufficiently that catheter passage can continue. Once the Fogarty has been manipulated to just proximal to the internal feeding opening, it is withdrawn while the balloon is intermittently inflated and deflated gently. The catheter should not be withdrawn while inflated, because it and the feeding tube tend to move as a unit.

The procedure may need to be repeated several times. Contrast injection to confirm tube position and integrity should be performed after declogging is completed. [94]

Irrigation with carbonated beverages and high-pressure irrigation with small-volume syringes have also been recommended as techniques for unclogging feeding tubes. Although irrigation seems like a straightforward and simple solution, these techniques are generally ineffective; furthermore, the possibility exists for dangerous tube ruptures with internal leakage. Broviac catheters are especially prone to tube aneurysms that can rupture under pressure. [75] Tubes unclogged by forceful irrigation or by deep luminal probing should be radiographed after injection of contrast to check tube integrity.

Enzymatic declogging of feeding tubes may sometimes be effective. Most enzymatic preparations are insoluble in an acidic environment and require mild alkalinity for effective action. Such precipitates can add to the obstructing material. One study suggested that two enzyme preparations can soften clog consistency sufficiently to enhance tube clearing with insufflation. [92] A crushed chymotrypsin tablet or two papain tablets (effervescent Allergan) can be dissolved in 2 mL of distilled water and irrigated into the drained feeding tube. A 12.5-cm catheter may make such irrigation more effective. The feeding tubes should be closed or clamped and left for 1 to 4 hours. One study attempted tube insufflation only at 4 hours, but clog changes were noted after one-half hour. [92] Insufflation of air with a 50-mL syringe also may clear the tube.

Most clogged tubes should be replaced. Tubes that have been unclogged by using force should be radiographed after water-soluble contrast injection (see section on transabdominal feeding tube replacement) to check for internal leaks. Regular tap water irrigations and selection of a feeding solution appropriate for the diameter of the feeding tube are vital to prevent tube clogging.

CONCLUSION

Emergency clinicians must commonly manage the patient who requires NG tube placement or management of a dislodged or malfunctioning feeding tube. Although placement of these tubes is generally straightforward in the cooperative patient, many patients requiring such tubes will be uncooperative or have an altered sensorium. Therefore, careful attention to the details recommended for these procedures is encouraged, as the complications associated with a misplaced tube can be disastrous.

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Chapter 42 - Balloon Tamponade of Gastroesophageal Varices

Edward A. Panacek

More than 2% of all hospital admissions in the United States and more than 5% of admissions from emergency departments (EDs) are related to acute gastrointestinal (GI) bleeding. In the vast majority of these patients, the bleeding source is in the upper GI tract. Bleeding from varices, both esophageal and gastric, is the third most common cause of upper GI bleeding, accounting for about 20% of cases.[14] Variceal hemorrhage is the most dramatic and life-threatening complication of portal hypertension. After one bleeding episode, the risk of a second is 70%, accounting for up to 50% of all deaths in patients with cirrhosis. [29] The risk of mortality from each bleeding episode ranges from 20% to 84% and depends on the etiology of the portal hypertension, the hepatic functional reserve of the patient, and the duration of hemorrhage prior to presentation.[25] Individuals with noncirrhotic portal hypertension and intact hepatic function generally tolerate the hemorrhage better than those patients with cirrhosis and advanced hepatic dysfunction. The greatest risk of death is in the first few days after the onset of variceal hemorrhage. [25] Therefore, prompt and effective therapy during this initial period is essential to maximize the patient's chance of survival.

BACKGROUND

The use of balloons to control variceal hemorrhage dates back to the 1930s, although the balloons were initially filled with water rather than air. [30] In 1950, Sengstaken and Blakemore first described the technique of a double-balloon tamponade system similar to what is currently used. During the subsequent 30 years, the Sengstaken-Blakemore tube underwent a number of modifications or refinements by Linton, Nachlas, Boyce, and Edlich. [3] [4] [11] These modifications generally carried the name of their originator and altered the size or shape of the respective balloons or added aspiration ports in the stomach or the esophagus to allow monitoring of ongoing bleeding.

Currently there are two types of gastroesophageal balloon tamponade (GEBT) tubes available: the three-lumen Sengstaken-Blakemore tube (gastric balloon, esophageal balloon, and gastric aspiration) and the four-lumen Minnesota tube (which adds an esophageal aspiration port). [2] [11] Figure 42-1 and Figure 42-2 show these two types of tubes. Institutions generally stock a single type of tube. Most hospitals also generally only stock adult sizes, although pediatric tamponade tubes do exist.[18] However, since the advent of endoscopy, such devices are rarely used. It would not be considered standard care that all emergency clinicians have experience, or expertise with there tubes. Although GEBT tubes are used less frequently today than previously, their use can still be temporizing or life-saving. Although these tubes are not usually placed in the ED, some emergency clinicians will encounter a GEBT tube during their professional career. This chapter attempts to make emergency clinicians familiar with their structure, placement, function, and management. This may be especially helpful in more remote settings, where an invasive gastroenterologist is not readily available.

INDICATIONS AND CONTRAINDICATIONS

A nasogastric (NG) tube should be placed in all patients in whom GI bleeding is suspected or known, regardless of the presumed source or degree of the bleeding. There is no evidence that passage of an NG tube in a patient with esophageal varices results in variceal trauma or increases bleeding. [21] Lavage should be carried out, ideally with the patient in the left lateral decubitus position, to determine if the upper intestinal bleeding is limited in extent (i.e., gastric aspiration becomes clear with about 1 to 2 L of fluid). Lavage is generally performed to decompress the stomach and minimize the risk for further emesis. Removal of large clots also allows gastric wall contraction in those cases where the upper GI bleed has a gastric origin (see Chapter 41). Ongoing lavage has little effect on gastric hemorrhage, and extended lavage with cold solutions does little for hemostasis and may induce hypothermia.

When the gastric aspirate does not clear with lavage, more aggressive therapeutic approaches must be considered. The approach to patients with acute hemorrhage from esophageal varices is substantially different from that used with other causes of upper GI bleeding. [21] The presence of advanced hepatic disease, evidence of portal hypertension, or prior known variceal hemorrhage greatly increases the likelihood of a variceal source for the bleeding. [14] However, in 25% to 50% of such cases, upper GI bleeding can still be due to nonvariceal sources. [10] For this reason, emergent endoscopy is indicated in such patients whenever available. [21] Endoscopic sclerotherapy and other techniques are the most effective therapy to stop active bleeding from varices and to stabilize varices that have recently bled. [27] [28]

The other early therapeutic option for variceal hemorrhage is IV octreotide, or somatostatin, potent nonselective vasoconstrictors. They can be used in conjunction with sclerotherapy or as the sole therapy when sclerotherapy is unsuccessful or unavailable. These drugs must be used with caution in patients with coronary artery or other vascular disease because of the risk of adverse ischemic effects. [26]

When sclerotherapy is unavailable or unsuccessful and vasoconstrictor therapy is not adequate, balloon tamponade is the next therapeutic option. Balloon therapy is less successful than sclerotherapy but can control bleeding acutely in up to 80% of cases. [6] [12] [20] The role of balloon tamponade in these other therapies is depicted in Figure 42-3 .

Some authors have advocated the use of esophageal balloon tamponade based upon transfusion requirements or ongoing hemorrhage exceeding a certain amount (usually 2000 mL of blood within a 24-hour period). [14] Other recommendations have focused on endoscopic findings, including active variceal bleeding that cannot be controlled with sclerotherapy or traumatic longitudinal tears of the esophagogastric junction (Mallory-Weiss tear), with active persistent bleeding. [20] However, these indications are generally more relevant for the inpatient setting. In the absence of endoscopy, the indication in the ED would be a patient with known portal

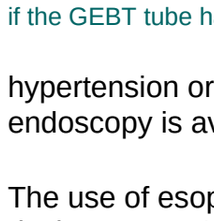

Figure 42-1 *A,* Photograph of the three-lumen Sengstaken-Blakemore tube with the balloons deflated. *B,* Diagram of GEBT tube with nasogastric tube attached. This is not required if the GEBT tube has esophageal aspiration ports.

hypertension or prior variceal hemorrhage who has substantial ongoing upper GI bleeding that does not clear with gastric lavage and vasoconstrictor therapy. When endoscopy is available, the indication for a GEBT tube is substantial ongoing variceal hemorrhage that cannot be controlled with endoscopic interventions. [20]

The use of esophageal balloon tamponade tubes is associated with a number of serious and potentially lethal complications. Although potentially lifesaving, these devices are not always easy or safe to use; but, their use is supported in critical situations. The overall complication rate is much higher than for most other procedures that emergency clinicians perform. [22] Therefore, clear indications for GEBT tube use and a complete understanding of the instructions for their placement are required. GEBT tubes are best placed by the individual most knowledgeable about their use.

GEBT tubes are meant only for temporary bleeding control. [20] Although most studies have shown that they provide initial hemostasis in up to 80% of patients with bleeding from esophageal varices, they have not measurably affected the long-term mortality of these patients. [6] Their success rate improves when used in conjunction with vasoconstrictor therapy. [23] However, these therapies often do not control hemorrhage definitively, and arrangements for more definitive endoscopic procedures should be initiated. [12] [20]

EQUIPMENT

Most hospitals stock only one type of GEBT tube. Ideally, the tubes are stocked in the ED itself or are readily available when needed. Multiple companies make these tubes, and each

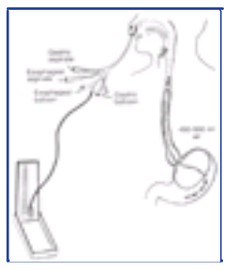

Figure 42-2 Diagrammatic representation of the four-lumen Minnesota tube with the balloons properly positioned and inflated. Note manometer attached to esophageal balloon port to measure pressures foam rubber cuff at nose as a fixation device, and the esophageal aspiration lumen.

company may include some of the items listed below. The equipment required for this procedure is as follows:

1. GEBT tube
2. Traction device or setup, including weights (discussed later)
3. Manual manometer or sphygmomanometer
4. Y tube connector, if not already part of the tamponade balloon ports
5. Vacuum suction device
6. Connectors for the suction tubing
7. Plastic tubing to connect to suction
8. Soft restraints
9. Topical anesthetic (spray and jelly)
10. Water-soluble lubricating jelly
11. 4 tube clamps (see text)
12. Large (e.g., 50 mL) catheter tip irrigating syringe
13. Surgical scissors for emergency balloon decompression

Figure 42-3 Algorithm for management of acute variceal hemorrhage, showing the role for balloon tamponade. After endoscopy banding and/or lauage have been used. Note that balloon tamponade can also be used after failed sclerotherapy as a temporizing measure pending emergency surgery, or between repeat attempts at sclerotherapy.

Optional: Standard NG Tube

There are multiple ways of providing a traction setup for the tube, and individual hospitals may have specific policies. [2] Tubes can be taped to the mouth guard of a football helmet, although currently it is extremely unusual to see that technique used. Even in those institutions that try to keep a helmet available, it can rarely be located when needed. More often traction is maintained with a cube of foam rubber that is generally included in the package with the tube itself, as shown in Figure 42-2 . The tube is inserted between the two halves of the foam, and the foam "cuff" is placed against the nose and taped.

The more common way of providing traction, and generally the way considered most effective, is through a pulley apparatus similar to the system used for orthopedic traction on lower extremity fractures.[15] It is a bit more cumbersome, and there are concerns regarding its use in disoriented or agitated patients, but it can effectively and consistently maintain the desired 0.45 to 0.91 kg (1 to 2 lb) of traction. The usual trapeze bar apparatus is used, but this requires two pulleys: one over the patient and one at the end of the bed. It also requires a longer rope (about 3 m [10 feet]), as well as the suspension hook and 0.23- to 0.45-kg (0.5- to 1.0-lb) weights.

Clamps are needed for the process of inflating the balloons, checking pressures, and maintaining balloon inflation. Never use bare standard hemostats for this purpose, as the serrations on the clamp jaws can puncture the rubber tubing. Specific tube clamps are best, if available (e.g., hosecock clamps). Alternatively, large surgical clamps or large hemostats can be modified by covering the clamping surfaces with sections of plastic or rubber tubing or multiple layers of tape.

If the GEBT tube does not have an esophageal aspiration lumen, secretion can be removed by attaching a standard NG tube along side the GEBT tube (Fig. 42-1B). This will decrease the risk of aspiration. This is not required if the GEBT has esophaged suction parts.

PROCEDURE AND TECHNIQUE

Patients requiring GEBT tubes are critically ill and must be closely monitored within the ED or in an intensive care unit. In the best of circumstances, these tubes can be difficult to place. However, these patients are often uncooperative and sometimes even combative due to encephalopathy. Control of the patient is imperative. Soft restraints should be used routinely, and sedating medications considered in most patients. If the patient is not awake, alert, and fully able to protect the airway, endotracheal intubation should precede placement of the GEBT tube (see Chapter 4). These patients are at extremely high risk for regurgitation and aspiration, and the threshold for endotracheal intubation should be lower than usual. [19]

The procedure itself is unfamiliar to most emergency clinicians and requires a number of items of equipment. Each of these should be checked prior to beginning the procedure. In particular, the balloons on the tamponade tube must be checked for patency and lack of leaks. If there is any question, the balloons should be submerged beneath water for checking. During testing, inflation of the gastric balloon is done in progressive steps (generally in 100-mL increments), up to the maximum recommended volume (generally 500 mL), while the pressure is monitored with the manometer. The pressure within the gastric balloon should not increase by more than 15 mm Hg with each progressive administration of 100 mL of air. The operator should note the pressure at full inflation of the gastric balloon. If the optimal NG tube is to be used, it is now tied along the course of the GEBT tube with sulk sutures. The tip of the NG tube should be 3–4 cm proximal to the esophageal balloon (Fig. 42-1B). The esophageal balloon is simply checked for patency. The balloons are then deflated and coated with water-soluble lubricating jelly, preferably with a topical anesthetic.

The patient is positioned for the procedure by having the head of the bed elevated to about 45 degrees, if possible. If the patient is unable to tolerate that position, the left lateral decubitus position is also acceptable. The posterior pharynx and nostrils are anesthetized with a topical anesthetic. The patient should already have undergone gastric lavage with a standard NG tube. At this point the stomach is maximally evacuated (to decrease regurgitation), and the NG tube is removed.

After suctioning all air from the gastric and esophageal balloons and ensuring that they are maximally collapsed, each of the balloon ports is clamped, or the plastic plugs (if provided with the tube) are inserted into the lumens. This maneuver maintains deflation of the balloons during placement. In awake and alert patients, passage through the nostril is an option, but passage through the mouth is preferred, especially in tracheally intubated patients. The alert patient can assist the process by taking sips of water through a straw. The tube is then passed to at least the 50-cm mark, or the maximum depth allowed by the length of the tube, in the same manner as a standard NG tube. Suction is then applied to the gastric and esophageal aspiration lumens to minimize chances of further regurgitation or aspiration. Aspiration of gastric juice or fresh blood from the gastric lumen provides further evidence for proper location of the tube. However, its position must be confirmed radiographically.

Some authors recommend initial inflation of the gastric balloon with 50 mL of air to assist with radiographic visualization to ensure that the gastric balloon is below the diaphragm.[20] However, this is not routinely necessary, and the position of the distal end of the tube is usually obvious on radiograph. Absolute confidence regarding tube location is extremely important to reduce the risk of esophageal perforation caused by inflation of a misplaced balloon. Once gastric positioning of the distal balloon is ensured, the tube clamps (and plastic plugs if used) are removed from the gastric balloon inflation ports.

The pressure monitoring outlet for the gastric balloon lumen is connected to a manometer, similar to Figure 42-2 and Figure 42-4 . Increments (generally 100 mL) of air are then introduced through the gastric balloon inflation lumen until the recommended total volume (generally 450 to 500 mL) fills the gastric balloon. Only air, never water or any liquid, is used to inflate the balloons. As the air is introduced into the gastric balloon, the intragastric balloon pressure is monitored. If the gastric balloon pressure at any step is 15 mm Hg greater than the intragastric balloon pressure at an identical volume noted before intubation (i.e., during testing), the gastric balloon may be located within the esophagus, and further inflation of the balloon could result in overdistention and rupture of the esophagus. If an elevated intragastric balloon pressure is recognized, the balloon should be deflated, advanced, and reintroduced into the stomach. A repeat radiograph is then

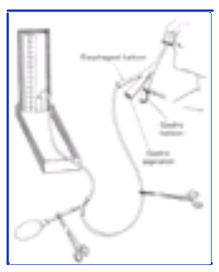

Figure 42-4 Esophageal balloon pressure measurement in the Sengstaken-Blakemore tube using a manometer. To inflate the esophageal balloon, the operator inserts one end of a Y connector to the esophageal balloon port, one end to a manometer, and one end to a bulb. At this point, no clamps are closed. The bulb is compressed until the manometer reads the desired pressure, and then the esophageal balloon port is clamped. Periodic readings to check the esophageal balloon pressure are subsequently taken by clamping the bulb port and unclamping the esophageal balloon port. The pressure in the esophageal balloon may vary with respirations and spasms, but the baseline pressure should not exceed 45 mm Hg. Most bleeding is controlled with pressures well below the maximum safe pressure. Note that standard toothed clamps are to be avoided (see text).

obtained to confirm proper positioning before restarting the inflation procedure.

When the gastric balloon, positioned correctly in the stomach, has been inflated with the recommended full volume (generally 450 to 500 mL) of air, the air inlet and pressure monitoring outlet of the gastric balloon are each clamped. The tube is then pulled back gently until the resistance of the diaphragm is firmly felt. With a small amount of tension on the tube, the proximal end is secured using a traction device. The tube kit generally includes a sponge rubber cuff, which can then be fixed at the nostril and taped, if that approach is used. If the tube has been passed orally, an external pulley traction device is preferred. Once the tube is secured in place, the gastric aspiration port is attached to high intermittent suction. A final check should be made by irrigating the tube with water under auscultation. Lavage the gastric port until clear.[15] If there is any question regarding correct tube position, obtain a repeat radiograph before proceeding.

If blood is detected continually in the gastric aspiration port (or in the esophageal aspiration port on a 4-lumen tube) despite lavage, the esophageal balloon should be inflated to the pressure recommended in the accompanying instructions (generally 30 to 45 mm Hg). Inflation of the esophageal balloon should be monitored continuously by a manometer attached to the esophageal balloon monitoring outlet. The esophageal balloon pressure should be maintained at the lowest level that will stop bleeding from each of the aspiration suction ports. In addition, the esophageal balloon pressure should not exceed the maximum listed in the instructions (generally 45 mm Hg). Once the balloon has been inflated to the desired amount, the port for the esophageal balloon is clamped, and periodic readings are taken.

If bleeding continues from the gastric aspiration port after full inflation of the gastric and esophageal balloons, it usually originates from a gastric rather than an esophageal varix. In this case, external traction on the tube should be increased. If a foam rubber block is used, the tube should be pulled to a more taut position and refixed to the nasal cuff at the point where the tube emerges from the nose. If the tube was passed through the mouth, and a trapeze-bar pulley system was used, adding traction is easy. An additional 0.45 to 0.91 kg (1 to 2 lb) of weight can be applied to the pulley system, in progressive steps (maximum of 1.1 kg [2.5 lb]). [15] The pulley system should be attached to the GEBT tube through use of a nonoccluding knot (e.g., bowline knot) passed around the area of the tube where all the respective ports separate. Tightening the knot directly onto the tube itself can occlude the ports.

Direct pressure from the tube can cause ulceration of mucosal surfaces within a matter of a few hours. Therefore, there should be frequent examinations to ensure that the tube is not placing excessive pressure on a given mucosal surface. If one is using an external pulley traction device, the angle of the tube, as it exits the mouth, is adjusted to minimize any such pressure by altering the location of the pulley. If the tube is passed through the mouth, precautions are also needed to preclude patient biting of the tube. A rubber or plastic mouthpiece should be placed in dentulous patients. Suction of the aspiration ports is then adjusted. The gastric

port should have intermittent 60- to 120-mm Hg suction, and the esophageal port (for 4-lumen tubes) should be attached to 120- to 200-mm Hg continuous suction. [15]

After bleeding has been controlled by the tamponade, the pressure in the esophageal balloon is generally reduced by 5 mm Hg every 3 hours (or as specified in product instructions), until an intraesophageal balloon pressure of 25 mm Hg (or as specified in instructions) is achieved without ongoing bleeding. Note that continual elevated balloon pressures (>30 mm Hg) on the esophagus for long periods of time can result in mucosal ischemia and may induce esophageal necrosis. [15] Therefore, it is recommended that periodic deflation of the esophageal balloon be undertaken for approximately 5 minutes every 6 hours. If bleeding can be controlled with an intraesophageal balloon pressure of 25 mm Hg, this pressure is generally maintained for the next 12 to 24 hours. [15] [20] The pressure in the esophageal balloon can transiently vary with respiration and esophageal spasm. This may result in intermittent increases in the measured pressure of 30 mm Hg above baseline, although this should only be for transient periods. If it remains high, air must be removed until the pressures are acceptable.

If a 3-lumen GEBT tube has been used, it will not have an esophageal aspiration port. Because the amount of oropharyngeal and esophageal secretions can exceed 1500 mL/day, additional suction proximal to the esophageal balloon must be provided. [15] This can be done with a standard 14 to 16 Fr NG tube passed to a position measured or calculated to be just above the esophageal balloon. This should be placed even if the esophageal balloon is not maintained in an inflated position, because an inflated gastric balloon will also interfere with the ability to swallow or pass secretions.

Once satisfactory positioning of the GEBT tube has been confirmed, the tube is generally then not disturbed for some 24 hours, unless necessary due to complications.[15] During this time, adjunctive therapy is ongoing and additional evaluation instituted. [20] These tubes are very uncomfortable, and patients should be provided with analgesics and sedation. In addition, soft restraints are needed on the arms to prevent the patient from dislodging the tube. If the bleeding does not remain controlled, other therapeutic interventions must be considered. These include endoscopic interventions with sclerotherapy, as well as emergency surgery. If these are not available, patient transfer may be necessary. [12]

COMPLICATIONS

Complications associated with the use of GEBT tubes are frequent and often very serious. [22] Mortality rates are high with patients in need of these devices. Table 42-1 lists both major and minor complications. Major complications have been reported to occur in 8% to 16% of patients. [2] Mortality directly related to use of these tubes is generally reported to be 3%. [15] [22] However, one study reported the GEBT tube directly caused death in 22% of the patients in which it was used. [7] This must be considered in the context that variceal hemorrhage itself carries a 20% to 80% overall mortality. [20] Therefore, use of a GEBT tube is sometimes viewed as a desperate measure for a desperate disease process. GEBT tubes can be temporizing or even life-saving, but the high associated complication rate requires an individual risk-benefit analysis for each patient in whom it is being considered.

Aspiration pneumonitis is probably the most frequent major complication. [22] This can result from aspiration of oral secretions, gastric contents, or, most commonly, blood. [2] The volume of aspiration can be substantial, and associated deaths

TABLE 42-1 -- Complications of Esophageal Balloon Tamponade Therapy

Complications	Common	Uncommon
Major	Aspiration pneumonia	Esophageal perforation
	Asphyxiation	Duodenal rupture
	Esophageal necrosis	Tracheobronchial rupture
		Periesophageal abscess
		Mediastinitis
Minor	Gastroesophageal ulceration	Epistaxis
		Pharyngeal erosions
	Regurgitation	Pressure necrosis of tongue
	Chest discomfort	
	Back pain	Hiccups
	Pressure necrosis of nose or lip	

have been reported. [2] [7] The likelihood of this complication can be decreased by evacuating the stomach prior to placement of the GEBT tube and having a low threshold for tracheal intubation to protect the airway. [19]

Asphyxia owing to airway obstruction has been reported to occur with dislodgment of the tube such that the esophageal balloon migrates into the oropharynx. [2] [5] This is more likely when the tube is passed through the nares such that the inflated esophageal balloon cannot pass through the nasopharynx, and it is one of the reasons that oral placement is preferred. Tube migration can be prevented by maintaining full inflation of the gastric balloon through periodic monitoring of pressures and radiographic reconfirmation. Of course, airway obstruction can be completely prevented by prophylactic tracheal intubation. If the tube suddenly migrates, resulting in an airway obstruction in a nontracheally intubated patient, the balloons must be immediately deflated and the tube extracted. This can be achieved most quickly by cutting across all of the tube lumens just distal to the bifurcation points. This will immediately vent all of the balloons, and the entire tube can be extracted. For this reason, it is recommended that surgical scissors be kept available at the patient's bedside whenever a GEBT tube is in place.

The other relatively common major complication is esophageal perforation or rupture. [7] [8] This occurs with overinflation of a misplaced gastric balloon and can be prevented through careful employment of the placement steps already outlined. However, this can also occur as a result of esophageal mucosal necrosis caused by excessive or prolonged pressure in the esophageal balloon. Crerar-Gilbert reported a case of massive esophageal rupture with the use of balloon tamponade and associated sclerotherapy. [9] The treatment is immediate removal of the GEBT and initiation of diagnostic studies (e.g., contrast swallow) and broad-spectrum antibiotics (for potential mediastinitis). Again, risk for this complication can be decreased by periodic deflation of the balloons at 6-hour intervals and limiting the amount of pressure in the esophageal balloon to the minimum amount necessary to control bleeding. This problem is also more common when the balloons are left inflated for more than 24 hours. [15]

Common minor complications include pain, discomfort, and local pressure effects of gastric or esophageal erosions or mucosal ulcers. [6] [22] These latter can be minimized by frequently checking and optimizing tube position to minimize pressure on the nasal or oral mucosa, the tongue, and other structures. Although less common, other complications can occur; these are also listed in Table 42-1 . [1] [13] [16] [17] [22]

INTERPRETATION

GEBT tubes are used to temporarily control bleeding varices. This is assessed by monitoring the rate of blood aspirated from the gastric and esophageal ports after tube placement. It is not uncommon for the GEBT tube to fail to control the hemorrhage.[14] [15] [20] When significant bleeding continues, consider correctable causes, which include malpositioned balloons, inadequate balloon pressures, and misdiagnosis of the site of bleeding (e.g., a duodenal ulcer instead of variceal source).[7] [15] When these have been addressed but bleeding continues, other therapeutic options must be considered, including sclerotherapy, angiographic embolization, and surgery (see Fig. 42-3).[12] [20]

CONCLUSION

Gastroesophageal balloon tamponade is an uncommon procedure in general, and particularly for emergency clinicians. Some emergency clinicians may not have performed this procedure previously. However, the procedure can temporarily control an exsanguinating hemorrhage from gastric or esophageal varices in about 80% of patients when other options are unsuccessful or unavailable. As such, the emergency clinician may periodically be called upon to place a balloon tamponade tube. Because of the high incidence of associated serious complications, the procedure should be used in carefully selected patients in whom the potential benefit is favorable, given the risk of exsanguination.

Each hospital is unlikely to stock more than one tube type, and this may not be the type most familiar to the clinician. However, currently most balloon tamponade tubes are fairly similar and are packaged with a complete set of instructions. These instructions must be carefully reviewed before tube placement. There can be great difficulty locating these tubes within the hospital when they are suddenly needed, so advance planning is recommended, when possible.

The most serious common complications are those of aspiration and airway occlusion. [22] A low threshold for prophylactic tracheal intubation is recommended. The most serious iatrogenic complication is due to balloon inflation of a misplaced tube, with resultant esophageal rupture. This is avoidable by carefully following the instructions and insisting on radiographic confirmation of tube position before fully inflating the balloons.

Acknowledgment

The author and editors acknowledge the contributions made to this chapter by Jonathan M. Glauser, MD, in earlier editions of this textbook.

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Chapter 43 - Decontamination of the Poisoned Patient

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Alexander B. Baer

The presentation of poisoned patients to the emergency department (ED) is a common occurrence. In 2000, the *Toxic Exposure Surveillance System* of the American Association of Poison Control Centers reported 2,168,248 toxic exposures and 920 resultant fatalities. [72] Of these total exposures, 264,526 (12.2%) were managed in a health care facility. A massive exposure to some very toxic agents (such as cyclic antidepressants, cardiovascular preparations, colchicine, cocaine, carbon monoxide, chloroquine, iron, cyanide, amanita mushrooms, paraquat, and many others) will likely result in severe morbidity or a fatality regardless of even the most sophisticated and timely medical intervention. With general supportive care and the use of a few specific antidotes, however, the mortality rate of unselected overdose patients is only 1% to 2% if the patient arrives at the hospital in time for the clinician to intervene.

The management of poisoned patients presenting to health care facilities initially focuses on confirming the diagnosis of a possible toxin exposure, providing standard cardiovascular and respiratory supportive care, and the use of a small cadre of specific antidotes. In selected instances, the prevention of further toxin absorption by various decontamination procedures may theoretically ameliorate morbidity or reduce mortality. Although a better final outcome from gastric decontamination may seem intuitively reasonable, there is no definitive evidence from prospective clinical trials proving that the use of various decontamination techniques positively alters the morbidity or mortality of the poisoned patient.

Before the availability of objective, experimental evidence addressing gastric emptying procedures, most clinicians instituted such procedures in the ED as a reflex response for the majority of patients suspected of drug overdose, often without much forethought, and certainly without confirming data. Significant controversy exists concerning the need for routine gastric emptying in the poisoned patient, and mounting evidence relegates any form of gastric decontamination to selected cases and individual specific scenarios. *At this time, it is the editors' opinion that there is no universally accepted standard of care that mandates any form of gastric decontamination as a routine medical intervention in the patient suspected or proved to have been exposed to a toxic substance.* (Note: This statement does not apply to ocular or dermal decontamination issues.)

Nonetheless, there may a selective role for gastric decontamination, and there will always be a role for clinical judgment. Since compelling circumstances may call for gastric decontamination, this chapter will discuss specific clinical procedures. These techniques include syrup of ipecac-induced emesis, gastric lavage, oral activated charcoal administration, and whole-bowel irrigation (WBI). Before performing these techniques, the clinician responsible for the care of the poisoned patient must clearly understand that these procedures are not without hazards, and any decision on their use must consider whether the benefit of decontamination outweighs any procedure-related harm.

GASTRIC DECONTAMINATION

Ipecac-Induced Emesis

Background

Syrup of ipecac is available as a nonprescription product in many countries, including the United States. It is prepared from the dried rhizome and roots of the *Cephaelis ipecacuanha* or *Cephaelis acuminata* plant, both of which contain the alkaloids emetine and cephaeline. These alkaloids are potent emetics inducing vomiting by both direct local gastrointestinal effects and central nervous system actions. Emesis following syrup of ipecac ingestion typically occurs within 20 minutes of ingestion and persists for 30 to 120 minutes. [91]

There have been numerous animal and human volunteer studies examining both the efficacy of syrup of ipecac to expel specific ingested agents from the stomach and its ability to decrease serum drug levels. [1] [20] [33] [35] [37] [47] [65] [84] [126] [129] [132] [137] [140] [146] In these studies, the amount of marker removed by syrup of ipecac was highly variable. Syrup of ipecac's efficacy at expelling experimental markers decreased as the administration time postingestion increased. Syrup of ipecac is of very limited benefit if more than 60 to 90 minutes have elapsed since the time of ingestion. Although syrup of ipecac may be effective in reducing the quantity of a drug absorbed, no studies have demonstrated that its use improves patient outcome. [65] [69] [87] [106] Saetta et al suggested in their study that syrup of ipecac may actually enhance gastric emptying and potentially facilitate drug absorption. [116] Ipecac use may delay the use of, or reduce the effectiveness of, other methods of decontamination.

Indications

The administration of ipecac in the ED is rarely indicated. Although theoretically some indications may arise in the ED, there is no standard of care that mandates its use in the management of the poisoned patient in the hospital. Even the historical use of ipecac-induced emesis in the home, prior to definitive medical intervention, has been questioned. The position statement written by the American Academy of Clinical Toxicologists and the European Association of Poisons Centres and Clinical Toxicologists (Fig. 43-1) declared that the routine administration of ipecac in the ED should be abandoned. [67] Bateman's review stated that "ipecac is effectively obsolete" in the management of the poisoned patient. [13] Whether specific subsets of poisoned (e.g., iron, lithium, mushroom) patients may benefit from syrup of ipecac has not been clearly delineated. If syrup of ipecac is administered to a patient, it should be given only to an alert, conscious patient who has ingested a potentially toxic amount of a poison no more than 60 minutes before administration.

Contraindications

The administration of syrup of ipecac is contraindicated in any person who demonstrates compromised airway protective reflexes or has the potential to lose such protective reflexes.[66] Its use should be avoided in persons who have ingested substances that could result in coma, seizures, cardiovascular collapse, or paralysis. Syrup of ipecac is also contraindicated in persons who have ingested corrosive substances (acids or

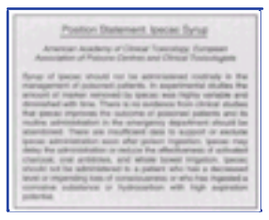

Figure 43-1 Position statement: ipecac syrup. *(From the American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Published in Clin Toxicol 35:699, 1997.)*

alkalis), hydrocarbons, or foreign bodies that could potentially result in airway obstruction. Caution should be exercised in using syrup of ipecac in patients who have medical conditions that could be further compromised by the induction of emesis, such as patients with bleeding diatheses.

Technique

Syrup of ipecac is sold in packages of 15 mL and 30 mL. It is administered orally in the following dosages:

- 0 to 6 months: No ipecac.
- 6 to 12 months: 10 mL ipecac plus 120 to 240 mL clear liquids.
- 1 year to 12 years: 15 mL ipecac plus 120 to 240 mL of clear liquids and repeat 15 mL ipecac if no vomiting occurs within 20 minutes of the first dose.
- Older than 12 years: 30 mL ipecac plus 240 mL of clear liquids and repeat 30 mL ipecac if no vomiting occurs within 20 minutes of the first dose.

Complications

The most commonly reported complications of ipecac administration include diarrhea, lethargy, and prolonged vomiting. [28] [36] [73] Other reported complications include pulmonary aspiration of gastric contents, bradycardia, cerebral hemorrhage, gastric rupture, gastric diaphragmatic herniation, Mallory-Weiss tear, and pneumomediastinum.[62] [63] [68] [115] [127] [135] [145] Prolonged emesis may result in the inability to subsequently administer charcoal.

Gastric Lavage

Background

Reports of the use of gastric lavage in the poisoned patient dates as far back as the early 19th century. [26] [58] Numerous animal and human volunteer studies have been conducted examining the effectiveness of gastric lavage in removing toxins from the stomach, especially in comparison to other gastrointestinal decontamination methods.[7] [10] [21] [25] [32] [33] [126] [129] [137] [143] [146] The reported efficacy of gastric lavage in removing markers from the stomach varies significantly in these studies. The difference in these study results is due in part to the variability of the methods used (different fluid instilled markers, animal models, positioning, amount of lavage and lavage tube sizes) and the time that elapsed from the instillation of the marker in the stomach until gastric lavage was performed. Even within individual studies, the range of effectiveness of gastric lavage to remove the marker varied considerably. For example, Tandberg et al performed gastric lavage 10 minutes after ingestion of the marker and reported its effectiveness to remove the marker varied from 18.9% to 67.7%.[126] There is a possible theoretical benefit of gastric lavage for the evacuation of a toxic liquid from the stomach. Grierson et al, however, were unable to demonstrate a significant benefit of an optimal lavage 1 hour after the ingestion of liquid acetaminophen, further casting doubt on the benefit of the procedure in the clinical scenario. [50]

Many of these studies do not replicate the typical clinical scenario encountered in emergency medicine. [79] The efficiency of gastric lavage to remove a marker significantly decreases with increasing time following ingestion. This is due to the fact that as time increases after ingestion, the more time there is for the marker to be absorbed and for the marker to pass out of the stomach. For example, Shrestha et al reported that greater than 70% of the marker used in their study passed out of the stomach by 60 minutes.[123] It is rare that gastric lavage can be performed within the first hour after toxic ingestion. Not only does it take time for these patients to present to the ED, but it also takes time for evaluation, stabilization, and for the gastric lavage to take place. For example, Watson et al reported that the mean time required by experienced emergency medicine nurses to perform lavage was 1.3 hours.[143] *Gastric lavage may also propel the marker from the stomach into the small*

intestine, decreasing the effectiveness of removing the toxin from the stomach and enhancing the rate of absorption.[116]

Gastric lavage is still considered as a potentially advantageous procedure because, to date, there have been no controlled studies that have included enough patients with confirmed life-threatening ingestions to adequately evaluate lavage versus no lavage. Only three major studies have been performed examining whether gastric lavage positively influences the outcome of poisoned patients. [69] [87] [106] In a study performed by Kulig et al, there was no difference in outcome among patients who received gastric lavage followed by charcoal vs charcoal alone when these were performed >1 hour after ingestion. [69] In patients who were treated within 1 hour of ingestion, gastric lavage followed by charcoal provided a small but statistically significant advantage over activated charcoal alone. Merigian et al, demonstrated that for symptomatic patients, the rate of intensive care admission and the need for intubation was significantly higher for those patients who received gastric lavage followed by charcoal than for those who received charcoal alone. [87] This increased admission and intubation rate was directly attributed to the aspiration of gastric contents owing to gastric lavage. Pond et al replicated the Kulig study. They found no difference in outcome between those who received gastric lavage followed by charcoal versus charcoal alone, regardless of time of performance of gastric lavage. [106] They concluded that "gastric emptying procedures can be omitted from the treatment regimen for adults after acute overdose, including those who present within 1 hour of overdose and those that manifest severe toxicity."

Indications

Based on the available literature, gastric lavage should not be routinely used in the management of poisoned patients [138] (Fig. 43-2). There is no universally accepted standard of care that can be applied to the use of gastric lavage in the unselected poisoned patient in the ED. Because there may be a theoretical benefit from gastric emptying, usually by lavage, under certain circumstances, a listing of circumstances that may increase the appropriateness of gastric emptying appears in Table 43-1 (Table Not Available) . It is stressed that these indications are theoretical, not evidenced based, and are the opinion of one toxicology textbook. Whether specific subsets of overdose patients may benefit from gastric lavage has not been clearly defined.

Only patients who have ingested a potentially life-threatening amount of poison where the procedure can be performed within 60 minutes should be considered candidates for gastric lavage. Oral charcoal alone is considered superior to gastric lavage if a drug is adsorbed by charcoal.

Contraindications

Although generally safe, gastric lavage is not an innocuous procedure. The performance of gastric lavage is contraindicated in any person who demonstrates compromised airway protective reflexes unless they are intubated. Many clinicians opt for lavage in a seriously ill patient who is intubated, because airway protection is already accomplished. Tracheal intubation, however, does not ensure a totally protected airway. *Paralyzing and intubating a patient merely to initiate gastric lavage is generally eschewed.* Gastric lavage is contraindicated in persons who have ingested corrosive substances (acids or alkalis), hydrocarbons (unless containing highly toxic substances such as paraquat, pesticides, heavy metals, halogenated and aromatic compounds), known esophageal strictures, or history of gastric bypass surgery. Caution should be exercised in performing gastric lavage in patients who have medical conditions that could be compromised by performing this procedure, such as patients with bleeding diatheses, and in combative patients.

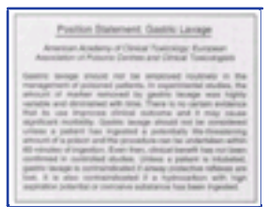

Figure 43-2 Position statement: gastric lavage. *(From the American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Published in Clin Toxicol 35:711, 1997.)*

TABLE 43-1 -- Factors that Cumulatively Increase the Appropriateness of Gastric Emptying

(Not Available)

From Smilkstein MJ: Techniques used to prevent gastrointestinal absorption of toxic compounds. Goldfrank's Toxicologic Emergencies, 7th ed., New York, NY. McGraw Hill, 2002.

Equipment and Preparation

If the decision is made to perform gastric lavage, careful attention to the details of the procedure results in increased safety for the patient and more effective removal of the ingested poison. Before lavage, the patient should have IV access secured and should have continuous cardiac monitoring and pulse oximetry. A large, rigid suction tip should be immediately available.

If the level of consciousness is significantly depressed or the patient's airway-protective reflexes are diminished, the airway should be protected with a cuffed endotracheal tube before initiation of gastric lavage. If the patient is highly anxious or agitated, small doses of a benzodiazepine (e.g., 1 to 2 mg midazolam IV) may be given. If the airway status is questionable, or has the potential to be compromised during the procedure, rapid-sequence induction and intubation should be considered. If patients are fully alert and awake, lavage may be done without tracheal intubation. The procedure should proceed deliberately without significant patient resistance. The procedure is intended to be therapeutic, not punitive. Antiquated arguments promulgating that a noxious lavage will keep patients from overdosing again should be abandoned.

The position of the patient during gastric lavage is important. All patients should be placed in the left lateral decubitus position with the head down (approximately 20° tilt on the table) (Fig. 43-3). This position diminishes the passage of gastric contents into the duodenum during lavage and decreases the risk of pulmonary aspiration of gastric contents

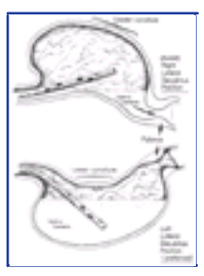

Figure 43-3 The effect of patient positioning on lavage. The left lateral decubitus position is preferred.

should vomiting or retching occur. The uncooperative patient's hands should be restrained to prevent removal of the gastric or endotracheal tube. Intubated patients on a ventilator may be lavaged in the supine position because of logistical reasons. *Under no circumstances should the nonintubated patient undergo lavage in the restrained supine position.* Such positioning invites aspiration and diminishes the patient's natural protective maneuvers, such as coughing and sitting up.

Most clinicians prefer the oral route for gastric lavage, but in selected circumstances, a standard large-bore nasogastric (NG) tube (Salem sump pump) may be used. Large-diameter gastric hoses with extra holes cut near the tip have been traditionally recommended for gastric lavage (Fig. 43-4). There are no convincing data on humans to refute or support this recommendation, and one study of a small number of dogs failed to show any difference in efficacy with lavage through a 32 Fr tube

compared with a 16 Fr lavage tube.[41] It is generally held that large-diameter NG or orogastric tubes (>1 cm) are more likely to retrieve particulate matter successfully. Smaller, more flexible tubes may kink and are significantly more difficult to pass. An NG tube may be passed through the mouth or nose, but orogastric hoses should not be passed through the nose. Because most pills disintegrate in the stomach in a few minutes, significant amounts of particulate matter may be retrieved with a large-bore NG tube, such as an 18 Fr Salem sump tube. Nasogastric tubes are considerably easier to pass, and are less traumatic for the patient (see Chapter 41). Nasogastric tubes are preferred for liquid ingestions.

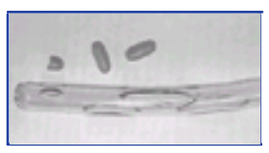

Figure 43-4 A large-diameter gastric tube. Note the extra side holes that have been cut near the tip. This is a theoretical advantage over a standard tube or a nasogastric tube. For small pill particles and liquids, a large-bore nasogastric tube may suffice.

In most cases a 36 to 40 Fr or 30 English gauge tube (external diameter 12 to 13.3 mm) should be used in adults and a 24 to 28 Fr gauge (diameter 7.8 to 9.3 mm) tube in children.[138] (See discussion of pediatric issues in Fig. 43-5 .) Before passage, the length of the tube required to enter the

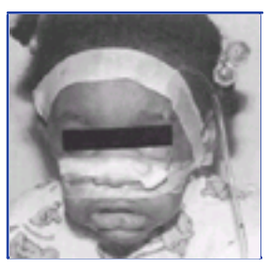

Figure 43-5 Gastric lavage in a child is always problematic. Obviously, an adult-sized large-bore oral gastric tube cannot be used, but a nasogastric (NG) tube may suffice. Some pediatric textbooks recommend a 24 Fr oral gastric tube for toddlers, and a 36 Fr tube for adolescents. In this case, a child was found with an open bottle of digoxin, and it could not be determined if ingestion had occurred. She would not drink charcoal. The 18 Fr NG tube was used to attempt to aspirate digoxin from the stomach (none was recovered) and to instill charcoal. Some would suggest the oral route for this tube, but it was passed rather easily through the nose. An NG tube is not ideal for some ingestants (iron, sustained-release products), but most pills quickly dissolve in the stomach and the small particles can easily be removed with an NG tube. Although lavage may have been reasonable in this scenario, a potent and safe antidote for digoxin does exist. The common routine practice of passing an NG tube in a child who is unwilling to drink charcoal is controversial and likely done far too often for benign ingestions. *(Reprinted with permission from Elsevier [The Lancet, vol 338 (8778), 1991, pp 1313–1315].)*

stomach should be estimated by approximating the distance from the corner of the mouth to the mid-epigastrium; premeasurement avoids the curling and kinking of excess hose in the stomach (Fig. 43-6). Passage of an excessive length of hose may cause gastric distention, bruising, and perforation, whereas passage of an insufficient length of hose may result in lavage of the esophagus and the increased risk for emesis and aspiration. Commercial lavage systems are available and often use either a gravity fill-and-empty system with a Y connector (e.g., Travenol, Ethox) or a closed irrigation syringe system. Alternatively, an irrigation syringe can be used for intermittent lavage fluid input and withdrawal.

Technique

In most cases gastric lavage should be performed through an orogastric tube. Use of a standard NG tube may be adequate if only small pill fragments are present, but an NG tube is associated with two problems: It is of insufficient bore to produce satisfactory lavage of large particulate matter, and the larger bore tubes may cause epistaxis or damage to the turbinates. The latter problem may be obviated by oral passage. The gastric tube should be lubricated and passed gently to avoid damage to the posterior pharynx. A bite block or an oral airway should be used to avoid chewing of the orogastric tube and biting of the fingers of the inserter. If the patient is obtunded or paralyzed, the clinician may extend the jaw to facilitate passage. *Force should never be used to pass the tube.* Putting the patient's chin on the chest can facilitate passage of the tube into the esophagus once the pharynx has been entered (Fig. 43-7). Cough, stridor, or cyanosis indicates that the tube has entered the trachea; the tube should be withdrawn immediately and passage reattempted. Once the tube is passed, its intragastric location should be confirmed. Intragastric placement is usually evident on clinical grounds, and confirmed by auscultation of the stomach during injection of air with a 50-mL syringe and aspiration of gastric contents. In

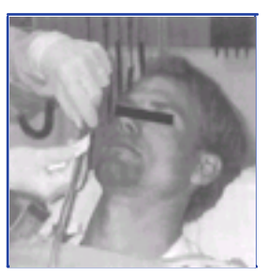

Figure 43-6 Failure to premeasure a lavage tube before passage is a common error. Here a piece of tape marks the depth of proper passage to ensure that the tip is in the stomach without excess tubing that may hinder fluid egress.

Figure 43-7 Positioning the patient's chin on the chest can facilitate passage of the tube into the esophagus once the pharynx has been entered. Once the tube is positioned, lavage is performed in the left lateral decubitus position. If the patient begins to vomit, the tube is immediately withdrawn.

the intubated or obtunded patient or the young child, some clinicians consider confirming tube position radiographically before lavaging, although this is not routinely performed. A misplaced tube may irrigate the esophagus with a tube that has doubled back on itself during passage. The most serious complication is inadvertent passage of the tube into the lungs. Tracheal passage of a lavage tube should be readily obvious in the awake patient prior to lavage, and obtunded patients are intubated, obviating this problem. *If an awake patient begins to vomit during the lavage, immediately remove the tube to allow the patient to protect the airway.*

Before gastric irrigation, the gastric contents should be removed by careful gastric aspiration with repeated repositioning of the tube tip. With the Y connector closed system, lavage is performed by clamping the drainage arm of the Y adapter and infusing aliquots of fluid into the stomach from a reservoir (Fig. 43-8). The reservoir arm of the Y is then clamped, and the drainage arm is opened to permit gravity drainage of the stomach contents. The procedure is then repeated. Some resistance is produced by the Y connector and tubing. Suction can be applied intermittently to the drainage tubing to enhance stomach emptying.

Lavage can be performed adequately with tap water in adults. Because electrolyte disturbance has occurred in children who were lavaged with tap water, prewarmed (45°C) normal saline is generally recommended for children. [11] [27] [104] Warmed lavage fluid increases the solubility of most substances, delays gastric emptying, and theoretically should increase the effectiveness of the procedure. [83] [113] Small aliquots of lavage solution (200 to 300 mL in adults and 10 mL/kg body weight in children up to a maximum of 300 mL) should be repeatedly introduced into the stomach and removed. If larger amounts of fluids are used, there is a potential for an increased risk of washing gastric contents into the duodenum or the lungs, and much smaller amounts are not clinically practical because of the dead space in the tubing (approximately 50 mL in the 36 Fr

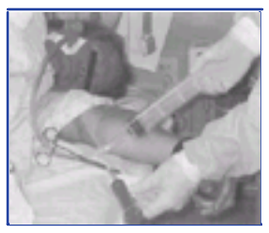

Figure 43-8 An example of the Y connector closed system with the patient in a left lateral decubitus position. Patients on a ventilator, or those who are intubated may be lavaged in the supine position due to logistic issues, but an awake nonintubated patient is never lavaged in the restrained supine position.

hose) and the increase in time that is required. The amount of fluid that is returned should approximate the amount that is introduced. Manual agitation of the patient's stomach by gently "kneading" the stomach with a hand placed on the abdomen may increase recovery. [83] Lavage should be continued until the lavage fluid becomes clear.

After gastric aspiration and lavage have been completed, a slurry of activated charcoal should be administered through the gastric tube. When no longer needed, the gastric tube should be pinched or clamped during its removal to avoid "dribbling" fluid into the airway. With the increasing use of repetitive doses of activated charcoal, the gastric tube is often left in place after the lavage procedure is completed. Because this large tube is irritating and may predispose the patient to gagging, drooling, or aspiration, it should be removed. The alert patient should take subsequent doses orally as necessary. The patient who remains obtunded may receive additional doses via a standard NG tube, although the high viscosity of the charcoal makes this route challenging. If endotracheal intubation was required prior to lavage tube placement, the endotracheal tube should *not* be removed until the patient is clearly awake and able to control his or her airway, because emesis is common after an overdose and in association with the procedure.

Complications

A correctly performed procedure is generally safe but there have been numerous complications associated with gastric lavage. The complications can be divided into those caused by mechanical trauma and those resulting from the lavage fluid.

Depending on the route selected for tube insertion, damage to the nasal mucosa, turbinates, pharynx, esophagus, and stomach have all been reported. [8] [34] [80] [142] After tube insertion, it is imperative to confirm correct placement. Scalzo et al found radiographically that 7 of 14 children had improper tube placement (too high or too low) despite positive gastric auscultation in all cases. [117] Although not a routine standard, radiographic confirmation of tube placement should be considered in young children and intubated patients. Instillation of lavage fluid and charcoal into the lungs through tubes inadvertently misplaced within the airways has been reported. [57]

During lavage, changes in cardiorespiratory function have been noted. Thompson et al, reported that during lavage, 36% of patients had atrial or ventricular ectopy, 4.8% had transient ST elevation, and 29% had a fall in oxygen tension to 60 torr or less. [134] Patients at greatest risk for these findings included the elderly, smokers, those with lung disease, or cyclic antidepressant overdose. Laryngospasm may also occur during gastric lavage. [138]

The lavage fluid itself is a potential source of complications. The large amount of fluid administered during lavage has been reported to cause patient fluid and electrolyte disturbances. These disturbances have been seen with both the use of hypertonic and hypotonic lavage fluids in the pediatric population. [11] [27] [104] Hypothermia is a possible complication if the lavage fluid is not pre-warmed.

Pulmonary aspiration of gastric contents or lavage fluid is the primary potential risk during gastric lavage, especially in patients with compromised airway protective reflexes.[81] Merigian et al, reported a 10% incidence of aspiration pneumonia in patients who received gastric lavage. [87] This risk is reduced by using small aliquots of lavage fluid, by adequately positioning the patient, and by intubating patients with compromised airway protective reflexes.

Excessive force should be avoided if the lavage tube cannot be easily removed. Kinking or knotting of the tube can occur, but occasionally a tube may become stuck because of lower esophageal spasm. If fluoroscopy demonstrates no deformation to the lavage tube, 1 to 2 mg of IV glucagon can be infused in an attempt to relieve lower esophageal spasm.[133] Surgical removal may be necessary if the gastric tube is deformed by kinking or knotting.

Activated Charcoal

Background

Activated charcoal is a carbon product that is subjected to heat and oxidized to increase the surface area and its capacity to adsorb substances onto the surface of the charcoal. A high surface charcoal, termed superactivated charcoal, is intermittently available to the clinician. Superactivated charcoal adsorbs more toxin per gram of charcoal and is recommended if available. Activated charcoal acts both by adsorbing a wide range of toxins present in the gastrointestinal tract and by enhancing toxin elimination, if systemic absorption has already occurred. It enhances elimination by creating a concentration gradient between the contents of the bowel and the circulation, but it also has the potential of interrupting enterohepatic circulation if the particular toxin is secreted in the bile and enters the gastrointestinal tract prior to reabsorption.[103] Oral activated charcoal is given as a single-dose or in multiple doses. The adsorptive capacity of charcoal depends on inherent properties of the toxin, and the local milieu, such as pH. Adsorption begins within minutes of contact with a toxin, but may not reach equilibrium for 20 to 30 minutes. Desorption of toxins from charcoal occurs over time, although this has little clinical significance for most patients and can be overcome by administering additional charcoal.

Indications

For years the administration of oral activated charcoal for essentially all overdoses has been routine. Clearly charcoal

binds many toxins in the gut, thereby decreasing some systemic absorption. Despite a lack of scientific data demonstrating a decrease in morbidity and mortality, and without firm evidence to support its widespread use, charcoal is a reasonable intervention for most poisoned patients presenting to the ED; if it can be easily and safely administered. The exact indications are not established, and no universally accepted standard of care has been promulgated. In the opinion of the editors, single-dose activated charcoal is indicated if the clinician estimates that a clinically significant fraction of the ingested substance remains in the GI tract, the toxin is adsorbed by charcoal, and further absorption may result in clinical deterioration. This will essentially always be a clinical decision, since adequated historical data may often be lacking. It may also be administered by multiple dosing, if the clinician anticipates that the charcoal will result in increased clearance of an already absorbed drug. In 1997, the American Academy of Clinical Toxicology released a position statement advising that activated charcoal should not be routinely administered but should be reserved for cases in which serious toxicity is anticipated [31] (Fig. 43-9). It is most effective within the first 60 minutes after oral overdose and decreases in effectiveness over time. Charcoal is generally considered to provide superior gut decontamination when compared to gastric lavage. Combining lavage and charcoal, although intuitively attractive, has no proven additional benefit.

Contraindications

The administration of charcoal is contraindicated in any person who demonstrates compromised airway protective reflexes, unless they are intubated. [31] It is contraindicated in persons who have ingested corrosive substances (acids or alkalis). Charcoal not only provides no benefit in a corrosive ingestion, but its administration could precipitate vomiting, obscure endoscopic visualization, and lead to complications if a perforation developed and charcoal entered the mediastinum, peritoneum, or pleural space. Charcoal should be avoided in cases of a pure aliphatic petroleum distillate ingestion. Hydrocarbons are not well adsorbed by activated charcoal and its administration

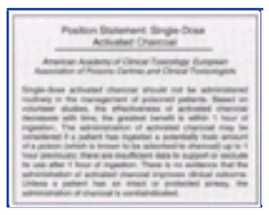

Figure 43-9 Position statement: single-dose activated charcoal. *(From the American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Published in Clin Toxicol 35:721, 1997.)*

could lead to further aspiration risk. Many hydrocarbons are potential systemic toxins (e.g., carbon tetrachloride and benzene) or are mixed with other potentially significant toxins such as pesticides. In these cases, data are lacking, but charcoal administration can be considered. Caution should be exercised in using charcoal in patients who have medical conditions that could be further compromised by charcoal ingestion, such as those with gastrointestinal perforation or bleeding. Charcoal is not indicated for isolated ingestions of ethanol, iron, or lithium because these substances are not adsorbed. If the airway is not secure, charcoal should be given with caution, to minimally symptomatic patients who have ingested a toxin that may suddenly induce seizures. Such as cyclic antidepressants and theophylline. Charcoal will adsorb cyanide, strychnine, and mercuric chloride. Because it is often impossible to determine the exact nature of an ingestion, a liberal use policy is advocated for potentially mixed overdoses.

The use of activated charcoal in the treatment of acetaminophen ingestion presents a special issue because the antidote, *N*-acetylcysteine, is approved only for oral use in the United States. Although activated charcoal could potentially prevent acetaminophen from reaching toxic concentrations in the blood, there are concerns that charcoal may also adsorb significant amounts of the antidote as well. Pharmacokinetic studies evaluating the effect of activated charcoal on *N*-acetylcysteine absorption have produced conflicting results. [39] [62] [97] The standard *N*-acetylcysteine dosing currently in use is much larger than needed to treat the vast majority of acetaminophen overdoses, and therefore no adjustment is required in this scenario. This issue is totally circumvented by the use of IV *N*-acetylcysteine. Although not FDA approved, the oral *N*-acetylcysteine preparations are universally used as an IV antidote, a technique recommended by the authors.

Charcoal administration by paramedics and other emergency response personnel should be performed with caution. The same indications and contraindications apply as for those patients who are in the hospital. The motion of the ambulance during transport may make the patient more prone to emesis. Either the spilling of charcoal or the vomiting of charcoal may result in significant contamination of the transport vehicle and subsequently place that vehicle out of commission until it can be cleaned.

Technique

There is no universally accurate dose for charcoal. A 10:1 ratio (charcoal:toxin) is recommended if the amount of ingestion is known. Charcoal dosing should be considered in light of the specific ingestion, but the recommended empiric doses of single-dose activated charcoal (standard aqueous products, such as Liqui-Char) are as follows[31] :

- Up to 1 year: 1 g/kg of body weight
- 1 year to 12 years: 25 to 50 g
- Older than 12 years: 25 to 100 g

If the ingestion were, for example, clonidine (0.1-mg tablets) or digoxin (0.25-mg tablets), this regimen would be more than adequate for even a massive overdose to achieve the desired 10:1 ratio. If the ingestion consisted of a large number of 325-mg aspirin tablets, or 240-mg verapamil tablets, the dosing regimen could be insufficient. If toxic medications with a high milligram dosage are ingested, it would be prudent to administer more charcoal than indicated by these guidelines. We recommend superactivated charcoal if it is commercially available. There is no known benefit of

mixing charcoal with a cathartic, and the combination is not suggested. Sorbitol increases the incidence of vomiting.

In many formulations the contents settle with time and vigorous shaking before administration is recommended. This should be followed by rinsing the container with a small amount of tap water prior to administering it to the patient, which will allow ingestion of the full dose. [66] Aqueous activated charcoal has a gritty texture that most patients find unpleasant and attempts have been made to improve the taste and texture. Mixing activated charcoal with chocolate milk, chocolate- or cherry-flavored syrup, or ice cream may increase palatability, but mixing with these additives has been suggested, though not proved, to cause a decrease in the adsorptive capacity of activated charcoal. [71] Rangan et al have recently reported no decrease in adsorption after mixing superactivated charcoal with a non-caffeinated cola. [111] Scharman et al demonstrated that a regular, sugared cola was favored by children over a diet cola, but it was very difficult to cajole even nonpoisoned children under age 3 years to drink a therapeutic amount of flavored charcoal. [119] It is very difficult to convince a toddler to drink a therapeutic dose of charcoal under the best of circumstances, questioning the long-held practice of keeping charcoal in the home to hasten the use of the antidote.

The patient should be given a brief (5- to 10-minute) period of time to drink the dose. If the dose is not consumed by that time, the clinician should consider giving the dose by NG tube to maximize charcoal efficacy. If a nasogatric tube is inserted, correct placement must be verified. Radiographic confirmation of tube placement should be considered in obtunded or intubated patients if doubts about placement exist (Fig. 43-10). Instillation of charcoal into the lungs through tubes inadvertently misplaced within the airways has been reported. [57]

Activated charcoal may be given orally if the patient is awake and cooperative or by NG tube if the patient is unconscious (Fig. 43-11). It may also be given through the lavage tube after gastric lavage (Fig. 43-12). The common tactic of passing an NG tube in the awake but uncooperative patient, merely to administer charcoal, is controversial. Such a scenario is more likely to result in trauma from the tube, a misplaced tube, or subsequent emesis from the rapid administration of charcoal. Given the unproven efficacy of charcoal, the editors advise against the *routine* insertion of an NG tube to simply administer charcoal in the awake and minimally symptomatic patient. Such a decision is, however, a clinical one that must be made based on the entire clinical milieu.

Complications

Charcoal is generally very safe and few adverse effects from the use of single-dose activated charcoal have been reported, despite its widespread use. There are no reports of gastrointestinal obstruction associated with single-dose activated charcoal. The most common complications of charcoal administration include constipation, diarrhea, and vomiting. [96] Pulmonary aspiration of activated charcoal is a dreaded complication that can result in pneumonitis, obstruction of the respiratory tree, and bronchiolitis obliterans. [14] [44] [80] [105] Aspiration of large amounts of charcoal can be fatal. [86] Risk factors for serious aspiration are large amounts of charcoal instilled over a short period of time, multiple dose charcoal in the setting of an ileus, charcoal administration in a patient who becomes obtunded, or the forced administration of charcoal via an NG tube, especially in a restrained supine

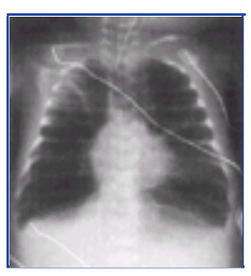

Figure 43-10 Radiographic confirmation of nasogastric tube placement before lavage or instillation of charcoal. Tracheal placement of a lavage tube is usually readily evident. Vomiting during lavage suggests that the tube has curved back into the esophagus. A confirmatory radiograph is suggested in the obtunded patient if gastric placement is questioned. Tracheal intubation precludes passage of a tube into the lungs, but it does not ensure proper gastric placement.

patient. Trivial aspirations of charcoal are common, even if the patient is intubated, and are usually innocuous.

Multiple Doses of Activated Charcoal

Indications

The use of multidose activated charcoal (MDAC) may be indicated in select cases. [30] Its use has been advocated for two purposes: first, to prevent continued absorption of a drug that may still be present within the gastrointestinal tract; second, to increase the clearance of a drug that has already been absorbed. The recommendations of various toxicology organizations for MDAC are presented in Figure 43-13 .

MDAC prevents continued absorption by either binding a drug that may be present throughout the gastrointestinal tract or binding a drug that exists as extended-release or enteric-coated preparations. MDAC enhances elimination of a drug by interrupting enterobiliary recirculation or augmenting enterocapillary exsorption.[96] By interrupting enterobiliary recirculation, charcoal binds to an active drug that is secreted by the biliary system, subsequently preventing reabsorption. By augmentation of enterocapillary exsorption, charcoal produces sink conditions that drive diffusion of drug from the capillaries into the entraluminal space, where it is subsequently eliminated. This process is called "intestinal dialysis". [70] Drug characteristics that are associated with enhanced systemic clearance with MDAC include a low intrinsic clearance, a prolonged distributive phase, low protein binding, and a small volume of distribution. [29]

Figure 43-11 If the overdose patient will voluntarily drink charcoal, there are few reasons to withhold it, even though a definite clinical benefit in the routine case cannot be proved. If a patient will not drink charcoal, patient management becomes controversial. Passing a nasogastric tube in a struggling patient or in a recalcitrant child merely to instill the unproven, but theoretically useful, antidote is not supported by scientific data. Nonetheless, it remains a common procedure. Although not always easy or pleasant, such an intervention is usually safe. Pulmonary aspiration, even in the awake patient, is the major downside. *Restrained supine patients are at greatest risk for aspiration, and that position should be avoided, even in the initially awake patient.*

MDAC has been shown to increase total body clearance of multiple drugs, including carbamazepine, [18] [90] [93] dapsone,[94] [95] phenobarbital,[15] [16] [19] [42] [47] [93] [107] [141] quinine, [74] [109] and theophylline.[5] [17] [30] [38] [43] [55] [76] [99] [101] [103] [110] [121] [122] [136] MDAC may be beneficial in the management of overdoses of the drugs listed in Table 43-2 . Despite the reported increase in drug clearance associated with the use of MDAC, improved clinical outcomes have not been definitively demonstrated. For example, Pond et al described 10 comatose patients following phenobarbital overdose who were randomized to receive either single-dose activated charcoal or MDAC.[107] Despite the fact that the MDAC group had a significantly shorter phenobarbital serum half-life, there was no difference between the groups in regard to the duration of intubation or hospitalization.

Contraindications

MDAC is contraindicated if there is evidence of bowel obstruction. An ileus is a relative contraindication. Many ill patients who develop an ileus may be selected candidates for MDAC if the airway is protected. The administration of MDAC is contraindicated in any patient who does not

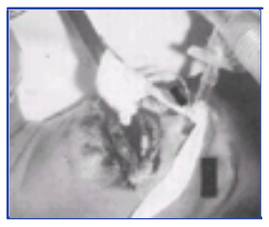

Figure 43-12 Charcoal that is voluntarily swallowed or instilled via an oral-gastric lavage tube or nasogastric tube can induce emesis. This occurs in both the obtunded and awake patient. In this instance, the patient was unconscious from the overdose and the airway was protected with prior tracheal intubation. Although the intubation procedure does not totally exclude pulmonary aspiration, and it carries some morbidity in its own right, it is recommended prior to charcoal use in the patient who is not able to fully protect the airway. Patients who initially are asymptomatic or minimally affected but have ingested drugs that have the potential to produce rapid deterioration, seizures, or loss of airway protection make decisions on the use of charcoal difficult for the clinician. In borderline cases, some experienced clinicians avoid the use of charcoal altogether.

have an intact or protected airway. MDAC should be avoided in patients who have repetitive emesis, especially when associated with decreased mental status or a decreased gag reflex. The concurrent use of cathartics with MDAC remains unproved and is not recommended. [108] MDAC with cathartics should not be administered to young children because of the propensity for laxatives to cause fluid and electrolyte imbalance. For example, MDAC with sorbitol has been associated with hypernatremia and dehydration[2] [82] and MDAC with magnesium cathartics has been associated with hypermagnesemia, neuromuscular weakness, and coma.[56] [124]

Technique

The first dose of activated charcoal should be 1 g/kg (maximum of 100 g). If a cathartic is used, it should be administered only with the first dose of charcoal to decrease the risk of cathartic-induced electrolyte abnormalities that can potentially develop, especially in children. [2] [56] [82] [124] The initial dose of charcoal is followed by 0.5 g/kg (up to 50 g) of activated charcoal every 4 hours. If repeat examination reveals an absence of bowel sounds or reveals a distended abdomen, then MDAC should be terminated and the clinician should consider placement of a NG tube on low intermittent suction. Patients receiving MDAC may be at increased risk for emesis because of the larger total dose of activated charcoal received. The use of antiemetics may help decrease the incidence of vomiting associated with MDAC. [5] [22] [114] Charcoal therapy should be continued until

Figure 43-13 Position statement and practice guidelines on the use of multi-dose activated charcoal in the treatment of acute poisoning. *(From the American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Published in Clin Toxicol 37:731, 1999.)*

there is clinical improvement and plasma drug levels have fallen to acceptable levels.

Complications

The complications encountered in single-dose activated charcoal are also encountered in multiple dose activated charcoal. In addition, there have been reports of gastrointestinal obstruction and perforation from MDAC therapy, especially in conjunction with the ingestion of drugs with anticholinergic properties. [9] [48] [49] [112] [144]

TABLE 43-2 -- Drugs Whose Serum Clearance May Be Enhanced by Multiple Doses of Activated Charcoal

Aspirin
Caffeine
Carbamazepine
Cyclosporine
Dapsone
Digoxin
Disopyramide
Nadolol
Phenobarbital
Phenytoin
Quinine
Sotalol
Sustained-release thallium
Theophylline
Valproate
Vancomycin

Cathartic Use

Background

The use of cathartics is intended to decrease the absorption of substances by accelerating the expulsion of the poison from the gastrointestinal tract. Theoretically, this would also minimize the possibility of desorption of drug bound to activated charcoal. The majority of data suggest negligible clinical benefit from cathartic use. [3] [89] There is little evidence that a single dose of aqueous activated charcoal is significantly constipating; however, cathartics are often given for this potential problem.

Indications

The routine administration of a cathartic in combination with activated charcoal is not endorsed by the American Academy of Clinical Toxicology or the European Association of Poison Centres and Clinical Toxicologists. [12] Figure 43-14 gives a consensus recommendation on the use of cathartics. In addition, the administration of a cathartic alone has no role in the management of the poisoned patient.

Contraindications

Cathartics are contraindicated if there is volume depletion, hypotension, significant electrolyte imbalance, corrosive ingestion, ileus, recent bowel surgery, intestinal obstruction or perforation. The administration of cathartics is also

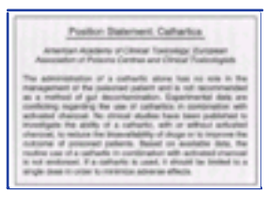

Figure 43-14 Position statement: cathartics. *(From the American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Published in Clin Toxicol 35:743, 1999.)*

contraindicated with patients who do not have an intact or protected airway. They should be avoided in patients who have repetitive emesis, especially when associated with decreased mental status or a decreased gag reflex. Cathartics should be used cautiously in young children and the elderly because of the propensity for laxatives to cause fluid and electrolyte imbalance.

Technique

There are two types of osmotic cathartics: saccharide cathartics (sorbitol) and saline cathartics (magnesium citrate, magnesium sulfate, and sodium sulfate). The optimal dose of sorbitol or magnesium citrate remains to be determined. The recommended dose of sorbitol is approximately 1 to 2 g/kg of body weight or 1 to 2 mL/kg of 70% sorbitol in adults and 4.3 mL/kg of 35% sorbitol in children (single administration only). [12] Many charcoal formulations come premixed with sorbitol, but there is considerable variation in sorbitol content: Liqui-Char with sorbitol contains 50 g of activated charcoal in 54 g of sorbitol; Actidose with sorbitol contains 50 g of activated charcoal in 96 g of sorbitol; activated charcoal USP with sorbitol contains 25 g of activated charcoal in 27 g of sorbitol; CharcoAid contains 30 g of activated charcoal in 110 g of sorbitol. The recommended dose of magnesium citrate is 250 mL of 10% solution in an adult and 4 mL/kg body weight of 10% solution in a child. Multiple doses of cathartics should be avoided.

Complications

The administration of sorbitol has been associated with vomiting, abdominal cramps, nausea, diaphoresis, and transient hypotension. [46] [55] [88] Because the sorbitol content varies between different charcoal/sorbitol combination products,

Figure 43-15 This "body packer" *(A)* attempted to smuggle more than 50 packets of heroin. All packets were passed intact after 12 hours of whole-bowel irrigation. Note the integrity of the carefully wrapped packets that were passed *(B)*.

attention should be paid to the sorbitol content in each brand to avoid excessive sorbitol administration. Multiple doses of sorbitol have been associated with volume depletion. [2] Multiple doses of magnesium-containing cathartics have been associated with severe hypermagnesemia. [56] [124] Children are particularly susceptible to the adverse affects of cathartics, and therefore cathartics should be used with caution, or totally avoided, in children.

Whole-Bowel Irrigation

Background

Whole-bowel irrigation (WBI) has emerged as the newest technique in gastrointestinal decontamination. It involves the enteral administration of an osmotically balanced polyethylene glycol-electrolyte solution (PEG-ES) in a sufficient amount and rate to physically flush ingested substances through the gastrointestinal tract, purging the toxin before absorption can occur (Fig. 43-15).[129] PEG-ES (CoLyte, GoLYTELY) is isosmotic, is not systemically absorbed, and will not cause electrolyte or fluid shifts. Available data suggest that the large volumes of this solution needed to mechanically propel pills, drug packets, or other substances through the gastrointestinal tract are safe, including in pregnancy and in young children. [128] [139]

Indications

WBI may be considered for ingestions of exceedingly large quantities of potentially toxic substances, ingestions of toxins that are poorly adsorbed to activated charcoal (e.g., iron, lithium), ingestions of delayed-release formulations, late presentation after ingestion of a toxin, pharmacobezoars,

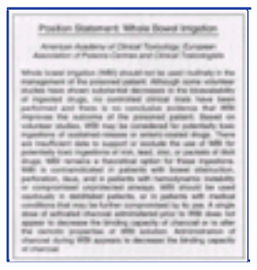

Figure 43-16 Position statement: whole-bowel irrigation. *(From the American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Published in Clin Toxicol 35:753, 1997.)*

and body stuffers or packers.[53] [60] [125] [129] WBI remains a theoretical option for these ingestions. There is no definitive evidence that WBI improves the outcome of the poisoned patient[131] (Fig. 43-16). Although not a proven procedure, WBI is often suggested by toxicologists. Its use is intuitively reasonable and supported by the editors. The most common indication for WBI in the ED is for the treatment of toxic sustained-release medications (such as calcium channel blockers, theophylline, and lithium) and iron tablets (Fig. 43-17).

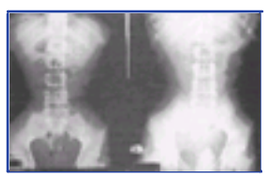

Figure 43-17 Whole-bowel irrigation is commonly recommended for the treatment of iron ingestion. These radiographs depict the effect of 5 hours of whole-bowel irrigation. Note the marked decrease of radiopaque pills in the gastrointestinal tract. Intact pills were recovered in the rectal effluent.

Contraindications

WBI is contraindicated in patients with gastrointestinal obstruction, perforation, ileus, and corrosive ingestion. It should also be avoided in patients with hemodynamic instability or an unprotected airway.[130] WBI should be avoided with patients who have repetitive emesis, especially when associated with decreased mental status or a decreased gag reflex. WBI should be used cautiously in debilitated patients.

Technique

PEG-ES is marketed in a powder form. Tap water is added to make a total volume of 4 L. The recommended rate of administration is as follows.[129]

- 9 months to 6 years: 500 mL/hour
- 6 years to 12 years: 1000 mL/hour
- Older than 12 years: 1500 to 2000 mL/hour

Cooperative patients with intact airway protective reflexes may drink the solution. The large volume and taste often limit even the most motivated patient's ability to comply. If the patient is unable or unwilling to drink this solution, it should be administered through a small-bore NG tube after placement is confirmed. Because it is common for WBI to be delayed while the patient and medical personnel attempt to administer the large volumes of WBI solution required to be effective, it is suggested that NG instillation be instituted early in the ED course (Fig. 43-18). Unconscious patients with protected airways may receive WBI. Pre-warming the irrigant to a temperature of approximately 37°C avoids the potential complication of hypothermia. To collect the waste products, the awake patient may need to be seated on a commode; a rectal tube will need to be placed in the obtunded patient. Many toxicologists recommend adding two to three bottles of activated charcoal to each liter of WBI solution. The benefit is unproved but there is little theoretical downside to this technique, and it is supported by the editors. The binding capacity of charcoal is decreased when combined with PEG-ES, but the clinical consequences of this observation are unknown. Empirically, metoclopramide may be coadministered to decrease nausea and facilitate GI passage.

The endpoint of WBI is until the arrival of clear rectal effluent and/or resolution of toxic effect. [130] There are rare case reports of late purging of drug packets, plant parts, and tablets

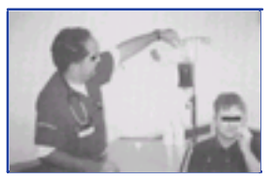

Figure 43-18 It is very difficult for even the most motivated patient to drink an effective volume of whole-bowel irrigation solution. To enhance compliance and to decrease vomiting, PEG-ES may be slowly and continuously administered via a nasogastric tube. An empty bag of saline is hung on an IV pole, the corner of the bag is removed, and the PEG-ES is poured into the bag. Standard IV tubing is connected to the proximal end of a nasogastric tube and the solution is infused continuously. In this picture, charcoal has been added to the whole-bowel irrigation solution. Metoclopramide was coadministered to reduce nausea.

after the arrival of clear effluent.[53] [118] Radiographic studies may also be beneficial to determine the endpoint in body packers or in patients who have ingested radiopaque medications.

Complications

There have been few reported complications from WBI therapy, especially pertaining to acute poisonings. Nausea, vomiting, abdominal cramps, and bloating have been described.[40] Nausea and vomiting may make administration of WBI difficult. Antiemetics and a 15- to 30-minute break followed by a slower rate may allow readministration. As discussed with the other methods of decontamination, attention should be directed to the airway and the potential for aspiration. Administration of a large amount of chilled or room temperature WBI fluid to pediatric patients could potentially cause hypothermia. Warmed fluid should be considered in these

patients. If activated charcoal is administered concurrently with WBI, there may be a desorption of toxin from charcoal. [52] [61] [77]

DERMAL DECONTAMINATION

Background

Numerous hazardous material (HAZMAT) incidents occur each year in the United States. The National Response Center Database, which tracks comparatively large incidents such as train derailments and industrial accidents, reported 34,360 HAZMAT incidents in 2001. [92] In 1996, 5502 HAZMAT events in 14 states involving 5887 substances were reported to the Hazardous Substances Emergency Events Surveillance System (HSEESS).[51] HAZMAT events frequently result in injuries, and the ED treatment of contaminated HAZMAT patients is not a rare event. Many of these patients transport themselves to the ED, including those involved in past terrorist events. For example, in the Tokyo sarin gas attack, 93% of 498 patients reporting to St. Luke's Hospital arrived by means other than ambulance. [100] The risk of injury to medical personnel incurred while treating contaminated patients is significant. Of the patients reported to HSEESS, emergency responders accounted for 10% of injuries and hospital personnel for 4.1% of injuries.[51] After the Tokyo attack, 13 of 15 clinicians (87%) reported symptoms while treating patients in the ED and 23% of involved hospital staff complained of acute poisoning symptoms.[96] Burgess et al reported that 13% of Washington state emergency care facilities had evacuated their ED or another part of the hospital for contamination during a 5-year period. [23] Ghilarducci et al (2000) surveyed Level 1 trauma centers in the United States and reported that only 6% had the necessary equipment required for safe decontamination, less than 36% of emergency medicine staff had received appropriate training in handling the contaminated patient, and 5.6% had experienced injuries to their staff due to contact with contaminated patients during a 1-year period. It is imperative that EDs have plans in place to handle patients who are exposed to potential toxins, provide adequate decontamination facilities, and ensure the safety of the treating medical staff.

Technique

There are a number of key components in the management of a hazardous materials incident and the care of the contaminated patients who present to the ED. [75] These components should include early recognition of a HAZMAT event, rapid activation of a plan to manage contaminated patients, initiation of primary triage, appropriate patient registration, patient decontamination, secondary triage, and final treatment.

First, the ED must be able to recognize that an event has occurred before contaminated patients gain entrance into the health care facility. Communication with local fire, police, and paramedics provides early detection of such events and allows preparation before patients arrive. Security should be arranged to prevent contaminated patients from entering the hospital, and a "lockdown" of the facility should be considered.

Second, the ED should have the authority to activate a plan expeditiously to prepare the decontamination facility and allow appropriate personnel to don personal protective equipment (PPE). If necessary, the hospital disaster plan should be activated quickly at the discretion of the ED attending clinician who is in contact with scene operations and incoming patients. Specific data to determine the appropriate level PPE to maintain hospital worker protection remains limited. Most chemical exposures do not pose a risk of secondary exposure. With unknown chemical and biologic exposures, level B and level A PPE, respectively, is recommended by OSHA. Fortunately, most chemical exposures are known. For those that occur in the workplace, Material Safety Data Sheets can be obtained and either the local poison center or the Agency for Toxic Substances and Disease Registry (ATSDR) can be contacted to obtain advice on what level of protection is appropriate.

Third, appropriate primary triage should occur. Contaminated patients should not enter the ED until proper decontamination has occurred to assure that the hospital staff will not get secondary contamination. Appropriate triage should then occur with experienced personnel performing an initial brief assessment of each patient.

Fourth, a brief sign-in process should capture the patient's name and date of birth with full registration to occur following decontamination. Contaminated clothing and valuables should be placed in an impervious bag to avoid potential off gassing. [54] [120]

Fifth, decontamination should be performed. The hospital ED should have preexisting hazardous materials incident protocols that designate the decontamination area and the triage and decontamination team. The decontamination area should meet several qualifications: (1) it should be secured to prevent spread to other areas of the hospital, (2) the ventilation system should be separate from the rest of the hospital or it should be shut off to prevent airborne spread of contaminants, and (3) provisions must be made to collect the rinsate from contaminated patients to prevent contamination of the facility and water supply. Some hospitals may have designated decontamination rooms. At most facilities, the best place to begin initial treatment and evaluation is outdoors (Fig. 43-19). Portable decontamination facilities are available, but their cost may be prohibitive for many institutions. A practical alternative is to have a warm shower nozzle, soap, and a wading pool available outside the entrance to the ED. A tent or screen can provide privacy. Decontamination should proceed by using soap and copious warm water irrigation. Starting from head to toe, irrigate the exposed skin and hair for 10 to 15 minutes and scrub with a soft surgical sponge with careful attention not to abrade the skin. Irrigate wounds for an additional 5 to 10 minutes with water or saline. Irrigate the eyes for 10 to 15 minutes with saline, directed away from the medial canthus to avoid forcing contaminants into the lacrimal duct. Longer irrigation times may be needed with strongly alkaline substances. Irrigate the nares and the ear canals with frequent suctioning if contamination is suspected. Clean underneath the nails with a brush. Stiff brushes and abrasives should be avoided as they may enhance dermal absorption of the toxin and can produce skin lesions that may be mistaken for chemical injuries. Sponges and disposable towels are effective alternatives.

Secondary triage should occur after decontamination. Patients with major or moderate casualties can then be transferred to areas designated for such cases. Those with minor or no injuries should be sent to appropriate holding areas for further evaluation. Medical care at this stage would then depend on the toxin to which the patient has been exposed and the potential toxicity of that agent.

Figure 43-19 Decontamination of personnel using copious water irrigation.

In order for the ED to care for the contaminated patient, protocols should be in place and regularly rehearsed by the facility. There are template protocols in both peer-reviewed literature and in the government literature. [24] [64] For example, guidelines for managing hazardous materials incidents are available from the Emergency Response and Consultation Branch (E57), Division of Health Assessment and Consultation, Agency for Toxic Substances and Disease Registry, 1600 Clifton Road NE, Atlanta, Georgia 30333.[78]

CONCLUSION

Gastric emptying techniques such as gastric lavage and ipecac-induced emesis are rarely being used to decontaminate the poisoned patient who presents to the ED. There may be future indications for these procedures with specific toxins. At this time the documented risks associated with these procedures should be carefully weighed in light of the rare indications. Activated charcoal as the sole means of gastric decontamination is increasing in popularity, but its efficacy has specific limitations. Protocols for the evaluation and treatment of asymptomatic or minimally symptomatic patients, who have not ingested significantly toxic substances, continue to evolve, and it has been suggested that these patients will do well regardless of therapy. The major issue currently facing the clinician is the choice of gastrointestinal decontamination in the significantly poisoned patient. The choice of decontamination method for these patients must be individualized using both evidence-based medicine and clinical acumen. No patient should undergo any of the available procedures unless it is anticipated that decontamination will provide clinical benefit.

Acknowledgment

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Chapter 44 - Peritoneal Procedures

John A. Marx

Paracentesis and diagnostic peritoneal lavage constitute the two primary intraperitoneal procedures. They are fundamentally similar in purpose and design. However, the former is generally reserved for medical concerns and the latter for traumatic pathology.

DIAGNOSTIC PERITONEAL LAVAGE

Root and colleagues introduced diagnostic peritoneal lavage in 1964. [1] It has withstood the passage of more than 3 decades and remains a mainstay in the management of penetrating torso trauma. Following a blunt mechanism of injury, its greatest utility is as a triage tool in the hemodynamically unstable multiply injured patient. Otherwise, it serves with computed tomography (CT) and with the intent of rapidly discovering or excluding the presence of intraperitoneal hemorrhage, a purpose identical with that of ultrasound (US) in the diagnostic armamentarium of blunt trauma. [2]

While commonly referred to as *diagnostic peritoneal lavage* (DPL), this procedure comprises two distinct components: peritoneal aspiration and peritoneal lavage. Peritoneal aspiration, in which an attempt is made to retrieve free intraperitoneal blood, precedes lavage. A finding of intraperitoneal blood presages intraperitoneal organ injury and precludes the need for subsequent lavage. In the lavage portion, normal saline is introduced by catheter into the peritoneal cavity, recovered by gravity, and analyzed.

Peritoneal lavage can be used as a therapeutic tool in hypothermia and as a means of removing toxins. [3] It has also been used as a diagnostic instrument for suspected intra-abdominal infection and nontraumatic sources of hemorrhage. [4] [5] However, its primary use is as a determinant for the need for laparotomy following trauma, and this chapter focuses on that use.

Indications

Blunt Trauma

Prior to the advent of CT and US, DPL was the sole diagnostic option to physical examination for predicting the need for operative intervention (Table 44-1). It was integral both to the reduction of unnecessary laparotomies and to the discovery of unsuspected and life-threatening intra-abdominal hemorrhage in patients with significant closed head injury. [6] [7]

In a number of respected centers in the United States, DPL continues to be a focal diagnostic instrument. It serves two primary functions. [8] First, it can rapidly determine or exclude the presence of intraperitoneal hemorrhage (Table 44-2). Thus, the patient with a critical closed head injury, the unstable motor vehicle crash victim with multiple potential sources of blood loss, or the patient with pelvic fracture and retroperitoneal hemorrhage can be appropriately routed to life-saving laparotomy. [9] [10] Furthermore, given its exquisite sensitivity, a negative peritoneal aspiration allows the clinician to proceed to alternative management steps and the patient to forego unnecessary laparotomy. Second, DPL can be used in less exigent circumstances as a means of predicting solid or hollow visceral injury requiring laparotomy. [11] [12] In this venue, its sensitivity to the presence of hemorrhage may prompt unnecessary laparotomy in a small percentage of patients with self-limited lacerations of the liver or spleen. [13] [14] [15] [16] CT scan specifically evaluates all intraperitoneal structures as well as the retroperitoneum, a region inaccessible to DPL. Because resolution and the speed with which it can be undertaken have vastly improved, CT has become an invaluable adjunct in the management of blunt trauma. [17] [18] It is most useful in the identification of injury to solid organs with accompanying intraperitoneal hemorrhage and greatly assists nonoperative management of those injuries. Using DPL and CT in a complementary fashion can mitigate the number of unnecessary laparotomies while prompting therapeutic operations in other cases. The ability of CT to discern hollow viscus and pancreatic pathology has improved but remains inconsistent. [19] [20] With regard to hollow viscus injury, it is when serial clinical evaluations cannot be performed that gut perforation leads to preventable mortality. It is for this express scenario that certain authorities recommend the performance of DPL: following a negative CT or particularly one demonstrating free fluid without evidence of solid organ damage. [21] [22]

Experience with US in North America is meager in comparison with that in Western Europe (notably Germany) and Asia (notably Japan). In the past, US in the United States had been used exclusively for the detection and serial examination of traumatic pancreatic pseudocysts. There are two paradigms that have brought US to the forefront. First, this modality has been adopted as the primary triage instrument, in lieu of DPL, for the detection of intraperitoneal hemorrhage on the basis of identifying which pouches and gutters are fluid-filled. [23] [24] [25] [26] Clinical success in this role has been mixed with reported sensitivities for intraperitoneal hemorrhage of 65 to 95%. [27] [28] [29] [30] [31] [32] [33] Additionally, to be useful in this role, a competent technician, interpreter, and equipment must be present in real-time. It has been demonstrated that emergency clinicians and surgeons can be trained in this technique to a level of competence sufficient for this need. [34] In centers that rely upon US, DPL should serve as a reliable study when US equipment is unavailable, the US is technically difficult, or when the results

TABLE 44-1 -- Clinical Indications for Laparotomy after Blunt Trauma

Manifestation	Pitfall
Unstable vital signs with strongly suspected abdominal injury	Alternate sources of shock
Unequivocal peritoneal irritation	Unreliable
Pneumoperitoneum	Insensitive; may be due to pulmonary source or invasive procedures (diagnostic peritoneal lavage, laparoscopy)
Evidence of diaphragmatic injury	Nonspecific
Significant gastrointestinal bleeding	Uncommon, unknown accuracy

From Marx J: Abdominal trauma. In Marx JA, Hockberger RS, Walls RM, et al (eds): Rosen's Emergency Medicine Concepts and Clinical Practice, 5th ed. Philadelphia, Mosby, 2002, p 433.

TABLE 44-2 -- Indications for Diagnostic Peritoneal Lavage Following Blunt Mechanism

Purpose	Circumstance	Alternate or Complementary Diagnostic
Rapidly determine presence of IPH	Hemodynamically unstable multiple blunt trauma	US
Determine presence of organ injury	Suspected or known blunt trauma with unreliable examination:	CT
	• Head injury with altered mental status	
	• Alcohol intoxication	
	• Drug intoxication	
	• Spinal cord injury	
Determine presence of IPH or injury	Multiple trauma patients who require general anesthesia for other injuries	CT, US

CT, computed tomography; IPH, intraperitoneal hemorrhage; US, ultrasound.

of the US are indeterminate, especially when the patient demonstrates hemodynamic compromise.

Second, US can determine injury to solid viscera such as the liver, spleen, kidneys, and pancreas. This requires considerably greater expertise, and in most centers

US has not supplanted CT for this purpose. [35]

DPL is a readily available procedure that can be conducted rapidly in the safe confines of the emergency department (ED). The ability to undertake CT, in particular, or to a lesser extent, US in a similar manner requires careful consideration of clinical circumstances, equipment location, and the capabilities of available personnel (Table 44-3) (Fig. 44-1).[36] [37]

Penetrating Trauma

The advent of DPL was seminal in the promotion of selective management for penetrating abdominal injury. Here its role is more dominant than for blunt trauma due to the far greater likelihood of occult injury to hollow viscera and the diaphragm following a penetrating mechanism. [38] [39]

Instruments and missiles may penetrate the abdominal cavity via the anterior abdominal wall, flank, back, or low chest. [40] The intraperitoneal space is vulnerable if penetration occurs as high as the fourth intercostal space anteriorly and the sixth or seventh laterally and posteriorly, as the diaphragm may rise to these levels in the expiratory phase of respiration. [41] Coincident thoracic penetration has occurred in up to 46% of abdominal injuries. [42] [43] [44] The likelihood of retroperitoneal

TABLE 44-3 -- Diagnostic Studies in Blunt Abdominal Trauma

Scenario	Study Purpose	Primary Study	Alternate/Compensatory
Hemodynamically unstable			
General	IPH	DPL, US	—
Pelvic fracture	IPH	DPL,* US	—
Hemodynamically stable			
General	OI†	CT	DPL, US‡
Nonoperative management§	OI	CT?	DPL¶ , US‡
CHI	OI, HVI	DPL¶ , CT?	US‡
BAD	IPH	DPL, US	CT£

BAD, blunt aortic disruption; CHI, closed head injury; CT, computed tomography; DPL, diagnostic peritoneal lavage; HVI, hollow viscus injury; IPH, intraperitoneal hemorrhage; OI, organ injury; US, ultrasound.

From Marx J: Abdominal trauma. In Marx JA, Hockberger RS, Walls RM, et al (eds): Rosen's Emergency Medicine Concepts and Clinical Practice, 5th ed. St. Louis, Mosby, 2002, p 431.

*+ peritoneal aspirate mandates laparotomy, + red blood cell count only, warrants attention to pelvic fracture.
†Specific organ damage or fluid/blood suggesting injury.
‡US for OI much less reliable than for IPH.
§Institutional capability should be carefully considered.
?CT less reliable for HVI than for solid visceral injury.
¶Complementary to CT if HVI suspected.
£May be more appropriate if can be rapidly acquired or if CT primary study for BAD.

injury increases when the entry site is over the flank or back, but the prospect of intraperitoneal pathology remains considerable with cited incidences of 21 to 44% for the flank and 7 to 14% for the back (Table 44-4).[45] [46] [47]

Stab wounds.

Because only one fourth to one third of patients who sustain stab wounds to the anterior abdomen require laparotomy, diagnostic algorithms are used to decrease the rate of unnecessary operation.[38] [43] [48] [49] An optimal approach would not sacrifice sensitivity for morbid intraperitoneal injury. A pathway using a combination of clinical mandates, local wound exploration, and DPL is well established (Fig. 44-2). [50] These clinical mandates are reasonably accurate predictors of significant intraperitoneal injury (Table 44-5). Thus, the presence of one or more mandates suggests the need for urgent laparotomy and precludes the undertaking of other diagnostic studies.

DPL fills three roles in the evaluation of patients with abdominal stab wounds (Table 44-6): (1) rapid determination of the presence of hemoperitoneum, (2) discovery of intraperitoneal injury requiring operation in stable patients, and (3) the establishment of diaphragmatic violation. As is the case in blunt trauma patients, DPL can be invaluable as a rapid triage tool when the source of hemodynamic instability is not known. Pericardial tamponade, intrathoracic hemorrhage, and intraperitoneal hemorrhage may be contributory to hemodynamic instability or wholly causal. Again, as for blunt trauma evaluation, US is the only diagnostic modality for

Figure 44-1 Blunt abdominal trauma algorithm. BAT, blunt abdominal trauma; CT, computed tomography; D/C, discharge; DPA, diagnostic peritoneal aspiration; DPL, diagnostic peritoneal lavage; IP, intraperitoneal; IPH, intraperitoneal hemorrhage; LAP, laparotomy; SPE, serial physical examinations; US, ultrasound. *Determined by unequivocal free intraperitoneal fluid on ultrasound or positive peritoneal aspiration on diagnostic peritoneal lavage. †Can be unreliable because of closed head injury, intoxicants, distracting injury, or spinal cord injury. ‡One or more studies may be indicated. §Need for laparotomy is based on clinical scenario, diagnostic studies, and institutional resources. *(From Marx J: Abdominal Trauma. In Marx JA, Hockberger RS, Walls RM, et al. [eds]: Rosen's Emergency Medicine Concepts and Clinical Practice, 5th ed. St. Louis, CV Mosby, 2002, p 432.)*

intraperitoneal hemorrhage that is competitive for this role and carries the added advantage of scanning for intrapericardial and intrathoracic hemorrhage as well. [44] In the determination of injury following stab wounds, DPL carries 90% accuracy. [51] [52] [53] Serial examinations,[54] [55] [56] CT, and laparoscopy[57] [58] [59] [60] are alternative modalities in specific circumstances and centers.[61] Diaphragmatic rents created by stab wounds are generally small; thus, at the outset, they do not create apparent clinical or radiologic abnormalities. [62] [63] However,

TABLE 44-4 -- Injury Likelihood by Entry Site

	Intraperitoneal	Retroperitoneal	Diaphragm
Anterior abdomen	++	+	+
Flank	+	++	+
Back	+	++	+
Low chest	+	+	++

From Marx JA: Diagnostic peritoneal lavage. In Ivatury RR, Cayten CG (eds): The Textbook of Penetrating Trauma. Baltimore, Williams & Wilkins, 1996, p 336.

morbidity due to delayed herniation of bowel is common and substantive. [64] DPL is currently the most sensitive means of discerning this injury in the immediate

post-trauma phase.[51] Physical examination and CT are notoriously insensitive. Laparoscopy has demonstrated promise in experienced hands. [57] [58] For these small diaphragmatic wounds, the advanced radiologic tool called magnetic resonance imaging (MRI) may be diagnostic, but due to safety and accessibility concerns, it should be reserved for the non-acute phase of management.

Gunshot wounds.

Multiple organ injury is the rule following gunshot wounds, and mortality is significantly greater when compared with that for stab wounds. [65] The diagnostic approach is more conservative for gunshot wounds because the likelihood of intraperitoneal injury requiring operative intervention exceeds 95% when the projectile has entered the intraperitoneal cavity (Fig. 44-3).[66] [67] [68] If clinical mandates are met (see Table 44-5) or if peritoneal violation has occurred, most centers proceed to laparotomy.[50] One series,

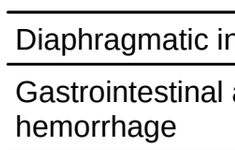

Figure 44-2 Anterior abdomen stab wound algorithm. *Plain films, ultrasound, laparoscopy, and computed tomography (CT) can assess peritoneal entry. †Laparoscopy or CT can complement or replace diagnostic peritoneal lavage. ‡Expectant management of injuries is infrequently attempted. D/C, discharge; DPL, diagnostic peritoneal lavage; LAP, laparotomy; LWE, local wound exploration; PEx, physical examination. *(From Marx J: Abdominal Trauma. In Marx JA, Hockberger RS, Walls RM, et al. [eds]: Rosen's Emergency Medicine Concepts and Clinical Practice, 5th ed. St. Louis, CV Mosby, 2002, p 427.)*

however, cited intra-abdominal injury in 70% to 80% of cases, supporting the contention that nonoperative management could be applied to a substantial percentage of patients. [69] DPL is reserved for two circumstances: (1) the wound tract is neither obviously superficial nor intraperitoneal, and (2) penetration is to the low chest, where diaphragmatic injury is more likely, yet the possibility of intraperitoneal injury exists.

Contraindications

Diagnostic peritoneal lavage can be undertaken in virtually any patient irrespective of age, pregnant state, or comorbid illness. Adjustment of the technique and site of performance allows relative contraindications to be overcome. Relative contraindications include prior abdominal surgery or infections,

TABLE 44-5 -- Clinical Indications for Laparotomy Following Penetrating Trauma

Manifestation	Premise	Pitfall
Hemodynamic instability	Major solid visceral or vascular injury	Thorax, mediastinum
Peritoneal signs	Intraperitoneal injury	Unreliable, especially immediately postinjury
Evisceration	Additional bowel, other injury	No injury in ¼ to ? of stab wound cases
Diaphragmatic injury	Diaphragmatic herniation	Rare clinical, radiographic findings
Gastrointestinal and vaginal hemorrhage	Proximal gut or uterine injury	Uncommon, unknown accuracy
Impalement in situ	Vascular impalement	High operative risk, pregnancy
Intraperitoneal air	Hollow viscus perforation	Insensitive; may be caused by intraperitoneal entry only or be due to cardiopulmonary source

Modified from Marx JA: Diagnostic peritoneal lavage. In Ivatury RR, Cayten CG (eds): The Textbook of Penetrating Trauma. Baltimore, Williams & Wilkins, 1996.

obesity, coagulopathy, and second- or third-trimester pregnancy. The sole absolute contraindication is when clinical mandates for urgent laparotomy already exist.

Technique

Preliminary Steps

The stomach and bladder should preferably be decompressed to prevent inadvertent injury. The patient is kept supine and administered sedation and analgesia as appropriate (see Chapter 34). This is most relevant when the semi-open technique is used and a trocar is passed through the peritoneum. DPL should be performed according to compliance with standards for body fluid precautions (see Chapter 71). Prior to making the skin incisions described later, the site of placement should be prepped with standard skin antiseptics (e.g., povidone-iodine) and appropriately draped. The operator should observe sterile precautions throughout the procedure. Prophylactic antibiotics are not indicated for routine DPL because local and systemic infections are rare. [52] [70]

Local anesthesia (1% lidocaine *with* epinephrine) should be infiltrated liberally into the area for incision and dissection (Fig. 44-4). It is best to delay the incision for more than 30 seconds following local anesthetic infiltration to permit local vasospasm, which minimizes wound bleeding during the procedure. Standard equipment for an open peritoneal lavage catheter placement is shown in Figure 44-5 .

Catheter Placement

DPL is performed via two basic methods: open and closed. The two open techniques are *semi-open* and *fully open*, and they typically require an assistant. DPL is clearly within the diagnostic armamentarium of the emergency clinician and surgeon. It may be undertaken by either or both in keeping with clinical policies established at the particular trauma center.

Open technique.

In the semi-open method, sharp then blunt dissection using a No. 11 scalpel and Army-Navy retractors, respectively, proceeds to the *rectus fascia* (Fig. 44-6*A* and *B*). The skin incision should be 4 to 6 cm in length. When the selected site is infraumbilical in the midline, the operator should reach the *linea alba*; its crossing bands of crural fibers may be apparent.[71] A small 2- to 3-mm opening is then made in the *linea alba*, preferably with a No. 15 scalpel blade (Fig. 44-6*C*). (The operator will notice a tough, gritty sensation when cutting the *linea alba* with the scalpel.) Towel clips can be placed through this opening to grasp each side of the *rectus fascia* (Fig. 44-6*D*). These two towel clips are then lifted to allow safe advancement of the catheter via trocar in a 45° to 60° caudad orientation through the peritoneum and into the peritoneal cavity (Fig. 44-6*E* and F).[72] [73]

TABLE 44-6 -- Indications for DPL Following Penetrating Mechanism

Purpose	Circumstance	Alternate or Complementary Diagnostic
Rapidly determine presence of IPH	Hemodynamically unstable SW or GSW to low chest, abdomen, flank, back	Ultrasound
Determine presence of organ injury	SW or GSW to low chest, abdomen, flank, back	Serial clinical examinations, CT, laparoscopy
Determine diaphragmatic violation	SW or GSW to low chest, upper abdomen	Laparoscopy

CT, computed tomography; GSW, gunshot wound; IPH, intraperitoneal hemorrhage; SW, stab wound.

To decrease the likelihood of penetrating underlying viscera, some operators advocate holding the fingers low on the catheter-trocar instrument such that on entering the abdominal peritoneum, the fingers will prevent deep penetration. Excessive pressure during trocar penetration is a common error. Steady "one-finger pressure" applied to the handle is sufficient to "pop" through the fascia and peritoneum. After controlled peritoneal penetration of 0.5 to 1.0 cm in the midline, the trocar is retracted 1.0 to 2.0 cm within the catheter, and the catheter is carefully advanced toward the pelvis. Some operators prefer to advance the catheter toward the right or left side of the pelvis. Use of a

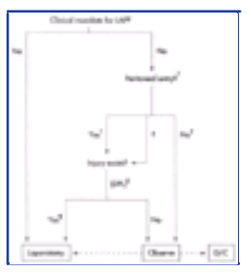

Figure 44-3 Gunshot wound algorithm. D/C, discharge; DPL, diagnostic peritoneal lavage; LAP, laparotomy. *Can be assessed by missile path, plain films, local wound exploration, ultrasound, and laparoscopy. †Most centers proceed to laparotomy if peritoneal entry is suspected. ‡Patients with documented superficial and low-velocity injuries can be discharged; unknown depth or high-velocity injuries require further tests or observation. §Serial examinations, laparoscopy, or a computed tomography scan can complement or replace diagnostic peritoneal lavage. ¶Expectant management of injuries caused by gunshot wounds is rarely attempted. *(From Marx J: Abdominal Trauma. In Marx JA, Hockberger RS, Walls RM, et al. [eds]: Rosen's Emergency Medicine Concepts and Clinical Practice, 5th ed. St. Louis, CV Mosby, 2002, p 430.)*

slight twisting motion during advancement may minimize visceral or omental injury.

The *fully open* technique extends the semi-open technique by one step. The opening in the *linea alba* is lengthened, and the peritoneum is opened to allow direct visualization during catheter placement into the peritoneal cavity.

The two open techniques can be accomplished with a single technician, but an additional pair of hands is advised in order to assist in retraction and handling of instruments. The fully open method is the more technically demanding and time-consuming. It is reserved for clinical circumstances in which the closed or even semi-open technique is not deemed safe or has been attempted and failed. These include pelvic fracture, pregnancy, prior abdominal surgery or infections, and obesity.

Closed technique.

With closed techniques, the catheter is introduced into the peritoneal space in a blind percutaneous fashion. [74] Formerly, this was accomplished via trocar. This has been abandoned in favor of the much safer but equally simple Seldinger (guide wire) method, in which a small-gauge guide needle is inserted into the peritoneal cavity in the infraumbilical midline (Fig. 44-7*A*). A flexible wire is then passed through the needle (Fig. 44-7*B*), and the needle is removed, allowing the over-the-wire placement of a soft catheter into the peritoneal cavity. A stab with a No. 11 scalpel at the entry site of the wire allows easier passage of the catheter through the abdominal wall (Fig. 44-7*C* and *D*). Rotating the catheter while pushing it over the guide wire is recommended to facilitate entry into the peritoneal cavity. The catheter is placed into the right or left pelvic gutter.

Figure 44-4 Local anesthesia is introduced at incision or puncture site. The patient is supine with the head of the bed elevated slightly.

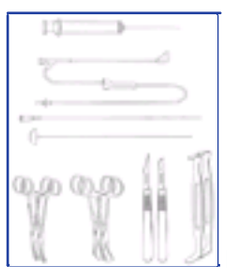

Figure 44-5 Standard equipment used for open lavage technique.

The wire is then withdrawn, and aspiration conducted, followed by lavage when necessary. The guide wire should always be controlled to avert intra-abdominal migration of the wire. Proponents of the guide wire technique promote its ease and rapidity. [75] [76] [77] [78] [79] Those who prefer the semi-open method argue that the time to peritoneal aspiration, the more critical interval, is minimally different, and that this method may have fewer complications and thus be more accurate than the guide wire technique. [80] [81] [82] [83] Note that time to aspiration for semi-open or closed approaches should entail no more than 2 to 5 minutes.

Site

The optimum location for DPL is at the infraumbilical ring, at the inferior border of the umbilicus (Table 44-7). Here, between the *rectus abdominis* muscles, there is adherence of the peritoneum and relative lack of vascularity and preperitoneal fat. [71] Closed DPL should always be conducted here. In the event of second- or third-trimester pregnancy, a suprauterine approach is used. If there is midline scarring, a fully open technique at the lateral border of the *rectus abdominis* in the left lower quadrant may be necessary. The left is preferred to avoid later confusion about whether an appendectomy has been performed. It is interesting to note that Moore and colleagues found no increase in complications or misclassified lavages when the closed technique was used in a small series of patients with prior abdominal surgery. [84] In the presence of a pelvic fracture, a fully open supraumbilical approach is recommended. This greatly decreases the likelihood of passing the catheter through retroperitoneal hematoma that has dissected from the fracture anteriorly and across the abdominal wall. [85] In penetrating trauma, DPL should not be conducted through the stab or missile entry site. Such an approach may contaminate the intraperitoneal cavity and potentially exacerbate abdominal wall bleeding, which could in turn lead to a false-positive result.

Aspiration and Lavage

Once the catheter has been placed successfully into the peritoneal cavity and the right-angle adapter, extension tubing, and a non-Luer-Lok syringe have been attached, aspiration is attempted (Fig. 44-8). The recovery of 10 mL of blood is considered positive, and the procedure terminated. In penetrating trauma, the acquisition of lesser amounts may be meaningful because of the tendency for the diaphragm and bowel to hemorrhage minimally when injured. However, there are no

established rules in this regard.

If little to no blood is aspirated, the peritoneal cavity is lavaged with either normal saline or lactated Ringer's solution (Fig. 44-9). A blood pressure cuff or blood infusion pump can be applied to the plastic intravenous (IV) bag to speed the influx (i.e., decrease lavage time), but they are rarely needed. Large-bore infusion tubing (e.g., urologic irrigation tubing sets, such as the Abbott No. 6544 cystoscopy/irrigation set) also shortens fluid influx time. The normal amount is 1 L in adults or 15 mL/kg in children. When possible, the patient is rolled or shifted from side to side after infusion to increase mixing. The IV bag or bottle is placed on the floor

(or below abdominal level), and the fluid is allowed to return by gravity.

The fluid may not continue to return because of several factors. Some IV tubing contains a one-way valve; if tubing with a valve was used in error, valveless tubing must be reinserted into the IV bag. Another reason for poor return is inadequate suction. This problem can be corrected by insertion of a needle into the second opening at the bottom of the IV bag or into the head of the IV bottle for aspiration of 10 mL of air. Alternatively, the catheter may be adherent to the peritoneum. If so, relieving some of the pressure in the IV bottle or gently wiggling and twisting the catheter as well as applying abdominal pressure may aid flow return.

It is generally accepted that the return of greater than or equal to 700 mL in the adult is adequate for interpretation of findings. However, as little as 10 to 20% of the infusate may give a representative sample for both gross and microscopic determinations. Only 10 mL of fluid from the return need be sent to the laboratory for cell count analysis; another 10 mL can be sent for enzyme analysis (see interpretation later in this chapter). Some operators prefer to leave the dialysis catheter in place until the returned fluid is analyzed. The clinician may wish to re-lavage when the initial results are borderline or an occult bowel perforation is suspected.

Complications

Local and Systemic

Local wound complications, including infection, hematoma, and dehiscence, have occurred in only 0.3% of patients in 2 large series. [51] [86] [87] Dehiscence with evisceration is likely an even more rare condition. [88] Systemic infection has been described rarely (Table 44-8).

Intraperitoneal

Iatrogenic intraperitoneal injury can be inflicted by the trocar, wire, and, rarely, the catheter. Virtually any structure in the peritoneal cavity can be breached, including the small and large bowel, the bladder, and major vessels. Typically, if the needle is the culprit, and even if the trocar is responsible, injury to these structures is minimal and self-limiting, and observation of the patient is sufficient.

Technical Failure

Inability to recover peritoneal aspirate or lavage fluid can result in a false-negative interpretation. This can occur in several circumstances. It follows unwitting placement of the catheter into the preperitoneal space, which is less likely to occur with either open technique. Compartmentalization of fluid by adhesions or obstructing omentum can impede egress of fluid. When a fully open supraumbilical or suprauterine technique is used, the catheter may be too short to access the depths of the intraperitoneal cavity. Finally, large diaphragmatic tears typical of blunt pathophysiology allow flow of lavage fluid from the intraperitoneal to the thoracic cavity. Saunders and colleagues compared percutaneous DPL versus the open technique in a prospective randomized trial. [89] Fluid obtained by the two techniques had similar test performance for intra-abdominal pathology. The open technique took on average more than 4 minutes longer to perform, but the percutaneous approach had an 11.2% (versus 3.8% with the open approach) technical failure rate.

False-positive findings can occur in two ways. First, iatrogenic misadventure can be responsible. Second, in penetrating trauma, particularly stab wounds, bleeding from the abdominal wall injury site into the peritoneal cavity can lead to positive findings when no injury to intraperitoneal structures has occurred. [52]

Interpretation

Gross Blood

The recovery of =10 mL blood via aspiration is considered a positive finding. Lesser-volume aspirates are generally discarded and are not factored into lavage analysis. Grossly bloody aspirates are typically indicative of solid visceral or vascular injury, with a positive predictive value >90%. [86] [90] Aspiration of blood is responsible for approximately 80% of true-positive DPL findings in blunt trauma and for 50% of those following stab wounds. [51]

A positive aspiration in the blunt trauma patient who is hemodynamically stable or has been resuscitated to apparent stability need not mandate urgent operation. Unnecessary laparotomy will occur if there has been minimal and self-limited damage to the liver, spleen, bowel serosa, or mesentery. [17] [91] In this situation, CT and clinical indicators should be used in concert with the DPL findings.

Red Blood Cell (RBC) Count

The recommended RBC threshold varies according to mechanism and, in the case of stab wounds, the external site of injury (Table 44-9). The optimum criterion will deliver excellent sensitivity, a high positive-predictive value, and, therefore, a minimum incidence of unnecessary laparotomy. Negative laparotomy incurs a prolongation of hospitalization and increases the cost of care, in addition to creating the potential for procedural complications. [92] [93] RBC counts greater than 10^5 /mm^3 (i.e., >10^5 /µL) are generally considered positive with a blunt mechanism or following stab wounds to the anterior abdomen, flank, or back. Counts of 20,000 to 100,000/mm^3 should be considered indeterminate. [51] [53] [94] [95] For stab wounds to the low chest, where the diaphragm is at increased risk of injury, the RBC criterion should be lowered to 5000/mm^3 to maximize sensitivity for isolated injury to this structure. [43] [51] [96] [97] With gunshot wounds to the abdomen or low chest, the same criterion is applied. This is intended to increase the sensitivity of the test, because intraperitoneal entry by a missile carries a likelihood of intraperitoneal injury of greater than or equal to 95%. [43] [69] [98] [99] An uncomplicated DPL should not create more than several hundred to several thousand RBCs in the peritoneal lavage fluid.

The incidence of false-positive RBC interpretation in the setting of pelvic fracture is considerable. However, aspiration of free blood in the critical pelvic fracture patient predicts active intraperitoneal hemorrhage in greater than 80% of cases. [100] A positive RBC count should generally prompt corroboration or refutation of intraperitoneal injury by CT. In this fashion, needed pelvic angiography and embolization will not be delayed unnecessarily should active intraperitoneal bleeding not be found (Fig. 44-10).

White Blood Cell (WBC) Count

An inflammatory peritoneal response occurs to a multitude of stimuli, including stool, blood, and enzymes. [101] The WBC count in lavage effluent was formerly touted to predict small

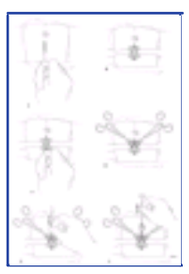

Figure 44-6 *A,* After bladder decompression (generally by Foley catheter placement), a 4- to 6-cm long vertical infraumbilical incision is made with a No. 11 scalpel. *B,* Blunt dissection using Army-Navy retractors is carried down to the *rectus fascia*. Crossing bands of crural fibers may be seen. *C,* A 2- to 3-mm incision is made through the *rectus fascia* in the midline (*linea alba*) with a No. 15 scalpel. *D,* Towel clips grasp each side of the *rectus fascia*, which is lifted prior to insertion of the trocar and diagnostic peritoneal lavage (DPL) catheter. *E,* The trocar with DPL catheter is passed at a 45° caudad angle into the fascial opening and through the peritoneum. Note that in the fully open method, the incision in the *rectus fascia* is extended, the peritoneum is directly visualized and incised, and the catheter alone is placed into the peritoneal cavity. *F,* As soon as the peritoneum has been entered, only the catheter is gently advanced into the peritoneal cavity while the trocar is withdrawn. It is often helpful to advance the catheter with a slight twisting motion and to direct it toward either the right or left pelvic gutter.

Figure 44-7 *A,* For the closed diagnostic peritoneal lavage (DPL) method using a guide wire (Seldinger technique), the needle is inserted into the peritoneal cavity in the midline just below the umbilicus and aimed slightly caudad. *B,* The flexible guide wire is passed through the needle and into the peritoneal cavity. Ideally, the wire should be directed toward the right or left pelvic gutter. The needle is withdrawn while the wire is stabilized with the operator's free hand at all times. *C,* A stab incision is made with a No. 11 scalpel immediately below the wire to permit easier passage of the DPL catheter. *D,* The DPL catheter is directed over the wire and into the peritoneal cavity using a slight twisting motion. The wire is stabilized by the operator at all times and removed after catheter placement. The catheter should be directed toward the right or left pelvic gutter when advanced.

TABLE 44-7 -- Preferred Site of Diagnostic Peritoneal Lavage

Clinical Circumstance	Site	Method
Standard adult	Infraumbilical midline	C or SO
Standard pediatric	Infraumbilical midline	C or SO
Second- and third-trimester pregnancy	Suprauterine	FO
Midline scarring	Left lower quadrant	FO
Pelvic fracture	Supraumbilical	FO
Penetrating trauma	Infraumbilical midline*	C or SO

C, Closed; FO, fully open; SO, semiopen.
*The stab wound or gunshot wound site should be avoided.

bowel injury but has since been proven unreliable.[102] It is insensitive in the immediate postinjury period, as 3 to 5 hours are necessary before the test becomes positive (Table 44-10).[103] [104] Moreover, a positive finding is likely to be falsely so. [103] [105] Therefore, the WBC level in and of itself should not determine the need for laparotomy.

Enzymes

Alkaline phosphatase is contained in intramural small bowel as well as in hepatobiliary secretions released into the proximal intestine. Amylase is contained in the latter only. Perforation of small bowel allows access of these two markers into the peritoneal cavity, where they can be recovered by peritoneal lavage. [106] [107] [108] While levels of the two markers usually rise in tandem, lavage amylase has been shown to be a more accurate marker than lavage alkaline phosphatase (see Table 44-10). In contradistinction to the WBC count, these tests will be positive in the immediate postinjury period. However, they may not be economical if used on a mandatory rather than a selective basis. Neither is helpful in discerning the presence of pancreatic pathology.

Miscellaneous

Routine bile staining, Gram stain, and microscopy to identify vegetable fibers are rarely productive and are of untested accuracy. Deck and Porter have reported that finding urine in the lavage fluid as evidenced by a straw color and creatinine in the peritoneal fluid should suggest an intraperitoneal bladder or collecting system injury.[109]

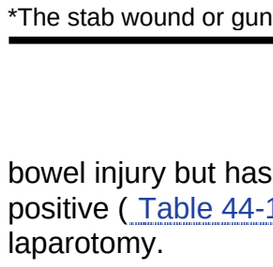

Figure 44-8 After attachment of the right angle connector and extension tubing, aspiration of the peritoneum is attempted.

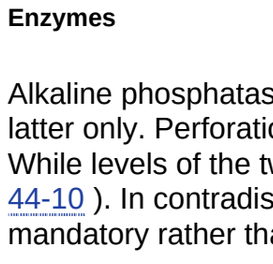

Figure 44-9 If the aspiration is negative, normal saline or Ringer's lactate solution is instilled through the catheter. *The IV tubing should have no valves in place.* After infusion, the fluid bag is placed on the floor and allowed to fill with peritoneal effluent via gravity.

Conclusion

DPL remains an invaluable diagnostic instrument in trauma. It should be used in common-sense fashion. Laboratory parameters are guidelines and should not be embraced to

TABLE 44-8 -- Diagnostic Peritoneal Lavaage Complications

Category	Comments
Local and systemic	
Hematoma-incision site	Local wound care
Dehiscence-incision site	Local wound care
Local wound infection	As indicated
Systemic infection	As indicated
Intraperitoneal injury	
Bowel	Observe, usually self-limited
Bladder	Observe, usually self-limited
Vascular	Observe, usually self-limited
Technical failure	
INABILITY TO RECOVER FLUID*	
Preperitoneal catheter placement	Repeat DPL
Compartmentalization of fluid	US, CT
Obstructed catheter	Gentle catheter manipulation
Diaphragm injury	Reverse Trendelenburg; consider US, CT
"Short" catheter (supraumbilical or suprauterine approach)	Trendelenburg
INTRAPERITONEAL HEMORRHAGE†	
Iatrogenic injury	As indicated by clinical markers
Stab wound abdominal wall bleed	As indicated by clinical markers
Pelvic fracture (RBC count)	Complementary CT

CT, computed tomography; DPL, diagnostic peritoneal lavage; RBC, red blood cell; US, ultrasound.

*May lead to false-negative DPL
†May lead to false-positive DPL

TABLE 44-9 -- Diagnostic Peritoneal Lavage Red Blood Cell Criteria (per mm^3)

	Positive	Indeterminate
Blunt trauma	100,000*	20–100,000
Stab wound		
Anterior abdomen	100,000	20,000–100,000
Flank	100,000	20,000–100,000
Back	100,000	20,000–100,000
Low chest	5000	1000–5000
Gunshot wound	5000	1000–5000

From Marx J: Abdominal trauma. In Marx JA, Hockberger RS, Walls RM, et al (eds): Rosen's Emergency Medicine Concepts and Clinical Practice, 5th ed. St. Louis, Mosby, 2002, p 425.

*In a hemodynamically stable patient with pelvic fracture and positive or equivocal red blood cell count, computed tomography should be obtained to corroborate or refute intraperitoneal injury.

the exclusion of pertinent clinical features. CT, US, or both can serve in lieu of or in addition to DPL in its various roles. [110] Optimal strategies depend largely on the capability of an institution's resources and personnel in each clinical scenario.

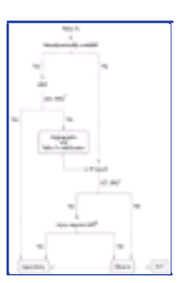

Figure 44-10 Pelvic fracture algorithm. CT, computed tomography; D/C, discharge; DPA, diagnostic peritoneal aspiration; DPL, diagnostic peritoneal lavage; IP, intraperitoneal; IPH, intraperitoneal hemorrhage; LAP, laparotomy; Pelvic Fx, pelvic fracture; US, ultrasound. *Determined by unequivocal free intraperitoneal fluid on ultrasound or positive peritoneal aspiration on diagnostic peritoneal lavage. †One or more studies may be indicated. ‡Need for laparotomy is based on clinical scenario, diagnostic studies, and institutional resources. *(From Marx J: Abdominal Trauma. In Marx JA, Hockberger RS, Walls RM, et al [eds]: Rosen's Emergency Medicine Concepts and Clinical Practice, 5th ed. St. Louis, CV Mosby, 2002, p 434.*

PARACENTESIS

Ascites connotes an abnormal accumulation of fluid within the peritoneal cavity. The word derives from the Greek *askos,* which means bag or sack. It is a symptom with important diagnostic, therapeutic, and prognostic implications.

Therapeutic abdominal paracentesis is one of the oldest medical procedures, dating to approximately 20 BC. Paracentesis was first described in modern medical literature by Saloman at the beginning of this century, and it became a valued decompressive therapy. [111] With the advent of diuretics in the early 1950s, paracentesis fell out of favor as a treatment option. Controlled clinical trials in the late 1980s up to the present have restored its reputation by demonstrating the safety and efficacy of large-volume paracentesis in adults and children. [112] [113] [114] [115] [116] [117] [118] Because this mode is invasive and consumes clinician hours, it is generally reserved for the treatment of patients with chronic ascites who have tense ascites or whose condition is refractory to diuretic therapy. [115] [119] However, paracentesis remains an important diagnostic agent for patients with new-onset ascites or to determine the presence

TABLE 44-10 -- Diagnostic Peritoneal Lavage Non-Red Blood Cell Criteria

	Positive	Indeterminate
LAM (IU/L)	=20	10–19
LAP (IU/L)	=3	NA
WBCs (per mm^3)	>500	250–500

LAM, lavage amylase; LAP, lavage alkaline phosphatase; NA, not applicable; WBCs, white blood cells.

From Marx JA: Diagnostic peritoneal lavage. In Ivatury RR, Cayten CG (eds): The Textbook of Penetrating Trauma. Baltimore, Williams & Wilkins, 1996, p 337.

of worrisome conditions, notably infection, in those with preexistent ascites. [120] [121]

Clinical Features

Determination of Ascites

Small amounts of ascites may be asymptomatic. Larger collections typically cause a sense of abdominal fullness, anorexia, early satiety, and perhaps nausea and abdominal pain. Considerable accumulations create symptoms of respiratory distress by virtue of restricting lung capacity. [122]

The most predictive history and physical findings for *excluding* the diagnosis of ascites are the absence of ankle swelling and increased abdominal girth and the inability to demonstrate bulging flanks, flank dullness, or shifting dullness. [123] [124] Positive predictors for the diagnosis are a positive fluid wave, shifting dullness, or peripheral edema. [125] [126]

Patients who lack obvious clinical markers may benefit from the performance of ultrasonography, which can discern the presence of as little as 100 mL fluid. [127] Endoscopic-guided ultrasound may discover just 10 mL. It is more sensitive than computed tomography in this respect and can assist in the identification of malignancy. [128] Additionally, it is a useful adjunct to determine the location of fluid that may be compartmentalized by preexistent infection or surgical adhesions.

Differential Diagnosis

The etiologies of ascites can be categorized in several ways. On a structural basis, these are divided into diseases of the peritoneum and diseases not involving the peritoneum. The former group includes infections, neoplasms, collagen vascular diseases, and idiopathic causes. The latter includes cirrhosis, congestive heart failure, nephrotic syndrome, protein-losing enteropathy, malnutrition, myxedema, pancreatic disease, ovarian disease, chylous effusion, Budd-Chiari syndrome, and hepatic venous occlusive disease. [121] [129] [130] Pathophysiologic categories are found in Table 44-11 . In this country, parenchymal liver pathology is overwhelmingly the most likely cause. Within this group, alcoholic liver disease is responsible for approximately 80% of cases (Table 44-12). [131] [132] Finally, ascites can be classified on the basis of a serum-ascites albumin gradient, that is, the difference between albumin values obtained simultaneously from serum and ascites samples (Table 44-13). [133]

Indications and Contraindications

Therapeutic paracentesis may be undertaken in the emergency setting to relieve the cardiorespiratory and gastrointestinal

TABLE 44-11 -- Pathophysiologic Classification of Ascites

I. Elevated hydrostatic pressure
A. Cirrhosis
B. Congestive heart failure
C. Constrictive pericarditis
D. Inferior vena cava obstruction
E. Hepatic vein obstruction (Budd-Chiari syndrome)
II. Decreased osmotic pressure
A. Nephrotic syndrome
B. Protein-losing enteropathy
C. Malnutrition
D. Cirrhosis or hepatic insufficiency
III. Fluid production exceeding resorptive capacity
A. Infections
1. Bacterial
2. Tuberculosis
3. Parasitic
B. Neoplasms

From Runyon BA: Diseases of the peritoneum. In Wyngaarden JB, Smith LH (eds): Cecil Textbook of Medicine, 18th ed. Philadelphia, WB Saunders, 1988, pp 790–793.

manifestations of tense ascites.[134] [135] [136] Diagnostic paracentesis is indicated in any patient whose ascites is of new onset or to disclose the presence of infection in patients with known or suspected ascites, particularly in the context of alcohol-related cirrhotic liver disease. [137] [138] Diagnostic paracentesis is also useful in the management of the AIDS patient, in whom the etiology of ascites will be non-AIDS related in three-quarters of cases. [139]

There are few relative contraindications to abdominal paracentesis. Certain systemic and anatomic risks should be considered.

Systemic

Given the predominance of alcohol-related cirrhotic liver disease as the cause for ascites, as many as two-thirds to three-quarters of patients subjected to paracentesis will have a coagulopathy. However, the only prospective study to evaluate the complications of paracentesis determined that transfusion-requiring abdominal hematomas occurred in less than 1% of cases despite the fact that 71% of patients had an abnormal prothrombin time. [140] Because transfusion-requiring hematoma is so unlikely, even in this population, prophylactic administration of fresh frozen plasma or platelets imposes

TABLE 44-12 -- Causes of Ascites*

Cause	% of Patients
Parenchymal liver disease	80
"Mixed"	5
Malignancy	10
Heart failure	5
Tuberculosis	2
Pancreatic	1
Nephrogenous ("dialysis ascites")	<1
Chlamydia	<1
Nephrotic	<1
Surgical peritonitis in the absence of liver disease	<1

From Runyon BA: Ascites and spontaneous bacterial peritonitis. In Sleisenger MH, Fordtran JS (eds): Gastrointestinal Disease: Pathophysiology/Diagnosis/Management, 5th ed. Philadelphia, WB Saunders, 1993, p 1977.

*Based on a series of 1500 paracenteses performed in a predominantly inpatient hepatology/general internal medicine setting (B.A. Runyon, unpublished observations).

TABLE 44-13 -- Classification of Ascites by Serum-Ascites Albumin Concentration Gradient

High gradient (=1.1 g/dL)
Cirrhosis
Alcoholic hepatitis
Cardiac ascites
Massive liver metastases
Fulminant hepatic failure
Budd-Chiari syndrome
Portal vein thrombosis
Veno-occlusive disease
Fatty liver of pregnancy
Myxedema
Mixed ascites
Low gradient (<1.1 g/dL)
Peritoneal carcinomatosis
Tuberculous peritonitis
Pancreatic ascites
Biliary ascites
Nephrotic syndrome
Serositis in connective tissue diseases

From Runyon BA: Ascites. In Schiff L, Schiff ER (eds): Diseases of the Liver, 7th ed. Philadelphia, Lippincott-Raven, 1993, p 997.

considerable cost, in addition to the risk of post-transfusion hepatitis, with little net gain. [141] These data countermand older and conservative recommendations to administer platelets to patients with levels less than 50,000/mm^3 (i.e., <50 × 10^9 /L), or to give fresh frozen plasma to those with a prothrombin time exceeding 20 seconds (1.5 times the therapeutic level).[142] These agents should be reserved for clinically evident fibrinolysis and disseminated intravascular coagulation.

Anatomic

Structural impediments to the safe introduction of a paracentesis needle can include the bladder, bowel, and pregnant uterus. The bladder is normally tucked into the recess of the pelvis. However, neuropathically distended bladders caused by pharmacologic agents or medical conditions should preferably be emptied by voiding or by catheterization to avoid puncture. Intestines typically float in ascitic fluid and will move safely away from a slowly advancing paracentesis needle. [142] Even if penetrated by an 18- to 22-ga needle, leakage of intestinal contents will not occur unless the intraluminal pressure is 5- to 10-fold greater than normal conditions. [143] Therefore, US guidance may be indicated in cases of suspected adhesions or bowel obstruction. In second- and third-term pregnancy, an open supraumbilical or US-assisted approach is preferred.

The abdomen should be carefully inspected for evidence of abdominal hematoma, engorged veins, or superficial infection, and these sites should be strictly avoided.

Technique

Preliminary Actions

The operator should comply with guidelines for body fluid precautions (see Chapter 71). The patient should be carefully draped and the skin thoroughly prepped in sterile fashion to prevent the iatrogenic introduction of bacteria into the abdominal wall tract or peritoneal cavity.

Site of Entry

The preferred site is approximately 2 cm below the umbilicus in the midline (Fig. 44-11). The *fasciae* of the *rectus abdominis* join to form the fibrous, thin, avascular *linea alba*. [142] Large collateral veins may occasionally be present and should be avoided, as should suspected areas of skin infection. If the patient has midline scarring, the preferred alternate site is in either lower quadrant, approximately 4 to 5 cm cephalad and medial to the anterior superior iliac spine (see Fig. 44-11). The importance of remaining lateral to the rectus sheath is to avoid the inferior epigastric artery. Patients with a large quantity of ascites can readily undergo the procedure in the supine position with the head of the bed slightly elevated. Those with lesser amounts of fluid may profit from a lateral decubitus position with introduction of the needle into the midline or dependent lower quadrant (Fig. 44-12). Some clinicians prefer to use the lateral decubitus position routinely because the bowel tends to float upward and away from the path of the needle. Rarely, patients may need to be placed in a facedown, hands-on-knees position. [140] In the patient with multiple abdominal scars or suspicion of compartmentalized abdominal fluid for any reason, US guidance is prudent. [144]

Procedure

Following sterile preparation of the skin, local anesthesia is administered at the paracentesis site. A standard 3.8 cm (1.5 in.) metal needle is sufficient in most cases.[129] An 8.9 cm (3.5 in.) spinal needle may be required in obese patients. Plastic sheath cannulas tend to kink and run the risk of being sheared off into the peritoneal cavity.[118] A steel needle can be left in the abdomen during a therapeutic tap for intervals of an hour or more without injury. A smaller-gauge needle (20- to

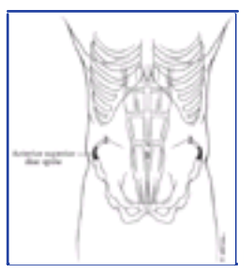

Figure 44-11 Preferred sites for paracentesis: *1,* Primary site is infraumbilical in midline through *linea alba*. *2,* Preferred alternate (lateral rectus) site is in either lower quadrant, approximately 4 to 5 cm cephalad and medial to the anterior superior iliac spine.

Figure 44-12 An alternative to the sitting or supine position for needle paracentesis is to place the patient in the lateral decubitus position. In this example the midline is aspirated, although the lateral rectus sites may also be used. Some prefer the lateral decubitus position routinely, because the bowel tends to float upward and out of the path of the needle.

22-ga) is appropriate for diagnostic taps, as these lessen the likelihood of postprocedural ascitic fluid leak through the wound site. However, large-volume therapeutic paracenteses benefit from 18-ga needles, as this permits expeditious outflow. [117] [145]

The needle can be inserted directly perpendicular at the preferred site. Certain authors prefer the "Z-tract" method wherein the skin is pulled approximately 2 cm caudad in relation to the deep abdominal wall by the non-needle-bearing hand while the paracentesis needle is being slowly inserted (Fig. 44-13).[146] The skin is not released until the needle has penetrated the peritoneum and fluid flows. When the needle is removed following the procedure, the skin will slide to its original position and help seal the tract. In any case, slow insertion of the needle in 5-mm increments is preferred. This

Figure 44-13 *A,* Z-tract method of paracentesis. The skin is pulled approximately 2 cm caudad in relation to the deep abdominal wall by the non-needle-bearing hand while the paracentesis needle is slowly being inserted directly perpendicular to the skin. *B,* After penetrating the peritoneum and obtaining fluid return, the skin is released. Note that the needle is now angulated caudally.

allows the operator to detect undesired entry of a vessel and helps prevent unnecessary puncture of small bowel. Continuous suction should be avoided as it may attract bowel or omentum to the end of the paracentesis needle with resultant occlusion. Once fluid is flowing, the needle should be stabilized to ensure a steady flow. If flow ceases, the needle should be gently rotated and advanced inward using 1- to 2-mm increments.

Complications

Complications can be divided into systemic, local, and intraperitoneal categories.

Systemic

An oft-cited but poorly documented concern is hemodynamic compromise caused by overzealous removal of ascitic fluid. Because upward of 6 L has been reportedly removed in less than 15 minutes without complication, certain authorities decry this issue as folklore. [147] [148] Others believe that rapid total paracentesis is accompanied by marked cardiovascular and humoral changes, some of which are explained by mechanical factors directly or indirectly related to relief of abdominal pressure.[149] [150] Other changes, including systemic vasodilation and humoral deactivation, are of a nonmechanical nature.

Because fluid and electrolyte shifts tend to be minimal following the removal of large amounts of fluid, [151] colloid infusion is considered strictly optional for paracentesis of more than 5 L and is not recommended for paracentesis of lesser volume. [120] [152] [153] When colloid is believed to be indicated, albumin has been the de facto choice. However, colloid dextran 70 is favored by some authorities due to cost and infection concerns. [154] [155] [156]

Local

Local complications include persistent ascitic fluid leak at the wound site, abdominal wall hematoma, and localized infection. Persistent fluid leak can be corrected with a single suture

at the site of puncture.[145] An abdominal wall hematoma requiring transfusion is very uncommon, but careful observation in such cases is necessary. [140]

Intraperitoneal

Intraperitoneal complications include perforation of vessels and viscera. [157] In experienced hands, these are uncommon, and in most circumstances they are self-sealing and clinically inconsequential. However, generalized peritonitis and abdominal wall abscess have been reported after paracentesis in rare cases. The most common cause of postparacentesis intraperitoneal hemorrhage is bleeding due to a coagulopathy, rather than large-vessel injury per se. [158]

Interpretation

Ascitic fluid should undergo gross inspection. Routine laboratory testing includes a differential cell count, albumin assay, and cultures (Table 44-14).

Inspection

Ascitic fluid is typically translucent and yellow. A dark greenish-brown hue may reflect biliary perforation. Cloudy fluid generally indicates particulate matter, including neutrophils: fluid with WBC counts greater than 5,000/µL (i.e., >5,000/mm ^{3}) are cloudy and those greater than 50,000/µL are purulent. An opaque, milky appearance may indicate elevated triglyceride levels. [159] A blood-tinged appearance requires at least 10,000/µL RBCs. This may

TABLE 44-14 -- Ascitic Fluid Laboratory Data to Be Obtained on Patients With Ascites

Routine
Cell count
Albumin
Culture in blood culture bottles
Optional
Total protein
Glucose
Lactate dehydrogenase
Amylase
Gram stain
Unusual
Tuberculosis smear and culture
Cytology
Triglyceride
Bilirubin
Unhelpful
pH
Lactate
Cholesterol
Fibronectin

From Runyon BA: Ascites. In Schiff L, Schiff ER (eds): Diseases of the Liver, 7th ed. Philadelphia, Lippincott-Raven, 1993, p 997.

reflect an iatrogenic complication, malignancy, hemorrhagic pancreatitis, or tuberculous peritonitis, although the last diagnosis creates hemorrhagic-appearing fluid in less than 5% of cases. [140]

Cell Count

Several milliliters of ascitic fluid are sufficient to obtain a differential cell count. Cirrhotic ascites should generally contain less than 250 WBCs/µL (Table 44-15). However, because cells may exit through the peritoneal cavity more

TABLE 44-15 -- Ascitic Fluid Characteristics in Various Disease States

				Cell Count		
Condition	**Gross Appearance**	**Specific Gravity**	**Protein (g/dL)**	***RBCs, >10,000/µL***	***WBCs/µL (WBCs/mm^3)***	**Other Tests**
Cirrhosis	Straw-colored or bile-stained	<1.016 (95%)*	<25 (95%)	1%	<250 (90%),* predominantly mesothelial	
Neoplasm	Straw-colored, hemorrhagic, mucinous, or chylous	Variable, >1.016 (45%)	>25 (75%)	20%	>1000 (50%); variable cell types	Cytology, cell block, peritoneal biopsy
Tuberculous peritonitis	Clear, turbid, hemorrhagic, or chylous	Variable, >1.016 (50%)	>25 (50%)	7%	>1000 (70%); usually >70% lymphocytes	Peritoneal biopsy, stain and culture for acid-fast bacilli
Pyogenic peritonitis	Turbid or purulent	If purulent, >1.016	If purulent, >2.5	Unusual	>250; mainly polymorphonuclear leukocytes	Positive Gram stain, culture
Congestive heart failure	Straw-colored	Variable, <1.016 (60%)	Variable, 15–53	10%	<1000 (90%); usually mesothelial, mononuclear	
Nephrosis	Straw-colored or chylous	<1.016	<25 (100%)	Unusual	<250; mesothelial, mononuclear	If chylous, ether extraction, Sudan staining
Pancreatic ascites (pancreatitis, pseudocyst)	Turbid, hemorrhagic, or chylous	Variable, often >1.016	Variable, often >25	Variable, may be blood-stained	Variable	Increased amylase in ascitic fluid and serum

RBC, red blood cell; WBC, white blood cell.

From Glickman RM, Isselbacher KJ: Abdominal swelling and ascites. In Isselbacher K, et al (eds): Harrison's Principles of Internal Medicine, 13th ed. New York, McGraw-Hill, 1994, p 234.

*Since the conditions of examining fluid and selecting patients were not identical in each series, the percentage figures (in parentheses) should be taken as an indication of the order of magnitude rather than as the precise incidence of any abnormal finding.

slowly than fluid does, the WBC count can rise in the ascitic fluid during the procedure. [160] Thus, an upper limit for uncomplicated cirrhotic ascites is reported as 500 cells/µL.[161] [162] [163] Lymphocytes should predominate, and clinical signs or symptoms of peritoneal infection should be absent. [164] In cases in which spontaneous bacterial peritonitis is a clinical consideration, the WBC criterion is 250 WBCs/µL with greater than 50% polymorphonuclear leukocytes. [134] [137] [138] [164] [165]

Albumin

A serum-ascites albumin gradient can be obtained by simultaneous measurement of ascitic and serum-ascites albumin gradient. A serum-ascites albumin gradient greater than 1.1 g/dL indicates portal hypertension with greater than 95% accuracy (see Table 44-13). [166] [167] [168]

Culture and Gram Stain

The most valuable method for determining the presence of infection is culture. The sensitivity of this test is markedly increased by the direct inoculation of blood culture bottles at the bedside in contrast to simply delivering the ascitic fluid to the laboratory. [146] [169] Approximately 10 bacteria/µL of fluid is required for a positive Gram stain. Thus, the Gram stain is notoriously insensitive in spontaneous bacterial peritonitis in which the medium concentration of bacteria is 10^{-3} organisms/µL of fluid.[169] The Gram stain can only be expected to be helpful in cases of free gut perforation.

Miscellaneous

Optional tests include measurement of total protein, glucose, lactate dehydrogenase, and amylase. These will be beneficial in selected circumstances and need not be obtained on a routine basis. Immunosuppressed patients, including those with AIDS, should undergo microbiologic testing for opportunistic infections, including tuberculosis.[139] Cytologic analysis is recommended in patients with suspicious constitutional symptoms and signs. [170] [171] Triglyceride and bilirubin studies are indicated if the gross appearance of the fluid is suggestive of increased levels. [172]

Conclusion

Abdominal paracentesis is a straightforward, commonly used procedure in the ED, with few relative contraindications. The imperative of paracentesis is to disclose the presence of infection in the peritoneal cavity. It is also useful in the diagnosis of other conditions and contributes to the relief of manifestations of tense ascites.

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Chapter 45 - Abdominal Hernia Reduction

David E. Manthey

When faced with a possible hernia, the emergency clinician must consider three issues: (1) Is the mass truly a hernia? (2) Is the hernia reducible or incarcerated? (3) Is the vascular supply to the bowel strangulated? An easily reducible hernia can be safely discharged for outpatient follow-up and repair while an incarcerated and/or strangulated hernia is a surgical urgency. Some seemingly incarcerated hernias can be reduced by persistent manipulation in the ED. The emergency clinician must differentiate among these entities.

Although there are many types of hernias, this chapter will address only those hernias amenable to reduction in the emergency department (ED). Groin hernias may be divided into inguinal and femoral. The inguinal hernias include direct, indirect, and pantaloon hernias. Abdominal wall (ventral) hernias include incisional, umbilical, epigastric, and spigelian hernias.

BACKGROUND

A hernia is defined as the protrusion of any viscus from its normal cavity through an abnormal opening. Approximately 5% of the male population and 2% of the female population develop hernias. [21] [24] Approximately 75% of all hernias occur in the groin and two thirds of these are indirect hernias. In fact, 90% of all cases in children and young adults are indirect inguinal hernias. [18] As the age of the patient increases, so does the incidence of acquired (direct) hernias. [17] Incisional and ventral hernias account for approximately 10% of all hernias. Hernias can develop along a congenital tract that fails to close (indirect inguinal hernia) or along a weakness in a muscular and fascial wall (direct inguinal hernia). This weakness may be due to aging and the accompanying loss of tissue elasticity, increased intra-abdominal pressure, or trauma to the wall itself. An abdominal wall hernia is defined as a protuberance of intra-abdominal contents through an abnormal opening in the wall.

CLASSIFICATION OF ABDOMINAL HERNIAS

A *reducible hernia* is one whose contents can be returned through the fascial defect back into the abdominal cavity.

An *incarcerated hernia* is one whose contents are not reducible back into the abdominal cavity. An associated swelling of the hernia sac contents may result.

A *strangulated hernia* is an incarcerated hernia whose blood supply to the herniated structures is compromised. Hernias with a small neck are more likely to strangulate. A strangulated hernia is a surgical emergency since gangrene will result if the vascular compromise is not relieved.

INGUINAL HERNIAS

Anatomy of the Inguinal Triangle

The inguinal (or Hesselbach's) triangle is formed by the inguinal ligament (on the inferior side), the inferior epigastric artery (on the supero-lateral side), and the lateral edge of the rectus abdominis muscle (on the medial side). There are two types of inguinal hernias (direct and indirect), both of which occur superior to the inguinal ligament. Femoral hernias are located inferior to the inguinal ligament.

Indirect Inguinal Hernia

When the hernia passes through the internal (deep) inguinal ring and into the inguinal canal, it is known as an indirect inguinal hernia (Fig. 45-1). It lies lateral to the inferior epigastric vessels. During fetal development the processus vaginalis allows for the descent of the testes into the scrotum. The processus vaginalis generally begins to close off shortly before birth. If it fails to be obliterated completely, a hernia or hydrocele may develop.

The indirect inguinal hernia is the most common hernia overall. Although there is a male to female ratio of 9:1, it is still the most frequently occurring hernia for either sex. It occurs more commonly in children and young adults. Approximately 3% to 5% of full-term infants and up to 30% of preterm infants will have an inguinal hernia.[11] [12] Of these, 80% to 90% occur in males. Incarceration occurs more commonly in the first 6 months of life, with an overall rate of 12%. [10] [22] Inguinal hernias that are successfully reduced should be considered for surgical repair within 24 to 48 hours. [2] [10] [14]

Direct Inguinal Hernia

A direct inguinal hernia is one that comes directly through the muscular and fascial wall of the abdomen. It is located within Hesselbach's triangle and therefore medial to the inferior epigastric vessels (Fig. 45-2). It can be differentiated from the indirect inguinal hernia because it does not travel along the inguinal canal.

The direct inguinal hernia is the second most common groin hernia. It is an acquired hernia and occurs later in life

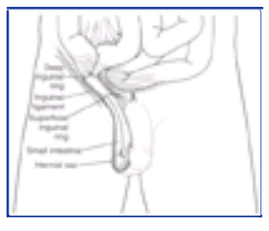

Figure 45-1 Indirect inguinal hernia.

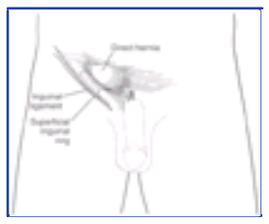

Figure 45-2 Direct inguinal hernia.

secondary to weakening of the fascial and muscular wall from aging and the repetitive stress of increased abdominal pressure. This hernia carries a minimal risk of incarceration because the neck of the hernial orifice is wide.

Pantaloon Hernia

The pantaloon hernia is a combination of a direct and indirect hernia, which resemble the two legs of a pair of pants. This hernia is difficult to diagnose in the ED and is often discovered during surgical exploration. Successful treatment depends upon correctly identifying the hernia followed by reduction of each hernia separately.

Femoral Hernia

A femoral hernia occurs inferior to the inguinal ligament through a defect in the transversalis fascia. The contents protrude into the potential space in the femoral canal located

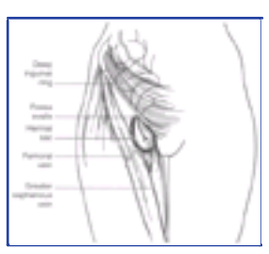

Figure 45-3 Femoral hernia.

medial to the femoral vein and lateral to the lacunar ligament (Fig. 45-3). Due to the small fascial defect and constriction by the inguinal ligament, this hernia frequently becomes incarcerated. The femoral hernia is relatively rare, and occurs more commonly in women than in men.

Pelvic Floor Hernias

A pelvic floor hernia occurs in the peri-rectal area as a result of acquired weakness in the pelvic muscular sling. This hernia is noted to occur in approximately 3 to 6% of pelvic exenterations.[1] [19]

Ventral Hernia

A ventral hernia may be an incisional, umbilical, epigastric or a Spigelian hernia (Fig. 45-4). The ventral hernia occurs due to weakening of the diastases of the anterior abdominal wall.

Incisional Hernia

An incisional hernia commonly follows abdominal surgery in an area of postincisional weakness in the abdominal wall (see Fig. 45-4*A*). Poor wound healing (owing to

infection, for example) increases the likelihood of forming this type of hernia. An incisional hernia occurs in 2% to 10% of all abdominal surgeries and carries recurrence rates of 20% to 45%. As the lines of tension pull this hernia open, the size of the defect is usually sufficient to prevent incarceration.

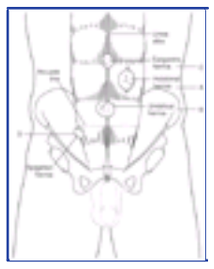

Figure 45-4 Ventral hernias. *A,* Incisional hernia. *B,* Umbilical hernia. *C,* Epigastric hernia. *D,* Spigelian hernia.

Umbilical Hernia

An umbilical hernia traverses the fibromuscular ring of the umbilicus (see Fig. 45-4*B*). This hernia is found commonly in infants, is congenital in origin, and usually resolves without treatment by age 2 years. If it persists beyond this age, is larger than 2 cm, or becomes incarcerated or strangulated, it may be repaired surgically. An acquired umbilical hernia may also be seen in an adult, particularly with increased abdominal pressure (such as in obesity, ascites, or pregnancy). An umbilical hernia is more prone to incarceration and strangulation in an adult than in a child.

Epigastric Hernia

This hernia occurs in the midline through the linea alba of the rectus sheath (see Fig. 45-4*C*). It is usually located in the epigastric region between the xiphoid and the umbilicus. Although previously considered rare in infants, one study found epigastric hernias in 4% of all pediatric patients seen for hernias. [6]

Spigelian Hernia

The Spigelian hernia is rare and courses through a defect at the lateral edge of the rectus muscle at the semilunar line (see Fig. 45-4*D*).

PATIENT PRESENTATION

An asymptomatic hernia may present as a mass that is found incidentally on physical exam. It requires no procedural intervention in the ED. A patient with a symptomatic hernia may describe swelling and/or pain. Many hernias, especially femoral hernias, present with abdominal pain. [25] The patient may report a history of heavy lifting. The clinician should inquire about signs of infection such as fever, chills, and malaise; and signs of bowel obstruction including nausea and vomiting. The patient's history should include a record of previous surgeries and hernia repairs (including the use of synthetic material).

The inguinal canal may be palpated in males by inverting the scrotal skin and passing a finger into the external ring. The Valsalva maneuver or coughing increases intraabdominal pressure and facilitates the detection of a hernia. The palpation of the external ring is more difficult in females because it is narrower.

An indirect inguinal hernia presents as a swelling in the area of the inguinal ligament or as a scrotal swelling. It is often painless and may be noted as an incidental finding. On examination, this hernia can be differentiated from a direct hernia in two distinct ways. First, it starts laterally to the inferior epigastric arteries. Second, on palpation of the inguinal canal, the contents of the hernia will strike the tip of the finger instead of the pad. This occurs as the hernia protrudes down the canal to meet the finger instead of across a fascial and muscular defect. This effect can be accentuated by applying pressure over the internal ring after hernia reduction. Bulging will reoccur with straining if the hernia is direct, but the pressure over the internal ring should block distention of the hernia into the inguinal canal. Similarly, a hernia that fills the scrotum is most likely an indirect hernia. The peritoneal contents may become incarcerated owing to swelling of the internal or external ring.

A child with an inguinal hernia may have a reducible inguinal or scrotal mass that occurs with straining or crying. A child may present with symptoms of vomiting, poor eating, lethargy, or irritability. The clinician must always consider incarcerated or strangulated hernias in the differential of such vague complaints.

Incarcerated Versus Strangulated

When one cannot reduce the contents of the hernia back into the abdominal cavity, the hernia is described as incarcerated. A patient with an incarcerated hernia usually presents with a mass that is now painful, discolored, enlarged, and no longer reducible. Associated symptoms may include nausea, vomiting, fever, scrotal pain, labial pain, and abdominal distention. Patients with incarcerated hernias do not necessarily have a bowel obstruction and those with a bowel obstruction are not necessarily incarcerated. A hernia may become incarcerated due to a small fascial defect. Constriction of the fascial defect by outside musculature or swelling of the defect or of the hernia contents may also lead to incarceration. Incarceration usually occurs with femoral hernias, small indirect inguinal hernias, or abdominal wall hernias.[3]

A strangulated hernia is one in which the vascular supply to the hernia is compromised, causing ischemia of the contents. Erythema, significant tenderness to palpation, purple or bluish discoloration, abdominal tenderness or peritoneal signs, or extra-luminal air (on x-ray) suggest ischemic injury to the bowel. [3] The patient may present with bowel obstruction, peritonitis, perforation, abscess, or even shock. A leukocytosis with a left shift is common but may be absent in the elderly.

It has long been suggested that strangulated hernias cannot be reduced. However, in rare instances, the hernia may inadvertently be reduced en masse to a pre-peritoneal location (Fig. 45-5), making the hernia sac and contents no longer palpable. [15] [16] [23] In this case, the hernia has not been reduced into the peritoneal cavity and the incarceration/ischemia has not been relieved. Because the clinician believes the hernia has been appropriately reduced, this can result in delay in the diagnosis of ischemic bowel. Fortunately, this is rare and occurs in less than 1% of hernias. [4]

Differential Diagnosis

The differential diagnosis for a groin mass is large. Table 45-1 lists a number of disease processes that may masquerade as a hernia.

Testicular torsion can be mistaken for a hernia, especially if there is an associated reactive hydrocele. The clinician must examine the testicle for tenderness, swelling, lie, and cremasteric reflex. If there is concern for a testicular torsion, urology should be notified immediately while simultaneous diagnostic studies are undertaken.

A hydrocele can be confused with a hernia because both can occupy the same anatomical space (Fig. 45-6). A hydrocele may transilluminate whereas a hernia generally does not.[21] Differentiation may be difficult and may require ultrasound to define the contents of the scrotum.

A retracted testis may be diagnosed by its absence in the scrotal sac. However, a mass felt in the inguinal canal may be mistaken for the testicle when it is actually a hernia.

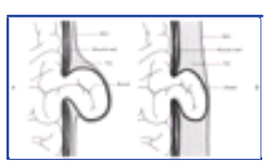

Figure 45-5 En masse reduction. *A,* When a hernia forms, it projects from the fascia into the subcutaneous fat. The object of reduction is to replace the hernia into the peritoneal cavity. *B,* If the hernia sac is partially reduced into the subcutaneous fat of an obese patient, it may appear reduced and may not be palpable owing to the patient's body habitus. However, the hernia is still susceptible to incarceration or ischemia, as it has not been returned to the peritoneal sac.

Disease processes remote to the inguinal canal can also cause a groin mass. There are reports of blood from a ruptured spleen [20] as well as appendicitis[5] presenting as a mass.

ABDOMINAL HERNIA REDUCTION

Indications and Contraindications

The indications for attempting to reduce a hernia are the presence of a hernia and the absence of strangulation. In some clinical practices, the surgeons prefer consultation before an attempt at reduction by the emergency clinician. Because many patients require sedation, it is ideal if the patient is sedated only once. This may be facilitated by discussing the

TABLE 45-1 -- Differential Diagnosis of Groin Masses

Hernia
Testicular torsion
Retracted or undescended testicle
Hydrocele
Spermatocele
Venous varix
Pseudoaneurysm
Lymphadenopathy
Lymphogranuloma venereum
Epididymitis
Hidradenitis suppurativa
Groin abscess
Hematoma
Lipoma
Epidermal inclusion cyst
Tumor
Appendicitis
Tracking of intraperitoneal blood

treatment plan with the consultant prior to reduction. The emergency clinician should not make repetitive attempts at reducing the hernia as this may cause increased swelling and limit the chances of a non-operative reduction by the surgeon.

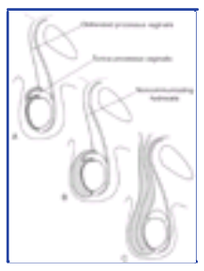

Figure 45-6 Hydrocele versus hernia. *A,* normal anatomy; *B,* noncommunicating hydrocele that may be confused with hernia; *C,* indirect hernia that can be palpated from inguinal ring to testicle.

Reduction of a strangulated hernia in the ED is contraindicated. In the case of a strangulated hernia, operative management is required. The presence of a bowel obstruction is a controversial contraindication for reduction in children younger than 2 years. [7]

The presence of a fever or an elevated white blood cell (WBC) count is a contraindication to reduction of a hernia; however, the absence of a fever or increased WBC count does not rule out the presence of ischemic bowel. [13]

When an undescended testis or ovary is trapped within an incarcerated hernia, it should be reduced surgically. Traumatic hernias also require surgical repair to allow for evaluation of the underlying organs.

PROCEDURE AND TECHNIQUE

An increase in intra-abdominal pressure will work in direct opposition to reducing a hernia. To limit this the patient should be positioned appropriately and steps should be taken to minimize patient discomfort. This in turn will decrease muscle guarding. The patient should be placed in a position so that gravity can work to pull the contents of the hernia sac back into the peritoneal cavity. For inguinal hernias, place the patient in the Trendelenburg position. For abdominal hernias place the patient in the supine position. Often, just leaving the patient in this position for 10 minutes will allow the hernia to reduce itself. In order to minimize tensing of the abdominal muscles, care must be taken not to hurt, tickle, startle, or chill the patient. To this end, using a slow approach with soft, ongoing dialogue and warm hands goes a long way.

Because swelling of the contents of the sac makes it more difficult to reduce, placing cool compresses on the hernia may help by producing vasoconstriction and by reducing the

Figure 45-7 Ballooning of contents. *A,* When reducing a hernia, the contents may ride up over the edge of the fascial defect. This leads to ballooning of the contents around the neck of the hernia, which hinders the reduction. *B,* By placing fingers along the edge of the hernia neck, one can direct the contents into instead of over the fascial defect.

volume of intraluminal bowel gas, thus decreasing the hernia sac swelling. Be sure to wrap the ice or ice water in a towel to prevent cold injury to the overlying skin. This should be done while the patient is positioned and preparations are being made to sedate the patient.

The patient should be relaxed and the pain of the procedure minimized. This can be accomplished by using sedation and analgesia before attempting reduction, not following a failed attempt. Although this author uses versed and fentanyl, other alternatives exist including etomidate and propofol. The techniques of analgesia and sedation are discussed in Chapter 34 .

To understand how to reduce a hernia, one must first consider its "parts." A hernia consists of a defect in the existing tissue (muscle and fascia) through which the visceral contents protrude. This defect makes up the neck of the hernia sac and it can be small or large. If it is small, the hernia will have a higher incidence of incarceration and strangulation. When attempting to reduce the hernia, care must be taken not to override the edge of the hernia orifice, since this will cause "ballooning" of the contents of the hernia sack around the hernia neck (Fig. 45-7). To prevent this, the examiner should attempt to find the edge of the hernia defect and position a hand or fingers opposite the reducing hand. After this preparation, the clinician may attempt to gently guide the hernia contents and sac back through the neck of the hernia. If the content of the hernia is small, gentle steady pressure will be all that is required. If the content of the hernia is large, a different approach may be needed. In this case gently guide the most proximal aspect of the hernia back through the neck. Reduce the hernia contents in the opposite order from which they protruded. Forcing the distal end of the hernia sac through the hernia opening causes the rest of the proximal contents to be displaced around the opening. This will

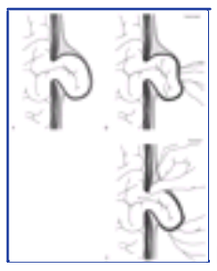

Figure 45-8 Guiding hernia into place. A, The hernia can be divided into several sections. The proximal portion is closest to the neck or fascial defect, through which the hernia protrudes. The distal portion is farthest from the neck. B, When attempting to reduce the hernia, be careful not to invaginate the distal portion first or the proximal portion may obstruct the opening as it is pushed over the sides. C, The last portion of the bowel that exits into the hernia sac should be the first returned to the peritoneal cavity.

obstruct the reduction of the more distal tissue. Gentle steady pressure on the tissue at the neck of the hernia (while preventing ballooning of the tissue over the edge of the hernia) will gradually reduce the hernia (Fig. 45-8 and Fig. 45-9).

With children, positioning is one of the most important aspects in the reduction of the inguinal hernia. For an inguinal hernia, the patient should be placed in a Trendelenburg position at about 20°. If spontaneous reduction does not occur, then manual reduction should be attempted. Spontaneous reduction has been reported in up to 80% of children with inguinal hernias over a 2-hour period without manipulation.

Secondly, place the patient in the "unilateral frog leg" position as described by Fraser (Fig. 45-10). [8] Stabilize the patient by grasping the anterior superior iliac spines to prevent lateral movement of the pelvis. Abduct the ipsilateral leg, externally rotate and flex the hip to obtain the classic "frog leg" position. The purpose of this position is to allow both the internal and external rings the greatest re-approximation. After achieving this position, use the fingers of one hand to prevent the hernia contents from overriding the external ring while the other hand provides steady but gentle pressure to the contents of the hernia sac. Repeated forceful attempts are not recommended.

COMPLICATIONS

Three major complications may occur during the reduction of hernias. The first is due to overzealous attempts at reduction by the clinician, thus causing injury to the underlying bowel. This may aggravate the swelling and make the hernia irreducible. Appropriate patient preparation, positioning,

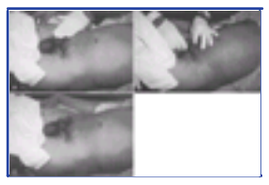

Figure 45-9 *A,* This patient presented with signs and symptoms of a bowel obstruction. It was not until the surgery consultant removed the patient's pants that an inguinal hernia was found to be the cause of the obstruction. *B,* After intravenous analgesia/sedation, the inguinal ring is stabilized with the left hand. *C,* Slow gentle, yet persistent pressure is applied to reduce the hernia. It required 5 minutes. The hernia was reduced. Although this was a reducible hernia, because of the bowel obstruction this patient should be admitted to the hospital for observation and next day surgery.

sedation, and careful reduction can reduce the risk of this complication. The second and third major complications occur following reduction of ischemic bowel. Complications may occur when the clinician ignores or does not search for

Figure 45-10 Frog leg technique.

clues of ischemic tissue and inadvertently reduces ischemic bowel back into the peritoneal cavity. Finally, complications may occur when the tissue is reduced en masse into the pre-peritoneal position as described earlier.

CONCLUSION

The clinician must make sure that all the factors which aid in reduction are adequately in place before attempting the procedure. This includes positioning, sedation and pain control, and proper technique. Haphazard attempts may work in easily reducible hernias, but they seldom do more than cause pain and swelling in more difficult cases.

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Chapter 46 - Anorectal Procedures

Christopher M. Strear
Wendy C. Coates

Patients with anorectal disorders frequently present to the emergency department (ED). In some cases, the condition is isolated, while in others, the anorectal complaint can be an outward manifestation of a more serious illness. A thorough history and physical examination must precede any procedure. Because of the nature of these complaints, extreme sensitivity and professionalism must be used during the care of the patients.

Patients may be quite anxious about an anorectal exam or associated procedure. Although sedation is rarely needed for a simple digital rectal exam (DRE), other anorectal procedures may be difficult, if not impossible, to perform without sedation, analgesia or both. Therefore, *the emergency clinician should consider the liberal use of analgesia and sedation with these anorectal procedures*. The use of a topical anesthetic on the anoscope is an effective lubricant, but provides inadequate analgesia in a patient with severe pain or anxiety. The use of parenteral analgesia and sedation is outlined in detail in Chapter 34 .

ANATOMY

The rectum and anus comprise the most distal portion of the gastrointestinal tract. The rectum is bounded by the sigmoid colon at the level of the third sacral vertebra and extends distally 12 to 15 cm. Here it joins the anal canal, which is the terminal 4 cm of the lower intestinal tract. Sensory innervation to the rectum is primarily visceral, while innervation to the anus is via cutaneous fibers. Therefore, patients are often unaware of rectal pathology since the pain associated with it is often vague or absent. By contrast, anal lesions are usually very painful and well localized by the patient. The dentate or pectinate line marks the transition from the anus to the rectum. At the line is a series of anal crypts where the openings of the submucosal anal glands lie. Infections of the anal glands are the likely etiology of many anorectal abscesses. The blood supply to the anorectum consists of superior, middle, and inferior hemorrhoidal arteries and veins. The venous system is formed by a series of plexi that are susceptible to overdistention and thrombosis [3] [18] [25] (Fig. 46-1).

DIGITAL RECTAL EXAMINATION

Indications and contraindications

The physical examination should take place in a private location and care should be taken to assure that the patient is completely draped and relaxed. Generally, a calm atmosphere and caring examiner should be sufficient. In some cases, analgesic or anxiolytic agents may be needed to facilitate a thorough examination. Some conditions, such as thrombosed or gangrenous hemorrhoids, produce so much pain that the examination may need to be postponed until the patient is anesthetized. A suspected sharp edged foreign body (e.g., metal blade or broken glass) warrants a preprocedure radiograph. If confirmed, the DRE should be deferred in favor of anoscopy or sigmoidoscopy under anesthesia.

Procedures

The patient is placed in the lateral decubitus position and the examiner's gloved finger is lubricated. Visual inspection of the perianal area may give important information regarding patient hygiene, trauma, or sexually transmitted diseases. The finger is placed firmly against the anal sphincter and the patient is asked to bear down. Prolapsing of hemorrhoids or rectal mucosa may be noted. The gloved finger is then inserted into the anus and a 360-degree sweep is performed to identify any irregularities. A prostate exam may be performed at the same time, if needed. After removing the finger from the anus, adherent stool or residual lubricating jelly can be applied to a stool guaiac card to examine for the presence of occult blood. [5]

Complications

Although DRE has been reported to cause a vasovagal response or cardiac dysrhythmias such as ventricular fibrillation, these complications are rare and do not negate the need for this examination.[23]

ANOSCOPY

Indications and Contraindications

Anoscopy may be used as an adjunct to the DRE when evaluating a patient for anal pathology. [25] Internal hemorrhoids, tears in the distal rectal mucosa, foreign bodies (FBs), and distal anorectal masses may be visualized. Imperforate anus is the only absolute contraindication to anoscopy; however, rectal pain, a common presenting complaint in the ED, may preclude awake anoscopic examination in the anxious patient.

Equipment and Setup

The anoscope is a clear plastic or stainless steel tube with a removable obturator inside. It may have an integrated light source or require an external light or head lamp. In addition, lubricant, an appropriate examination table, topical anesthetics, and gauze with forceps may be required (Fig. 46-2*A, B* and Fig. 46-3*A, B*).

Positioning

Ideally, the patient should be placed on a proctoscopic examination table or supine in the knee-chest position. The prone or lateral decubitus position with knees and hips flexed may also be adequate, and is sometimes better tolerated by the patient. In the decubitus position, the patient should be placed on the left side if the examiner is right-handed (Fig. 46-4).

Procedure

Although most patients do not require intravenous sedation and analgesia, these agents can be administered as needed to keep the patient relaxed and comfortable. A topical anesthetic

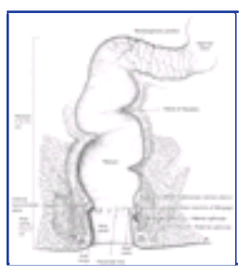

Figure 46-1 Anatomy of the terminal gastrointestinal tract. *(Redrawn from Abrahams PH, Webb PJ: Clinical Anatomy of Practical Procedures. London, Pitman, 1975.)*

such as 2% lidocaine jelly can be applied for painful conditions such as thrombosed hemorrhoids or anal fissures. Topical agents should be applied at least 10 minutes before anoscopy for optimal effect, although complete relief of pain should not be expected. Topical EMLA cream may serve as an alternative to lidocaine. Patients who do not tolerate anoscopy because of pain, or whose conditions are not limited to the anus, may be referred to a specialist for a more thorough examination (e.g., sigmoidoscopy, colonoscopy, examination under anesthesia).

Prior to anoscopy, a routine DRE should be performed to check for bleeding, identify a mass, or help localize pain. The axis of the anal canal should also be noted to properly direct the anoscope. After the DRE, the anoscope is introduced with the obturator inserted completely into the anoscope. The examiner gently advances the instrument while the patient bears down slightly. Gentle, constant pressure will overcome resistance from involuntary contraction of the external anal sphincter, as tonic contraction is exhausted after 2 minutes. The anoscope is passed gently into the anorectum while the examiner's thumb maintains the obturator in place. If the obturator falls back during insertion, the examiner must remove the anoscope completely and then replace the obturator. This will avoid pinching the anal mucosa. The instrument should be inserted until the outer flange impinges on the anal verge (Fig. 46-5).

Once the anoscope is fully inserted, the obturator is removed. The anal canal is then visualized as the anoscope is gradually withdrawn. Blood or debris can be swabbed to aid visualization, and discharge can be cultured. The anoscope should be withdrawn slowly as the entire circumference of mucosa is inspected. Near the last stage of withdrawal, reflex

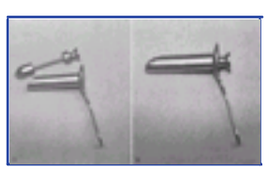

Figure 46-2 Stainless steel anoscope. *A,* Anoscope with obturator. *B,* Anoscope prepared for insertion.

spasm of the anal sphincters may cause the anoscope to be expelled quickly. Firm counterpressure may prevent such rapid expulsion, although it is not uncommon to need to repeat the procedure to obtain an adequate view of the anal verge.

Figure 46-3 Anoscope with integrated light source. *A,* Scope with obturator in place for insertion. *B,* Scope attached to light source, also with obturator in place. Generally the scope is inserted before attaching it to the light source.

Complications

Anoscopy is a safe procedure and complications are rare. Local mucosal irritation with subsequent bleeding is the most common complication. Transmission of infectious diseases

Figure 46-4 Positions for performing sigmoidoscopy or anoscopy. *(From Hill GJ II: Outpatient Surgery, 3rd ed. Philadelphia, WB Saunders, 1988.)*

is possible. Therefore, instruments must be disposed of or sterilized after each use.

MANAGEMENT OF HEMORRHOIDS

Hemorrhoids are a common affliction and have been described and treated for more than 4000 years. [18] Thrombosed hemorrhoids may have been responsible for Napoleon's defeat at Waterloo. [33] The refined, low-fiber diet

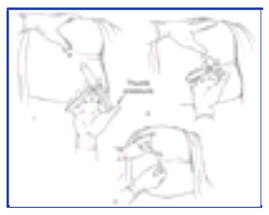

Figure 46-5 Anoscopy. Initially, with the thumb used to keep the obturator fully inserted during passage of the instruments, the anoscope is directed toward the umbilicus, and the obturator is not withdrawn until the scope has been passed to the hilt. Detailed examination is performed on withdrawal of the instrument. A penlight is used in this illustration for illumination of the deep anus.

of Western nations makes hemorrhoids extremely common in the United States [25] where 1 in 25 to 30 individuals are afflicted. [7] An estimated one third of these people, or 1 million patients annually, [2] have sought medical attention for their condition. [18] Many of these visits are to the ED.

The composition of hemorrhoidal tissue is thought to consist of vascular, mucosal, and muscular tissues. Although frequently attributed to varicosities, it is likely that all three elements comprise the hemorrhoid. [31] There are two types of hemorrhoids: internal and external. [18] [19] Internal hemorrhoids

originate above the dentate line. [3] [25] They are covered with mucosa and lack sensory innervation. [19] Internal hemorrhoidal prolapse is painless, [19] [25] and acutely thrombosed internal hemorrhoids may only be mildly tender. [3] Gangrenous, thrombosed internal hemorrhoids, however, may be exquisitely painful.

Internal hemorrhoids can be further classified as first through fourth degree. First-degree hemorrhoids do not prolapse but can be identified on anoscopic examination. Second-degree hemorrhoids prolapse upon straining, but reduce spontaneously. Third-degree internal hemorrhoids prolapse upon straining, and can be reduced manually, whereas fourth-degree internal hemorrhoids prolapse and are irreducible. Fourth-degree hemorrhoids are prone to thrombosis (Fig. 46-6). [18]

External hemorrhoids originate below the dentate line [3] [25] and are covered with squamous epithelium. They are innervated

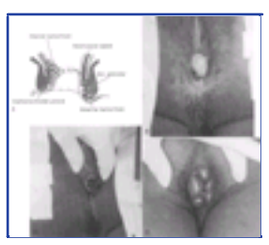

Figure 46-6 *A*, Anatomic location of internal and external hemorrhoids. *B*, Large thrombosed external hemorrhoid. *C*, This thrombosed external hemorrhoid spontaneously ruptured. This demonstrates the concept that a thrombosed hemorrhoid is actually a hematoma into soft tissues. *D*, Thrombosed prolapsed internal hemorrhoids. These hemorrhoids cannot be reduced and are quite painful. They should not be incised in the ED, a formal hemorrhoidectomy is required if conservative measures are not successful. They are often mistaken for a partial "rectal prolapse". *(A from Hill GJ II: Outpatient Surgery. 2nd ed. Philadelphia, WB Saunders, 1980. Reproduced by permission.)*

by the inferior rectal nerve. [18] [19] A thrombosed external hemorrhoid appears as a bluish mass covered by epidermis. [19] [25] What is commonly referred to as a thrombosed external hemorrhoid often represents an actual rupture of one of the veins of the external hemorrhoidal plexus into the surrounding tissue. This condition is more accurately termed an anal hematoma or external anal thrombosis. Acute thrombosis occurs suddenly and is usually very painful, and pain is the presenting complaint of most patients with this condition. [3] [25] Many patients will feel a tender mass and state that it hurts to sit. Significant bleeding is uncommon but may occur if spontaneous rupture occurs. [25]

Increased pressures from straining or trauma from constipation or diarrhea may exacerbate external hemorrhoids. [3] [25] Distention and trauma predispose the hemorrhoidal venous plexus to stasis with ensuing clot formation and edema. [12] [26]

Indications and Contraindications

ED management of prolapsed internal hemorrhoids is conservative [2] and includes sitz baths, stool softeners, fiber supplement, and increased fluid intake. [25] A useful mnemonic for conservative therapy is WASH: *w*ater (increase fluid intake, sitz baths in water), *a*nalgesics, *s*tool softeners, *h*igh fiber diet. [5] Referral for outpatient surgical consultation can be considered. [19] An important historical question is whether the patient "must push the hemorrhoids back in after a bowel movement." An affirmative answer suggests that the patient has symptomatic third-degree internal hemorrhoids, which would benefit from elective surgical referral. This condition can be easily demonstrated by having the patient strain before the DRE. Surgical consultation should be obtained in the ED for very distressing and painful fourth-degree hemorrhoids [25] and for profuse bleeding that is hemodynamically significant.

Without treatment, thrombosed external hemorrhoids will resolve spontaneously over 1 to 3 weeks, [19] [26] usually without complication (except for an occasional skin tag). [25] Small hemorrhoids that present acutely with minimal discomfort may be managed conservatively. [14] This includes pain medication, sitz baths, stool softeners, and a dietary fiber supplement. [3] The pain is most severe within the first 48 hours, [25] and patients presenting within this time window are most likely to benefit from excision (not incision and drainage) of their thrombosed external hemorrhoid. Patients who present after this time are usually best managed with conservative treatment. [14] [19] Bleeding disorders, serious systemic illness, or hemodynamic instability are all relative contraindications to excision in the ED.

Procedure

The patient is placed in the prone or lateral decubitus position. [25] [28] The buttocks can be taped apart to aid in visualization (Fig. 46-7). Parenteral analgesia and sedation can be given as an adjunct to local anesthesia. Local infiltration with 0.5% bupivacaine or 1% lidocaine with epinephrine at 1:100,000 [3] may be administered just under the skin, above the hemorrhoid [19] (Fig. 46-8). The overlying skin should blanch, indicating that anesthesia has been introduced at the appropriate depth. This is usually all the anesthesia that is required. A

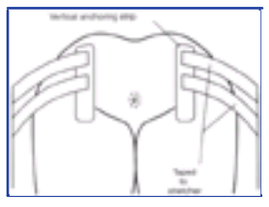

Figure 46-7 Taping the buttocks to gain exposure.

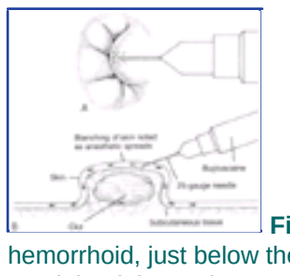

Figure 46-8 Very adequate anesthesia often can be obtained by a single injection of long-acting bupivacaine. *A,* A 25-ga needle is inserted in the middle of the swollen hemorrhoid, just below the skin surface. *B,* With the injection, the anesthetic spreads over the surface of the dome and into the surrounding tissue. A field block about the hemorrhoid can also be used, but it is much more painful, usually not required, and not advised as a first attempt at anesthesia.

field block may be necessary to anesthetize a larger area or multiple hemorrhoids but this is quite painful and rarely required. This can be accomplished by extending the injection circumferentially around the base of the hemorrhoid into the submucosal surface (Fig. 46-9).

The skin overlying the thrombosis is elevated with forceps. An elliptical incision is made around the clot and directed radially from the anal orifice (Fig. 46-10). The skin edges are then elevated with the forceps and excised to expose the underlying thrombosis. [19] The clot is removed with forceps[3] or by applying digital pressure (Fig. 46-11). Often multiple individual clots will be present. If any skin ulceration is noted over the hemorrhoid, it can be included in the excised portion. The wound is then packed loosely to prevent the skin edges from reapproximating prematurely. [3] Packing may be done with standard cotton gauze (e.g., NuGauze) or Gel-Foam.

For the trip home, a gauze pad can be placed between the buttocks and the buttocks taped together to hold it in place.

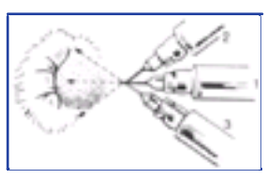

Figure 46-9 Subcutaneous injection of a long-acting local anesthetic using a field block will provide complete anesthesia during a procedure, but is rarely required.

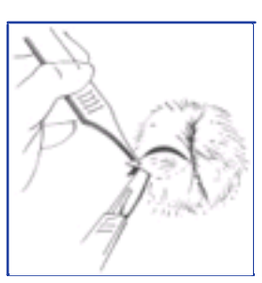

Figure 46-10 An unroofing technique uses an elliptic or triangular incision that removes a piece of the overlying skin. A simple linear incision should not be used.

The patient should be advised to avoid prolonged standing for the next few days. Sitz baths can be started as soon as the anesthetic has worn off. If the gauze has not fallen out, it can be removed by the patient after a sitz bath in 24–36 hours. Gelfoam packing avoids the need for this step, and is preferred as a packing agent. Minor bleeding can be expected, but it should be mimimal. After the packing has been removed, the patient may apply a soothing cream to the area for a few days (such as Preparation H, Anusol HC, lidocaine ointment). Routine stool softeners are unlikely to be of value for this episode, but may help prevent further episodes. Antibiotics are not indicated. Following a bowel movement the patient should avoid toilet paper for a few days and may wash the area with mild soap and water in the shower. Most patients with a straightforward case do not need a routine wound check and they should be relatively asymptomatic in 48 hours. If pain or bleeding persist, a wound check is necessary. If the clot has been removed a recurrence of this thrombosis is unlikely, but such patients are predisposed

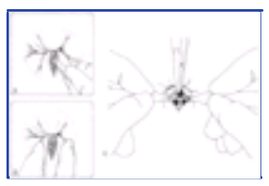

Figure 46-11 Blood clots are removed with forceps *(A)* or expressed with the fingers *(B)*. Often multiple clots are present, and all should be removed. *C,* After the initial clot is removed, an assistant spreads the incision to expose the base of the hemorrhoid and allow other smaller clots to be removed individually with forceps or with a small hemostat.

to future episodes. Permanent skin tags may be felt after the area has healed.

Long-term therapy should be directed toward avoiding constipation by increasing dietary fiber and fluid intake. [25]

If an invasive procedure is not well-tolerated by the patient, alternate nonoperative treatments using topical nitrates [13] [19] or topical nifedipine have been proposed. [1] [26] Applied to the thrombosed hemorrhoid, these creams relax the anal sphincter, relieve pain, and promote healing. Systemic absorption is minimal and the application is usually well-tolerated.

Complications

While complications are rare, bleeding and infection do occur. [19] Bleeding is usually simple skin oozing and stops with direct pressure. [3] When a simple incision and drainage (I&D) is performed instead of an elliptical excision, or when the ellipse of skin is not completely removed, the skin edges can close prematurely and cause infection and a permanent perianal skin tag. [3] [25] [26] Premature closure can also result in an incomplete evacuation of a developing clot.

MANAGEMENT OF ANORECTAL ABSCESS AND PILONIDAL CYST/ABSCESS

These topics are covered in detail in chapter 38 .

MANAGEMENT OF RECTAL FOREIGN BODIES

The emergency clinician is often the primary care provider for patients presenting with rectal FBs. Many of these objects can be removed successfully in the ED. [34] By following some simple guidelines, outpatient treatment can be practical and cost-effective.

The etiology of rectal FBs includes autoeroticism (most common), [20] iatrogenic placement (e.g., thermometer, enema tip), assault, self-administered treatment (e.g., enema), accidental ingestion, and concealment (e.g., body packing). The myriad of objects that have been removed include vibrators, sex toy phalluses, aerosol cans, light bulbs, glass bottles, billiard balls, fruits and vegetables, and small animals. [8] [11] [20] [21] Most objects are cylindrical.

Although the early medical literature stressed inpatient treatment because of isolated complications from difficult retrievals, the most recent series show a high success rate for the outpatient management of rectal FBs, if proper guidelines for evaluation and treatment are followed. [20] [34]

Diagnosis of a rectal FB can usually be made from the history. The physical examination should, therefore, concentrate on excluding anorectal or intestinal perforation and determining which objects will be accessible in the ED. [34] DRE will identify objects that are low-lying or palpable. These are most likely to be removed successfully in the outpatient setting. [20] [34] Plain radiographs can supplement the examination by delineating the shape, position, and number of the object(s). [11] [20] If a FB with a sharp edge is suspected from the history, the clinician is advised to forego the DRE.

Indications and Contraindications

All rectal FBs should be removed when they are diagnosed. Although some objects may pass spontaneously, delayed removal may lead to obstipation, pain, and infection, with or without perforation. An algorithm for the removal of FBs in the ED has been outlined (Fig. 46-12).

FB removal in the ED is contraindicated in patients who have severe abdominal pain or signs of perforation, a

Figure 46-12 Emergency approach to the removal of rectal foreign bodies. Foreign bodies that are fragile or are associated with rectal spasm are generally managed with regional or general anesthesia. The use of supplemental analgesic, anxiolytic, and local anesthetic medications is highly recommended.

nonpalpable FB, or broken glass in the rectum. Other situations precluding ED removal include a FB that is unusually difficult to remove (a set time limit has elapsed or the patient cannot tolerate removal), or when there is insufficient experience or equipment to perform the procedure. [34] Patients who present under these conditions require surgical consultation and admission to the hospital.

Equipment

The specific equipment required will often depend on the nature of the FB. In general, the clinician will need anesthesia and analgesia as described earlier, a speculum with a light source, and an instrument to grasp the FB. The speculum can be an anoscope, a rigid sigmoidoscope, a vaginal speculum, or a retractor (Fig. 46-13). Instruments useful for grasping the FB include ring forceps, tenaculum forceps, or obstetric forceps. In some instances, a Foley catheter or endotracheal tube will be helpful. A suction dart, [9] vacuum extractor,[20] and plaster of Paris have also been used to aid in FB retrieval. Individual situations may lead to creative use of standard medical equipment, but one must ensure safety before using a device to remove a FB.

Procedure

The technique for removal depends on the size, location, orientation, and composition of the FB. The patient can lie prone in the knee-chest position, or in a lateral decubitus position. Alternatively, the patient can be placed in the lithotomy position where pressure can be placed on the abdomen to help maneuver the FB toward the distal rectum. Parenteral analgesia is often required to relieve pain from anal stretching and manipulation. Intravenous sedation is almost always

Figure 46-13 *A,* Parks retractor (unassembled). *B,* Assembled Parks retractor. *C,* A Parks retractor inserted into the anal canal to visualize the foreign body. *(C from Sohn N, Weinstein M: Office removal of foreign bodies in the rectum. Surg Gynecol Obstet 146:209, 1978.)*

required to calm the patient and facilitate relaxation of the anal sphincter. A perianal block will also allow greater dilation of the sphincter. Local infiltration with 0.5% bupivacaine or 1% lidocaine with epinephrine at 1:100,000 may be administered circumferentially around the anus in the submucosal tissue [34] (see Fig. 46-9).

After analgesia and sedation are administered, the clinician performs a DRE to gauge the position and orientation of the FB. Suprapubic pressure from above, the examiner's finger from below, and the patient performing a Valsalva maneuver may successfully deliver the object without instrumentation. [34] The FB is often lodged against the sacrum posteriorly[20] and may require gentle redirection. This can be accomplished by cradling the posterior aspect of the object between two fingers, directing the object slightly proximally and anteriorly, and having the patient bear down gently.

If this is unsuccessful, or if the DRE reveals that the object has an accessible edge or lip, the FB can be extracted under direct visualization using an instrument. An anoscope, rigid sigmoidoscope, vaginal speculum, or retractor is first inserted into the anus as described above (see section on anoscopy). If the object is visualized clearly, a blunt instrument can be used to secure it. Gentle traction is then applied to remove the object, the instrument, and the scope or speculum as a single unit. The object must be grasped under direct visualization to avoid pinching or tearing the mucosa. [34]

Rigid sigmoidoscopes offer a unique advantage as air can be insufflated into the rectum around the FB. This technique

can be particularly helpful when retrieving glass objects. Glass rectal FBs often create a vacuum in the segment of bowel just proximal to where they lie. This makes removal with simple traction almost impossible. The vacuum can be released by distending the rectal wall around the object with air. If a sigmoidoscope cannot be used to retrieve a glass object, one or two Foley catheters or an endotracheal tube can be passed beyond the FB and the balloon or cuff inflated, respectively. This may also release any vacuum. The object can then be removed using the inflated balloons with gentle traction (Fig. 46-14). Often specific equipment is not available to remove all foreign bodies, and the clinician must improvise based on the circumstances. Something as simple as 2 large spoons or an endotrachael tube may be used in lieu of complicated forceps and clamps (Fig. 46-15 (Figure Not Available)).

Besides creating a vacuum, glass objects are especially difficult to remove because they can break and cause a tear or perforation in the rectal wall. If forceps are used for retrieval of a glass object, the grasping edge should be coated in rubber [29] or plastic, or should be padded with gauze. Plaster of

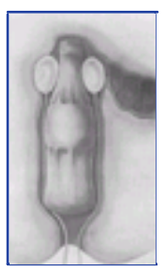

Figure 46-14 Use of Foley catheters to remove a rectal foreign body made of glass. *(From Eftaiha M, Hambrick E, Abcarian H: Principles of management of colorectal foreign bodies. Arch Surg 112:693, 1977.)*

Paris has been used to remove a hollow glass object if the object has an open end facing distally. A hollow tube (e.g., an endotracheal tube or small chest tube) is inserted into the open end. The FB is filled with plaster using a large irrigation syringe to inject plaster through the hollow tube. The plaster then cools around the tube, which can be used as a handle to remove the object using gentle traction. Care must be taken with this technique to avoid leaking plaster onto the mucosa. Additionally, heat is released as the plaster hardens, and may cause the glass to crack or shatter. After removal, sigmoidoscopy is commonly recommended to evaluate for edema and possible perforation of the mucosa. [20] [34] Patients with normal postextraction examinations and no evidence of perforation may be safely released home after a period of observation.

Observation in the ED to allow small, high-lying or non-palpable FBs to pass into the lower rectum has occasionally been successful. [34] However, this may not be a feasible use of ED resources as there is no way to gauge how much time will be required or which cases will be successful. All cases of nonpalpable rectal FBs warrant surgical consultation in the ED. Enemas or cathartics should not be used, because they may increase the impaction of a FB or cause it to move higher into the colon.

Complications

Perhaps the most common complication of this procedure is failure to remove the rectal FB. This should prompt a surgical consultation in the ED. The most serious complication of rectal FB retrieval is perforation or deep mucosal tear. Both of these require hospitalization and may necessitate surgery. Cracking or shattering of glass may also require surgical exploration and retrieval. Mild mucosal edema and rectal bleeding are common complications of prolonged rectal FB placement and of retrieval. [11] These may not require any specific treatment. However, when post-extraction sigmoidoscopy is warranted and the emergency clinician is not skilled in this procedure or when other findings are present (e.g., post-procedural abdominal pain, fever, rectal bleeding, rectal discharge), surgical consultation is recommended.

MANAGEMENT OF RECTAL PROLAPSE

Rectal prolapse is the protrusion of some or all of the layers of the rectal wall through the anal orifice. [10] Prolapse is usually not an emergency, but manual reduction is usually easily accomplished in the ED. The most common presenting complaint is protrusion of a rectal "mass" or tissue. [6] Patients may also complain of "hemorrhoids," pain on defecation, itching, incomplete evacuation, incontinence, or bloody mucosal discharge. Rectal prolapse is diagnosed by visual inspection of the anus and DRE. The differential diagnosis includes hemorrhoids, polyps, cystocele, or carcinoma. [15]

There are three types of prolapse: (1) Complete prolapse, or procidentia, involves all layers of the rectum protruding through the anal orifice (Fig. 46-16 and Fig. 46-17). (2) Incomplete, or occult prolapse, describes internal prolapse that does not reach the orifice. This type is difficult to diagnose in the ED and requires no emergency intervention. (3) Mucosal prolapse is limited to mucosal protrusion through the anal opening. [10] [15] [32]

Figure 46-15 (Figure Not Available) *A*, Large spoons grasping a fragile foreign body in the rectum. *B*, Use of an endotracheal tube to remove a smooth foreign body made of glass. An advantage of this technique and the Foley catheter technique is that air is introduced above the obstruction to overcome the vacuum created by the foreign body. *(A, From Rosen P, Baker FJ, Barkin RM, et al (eds): Emergency Medicine: Concepts and Clinical Practice. St. Louis, CV Mosby, 1988. B, From Garber H, Rubin R, Eisenstat T: Removal of a glass foreign body from the rectum. Dis Colon Rectum 24:323, 1981. Reproduced by permission.)*

Complete and partial prolapse can be distinguished from each other by digital palpation. A thick muscular layer of tissue between the examiner's thumb and forefinger suggests complete prolapse. Partial or mucosal prolapse may demonstrate radial rectal folds protruding through the rectum and rarely extends more than 3 to 4 cm from the anus.[3] [6] Complete prolapse can extend 10 to 15 cm outside the anal verge.[3] [10]

Rectal prolapse can occur in children or adults. Prolapse in children is typically incomplete or mucosal. It usually affects children younger than age 3 years, and is often associated with

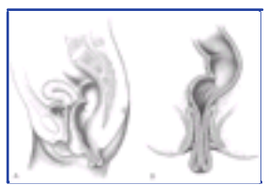

Figure 46-16 *A,* Type I procidentia (rectal prolapse). *B,* Intussusception of sigmoid colon beyond the anus. *(From Kratzer GL, Demarest RJ: Office Management of Colon and Rectal Disease. Philadelphia, WB Saunders, 1985, pp 221–333.)*

cystic fibrosis, parasitic infection, chronic diarrhea, malnutrition, or as a sequelae of chronic neurological disease. Prolapse is usually self-limited and outpatient management (after manual reduction) includes correcting constipation, [3] avoiding straining, and referring for testing to exclude cystic fibrosis. [15] [17] Rectal prolapse in adults occurs most often in older women. Nursing home patients may have recurrent prolapses. The etiology is poorly understood, but is associated with chronic constipation, chronic neurological conditions, or pudendal neuropathies that weaken the anal sphincter. [10] [17] [32]

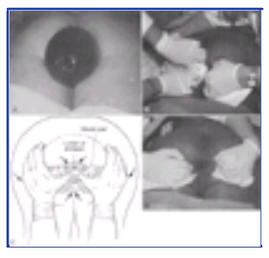

Figure 46-17 *A,* Complete (recurrent) rectal prolapse in a nursing home patient. This can be very distressful and painful, or relatively asymptomatic. *B and C,* To reduce the prolapse, an assistant spreads the buttocks and the clinician's hands encircle the prolapse. The key to success is adequate sedation/analgesia and a slow steady gentle pressure. *D,* Successful reduction.

Indications for Reduction

Rectal prolapse may be reduced in the ED. If successful, outpatient surgical referral is appropriate. Definitive surgery may be attempted but occasional prolapses in debilitated patients are usually treated conservatively. [15] (Referral for outpatient proctoscopy should be made to search for a polyp or malignancy that may have acted as a lead point.[17] If the prolapse is incarcerated, a surgical consultation should be obtained in the ED. [15]

Procedure

Reduction of a mucosal prolapse can usually be accomplished easily with a few minutes of gentle, constant pressure on the mass. [15] In children, intravenous sedation may be necessary to allow reduction. Children are often more relaxed if they are allowed to remain in the parent's lap during the procedure. After reduction, the child is sent home with a pressure dressing and stool softeners. The parents are counseled on the use of dietary fiber and increased fluid intake to prevent constipation and straining.[15] The child is then referred for outpatient follow-up as described earlier. [16] [17] [24]

Reduction of a complete prolapse is accomplished with the patient in the prone or lateral decubitus position. Parenteral sedation may be required if the patient is anxious or is having difficulty relaxing the sphincteric muscles. The buttocks can be taped apart to aid in reduction (see Fig. 46-6). Constant, gentle circumferential pressure is applied to the prolapsed area, beginning with the portion closest to the lumen (the most distal segment). The examiner's thumbs are placed on either side of the lumen while the fingers grasp the exterior walls. The thumbs then apply pressure as the walls are rolled inward to force the prolapse back through the anus. [15] A DRE should be performed gently after reduction to search for a mass that may have acted as a lead point. [6]

Complications

Complications after a successful reduction are uncommon, but may include bleeding and ulceration. Failure to reduce a prolapse is a serious complication that requires surgical consultation. A persistently prolapsed rectum can result in ulceration, strangulation, and perforation of the bowel wall. [6] Moreover, the possibility of anal sphincter tone loss and

incontinence increases with delays in reduction of rectal prolapse. [15] [17]

CONCLUSION

Prompt attention and management of anorectal complaints in the ED may reduce delay in the diagnosis and management of diseases that involve this area. A caring clinician who is sensitive to the patient's concerns will facilitate treatment. Many conditions affecting the anorectum have associated medical or surgical issues, and a cooperative approach to their management will optimize patient care.

Acknowledgments

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Section VIII - Musculoskeletal Procedures

Chapter 47 - Prehospital Splinting

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Brett S. Greenfield

SPINE IMMOBILIZATION

Despite a lack of data to show that it improves outcomes, spinal immobilization remains one of the most frequently performed prehospital procedures. [1] [2] Recommendations for spinal immobilization date back to 1971 when the American Academy of Orthopedic Surgeons [3] published the first set of guidelines for spinal immobilization following blunt trauma. In these guidelines, the presence of signs (e.g., weakness, paralysis) or symptoms (e.g., neck pain, paresthesias) of spine injury were the primary indications for spine immobilization. [3] Since then, recommendations for spine immobilization have evolved considerably. Today, indications for spine immobilization are based primarily on mechanism of injury, regardless of the presence or absence of symptoms or physical findings suggestive of a spine injury. [2] [4] This has resulted in routine prehospital spinal immobilization in all but the most trivial injuries.

While mechanism of injury is widely accepted as an indication for prehospital spine immobilization, recent focus on evidence-based medicine has led some investigators to question this practice. [4] [5] [6] The results of several trials support the use of clinical criteria for cervical spine clearance by prehospital care providers. [4] [7] [8] [9] [10] A few studies have found that unnecessary spine immobilization may be associated with increased morbidity and cost. [2] [11] [12] While large prospective studies will be needed before prehospital spine clearance gains widespread acceptance, studies to date have prompted several emergency medical service (EMS) systems across the country to establish specific clinical criteria for prehospital spine immobilization (Table 47-1). [2] [4] [9] [10] [13]

Despite the growing controversy regarding the practice of routine prehospital spine immobilization, mechanism of injury remains the primary indication for spine immobilization in the prehospital setting. This section outlines the techniques and equipment used by most prehospital care providers in the United States. All emergency care providers should be well trained on their use, limitations, and application.

TABLE 47-1 -- Clinical Criteria for Prehospital Spine Immobilization

Mechanism of injury
Spine pain or tenderness
Focal neurologic deficit
Unreliable patient examination (not awake, alert, oriented, calm, and cooperative)
Head injury (including severe head and facial trauma)
Altered mental status:
no available history;
found in the setting of possible trauma (e.g., lying at the bottom of a staircase); or
near drowning with a history or high probability of a diving injury
Distracting injury
Communication barriers
Extremes of age

CERVICAL SPINE

Trauma patients frequently sustain injuries to the cervical spine. These injuries are particularly devastating when associated with injury to the spinal cord. In the United States approximately 10,000 to 12,000 spinal cord injuries occur each year. [1] [14] Of these, more than half are in the cervical region, and most are a result of motor vehicle crashes.[14] The remainder are due to falls, recreational activities (e.g., diving, contact sports), and penetrating trauma. Therefore, it is critically important that the unstable cervical spine be immobilized early and effectively.

Because the minimum degree of motion required to cause spinal cord injury has not been defined, the goal of immobilization is to maintain the head in a neutral position with zero degrees of motion in all directions. [15] To immobilize the entire cervical spine, an orthotic device must fix the head, hold the occiput and mandible, and restrict motion at the cervicothoracic junction.[15A] This approach follows the basic orthopedic principle of immobilizing the joint above and below a suspected injury. In addition, to be useful in the prehospital setting, a cervical immobilization device must be portable and easy to apply, and must allow access to the upper airway.

At present, the standard technique of spinal immobilization involves early manual stabilization of the head and neck relative to the long axis of the body. Manual stabilization is usually followed by the application of a cervical collar. Although its use is controversial, a cervical collar has value as an early adjunct in the sometimes complex process of immobilization and extrication. It is extremely important to remember, however, that even the best-supporting collars *do not* provide adequate immobilization when used independently. For complete cervical spine immobilization, the patient must also be secured to an intermediate spine-immobilizing device (e.g., short spine board, KED) or a full-length spine board (e.g., backboard, scoop stretcher, full body splint), or both.

Complete cervical immobilization must also incorporate lateral stabilization of the head in the form of lightweight bulky objects such as foam blocks, towel rolls, blanket rolls, or cushions and tape. This can also be accomplished using factory-made devices such as the HeadBed (Laerdal Medical Corp., Wappingers Falls, NY) or Bashaw Cervical Immobilization Device (CID) (Bashaw Medical, Inc., Pensacola, FL).

Background

Since 1965, an entire industry aimed at prehospital preservation of life and limb has evolved. Specially manufactured spinal immobilizers were developed for field use during the late 1960s and early 1970s. Numerous variations of these early devices have been developed to solve specific prehospital problems and to meet the need for lightweight, durable, adaptable, and affordable equipment. Many of the latest designs have been possible by advances in composite

technology that have produced more versatile plastics and stronger adhesives.

It should be stressed that although there is widespread agreement about the basic steps of spinal immobilization, prehospital care is a difficult setting for individuals who cannot easily adapt. Because of the variety of circumstances that confront prehospital care providers on a daily basis, flexibility is mandatory.

In 1965, Louis Kossuth was the first clinician to support the need for extrication standards, including immobilization of the cervical spine. [16] J.D. Farrington, a pioneer in the use of the spine board for cervical immobilization, is credited with thrusting the concept of prehospital spinal immobilization into the venue of conventional medicine.[17] [18] Farrington emphasized that spinal fractures were frequently mishandled and made worse by rough or hasty movement at the scene. He outlined the use of a backboard, sandbags, and tape for the prehospital extrication and care of patients with suspected spinal injuries. He fashioned extrication collars using universal dressings held in place by soft roller bandages and advocated manual traction during the extrication phase. [18]

Manual *traction* is no longer recommended, because it may aggravate an underlying spinal injury. [19] In addition, although sandbags are effective devices for lateral immobilization, they may cause significant movement of the neck if the board is suddenly tilted (e.g., to decrease the risk of aspiration in a vomiting patient). As a result, sandbags have been replaced by more ergonomic and lighter weight devices such as foam blocks, the HeadBed, or Bashaw CID. Despite these modifications, the original Farrington method of splinting the head and torso to a rigid object remains the preferred technique for effective spinal immobilization.

Indications

An extrication collar should be used as a primary adjunct in cases involving trauma to the head and neck. In the absence of visible trauma, patients should be immobilized whenever the mechanism for a potential cervical spine injury exists. The most common mechanism of injury involves sudden deceleration of an automobile, resulting in hyperflexion and hyperextension forces. Patients under the influence of alcohol or drugs lack the self-awareness to recognize their own spinal injury and should be immobilized routinely. Likewise, every unconscious trauma patient should be immobilized to avoid aggravating an underlying spinal injury. Any awake and alert trauma patient who complains of spine pain, paresthesia, weakness, or absent movement should be immobilized carefully to avoid secondary injury to the spinal cord. Also, extremes of age and the presence of communication barriers (e.g., language, hearing impairment) may impact the ability to accurately assess the patient's perception and communication of pain, and should lower one's threshold for spinal immobilization. [2] [4]

It is not uncommon to encounter patients in the field who are both conscious and oriented but unaware of their own neck injuries. The sensory impact of having one's automobile forcibly disassembled by rescue personnel who are using modern tools that produce noise in excess of 80 dB may result in considerable distraction. In addition, the presence of other painful injuries or concern over other victims can easily mask the manifestations of an occult cervical spine injury.

Serious cervical cord injuries can also occur in the absence of demonstrable fractures. Spinal cord injury is common in elderly patients with cervical spondylosis, in whom an arthritic osteophyte may sever a portion of the cord as permanently as a fracture or dislocation. In such cases, there is often little or no subjective pain, and the mechanism of injury may appear seemingly minor.[20]

Therefore, a high index of suspicion for possible spinal injury must be maintained at all times. In all cases, the medical record must include documentation of the neurologic examination before and after spinal immobilization.

The purpose of an extrication collar is to assist in splinting the head and neck either therapeutically or prophylactically in a neutral position. [15A] The collar is useful for the following reasons:

1. It provides airway protection by limiting flexion in a patient whose unsupported jaw and neck position threatens patency.
2. It helps reduce cervical spine motion, especially flexion, but also rotation, lateral bending, and extension. In this regard, however, it serves only as an adjunct.
3. If properly chosen, it can support the weight of the head while the patient is sitting and help to maintain the alignment of the cervical spine once the patient has been moved to a supine position.
4. An equally important function is to serve as a reminder to the patient and rescuers that the integrity of the basilar skull and cervical spine are suspect because of the mechanism of injury.

The cervical collar does not provide complete immobilization of the head and neck. The collar was designed as an adjunct and was never intended to provide definitive immobilization in itself. Complete immobilization is not possible until the patient is properly secured to a long backboard-type device. Nonetheless, the collar should go on first and remain in place during the entire procedure.

Contraindications

There are few circumstances that preclude the use of an extrication collar. The presence of a surgical airway (i.e., cricothyroidotomy or tracheotomy) may require modification of the cervical immobilization technique.

In addition, cervical dislocation with fixed angulation or a preexisting anatomic abnormality may prevent effective application of factory-made collars. This situation is rarely encountered and can be managed with an improvised cervical immobilizer, such as a version of a horse collar or prolonged manual positioning without traction.[21]

A third circumstance that could preclude the use of a collar is massive cervical swelling (e.g., secondary to hemorrhage or tracheal injury). In these circumstances the compressive effect of a collar may impede air exchange, decrease cerebral perfusion, or increase intracranial pressure. [22] [23] [24] Finally, the presence of an impaled foreign body such as a knife, a piece of glass, or metal can also make cervical spine immobilization using extrication collars difficult.

Note that improvised cervical support may work better than a manufactured collar, depending on the size and shape of the patient. This is particularly true in the pediatric population, as collars come in limited sizes and may not be well tolerated. In such circumstances, prolonged manual in-line

stabilization is often necessary. Again, adaptability is a trait of the well-trained and experienced prehospital provider.

Equipment

There are three types of cervical collars: cervical, head-cervical, and head-cervical-thoracic. Traditionally, cervical collars have used a four-point support structure at the bottom of the collar: namely at the two trapezius muscles posteriorly and at the two clavicles anteriorly (Fig. 47-1). Most modern collars are modified rigid head-cervical-thoracic devices that use the sternum as a fifth support structure (Fig. 47-2). Current collar designs support the head with wing-like flaps on the collar's upper posterior edges. Anteriorly, the collar supports the mandible. The collar's flaring design generally prevents compression of the thyroid cartilage and cervical vessels, even when applied firmly.

Soft collars, although comfortable, have no role in spinal immobilization, because they provide minimal support and do not reduce cervical motion to any significant degree.[25] [26] However, the semirigid collar must be comfortable to ensure patient compliance. Dick and Land described the characteristics of an "ideal collar." [27] These features included the following:

1. It should support the weight of the head in a neutral position.
2. It should prevent lateral, rotational, and anteroposterior movement of the head.
3. It should be comfortable, translucent on radiographs, and compact.
4. It should be easy to apply.
5. Its price should be such that it can be carried in sufficient numbers in various sizes by any ambulance.
6. It should not interfere with the position or function of important airway structures, nor adversely affect cerebral circulation in any way.
7. Simplicity of design should permit its application by 2 rescuers in less than 60 seconds, in darkness, rain, or cold weather, without manipulation of the head or neck.
8. It should be available in the smallest number of sizes possible.

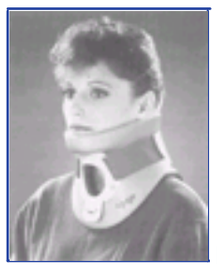

Figure 47-1 Philadelphia collar. This is a two-piece, high-type collar that comes in four sizes. The collar supports the head in a dish-shaped contour that is formed when the front and rear halves are joined by Velcro fasteners. When properly sized for a patient, this collar provides excellent support. When applied too tightly, it tends to force the mandible backward and can cause thyroid compression in some patients. It is extremely comfortable. *(Courtesy of Philadelphia Cervical Collar Company, Westville, NJ.)*

Figure 47-2 Stifneck collar. This collar is made of high-density polyethylene (a hard material) and padded with semiflexible foam margins. Note the low-reaching anterior panel, which contacts the sternum for additional support. *(Courtesy of Laerdal Medical Corporation, Wappingers Falls, NY.)*

Other investigators have attempted to evaluate cervical collars in an objective fashion. The accepted gold standard for comparison is the halo brace, which restricts motion to 4% flexion-extension, 1% rotation, and 4% lateral bending. [15A] Unfortunately, even the best cervical collars (when used independently) restrict flexion and extension by only 70% to 75%, and overall neck movement by less than or equal to 50%. [28] A number of studies have evaluated neck motion in volunteers immobilized supine on a backboard with various collars in place. [1] [25] [26] [29] [30] [31] [32] [33] [34] While these studies demonstrated small differences among some of the collars, overall they merely confirm the fact that cervical collars alone are inadequate to immobilize the cervical spine completely. Thus, it is important to keep in mind that for effective cervical spine

immobilization, differences among various types of cervical collars are less important than proper application, fit, and most importantly, use of adjunctive equipment.

Little information is available regarding the proper selection and application of spinal immobilization devices for children. Most of the available data were derived from studies of adults and may not be applicable to children. Half of the total growth in head circumference is achieved by the age of 18 months, giving children a disproportionately large head compared with the rest of the body. Prior to age 8, these anatomic and developmental differences result in a higher incidence of upper cervical spine injuries (C1–2). Because injuries in this area are frequently unstable, proper cervical immobilization in the *neutral* position is critically important. Schriger defined the "neutral" position as the normal position of the head, neck, and torso when the patient is upright and looking directly ahead. [35] In this position, the pediatric cervical spine is normally lordotic or extended. [24] However, because the head is large, positioning the child's body on a standard backboard may force the neck into flexion or a relative kyphosis. The clinical significance of this is currently unclear, but theoretically may be hazardous for young children. Therefore, the standard backboard should be modified to adapt to the child's larger head size. As a rough guide, the external auditory meatus should be on the same level as the mid-shoulder. Suggested modifications include a cutout in the backboard that accommodates the occiput or a pad under the back at the level of the chest (Fig. 47-3). If not modified, the standard backboard in conjunction with the disproportionately large head of a child may force the neck into hyperflexion, potentially aggravating an underlying cervical spine injury. [36] Nypaver and Treloar showed that all children required elevation of the back (mean height, 25.4 ± 6.7 mm) for correct neutral position on a spine board. Children younger than 4 years old required more elevation than those 4 years old or older. [37] It must be pointed out, however, that

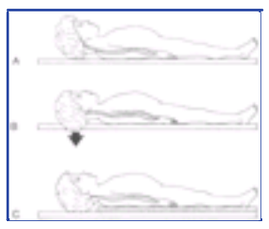

Figure 47-3 *A,* Young child immobilized on a standard backboard; note how the large head forces the neck into flexion. Backboards can be modified by an occiput cutout (*B*) or a double mattress pad (*C*) to raise the chest. *(Adapted from Herzenberg JE, Hensinger RN, Dedrick DK, et al: Emergency transport and positioning of young children who have an injury of the cervical spine. J Bone Joint Surg Am 71:15, 1989.)*

Figure 47-4 Pediatric high-cut extrication collar. *(Courtesy of Laerdal Medical Corporation, Wappingers Falls, NY.)*

there have been no published reports of a cord lesion resulting from the use of standard immobilization techniques and equipment in children. [24]

In a study addressing pediatric cervical spine immobilization, Huerta and coworkers evaluated the performance of infant and pediatric collars using a mannequin model.[38] They concluded that the high-cut collar in conjunction with a rigid backboard and lateral neck stabilizers provided the most effective spine immobilization (Fig. 47-4 and Fig. 47-5). In some children, these devices may provoke struggling because of fear, which has the potential to aggravate an underlying injury. In these situations, manual immobilization may be more effective and less frightening for the child. [39]

Procedure

Application of an extrication collar is a straightforward procedure (Fig. 47-6). A collar should be treated as a splint. The normal axiom in splinting is to immobilize the joint above and below the area of injury. Because no collar performs this function perfectly, a rescuer should be charged with maintaining manual in-line cervical stabilization in the neutral position during collar application and until the patient can be fully immobilized in an intermediate stage corset-type device or on a full backboard. The rescuer's intentions should be thoroughly explained to the patient throughout the procedure.

The neck should be examined before application of the collar for swelling, ecchymosis, deformity, or penetrating wounds. Once the collar is in place, a conscious patient should be cautioned repeatedly against movement of the head. Any persistent complaints of pain or dyspnea by the patient

Figure 47-5 LSP pediatric immobilization board with cervical collar. *(Courtesy of Allmed, St. Louis.)*

should be investigated by removal and possible replacement of the device while manual stabilization is maintained. The collar size should be determined using the manufacturer's suggested guidelines. For example, the Stifneck collar (Laerdal Medical Corp., Wappingers Falls, NY) is available in various sizes and uses the distance from the top of the shoulder to the chin to determine size (Fig. 47-7). The tallest collar that does not cause hyperextension should be used. For extremely short necks, a special extrication collar such as the No-Neck (Laerdal Medical Corp., Wappingers Falls, NY) is recommended (Fig. 47-8).

In cases in which an extrication collar of the proper size is not available, an improvised device should be made from available materials (Fig. 47-9). Once the patient is in the supine position and firmly secured to a backboard, lateral stabilization should be added, using foam blocks and tape or a factory-made lateral neck stabilizer (Fig. 47-10). Because immobilization on a flat backboard has been shown to place most adult patients into relative cervical extension, it is recommended that occipital padding (mean, 3.8 cm) be added to restore neutral position in adults. [35]

It should also be remembered that application of a cervical collar should not be attempted until the patient's head has been brought into a neutral position, and manual in-line stabilization has been applied. [28] If the patient experiences cervical muscle spasm, increased pain, neurologic complaints (e.g., paresthesia, weakness), or airway compromise, movement of the head and neck should be halted immediately. In these situations, patients should be immobilized in the position they are found using an alternative technique (e.g., blanket, towel roll).

Complications

Improper application of an extrication collar can occur if the wrong size is used or too little care is exercised during placement. The best means of preventing either error is strong clinician involvement in the training and continuing education of rescue crews, with vigorous feedback in cases of correct and incorrect application. In addition, adherence to the manufacturer's collar-specific recommendations for size and application should be emphasized.

A collar that is too small for a patient may be either too tight for the girth of the neck (with obvious complications) or too short to provide adequate immobilization. Too large a collar commonly results in hyperextension, which can exacerbate a preexisting spinal injury.

Improper or prolonged application of an extrication collar may impede venous return and raise intracranial pressure (ICP). [40] This is sometimes manifested by facial flushing and is more common with the short collar types.[22] While the clinical significance of increased ICP produced by cervical immobilization is still unknown, two studies have confirmed that the application of a rigid cervical collar causes a statistically significant and sustained rise in ICP. [41] [42] Kolb and coworkers reported a 24.8 cm H_2O increase in cerebrospinal fluid (CSF) pressure in 20 adult patients undergoing lumbar puncture. Hunt and associates reported a 4.6 mm Hg mean rise in ICP in 30 patients with severe traumatic brain injury. The largest rise in ICP was noted in patients with a baseline ICP greater than 15 mm Hg. The authors of these studies concluded that elevation of ICP produced by cervical immobilization might have deleterious effects in patients with acute or sustained intracranial hypertension.[41] [42]

The long-term use of the Philadelphia extrication collar as part of the treatment plan for an underlying cervical spine injury has been associated with pressure ulcers of the scalp.[43] Because some collars (e.g., Philadelphia [Philadelphia Cervical Collar Co., Westville, NJ] and Stifneck) have been shown to exert higher capillary closing pressures at contact points, it is suggested that collars with favorable skin pressure patterns and superior patient comfort (e.g., NECLOC [Jerome Medical, Moorestown, NJ]) be used in these settings.

One final complication should be mentioned. The patient who, for whatever reason, actively resists placement of an extrication collar or other splint should not be forced to wear it. Immobilization of the combative patient cannot be accomplished without considerable muscular exertion not only by rescuers but also by the patient.

If fractures do exist, it is possible that struggling can cause further damage. If the patient permits manual stabilization, this should be maintained as an alternative.

Conclusion

Cervical spine immobilization is an important skill for all emergency care providers. The use of a rigid cervical collar is an important first step in the immobilization of patients with potential cervical spine injuries. However, the use of a cervical collar alone does not provide complete immobilization. Proper cervical spine immobilization requires the use of a cervical collar, backboard, and lateral support device to ensure complete immobilization of the head and neck.

Figure 47-6 Technique for the application of an extrication collar. *(Courtesy of Laerdal Medical Corporation, Wappingers Falls, NY.)*

Figure 47-7 Method for predetermining the correct size for an extrication collar. *(Courtesy of Laerdal Medical Corporation, Wappingers Falls, NY.)*

Figure 47-8 No-Neck extrication collar. Designed for individuals with extremely short necks. *(Courtesy of Laerdal Medical Corporation, Wappingers Falls, NY.)*

Figure 47-9 Horse collar. Most extrication collars are available in three to five factory sizes. If a collar is not sized properly to fit a particular patient, it performs no function. Patients with extremely long necks or especially short ones can be immobilized by means of a horse collar fashioned from a bulky rescue blanket. The blanket is rolled to the thickness desired and slid under the patient's neck while a bystander applies manual stabilization; the ends of the blanket are then brought across the patient's anterior chest. The patient's forearms are fastened as shown to stabilize the "tails" of the collar. *(From Dick T: Tricks of the trade: Horse sense, immobilizing necks that don't fit collars. J Emerg Med Serv 7:23, 1982. Reproduced with permission.)*

Figure 47-10 Extrication collar combined with a cervical immobilization device. *(Courtesy of Laerdal Medical Corporation, Wappingers Falls, NY.)*

THORACOLUMBAR SPINE

Adequate full-body thoracolumbar spine immobilization is best accomplished by means of a full-length spine board (also called a *backboard*). Full-body spinal immobilization includes early application of a cervical collar, lateral immobilization of the head and neck, and ample strapping of the entire body to the backboard. Proper strapping minimizes movement whenever the backboard is used to transport the patient over long distances. In addition, this will help limit spinal movement associated with backboard tilting, which may be necessary in the likelihood of emesis or during transport of pregnant females in the second or third trimester.

Transferring a victim from a location and position of origin to a backboard may require the use of an intermediate-stage immobilization device such as a short spine board or a corset-type device. Corset-type devices have extensions that engage the head and neck and are equipped with weight-bearing loops that allow easier handling of the patient. Intermediate immobilizers or extrication splints should be used when a patient must be removed from a confined environment or when circumstances require movement in or from a sitting position.

In some circumstances, a threatening environment (fire, hazardous material incident, extreme weather) or patient condition (compromised airway, shock) may necessitate *rapid extrication*. Rapid extrication is the process of patient removal and spinal immobilization using an abbreviated manual technique. [1] It is performed by first bringing the patient's head into the neutral position. A cervical collar is then applied and the patient is transferred to a backboard without using an intermediate-stage device. Manual in-line stabilization of the spine is maintained throughout the procedure.

When extrication is not required by a patient's location, position of origin, or route of egress, the patient is most often found lying at ground level. With an extrication collar in place and in-line manual cervical immobilization, the patient can be logrolled onto a backboard. [44] Visual inspection of the back should be carried out during the logrolling process while the body is kept in a single plane.

McGuire and colleagues evaluated the safety of the logroll maneuver in three different settings: a volunteer with a stable spine, a cadaver with a surgically created unstable thoracolumbar injury, and a patient with a T12–L1 fracture-dislocation. [45] Radiographic studies on the volunteer demonstrated mild lateral movement during logrolling. The cadaver demonstrated a 2.1-cm anteroposterior displacement and a 5-mm lateral displacement during the logroll. The injured patient demonstrated no anteroposterior displacement but did experience a 7-mm lateral displacement when logrolled onto the side. Suter and coworkers studied variations of the logroll maneuver in healthy volunteers to determine the effect of axial traction and subject arm positioning on thoracolumbar movement. [46] Their study demonstrated significant thoracolumbar movement during the logroll maneuver that was consistently minimized by positioning subjects with the arms extended at the sides and the palms resting on the thighs. These studies raise concerns about the safety of the logroll maneuver and suggest that extreme caution be used during transfer of patients with suspected spinal injuries. Despite these concerns, there have been no reports of neurologic deterioration following a logrolling maneuver. Consequently, logrolling remains a widely accepted method of transferring patients on the ground to a backboard.

An optional but effective means of moving a supine patient is provided by a type of stretcher that breaks apart longitudinally and can be slid beneath a victim without disturbing his or her position. The halves of this scoop stretcher are anatomically contoured to enhance comfort and limit lateral movement of the immobilized patient. Unfortunately, visual inspection of the back is not possible with the use of a scoop stretcher.

A third means of both immobilizing and moving a trauma victim consists of a specially designed full-body splint, complete with factory-made straps or harnesses. There are several such immobilizers, most of which are highly effective and provide good lateral stability as well as anatomic conformity. One such device that is popular in European countries is the vacuum stretcher. A full-body

Figure 47-11 The Evac-U-Splint mattress. *(Courtesy of Hartwell Medical, Carlsbad, CA.)*

splint marketed in the United States (Evac-U-Splint [Hartwell Medical, Carlsbad, CA]) offers fast, full-body immobilization that supports the entire patient without creating pressure points (Fig. 47-11). In general, the patient must either be logrolled or lifted, using a scoop stretcher or full backboard, onto one of these devices.

Background

Until 1965, the principle of "rapid transportation above all" held widespread acceptance among rescuers, who had little or no orthopedic training, and among clinicians whose emergency care experience, by modern standards, was just as limited. The most commonly agreed-on means of getting a sitting patient out of a wrecked automobile was to use some version of a chair-carry. If the patient originated in a position other than the sitting position, the patient was first placed into a sitting position and *then* moved by means of a chair-carry or simply dragged out of the vehicle.

Col. Louis Kossuth, commander of the U.S. Air Force's Medical Service School at Gunter Air Force Base in Alabama, made note of several automobile crashes in which he thought patients were handled roughly by bystanders who were trying to help. [16] Finding the medical literature lacking at that time, he made recommendations regarding how these victims should be handled. He experimented with a set of canvas splints reinforced with semirigid steel stays as slats, similar to the modern Kendrick extrication device (KED) (Ferno Model 125, Ferno-Washington, Inc., Wilmington, OH). In addition, Kossuth has been credited with developing the first modern-type spine board. [18]

In 1967 and 1968, Farrington wrote two classic articles that showed the use of an extrication collar, spinal traction, nine-foot webbing straps, and both short and long spine boards to remove people in every conceivable position from automobiles. [17] [18] Much of today's extrication theory is essentially identical to what was taught by Farrington.

In 1967, the Committee on Trauma of the American College of Surgeons listed the minimum amount and type of equipment that should be carried in ambulances. This list included both short and long spine boards with accessories. [47] A similar document published the following year by the National Academy of Science also listed medical requirements for ambulance equipment and recommended short and long spine boards. [48] Since then, the list of ambulance supplies and equipment has evolved greatly, but the requirement for both short and long spine boards (or their equivalent) has not changed. [49]

Indications and Contraindications

Any mechanism capable of causing injury to the cervical spine should prompt rescuers to immobilize not only the head and neck, but also the entire body. The motion of any one vertebral joint is impossible to isolate. For complete spinal immobilization, the head, neck, and torso must be fastened into a single common plane. Extrication devices (e.g., short spine boards) will accomplish this to some extent, although movement of nonimmobilized lower extremities can lead to secondary movement of the pelvis and lumbar spine. Movement of the lumbar spine may also induce thoracic movement to some extent. Considering the fact that the most

feasible position for transport is the supine position, full-body immobilization is best achieved using the long spine board with the extremities securely fastened to the board.

Mechanisms that arouse suspicion of injury to the thoracolumbar spine should also prompt full-body spinal immobilization. These include penetrating and blunt injuries to the thorax, abdomen, pelvis, and spine. Full-body immobilization should be considered whenever the mechanism for spinal injury exists, even in the absence of signs and symptoms. In such cases, the possibility of occult injury is best ruled out through clinical or radiographic examination, or both, at the hospital.

The only contraindication to full-body immobilization of a patient whose mechanism of injury suggests spinal injury is *the existence of a greater threat*. The threat to a patient's life may exceed the threat of *possible* spinal injury under the following circumstances:

- *Hazards on the scene.* Problems with traffic control in the direct vicinity of the patient's location such that the patient or rescuers are likely to be injured. Such hazardous situations include leaks of fuel or other flammable substances, fire, hostile crowds, partially collapsed structures, or unstable vehicles.
- *Ongoing gunfire at the scene.* Gunfire is always considered an indication for rescue personnel to stay away from a scene.
- *Overwhelming casualties.* Rescuers may have to improvise in cases in which casualties exceed available resources. In such cases, proper spinal immobilization may merit a lower priority than usual.
- *Weather extremes.* Under conditions of extremely adverse weather, the urgency to move the victim may supersede the priority of normal treatment, including immobilization.
- *Patient noncompliance.* A competent rescuer can do much to make an immobilization device comfortable by means of padding and reassurance. If this fails, immobilization that is applied by force may cause more harm than no immobilization at all.

Equipment

Cervical Extrication Splints

There is a large variety of short spine boards (Fig. 47-12) and intermediate-stage extrication devices available for prehospital use. Generally, these devices are manufactured using rigid lightweight materials. They have a narrow board design that permits easy application in small automobiles or confined spaces, and are constructed with multiple openings along the edges to allow for a variety of strapping options. Although these devices have not been compared scientifically, some useful and important features of cervical extrication splints have been identified.

The device should not produce unnecessary movement or change the position of the head, neck, shoulders, or torso during application. In conjunction with a good cervical collar, a properly applied cervical extrication splint should effectively limit flexion, extension, lateral, and rotational motion of the head, neck, and torso. A cervical extrication splint should be comfortable, since it may be left on a patient for prolonged periods. Simplicity of design should allow rapid placement of the device by two rescuers in any situation, without causing movement of the head, neck, or torso.

The cervical extrication splint should be compact enough to allow its use in many types of extrication situations. Cost should be reasonable so that at least one device can be carried in every ambulance. Ideally, the device should also be translucent so that radiographs can be readily obtained in the emergency department (ED). Lastly, the cervical extrication splint should be designed to allow for repeated use and easy cleanup.

One commonly used device that meets all of these criteria is the KED (Fig. 47-13). The KED was developed by Rick Kendrick, an emergency medical technician (EMT)-fire-fighter from El Cajon, CA, in response to frustrated rescuers who experienced difficulty when removing victims from wrecked racecars. The device consists of two layers of nylon mesh impregnated with plastic and sewn over plywood slats to provide rigidity. It has a nylon loop behind the patient's head that is continuous with the pelvic support straps for additional strength. Part of its anterior thoracic panels can be folded backward to fit the obese, pregnant, or pediatric

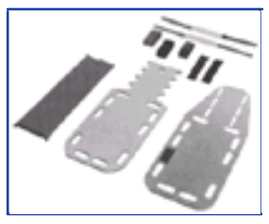

Figure 47-12 Rigid short boards. *(Courtesy of Ferno-Washington, Inc., Wilmington, OH.)*

Figure 47-13 The Kendrick extrication device (KED). Note the presence of a cervical collar, applied before the KED. *(Courtesy of MediXchoice, El Cajon, CA.)*

patient.[50] Properly applied, the KED is a snug-fitting, highly adaptable immobilizer that can be used under even the most adverse of circumstances.

Full-Body Spine Immobilizers

There are three basic classes of full-body spinal immobilizers, each with its own advantages and disadvantages. [27] It should be stressed that it is more important for a rescuer to achieve results (e.g., rapid, efficient, and complete spinal immobilization) than to be particular about using a specific piece of equipment.

Full-body spine boards (Backboards).

Backboards are made from wood or plastic composites, and can be either rectangular or tapered in shape (Fig. 47-14). Most rescuers prefer the tapered type because it takes up less horizontal room when angled into a narrow opening or doorway (such as that of an automobile). In addition, the slight narrowing of these boards on either end enhances the effectiveness of strapping.

Most backboards have strategically placed openings along the edges that can be used to secure head-stabilizing devices, strap the patient to the board, or lift. Many also feature runners, usually about 2.5 cm thick, on their undersides that serve both as stiffeners and as spacers. These raise the

Figure 47-14 The Bound Tree spine board. An example of a commercial plywood backboard. *(Courtesy of Bound Tree ALS Medical Products Corporation, Phoenix, AZ.)*

board slightly off the ground so that rescuers can get their fingers under the board during lifting. The runners, however, may make it more difficult to slide a patient onto the board.

Advantages of boards over full-body splints include their ease of storage, low cost, and extreme versatility. The backboard can be used to slide a victim out of an automobile or to protect a victim during removal of a windshield. Boards provide insulation against electrical hazards and can be used as improvised shelter in bad weather. Finally, they may be useful as ramps in muddy conditions.

Disadvantages of boards as immobilizers, although few, deserve mention. Board-like splints, as a class, are the least comfortable of all immobilizers. One prospective study demonstrated that standard spinal immobilization (hard backboard, rigid cervical collar, lateral immobilization device) of healthy volunteers was associated with a variety of symptoms, including headache, backache, and jaw pain.[11] In another study using EMT trainees, Cross and Bakerville found that the occiput, lower back, and sacrum are the three most common locations for pressure pain, and that pain in these areas is greater on a long backboard than with vacuum-type mattresses. [51] Pain in these areas may become severe as patients are frequently left immobilized on these boards for extended periods of time in the ED. [12] [52] [53] These findings may also have clinical implications, since pain generated by the application of a backboard can be difficult to separate from other sources of pain in the trauma patient, and may lead to unnecessary and costly radiographs.[4] Discomfort may be minimized by using padding at points of contact between a bony prominence and the board or the cervical collar. Alternatively, patient discomfort can be reduced, without compromising cervical spine immobilization, with the use of a pad (1.3 cm closed-cell foam padding) along the entire board. [54] This concept was reaffirmed by Hauswald and colleagues, who found that increasing the amount of padding on a backboard decreased the amount of ischemic pain caused by immobilization. [55]

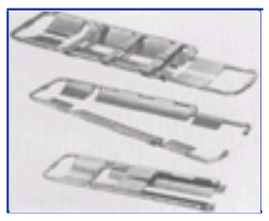

Figure 47-15 The Ferno-Washington model 65 orthopedic (scoop) stretcher. *(Courtesy of Ferno-Washington, Inc., Wilmington, OH.)*

Backboards have also been shown to produce higher sacral interface pressures and higher self-reported pain scale scores on the occiput, low back, and sacrum than a conforming support surface such as the vacuum-type splint.[51] [56] The duration and degree of contact pressure are considered important determinants of pressure necrosis in spinal cord injuries. [57] A further disadvantage of the backboard is its slippery surface, which makes it an unstable carrying device in rough terrain.

Scoop stretchers.

If a trauma victim has to be extricated from a tight location, a smooth backboard is probably the best immobilizer. If the victim is not in a tight location, the scoop stretcher is an ideal field immobilizer (Fig. 47-15). The scoop stretcher is designed to split into two or four pieces. It is comfortable, rigid, adaptable to patients of various lengths, and provides unobstructed radiographic transparency of the entire spine. If necessary, it can be almost instantly applied or removed without disturbing the position of the victim. The stretcher also provides good lateral stability due to the troughlike shape of its top surface, and it is stable enough to be used for carrying purposes. For optimal protection of a potential spinal injury, the spine should be completely immobilized (e.g., cervical collar, lateral supports, and secure strapping) and the scoop stretcher should be placed on a backboard before moving the patient. In addition, the scoop stretcher should be carefully reassembled to avoid trapping clothes, skin, or other objects between interlocking parts.

The scoop interferes slightly with the ischial section of a half-ring traction splint but works well with Sager-type devices. The scoop has no adverse effect on other immobilizers and does not interfere with cardiopulmonary resuscitation (CPR). The Ferno-Washington model 65 scoop (Ferno-Washington, Inc., Wilmington, OH) is the most widely used stretcher of this type. Newer devices such as the CombiCarrier (Hartwell Medical, Carlsbad, CA) and the Scoop EXL (Ferno-Washington, Inc., Wilmington, OH) offer lightweight polymer construction and additional spine support (Fig. 47-16 *A&B*).

Full-body splints.

Various devices take the concept of full-body immobilization one step further than the spine board. Los Angeles County Fire Department paramedic Larry Miller designed a narrow spine board shaped like the human body, with handles on both long edges and a system of harnesses to provide immobilization. The Miller body splint consists of a polyethylene shell injected with closed-cell foam that is radiographically translucent and provides buoyancy in water (Fig. 47-17). The full-body splint features a removable head harness, a thoracic harness, and pelvic as well as lower extremity belts. The space between its lower extremities facilitates wrapping with bandage material in the event of fractures. In addition, it is shaped so that it can easily fit into a basket-type rescue stretcher. Similar spine immobilization systems are now available for pediatric patients (e.g., Pedi-Pac [Ferno-Washington, Inc., Wilmington, OH]), making immobilization, extrication, and transport of traumatized children easier and safer for prehospital care providers.

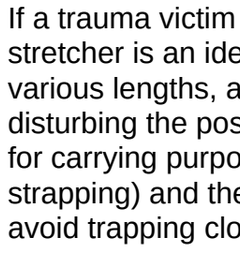

Figure 47-16 *A*, CombiCarrier. *(Courtesy of Hartwell Medical, Carlsbad, CA.) B*, Scoop EXL. *(Courtesy of Ferno-Washington, Inc., Wilmington, OH.)*

Figure 47-17 The Miller body splint. *(Courtesy of Life Support Products, Inc., Irvine, CA.)*

An important innovation in the area of spine immobilization in the United States has been the vacuum mattress splint (e.g., EVAC-U-Splint [Hartwell Medical, Carlsbad, CA] and Immobile-VAC [MDI, Gurnee, IL]) (see Fig. 47-11). It consists of a vinyl-coated polyester envelope filled with thousands of 1.1 mm diameter polyester foam spheres. A manual or electric vacuum pump is used to evacuate the interior to a pressure of about one-fourth atmosphere. This reduction in internal pressure causes the mattress to conform to the contours of the patient's body. Vacuum splints are more comfortable, quicker to apply, and allow less slippage on lateral tilting than wooden spine boards. [58] [59] The vacuum splint has also been shown to produce lower sacral interface pressure and lower mean pain scores than traditional hard

Figure 47-18 The HeadBed, a cervical immobilization device made of a water-resistant corrugated board. *(Courtesy of Laerdal Medical Corporation, Wappingers Falls, NY.)*

backboards,[51] [56] and may provide better immobilization in patients with known spinal cord injuries. [60] [61] It should also be pointed out, however, that vacuum splints are larger and more cumbersome than backboards, making ambulance storage more difficult. In addition, cleaning, durability, and cost remain important concerns. [51]

Lateral neck stabilizers.

Sandbags, which were used as lateral neck stabilizers for years, are no longer recommended for use in the field. Lighter objects such as blocks (10 cm × 10 cm × 15 cm) made of medium-density foam rubber are commonly used. Foam blocks are lightweight, inexpensive, and disposable, and do not slip on the backboard. More recently, cardboard devices that have the same advantages as foam blocks have been developed to provide lateral stabilization (Fig. 47-18).

Another commercial device is the Bashaw CID. It is a lateral neck stabilizer designed to quickly and easily fasten the patient's head to a scoop stretcher or spine board (Fig. 47-19). The CID is made of a Herculite nylon and

Figure 47-19 The Bashaw cervical immobilization device (CID). *(Courtesy of Bashaw Medical, Inc., Pensacola, FL.)*

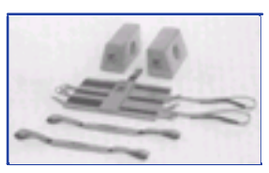

Figure 47-20 Ferno Universal Head Immobilizer. *(Courtesy of Ferno-Washington, Inc., Wilmington, OH.)*

polyethylene foam platform that is fastened to the stretcher, either by elastic belts or by nonelastic belts with buckled closures. Its pillows are then attached to the nylon platform by means of large Velcro interfaces. The Universal Head Immobilizer is a similar device manufactured by Ferno (Fig. 47-20).

Mosesso and colleagues compared six prehospital cervical immobilization devices and concluded that the devices were similar in their ability to immobilize the cervical spine, although the Bashaw CID was less effective than the other five devices in limiting rotational movement. [62]

Procedure

Despite the presence of a field cervical collar, manual in-line cervical stabilization should be continued until the patient is fully immobilized in either a cervical extrication splint or a full-body splint (e.g., a backboard or vacuum stretcher). The immobilization technique used will depend on the patient's position of origin.

Sitting Position

The extrication splint should be stored so that its straps are secured in their individual retainers, to reduce their likelihood of becoming entangled during application. At least two rescuers should be used to apply an extrication splint to a sitting patient.

When used, the device is opened, butterfly style, and gently slid behind the victim with a rocking motion. If necessary, the patient can be very carefully rocked forward a few degrees to facilitate placement of the splint.

Once behind the victim, the splint's pelvic support straps should be freed from their retainers and allowed to dangle at the patient's sides. Next, the lateral thoracic panels are brought around the chest just beneath the patient's shoulders. While grasping these panels, a rescuer *slides the splint upward until the top edges of the panels firmly engage the patient's axillae*.

Now the thoracic straps can be used to secure the splint, beginning with the middle strap, then the bottom strap, and finally the top strap. This procedure may need to be modified depending on injuries and preexisting conditions. For example, patients with pelvic fractures may not tolerate placement of the pelvic support and bottom straps, and the gravid abdomen of the pregnant patient may prevent placement of the middle strap. The straps should be fastened snugly but not so tightly as to interfere with respiration.

The pelvic support straps are fastened next. They can be slipped, one at a time, beneath the patient's lower extremities and *brought directly beneath the pelvis using a back-and-forth motion*. If the pelvic straps are not applied properly, they allow considerable slippage when the patient is lifted. The free end of each of these straps mates with a buckle located at the patient's hip on the outside of the splint. Once a strap is ready to be buckled, it can either be attached to the buckle on its own side or moved across the patient's lap and engaged with the opposite buckle. Most prehospital care providers prefer the latter method because it allows the patient's knees to remain together without discomfort to the patient. It is also a good idea to pad the groin area when placing the pelvic support straps as these may cause the patient considerable discomfort.

Next, the head is fastened. When using the KED, the head panels are wrapped snugly around the head and neck by one rescuer while another rescuer applies the diagonal head straps. It may be necessary to place padding behind the head to maintain a neutral position. The forehead can be used as a point of engagement for one strap, and the cervical collar itself can be used for the other.

Finally, all buckles should be tightened until the entire splint is firmly in place, while avoiding respiratory discomfort. The patient can now be moved (see Fig. 47-13). If the patient is to be lifted from a vehicle, the ambulance cot, with a spine board on it, should be brought as close as possible. While one rescuer supports the patient's knees, the other rescuer uses the handholds on the splint to lift the patient. The patient should be rotated and laid in a supine position onto a backboard.

The pelvic straps should then be loosened to allow the legs to be lowered onto the backboard. The legs can then be extended and secured to the backboard or left in the flexed position with a pillow placed under the knees for support.

Some type of lateral immobilizer should be applied for the head and neck, and the body should be belted into place on the board. Once the patient is on the board, the thoracic straps of the cervical extrication splint may need to be readjusted.

Recumbent Position

A patient who is found in a recumbent position should be placed in a supine position, if not already in one. If repositioning is necessary, the back should be examined in the process. Physical examination, spinal immobilization, airway management, and transport are easier to accomplish with the patient in the supine position.

Patients who are found supine do not require the use of a cervical extrication splint. They should, however, receive initial manual in-line cervical stabilization and an extrication collar. The patient should then be fastened to a full-body spinal immobilizer, such as a scoop stretcher, a backboard, or a full-body splint.

Scoop stretcher.

A patient who is in a supine position can be moved by means of a scoop stretcher. In the conscious patient, rescuers should explain that they are about to apply a

scoop-type stretcher, which may be cold to the touch, beneath the patient's body. An extrication collar is applied, and manual in-line cervical stabilization is maintained until the patient is completely secured to the stretcher. Another rescuer places the scoop on the ground next to the patient and opens the latches that regulate its length. The length should be adjusted

so that the scoop stretcher fits the full length of the patient's body. The latches that regulate the length of the device should then be engaged.

Next, the latches at each end should be released, allowing rescuers to separate the stretcher into two halves. Each half is then placed next to the patient. One rescuer then gently pushes half the stretcher under one side of the patient. In some cases it may be necessary to have another rescuer rock the patient to allow proper positioning. The same procedure takes place with the opposite half of the scoop until both halves are aligned beneath the patient. The latch at the head of the device should be engaged first. The lower end of the stretcher is then brought together, and the foot latch is engaged to complete the integrity of the stretcher. The patient's torso should be strapped into place and the head immobilized using a suitable lateral neck stabilizer. The patient can then be lifted onto another device (e.g., Stokes stretcher or backboard) for transport. After placement on another device, the scoop stretcher can be removed without disturbing the patient's position, if necessary.

Full-body spine boards (Backboards).

There are several ways of placing a patient onto a spine board. The precise technique used will depend on the space available and the position of the patient within that space.

For *lengthwise extrication*, as from an automobile seat, the patient can be slid, either feet first or head first, onto the backboard. It is important that the patient be moved as a unit during this process.

The end of the backboard should first be placed on the seat or doorsill of the automobile. One rescuer should then stabilize and maintain the backboard level at its opposite end, while other rescuers (at least two) lift and slide the body onto the board. Manual cervical in-line stabilization should be maintained throughout the procedure, and rescuers should avoid spinal compression or traction. Once the patient is completely on the board, the board can be slid out and placed on a waiting stretcher.

When space permits, *lateral extraction* is preferred. With the patient in the recumbent position, rescuers may logroll or slide the patient onto the board. As mentioned previously, extreme caution should be used with the logroll maneuver. [45] [46] The logroll maneuver requires the presence of at least three rescuers. One rescuer is positioned at the patient's head and applies manual in-line cervical stabilization. It will be this person's responsibility to oversee and direct body movement throughout the procedure. The backboard is then positioned next to the body. To minimize thoracolumbar movement, the patient's arms should be extended at the sides with the palms resting on the lateral thighs.[46] Some operators prefer to have the patient cross his/her arms across the thorax. If one arm is injured, the backboard should be placed against this side, so that the patient can be rolled onto the uninjured extremity. The other rescuers should be positioned on the side that the patient will be rolled *toward,* with one rescuer at the mid chest and the other at the legs. The rescuer at the chest should reach across the victim, taking hold of the shoulder and hips, while the other rescuer grasps the hips and lower legs. When everyone is ready, the rescuer at the head gives the command to roll the patient. The patient's back should be examined at this point. The backboard is then slid under the patient, and when everyone is ready, the rescuer at the head gives the command to lower the patient onto the board (Fig. 47-21). The patient should then be centered and securely strapped to the board. Alternatively,

Figure 47-21 Logroll maneuver. *(Courtesy of Albert Einstein Medical Center Emergency Medical Services Education, Philadelphia.)*

during lateral extraction, a recumbent patient can be slid sideways onto the spine board. This improvised technique also requires the presence of three or four rescuers, one of whom can maintain control of the patient's head and neck.

Various techniques can be used to secure a patient onto the backboard. Mazolewski and Manix evaluated the effectiveness of four different strapping techniques in reducing lateral motion of volunteers restrained on a backboard. [63] These investigators found that the addition of an abdominal strap significantly reduced lateral motion without compromising respiration. In addition, proper strap placement and firm contact between the straps and the patient are also important in limiting lateral motion.[64] Aside from this study, there is little scientific basis for the various strapping techniques, and in many cases the methods used are determined by local protocol.

After the body has been strapped to the board, the head can be secured. If necessary, padding should be placed under the occiput to maintain the head in neutral position. A lateral neck stabilizer (e.g., foam blocks or a Headbed device) is then applied and the head secured in place using tape or straps. Most taping techniques involve the use of one piece across the forehead and one piece across the cervical collar. Note that this method of securing a patient to a backboard is designed for horizontal lifting only.

Standing Position

The standing patient with a potential spine injury must be immobilized and placed in the supine position. One technique for placing these patients on a backboard that is quick, safe, and effective is presented here (Fig. 47-22).[65] The tallest rescuer should be positioned behind the patient to manually stabilize the head while a second rescuer applies an extrication collar. The first rescuer must maintain manual in-line cervical stabilization until the patient is completely secured to the board. The backboard should be centered behind the patient between the arms of the rescuer who is stabilizing the neck. Facing the patient, one rescuer on each side should each reach under the patient's arm and grab the backboard by a handhold at or above the patient's axillae. The patient's elbows are then brought closer to the body. If an additional rescuer is available, this rescuer should be positioned at the feet to prevent the board from sliding out, particularly on slippery surfaces.

Figure 47-22 Backboarding the standing patient. *A,* Step 1: Manual stabilization. *B,* Step 2: Apply a rigid collar. *C,* Step 3: Insert a long backboard. *D,* Step 4: Center the backboard. *E,* Step 5: Emergency medical technicians grasp the board, using a handle higher than the patient's armpit *(F)*. *G,* Step 6: Slowly lower the patient. *H,* Step 7: Fully immobilize the torso, then the head and neck. *(From Elling R, Politis J: Backboarding the standing patient. J Emerg Med Serv 12:9, 1987. Reproduced with permission.)*

The patient should be slowly tilted back by lowering the head of the backboard. The rescuer at the head should step back during this process while maintaining the patient's head and neck in neutral alignment. When the backboard is completely horizontal, the patient can be secured to the backboard in the normal fashion.

Complications

In general, complications are more likely to occur as a result of failure to immobilize spinal injuries before movement than from the technique of immobilization. When complications do arise, they may be related to improper choice or use of equipment. The variety of situations routinely encountered in the field call for a

common-sense approach to spinal immobilization.

Victims are generally belted in place on a spine board to prevent sliding during transport. If too few straps are used or if the straps are loosely applied, motion during transport can occur. Patients who are strapped too firmly in place may complain of extreme discomfort and even panic. Excessive strapping can interfere with respiratory function in both children and adults. [66] [67] [68] Totten and colleagues evaluated the effect of two spinal immobilization methods (wooden backboard and vacuum mattress) on eight respiratory function measurements in healthy volunteers. In comparing baselines for each method, six of the eight (FVC, FVC%, FEV_1, FEV_1 %, PEF, and FEF_{25} % to FEF_{75} %) measures showed respiratory

function to be restricted an average of 15%. [69] While this may not be a problem in healthy volunteers, the effects on patients with chest trauma or preexisting respiratory disease may be significant. [69]

Once strapped into place, an unresponsive patient who vomits should be protected from aspiration. The traditional means is to logroll the board and patient as a unit to the side. Although this procedure may be associated with some spinal movement, the airway takes higher priority.

Conclusion

The wide variety of circumstances in which a traumatized patient is likely to be involved mandates the need for many approaches to full-body spinal immobilization. The overall goal of rescuers is to immobilize the entire spine by fastening the victim to a board-like full-body immobilizer. After early application of an effective cervical *collar*, this process involves the following steps:

1. Application of a cervical *extrication splint* that serves to immobilize the cervical and thoracic spine. This sort of device is often used when the patient is first encountered in a confined environment, such as a wrecked automobile or bathtub.
2. Placement of the patient on a full-body spine board, a scoop stretcher, or a factory-designed full-body immobilizer.
3. Application of lateral immobilization support devices for the head.
4. Use of straps to fasten the patient securely to the backboard.
5. Placement of the immobilized patient into a rescue litter, such as a Stokes basket stretcher, if complex terrain must be traversed.

UPPER EXTREMITY SPLINTING

Fractures and dislocations of the upper extremity are extremely common injuries. Although upper extremity injuries are rarely life threatening, it is important to assess and manage these injuries properly. Splinting has constituted a fundamental component of orthopedic care since 2500 BC, when the Egyptians used palm fibers and reed bundles to immobilize injured extremities. [70] Appropriate splinting of a minor fracture or dislocation, in addition to decreasing pain, reduces the incidence of serious complications and the risk of permanent disability.

The rescuer must not let obvious injuries to the extremities be a distraction to the care of more life-threatening injuries. In some situations it may be necessary to rapidly secure the patient to a long backboard that supports and splints every bone and joint of the body in one efficient step. [44] Injuries to nerves or blood vessels are a frequent complication of upper extremity trauma. Circulation, motor function, and sensation distal to the injury must be assessed early and monitored closely.

The purpose of splinting is to prevent motion of broken or dislocated bone ends. Carefully applied splints decrease pain while minimizing further damage to muscles, nerves, and blood vessels. Splinting also reduces the risk of converting a closed injury to an open one. [71]

Indications and Contraindications

Indications for splinting an extremity are usually clear. Pain with or without deformity following trauma should arouse suspicion for underlying bone or joint injury. Other signs include swelling, discoloration, deformity, *crepitus*, or loss of neurovascular function. The absence of these findings does not always rule out an underlying fracture or dislocation. Whenever a musculoskeletal injury is suspected, a prophylactic splint should be applied and maintained. The old axiom, "if in doubt, splint," should be followed.

There are no contraindications to splinting suspected upper extremity fractures or dislocations. However, in the setting of multisystem trauma with life-threatening injuries, rapid transport may be more important than extremity splinting. Averting loss of life takes precedence over averting loss of limb.

Equipment

Various splints are currently available for immobilizing upper extremity injuries. They can be divided into two basic types: rigid and soft. [44]

Rigid splints are made of many different materials, including cardboard, plastic, aluminum, wire, and wood. These splints must be fastened to the injured extremity using tape, gauze, cravats, or Velcro straps. They are nonflexible and, when applied, immobilize the limb in a rigid fashion to maintain stability. Although some rigid splints are prepadded, many others require the use of some additional soft material to cushion the splint. When applying rigid splints, the fingertips should be left exposed so that distal circulation can be monitored.

Cardboard splints are excellent for long-bone fractures of the upper arm. They can be formed into any desired shape and are inexpensive. Plastic, aluminum, wire, and wood splints, although less malleable, are also good choices. An inexpensive aluminum splint that is popular in wilderness medical kits is the SAM splint (The Seaberg Company, Inc., South Beach, OR). The SAM splint is foam-padded for comfort, water-resistant, lightweight, radiolucent, and reusable. These characteristics also make it an ideal tool for disaster medicine. [72]

Vacuum splints (Fig. 47-23) are a special type of rigid splint in which the air is evacuated from a closed bag

Figure 47-23 Upper extremity vacuum splint. *(Courtesy of Hartwell Medical, Carlsbad, CA.)*

containing tiny foam beads. This compresses the contents into a solid mass, resulting in a rigid splint. Injuries can be encased and immobilized in the position in which they are found, thereby reducing patient discomfort. Flexibility of the splint before removal of air allows molding of the splint to conform to the patient's position. Vacuum splints are radiolucent and do not apply external pressure to the injured extremity.

Soft splints include air splints, pillows, slings, and swaths. Immobilization with pillows, slings, or swaths alone is usually inadequate because these splints allow significant flexibility and motion. Therefore, they are most effective when used with some form of a rigid device.

Air splints are soft splints that become rigid when inflated. Besides providing immobilization, they help compress underlying soft tissue to reduce local hemorrhage. These devices are sensitive to differences in atmospheric pressure and temperature. Therefore, their inflation must be constantly monitored to ensure that the underlying tissue is not subject to pressure-induced ischemia. One study suggests a maximum splint pressure of 15 mm Hg to reduce the risk of ischemia. [73] With long ambulance transports, the splint should be deflated for 5 minutes every 1.5 hours. [74] A significant disadvantage is that pulses can no longer be monitored once the air splint is in place. Air splints are designed to conform to a specific shape when inflated and should not be used on angulated fractures. In addition to being radiolucent, some types can be inflated with a refrigerant to provide concurrent cooling.

Pillow splints (Fig. 47-24) can be fashioned from any soft bulky material and are excellent choices for hand or wrist injuries. These splints are extremely comfortable and can be easily applied.

Slings and swaths are usually used in combination with a rigid or soft splint. When used alone, they can effectively immobilize injuries to the shoulder, clavicle, or humerus.

Procedures

To apply a splint properly to an injured extremity, several general rules must be followed. Communication is important to ensure that the patient understands what is being done at all times. If necessary, clothing should be removed to adequately visualize the injured extremity. Manual stabilization of the

Figure 47-24 Hand/wrist pillow splint.

TABLE 47-2 -- Management of Specific Upper Extremity Orthopedic Injuries

Site	Suggested Immobilization Techniques
Clavicle	Sling and swath
Shoulder	Sling and swath as it lies
Humerus	Cardboard or vacuum splint with sling and swath
Elbow	Cardboard or vacuum splint as it lies
Forearm	Cardboard, malleable metal, air, or vacuum splint with sling and swath
Wrist	Pillow, cardboard, malleable metal, or vacuum splint applied in position of presentation
Hand	Pillow, cardboard, or malleable metal splint in position of function
Finger	Tongue depressor or small malleable metal splint

fracture site helps limit unnecessary movement and prevent further injury. The neurovascular status (i.e., pulse, motor, and sensation) should be checked before and after the application of a splint. With a severely angulated extremity, traction not exceeding 10 lb of pressure may be applied to reduce the deformity. If resistance or pain is encountered, the extremity should be splinted in the position found. Open wounds should be covered with a sterile dressing before a splint is applied. The splint (Table 47-2) should be applied using the orthopedic principle of immobilizing the joint above and below a suspected fracture site. Cooling and elevation of the injured area may help reduce local swelling. Once the splint has been applied, the distal neurovascular status should be assessed frequently. Any deterioration requires immediate evaluation of the splint to determine if excess pressure is being applied.

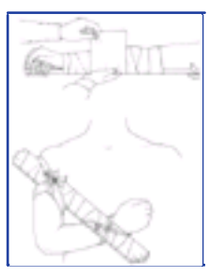

Figure 47-25 Examples of rigid splints.

Rigid Splints

To apply a rigid splint, an assistant should provide support and gentle traction above and below the injury. The splint is then applied on the side of the extremity away from any open wounds. The splint should be large enough to immobilize the joint above and below a fracture or the bone above and below a dislocation. The splint should be well padded to reduce the risk of pressure necrosis. The splint is then secured to the extremity using gauze or tape (Fig. 47-25).

Vacuum splints are applied in much the same manner as other rigid splints. While an assistant stabilizes the injured site and applies traction, the splint should be wrapped around the extremity and secured with the attached straps. The air is then evacuated from the splint by means of a hand pump until the splint becomes rigid.

Soft Splints

The application procedure for an air splint depends on whether the splint is equipped with a zipper. If the splint does not have a zipper, it must first be placed on the rescuer's arm until the bottom edge lies above the wrist. Next, the rescuer grasps the hand of the patient's injured extremity, while the free hand is used to provide support and gentle traction above the injury (Fig. 47-26*A*). An assistant should then slide the splint onto the patient's arm (see Fig. 47-26*B*). After making

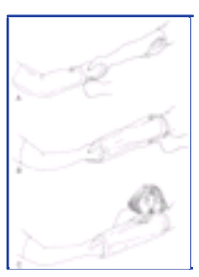

Figure 47-26 Application of an air splint. *A*, The rescuer supports the injured extremity with one hand and places the air splint on the other arm. *B*, An assistant slides the splint onto the patient's arm. *C*, The air splint is inflated until finger pressure makes a slight dent.

sure that the splint is not wrinkled, it should be inflated until finger pressure makes a slight dent (see Fig. 47-26*C*). Zippered air splints should be opened and placed around the injured area. The zipper should be closed and inflation accomplished as discussed previously. With air splints that completely enclose the hand, distal circulation must be assessed by checking capillary refill.

Pillow splints are applied by encasing the injury in the pillow and securing with tape, cravats, or gauze (see Fig. 47-24). If possible, the nailbeds should remain exposed to allow for assessment of circulation.

To apply a sling, an assistant should support the injured arm in a flexed position across the patient's chest. The long edge of the triangular bandage should then be placed lengthwise along the patient's side opposite the injury, with its tip over the uninjured shoulder (Fig. 47-27). The other tip is then brought over the injured shoulder to enclose the arm in the sling. The sling should be adjusted so that the arm rests comfortably with the hand higher than the elbow. The sling is then tied together at the side of the neck, and the knot is padded for patient comfort. The point of the sling at the elbow should be drawn around to the front and pinned. With the sling properly applied, the patient's arm rests comfortably against the chest with the fingertips exposed (Fig. 47-28).

To apply a swath, a cravat of sufficient length should be placed under the uninjured arm and over the injured arm at the level of the mid-humerus. This should then be fastened circumferentially around the thorax so that the injured extremity is secured snugly to the chest (Fig. 47-29). In adults, two

Figure 47-27 Stepwise application of a triangular bandage. 1, Place tip A over the uninjured shoulder. 2, Bring tip B over the injured shoulder to enclose the arm. 3, Draw tip C around the front and pin.

Figure 47-28 Completed triangular bandage.

cravats may have to be tied together in an end-to-end fashion to produce a swath of sufficient length.

Complications

Potential complications of upper extremity splinting include pressure necrosis, conversion of a closed injury into an open one, and loss of neurovascular function. With the use of air splints, there is the additional risk of pressure-induced tissue ischemia and compartment syndrome. [75]

Figure 47-29 Sling with swath.

Conclusion

Injuries to the upper extremities, although not life threatening, can have significant immediate or long-term effects. A high index of suspicion for underlying neurovascular injury should always be maintained. Neurovascular status must be checked before and after application of splints and monitored frequently throughout transport.

LOWER EXTREMITY SPLINTING

Many of the principles, techniques, and complications discussed with upper extremity splinting also apply to injuries of the lower extremity (Table 47-3); the pillow splint is one example (Fig. 47-30). In addition, the pneumatic anti-shock garment (PASG) can also be used as an effective splinting device for lower extremity injuries (see Pneumatic Anti-Shock

TABLE 47-3 -- Management of Specific Lower Extremity Orthopedic Injuries

Site	Suggested Immobilization Techniques
Pelvis	Pneumatic anti-shock garment (PASG), long backboard
Hip	Traction splint and long backboard, or secure injured leg to uninjured leg
	Long backboard with limb supported by pillows
Femur	Traction splint or PASG
Knee	Cardboard or vacuum splint in position found
Tibia/fibula	Cardboard, air, or vacuum splint
Ankle	Pillow or air splint
Foot	Pillow or air splint
Toe	Tape to adjacent toe

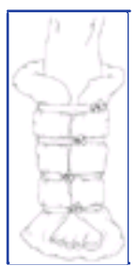

Figure 47-30 Lower leg pillow splint.

Garment later in this chapter). Prolonged use of the PASG as a splinting device is discouraged, however, since it has been associated with an increased incidence of compartment syndrome.[75] The following discussion is limited to lower extremity traction splinting.

The use of traction and countertraction for the alignment and reduction of fractures dates from the time of Hippocrates. [76] In the late 1800s, Sir Hugh Owen Thomas developed the first full-ring traction splint for the definitive management of fractured femurs. [76] Since the Thomas full-ring splint was not an emergency treatment device, it was later modified by his nephew, Sir Robert Jones, and other surgeons to a half-ring splint that made it easier to apply in the field. During World War I, the modified splint was credited with reducing the mortality rate associated with fractured femurs from 80% to 15%. [77] Since then, several additional modifications that carry the name of their inventors (e.g., Glenn Hare, Joseph Sager, Allen Klippel) have furthered the development of lower extremity traction splints.

In the setting of a fractured femur, muscle spasm and fragment overlap may cause the thigh to lose its cylindrical shape and adopt a more spherical appearance. [76] The resultant decreased tissue pressure and increased volume may allow 1 to 2 L of blood to accumulate at the fracture site. Traction splints are designed to restore the cylindrical shape of the thigh, which in turn increases tissue pressure and inhibits further hemorrhage.

Indications and Contraindications

Application of a lower extremity traction splint is indicated whenever a fractured femur is suspected. This should be clinically suspected when there is shortening, angulation, crepitus, swelling, or ecchymosis of the thigh with pain. The use of a traction splint will help align the fracture fragments, relieve pain, prevent damage to neurovascular structures, and reduce blood loss.

Lower extremity traction splints should not be used to immobilize fractures or dislocations of the pelvis, hip, knee, and foreleg. [44] It is controversial whether traction should be applied to an open femur fracture. Concern has been expressed that the use of traction may allow contaminated bone fragments to retract into the wound. Should traction produce bony retraction into the wound, this information must be relayed to the receiving clinician. A workable compromise is to use the splint to apply sufficient traction to achieve stabilization without retraction. Alternatively, a vacuum splint or the PASG can be used to immobilize the bony fragments in the position of presentation. In any case, stabilization of the fracture site to prevent further hemorrhage, neurovascular damage, or soft tissue injury should take precedence over the theoretical risk of increased contamination.

Equipment

Currently, there are a variety of commercially available lower extremity traction splints (e.g., Hare Traction Splint, FernoTrac, Sager-type devices, Kendrick Traction Device), each with its own unique method of application. Despite the differences among these splints, each is designed to align and immobilize a fractured femur. Except for Sager-type devices, most traction splints produce flexion at the hip joint because of their half-ring design. This flexion of up to 30° does not allow complete fracture alignment, unless the patient is in a reclining position about 30° from horizontal or the injured extremity is elevated to create the same angle.

Procedure

Application of the FernoTrac Traction Splint (Ferno-Washington, Wilmington, OH) and the Sager Emergency Traction Splint (Minto Research and Development, Redding, CA) are illustrated in Figure 47-31 and Figure 47-32 Figure 47-32 Figure 47-32 , respectively.

When possible, the splinting procedure should be explained to the patient. There is always pain associated with the application of a traction splint, but the patient should be reassured that the resultant stabilization of the fracture site will help reduce subsequent discomfort. The area of injury should be exposed and the distal neurovascular status assessed before application. Open fractures should be managed as discussed previously. If the injured leg is markedly deformed, an assistant should first straighten it using manual traction and maintain that position until a splint has been applied. The amount of traction necessary to straighten a badly deformed extremity will vary, but rarely exceeds 15 lb. Also, keep in mind that the initial application of traction is often quite painful, but usually subsides quickly as the injured leg is aligned and stabilized. If the patient strongly resists while traction is being applied, the emergency care provider should stop and splint the limb in the position it was found.

If the splint has an adjustable bar, the noninjured extremity can be used for length adjustment. The splint should extend approximately 25 to 30 cm beyond the heel. With the extremity slightly elevated, the *traction-splint* (e.g., FernoTrac Traction Splint) is placed under the injured leg and brought to rest firmly against the ischial

tuberosity. The heel stand should be unfolded and locked in place to support the end of the splint. This will ensure that the injured extremity will remain elevated once manual traction has been released. *Sager-type devices* (e.g., Sager Emergency Traction Splint) should be placed either against the symphysis pubis or positioned laterally against the greater trochanter of the femur. When the padded end of a Sager-type device is placed in the groin, one should ensure that the genitalia are carefully protected. Once the splint is properly positioned, the thigh strap should be firmly secured.

A harness is then placed around the ankle immediately above the medial and lateral malleoli and attached to the distal end of the traction splint. Traction is then applied gradually to approximately 10% of body weight or a maximum of 22 to 25 lb (10 to 12 kg). The goal is to stabilize the fracture and maintain proper limb alignment; the least amount of force needed to accomplish this should be used. [78] For *Sager-type devices*, the inner shaft of the splint must be extended until the desired amount of traction is achieved, whereas the *FernoTrac Traction Splint* uses a ratchet mechanism to apply traction to the ankle strap.

Before movement, supportive straps are applied around the thigh, knee, and distal leg to vertically stabilize the extremity. After application of the splint, the distal neurovascular status should be rechecked. The patient and splint should be firmly secured on a backboard. Extra care should be taken while moving the patient and when closing the ambulance door to avoid any unnecessary movement. If the splint extends beyond the dimensions of the backboard or stretcher, additional support for the splint may be needed (e.g., short spine board) to ensure the injured extremity remains elevated

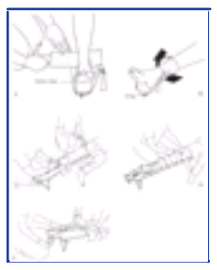

Figure 47-31 Application of the Ferno traction splint. *A–B*, Applying the ankle wrap. *C–E*, Applying the splint. *(Reproduced and modified with permission. Ferno-Washington, Inc., Wilmington, OH.)*

throughout transport. The loss of pulses with application of a traction splint requires that the position of straps and the amount of applied traction be reassessed immediately. Once the splint is applied, the position of the splint and the patient's neurovascular status should be rechecked following any patient movement. Removal of a traction splint should be done in reverse order of application.

Special Considerations

The traction splints in common use can be applied before application of the PASG. However, except for Sager-type devices, application of the PASG over a traction splint is awkward and associated with uneven pressure distribution. Sager-type devices are advantageous in that they can be

Figure 47-32a Application of the Sager emergency traction splint. *A–J*, Standard application.

applied *before* or *after* application of the PASG. In addition, the Sager splint can immobilize both legs using only one splint.

Unless the patient is found in an extremely cold environment, the shoe on the injured extremity should be removed before splinting to allow for assessment of neurovascular status. Furthermore, this facilitates monitoring of changes in color, temperature, or pulse distal to the injury en route to the hospital. The shoe can be removed by cutting the shoelace and pulling forward on the tongue of the shoe. The underlying sock can then be removed using scissors to prevent movement of the fracture site. Removal of the shoe and sock of the noninjured foot will allow comparison of the distal extremities.

The use of Sager-type devices is not recommended in the presence of an associated distal tibia-fibula or ankle fracture in the same extremity. In these circumstances, the amount of traction required to realign the fractured femur can distract the distal fracture site. Similarly, splints that incorporate part of the foot in their design may not be usable in the presence of an unstable foot fracture in the same extremity. A cardboard splint, pillow splint, or the PASG should be considered in these settings.

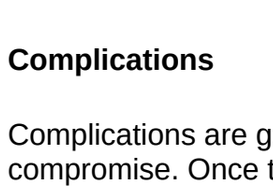

Figure 47-32b

Complications

Complications are generally the result of incorrect application and include ongoing hemorrhage, perineal injury, movement at the fracture site, or neurovascular compromise. Once the fracture site is stabilized, additional traction is unnecessary and potentially dangerous.

Conclusion

A properly applied traction splint will limit hemorrhage and movement associated with femur fractures. Careful monitoring of distal neurovascular status is imperative with the use of these splints.

PNEUMATIC ANTI-SHOCK GARMENT

The PASG, introduced earlier in this chapter, has been the source of much controversy since its introduction to medical practice. [79] In the late 1970s and 1980s, prehospital care providers and ED clinicians used the PASG routinely for patients with shock or hypotension from almost any cause. It was thought particularly useful in the management of blunt and penetrating trauma. However, the 1990s brought about a marked decline in use of the PASG as numerous studies failed to show improved outcome in patients with penetrating or blunt trauma. [80] [81] [82] [83] Today, the PASG is used primarily to stabilize fractured bone ends and control hemorrhage associated with fractures of the lower extremities and pelvis. [84]

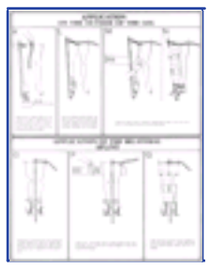

Figure 47-32c *K–N*, Application on the outside of the leg. *O–Q*, Application of the bilateral splint.

Figure 47-33 Assembled pneumatic anti-shock garment and inflation pump. *(Courtesy of the American College of Surgeons, Committee on Trauma.)*

The PASG resembles a pair of high-waisted inflatable trousers. It is constructed from two layers of an opaque, air-tight fabric sewn into three independently inflatable chambers (Fig. 47-33 and Fig. 47-34). It is colored and labeled to signal the internal and external sides.

This device has been called many names. The most common are military anti-shock trousers (MAST), medical anti-shock trousers, pneumatic anti-shock garment (PASG), and G-suit. Because some of these names (e.g., MAST) are also registered commercial trademarks and because pneumatic anti-shock garment is the term adopted by the American College of Surgeons for its Advanced Trauma Life Support course, [84] PASG is used in this chapter to refer to the devices that are currently available.

Background

Regardless of the name, the device has changed little in its design or application since 1903, when Crile created a

Figure 47-34 Pneumatic anti-shock garment applied to a patient and inflated. *(Courtesy of the American College of Surgeons, Committee on Trauma.)*

"pneumatic suit" from a double layer of rubber. [85] One or both legs or the abdominal compartment could be inflated separately with a bicycle pump. He designed the device to manipulate blood pressure during head and neck surgery in the sitting position. The pneumatic suit was subsequently applied to the management of the trauma patient. [86] Leaks in the material, however, inhibited widespread use of the device. The principle of external counterpressure lay dormant until its rediscovery by the military (allegedly at Crile's suggestion) during World War II. [87] Reincarnated as the G-suit, the garment was used to provide momentary compression of up to 100 mm Hg to counteract the cerebral and retinal ischemia (with resultant loss of consciousness or vision) that occurred during certain maneuvers in high-speed aircraft. Medical interest was renewed with investigations performed by Gardner and Dohn. [88] They used a homemade G-suit in patients who were likely to experience postural hypotension. Their device subsequently became commercially available and consisted of a double-layered rectangular blanket wrapped completely around the patient from xiphoid to ankles. For the next decade or more, external counterpressure was used only in the hospital setting and usually as a last resort in cases of uncontrollable postoperative hemorrhage (Table 47-4).

The military G-suit experience during the Vietnam War was the first recorded routine use of the external counterpressure principle in the preoperative stabilization of trauma patients. [89] The Army continued to develop the device until the current pants-like form was achieved. [90] The inventor of the modern-era trousers, B. H. Kaplan, became the first to adapt them to their next area of extensive use: civilian prehospital application by paramedics and emergency medical technicians. [91]

Mechanism of Action

The PASG's mechanism of action has been the object of many experimental studies. Interpretation and comparison of results from these studies require attention to several factors: species of subjects, volume status, design of garment, inflation pressures, methods of hemodynamic measurements, and position of subjects (supine or tilted). The increase in blood pressure that results from application of the PASG is due to at least three effects: enhanced venous return (autotransfusion),

TABLE 47-4 -- Early Uses of the Pneumatic Anti-shock Garment Device

Retroperitoneal bleeding from massive pelvic trauma
Postoperative bleeding from coagulopathy
Postural hypotension
Spontaneous rupture of the liver
Postoperative hemorrhage after:
Abdominal procedures
Nephrectomy

Prostatectomy
Renal biopsy
Tubal ligation
Hysterectomy
Leaking and ruptured abdominal aortic aneurysms
Lower extremity fractures
Placenta previa
Gastrointestinal bleeding
Ruptured ectopic pregnancy

TABLE 47-5 -- Pneumatic Anti-shock Garment: Proposed Mechanisms of Action

Blood pressure elevation
Increased total peripheral resistance
"Autotransfusion"
Control of bleeding
Direct pressure
Fracture stabilization
Effects on bleeding vessels
Decreased transmural pressure

increased total peripheral resistance, and reduced volume loss from control of hemorrhage (Table 47-5).

Enhanced Venous Return

The improvement in blood pressure seen during early PASG device studies was assumed to result from "autotransfusion" of blood from the venous system of the lower extremities and the splanchnic bed to circulation above the diaphragm. Increased central blood volume in humans after PASG application has been demonstrated by changes in thoracic radioactivity after ^{131}I-labeled albumin injection [92] and using a measurement of ^{99m}Tc-labeled albumin.[93] Attempts at quantification of "autotransfusion" in humans using nuclear scanning after radioactive red blood cell injection [94] have estimated that only 150 to 300 mL is actually autotransfused.

Increased Total Peripheral Resistance

Another explanation for the increase in blood pressure is an increase in total peripheral resistance after PASG application. This phenomenon has been well characterized in normovolemic and hypovolemic animal models.[95] [96] Using a hemorrhagic canine model, Niemann and colleagues demonstrated progressive increase in total peripheral resistance with inflation pressures of 40, 60, and 100 mm Hg. [96] Greater increases in total peripheral resistance occurred in the normovolemic group. Cardiac output increased only in the hemorrhagic hypovolemic group.

Control of Hemorrhage

The PASG can serve as a pressure dressing over otherwise uncontrollable internal and external bleeding sites. Acting as a pneumatic splint, the device prevents continued bleeding provoked by motion at fracture sites. This is particularly efficacious with lower extremity long-bone fractures and with retroperitoneal bleeding from pelvic fractures. In the management of hemorrhage associated with major pelvic fractures, the use of the PASG has been recommended as part of the initial management, followed by external fixation, surgery, or selective angiographic embolization. [97] [98] The application of the PASG in the prehospital setting is an efficacious means of initial tamponade and stabilization during the patient transport. One review of pelvic fractures has demonstrated radiographic realignment of an open-book, diastasis-type pelvic fracture after inflation of the PASG. [99] The prehospital application of the PASG, however, has the potential to mask an open-book pelvic fracture.[100] The PASG, combined with other nonsurgical therapy, has also been effective in controlling nontraumatic pelvic hemorrhage in obstetric and gynecologic patients.[101]

Indications

Today, the main indication for use of the PASG is fracture stabilization and hemorrhage control in patients with pelvic or

TABLE 47-6 -- Indications for Pneumatic Anti-shock Garment Application

Hypovolemic shock in selected trauma patients (*see text*)
Stabilization of fractures (pelvic and lower extremity)
"Prophylactic" uses (gastrointestinal bleeding, aortic aneurysm)
Compression of external bleeding
Non-traumatic pelvic hemorrhage

lower extremity fractures (Table 47-6). [84] [98] [99] [102] [103] [104] External pressure exerted by the PASG can improve pelvic alignment in open-book fractures, [95] [105] and provide prompt hemostasis in patients with severe hemorrhage from pelvic fractures. [106] It should be noted that the PASG could potentially cause a compartment syndrome.[105] [107] [108] [109] However, if used as a temporizing measure, the incidence appears to be very low, and most complications can be avoided.

PASG use has not been shown to improve outcome in victims of blunt or penetrating abdominal trauma (especially those with short transport times) and is no longer recommended in this setting.[80] [81] [83] Moreover, PASG use is contraindicated in patients with penetrating thoracic trauma as it has been shown to increase mortality.[82] [110]

While most prehospital providers have abandoned use of the PASG for trauma patients without pelvic or lower extremity fractures, there may be a few circumstances in which it is beneficial. A retrospective study of 142 patients who were severely hypotensive (systolic blood pressure [SBP] less than or equal to 50 mm Hg) as a result of both blunt and penetrating trauma found an improvement in survival with PASG use. [111] In another retrospective study comparing air and ground transport of patients with primarily blunt multi-organ trauma, Moylan and associates identified PASG use as one of several interventions in the air-transported group that were associated with improved survival.[112] Hence, PASG use may be considered for selected patients with blunt or penetrating abdominal trauma and severe hypotension who will require long transport times, especially when prehospital IV fluid therapy is not available.

"Prophylactic" application (with or without inflation) may be helpful in potentially hypovolemic or hypotensive patients. Examples are patients with gastrointestinal bleeding or those with leaking abdominal aortic aneurysms. Ali and associates examined outcomes in 18 patients with ruptured abdominal aortic aneurysms and SBPs

less than 80 mm Hg who were allocated to control and to PASG-treatment arms. The PASG group had a lower average SBP (54 mm Hg vs 76 mm Hg), but had increased survival to the operating room (88% vs 60%), and increased postoperative survival (75% vs 0%). Although the PASG-treatment group appeared more gravely ill, their outcomes were much improved.[113] In another study using a porcine model to study uncontrolled hemorrhage from the abdominal aorta, the group treated with the PASG survived longer than either the control group or the group treated with aggressive volumes of intravenous (IV) fluids. In addition, the use of the PASG was associated with a fourfold reduction in blood loss.[114] If definitive surgery is unavailable or delayed, PASG application may be indicated. In patients with leaking abdominal aortic aneurysms, one should maintain the SBP at approximately 100 mm Hg to avoid contributing to further hemorrhage.[114] [115]

Contraindications

Pulmonary edema, congestive heart failure, and penetrating thoracic trauma are the current absolute contraindications to the use of PASGs (Table 47-7). The increased venous return, decreased vital capacity, and elevation in pulmonary wedge pressure produced by these devices may aggravate preexisting pulmonary congestion.

External pressure applied below the diaphragm can increase the rate of blood loss from thoracic injuries. In a porcine model of thoracic aorta laceration, PASG inflation increased hemorrhage and mortality.[116] A retrospective review of 70 patients with penetrating cardiac wounds demonstrated a significantly lower survival rate in patients treated with PASG.[110] In addition, Mattox and colleagues, in a large clinical trial, found that PASG use coupled with aggressive fluid resuscitation in hypotensive patients with penetrating thoracic injury was deleterious.[82] There has also been a reported case of a traumatic diaphragmatic rupture that was exacerbated by the application of a PASG.[117] Therefore, PASG use in the setting of penetrating thoracic injury is contraindicated.

Other relative contraindications that have been proposed include pregnancy, evisceration of abdominal contents, a foreign body impaled in the abdomen, and lumbar spine injury. For these situations, the leg chambers may be inflated without additional risk, and the relative risks and benefits of inflation of the abdominal binder can then be assessed. Circumferential burns and other injuries that suggest lower extremity compartmental injury also represent significant relative contraindications. Application of external counterpressure elevates the limb compartment pressure and increases muscle ischemia.

Finally, advanced age represents a relative contraindication to PASG use. PASG application and inflation were associated with increased mean arterial pressure and total peripheral resistance in elderly patients without preexisting cardiac disease. Diminished left ventricular function, stroke volume, and cardiac output were observed and were greatest when higher inflation pressures were used and inflation times exceeded 10 to 15 minutes.[118] There are a number of situations in which PASG use has been discouraged by early authors (e.g., head injuries, cardiac tamponade, and tension pneumothorax). Although few data address these circumstances, some general review articles have discussed the judicious use of external counterpressure in these controversial settings.[119] [120] [121]

Equipment

PASGs are manufactured in two different sizes: adult and pediatric. Some features include x-ray transparency, perineal

TABLE 47-7 -- Proposed Contraindications to Pneumatic Anti-shock Garment Application

Absolute Contraindications	Relative Contraindications
Congestive heart failure	Pregnancy
Pulmonary edema	Evisceration
Penetrating thoracic injuries	Impaled foreign body
	Lower extremity compartmental injury
	Circumferential lower extremity burns
	Lumbar spine instability
	Advanced age

opening, and the ability to fit over most and under some (e.g., Sager-type) traction splints. The trousers come with or without a gauge for monitoring air pressure. Advantages of using models with pressure gauges include the ability to (1) assess PASG inflation pressure during deflation for removal; (2) monitor garment pressure during air transport of the patient (in unpressurized aircraft); (3) evaluate PASG pressure in settings of extreme temperatures; and (4) detect damaged air chambers, tubing, and the like.

Procedure

Before PASG application, it is useful to inspect the device and to establish the proper orientation (Fig. 47-35). The device is labeled and color-coded to identify the inside and outside. If there is potential for a cervical spine injury, proper cervical spine stabilization should be maintained throughout the garment application process. The patient can be logrolled onto the opened garment. Alternatively, with one person standing on either side and elevating the patient's legs, the garment can be slid beneath to the buttocks. Then the patient's hips are elevated slightly, and the upper border of the garment is placed at the costal margin. The medial portion on each leg binder is brought between the legs, and the Velcro fasteners are closed over each leg and over the abdomen.

After device application, the foot pump hoses are attached to the stopcocks, and the foot pump is used to inflate the two leg compartments and then the abdomen. The operator can accomplish inflation faster if the compartments are initially filled by blowing into them. After resistance to filling is met, the pump becomes more efficient at increasing trouser pressures.[122] The compartments should be inflated until the SBP reaches 100 mm Hg, the pop-off valves engage, or the Velcro straps begin to snap. The pressure-relief valves are set at 104 mm Hg to prevent overinflation. Because the PASG is most often used to treat patients with pelvic fractures, all three compartments are typically inflated. In the rare event that it is used for hypotension, Hanke and associates found no difference in redistribution of blood volume between simultaneous and sequential inflation of the leg and abdominal compartments.[123] Jennings and coworkers similarly found no hemodynamic benefit to simultaneous device inflation.[124] Therefore, it seems prudent when using the device for hypotension to inflate the leg compartments first and then check the blood pressure. If an adequate blood pressure is obtained, the abdominal compartment does not need inflation. If the blood pressure is inadequate, the abdominal compartment is inflated. Blood pressure and pulse should be closely monitored during the procedure, and inflation should be stopped if the SBP exceeds 100 mm Hg.

The PASG alone can passively splint lower extremity fractures. Of the commonly available traction devices, only the Sager traction splint can be applied after the trousers are in place and inflated. The Sager splint can be used either inside or outside the device to splint one or both legs at the same time. The Ferno traction splint, Hare traction splint, and the Thomas splint are significantly more awkward to use with the PASG inflated. These traction splints also present some risk of damage to the trousers themselves and result in uneven application of circumferential pressure. It should be noted that the application of traction in combination with external counterpressure exacerbates compartmental pressures in the lower extremities.[125]

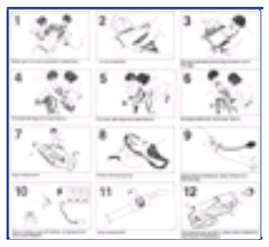

Figure 47-35 Pneumatic anti-shock garment application. *(Courtesy of the American College of Surgeons, Committee on Trauma.)*

Since PASGs are used primarily in patients with pelvic and lower extremity fractures, inflation is generally performed rapidly. Titrating the inflation pressure to achieve an SBP greater than 100 mm Hg is rarely necessary any longer. If the PASG is equipped with gauges, they should be checked periodically for accuracy. Most side effects and complications of these devices are proportional to the magnitude of PASG pressure and duration of pressure application. Most animal experiments and some clinical studies have suggested that hemorrhage control (presumably control of venous bleeding) in otherwise stable (often postoperative) patients is often accomplished with less than 40 mm Hg of inflation pressure.[126] The ultimate goal is to achieve fracture stabilization and hemorrhage control, or in selected hypotensive trauma patients, an SBP of approximately 100 mm Hg at the lowest inflation pressure possible.

For patients requiring pelvic stabilization, the PASG should only be deflated when a more definitive means of orthopedic stabilization becomes available. In other cases, deflation may be considered when the combination of PASG and other resuscitative measures (e.g., fluids, hemorrhage control) has resulted in restoration of satisfactory vital signs. The gradual deflation procedure outlined later in this section should be followed. The presence of any contraindications to continued use (e.g., congestive heart failure, renal disease, pulmonary disease) should also be considered. The presence of a coagulopathy may be a relative contraindication to removing the device.

If emergent surgical intervention is indicated, the operating room may be the best place to deflate the PASG. Once the anesthesiologist is prepared to monitor the patient and the surgeon is prepared to operate, the abdominal compartment can be slowly deflated. The leg compartments can remain inflated until any internal hemorrhage is controlled. In most situations, after adequate volume resuscitation and hemorrhage control, slow and systematic PASG deflation may be safely performed in the ED.

The PASG should be deflated gradually. Rapid deflation of the PASG in the hypovolemic patient can result in a

catastrophic drop in blood pressure. The hemodynamic changes seen during deflation result from a rapid reduction in left ventricular afterload followed by a decrease in preload.[127] This process may be exacerbated by the sudden release of lactic acid and other vasoactive chemicals pooled in the abdomen and the lower extremities.[128] Hence, deflation, particularly in the setting of prolonged PASG use, should be approached with caution.

The patient's hemodynamic status must be reassessed with each deflation attempt in order to identify any deterioration in the patient's condition. Deflation of the abdominal compartment should occur first. A small amount of air is released and the patient's blood pressure is then rechecked. If the SBP falls more than 5 mm Hg, deflation should be stopped immediately, and additional IV fluids should be infused until the SBP is restored. This process should be followed throughout the entire deflation procedure of the PASG. After the abdominal compartment is deflated, the same procedure should be followed for each individual leg compartment.

Complications and Disadvantages

A number of specific adverse effects have been noted in association with PASG use (Table 47-8). In addition, patients with penetrating abdominal and thoracic trauma whose management included PASG use in conjunction with vigorous prehospital fluid resuscitation reportedly had an increased mortality rate when compared with patients treated with fluid resuscitation alone in one urban prehospital setting. [80] [82] As noted previously, the use of PASG in patients with penetrating truncal injuries in the setting of rapid transport times or IV fluid therapy does not appear to be warranted.

Clinical experience has shown that the major life-threatening complication resulting from the use of the PASG in hypovolemic patients is sudden and severe hypotension resulting from precipitous removal of the device in the absence of adequate fluid resuscitation. [89] [129] Although ED

TABLE 47-8 -- Complications and Disadvantages of Pneumatic Anti-shock Garment Application

Hypotension after removal
Metabolic acidosis
Respiratory compromise
Decreased renal perfusion
Other (infrequent) complications
Pulmonary edema, congestive heart failure
Compartment syndromes
Increased wound bleeding
Urination, defecation, vomiting
Skin breakdown
Lumbar spine movement
Mechanical problems and disadvantages
Limitation of diagnostic and therapeutic procedures
Physical examination
Urinary catheterization
Peritoneal lavage
Vascular access
Environmental influences
Barometric pressure
Temperature

clinicians and emergency medical technicians are well aware of the problem, many consultants are not. The knowledgeable emergency clinician should prevent the consultants from making the potentially disastrous mistake of rapidly removing the PASG.

Abdominal binding invariably produces subjective effects on respiration; alert patients frequently complain of shortness of breath when the abdominal compartment is inflated. However, McCabe and colleagues found no changes in inspiratory and expiratory reserve volumes, maximum breathing capacity, or tidal volume in their volunteers.[130] Similarly, Batalden and coworkers found no pulmonary complications in 10 patients with external counterpressure garments who underwent positive-pressure ventilation for 24 to 48 hours. [131] However, Burdick and colleagues found atelectasis, pulmonary edema, or pneumonia in 14 of 28 similar patients.[132] Cogbill and coworkers demonstrated mild impairment of pulmonary function in healthy individuals and those with airflow obstruction when the PASG was inflated.[133] The impairment was restrictive rather than obstructive and was not clinically significant with inflation pressures less than 50 mm Hg. Some studies suggest

that impaired pulmonary function results from mechanical binding of the thorax and decreased excursion of the diaphragm. [130] [134] Newer designs of the device incorporate smaller abdominal compartments, which should not be applied at or above the costal margin, and have resulted in less restriction of vital capacity. [130] In general, investigators concerned about respiratory compromise have recommended careful attention to arterial blood gases combined with controlled positive-pressure ventilation in those patients requiring prolonged external counterpressure. [132] [135]

A number of case reports document some infrequent but serious complications with the application of the PASG.

Lower extremity compartment syndromes following use of PASG have been reviewed elsewhere.[136] Increased pressure within the limits of a fascial compartment results in impaired microcirculation. The compartment pressure under the PASG is dependent on the PASG inflation pressure, the mean arterial pressure, and the application of passive traction. [125] [137] During hypotension, with full PASG inflation, the compartment pressure may exceed the mean arterial pressure. [137] Many factors contribute to the development of a compartment syndrome, including prolonged shock, inflation pressure (and duration of inflation), reperfusion edema, and local tissue injury. [136] It is not completely understood which of the multiple factors are most significant in the compartment syndrome associated with PASG use. Some cases are related to a lower extremity fracture with prolonged application of PASG, [109] but other cases have occurred in the absence of lower extremity injury.[138] Improper deflation technique (i.e., leaving the abdominal compartment inflated after deflation of the extremity compartments) may facilitate the syndrome. [107] The overall frequency of this complication seems quite low. In their review of 1120 patients, Wayne and MacDonald reported no cases of compartment syndrome. [139] Mattox and associates found an incidence of anterior compartment syndrome in approximately 1% of their PASG patients and none of the patients without PASG. [81]

Skin breakdown at pressure points with prolonged use has prompted the suggestion to pad bony prominences.[131]

Diaphragmatic herniation has been reported following PASG inflation.[117] A sudden deterioration in blood pressure or respiratory status following PASG inflation should alert the clinician to this possibility. Using a porcine model of diaphragmatic injury, Ali and Qi demonstrated a significant increase in mortality rate after PASG as a result of cardiorespiratory deterioration caused by the increase in intra-abdominal pressure. [140] If significant herniation of abdominal contents into the thoracic space has occurred, deflation of the device may not fully resolve the clinical deterioration.

Metabolic acidosis may occur with prolonged use of the PASG. Close monitoring of the arterial blood gas and correction with IV fluids and bicarbonate as needed should correct the potential problem.

Environmental Influences on Device Pressures

With the current popularity and availability of helicopter ambulance transport, the role of Boyle's law (i.e., the volume of gas is inversely proportional to its pressure) must be reemphasized.[141] [142] As the helicopter rises, the air in the suit expands, increasing the trouser pressure. Decreasing altitude has the opposite effect. Similarly, as ambient temperature increases, trouser pressure rises, and vice versa. This is more of a concern with higher altitude fixed-wing airplane transports than with helicopter transports. Most helicopter transports occur at sufficiently low altitudes that the effects of altitude are not of concern, but when the "optimal" inflation pressure is determined at the scene of the injury, changes produced by movement into the controlled climate of the ambulance or hospital must be predicted and recognized.

An Alternative to the Pneumatic Anti-shock Garment for Stabilizing Pelvic Fractures

Based on some of the literature cited earlier, many prehospital and hospital emergency care providers have chosen to no longer have a PASG available. Thus, an alternative means of emergent pelvic stabilization is needed. A simple and cost-effective alternative is wrapping the pelvis with bed sheets (Fig. 47-36). In a recent study of hemodynamically compromised patients with a biomechanically unstable pelvic fracture, Biffl and associates demonstrated an immediate reduction of the pelvic volume by wrapping the pelvis with a sheet and binding the knees and ankles. [143] This simple maneuver can be performed quickly and easily in the ED, and is recommended for use by prehospital personnel to help stabilize patients for transport. [143]

Conclusion

The PASG is a useful adjunctive for temporary stabilization and hemorrhage control in patients with pelvic or lower fractures. When the garment is used, improvements in blood pressure, pulse, and pelvic fracture stabilization are often dramatic. If the PASG is not available, wrapping the pelvis with a sheet and binding the knees and ankles is a simple and inexpensive alternative. Although the PASG is not helpful in the management of urban trauma victims, it may be indicated

Figure 47-36 Stabilization of a pelvic fracture using a sheet.

for a select group of patients with blunt and penetrating abdominal trauma and severe hypotension who require long transport times, especially when prehospital IV fluid therapy is not available. It may also be helpful when surgery is unavailable or delayed in patients with a leaking abdominal aortic aneurysm.

HELMET REMOVAL

Although originally developed for protection of the head during combat, helmets are commonly worn by motorcyclists, athletes in many sports (e.g., football, hockey, lacrosse, motorsports), and individuals participating in a host of recreational activities (e.g., kayaking, rollerblading, skate-boarding). Hence, emergency care providers must be able to remove a helmet safely. The use of helmets has been shown to reduce the incidence and severity of head injuries associated with motorcycle crashes.[77] [144] [145] Most modern helmets consist of an inner layer of foam material covered by a hard plastic shell. Helmets may be modified with additional padding so that they conform tightly to the individual's head. Although earlier studies [77] [146] suggested that the use of motorcycle helmets might be associated with an increased incidence of cervical spine injury, this concern has not been substantiated. [145] [147] [148]

Helmet removal requires a careful, methodical approach to avoid compounding a suspected injury to the spinal cord. [149] Fluoroscopic studies have detected spinal motion even in the best of circumstances when removing hockey and football helmets. [150] [151] Prehospital care providers should understand that they can achieve proper cervical spine immobilization of an athlete wearing a well-fitted helmet and achieve adequate airway control by removing only the facemask. [152] A proper fitting helmet should hold

the head securely in a neutral position of alignment, minimizing any motion, provided that the athlete is wearing shoulder pads. Therefore, the first priority of field management is to stabilize the cervical spine and keep the helmet on, if possible.

Indications and Contraindications

When dealing with athletes and recreational helmet users, two schools of thought predominate with regard to helmet removal in the prehospital setting. One theory suggests that removal of helmets is rarely necessary, since the helmet itself provides for excellent spinal immobilization. The other philosophy recommends routine removal of the helmet so that the patient is completely exposed and the rescuer has full access to the head and neck for airway management, hemorrhage control, and cervical spine stabilization. This controversy prompted the formation of an Inter-Association Task Force for the "Appropriate Care of the Spine-Injured Athlete" in November 1999. The task force consensus guidelines recommend helmet removal only in the following situations [153] :

- If the helmet and chin strap fail to hold the head securely, such that immobilizing the helmet does not also adequately immobilize the head.
- If the helmet and chin strap design prevent adequate airway control, even after removal of the facemask.
- If the facemask cannot be removed after a reasonable amount of time.
- If the helmet prevents proper immobilization for transport.

If the helmet is not removed, cervical spine immobilization can usually be maintained with a properly fitting helmet by using tape, foam blocks, and a backboard. If it becomes necessary to remove the helmet, the shoulder pads (if present) should be removed at the same time to avoid hyperextension of the cervical spine. [150] [152] In the ED, stable patients typically undergo imaging studies of the cervical spine before helmet removal. However, a study by Davidson and coworkers found that the helmet and shoulder pads worn by football players interfere with cervical spine radiographic evaluation. They recommend incorporating procedures for controlled and cautious removal of equipment before initial radiographic evaluation. [154]

In contrast, motorcycle helmets often must be removed in the prehospital setting. [44] The motorcycle helmet with a full-face guard makes it very difficult to assess and manage the airway and to evaluate injuries to the head and neck. In addition, the helmet's large size and design may cause significant neck flexion if left in place when the patient is placed on a backboard.

The only absolute contraindication to helmet removal is neck pain or paresthesias associated with the procedure. Relative contraindications to helmet removal include unfamiliarity with the technique and lack of sufficient assistance. [147]

Procedure

In 1980, the American College of Surgeons endorsed the helmet removal technique discussed in this chapter. [155] Proper removal of a helmet requires at least two individuals along with manual stabilization of the cervical spine throughout the procedure. [147] [155] Although a one-person technique of helmet removal has been described, it is best for at least two rescuers (preferably three or four) to remove the helmet and shoulder pads, especially in the setting of an unconscious or uncooperative patient.

If applicable, the facemask should be removed and not retracted. Studies indicate more head and neck movement with this procedure than with complete facemask removal.[152] One rescuer establishes neutral position by placing his or her hands on each side of the helmet with the fingers on the victim's mandible (Fig. 47-37). The second rescuer then cuts or releases the chin strap and assumes manual in-line stabilization by placing one hand on the patient's mandible and the other hand on the occiput. The first rescuer then gently removes the helmet by grasping it at the base and expanding it laterally to clear the ears. Some helmets may need to be titled backward to allow the chin guard to clear the nose. The second rescuer must be prepared to assume the weight of the head as the helmet is removed. As soon as the helmet is removed, it is essential to place under the head a firm cushion or support thick enough to prevent hyperextension of the cervical spine. [149] The first rescuer then reassumes the responsibility for manual in-line stabilization and neutral positioning while the second rescuer applies a rigid extrication collar. Both rescuers should avoid in-line traction during helmet removal to reduce the risk of subluxation or distraction of an unstable cervical spine. [19]

In a volunteer study, Meyer and Daniel have shown limited motion of the cervical spine with this removal technique. [156] The addition of a folded sheet or jacket placed behind the patient's shoulders was also found to help limit any cervical motion associated with helmet removal. [156] In contrast, Aprahamian and colleagues demonstrated that manual helmet removal adversely affected a surgically created unstable cervical spine injury in a cadaver model. They recommended the use of a cast-cutter saw to divide the helmet into two pieces in the coronal plane and thereby facilitate removal. Following division of the helmet shell, the inner foam material may be cut with a scalpel or other sharp blade. Although this approach does provide an alternate method of removing the helmet, the intense vibrations produced during use of the cast cutter may exacerbate an underlying spinal injury. In addition, the technique would be slow and difficult with modern, well-fitting, high-quality helmets.[147] When necessary, removal of the full-face guard can be accomplished using a cast-cutter or other saw permitting rapid access to the airway without aggravating a possible cervical spine injury. This can then be followed by slow and careful removal of the entire helmet. [147] Donaldson and colleagues demonstrated in a cadaveric model that even in the best clinical setting there is a significant amount of motion during helmet and shoulder pad removal. [157] It is still unclear whether the results of any of these studies are applicable to patients with cervical spine injuries.

Complications

Underlying cervical spine injuries may be exacerbated by failure to adhere to proper helmet removal techniques. Although no controlled studies have demonstrated the safety of manual cervical stabilization, its efficacy during endotracheal intubation of the trauma patient suggests that minimal movement is likely during helmet removal.[158]

Conclusion

Airway management and cervical spine stabilization may be complicated by the presence of a helmet. If necessary,

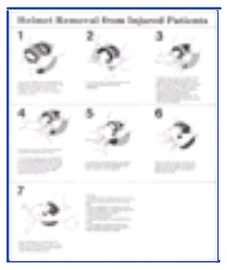

Figure 47-37 Helmet removal technique. *(Modified from McSwain N: Techniques of helmet removal from injured patients. Bull Am Coll Surg 65:20, 1980.)*

prehospital helmet removal can be accomplished in a safe and effective manner using a two-person technique. Current guidelines call for the removal of both the helmet and shoulder pads together in a controlled ED setting by qualified clinicians.

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Chapter 48 - Management of Amputations

William C. Dalsey
Jeffrey Luk

"The first person caring for an injured hand will probably determine the ultimate stage of its usefulness." [1] Consequently, rapid and appropriate emergency care of a patient with an amputated part is crucial to the salvage and preservation of function. This chapter discusses the acute care of amputated parts before they are replanted and specifically addresses the management of distal digit amputations and dermal "slice" wounds.

Amputation may be partial or complete. Injuries with interconnecting tissue between the distal and proximal portions, even if there is only a small piece of bridging skin, technically are considered incomplete (or partial) amputations. Complete amputations are replanted, whereas partial amputations are revascularized. This distinction is arbitrary; for emergency clinicians, treatment for both injuries is very similar. The prognosis and outcome of both types of amputations are similar, although partial amputations often have better venous and lymphatic drainage, and functional recovery may be more complete if there is less anatomic damage.

The peak incidence of traumatic amputations occurs between the ages of 20 and 40 years, [2] [3] and men predominate over women at a ratio of 4:1. Local crush injuries are the most common mechanism of injury, and sharp guillotine amputations are the least common. [4] [5] [6] Partial amputations occur as often as total amputations. [7] Power saws and lawn mowers are frequently the instruments of destruction. [7] Proximal amputations are less common than distal amputations.

The media have exaggerated somewhat the success of replantation and have often generated unrealistic expectations from the public. The technical limitations of successful repair of vessels that are less than 0.3 mm in diameter usually preclude replantation of digits distal to the distal interphalangeal joint. [8] Successful revascularization of amputated parts often ensures viability, but neurologic, osseous, and tendinous healing are critical for ultimate function. If there is incomplete neurologic recovery, limited range of motion, and intolerance to cold, the replanted part may have little functional value for the patient. Rehabilitation from replantation surgery may be prolonged, often requiring more than 1 year and repeated surgical procedures. The emergency clinician should be aware of the limitations of replantation surgery and should not encourage unrealistic expectations in injured patients or their families.

BACKGROUND

The possibility of restoring viability and function to traumatically severed parts has fascinated clinicians for centuries. Clinicians have attempted to replant parts with little more than a few sutures and secure bandaging and occasionally have had spectacular results. One of the earliest medical reports was by Fiorvanti, who in 1570 reported the successful replantation of a soldier's nose, which was severed by a saber, after first cleansing it with urine and then carefully bandaging it. [9] [10] In 1814, Balfour reported the successful replantation of a finger, which was severed by a hatchet, [11] using only meticulous alignment and secure bandaging.

The ability to consistently replant amputated parts awaited the development of modern microvascular surgical techniques. The first reported successful upper limb replantation was by Malt and McKhann in 1962. [12] Later that year, a successful replantation of a hand and arm was reported by Chen and Pao. [13] Developments in microsurgical techniques, advanced optics, and microsurgical instruments have created the ability to consistently replant amputated parts with a high degree of success. Since 1965, when Kleinert and Kasdan [14] reported the first successful microvascular anastomosis of a digital vessel, there have been several large series of replantations, with success rates ranging from 70% to 90%. [6] [7] [15] [16] [17] [18] [19] [20] [21] [22] [23] [24] [25] [26] [27] To the original pioneers in replant surgery, survival of the replanted tissue was the criterion for success, but with further technological and surgical refinements, today's surgeons emphasize functional recovery as well as viability. The replantation of a part that is painful or useless or that interferes with function is a disservice to the patient and is less desirable than early restoration of function without replantation.

INDICATIONS

Preservation of the amputated part is generally indicated when replantation or revascularization is a potential therapeutic method for care of the injured part. Revascularization and reanastomosis of partially and completely amputated parts should be provided when there is hope of preservation or restoration of function. Aesthetic considerations, patient avocations, and occasional religious or social customs may also influence the decision to proceed with surgery. [16] [28] [29] Ultimately, the decision must be reached by both the operating microsurgical team and the patient after a rational explanation of potential results and successes.

Indications for replantation of fingers and hands have been proposed and are generally accepted, although they should not be applied rigidly to all circumstances. Successful functional recovery is more likely in distal than in proximal extremity amputations and more likely in multidigit amputations, single-digit thumb amputations, or transmetacarpal amputations.[30] [31] Generally these are indications for replantation (Table 48-1). Single digits that are both proximal to the distal interphalangeal joint and distal to the flexor digitorum superficialis may be replanted successfully, with good functional recovery.

Successful replantations have been reported in patients from the ages of 1 to 84. [32] [33] There are no fixed age limits for replantation, although particularly good results have been reported in children owing to their regenerative capacity and adaptability to rehabilitation. [2] [34] [35] [36] [37] [38] The decision to replant is made on a case-by-case basis by the microsurgical team, who must weigh all the factors involved.

CONTRAINDICATIONS

There is no contraindication to managing the amputated part and stump as though replantation were going to occur, even

TABLE 48-1 -- Replantation of the Amputated Extremity

Indications
Young stable patient
Thumb
Multiple digits injured
Sharp wounds with little associated damage
Upper extremity (children)
Absolute Contraindications
Associated life threats
Severe crush injuries
Inability to withstand prolonged surgery
Relative Contraindications*
Single digit, unless thumb
Avulsion injury
Prolonged warm ischemia (=12 hr)
Gross contamination
Prior injury or surgery to part
Emotionally unstable patients
Lower extremity

*If the victim is a child or if there are multiple losses, salvage replantations are attempted, and the relative contraindications are ignored.

when replantation is considered unlikely by the emergency clinician. In addition, the requirements of the amputated part and stump can often be handled by ancillary personnel during resuscitation and transportation of the patient. However, the care of the entire patient must take precedence over that of the amputated part. Contraindications to replantation are listed in Table 48-1 and are discussed in the following sections. Note that even when replantation is contraindicated, tissue (skin, bone, tendon) from the amputated part may be useful in restoring function to other damaged parts. *Never discard amputated tissue until all possible uses of the severed parts are considered.* For example, even though an amputated fingertip is not suitable for replantation, the skin may be an ideal donor source for a skin graft to the stump.

GENERAL CONSIDERATIONS

Mechanism of Injury

The potential for successful replantation in terms of survival as well as useful function is directly related to the mechanism of injury. Guillotine injuries, which are sharp, are the least common, but have the best prognosis owing to the limited area of destruction. Crush injuries, which are the most common, produce more tissue injury and therefore have a poorer prognosis. The avulsion injury has the worst prognosis because a significant amount of vessel, nerve, tendon, and soft tissue injury invariably occurs. [2] [5] [6] [7] [39]

Ischemia Time

The time that an amputated part can survive before replantation has not been determined. After 6 hours, additional delay may decrease the success rate of revascularization and lead to diminished function. Skin, bone, tendons, and ligaments tolerate ischemia much better than do muscle and connective tissue. Therefore, as a general rule, the more proximal the amputation, the less ischemia time the amputated part can tolerate. Attempts to extend viability during ischemia have shown that the most important controllable factor is the *temperature* of the amputated part. Warm ischemia may be tolerated for 6 to 8 hours. [40] When the part is cooled properly to 4°C, 12 to 24 hours of ischemia may be tolerated with distal amputations. [2] [5] [7] [15] [16] [17] [19] [20] [21] [22] [23] [24] [25] [26] [27] [39] [41] [42] There is a report of a successful digital replantation after 33 hours of cold ischemia. [42] It has been postulated that hypothermia may limit metabolic demand, thereby preserving intracellular energy. [43] [44] [45] [46] Other investigations suggest that the effect is due to the retardation in development of an acidotic pH. [47] Hypothermia may also prevent the no-reflow phenomenon that can follow low-flow states. [48]

Delay in the replantation of proximal arm and leg amputations containing significant amounts of muscle tissue can lead to the buildup of toxic products. In such cases, when blood supply is restored, the absorbed toxins have been reported to cause respiratory failure, renal failure, cardiovascular collapse, and even death. [29] [41] [49] [50] [51] [52] [53] [54]

Perfusion techniques such as those used in organ transplants to extend anoxic time have not yet been developed but are under investigation. In the past, surgical teams used intraoperative perfusion as a technique to help cool the amputated part. The benefits of intraoperative perfusion with cold hypertonic solutions are currently being investigated. Perfusion should not be attempted by emergency clinicians. The risk of damage to vessels as well as the potential delay in care and in rapid transport of the patient and the amputated part override the theoretic benefits of emergency department (ED) cold perfusion at this time. [52] [55]

ASSESSMENT OF THE PATIENT

The initial care and treatment of the patient who has had a body part amputated are the same as those for any trauma patient. The clinician must not be distracted by the amputated extremity or the excitement of others from assessing and stabilizing the patient's airway, breathing, and circulation. Amputations are generally not life-threatening injuries, and other potentially more serious injuries must first be assessed and treated. Hemorrhage from amputated limbs is often limited by the retraction and spasm of severed vessels. Therefore, partial amputations may result in more serious hemorrhage than if the vessels were totally severed. Usually hemorrhage can be controlled adequately with direct pressure and elevation. Vascular clamps and hemostats have no role in the ED management of these injuries and may cause additional injury that may make replantation impossible. A proximally placed blood pressure cuff inflated 30 mm Hg above systolic pressure can be used for short periods of time (<30 minutes) to control severe bleeding, if necessary.

After the initial primary assessment and treatment and subsequent stabilization of the patient, care of the stump and amputated part can be initiated safely. In addition to the general history obtained from all trauma patients, particular attention should be focused on the exact mechanism of injury, the time and duration of injury, handedness, allergies, medications, illness, prior injury to the affected part, care of the stump and amputated part before arrival in the ED, occupation, avocations, and tetanus history.

Tetanus prophylaxis and broad-spectrum systemic antibiotic therapy (e.g., cephalosporins) should be initiated; however, the role for routine prophylactic antibiotics has not been proven. Analgesic medications may be necessary, especially

with crushing injuries, for managing patient discomfort. The dose of IV opioids should be titrated to the clinical condition. In fingertip amputations, digital or regional nerve blocks are ideal for pain relief but may make functional and neurological evaluation by a consultant impossible. Some clinicians recommend the early use of aspirin, low-molecular-weight dextran, or both for amputation patients, but such attempts to maintain small vessel perfusion are controversial and have not proved efficacious in preoperative treatment.

Amputation patients often experience denial, shock, disbelief, and feelings of hopelessness about their injury; some have even become suicidal. Patients should be treated with supportive and realistic reassurance, but unrealistic medical promises should be avoided. It is important that the emergency clinician (or other non-replantation specialist) does not speculate on the specifics of the ultimate prognosis.

Examination of the stump may be brief and should primarily be an assessment of the degree of damage to the surrounding tissue. Gross contamination can be removed by irrigation with normal saline. Local antiseptics, especially hydrogen peroxide or alcohol, should *not* be used because they may damage viable tissues. Similarly, tissues should not be manipulated, clamped, tagged, or further traumatized in any way. It is important to assess the degree of contamination, the level of injury, and any concomitant injury, such as crushing or multiple levels of injury or amputation. The amputated part should also be examined for the degree of tissue injury, contamination, and possible distal injuries. Radiographs of the amputated part and proximal stump to the level of at least one joint proximal to an extremity injury should be obtained. Preoperative laboratory studies and IV access in an uninjured extremity should also be initiated.

The neurological status of the stump or distal extremity in partial amputations should be assessed by pinprick and two-point discrimination tests. The presence of sweat may indicate autonomic-neurologic functioning. Vascular competence can be assessed by noting the color, temperature, capillary refill, and presence of pulses. An Allen test at the wrist or a modified Allen test at each digit may aid in determining the existence of an arterial injury (see Chapter 20). The neurovascular status should be carefully and clearly documented in the medical record. Motor and tendon function should be evaluated immediately. The regional microvascular resource center should be contacted as soon as possible to arrange transportation and to provide adequate time for mobilization of the replantation team.

CARE OF THE STUMP AND AMPUTATED PART

The stump can be evaluated, and primary care can be rendered during the secondary assessment of the trauma victim (Table 48-2). If replantation is proposed, the goals of initial care include control of hemorrhage and prevention of further injury or contamination. All jewelry should be removed. The stump should be irrigated with normal saline to remove gross contamination. Debridement and dissection should be done by a specialist. Do not clamp arterial bleeders. The stump wound should then be covered with a *saline-moistened* sterile dressing to prevent further contamination and to limit damage from desiccation. The stump should be splinted for protection and for the prevention of further injury from concomitant

TABLE 48-2 -- Axioms for Care of Amputations

Do's	Dont's
Splint and elevate	Apply dry ice or freeze tissue
Apply pressure dressing	Place tags on tissue
Protect from further trauma or injury	Place sutures in tissue
	Sever skin bridges
Protect from further contamination	Initiate perfusion of amputated part
Provide analgesia	Place tissue in formalin or water
Supply tetanus prophylaxis and antibiotic therapy	
Obtain radiographs	

fractures or compromise of blood flow owing to change in position. Splinting and elevation may reduce the extent of edema and help control bleeding.

Care of the amputated part follows the same general guidelines as that for the stump. Gross contamination can be eliminated by irrigation with saline. All jewelry should be removed. The amputated part should be handled minimally to prevent further damage and should be wrapped in a salinemoistened sterile dressing. *Direct prolonged immersion in saline or hypotonic fluids should be avoided* because it may cause severe maceration of tissue and may make replantation more difficult technically. The amputated part should be cooled as soon as possible. The ideal temperature is 4°C. Care must be taken to prevent the freezing of tissues. Amputated parts *should not be placed directly on ice* because tissue that is in direct contact with the ice may freeze. Currently, the recommended method for cooling amputated parts is to place the part, which is wrapped in saline-moistened gauze, in a watertight plastic bag and immerse the bag in a container of ice water (Fig. 48-1). A guideline is to use half water and half ice; excessive ice should be avoided. Cooling coils and refrigeration devices have occasionally been used but are generally not available and offer no significant advantages. The tissue containers should be labeled with the patient's name, the amputated part contained within, the time of the original injury, and the time that cooling began.

Treatment for *partial amputations* with vascular compromise is the same as that just described. Clean the wound with normal saline irrigation. Place a saline-moistened sponge on the open tissue, and wrap the injury in a sterile dressing, incorporating a splint to protect it from further injury. Ice packs or commercial cold packs should be applied over the dressing to cool the devascularized area (Fig. 48-2).

SPECIAL CONSIDERATIONS

Hand Function

Hand function is often determined in part by pinch and grasp functions. If the index finger is removed, the pinching function of the index finger is adequately provided by the middle finger. Power in grasping and gripping is mainly considered an ulnar function of the fourth and fifth digits. An effective grip that provides the ability to hold a variety of objects is a central function of the ring and middle fingers. In addition to its function in pinching, the thumb is the major opposing

Figure 48-1 Evaluate the patient's condition to ensure that resuscitation is not necessary before transfer. *A,* The wound should be rinsed with saline solution. *Do not scrub or apply antiseptic solution to the wound.* Apply saline moistened sterile dressing, wrap in Kling or Kerlix for pressure, and elevate. *B,* The amputated part should be rinsed with saline. *Do not scrub or apply antiseptic solution to the amputated part.* Wrap it in moist sterile gauze or a towel, depending on its size, and place it in a plastic bag or plastic container. *Do not place the amputated part directly in saline. C,* The part is then put in a container, preferably Styrofoam, and cooled by separate plastic bags containing ice or in a container of ice water. Do not pack the bagged, injured part in ice, but it can be immersed in half water, half ice. *(From Hand Trauma: Emergency Care. Baltimore, MD, Emergency Services.)*

force for successful grip and grasp. The thumb is the most important digit for adequate hand function, and its loss results in 40 to 50% disability. Such disability requires aggressive attempts to replant amputated thumbs. If this is impossible or unsuccessful, secondary alternatives are pollicization of other digits or toe transfers.[56] [57] [58] [59] [60]

Lower Extremity Amputations

There are few reports of successful replantation of amputated parts of the lower extremity. [61] [62] Indications for replantation of amputated parts of the lower extremity are different from those for replantation of amputated parts of the upper extremity. The goal of all replantation is restored function. If this

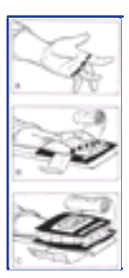

Figure 48-2 For a *partial amputation*, rinse with saline (*A*); then place part(s) in a functional position, apply a saline moistened sterile dressing, and splint and elevate *(B)*. Apply coolant bags to the *outside* of the dressing *(C)*. *Do not scrub or apply antiseptic solution to the wound.* Control any bleeding with pressure. If a tourniquet is necessary, place it close to the amputation site. *(From Hand Trauma: Emergency Care. Baltimore, MD, Emergency Services.)*

cannot be achieved, a patient is substantially better off with a prosthesis.

The lower extremity is primarily used for weight bearing and allows the individual to ambulate. Lower limb prostheses, especially those used below the knee, are well tolerated and functional. Prostheses provide a secure stance and permit locomotion. Lower extremity replantation generally requires skeletal shortening, and distal nerve regeneration is often imperfect. Both deficits may produce dysfunction. A patient with a replanted lower extremity with significant shortening and without sensation would function better with a prosthesis. This is not necessarily true of someone with an upper extremity replant. For these reasons, lower limbs are not generally replanted except under ideal circumstances, usually in children, although there have been documented cases of successful lower extremity replantation in adults. [61] [62] The final decision regarding replantation should be left to the replantation team.

Transplants of Upper Extremities

Advances in controlling tissue rejection have led to attempts at transplanting cadaveric hands in patients with complete amputations. This has been attempted in several patients with encouraging results. Many of the transplants have been

temporarily viable and those that have not been rejected have demonstrated both enervation and functional recovery. Patients must take immunosuppressive medications to prevent transplant rejection. These medications appear to facilitate reenervation and are being investigated as possible adjuncts for replantations.

Fingertip Amputations and Dermal "Slice" Wounds

Proper treatment of distal fingertip injuries is controversial. Fingertip amputations often heal by normal wound contracture, but occasionally this practice may result in the loss of functional ability to palpate. The basic goals of treatment are to provide tissue coverage, an acceptable cosmetic result, and an early functional recovery. In distal amputations in which the wound area is less than 10 mm^2 , this is not a problem (Fig. 48-3 (Figure Not Available)). Larger dorsal wounds also heal well by secondary intention. The challenging problem of fingertip injuries occurs when loss of skin and soft tissue from the fingerpad is significant. Volar skin is unique in its combination of toughness and sensitivity. Wounds with significant volar tissue loss frequently require additional treatment. Children, with their regenerative capacity, often progress very well when significant volar wounds are allowed to heal primarily. For older people and for amputations that involve a more significant amount of the distal digit, a wide variety of techniques for managing the injured fingertip have been advocated, including partial-thickness skin grafts; full-thickness skin grafts;

Figure 48-3 (Figure Not Available) Clinical classification of fingertip injuries and treatments for each type. *(From Newmeyer WL: Managing fingertip injuries on an outpatient basis. J Musculoskeletal Med 2:17, 1985.)*

V-Y, Kutler, Kleinert, and island advancement flaps; and various local and distal flap coverage techniques. These procedures are designed to preserve length and to provide soft tissue coverage of exposed bone and sensation to the fingerpad. Each of these procedures has its own indications, complications, and limitations. [63] [64] [65] [66] [67] [68] Discussion of these procedures is beyond the scope of this chapter. Most of these techniques are best performed by a specialist in the operating room as primary procedures under ideal circumstances or as delayed procedures when necessary.

In most complete fingertip amputations distal to the distal interphalangeal joint, the emergency clinician can provide adequate care initially with conservative wound management. Although thinking has changed significantly over the years, many hand surgeons still advise skin grafting to shorten the time for wound healing.

Although complete transverse amputations could be handled conservatively, wound healing may take several weeks, and these patients may benefit from operative treatments. Patients with complete transections should be referred for consultation to coordinate their initial care and subsequent follow-up. Regardless of the type of injury, definitive care is usually provided on a delayed basis, and ED intervention is conservative with subsequent consultation being considered standard.

Incomplete transections and small distal amputations without significant soft tissue loss may heal well with conservative therapy started by the emergency clinician. Nonoperative treatment in selected patients provides excellent functional and cosmetic results, minimizes recovery time, and

has few complications.[63] [66] [67] [69] [70] [71] [72] Children have excellent regenerative capacity and also respond extremely well to conservative treatment. Necrotic or grossly contaminated tissue should be debrided, and the wound should be irrigated thoroughly. If bone is left exposed without soft tissue coverage, the patient will need an operative procedure; alternatively, the bone may be rongeured (shortened) to allow soft tissue coverage and primary healing with better functional recovery. The nailbed tissues should be preserved, because the presence of a nail affects the cosmetic result. After cleansing and cautious debridement, an occlusive dressing is placed directly over the wound. Tetanus prophylaxis, wound dressings, and bandages, along with placement of a protective splint, complete the initial management of these injuries. Amputations that involve the distal phalanx are frequently treated as contaminated open fractures. IV antibiotics are often given initially, followed by a course of oral antibiotics, but the value of this intervention is unproved. [73] Wounds managed conservatively must have serial dressing changes and cleansing. Soaking wounds, cleansing, and replacement of dressings help provide superficial debridement, which may aid healing and minimize the chance of secondary infection. Wound contraction and healing usually result in acceptable cosmetic and functional recovery in 2 to 3 weeks. Patients should have appropriate follow-up to ensure adequate healing and recovery.

Partial fingertip amputations distal to the distal interphalangeal joint can also be managed successfully by the emergency clinician. These wounds are treated in a manner similar to that for complete amputations. However, when the amputation has substantial undamaged tissue connecting the fingertip, careful alignment and stabilization are provided by sutures or bandaging and protective splinting. Partially amputated fingertips, especially in children, may occasionally survive and regain vascularization and sensation. If the distal tissue becomes ischemic and necrotic, the amputation becomes complete.

Injury to the nailbed requires special attention to ensure proper alignment. If the nailbed tissues are not aligned properly, permanently disfigured nails may result. Removal of the nail helps provide adequate visualization for the placement of sutures and also minimizes the risk of wound contamination.

Dermal "slice" wounds (see type 1 in Fig. 48-3 (Figure Not Available)) are managed by gentle wound cleansing and application of an antibiotic ointment and a nonadherent dressing, followed by a pressure dressing (e.g., tube gauze). At a dressing change 48 to 72 hours after the initial treatment, the patient can be instructed on daily changes of nonadherent dressings for 10 to 14 days until functional epithelialization of the wound occurs. A protective finger splint or guard also minimizes the risk of further injury and pain from trauma to the sensitive wound area. Protection allows an earlier return to function and employment. Wounds larger than 10 mm^2 and those with deep loss of digit pulp tissue may be candidates for skin grafting.

Penis, Ear, and Nose Amputations

Replantation of the penis, ear, and nose generally results in better function and cosmesis than a prosthesis or reconstructive surgery. The amputated parts and wounds should be handled in the same manner as digital replantations.

Penile amputations are an uncommon problem. Most cases result from self-inflicted trauma in patients who are severely psychologically disturbed. Successful replantation has been reported using microsurgical techniques. Preservation or reconstruction of the urethra to maintain a competent urinary stream is critical for success.[74] [75] Ears and noses frequently are partially amputated and occasionally are totally amputated. Whenever possible, these body parts should be replanted unless they are severely traumatized and there is gross contamination. These wounds frequently heal well, and patients with such wounds have a high tissue survival rate and a low incidence of total necrosis. Replantation of these parts requires good suture technique and careful placement but does not necessarily require skill in microsurgical techniques. [75] [76] [77]

COMPLICATIONS

The care of amputated parts should not lead to avoidable complications if the aforementioned principles are followed. Improper management of the parts or stump with subsequent additional injury of the tissue from overzealous hemostasis or cleansing should be avoided. Furthermore, desiccation, maceration, or freezing of tissue from improper storage should not occur. The clinician must consider expediting the preoperative work-up of the patient and immediate notification of the replantation team as crucial factors in the patient's care.

Despite optimal initial care, replantation itself may be associated with acute or long-term complications. There is the usual risk of anesthesia and protracted surgery. Postoperative complications include vascular thrombosis, hemorrhage, infection, and reaction to accumulated toxins. It is not unusual for second and third emergency operations to be required to reestablish adequate blood flow. Patients are often placed on anticoagulants, which create an additional risk. Toxins accumulate in ischemic amputated parts, despite cooling. The amount of toxin is directly proportional to the amount of muscle mass and the duration of ischemia. Reports of significant pulmonary failure, electrolyte disturbance, and even death have been reported in replantation efforts.

Later complications include a significant percentage (60%) of patients with cold intolerance, limited function, anesthesia, pain, paresthesias, malunions, and nonunions. Repeated operative procedures may be required to obtain a functionally useful result. To minimize the morbidity from amputations, proper initial care is essential and may be the most important determining factor in the patient's eventual outcome.

COMMENTS

The emergency clinician will frequently encounter distal fingertip injuries that patients often expect will be replanted. These injuries heal well with conservative management and frequently the greatest clinical challenge is to reassure the patient or parents and convince them that replantation is not indicated and will not result in a better outcome. When a distal amputation has exposed bone, shortening can be easily accomplished with a rongeur so that healing will result in a better functional outcome. Bleeding from complete amputations is usually not life-threatening but partial amputations that don't allow retraction and spasm of blood vessels can bleed profusely. In such cases a proximally placed blood

pressure cuff inflated to 30 mm above the systolic blood pressure has been invaluable in initially controlling hemorrhage. Early notification of the transplant team is important to allow for timely referral and to ensure optimal outcomes. Consultation to determine the possibility of replantation also helps to communicate realistic expectations to the patient. Frequently, other ED personnel care for the amputated tissue. The emergency clinician should ensure that the tissue is appropriately cared for and not placed directly on ice, which might freeze tissue, or in saline, which will result in macerated tissue.

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Chapter 49 - Extensor and Flexor Tendon Injuries in the Hand, Wrist, and Foot

Peter E. Sokolove

EXTENSOR TENDONS

Extensor tendons are quite superficial, covered only by skin and a thin layer of fascia, and are thus highly susceptible to injury. These injuries may result from lacerations, bites, or burns, but they also may be caused by closed injury with even seemingly superficial lacerations. While some extensor tendon injuries must be managed by a hand surgeon, others may be treated in the emergency department (ED). Extensor tendon injuries are not benign and must be managed with care. The emergency clinician must understand the anatomy,

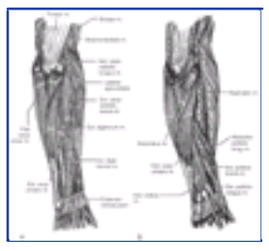

Figure 49-1 Extensor muscles and tendons of the RIGHT forearm and wrist. *A,* superficial layer. *B,* deep layer *(Modified from Pansky B: Upper extremity. In Review of Gross Anatomy, 4th ed. New York, Macmillan Publishing, 1979, pp 219 and 243.)*

principles of treatment, repair technique, and postrepair care of these injuries to ensure the best possible patient outcome.

FUNCTIONAL ANATOMY

There are 12 extrinsic extensors of the wrist and digits, all of which are innervated by the radial nerve. The muscles that give rise to these tendons originate in the forearm and elbow (Fig. 49-1). The extrinsic extensor tendons reach the hand and digits by passing through a fibro-osseous tendon sheath (retinaculum) located at the dorsal wrist. This synovium-lined sheath provides for smooth gliding of the tendons and prevents bowstringing when the wrist is extended. [19] The dorsal retinaculum contains six compartments or subdivisions (Fig. 49-2). These compartments are numbered from the radial to the ulnar side of the wrist.

The first compartment contains two tendons, abductor pollicis longus (APL) and extensor pollicis brevis (EPB). The APL tendon, the most radial of the extensor tendons, inserts on the base of the first metacarpal. It can be palpated just distal to the radial tubercle. The APL tendon causes thumb abduction and extension and some radial wrist deviation. The EPB travels with the APL through the first compartment but inserts at the base of the proximal phalanx of the thumb. The EPB tendon can be palpated over the dorsum of the first

Figure 49-2 *A,* The extensor mechanism at the wrist and dorsum of the right hand. The six extensor compartments at the wrist contain *(1)* the abductor pollicis longus (APL) and extensor pollicis brevis (EPB); *(2)* the extensor carpi radialis longus (ECRL) and brevis (ECRB); *(3)* the extensor pollicis longus (EPL); *(4)* the extensor digitorum communis (EDC) II–V and extensor indicis proprius (EIP); *(5)* the extensor digiti quinti (EDQ); and *(6)* the extensor carpi ulnaris (ECU). An important anatomic detail is the presence of a synovial sheath around each tendon unit within each fibro-osseous canal. *(From Thomas JS, Peimer CA: Extensor tendon injuries: Acute repair and late reconstruction. In Chapman MW [ed]: Operative Orthopaedics, 3rd ed. Philadelphia, JB Lippincott, 2001, p 1497.) B,* Cadaver model demonstrating the dorsal retinaculum of the left wrist with a probe passing through the fourth dorsal compartment. Note that the EDQ is also called the extensor digiti minimi (EDM) by some authors.

metacarpal when the thumb is extended against resistance. Both tendons can be tested by having the patient spread the fingers apart against resistance.

The second compartment also contains two tendons: the extensor carpi radialis brevis (ECRB) and the extensor carpi radialis longus (ECRL). These two tendons arise from the lateral epicondyle of the elbow. The ECRL inserts on the base of the second metacarpal, while the ECRB inserts on the base of the third metacarpal. Both tendons are powerful wrist extensors, and the ECRL also allows some radial wrist deviation. Wrist extension plays an especially important role in the mechanics of the hand, because hand grip strength is maximal only when the wrist is extended.

The third compartment contains only one extensor tendon: the extensor pollicis longus (EPL). This tendon crosses over the ECRB and ECRL and travels along the dorsum of the

thumb to insert on the distal phalanx. The EPL forms the top of the anatomic "snuff box," while the bottom is formed by the EPB. The EPL can be visualized when the thumb is extended, and its strength can be tested by having the patient hyperextend at the interphalangeal (IP) joint against resistance. The intrinsic extensor of the thumb can provide some degree of extension at the IP joint. Therefore, if EPL injury is suspected, it is important to compare extension at the IP joint with that of the unaffected thumb.

The fourth and fifth compartments contain the six tendons that extend the index through little fingers. Each finger has its own extensor digitorum communis (EDC) tendon. The index and little fingers have an additional independent extensor tendon—the extensor indicis proprius (EIP) for the index finger and the extensor digiti minimi (EDM) for the little finger. The fourth compartment contains the EIP and EDC tendons, while the fifth compartment contains only the EDM tendon. These six tendons can be seen over the dorsum of the hand, where they are poorly protected and prone to injury. In this region there are tendinous, ligamentous, and fascial connections between these tendons known as the *juncturae tendini. Because of these interconnections, a patient may be able to extend a digit, albeit weakly, even when there is a complete laceration of its EDC tendon.* To avoid missing a tendon injury on the dorsum of the hand, it is important that the examiner test for tendon *strength* and not just for active extension.

The course of the extensor tendons along the fingers is more complex, but a basic understanding of this anatomy is essential for the emergency clinician to evaluate and treat extensor tendon injuries (Fig. 49-3). The EIP tendon joins the EDC tendon at the level of the metacarpophalangeal (MCP) joint in the index finger. The EDM tendon parallels the course of the EDC tendon; the four EDC tendons eventually insert at the base of the proximal, middle, and distal phalanges. The most proximal insertion of the EDC tendon is at the level of

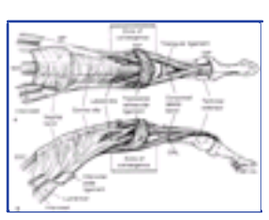

Figure 49-3 Digital extensor mechanism. *A,* Dorsal view. *B,* Lateral view. ORL, oblique retinacular ligament. See text and Figure 49-1 legend for other abbreviations. *(From Thomas JS, Peimer CA: Extensor tendon injuries: Acute repair and late reconstruction. In Chapman MW [ed]: Operative Orthopaedics, 3rd ed. Philadelphia, JB Lippincott, 2001, p 1498.)*

the base of the proximal phalanx. The tendon actually inserts in two ways. First, there is a loose dorsal insertion just distal to the MCP joint. In addition, the EDC tendon inserts into the volar plate via the sagittal bands. The sagittal bands are circumferential structures at the level of the metacarpal head that serve to keep the EDC tendon centered over the metacarpal head, as well as to provide a stable connection with the volar plate located on the palmar side of the hand. After its primary insertion at the level of the MCP joint, the EDC tendon then extends dorsally along the digit. The EDC trifurcates over the proximal phalanx (Fig. 49-4). Its major central slip inserts on the base of the middle phalanx (Fig. 49-5). The lateral branches of the EDC tendon join with the lateral bands from the interossei and lumbricals to form the co-joined lateral bands. The two co-joined lateral bands then fuse together over the middle phalanx to form the terminal extensor mechanism (TEM), which inserts into the base of the distal phalanx (Fig. 49-6). The triangular ligament is a connection between the two co-joined lateral bands that assists in keeping these structures on the dorsal aspect of the digit.

The sixth dorsal compartment of the wrist contains only one tendon: the extensor carpi ulnaris (ECU). This tendon originates at the lateral epicondyle of the elbow and inserts at the base of the fifth metacarpal. The ECU functions as a wrist extensor and ulnar deviator. It can be palpated just distal to the tip of the ulna, and its strength can be tested by forced ulnar deviation of the wrist.

GENERAL APPROACH TO EXTENSOR TENDON INJURIES

The key to detecting extensor tendon injuries in the ED is to perform a careful and thorough history and physical examination. Closed injuries may appear innocuous at first but may

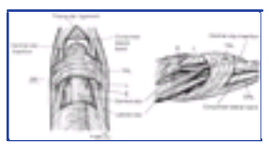

Figure 49-4 The zone of convergence of the digital extensor mechanism, which begins at about the midportion of the proximal phalanx and ends at the level of the central slip insertion into the dorsal base of the middle phalanx. Proximal to the zone of convergence, the extrinsic and intrinsic components of the extensor mechanism are separate: the central slip is extrinsic, whereas the lateral slips are intrinsic. Within the zone of convergence there is complete reciprocal crossover of fibers from the central slip and lateral slips. The products of the completed convergence are the central slip insertion and the conjoined lateral bands, both of which have dual muscular activity. PIP, proximal interphalangeal joint; TRL, transverse retinacular ligament; ORL, oblique retinacular ligament; E, extrinsic contribution to conjoined lateral bands; I, intrinsic contribution to central slip insertion. *(From Thomas JS, Peimer CA: Extensor tendon injuries: Acute repair and late reconstruction. In Chapman MW [ed]: Operative Orthopaedics, 3rd ed. Philadelphia, JB Lippincott, 2001, p 1500.)*

result in tendon injuries that often lead to severe deformities or dysfunction if undetected. Closed injuries are also commonly associated with fractures. A hand radiograph is recommended in closed hand injuries when a fracture is suspected or in open-hand injuries where fracture or a foreign body is suspected. It is generally accepted that all open injuries that result from glass should be radiographed. Plain radiographs have a sensitivity of approximately 98% for detecting radiopaque foreign bodies (e.g., gravel, glass, metal). [14]

Figure 49-5 The extensor mechanism on the dorsum of a finger. *Arrows* point to the radial and ulnar lateral band portions of the extensor mechanism, and the probe lifts the entire structure up off the phalanx.

Injuries to extensor tendons from lacerations are quite common, especially on the dorsum of the hand, where they are superficially located. *All dorsal wrist, hand, and digit lacerations should be assumed to have an underlying tendon laceration until proven otherwise* (Fig. 49-7). Digital extension, albeit it weak, can still occur with partial tendon lacerations of up to 90%, so visualization of the tendon and careful strength testing are required to definitively rule out a partial injury. In some cases, the specific diagnosis simply cannot be

Figure 49-6 The terminal extensor mechanism.

made on the first examination (see below). Complete lacerations of an EDC tendon on the dorsum of a hand can also still allow digital extension through the juncturae tendini.

After assessing the strength and neurovascular status of the injured hand, it is imperative that the emergency clinician visually inspect the wound thoroughly. Inspection should include an assessment of the degree of wound contamination,

Figure 49-7 Because of their superficial location, it is difficult to avoid at least partial injury to extensor tendons in even superficial lacerations of the dorsum of the wrist, hand, or fingers. In some cases *(A)* the injury is obvious. In other cases, a partial tendon laceration is not appreciated, but becomes obvious when complete rupture occurs *(B)*. In this case the patient seemingly had full tendon function during the initial ED evaluation, but the entire tendon could not be visualized due to an uncooperative patient. An unappreciated partial laceration progressed to complete rupture by the time of suture removal. The injury was diagnosed and an expeditious delayed primary repair resulted in a good outcome. Note: Inability to extend index finger is evident in both photos.

as well as a search for foreign bodies and occult tendon lacerations. It is often necessary to extend the skin laceration to aid in the visualization of a possible tendon injury. Because an extensor tendon is a mobile structure, it is imperative that if it is exposed, it is visualized in its entirety through a full range of motion. *It is especially important to examine the tendon in the position of injury, because the tendon injury frequently does not lie directly under the skin wound* (Fig. 49-8).

The definitive examination of any wound must occur under the best possible conditions—with a good light source, a bloodless field, adequate local anesthesia, and a cooperative patient (Fig. 49-9). It may be impossible to adequately assess some patients completely during the first ED visit. Final diagnosis must be delayed until the proper circumstances permit the required conditions. Occasionally, however, patient noncompliance thwarts even the most carefully planned follow-up. Often the patient's pain, swelling, anxiety, or degree of intoxication/altered sensorium limits the clinician's diagnostic ability; therefore, *it would not be considered standard of care to diagnose the presence of, or the full extent of, all extensor tendon injuries immediately*. Whenever logistically possible it is suggested that a specialist be consulted when an extensor tendon injury is suspected, either by mechanism or location of the wound, or tendon dysfunction. Under most circumstances, however, there is no value in obtaining an immediate onsite consultation with a hand surgeon/orthopedic surgeon, since the intrinsic scenario would similarly limit any clinician's diagnostic acumen.

If the examining clinician suspects, but is unable to locate, a tendon laceration, or if a patient is uncooperative with the examination and the circumstances prohibit ideal initial care, the patient should be referred for follow-up in 1 to 3 days for a repeat examination. Interim wound care with skin closure and splint application is advised. A delay of a few days for definitive diagnosis or surgical repair or both does not result in any significant alteration in final outcome. Delayed primary repair, without the need for tendon grafting or tendon transfer, is a well accepted technique. In fact, many

Figure 49-8 The location and depth of this laceration suggests an extensor tendon injury. On examination, the patient had full extension. Note that an intact tendon is visualized when the laceration is examined with the fingers in extension *(A)*. The injury occurred with a closed fist, and when the fingers were fully flexed, an 80% laceration of the extensor tendon could be viewed in the depths of the wound *(B)*. Note: Contrary to this figure, gloves should be worn by the examiner.

hand surgeons are reluctant to immediately repair even a complete extensor tendon laceration in a contused, potentially contaminated, wound. The exact time frame under which such delayed repair results in an outcome similar to immediate repair is not well defined and depends on the clinical scenario. Usually, delayed repair up to 7 to 10 days will ensure an outcome similar to an immediate repair, but this is variable depending on the injury. Inability to rule out a tendon injury in the ED and the mandate for follow-up, *with a specific following time frame indicated*, should be clearly documented on the medical record and discharge instructions.

Antibiotic Use

There are no data to support or refute the use of prophylactic antibiotics as a routine adjunct following tendon injury. In

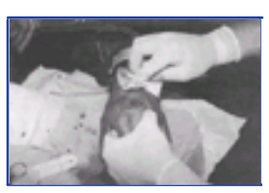

Figure 49-9 With this injury on the dorsum of the hand, the clinician should expect to find an extensor tendon injury. The proper way to examine this wound includes a bloodless field (often with the use of a blood pressure cuff tourniquet), good light, a cooperative patient, excellent anesthesia, and extending the skin laceration to visualize the depths of the laceration. Note that the tendon is visualized with the fingers manipulated from full extension to full flexion.

general, prophylactic antibiotics have not been demonstrated to reduce infection rates following soft tissue injury in the setting of proper wound cleaning. Neither have they been proven to reduce infection rates in the absence of gross contamination, retained foreign material, or extensive contusion, or after a delay in cleaning. Many clinicians opt for antibiotics with gram-positive (including antistaphylococcal) coverage if the tendon has been injured or sutured, but no universally accepted standard of care exists. An individualized approach is advocated. Prophylaxis is generally only used for 3 to 5 days post injury unless extenuating circumstances exist (such as immunocompromise, human bite, unusual source of contamination, or peripheral vascular disease). If there is doubt about the sterility of a wound, tendon repair should not be attempted.

PREPARATION FOR REPAIR

Prior to attempting repair of an open extensor tendon injury in the ED, it is essential that the treating clinician be prepared and has the proper equipment available. Patients should be placed supine on a gurney that ideally has an arm board attached. Bright overhead lighting is important for wound exploration so that the presence of tendon injuries and foreign bodies can be adequately assessed. Instruments should include, at a minimum, a needle holder; two skin hooks and retractors; sharp (i.e., "iris") and blunt-nosed scissors; several small hemostats; and one pair of small, single-toothed (i.e., Adson) forceps.

The choice of suture material depends on the location of the tendon injury. For the repair of *complete* tendon injuries on the dorsum of the hand, nonabsorbable, synthetic, braided sutures are preferred. [25] Polyester sutures, such as Ethibond or Mersilene, are recommended. Nylon sutures are acceptable but are less ideal, as colored nylon may be visible under the skin. Chromic and plain gut should be avoided because they will dissolve before adequate tendon healing has occurred. Silk is not desirable because of its reactivity. Most extensor tendons on the dorsum of the hand will accommodate 4-0 sutures, but 5-0 suture material may be needed for smaller

tendons. Small, "plastic repair," tapered needles should be used to avoid tearing the tendon. *Partial* tendon injuries of the digits are best repaired with fine, synthetic, absorbable sutures such as polyglactin (i.e., Vicryl).

It is imperative that the clinician use adequate anesthesia so that thorough wound exploration can occur. A field block or regional nerve block can be used on the dorsum of the hand, while local anesthesia or a digital nerve block can be used on the fingers (see Chapter 30 and Chapter 32). Many clinicians readily use lidocaine with epinephrine in the hand except in areas supplied by end arteries. It is important to liberally anesthetize the area around the wound, since many lacerations *must be extended to afford access to the surgical field*. It is a common error to neglect to extend a laceration and to attempt examination, cleaning, or repair through a small initial skin laceration.

Following the administration of anesthesia, a tourniquet may be placed on the involved limb if hemostasis is problematic. It is absolutely essential that adequate control of blood flow be obtained before attempting to repair a tendon laceration. It is very difficult to find the proximal end of a retracted tendon in a bloody field. Before application of a tourniquet, the patient's arm should be wrapped in several layers of cast padding as a comfort measure, and the arm should be elevated for at least 1 minute to allow blood to drain by gravity. A blood pressure cuff is placed on the mid to upper arm, wrapped in several more layers of cast padding, then inflated to 260 to 280 mm Hg. Once inflated, the tubes are clamped tightly using a hemostat. The use of cast padding during inflation helps avoid inadvertent unraveling of the cuff. The use of a hemostat to clamp the blood pressure cuff tubes helps avoid a slow leak in the cuff with resultant deflation. A blood pressure cuff tourniquet is generally well tolerated by patients for approximately 15 to 20 minutes. If tendon repair cannot be accomplished in this amount of time, it is likely that the injury is too complex for repair in the ED. When necessary, the use of parenteral sedation may allow the patient to tolerate a longer tourniquet time (see Chapter 34).

Atraumatic technique is essential for minimizing adhesions and scar tissue formation. Tendons should be handled delicately, avoiding crushing forces or excessive punctures with forceps and needles. Forceps should only be used on the exposed, cut end of the tendon whenever possible. [13]

PATTERNS OF INJURY AND MANAGEMENT

The treatment for extensor tendon injury depends primarily on whether the injury is open or closed, as well as the anatomic location of the injury. The most widely accepted classification system is that developed by Verdan, which divides the hand and wrist into eight anatomically based zones (Fig. 49-10).[26] It is quite useful for emergency clinicians to become familiar with this classification, because in many instances the zone of injury can help determine whether tendon repair should be attempted in the ED. One must keep in mind that repair of lacerated extensor tendons within 72 hours of injury is still considered primary closure. Therefore, while emergency clinicians may repair many extensor tendon injuries immediately, some injuries are best managed with delayed repair. In these cases, initial care in the ED should consist of sterile skin prep, copious wound irrigation and inspection for foreign bodies, skin closure, splint application,

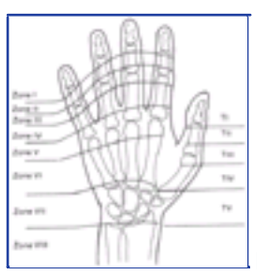

Figure 49-10 Dorsum of left hand. The injury classification system recommended by Verdan includes eight anatomically based zones. *(From Blair WF, Steyers CM: Extensor tendon injuries. Orthop Clin North Am 23:142, 1992.)*

and a referral to a hand specialist for further care in 1 to 5 days. A dorsal plaster/fiberglass splint, incorporating a metal foam finger splint, is an ideal way to totally immobilize a finger (Fig. 49-11). See an additional discussion in the previous section on the general approach to extensor tendon injuries.

Zone 7 and 8 Injuries

Zones 7 and 8 consist of the area over the wrist and dorsal forearm, respectively. [19] Extensor tendon lacerations in these regions can be quite complex and are therefore not repaired in the ED. Because of the close proximity of extensor tendons in the distal forearm, lacerations such as stab wounds may appear innocuous but often result in multiple tendon lacerations. At the wrist level, extensor tendons are covered by a retinaculum that is lined with synovium. While this tissue allows smooth gliding of tendons during normal activity, the presence of synovium increases the risk for adhesions following tendon repair. In addition, lacerated tendons in the wrist and distal forearm may retract away from the site of initial injury. This may make tendon retrieval and repair quite difficult and may necessitate incision of the retinaculum and exploration of 1 or more of the compartments.

As a result of the potential complexity of these injuries, all tendon lacerations in zones 7 and 8 require formal surgical exploration and repair. ED management of these patients includes local wound care (see Chapter 35) with primary repair of the skin (see Chapter 36) and placement of a volar splint with 35° of extension at the wrist and 10°–15° of flexion at the MCP joints (see Chapter 51). Many hand surgeons recommend prophylactic antibiotics for all open tendon lacerations, while others prescribe them selectively for patients with wounds where contamination is suspected or significant injury to the surrounding tissues is present. There are no prospective data proving or disproving the value of prophylactic antibiotics in either circumstance. These patients should

Figure 49-11 An effective way to fully immobilize a finger with a tendon laceration is to incorporate an aluminum foam splint into the middle of a standard dorsal plaster/fiberglass splint.

be promptly referred to a hand surgeon so that repair may be undertaken within 1 week of injury.

Zone 6 Injuries[7] [19] [25]

Zone 6 consists of the area over the dorsum of the hand. Extensor tendon injuries in this region frequently result from lacerations owing to broken glass or another sharp object. Common pitfalls in ED management of these injuries are usually related to failure to recognize that the tendon has been injured. It is important to remember that these tendons are superficially located, partial tendon lacerations may occur, and weak extension of a digit is possible in the presence of a complete tendon laceration because of transfer of extensor function through the juncturae tendini. Lacerations of the EIP or EDM tendons are evidenced by an inability to independently extend

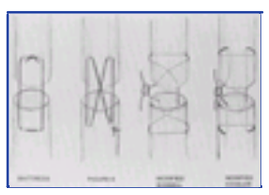

Figure 49-12 Suture techniques used in extensor tendon repair. *(From Newport ML, Williams CD: Biomechanical characteristics of extensor tendon suture techniques. J Hand Surg 17:1117, 1992.)*

the index or little fingers, respectively. All of these pitfalls can be avoided if a careful physical examination is performed, including a thorough wound exploration under sterile conditions using a tourniquet, adequate local anesthesia, and good lighting.

Extensor tendon injuries in zone 6 are usually appropriate for repair in the ED. Because of the juncturae tendini, extensor tendons in zone 6 are less likely to retract than those in zone 7 or 8; however, the severed tendon may retract when the injury is more proximal. The distal end of a severed tendon is usually easy to find by passively extending the patient's affected digit to bring the end into view. Retrieval of the proximal portion of a severed tendon is sometimes required and usually can be accomplished in the ED. Before searching for the proximal end of the tendon, the clinician should have a 4-0 nylon suture loaded onto a needle holder. When the proximal end is located, this suture should be placed as a holding suture as far proximal as possible so that the tendon is not lost again. It is often necessary to use a scalpel to extend the wound proximally in a direction parallel to the course of the injured tendon to obtain adequate exposure. One should then begin to search for the tendon by lifting up this overlying skin with a forceps and inspecting the proximal portion of the wound. Sometimes the blood-stained end of a tunnel can be seen; this may contain the proximal end of the tendon. By gently placing a small hemostat or toothed forceps up this tunnel, the tendon stump can often be pulled into view.

Once both ends of the injured tendon have been located, the technique used for repair is dependent on the size and shape of the tendon. While larger, round tendons can accommodate sutures that pass through the core of the tendon, smaller or flat tendons are difficult to repair using this technique. Most of the tendons in zone 6 can be repaired with either a modified Kessler or a modified Bunnell core suture technique, using 4-0 nonabsorbable suture (Fig. 49-12). Both of these techniques involve first placing a single suture into

one half of the cut tendon. The suture is placed into the tendon core by inserting the suture needle into the exposed, cut end, and then weaving the suture through the lateral tendon margins. Next, the same suture is placed through the core of the opposite half of the cut tendon. The suture ends are tied in a square knot in between the cut ends of the tendon, bringing the two halves together.

Smaller tendons may be repaired using a figure eight or horizontal mattress suture (see Fig. 49-12). Small, tapered needles should be used to avoid tearing the tendon. In a cadaver study comparing these multiple suture techniques, it was found that the modified Bunnell technique provided the strongest extensor tendon repair.[17] In addition, this technique produced no gaping between the repaired tendon ends and minimized the postrepair restriction of flexion at the MCP and proximal interphalangeal (PIP) joints. It is important to passively test the degree of flexion at the MCP joint after a zone 6 tendon repair to be certain that the tendon has not been excessively shortened.

To improve the tensile strength of the repair, a number of other suture techniques may be used. [13] One option is to increase the number of suture strands that cross the repair site (e.g., four strands rather than two). A cadaver study that compared various four-strand tendon repair techniques concluded that the MGH technique was more resistant to gap formation than either the Krackow-Thomas or four-strand modified Bunnell technique (Fig. 49-13).[9] Another way to improve tensile strength is to place a peripheral suture in addition to the core suture. A running suture can be placed circumferentially around the repair site using synthetic, absorbable

Figure 49-13 Four-strand suture techniques used in extensor tendon repair. *(Adapted from Howard RF, Ondrovic L, Greenwald DP: Biomechanical analysis of four-strand extensor tendon repair techniques. J Hand Surg 22:839, 1997.)*

material (e.g., polyglycolic acid, polyglactin, polydioxanone). Alternatively, sutures may be placed laterally along both sides of the tendon, starting at about 1 cm on either side of the repair site. The ultimate choice of repair technique will depend largely upon the treating clinician's familiarity with extensor tendon repair, as well as the size of the tendon.

The approach to partial extensor tendon lacerations is not well-defined, and no definitive standard of care exists. One evidence-based analysis identified 141 papers in its literature search, but none was relevant to the question of partial extensor tendon injury repair. [23] The authors concluded that there is no direct evidence to assist in answering this question. Given the lack of literature on the subject, a reasonable approach may be to extrapolate from data on flexor tendon injuries. It has been demonstrated that many partial *flexor* tendon lacerations do well without repair, [16] but disagreement still remains among hand surgeons concerning the need for repair of these injuries. In a survey of hand surgeons, 30% of respondents repaired *all* partial flexor tendon lacerations, while 45% of respondents repaired *only* lacerations with greater than 50% cross-sectional area involvement. [15] Except at the wrist level, extensor tendons are not covered with synovium and are less likely than flexor tendons to develop adhesions after repair. This encourages some authors to recommend repair of most partial extensor tendon lacerations. While the ideal approach to these injuries is not known, it is reasonable to consider repair of partial extensor tendon lacerations to be *optional* if less than 50% of the cross-sectional area is involved. However, such injuries must be splinted for 3 to 4 weeks to ensure that a partial laceration is not converted into a complete injury. Skin closure, splinting, and referral for follow-up is a standard approach to unsutured partial extensor tendon lacerations.

After repair of a lacerated EDC tendon in zone 6, a plaster or fiberglass volar splint should be applied (see Chapter 51) so that the wrist is in 45° of extension, the affected MCP joint is in neutral (0° of flexion), and the unaffected MCP joints are in 15° of flexion. The PIP and distal interphalangeal (DIP) joints should be allowed full range of motion. After 10 days, the MCP joints are allowed 20°–30° of flexion. If there is an isolated EIP or EDM tendon injury, then only the index or little finger must be included in this splint. Dynamic extension splinting may be used as early as 3 days after tendon repair, so close follow-up is recommended. [1]

Zone 5 Injuries[5] [22]

Zone 5 consists of the area over the MCP joint. Open injuries in this region should be considered secondary to a human tooth bite until proven otherwise. This is especially true if the injury occurs over the first or second MCP joint, as this is frequently the location of a clenched-fist ("fight-bite") injury. ED evaluation must begin with a careful and persistent history and physical examination, although patients' reluctance to admit to punching someone in the mouth is notorious. The wound should be inspected through its full range of motion, since the position of the EDC tendon changes with hand position. It is generally recommended that radiographs be obtained in all of these injuries to evaluate for metacarpal head fractures, air in the joint space, or presence of a foreign body, such as a tooth fragment [1] (Fig. 49-14).

If, after a thorough evaluation, it is determined that a human bite to this region has resulted in a superficial skin

Figure 49-14 *A,* Regardless of this patient's history, this wound is highly suggestive of a human bite injury. Human bites cause extensor tendon injuries, fractures, and joint capsule injuries, and can harbor foreign bodies. *B,* This patient stated that he cut his hand on a piece of metal at work (expecting a Workers Compensation claim), but was unable to explain the piece of tooth that was found in the wound on exploration.

laceration only, without injury to the underlying tendon or joint, outpatient management is appropriate. The wound should be copiously irrigated and left open. A volar splint is applied (see Chapter 51) with the wrist in 45° of extension, the MCP joints in the neutral position (0° of flexion), and the hand dressed with a bulky dressing. Most authors recommend that 3 to 5 days of prophylactic antibiotics be given to these patients, and patients should be seen in 24 to 36 hours for a repeat examination to evaluate for wound infection. As mentioned previously, there are inadequate prospective data to prove or disprove the value of prophylactic antibiotics for extensor tendon injuries, and no universally accepted standard of care exists. It would be prudent to err on the side of caution if doubt exists concerning the degree of contamination and potential for infection.

If a human bite results in tendon damage, including partial or complete laceration, some clinicians opt for admission and IV antibiotics. However, no specific standard of care exists. Outpatient therapy is acceptable in the reliable patient who has access to follow-up. Delayed closure with tendon evaluation/repair should be undertaken by a hand surgeon after 5 to 10 days of antibiotics. [1] [25] Primary closure of even seemingly clean and well-irrigated human bites in this region is not advisable because of the increased risk of wound infection, as well as the potential for septic destruction of the MCP joint if it is violated. If an *open joint* is noted by physical examination, a more aggressive approach is warranted. Such patients are generally admitted for IV antibiotics, following copious irrigation. [1] [25] However, after initial ED treatment (aggressive wound care and initial IV antibiotics), there may be a role for outpatient therapy in selected cases. If a patient suffers a zone 5 tendon injury, and it can be determined with complete certainty that it was caused by a relatively clean, sharp object rather than by a human bite, primary closure is appropriate. Referral of these injuries to a hand surgeon is a common practice, given the complexities of the injury and the sequelae. Careful repair of lacerations to both the EDC tendon and the sagittal bands is necessary to prevent subluxation of the EDC tendon away from the center of the metacarpal head. Initial ED management of non-human bite injuries is often limited to skin closure, splinting as described earlier, and referral to a hand surgeon within 1 to 5 days.

Closed extensor tendon injuries in zone 5 usually result from the acute or recurrent application of compressive forces to the MCP joint capsule. Closed injuries in this region are sometimes referred to as *boxer's knuckle*. Repetitive closed injury to the MCP joint region can produce small tears of the EDC tendon, the sagittal bands, or

the joint capsule. These patients tend to present with chronic and recurrent pain and swelling at the MCP joint region and usually have normal radiographs. Acute trauma may result in the same injuries or cause more severe damage to the extensor hood. Such patients may have complete disruption of the extensor mechanism, including damage to the central tendon and the sagittal bands. The MCP joint is swollen, has decreased mobility, and may exhibit an extensor lag. Traumatic subluxation of the EDC tendon may be present, and usually involves the middle finger with subluxation to the ulnar side (Fig. 49-15). The subluxation becomes more prominent with flexion at the MCP joint. There is controversy regarding the initial management of closed injuries to this region. While some authors prefer initial surgical repair,[22] others use an initial trial of extension splinting in some or all cases. [1] [5] [25] Splinting the MCP joint in neutral or slight flexion for 6 weeks has been recommended for dislocations presenting within 2 weeks of injury, with operative repair reserved for more delayed presentations. [1]

Zone 4 Injuries[5]

Zone 4 consists of the area over the dorsal aspect of the proximal phalanx between the MCP and PIP joints. The extensor tendon is a broad, flat structure in this region and is relatively easy to repair. Because the extensor tendon is flat and conforms to the roundness of the proximal phalanx, tendon injuries in this area usually result from a laceration and are almost always incomplete. As a result, extension at the PIP joint is usually not impaired. It is therefore imperative that all

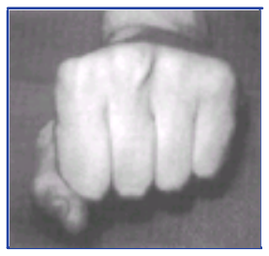

Figure 49-15 Ulnar dislocation of the extensor tendon of metacarpophalangeal joint of the middle finger. *(From Leddy JP, Dennis TR: Tendon injuries. In Strickland JW, Rettic AC [eds]: Hand Injuries in Athletes. Philadelphia, WB Saunders, 1992, p 196.)*

of these wounds be explored carefully, remembering that the extensor tendon lies immediately beneath the thin overlying skin. Tendons tend not to retract in this area, so close inspection will usually result in location of the injured tendon.

A hand surgeon usually repairs central slip lacerations or any laceration that results in an extension lag at the PIP joint. The decision regarding whether to repair a partial tendon laceration and whether it should be repaired by the emergency clinician in this zone is best discussed with the consulting hand surgeon. In general, because of the duality of the extensor system in this region, lacerations of a single lateral slip can either be repaired with 5-0 nonabsorbable sutures or be left unrepaired and splinted. A running suture or simple interrupted sutures with buried knots are appropriate for this area. Postrepair splinting depends on the presence of tension at the repair site. Minor lacerations in zone 4 that do not result in tension on the repair site can be treated with a finger guard for 7 to 10 days and early range of motion. Larger lacerations or those that result in tension at the repair site are usually treated in a splint that extends from the forearm to the digit for 3 to 6 weeks. The splint should be applied so that the wrist is in 30° of extension, the MCP joints at 30° of flexion, and the PIP joint in neutral position. Fingers should be grouped so that either digits 2 and 3 or digits 3 through 5 are immobilized (see Chapter 51).

It is important to recognize that *complex partial* tendon lacerations (e.g., a laceration of a lateral slip resulting from a saw) in zone 4 may result in damage to the gliding layer located between the tendon and the bone. If the patient is still able to actively extend the digit at the PIP joint, then these *complex partial* tendon lacerations are best managed by debriding the frayed tendon ends and splinting the digit in extension rather than attempting to suture the damaged tendon. The splint should be worn for 10 days, followed by active range of motion.

Zone 3 Injuries[1] [19] [22]

Zone 3, the area over the PIP joint, is a common site of both closed and open injury. Open injury usually results from laceration with a sharp object. It is imperative that these wounds be carefully explored in the ED to rule out penetration of the joint capsule. Patients with wounds that are suspected of penetrating the joint are generally taken to the operating room for surgical exploration, irrigation, and treatment with IV antibiotics, but protocols vary.

Zone 3 tendon lacerations can result in long-term deformity if not carefully repaired, and patients with such injuries are commonly referred to a hand surgeon. Partial lacerations of the central slip or lateral bands are managed variably, and it is advisable to discuss these injuries with the consulting hand surgeon. Lacerations in this area may sometimes result in a complete central slip injury. This may present as an acute boutonnière ("buttonhole") deformity, where the PIP joint rests in 60° of flexion. The presentation may be subtler, however, and may only be noticeable by weakened extension at the PIP joint or incomplete extension by only a few degrees.

The boutonnière deformity develops when the central slip is ruptured by an open or closed mechanism, leading to unopposed action of the flexor digitorum superficialis tendon (Fig. 49-16). This results in flexion at the PIP joint, protrusion of the head of the proximal phalanx between the two lateral bands, and disruption of the triangular ligament. When this occurs, the lateral bands are displaced volar to the axis of motion of the PIP joint. The lateral bands then paradoxically become flexors of the PIP joint. In addition, the extensor hood mechanism is pulled more proximally, resulting in increased tension on the terminal extensor mechanism and hyperextension at the DIP joint. Thus, the boutonnière deformity consists of flexion of the PIP joint with hyperextension at the DIP joint.

Open central slip injuries are usually managed operatively, and complex injuries may require direct attachment of the tendon to bone or tendon reconstruction. If the consulting hand surgeon chooses not to repair the tendon injury immediately, the skin should be closed and a plaster splint applied in the same fashion as described for zone 4 injuries (see Chapter 51). Thermoplastic splints allow splinting of the hand without involvement of the wrist but are generally not available in the

Figure 49-16 Boutonnière deformity. This can be an open or closed injury. Note flexion of the PIP joint and extension of the DIP joint, from a laceration of the central slip mechanism.

ED setting. These patients should be promptly referred to a hand surgeon so that repair may be undertaken within 1 week of injury.

Patients with closed injuries to zone 3 present commonly to the ED. They may complain of a direct blow to the dorsal PIP joint, or they may complain of a "jammed" finger. This injury occurs when an object such as a ball delivers a sudden axial loading force with forced flexion of the PIP joint while it is extended. These patients commonly complain of a painful, swollen PIP joint, which often makes the examination difficult. Some of these injuries may represent PIP joint dislocations that were spontaneously or manually reduced before the patient's ED presentation (see Chapter 50). The tendon injury that is important to recognize in this setting is an occult isolated central slip rupture. Patients may have decreased extension at the PIP joint, but extension is usually normal because the lateral bands are the primary extenders of this joint. With forced extension against resistance, patients usually have pain and may have decreased strength. To eliminate pain as the cause of decreased mobility, it may be helpful to test PIP extension against resistance after performing a digital block. With acute central slip rupture, PIP joint extension may be particularly weak when the MCP and wrist joints are held in maximal flexion. In this position, a 15° or greater loss in active extension is highly suggestive of a central slip injury. [22] The Elson test may also help identify this injury [2] (Fig. 49-17 (Figure Not Available)).

The boutonnière deformity usually does not develop in patients with closed zone 3 injuries until 10 to 21 days after injury. The only way to prevent this deformity is to have a high index of suspicion for its presence and treat these patients conservatively. It is advisable that all patients with a swollen, tender PIP joint and pain with flexion or extension be splinted and referred for close follow-up. A dorsal splint should be applied overlying the PIP joint, keeping it in full extension. This can be

accomplished using an aluminum foam-backed splint or a Bunnell ("safety pin") splint, although the latter may not be available in the ED [1] (see Chapter 51). The MCP and DIP joints should be left free to have full, active range of motion (Fig. 49-18). If a central slip attachment fracture is present, orthopedic consultation is recommended, as these patients may require surgical internal fixation. [10]

Zone 1 and 2 Injuries[1] [19] [22] [25]

Zones 1 and 2 consist of the area over the DIP joint and the middle phalanx, respectively. In zone 2 the co-joined lateral bands come together to form the terminal extensor mechanism (TEM) and are held together, in part, by the triangular ligament. The TEM inserts on the base of the distal phalanx and allows extension at the DIP joint. Complete disruption of the TEM results in inability to extend at the DIP joint. Because of the unopposed action of the flexor digitorum profundus (FDP) tendon, the DIP joint rests in the flexed position. This is known as a mallet deformity of the finger (Fig. 49-19).

Tendon *lacerations* in zones 1 or 2 that result in a partial or complete mallet deformity generally warrant discussion with a hand surgeon. Management consists of repair of the lacerated tendon and post-repair immobilization. Some surgeons will use only an external splint, while others prefer placement of a K-wire through the distal phalanx into the

Figure 49-17 (Figure Not Available) *A,* Diagrammatic explanation of a boutonnière deformity. *(From Newmeyer WL: Primary Care of Hand Injuries. Philadelphia, Lea & Febiger, 1979.) B–E,* The Elson test for early diagnosis of an acute rupture of the central slip of the extensor digitorum communis tendon. Such rupture results in boutonnière deformity, in which the distal interphalangeal (DIP) joint is hyperextended, as shown. With the patient's finger flexed (over a straight edge) at the PIP joint, the examiner palpates the dorsal surface of the middle phalanx *(B)*. If the central slip is intact *(C),* PIP joint flexion causes the slip to tighten distally, thereby relaxing the lateral bands and leaving distal phalanx flail *(arrows)*. Thus, when the patient is asked to extend the digit, the examiner feels pressure that is necessarily being exerted by an intact central slip. If the central slip is disrupted, however, the examiner feels no pressure on the dorsum of the middle phalanx as the patient tries to extend the digit *(D)*. It is possible for the patient to extend the injured finger successfully only by hyperextending (by action of the lateral bands) *(arrows, D and E)*. *(From Connolly JF: What to do for a "jammed" finger. J Musculoskeletal Med 99, 1988 May.)*

middle phalanx to help stabilize the joint. One technique for tendon repair involves placement of a roll-type suture (dermatotenodesis), which incorporates the tendon and overlying skin into a single suture [1] [19] (Fig. 49-20). The DIP joint is then splinted in full extension for at least 6 weeks. Occult partial tendon lacerations are important to recognize to prevent development of a mallet deformity. If there is a partial tendon laceration in zone 1 or 2 that does not result in any extension lag, the approach to repair is variable, and it is advisable to discuss the repair with the consulting hand surgeon. In general, partial tendon lacerations of less than 50% of the tendon area that do not result in an extension lag may be splinted in extension for 7 to 10 days with or without repair of the tendon itself. [1] Partial tendon lacerations of more than

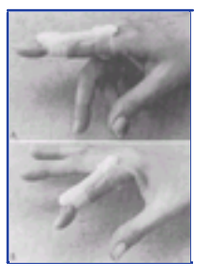

Figure 49-18 *A,* Boutonnière splint. *B,* This splint allows active flexion at the metacarpophalangeal and distal interphalangeal joints.

50% that do not result in an extension lag may be repaired by a hand surgeon or an emergency clinician who is experienced in the repair of these injuries. In either case, it is advisable to discuss whether tendon repair will occur in the ED or the operating room with the consultant hand surgeon.

If the zone 1 or 2 tendon laceration is repaired in the ED, it can be approximated using a combination of running and cross-stitch sutures, [1] using 5-0 nonabsorbable suture material (Fig. 49-21). It is important that the tendon ends be approximated but not pulled too tightly; otherwise joint stiffness and limitation of flexion will occur. After repair of a partial tendon laceration, the DIP joint should be splinted in extension for 6 weeks, followed by 2 to 4 weeks of night splinting

Figure 49-19 Mallet finger deformity. *(From Leddy JP, Dennis TR: Tendon injuries. In Strickland JW, Rettic AC [eds]: Hand Injuries in Athletes. Philadelphia, WB Saunders, 1992, p 180.)*

and active range-of-motion exercises. Patients should be warned after tendon repair that there is likely to be some residual loss of flexion at the DIP joint, even in the best case.

Closed injuries in zones 1 and 2 may result in a partial or complete mallet deformity depending on the injury pattern. These injuries are usually caused by an axial loading force with forced flexion of the DIP joint while it is being held in extension. A common ED presentation of this injury is a patient who complains of pain and swelling at the DIP joint after a ball strikes his or her fingertip.

Closed tendon injuries in this region can generally be classified into three types. In the first type of injury, there is a closed rupture of the TEM. The second type of injury is an avulsion fracture of the dorsal lip of the distal phalanx. This fracture is intra-articular, but there is no volar displacement of the remaining portion of the distal phalanx. Both type 1 and type 2 injuries can be treated by splinting in full extension for 6 weeks. The splint should hold the DIP joint in extension while allowing free range of motion of the PIP joint (Fig. 49-22). The splint can be constructed from an aluminum, foam-backed splint or from a prefabricated Stack splint (see Chapter 51).

The third type of closed injury is an intra-articular avulsion fracture of the dorsal lip of the distal phalanx *with* volar displacement of the remaining portion of the distal phalanx (Fig. 49-23). Normally the DIP collateral ligaments hold the distal phalanx in place; however, if there is a large enough fracture fragment (usually more than 50% of the articular surface), then the remaining distal phalanx fragment displaces in the volar direction secondary to unopposed action of the FDP tendon. When volar displacement of the distal phalanx occurs, this injury may require more aggressive treatment for an optimal outcome. [22] Operative repair usually involves placement of a K-wire and open reduction and internal fixation of the fracture. It is important to remember that it is the presence of volar subluxation, not the size of the avulsion fracture, that determines the need to consider operative management.

Any injury, whether open or closed, that results in a complete disruption of the TEM may result in a swan-neck deformity (Fig. 49-24). This deformity consists of flexion at the DIP (a mallet finger) and hyperextension at the PIP joint. This results from a dorsal and proximal displacement of the lateral bands causing increased extension forces on the middle phalanx. This complication often can be avoided if disruption of the TEM is diagnosed and treated correctly in the ED.

Figure 49-20 Dermatotenodesis technique for zone I extensor tendon repair. *A,* Fresh lacerations of the extensor mechanism over the distal joint with mallet finger deformity are

repaired by a running-type suture, which simultaneously approximates the skin and tendon *(B and C)*. A small dressing is applied along with a splint, which maintains the joint in full extension. The sutures are removed at 10 to 12 days, but the splint is continued for a total of 6 weeks. *(Adapted from Doyle JR: Extensor tendons: Acute injuries. In Green DP [ed]: Operative Hand Surgery, 4th ed. New York, Churchill Livingstone, 1999, p 1968.)*

COMPLICATIONS

All extensor tendon repairs are subject to the usual complications of wound infection and skin breakdown secondary to prolonged splinting. Tendon rupture is a rare complication following tendon repair and may result from inadequate suture technique or premature motion against resistance. It is important when extensor tendons are repaired for at least five throws to be used and a square knot to be tied. All extensor tendon repairs require some period of complete immobilization during tendon healing, and it is important for the emergency clinician to stress the necessity for patient compliance.

Extensor tendon injuries in zone 7 tend to have the worst prognosis. Because of the presence of a synovial lining, postrepair adhesions may occur. The adhesions may lead to decreased excursion of the extensor tendons with resultant decreased mobility at the wrist. There may also be limitation of finger flexion when the wrist is flexed, as well as finger extension when the wrist is extended. Because of the lack of synovium, the relatively simple anatomy, and the usual lack of associated injuries, zone 6 tendon injuries tend to have fewer complications than other areas of the hand. The tendons in zone 6, however, do have a tendency to shorten if the tendon ends are approximated too tightly. This may result in a restriction of PIP and MCP joint flexion.

Zone 5 injuries are particularly prone to infection, as injuries in this region commonly occur from a human bite. In addition, if the extensor hood covering the MCP joint is not carefully repaired, subluxation of the EDC tendon may occur. [7] If complex partial tendon lacerations in zone 4 are managed too aggressively, then tendon shortening and stiffness may result. As discussed previously, these injuries are often

Figure 49-21 Repair of zone II extensor tendon lacerations *(A)*. Zone II lacerations may be repaired with a running stitch *(B)* near the edge of the laceration, followed by the cross-stitch *(C)*. The extensor tendon in this zone is comparatively thin, and this dual-suture technique has sufficient grasping capability to restore and maintain the normal length of the extensor. The repair is protected by 6 weeks of DIP joint splinting, as for zone I injuries. *(Adapted from Doyle JR: Extensor tendons—Acute injuries. In Green DP [ed]: Operative Hand Surgery, 4th ed. New York, Churchill Livingstone, 1999, p 1971.)*

best managed with splinting alone. A common complication of zone 3 extensor tendon injuries is development of a boutonnière deformity. This usually results from failure to diagnose or adequately immobilize a central slip injury. Similarly, undiagnosed or improperly treated extensor tendon injuries in zones 1 and 2 may lead to either a swan-neck or chronic mallet deformity of the digit. DIP joint splinting itself may result in skin ulceration or tape allergy, usually presenting in the second week of treatment.[1] Skin breakdown may be encouraged if the DIP joint is splinted in hyperextension, because of decreased skin perfusion.

POSTREPAIR CARE AND REHABILITATION

Proper care after diagnosis and repair of an extensor tendon injury is extremely important for optimal patient outcome. Even the best initial tendon repair can have a poor result if subsequently treated improperly. The rehabilitation of tendon injuries has evolved since 1980 to include dynamic splinting and active range-of-motion exercises to obtain maximal motion of the affected digit.

Zone 1 and 2 injuries are usually treated with static splinting as described previously. After 6 weeks, active range-of-motion exercises should begin. Night splinting is recommended for an additional 2 weeks.[19] [22] Some authors also recommend wearing the splint during the day between exercises.[1] It is advisable to give the patient a number of extra splints so that the patient (or family) can change the splint

Figure 49-22 A proper mallet finger splint. Either a commercially available volar plastic splint (Stack mallet finger splint) *(A–C),* or a volar or dorsal aluminum foam splint may be used *(D and E)*. The splint should allow easy motion of the proximal interphalangeal joint. *(Adapted from Doyle JR: Extensor tendons—Acute injuries. In Green DP [ed]: Operative Hand Surgery, 4th ed. New York, Churchill Livingstone, 1999, p 1967.)*

frequently to avoid pressure injury. During splint changes, it is important that the DIP joint be held in full extension either by using the other hand or by placing the finger against a table. If an extension lag develops at any time, continuous splinting must be repeated. Closed injuries of the central slip (zone 3) are often treated with a boutonnière splint for 4 weeks, followed by 2 weeks of gradual flexion exercises and night splinting. During the initial 4 weeks, the patient should be instructed to passively flex the DIP joint every hour to maintain gliding and proper position of the lateral bands.

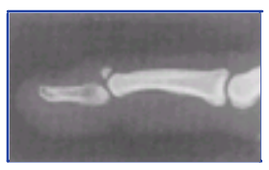

Figure 49-23 A mallet fracture with volar subluxation of the distal phalanx.

Figure 49-24 Swan neck deformity. *(Courtesy of Raymond G. Hart and Joseph E. Kutz.)*

Lacerations in zones 3 and 4 have traditionally been treated with static splinting from the forearm to digits. An alternative approach is to splint only the DIP and PIP joints in extension, and begin a "short-arc-motion" protocol within 1 to 2 days of repair.[3] This consists of active motion at the PIP joint progressing from 0° to 30° the first 2 weeks to 0° to 50° the fourth week. When compared to static splinting, this protocol may lead to better PIP and DIP joint flexion, without resulting in tendon rupture or boutonnière deformity. Dynamic extension splints are also proving to be useful for zone 3 and 4 tendon injury rehabilitation.[1] [3] [11]

Early motion following extensor tendon repair has been found to be most useful in zones 5 through 7. A dynamic extension splint is commonly used in which the wrist is extended 45°, and all finger joints rest in the neutral position (Fig. 49-25). A volar block allows 30° to 40° of MCP joint flexion, while a dynamic traction mechanism passively extends the digits. Dynamic splinting is started 1 to 3 days following repair. Active motion is added at 3 to 4 weeks, and resistance is added at 7 weeks. A short-arc-motion protocol with controlled active motion at the MCP joint has also been shown to be safe and effective when started 24 to 48 hours after repair.[3] One comparative trial reported that dynamic extension splinting and controlled active mobilization worked equally well for zones 5 and 6 tendon injuries.[12] Both protocols are most beneficial when managed closely by a skilled hand therapist. Patients must be reliable and motivated to take advantage of these techniques. It is best to refer patients to a hand surgeon or hand therapist as soon as possible following repair so that rehabilitation can begin in a timely manner.

EXTENSOR TENDON INJURIES OF THE FOOT

The extensor tendons of the foot are less commonly injured than the extensor tendons of the hand and wrist. The most important extensors of the foot and ankle that may be presented with injuries in the ED are the tibialis anterior, extensor hallucis longus, and extensor digitorum longus tendons.

The tibialis anterior muscle originates on the shaft of the tibia and interosseous membrane and inserts on the medial cuneiform and the base of the first metatarsal. The tibialis anterior extends the foot at the ankle joint and inverts the foot at the subtalar and transverse tarsal joints. Spontaneous rupture of the tibialis anterior tendon may be seen in both elderly and young patients who have been injured during athletic activity. Injury to this tendon commonly results from forceful attempted dorsiflexion of the ankle while it is held fixed in the

Figure 49-25 A dynamic extension splint allows for early motion following extensor tendon repair. Elastic retraction maintains the fingers in extension. Excursion of the repaired tendon is achieved by active flexion. *(Adapted from Doyle JR: Extensor tendons—Acute injuries. In Green DP [ed]: Operative Hand Surgery, 4th ed. New York, Churchill Livingstone, 1999, p 1957.)*

plantar-flexed position.[18] Patients generally present with decreased strength of foot dorsiflexion, because toe extensors are used to accomplish this motion. Ruptures or lacerations of the tibialis anterior tendon should be referred promptly to an orthopedic surgeon for consideration of formal operative repair.

The extensor digitorum longus (EDL) and extensor hallucis longus (EHL) tendons both originate from the shaft of the fibula and interosseous membrane. The EHL tendon inserts into the base of the distal phalanx of the great toe, while the EDL tendon divides into four branches that insert on toes 2 through 5 (Fig. 49-26). Both the EHL and EDL tendons primarily result in extension of the toes and dorsiflexion at the ankle. The extensor digitorum brevis (EDB) and extensor hallucis brevis (EHB) muscles originate from the upper part of the calcaneus. The EHB tendon joins the lateral aspect of the EHL tendon prior to inserting on the great toe. The EDB muscle gives rise to three tendons that join the lateral side of the EDL tendons going to toes 2 through 4 (see Fig. 49-26).

Injury to the EHL and EDL tendons may result from a sharp object lacerating the dorsum of the foot. Patients may present with weakness of, or an inability to extend, the involved toe. The examiner may be unable to palpate the injured tendon. Whether one should repair EHL or EDL tendon lacerations is controversial. However, many authors favor repair; failure to repair EDL tendons may result in a claw deformity of the adjacent toes. [4] Lacerations of EHL and EDL at the level of the ankle are usually repaired, while lacerations on the dorsum of the foot and the toe are managed variably. If the patient has significant pain or has any flexion deformity of the involved toe, one should probably repair the lacerated tendon. Repair is also favored when both tendon ends are easily visualized in the wound, and the patient is willing to undergo

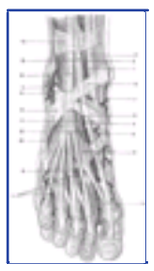

Figure 49-26 First layer of the dorsum of the foot and ankle. *1,* anterior tibial artery; *2,* anterior medial malleolar artery; *3,* anterior lateral malleolar artery; *4,* dorsalis pedis artery; *5,* first dorsal metatarsal artery; *6,* arcuate artery; *7,* dorsal metatarsal arteries; *8,* medial tarsal artery; *9, 10,* deep peroneal nerve; *11,* motor nerve branch to extensor digitorum brevis; *12,* inferior extensor retinaculum; *13,* superomedial band of inferior extensor retinaculum; *14,* inferomedial band of inferior extensor retinaculum; *15,* superolateral band of inferior extensor retinaculum; *16,* superior extensor retinaculum; *17,* tibialis anterior tendon; *18,* extensor hallucis longus tendon; *19,* extensor digitorum longus tendon; *20,* extensor digitorum brevis muscle to toes; *21,* extensor hallucis brevis muscle. *(From Sarrafian SK: Functional Anatomy of the Foot and Ankle: Descriptive, Topographic, Functional, 2nd ed. Philadelphia, JB Lippincott, p 423.)*

prolonged postrepair immobilization.[20] Because management of these injuries is controversial, it is advisable to discuss the care of these patients with the consulting orthopedic surgeon. Extensor tendon repair of the foot is usually not performed in the ED setting. Superficial cutaneous nerves are easily injured on the dorsum of the foot during wound exploration, which

can lead to the formation of a chronic, painful neuroma. If the injury is repaired in the ED, the technique for repair is similar to that used for the dorsum of the hand (zone 6). A posterior splint that includes the toes should be applied following tendon repair. The ankle should be splinted at 90° with the toes in the neutral position (see Chapter 51).

FLEXOR TENDON INJURIES

Flexor tendon injuries are more difficult to diagnose and more challenging to treat than extensor tendon injuries. In general,

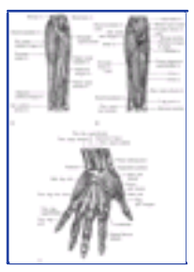

Figure 49-27 Flexor tendons of the right forearm, wrist, and hand. *A,* Superficial forearm layer. *B,* Deep forearm layer. *C,* Left wrist and palm. Proper function of the flexor tendons requires defect-free tendons and smooth tendon sheaths for easy gliding, and an intact pulley system; hence, flexor tendon lacerations are seldom repaired in the ED. *(Modified from Pansky B: Upper extremity. In Pansky B [ed]: Review of Gross Anatomy, 4th ed. New York, Macmillan Publishing, 1979, pp 219, 243.)*

ED repair of flexor tendons is not performed by emergency clinicians. Anatomic and biomechanic issues, the physiology of flexor tendons and tendon healing, and follow-up rehabilitation/physical therapy issues are complex and formidable. A satisfactory outcome of an injured flexor tendon is more difficult to achieve than is a similar degree of injury to an extensor tendon. Unlike extensor tendons, flexor tendons are influenced by a number of pulley mechanisms. The tendon must glide through delicate tendon sheaths, so even a minor defect in tendon integrity is physiologically magnified (Fig. 49-27). In addition, flexor tendon injuries are often associated with nerve and vascular injuries.

Figure 49-28 Deep puncture wounds of the palm may injure flexor tendons. The depth of this wound precludes extensive exploration to visualize the tendon. Partial tendon lacerations may still initially allow full function. Clues to a partial tendon laceration include weakness of flexion or pain with attempts at flexion against resistance, but many partial lacerations are clinically silent. Despite full function this wound's location and depth suggest the possibility of at least a partial tendon injury. The prudent course would include meticulous wound care, splinting, skin closure, and contact with a hand specialist to arrange re-examination in a few days, while cautioning the patient that further care may be required.

The main clinical mandates for emergency clinicians are to diagnose, or consider, flexor tendon injuries; provide initial proper wound care; and expedite appropriate consultation and follow-up. Unlike the more superficial extensor tendons, flexor tendons are often buried deep within the hand and forearm, and it is often not possible to readily visualize the tendon in the recesses of a wound. Puncture wounds of the palm often injure flexor tendons, but deep puncture wounds prohibit visualization of the injured structures (Fig. 49-28). Therefore, a partial flexor tendon injury may be totally

Figure 49-29 *A,* The flexor digitorum profundus tendon is examined by immobilizing the digit in question and asking the patient to flex the distal interphalangeal joint against resistance. *B,* The flexor digitorum superficialis tendon is examined by immobilizing the digits not being tested and asking the patient to flex the proximal interphalangeal joint against resistance. Pain and weakness associated with flexion against resistance may suggest a partial tendon laceration, but this is often a very subtle or inaccurate evaluation that must be repeated when pain and swelling have subsided.

clinically silent until rupture occurs days or weeks later. Delayed repair of undiagnosed flexor tendons may be complicated by tendon retraction or scar formation, and tendon transfer/grafting may be necessary.

It may not be possible for the emergency clinician to diagnose the presence of all flexor tendon injuries, nor the full extent of such injuries, on the initial visit. Help may be obtained from a specialist if logistically possible, but generally there is no mandate for such immediate on-site examination when there are questions about tendon integrity. While consultation is advised prior to definitive disposition, the same limitations in the examination would similarly confront a specialist. Individual scenarios and local protocols will guide the timing and degree of consultation in the ED.

Complete flexor tendon injuries are often readily apparent on physical examination, either by individual tendon testing or by the resting posture of the injured hand. Partial tendon lacerations are commonly clinically unappreciated since no functional deficit is evident. Clinical clues to a potential flexor tendon injury are weakness of flexor tendon function (difficult to evaluate in the acutely injured extremity), pain at the site of injury when performing active range of motion against resistance, or an abnormal resting posture of the hand (Fig. 49-29). However, the clinician may not ever be able to arrive at a complete or accurate diagnosis without surgical exploration. *It is counterproductive, and potentially harmful, to attempt extensive exploration of the deep recesses of the hand or forearm in the ED merely to visualize a suspected flexor tendon injury.*

Completely transected flexor tendons are surgically repaired by a consultant, usually on an elective basis. Most hand surgeons are reluctant to perform a primary repair of a flexor tendon injury on ED patients, and prefer to have the wound cleaned, the skin closed, and the patient schedule a subsequent definitive repair. The final outcome of flexor tendon surgery depends on multiple factors; however, surgical repair of most flexor tendons that is accomplished within 10 to 21 days of injury (delayed primary repair) generally

manifests final outcomes similar to immediate repair.[6] [21] [24] Therefore, if a partial tendon laceration is not diagnosed on the initial visit, and rupture is noted at the time of skin suture removal or wound check, immediate referral to a hand surgeon would be expected to produce a similar result as expected had the injury been diagnosed at the time of injury.

Partial flexor tendon lacerations, if appreciated, are usually treated by careful wound cleaning, skin closure, splinting, and referral for reevaluation in 1 to 5 days. The definitive treatment of partial lacerations remains quite controversial. Some surgeons will explore partial tendon lacerations. There is some experimental evidence that surgical repair of partially lacerated tendons results in weaker tendons than if the tendons were not surgically repaired. [28] To further complicate the issues, some authors suggest no suturing and early mobilization of lacerated tendons of 25 to 95% cross-sectional area. [27] As a guideline, tendon lacerations of greater than 50% cross-sectional area are often sutured with specific surgical techniques, 25 to 50% lacerations are repaired with simple or special sutures, and injuries of less than 25% are trimmed to promote normal gliding function.[24] All decisions concerning the type and timing of repair should be made in concert with a consultant. Some decisions concerning surgical repair of partial injuries cannot be made for weeks or months.

Following evaluation of a known or suspected flexor tendon injury, the skin is sutured and the hand splinted to protect the tendon and minimize retraction. Techniques vary, but splinting with the wrist in 30° of flexion, the MCP joints in 70° of flexion, and the interphalangeal joints flexed at 10 to 15% has been recommended. [8] There are no data to support or refute the value of prophylactic antibiotics in any soft tissue injury that has been properly cleaned. Although no definitive standard of care is promulgated, many clinicians prescribe 3 to 5 days of antibiotics effective against gram-positive organisms (including *Staphylococcus aureus*) if the tendon is injured.

Antibiotics are recommended if the degree of contamination is significant, there has been delay in cleaning, there are unusual sources of injury, or if the patient is immunocompromised. Specific written instructions, *with a definite follow-up time frame outlined,* and help with patient referral will likely improve final outcome, but flexor tendon injuries often produce life-long disability despite even ideal care in the ED.

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Chapter 50 - Management of Common Dislocations

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Robert McNamara

Joint dislocations are frequently encountered among patients presenting to the emergency department (ED). They can range from a simple finger injury to limb- or life-threatening consequences of high-energy trauma. Although the dislocated joint is most often clinically obvious, the presentation may be obscure or masked by other injuries. Emergency clinicians must be capable of detecting and managing these injuries; appropriate timely referral to a consultant is generally required for complex dislocation injuries.

This chapter addresses the diagnosis and management of joint dislocations. Keys to the clinical assessment and radiographic evaluation of these injuries are discussed along with methods of reduction. The emphasis of the chapter is on simple dislocations that should be diagnosed and generally managed in the ED. Fracture-dislocations that commonly require operative intervention and emergency orthopedic consultation are not discussed.

PREPARATION OF THE PATIENT

Although many authors claim their reduction method is well tolerated without premedication, they generally have not quantitatively measured the discomfort of their patients.[1] [2] [3] [4] [5] There are no rigid generally accepted guidelines for the use of pharmacologic adjuncts in the management of dislocations. Each patient and presentation is unique and the treating clinician must use judgment as to whether premedication is required, which agent or agents to use, and what dose to give. *In general, the editors suggest the judicious use of analgesia/sedation for the majority of reductions performed in the ED.* The calm, cooperative patient may tolerate gentle reduction attempts of a major joint such as the shoulder, but even the most stoic of patients may be quite uncomfortable with the manipulations necessary for reduction of a dislocated finger. A radial head dislocation in a child is usually easily accomplished without analgesia; however, the reduction of a hip dislocation is rarely successful without a significant amount of anesthesia/analgesia. Attempting any reduction technique in an extremely anxious patient without premedication will generally frustrate the operator and further upset the patient, and it may hinder a successful outcome. When multiple attempts are required, and significant force must be exerted due to muscle spasm or an uncooperative patient, there is additional chance of producing complications during the reduction.

Verbal techniques for alleviating anxiety and discomfort are not to be discounted as they can be of great assistance during joint reduction. In field settings, simple hypnosis techniques have been successfully used for major joint dislocations.[6] In the ED, verbal reassurance and distracting conversation are useful adjuncts.

In most circumstances, analgesia or sedation of some sort, or both, will be required; generally the intravenous (IV) route for drug administration is the method of choice, as it allows for rapid relief of patient discomfort and facilitates repetitive dosing for titration to the desired effect. Chapter 34 provides an in-depth discussion of procedural sedation and analgesia for a wide variety of ED procedures, including joint reduction. Alternatives to procedural sedation and analgesia include intra-articular injection of lidocaine, hematoma blocks (see Chapter 30), peripheral nerve blocks (see Chapter 32), and regional anesthesia (see Chapter 33).

GENERAL PRINCIPLES

The clinical assessment of the patient with a dislocation must include a search for other injuries, especially if the mechanism was of high energy. This is generally most important for hip, knee, and posterior sternoclavicular dislocations. For all dislocations, a detailed extremity neurovascular examination should be conducted and appropriately documented prior to focusing attention on the injured joint.

Although many dislocations are clinically obvious, some may escape detection for some time while other injuries or issues dominate the clinical picture. A knee dislocation may be quite obvious in a 170-pound man who displays a deformity of the knee, but in a 400-pound obese patient, the knee may look deceivingly normal on first glance. The history and mechanism of injury can be quite helpful in certain circumstances. For example, a painful shoulder joint in a seizure patient should prompt assessment for a posterior shoulder dislocation, whereas a history of the knee striking the dashboard will clue one to the potential for a hip dislocation. Some dislocations will have been reduced prior to clinician assessment. A careful history will uncover these injuries and prompt the necessary assessment of the ligamentous integrity of the joint and guide proper immobilization and follow-up care. A dislocated, then spontaneously reduced, knee has escaped detection by even the seasoned clinician's initial evaluation. Other dislocations that commonly present in a reduced state include finger dislocations, knee dislocations, patellar dislocations, and radial head subluxations.

Although the chance of a gentle reduction attempt causing a fracture or neurovascular injury is extremely low, *careful evaluations before and after reduction, as well as documentation of the neurovascular status, are prudent*. Often the initial pain of the dislocation is distracting, and paresthesias or a weak pulse may not be readily apparent until the joint has been replaced. When the integrity of the pulse is in question, the blood pressure at the wrist or foot may be compared to the uninjured extremity, or a pulse oximeter may be applied to the distal fingers (Fig. 50-1). *Prereduction radiographs of dislocated joints are generally recommended.* Reasons for this include the difficulty in distinguishing a fracture—dislocation by clinical examination and the potential for medicolegal problems if the fracture is not identified prior to reduction attempts. More important, certain associated fractures predict a poor outcome from closed reduction and make orthopedic consultation a consideration prior to such attempts. The obvious exceptions to this rule include suspected radial head subluxation in young children, clinical circumstances in which radiographs are not readily available (e.g., in the wilderness), minimally symptomatic patients with recurrent shoulder dislocations with a history of minor to no trauma, and clinical conditions (i.e., vascular compromise or

Figure 50-1 Significant vascular injuries from dislocations, such as the knee, are usually obvious, but some reduction in distal circulation may be subtle due to partial vascular compromise that heralds subsequent ischemia. The standard techniques to assess vascular injury are assessing the strength of the pulse and capillary refill, but other techniques may be helpful. While these procedures are neither well studied nor quantified, taking the blood pressure distal to the injury with a cuff and Doppler (shown here) or applying a pulse oximeter distal to the injury and comparing the results to the uninjured extremity may give some helpful clues to underlying vascular injuries. Calculating a brachial/ankle blood pressure index (see Chapter 1) is also more accurate than simple palpation.

threatened skin penetration) that dictate the need for immediate reduction. Some also question the need for prereduction films in certain patients with anterior shoulder dislocations.[7] [8] *The editors strongly suggest postreduction films in virtually all patients who have had a dislocation reduced in the ED.* Although postreduction radiographs are traditionally obtained, the need for this in a clinically obvious successful shoulder joint relocation also has been questioned. [8] [9] Although postreduction films are often not clinically useful and may not be cost-effective, they are recommended as prudent clinical practice and for medicolegal purposes.

Patients who have received sedatives and opioids may not remember the actual successful reduction or the immediate postreduction period. A reinjury after release from the ED without radiographic corroboration of a successful reduction can raise questions about the adequacy of the initial procedure. Occasionally a fracture is detected on postreduction radiographs that was not obvious on the initial films, or a previously noted minor fracture may be found to reside in an intra-articular location.

The proper terminology for dislocations describes the relationship of the distal (or displaced) segment relative to the proximal bone or the normal anatomic structure. The terms *anterior* and *posterior* are used in most dislocations. Therefore, if the head of the humerus lies anterior to the glenoid fossa, the injury is an *anterior shoulder dislocation*. Similarly, if the olecranon lies behind the distal end of the humerus, the injury is a *posterior elbow dislocation*. In the hand, wrist, and foot, one uses the terms *dorsal* and *volar. Palmar* and *plantar* are sometimes used in place of volar to describe the position of the dislocated part. Dislocations can be open or closed and may have associated fractures requiring separate description.

It is generally accepted that the sooner a dislocation is reduced, the better. This alleviates the patient's discomfort and corrects the distortion of surrounding soft tissue structures. In some studies the success rate of relocation (reduction) is higher when attempted closer to the time of injury. [2] However, there is no reason to forego an attempt at a closed reduction due to "old injuries" in the vast majority of dislocations. Chronic dislocations of several days, weeks, or more are often difficult to reduce in a closed manner, but such presentations are infrequent.

General points about the reduction itself include the need for patience on the operator's part and the avoidance of excessive or abrupt applications of force. Gentle and gradual application of the various reduction techniques lessen the risk of complications. The operator must clearly understand the technique to be applied, and one should not hesitate to review a description of the procedure on a regular basis. Review of the technique is most important for uncommon dislocations, but it is a good habit even for the more common dislocations performed by seasoned clinicians.

A certain percentage of all types of dislocations are not amenable to closed reduction. Inability to complete a closed reduction is generally a result of the interposition of soft tissue structures or fracture fragments and not necessarily due to improper technique. If one has achieved sedation/analgesia adequate to permit relaxation of the patient's muscle tone, reduction should be relatively straightforward. When reduction under adequate sedation/analgesia is unsuccessful, multiple attempts at closed reduction are inappropriate. Generally, orthopedic consultation should be considered after two failed attempts.

Once an attempt at reduction is completed, the operator should recheck the neurovascular status that was documented before the reduction was performed. For the elbow, hand, and forefoot joints, passive range of motion is performed to assess the stability of the reduction and to ensure a smoothly gliding joint that is free of intra-articular obstruction. In addition to close monitoring of the medicated patient, proper aftercare involves adequate immobilization of the injured joint for comfort and to prevent repeat dislocation. Recommendations for follow-up care are dependent on the injury and its severity.

Timing of Reductions

Questions often arise concerning the necessity of immediate reduction vs delayed reduction, with the clinician fearing disastrous neurovascular consequence if a dislocation is not manipulated immediately upon arrival. In reality, there is rarely an instance where some prereduction radiographs, even portable films, cannot be obtained prior to treatment. Even if the pulse is weak, or the fingers are numb, a few minutes' delay is usually acceptable in order to gain important radiographic information on the type of fracture and for documentation for the follow-up clinicians. Important clinical information may be difficult to obtain or the specific initial injury may be impossible to reconstruct once the joint has been reduced (Fig. 50-2). Of equal importance, dislocation with concomitant neurovascular injuries should be

reduced with the least amount of trauma possible, often requiring a

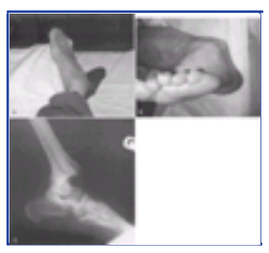

Figure 50-2 *A* and *B*, Because the distal pulse is weak and the toes are numb, it may be tempting to immediately reduce these obvious dislocations while the patient is still on the ambulance stretcher. Some clinicians prefer to first obtain proper analgesia/sedation to allow a less traumatic reduction. *C*, Prereduction radiographs (even limited-view portable films are acceptable) can be helpful since once the reduction is accomplished, the specific initial injury may be impossible to reconstruct from the physical examination alone. The few minutes required to properly prepare the patient for reduction and to document the initial injury should not result in a more adverse outcome than has been prognosticated by the initial injury. However, when the patient has sustained multiple trauma and extremity films are a low priority, early reduction without radiographs may be warranted.

few minutes for the induction of analgesia/sedation, a time during which radiographs can be obtained. *If a vascular or neurologic abnormality is documented prior to reduction, the joint should be reduced by the most timely and least traumatic procedure available.* Each case should be handled individually, considering the specific injury, available resources, and experience of the clinician. Although multiple unsuccessful or forceful attempts at reduction in the ED should be avoided with all dislocations, this is especially important if there is vascular or neurologic compromise. Occasionally, the more prudent course is reduction under general anesthesia, but this decision must be analyzed given the availability of consultation and other resources.

This chapter covers dislocations of the various joints with the exception of wrist dislocations, which are complex and require orthopedic consultation, and temporomandibular joint dislocations, which are discussed in Chapter 65 . Assessment and management principles, including reduction methods, are presented and aftercare is discussed.

SHOULDER DISLOCATIONS

The human shoulder joint is remarkable for its degree of possible motion. The anatomic features that allow for this mobility, however, contribute to its instability. The glenohumeral joint has the greatest range of motion of any joint in the body, largely due to the loose joint capsule and the shallow nature of the glenoid fossa. [10] Posterior dislocation is uncommon, largely due to the anatomic support of the scapula and the thick muscular support in this area. The anterior support is less pronounced, with the inferior glenohumeral ligament serving as the primary restraint to anterior dislocation. [11] The depth of the glenoid fossa is somewhat increased by the fibrocartilaginous glenoid labrum, which forms the rim of this structure.

Most shoulder dislocations are anterior (i.e., the humeral head becomes situated in front of the glenoid fossa). Posterior dislocations are the next most common, but they generally account for less than 4% of shoulder dislocations. [12]

Uncommon variations include inferior (luxatio erecta), superior, and intrathoracic dislocations. Dislocations of all types, including the shoulder, are less common in children due to the relative weakness of the epiphyseal plate as compared to the ligamentous support of the joint.

Anterior Shoulder Dislocations

Anterior dislocations of the shoulder are the most common major joint dislocation encountered in the ED. The usual mechanism of injury is indirect, with a combination of abduction, extension, and external rotation. [10] [11] Only rarely is the mechanism a direct blow to the posterior aspect of the shoulder. Occasionally, especially with recurrent dislocations, the mechanism is surprisingly minor, such as mere external rotation of the shoulder while rolling over in bed or raising the arm overhead. The occurrence of a first dislocation at a younger age is associated with a higher recurrence rate; 80% to 92% with a first dislocation before age 20 years vs 10% to 15% in patients with a first dislocation after age 40 years. [10] Rotator cuff injuries, however, occur more frequently in older patients with anterior shoulder dislocations. [13]

The 4 types of anterior dislocations are subcoracoid (accounting for >75% of anterior dislocations), subglenoid, and the uncommon subclavicular and intrathoracic. [10] These are classified according to where the humeral head comes to rest (Fig. 50-3).

Clinical Assessment

The presentation of anterior shoulder dislocation is usually obvious (Fig. 50-4). Posterior dislocations are more subtle on both clinical presentation and radiographic manifestations, and can be misdiagnosed as a severe contusion (Table 50-1). The patient supports the injured extremity and leans toward the injured side, holding the arm in *abduction* with slight external rotation. The patient cannot *adduct* or internally rotate the shoulder. Visual inspection reveals loss of the rounded appearance of the shoulder due to the absence of the humeral head beneath the deltoid region. The acromion is prominent and an abrupt drop-off below the acromion can be seen or palpated. An anterior fullness in the subclavicular region is visible in thinner individuals and is easily palpable in most others. Comparison to the uninjured side is a useful aid for both visual examination and palpation. Any attempt at internal rotation is quite painful and is resisted by the patient. The inability to place the palm from the injured extremity on the uninjured shoulder is consistent with anterior shoulder dislocation; postreduction, this maneuver should be possible.

A careful assessment of the neurovascular status of the affected extremity is essential. Injury to the axillary artery is rare, usually occurring in the elderly [13] and can be quickly assessed by palpation of the radial pulse or the presence of an expanding hematoma. It is important to assess the status of the axillary nerve, as this is the most common nerve lesion resulting from anterior dislocations. [14] The sensory component of the axillary nerve is assessed by testing for sensation over the lateral aspect of the upper arm (Fig. 50-5). The motor component of the axillary nerve would be tested by assessing the strength of the deltoid muscle, a difficult undertaking in the patient with a dislocated shoulder. Less commonly, the brachial plexus may be injured by a stretch injury, producing variable nerve deficits. The neurologic examination should include a complete assessment of all major nerves to the arm,

Figure 50-3 Types of anterior dislocations. These types of anterior dislocations should receive the same treatment. *A,* Subglenoid dislocation (rare type). *B,* Subcoracoid dislocation (most common type). *C,* Subclavicular dislocation (rare type). *(From DePalma AF: Management of Fractures and Dislocations: An Atlas. Philadelphia, WB Saunders, 1970, p 617. Reproduced by permission.)*

as other nerve injuries such as to the ulnar and radial nerve may occur. [14] The presence of a neurologic deficit does not preclude closed reduction, *but in the presence of a nerve injury, multiple forceful attempts at reduction should be avoided*. Brachial plexus injuries require an especially atraumatic reduction. If generous sedation/analgesia does not permit an easy reduction in the ED, reduction of the dislocation with a nerve injury may be more prudently performed in the operating room under general anesthesia. Nerve injuries in this setting generally have a good prognosis, but the patient should be informed of the findings and the need for follow-up. Symptoms may require many months to resolve.

The rare vascular injuries, such as axillary artery disruption, are usually quite obvious, producing dysesthesias and coolness of the involved arm. An expanding axillary hematoma, pulse deficit, peripheral cyanosis, and pallor can be seen. Collateral circulation may produce a faint pulse in the extremity, so comparison blood pressure of the uninjured side may be helpful. Specific lesions include complete disruption, linear tears, or thrombus. Axillary artery injuries can occur in all ages, although they are more prominent in the

Figure 50-4 *A*, Typical presentation of an anterior right shoulder dislocation. The shoulder is very painful; thus, the patient resists movement. The outer round contour of the shoulder is flattened, and the displaced humeral head may be appreciated in the subcoracoid area. Often the patient abducts the arm slightly, bends the torso toward the injured side, and supports the flexed elbow on the injured side with the other hand. *B*, Another example of an obvious left shoulder dislocation. This chronic dislocation occurred frequently with minimal trauma, in this case from rolling over in bed.

elderly. The artery is at risk with anterior dislocations, and a dislocation-spontaneous reduction can produce the injury. Arteriography with surgical repair of the artery is required, occasionally with fasciotomy of the forearm if ischemia is longstanding. [15]

Some portion of the rotator cuff will be injured in many shoulder dislocations. Rotator cuff tears are easier to evaluate after reduction, often days later when pain and swelling have subsided.

Radiologic Examination

Associated fractures are detected in 15% to 35% of anterior shoulder dislocations, with fractures of the greater tuberosity being the most common. [10] The presence of a fracture of the greater tuberosity does not change the initial management of anterior shoulder dislocations, and these fractures usually heal well after closed reduction in the routine fashion. [10] The Hill-Sachs deformity, a sign of repeated dislocations, produces a groove in the posterolateral aspect of the humeral head and

TABLE 50-1 -- Comparison of Anterior and Posterior Shoulder Dislocations: Classified According to the Displacement of the Humeral Head

Type of Dislocation	Patient Presentation	Other Clinical Clues	Radiographs
Anterior	• Arm held in *abduction* and *slight external rotation* (abduction more prominent in subglenoid dislocation)	Seen from the front, shoulder appears "squared off"	*On AP view*: obvious dislocation
99% subcoracoid and subglenoid			
Humeral head is *anterior* to the glenoid		Distal acromion prominent from side view	*On lateral or "Y" view*: humeral head appears *anterior* to glenoid fossa
	• Patient cannot adduct or *internally* rotate shoulder		
Posterior	• Arm held in sling position, with adduction and internal rotation	Coracoid process prominent, glenoid fossa empty anteriorly and humeral head bulging posteriorly	*On AP view*: vacant glenoid sign, 6-mm sign, light bulb sign
95% subacromial			
5% subglenoid and subspinous			
Humeral head is *posterior* to the glenoid	• Attempts at *abduction* and *external rotation* cause extreme pain		*On lateral or "Y" view*: humeral head appears *posterior* to glenoid fossa

may be seen on prereduction or postreduction films (Fig. 50-6). It is caused by impaction of the humeral head against the glenoid rim after dislocation. It rarely has clinical significance, but may result in a loose body within the joint. [13] Impaction of the humeral head against the glenoid during dislocation may cause a disruption of the glenoid rim, known as a "Bankart lesion." This has been implicated as one cause of recurrent dislocations, but does not affect immediate ED management. [13]

Fractures of the humeral neck are frequently displaced with attempts at closed reduction, the result of which is often avascular necrosis of the humeral head. [16] The fact that humeral neck fractures are a known complication of shoulder relocation [10] suggests the value of prereduction radiographs in anterior shoulder dislocations. However, some argue that *clinically obvious recurrent dislocations and clinically obvious anterior dislocations without a blunt trauma injury mechanism* (information usually offered by the patient) can be reduced without prior radiographs, as fracture is quite unlikely in these situations. [7] [8]

Figure 50-5 Evaluation of the upper extremity with a shoulder dislocation. Axillary (circumflex) nerve palsy is the most common neurologic complication. The axillary nerve has a sensory and motor function. Test the integrity of the nerve by assessing sensation to pin prick (*1)* in its distribution over the "regimental badge" area. (The shoulder is usually too painful to allow assessment of deltoid activity with certainty.) Look for other (rare) involvement of the radial portion of the posterior cord (*2)* and involvement of the axillary artery (*3)*. *(From McRae R: Practical Fracture Treatment. Edinburgh, Churchill Livingstone, 1981, p 84. Reproduced by permission.)*

Anterior dislocations are not subtle on the routine anteroposterior (AP) radiograph, and this view detects the most important fracture to identify, that of the humeral neck. An adequate AP view, when combined with the typical clinical examination, allows for successful management of most anterior shoulder dislocations. The true AP view of the shoulder is taken at a right angle to the scapula, requiring rotation of the patient to 30 to 45° as shown in Figure 50-7 .

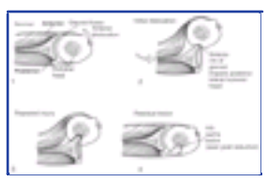

Figure 50-6 With repeated anterior shoulder dislocations, a Hill-Sachs lesion may form. During the dislocation the humeral head is damaged by the sharp anterior rim of the glenoid *(2)*. With repeated dislocation the lesion, called the "hatchet sign" develops *(3)*. On the reduction film the lesion is apparent *(4)*.

The typical lateral views obtained include the scapular Y view (Fig. 50-8), the transthoracic view, and the axillary view. These views rarely add to the AP in the obvious anterior dislocation, but they are of value in *posterior* dislocations. The usefulness of additional views in anterior shoulder dislocations is primarily to detect fractures, and the previously mentioned lateral views (especially the transthoracic view) are quite limited in this respect. [17] The apical oblique view has been found to be more valuable in acute shoulder trauma than the oblique scapular projection. [17] This view is obtained by angling the beam 45° caudad with the patient in a 45° oblique position (Fig. 50-9*A* and *B*).

Postreduction radiographs are obtained to document the success of the reduction. Occasionally they will reveal a fracture not detected on the prereduction radiographs.

Reduction Techniques

Hippocrates (450 B.C.) is generally credited with the first detailed description of reduction techniques, and it is believed that a drawing in the tomb of Upuy (1200 B.C.) is the earliest depiction of such a method. [10] The Hippocratic technique involves placement of the operator's foot in the axilla to effect countertraction. This technique is problematic and is not recommended by recent authors. [3] [11] Likewise, the Kocher method, which involves forceful leverage of the humerus, has an increased rate of complications and is generally discouraged in favor of other techniques. [10] [11]

This section discusses several methods of reduction that are well studied, proven to be safe, and easy to master. Regardless of the reduction technique used, gradual, gentle application of the technique is essential. Although all of the techniques discussed are generally acceptable and many authors state that their techniques are quite painless, [1] [2] [3] [4] [5] few studies have quantified the actual pain reported by patients. [18] McNamara found that scapular manipulation was generally well tolerated; 62% of patients not receiving premedication reported no or only mild pain during the reduction.

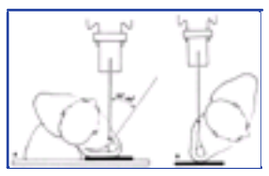

Figure 50-7 Trauma series includes two views of the shoulder made perpendicular and parallel to the scapular plane. This provides an anteroposterior (*A*) and a scapular Y (*B*) view. The advantage is that roentgenograms may be obtained without moving the patient or removing the arm from the sling. *(From Heppenstall RB: Fracture Treatment and Healing. Philadelphia, WB Saunders, 1980, p 374. Reproduced by permission.)*

Additionally, pain ratings were not lessened in the premedicated group. [18] As noted previously, intra-articular lidocaine also may be used to reduce the pain of reduction (Fig. 50-10). In studies by Matthews and Kosnick, the use of intra-articular lidocaine was found to offer significant pain relief during reduction of anterior shoulder dislocations, making it a useful alternative to procedural sedation and analgesia. [19] [20] Note that 10 to 20 mL of 1% lidocaine has been used with the intra-articular technique, and 15 to 20 minutes postinjection is

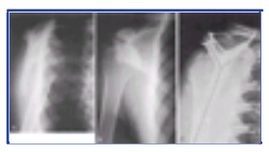

Figure 50-8 In the trauma series, a lateral view of the scapula (also called scapular Y view) demonstrates the head of the humerus displaced inferiorly and medially, the most common position for an anterior dislocation *(A)*. An anterior dislocation is shown on the anteroposterior projection *(B)*. A posterior dislocation: Transscapular projection showing the dislocated humeral head, posterior in relationship to the intersecting limbs of the Y *(C)*. *(A and B from Heppenstall RB: Fracture Treatment and Healing. Philadelphia, WB Saunders, 1980, p 392; C from Greenbaum E (ed): Radiology of the Emergency Patient. New York, John Wiley & Sons, 1982, p 512.)*

required to expect analgesia. Joint injection does not produce muscle relaxation, but it does obviate the need for intravenous access and prolonged observation. Operator judgment is an important part of the decision as to whether reduction should be attempted without premedication. The advantages of such an approach include the avoidance of potential complications from drug therapy, reduced staff requirements, and theoretically, a more rapid patient disposition. Certainly the patient who is markedly intoxicated may require little if any supplemental

Figure 50-9 *A,* Positioning for apical oblique view. The affected shoulder is placed at a 45° oblique position and the central ray is angled 45° caudad. The affected arm is adducted. *B,* Normal apical oblique view. *(A and B from Heppenstall RB: Fracture Treatment and Healing. Philadelphia, WB Saunders, 1980, p 392. Reproduced by permission.)*

sedative therapy. However, all patients who are more than mildly anxious or who are reluctant to cooperate with an attempt at reduction without medication, and those with a high degree of muscle spasm, should receive premedication. Generally, only one attempt is made and, if unsuccessful, reduction is attempted with the use of medication. When in doubt, it is best to use pharmacologic adjuncts (see Chapter 30 , Chapter 33 and Chapter 34).

Several factors will help decide which technique is best in each situation. One factor is whether the patient will tolerate a reduction attempt without sedation, as attempts without sedation should not use forceful techniques such as traction-countertraction. The comfort level of the clinician with a given technique is always a factor, as the greatest success rates will likely result from techniques with which the clinician is most familiar. The time and resources available to the clinician must be considered, as methods such as the Stimson maneuver require greater time and the availability of weights and straps. Additionally, certain reduction techniques can be performed without assistance, while others require an additional person to apply countertraction or to help with manipulation of the scapula or humeral head. Ideally, the emergency clinician should become familiar with a number of different techniques for reducing anterior dislocations of the shoulder, as no single method has a 100% success rate nor is any technique ideal in every situation.

Stimson maneuver.

The Stimson maneuver (Fig. 50-11) is a classic technique that offers the advantage of not requiring an assistant. The patient is placed prone on an elevated stretcher and about 2.5 to 5.0 kg (5 to 10 lb) of weight is suspended from the wrist. [10] [11] The weights can be strapped to the wrist, or a commercially available Velcro wrist splint can be placed and the weights hung from this with a hook. [21] The slow, steady traction of this method often permits reduction, but it may take 15 to 20 minutes. Reduction may be facilitated by gentle external rotation of the extended arm.

Variations of this method include the recommendation for flexion of the elbow to further relax the biceps tendon and the application of manual traction instead of weights. [22] [23] Rollinson allowed the arm to hang under its own weight after a supraclavicular block and reported a 91% success rate with usually no more than a gentle pull on the arm after 20 minutes in this position. [24] Each variation of the Stimson method can be used in combination with the scapular manipulation technique described later. Indeed, a success rate of 96% has been reported using the combined prone position, hanging weights, IV drug therapy, and scapular manipulation. [21]

Disadvantages of the Stimson method include the time required and the danger of patients slipping off the elevated bed. A "seatbelt" strap or bedsheet may be placed around the patient and stretcher to avoid patient movement off the stretcher. Additionally, a bed that elevates to a suitable height for the patient's arm length, a convenient method to hang the weights, the weights themselves, and adequate staff to monitor the patient are often difficult to locate and organize in a busy ED.

Scapular manipulation technique.

This method is popular due to its ease of performance, reported safety, and acceptability to patients. To date, no complications from this technique have been reported in the literature. [18] [21] [25] Shoulder reduction using this method focuses on repositioning the glenoid fossa rather than the humeral head, and it requires less force than other methods. [21] The success rate is high, generally > 90% in experienced hands. [21] [25]

The initial maneuver for scapular manipulation is traction on the arm as it is held in 90° of forward flexion. This may be performed with the patient prone and the arm hanging down as described in the Stimson method, with or without flexion of the elbow to 90° (Fig. 50-12*A*). Alternatively, this traction may be applied by the operator placing an outstretched arm over the seated patient's mid-clavicle while pulling the injured extremity with the other arm (Fig. 50-12*B*). Regardless of the

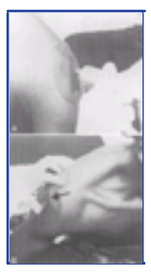

Figure 50-10 Intra-articular injection for the reduction of an acute anterior shoulder reduction. *A,* After aspirating blood from the joint, 10 to 20 mL of 1% plain lidocaine is slowly injected through the lateral sulcus, aiming slightly caudad. *B,* Anterior view. Allow 15 to 20 minutes for the lidocaine to take effect. *(From Matthews DE, Roberts T: Intraarticular lidocaine versus intravenous analgesic for reduction of acute anterior shoulder dislocations. Am J Sports Med 23:54, 1995. Reproduced by permission).*

means of arm traction, slight external rotation of the humerus may facilitate reduction by releasing the superior glenohumeral ligament and presenting a favorable

profile of the humeral head to the glenoid fossa. [26]

The prone patient position is recommended for those not familiar with the technique, as it facilitates identification of the scapula for manipulation (medial rotation of the tip). Nonetheless, the technique can be performed with the patient supine, given that the patient's shoulder is flexed to 90° and the scapula is exposed during gentle upward traction on the humerus. [27] Although seated scapular manipulation offers the advantage of not requiring the patient to go through the awkward and potentially uncomfortable assumption of the prone position, it is a technically more difficult variation of scapular manipulation. When placing the patient in the prone position it is important to place the injured shoulder over the edge of the bed to allow the arm to hang in a perpendicular manner for the application of traction. [25]

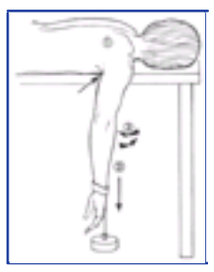

Figure 50-11 Stimson technique. This technique is often tried first, because it is the least traumatic if the patient can relax the shoulder muscles. *1,* The patient is lying prone on the edge of the table. One must be careful that the sedated or intoxicated patient does not fall off the table. Belts or sheets can be used to secure the patient to the stretcher. *2,* 5-kg weights are attached to the arm, and the patient maintains this position for 20 to 30 minutes, if necessary. *3,* Occasionally, gentle external and internal rotation of the shoulder with manual traction aids reduction. *(From DePalma AF: Management of Fractures and Dislocations: An Atlas. Philadelphia, WB Saunders, 1970, p 618. Reproduced by permission.)*

After application of traction, the scapula is then manipulated to complete the reduction. Anderson and coworkers recommend manipulation of the scapula after the patient's arm is relaxed [25] ; however, success is possible with no delay in the performance of this second step. [16] Manipulation of the scapula is carried out by stabilizing the superior aspect of the scapula with one hand and pushing the inferior tip of the scapula medially toward the spine (see Fig. 50-12*A*). The thumb of the hand stabilizing the superior aspect of the scapula can be placed along the lateral border of the scapula and used to assist the pressure applied by the thumb of the other hand. A small degree of dorsal displacement of the scapular tip is recommended as it is being pushed as far as possible in the medial direction. [25]

When the patient is properly positioned, with the affected arm hanging in a perpendicular fashion, the lateral border of the scapula may be difficult to find in larger subjects. This border is generally located quite lateral with the patient in this position, and it must be properly located prior to any reduction attempt. The reduction itself is occasionally so subtle that it may be missed by both the patient and the operator. A minor shift of the arm may be the only clue to the successful reduction. Careful palpation of the subclavicular area prior to repositioning the patient may be used to determine the success of the reduction.

External rotation method.

This method offers the advantage of requiring only one person and no special equipment. The technique requires no strength or endurance on the part of the operator. Additionally, it has been reported to be well tolerated by patients. The actual pain experienced by patients with this technique has not been quantified, but

Figure 50-12 Scapular manipulation technique. *A,* The inferior tip of the scapula is pushed medially and dorsally with the thumbs while the superior aspect of the scapula is stabilized with the fingers of the superior hand. Weights may be attached to the hand to apply hanging traction. *B,* While the patient is seated, the operator applies traction with one hand and countertraction with the other, while an assistant rotates the scapula in the same manner as in *A*. *(From McNamara RM: Reduction of anterior shoulder dislocations by scapular manipulation. Ann Emerg Med 22:1140, 1995. Reproduced by permission.)*

Plummer and Clinton state it can be performed with "little, if any sedation." [3]

In this technique the basic maneuver is *slow, gentle* external rotation of the fully adducted arm. In 1957, Parvin described a self-reduction external rotation technique in which the patient sits on a swivel-top chair and grasps a fixed post at waist height and slowly turns the body to enact external rotation. Parvin reported that the reduction usually takes place at 70 to 110° of external rotation. [28]

Since Parvin's initial study, this method has been described with the patient supine and the affected arm adducted tightly to the side of the patient. [1] [29] The elbow is flexed to 90° and held in the adducted position with the operator's hand closest to the patient. The other hand holds the patient's wrist and guides the arm into slow and gentle external rotation (Fig. 50-13). The procedure may require several minutes, because each time the patient experiences pain, the procedure is momentarily halted. Although the report of Mirick and colleagues mentioned using the forearm as "a lever," [1] a later description clearly recommends allowing the forearm to "fall" under its own weight. [3] No additional force should be applied to the forearm and no traction is exerted on the arm.

The end point of the reduction may be difficult to identify, as reduction is frequently very subtle. It is therefore recommended to continue the external rotation until the forearm is near the coronal plane (lying on the bed, perpendicular to the body), a process that usually takes 5 to 10 minutes. [3] If the patient notes persistent dislocation with full external rotation, steady traction at the elbow may be added at this time. Reduction may occasionally be noted when the arm is rotated back internally. [29] The success rate of this technique in 3 series performed by emergency clinicians was around 80%. [1] [29] [30]

Milch technique.

Proponents of this method praise its gentle nature, high success rate, lack of complications, and tolerance by patients. [2] [5] It can be described as "reaching up to pull an apple from a tree." The basic steps of this technique are abduction, external rotation, and gentle traction of the affected arm. Finally, if needed, the humeral head is pushed into the glenoid fossa with the thumb or fingers (Fig. 50-14).

Milch, in describing this technique, wrote that the fully abducted arm was in a natural position in which there was little tension on the muscles of the shoulder girdle. [31] He postulated that this was related to our ancestral "arboreal brachiation" (swinging from trees). The primary step in this technique is to have the affected arm abducted to an overhead position. Russell and coworkers had their patients raise the arm and put the hand behind the head as a first step. [26] Although this seems odd, patients can usually do this quite readily with little assistance and be quite comfortable in this position. Alternatively, the operator may abduct the arm by grasping the patient's arm at the elbow or the wrist. Lacey and Crawford found that the prone position, with the patient's shoulder close to the end of the bed, facilitated this step. [32]

Once the arm is fully abducted, gentle longitudinal traction is applied with slight external rotation. If reduction does not occur quickly, the humeral head can be pushed upward into the glenoid fossa using the thumb or fingers of the other hand. Beattie and associates reported a success rate of 70% with the Milch technique, [2] but others report success rates of = 90%. [5] [26]

Traction-countertraction.

This method is commonly used in the ED, largely out of tradition, as it has a high rate of success and many emergency clinicians are most comfortable with it. Familiarity is an advantage of this technique, but it requires more than one operator, some degree of force, and, occasionally, endurance. This technique is usually quite uncomfortable for the patient, and premedication is recommended prior to any attempt.

With the patient supine, a sheet or strap is wrapped around the upper chest and under the axilla of the affected shoulder (Fig. 50-15). An assistant holds this sheet so as to apply the countertraction. The operator's foot should *not* be used in the axilla to provide countertraction. Traction may then be applied to the extended arm,

but this generally results in operator fatigue, especially if the operator relies on biceps

Figure 50-13 External rotation method. No traction is applied and a slow, gentle approach is essential. *A,* Arm is adducted to the patient's side. In one hand, the elbow is held flexed at 90° while the other hand grasps the wrist. *B,* Slowly and gently, the forearm is used as a lever to rotate the arm externally. *C,* Usually by the time the forearm has reached the coronal plane, the shoulder will have been reduced. *(From Mirick MJ, Clinton J, Ruiz E: External rotation method of shoulder dislocation reduction. JACEP 8:529, 1979. Reproduced by permission.)*

strength to provide continuous traction. Preferably, the elbow of the affected side is flexed to 90° and a sheet or strap is wrapped around the proximal forearm and then around the operator's back. The bed should be elevated to a point at which the sheet can sit at the level of the operator's ischial tuberosities. This allows the operator to comfortably lean back and use the body weight to supply the force of traction, eliminating the possibility of operator fatigue. The portion of the sheet that is positioned on the patient's forearm has a tendency to ride up; flexion of the elbow beyond 90° will minimize this problem. Alternatively, the operator merely leans backward with the arms fully extended, again using the continuous weight of the body rather than the strength of the biceps to provide constant traction.

Once traction is applied, the operator must be patient, as the procedure may take a number of minutes to be successful. Inadequate premedication is noted by the patient who resists the procedure or is notably uncomfortable during the reduction attempt. The operator should not hesitate to order supplementary medications. Gentle, limited external rotation is sometimes useful to speed reduction. [10] Applying traction to an arm that is slightly abducted from the patient's body is often successful, but some operators prefer to slowly bring the arm medial to the patient's midline while maintaining traction or to have an assistant apply a gentle lateral force to the mid-humerus to direct the humeral head laterally. Successful reduction is usually presaged by slight lengthening of the arm as relaxation occurs, and a noticeable "clunk" may occur at the point of reduction. A brief fasciculation wave in the deltoid may also be seen at the time of reduction.

Spaso technique.

This technique was first reported by Spaso Miljesic as a simple, single operator technique that requires minimal force. [33] One published series reported an 87.5% success rate among premedicated patients when performed by junior house officers. [34] The patient is placed in a supine position and the operator grasps the affected arm around the wrist or distal forearm. The affected arm is gently lifted vertically toward the ceiling, applying gentle vertical traction. While continuing to maintain traction, the arm is externally rotated (Fig. 50-16). Reduction may be subtle, but is generally signaled by hearing or feeling a "clunk." Completion of this technique may require several minutes of gentle traction, allowing the muscles of the patient's shoulder to relax. [34]

Figure 50-14 *A*, Milch technique. Slow, steady abduction with overhead-traction, external rotation (not shown), and direct pressure over the humeral head are the steps of the Milch technique. The procedure may take 3 to 4 minutes to complete, and the operator should avoid sudden, jerky manipulations. It may help to ask the patient to make a motion as if he or she is reaching up and picking an apple from a tree. *B*, The Milch method diagrammed: reduction of an anterior shoulder dislocation includes *(1)* abduction and external rotation, and *(2)* slow and steady gentle traction. When reduced, the arm is adducted *(3)*. Pressure to the humeral head with the operator's hand during traction *(4)* may aid the reduction.

Other methods.

Poulsen reported a method termed the *Eskimo technique*, which may be performed in field settings. In this technique, the patient lies on the unaffected side and is lifted a short distance off the ground by grasping the abducted arm of the injured side. The patient's body weight acts to effect the reduction. Poulsen's success rate was 74% in a series of 23 patients, all of whom were premedicated. [35] Poulsen also postulated that this technique could place undue stress on the brachial plexus or axillary vessels. Use of this technique, when other options are available, should probably be reserved until a larger experience is reported.

Noordeen and associates reported a simple method in which the patient sits sideways in a chair, with the affected arm draped over the backrest. The operator holds the arm with the wrist supinated, and the patient is instructed to stand up. The success rate was 72% in 32 patients treated in this manner. [36] A variation of the chair technique, which was successful in 97% of 188 anterior shoulder dislocations, involves operator-applied traction to the patient's flexed elbow by means of a cloth loop or stockinette. [37] While standing beside the patient, the operator supports the involved elbow, holding the cloth loop in 90° of flexion while stepping down on the cloth loop. The patient sits in the chair, and an assistant may help support the patient by applying countertraction under the involved arm.

Waldron described a technique, without detailing the success rate, which is essentially a reverse of the Stimson method. The patient is placed supine, the affected arm is forward flexed to 90° and upward traction is applied to the distal humerus with the support of the epicondyles. The elbow is allowed to flex passively, and gentle internal and external rotation is applied through an arc of 20° total. [38]

Postreduction Care

After an attempt at reduction, the neurovascular status of the affected extremity should be rechecked and the results documented on the patient record. Indirect evidence that the reduction has been successful includes an immediate reduction

Figure 50-15 Traction-countertraction method. This simple technique for reducing the dislocated shoulder applies gradual and steady traction along the axis of the dislocated limb. A bedsheet, wrapped around the supine patient's upper chest wall and over the unaffected shoulder, is either tied or held by an assistant and acts as a fixed counterforce. A second bedsheet is placed around the patient's flexed forearm, just distal to the flexed elbow, and securely tied behind the operator's back. Note that a significant skin avulsion or friction burn may occur if there is excessive motion of the sheets, especially in the elderly patient with thin, delicate skin. With the patient's forearm held in a neutral rotation and the hand in a vertical position, the operator applies traction by leaning back, rather than using the biceps to apply traction. *(From Respet PB: A practical technique for reducing shoulder dislocations. J Musculoskel Med 5:29, 1988.)*

in pain, restoration of the round shoulder contour, and increased passive mobility of the shoulder. No harm is done by putting the joint through a limited range of motion. If the patient can tolerate placement of the palm from the injured arm on the opposite shoulder, it is quite likely that the shoulder reduction was successful (Fig. 50-17).

Postreduction radiographs are often recommended, with a careful search for new fractures. Although most greater tuberosity fractures do not alter patient management, patients with greater tuberosity fractures displaced > 1 cm after closed reduction are almost always associated with a rotator cuff tear, [39] and should receive prompt orthopedic consultation, as they may require operative repair.

It is important to prevent further external rotation or abduction of the reduced shoulder; adequate immobilization can be obtained by a commercially available shoulder immobilizer or a sling and swath (see Chapter 51). Orthopedic follow-up is recommended for all anterior shoulder dislocations. The incidence of rotator cuff injury is as high as 38%[40] and may complicate restoration of normal function. Younger patients will generally be immobilized for approximately 3 weeks and can be instructed to follow up within 1 or 2 weeks of the event. The older the patient, the shorter the time of immobilization. [10] Those older than 60 years should have early follow-up (5 to 7 days) to allow for early mobilization and avoidance of shoulder joint stiffness.

It is appropriate to prescribe oral analgesics (either nonsteroidal antiinflammatory drugs or narcotics) appropriate for the amount of patient discomfort at the time of disposition and to instruct the patient to return for any worsening of the clinical condition. Periodically one may encounter a return visit from a successfully treated patient who is in severe pain from a hemarthrosis. Trimmings reported excellent relief of pain by aspiration of the hemarthrosis 24 to 48 hours

Figure 50-16 Spaso technique. While maintaining gentle vertical traction, the affected arm is externally rotated by grasping the wrist or forearm. Reduction may be subtle.

after shoulder reduction in a series of patients older than 60 years. [41] This can be accomplished using the technique of arthrocentesis described in Chapter 54 . In addition, intra-articular instillation of 10 to 20 mL of 1% lidocaine as has been recommended for shoulder reduction may be helpful for further pain relief.

Posterior Shoulder Dislocations

Posterior shoulder dislocations account for less than 4% of all shoulder dislocations. [12] Because they are so uncommon, posterior dislocations are easily overlooked and the emergency clinician must be knowledgeable about these injuries to avoid a misdiagnosis. Delays in diagnosis for weeks to months have been reported with posterior dislocations.[42] [43] This may lead

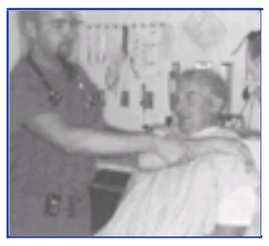

Figure 50-17 If a patient with a shoulder injury can place the palm of the injured arm on top of the contralateral shoulder, it is unlikely that a shoulder dislocation is present. Alternatively, completion of this maneuver after a reduction attempt provides strong evidence that the reduction was successful.

to increased rates of dislocation arthropathy and chronic pain. [13] The mechanism of injury is almost always indirect, with a combination of internal rotation, adduction, and flexion.[10] *Classic precipitating events include seizure, electrical shock, and falls.* The patient may also present at a point well past the original event. [43] Patients with seizures may not experience obvious problems in the immediate postictal period due to their altered mental status.

Clinical Assessment

While clinically less obvious than anterior dislocations, posterior shoulder dislocations do present in a typical, recognizable manner. Mistakes may be made if the clinician is overly reliant on the AP radiographs, which are potentially misleading, [43] and may result in misdiagnosing the injury as a soft tissue contusion or acromioclavicular (AC) strain. *The principal sign of posterior dislocation is an arm that is somewhat fixed in adduction and internal rotation.* Abduction and external rotation are limited, and attempts to perform these movements generally elicit pain (Fig. 50-18).[10] [12] Inspection and palpation reveal a loss of the normal anterior contour of the shoulder and a prominent coracoid and acromion. The shoulder is flattened anteriorly and rounded posteriorly, where the humeral head may be palpable.[10] [12]

Comparison to the opposite shoulder should be undertaken with the understanding that this injury may occasionally occur bilaterally. Neurovascular assessment is performed in the standard manner, although such complications are unusual with posterior dislocations.

Radiologic Examination

The key point regarding radiographs for posterior shoulder dislocations is the subtle nature of this dislocation on a single AP radiograph (Fig. 50-19*A and B*) and the diagnostic value of the scapular Y view (Fig. 50-19*C*) or the axillary view (Fig. 50-19*C*). The diagnosis of posterior shoulder dislocation using the axillary view is quite easy, whereas the routine AP and lateral views are difficult to interpret in around half of cases. [43] The axillary view is generally available in the radiology department and can be obtained with as little as 20–30° of abduction, with the plate placed on the shoulder. [43] In addition to easy visualization of the *posteriorly* situated humeral head, the axillary view often reveals an impression fracture of the humeral head (Fig. 50-19*D*). The humeral head is *anterior* to the glenoid.

Whereas the axillary view is diagnostic, clues to posterior dislocation do exist on the AP film. The internally rotated humeral head appears symmetrical on the AP film in the shape of a light bulb as opposed to the normal club-shaped appearance created by the greater tuberosity. [44] With posterior dislocation, the space between the articular surface of the humeral head and the anterior glenoid rim is widened, and there is a decrease in the half-moon-shaped overlap of the head and the fossa (Fig. 50-20 and Fig. 50-21). [42] [44] There may also be a compression fracture of the medial aspect of the humeral head, indicated by a dense line. This is known as the "trough" sign.[44] A fracture of the lesser tuberosity should always prompt a search for the presence of a posterior shoulder dislocation. [42]

Reduction Technique

An acute posterior dislocation may be reduced by traction on the internally rotated and adducted arm combined with posterior pressure on the humeral head (Fig. 50-22).[10] [43]

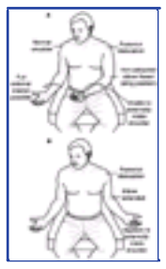

Figure 50-18 *A,* A clue to a posterior shoulder dislocation is the arm locked in adduction and internal rotation, with patient's inability to rotate the shoulder externally with the elbow flexed at 90°. *B,* Note that extension of the elbow with supination of the forearm may obscure loss of the external rotation.

Premedication is generally indicated (see Chapter 34) and countertraction may be applied with a sheet looped in the affected axilla much as described for anterior dislocations. Rockwood and Wirth recommend applying lateral traction on the upper humerus if the humeral head is locked on the posterior glenoid. [10] Hawkins and coworkers suggest that posterior dislocations with an impression defect of the humeral head that is greater than 20% of the articular surface require open reduction. [43] Posterior dislocations that have been diagnosed late are difficult to reduce in a closed manner, but an attempt with adequate premedication is generally indicated. [43]

Postreduction Care

As with anterior dislocations, a repeat neurovascular examination and radiographs are obtained after reduction attempts. As before, the patient's ability to place the palm of the injured arm on the opposite shoulder is suggestive of a successful reduction. Given the rarity of these injuries, orthopedic consultation is often sought early in the care of these patients. Certainly in a training environment, involvement of an orthopedic resident benefits his or her education and should be considered early on. After successful reduction, immobilization with application of a "hand-shake" cast in neutral rotation and slight extension is indicated (Fig. 50-23).[10] This relaxes the injured structures to allow healing.

Unusual Shoulder Dislocations

Inferior dislocations of the shoulder, known as *luxatio erecta*, are quite rare, but also quite obvious. The patient presents with the arm locked in marked abduction with the flexed forearm lying on or behind the head. [45] Occasionally, the humerus may have less abduction, thus potentially obscuring the diagnosis. [46] The humeral head can be palpated along the lateral chest wall. Neurovascular compression may be present, but this is usually reversed once reduction is accomplished. [10] Overhead traction (generally with the arm in full abduction) is applied in the longitudinal direction of the arm and cephalad pressure can be exerted over the humeral head much as in the Milch technique. [10] [46] Countertraction toward the patient's feet can be applied using a sheet placed over the injured shoulder. After reduction, the abducted arm is brought into adduction against the body and the forearm supinated. [47]

Scapular dislocation or "locked scapula" is a rare condition that presents with an obvious protrusion of the lateral border of the scapula and significant swelling of the medial border due to tearing of the musculature. [48] Reduction is accomplished by traction on the abducted arm and medial pressure on the scapula. [48]

ACROMIOCLAVICULAR SUBLUXATION AND DISLOCATIONS

The AC joint is a true diarthrodial joint with a synovial cavity surrounded by a relatively lax capsule and the weak AC ligament. This structure allows for the gliding motion necessary for shoulder movement. The major stability of the AC joint comes from the coracoclavicular ligament, which has posterior (conoid) and anterior (trapezoid) components. The mechanism of injury is generally from a direct force such as a fall on the point of the shoulder with the arm adducted. [49] There are six grades of injury to the AC joint; they are classified by degree or type (I through VI) (Fig. 50-24).

First degree (type I).

This injury consists of a minor tear in the AC ligament. The coracoclavicular ligament is intact. The clinical findings are limited to tenderness in the area of the AC joint. Radiographs show little if any change in the position of the clavicle in relation to the acromion. [39] The management of this condition consists of a sling for comfort, ice, and mild analgesics. Generally, symptoms subside with 7 to 10 days of rest. [10] Orthopedic referral is generally not necessary unless return to normal function is delayed beyond 2 weeks.

Second degree (type II).

In addition to a complete tear of the AC ligament, the coracoclavicular ligament is stretched or incompletely torn. [39] The patient generally supports the injured arm and has slight swelling and definite tenderness over the AC joint. Radiographs demonstrate a definite change in the relationship of the distal clavicle to the acromion.

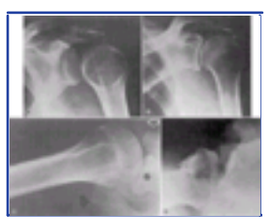

Figure 50-19 *A,* This patient has a posterior dislocation of the humerus. Because the dislocation is directly posterior, there is no superior or inferior displacement of the humeral head. On superficial observation, the head of the humerus appears to maintain a normal relationship with the glenoid fossa and the acromion process. However, definite abnormalities exist in this film. The space between the humeral head and the glenoid fossa is abnormally wide, and because of the extreme internal rotation of the humerus, the head and neck are seen end on. In this projection, the humeral head resembles a light bulb. Compare this film with the same patient's normal opposite shoulder (note that the film is reversed for illustrative purposes) *(B). C,* The normal axillary view of the shoulder. The *asterisk* indicates the glenoid process. The *open arrow* indicates the coracoid process of the scapula and the *closed arrow* indicates the acromion process of the scapula. *D,* Axillary view of a posterior shoulder dislocation with an impression fracture of the humeral head. *(From Harris JH, Harris WH (eds): The Radiology of Emergency Medicine. Baltimore, Williams & Wilkins, 1971.)*

However, in type II injuries, the inferior edge of the clavicle should not be separated from the acromion by more than one-half its diameter, [39] and on radiographic examination, the coracoclavicular distance is the same as the uninjured side. [10] This injury can be treated in a closed fashion with a sling. [10] Orthopedic referral is recommended, and some will use a sling-strap device that elevates the arm and depresses the clavicle for these injuries. [39]

Third degree (type III).

In this injury, the distal end of the clavicle is essentially free floating, as both the AC and coracoclavicular ligament are completely disrupted. [39] The arm is supported by an uncomfortable patient and the distal clavicle is usually seen to be riding high above the acromion. The diagnosis is generally obvious, and radiographs are mainly used to rule out an associated fracture. Radiographic criteria for this degree of injury include an inferior border of the clavicle above the acromion, or a discrepancy in the coracoclavicular distance as compared with the normal side. [10] These injuries require orthopedic referral, and a fair bit of controversy exists regarding their subsequent management.[10] Larsen and colleagues conducted a prospective, randomized trial of conservative vs operative management for significant AC separations and concluded that conservative management was generally better, with possible exceptions made for patients with significant cosmetic deformity and for those who frequently keep the arm at 90° of abduction. [50] While optimal therapy is still unclear, a logical approach would include ED treatment with a sling and early orthopedic referral.

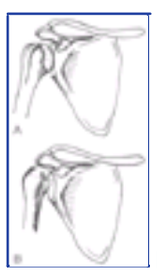

Figure 50-20 *A,* Note the normal elliptical pattern of overlap produced by the head of the humerus and the glenoid fossa. *B,* In the patient with a posterior dislocation, this pattern is lost, and internal rotation of the greater tuberosity is also noted. *(From Simon R, Koenigsknecht S: Orthopedics in Emergency Medicine. New York, Appleton-Century-Crofts, 1982, p 344. Reproduced by permission.)*

Fourth, fifth, and sixth degree (type IV to VI).

In type IV injury, the distal clavicle is free floating and posteriorly displaced into the mass of the trapezius muscle. Type V injury is characterized by inferior displacement of the scapula

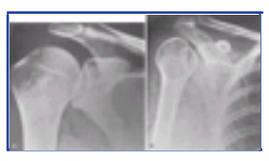

Figure 50-21 Posterior dislocation of the humeral head is a subtle, easily missed finding on the anteroposterior view. A key finding is an abnormal overlap of the humeral head with the glenoid fossa, but additional views are usually needed to confirm the dislocation. Comparison with the normal shoulder may also help. *A,* This AP film showing a posterior dislocation was initially read as normal, but the dislocation was obvious on an axillary view (see Fig. 50-19). *B,* "Light-bulb" appearance of the humeral head in a posterior dislocation. Posterior dislocation should be suspected in patients with significant pain and dysfunction after trauma, especially following seizures and electrical shock injuries. Occasionally the dislocations are bilateral. *(From Riddervold HO: Easily Missed Fractures and Corner Signs in Radiology. Mt Kisco, NY, Futura, 1991.)*

with a marked increase (two to three times normal) in the coracoclavicular interspace. [10] Type IV and V dislocations generally require surgery, and orthopedic referral is required. Type VI injury involves dislocation of the distal clavicle inferiorly. Because this is usually the result of major trauma, multiple other fractures are often seen.[10]

Radiographic Examination

The diagnosis is usually made clinically, with pain and local tenderness at the AC joint in the absence of other findings. Radiographs are generally indicated to rule out associated fractures and to aid in assessing the degree of injury. A single radiograph of the injured shoulder often suffices, but some clinicians prefer to obtain comparison views of the opposite shoulder. While their efficacy has never been proven, it has been traditionally recommended that "weighted" films be obtained in suspected type I or II injuries. Weighted films are generally performed after routine "unweighted" radiographs and are obtained by strapping about 4.5 to 7.0 kg (10 to 15 lb) of weight to the patient's wrists and repeating the radiographs. It is important that the patient keep the shoulders as relaxed as possible during this study, and the patient should not be asked to hold the weights (rather, they are strapped to the wrists). As expected, this study may cause significant discomfort for the patient. Weighted films are of questionable value in mild injuries, and superfluous in obvious type III to VI injuries.

The value of comparison views as well as weighted films may remain controversial by some authors, [51] but their use has been essentially abandoned in current day practice. Bossart and others examined the routine use of "weighted" studies of

Figure 50-22 Reduction of posterior shoulder dislocation—with countertraction being applied, traction on the internally rotated and adducted arm is combined with posterior pressure on the humeral head to effect reduction.

the AC joints and recommended abandoning their use in the ED. [52] In a prospective study of 70 type I or II injuries, the use of weights was associated with *less evident separation* in 7 cases, essentially producing a false-negative study compared to plain unweighted films. Only three injuries were re-categorized as type III after the performance of weighted films. [52] This yield is not necessarily inconsequential if subsequent management would entail a change in therapy for these three patients. However, Bossart and colleagues noted that surgery is often not recommended with type III injuries. [52] For the majority of cases, the editors consider weighted radiographs unnecessary, but they may be helpful for athletes or

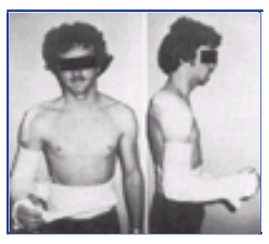

Figure 50-23 Handshake cast—after successful reduction of an acute posterior shoulder dislocation, this cast is applied in neutral rotation, slight extension, and 15 to 20 degrees of abduction. *(From Rockwood CA, Wirth MA: Subluxations and Dislocations about the Glenohumeral Joint. In Rockwood CA, Green DP, Bucholz RW, et al (eds): Rockwood and Green's Fractures in Adults, vol 2, 4th ed. Philadelphia, Lippincott-Raven, 1996, p 1291. Reproduced by permission.)*

in other selected cases. When in doubt, consultation with the referring orthopedic surgeon is advised.

STERNOCLAVICULAR DISLOCATIONS

Despite the fact that the sternoclavicular joint is the least stable joint in the body, sternoclavicular dislocations are rare. [53] The primary supports of this joint are the sternoclavicular and costoclavicular ligaments. Anterior dislocations are much more common and are usually the result of an indirect mechanism involving a blow thrusting the shoulder forward, [39]

Figure 50-24 Grades I to III of acromioclavicular separation. (See text for description.) *(From Heppenstall RB: Fractures and dislocations of the distal clavicle. Orthop Clin North Am 6:480, 1975.)*

or they may be atraumatic due to ligamentous laxity in teens and young adults. [53] Posterior dislocations also usually result from a blow to the shoulder but can also be the result of a direct superior sternal or medial clavicular blow. [53] Posterior sternoclavicular dislocation (also known as retrosternal dislocation as the medial end of the clavicle dislocates both posteriorly and medially) is potentially life threatening, as injury to the great vessels or compression of the airway may occur. [53] Any suggestion of these complications should prompt immediate surgical consultation.

The presentation of these injuries is usually straightforward, with pain, swelling, tenderness, and deformity of the joint. Plain radiographs of this joint are difficult to interpret and generally include an apical lordotic-type view with the radiographic tube angled at 45° cephalad. Confirmation of the diagnosis is best made using a thoracic computed tomographic (CT) scan, which may also identify high rib fractures, pulmonary contusion, or pneumothorax. [53] [54] Children may have epiphyseal disruption with retrosternal displacement of the medial clavicle. [55]

Closed reduction of both types of sternoclavicular dislocation involves placing a rolled blanket or a sandbag between the scapula and applying traction to the 90° abducted arm in line with the clavicle. The clavicle can then be pushed (anterior) or lifted (posterior) back into position. [53] Posterior dislocations may be difficult to reduce and to maintain reduced in a closed manner. Therefore, some authors recommend reduction in an operating suite unless complications necessitate immediate reduction.[53] Given the rarity of this injury and the potential for major underlying complications, early consultation is recommended in suspected posterior sternoclavicular dislocations.

ELBOW DISLOCATIONS

The elbow is second only to the shoulder as a site for major joint dislocations in adults, and it is the most commonly dislocated joint in children. Anatomically, the principle articulation of the humerus and ulna is a stable hinge joint with the intercondylar groove of the distal humerus nestled in the olecranon fossa. Due to the stability of the elbow, any dislocation is expected to be accompanied by significant soft tissue damage, and associated fractures are common. Elbow dislocations are often simply divided into posterior and anterior dislocations (Fig. 50-25). However, there are actually several different types of elbow dislocations in addition to posterior and anterior. These include lateral, divergent, and isolated dislocations of the radius. [56] In the rare divergent dislocations, the radius and ulna are dislocated in opposite directions, either anterior and posterior or medial and lateral. [56]

The most serious complication of elbow dislocation is a brachial artery injury. This injury is possible with any type of

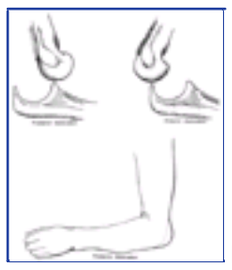

Figure 50-25 Classification of elbow dislocations. *(From Simon R, Koenigsknecht S: Orthopedics in Emergency Medicine. New York, Appleton-Century-Crofts, 1982, p 333. Reproduced by permission.)*

elbow dislocation and is a frequent occurrence in open dislocations. [56] Vascular compromise can be delayed in onset, resulting from either unsuspected arterial injury or progressive soft tissue swelling. The circulatory status of the arm must be carefully monitored even after successful reduction. [56] Although not absolute, patients with these injuries who manifest significant or immediate soft swelling, hematoma formation, or have questionable vascular integrity or neurologic findings are often admitted to the hospital or ED observation unit for observation. In most cases, orthopedic consultation should be sought prior to disposition.

Injury to the median and ulnar nerves may be the result of stretch, severance, or entrapment. It is difficult to clinically distinguish these etiologies, and management of nerve injuries is frequently expectant.[56] It is imperative to conduct a careful neurologic examination before and after reduction, as any increase in findings may indicate entrapment and the need for surgical intervention. [56] Myositis ossificans is also a potential complication of this injury, which underscores the advisability of orthopedic consultation early in the course of care.

Posterior Dislocations

Posterior dislocations make up the vast majority of elbow dislocations. [39] The usual mechanism is a fall on the outstretched hand, with the arm in extension. The clinical examination is usually diagnostic unless severe soft tissue swelling is present. The patient presents with a shortened forearm that is held in flexion, and the olecranon is prominent posteriorly (see Fig. 50-25). The normally tight triangular relationship of the olecranon and the epicondyles of the distal humerus is disturbed in a posterior dislocation. A defect may also be palpated above the prominence of the olecranon.

Radiologic Examination

Two radiographic views, an AP and a true lateral view, should be obtained. The diagnosis is obvious with proper radiographs. A

Figure 50-26 Manipulative reduction of posterior elbow dislocation. While an assistant holds the arm and makes steady countertraction *(1)*, grasp the wrist with one hand and apply steady traction on the forearm in the position in which it lies *(2)*. While traction is maintained, correct any lateral displacement with the other hand *(3)*. While traction is maintained *(4)*, gently flex the forearm *(5)*. Note that with reduction, a clunk is usually felt and heard as the olecranon engages the articular surface of the humerus. *(From DePalma AF: Management of Fractures and Dislocations: An Atlas. Philadelphia, WB Saunders, 1970, pp 793 and 794. Reproduced by permission.)*

careful search for fractures of the distal humerus, radial head, and coronoid process must be undertaken, as they occur commonly in this injury. [56] In children younger than 14 years, the fracture is usually a medial epicondyle separation, as the epiphyseal plate gives way before the medial collateral ligament of the elbow. [56] Postreduction radiographs are also necessary to confirm reduction and to disclose any associated fractures. [57]

Reduction Techniques and Postreduction Care

Although, as with shoulder reduction, some authors claim that their method of reduction is virtually painless, [28] [58] this has not been objectively documented. In general, patients with posterior elbow dislocations are quite uncomfortable, and it is beneficial to administer IV analgesics early in the course of care, preferably prior to positioning for radiographs (see Chapter 34).

In addition to, or in lieu of parenteral sedation and analgesia, some clinicians inject the elbow joint with a local anesthetic (e.g., 3 to 5 mL of 2% plain lidocaine) prior to attempting reduction. Prior to injection, the joint should be aspirated to remove blood (see Chapter 54).

Traditional traction method.

The traditional method[59] of reducing a posterior dislocation is to place the patient in the supine position and have an assistant stabilize the humerus with both hands (Fig. 50-26). The operator then grasps the wrist and applies slow and steady in-line traction. The elbow is slightly flexed to keep the triceps mechanism loose, and the wrist is held supinated as traction is applied. Reduction is usually signified by a "clunk" that is heard or felt. If this method is not successful after a reasonable period of traction (10 minutes), the forearm may be gently flexed to try and effect reduction. Alternatively, downward pressure on the proximal volar surface of the forearm may help free up the coronoid process.

Alternatives.

Several authors have described variations of a prone method of reduction that are reportedly well tolerated by patients. [28] [58] [59] In the method described by Minford,

the patient is positioned with the arm hanging over the padded back of a chair or over the edge of the bed. The clinician then simply applies pressure to the prominent posterior aspect of the olecranon to achieve reduction. [56] Lavine applied traction with the elbow flexed over the edge of a chair by pulling down on the hand while

using the thumb to guide the olecranon into place (Fig. 50-27*A*).[59] Parvin positioned the patient as for the Stimson method of shoulder relocation and applied gentle downward traction to the wrist.[28] When the olecranon was felt to ride distally on the humerus, lifting the humerus completed the reduction.

Recommended initial approach.

A prone technique is advantageous, as patients tolerate this position quite well. The elbow is allowed to hang flexed over the edge of the bed, and an assistant is positioned with his or her back toward the patient such that the humerus can be encircled with both

Figure 50-27 *A,* Hanging method of elbow dislocation reduction. Downward traction is applied to the hand while the olecranon is guided into place. *(From Lavine LS: A simple method of reducing dislocations of the elbow joint. J Bone Joint Surg Am 35:785, 1953.) B,* Reduction of posterior elbow dislocation. An assistant encircles the distal humerus and applies pressure to the olecranon. Traction is applied with the elbow in slight flexion to relax the triceps. The reduction can be performed with the patient reclining *supine* as shown. Having the patient recline *prone* with the upper arm supported by the stretcher can often enhance muscle relaxation.

hands and pressure applied with the thumbs to the posterior aspect of the olecranon. This pressure on the olecranon is intended to lift it up and away from the humerus. The operator applies longitudinal traction to the arm with the elbow in slight flexion. If reduction is not succeeding, an attempt may be made to flex the elbow or the assistant can be instructed to lift the humerus (Fig. 50-27*B*). Reduction is generally noted by a definite "clunk."

Postreduction care.

Once reduction is achieved, the elbow should immediately be put through a gentle range of motion to ensure that the reduction is stable and that there is no mechanical block to movement.[56] An inability to move the elbow through a smooth range of motion after reduction is often caused by an entrapped medial epicondyle fracture fragment that requires operative intervention. [56] The elbow is generally immobilized in at least 90° of flexion with a long-arm posterior splint. A complete recheck of the neurovascular status is performed along with postreduction radiographs.

Following reduction, any signs of delayed vascular compromise are first addressed by loosening the splint and decreasing the degree of flexion. This may restore the pulse.[56] If not, immediate surgical consultation is necessary for emergent arteriogram or exploration of the brachial artery, or both. [57] The risk of vascular compromise is a reason to consider in-hospital observation. Alternatively, some clinicians observe the patient in the ED or ED Observation Unit for 2 to 3 hours postreduction, evaluating for delayed neurovascular compromise prior to discharge.

Anterior Dislocations

Anterior dislocations of the elbow are quite rare; they usually result from a direct posterior blow to the olecranon with the elbow flexed. [56] On physical examination, the arm is extended and there is anterior tenting of the proximal forearm with prominence of the distal humerus posteriorly. [56] These injuries are the result of a great deal of force; they are frequently open and accompanied by significant neurovascular injury. An avulsion of the triceps mechanism may also occur. [56]

Reduction of an anterior dislocation of the elbow involves in-line traction and backward pressure on the proximal forearm (Fig. 50-28). An assistant provides countertraction by grasping the humerus with both hands. Given the infrequent nature of anterior dislocations and the high probability of a severe associated injury, the emergency clinician should consider early orthopedic consultation in such dislocations.

Radial Head Subluxation (Nursemaid's Elbow)

Radial head subluxation is a common pediatric presentation generally occurring between the ages of 1 and 3 years. The mean age of presentation is just older than 2 years, but this entity has been reported in infants younger than 6 months [60] [61] and in older children up to the preteen years. [62] There is a slight predilection for this injury to occur in girls and in the left arm. [61] [63] The classic mechanism of injury is longitudinal traction on the arm with the wrist in pronation, as occurs when the child is lifted up by the wrist.[61] There is no support for the common assumption that a relatively small head of the radius as compared to the neck of the radius predisposes the young to this injury.[64] The pathologic lesion is generally a tear in the

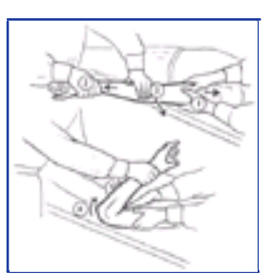

Figure 50-28 Manipulative reduction of anterior elbow dislocation. Reduction is performed with the patient under local or general anesthesia. *1*, An assistant grasps the arm and provides countertraction. *2*, The operator grasps the wrist with one hand and applies traction in the line of the arm, and with the other hand applies firm, steady pressure downward and backward on the upper end of the forearm *(3)*. A clunk usually indicates that reduction is achieved. *4*, The arm is flexed to 45° beyond a right angle. *(From DePalma AF: Management of Fractures and Dislocations: An Atlas. Philadelphia, WB Saunders, 1970, p 796. Reproduced by permission.)*

attachment of the annular ligament to the periosteum of the radial neck, with the detached portion becoming trapped between the head of the radius and the capitellum. [64]

Clinical Assessment

The history offered by the caretaker may not be that of the classic pulling type mechanism. Schunk, in a series of 83 patients, reported that only 51% described such a mechanism, whereas 22% reported a fall.[61] In patients younger than 6 months, the mechanism in the majority is simply rolling over in bed. [60] It is important to remember this and not to proceed with a child abuse investigation unless other suggestive features are present. The typical patient with a nursemaid's elbow presents in no distress with the arm held slightly flexed and pronated at the side (Fig. 50-29). This has been termed the *nursemaid's position*. [65] The exact area of pain is often difficult to locate. The child will refuse to use the arm, and this may be the chief complaint. [62] The older child will usually point to the dorsal aspect of the distal forearm when asked where it hurts. This may mislead one to suspect a buckle fracture of the distal radius.

Although tenderness about the elbow has been reported occasionally, there is often little tenderness or swelling of the elbow region. [61] [62] In the cooperative child, the arm and shoulder are carefully palpated to discern any tenderness. Areas of focus on palpation should include the clavicle and the distal radius, as these are common sites of pediatric fractures. When patient anxiety interferes with a reliable assessment of tenderness in a child whose arm is in the classic nursemaid's position, the examiner can stand at a distance and have the parent or caretaker palpate the extremity to ascertain tenderness. This may also be done in the cooperative patient

Figure 50-29 Typical presentation of a child with a subluxation of the radial head (nursemaid's elbow). It may be difficult to determine exactly where the pathology exists, and often the wrist is thought to be the culprit. This child will not use the injured arm but has minimal discomfort as long as the elbow is not manipulated. *A*, The affected arm hangs down at the side, slightly flexed and pronated. *B*, Once reduced, full activity is generally regained in a matter of minutes.

to reassure the doubtful parent regarding the absence of a fracture. If no tenderness is noted by palpation, it is appropriate to attempt a reduction without prior radiographs. [66]

Although resistance to or pain with supination is a frequent finding in such patients, [61] one need not test for this finding until the time of reduction.

Radiographic Examination

Radiographs are generally not needed in a child presenting with an arm in the nursemaid's position that is nontender (or minimally tender in the radial head area) on palpation, regardless of the history. [66] In these cases, radiographs are generally normal, and if obtained, the positioning of the child's arm by the x-ray technician often effects reduction.[61] However, Frumkin described three cases of nursemaid's elbow in which a line drawn through the longitudinal axis of the radius did not normally bisect the capitellum on prereduction radiographs, but did so after reduction. [64] Radiographs are sometimes recommended if the child is not moving the arm normally 15 minutes after reduction.[65] However, this time frame may be too short, as reuse can be delayed for more than 30 minutes, particularly in children who present some time after the injury. Quan and Marcuse recommend an approach in which no radiographs are obtained on the first visit, including in those children released from the ED prior to full use of the arm. At the time of a 24-hour follow-up visit, radiographs are obtained only if repeat attempts at manipulation are not successful. [62]

While this condition does not generally require x-rays, they can be valuable if external signs of trauma are present (e.g., swelling, abrasions, ecchymoses), or if the child does not use the arm normally within 24 hours after the subluxation is considered reduced. Other less common conditions that can present with similar findings are fractures, joint infections, tumors, or osteomyelitis.

Reduction Technique

Supination method.

Reduction of a nursemaid's elbow (Fig. 50-30) is generally performed without premedication. If the subluxation has been present for hours, oral or nasal midazolam (see Chapter 34) can be a useful adjunct to overcome the child's anxiety related to manipulation. It is

Figure 50-30 Radial head subluxation. *A*, Anatomically, this injury represents interposition of the torn annular ligament between the radial head and the capitellum. *B*, The supination method of reduction is performed by grasping the arm about the wrist and placing the other hand about the elbow with the thumb over the radial head. The forearm is then supinated *(C)* and then the arm is flexed *(D)* in one continuous motion. *(From Fleisher GR, Ludwig S: Textbook of Pediatric Emergency Medicine. Baltimore, Williams & Wilkins, 1988, p 1322. Reproduced by permission).*

important to explain to the caretaker that the reduction will likely cause the child discomfort, but that this is transient and a clue to the diagnosis. The child is positioned seated on the lap of an assistant (often the parent) who stabilizes the arm by holding the humerus adducted to the side. The operator then grasps the elbow with one hand placing the thumb over the

region of the radial head. Although it has been stated that the thumb can apply pressure to the radial head, this positioning is mainly useful for palpation of the reduction "click." The other hand grasps the wrist and is then used to supinate the extended forearm in a steady, deliberate manner. Slight traction prior to supination is generally recommended, but it is unclear whether this increases the likelihood of successful reduction. Once supinated, the arm can be flexed or extended; however, flexion is the most common maneuver and may actually be somewhat more successful than extension.[61] An audible or palpable click signifies successful reduction, but it is not always noted. Once the reduction has been performed, the child usually cries for a few minutes. Generally the operator should leave the room and then return in 10 to 15 minutes to do a repeat examination. Full use of the arm should be evident (see Fig. 50-29B).

Pronation method.

This technique is performed with the child positioned as in the supination method. However, the forearm is not supinated. Instead, the forearm is rapidly hyper-pronated and flexed. A recent study by McDonald reported equal success rates using this technique as compared with the supination technique. [63]

After attempted reduction.

If a click is detected, the child will generally regain use of the arm quickly (almost always by 30 minutes). [62] Therefore, if a definite click is detected, it is reasonable to observe the child for up to 30 minutes prior to further intervention. If there is still no use at 30 minutes, the operator may try to determine if supination is still painful, which would suggest the need for a repeat attempt. In those in whom a click is not detected, the majority will not use the arm by 30 minutes. [62] In these children, a repeat attempt at reduction is recommended after 10 to 15 minutes of nonuse. Two or more attempts are required to produce the click in up to 30% of patients. [62]

If the child has not regained the use of the arm after a few attempts and a reasonable period of time, some authors recommend that radiographs be performed. [65] X-ray films also may help relieve parental anxiety. Alternatively, instructions should be given for 24-hour follow-up if normal function is not restored, with consideration for radiographs at the time of follow-up. [62] In 2 series of patients with nursemaid's elbow, of 10 patients released without normal arm use, 6 had spontaneous restoration of function, and the other 4 required remanipulation, which successfully restored function. [61] [62] The use of a posterior splint to protect the elbow of the child who refuses to use the arm after a presumed reduction is of uncertain value. However, some form of immobilization (e.g., splint, sling, or both) may be valuable in the child with significant residual discomfort following a prolonged period of subluxation or in whom recurrent subluxations have occurred. On occasion, a successful reduction painfully resubluxates with movement; in this case, immobilization and referral may be necessary. [64] If reduction has been achieved clinically and maintained in the ED, analgesics or a follow-up visit are unnecessary. Because other pathology can *rarely* mimic this condition (e.g., occult fractures, osteomyelitis, joint infection, tumors), full, unrestricted, and painless use of the arm must be evident by 24 hours. If not, further assessment is indicated.

HAND INJURIES

The hand is an extremely common site of injury due to the demands placed on it and the exposed nature of its region. Proper motion and function of the hand are intimately related to normal anatomic alignment.[67] The emergency clinician must therefore be skilled in the diagnosis and management of dislocations about the hand. An improperly managed hand injury can result in significant disability that the patient is reminded of on a daily basis.

Anatomically, the joints of the digits are quite similar and consist of a hinge joint with a tongue-in-groove type arrangement. [67] The soft tissue support includes 2 collateral ligaments that are attached to a volar plate (Fig. 50-31). The volar plate is dense fibrous connective tissue that is thickened at its distal attachment and thinner at its proximal attachment, to allow for folding with joint flexion. [67] [68] Dorsal dislocation of a digit requires failure of the volar plate, whereas lateral dislocation disrupts a collateral ligament and induces at least a partial tear in the volar plate (see Fig. 50-31).

Radiographic examination of all hand injuries is relatively straightforward, including at least two views (AP and lateral) of the injured area. The most important radiographic error in evaluating joint injuries of the hand is failing to get a true lateral view of the injured joint. [68] This may lead to missing a fracture or a loose body in the joint.

Anesthesia is generally required for the proper management of dislocations about the hand. This is most often accomplished by finger or wrist block, although a more proximal regional or Bier block may be used on occasion (see Chapter 32 and Chapter 33). Getting a secure grip on the digits may

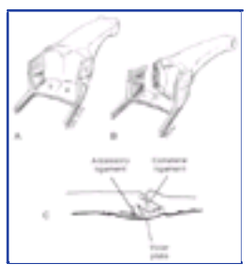

Figure 50-31 *A* and *B,* The collateral ligament-volar plate relationship. The metacarpophalangeal (MCP) and interphalangeal (IP) joints derive their strength from a combination of the two collateral ligaments and the volar plate. Dislocations of these joints require tearing of at least two parts of this three-part structure. *(From Carter P [ed]: Common Hand Injuries and Infections. Philadelphia, WB Saunders, 1983, p 114. Reproduced by permission.) C,* Lateral view demonstrating collateral ligament-volar plate relationship. *(Redrawn from Eaton RG: Joint Injuries of the Hand. Springfield, IL, Charles C Thomas, 1972.)*

be difficult and may complicate the reduction. Wearing rubber gloves or wrapping gauze around the fingers may be useful.

Thumb Dislocations

The opposable thumb is an essential structure for countless activities. Despite its strong ligamentous and capsular support, the exposed positioning of the thumb makes it a frequent site of dislocations and subluxations. The metacarpophalangeal (MCP) joint is similar to those of the fingers but has a stronger volar plate and collateral ligaments.[68]

Interphalangeal (IP) Joint Dislocation of the Thumb

The single interphalangeal (IP) joint of the thumb has strong cutaneous-periosteal attachments, and dislocations of this type are therefore frequently open. [68] Dislocations are generally dorsal and can be reduced in a manner similar to IP dislocations of the finger (Fig. 50-32). The mechanism of injury is recreated by longitudinal traction and hyperextension to distract the phalanges. Reduction is accomplished by flexing the IP joint with continued traction and by applying direct pressure to the base of the distal phalanx.[68]

Following reduction, the range of motion is tested and the stability of the reduction is ascertained. An adequate reduction that is documented on postreduction films is then splinted in slight flexion for 3 weeks.[68] Orthopedic referral is advisable.

Metacarpophalangeal Joint Injury of the Thumb

Dorsal dislocation.

The MCP joint of the thumb can be dorsally dislocated by a hyperextension injury. The proximal phalanx will come to rest in a position dorsal to the first metacarpal (Fig. 50-33). There are two basic types of MCP dislocations (this applies to the fingers also): simple and complex. In a complex MCP dislocation, the volar plate becomes entrapped dorsal to the metacarpal head (Fig. 50-34) with the flexor tendons and lumbricals acting to completely entrap the

Figure 50-32 *A–D,* Traction method of joint reduction. Complete anesthesia using a regional block should precede reduction attempts. *E–H,* Exaggeration of existing deformity method. First, exaggerate the deformity that is present. Then, in addition to steady traction, push the joint back into position. *(From Carter P [ed]: Common Hand Injuries and Infections. Philadelphia, WB Saunders, 1983, pp 109 and 110. Reproduced by permission.)*

metacarpal head.[68] The simple type is amenable to closed reduction, whereas the complex type requires operative reduction due to interposed soft tissue. [67] [68] A simple MCP dislocation can be converted into a complex one during reduction. [67]

Clinical features that suggest a complex MCP dislocation include a proximal phalanx that is less acutely angulated than with a simple dislocation (i.e., <60°). [68] There may also be dimpling noted over the thenar eminence due to pressure from the entrapped metacarpal head. [67] On radiographic studies of simple dislocations, the joint surfaces are in close contact, whereas they are separated in complex dislocations. The presence of a sesamoid bone in the joint space is diagnostic of a complex MCP dislocation (Fig. 50-35). [67]

Reduction of a simple MCP dislocation (Fig. 50-36) involves hyperextension of the joint as far as possible with the wrist in flexion to relax the tendons. Once maximal hyperextension is achieved, the base of the proximal phalanx is pushed distally while the joint is brought back into flexion. [67] Applying simple traction alone as an initial maneuver risks trapping the volar plate and creating a complex dislocation. [67] After reduction, stability of the joint is tested by putting the joint through a full range of motion. The integrity of the collateral ligaments is assessed with the MCP joint in flexion (see later). Simple MCP dislocation injuries generally require casting for 3 weeks with the joints in moderate flexion.[68]

Volar dislocation.

Volar dislocations are rare, and are generally associated with collateral ligament ruptures. They are commonly irreducible due to interposition of one or both extensor

tendons and the dorsal capsule.[67] Orthopedic consultation is required.

Ulnar collateral ligament rupture.

Also known as *gamekeeper's* or *skier's thumb,* this injury results from a laterally directed force at the thumb MCP joint causing a rupture of the ulnar collateral ligament (Fig. 50-37). The usual mechanisms include falling with a ski pole in hand or having the

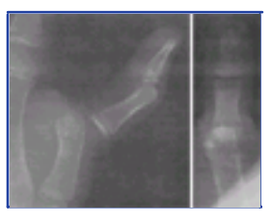

Figure 50-33 Complete dorsal dislocation at the metacarpophalangeal (MCP) joint of the thumb. There is neither associated fracture nor epiphyseal separation. *(From Harris JH Jr, Harris WH: Radiology of Emergency Medicine. 2nd ed. Baltimore, Williams & Wilkins, 1981, p 239. Reproduced by permission.)*

thumb alone draped over the steering wheel in an auto crash. These injuries most often present in the reduced state with just the complaint of pain in the area. Early recognition of this injury is essential in preventing further disability, as this ligament is important for the grasping function of the thumb.

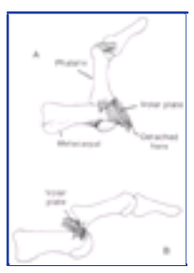

Figure 50-34 *A,* In a simple dorsal metacarpophalangeal (MCP) joint dislocation (note right angle between phalanx and metacarpal), the volar plate remains in front of the metacarpal head, although it is detached from its weaker metacarpal insertion. *B,* In a complex dislocation (note more parallel alignment between phalanx and metacarpal), the volar plate becomes entrapped in the joint and results in an irreducible reduction by closed methods. *(From DePalma AF: Management of Fractures and Dislocations: An Atlas. Philadelphia, WB Saunders, 1970, p 1177. Reproduced by permission.)*

The diagnosis is generally made through stress testing of the MCP joint (Fig. 50-38). Radiographs occasionally demonstrate an avulsion-type fracture. The exact positioning of the thumb for stress testing is debatable, but the metacarpal should be stabilized with the thumb and index finger of one hand and stress applied with the other hand. Louis and coworkers recommend stressing the joint in full flexion, as virtually no lateral movement of the MCP joint should be noted in this position. Instability in full flexion of > 35° is indicative of complete rupture. [69] Hossfeld suggests testing the MCP joint in 20–30° of flexion to lessen the stabilizing effects of the volar plate; the results should be compared with stability on the other side. [68]

Partial injuries to the ulnar collateral ligament are generally casted for 3 weeks; complete rupture usually requires operative repair. [69] An associated nondisplaced fracture may be treated closed, while a displaced fracture is an indication for operative repair. [69]

Carpometacarpal Dislocations of the Thumb

Carpometacarpal dislocations of the thumb are uncommon; when present, they often occur with an associated fracture. Closed reduction is generally unstable, usually necessitating operative stabilization through the percutaneous placement of Kirschner wires. [68]

Finger Dislocations

The basic anatomic structure of the fingers is similar to that of the thumb with the exception that there is more lateral support of the MCP joints, making collateral ligament injury here much less common than in the thumb. The treatment principles are also similar. It is advisable to order radiographs for a specific finger (not just "hand" films). Complete views of the finger will include an AP view, a true lateral view, and an oblique view. The true lateral view is extremely important for detection of subtle dislocations or small avulsion fractures on the volar surface (Fig. 50-39*A* and *B*).

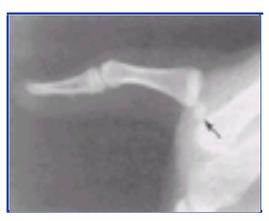

Figure 50-35 Irreducible metacarpophalangeal (MCP) joint dislocation of the thumb. Note the sesamoid bone *(arrow)*, indicating volar plate interposition between the two bone ends, which may prevent closed reduction. *(From Carter P [ed]: Common Hand Injuries and Infections. Philadelphia, WB Saunders, 1983, p 115. Reproduced by permission.)*

Proximal Interphalangeal Dislocations

The proximal interphalangeal (PIP) joint is extremely important, and any loss of motion in this joint may severely restrict normal function. [67] This joint is also prone to stiffness, so careful treatment of injuries to this area is essential. Injuries to the PIP joint are generally slow to heal and often result in an increase in joint size due to scar tissue formation.[67] Because of this propensity for a less-than-perfect outcome, it is advisable to refer PIP injuries after emergency care.

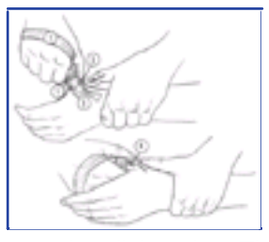

Figure 50-36 If a simple thumb metacarpophalangeal (MCP) dislocation is treated with traction alone, the forces will often interpose the volar plate and result in an irreducible complex dislocation. The proper technique for reduction includes *(1)* a good hold on the patient's distal thumb; *(2)* initial hyperextension of the dislocated phalanx; *(3)* pushing the base of the dislocated phalanx, rather than using traction alone; and *(4)* flexing the thumb. *(From DePalma AF: Management of Fractures and Dislocations: An Atlas. Philadelphia, WB Saunders, 1970, p 1178. Reproduced by permission.)*

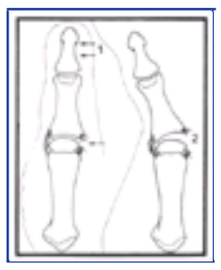

Figure 50-37 Rupture of the ulnar collateral ligament (gamekeeper's thumb). *1,* This injury is caused by forcible abduction. If unrecognized and untreated, progressive metacarpophalangeal (MCP) subluxation may occur *(2)* with interference during grasp, causing significant permanent disability. Suspect this injury when there is a complaint of pain in this region. Look for tenderness on the medial side of the MCP joint. *(From McRae R: Practical Fracture Treatment. Edinburgh, Churchill Livingstone, 1981, p 162. Reproduced by permission.)*

Proper examination of the PIP joint (Fig. 50-40) includes ulnar and radial stress to test the integrity of the collateral ligaments and hyperextension to determine the integrity of the volar plate. The inability to actively extend the flexed PIP joint against resistance suggests a central slip rupture, which may progress to a boutonniere deformity (see Chapter 49).[67] Such an examination should be carried out after any successful joint reduction. This examination should also be conducted in the painful PIP joint that is radiographically normal to detect soft tissue injury in a spontaneously or field-reduced

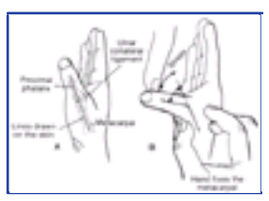

Figure 50-38 Stress testing of the ulnar collateral ligament of the thumb. This is done both clinically and with an anteroposterior radiograph. *A*, A line is drawn on the skin with a pen. The line is along the long axis of the metacarpal and the proximal phalanx. *B*, Deviation of the straight line during stress indicates instability. The metacarpal is fixed with the operator's other hand.

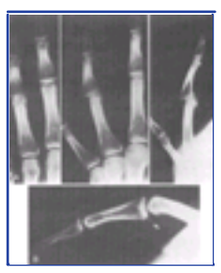

Figure 50-39 *A*, This fracture-dislocation is only fully appreciated on the true lateral film. *B*, A small fragment of bone was avulsed with the volar plate. This frequently is appreciated only with the postreduction film in a true lateral projection. *(From Carter P [ed]: Common Hand Injuries and Infections. Philadelphia, WB Saunders, 1983, p 113. Reproduced by permission.)*

dislocation. This is extremely important in athletes, as coaches often reduce these injuries. [67]

Dorsal PIP dislocations.

These are among the most common types of dislocations encountered in the ED. The

Figure 50-40 Postreduction stress of proximal interphalangeal dislocation. *A,* If the volar plate has been completely disrupted, the proximal interphalangeal joint will hyperextend with both passive and active motion. *B,* An inability to actively extend the proximal interphalangeal joint indicates a rupture of the central slip of the extensor tendon. *C,* Passive lateral stress is performed to check integrity of collateral ligaments. *(From DePalma AF: Management of Fractures and Dislocations: An Atlas. Philadelphia, WB Saunders 1970, pp 1203 and 1204. Reproduced by permission.)*

mechanism is usually a blow to the end of the finger, such as from a thrown ball, which creates an axial load and hyperextends the finger. [68] The middle phalanx comes to rest dorsal to the proximal phalanx (Fig. 50-41). There is always an associated disruption of the volar plate. [67] [68] The deformity is obvious on clinical examination, and radiographs clearly demonstrate the injury. An associated fracture of the volar lip may be detected. If this fracture affects > 33% of the joint surface, a closed reduction will be unstable and operative repair will be necessary, as the collateral ligament is attached to the bony fragment. [68]

A dorsal PIP dislocation can be reduced after a finger block. The usual method (see Fig. 50-32*E–H*) is to exaggerate the injury by slight traction and hyperextension, thereby distracting the middle phalanx. One can then apply pressure to the base of the middle phalanx as the finger is brought into flexion. These injuries usually reduce fairly easily, and failure of routine attempts should raise the suspicion of interposed soft tissue, for which an orthopedic consultation should be sought.

After reduction is completed, the joint should be placed through a range of motion to ensure stability of the reduction. If stable, the joint may be splinted in 20–30° of flexion for 3 weeks.[67] [68] Alternatively, buddy taping for 3 to 6 weeks allows early active motion and prevents hyperextension, which should be avoided. [67] As PIP injuries can be slow to heal and are complicated by stiffness, it is advisable to refer patients with these injuries for orthopedic follow-up.

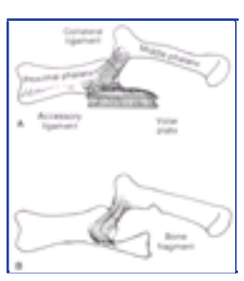

Figure 50-41 A dorsal proximal interphalangeal joint dislocation may involve rupture of the volar plate itself *(A)* or may involve an avulsion of varying amounts of bone from the middle phalanx *(B)*. If a large fragment of bone is avulsed from the base of the phalanx, the dislocation is unstable after reduction. The collateral ligaments will tear in varying degrees and should be assessed with stress testing following reduction.

Volar PIP dislocations.

These are uncommon injuries and virtually always are accompanied by an injury to the central slip of the extensor tendons. If the dislocation is reduced prior to the ED visit and there is no indication that the dislocation was volar, this injury may be incorrectly treated with splinting in mild flexion as if it were the more common dorsal dislocation. However, with disruption of the central slip of the extensor tendons, immobilization in flexion will lead to the development of a boutonniere deformity (see Chapter 49).[67] [70] Even when recognized and treated properly, some impairment of mobility may occur. [70] It is generally best to seek early orthopedic consultation for these injuries, as some require operative repair. If the emergency clinician accomplishes a closed reduction, postreduction films must demonstrate normal congruity of the joint surfaces and a central slip attachment fracture must be excluded. [70] If so, splinting of only the PIP joint in full extension should be undertaken for 3 weeks,

and early orthopedic follow-up ensured. [67]

Lateral PIP dislocations.

These injuries are fairly common and are often reduced in the field. The patient will often dramatically describe how the finger was pointing in an unnatural manner. The injury can be detected by ulnar and radial stress of the PIP joint. If still dislocated, reduction is usually easily accomplished by recreating the injury and applying longitudinal traction to the finger. Partial tears of the collateral ligaments can be treated by buddy taping the finger for 3 to 6 weeks. [67] The management of complete tears is controversial, with some using operative therapy for all such injuries, and others using varying durations of immobilization or buddy taping. [67] Referral is suggested for all but the mildest of PIP collateral ligament injuries.

Distal Interphalangeal Dislocations

As in the thumb, the distal phalanx is attached firmly to skin and subcutaneous tissue by osteocutaneous fibers. For this reason, dislocations of the distal interphalangeal (DIP) joint are frequently open. [67] A DIP dislocation is usually dorsal, and the mechanism is a blow to the end of the finger. Despite the dislocation, the DIP joint may retain some range of motion, so it is important not to overlook these injuries. [71] Lateral radiographs are diagnostic.

Management of the dorsal DIP dislocation involves reduction in a fashion similar to that described for other IP joint injuries. The injury is distracted by traction and hyperextension and relocated by pressure on the base of the distal phalanx during flexion. The joint should be checked for stability, and subsequent management involves application of a dorsal splint for 10 to 12 days of immobilization. [67]

An injury to the DIP joint that may be confused with a dislocation is the mallet finger (Fig. 50-42). This injury is often caused by blunt trauma to the end of the finger (e.g., struck by a baseball). The patient presents with an inability to extend the fingertip, but the joint appears normal on passive extension by the examiner. The injury is a rupture of the extensor tendon, with or without avulsion of a small piece of bone. Unless the injury is properly splinted or surgically immobilized, permanent deformity will occur (see Chapter 49 and Chapter 51).

Metacarpophalangeal Dislocations

The pathology and management of finger MCP dislocations are identical to those of the thumb, as discussed earlier. The same classification of simple and complex applies; the complex type requires operative repair. Dimpling on the palmar surface suggests the presence of a complex dislocation. It is important to remember that the application of traction alone for a simple MCP dislocation may convert it to a complex dislocation. For dorsal dislocations, the wrist is flexed to

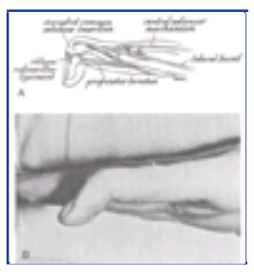

Figure 50-42 *A*, The mallet finger injury is not a dislocation; it is a rupture of the extensor tendon to the distal phalanx. *B*, This mallet deformity was caused by a baseball striking the fingertip end on, producing acute flexion of the joint.

relax the tendons. The next maneuver in reduction should be hyperextension as far as possible. This is followed by pressure on the base of the proximal phalanx to effect reduction.[67] After reduction of a simple dorsal MCP dislocation, buddy taping is generally sufficient to secure the reduction. [67] Volar dislocations are rare and require orthopedic consultation.

Carpometacarpal Dislocations

Carpometacarpal dislocations are rare injuries that are frequently misdiagnosed. The usual site of injury is the fifth carpometacarpal joint, which is dorsally dislocated.[68] The injury is usually the result of a high-energy mechanism, such as a motor vehicle crash or a fall. The diagnosis can be quite difficult, as it may be subtle even on the lateral radiograph. Associated fractures and other injuries are frequently present, and percutaneous fixation is usually required. [68]

HIP DISLOCATIONS

The hip is generally a stable ball-and-socket joint. The head of the femur is deeply situated in the acetabulum, and ligamentous and muscular support is very strong. Hip dislocations are therefore usually the result of significant forces, and a careful search for other limb- or life-threatening injuries must be undertaken. Common mechanisms of hip dislocation include motorcycle crashes, car crashes, and falls. [72]

Associated fractures are quite common with hip dislocations. In fact, up to 88% of hip dislocations present with an associated fracture. [73] When a fracture complicates the dislocation, orthopedic consultation is generally indicated. However, the emergency clinician should be able to reduce simple hip dislocations, which are dislocations without an associated fracture or with a very minor fracture. [74]

Hip dislocations may occasionally be missed in the setting of severe trauma, as other injuries garner more attention. A missed diagnosis can also occur when a femur fracture obscures the clinical picture of hip dislocation. [74] Common complications of hip dislocation include sciatic nerve injuries and avascular necrosis of the femoral head. Sciatic nerve injuries are seen in 10% to 14% of posterior hip dislocations. [74] Avascular necrosis of the femoral head is one of the more disabling complications associated with hip dislocation. Although it is generally stated that early reduction will reduce the frequency of this complication, evidence for this statement is hard to find. Dreinhofer and coworkers noted poor outcomes despite early (i.e., < 6 hours) reduction of type I hip dislocations. [73] Yang and colleagues found that reduction beyond 24 hours was associated with a worse prognosis, but they could not find a significant time factor for those reduced in < 24 hours. [72] However, it is still advisable to reduce hip dislocations as soon as feasible, to decrease soft tissue distortion. If evidence of nerve injury exists, the dislocation should be treated as an emergency and should be reduced as early as possible.

Radiographic Examination

Dislocation of the hip is generally obvious on the standard AP pelvic film that is often taken during trauma resuscitations. The use of a lateral or oblique view may help clarify the type of dislocation, but this can usually be deduced through clinical examination.

Analgesia and Anesthesia

Dislocation of a prosthetic hip can usually be managed with moderate amounts of IV premedication in the ED. Premedication recommendations for acute traumatic dislocations run the gamut from general anesthesia for all reductions [73] to IV sedation only.[75] Most clinicians would agree that some type of IV premedication is necessary, and patients often require deep sedation if the procedure is to be successful in the ED (see Chapter 34). One should not hesitate to opt for spinal or general anesthesia if a reasonable attempt at reduction fails in the ED.

Posterior Hip Dislocations

A posterior hip dislocation is most common and is usually the result of a blow to the flexed knee with the hip in varying degrees of flexion. The greater the amount of flexion of the hip at the time of the injury, the less the chance of an associated fracture. [74] The femoral head is forced out of the acetabulum and rests behind it (Fig. 50-43*A* and *B*). The sciatic nerve is located just behind the hip joint and may be injured with posterior hip dislocation. The clinical picture includes a shortened, internally rotated, and adducted leg.

Reduction Techniques

Several basic methods for hip reduction have been reported in the literature. In the prone or gravity method described by Stimson (Fig. 50-44 and Fig. 50-45), the patient is placed so that the distal pelvis overhangs the edge of the stretcher. The hip, knee, and ankle are all flexed to 90°, and downward pressure is applied to the proximal posterior tibia.[74] The hip can be gently internally and externally rotated to facilitate reduction, and if needed, direct downward pressure may be applied to the femoral head by an assistant. An alternative and more comfortable way to provide downward pressure on the tibia is for the operator to grasp the patient's ankle and place his or her own knee on the patient's calf, applying the body weight for pressure. [75] This method is believed to be the least traumatic; however, associated injuries or deep sedation may prevent the required prone position. [74]

Other techniques involve placing the patient in a supine position with downward stabilization of the pelvis performed by an assistant. In the Allis technique (Fig. 50-46), upward traction is exerted in line with the deformity, and the hip is flexed to 90°. The hip can be gently rotated internally and externally until it is reduced. [74] Howard suggests modifying this technique by applying lateral traction to the flexed upper femur to disengage the head of the femur from the outer lip of the acetabulum. [76]

A newer method known as the Whistler technique was described by Walden and Hamer. [77] The patient is placed in the supine position with both knees flexed to 130°. While an assistant stabilizes the pelvis, the operator stands beside the affected limb, placing an arm under the affected knee to grasp the unaffected knee. With the other hand, the operator anchors the ankle of the affected leg firmly against the stretcher (Fig. 50-47). Using the arm placed under the knee as a lever, the clinician raises the shoulder, elevating the affected knee. This allows the femoral head to move anteriorly around the acetabular rim to relocate. [77] Although there is only a limited experience with this technique, it appears to be a promising, gentle reduction method.

Once reduction is achieved, the legs are immobilized in slight abduction through the placement of pillows or another object between the knees. Reduction is confirmed by repeat radiographs, and the patient is admitted to the hospital.

Anterior Hip Dislocation

Anterior hip dislocation is a less common injury than posterior dislocation, constituting 10% to 15% of all hip dislocations. [74] There are three general types of anterior hip dislocations, which are defined by where the femoral head comes to rest (Fig. 50-48): the iliac or subspinous, the pubic,

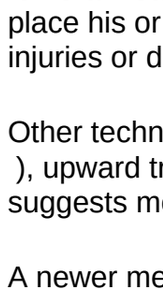

Figure 50-43 *A*, Posterior dislocation of the hip. *B,* In a severely injured patient, a hip dislocation may be initially missed while more pressing or obvious injuries are managed. In this posterior dislocation, the femur is adducted, internally rotated, and superiorly displaced, and an associated posterior acetabular fracture is present. A Malgaigne fracture of the pelvis is also present, with diastasis of the public symphysis and left sacroiliac joint. *(A from Simon R, Koenigsknecht S: Orthopedics in Emergency Medicine. New York, Appleton-Century-Crofts, 1982, p 366. B from Greenbaum E: Radiology of the Emergency Patient. New York, John Wiley & Sons, 1982, p 563. Reproduced by permission.)*

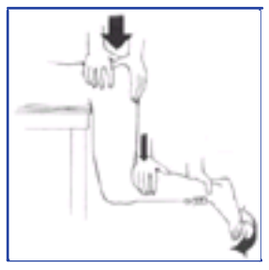

Figure 50-44 Stimson method of reduction for posterior dislocation of the hip (see text for description). *(From DeLee JC: Fractures and dislocations of the hip. In Rockwood CA, Green DP [eds]: Fractures in Adults, vol 2. Philadelphia, JB Lippincott, 1991, p 1588. Reproduced by permission.)*

and the inferior or obturator dislocation. Anterior hip dislocations generally result from a forced abduction of the thigh, which may occur in a fall or motor vehicle crash.[74] The clinical picture varies with the type of dislocation. With the obturator (inferior) type, the leg is abducted and externally rotated with varying degrees of flexion. In the other types, the hip is usually extended and externally rotated. [74]

Reduction Techniques

The Stimson gravity method may work for anterior hip dislocation, although it is not recommended for the pubic type. [74] Alternatively, the Allis maneuver is applied in a modified fashion (Fig. 50-49). The patient is placed in a supine position and an assistant stabilizes the pelvis and applies lateral countertraction to the thigh. Traction by the operator is applied in the long axis of the femur with the hip slightly flexed. The leg is then gently adducted and internally rotated to effect reduction. [74] This is the preferred method.[74]

In the reverse Bigelow technique, the hip is held in partial flexion and abduction. Traction is applied in the line of the deformity, and the hip is then adducted, sharply internally rotated, and extended. Caution should be exercised in using this technique, as the sharp internal rotation may result in femoral neck fracture in patients with osteoporotic bone.[74]

As with posterior dislocations, admission to the hospital is required for patients with these injuries.

Prosthetic Hip Dislocation

Prosthetic hip dislocations may occur with minimal trauma. The three major causes of prosthetic hip dislocations include: 1) the patient assumes a position that exceeds the stability of the prosthesis, 2) soft tissue imbalances, and 3) component malposition. [76] The clinical findings, radiography, and management of prosthetic hip dislocations are similar to those detailed earlier. Orthopedic consultation should be considered for patients with prosthetic hip dislocation.

Figure 50-45 Usually a hip dislocation is the result of significant trauma. *A*, An anterior dislocation of the hip occurred in this elderly patient with a hip prosthesis from minimal trauma (rolling over in bed). *B*, Note the position of the left leg, with the hip abducted, externally rotated, and extended. This is the same position assumed by a patient with an intertrochanteric hip fracture. The more common posterior dislocation positions the hip as adducted and internally rotated. *C* and *D*, Some clinicians prefer to climb on the bed to effect reduction. The assistant may lie over the pelvis or hold the pelvis for countertraction. Care must be taken not to disrupt the prosthesis by using excessive force. Also note that osteoporotic bones can fracture during a forceful reduction. *E*, Following relocation, a knee immobilizer usually prevents the motions required to produce a recurrent dislocation.

Dislocations of Hip Prostheses

The dislocation of a hip prosthesis is a separate issue (see Fig. 50-45). Unlike primary dislocations that require significant trauma, a prosthetic hip may dislocate with minimal force, such as rolling over in bed or trying to get out of a chair. Most dislocations occur in the first 3 to 4 months after surgery, but recurrent dislocation may occur much later. The majority of dislocations are posterior. Reduction techniques are similar to those described earlier; however, the urgency is not paramount since the problems with bone necrosis do not exist. *The clinician must be aware that forceful reduction of the dislocated hip prosthesis may dislodge the acetabular cup, fracture underlying osteoporotic bone, or loosen the prosthesis.*

KNEE (FEMUR/TIBIA) DISLOCATIONS

Although the knee is a simple hinge joint, dislocations are quite rare due to its strong ligamentous support. The major

Figure 50-46 Allis method of reducing posterior dislocation of the hip (see text for description). *(From DeLee JC: Fractures and dislocations of the hip. In Rockwood CA, Green DP [eds]: Fractures in Adults, vol 2. Philadelphia, JB Lippincott, 1991, p 1594. Reproduced by permission.)*

ligaments include the anterior and posterior cruciate and the collateral ligaments. The usual mechanism of a knee dislocation involves a great deal of force, such as a motor vehicle crash or a sporting injury. However, knee dislocation has been reported after minor mechanisms, such as stepping off a curb or into a hole, usually associated with a twisting action.[79] Obese patients may be more likely to dislocate a knee with surprisingly minor trauma, with stepping in a hole with a twisting mechanism being a common mechanism (Fig. 50-50). There are five general types of knee dislocations, including anterior, posterior, medial, lateral, and rotatory. The more common types are shown in Fig. 50-51 . Rotatory

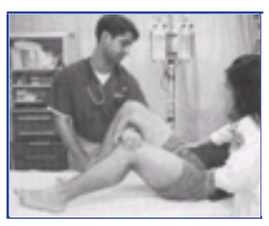

Figure 50-47 Whistler technique of reducing posterior hip dislocation. The operator stabilizes the affected ankle with one hand while placing the other arm under the affected knee to grasp the unaffected knee. While an assistant stabilizes the pelvis, the operator raises the shoulder, elevating the knee to reduce the dislocation.

dislocations may be either posterolateral or posteromedial. Knee dislocations are described with respect to the position of the tibia in relation to the femur. [80]

Clinical Assessment

Knee dislocations are usually clinically obvious; however, dislocation may have been spontaneously reduced prior to ED evaluation, presenting only as a severe knee sprain/strain with hemarthrosis. It is the spontaneously reduced knee dislocation, often one that is associated with other major trauma, that thwarts initial diagnosis. Obese patients may exhibit a seemingly normal appearance to the knee (see Fig. 50-50), but an obvious deformity is often appreciated on initial examination. A grossly unstable knee is probably a reduced dislocation and carries the same risk of vascular and other complications as a dislocated knee. [81] The severely unstable knee can be defined as one that has > 30° of recurvatum (hyperextension) [81] or one that has gross instability after reduction. [80] Because of pain and muscle spasm that limit the physical examination for stability, a knee hemarthrosis, usually a large one with signs of posterior or calf hemorrhage, is a potential tipoff to a reduced dislocation.

An impressive effusion may not be present in knee dislocation, as the joint capsule is often disrupted and extravasation occurs into the surrounding tissue, usually posteriorly. The most important part of the clinical assessment is the vascular status of the extremity (see later). Nerve injury is less common, but peroneal nerve injury is a recognized complication, particularly of a posterolateral dislocation. [80] Posterolateral dislocations may be irreducible, as the medial femoral condyle buttonholes through the joint capsule. [80] A clue to this injury is the presence of a dimple sign at the medial joint line.

Vascular Injury

The most feared complication of a knee dislocation is severance or internal injury of the popliteal artery. Injury to the popliteal artery may complicate both anterior and posterior

Figure 50-48 Anterior dislocations of the hip: obturator, pubic, and iliac. *(From Simon R, Koenigsknecht S: Orthopedics in Emergency Medicine. New York, Appleton-Century-Crofts, 1982, p 367. Reproduced by permission.)*

knee dislocation, and occurs because the artery is relatively fixed both proximally and distally. [80] In addition, Varnell and others noted that vascular injury was as common in the severely unstable knee (e.g., field reduced) as in an acutely dislocated knee. [81] The incidence of popliteal artery injury in a dislocated knee is around 20% in most series.[81] [82] The seriousness of this complication is largely due to the fact that collateral circulation about the knee is poor, [39] and amputation may be the end result of popliteal artery (or vein) injury.

It has been previously stated that popliteal artery disruption can occur despite the presence of a normal pulse. [83] Such statements have led to recommendations to perform arteriography or exploration in all knee dislocations. [80] However, recent studies question that perspective. Varnell and coworkers reported a pulse deficit or absent pulse in all patients with

Figure 50-49 Modified Allis maneuver for reduction of anterior hip dislocation (see text for description). *(From DeLee JC: Fractures and dislocations of the hip. In Rockwood CA, Green DP (eds): Fractures in Adults, vol 2. Philadelphia, JB Lippincott, 1991, p 1588. Reproduced by permission.)*

vascular injury.[81] Kendall and colleagues also reported clear clinical evidence for all popliteal artery injuries in knee dislocations. [82] This group recommended exploration for obvious ischemia, angiography for those with ischemia who have pulse restoration after relocation, and observation for all others. [82] Dennis and colleagues reported that physical examination alone showed 100% accuracy in predicting the need for surgical intervention among patients with posterior knee dislocations. [84] Miranda et al.[85] reported that popliteal artery injury can be safely and reliably predicted by a physical examination that includes specific evaluation for active posterior hemorrhage, expanding hematoma, absent pulse, or the presence of a thrill/bruit. However, it is noted that the hard physical signs of arterial injury may be delayed for 24 to 48 hours. While the focused clinician examination may be quite accurate in the vast majority of cases, popliteal artery injury is subtle enough, or occasionally delayed, that any dislocated knee should prompt serious concern about the vascular integrity of the leg. Simple palpation of the artery may not be sensitive enough to detect a decreased pulse, so a brachial/ankle arterial pressure index, or comparison of the blood pressure at the ankle of the uninjured leg should be considered (see Fig. 50-1). Since all knee dislocations will require orthopedic evaluation, it is the dislocated/spontaneously reduced knee that is problematic for the emergency clinician to diagnose. Internal derangement with a knee hemarthrosis (often of the size noted with an anterior cruciate ligament tear) is a common first impression in the spontaneously reduced knee dislocation. Therefore, all knee injuries with significant swelling, hemarthrosis, or a dislocating mechanism of injury should be evaluated with a specific intent on ruling out vascular injury.

If vascular compromise is detected on clinical assessment, it is appropriate to reduce the knee dislocation without obtaining radiographs, although a few minutes to obtain portable x-rays and administer IV medication would probably rarely make a difference in final outcome. [80] Use of Doppler ultrasound for pulse checks and ankle-brachial pressure indices should also be considered in these injuries (see Chapter 1). Early consultation should be sought in knee dislocations due to the high incidence of complications and frequent need for operative intervention.

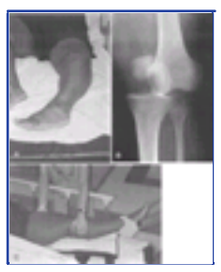

Figure 50-50 *A*, In an obese patient a dislocated knee may not be obvious on initial inspection. This patient stated that she stepped into a hole and twisted the knee (a classic mechanism for dislocation), causing the clinician to suspect only a sprain. *B*, An x-ray demonstrated the seriousness of this seemingly benign injury. *If a spontaneous reduction occurs prior to ED evaluation, this diagnosis may not even be considered. C*, The dislocation is usually readily reduced by traction-countertraction. Tibial manipulation concurrent with traction-countertraction is often helpful.

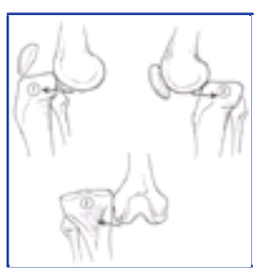

Figure 50-51 Types of knee dislocations. Anterior (*1*), posterior (*2*), and lateral (*3*). *(From DePalma AF: Management of Fractures and Dislocations: An Atlas. Philadelphia, WB Saunders, 1970, p 1621. Reproduced by permission.)*

Reduction Technique

The need for IV sedation and analgesia is dependent on the clinical situation, but it should be considered whenever possible (see Chapter 34). The basic initial approach for all types of knee dislocations is to apply traction to the extremity (Fig. 50-52). This alone is often all that is required for reduction. [57] For anterior dislocations the distal femur is lifted to effect reduction. For posterior dislocations the proximal tibia is lifted to complete the reduction. [39] For medial, lateral, and rotatory dislocations, a similar approach is acceptable, with pressure being exerted as needed in the medial or lateral direction. Posterolateral rotatory dislocations may be irreducible, and will then require operative reduction. [39]

After reduction the extremity is splinted in 15° of flexion. The posterolateral dislocation may be irreducible, and operative intervention should be considered if reduction is not easily accomplished.

Postreduction Care

Appropriate aftercare for knee dislocations involves serial reassessment of the neurovascular status of the extremity. Postreduction radiographs are performed and the patient is

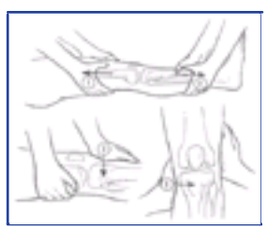

Figure 50-52 Manipulative reduction of a knee dislocation. *1,* An assistant fixes and provides countertraction on the thigh. *2,* Another assistant provides straight traction on the leg (this usually reduces the dislocation). *3,* The operator puts direct pressure over the displaced bones. *(From DePalma AF: Management of Fractures and Dislocations: An Atlas. Philadelphia, WB Saunders, 1970, p 1623. Reproduced by permission.)*

admitted to the hospital. The application of a knee immobilizer will provide stabilization and comfort. These injuries cause severe ligamentous and other derangements in the knee and generally require operative stabilization, with a long period of recovery and physical therapy.

DISLOCATIONS OF THE FIBULAR HEAD

The fibula can be dislocated at its proximal articulation in the knee joint. This is most commonly an anterolateral

Figure 50-53 Anterolateral fibular head dislocation compared to the normal knee. The interosseous distance is widened and the proximal fibula is displaced laterally. *A*, normal anteroposterior projection of knee. *B*, lateral displacement of proximal fibula. *C*, use of bilateral comparison views to highlight fibular displacement.

dislocation.[80] The fibular head is normally nestled in a stable manner behind the lateral tibial condyle with two supporting tibiofibular ligaments. [86] The tibiofibular joint has a separate synovial cavity, and therefore a typical knee joint effusion will not be seen with this dislocation. When the knee is flexed, the stability of this joint is decreased due to relaxation of the fibular collateral ligament. [86] The typical mechanism of injury is a fall on the flexed, adducted leg, often combined with ankle inversion. This mechanism is seen in sports and parachute landings. [86] Posterior dislocations can occur from a twisting mechanism or a direct blow to the area while the knee is flexed.[80]

Anterolateral dislocation is the most common type. It is accompanied by obvious prominence of the fibular head anteriorly; no associated neurovascular problems are noted. The less common posterior dislocation may be accompanied by peroneal nerve injury. [80] Patients present with varying degrees of disability, and some may walk on the leg with only mild discomfort. [86] On radiographic examination, the three cardinal signs of anterolateral dislocation are lateral displacement of the fibula on the AP film, a widened proximal interosseous space, and anterior displacement of the fibular head on the lateral view (Fig. 50-53).[86]

Reduction Technique

The position for reduction of an anterior fibular head dislocation is to place the patient supine and flex the knee to 90° in order to relax the biceps femoris tendon. Direct pressure is then applied to the fibular head and reduction is usually signified by a snap. [80] The method for a posterior dislocation is the same except that the direct pressure is applied in forward manner. Patients should not bear weight for 2 weeks and should receive orthopedic referral. Immobilization is probably unnecessary.[80]

PATELLAR DISLOCATION

Patellar dislocations are fairly common, especially among adolescents. The usual mechanism is a powerful quadriceps contraction combined with a strong valgus and external rotation component.[87] This may be seen in activities such as making a "cut" in sports or with dancing. The patella may

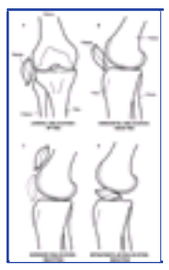

Figure 50-54 Various types of patellar dislocation. The lateral dislocation is the most common.

also dislocate from a direct blow to the flexed knee.[39] Predisposing factors to patellar dislocation include chronic patellofemoral abnormalities such as genu valgum and femoral anteversion.[87] The patellar dislocation is described by the relation of the patella to the knee joint. Lateral dislocations are the most common by far. Other types include superior, medial, and intra-articular (Fig. 50-54).

Figure 50-55 *A*, Lateral dislocation of the patella. *B*, To reduce a lateral dislocation, the knee is extended while the patella is directed medially using slight anteriorly directed elevation.

Clinical Assessment

Lateral dislocation of the patella is generally clinically obvious (Fig. 50-55). The knee is held in some degree of flexion and the patella can be easily seen and palpated on the lateral side of the knee. A tenting-type action of the patella is often detectable unless significant soft tissue swelling is present.

The patella may be spontaneously reduced in the field with simple leg straightening. The patient will report that the leg "went out" and may describe actually seeing the lateral deformity caused by the displaced patella. Clinical clues to the spontaneously reduced patella include the presence of a knee effusion and tenderness along the medial edge of the patella. Fairbank's test or the patellar "apprehension" sign is elicited when the patella is pushed laterally and the patient grabs for the knee, indicating the sensation of repeat injury.[39]

Radiographs

Prereduction films are difficult to obtain as the patient is usually in flexion. Some recommend prereduction films when possible in all patients [39] ; however, it is an easy matter to reduce these injuries prior to radiography. The diagnosis is usually obvious and there are no reports in the literature of complications from gentle reduction. Osteochondral fractures are detectable in about half of patients with patellar dislocations, but many of these are only visible on arthroscopy. [87] Postreduction radiographs are recommended, as are prereduction studies, when the diagnosis is uncertain. The clinical diagnosis of patellar dislocation in an older patient should be made with caution, as these are primarily injuries of the young.

Reduction Technique and Postreduction Care

Reduction of a lateral patellar dislocation is usually quite simple. Premedication is often not required if the patient can be verbally reassured. If the patient is anxious or in great discomfort, premedication should be considered (see Chapter 34). The two basic maneuvers for patellar relocation are extension of the knee and gentle medial pressure applied to the patella, lifting the most lateral edge of the patella over the femoral condyle (Fig. 50-56). [39]

The leg is then immobilized in extension. This may be done by casting or application of a commercially available knee immobilizer (see Chapter 51). Orthopedic follow-up is necessary because of the need for physical therapy and the high rate of persistent instability. [87] However, hospitalization is not required for routine lateral dislocations of the patella. Recurrent dislocation, and those associated with an osteochondral fracture, may require operative repair.

Patellar dislocations in other locations are often irreducible, and orthopedic consultation should be sought in these circumstances. Intracondylar and superior dislocations are extremely rare and require operative reduction. The rare horizontal dislocation may relocate with closed reduction but surgical reduction is often necessary.

ANKLE DISLOCATIONS

The ankle joint is a modified saddle joint in which the talus is nestled in the mortise formed by the distal tibia and fibula. [39]

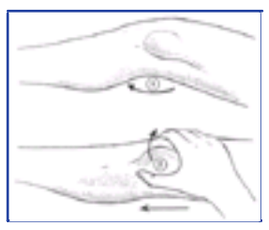

Figure 50-56 Manipulative reduction of a lateral patellar dislocation. Extend the knee gradually (*1*) while applying medially directed pressure on the patella *(2)*, pushing it over the lateral femoral condyle. *(From DePalma AF: Management of Fractures and Dislocations. Philadelphia, WB Saunders, 1970, p 1665. Reproduced by permission.)*

The ligamentous support of the ankle is quite strong, and pure dislocations are uncommon. Usually there are associated fractures of the ankle joint (Fig. 50-57). Ankle dislocations are described by the relation of the talus to the tibia. Posterior dislocations of the ankle are more common than are anterior dislocations, and they usually result from a fall on a plantar flexed foot. Patients with posterior dislocations often have an associated fracture of one or more of the malleoli (Fig. 50-58).[39] The clinical picture is usually one of significant deformity and disability.

Anterior dislocations generally result from forced dorsiflexion or a blow directed posteriorly to the distal tibia while the foot is fixed. The talus is prominent anteriorly and the dorsalis pedis pulse may be lost secondary to pressure from the talus. Superior dislocations are uncommon and result in diastasis of the tibiofibular joint. These injuries are usually the result of a significant axial force. Lateral dislocations of the

Figure 50-57 The types of dislocations of the ankle. *(From Simon R, Koenigsknecht S: Orthopedics in Emergency Medicine. New York, Appleton-Century-Crofts, 1982, p 419. Reproduced by permission.)*

Figure 50-58 Isolated posterior tibial lip fracture *(open arrow),* seen after reduction of posterior ankle dislocation. *(From Harris JH Jr, Harris WH: Radiology of Emergency Medicine, 2nd ed. Baltimore, Williams & Wilkins, 1981, p 629. Reproduced by permission.)*

ankle are always associated with fractures of the malleoli or distal fibula.

Radiographic Examination

Because of the high rate of associated fractures and the clinical difficulty in assessing for the presence or the exact nature of a dislocation, it is recommended that prereduction radiographs be obtained in all suspected ankle dislocations. It is acceptable to reduce the dislocation without a radiograph if severe vascular compromise is present, but for the vast majority of cases, the few minutes taken to obtain bedside radiographs and administer IV medications rarely impact final outcome. It may be impossible to accurately determine the exact type of dislocation unless prereduction films are obtained. An AP and lateral view usually suffice for emergency management, and other views can be ordered if necessary after the joint is relocated.

Reduction Techniques

Unless a strong contraindication is present, it is advisable to administer IV sedation and analgesia to patients with ankle dislocation early in their care, preferably before conducting any manipulations or radiologic studies. Reduction is always painful in the awake patient, and sufficient premedication must be administered (see Chapter 34). For posterior dislocations, the patient is placed supine and the knee is flexed to relax the Achilles tendon. This can be done by an assistant, or

Figure 50-59 Manipulative reduction of a posterior ankle dislocation. *1,* The knee is flexed. *2,* The assistant provides countertraction on the leg. *3,* The forefoot is grasped with one hand and the heel with the other hand. *4,* The foot is slightly plantar flexed. *5,* Apply straight downward traction on the plantar flexed foot, then pull the foot forward *(6)* while a second assistant provides counterpressure on the front of the lower leg *(7). (From DePalma AF: Management of Fractures and Dislocations. Philadelphia, WB Saunders, 1970, pp 1916 and 1917. Reproduced by permission.)*

the patient can be brought to where the knee hangs over the end of the bed. The operator then grasps the foot with both hands, placing one hand on the heel and the other on the forefoot (Fig. 50-59). The foot is flexed slightly plantar, and traction is applied to the foot. A second assistant can then apply downward pressure on the distal tibia as the operator moves the heel anteriorly to effect reduction. [88]

For anterior dislocations, the initial steps and positioning are the same as for posterior dislocation (Fig. 50-60). However, instead of plantar flexion, the foot is dorsiflexed to free the talus. The second assistant applies upward pressure to the distal tibia while the operator applies traction and pushes the foot in a posterior direction. [39]

Lateral dislocations are really fracture-dislocations, and orthopedic consultation is generally required as part of the ED course. The emergency clinician will often need to reduce these injuries due to the extreme lateral deformity and the occasional compromise of the dorsalis pedis artery by stretch. Open dislocations (in the absence of vascular compromise) may be better handled by cleaning in the operating room prior to attempts at reduction. If a lateral fracture-dislocation is to be reduced in the ED, the approach is quite similar to that for posterior ankle dislocation. However, instead of pressure in the AP direction, the foot is moved medially

after application of traction. [39]

Postreduction Care

The ankle is splinted at 90° with a long-leg posterior splint; necessity for admission to the hospital must be determined in consultation with an orthopedic surgeon. Many patients with these injuries have associated fractures that necessitate surgical intervention.

DISLOCATIONS OF THE FOOT

The importance of the foot is recognized by anyone who has had to spend time ambulating with an injury to this area. For the purposes of discussion, injuries to the foot can be divided into those of the hindfoot and those of the forefoot.

Hindfoot Injuries

Injuries to this area are uncommon and usually require high-energy transfer. The major dislocations are the subtalar and talar dislocations, and midtarsal fracture-dislocations (Lisfranc injury). Lisfranc injury is complex and is always a fracture-dislocation because of the rigid nature of the region. These injuries require orthopedic management and are not discussed here.

Subtalar Dislocation

This uncommon injury generally occurs secondary to sports, falls from heights, or motor vehicle crashes. The calcaneus, navicular, and forefoot are displaced from the talus.[88] The primary mechanism is severe inversion causing a medial dislocation, or severe eversion resulting in a lateral dislocation. The medial type occurs so commonly during basketball that it has been termed *basketball foot*.[89] This is an injury usually seen in young adult males. Medial dislocations constitute the majority (85%) of these injuries, with lateral dislocations making up the rest. [89]

The diagnosis is usually obvious, as the talus is prominent and often tents the skin of the proximal foot. The medial type has been termed an "acquired clubfoot," whereas the lateral appears as an "acquired flatfoot." [89]

Some authors recommend spinal or general anesthesia for all such injuries [89] ; however, it is usually possible to reduce these injuries with IV premedication (see Chapter 34). The patient is positioned in a supine fashion and the hip and knee are flexed much as in posterior dislocation of the ankle. One hand is placed on the forefoot and the other grasps the heel. Firm longitudinal traction is required to effect reduction, and dangling the leg over the end of the bed allows the operator to use his or her body weight to assist in traction. Once traction is applied, the deformity is initially increased (inversion for medial; eversion for lateral) and then reversed to effect reduction.[89]

Dislocation of the Talus

In this extremely rare injury, the talus is essentially extruded from its normal position, coming to lie anteriorly. This injury is generally open, [89] is not amenable to closed reduction, and virtually always progresses to avascular necrosis. [90] Talectomy and arthrodesis are generally required, [89] and orthopedic referral should be emergently undertaken if vascular compromise of the foot is present.

Forefoot Dislocations

Much of what is pertinent to the diagnosis and management of forefoot dislocations has already been discussed in the

Figure 50-60 Manipulative reduction of anterior ankle dislocation. *1,* The knee is flexed. *2,* The operator grasps the forefoot with one hand and the heel with the other hand. *3,* Dorsiflexion of the foot is slightly increased (to disengage the talus). *4,* An assistant provides countertraction on the leg. *5,* Straight longitudinal traction is applied, then the foot is pushed directly backward *(6)* while a second assistant provides countertraction on the back of the lower leg *(7). (From DePalma AF: Management of Fractures and Dislocations. Philadelphia, WB Saunders, 1970, pp 1918 and 1919. Reproduced by permission.)*

management of dislocations of the fingers and hand MCP joints. Anatomically, the joints are quite similar.

Metatarsophalangeal (MTP) Dislocations

These uncommon injuries are generally the result of hyperextension resulting in a dorsal dislocation of the great toe MTP joint. [91] Among the lesser toe MTP joints, lateral or medial displacement of the digit on the metatarsal head is more common, and usually is the result of jamming the toe on a piece of furniture. [89] As with MCP dislocations, these can be simple or complex. Complex dislocations of the first toe can be suspected by the presence of sesamoid bones in the joint space in the prereduction radiographs. [89] Complex MTP dislocations are irreducible.

For simple MTP dislocations, reduction is accomplished by increasing the deformity through hyperextension and then applying traction while applying thumb pressure over the base of the dislocated proximal phalanx. Plantar flexion of the foot may be used to relax the flexor tendons. [88] Operative intervention is required after reduction if crepitus is present on motion, the joint is unstable, or an intra-articular loose body is noted on the postreduction radiographs. [89]

Interphalangeal Dislocations

In the foot, IP dislocations result from an axial load to the toe, such as from kicking a wall. These dislocations are generally dorsal and can be reduced as in the hand. Dislocations of the first toe interphalangeal joint are usually buddy taped to the second toe for 2 to 3 weeks, whereas those of lesser toes can be taped for 10 to 14 days.[89] As in the hand, complex dislocations may occur and require open reduction.

CONCLUSION

The following points are important regarding the assessment and management of dislocations:

1. A search for other more serious injuries should be undertaken when there is a high-energy mechanism of injury.
2. A neurovascular assessment should be performed early in the evaluation and appropriately documented.
3. Radiographs and IV premedication are generally indicated prior to reduction attempts.
4. Reduction attempts should involve the gentle, gradual application of forces and patience on the operator's part.
5. After completion of reduction, the operator should recheck the neurovascular status, request postreduction radiographs (except with radial head subluxations), and, in certain circumstances, assess the stability and the range of motion of the joint.
6. A definite percentage of dislocations are irreducible, and the need for multiple attempts should halt prolonged and forcefull attempts in the ED and prompt orthopedic consultation.
7. Reductions that present with neurologic injury should be reduced by the most expeditious and least traumatic method.

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Chapter 51 - Splinting Techniques

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Stacie Byers

Splints are frequently used in the emergency department (ED) for temporary immobilization of fractures and dislocations and for definitive therapy of soft tissue injuries.[1] [2] Patients with acute gouty arthritis of the wrist acknowledge reduced pain with short-term use of a short arm splint while medical therapy is initiated.

Immobilization is the mainstay of fracture therapy, but it is difficult to find firm scientific data that support the use of splinting for soft tissue injuries. [3] [4] Although the general principle of immobilizing sprains and contusions is strongly supported by custom and personal preference, its exact influence on healing, number of complications, and ultimate return to normal activity is not known. In most studies of ankle sprains, for example, the function and pain of the injured joint are similar at 6 weeks' follow-up, regardless of whether treatment consisted of ad lib walking, a simple elastic bandage, a posterior splint, or a formal cast. [5] [6] A systematic review of 22 random clinical trials comparing various treatments for lateral acute ankle sprains (cast, splint, or early immobilization with support) found no favorable effect of immobilization. The current data support functional management for most cases of acute ankle sprains. [7] Similar concepts have evolved for acute cervical strain and low back strain. Although a strict standard of care cannot be promulgated, the use of short-term splinting in the ED for acutely painful conditions remains a common practice.

Most splinting techniques are handed down from house staff or experienced clinicians, but the procedure is often suboptimal and haphazard. [8] This chapter presents guidelines for the adequate immobilization of injuries that are commonly encountered by emergency clinicians.

Patients routinely present to the ED with injuries that are amenable to splinting to relieve pain and to augment healing (Table 51-1). Emergency clinicians have virtually abandoned the use of circumferential casts in favor of premade commercial immobilizing devices or splints made from plaster of Paris or fiberglass. The impetus for this change is primarily related to the complications occasionally associated with circumferential casts, liability issues, and ease of application

TABLE 51-1 -- Conditions That Benefit from Immobilization

Acute arthritis
Severe contusions and abrasions
Skin lacerations that cross joints
Tendon lacerations
Tenosynovitis
Puncture wounds to the hands, feet, and joints
Animal bites to the hands or feet
Deep space infections of the hands and feet
Joint infections
Fractures and sprains
Reduced joint dislocations

brought about by new technology. In most instances, properly applied splints provide short-term immobilization equal to that of casts, while allowing for continued swelling, thus reducing the risk of ischemic injury. Other obvious advantages of splints are that patients can take them off when immobilization is no longer needed or can remove them temporarily to bathe, exercise the injured part, or perform wound care.

INDICATIONS

Theoretically, immobilization facilitates the healing process by decreasing pain and protecting the extremity from further injury. Other benefits of splinting are specific to the particular injury or the problem that is being treated. For example, in the treatment of fractures, splinting helps maintain bony alignment. Splinting deep lacerations that cross joints reduces tension on the wound and helps prevent wound dehiscence. Immobilizing tendon lacerations may facilitate the healing process by relieving stress on the repaired tendon. The discomfort of inflammatory disorders such as tenosynovitis or acute gout is greatly reduced by immobilization. Deep space infections of the hands or feet as well as cellulitis over any joint should similarly be immobilized for comfort. Limiting early motion also may reduce edema and theoretically improve the immune system's ability to combat infection. Hence, select puncture wounds and mammalian bites of the hands and feet may be immobilized until the risk of infection has passed. Splinting large abrasions that cross joint surfaces prevents movement of the injured extremity and reduces the pain that is produced when the injured skin is stretched. Finally, patients with multiple trauma should have fractures and reduced dislocations adequately splinted while other diagnostic and therapeutic procedures (e.g., peritoneal lavage, computed tomography scan) are completed. Immobilization decreases blood loss, minimizes the potential for further neurovascular injury, decreases the need for opioid analgesia, and may decrease the risk of fat emboli from long bone fractures.

EQUIPMENT

Support Materials

Plaster of Paris

Plaster of Paris is the most widely used material for ED splinting. [9] Its name originated from the fact that it was first prepared from the gypsum of Paris, France. When gypsum is heated to approximately 128°C, most of the water of crystallization is driven off, leaving behind a fine white powder—plaster of Paris. When water is added to plaster, the reaction is reversed, and the plaster recrystallizes or sets by incorporating water molecules into the crystalline lattice of the calcium sulfate dehydrate molecules.

Today, plaster is impregnated into strips or rolls (2-, 3-, 4-, or 6-in widths) of a crinoline-type material. The crinoline allows for easy application, helps keep the plaster molded to the proper form during the setting process, and adds support to the finished splint. Plaster rolls and sheets are available in a variety of setting times and widths. The distinct advantage of plaster over commercially available, premade splints is that plaster can be more easily molded and tailored to the individual's anatomy, negating the "one-size-fits-all" approach. Also, plaster is generally less expensive than premade splints.

Prefabricated Splint Rolls

The use of plaster splints in the form of prefabricated splint rolls (e.g., OCL) are very popular among emergency clinicians. These splint rolls have 10 to 20 sheets of plaster enclosed between a thick layer of protective foam padding on one side, and a thin layer of cloth on the other side. Like custom-constructed splints, they are secured to the extremity with an elastic bandage. The major advantage of prefabricated splint rolls is that significant time is saved because the splint and padding come ready to apply. In addition, prefabricated splint rolls are ideal for intermittent splinting and can be removed and reapplied by the patient as needed. However, prefabricated plaster splint rolls are more expensive than simple plaster rolls, and they lack some of the versatility and custom-fit qualities of self-made plaster splints. The application of premade splints is shown in Figure 51-1 .

Prefabricated splint rolls using layers of fiberglass between polypropylene padding (e.g., Ortho-Glass, 3M) are now commonplace in many EDs (Fig. 51-2). Fiberglass splint rolls offer the same timesaving aspect of prefabricated plaster splint rolls, but require only 3 minutes to set, making application faster. In addition, splints made from prefabricated fiberglass rolls cure more rapidly (20 minutes), have no messy

Figure 51-1 Some clinicians prefer to use premade plaster splints for temporary immobilization (e.g., OCL, J-Splint). There is less latitude for custom fitting with these products compared with the technique demonstrated in Figure 51-3 . *A,* Measure the appropriate length. (Note that plain plaster slabs shrink several centimeters when wet. Cut the material slightly long, since the excess may be folded back on itself if necessary). *B,* Submerge the plaster until bubbling stops (do not oversoak). *C,* Roll and squeeze excess water from the premade splint. *Inset,* If using plain plaster slabs, allow the water to drip off. *D,* Smooth the sheets to remove wrinkles and mix the plaster throughout the layers. *E,* Apply the splint and secure it with an elastic bandage. *F,* Mold the splint to fit the contour of the extremity—an important step. *(Courtesy of Johnson & Johnson Products, Inc., New Brunswick, NJ.)*

residue (i.e., they can be hydrated using a conventional sink without a special trap), can be washed and reapplied, and are stronger and lighter than splints constructed from prefabricated plaster rolls. Another advantage is the polypropylene padding, which wicks moisture away from the skin better than polyester, nylon, or cotton padding. [10] Prefabricated fiberglass splint rolls are more expensive than both simple plaster rolls and prefabricated plaster splint rolls, and like prefabricated plaster splints, lack some of the versatility and custom-fit qualities of self-made plaster splints.

Protective and Miscellaneous Equipment

Stockinette

A single layer of stockinette is commonly used under circumferential casts and splints. It protects the skin and, when folded back over the ends of the plaster, creates a smooth, professional-looking, padded rim. Stockinette is available in 2-, 3-, 4-, 8-, 10-, and 12-in widths.

Padding

Padding under the splint protects the skin and bony prominences and allows for swelling of the injured extremity. Most

Figure 51-2 Premade fiberglass splints are also popular for temporary immobilization. Fiberglass splints set and cure more rapidly, have no messy residue (i.e., can be hydrated in a conventional sink without a special trap), can be washed and reapplied, and are lighter and stronger than plaster splints. *A*, Open the pouch and cut the splint to the desired length, stretching the padding to cover the exposed edge. Dip into cool water and squeeze three or four times (Note: Prefabricated fiberglass splints that do not contain water-repellant felt are hydrated in a different manner—Always follow the manufacturer's instructions). Remove the splint from the bucket and squeeze to remove any excess water (this allows a curing time of 3 to 4 minutes). *B*, Place the splint on an absorbent towel to remove excess water. *C,* Apply the splint and secure it with an elastic bandage. When applying the splint at a right angle (e.g., posterior ankle splint), pinch and fold over any extra splinting material). *(Adapted, courtesy of 3M Medical Division, 3M Health Care Markets, St. Paul, MN.)*

commercially available splints contain adequate padding in the premade product, but in some instances additional padding is prudent. In general, the older thin cotton padding known as sheet wadding has been replaced by newer materials such as Webril (Curity) or Specialist (Johnson & Johnson) cast padding. Webril is soft cotton with a much coarser weave than sheet wadding; consequently it has greater tensile strength, adheres better, and can be applied more evenly. Specialist padding uses micropleated cotton fibers that relax when moistened. This results in uniform, felt-like padding that conforms to the surface being wrapped. Felt (0.5-in thick) also may be used to pad bony prominences.

Elastic Bandages

Elastic bandages are used to secure the splint in place. Elastic bandages are available in 2-, 3-, 4-, and 6-in widths. Some bandages use metal clips. Others use a Velcro-type arrangement at the end of the roll.

Adhesive Tape

Adhesive tape is used to prevent slippage of the elastic bandages, to line the cut edges of a bivalved cast, and to "buddy tape" digits. Coban tape can be used in a similar manner and has the advantage of only adhering to itself.

Utility Knife, Scalpel, and Plaster Scissors

A utility knife, a No. 10 scalpel blade, or plaster scissors can be used to cut and shape dry plaster.

Bucket

A large bucket (preferably stainless steel) is used for wetting plaster. Plaster should not be prepared in the sink because the residue quickly clogs the drain. A special drain is required to accept plaster residue. A bucket is not required for the minimal amount of water used to soften fiberglass premade splints—they can be placed directly under the faucet.

Protective Gear

Gowns or sheets prevent soilage of both the patient's and the clinician's clothing. Gloves (vinyl or latex) and safety glasses are recommended to prevent skin or eye damage from plaster dust, wet plaster, or uncured fiberglass polymer. Wearing gloves also decreases clean-up time for the clinician.

GENERAL PROCEDURE OF CUSTOM SPLINT APPLICATION

The following section refers to the application of custom-made plaster splints (Fig. 51-3), unless otherwise stated. If periodic wound care is required, a more easily removable splint (e.g., OCL, Ortho-Glass, Velcro-type splint) should be applied in lieu of the standard splint to be described. The issue of removability should be addressed before the splint is applied. In addition, use of Webril (Curity) cast padding is described, but other suitable cast padding may be substituted.

Patient Preparation

If the clinical situation permits, the patient should be covered with a sheet or gown to protect clothing and the surrounding area from water and plaster. Nursing staff and housekeeping also appreciate this courtesy. The involved extremity should be inspected carefully before splinting. All skin lesions and soft tissue injuries should be examined and documented

Figure 51-3 Principles of custom splint application. *A*, Stockinette is applied to extend 2 or 3 in beyond the plaster. *B*, Two to three layers of Webril are evenly and smoothly applied over the area to be splinted. *C*, The plaster slab is positioned over the area to be immobilized and the stockinette and Webril are folded back to help secure the slab in place and to form smooth, rounded ends. *D*, The elastic bandage is applied to secure the splint. *E*, While still wet, the plaster is molded to conform to the shape of the extremity. This is an important step that is often overlooked.

clearly on the ED record. All wounds should be cleaned, repaired, and dressed in the usual manner. When immobilizing open fractures or joints, the soft tissue defect should be covered with saline-moistened sterile gauze.

Padding

When the splint involves the digits, padding must be placed between the fingers and toes to prevent maceration of the skin. This can be done with pieces of Webril or gauze cut to the appropriate length.

Following placement of padding between the fingers and toes, stockinette is often used as the next protective layer in self-made splints (see Fig. 51-3*A*). The stockinette should extend at least 10 to 15 cm beyond the area to be splinted at both ends of the extremity. Later, after plaster has been applied, the stockinette can be folded back over the ends of the splint to create smooth, padded rims. Folding back the stockinette can also help hold the splint in place when applying elastic bandages (see Fig. 51-3*C*). Care is needed to avoid pressure damage from pulling the stockinette too tightly over bony prominences, such as the heel. Wrinkling over flexion creases should also be avoided by slitting and overlapping the stockinette at bony prominences. One may also use two separate pieces of stockinette (one at each end of the splint) to produce the smooth padded rims. As a general rule, a 3-in-wide stockinette is used for the upper extremity, whereas a 4-in-wide one is used for the lower extremity.

After the stockinette has been properly positioned, Webril should be wrapped around the entire area that will be exposed to plaster. The Webril should be at least 2 to 3 layers thick and each turn should overlap the previous turn by 25% to 50% of its width (see Fig. 51-3*B*). In addition, the Webril should extend 2.5 to 5.0 cm beyond the ends of the splint so that it, too, can be folded back over the splint to help create smooth, well-padded edges (see Fig. 51-3*C*). Extra padding should be placed over areas of bony prominence, such as the radial condyle or the malleoli (Table 51-2). If significant swelling is anticipated, three to four layers of Webril should be applied as padding. Care should be taken to avoid wrinkling because it can result in significant skin pressure when a tight splint is used for a long period. Wrinkles can be eliminated by proportionately stretching or even tearing the side of the Webril that must wrap around the bigger portion of an extremity. Joints that must be immobilized in a 90° position, such as the ankle, make continuous Webril wrapping difficult. To avoid wrinkles in the area of the ankle, the joint should be placed in the proper position before padding. Webril is then wrapped around the malleolar and midtarsal regions first. The

TABLE 51-2 -- Areas of the Upper and Lower Extremity That Require Additional Padding

Upper Extremity
Olecranon
Radial styloid
Ulnar styloid
Lower Extremity
Upper portion of the inner thigh
Patella
Fibular head
Achilles tendon
Medial and lateral malleoli

bare calcaneal region can then be covered with overlapping vertical and horizontal Webril strips until the entire heel region is evenly padded. The same approach can be used in similar areas such as the elbow. The width of Webril that should be used varies depending on the extremity to be splinted. In general, the 2-in width should be used for hands and feet, the 3- to 4-in width for the upper extremity, and the 4- to 6-in width for the lower extremity.

A final caveat when using Webril is to be aware of the potential for ischemic injury. This rare complication is most likely to occur in an extremity that continues to have significant swelling after the patient is released from the ED. Ischemia may result because the concentrically placed Webril can become a constricting band. If this situation is anticipated, it can be prevented easily by cutting through the Webril along the side of the extremity opposite to the plaster splint. The splint is then secured to the extremity in the usual manner. Alternatively, two to three layers of Webril (the same diameter as the plaster) are placed directly over the wet plaster (Fig. 51-4). The Webril-lined splint is then positioned over the area to be immobilized and secured with an elastic bandage.

Plaster Preparation

The choice of plaster setting time depends on the nature of the injury and the expertise of the clinician. Extra-fast-setting plaster is typically used when rapid hardening is desired to help maintain alignment of an acutely reduced fracture. However, for the majority of ED splints, plaster with slower setting times (e.g., Specialist fast-drying) is recommended.[11] Plaster that sets more slowly is easier for some clinicians to use because it affords more leeway in applying and molding

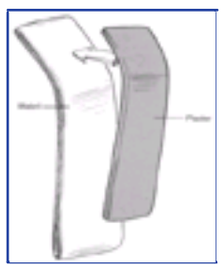

Figure 51-4 Alternative method of Webril application. If significant swelling is anticipated, Webril (the same diameter as the plaster) may be placed directly over the wet plaster, rather than wrapping it around the extremity. The Webril-lined splint is then positioned over the area to be immobilized and secured with an elastic bandage. Sandwiching the plaster strips with Webril will also minimize rigid adherence of the plaster to the elastic wrap.

TABLE 51-3 -- Setting Times of Fast- and Extra-Fast-Drying Plaster

Plaster	Setting Time (min)
Fast-drying	5–8
Extra-fast-drying	2–4

the splint. Furthermore, plaster with a longer setting time produces less heat, thus reducing both patient discomfort and the risk of serious burns. [12] Table 51-3 lists the setting times for commonly used plaster. These setting times are created by adding different substances to the plaster during the production process (Table 51-4). Given plaster with equal setting times, the most important variable affecting the rate of crystallization is water temperature. *Warm water hardens a splint faster than cold water* and should not be used when extra time is needed for splint application.

The ideal length and width of plaster depends on the body part to be splinted and the amount of immobilization required. The best way to estimate length is to lay the dry splint next to the area to be splinted. It is best to use a generous length because wet plaster shrinks slightly from its dry length. Also, if the wet splint is too long, the ends can be folded back easily. The plaster width varies according to the type of splint being made and the body part that is injured, but generally it should be slightly greater than the diameter of the limb to be splinted. Specific recommendations regarding splint length and width are made in sections describing individual splints.

The thickness of a splint depends on the size of the patient, the extremity that is injured, and the desired strength of the final product. An ankle splint may crack quickly and become useless if only eight layers are used, but this thickness may be ideal for a wrist splint. In general it is best to use the minimum number of layers that are necessary to achieve adequate strength. Thicker splints are heavier and more uncomfortable. It is also important to note that plaster thickness is a major determinant of the amount of heat given off during the setting process. With more than 12 sheets of plaster, there is an increased risk of significant burns, especially when using extra-fast-drying plaster, using dipping water with a temperature greater than 24°C, or placing a pillow under or around the extremity for support during the setting process (Table 51-5). For an average-sized adult, upper extremities should be splinted with 8 sheets of plaster, whereas lower extremity injuries generally require 12 to 15 sheets. This layering usually gives the strength necessary for adequate immobilization

TABLE 51-4 -- Effect of Water Temperature and Different Additives on Setting Time of Plaster

Accelerates Setting Time	Slows Setting Time
Reusing dip water	Cool dip water
Higher dip water temperature	Glue
Salicylic acid	Gum
Zinc	Borax
Magnesium	
Copper	
Iron	
Aluminum	
Salt	
Alum	

TABLE 51-5 -- Variables That Increase Heat Production During Crystallization

Major	Minor
Increased splint thickness	High humidity
Setting time*	High ambient temperature
High dip water temperature†	Reusing dip water
Wrapping the extremity for support while drying	

*Faster setting times produce more heat.
†Dip water temperature has been a minor determinant of heat production in some studies.

while reducing the patient's discomfort and the risk of significant burns. In a 136 kg (300 lb) patient, however, up to 20 layers may be required to make a durable ankle splint.

The dipping water should be kept clean and fresh. The use of water that has been used previously for wetting plaster increases the amount of heat given off during crystallization and causes plaster to set more quickly. As a rule of thumb, the temperature of the water should be kept around 24°C. This temperature allows for a workable setting time and has not been associated with an increased risk of significant burns. As the temperature of the dipping water approaches 40°C, the potential for serious burns increases, even at splint thicknesses of less than 12 plies. It is interesting to note that water temperature has been shown to be only a minor consideration in heat production in some studies (see Table 51-5).

Splint Application

The dry splint should be completely submerged in the water until bubbling stops. The splint is removed and excess water is gently squeezed out until the plaster has a wet and sloppy consistency. The splint is placed on a hard table or countertop (a protective covering is recommended to prevent water or plaster damage) and

smoothed out to remove any wrinkles and to ensure uniform lamination of all layers. Lamination helps to increase the final strength of the splint. The splint is placed over the Webril and gently smoothed over the extremity. Plaster is usually somewhat adherent to Webril, but an assistant may be required to hold the splint in place. Once the splint has been properly positioned over the extremity, folding back the underlying stockinette and Webril also helps hold it in place. The splint is secured with an appropriately sized elastic bandage by wrapping in a distal to proximal direction. Finally, the extremity is placed in the desired position and the wet plaster is molded to the contour of the extremity using only the palms of the hand. Finger indentations may cause a ridge that will produce a pressure point.

Molding the wet splint to conform to the body's anatomy is probably the most important, yet the most frequently overlooked, step to ensure adequate immobilization (see Fig. 51-3*E*). The act of molding may cause some pain, and the patient should be forewarned. All manipulation of the wet plaster should be completed before it reaches a thick, creamy consistency. Any movement after this time, also known as the critical period, results in an imperfect crystalline network of calcium sulfate molecules and greatly weakens the ultimate strength of the splint. *While the plaster is setting*, a pillow or blanket should *NOT* be wrapped around the extremity for support. This leads to inadequate ventilation around the splint and greatly increases the amount of heat produced (see Table 51-5).

If an elastic bandage is applied directly over wet plaster, the elastic bandage may be incorporated into the drying plaster, making subsequent removal of the bandage difficult. To make it easier for patients to remove and reapply the splint, a single layer of Webril or roll gauze can be wrapped around the wet plaster loosely before application of the elastic bandage. This prevents the wet plaster from becoming stuck to the elastic bandage. Only one layer of Webril should be used over the plaster because multiple layers have been associated with high drying temperatures.

Before the patient is released from the ED, the splint should be checked for adequate immobilization, and the patient should be observed for any evidence of vascular compromise or significant discomfort. If either occurs, the elastic bandage should be loosened. If the discomfort persists, additional padding should be placed over the painful areas. If this measure, too, is unsuccessful, a new splint should be made, and special attention should be paid to proper molding so that the wet plaster does not become indented. By resting tender tissue, splinting usually relieves discomfort quickly, and patients generally say that they feel better immediately after the splint has been applied. Never release a patient who complains of *increased* pain after a splint has been placed.

After a proper-fitting, comfortable splint has been applied, one may place two strips of tape along each side of the splint to prevent the elastic bandage from slipping. Tape should always be applied over the metal fasteners used to secure the elastic bandages. Note that these objects can be easily swallowed or aspirated by infants and small children. Finally, a sling should be provided for upper extremity injuries, and, if required, crutches should be dispensed (and instructions given for their proper use) for lower extremity injuries.

Patient Instructions

Patients should receive both verbal and written instructions on splint care and precautions. The importance of elevation in helping to decrease pain and swelling should be stressed and demonstrated (most patients do not understand the medical definition of elevation). *At night* a pillow wrapped and secured around a hand or foot will help the patient keep the injured extremity satisfactorily elevated. If the injury is less than 24 hours old, the application of ice bags or cold packs also should be encouraged. It is useless to apply cold packs over plaster, but it can be beneficial if it is applied over Webril and an elastic bandage or directly over an injury if the splint is removed. In theory, cold therapy stiffens collagen and thus reduces the tendency for ligaments and tendons to deform. Cold therapy also decreases muscle spasm and excitability, decreases blood flow (thus limiting hemorrhage and edema), increases the pain threshold, and decreases inflammation. Because the thermal conductivity of subcutaneous tissue is poor, cold packs should be applied for at least 30 minutes at a time. This guideline is in contrast to the popular recommendation of "ice 20 minutes on, 20 minutes off," which does nothing more than cool the skin. Cold packs should not be applied for more than the first 24 to 48 hours because cold can interfere with long-term healing. The patient should be instructed not to stress the splint for at least 24 hours because plaster does not approach optimal strength until evaporation has reduced the water content of the plaster to approximately 21% of its initial hydrated level. This process of removing excess water by evaporation is called *curing* and it generally takes several days to be completed. However, by 24 hours the water content of the plaster has usually been reduced enough to produce a strong resilient splint. In addition, because the chemical process involved in the formation of plaster is reversible, the patient should avoid getting the splint wet. If the injury permits, the splint can be removed for showering and then reapplied. Alternatively, one or more plastic bags may be placed over a splint before showering.

Splints may crack, break, or disintegrate with wear, and such a useless splint should be removed or replaced. Patients should be given general guidelines for length of immobilization and appropriate follow-up care. Long-term immobilization, particularly in the elderly, can produce permanent disability.

It is extremely important for the patient to continue to check for signs of vascular compromise. If the patient experiences a significant increase in pain, any numbness or tingling of the digits, pallor of the distal extremity, decreased capillary refill, or weakness, he or she should be instructed to return to the ED or to see the primary clinician without delay. As with casting, increased pain after splinting is a warning sign that should prompt a return visit— *not telephone advice*. Strong opioids should be avoided during the first 2 to 3 days after splinting to allow pain to prompt a follow-up visit.

UPPER EXTREMITY SPLINTS

Long Arm Splints

Long Arm Posterior Splint

Indications.

The long arm posterior splint (Fig. 51-5) is used to immobilize injuries of the elbow and proximal forearm. It completely eliminates flexion and extension of the elbow, but it does not entirely prevent pronation and supination of the forearm. Therefore, it is not recommended for immobilization of complex or unstable distal forearm fractures unless used in conjunction with a long arm anterior splint. Alternatively, a double "sugar-tong" splint can be applied.

Construction.

The long arm posterior splint is constructed using 8 to 10 layers of 4- to 6-in wide plaster. The splint starts on the posterior aspect of the proximal arm. It runs down the arm to the elbow and then continues along the ulnar aspect of the forearm and hand to the level of the metacarpophalangeal joints. The anterior splint is constructed in the same manner. It mirrors the posterior splint by running down the anterior aspect of the arm to the antecubital fossa, where it continues along the radial aspect of the forearm to the distal radius. The anterior splint is never used alone, but rather is used as an adjunct to the long arm posterior splint to improve immobilization by increasing stability and preventing pronation and supination of the forearm.

Application.

Stockinette and Webril are applied as described previously. A hole should be cut in the stockinette to expose the thumb and extra padding should be placed over the olecranon to prevent pressure injury. The arm is positioned with the elbow flexed to 90°, the forearm neutral (thumb upward), and the wrist neutral or extended slightly (10°–20°). It is helpful to have an assistant hold the wet splint in place, particularly when applying both a posterior and anterior splint. Once the splint has been properly positioned, the ends of the stockinette and Webril are folded back and

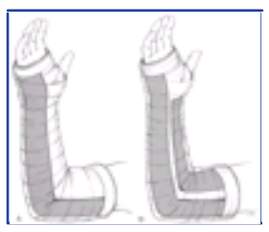

Figure 51-5 Application of a long arm posterior splint. *A*, The posterior portion of the splint begins on the posterior aspect of the proximal humerus. It runs down the arm to the elbow and then continues along the ulnar aspect of the forearm and hand to the distal metacarpals. The elbow is flexed at a 90° angle, the forearm is in the neutral (thumb up) position, and the wrist is in a neutral position or slightly (10°–20°) extended. *B*, Adding an anterior splint. The anterior splint mirrors the posterior splint by running down the anterior aspect of the arm to the antecubital fossa, where it continues along the radial aspect of the forearm and hand to the distal radius. The anterior splint is never used alone, but rather as an adjunct to the long arm posterior splint. It improves immobilization by increasing stability and preventing pronation and supination of the forearm.

the splint is secured in place using 2-, 3- to 4-in elastic bandages. Finally, the sides of the splint are folded up to create a gutter configuration and the splint is carefully molded using the palms of the hand. The fingers and thumb should remain free to prevent stiffness from unnecessary immobilization.

Double Sugar-Tong Splint

Indications.

Like the long arm posterior splint, the double sugar-tong splint (Fig. 51-6) is used to immobilize injuries of the elbow and forearm. However, because it prevents pronation and supination of the forearm, it is preferable for some fractures of the distal forearm and elbow.

Construction.

The splint consists of two separate pieces of plaster, a forearm splint and an arm splint. Each piece is constructed using 8 layers of 3- to 4-in plaster. The forearm portion of the splint runs from the metacarpal heads on the dorsum of the hand, along the dorsal surface of the forearm around the elbow. It continues along the volar surface of the forearm to the palm of the hand, stopping at the level of the metacarpophalangeal joints. The arm portion of the splint begins on the anterior aspect of the proximal humerus. It runs down the arm over the forearm splint and around the elbow. It then continues up the posterior aspect of the arm, once again going over the forearm splint, until it reaches the starting point.

Application.

Use of stockinette, Webril, and positioning are similar to those described for application of a long arm posterior splint. The two splints are secured in place using two 3- to 4-in elastic bandages starting at the forearm splint at the hand. Once secure, the arm portion of the splint is wrapped beginning at the proximal end. The fingers and thumb should remain free to avoid stiffness.

Forearm and Hand Splints

Volar Splint

Indications.

The volar splint (Fig. 51-7*A*) is used to immobilize a variety of soft tissue injuries of the hand and wrist. It is also used for temporary immobilization of triquetral fractures, lunate and perilunate dislocations, and second through fifth metacarpal head fractures. For these more serious injuries, some clinicians prefer to add a dorsal splint to create a more stable bivalve effect (Fig. 51-7*B*). Because the volar splint does not completely eliminate pronation and supination of the forearm, it may not be ideal for fractures of the distal radius and ulnar, although many clinicians use this splint for nondisplaced or minimally displaced distal ulnar and radial fractures.

Construction.

The splint is constructed using 8 to 10 layers of 3- to 4-in wide plaster. The splint begins in the palm at the metacarpal heads and extends along the volar surface of the forearm to just proximal to the elbow. If there is an injury to any of the fingers, the splint may be extended to incorporate the involved digit.

Application.

Stockinette and Webril should be applied as described previously. A hole should be cut in the stockinette to expose the thumb. In addition, Webril or gauze should be placed between any digits that are going to be immobilized. The forearm is placed in the neutral position (thumb upward) with the wrist extended slightly (10°–20°). Wrist flexion should be avoided. After the wet plaster has been properly positioned, the ends of the stockinette and Webril are folded back and a 3- or 4-in elastic bandage is used to hold the splint in place. The sides of the splint are folded up, creating a gutter effect, and the plaster is carefully molded to conform to the contours of the palm and wrist. Some

Figure 51-6 An alternative to the long arm posterior splint is a double sugar-tong splint. This splint immobilizes the elbow and prevents pronation and supination of the forearm. The splint consists of two separate pieces of 4-in plaster, a forearm splint, and an arm splint. The elbow is flexed at a 90° angle, the forearm is in the neutral (thumb up) position, and the wrist is in a neutral position or slightly (10°–20°) extended. The forearm portion of the splint is applied first. It runs from the metacarpal heads on the dorsum of the hand, along the dorsal surface of the forearm, and around the elbow. It continues along the volar surface of the forearm, stopping at the level of the metacarpophalangeal joints. The arm portion of this splint begins on the anterior aspect of the proximal arm. It runs down the arm over the forearm splint and around the elbow. It then continues up the posterior aspect of the arm, once again going over the forearm splint until it reaches the starting point. The fingers and thumb should remain free to avoid stiffness.

clinicians prefer to extend the splint to the fingertips and then fold the wet plaster back toward the palm, allowing the fingers to "grasp" the rounded distal end when at rest. In any event, the thumb and fingers should be free to move unless they are injured and are being intentionally immobilized by the splint.

Sugar-Tong Splint

Indications.

The sugar-tong splint (Fig. 51-8) is used for fractures of the distal radius and ulna. The advantage of this splint over the volar splint is prevention of pronation and supination of the forearm. In addition, it immobilizes the elbow, which is desirable for the first few days following a distal forearm fracture.

Construction and Application.

The splint is constructed and applied the same way as the forearm portion of the double sugar-tong splint, described earlier.

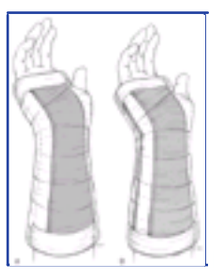

Figure 51-7 Application of a volar splint. *A*, The splint begins in the palm at the metacarpal heads and extends along the volar surface of the forearm to a point just proximal to the elbow. If any of the fingers are injured, the splint may be extended to incorporate the involved digits. The forearm is placed in the neutral position (thumb upward) with the wrist slightly (10°–20°) extended. Wrist flexion should be avoided. *B*, For more serious injuries, an additional dorsal slab may be used to create a bivalve splint.

Thumb Spica Splint

Indications.

The thumb spica splint (Fig. 51-9) is used to immobilize injuries to the scaphoid, lunate, and thumb and fractures of the first metacarpal. It is also used in the treatment of de Quervain tenosynovitis. Traditionally, a thumb spica splint or cast was thought to be a requirement to properly immobilize scaphoid fractures; however, there is no totally agreed-upon standard. Clay and coworkers state that the optimal method of casting scaphoid fractures has not been definitively established. They were unable to prove a difference in patient comfort, recovery of function, or incidence of nonunion between a Colles' cast and a traditional scaphoid cast that included the thumb.[13]

The incidence of nonunion of scaphoid fractures is about 10%, regardless of the type of immobilization in the ED, but it is greatest with unstable proximal pole fractures. Since some scaphoid fractures heal poorly under the best of circumstances, it seems prudent to provide thumb immobilization in the initial splinting. Failure to do so, such as when a "sprained wrist" is suspected, should not be construed as beneath the accepted standard of care. Most volar splints will at least partly immobilize the base of the thumb, so the discussion may be moot.

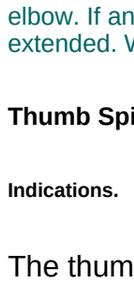

Figure 51-8 Application of a forearm sugar-tong splint. The splint runs from the metacarpal heads on the dorsum of the hand, along the dorsal surface of the forearm, and around the elbow. It continues along the volar surface of the forearm stopping at the level of the metacarpophalangeal joints. The elbow is flexed at a 90° angle, the forearm is in the neutral (thumb up) position, and the wrist is in a neutral position or slightly (10°–20°) extended. The advantage of this splint over the volar splint is immobilization of the elbow and prevention of pronation and supination of the forearm.

Construction.

The splint is constructed using 8 layers of 3-in wide plaster. The splint extends from just distal to the interphalangeal joint of the thumb to the mid-forearm.

Application.

The forearm is placed in the neutral position with the wrist extended 25° and the thumb in the wine glass position (Fig. 51-10). Stockinette and Webril are applied from the base of the palm to the mid-forearm. It may be difficult to place stockinette around the thumb. Instead, a hole can be cut in the stockinette to expose the thumb. The thumb is then padded with small vertical strips of Webril or wrapped with 2-in Webril. The dry plaster is then placed over the radial aspect of the forearm

from just beyond the thumb interphalangeal joint to the mid-forearm. Once in position, the location of the first metacarpophalangeal joint is marked and a small (1 to 2 cm) perpendicular cut is made 1 cm distal to the mark on each edge of the plaster (see Fig. 51-9 inset). If necessary the plaster distal to the notch may be tapered slightly. This will allow the splint to be molded around the thumb without creating a buckle in the plaster. The plaster is then dipped and secured in place with a 2- or 3-in elastic bandage. It is important to carefully mold the wet plaster around the thumb and palm and to maintain the thumb in the wine glass position while the plaster is drying.

Figure 51-9 Application of a thumb spica splint. The splint extends from just distal to the interphalangeal joint of the thumb to the mid-forearm. The forearm is placed in the neutral position with the wrist extended 25° and the thumb in the wine glass position (see Fig. 51-10). *Inset, A* small (1- to 2-cm) perpendicular cut is made 1 cm distal to the first metacarpophalangeal joint on each edge of the plaster to allow molding of the splint around the thumb without creating a buckle in the plaster.

Ulnar Gutter Splint

Indications.

The ulnar gutter splint (Fig. 51-11) is used to immobilize fractures and serious soft tissue injuries of the little and ring fingers and fractures of the neck, shaft, and base of the fourth and fifth metacarpals.

Construction.

The splint is made using 6 to 8 layers of 3- to 4-in plaster. It incorporates both the little and ring fingers. It runs along the ulnar aspect of the forearm from just beyond the distal interphalangeal joint of the little finger to the mid-forearm.

Application.

Stockinette and Webril are applied as usual. Additional Webril or gauze should be placed between the little and ring fingers to prevent maceration of the skin. The forearm is in the neutral position with the wrist in slight extension (10°–20°), the metacarpophalangeal joints in 50° of flexion, and the proximal and distal interphalangeal joints in slight (10°–15°) flexion. When immobilizing a metacarpal neck fracture (i.e., boxer's fracture), the metacarpophalangeal joint should be flexed to 90°. Once in the proper position, the sides of the splint are folded up to form a gutter. The ends of the stockinette and Webril are then folded back to help hold the splint while it is secured in place with a 2- or 3-in elastic bandage.

Figure 51-10 The wine glass position, which is a safe splint position for the hand. The wrist should allow alignment of the thumb with the forearm, the metacarpophalangeal joint should be moderately flexed, and the interphalangeal joints should be only slightly flexed. The thumb should be abdicted away from the palm.

Radial Gutter Splint

Indications.

The radial gutter splint (Fig. 51-12) is used to immobilize fractures and serious soft tissue injuries of the index and long fingers and fractures of the neck, shaft, and base of the second and third metacarpals.

Construction.

The splint is made using 6 to 8 layers of 3- to 4-in plaster. It runs along the radial aspect of the forearm from just beyond the distal interphalangeal joint of the index finger to the mid-forearm.

Application.

Stockinette (with a hole cut to expose the thumb) and Webril are applied as previously described. Additional Webril or gauze should be placed between the index and long fingers to prevent maceration of the skin. The forearm is in the neutral position with the wrist in slight extension (10°–20°), the metacarpophalangeal joints in 50° of flexion, and the proximal and distal interphalangeal joints in slight (10°–15°) flexion. When immobilizing a metacarpal neck fracture, the metacarpophalangeal joint should be flexed to 90°. The dry plaster is placed over the extremity and the location of the thumb is marked. A hole is cut in the dry plaster to expose the thumb. The plaster is then dipped and positioned over the extremity. The ends of the stockinette and Webril are folded back to help hold the splint while it is secured in place with a 2- or 3-in elastic bandage.

Figure 51-11 Application of an ulnar gutter splint. The ulnar gutter splint incorporates both the little and ring fingers. Webril or gauze should be placed between the digits to prevent maceration of the skin. The splint runs along the ulnar aspect of the forearm from just beyond the distal interphalangeal joint of the little finger to the mid-forearm. The forearm is in the neutral position with the wrist in slight extension (10°–20°), the metacarpophalangeal joint in 50° of flexion, and the proximal and distal interphalangeal joint in slight (10°–15°) flexion. When immobilizing a metacarpal neck fracture, the metacarpophalangeal joint should be flexed to 90°.

Finger Splints

Fingers are splinted following sprains, fractures, tendon repair, or infection. Minor finger sprains can often be managed with dynamic splinting (e.g., buddy taping) (Fig. 51-13) or a commercially available foam splint with aluminum backing (one-surface splint) (Fig. 51-14), but fractures, tendon repairs, and some soft tissue injuries benefit from formal splinting (e.g., thumb spica, ulnar and radial gutter splints). Specific conditions, such as mallet finger, require a specialized splint (Fig. 51-15). When complete immobilization of a finger is required (e.g., unstable phalangeal fractures), an "outrigger" finger splint that incorporates the wrist may be used (Fig. 51-16).

Sling, Swathe and Sling, Shoulder Immobilizer

Sling

The sling is used to maintain elevation and provide immobilization of the hand, forearm, and elbow. It is most often used in conjunction with a plaster splint or cast. There are a number of commercially available slings to choose from. Many of these are fairly economical and simple to use, while others are very expensive and do not allow the versatility of a simple, inexpensive triangular muslin bandage. When applying a sling, it is important to have adequate support of the wrist and hand (Fig. 51-17). A sling that is too short will allow the wrist and hand to hang down (ulnar deviate) and can result in ulnar nerve injury.

Swathe and Sling, Shoulder Immobilizer

The swathe and sling is the treatment of choice for most proximal humerus fractures and shoulder injuries, such as reduced dislocations. The sling supports the weight of the arm, and the swathe immobilizes the arm against the chest wall to minimize shoulder motion. In most EDs, the swathe and sling has been replaced by the commercially available shoulder immobilizer (Fig. 51-18). Its advantage is that it may be removed for showering and range-of-motion exercises and is easily reapplied by the patient (a desirable option in the care of a shoulder dislocation). If used for more than a few days the axilla should be padded to absorb moisture and decrease skin chafing.

Figure 51-12 Application of a radial gutter splint. The radial gutter splint incorporates both the index and long fingers. Webril or gauze should be placed between the digits to prevent maceration of the skin. The splint runs along the radial aspect of the forearm from just beyond the distal interphalangeal joint of the index finger to the mid-forearm. The forearm is in the neutral position with the wrist in slight extension (10°–20°), the metacarpophalangeal joint in 50° of flexion, and the proximal and distal interphalangeal joint in slight (10°–15°) flexion. When immobilizing a metacarpal neck fracture, the metacarpophalangeal joint should be flexed to 90°.

The Velpeau bandage is a sling and swathe device that positions the forearm diagonally rather than horizontally across the chest with the hand elevated to the level of the shoulder. This offers no particular advantage over a standard sling and swathe, is difficult to apply, cannot be removed easily, and is not well tolerated for prolonged immobilization.

Figure-of-8 Clavicle Strap

Clavicle fractures have been traditionally treated with an uncomfortable and complex figure-of-8 bandage. Despite its widespread use, this device has never been proved superior to a simple sling (in terms of cosmesis, functional outcome, or pain relief). [14] [15] Indeed, use of the figure-of-8 dressing should be discouraged because it may actually promote nonunion or increase the deformity at the fracture site; it is very uncomfortable; it prohibits bathing, often causing chafing and discomfort in the axilla; and it may predispose to axillary vein thrombosis. [4] Although some orthopedists continue to recommend the figure-of-8 bandage, a simple sling is sufficient to treat most clavicular fractures.

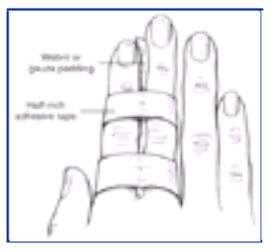

Figure 51-13 "Buddy tape" technique. Taping between the digital joints allows the normal adjacent finger to protect the collateral ligament of its injured neighbor. Webril should be placed between the digits to prevent maceration of the skin.

Pitfalls of Hand Dressings and Splints

The two most common problems with hand dressings are putting them on too tightly and leaving them on too long (Table 51-6). One must be especially careful to avoid wrapping elastic bandages too snugly. The patient should be instructed to loosen an elastic bandage if it feels too tight. The patient should always have access to emergency follow-up care. It is often advisable to start patients on a regimen of early protected motion. This means that the patient removes the splint for a specified period, does a prescribed exercise, and then replaces the splint. A splint is not an all-or-none device, and the patient is generally weaned slowly from it before it is discarded entirely. A stiff hand is a nonfunctional

Figure 51-14 Dorsal aluminum foam splint. The bone is subcutaneous dorsally, and splints here afford better immobilization of the digit. The dorsal splint also allows preservation and use of tactile sense, which encourages function and better splint acceptance on the part of the patient.

Figure 51-15 Splinting a mallet finger. The dorsal splint immobilizes only the distal interphalangeal joint. This allows use of the finger. Hyperextension of this joint predisposes to skin sloughing and should be avoided. The patient should be advised not to flex the joint during splint changes.

one, and stiffness is often a consequence of prolonged immobilization. It is important for the patient to be made aware of his or her responsibility for the injured hand.

LOWER EXTREMITY SPLINTS

Knee Splints

Knee Immobilizer

Indications.

The knee immobilizer (Fig. 51-19) is commonly used for mild to moderate ligamentous and soft tissue injuries of the knee. It is removable and extremely easy to apply, making it popular among patients and clinicians alike. In many EDs it has almost totally replaced the more bulky plaster splint. Its use should be restricted to injuries that do not require immediate surgical intervention, traction, or

Figure 51-16 "Outrigger" finger splint for complete immobilization of the finger. A padded aluminum splint is incorporated into the middle of a plaster splint, forming an outrigger configuration. The plaster splint is applied to the dorsum of the hand and wrist with an elastic bandage; the finger is then taped to the aluminum splint.

casting. For these injuries, in which temporary but more complete immobilization is needed, a plaster knee splint can be used because it provides better stabilization and costs much less than a knee immobilizer. The exact scientific benefit of the knee immobilizer is poorly studied and difficult to document. However, it clearly helps relieve pain and, at least theoretically, hastens healing.

Application.

The knee immobilizer is available in small, medium, large, and extra-large sizes. To choose the appropriate size, the knee immobilizer is placed next to the injured leg so that the tapered end lies distal to the patient's knee; if present, the cutout patellar area on the anterior aspect of the splint lies adjacent to the knee. In this position, the splint should extend distally to within a few inches of the malleoli and proximally to just below the buttocks crease. To apply the knee immobilizer, the open splint is slid under the injured extremity and firmly secured in place using the Velcro straps. The knee immobilizer can be applied directly over clothing, obviating the need to remove or cut the patient's pants.

Posterior Knee Splint

Indications.

In many EDs, the knee immobilizer has virtually replaced the plaster knee splint for mild to moderate injuries to the knee. However, the plaster knee splint can be particularly useful in patients whose extremities are too large for the knee immobilizer, in the treatment of angulated fractures, or for temporarily immobilizing other knee injuries that require immediate operative intervention or orthopedic referral. The posterior (gutter) knee splint (Fig. 51-20*A*) is the type most commonly applied, but as an alternative, two parallel splints can be placed along each side of the leg and foreleg, creating a bivalve effect (see Fig. 51-20*B*), or a long leg U-splint can be applied (see later). The bilateral knee splint is slightly more difficult to apply than the posterior knee splint, but it may provide better immobilization of the lateral and medial collateral ligaments and can be used for injuries to these structures.

Construction.

The knee splint is made with 12 to 15 layers of 6-in plaster. It should run from just below the buttocks crease to approximately 5–8 cm above the malleoli. The sides of the splint are folded upward to form a gutter configuration.

Application.

A stockinette should be placed in the usual manner, and the leg should be well padded with 4- to 6-in Webril. If available, an assistant can help elevate the leg and hold the splint in place while it is being secured with 4- or 6-in elastic bandages. If no aide is available, the patient can be placed in the prone position and the splint laid on the posterior surface of the extremity. The leg is then wrapped in the usual manner without the need for special support of the applied plaster. Also, while in the prone position, the patient's toes can elevate the lower part of the leg off the bed, allowing sufficient room to wrap the Webril and elastic bandages around the injured extremity.

Jones Compression Dressing

Indications.

A Jones compression dressing is commonly used for short-term immobilization of soft tissue injuries of the knee. It immobilizes and compresses the knee, reducing both pain and swelling. However, because it does allow slight flexion and extension of the knee, it should not be used for injuries that require strict immobilization. In addition, it is difficult to maintain the splint for more than a few days.

Figure 51-17 *A*, Step-wise application of a triangular muslin sling. (1) Place tip X over the uninjured shoulder. (2) Bring tip Y over the injured shoulder to enclose the arm. (3) Draw tip Z around the front and pin. *B*, Completed triangular muslin sling (Note: When applying a sling, it is important to have adequate support of the wrist and hand. A sling that is too short will allow the wrist and hand to hang down [ulnar deviate] and can result in ulnar nerve injury).

Figure 51-18 The shoulder immobilizer is used for most proximal humerus fractures and shoulder injuries. It may be removed for showering and range-of-motion exercises and is easily reapplied by the patient.

Construction.

A Jones dressing is made using 6-in Webril and elastic bandages.

Application.

To apply a Jones dressing, the patient is placed on a stretcher, lying supine. If available, an assistant can elevate the patient's leg to facilitate wrapping. If no help is available, a pillow placed under the patient's heel should suffice. Webril is then wrapped around the extremity from the groin to a few inches above the malleoli. Two or 3 layers of Webril can be used, and each turn should overlap the previous turn by 25% to 50%. The elastic bandage (two are usually required) is then wrapped around the Webril. If more support is required, the process can be repeated with another two to three layers of Webril held in place by additional elastic bandages.

Ankle Splints

Posterior Splint

Indications.

The posterior ankle splint (Fig. 51-21) is one of the most common splints applied in the ED. As noted in the introduction, the entire concept of splinting an acutely sprained ankle has been questioned, with no firm evidence to support a better outcome of any type of splinting or casting versus functional management (early mobilization with an external support). Nonetheless, an acutely sprained ankle is painful, and if nothing else, splinting for a few days will alleviate pain.

The posterior splint is used primarily to immobilize severe ankle sprains, fractures of the distal fibula and tibia, and reduced ankle dislocations. It can also be used for

TABLE 51-6 -- Useful Estimates of Splint Times for Various Hand Problems

Injury	Splint Type	Immobilization Time*
Mallet finger	FIN†	8 wk
Boutonnière deformity	FIN	6 wk
Distal phalanx—soft tissue	FIN	1 to 2 wk
Extensor tendon	DHWF‡	3 wk
Sprain-strain§		
Interphalangeal joint	FIN	1–2 wk
Wrist	DHWF	1–2 wk
Hand burn	DHWF	5–7 wk
Infection		
Digit	DHWF	5–7 day
Hand	DHWF	5–7 day
Severe hand contusion	DHWF	5–7 day
Fracture		
Distal phalanx	FIN	2–3 wk
Middle phalanx	FIN	2–3 wk
Proximal phalanx	DHWF	2–3 wk
Metacarpal	DHWF	2–3 wk
Carpal tunnel	DHWF	Night only
de Quervain disease	DHWF	2–3 wk
Trigger finger	FIN	Night only

*These are average times only. Every patient is treated as an individual when a splint is used. Clinical judgment is critical.
†Finger splint.
‡Digit-hand-wrist-forearm splint.
§The diagnosis of a sprain should be made only after a thorough effort has been made to rule out a fracture or dislocation. This is particularly true in the wrist.

fractures of the tarsal and metatarsal bones or for other foot conditions that require immobilization. In particularly severe or unstable injuries, an additional anterior splint may be used to provide extra immobilization resembling that of a formal cast (Fig. 51-22). For severe lateral or bilateral ligamentous injuries, a U-splint or stirrup splint (see later) may be added to the posterior splint for increased immobilization. With minor soft tissue injuries, patients may have partial weight bearing on ankle splints after 24 hours. If the patient will be bearing weight, a cast shoe over the splint makes it easier to walk. In addition, a cast shoe increases the longevity of the splint because walking on an unprotected splint quickly destroys the device. Generally, walking on the splint is prohibited if immobilization for more than 2 or 3 days is desired.

Construction.

The posterior splint is made using 4- to 6-in-wide plaster strips. It should extend from the plantar surface of the great toe or metatarsal heads along the posterior surface of the foreleg to the level of the fibular head. If it hurts to move the toes, they should be incorporated into the splint (after padding is placed between the digits). It is a common mistake to apply a posterior splint that does not extend far enough to support the ball of the foot. Fifteen to 20 layers should be used if partial weight bearing is allowed because this splint frequently breaks or cracks when walked on. [16]

Application.

The easiest way to apply a posterior splint is to place the patient in the prone position with the knee and ankle flexed at a 90° angle. Failure to place the ankle in a 90° angle results in a plantar-flexed splint. The supine patient may help maintain the ankle in a 90° angle by pulling up on the foot with a wide stockinette stirrup. Flexing the knee to a 90° angle relaxes the gastrocnemius muscle and facilitates ankle motion. With the knee and ankle in the proper position, stockinette may be applied and the foot and leg padded with Webril as described earlier. Extra padding is used

Figure 51-19 The Velcro strap bulky knee immobilizer is easily removed and readily applied by the patient. It can be worn over clothes.

over bony prominences, particularly the malleoli. Again, Webril or gauze is placed between the toes if they are to be included in the splint. The wet plaster is then laid over the plantar surface of the foot and secured in place by folding back the ends of the stockinette and wrapping with one or two 4-in-wide elastic bandages. The wet plaster is carefully molded around the malleoli and instep to ensure maximum comfort and immobilization. The toes should be left partially exposed for later examination of color and capillary refill.

Anterior-Posterior Splint

Indications.

The anterior splint is never used by itself, but it can augment a posterior splint, creating a bivalve effect (see Fig. 51-22). It is used for serious fractures and soft tissue injuries of the ankle.

Construction.

A piece of plaster should be cut several centimeters shorter than the one used for the posterior splint, but because this splint does not bear weight, only 8 to 10 layers are required.

Application.

The patient should be positioned and padded as for the posterior splint. After the wet posterior splint has been applied, the anterior splint is placed over the anterior aspect of the ankle and foreleg parallel to the posterior splint. The two are then held in place with elastic bandages as described earlier for the posterior splint alone. An assistant is needed to apply the anterior-posterior splint because it is extremely difficult to hold both splints in place

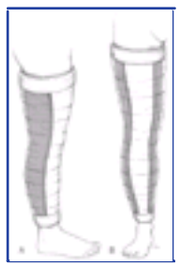

Figure 51-20 Application of a posterior knee splint. *A*, The posterior knee splint runs from just below the buttocks crease to approximately 2 to 3 cm above the malleoli. *B*, Alternatively, two parallel splints can be placed along each side of the leg and foreleg, creating a bivalve effect.

while wrapping the elastic bandages. Once secured, both splints are carefully molded over the instep and ankle joint.

U-Splint (Stirrup Splint)

Indications.

The U-splint or stirrup splint (Fig. 51-23) is used primarily for injuries to the ankle. It functions like the posterior splint, and either of the two provides satisfactory ankle immobilization. In one study that compared these splints in normal volunteers, the U-splint allowed less plantar flexion and broke less often with plantar flexion than the posterior splint.[16] Also, because it actually covers the malleoli, the U-splint may protect the medial and lateral ligamentous area from further injury better than the posterior splint.

Construction.

The U-splint is made using 4- or 6-in-wide plaster strips. The splint passes under the plantar surface of the foot from the calcaneus to the metatarsal heads and extends up the medial and lateral sides of the foreleg to just below the level of the fibular head.

Application.

The patient is positioned, and the extremity is padded as described for the posterior splint. If both posterior and U-splints are used, the posterior splint is applied first. The wet plaster is laid across the plantar surface of the foot between the calcaneus and metatarsal heads with the sides extending up the lateral and medial aspects of the foreleg. The plaster is secured in place with 4-in elastic bandages. The elastic bandage should be wrapped around the extremity starting at the metatarsal heads and continuing around the ankle using a figure-of-8 configuration. Once the ankle has been wrapped, another 4- or 6-in elastic bandage can be used to secure the remainder of the splint in place. The splint should be carefully molded around the malleoli. The plaster may overlap on the anterior aspect of the ankle; this overlap does not interfere with the splint's ability to accommodate further swelling.

Semirigid Orthosis

Indications.

In patients with sprains of the lateral ankle associated with a stable joint, the use of a functional brace with early mobilization is frequently more comfortable, and results in an earlier return to normal function than complete immobilization in a plaster splint or cast. [17] [18] [19] [20] [21] [22] [23] Consequently, functional bracing with early mobilization has become the standard of care. However, it should be pointed out that there is no documented difference in long-term outcome between the two methods of treatment.

Application.

Most functional ankle braces resemble a U-splint with air bladders (Aircast, Inc., Summit, NJ) or foam padding (DeRoyal Inc., Powell, TN) for cushioning the malleoli. The braces are secured about the ankle by Velcro straps. The device is worn within the patient's shoe over a sock and appears to eliminate ankle instability.

Hard Shoe (Cast or Reese Shoe)

Indications.

A hard shoe can help reduce the pain associated with ambulation in patients with fractures or soft tissue injuries to the foot. This device can also be used over a splint or cast to allow partial weight bearing.

Application.

If the cast shoe is going to be used by a patient with a fractured toe, the injured digit should first be "buddy taped" to the adjacent toe. After this is done, the patient merely slips on the hard shoe like a sandal. The shoe is then fastened with ties or Velcro straps.

Soft Cast

Indications.

A "soft cast" is basically a modified Jones compression dressing. It is useful for minor ligamentous and soft tissue injuries of the foot and ankle that do not require prolonged or complete immobilization. A soft cast can help reduce the pain and swelling often associated with mild ankle sprains and gives support for early weight bearing.

Construction.

A soft cast is made using 3- or 4-in Webril and elastic bandages.

Application.

A soft cast is as simple to apply as the Jones compression dressing. To begin, the patient is placed in a supine position with the foot and ankle extending off the end of the stretcher. Alternatively, the leg can be elevated by an assistant or by placing pillows under the knee and foreleg. The ankle and foot are then wrapped with two to three layers of Webril, starting at the metatarsal heads and continuing around the ankle in a figure-of-8 configuration. The Webril should extend 5 to 7 cm above the malleoli and, as discussed earlier, should overlap by 25% to 50% of its width. After the Webril is in place, an elastic bandage is wrapped around the foot and ankle in a similar fashion. Additional layers of Webril and elastic bandages are seldom required.

Unna Boot Dressing

Ligamentous injuries of the ankle may be treated with a cloth dressing impregnated with calamine gelatin-zinc oxide (Dome-Paste or Unna Boot) (Fig. 51-24).[24] This moist dressing is applied like that for a rolled cast in a figure-of-8

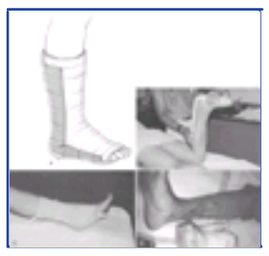

Figure 51-21 Posterior ankle pitfalls. *A*, Proper application of a posterior ankle splint. This splint extends from the plantar surface of the great toe (or metatarsal heads) along the posterior surface of the foreleg to the level of the fibular head. The ankle should be at a 90° angle. *B,* The most convenient way to apply an ankle splint is to have the patient lie prone and bend the knee to a 90° angle, thereby relaxing the calf muscles. The ankle should be at a 90° angle so that the foot is flat for partial weight bearing. *C, Incorrect splint application.* There are three things wrong with this posterior ankle splint: (1) It does not extend distally enough to support the entire foot. (2) The ankle is not maintained at a 90° angle. (3) The edges and ankle area are not molded. Overall the splint is sloppy and ineffective. *D*, The problem with this splint is that it was intended to be used for only a few days, but the patient wore it for 3 weeks. Note the resultant full-thickness skin loss. No padding was used under the premade splint. Skin grafting was eventually required.

design, from the metatarsal heads to the distal foreleg. This dressing remains moist for a few days (it should be covered with a gauze roll or an elastic bandage) and dries to a leathery consistency. It requires minimal maintenance or patient compliance, is lightweight, can be worn under a shoe, and is associated with few complications. It is cut off with scissors by the patient after 5 to 10 days, depending on symptoms. This dressing is nonyielding; hence, evaluation for ischemia is essential in the edema-prone extremity. The dressing (unna boot) is also useful for covering chronic stasis ulcers and averting patient manipulation of lower extremity wounds.

COMPLICATIONS OF SPLINTS

Ischemia

A compartment syndrome leading to ischemic injury and ultimately to a Volkmann ischemic contracture is the most worrisome complication of cylindrical casts. Although the risk of ischemia is drastically reduced with splinting, Webril or elastic bandages can cause significant constriction. To reduce the likelihood of this occurring, the elastic bandage should not be excessively tight. If the patient has a high-risk injury, the

Figure 51-22 Application of an anterior-posterior ankle splint. In particularly severe or unstable injuries, an anterior splint may be added to a posterior ankle splint to provide extra immobilization resembling that of a formal cast. The anterior splint is never used by itself, but it can augment a posterior splint, creating a bivalve effect. The ankle should be at a 90° angle.

Webril may be cut lengthwise before the plaster is applied. Elevation, no weight bearing, and application of cold packs should be stressed to each patient. Furthermore, signs and symptoms of vascular compromise should be explained carefully, and all patients whose injuries have the potential for significant swelling or loss of vascular integrity should receive follow-up in the first 24 to 48 hours. *Complaints of increasing pain under a splint should not be ignored by the clinician. Patients with splint-related discomfort should re-evaluated clinically and should not be treated with a telephone prescription for opioid analgesics.*

Heat Injury

Fiberglass splints produce minimal heat when drying, but plaster generates considerable heat as it hardens. Many clinicians are unaware of the potential for drying plaster to produce second-degree burns.[25] Thermal injury can occur with both cylindrical casts and plaster splints. Some clinicians have reported a higher incidence of burns with the use of plaster splints, although the reasons for this are unclear.[25] [26] Table 51-5 lists factors that can increase the amount of heat produced during plaster recrystallization. Their effects are additive, and this fact should be taken into account when applying a splint. For example, if 15 sheets of plaster are needed for strength in a particular splint, one should not increase the heat production further by using extra-fast-drying plaster or by reusing warm dip water. To avoid plaster burns, use only 8 to 12 sheets of plaster when possible, use fresh dip water with a

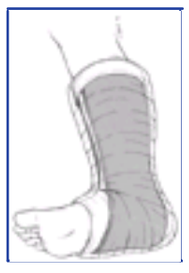

Figure 51-23 The U-splint (AKA sugar-tong or stirrup splint) is also used primarily for injuries to the ankle. The splint passes under the plantar surface of the foot, extending up the medial and lateral sides of the foreleg to just below the level of the fibular head. The ankle should be at a 90° angle. For immobilization of the knee, the sides of the splint may be extended proximally to the groin, creating a long leg splint.

temperature near 24°C, and never wrap the extremity in a sheet or pillow during the setting process. Peak temperatures usually occur between 5 and 15 minutes after plaster wetting.

The patient should be warned that the hardening process produces warmth. The heat of drying may produce pain in patients with hemophilia-related hemarthroses. Splinting these patients may require that the plaster splint only be placed long enough to verify proper fit; the splint is then reapplied after setting (and cooling) of the plaster. *If any patient complains of significant burning while the plaster is drying, do not ignore this complaint!* Immediately remove the splint, and promptly cool the area with cold packs or cool water. Patients with vascular insufficiency or sensory deficits (e.g., diabetic neuropathy, stroke) are at high risk for plaster burns and require close observation during the drying process.

Pressure Sores

Pressure sores are an uncommon complication of short-term splinting.[27] They can result from stockinette wrinkles, irregular wadding of Webril, incorrectly padded or unpadded bony prominences, irregular splint ends, plaster ridges, or indentations produced from using the fingers rather than the palms to smooth and mold the wet plaster. Attention to detail during padding and splinting reduces the incidence of pressure sores. However, whenever a patient complains of a persistent pain or burning sensation under any part of a splint, the splint should be removed and the symptomatic area inspected closely. The padding that is incorporated in premade plaster and fiberglass splints is generally all that is needed for safe short-term splinting. However, the life of a splint applied in the ED may be longer than intended by the clinician; therefore, it is

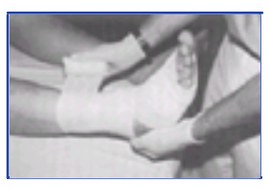

Figure 51-24 The Unna boot provides effective immobilization of an ankle soft tissue injury. It is applied from a semisolid paste roll. The wrap is then covered with gauze or an elastic bandage. The entire dressing can be cut off by the patient at home. For similar short-term immobilization without plaster, a modified Jones dressing can be used. Webril is wrapped around the ankle and it is covered with an elastic bandage.

prudent to err on the side of additional padding when putting splints on patients who will overuse the splint, such as those who will not use crutches, or for those who may not have ready access to follow-up.

Infection

Bacterial and fungal infections can occur under a splint.[28] [29] Infection is more common in the presence of an open wound but may occur with intact skin or develop in a skin lesion produced by prolonged splinting. The moist, warm, and dark environment created by the splint is an excellent nidus for infection. Toxic shock syndrome has been rarely reported from a staphylococcal skin infection that has clandestinely developed under a splint or cast. Also, it has been shown that bacteria can

multiply in slowly drying plaster. To avoid infection, all wounds should be cleaned and debrided before splint application, and clean, fresh tap water should be used for plaster wetting. In some instances, it is preferable to apply a removable splint that allows for periodic wound inspection or local wound care.

Dermatitis

Occasionally patients develop a rash under a plaster cast or splint. [30] [31] [32] [33] [34] Allergy to plaster is exceedingly rare, but there are several reports of contact dermatitis when formaldehyde and melamine resins are added to the plaster. [32] [33] The rash is usually pruritic, with weeping papular or vesicular lesions. Because these resins are unnecessary for ED splints, their use should be avoided whenever possible. Dermatitis has also been reported with the use of fiberglass splinting materials.[35]

Joint Stiffness

Some degree of joint stiffness is an invariable consequence of immobilization. It can range in severity from mild to incapacitating and can result in transient, prolonged, or, in some cases, permanent loss of function. Stiffness appears to be worse with prolonged periods of immobilization, in elderly

TABLE 51-7 -- Suggested Length of Immobilization for Conditions That Frequently Require Splinting

Condition	Length of Immobilization (Days)
Contusions	1–3
Abrasions	1–3
Soft tissue lacerations	5–7
Tendon lacerations	Variable*
Tendonitis	5–7
Puncture wounds and bites	3–4
Deep space infections and cellulitis	3–5
Mild sprains	5–7
Fractures and severe sprains	Variable†

*There is considerable controversy surrounding the length of immobilization for tendon lacerations, and duration therefore is best left to the orthopedic or plastic surgeon.
†Usually requires prolonged immobilization; best determined by an orthopedic surgeon.

patients, and in patients with preexisting joint diseases such as rheumatoid arthritis or osteoarthritis. Thus, splints should be left on only for that period of time necessary for adequate healing. Table 51-7 lists several injuries that commonly require splinting, along with some suggestions for length of immobilization. Fractures, dislocations, or other conditions that require prolonged immobilization (more than 7 days) should have orthopedic follow-up. Patients must be told that a splint is only a short-term device and that prolonged immobilization can be detrimental. For minor injuries, the clinician can suggest that the patient use his or her own judgment about when to remove the splint, but a definite end point should be set.

CAST PAIN

Cast-related pain is a common complaint that brings patients to the ED. Because of the potential for ischemia with circumferential casts, all complaints should be fully investigated, and vascular compromise must be ruled out. A detailed history and physical examination should be performed on all such patients. The nature and onset of the pain is of particular importance. A dull, nonspecific pain that has worsened gradually since the time of injury may be the only clue to an early compartment syndrome (see Chapter 55). The sudden onset of throbbing pain associated with swelling and redness suggests a possible deep venous thrombosis. In both these cases, rapid intervention is the key to decreasing morbidity and mortality. The physical examination should pay particular attention to the areas of tenderness and the effect of active and passive movement on the severity of pain.

With a *compartment syndrome*, tenderness over the involved compartment is a common finding; stretching or contracting ischemic muscle also elicits significant pain. The examination should also evaluate the presence and quality of distal pulses, amount of edema fluid present, distal sensation, capillary refill, and color and temperature of the digits. The finding of pain, pallor, paresthesias, paralysis, and pulselessness (the five Ps) are said to be pathognomonic for ischemia. Unfortunately, they seldom occur simultaneously, and their presence together is usually a late finding that carries a poor prognosis. Hence, the emergency clinician must maintain a high index of suspicion for possible ischemia and remove the

Figure 51-25 *A,* The cast saw vibrates; it does not rotate. The blade is controlled by placing the thumb on the splint and lowering the saw to the plaster. The blade is raised and lowered for each cut; it is *not* drawn across the plaster like a knife. *B,* This cast was too tight, and it was therefore bivalved from cast to forefoot with a cast saw. After separation of the edges of the cut cast, the anterior and posterior components were secured in place with an elastic bandage. Note that the underlying Webril padding was cut to relieve pressure but was not removed *(inset)*. A bivalved cast provides temporary immobilization equal to that of an intact cast.

cast if any possibility of vascular compromise exists. Almost any cast can be bivalved and reapplied after inspection without significant loss of short-term immobilization.

To loosen a cast, an oscillating cast saw is used to cut along the medial and lateral aspects of the cast (Fig. 51-25). This is called *bivalving* the cast, and it allows the halves to be spread and reapplied in a less constricting manner while still maintaining proper immobilization. To use the oscillating power saw, proceed in a series of downward cutting movements facilitated by wrist supination, removing the blade between cuts. The blade is removed between cuts to prevent it from getting hot enough to burn the skin. This is particularly important if synthetic materials have been used in the cast. Also, the blade should not be allowed to slide along the skin, and the saw should never be used on unpadded plaster. With an apprehensive patient, the clinician can demonstrate that the cast saw blade only vibrates (it does not turn) and that it does not cut the skin.

After completely cutting through the medial and lateral sides of the cast, the two halves are separated using a cast spreader, and the padding is cut lengthwise with scissors. This may be sufficient to relieve early ischemia if the problem is simple postinjury swelling, but both the padding and cast can be removed totally to inspect the injured area if necessary. If ischemia cannot be ruled out, compartment pressures should be measured (see Chapter 55), and an orthopedic consultation should be obtained.

If vascular integrity is established and no other problems are found, the bivalved cast can be replaced. First, the extremity should be padded in the usual manner using fresh Webril. The cut ends of the bivalved cast are then lined with white adhesive tape, and the cast is replaced around the extremity. Finally, the cast is secured in place using elastic bandages.

If plaster sores are causing the patient's discomfort, the clinician who placed the cast should be consulted. In some cases, additional padding is all that is needed, but in others a window should be cut out over the problem area. Because pressure sores can lead to significant tissue necrosis, the patient should receive follow-up care within 24 hours.

If the patient's problem is plaster (or more likely, resin) dermatitis, treatment generally consists of topical or oral steroids and antihistamines. Therapy should be done in concert with an orthopedic surgeon because the patient may require admission for other forms of immobilization until the cast can be replaced. With mild cases, changing the cast or splint and using antihistamines for symptomatic relief may suffice. All of these patients should receive close follow-up, and if the condition does not improve, the cast must be removed.

CONCLUSION

Splinting represents an important means of temporary fracture immobilization and provides protection and comfort for a variety of soft tissue injuries. The clinician should be aware of potential complications including ischemia, thermal injury, and pressure sores, which can occur with improper splint application. Proper technique should minimize the risk of these adverse outcomes. The emergency clinician also should be facile in the release of circumferential cast and splint materials when ischemia is suspected.

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Chapter 52 - Podiatric Procedures

Douglas McGee

Patients with painful or infectious conditions of the feet often seek medical attention since normal daily activities cannot be easily accomplished without walking. This chapter focuses on procedures performed for common maladies of the foot. Other procedures of the foot are described elsewhere in this text, including anesthesia of the foot and ankle (see Chapter 30 and Chapter 32), management of nailbed injuries (see Chapter 36 and Chapter 38), incision and drainage of paronychia (see Chapter 38), joint fluid analysis (see Chapter 54) the management of common dislocations of the foot (see Chapter 50), and splinting (see Chapter 51).

COMMON NONTRAUMATIC CONDITIONS OF THE FOOT

Many painful conditions of the foot are chronic and usually do not require definitive treatment in the emergency department (ED); however, patients often present with common conditions that require evaluation and proper referral. To accomplish this, the clinician must be cognizant of basic podiatric issues. Typical conditions include painful lesions over bony prominences, heel pain, foot infections, and pain on the plantar surface of the foot.

Footpad Use

Footpads redistribute pressure over an inflamed, tender area of the foot. The particular type of footpad and its placement depend on the condition being treated (Fig. 52-1). Commercially available aperture footpads are recommended

Figure 52-1 Use of aperture pads to redistribute pressure from painful areas to surrounding structures. *(Courtesy of Kenneth R. Walker, DPM.)*

for temporary relief of warts, corns, hyperkeratoses, and bunions. Verruca virus introduced into the plantar surface of the foot may produce a painful hyperkeratotic lesion, commonly referred to as a "plantar wart," on the sole of the foot. Simple callus may be painful and result in the formation of a "hard corn" when formed over the bony prominence of a digit. Once recognized, and after other conditions are ruled out, definitive care of these lesions is rarely indicated in the ED.

When tenderness is elicited over more than one metatarsal head, the diagnosis is metatarsalgia. Pain that is progressively worse while walking but relieved by rest, often beneath the second or third metatarsal heads, is typical in this case. A pad placed under the first metatarsal head to raise the second and third metatarsals may provide some relief.

A bunion develops when unbalanced forces applied to the first metatarsal cause lateral displacement of the distal hallux. Bunions typically form in women wearing heeled shoes with narrow toe boxes. The patient may complain of numbness over the distal, medial aspect of the first toe owing to compression of the terminal branch of the medial dorsal cutaneous nerve. The mechanical forces that precipitate bunion formation may also cause other painful conditions including intermetatarsal neuroma, hammertoes, ingrown toenails, corns, and callus. Bursitis may develop over the medial bony prominence of the first metatarsal phalangeal (MTP) joint. Self-adherent bunion pads placed over the first MTP joint may provide temporary relief (Fig. 52-2). In the ED, treat patients suffering from these common disorders with analgesics and footpads followed by referral for definitive care. When appropriate, recommend that the patient avoid the offending shoes. Also, remember to consider gout when evaluating pain over the first MTP joint, particularly in the presence of other signs of inflammation (e.g., redness, swelling, warmth).

Heel Pain Syndromes

Bony spurs on the plantar surface of the calcaneus, retrocalcaneal bursitis, calcaneal apophysitis, and other conditions may cause heel pain. Treat most of these conditions with rest, nonsteroidal analgesics, modification of physical activities or shoewear, footpads, and orthoses. Some clinicians also include the injection of anesthetics or steroids for these conditions.

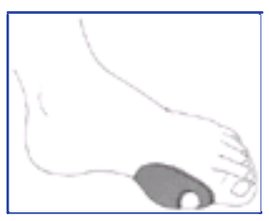

Figure 52-2 Use of a self-adherent bunion pad. *(Courtesy of Kenneth R. Walker, DPM.)*

Heel spur pain can be quite bothersome and chronic. This condition is not easily remedied in the ED, and after other conditions are ruled out, minimal intervention with podiatric referral is often the best course of action. Patients typically present with pain over the medial border of the plantar aspect of the calcaneus. The pain gradually worsens over months. A bony prominence that begins as periostitis extends from the medial aspect of the calcaneal tuberosity into the central plantar fascia and may be demonstrated on radiographs. Radiographs of the calcaneus that do not demonstrate a bony spur suggest plantar fascitis (see Painful Conditions on the Plantar Surface of the Foot later in this chapter).

Shoe supports using a heel pad or cup, or a donut-shaped orthosis often help reduce the discomfort by redistributing weight. Injection of the painful site is done with 10 to 20 mg of methylprednisolone injected from the medial aspect of the foot, avoiding the sensitive plantar surface. Few randomized controlled trials have evaluated steroid therapy; those that have do not provide substantial evidence supporting its long-term efficacy. [1] [2] Some evidence suggests that 25 mg prednisolone acetate injected into the medial heel provides partial pain relief at 1 month compared with lidocaine only, but no advantage can be detected at 3 months. [3] A short leg walking cast may be effective in some patients with recalcitrant heel pain. [4]

Retrocalcaneal Bursitis, Achilles Tendonitis, and Calcaneal Apophysitis

Although retrocalcaneal bursitis and Achilles tendonitis are anatomically distinct, the clinical presentation is similar. Pain at the insertion of the Achilles tendon is worsened with prolonged standing or walking and is aggravated by passive or active range of motion in both conditions. Directed palpation can distinguish one entity from the other but both are treated similarly. Tenderness of the Achilles tendon suggests tendonitis, whereas tenderness between the tendon and calcaneus suggests retrocalcaneal bursitis. Rest, elevation, ice, nonsteroidal anti-inflammatory medications, heel pads, and an open-back shoe provide relief in the majority of patients. A corticosteroid injection may provide some relief although its superiority to conservative measures is unproven. Repeated steroid injection is associated with Achilles tendon rupture. [5] Osteochondrosis of the posterior calcaneal apophysis may cause pain worsened by activity in the child between 7 and 10 years old. It is thought to represent an overuse syndrome in the athletically active child with a tender posterior heel. [5] [6] Treat this self-limited condition with rest, ice, and heel pads. Activity is resumed when the pain abates.

Painful Conditions of the Plantar Surface of the Foot

Plantar Fascitis

Repeated microtrauma to the plantar aponeurosis causes pain on the plantar surface of the foot. Plantar fascitis is typically unilateral and found in women who wear high-heeled shoes. The pain is maximally severe in the morning or after prolonged sitting and improves after walking. Some patients with plantar fascitis may also have a calcaneal heel spur. Pain is elicited with palpation, toe walking, or passive stretching of the plantar aponeurosis. Often this annoying condition resolves on its own, but resolution is slow. Conservative therapy, including rest, elevation, ice, and nonsteroidal anti-inflammatory medications, results in a satisfactory outcome after

6 to 8 weeks in 90% of patients.[7] Corticosteroid injection is used by some clinicians; the benefit remains unproven. Repeated injections with corticosteroids have been associated with rupture of the plantar fascia.[8]

Forefoot Neuroma

A forefoot neuroma, also know as a Morton neuroma, is a painful condition of the plantar surface of the foot. It most commonly affects women who wear high-heeled shoes. The neuroma forms after chronic irritation to the sensory nerve between the metatarsals. The neuroma frequently occurs in the third interspace but may be found in the second space. Patients report the sensation of a lump or cord in the interspace and describe paresthesia or numbness in the third or fourth toes. Rest, elevation, ice, and nonsteroidal anti-inflammatory medications may result in some improvement, but surgical excision is often required.[9] [10] Little data support the use of corticosteroid injections. In one study, less than 50% of patients with a foot neuroma had any benefit from injected corticosteroids.[9]

Ganglion Cyst of the Foot

A ganglion cyst is histologically similar to the synovial sheath and contains synovial fluid. The diagnosis is easy to make when the cyst is located over a tendon on the dorsum of the foot, but may be difficult when located among the compact structures of the plantar forefoot. The ganglion cyst usually causes edema located along the involved tendon sheath. The mass should roll under the examiner's finger; a painless, immovable mass suggests a soft tissue neoplasm. Painful ganglion cysts are treated with aspiration with or without the injection of corticosteroid (see Chapter 53). After local or regional anesthesia (see Chapter 30 and Chapter 32), insert a 20-ga needle into the cyst and withdraw yellow, thick, synovial fluid. Manually express any remaining synovial fluid after the needle is withdrawn. Corticosteroid injection is often advocated for ganglion cysts but recurrence is common after aspiration and corticosteroid injection—as high as 57% in one study.[11] Recurrence, chronic pain, neuritis, stiffness and infection are not uncommon even after surgical excision.[12]

TRAUMATIC CONDITIONS OF THE FOOT

Trauma to the feet and toes is common and covers a broad spectrum of injury. Lacerations, fractures, dislocations, compartment syndromes, and nailbed injuries are described in other chapters of this text. Two specific injuries, toe fractures and puncture wounds to the plantar surface of the foot, are discussed in detail here.

Toe Fractures and Fractures of the Sesamoid Bones

Many clinicians and laypersons shrug their shoulders at a toe fracture, believing nothing can be done. Something *can and should be done* to relieve the pain and encourage healing. As with any other fracture, pay attention to the possibility of disrupted joint cartilage, hypermobility of fracture segments, and malposition or malunion of the fracture fragments. Aggressive reduction is indicated for fractures of the proximal phalanx of the great toe, as it represents the main propulsive segment of the forefoot. A plaster cast alone without anatomic

reduction is insufficient treatment. Displacement suggests axial rotation or abnormal biomechanical interaction between the hallux and its own interphalangeal or MTP joint.

In the acute setting, a non-weight-bearing ankle splint that extends beyond the great toe (see Chapter 51) provides protection until the patient with a complicated great toe fracture obtains follow-up with a foot and ankle surgeon. Open fractures mandate immediate referral for debridement and antibiotic therapy. Fractures of the lesser toes usually result from jamming the toe into a nightstand or bedpost while barefoot. Radiographs of the lesser phalanges confirm the suspected fracture and may occasionally reveal an unsuspected interphalangeal or metatarsal phalangeal dislocation. However, x-rays are generally not required and the injured digit is easily reduced.

Treat closed, lesser phalangeal fractures with "immobilization" for 6 weeks. After the fracture is reduced, splint the injured toe against an adjacent noninjured toe. Place a soft corn pad or other suitable material between the toes to prevent skin maceration, and hold the toes together with adhesive tape or a self-adherent wrap like Coban (Fig. 52-3). Demonstrate the procedure to the patient or family and dispense or prescribe enough material so that the splint can be changed every 2 to 3 days at home. Put the patient in a less restrictive,

Figure 52-3 "Buddy taping" of a fractured lesser toe. *A,* a pad is placed between the injured toe and an adjacent toe. *B,* the toes are secured together with tape or self-adherent wrap. *(Courtesy of Kenneth R. Walker, DPM.)*

stiff-soled shoe. A postoperative shoe (or similar footwear) may be a comfortable alternative for the first several days.

Jumping from a height can result in a fracture of the first MTP joint sesamoid bone. The great toe sesamoid bones lie in grooves on the bottom of the metatarsal head. Each bone lies within the tendon of its respective flexor *hallucis brevis* muscle belly. There is localized pain on the plantar aspect of the first metatarsal head with a sesamoid bone fracture. Bipartite sesamoids (tibial more frequently than fibular) are common. Comparison radiographs clarify whether the radiographic abnormality represents a fracture.

For a tibial sesamoid injury, an aperture bunion-type pad, reinforced medially, of 0.5- to 0.75-cm-thick felt protects the sesamoid and transfers weight bearing to the surrounding structures (Fig. 52-4). A hard-soled shoe and nonsteroidal anti-inflammatory drugs are also helpful. Subsequent radiographs rarely show bony consolidation, but the fracture interface appears smoother.

Plantar Puncture Wounds

Plantar puncture wounds present a diagnostic and therapeutic challenge for the clinician (Fig. 52-5). Considerable controversy exists regarding the proper initial management of puncture wounds to the plantar surface of the foot, and no universally accepted standard of care exists. Treatment recommendations range from simple wound cleaning to aggressive débridement (see also Chapter 37 and Chapter 38). No single approach has been demonstrated to be superior. The editors support an initial conservative approach, but an aggressive one if the patient returns or presents with an infection, or if pain persists for more than a few days.

Although nails produce many such wounds, various other objects may cause them, too, including other metal objects,

Figure 52-4 Bunion shields redistribute pressure away from fractured sesamoid bones. *(Courtesy of Kenneth R. Walker, DPM.)*

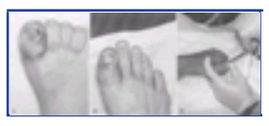

Figure 52-5 *A,* An ingrown toenail of this degree requires removal of a portion of the nail and debridement of inflamed tissue. *B,* Two days later, there is marked improvement. *C,* To remove a toenail, small scissors are held parallel to the nail bed, advanced, and spread. This will produce the least damage to the nail bed.

wood, and glass. Patient response to the injury depends on the penetrating material, location and circumstances of the wound, depth of penetration, footwear, time from injury until presentation, and underlying health. Because superficial puncture wounds generally do well, depth of penetration may be a primary determinant of outcome. [13] Since one's reflexes are not fast enough to pull back when stepping on a sharp object, the clinician should assume that the entire length of a protruding nail has entered the foot (minus, of course, the thickness of footwear). Stepping on an unknown object in a field or while walking in a stream requires a more cautious approach than does a simple puncture from a known object, such as a protruding nail.

The vast majority of patients who "step on a nail" suffer nothing more than transient pain and never seek medical attention. Since most minor puncture wounds are not seen in the ED, the true risk of infection, in particular osteomyelitis, is unknown. Consequently, reported infection rates vastly overstate the actual incidence. One review of these wounds suggests that no more than 2% to 8% of puncture wounds become infected and only a small percentage of these develop osteomyelitis. [13] A prospective series suggests that only the presence of symptoms (e.g., redness, tenderness, increased swelling) at 48 hours was associated with the risk of infection or potential retained foreign body. [14] Retained foreign material (e.g., a portion of a tennis shoe sole) in the wound is an important factor in persistent infection. Since no single test detects all possible foreign bodies, tailor the evaluation of a suspected foreign body to the suspected object.

Evaluation

The approach to the patient is dependent on several factors, including the time from injury to presentation, the suspicion of a foreign body, and the presence of infection. The extent to which a foreign body is pursued depends on the history and physical findings. When the patient clearly states that a needle, pin, or nail was removed intact, radiograph or local wound exploration for a retained metallic foreign body are not needed. If there is any debate regarding a retained metallic object, plain radiographs readily demonstrate their presence. Plain radiographs also demonstrate other radiopaque objects such as glass, gravel, bone, and teeth. If the patient steps on an unknown object, a radiograph is usually indicated unless the entire depth of the wound can be ascertained and inspected. Ultrasonography is noninvasive and does not use radiation, making it potentially useful for radiolucent foreign bodies, but soft tissue air or calcifications may suggest a retained foreign body when there is not one (see Chapter 69). Computed tomography can demonstrate radiopaque and radiolucent objects but its expense, limited availability, and greater radiation exposure as compared to plain films make it unsuitable as a screening tool. Use CT scanning when other screening tools have failed to demonstrate a suspected foreign object, when infection is present, or when joint penetration is suspected. Magnetic resonance imaging provides no additional benefit and cannot be used when metallic objects are retained. General management of suspected foreign bodies is discussed in further detail in Chapter 37 .

Treatment

As a general rule, the plantar surface of the foot should be examined under good lighting and in a bloodless field. This is best accomplished with the patient in a prone position (Fig. 52-6 and Fig. 52-7). Plantar puncture wounds explored for persistent infection often have contained foreign material (Fig. 52-8). Since most patients do not develop persistent infection, initial deep exploration in the absence of evidence or strong suspicion for retained material cannot be advocated. [13] Routine initial deep wound exploration, including coring of the wound, is not supported by scientific research and is not recommended for simple, noninfected puncture wounds. [13]

However, select wounds may benefit from exploration to facilitate a search for foreign objects and to promote irrigation, cleansing, and drainage. When the wound is large and retained organic material is suspected, local wound exploration may be warranted. Patients wearing rubber-soled shoes

Figure 52-6 *A,* The best way to examine a puncture wound of the foot is to place the patient prone on a stretcher and obtain good lighting and a bloodless field (in this case, a blood pressure cuff was applied to the lower leg). *B,* When the overlying skin was unroofed, a small piece of rubber from the patient's sneaker was found imbedded in the wound. It was removed by a coring technique and packed.

during plantar puncture wounds may retain a portion of the shoe in the wound (Fig. 52-9). Exploration of the wound is most productive after local anesthesia or a regional anesthetic block (see Chapter 32) and excision of the epidermal flap. An incision may be required to facilitate foreign body removal, but extensive removal of surrounding tissue is not proven to increase successful removal of retained material. [13] Some clinicians favor a coring technique when a foreign body is found or suspected. Although this may be aggressive for many wounds, it is the best way to remove particulate matter as a block. To accomplish coring, a No. 11 blade is advanced to the hilt and a 2 to 3 mm core is excised. This tract can be packed with gauze for a few days or left open. Occasionally, blunt probes may facilitate wound exploration. It is generally impossible, and likely counterproductive, to attempt to probe or visualize the entire length of the puncture tract.

Patients presenting within 24 hours and *without* signs of infection generally require only simple wound care. Although irrigation of all exposed dermal tissue is recommended, high-pressure irrigation of deep tissues with distention of the soft tissues is unlikely to be helpful and not recommended. Schwab described a case series of uncomplicated puncture wounds in healthy individuals who had conservative treatment with cleansing and crutches. Radiographs were obtained at the discretion of the treating clinician; antibiotics were not given. [14] At their 6-month follow-ups, 88% of all patients had healed without complication. The remaining 12% developed

Figure 52-7 *A,* Patients who present with lymphangitis, obvious cellulitis, or both within a few days of stepping on a nail usually have a gram-positive soft tissue infection. *B,* In this case, the puncture wound was excised by a coring technique, yielding a few bits of foreign material. A pack was placed in the wound, oral antibiotics were given, and the patient did well.

complications including wound infection due to retained foreign body. No findings on initial presentation predicted a subsequent infection. Initial antibiotic therapy remains controversial, but there is no evidence to suggest that prophylactic antibiotics reduce the already low rate of infection.

Symptomatic patients who present later (24 hours or longer) have an increased risk for a retained foreign body, since delayed presentation is often due to the development of inflammation or infection. Local wound exploration is warranted unless the patient presents without infection or with a clearly superficial cellulitis expected to respond to simple oral antibiotics (see Fig. 52-7 and Fig. 52-8). Recurrent infection, deep soft tissue tenderness, and increasing soft tissue swelling suggest a retained foreign body or deep space infection, such as osteomyelitis. Such patients require prompt specialty referral or additional diagnostic studies in the ED.

Patients with obvious signs of infection within a few days of a puncture wound usually have a simple cellulitis (with or without a foreign body) with a gram-positive organism. In a patient with persistent pain or swelling days to weeks after a puncture wound, *the presence or absence of a deep soft tissue infection, or a low-grade osteomyelitis cannot be ruled in, or ruled out, by physical examination, plain radiographs, or laboratory tests (such as a sedimentation rate, complete blood count, or wound cultures)*. A high index of suspicion, coupled with additional investigation, is the prudent approach to the patient with minimal physical findings, normal laboratory

Figure 52-8 This 4-day-old inflamed puncture wound tract likely harbors a foreign body. Excision of a core of the tract found a few pieces of the patient's sock embedded in the wound.

tests, and continued pain or swelling following a seemingly simple puncture wound to the bottom of the foot. This usually entails a computed tomography scan, bone scan, or magnetic resonance imaging.

Plantar puncture wounds with *Pseudomonas* osteomyelitis and osteochondritis are clinically clandestine and particularly devastating, and have been described for nearly 40 years. Some investigators have cultured *Pseudomonas* from the sole of tennis shoes, suggesting that puncture wounds made through athletic footwear may be inoculated with *Pseudomonas*.[15] There is no evidence that prophylactic anti-*Pseudomonas* antibiotics prescribed on an initial evaluation of an uncomplicated wound will prevent infection among patients with subsequent deep

Figure 52-9 A patient stepped on a nail while wearing this shoe. An initial ED evaluation 3 weeks previous found no foreign body or infection. A week previous, the patient began taking antibiotics, but did not improve. The physical examination was quite benign, but the continued aching pain and minimal swelling suggested a deep infection. The complete blood count, sedimentation rate, and plain film were negative. A magnetic resonance imaging scan demonstrated osteomyelitis. *Pseudomonas* is often the offending organism in this scenario.

space or bone infections.[15] Some argue that prophylactic antibiotics, at the time of puncture, may select out resistant organisms. Surgical debridement and prolonged intravenous antibiotics are often required for established infections. In Figure 52-10 , an algorithm is offered to guide evaluation and management of plantar puncture wounds.

INGROWN TOENAIL

An "ingrown toenail" is characterized by progressive curving or excessive widening of the lateral margin of the toenail and impingement of the nail into the periungual soft tissue (see Fig. 52-5). The toenail normally grows distally in an unimpeded manner, allowing the nail to pass beyond the lateral nail fold. Nail deformity, tight fitting shoes, and rotational deformity of the toes increases the friction between the nail and nail fold. Toenails that have been trimmed in a curve increase the likelihood that the lateral nail margin will impinge on the lateral nail fold. The resulting soft tissue injury may lead to hyperkeratosis, edema, and erythema of the nail fold or frank infection (Fig. 52-11). Although an ingrown toenail can be found on any toe, the majority occur in the great toe.

Evaluation

The patient presents with pain, edema, and erythema of the lateral nail fold. Pressure over the nail margins increases the pain. Since intense pain often precipitates an early visit to the ED, most inflammatory or infectious responses are confined locally. Recurrent ingrown toenails or those in patients with circulatory dysfunction, neuropathy, or diabetes may have underlying osteomyelitis. Cleanse the toe gently to make visualization of periungual debris easier. When the free edge of the lateral nail can be easily visualized as separate from the lateral nail fold, consider other painful conditions of the toe like trauma, gout, paronychia, and cellulitis.

Treatment

Because the toe is exquisitely tender, additional treatment will usually require a digital block as described in Chapter 32 . The decision to treat and what course of action to take in the ED depends on the patient's degree of discomfort. Two general courses of action may be needed: removal of the offending nail spicule or removal of the spicule and some portion of the nail. Any degree of nail removal is usually followed with ablation of the nail bed. [16]

Removal of the Nail Spicule and Debridement of Hyperkeratosis

When the amount of inflammation and pain and the degree of nail deformity are both minimal, removal of the impacted nail spicule is indicated. After a digital block and thorough cleansing, remove an oblique segment of the nail about one third to one half of the way to the proximal nail fold (Fig. 52-12). The ideal instrument is an English anvil nail splitter designed to cut the nail while minimizing trauma to the underlying nail matrix (Fig. 52-13). Sharp, pointed scissors may be substituted if care is taken to minimize nailbed injury by maintaining upward pressure while cutting the nail. Some clinicians use a disposable electric cautery device to cut the nail after it has been softened by soaking in warm soapy water.

Figure 52-10 Plantar puncture wound management. *A,* Management within 24 hours of wounding. *B,* Management after 72 hours. *(From Chisolm CD, Schlesser JF: Plantar puncture wounds: Controversies and treatment recommendations. Ann Emerg Med 18:1352, 1989.)*

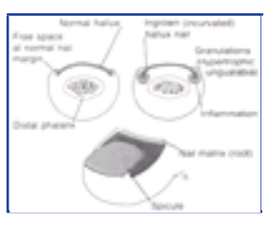

Figure 52-11 Pathology of an ingrown toenail. The normal free space at the nail margin is obliterated by inflammation and granulation tissue, which is caused by improper nail trimming, trauma to the matrix, and faulty footwear. *(From Hill GL II: Outpatient Surgery, 3rd ed. Philadelphia, WB Saunders, 1988. Reproduced by permission.)*

After cutting the nail spicule free from the bulk of the nail, grasp the free edge of the nail with forceps or hemostats and remove it. The nail fold typically contains impacted debris that must be removed after the nail fold has been gently retracted away from the nail. Remove debris until the epidermis or dermis is uncovered, taking care to avoid aggressive debridement that causes bleeding. Dress the area with antibiotic ointment and a nonadherent dressing. The wound should be reinspected for signs of infection at 48 hours and referral made for definitive podiatric care. Instruct patients to wear less constricting shoes and to trim the nail straight across. Like most foreign body reactions, removal of the nail spicule resolves the inflammation and infection. Antibiotics given without removal of the nail spicule will not ensure a satisfactory result or add benefit after spicule removal. [17] When the ingrown toenail is caused by nail deformity, a podiatrist or primary clinician can accomplish definitive removal during follow-up evaluation.

Toenail Removal

Toenail removal may be total or partial. Total nail removal is rarely needed but may be used when infection of both lateral

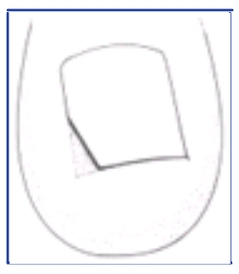

Figure 52-12 An oblique wedge of nail is trimmed from the lateral margin of the nail to free it from the hyperkeratotic area. *(Courtesy of Kenneth R. Walker, DPM.)*

Figure 52-13 English nail anvil used to divide the nail. *(Courtesy of Gill Podiatry Supply Company, Middleburg Heights, OH.)*

nail folds is present, particularly if the condition is present for more than a month. Consider partially removing the toenail when ingrown toenails are associated with chronic inflammation/infection or severe pain. Partial nail removal accomplishes two things: removal of the offending portion of nail and destruction of the underlying nail matrix to prevent nail regrowth. Phenol, the most commonly applied chemical, causes neurolysis of nerve endings and necrosis of the nail matrix in a procedure

called matrixectomy. After digital block, exsanguinate the toe by squeezing or wrapping, and apply a tourniquet at the base of the toe. A dry nail matrix after nail removal is needed prior to application of phenol.

Stabilize the toe in the nondominant hand and remove the lateral quarter of the nail by splitting the nail toward the cuticle. An English anvil nail splitter is desirable to begin the procedure, but sharp scissors, a No. 11 blade, or electric cautery device as described previously for removal of the nail spicule will work, too. When a nail splitter is used, divide the nail about one half to two thirds of the way back. Then divide the remainder of the nail using scissors or a scalpel. Take care to perform a controlled division along the longitudinal lines of the nail until resistance is no longer felt. The nail should be divided several millimeters past the proximal nail fold. Carefully separate the lateral portion of the nail from the underlying matrix and grasp it with a hemostat or forceps. Remove the free piece of nail by twisting it toward the remaining nail. Inspect the remnant to be certain that the entire piece of nail has been removed as desired. Curette the posterior and lateral nail grooves to remove any remaining hyperkeratotic debris. Take great care to avoid hemorrhage; a dry nailbed is mandatory before phenol application. Adjust the tourniquet if needed to achieve a bloodless field.

Apply a 70% to 90% solution of aqueous phenol to the nail matrix beneath the involved area of the lateral nail groove and proximal nail fold using cotton wound applicators. A 1% phenol solution can be prepared by diluting a 70% to 90% aqueous phenol solution in an 80:1 ratio (e.g., 8 mL distilled water to 0.1 mL phenol). Another alternative is to desiccate the exposed nailbed with silver nitrate sticks. Remove some of the cotton if the applicator is too bulky to concentrate the solution beneath the proximal nail fold and lateral nail groove. Apply thoroughly moistened (but not saturated) applicators, using three 30-second applications. Avoid forcing phenol under the remaining nail by rolling the applicator so that it rolls over the matrix and over the nail surface rather than against the split edge of the nail. Do not allow excess phenol to contact the exposed nail bed or surrounding healthy tissue. After the third application, the cauterized tissue appears

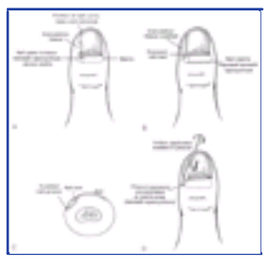

Figure 52-14 The nail ablation technique for treatment of an ingrown toenail. The lateral portion of the nail is cut and removed (*A*), exposing the nailbed. Granulation tissue is curetted (*B* and *C*) and the nail matrix is cauterized with absolute phenol (*D*) (see text).

brown tinged or gray. Alternatively, a 1% phenol solution can be applied for 5 minutes. Thoroughly irrigate the cauterized nailbed with water and rub the area with a gloved finger to remove traces of phenol. Snip away any remaining debris or dead skin with scissors. The technique is illustrated in Figure 52-14 .

Dress the wound with antibiotic ointment and a non-adherent dressing, followed by a dry sterile wrap. Do not forget to remove the tourniquet after the dressing has been applied. Instruct the patient to wash the wound twice daily followed by dry dressing changes. Antibiotics do not hasten wound healing and are not necessary in most cases.[17] The wound will heal in 2 to 4 weeks and may be accompanied by serous drainage for 2 weeks. The patient should be informed of this possibility. Complications include nail regrowth, infection, growth of an inclusion cyst, or delayed healing. The procedure successfully ablates the removed nail in more than 90% of cases.

CONCLUSION

Many common maladies of the foot can be treated in the ED with rest, analgesia, and supportive measures such as footpad use. Fractures of the lesser toes are easily treated with dynamic splinting, but the great toe may require definitive treatment by a foot and ankle surgeon. Most plantar puncture wounds can be treated like other soft tissue injuries but retained foreign bodies, deep space inoculation, and osteomyelitis can occur. Most ingrown toenails can be simply treated with removal of the offending nail spicule, although nail removal and ablation may be needed in severe or recurrent circumstances.

Acknowledgment

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Chapter 53 - Injection Therapy of Bursitis and Tendinitis

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Bursitis and *tendinitis* are terms frequently used to describe a variety of regional musculoskeletal conditions that are characterized chiefly by pain and disability at the involved site. Bursitis of the shoulder may be considered the prototypical disorder. All too often, in cases of ill-defined regional soft tissue rheumatic problems, bursitis or tendinitis is used as a "wastebasket" diagnosis. The pain and dysfunction of tendinitis and bursitis are variable, and not always reproducible by each examination. In addition, many muscular, neurologic, and even infectious conditions present similarly to these syndromes. For purposes of this chapter, in consideration of the accurate diagnosis that is necessary to institute appropriate therapy, the terms are reserved for well-defined, specific clinical entities. The clinician must, however, appreciate the fact that these are largely clinical diagnoses that cannot be verified by objective data, radiographs, or laboratory parameters.

GENERAL ANATOMIC CONSIDERATIONS OF BURSAE AND TENDON SHEATHS

Bursae are potential spaces or sacs, subcutaneous or deep, that develop in relation to friction and facilitate the gliding motion of tendons and muscles. There are approximately 78 bursae on each side of the body. These were well described in the classic atlas of anatomy by Monro in 1788 [1] and were later elaborated in greater detail in the atlas of Spalteholz in 1932. [2]

The normal bursal wall is lined with a thin layer of synovial cells that appear to be similar to those of joint synovial membrane when examined by electron microscopy.[3] When a bursa becomes subacutely or chronically inflamed, the normally thin surface of sparse cells may thicken to 1 to 2 mm. *Bursitis* may be caused by trauma, infection, crystal deposition, chronic friction from overuse, or a systemic inflammatory arthropathy. In addition, so-called adventitial bursae may form in response to abnormal shearing stress at sites subjected to chronic pressure; an example is a bunion over the head of the metatarsal bone of the great toe.

Involvement of the synovial lining of bursae and tendon sheaths may also result from underlying systemic diseases, including rheumatoid arthritis, ankylosing spondylitis, psoriatic arthropathy, and gout. The most common bursal lesions in these systemic inflammatory arthropathies involve the olecranon at the elbow and the trochanter region of the hip. Smaller bursae, especially those around the Achilles tendon, also may be affected. *Tendinitis* and *tenosynovitis* are useful terms that describe inflammatory reactions in tendons and tendon sheaths. Tendon sheaths are relatively long and tubular, whereas bursae are round and flat. Except for their shape, however, the structures are similar. In fact, because of the adjacent location of bursae and tendons, an inflammatory process in one may also involve the other.[4] Common sites of tendinitis in the body are depicted in Figure 53-1 (Figure Not Available) .

Bursitis and tendinitis embrace a variety of conditions that may be grouped together on a regional basis for the sake of a simple and convenient classification (Table 53-1).

In rare cases, the etiologies of tendinitis/bursitis syndromes can be exotic, such as the tendinitis caused by quinolone antibiotics or the bursitis noted in dialysis patients.[5] Tendon rupture (especially of the Achilles tendon) has been associated with the use of fluoroquinolone antibiotics (especially ciprofloxacin), but this rare condition is not appreciated by most clinicians and usually goes undiagnosed. [6] Steroid injection may facilitate tendon rupture if fluoroquinolone tendonopathy is present. Gonorrheal tenosynovitis may mimic various forms of tendinitis and is not always associated with overt vaginitis or urethritis or other signs or symptoms of bacteremia.

RATIONALE FOR STEROID INJECTIONS

The management of pain and dysfunction resulting from bursitis and tendinitis may be greatly enhanced by the proper selection and administration of local injections. Successful application of local injection and intrasynovial (bursa and tendon sheath) therapy requires an understanding of the diagnosis, accurate localization of the pathologic condition, and the appropriate choice of suitable injection techniques. Not infrequently, injections of lidocaine or corticosteroid preparations provide the additional aid that, alone or as an important adjunct to the management program, overcomes the refractory pain. While steroid injections are universally administered, often with great success, there are no convincing prospective data to support or refute the specific therapeutic benefit of this

Figure 53-1 (Figure Not Available) Common sites of tendinitis. *(From Walker LG, Meals RM: Tendinitis: A practical approach to diagnosis and management. J Musculoskel Med 6:24, 1989. Reproduced with permission.)*

TABLE 53-1 -- Classification of Bursitis and Tendinitis (Regional)

Upper Extremity Disorders
Elbow
Radiohumeral bursitis, olecranon bursitis, epicondylitis
Shoulder
Bicipital tendinitis, calcareous tendinitis (subacromial, subdeltoid bursitis), rotator cuff tendinitis
Wrist and Hand
Stenosing tenosynovitis ("trigger finger" syndrome), de Quervain disease
Lower Extremity Disorders
Hip
Trochanteric bursitis, ischiogluteal bursitis
Knee
Prepatellar, suprapatellar, and anserine bursitis
Ankle, Foot, and Heel
Ankle tendinitis, bunion bursitis, calcaneal bursitis (with heel spur)

therapy. Many painful conditions wax and wane, there is a significant placebo effect from injections, and there is a natural regression of pain syndromes due to a variety of factors. Although the emergency clinician may administer a local injection with success, in some instances a short course of an oral steroid may produce statistically similar results. In fact, the primary goal of corticosteroid injection therapy is the relief of pain so that the patient can not only function but, more importantly, participate in a physical rehabilitation program. [7] In many cases, a single injection may be all that is required to ameliorate a painful condition. However, injection therapy is best viewed as an adjunct in the management of painful tendinitis/bursitis syndromes. It should not be viewed as a single quick fix, but more of a method of facilitation of other modalities.

The precise mechanism of the lasting analgesia and the beneficial therapeutic effects of local injection therapy have not been clarified. Few clinical trials have adequately measured the efficacy of corticosteroid therapy. Although steroids are known to reduce inflammation, it is unclear if the anti-inflammatory effect is responsible for the increased range of motion and relief of pain that the patient usually experiences. There are histological studies of chronic tenosynovitis lesions that demonstrate degeneration, but not inflammation. [7] It is therefore possible that the pain experienced with tendinitis or bursitis occurs from other mechanisms besides inflammation, such as mechanoreceptor stimulation by shearing or traction, or activation of nociceptive receptors by substance P and chondroitin sulphate. [7]

Injection therapy should be considered an adjunct to a variety of treatment modalities including pain control, physical therapy, occupational therapy, relative rest, immobilization, and exercise. Additional pain control can be achieved with such options as nonsteroidal anti-inflammatory agents, acupuncture, ultrasound, ice, heat, and electrical nerve stimulation. [7] [8] [9] Besides pain relief, early participation by the patient in rehabilitative activities and exercises can be an important aspect of patient recovery. Patients receiving only analgesics may have poor outcomes compared to those who also have exercise as part of their treatment. [4] Any factors that provoke the initial injury should also be identified, as failure to eliminate these provoking factors can contribute to the injury becoming chronic. [7]

Although there are differing opinions in the literature, we recommend that corticosteroid injections not be repeated in the same site unless there has been at least a partial clinical response. There should be at least 6 weeks between injections in the same site, and an injection should not be repeated more than once every 3 to 4 months. [7] [8] [9] [10] Although there is little data on the outcome of repeated procedures, these recommendations are generally accepted and may limit the risk of adverse effects.

INDICATIONS AND CONTRAINDICATIONS

Local injection therapy with corticosteroids or local anesthetics may be effective treatment and provide relief of symptoms in a variety of acute or subacute bursitides and other painful soft tissue conditions. The indications for steroid injection are twofold: therapy and diagnosis. Injection therapy not only offers pain relief, particularly when a local anesthetic is concurrently used, but also offers a medium to deliver therapeutic agents. In addition to relieving pain, injection therapy may aid with a diagnosis. When injecting a bursa, for example, bursal fluid is sometimes collected for laboratory analysis. Finally, the relief of pain helps differentiate a localized site of injury from referred or visceral pain. [9]

Absolute contraindications to local injection therapy are limited and include specific infections such as bacteremia, infectious arthritis, periarticular cellulitis or ulceration, or adjacent osteomyelitis (Table 53-2). The procedure is also contraindicated in patients with significant bleeding disorders. A history of hypersensitivity, either to the corticosteroid or to the vehicle by which it is delivered, is an absolute contraindication. Finally, corticosteroid injections should not be performed in a patient who has a documented osteochondral fracture. However, because stress fractures and nonvisualizing fractures may result from the same trauma or mechanisms that have been implicated in producing an inflammatory condition, it is inevitable that some bony injuries will inadvertently be diagnosed as tendinitis or bursitis.

Relative contraindications depend on both the practitioner's experience and the indication for the injection. A violation of the integrity of the skin or chronic foci of infection, either locally or in the vicinity of the site of involvement, is a

TABLE 53-2 -- Contraindications to Local Injection Therapy

Absolute
Infection (bacteremia, infectious arthritis, periarticular cellulitis/ulceration, adjacent osteomyelitis)
Uncontrolled bleeding disorder
Hypersensitivity to corticosteroid or vehicle
Osteochondral fracture
Relative
Anticoagulant therapy
Joint instability
Poorly controlled diabetes
Hemarthrosis
Decubitus ulcers
Joint prosthesis
Adjacent abraded skin
Chronic foci of infection
Internal joint derangement
Partial tendon rupture

relative contraindication. The procedure is also relatively contraindicated in patients on anticoagulants, with poorly controlled diabetes, and with internal joint derangements or hemarthrosis. The patient with a preexisting tendon injury may be subject to tendon rupture when the corticosteroid injection removes pain and full activity is resumed. Hence, partial tendon rupture is a relative contraindication.

AVAILABLE PREPARATIONS AND CHOICE OF COMPOUND

Hydrocortisone and a variety of available corticosteroid repository preparations are described in Table 53-3 . Local anesthetics, such as lidocaine or bupivacaine, can be mixed with the corticosteroid preparation in the same syringe. All corticosteroid suspensions, with the exception of cortisone and prednisone, can produce a significant and rapid anti-inflammatory effect (in synovial spaces). These preparations are categorized based on their solubility and on their relative potency. Solutions that are more soluble have a shorter duration of action, primarily because these soluble preparations are absorbed and dispersed more rapidly. The addition of tertiary butyl acetate (TBA) to the solution causes decreased solubility, and therefore a longer duration of action. For example, triamcinolone hexacetonide, the least soluble preparation, has the longest duration of action. [10] Because of this and its greater potential for subcutaneous atrophy, however, some authors use this preparation only for intra-articular injections. [9] [10]

There have been attempts to incorporate steroid esters into liposomes to create an even less soluble preparation. This has been shown in both human and animal studies to offer long-lasting relief with minimal drug absorption; however, further research is necessary before this method of delivery is fully developed. [8] [10] [11]

There is little consensus in the literature regarding which corticosteroid should be used and what dosage is most appropriate for a given s[7] [11] [12]

Centeno and Moore noted that the choice of injection agent seemed dependent on the institution where the clinician trained. [13] In 1995, a survey of 172 rheumatologists found differing opinions regarding almost every facet of soft tissue and intra-articular injection, including

TABLE 53-3 -- Injectable Corticosteroids

Intrasynovial Preparations	Potency*	Range of Usual Dosage	Solubility
Short-Acting			
Hydrocortisone acetate	1	12.5–75 mg	High
Cortisone	0.8	15–25 mg	High
Intermediate-Acting			
Prednisone	3.5	2.5–5 mg	Medium
Prednisolone acetate	4	5–30 mg	NA
Methylprednisolone acetate	5	5–40 mg	Medium
Long-Acting†			
Triamcinolone acetonide	5	5–40 mg	Low
Triamcinolone diacetate	5	4–40 mg	Low
Triamcinolone hexacetonide	5	4–25 mg	Low
Betamethasone acetate and disodium phosphatate	25	1.5–6 mg	Low
Dexamethasone acetate	25	0.8–6 mg	Low

*Hydrocortisone equivalents (per mg).
†Best used for intra-articular injection only.

patient preparation, choice of corticosteroid, and post-injection advice. [14] In general, it is recommended that for an acute or subacute diagnosis such as bursitis/tendinitis, it is prudent to use a short- or intermediate-acting agent, while longer-acting agents, such as triamcinolone, should be reserved for chronic and prolonged conditions, including arthritis. [8] [10] Some clinicians, however, advocate mixing both shorter- and longer-acting corticosteroids in the same syringe, with little consideration for the location or category of the condition. [9] [10] We recommend that longer-acting corticosteroids not be used for any soft-tissue injections, particularly because of the increased risk of associated atrophy [7] [9] including atrophy of surrounding structures, such as ligaments and fascia. [15] Methylprednisolone acetate (Depo-Medrol) is a reasonable first choice for most ED indications.

DOSAGE AND ADMINISTRATION

The dose of any corticosteroid suspension used for intrasynovial injection may be arbitrarily selected. Factors that influence the dosage and expected response include the size of the affected area, the presence or absence of synovial fluid or edema, the severity and extent of any synovitis, and the steroid preparation selected for injection.

A useful guideline for estimating dosage is as follows: For relatively large spaces such as subacromial, olecranon, and trochanteric bursae, 40–60 mg of methylprednisolone acetate or equivalent; for medium- or intermediate-sized bursae and ganglia formation at the wrists, knees, and heels, 10 to 20 mg; and for tendon sheaths, such as flexor tenosynovitis of digits and the abductor tendon of the thumb (de Quervain disease), 5 to 15 mg. Sometimes it may be necessary to give larger doses for optimal response. Intrabursal therapy of elbow (olecranon) or knee (prepatellar) bursae containing considerable fluid may require 30- to 40-mg doses. Doses greater than discussed will likely not be detrimental in the vast majority of patients.

Unlike intra-articular injections for synovitis in chronic joint disease, repeat infiltrations for soft tissue conditions such as bursitis and tendinitis are generally not recommended or required. However, if only a partial response occurs or if recurrence develops, a single repeat injection can be given as long as one waits at least 6 to 12 weeks between injections.[7] [8] [9] [10]

PREPARATION OF THE SITE

Preparation of the site before injection requires meticulous adherence to aseptic technique. Anatomic landmarks may be outlined with a black or red skin pencil. Tincture of iodine or thimerosal (Merthiolate) applied with a sterile swab can be used in place of the skin pencil. The point of entry is cleansed with povidone-iodine (Betadine) and alcohol. It is important that the injection site and needle tip remain sterile, using the "no touch" technique, although sterile drapes are not generally considered necessary.[7] [12] For operator protection, universal precautions should be followed, including the use of sterile examination gloves. [9]

TECHNIQUES

General Considerations

Radiographs are not required prior to injection if the diagnosis is clinically well supported. Radiographs may occasionally demonstrate calcium deposits, especially around the shoulder, but the often described spurs associated with olecranon bursitis are of questionable significance. If a patient has an atypical presentation or systemic symptoms (such as fever, chills, nausea), a history of cancer, soft tissue swelling, or suspected bone disease, a radiograph would be prudent. Also, if the patient has had previous unsuccessful injections, a radiograph of the area may be helpful in further clarifying the diagnosis. In the elderly, metastatic disease or fractures without significant trauma may mimic tendinitis/bursitis.

Before beginning the injection, the patient should be informed of the specific indication(s) for treatment. The practitioner should describe the details of the procedure, including risks and complications, and obtain informed consent. Subsequently, the details of the procedure should be documented in the medical record. Use of a written consent form is optional.

Materials that are required beyond the aseptic preparation technique include needles, syringes, a hemostat, culture and laboratory supplies, bandages, and sterile gauze. Disposable needles and syringes are convenient and adequate for emergency department (ED) use. Special trays may also be stocked for this purpose. The usual sizes of needles for injection sites are listed in Table 53-4 .

TABLE 53-4 -- A Guide for Needle Size and Dosage for Injection of Common Regional Disorders

Disorder or Injection Site	Needle Size	Usual Dosage of Methylprednisolone acetate*
Bicipital tendinitis	1.5–2 in., 22–25 ga	20–40 mg
Calcareous tendinitis	1.5–2 in., 22–25 ga	20–60 mg
Subacromial bursitis		
Radiohumeral bursitis	1.5 in., 22–25 ga	20–40 mg
Epicondylitis		
Olecranon bursitis	1–1.5 in., 20 ga	15–30 mg
Ganglia on wrist	1 in., 18–20 ga	10–15 mg
de Quervain disease	7/8 in., 22–25 ga	10–20 mg
Carpal tunnel syndrome	1–1.5 in., 22–25 ga	20–40 mg
Digital flexor tenosynovitis	7/8 in., 22–25 ga	00–05 mg
Trochanteric bursitis	1.5–2 in., 22–25 ga	20–40 mg
Prepatellar bursitis	1–1.5 in., 22–25 ga	15–20 mg
Anserine bursitis	1–1.5 in., 22–25 ga	20–40 mg
Bunion bursitis	1 in., 22–25 ga	5–10 mg
Calcaneal bursitis	1 in., 22–25 ga	10–20 mg

*Empiric dose. Larger or smaller dose may be used dependent upon clinical scenario.

Once the point of entry has been determined and the site is prepared, either a superficial skin wheal is made with 1% lidocaine (Xylocaine) or 0.25% bupivacaine (Marcaine, Sensorcaine) using a 25-ga needle. Some clinicians do not use a skin wheal when the steroid is mixed with a rapid-acting local anesthetic. Topical vapocoolants (e.g., Fluori-Methane) may be helpful in these circumstances. Ordinarily, procedural sedation and analgesia is not necessary, but occasionally, in highly nervous or agitated individuals, it may be advisable (see Chapter 34).

Local injections can be administered with corticosteroids and local anesthetics mixed together in the same syringe. This is commonly done with peritendinous injections using the *Z-tract technique*, which limits the risk of a fistulous tract in the soft tissue. Because the steroid may theoretically precipitate or layer in the barrel of the syringe during the injection, the syringe should be agitated immediately prior to use to optimize its distribution. In addition to minimizing the pain associated with the injection, mixing the anesthetic with the corticosteroid also produces a larger volume for delivery into the soft tissue. [7]

The local anesthetic can also be given alone, before the injection of the corticosteroid. This is commonly seen when injecting bursae using the *two-syringe* technique. After making a wheal, a needle is introduced into the bursa and approximately half of the anesthetic is injected as the needle is inserted. Once the needle reaches the target site, the syringes are changed. Then, as much fluid as possible is aspirated before instillation of the corticosteroid suspension to reduce the dilution factor and to collect bursal fluid for laboratory analysis. Upon changing syringes again, the steroid is introduced without anesthetic. Often syringes are changed once more, and the remaining anesthetic is used to flush out the needle, frequently causing injection and deposition of several milliliters of the local anesthetic. There is no evidence, however, that the injection of the anesthetic before the steroid provides a benefit, or that there is a difference in later outcome with the use of short-acting versus long-acting steroids. [7] [8]

The duration of action of lidocaine is approximately 100 minutes whereas bupivacaine may last several hours. The

patient should be cautioned that *the local anesthetic effect may "wear off" within a couple of hours* and that *the beneficial effects of the corticosteroid may be delayed*.

The most important aspect of a successful technique is accurate positioning of the needle. The needle must "hit the mark" or the results will be disappointing. Injecting an inflamed synovial space, such as a bursa-containing fluid, may be as simple as puncturing a balloon. Aspiration of the fluid confirms that the needle has correctly entered the sac. On the other hand, direct injection into a painful soft tissue lesion requires additional skill that can be acquired only with experience. Sometimes it is advisable to reaspirate and reinject several times within the barrel of the syringe, a procedure called *barbotage*, to obtain heterogeneous mixing and maximal dispersion of the steroid throughout the synovial cavity. Although it is desirable to inject directly into a bursa, direct injection into the tendon itself is best avoided. If the injection requires significant pressure, the needle may be in the tendon, and it should be withdrawn or advanced a few millimeters. The patient may commonly feel some myofascial radiating pain during the injection, but true paresthesias should not be elicited. "Electric shocks" felt with an injection may signal that the needle is in a nerve.

While an accurate injection is desirable, injections are largely blind procedures aimed at depositing the medications near or around inflamed structures. Using a generous volume of anesthetic (3 to 6 mL) to dilute and hence disperse the steroid can compensate for a less than perfect injection. As a general rule, one should

allow the patient to localize the area of maximum tenderness, and the injection should begin in that area. Asking the patient to use one finger to localize the area of maximum pain and tenderness is the best way to ensure the most accurate positioning of the needle. In general, if the patient cannot localize a specific area of tenderness, the diagnosis should be reconsidered or the expectations of success lowered. Diffuse pain, such as throughout the entire shoulder or knee, is probably not tendinitis or bursitis, and will not likely be cured with a local injection. In such instances where the diffuse pain is inflammatory in nature, an oral corticosteroid preparation may be more appropriate.

The development of ultrasound-guided therapy has led to improvement in the accuracy of corticosteroid injections. Used to evaluate musculoskeletal pathology since the 1970s, ultrasound is now steadily becoming an important tool for both diagnosis and therapy by identifying soft tissue structures such as bursae, tendons, and joint capsules.[16] [17] In addition, cartilage, muscles, nerves, and blood vessels can now be easily visualized using this technique. [16] Chapter 69 provides a detailed description of ultrasound-guided procedures.

Specific Regions and Clinical Entities

Shoulder Region

Pain associated with disability may result from any of the intrinsic shoulder disorders, including bicipital tendinitis, calcareous tendinitis, and subacromial bursitis (Fig. 53-2 (Figure Not Available)). These areas may be frequently injected because it is safe and easy to do so, and one gets a consistently good response to therapy. The danger of an untreated persistent inflammation is the development of a "frozen shoulder."

Bicipital tendinitis (Tenosynovitis).

This is a nonspecific low-grade inflammation of the biceps tendon or its sheath, or both, and is more common in those who repeatedly

Figure 53-2 (Figure Not Available) Pain in the shoulder is often caused by biceps tendinitis or subacromial bursitis. *(From Walker LG, Meals RM: Tendinitis: A practical approach to diagnosis and management. J Musculoskel Med 6:24, 1989. Reproduced with permission.)*

flex at the elbow against resistance, such as weightlifters and swimmers.[4] [18] The tendon courses through the joint and along the bicipital (intertubercular) groove, which can be appreciated as the elbow is held at 90° of flexion, and the arm is internally and externally rotated. [18] Patients may have restricted or normal range of motion and normal strength; however, they usually complain of tenderness to palpation over the bicipital groove. [4] Efforts to elevate the shoulder, reach the hip pocket, or pull a back zipper all aggravate the symptoms. Tenderness over the bicipital groove does not confirm the diagnosis; however, as the supraspinatus tendon is in such close proximity to the bicipital tendon insertion. [18] Other diagnostic clues include the Lipman test, in which "rolling" the bicipital tendon produces localized tenderness. The Yergason test elicits pain along the bicipital groove when the patient attempts supination of the forearm against resistance, holding the elbow flexed at a 90° angle against the side of the body (Fig. 53-3 (Figure Not Available)). Radiographs are normal and are not required if the clinical diagnosis is supported.

Approach.

The point of maximal tenderness of the bicipital tendon is located. Entry is made with a 22- or 25-ga, 3.9- to 5.0-cm needle through a lidocaine skin wheal (Fig. 53-4). It is wise to *avoid an actual intratendinous injection*, which may cause weakening of the tendon and predispose the patient to tendon rupture. The needle is brought in along the side of the tendon, aimed at one border of the bicipital groove to give a *peritendinous infiltration*. One third of the injection is administered at this point. The needle is then withdrawn slightly but is kept subcutaneous. It is redirected upward approximately 2.5 cm for another one third of the injection, withdrawn again, and redirected downward, touching the bicipital border gently; the remainder of the drug is deposited at this point. If the two-syringe technique is used, 1 to 1.5 mL of an intermediate-acting corticosteroid suspension, such as prednisolone tebutate, is usually instilled at the maximum area of tenderness. The anesthetic is injected along the upper and lower borders of the tendon.

Figure 53-3 (Figure Not Available) The Yergason test helps determine the stability of the long head of the biceps tendon in the bicipital groove. This test, which involves resisted supination of the forearm with the elbow flexed to 90°, may accurately reproduce symptoms of bicipital tendinitis. *(From Walker LG, Meals RM: Tendinitis: A practical approach to diagnosis and management. J Musculoskel Med 6:24, 1989. Reproduced with permission.)*

Calcareous tendinitis, supraspinatus tendinitis, and subacromial bursitis.

These inflammations are so clinically similar that their symptoms and signs are difficult to differentiate. The musculotendinous rotator cuff is composed of the supraspinatus, infraspinatus, teres minor, and subscapularis muscles, which insert as the conjoined tendon into the greater tuberosity of the humerus. The subacromial bursa lies just superior and lateral to the supraspinatus tendon (Fig. 53-5). Both the tendon and the bursa are located in the space between the acromion process and the head of the humerus, and are particularly prone to impingement in this "critical zone." Injections into either the bursa or tendon sheath area are commonly performed secondary to inflammation and overuse.

In *calcareous* (or calcific) tendinitis of the shoulder, there is a calcific deposit (hydroxyapatite) within the substance of one or more of the rotator cuff tendons (commonly the

Figure 53-4 *A,* Anatomic site for injection into the bicipital groove for treating bicipital tendinitis. The anesthetic is infiltrated around the biceps tendon sheath in a fan-wise distribution, avoiding intratendinous injection. *B,* Actual peritendon injection of bicipital tendinitis: The patient is seated with the forearm and hand resting in the lap and externally rotated.

supraspinatus).[19] These calcium crystals can occasionally rupture into the adjacent subacromial bursa, causing acute pain and tenderness in the deltoid area (Fig. 53-6). The bursae in relation to the greater tuberosity and the subdeltoid (subacromial) bursa are the most common sites of calcific deposits.

During the acute or hyperacute stage, the patient holds the arm in a protective fashion against the chest wall. Pain may be incapacitating, and all ranges of motion are disturbed, with internal rotation markedly limited. Tenderness is often diffuse over the perihumeral region. The patient may also complain of pain at night when lying on the affected side and with abduction of the arm. Constitutional symptoms are rare, but sometimes in the hyperacute form, actual swelling may be visible, and even fever and an accelerated sedimentation rate may develop. When shoulder radiographs demonstrate a calcific deposit, the shadow appears "hazy" with lightening of the periphery caused by the pressure of inflammatory edema. Night pain may be intolerable, requiring opioids for control.

Anterior approach.

In calcific tendinitis or supraspinatus tendinitis without calcification, the injection may be given by the anterior (subcoracoid) approach. The patient is asked to rest the extremity on the lap, and the arm is rotated externally about 15°. The point of insertion is over the depression that is palpable inferior and lateral to the coracoid process, and medial to the head of the humerus (Fig. 53-7).

Posterolateral approach.

With the patient sitting and the lower part of the extremity resting on the lap, a lidocaine skin wheal is made at the depression about 1 cm inferior to the posterolateral tip of the acromion, located between the head of the humerus and the acromion. A 3.9- to 5.0-cm, 22- or 25-ga needle is then directed toward the center of the head of the humerus and upward at an angle of approximately 10° (Fig. 53-8). After the site has been penetrated 2 to 3 cm, aspiration is carried out for any fluid or calcific material. The syringe is then removed, leaving the needle in position. Another syringe containing 20 to 40 mg of methylprednisolone suspension or equivalent intermediate-acting steroid is attached, and the medication is instilled. There should be little resistance when injecting the medication. If resistance is appreciated,

then the needle should be repositioned, as it may be in the tendon substance of the rotator cuff. This injection can be followed with 1 to 5 mL of 1% lidocaine (or a similar volume of 0.25% bupivacaine). Alternatively, local anesthetic can be given combined with the steroid in the same syringe. One should be generous with the volume of local anesthetic

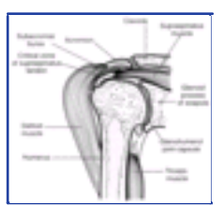

Figure 53-5 The "critical zone." Note the close relationship of the supraspinatus tendon and subacromial bursa to the humeral head and acromion.

to ensure adequate dispersion of the steroid. A single treatment relieves the majority of acute disorders. An injection into the peritendinous space is similar to that described earlier, except that the needle is advanced deeper than with a subacromial bursal injection. [20]

If calcific tendinitis is suspected, some recommend attempting to aspirate the calcium deposits. After the bursa has been anesthetized, an 18-ga needle can be used to penetrate the calcium, often creating a "gritty" sensation. [19] In addition, the practitioner may consider the technique of barbotage to facilitate the cleavage of calcium deposits. Using this method as described previously, the steroid or anesthetic/steroid combination is aspirated and re-injected repeatedly. [19]

Sometimes a painful reaction may follow when the analgesic has worn off. To avoid severe pain, the patient should be warned about this possibility and given appropriate analgesia. A sling may provide additional relief, and short-term use of opioids is appropriate. While some authors claim that patients who do not also undergo physical therapy after corticosteroid injection have satisfactory results, there is evidence to support the importance of close patient follow-up, range-of-motion exercises, and physical therapy to total recovery. [18]

Acromioclavicular joint inflammation.

Pain arising in the acromioclavicular (AC) joint is frequently an aftermath of an acute injury, such as falling on an outstretched hand or weightlifting. With this injury, all ranges of motion of the shoulder cause pain, and the joint is tender but rarely swollen. The practitioner should be aware of an obvious deformity or mechanism of injury that may suggest an AC separation or dislocation. With AC joint inflammation, crepitus is not uncommon. [18] Adduction of the arm across the body with forward elevation to 90° (the cross arm test) may also exacerbate the pain, as the AC joint is compressed with such motion. [18]

Approach.

Entry is made through a cutaneous lidocaine wheal over the interosseous groove at the point of greatest tenderness. This is usually just at the AC joint, which is palpated as a small "V"-shaped depression posteriorly at the

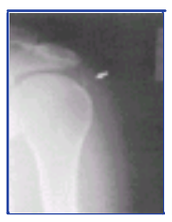

Figure 53-6 Abnormal calcific deposits in calcareous tendinitis of the shoulder are usually demonstrated roentgenographically in the suprahumeral region or adjacent to the greater tuberosity. When bursal calcifications appear dense (as in this x-ray film), they are frequently asymptomatic, whereas lightening at the margins of the calcium deposit is compatible with the presence of inflammatory edema in the rotator cuff, which produces pain and tenderness in the shoulder region. The location of the calcific deposit in the radiograph may be a useful guide for the point of entry of aspiration and injection. The needle is directed to the calcareous deposit, aspiration is carried out, and a portion of the steroid medication is deposited there. The calcium may subsequently disappear. Not infrequently, it does so spontaneously, without a needling procedure.

most lateral aspect of the clavicle (Fig. 53-9).[19] The joint line is relatively superficial, and a 2.2- to 2.5-cm, 22- or 25-ga needle is usually only advanced approximately 5 mm. One to 2 mL of lidocaine and 5 to 10 mg of a methylprednisolone suspension are injected. It is not necessary to advance the needle beyond the proximal margin of the joint surface.

Elbow Region

The elbow is subject to frequently occurring, characteristic, extra-articular disorders. These include radiohumeral bursitis, lateral and medial epicondylitis ("tennis elbow" and "golfer's elbow"), and olecranon bursitis ("barfly's elbow").

Radiohumeral bursitis, lateral epicondylitis, and medial epicondylitis.

Radiohumeral bursitis occurs at the juncture of the radial head and the lateral epicondyle of the elbow. This condition is commonly found in combination with lateral epicondylitis ("tennis elbow"), which is an inflammation at the insertion of the *extensor carpi radialis* and *extensor digitorum* muscles. The symptoms of the two adjacent problems are indistinguishable, but tenderness in bursitis overlies the site of the radiohumeral groove, whereas

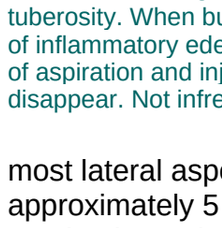

Figure 53-7 *A,* Anterior approach to the subacromial bursa or supraspinatus tendon. The needle is inserted into the depression located inferolateral to the coracoid process and medial to the humeral head.

tenderness in tennis elbow occurs chiefly at the lateral epicondyle (Fig. 53-10 (Figure Not Available)). There is often a history of repetitive motion, such as while golfing, gardening, or using tools. [4] A clinical sign supporting the diagnosis of tennis elbow is the provocation of pain when the patient attempts elevation of the middle finger against resistance, with the wrist and the elbow held in extension. Alternatively, pain is reproduced at the elbow when the patient is asked to extend the wrist against resistance (Fig. 53-11).

Medial epicondylitis ("golfer's elbow") is a similar condition, although it occurs on the opposite side as that of lateral epicondylitis and is much less common. [4] On physical examination, the patient will usually complain of pain when the wrist is *flexed* against resistance. Palpation of the medial epicondyle also elicits tenderness.

There is evidence to support the efficacy of corticosteroid injection for these conditions, particularly in the short term. In 1999 Hay and associates compared local corticosteroid injection, oral nonsteroidal anti-inflammatory medication, and other analgesics in 164 patients with lateral epicondylitis. [21] In 4 weeks, the injection group had the highest treatment success (defined as "better") rate (82%), versus the nonsteroidal

Figure 53-8 Posterolateral approach. *A,* The needle is inserted in the depression just inferior to the posterolateral tip of the acromion and superior to the head of the humerus. *B,* Injection using the posterolateral approach. The angle of entry should be approximately 15°.

anti-inflammatory (48%) and other analgesics group (50%). It should be noted, however, that after 1 year, the results were similar for all three groups (84% vs 85% vs 82%). Similarly, Stahl and colleagues studied 58 patients with medial epicondylitis who were first injected with methylprednisolone and then treated with both physical therapy and nonsteroidal anti-inflammatory agents. [22] In 6 weeks, the patients who received steroid injections reported significantly less pain compared to the placebo group. However, at 3 months and 1 year after the initial injection therapy, the two groups did not differ with regard to pain.

Approach.

With the elbow flexed at 90°, the radial head can be palpated as a bony protrusion just distal to the lateral epicondyle, and confirmed by rotation of the patient's forearm. The entry site is the point of maximal tenderness, usually found at a point slightly distal to the lateral epicondyle (Fig. 53-12). The injection is made through a skin wheal with a 3.9-cm, 22-ga needle, depositing 20 to 30 mg of methylprednisolone or equivalent intermediate-acting steroid mixed with anesthetic. Alternatively, the steroid is followed with 1 to 3 mL of local anesthetic. The injection should be infiltrated in a fan-like distribution, avoiding direct tendon injection. For radiohumeral bursitis, part of the repository preparation may be instilled into the radiohumeral bursa and part at the lateral epicondyle. In medial epicondylitis (golfer's elbow), a similar technique can be used, but it is important to remember to avoid the ulnar nerve, which lies in a groove behind the medial epicondyle. Damage to this nerve during steroid injection has been reported. [23]

Olecranon bursitis (Aseptic).

"Student's elbow," or "barfly elbow," is a common condition that is frequently idiopathic or provoked by minor trauma (Fig. 53-13*A* and *B*). [4] It is common for patients to present following some minor, often unrelated, trauma to the elbow region, and may also be seen in activities whereby the patient often leans on the elbow, such as gardening.[4] Acute trauma may result in a bursal hematoma from bleeding into the olecranon bursa, simulating acute bursitis. Although most cases of olecranon bursitis are sterile, the olecranon bursa is the most frequent site of septic bursitis. [24] The diagnosis of aseptic olecranon bursitis is obvious when the elbow is inspected and examined during flexion and extension. Radiographs are normal, but bony spurs (of questionable relationship) may be noted at the olecranon process, and amorphous calcific deposits may be seen. Occasionally in rheumatoid arthritis and gout, nodules, or tophi, may be readily palpated within the bursal sac. The olecranon bursa that is most commonly involved lies between the skin and the olecranon process. Motion at the elbow joint

Figure 53-9 Injection of the acromioclavicular joint. Entry is made at the "V"-shaped depression at the most lateral aspect of the clavicle.

remains complete and painless in aseptic bursitis. [4] In contrast, all motion is usually painful with septic arthritis.

Aseptic olecranon bursitis is cosmetically bothersome to the patient but does not cause discomfort and may resolve spontaneously unless there has been bleeding into the bursa or the effusion is extremely tense. When the bursa is large and subject to trauma or is tender and inflamed, provided that infection is excluded, aspiration and a steroid injection will expedite resolution. Smith and associates demonstrated the superiority of intrabursal methylprednisolone acetate over oral

Figure 53-10 (Figure Not Available) Lateral epicondylitis—commonly known as "tennis elbow"—is the result of microscopic rupture and incomplete tendinous repair of the *extensor carpi radialis brevis* origin on the lateral epicondyle of the humerus. Pain usually occurs over the lateral humeral epicondyle during work or recreation. *(From Walker LG, Meals RM: Tendinitis. A practical approach to diagnosis and management. J Musculoskel Med 6:24, 1989. Reproduced with permission.)*

naproxen or placebo at 6 months, noting faster resolution and less reaccumulation of fluid with the steroid injection. [25] The addition of a course of an oral nonsteroidal drug after steroid injection did not affect the outcome. Aspiration without injection is often followed by recurrence in a few days or weeks.

Septic bursitis.

Septic bursitis is most common in the olecranon and prepatellar bursae. [24] Infection of the other bursae is exceedingly rare. The infection is probably secondary to acute trauma or skin breakage resulting in direct penetration of the bursa by common skin pathogens, rather than by hematogenous spread. [26] [27] It would be impossible to clinically differentiate acute gouty olecranon bursitis from septic bursitis without laboratory analysis. Some unusual conditions that simulate bacterial septic olecranon bursitis include acute rheumatoid bursitis, aseptic bursitis secondary to oxalosis induced by dialysis, or infectious bursitis secondary to unusual organisms such as Mycobacterium, Serratia marcescens, or fungal organisms.

Septic bursitis is not associated with septic arthritis of the underlying joint, although, rarely, adjacent bone may become involved. [28] Many patients have an underlying predisposition to infection (e.g., diabetes mellitus, alcoholism, uremia, or gout). Rarely, an infection may follow an injection of corticosteroids into the bursa.

In most cases the diagnosis of septic bursitis is obvious (Fig. 53-14). The onset of pain and swelling may be quite rapid (over 8 to 24 hours), as compared with the more gradual onset of aseptic bursitis. The bursa is erythematous, tense, swollen, and very painful. Furthermore, the infected bursa is generally = 2.2°C warmer than the opposite elbow.[28A] The diagnosis of septic bursitis is confirmed by aspirating infected bursal fluid and culturing bacteria from the aspirate. Fluid is usually easily obtained from the tense bursa and (in the case of an established infection) is cloudy or grossly purulent.

The white blood cell count of the fluid in septic bursitis is usually 5000 to 100,000 cells/mm ^{3} or greater, and polymorphonuclear cells usually exceed 90%. Neither fever nor systemic leukocytosis are considered sensitive or specific for the disease. However, in immunocompromised patients (e.g., diabetes mellitus, alcoholism), the white blood cell count in the bursal fluid tends to be higher. [29] Because of some overlap in the leukocyte count of bursal fluid in septic versus nonseptic bursitis, it is important to remember that a low bursal white blood cell count does not exclude a septic etiology. Thus, Gram stain and culture are required in confusing cases. [24] Gram-positive organisms may be seen on Gram stain. The majority of septic bursitis cases are caused by penicillin-resistant *Staphylococcus aureus* (78%) in both immunocompromised and non-immunocompromised patients, followed by a small percentage of streptococcal organisms.[29]

Treatment of septic bursitis includes aspiration of the bursa and the use of antibiotics directed against penicillinase-producing *Staphylococcus*, splinting, hot soaks, and drainage of the bursa. Drainage may be adequately performed with one or more needle aspirations, but open incision and drainage may be required, particularly if the infection is recurrent or refractory. [26] *Corticosteroid injections should not be performed in infected tissue. They should also be withheld in the setting of high clinical suspicion for infection, pending negative bacteriology findings.* Outpatient therapy with oral antibiotics is generally acceptable, although this approach has been challenged for immunocompromised patients.[29]


Figure 53-11 Clinical test for lateral epicondylitis. The patient extends the wrist against resistance, while the examiner localizes the site of maximal tenderness at the lateral epicondyle.

Toxic-appearing, unreliable, or immunocompromised patients may be initially treated with intravenous antibiotics as inpatients, but no universally accepted standard exists. This decision is generally guided by the clinical appearance of the bursa, as well as associated comorbidities, compliance, and other factors regarding patient care. In general, the length of antibiotic therapy is often correlated with the length of time that the patient had symptoms before antibiotics were initiated. [29] Response to antibiotic therapy may be slow. Thus, it is important to initiate antimicrobial treatment once there is clinical suspicion for


Figure 53-12 *A,* Injection at lateral epicondyle for treatment of "tennis elbow." *(Redrawn from steinbrocker O, Neustadt DH: Aspiration and Injection Therapy in rthritis and Musculoskeletal Disorders: A Handbook on Technique and Management. Hagerstown, MD, Harper & Row, 1972. Reproduced with permission.) B,* The needle is inserted in the area of maximal tenderness, avoiding injection directly into the tendon.

septic bursitis. Additional treatment with oral nonsteroidal anti-inflammatory agents may also be effective. Standard gout medications will generally resolve acute gouty bursitis.

Approach.

A 2.5- to 3.9-cm, 20-ga needle is introduced at a dependent aspect of the bursal sac through a lidocaine skin wheal, or the skin may remain unanesthetized (Fig. 53-13C–E). If infected or inspissated fluid is anticipated, a 16- to 18-ga needle may be necessary for aspiration of the viscous contents. After as much fluid as possible is aspirated, 15 to 30 mg of methylprednisolone or an equivalent

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Figure 53-13 *A,* Anatomy showing location of the olecranon bursa superficial to the ulnar olecranon. *B,* traumatic painless swelling over the posterior elbow is characteristic of nonseptic olecranon bursitis. The mass is soft, movable, and fluctuant and is usually filled with serosanguineous fluid. *C,* Using sterile preparation, a 20-ga needle on a 10-mL syringe is advanced parallel to the forearm, and the bursal fluid is completely aspirated. *D,* The aspirating syringe is changed while the needle remains in the bursal sac, and a long-acting steroid preparation (such as 40 mg of methylprednisolone) is injected. An elastic bandage provides compression for 12 hours. *E,* An alternative posterior approach to the bursa. *F,* An extreme case of tophaceous gout involving the olecranon bursa. Most cases of acute gout bursitis resemble septic bursitis.

intermediate-acting preparation is injected. In aseptic bursitis the aspirated fluid may be yellow and clear, but often it is mildly serosanguineous in appearance. There are no compelling reasons to routinely send aspirated fluid for laboratory analysis or culture. The leukocyte count of aspirated fluid of aseptic bursitis should be less than 1000/mm^3 . Counts above 2000 to 10,000/mm^3 are associated with a higher incidence of infection or acute gout. The elbow is wrapped with an elastic compression bandage for 5 to 7 days following aspiration and injection. *Again, if septic olecranon bursitis is suspected, corticosteroid injections should not be used.*

Wrist and Hand Region

Ganglion cysts of wrist or hand.

These are cystic swellings occurring frequently on the hands, especially on the dorsal aspect of the wrist. Ganglion cysts are common and make up approximately 60% of all soft tissue tumors affecting the wrist and hand. They usually develop spontaneously in

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Figure 53-14 Fully developed septic olecranon bursitis is easily distinguished from nonseptic bursitis by clinical parameters. In the septic variety, there is diffuse swelling, as opposed to a discrete mass; the area is red, warm, and quite tender. In addition, bursal fluid leukocytosis is present.

adults between 20 and 50 years old, with a female-to-male ratio of 3:1. Ganglion cysts may also be seen on the foot and ankle, generally on the extensor surface.

The etiology of ganglia remains obscure. The word *ganglion* is derived from the Greek word meaning "cystic tumor." The mesothelium- or synovium-lined cystic structures are attached to or may arise from tendon sheaths or near the joint capsule and do not extend into the joint itself. [30] Attachment is often by a pedicle. The wall of the ganglia is smooth, fibrous, and of variable thickness. The cyst is filled with a clear, gelatinous, sticky, or mucoid fluid of great density. The viscous fluid in the cyst may sometimes represent almost pure hyaluronic acid.

The types of ganglia vary with their location. The most common ganglion is the dorsal wrist variety, which arises from the scapholunate joint and constitutes approximately 65% of ganglia. The volar wrist ganglion, which arises over


Figure 53-15 A typical volar ganglion cyst of the wrist (*A*) and dorsum of the foot (*B*).

the distal aspect of the radius, constitutes another approximately 20 to 25% of ganglia (Fig. 53-15). Flexor tendon sheath ganglia make up the remaining 10 to 15% found on the hand and wrist.

Most ganglia are of no great clinical significance and most do not require treatment. Spontaneous regression is common. However, if the appearance of the cyst is disturbing to the patient or if the ganglion is painful or tender (from soft tissue or nerve compression, or bone erosion), then simple aspiration with or without injection of a corticosteroid suspension is usually an effective approach. Up to three aspirations may be required before the technique is termed a failure. In one study, 69% of 116 patients required only a single aspiration for successful treatment. [31] Two or three aspirations were required for 19%, and only 12% of patients ultimately needed surgical excision. Aspiration with or without steroid injection is associated with considerable cost savings when compared with surgery. [25] The "old" treatment of attempting to rupture the ganglion with a heavy book is not advised due to the potential for local injury. In the event of failure of nonsurgical treatment, surgical

excision may be indicated. Reported recurrence rates after surgical removal range from 6 to 50%.

Approach.

Following instillation of 1% lidocaine for local anesthesia, a 2.5-cm, relatively large-bore needle (17- to 18-ga) is inserted into the center of the ganglion. The cyst is aspirated, with an attempt to achieve maximum evacuation of the contents. Usually 1 to 2 mL of mucinous fluid can be aspirated. Milking the contents of the cyst toward the aspiration needle can maximize the volume that is removed.[32] After localizing the cyst by aspiration, another smaller needle is used to simultaneously administer 10 to 15 mg of methylprednisolone (or equivalent intermediate-acting steroid). The use of steroids instilled into the cyst is a common procedure that has been proven to significantly augment the success of simple aspiration. [33] Following aspiration, a splint is usually not required, and activity need not be restricted. There is no proven role for routine nonsteroidal anti-inflammatory drugs or oral steroid therapy.

DeQuervain disease and intersection syndrome.

DeQuervain disease, a relatively common disorder, is a stenosing tenovaginitis of the short extensor tendon (*extensor pollicis brevis* [EPB]) and long abductor tendon (*abductor pollicis longus* [APL]) of the thumb. Although commonly referred to as a tenosynovitis, denoting inflammation of synovial sheaths of the tendons of the EPB and APL, this condition is more accurately described as a tenovaginitis. [34] Tenovaginitis refers to thickening of the fibrous sheath of the first extensor compartment. In 1912, deQuervain described "thickening of the dense fibrous connective tissues without any fresh sign of inflammation, neither round cell inflammation nor increase in numbers of cells".[34]

It is commonly thought that the disorder more often occurs following repetitive use of the wrists, especially with a wringing motion. The syndrome has been called "washerwoman's sprain," and often no specific etiology is apparent. However, a study by Kay and colleagues challenges the association

Figure 53-16 (Figure Not Available) *A,* de Quervain tenosynovitis occurs in the first dorsal compartment of the wrist secondary to tenosynovitis of the *abductor pollicis longus* and *extensor pollicis brevis* tendons. Symptoms, which include pain over the radial styloid, are generally caused by overuse. The Finkelstein test is positive when pain is reproduced by ulnar deviation of the wrist while the patient grasps the thumb with the fingers. *B,* Occasionally, crepitus may be palpated over the involved area. To elicit this sign, the examiner's fingers are placed over the painful area, and the patient's wrist is placed in ulnar deviation, which produces the characteristic sensation. *C,* Signs and symptoms of tenosynovitis of the wrist in a 25-year-old woman, which were initially thought to be de Quervain disease. The hemorrhagic papules found on the hand are classic for gonococcal bacteremia, and one aspect of this sexually transmitted disease is tenosynovitis. Cervical cultures were positive for *Neisseria gonorrhoeae*, despite the absence of vaginal symptoms. *(A from Walker LG, Meals RM: Tendinitis: A practical approach to diagnosis and management. J Musculoskel Med 6:24, 1989.)*

between repetitive motion and deQuervain disease. [34] In a retrospective study of 100 cases, there was not a strong correlation with occupation or history of repetitive activities in patients diagnosed with deQuervain disease. Thus, it may be possible that repetitive activities exacerbate the pain associated with a condition for which the etiology is unclear.

Tenderness and occasionally palpable crepitation is elicited just distal to the radial styloid process where both tendons come together in an osseofibrous tunnel. Patients usually present with wrist pain and often mistakenly attribute the discomfort to some distant, albeit irrelevant, trauma. The condition may be confused with first carpometacarpal arthrosis (osteoarthritis of the thumb) and intersection syndrome. Radiographs will be normal in deQuervain disease but may be appropriate to rule out other pathologic abnormalities. Rarely, gonococcal tenosynovitis may simulate this inflammatory condition.

A useful specific clinical maneuver that indicates deQuervain disease is the Finkelstein test (Fig. 53-16 *A* (Figure Not Available)),

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performed by abducting the patient's thumb into the palm of the hand and folding the fingers over the thumb. Forcible ulnar deviation at the wrist is then carried out, with severe pain at the site of the affected tendon sheaths indicating a positive test. When performing the Finkelstein test, one should also evaluate the tender area for palpable crepitus (see Fig. 53-16 *B* (Figure Not Available)). If axial traction or compression (the "carpometacarpal grind test") and rotation of the thumb produce pain, the condition is most likely due to degenerative changes of the carpometacarpal joint of the thumb rather than from deQuervain disease. It should be noted that gonococcal tenosynovitis of the wrist may mimic deQuervain disease, and one should inquire about other symptoms (e.g., sore throat, penile or vaginal discharge, or fever) and carefully look for the characteristic rash of this sexually transmitted disease (see Fig. 53-16 *C* (Figure Not Available)).

In a study comparing steroid injection of deQuervain disease with a splint and oral nonsteroidal anti-inflammatory therapy alone, the latter traditionally accepted initial treatment was effective only in a small group of patients with minimal symptoms.[35] Steroid injection therapy was recommended as the initial treatment once the disease began to cause an interference with activities of daily living or if symptoms were more severe. Overall, there is increasing evidence supporting the effectiveness of local corticosteroid injection as the therapeutic preference for deQuervain tenovaginitis. In 1 report, 90% of 56 cases were effectively managed either with a single injection (58%) or with repeat injections (33%), with only 10% requiring surgical treatment. [20] These data support a previous report describing an 83% rate of successful response to injections in 63 wrists with deQuervain tenosynovitis. [36] The strikingly favorable response to local injection therapy suggests that surgery to release the tendon sheaths is seldom needed.

Accurate injection of the corticosteroid has been found to be an important aspect of the patient's response to treatment. [37] Using radiographic dye to verify correct corticosteroid placement, researchers have found that when the corticosteroid and anesthetic injection did not successfully reach either the EPB or APL compartment, the patient did not experience relief of pain. Conversely, when the medication was accurately injected, most patients reported an improvement of symptoms.

Intersection syndrome is a condition that may be easily confused with deQuervain disease. Because the treatment approach and clinical course of intersection syndrome differ from that of deQuervain tenovaginitis, it is important that the clinician is also familiar with this entity. First described in 1841 by Velpea, intersection syndrome describes a clinical entity approximately 4 to 8 cm proximal to the location of deQuervain disease. [38] Although the etiology is not yet clear, some authors believe it to be an inflammation of the second dorsal compartment of the wrist, which houses the *extensor carpi radialis longus* (ECRL) and *extensor carpi radialis brevis* (ECRB) tendons.[39] Others report that the primary site of pathology lies in the compression of the ECRL and ECRB muscles by the muscle bellies of the APL and EPB (Fig. 53-17). [40]

Intersection syndrome presents with pain, tenderness, edema, and occasional crepitus 4 to 8 cm proximal to the radial styloid (Fig. 53-18). Treatment includes rest, nonsteroidal anti-inflammatories, and immobilization with a thumb spica splint (see Chapter 51). Corticosteroid injection

Figure 53-17 *A,* Site of de Quervain disease at the *extensor pollicis brevis* (EPB) and *abductor pollicus longus* (APL) tendons. *B,* Site of intersection syndrome at the *extensor carpi radialis longus* (ECRL) and *extensor carpi radialis brevis* (ECRB) tendons.

therapy is recommended only after a 2 to 3 week trial of splinting. Approximately 60% of patients will respond with nonoperative treatment. [39] Surgery for this condition is reserved for refractory cases.[38] In contrast, steroid injection therapy is recommended earlier in the course of deQuervain disease. Some authors, in fact, recommend corticosteroid injection therapy on initial presentation of deQuervain disease. [38]

Approach.

For deQuervain disease, the hand is positioned with the ulnar side of the wrist on the table and the radial side facing upward. A 2.2-cm, 25-ga needle is introduced at the most tender point (about 1 cm distal to the radial styloid) through a skin wheal, and 10 to 20 mg of prednisolone or equivalent intermediate-acting steroid suspension mixed with 4 to 5 mL of lidocaine 1% are deposited adjacent and parallel to the tendon sheath (peritendinous infiltration) (Fig. 53-19*A* and *B*). The

injection should be under the edge of the first dorsal compartment retinaculum, within the first extensor compartment. If firm resistance is appreciated, the needle should be redirected to prevent intratendinous injection. Because there are many superficial vessels in this area, it is important to aspirate before injecting, to verify that the needle is not in a blood vessel. [37] One should be generous with the injection volume, since a common reason for failure is the inability to get medication into both tendon sheaths. This may

Figure 53-18 Photograph of a patient's wrist with intersection syndrome. Note that the swelling (arrow) is several centimeters *proximal* to the radial styloid, overlying the compartment of the common wrist extensors (*extensor carpi radialis longus* and *extensor carpi radialis brevis*). *(From Hanlon DP, Luellen JR: Intersection Syndrome: A case report and review of the literature. J Emerg Med 17:970, 1999. Reproduced with permission.)*

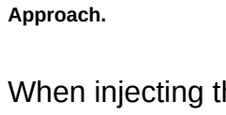

Figure 53-19 Injection of abductor tendon sheath (de Quervain disease). When giving an injection for de Quervain disease, it is desirable to avoid direct tendon injection and to instead aim for a peritendinous infiltration (*A*). If 3 to 4 mL of a local anesthetic is added to the steroid, generous infiltration over a diffuse area can compensate for a less than perfect localization (*B*). *(A from Steinbrocker O, Neustadt DH: Aspiration and Injection Therapy in Arthritis and Musculoskeletal Disorders: A Handbook on Technique and Management. Hagerstown, MD, Harper & Row, 1972. B from Zingas C, Failla JM, Holsbeeck MV: Injection accuracy and clinical relief of de Quervain tendinitis. J Hand Surg 23a:89, 1998. Reproduced with permission.)*

be partially overcome by increasing the volume of steroid-lidocaine injected in a more diffuse area. Often, there is visible edema at the radial aspect of the first metacarpal base and at the thumb metacarpophalangeal joint dorsally after successful injection. [37] A lightweight thumb or wrist splint for wrist support and protection may be used at night for several weeks after the injection, but routine splinting following injection is not required. [38] Oral nonsteroidal anti-inflammatory agents may be prescribed for analgesia but will likely not effect a cure by themselves. There is no proven role for oral corticosteroids.

Injection for intersection syndrome is similar to that of deQuervain disease, except that the target site is at the point of maximal tenderness, which is usually 4 to 8 cm proximal to the radial styloid.

Carpal tunnel syndrome.

Carpal tunnel syndrome is a common nerve entrapment caused by median nerve compression in the fibro-osseous tunnel of the wrist. It is characterized by pain at the wrist that sometimes radiates upward into the forearm and is associated with tingling and paresthesias of the palmar side of the index and middle fingers and the radial half of the ring finger. Typically, the patient wakes during the night with burning or aching pain, numbness, and tingling. Occasionally the discomfort is extremely severe, causing the patient to seek emergency care. Clinical signs supporting the diagnosis include a positive Tinel sign, elicited by reproducing the tingling and paresthesias by tapping (with a reflex hammer) over the median nerve at the volar crease of the wrist. [41] In addition, one can perform the Phalen test, described as flexed wrists held against each other for several minutes in an effort to provoke the symptoms in the median nerve distribution. [41] Severe muscle atrophy of the thenar eminence may develop in advanced or neglected cases. In many cases the disturbance is idiopathic, without a recognizable underlying cause. Other causes of carpal tunnel syndrome include rheumatoid arthritis (sometimes as the presenting manifestation), pregnancy, hypothyroidism, diabetes, and acromegaly.

Approach.

When injecting the carpal canal, one should insert the needle through a skin wheal at a site just radial or ulnar to the *palmaris longus* tendon and proximal to the distal crease at the wrist. The *palmaris longus* tendon can be appreciated by having the patient oppose the thumb and fifth digit while flexing the wrist (Fig. 53-20).[42] Injecting medial (ulnar) to the *palmaris longus* is preferred because it avoids direct injection of the median nerve and superficial veins. A 2.5- to 3.9-cm, 25-ga needle is directed at an angle of approximately 60° to the skin surface, pointing toward the tip of the middle finger. The needle is advanced 1 to 2 cm, and 20 to 40 mg of methylprednisolone (or other intermediate-acting steroid equivalent) with or without lidocaine is injected along the track and into the tissue space. Up to 2 weeks may be required for paresthesias to abate significantly, although it usually only takes a few days for an improvement in nocturnal pain. [43] A lightweight wrist splint may hasten recovery. Repeat injections may be given, but if a response is not elicited or permanent after two or three injections, decompressive surgery should be considered. Surgery is reserved for medically recalcitrant cases, thenar atrophy, or patients with significant sensory loss. [10] [42] Nerve compression should be confirmed by nerve conduction studies before surgery. [37]

Digital flexor tenosynovitis ("Trigger finger").

A "trigger" or "snapping" finger is characterized by a stenosed tendon sheath on the palmar surface over the base of the metacarpal head. The area of stenosis leads to intermittent catching of the tendon. Locking occurs when the involved digit is in flexion and is especially troublesome when the patient awakens in the morning. This can occur in any finger but is seen most frequently in the ring and middle fingers.

Besides a thickening and stenosing of the tendon sheath, trigger finger may also be characterized by flexor tendon synovitis.

The tendon sheaths are long and tubular, and the walls are lined with a thin layer of synovial cells. Symptoms develop when the tendon becomes trapped and is unable to glide within the tendon sheath (Fig. 53-21 (Figure Not Available)). A nodule or fibrinous deposit may form at a site in the tendon sheath, usually over or just distal to the metacarpal head in trigger finger. Carpal tunnel syndrome commonly coexists with trigger finger and may be caused by tenosynovitis. The common causes of tenosynovitis include trauma, diabetes mellitus, and rheumatoid arthritis, although it may also be a primary and idiopathic disorder. [39]

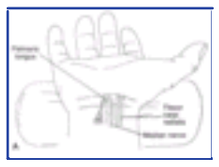

Figure 53-20 *A,* Injection of the median carpal tunnel with the wrist dorsiflexed over a rolled towel. To avoid direct injection of the median nerve, the needle is introduced just medial (ulnar) to the *palmaris longus* tendon. *B,* A palpable bulge at the distal edge of this ligament indicates correct placement of the injection. Direct injection into the median or ulnar nerve is to be avoided. *(A redrawn from Steinbrocker O, Neustadt DH: Aspiration and Injection Therapy in Arthritis and Musculoskeletal Disorders: A Handbook on Technique and Management. Hagerstown, MD, Harper & Row, 1972. B from Dehaan MR, Wilson RL: Diagnosis and management of carpal tunnel syndrome. J Musculoskel Med 6:47, 1989. Reproduced with permission.)*

Conservative treatment, including local rest or splinting, application of moist heat, and non steroidal anti-inflammatory therapy is the usual initial therapeutic approach for symptomatic tenosynovitis. If these simple measures fail to control symptoms, then corticosteroid injection, appropriately administered to the involved tendon sheath, is indicated. One double-blind, placebo-controlled, randomized study of trigger finger demonstrated that steroid injection was significantly more effective than placebo.[44] At follow-up 3 weeks post treatment, 9 of the 14 patients receiving a combination lidocaine and steroid injection were asymptomatic, versus 2 of the 10 patients receiving lidocaine injections alone. A similar study

Figure 53-21 (Figure Not Available) Trigger finger (stenosing tenosynovitis) can affect any digit, including the thumb, as shown here, but it is most common in the ring and middle fingers. In advanced cases, inflammation at the proximal (A1) pulley of the flexor tendon sheath overlying the metacarpophalangeal joint may hold the digit in either a flexed or an extended position. Generally, the tendon becomes thickened either proximal or distal to the pulley, causing a snapping or a locking phenomenon with finger flexion or extension. Palpation of the flexor tendon sheath over the metacarpophalangeal joint often reproduces symptoms. *(From Walker LG, Meals RA: Tendinitis: A practical approach to diagnosis and management. J Musculoskel Med 6:41, 1989. Reproduced with permission.)*

showed improvement in 1 month in 60% of patients receiving a corticosteroid and lidocaine injection, compared with a 16% improvement rate in a control group that

received lidocaine alone.[45] Alternatively, there is evidence to suggest that the efficacy of steroid injections for trigger finger diminishes with each subsequent injection in the same site.[46] This suggests that surgery may be an acceptable alternative if the patient does not obtain relief of symptoms after the first injection. [46] In particular, patients with diabetes mellitus more commonly require surgical release for trigger finger compared with non-insulin-dependent diabetic patients. [47] Division of the first annular pulley, digital nerve injury, scarring, and recurrence are well-known complications of surgical release. [46]

Approach.

Preparation of the site before the injection requires meticulous adherence to aseptic technique. The technique for injecting flexor tenosynovitis includes placing the palm in an upward direction and locating the tendon point at the base of the finger flexion crease, which is located between the A1 and A2 tendon pulleys. Approximately 0.25 to 0.35 mL of an intermediate-acting corticosteroid suspension mixed with 1.5–3 mL of anesthetic is subcutaneously injected with a 2.2-cm, 25-ga needle angled slightly proximally into the involved tendon sheath (Fig 53-22). The needle enters the

Figure 53-22 Injection of flexor tendon sheath in digital tenosynovitis.

skin at an angle of approximately 30° and is inserted into the tendon sheath, parallel to the tendon fibers. If resistance is felt on needle insertion, this suggests an intratendinous location, and the needle should be slightly withdrawn prior to injection. This injection can be quite painful. Local epidermal and SQ lidocaine may make the more critical tendon sheath placement tolerable. Similar injections can be administered to the base of the thumb metacarpal for a "snapping" thumb. If relapses are frequent or the clinical response is not satisfactory, then surgical release is indicated.

Hip Region

Trochanteric bursitis.

Trochanteric bursitis is the second leading cause of lateral hip pain after osteoarthritis. [48] However, the condition can be confused with other conditions at or about the hip, such as sacro-lumbar disease, hip/femur pathology, and metastases. [28] [49] Patients with trochanteric bursitis, however, usually demonstrate discrete tenderness to deep palpation at or adjacent to the greater trochanter, and relief of symptoms with a proper corticosteroid injection can help confirm the diagnosis. [49]

The principal bursa lies between the gluteus maximus and the posterolateral prominence of the greater trochanter, although other bursae of the hip are also commonly affected (Fig. 53-23). Pain may be acute but is more often subacute or chronic; often patients have tried nonsteroidal anti-inflammatory agents without success and have been given a number of incorrect diagnoses. The chief locus of the pathologic condition is in the abductor mechanism of the hip. Pain occurs near the greater trochanter and may radiate down the lateral or posterolateral aspect of the thigh. Pain is described as deep, dull, and aching, and it often interferes with sleep.

Lying on the side of the hip, stepping from curbs, and descending steps provokes pain. Tenderness may be elicited over and adjacent to the greater trochanter. In contrast with true hip involvement, the Patrick *fabere* sign (*f*lexion, *ab*duction, *e*xternal *r*otation, and *e*xtension) may be negative, and complete passive range of motion is relatively painless. Active abduction when the patient lies on the opposite side typically intensifies the discomfort, and sharp external rotation may accentuate the symptoms. Internal rotation does not usually affect the level of pain. [49] Hip radiographs may demonstrate a

Figure 53-23 Trochanteric bursitis. The principal bursa lies between the gluteus maximus and the greater trochanter, although other bursae may also be involved.

calcific deposit adjacent to the trochanter; however, the incidence of this is low. [28]

Corticosteroid injection for trochanteric bursitis is often an effective therapy. [28] In one study 77% of patients reported improvement of their pain after an injection of betamethasone mixed with lidocaine.[50] In addition, 61% of patients reported improvement of pain 26 weeks after receiving the injection.

Approach.

Intrabursal injection uses the site of maximum tenderness for the entry point. A 3.9- to 5.0-cm, 20- or 21-ga needle is advanced until the needle tip reaches the trochanter. The needle is then withdrawn slightly, and the site is infiltrated fairly widely with 3 to 10 mL of lidocaine and 20 to 40 mg of methylprednisolone or the equivalent.

Ischiogluteal bursitis.

"Weaver's bottom" is a painful disorder characterized by pain over the center of the buttocks with radiation down the back of the leg. [28] This condition is rarely initially diagnosed, often being mistaken for lumbosacral strain, a herniated disk, or spinal cord tumor. When it is recognized, a skillful intrabursal injection, coupled with a few days' rest, usually relieves the extreme pain. The ischial or ischiogluteal bursa is adjacent to the ischial tuberosity and overlies the sciatic and posterior femoral cutaneous nerves. Sitting on hard surfaces, bending forward, and standing on tiptoe provokes pain. Tenderness is present over the ischial tuberosity.

Approach.

The usual technique for injection requires that the patient lie in a prone position. A 5.0-cm, 20- to 22-ga needle is inserted through a skin wheal and is advanced cautiously in an effort to avoid the sciatic nerve, which lies at a depth of approximately 6.5 to 7.5 cm. Paresthesias occur on striking the nerve, and if this occurs, the needle should be withdrawn from the nerve. Generally, 5 to 10 mL of lidocaine and 20 to 40 mg of methylprednisolone are introduced into the bursa.

Knee Region

Prepatellar bursitis.

"Housemaid's knee," or "nun's knee," is characterized by swelling with effusion of the superficial bursa overlying the lower pole of the patella (Fig. 53-24). Passive motion is fully preserved, and pain is

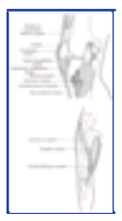

Figure 53-24 Prepatellar bursitis and *pes anserine* bursitis. The prepatellar bursa lies superior to the patella, while the *pes anserine* bursa lies deep to the insertion of the sartorius, gracilis, and semitendinosus tendons.

generally mild, except during extreme knee flexion or direct pressure. Although the disorder is usually caused by pressure from repetitive kneeling on a firm surface ("rug cutter's knee"), rarely it can develop after direct trauma, and occasionally it is a manifestation of rheumatoid arthritis. Although uncommon, the prepatellar bursa is one of the most common sites of septic bursitis.[4] The bursal aspirate should therefore always be sent for laboratory analysis, particularly if infection is suspected. [4]

Approach.

The patient should be in a supine position with the leg extended. The bursa is located superficially, between the skin and the patella, and can oftentimes be "milked" during the procedure to facilitate aspiration. This aspiration often yields a surprisingly scant amount of clear, serous fluid because the prepatellar bursa is a multilocular structure rather than the usual single cavity. Once aspiration is complete, the instillation of 1 to 2 mL of lidocaine with 15 to 20 mg of a prednisolone (or equivalent) suspension with a 2.5-cm, 20- to 21-ga needle is usually sufficient to cause the swelling to abate. In some cases the procedure may need to be repeated more than once (in 6- to 8-week intervals) to obtain a lasting result. The provocative activity should be discontinued.

Suprapatellar bursitis.

Suprapatellar bursitis usually is associated with synovitis of the knees. On occasion the bursa is largely separated from the synovial cavity with only a very minor communication, and the swelling and effusion are chiefly confined to the suprapatellar area. This may be traumatic in origin or an associated manifestation of an inflammatory arthropathy.

Approach.

The procedure for aspiration and injection of the suprapatellar area is similar to that for the knee (see Chapter 54).

Anserine bursitis.

"Cavalryman's disease" now mainly occurs in obese women with disproportionately large thighs in association with osteoarthritis of the knee, although this entity is also seen in athletes who are involved with running, baseball, and racquet sports. [28] The bursa is on the anteromedial side of the knee, inferior to the joint line at the site of the insertion of the conjoined tendons of the sartorius, semitendinous, and gracilis muscles and superficial to the medial collateral ligament (see Fig. 53-24). The entity is characterized by a relatively abrupt onset of knee pain, with localized tenderness and a puffy sensation in the vicinity of the anserine bursa about 4 to 5 cm below the medial aspect of the tibial plateau. Pain is exacerbated by flexion of the knee.

Approach.

The patient is positioned with the knee flexed at 90°. An injection of 2 to 4 mL of lidocaine, with or followed by approximately 20 to 40 mg of a corticosteroid suspension, is given using an anterior or medial approach with a 2.5- to 3.9-cm, 22-ga needle (Fig. 53-25). At the point of greatest tenderness, the needle is gently advanced until the tibia is reached, the needle is withdrawn 2 to 3 mm, and the steroid is then injected. Prompt symptomatic relief is frequently obtained, but the duration of benefit is variable and probably correlates with the patient's weight-bearing activities. It is important to avoid direct injection of the corticosteroid suspension into the nearby tendons.

Medial collateral ligament bursa.

This bursa is located anterior to the tibia and posterior to the medial collateral ligament (MCL). Injury to this bursa often occurs when the patient undergoes a twisting motion, with concurrent external rotation of the tibia. Tenderness may be appreciated along the anteroinferior aspect of the medial collateral ligament, and the pain is exacerbated with extension of the knee. This condition may sometimes be confused with a medial meniscus tear, and magnetic resonance imaging may be necessary to differentiate the two conditions. Treatment is usually successful with conservative measures, including relative rest, compression, and nonsteroidal anti-inflammatory agents.[28]

Popliteal cyst.

"Baker cysts" are herniated fluid-filled sacs of the articular synovial membrane that extend into the *popliteal fossa*, sometimes through the natural communication between the bursa of the posterior knee and the joint itself. These cysts may also be due to swelling of the medial gastrocnemius or semimembranosus bursae alone. Baker's cysts can occur secondary to trauma, although they are also seen in patients with rheumatoid arthritis, gout, and osteoarthritis. [28] The patient will often complain of *popliteal fossa* tenderness and swelling that may extend into the calf. Activities that involve active flexion of the knee, such as walking or jumping, will exacerbate the symptoms.

Figure 53-25 Injection of the anserine bursa of the knee. Note that the point of entry, which is the point of maximal tenderness, is inferior to the patella and medial to the tibial tuberosity.

The clinical presentation of Baker cysts can mimic that of a deep venous thrombosis (DVT), and it is important to take great care in differentiating the two. For this reason, symptomatic Baker cysts are also known as the pseudothrombophlebitis syndrome.[51] In most studies, 2% to 6% of patients suspected of having a DVT actually have a popliteal cyst as the cause of their knee/calf pain. Ultrasound is an important tool in this diagnosis.

Many cases of Baker cysts resolve spontaneously over a few weeks. However, treatment of Baker cysts may require surgery to correct any articular injury or to remove the cyst. Steroid injections are not usually performed because of the risk of neurovascular injury.

Patellar tendinitis.

The patellar tendon (also known as patellar ligament because it connects two bones) should not be injected in the ED since this weight-bearing tendon is subject to rupture with repeated injections. Referral to an appropriate specialist is recommended. Patellar tendinitis can be a manifestation of gout.

Quadriceps tendinitis.

The quadriceps tendon apparatus should not be injected in the ED since this weight-bearing structure is subject to spontaneous rupture and rupture secondary to repeated injections.

Ankle, Foot, and Heel Region

Ankle tendinitis.

This is a relatively uncommon condition. It may result from unusual repetitive activity or, rarely, from acute trauma. The disorder is differentiated from ankle joint involvement by the lack of pain or restricted motion during passive flexion and extension of the ankle. Active flexion and extension of the toes does produce pain. Local tenderness is elicited along the flexor and extensor tendons. Injection of the tendon sheaths is useful, producing considerable relief of symptoms. Tenosynovitis of the dorsum of the foot occurs in acute gout and can mimic acute tendinitis or cellulitis.

Approach.

A 2.5- to 3.9-cm, 22- or 25-ga needle is used. One makes a tangential entry to the enlarged tendon sheath, distending the sheath with approximately 2 to 4 mL of a mixture of corticosteroid (e.g., 20–40 mg of methylprednisolone) and lidocaine.

Bunion bursitis.

It is common for bunion bursitis to overlie the first metatarsophalangeal joint at its medial surface on the great toe. On occasion, tense swelling occurs, and decompression is required. Aspiration with culture of the fluid should be performed.

Approach.

If no infection is present, the bursa is injected with 5 to 10 mg of methylprednisolone with a 2.5-cm, 20-ga needle. Special shoes or an orthopedic correction will be needed if the swelling recurs.

Heel pain.

"Talalgia" has many different causes, including Achilles tendinitis, retrocalcaneal bursitis, or plantar fasciitis (Fig. 53-26). The bursae of clinical significance around the heel include the space between the skin and the Achilles tendon, the retrocalcaneal bursa, and the subcalcaneal bursa. Achilles tendinitis or bursitis may be traumatic in origin but is more apt to be part of a systemic disease, such as rheumatoid or gouty arthritis. Although the normal Achilles tendon is thick and strong, when affected by an inflammatory arthropathy, it is predisposed to degeneration, and since the Achilles tendon is not invested by a full synovial sheath, it is more vulnerable to intratendon instillation. Because of the potential hazard of tendon rupture after local steroid injection, it is wise to avoid infiltration of steroids into this area. In a double-blind, randomized, controlled trial, it was shown that the injection of methylprednisolone and Marcaine had no benefit over injecting Marcaine alone.[7] It is preferable to treat Achilles tendinitis with rest, splinting, and oral medication.

Retrocalcaneal bursitis is often seen in association with Haglund disease, which is a bony ridge on the posterosuperior aspect of the calcaneus. The bursa lies anterior to the Achilles tendon, and posterior to the calcaneus. Local swelling and tenderness at the posterior heel, proximal (and sometimes lateral) to the insertion of the Achilles tendon, characterize this bursitis. Treatment is focused on minimizing pressure on the bony ridge, which includes open heels (clogs), bare feet, sandals, or a heel lift. Conservative measures such as ice, nonsteroidal anti-inflammatory medications, and rest are other common treatment modalities. See Chapter 52 for more discussion of these conditions.

The condition in this region most amenable to injection therapy is plantar fasciitis, which is frequently associated with painful heel spurs ("policeman's heel" or "soldier's heel"). Plantar fasciitis involves collagen degeneration of the plantar fascial band at its origin at the medial aspect of the calcaneous. [52] The familiar signs of this condition include pain on the plantar medial aspect of the heel, which is often worse in the morning and after long periods of rest. The pain may be relieved with activity. Although patients may have a heel spur, many symptomatic patients do not. The pathology is thought to originate from microtears in the fascia, often due to

Figure 53-26 Heel pain. Talalgia may involve the tendons, bursa, or fascia around the heel.

overuse.[52] [53] Treatment begins with elimination of precipitating activity, relative rest, strength and stretching exercises, arch supports, and night splints. If these conservative measures are not effective, injection of the painful heel is sometimes beneficial. Studies have demonstrated a 70% success rate with steroid injections. [52] However, there is a risk of plantar fascia rupture and fat pad atrophy. [52] Since this tends to be a chronic or recurring condition, referral to the appropriate specialist is recommended following initial injection therapy. Orthotic devices in the shoes are often prescribed with good success.

Approach.

At the spot of maximal tenderness, a 2.5-cm, 22- to 24-ga needle enters the plantar surface at 90°, sliding into the space at the midpoint of the calcaneus. The tip of the needle lies in the aponeurosis of the attachment to the os calcis (Fig. 53-27). One milliliter of lidocaine and 10 to 20 mg of methylprednisolone are instilled.

HAZARDS AND COMPLICATIONS

Local anesthetics are often mixed with a corticosteroid preparation to increase volume, decrease postinjection pain, and assess the accuracy of the injection. Local anesthetics may also be used alone, before injection of the corticosteroid. The major hazards in the use of local anesthetics are hypersensitivity and accidental intravenous or intra-arterial introduction. Serious or fatal hypersensitivity to procaine and other regional anesthetic compounds is encountered very rarely; the possibility is usually suggested by a history of previous reactions. Lidocaine or one of the newer amide derivatives may be used to avoid sensitivity reactions to procaine (or other ester derivatives). Bupivacaine is an excellent choice because of its safety and long duration of action. When there is a definite history of sensitivity, the use of anesthetic agents during injection therapy is absolutely contraindicated.

In the event of accidental intravenous injection of an amide drug or if symptoms of hypersensitivity arise from these compounds, such as a significant slowing of the pulse rate or a seizure, a benzodiazepine or a rapidly acting barbiturate (e.g., sodium pentobarbital) should be given promptly in accordance with the reaction and response (see

Figure 53-27 Injection of calcaneal bursitis with heel spur. *(Redrawn from Steinbrocker O, Neustadt DH: Aspiration and Injection Therapy in Arthritis and Musculoskeletal Disorders: A Handbook on Technique and Management. Hagerstown, MD, Harper & Row, 1972.)*

Chapter 30). In addition, the patient should be placed on a cardiac monitor, oxygen should be administered, and a patent airway maintained. Severe reactions from accidental vascular injection of these drugs in the doses usually given are very rare.

No serious reaction to a depot corticosteroid preparation given intravenously has been reported. The possibility of an allergic reaction caused by corticosteroids is highly unlikely. However, an unusual skin rash following an intra-articular methylprednisolone injection, which appears to be consistent with a delayed type of hypersensitivity, has been reported. [11]

Minor reactions occasionally seen after injection of amide preparations include light-headedness or dizziness, pallor, weakness, sweating, nausea, and (rarely) fainting and tachycardia. These symptoms usually disappear within a few minutes after the injection and rarely require any treatment except reassurance and a cold compress to the forehead of the patient. Often it is difficult to decide whether the symptoms are the result of sensitivity to the drug or a fright (vasovagal) reaction. *The patient should always be in a supine, prone, or reclining position during the injection to minimize the effect of any vasovagal reaction.*

The obvious ways to prevent entering a blood vessel are an awareness of the local anatomy and aspiration after every 1 to 2 mL of solution are injected. Penetration into or striking a nerve may cause sharp pain or paresthesias, and the patient should be warned of this possibility in advance.

Corticosteroid injections have been found to be safe procedures with few complications (Table 53-5). [54] Although the possibility of introducing infection is one of the most serious potential complications, infections occurring as an aftermath of intrasynovial injections are extremely rare. In a study at the Mayo Clinic, no infections were reported for 3000 injections given in 1 year. [11] Others have found the infection risk to be 4.6 per 100,000 intra-articular injections. [55]

Although the problem of infection is usually avoided with meticulous attention to aseptic technique, the patient should be cautioned to report the development of any significant pain, redness, or swelling after any local injection. We do not recommend routine prophylactic antibiotic administration following corticosteroid injection.

Local undesirable reactions are usually minor and reversible. After steroid injection, about 2% of patients may experience an acute synovitis otherwise known as "post injection" flare. [10] [12] Characterized by an increase in pain and joint swelling, symptoms usually begin a few hours after steroid injection and can last as long as 3 days. Histologically, steroid

TABLE 53-5 -- Complications of Local Injection Therapy

Postinjection flare
Afterpain
Bleeding (local)
Cutaneous atrophy (local)
Depigmentation
Tendon rupture
Intraneural steroid deposit
Facial flushing
Hyperglycemia
Intravascular injection
Adrenal suppression
Infection

crystals within polymorphonuclear cells have been demonstrated, making them true synovitises. [10] This reaction may be difficult to differentiate from an infection, and infection must be ruled out if symptoms last greater than 48 hours or are associated with fever, warmth, or other suspicious signs of infection. The incidence of postinjection flare appears to be more likely with more soluble (shorter-acting) steroid solutions, and may be related to the carrier in which the steroid is manufactured. [8] [11] Usually, the reaction is mild and can be adequately controlled with application of ice or cold compresses and analgesics as needed.

Rarely following injections, "after pain" may occur up to several hours. Although the cause is obscure, this phenomenon may result from the trauma of needle insertion, penetration of inflamed tissue, or pressure on adjacent nerves from local swelling or bleeding. "After pain" is usually relieved by application of moist or dry heat and analgesics until the pain abates but is best prevented by mixing a long-acting anesthetic such as bupivacaine with the steroid preparation.

Occasional subcutaneous bleeding at the site of injection may occur with penetration of a venule, an arteriole, or a capillary. The patient should be warned that this may occur and should be reassured that the discoloration or hematoma will disappear spontaneously. Ice packs or cold compresses applied to the involved area for the first 24 hours are commonly advised.

Another relatively minor complication is localized subcutaneous or cutaneous atrophy at the site of the injection. [10] This problem is chiefly of cosmetic concern and is recognized as a small depression in the skin frequently associated with depigmentation, transparency, and occasionally the formation of telangiectasia. These

changes in the skin occur when injections are made near the surface and some of the injected steroid leaks back along the needle track. The skin depression usually recedes and the skin returns to normal with time, when the crystals of the steroid have been completely absorbed. These changes are usually evident 6 weeks to 3 months after the initial injection, and usually resolve within 6 months, although these changes can be permanent. [8] [10] The *two-syringe technique*, in which the anesthetic is first injected, the needle advanced to the bursa/peritendon area, and the syringe then exchanged for another in order to inject steroid, helps prevents this complication by avoiding any leaking of the steroid suspension to the skin surface. [12] A small amount of lidocaine or normal saline can be used to flush the needle of the suspension before removing it. The *Z-tract technique* is a method of creating an indirect route from puncture of skin to the ultimate site of steroid injection. [12] Using this method, the needle is introduced at a site 0.5 to 1.0 cm from the actual target site and is redirected when halfway through the fat tissue to the target site of injection. This is followed by injection of both anesthetic and corticosteroid. Injection site atrophy is more likely in preparations that are less soluble and thus longer-acting. [8]

One of the most serious complications following local steroid injection is tendon rupture. In general, the risk is very low (<1%) and appears to be dose-related. [8] [10] It is believed by some that steroids injected directly into the tendon lead to a decrease in the tendon tensile strength. [10] [11] [56] [57] However, Gray and coworkers noted no cases of tendon rupture after more than 300 tendon sheath injections. [10] We still advise being diligent about injection into the surrounding area of the tendon sheath and not into the tendon substance. Also, by using one size needle and syringe, the operator is more likely to appreciate the increase in resistance when injecting the needle directly into the tendon. We also suggest limiting the number of injections to no more than once every 3 to 4 months. [7] [8] [9] [10] Tendon rupture is especially more likely in major stress-bearing tendons, such as the Achilles tendon and the patellar tendons, in athletes. The injection of corticosteroids in these areas should be avoided. [12]

There have been reports of accidental nerve injury following corticosteroid injection, particularly of the ulnar nerve (in treating medial epicondylitis) and median nerve (in treating carpal tunnel syndrome). [22] Also, up to 42% of patients undergoing local steroid therapy develop pericapsular calcifications, although these are generally asymptomatic. [10] [12] Finally, within minutes to hours of injection, approximately 1% of patients may experience facial and neck flushing. This reaction may last a few days, but it is usually a benign and self-limited reaction. Facial flushing seems to be more common with triamcinolone preparations. [8] [10]

Systemic absorption of local corticosteroid injections does occur, although at a slower rate than with oral steroids. [9] As a result, patients are at a low risk for systemic complications, but they do occur. Specifically, intrasynovial injections of steroids have been shown to suppress the hypopituitary-adrenal axis for 2 to 7 days. [8] Corticosteroids can also exacerbate hyperglycemia in diabetes. [9] [12] Abnormal uterine bleeding has also been reported. [8] [9] [12]

CONCLUSION

Local injection therapy for painful nonarticular rheumatic disorders is a relatively simple, safe, and effective form of treatment. In association with other treatment measures, the patient may experience rapid relief of pain and swelling and on occasion may return to work and normal activity after a single injection. Additionally, steroid injections may allow the patient to participate more fully in a physical rehabilitation program. [7] Injection therapy may also avoid surgical intervention in some injuries.

The local introduction of a corticosteroid suspension is associated with side effects that should be considered with each patient. It is recommended that an injection should not be repeated more than once every 3 to 4 months, and injection about the Achilles and patellar tendons should be avoided. [7] [8] [9] [10] [12] However, local undesirable reactions are usually minor and reversible. In general, the injection of corticosteroids is a safe procedure with few complications. [54]

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Chapter 54 - Arthrocentesis

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Jonathan Fisher

Arthrocentesis, the puncture and aspiration of a joint, is an acknowledged, useful procedure that is easily performed in the emergency department (ED). It has been established as both a diagnostic and a therapeutic tool for various clinical situations. Many clinicians are wary of joint fluid aspiration because of a lack of experience and because of the fear of introducing infection. When performed properly, however, the procedure offers a wealth of clinical information and is associated with few complications. In the ED it is difficult to make an accurate assessment of an acutely painful, hot, and swollen joint without performing arthrocentesis.

INDICATIONS AND CONTRAINDICATIONS

The indications for arthrocentesis include the following [1] :

1. Diagnosis of nontraumatic joint disease by synovial fluid analysis (septic joint or crystal-induced arthritis).
2. Diagnosis of ligamentous or bony injury by confirmation of the presence of blood in the joint. Arthrocentesis may be needed to differentiate a traumatic joint effusion from an inflammatory process.
3. Establishment of the existence of an intra-articular fracture by the presence of blood with fat globules in the joint.
4. Relief of the pain of an acute hemarthrosis or a tense effusion. Although a minor hemarthrosis need not be drained, arthrocentesis can reduce the pain associated with large effusions and examination of an injured joint.
5. Local instillation of medications in acute and chronic inflammatory arthritides. Instillation of lidocaine into an injured joint also makes the initial examination of a traumatic injury less painful.
6. Obtaining fluid for culture, Gram staining, immunologic studies, and cell count in cases of suspected joint infection.
7. Determining if a laceration communicates with the joint space.

Infection in the tissues overlying the site to be punctured (e.g., an abscess or a frank cellulitis) is generally considered an absolute contraindication to arthrocentesis. However, inflammation with warmth, swelling, and tenderness may overlie an acutely arthritic joint, and this condition may mimic a soft tissue infection. Once convinced that cellulitis does not exist, the clinician should not hesitate to obtain the necessary diagnostic joint fluid. Known bacteremia is a relative contraindication as a hematogenous spread of bacteria into the joint after arthrocentesis because, with or without hemorrhage, it may lead to infection.

Bleeding diatheses are rarely a relative contraindication, and arthrocentesis to relieve a tense hemarthrosis in bleeding disorders, such as hemophilia, is an accepted practice following infusion of the appropriate clotting factors. There is little data regarding the safety or dangers of arthrocentesis in a patient receiving anticoagulants or platelet inhibitors. One prospective trial of 32 patients on warfarin found no complications after arthrocentesis. [2] Hence, when necessary (e.g., to rule out infection), arthrocentesis should be performed in patients with a coagulopathy. The value of reversing a coagulopathy prior to the procedure with blood components is not proven, and clinical judgment should prevail. Prosthetic joints are at high risk for infection and arthrocentesis should be avoided whenever possible. However, if an infected prosthesis is suspected, arthrocentesis should be performed.

Articular versus Periarticular Disease

Periarticular diseases such as trauma, tendonitis, bursitis, contusion, cellulitis, or phlebitis may mimic articular disease and suggest the need for arthrocentesis. Therefore, administration of the correct therapy for acute joint disease requires that the clinician first determine whether the patient's constellation of signs and symptoms derives from the joint itself and not from some other musculoskeletal or periarticular structure. Such a distinction, however, may be difficult, if not impossible, to make. No specific test or physical finding has high specificity for solving this dilemma.

If swelling is secondary to joint effusion or inflammation, then the entire articular capsule will be inflamed and distended, and fluid can often be palpated within the joint. In the knee, this condition must be differentiated from effusion into the prepatellar bursa, where swelling distends the bursa that lies mainly over the lower portion of the patella, between it and the skin. Effusion into the joint occurs posterior to the patella, whereas bursal swelling occurs anterior to it (Fig. 54-1). When there is considerable articular effusion, the capsule of the joint is distended, and an inverted U-shaped swelling of the joint occurs. This characteristic shape occurs because the dense patellar ligament prevents distention of the capsule along its inferior border. Also, with the knee extended, a large effusion causes the patella to "float" or lift away from the femoral condyles. Complete extension and flexion are often impossible because of the joint tension produced by the effusion.

Joint effusion causes limited movement of the joint in all directions with active and passive motion producing pain. The pain arising from a pathologic condition involving a joint may be diffuse or clearly localized to the joint, or it may radiate. Hip pain, for example, frequently radiates into the groin or down the front of the thigh into the knee. Shoulder joint pain commonly radiates into the elbow or the neck. Therefore, complete examination of contiguous structures is essential for adequate diagnosis.

In contrast, pain from a periarticular process is often more localized, and tenderness can be elicited only with certain specific movements or at specific points around the joint. In periarticular inflammation, one can often passively lead a joint through a range of motion with minimal discomfort, yet pain is significant when the patient attempts active motion. Crepitus may be elicited in tendinitis, or the pain may be traced along the course of a specific tendon.

Septic Arthritis

Acute monoarticular arthritis is a common problem in emergency medicine. Although there are many causes of acute monoarticular arthritis, the one requiring most urgent diagnosis and treatment is septic arthritis. Infectious arthritis is still relatively frequent, and suspicion of a septic process in the

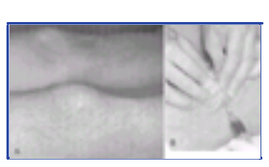

Figure 54-1 Periarticular problems may mimic an intra-articular process. *A*, This patient had trauma to the knee and presented with anterior soft tissue swelling and fluctuance, representing a hematoma of the prepatellar bursa. *B*, While an assistant (gloves should be worn) compresses the fluid, the bursa is aspirated of all blood.

joint is the first step in appropriate management; confirmation requires arthrocentesis and synovial fluid culture. Although repeated arthrocentesis may be needed when treating a septic joint, such therapy is usually performed on an inpatient basis.

Infection of a joint occurs by one of these mechanisms: 1) hematogenous spread, 2) spread from a contiguous source of infection, 3) direct implantation, or 4) postoperative contamination. The joint distribution of septic arthritis is typically monoarticular with a swollen, erythematous, and painful joint. The noninfectious differential diagnosis includes limited rheumatoid arthritis, gout, pseudogout, synovial osteochondromatosis, pigmented villonodular synovitis, and trauma. In many instances an acutely inflamed joint from gout or other arthritides simply cannot be clinically distinguished from infection. Early diagnosis is essential to prevent complications such as growth impairment, articular destruction with ankylosis, osteomyelitis, or soft tissue extension. [3]

Because an acutely swollen joint may be indicative of a number of disease entities, a thorough history and physical examination are the cornerstones of evaluation, followed by arthrocentesis (Fig. 54-2). Laboratory findings can be useful in diagnosis, as can response to therapy (e.g., response to penicillin in gonococcal arthritis is often the only criterion for diagnosis, as the organism is difficult to culture). Patients with malignancy (especially leukemia) or who are immunosuppressed or otherwise debilitated are at particular risk for a septic etiology. Infectious arthritis should be the first potential cause looked for in these patients, as well as in patients with such preexisting joint diseases as rheumatoid arthritis. In general, a swollen joint is usually not injected with corticosteroids until the possibility of infection has been eliminated. [4]

Neisseria gonorrhoeae, *Staphylococcus,* and *Streptococcus* are the most frequently identified etiologic agents. *N. gonorrhoeae* is the most common organism causing septic arthritis among adolescents and young adults. Patients older than 40 years and those with other medical illnesses are more likely to have *Staphylococcus* joint infections. In children, *Staphyloccus*, *Streptococcus, and Escherichia coli* predominate. *Haemophilus influenzae* was a common cause of septic arthritis in the past, but

widespread use of the conjugate vaccine has reduced *H. Influenzae* infection rates to near zero.[5] In neonates, *staphylococci, enterobacteriaceae*, *Group B Streptococcus*, and *N. gonorrhoeae* are the most likely organisms. Intravenous drug abusers commonly develop staphylococcal or *Pseudomonas* infections. *Salmonella* arthritis is seen in patients with sickle cell disease. Prosthetic joints or postoperative infections have high rates of *methicillin-resistant staph aureus*, *enterobacteriaceae*, and *Pseudomonas*.[6]

Although precise incidences for nongonococcal septic arthritis have not been established, predisposing factors have been described. These include chronic debilitating disease; prior antibiotic or immunosuppressive medications; a previous history of joint damage, such as from rheumatoid arthritis or degenerative joint disease; and prosthetic joints.[7] [8] [9] In a study by Sharp and coworkers, 22 of 113 patients (19%) with septic arthritis had other arthritides, the most common being rheumatoid arthritis.[3] Elderly patients with a debilitating arthritis and minor skin infections seem to be the most susceptible; the infection may be overlooked and the patient's findings thought to be an exacerbation of the rheumatoid arthritis. [10]

The simultaneous occurrence of gout and septic arthritis is possible, and one should not allow the establishment of a diagnosis of crystal-induced disease to stop a thorough search for infection.[11]

Because *N. gonorrhoeae* is the most common organism causing septic arthritis, gonococcal arthritis deserves special mention (Fig. 54-3). Disseminated gonococcal infection occurs in 0.5% to 3% of cases of mucosal infection. Gonococcal septic arthritis is more common in women, especially during pregnancy or after menstruation, because women are more likely to be asymptomatic. Time for local infection to disseminate can vary for several days to weeks. Patients will often experience systemic symptoms including fevers, chills, and malaise as well as migratory polyarthralgias. Tenosynovitis occurs in two thirds of patients. A dermatitis is also present in two thirds of patients (see Fig. 54-3). The most common rash is scattered painless, nonpruritic 0.5- to 0.75-cm macules or papules with necrotic or pustular centers distributed on the extremities and trunk. Eventually, the infection settles into one or two large joints, yielding a purulent arthritis. [12]

Whereas *N. gonorrhoeae*-infected joint fluid is usually "septic" in character, the yield of positive synovial fluid cultures has ranged from 25% to 50%. Blood cultures appear to be less helpful, being positive in only 20% to 30% of cases. However, cultures from the primary mucosal site are positive in up to 80% of infected patients.[12] A positive Gram stain is immediately diagnostic of septic arthritis. Gram staining is positive in only 65% of cases of septic arthritis, and therefore a negative Gram stain does not rule out an infectious process.

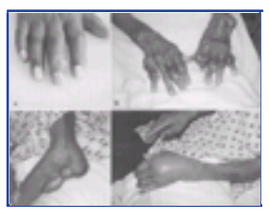

Figure 54-2 *A*, *B*, and *C*, Dramatic representation of tophaceous gout. These nodules are painless and full of uric acid crystals. An acutely swollen and painful joint in this patient is most likely acute gouty arthritis. *D*, This markedly swollen wrist is secondary to acute gouty arthritis. The patient had a fever and leukocytosis, and arthrocentesis with joint fluid analysis was the only way to differentiate gout from a septic joint.

A white blood cell (WBC) count and a synovial fluid glucose reduction may give confirmatory data. However, the synovial fluid WBC count in gonococcal arthritis is often <50,000 cells/mm^3 .[13] Although mild leukocytosis and elevated erythrocyte sedimentation rate (ESR) may occur, normal laboratory values do not exclude infection.[12] Counter-immunoelectrophoresis and latex agglutination have shown promise for early identification of infection. [14]

Hemarthrosis

Isolated nontraumatic hemarthrosis may occasionally be seen by the emergency clinician. An inflammatory reaction may follow an intracapsular bleed, and the proliferative reaction and the hyperplastic synovium formed may predispose the patient to recurrent hemorrhage in that joint, especially patients with bleeding diatheses. The knee is the most commonly affected joint (whether the cause is hemophilia or oral anticoagulants), followed by the foot and the ankle. [15]

The most common cause of intra-articular hemorrhage in the setting of no trauma or minor trauma is a hereditary clotting factor deficiency, as in hemophilia. Hemarthrosis is an infrequent complication of oral anticoagulant therapy but may occur even with prothrombin times within the normal range. [16] Chronic arthritis does not appear to be a long-term complication in patients with intra-articular bleeding from oral anticoagulant therapy. Hemarthrosis may also be a complication of sickle cell anemia, pseudogout, amyloidosis, pigmented villonodular synovitis, synovial hemangioma, rheumatoid arthritis, and infection. [15]

Management of acute hemarthrosis depends on the cause. Studies by Jaffer and Schmid and Wild and Zvaifler from the 1970s showed that the synovitis associated with oral anticoagulant therapy improves only after the oral anticoagulant is discontinued and the prothrombin time returns to normal. [16] [17] However, before stopping anticoagulation therapy, one must consider why the patient was anticoagulated, and weigh the risks and benefits of such a decision. Hemarthrosis following trauma is a frequent occurrence. It is most common in the knee and often denotes significant internal damage. A massively swollen knee after trauma is seen with knee dislocation (occasionally with spontaneous relocation) and a tear of the anterior cruciate ligament.

Distention of the joint by effusion or hemorrhage causes considerable pain and disability. If the exudate or the blood is not removed, it is partly absorbed, but part of it may undergo organization, resulting in formation of adhesions or bands in the joint. This is one argument for drainage of the joint. [18] Some believe that in an otherwise healthy joint that is subjected to a single traumatic event, even a relatively large hemarthrosis will be spontaneously reabsorbed without significant sequelae and therefore presents no pressing need to drain.[16] [17] Unfortunately, there is little literature to guide the best approach.

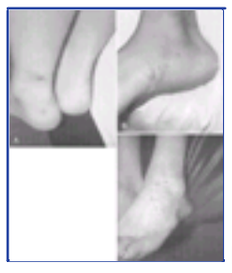

Figure 54-3 *A*, Skin lesions, ranging single or multiple petechiae to pustules may be seen in patients with acutely swollen joints infected with *Neisseria gonorrhoeae*. Skin lesions are usually found on the extremities, especially the feet and hands, and may be present before a large joint effusion accumulates, or with gonococcal tenosynovitis.

Nonetheless, a large, tense, traumatic effusion is quite painful, and its presence precludes proper evaluation of an injured joint. Therapeutic arthrocentesis to drain a symptomatic traumatic effusion is a well accepted practice.[19] The source of blood following trauma is frequently (1) a tear in a ligamentous structure, capsule, or synovium or (2) a fracture. Cruciate (especially anterior) ligament injury is the most common cause of significant hemarthrosis following trauma to the knee. [20] A joint effusion that develops 1 to 5 days following trauma may be secondary to a slow hemorrhage or a reinjury, but the swelling is often caused by a nonhemorrhagic irritative synovial effusion.

Occasionally one will diagnose an occult fracture by the presence of fat globules in the arthrocentesis blood. This may be appreciated when the bloody effusion is placed in a clear container (e.g., test tube or specimen jar) and held to the light. If the history of trauma is vague, arthrocentesis may be required to differentiate hemorrhage from other causes of joint effusion. Following therapeutic arthrocentesis for a hemarthrosis, it may be desirable to inject 2 to 15 mL of a local anesthetic, depending on joint size, into the joint to facilitate examination or provide temporary symptomatic relief. [19]

Intra-articular Corticosteroid Injections

In 1951 Hollander and coworkers first demonstrated that intra-articular corticosteroid injections were useful for symptomatic relief in patients with severe rheumatoid arthritis.[21] The use of steroids has proved to be a dependable method for providing rapid relief from pain and swelling of inflamed joints, although it is strictly local, usually temporary, and rarely curative.[22] [23] It is not commonly performed in the emergency setting.

Corticosteroid injections are most helpful when only a few of a patient's joints are actively inflamed. The most frequently used corticosteroids for intra-articular injection are shown in Table 54-1 .[24] Diminution of joint pain, swelling, effusion, and warmth is usually evident within 6 to 12 hours after injection.

Although very rare, the most serious complication of this practice is intra-articular infection. Therefore, steroids should not be injected into a joint if there is suspicion of a joint space infection. Repeated injections into one joint carry the risk of necrosis of juxta-articular bone with subsequent joint destruction and instability. Other complications include local soft tissue atrophy and calcification, tendon rupture, intra-articular bleeding, and transient nerve palsy. [23] Deposition of steroid crystals on the synovium may give rise to a transient, self-limited flare-up of a synovitis.[25]

It is always important to ascertain whether local corticosteroid therapy has been used previously, not only to consider the array of clinical conditions associated with steroid use, but also because crystalline corticosteroid material can hinder proper interpretation of crystals found in synovial fluid. [25]

TABLE 54-1 -- Intrasynovial Corticosteroid Preparations*

Preparation	Large-Joint Dose (mg)	Small-Joint Dose (mg)†
Triamcinolone hexacetonide	20	2–6
Triamcinolone acetonide	20	2–6
Prednisolone tebutate	25	2.5–7.5
Methylprednisolone acetate	40	3.5–10.5
Triamcinolone diacetate	20	2–6
Prednisolone acetate	30	3–9
Dexamethasone acetate	5	0.5–1.5

From Gray RG, Gottlieb NL: Corticosteroid injections in RA: Appraisal of a neglected therapy. J Musculoskeletal Med 7(10):53, 1990. Reproduced by permission.

*Listed in approximate descending order of duration of action.
†Dose will depend on joint size, capsular distensibility, and degree of inflammation.

EQUIPMENT

Necessary materials for arthrocentesis include skin preparation solutions (usually povidone-iodine followed by alcohol), sterile gloves and drapes, local or topical anesthetics, a syringe, and various-sized needles (Table 54-2). Fluid for cell count should be collected in a lavender-topped tube with ethylenediaminetetraacetic acid (EDTA) anticoagulant; however, glucose and viscosity determinations do not require anticoagulants. Immediately examine fresh synovial fluid in its unadulterated form for crystals. Calcium oxalate and lithium heparin anticoagulants have been reported to introduce artifactual crystals into the fluid. Joint fluid to be analyzed for crystals should be collected in a green-topped tube containing sodium heparin. If culturing for *N. gonorrhoeae*, the fluid should be immediately placed on proper medium and stored in a low-oxygen environment in the ED.

GENERAL ARTHROCENTESIS TECHNIQUE

Joint fluid may be obtained even when there is little clinical evidence of an effusion. Although you may successfully

TABLE 54-2 -- Emergency Department Arthrocentesis Tray

Gloves
Surgical scrub or plain green soap
Povidone-iodine solution
Alcohol sponges
Sterile towel (with center perforation)
Vapor coolant (e.g., fluoromethane solution)
1% or 2% lidocaine
Sterile saline for injection
Sterile gauze dressings (2 in. × 2 in.)
Sterile syringes (2 mL, 10 mL, 20 mL) Luer-Lok
Needles (18, 20, 22, and 25 ga) or over-the-needle intravenous catheters
Hemostat
Three-way stopcock (optional)
Plastic adhesive bandages
Sterile basin
Plain test tubes
Test tubes with liquid anticoagulant
Microscope slides and coverslips
Clear nail polish (to seal coverslip over material)

aspirate where the joint bulges maximally, certain landmarks are important. The most crucial part of arthrocentesis is spending adequate time in defining the joint anatomy by palpating the bony landmarks as a guide. These are described in detail later in this chapter. A puncture site and an approach to the joint should be selected; tendons, major vessels, and major nerve branches should be avoided. In most instances, the approach is via the extensor surfaces of joints, because most major vessels and nerves are found in flexor surfaces. Also, the synovial pouch is usually more superficial on the extensor side of a joint. If available, the use of ultrasound to help guide needle placement can be very helpful in difficult cases. Ultrasound can also help locate small effusions (see Chapter 69).

Aseptic technique is essential to avoid infection. Only sterile instruments should be used. Arthrocentesis should not be attempted if the skin or subcutaneous tissue overlying the joint is infected. Sterile gloves should be worn for the remainder of the preparation. The skin is thoroughly scrubbed with a surgical scrub, such as povidone-iodine scrub, and the skin is painted with an iodinated solution, such as povidone-iodine (Betadine). This antiseptic solution should be allowed to dry for several minutes, because the bactericidal effects of iodine are both concentration- and time-dependent. The iodine solution is then removed with an alcohol sponge to prevent transference of iodine into the joint space with a resultant inflammatory process. Although the utility of draping is unproven, and may obscure the site, a sterile perforated drape may be placed over the joint.

With appropriate local anesthesia, arthrocentesis should be a relatively painless procedure; without anesthesia, it may be quite painful and distressing to the patient. The synovial membrane itself has pain fibers associated with blood vessels, and the articular capsule and bone periosteum are richly supplied with nerve fibers and are very sensitive. The articular cartilage has no intrinsic pain fibers. It is important to have the patient relax during the procedure. Tense muscles narrow the joint space and make the procedure more difficult, requiring repeated attempts at aspiration, or result in inadequate drainage. Distraction of the joint may enhance the target area, especially in areas such as the wrist and the finger joints. Traction not only increases the chance of entering the joint, it also lessens the chance of scoring the articular cartilage with the needle.

Anesthesia is best accomplished by infiltrating the skin down to the area of the joint capsule using a local anesthetic agent such as 1% or 2% lidocaine (Xylocaine) with a 25- to

27-ga needle. For extremely painful joints, a regional nerve block before the procedure (see Chapter 32) is often greatly appreciated by the patient.

The landmarks described under Specific Arthrocentesis Techniques later in this chapter should be used, and care should be taken not to bounce the needle off bony structures as a means of finding the joint space, because this may damage the cartilage. [13] An 18- to 22-ga needle or intravenous (IV) catheter and needle set of appropriate length attached to a syringe is inserted at the desired anatomic point through the skin and subcutaneous tissue into the joint space. The largest needle that is practical is used to avoid obstructing the lumen with debris or clot. In large joints such as the knee, which can accommodate large effusions, it is suggested that one use a 30- to 60-mL syringe, because it may be difficult to change a syringe when the needle is within the joint cavity (Fig. 54-4). A three-way stopcock placed between the needle and syringe is an option for draining large effusions. If the syringe must be changed during the procedure, the hub of the needle should be grasped with a hemostat and held tightly while the syringe is removed. If an IV catheter and needle set is used, the needle is removed, leaving the outer atraumatic plastic catheter in the joint space. The syringe is then attached to the catheter for aspiration. Now manipulation of the joint or catheter can occur with little threat of tissue injury.

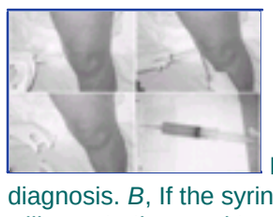

Figure 54-4 *A*, A chronic miniscus tear can produce a large effusion of clear yellow fluid. The miniscus has no blood supply, and a hemarthrosis suggests another or additional diagnosis. *B*, If the syringe is too small to accommodate all of the fluid, grasp the needle hub with a hemostat and remove the syringe without disturbing the correct position of the needle. A stopcock will negate the need to change the syringe. *C*, With the needle still held in place, use another syringe to instill a steroid when the aspirate is a clear yellow. *D*, Cloudy fluid such as this is from an inflammatory source, such as gout or a septic joint. Intra-articular steroids should not be administered in the setting of a cloudy aspirate.

Aspiration of synovial fluid and the easy injection and return of fluid indicate intra-articular placement of the needle tip. As a general rule, *one should try to remove as much fluid or blood as possible*. If the fluid stops flowing, this is a sign that the joint has been drained completely, the needle tip has become dislodged, or debris or clot is obstructing the needle. One should slightly advance or retract the tip of the needle, rotate the bevel, or ease up on the force of aspiration. Occasionally,

reinjecting a small amount of fluid back into the joint space confirms the needle placement and may clear the needle. If fluid flows freely back into the joint and is easily reaspirated, one has probably removed all the fluid. If resistance is met, the needle has probably been jarred from the joint space and is lodged in the soft tissue. In some instances, minor position changes produced by flexion or extension of the joint may allow the fluid to flow more freely. Scraping or shearing the articular cartilage with the needle should be avoided, since this may produce permanent cartilage damage. One should enter the joint in a straight line and avoid unnecessary side-to-side motion of the syringe. After aspiration is complete, the needle is removed, and a sterile dressing is applied over the puncture site.

Synovial fluid should be sent for studies as indicated by the clinical situation. Studies usually obtained include cell count with differential, crystal analysis, Gram staining,

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bacterial culture and sensitivity analysis, and synovial fluid glucose measurement. Less frequently obtained studies include synovial fluid protein measurement, rheumatoid factor analysis, lupus erythematosus (LE) cell preparation, viscosity analysis, mucin clot, fibrin clot, fungal and acid-fast stains, lyme titer, fungal and tuberculous culture, and synovial fluid complement analysis. If the arthrocentesis is performed for the relief of a hemarthrosis, the fluid need not be sent for analysis. One should be selective in ordering tests. There is no need to order a large battery of tests routinely on all fluids. If the volume of fluid collected is low, culture and examination of the "wet prep" under regular and polarizing microscopy have the highest priority. To avoid misdiagnosing borderline inflammatory fluids, missing crystals that dissolve with time, or overinterpreting the findings because of new artifactual crystals that appear over a prolonged time, prompt examination of synovial fluid specimens should be performed.[26]

COMPLICATIONS

Significant complications are rare with arthrocentesis and include the following:

1. *Infection.* Skin bacteria may be introduced into the joint space during needle puncture. This is a rare occurrence. One can, of course, limit this complication by maintaining rigorous sterile technique and avoiding inserting the needle through obviously infected skin or subcutaneous tissue. The chance of introducing infection with arthrocentesis through a noninfected area is very minimal if proper attention is paid to technique. Various studies report the incidence of infection following routine arthrocentesis to be in the range of 1 per 10,000 aspirations. [27] Joint aspiration in the presence of a bacteremia has been discussed previously.
2. *Bleeding.* Bleeding with subsequent hemarthrosis is rarely a complication, except in the patient with a bleeding diathesis. If a patient has a bleeding diathesis, such as hemophilia, arthrocentesis should be delayed until clotting competence has been enhanced by the infusion of specific clotting factors. Occasionally, a small quantity of blood may be aspirated along with the synovial fluid. This happens most often when the joint is nearly emptied. A small amount of blood-tinged fluid is generally the result of nicking a small synovial blood vessel; this is usually inconsequential. A grossly bloody effusion must be investigated. [4]
3. *Allergic reaction.* Hypersensitivity to the local anesthetic that is used can usually be prevented by thorough history taking. Fainting during the procedure is not uncommon and is most often the result of vasovagal influences.
4. *Corticosteroid-induced complications.* See Intra-articular Corticosteroid Injections earlier in this chapter.

SPECIFIC ARTHROCENTESIS TECHNIQUES

Arthrocentesis of the hip is generally performed by an orthopedic surgeon under fluoroscopic guidance and will not be discussed here. If available, fluoroscopy or ultrasound can also be used to guide aspiration of other joints, but these imaging adjuncts are generally not required.

First Carpometacarpal Joint (Fig. 54-5)

Landmarks.

The radial aspect of the proximal end of the first metacarpal is the arthrocentesis landmark for this joint. The abductor pollicis longus tendon is located by active extension of the tendon.

Position.

The thumb is opposed against the little finger so that the proximal end of the first metacarpal is palpable. Traction is applied to the thumb in order to widen the joint space between the first metacarpal and the greater multangular bone.

Needle insertion.

A 22- to 23-ga needle is inserted at a point proximal to the prominence at the base of the first metacarpal, on the palmar side of the abductor pollicis longus tendon.

Comments.

Degenerative arthritis commonly affects this joint. Arthrocentesis is of moderate difficulty. The anatomic "snuff box" (located more proximally and on the dorsal side of the abductor pollicis longus tendon) should be avoided, because it contains the radial artery and superficial radial nerve. A more dorsal approach may also be used.

Interphalangeal and Metacarpophalangeal Joints (Fig. 54-6)

Landmarks.

The landmarks are on the dorsal surface—the prominence at the proximal end of the proximal phalanx for metacarpophalangeal joints and the prominence at the proximal end of the middle or distal phalanx for interphalangeal joints. The extensor tendon runs down the midline.

Position.

The fingers are flexed to approximately 15°–20° and traction is applied.

Needle insertion.

A 22- to 25-ga needle is inserted into the joint space dorsally, just medial or lateral to the central slip of the extensor tendon.

Comments.

Synovitis causes these joints to bulge dorsally. Normally, it is unusual to obtain fluid in the absence of a significant pathologic condition.

Radiocarpal Joint (Wrist) (Fig. 54-7)

Landmarks.

The dorsal radial tubercle (Lister tubercle) is an elevation found in the center of the dorsal aspect of the distal end of the radius. The extensor pollicis longus tendon runs in a groove on the radial side of the tubercle. The tendon can be palpated by active extension of the wrist and thumb.

Figure 54-5 Landmarks for arthrocentesis of first carpometacarpal joint (see text). *(From Akins CM: Aspiration and injection of joints, bursae, and tendons. In Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

Figure 54-6 Landmarks for arthrocentesis of interphalangeal and metacarpophalangeal joints (see text). *(From Akins CM: Aspiration and injection of joints, bursae, and tendons. In Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

Position.

The wrist should be positioned in approximately 20°–30° of flexion and accompanying ulnar deviation. Traction is applied to the hand.

Needle insertion.

A 22-ga needle is inserted dorsally, just distal to the dorsal tubercle and on the ulnar side of the extensor pollicis longus tendon. The anatomic snuff box, located more radially, should be avoided.

Radiohumeral Joint (Elbow) (Fig. 54-8)

Landmarks.

The lateral epicondyle of the humerus and the head of the radius are the arthrocentesis landmarks for the radiohumeral joint. With the elbow extended, the depression between the radial head and the lateral epicondyle of the humerus is palpated.

Position.

With the palpating finger still touching the radial head, the elbow is flexed to 90°. The forearm is pronated, and the palm is placed down flat on a table.

Needle insertion.

A 22-ga needle is inserted from the lateral aspect just distal to the lateral epicondyle and is directed medially.

Comments.

Elevation of the anterior fat pad, or the presence of a posterior fat pad, on a lateral soft tissue elbow

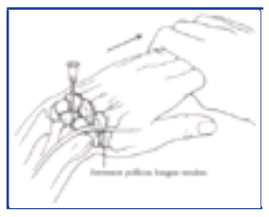

Figure 54-7 Landmarks for arthrocentesis of the radiocarpal joint (see text). Traction is applied with slight flexion. Contrary to the figure, *no* radial deviation is applied. *(From Akins CM: Aspiration and injection of joints, bursae, and tendons. In Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

radiograph signifies blood, pus, or fluid in the elbow joint (see Fig. 54-8*A*). Effusions in the elbow joint may bulge and be readily palpated (see Fig. 54-8*C*). Often the effusion appears inferior to the lateral epicondyle. The bulge can then be aspirated from a posterior approach on the lateral side (see Fig. 54-8*D*). A medial approach is not recommended, because the ulnar nerve and the superior ulnar collateral artery may be damaged. This is a common joint for gout or septic arthritis. The most common cause of an elbow hemarthrosis following trauma, with no obvious fracture, is a nondisplaced radial head fracture. A small hemarthrosis need not be aspirated, but removal of blood from a tense elbow joint will significantly hasten recovery and facilitate range of motion in patients with a radial head fracture.

Glenohumeral Joint (Shoulder), Anterior Approach (Fig. 54-9)

Landmarks.

The coracoid process medially and the proximal humerus laterally are palpated anteriorly.

Position.

The patient should sit upright with the arm at the side and his or her hand in the lap.

Needle insertion.

A 20-ga needle is inserted at a point inferior and lateral to the coracoid process and is directed posteriorly toward the glenoid rim.

Comments.

Arthrocentesis of this joint is of moderate difficulty. Other approaches have been suggested but are less well accepted. Ultrasound guidance may be helpful.

Knee Joint, Anteromedial Approach (Fig. 54-10*A*)

Landmarks.

The medial surface of the patella at the middle or superior portion of the patella is the landmark for the knee joint.

Position.

It is usually recommended that the knee be fully extended as far as possible. Alternatively, some practitioners prefer to flex the knee 15°–20° by placing a towel under the popliteal region in order to open up the joint space. Relaxation of the quadriceps muscle greatly facilitates needle placement. The foot is kept perpendicular to the floor.

Needle insertion.

An 18-ga needle or catheter and needle set is inserted at the midpoint or superior portion of the patella approximately 1 cm medial to the anteromedial patellar edge. The needle is directed between the posterior surface of the patella and the intercondylar femoral notch. The patella may be grasped with the hand and elevated to aid needle entry into the joint.

Comments.

If the patient is tense, contraction of the quadriceps will greatly hinder entering the joint. However, the knee is probably the easiest joint to enter and removal of a tense hemarthrosis will relieve pain and facilitate examination for ligamentous injury. If fluid stops flowing, the operator or assistant should squeeze the soft tissue area of the suprapatellar region to "milk" the suprapatellar pouch of fluid (see Fig. 54-10*B*). Alternatively, the patient's thigh can be wrapped with a 6-in elastic bandage from the groin to the suprapatellar area prior to beginning the procedure. The knee can easily accommodate 50 to 70 mL of fluid, and the clinician should therefore use a large syringe or be prepared to change syringes during the procedure, or both. Holding/securing the hub of the needle with a hemostat allows the clinician to remove the syringe without changing the position of the

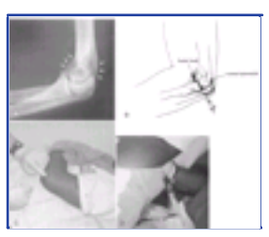

Figure 54-8 *A*, On a lateral elbow radiograph, displacement of the anterior fat pad (arrows) or the presence of a posterior fat pad (arrows) indicate blood, pus, or fluid in the joint. *B*, Landmarks for arthrocentesis of the radiohumeral joint (see text). *(From Akins CM: Aspiration and injection of joints, bursae, and tendons. In Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.) C*, An effusion in the elbow joint can usually be readily palpated. The index finger is placed over the lateral epicondyle

and slid posteriorly toward the olecranon. Usually a depression is felt as the finger leaves the epicondyle, but a bulge is appreciated if there is a joint effusion. *D*, Removal of only a few milliliters of blood will reduce pain and hasten recovery of the range of motion. The most common pathology following trauma with an x-ray negative for fracture, but positive for a hemarthosis, is a nondisplaced radial head fracture.

intra-articular needle. Alternatively, a stopcock on the needle will allow for complete removal of fluid without changing the position of the needle. The knee is a common site for septic arthritis (especially gonococcal) and various inflammatory or degenerative diseases. An anterolateral approach can be accomplished in a similar manner if the patella is approached laterally.

Tibiotalar Joint (Ankle) (Fig. 54-11)

Landmarks.

The medial malleolar sulcus is bordered medially by the medial malleolus and laterally by the anterior tibial tendon. The tendon can be easily identified by active dorsiflexion of the foot.

Position.

With the patient lying supine on the table, the foot is plantar flexed.

Needle insertion.

A 20- to 22-ga needle is inserted at a point just medial to the anterior tibial tendon and directed into the hollow at the anterior edge of the medial malleolus. The needle must be inserted 2 to 3 cm to penetrate the joint space.

Comments.

If the joint bulges medially, one may use an approach that is more medial than anterior, entering at a point just anterior to the medial malleolus. The needle may have to be advanced 2 to 4 cm with this approach.

Metatarsophalangeal and Interphalangeal Joints (Fig. 54-12)

Landmarks.

For the first digit, landmarks are the distal metatarsal head and the proximal base of the first phalanx. For the other toes, the landmarks are the prominences at the proximal interphalangeal and distal interphalangeal joints. The extensor tendon of the great toe can be located by active extension of the toe.

Position.

With the patient supine on the table, the toes should be flexed 15°–20°. *Traction is then applied.*

Needle insertion.

A 22-ga needle is inserted on the dorsal surface at a point just medial or lateral to the central slip of the extensor tendon.

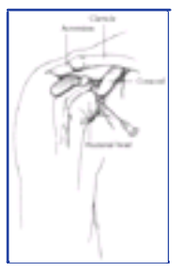

Figure 54-9 Landmarks for arthrocentesis of glenohumeral joint (see text). *(From Akins CM: Aspiration and injection of joints, bursae, and tendons. In Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

SYNOVIAL FLUID INTERPRETATION

Synovial fluid examination is essential for the diagnosis of septic arthritis, gout, and pseudogout. Inflammatory joint disease of previously unknown etiology can often be diagnosed

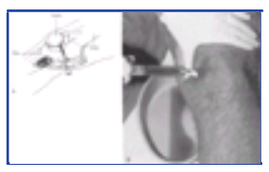
Figure 54-10 *A*, Landmarks for arthrocentesis of the knee joint, medial approach (see text). *B*, Note the use of a stopcock on the syringe to allow complete drainage without repositioning the needle. Compression of the suprapatellar region by the operator or an assistant will facilitate complete aspiration. For the knee, a 60 mL syringe should be used to drain large effusions. *(From Akins CM: Aspiration and injection of joints, bursae, and tendons. In Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

precisely by synovial fluid studies. Joint fluid is a dialysate of plasma that contains protein and hyaluronic acid. Normal fluid is clear enough to allow newsprint to be read through it, and it will not clot. Normal fluid is straw-colored and flows freely, with the consistency of machine oil. Normal fluid produces a good mucin clot and gives a positive "string sign" (see next section). The uric acid level of joint fluid approaches that of serum, and the glucose concentration is normally at least 80% that of serum. A normal joint contains only a few milliliters of fluid. Clarity of fluid reflects the leukocyte count. High leukocyte counts result in opacity, the degree of which generally correlates with the degree of elevated synovial fluid leukocytes.

String Sign

Viscosity correlates with the concentration of hyaluronate in the synovial fluid. Any inflammation degrades hyaluronate, characteristically resulting in low-viscosity synovial fluids. The string sign is a simple test for assessing viscosity. The practitioner measures the length of the "string" formed by a falling drop extruded from a syringe of synovial fluid. Normal joint fluid produces a string of 5 to 10 cm (Fig. 54-13). If viscosity is reduced, as in inflammatory conditions, the synovial fluid forms a shorter string or falls in drops.

Mucin Clot Test

The mucin clot test also corresponds to viscosity and inflammation. The greater the inflammatory response, the poorer the mucin clot and the lower the viscosity. The mucin clot test is rarely performed in emergency medical practice, and it is


Figure 54-11 Landmarks for arthrocentesis of the tibiotalar joint (see text). *(From Akins CM: Aspiration and injection of joints, bursae, and tendons. In Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

mentioned for completeness. This test may be useful to define the degree of polymerization of hyaluronate. Mucin clots are produced by mixing 1 part joint fluid with 4 parts 2% acetic acid. A good clot indicates a high degree of polymerization and correlates with normal high viscosity. In inflammatory synovial fluid, such as that seen in osteoarthritis and rheumatoid arthritis-related effusions, the mucin clot is poor. Many do not consider this test diagnostically useful. [28]

Cell Count and Glucose

A leukocytosis consisting predominantly of neutrophils is usually seen with inflammatory arthritides; a WBC count >50,000/mm ^{3} (i.e., >50,000/muL) is highly suggestive of a

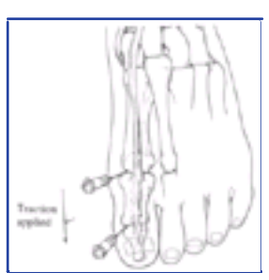
Figure 54-12 Landmarks for metatarsophalangeal and interphalangeal joints (see text). *(From Akins CM: Aspiration and injection of joints, bursae, and tendons. In Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*


Figure 54-13 *A*, Ability of normal synovial fluid to form a long tenacious string. (Note: Gloves should be worn during procedure.) *B*, Bloody joint fluid from recent trauma forms a normal string sign.

septic joint. Joint fluid glucose usually decreases as inflammation increases, but a proper interpretation requires a simultaneous blood glucose evaluation. A joint fluid-to-serum glucose ratio of less than 50% suggests a septic joint. Shmerling and coworkers have found a WBC count of >2000/mm ^{3} to be 84% sensitive and 84% specific for *all* inflammatory arthritides. Of their septic arthritis patients, 37% had a total WBC count <50,000/mm ^{3} . However, 89% of their patients with a total WBC count >50,000/mm^3 had a septic joint.[29]

Serology

Although available, most of the serological tests are not likely to be useful in the emergency setting. The detection of

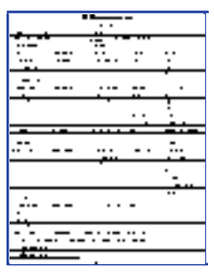

Figure 54-14 Data form and summary of specimens needed for joint fluid examination. *(Courtesy of Alexander Trott, MD.)*

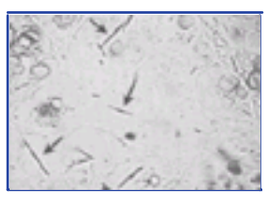

Figure 54-15 Synovial fluid with needle-shaped uric acid crystals *(arrows)*. Many crystals are characteristically engulfed by leukocytes. *(From Schumacher HR, Finkinson CA, Weiss JJ: Guidelines for obtaining and analyzing synovial fluid. ER Reports 4:40, 1983. Reproduced by permission.)*

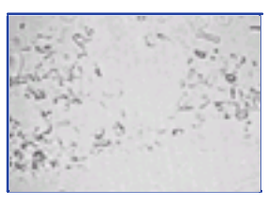

Figure 54-16 Synovial fluid with calcium pyrophosphate crystals. *(From Schumacher HR, Finkinson CA, Weiss JJ: Guidelines for obtaining and analyzing synovial fluid. ER Reports 4:40, 1983. Reproduced by permission.)*

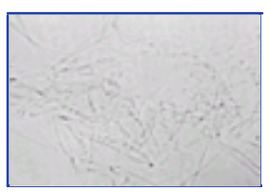

Figure 54-17 Appearance of cartilage fragments. *(From Schumacher HR, Finkinson CA, Weiss JJ: Guidelines for obtaining and analyzing synovial fluid. ER Reports 4:40, 1983. Reproduced by permission.)*

Figure 54-18 Appearance of cholesterol crystals. *(From Schumacher HR, Finkinson CA, Weiss JJ: Guidelines for obtaining and analyzing synovial fluid. ER Reports 4:40, 1983. Reproduced by permission.)*

Figure 54-19 Microscopic and gross appearance of fat globules in synovial fluid. This finding suggests a fracture extending into the joint. *(From Schumacher HR, Finkinson CA, Weiss JJ: Guidelines for obtaining and analyzing synovial fluid. ER Reports 4:40, 1983. Reproduced by permission.)*

TABLE 54-3 -- Synovial Fluid Interpretation

Diagnosis	Appearance	WBCs/mm^3	Polymorphonuclear Leukocytes	Glucose: % Blood Level	Crystals Under Polarized Light	Culture
Normal	Clear	<200	<25	95–100	None	Negative
Degenerative joint disease	Clear	<4000	<25	95–100	None	Negative
Traumatic arthritis	Straw-colored, bloody, xanthochromic, occasionally with fat droplets	<4000	<25	95–100	None	Negative
Acute gout	Turbid	2000–50,000	>75	80–100	Negative birefringence;* needle-like crystals	Negative†
Pseudogout	Turbid	2000–50,000	>75	80–100	Positive birefringence;* rhomboid crystals	Negative
Septic arthritis	Purulent/turbid	5000–>50,000	>75	<50	None	Positive (usually)
Rheumatoid arthritis/seronegative arthritis (Reiter disease, psoriatic arthritis, ankylosing spondylitis, inflammatory bowel disease)	Turbid	2000–50,000	50–75	~75	None	Negative†

WBC, White blood cell.

*Negative birefringence means that crystals appear yellow when lying parallel to the axis of the slow vibration of light of the first-order red compensator. With the same orientation to the compensator, positive birefringence crystals appear blue. When the crystals lie perpendicular to the axis, the opposite is true—that is, negative birefringence crystals are blue, and positive ones are yellow. A polarizing microscope is necessary for this distinction to be made.
†May be coexisting infection.

succinic acid is helpful in identifying patients with septic arthritis who have received antibiotic treatment before arthrocentesis. [30] Gas-liquid chromatography, a rapid and sensitive method for the detection of short-chain fatty acids, may complement the currently available methods used to diagnose septic arthritis of synovial fluid. [31]

Counterimmunoelectrophoresis and latex agglutination also are useful and are available in some centers on an emergency basis. Other immunologic markers such as complement, rheumatoid factor, and antinuclear antibodies have little diagnostic value in the acute setting but may be useful to the clinician providing follow-up care when compared to serum levels.

Fluid Processing

Proper collection of the joint fluid is essential for examination and testing. Tests for viscosity, serology, and chemistries are done on fluid collected in a red-topped (clot) tube, whereas cytology samples are collected in tubes with an anticoagulant (purple top). One should always transfer the fluid for crystal examination into a tube with liquid heparin (green top), because undissolved heparin crystals from powdered anticoagulant tubes can be seen on microscopy. Early transfer of synovial fluid to this green-topped tube is essential to prevent clotting. Culture requirements for transport and processing should be accessed before the procedure to ensure appropriate processing or plating of specimens. A useful data form and summary of specimens developed by Alexander Trott, MD, of the University of Cincinnati is shown in Figure 54-14 .

Polarizing Microscope

No synovial fluid analysis is complete until the fluid has been examined under a polarizing light microscope for crystals. The polarizing microscope used for crystal identification differs from the ordinary light microscope because it contains two identical polarizing prisms or filters. One filter, called the *polarizer,* is positioned below the condenser. The other filter is called the *analyzer* and is inserted at some point above the objective. A detailed discussion of the technique is beyond the scope of this textbook and is described elsewhere. [32] [33] The procedure is generally performed by trained laboratory personnel.

Polarization Physics

The polarizer allows passage of light in only one specific orientation. The analyzer acts as a crossed filter, removing all light in the light path unless the material being examined rotates the beam from the polarizer into the plane of the analyzer. The compensator functions by imparting color of a certain wavelength (red at about 550 nm). Birefringent materials change the wavelength to blue or yellow, depending on the direction (negative or positive) of refringence.

Microscopic Analysis

When examining crystals under polarized microscopy, the technician orients crystals on a stage according to 2 axes referred to as X and Z. If the long axis of the crystals is blue when parallel to the Z axis and yellow when perpendicular to it, it is calcium pyrophosphate and is termed *positively birefringent*. If the long axis of the crystal is yellow when parallel to the Z axis and blue when perpendicular to it, it is monosodium urate and is termed *negatively birefringent*. Urate crystals are 2 to 10 µm and are usually needle-shaped. Calcium pyrophosphate crystals range from 10 µm down to tiny crystals that have to be examined with the oil objective; they appear as rods, rhomboids, plates, or needle-like forms and are weakly birefringent. Cholesterol crystals are sometimes seen and are large, very bright, square, or rectangular plates with broken corners. [32]

Items found in synovial fluid that can be confused with sodium urate (Fig. 54-15) or calcium pyrophosphate crystals (Fig. 54-16) include collagen fibrils, cartilage fragments (Fig. 54-17), cholesterol crystals (Fig. 54-18), metallic fragments from prosthetic arthroplasty, and corticosteroid esters. [31] One may also identify fat globules (Fig. 54-19 *A* and *B*). Note that rare cases of uric acid spherulites in gouty synovia have been reported. [34] The spherulites are birefringent and do not take up fat stains.

Table 54-3 summarizes synovial fluid features for the joint diseases commonly encountered and studies commonly performed in the ED.

CONCLUSION

When performed correctly, arthrocentesis is a relatively safe procedure that is used to obtain valuable diagnostic information as well as to provide therapy for acute joint disease. The key to success is strict adherence to sterile technique, observance of anatomic landmarks, and proper preparation of the synovial fluid for examination.

Acknowledgment

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Chapter 55 - Compartment Syndrome Evaluation

Neal R. Frankel
L. Albert Villarin Jr.

Compartment syndrome, a condition of increased pressure within a limited space, results in compromised tissue perfusion and ultimate dysfunction of neural and muscular structures contained within that space.[1] Clinical hallmarks include burning, deep-seated pain, worsening of the pain with passive stretch of the involved muscles, paresthesias in the distribution of the involved nerves, paresis or paralysis of the involved limb, and palpable tenseness of the involved compartment. It should be stressed that severe pain, *out of proportion to the injury, may be the only early symptom of acute compartment syndrome* .[2] When pain is the only symptom, however, this diagnosis may be impossible to make on clinical grounds. Several theories have been proposed to account for the tissue ischemia associated with compartment syndrome. These include the "arteriovenous gradient,"[3] "critical closure,"[4] and "venous occlusion"[5] theories. The central theme in each of these is a significant rise in intracompartmental pressure, which results in reduced blood flow and muscle perfusion to a level below the metabolic demands of the tissue. The relationship between intracompartmental pressure and general circulatory status is one of the most important factors in the development of a compartment syndrome. Knowledge of this relationship is very important in determining the appropriate time to perform decompressive fasciotomy.[2] A high index of suspicion should be maintained, especially in the multi-trauma patient or obtunded patient, since prompt recognition and treatment are essential for optimal outcome.

Although compartment syndrome is relatively uncommon, recognition of this process has increased with greater clinician awareness of its clinical features. The lower leg and forearm are the most frequent locations of compartment syndrome; however, gluteal, hand, foot, upper arm, thigh, and back compartment syndromes are not uncommon.[1] Causes of compartment syndrome have been categorized into those that decrease compartment volume, increase compartment content, or create externally applied pressure.[1] If left untreated, compartment syndrome may result in permanent neurologic-muscular dysfunction and extremity deformity or lead to shock or renal failure if myonecrosis is extensive.[6]

The differential diagnosis of compartment syndrome includes neuropraxia, reflex sympathetic dystrophy (RSD), arterial injury, cellulitis, osteomyelitis, tenosynovitis, synovitis, and thrombophlebitis. Nerve and arterial injury may coexist with compartment syndrome. Differentiating compartment syndrome from these disease processes may be difficult even for the experienced clinician. Although compartment syndrome is a clinical diagnosis, objective measurement of compartment tissue pressure may help confirm the diagnosis and determine if operative treatment is required. This chapter discusses the indications, complications, and interpretation of compartment pressure monitoring. The equipment and techniques required to perform compartment pressure monitoring are described.

BACKGROUND

Postischemic myoneural dysfunction and contractures were first described in the 1870s by Von Volkmann. [7] In 1935, Henderson and associates developed an open-needle technique for measuring muscle "tonus." [8] Their method consisted of the three-way connection of a syringe, a manometer, and a needle placed into the muscle itself. In the 1960s, the technique was applied to muscle compartment pressure measurement. In 1975, Whitesides and colleagues refined the technique and described its ability to accurately reflect muscle compartment pressures. [9] They also related elevated pressures to a need for fasciotomy to relieve a compartment syndrome.

In an effort to improve the accuracy and reproducibility of intermittent pressure measurements, Matsen and coworkers modified the "simple needle" technique to include a constant infusion pump that allowed for prolonged, continuous monitoring. [10] While this proved more accurate than intermittent measurements, concerns were raised over injecting more fluid into an already compromised compartment. As a result, new methods were developed to continuously measure intracompartmental pressure without the risk of further pressures increases. One method used a biodegradable "wick" catheter attached to an extracorporeal transducer. [11] [12] Despite demonstrated accuracy, [12] [13] fears of catheter breakdown leading to measurement errors and retained foreign bodies resulted in development of the "slit" catheter. [14] Accuracy and reproducibility of the wick catheter and slit catheter have been similar. [13] [15] In the mid-1980s, a solid-state transducer intracompartmental (STIC) catheter was developed. This device, produced by Millar Instruments (Houston, TX), does not require saline-filled tubing to transmit the compartment pressure to an external transducer. [16] The Stryker intracompartmental pressure monitoring system (Kalamazoo, MI) is currently the most widely recognized solid-state handheld device used for temporary or continuous compartment pressure monitoring (Fig. 55-1). Arterial line transducer monitoring systems have also been used for acute monitoring of compartment pressure, although no study has been conducted to support the accuracy of this technique.

Noninfusion systems like the transducer-tipped fiberoptic system offer a distinct advantage over the conventional fluid-filled systems since they do not produce hydrostatic pressure

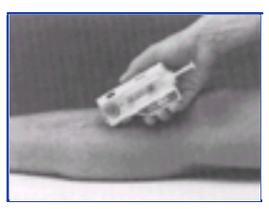

Figure 55-1 The Stryker 295 intracompartmental pressure monitor system. *(Courtesy of Stryker Instruments, Kalamazoo, MI.)*

Figure 55-2 The HANDY electronic transducer-tipped catheter system *(Courtesy of Mammendorfer Institute for Physics and Medicine LTD. Munich, Germany.)*

artifacts or require injections of fluid for long-term measurement. However, the fiberoptic transducer is relatively large and must be attached to an intracath sheath (2.1 mm outer diameter), which may cause patient discomfort during compartment pressure measurement. [17] In the late 1990s the HANDY (Mammendorfer Institute for Physics and Medicine LTD. Munich, Germany), a noninfusion electronic transducer-tipped catheter system, was introduced. This system consists of a reusable probe attached to a separate portable, battery-powered handheld device (Fig. 55-2). The electronic transducer-tipped catheter system is self-calibrating, and except for sterilization of the reusable probe, requires minimal setup and maintenance. One study has shown that this system accurately measures intramuscular pressure and can be used for both acute and long-term monitoring. [17]

Development of miniature transducer-tipped catheters and noninvasive techniques are ongoing. Until recently, noninvasive technologies like near-infrared spectroscopy and magnetic resonance imaging have mainly been used for the diagnosis of chronic compartment syndrome. [18] [19] [20] Further investigation will determine the utility of these non-invasive technologies as diagnostic tools for acute compartment syndrome.

PATHOPHYSIOLOGY OF COMPARTMENT SYNDROME

Whitesides and Heckman report that the most common cause of compartment syndrome is muscle injury edema, which is usually proportional to tissue damage. [2] Because the extremities are composed of relatively nonyielding fascial compartments, circulatory impedance ultimately occurs as tissue pressure rises. [2] A "vicious cycle" involving hydrostatic and osmotic forces ensues. When vascular perfusion falls below the metabolic demands of the tissue, cellular disruption creates more interstitial edema, increasing compartment pressure and leading to progressive ischemia. [21] Other factors, such as associated hematoma formation (with or without fracture), which can occur at the time of injury, may further exacerbate an early or existing compartment syndrome. Coagulopathies may also aggravate an existing compartment syndrome and may require different approaches to treatment.[22]

Pressure studies clearly show a linear relationship between increased tissue pressure and decreased blood flow [1] ; however, the complex relationship between systemic and venous pressures is not completely understood and how capillary blood flow ultimately becomes compromised depends on a number of clinical variables.[23]

Heppenstall and colleagues used MRI analysis of muscle cell phosphorus metabolism to confirm the relationship between perfusion pressure and cellular ischemia. [24] That investigation was followed by another study comparing muscle tolerance to ischemia induced by tourniquet application to ischemia caused by acute high-pressure compartment syndrome.[25] This comparative study was initiated because previous discussions of ischemic injury in compartment syndrome frequently used the tourniquet technique as a pathophysiologic reference. However, there was no evidence that ischemia was the *only* cause of muscle injury in the acute compartment syndrome.[26] It was noted that during ischemia, cellular levels of phosphocreatine decreased equally between both groups. In contrast, the levels of adenosine triphosphate (ATP) diminished rapidly in the compartment syndrome group alone. Moreover, phosphocreatine, ATP, and pH normalized within 15 minutes of releasing the tourniquet, whereas these values remained depressed for a significant period of time, even after fasciotomy, in the compartment syndrome group. These results indicate that elevated tissue pressure acts synergistically with ischemia to produce more severe cellular deterioration than ischemia alone. [26]

Early in compartment syndrome, the dysfunction may be reversible if conditions are improved. Some evidence shows that skeletal muscle at body temperature can survive for as long as 4 hours without irreversible damage, whereas complete irreversible changes occur with total ischemia of 8 hours. [27] [28] Other investigators, however, have noted that muscle necrosis may occur as early as 2 hours post trauma.[29] This observation is important since *even the most expeditious diagnosis may not always result in a positive outcome.* Variable results occurred after 6 hours. Peripheral nerves experience similar susceptibility to ischemia but, while dose-responsive relationship effects have been observed in some studies, the critical values for pressure and duration with respect to nerve injury are unknown. [30] Interestingly, a study done on healthy volunteers that measured nerve function with carpal tunnel compression found the critical extraneural value above which nerve function was blocked was pressure 30 mm Hg below diastolic pressure. [31]

Although lowering or raising the systemic blood pressure may affect the level of direct compartment pressure that results in ischemic compromise, two points are stressed: (1) tissue that has been damaged by injury is more sensitive to ischemia and, therefore, the threshold for definitive intervention is lessened and (2) in severely traumatized limbs or clinical instances where the diagnosis is clear-cut, fasciotomy may be justified regardless of the tissue pressures documented. [2] [26]

CLINICAL PRESENTATION

Even experienced clinicians find it difficult to evaluate a potential compartment syndrome, and no specific standard of

care exists with regard to a time interval from injury to definitive treatment. In the unconscious patient, or for those with other life-threatening conditions that mandate other priorities, the clinical scenario simply does not allow for a diagnosis to be made in a timely fashion. In the nontrauma case, or for the patient unable to voice pain or cooperate with an examination, this diagnosis is often not considered, or the diagnosis is delayed. Delays in diagnosis occur because the time of onset is extremely variable and because of other clinical variables (especially altered mental status and the absence of known trauma). Matsen and Clawson have found the onset of symptoms to range from 2 hours to as long as 6 days after the insult.[32] The peak time seems to be 15 to 30 hours (Fig. 55-3). The difficulty in diagnosing an acute compartment syndrome is further emphasized in a report by Vaillancourt et al.[29] In a retrospective review of 76 patients who underwent fasciotomy at major university trauma centers/teaching hospitals, the interval from initial patient assessment to diagnosis of a compartment syndrome was up to 8 hours, being most prolonged in nontraumatic cases. The interval from the precipitating event to definitive surgery was up to 35 hours, reflecting the difficulty in suspecting this diagnosis and instituting definitive therapy.

The limiting envelope required to produce a compartment syndrome may include fascia, skin, casts, external dressings, or even epimysium alone.[1] General categories for the many sources of increased pressure within these envelopes include decreased compartment volume, increased compartment contents, and externally applied pressure. Table 55-1 lists the reported causes in each category. Because of the nature of the limiting envelopes and the acknowledged causes, compartment syndromes are most commonly seen in the extremities. The lower leg is at high risk because of its propensity for injury and the existence of several low-volume compartments. It is interesting to note that lumbar paraspinal

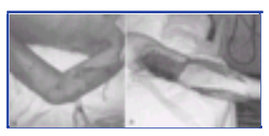

Figure 55-3 The diagnosis of a compartment syndrome is not always straightforward. This young man presented in coma from a drug overdose and had been lying on his arm for a number of hours. The entire arm was swollen and rhabdomyolysis was correctly suspected and treated *(A)*. Because of the coma he was unable to voice any complaint of pain. When he awakened 20 hours later the pain was severe, and compartment pressures demonstrated the need for fasciotomy *(B)*.

compartment syndrome may represent an unusual presentation of low back pain.[33]

Clinical history is the first step in the evaluation of an injury or condition that may produce a compartment syndrome, and it may reveal the etiology (see Table 55-1). Clinical examination confirms the diagnosis when there is clear evidence of increased tissue pressure, inadequate tissue perfusion, and loss of tissue function. The diagnosis is less certain when one or more of these factors are absent.[1]

Often the first symptom described by the patient is pain greater than expected for a given clinical situation. Pain often increases with the passive stretching of muscles in the involved compartment. The muscles also may be weak in comparison to normal. Hypesthesia may be present in the distribution of nerves and tenseness may be palpable in the involved compartment. Decreased vibratory sensation may be one of the earliest sensory findings.[34] [35] These findings usually progress during a period of observation. As a rule, *the presence or absence of arterial pulsation is not an accurate indicator of increased tissue pressure*; pulses may be present in a severely compromised extremity compartment.[2] When pulses are obliterated distally, irreversible damage has often occurred. Table 55-2 lists symptoms and signs of the respective compartments when compartment syndrome is suspected.

Patients who have an altered mental status and younger, uncooperative patients may make the interpretation of neuromuscular signs difficult. In addition, casts or bulky dressings may make careful examination impossible. Attributing the signs and symptoms to other pathologic entities is also a problem (Table 55-3). Primary nerve and muscle injuries can produce similar findings, but the deficit should be maximal initially and should not progress. Arterial injuries and subsequent ischemia may produce pain and dysfunction, although neurologic changes may be less pronounced unless secondary edema produces a compartment syndrome. The acute phase of

TABLE 55-1 -- Etiologies of Compartment Syndrome

Decreased compartmental volume
Closure of fascial defects
Application of excessive traction to fractured limbs
Increased compartmental content
Bleeding
Major vascular injury
Coagulation defect
Bleeding disorder
Anticoagulation therapy
Thrombolytic therapy
Postarterial line placement
Increased capillary filtration
Reperfusion after ischemia
Arterial bypass grafting
Embolectomy
Ergotamine ingestion
Cardiac catheterization
Lying on limb
Trauma
Fracture

Contusion
Intensive use of muscles
Exercise
Seizures
Eclampsia
Tetany
Burns
Thermal
Electric
Intra-arterial drug injection
Cold
Orthopedic surgery
Tibial osteotomy
Hauser procedure
Reduction and internal fixation of fractures
Snakebite
Increased capillary pressure
Intensive use of muscles
Venous obstruction
Phlegmasia cerullea dolens
Ill-fitting leg brace
Venous ligation
Diminished serum osmolarity, nephrotic syndrome
Other causes of increased compartmental contents
Infiltrated infusion
Pressure transfusion
Leaky dialysis cannula
Muscle hypertrophy
Popliteal cyst
Carbon monoxide poisoning
Externally applied pressure
Tight casts, dressings, or air splints
Lying on limb
Pneumatic anti-shock garment
Congenital bands

Modified from Matsen FA: Compartmental Syndromes. New York, Grune & Stratton, 1980.

RSD may present similarly; however, a history of accidental trauma is typically more remote (days to months).[23] Thrombophlebitis[36] and cellulitis[32] must also be considered in the differential diagnosis. Pain out of proportion to obvious injury may raise the issue of drug-seeking behavior, but a focused evaluation for potential compartment syndrome should precede such a diagnosis.

INDICATIONS FOR INVASIVE (COMPARTMENT PRESSURE) MONITORING

The earliest manifestation of acute compartment syndrome is an elevated tissue pressure. Signs and symptoms occur after tissue pressure has been elevated beyond a critical time period. Measurement of this pressure allows earlier diagnosis and treatment. In some patients, the diagnosis of compartment syndrome is clinically obvious, and one can proceed directly to fasciotomy. However, when clinical findings are equivocal or difficult to interpret, tissue pressure measurement may help guide treatment (Fig. 55-4). Tissue pressure measurements may suggest a compartment syndrome, but equivocal measurements will still require clinical judgment. Three groups of patients for whom clinical findings are difficult to interpret, and who may benefit from compartment pressure measurement, are unresponsive patients, uncooperative patients (such as children or intoxicated patients), and patients with peripheral nerve deficits attributable to other causes (e.g., tibial fracture with peroneal nerve injury).

PATIENT PREPARATION

Patient preparation includes positioning the patient and extremity for optimal results, performing skin preparation at the site of needle introduction, local anesthesia, and procedural sedation when necessary. Because compartment pressure monitoring is an invasive procedure with potential complications and may require procedural sedation and possibly an operative procedure, informed consent should be obtained from the patient or guardian. Details of the consent should be subsequently documented by a procedural note and where appropriate using standard hospital forms. The patient's clinical status and lack of availability of family will often prohibit formal written consent. Such circumstances should be clearly documented in the emergency department (ED) record.

Patient and extremity position for compartment pressure measurement depends on the extremity being studied, the compartment being studied, and presence of coexisting injury. Patients should be comfortable, usually in the supine or prone position, and the compartment to be measured should be at the same level as the heart. The patient and extremity should be positioned to allow the needle to be introduced perpendicular to the muscular compartment being measured. Any obstructions to the needle entry point into the skin and all structures that may put pressure on the compartment and falsely elevate the pressure should be removed. That is, the extremity of the compartment being measured should be positioned so that no external pressure is applied to that compartment. This may require that an assistant hold the extremity slightly above the stretcher.

Once the location for needle placement has been identified, preparation of the skin should be conducted as for any sterile procedure. Care should be taken to avoid needle placement in areas in which the overlying skin is possibly infected, to avoid introduction of bacteria into deep tissues. If an overlying cast is present, it should be bivalved, and if necessary, a window overlying the desired area of needle penetration should be cut from the cast (see Chapter 51). The skin should be anesthetized with a small amount of local anesthetic, with care taken to avoid injection into muscle or fascia. Excessive,

TABLE 55-2 -- Compartment Syndromes and Associated Physical Signs

Compartment	Sensory Loss	Muscles Weakened	Painful Passive Motion	Tenseness Location
Forearm				
Dorsal	—	Digital extensors	Digital flexion	Dorsal forearm
Volar	Ulnar/median nerves	Digital flexors	Digital extension	Volar forearm
Hand				
Interosseus		Interosseus	Abduct/adduct (metacarpophalangeal joints)	Dorsum hand between metacarpals
Leg				
Anterior	Deep peroneal nerve	Toe extensors	Toe flexion	Anterior aspect leg
		Tibialis anterior		
Superficial posterior	—	Soleus and gastrocnemius	Foot dorsiflexion	Calf
Deep posterior	Posterior tibial nerve	Toe flexors	Toe extension	Distal medial leg, between Achilles tendon and tibia
		Tibialis posterior		
Gluteal	(Rarely sciatic)	Gluteals, piriformis, or tensor fascia lata	Hip flexion	Buttock
Upper arm				
Flexor	Ulnar/median nerves	Biceps and distal flexors	Elbow extension	Anterior upper arm
Extensor	Radial nerves	Triceps and forearm extensors	Elbow flexion	Posterior upper arm
Foot	Digital nerves	Foot intrinsics	Toe flexion/extension	Dorsal/plantar foot
Lumbar	—	Erector spinae	Lumbar flexion	Paraspinous

deep infiltration may falsely elevate the compartment pressure reading.

Procedural sedation and analgesia should be considered in any patient who may be uncooperative and unable to hold the extremity still during the procedure (see Chapter 34). Any struggling or movement that requires restraint of the extremity may falsely elevate the compartment pressure. Caution should be taken as parenteral sedation and analgesia may blunt the pain and paresthesias that are paramount in the diagnosis of compartment syndrome. The benefits of accurate pressure measurement must be weighed against obliterating these clinical findings for the duration of sedation.

TISSUE PRESSURE MONITORING

The methods described in this chapter focus on the acute compartment pressure measurement with equipment commonly found in the ED or intensive care setting. These include the mercury manometer system,[37] arterial line system, and the Stryker 295 intracompartmental pressure monitor system (see Fig. 55-1). The described methods provide rapid

TABLE 55-3 -- Clinical Findings of Compartment Syndrome, Arterial Occlusion, and Neuropraxia

	Compartmental Syndrome	Arterial Occlusion	Neuropraxia
Pressure increased in the compartment	+	-	-
Pain with stretch	+	+	-
Paresthesia or anesthesia	+	+	+
Paresis or paralysis	+	+	+
Pulses intact	+	-	+

From Mubarak S, Carroll N: Volkman's contracture in children: Etiology and prevention. J Bone Joint Surg Br 61:290, 1979.

compartment pressure measurement with reasonable accuracy. Although the mercury manometer system is the least expensive and most readily available, it is also the least accurate and its results are the least reproducible. [1] Continuous monitoring of compartment pressures has been used to map out the changes in pressure over time, but this technique has not been widely accepted. [38] Note that *isolated, elevated tissue pressures do not mandate surgical intervention*. The entire clinical scenario must be cnsidered.

Needles commonly available for placement into the compartment for pressure measurement include a simple 18-ga needle, an 18-ga spinal needle (for deep compartments), and the side-port needle (Stryker Instruments, Kalamazoo, MI). The side-port needle and slit catheter have comparable efficacy when used for compartment pressure measurement, whereas a simple 18-ga needle is less precise.[39] The HANDY electronic transducer-tipped catheter system (Mammendorfer Institute for Physics and Medicine LTD) is a newer device, which may not be widely available (see Fig. 55-2). The wick catheter, slit catheter, and the STIC (described previously in the Background section) generally require specialized, cumbersome equipment that is not available in most EDs and are therefore not described.

Mercury Manometer System

Equipment

2 18-ga needles (or spinal needles)

2 plastic extension tubes

1 20-mL syringe

1 3-way stopcock

1 vial of sterile normal saline

1 mercury manometer

Setup and Procedure

1. The patient and extremity are prepared according to the guidelines described earlier.

2. The syringe, tubing, extension tubing, and needle are assembled as shown in Figure 55-5*A* .
3. The needle is inserted into a vented vial of sterile saline. A column of saline is aspirated into the tubing halfway to the stopcock; care should be taken to avoid bubble formation. The 3-way stopcock is turned to close off this tube to avoid losing saline during movement of the needle into the patient.
4. The needle is inserted into the muscle of the desired compartment (see Needle Placement Techniques later in this chapter for details).
5. The second extension tubing is attached to the monitor and to the third port of the 3-way stopcock. The stopcock is turned so that the syringe is open to both extension tubes (Fig. 55-5*B*). This closed system has equal pressure in both extension tubes.
6. The pressure in the system is increased gradually by *slowly* depressing the syringe plunger while simultaneously watching the column of saline. The mercury manometer will rise as the pressure in the system rises. When the pressure in the system exceeds the tissue pressure, saline is injected into the compartment, causing the saline column to move. The manometer is read at the time at which the saline moves. This reading corresponds to the tissue pressure in millimeters of mercury.
7. Completely remove the needle and repeat steps 4 to 6 to get a second measurement. A third measurement may be necessary to get two readings in agreement. The needle should be checked between readings for tissue plugs and blood clots.

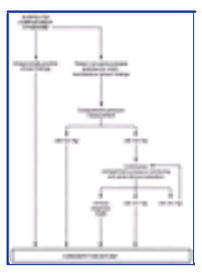

Figure 55-4 Algorithm for management of a patient with suspected compartment syndrome. Pressure thresholds (*) are based on published case series. *Clinical correlation is paramount to proper interpretation of compartment pressure measurements. (From Rorabeck CH: Compartment syndromes. In Browner BD, Jupiter JB, Levine AM, Trafton PG (eds): Skeletal Trauma: Fractures, Dislocations, Ligamentous Injuries, vol 1, 2nd ed. Philadelphia, WB Saunders, 1992, p 290.)*

Procedural Pearls

The most common error with this system is depressing the syringe plunger too quickly. Only when the saline is slowly injected into the compartment will the mercury column (which has greater inertia) accurately reflect the compartment pressure. Another source of error with this system is obstruction of the needle with a plug of tissue if the syringe plunger is pulled back. Finally, aneroid manometers are not calibrated at lower pressure ranges and should not be substituted for the more accurate mercury manometers.

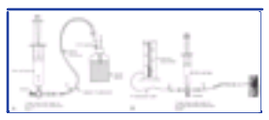
Figure 55-5 *A* and *B*, Mercury monitor technique for compartmental pressure monitoring. *(From Whitesides TE, Haney TC, Morimoto K, et al: Tissue pressure measurements as a determinant for the need of fasciotomy. Clin Orthop 113:43, 1975.)*

Arterial Line System

Equipment

Intracompartment needle

High-pressure tubing

Pressure transducer with cable

Pressure monitor/module

Sterile saline

Transducer stand that allows variable height

2 3-way stopcocks

1 20-mL syringe

Setup and Procedure

1. The patient and extremity are prepared according to the guidelines described earlier.
2. The transducer cable is connected to the pressure monitor/module.
3. The stopcocks, transducer, transducer cable, syringe, high-pressure tubing, and needle are assembled as in Figure 55-6 .
4. The syringe is filled with 15 mL of sterile saline and placed on 1 stopcock. The stopcocks are turned to allow filling of the transducer, high-pressure tubing, and needle. The stopcock to the high-pressure tubing is then closed.
5. The top stopcock is opened to air and the transducer placed at the height of the compartment to be measured. The transducer is calibrated to zero and the top stopcock closed.
6. The lower stopcock to the high-pressure tubing is opened and the needle is inserted into the muscle of the desired compartment (see Needle Placement Techniques later in this chapter for details). Digitally compressing the compartment or passively moving the muscles within the compartment should result in a pressure spike suggesting appropriate placement. The compartment is allowed to equilibrate for several seconds after this maneuver, and the *mean* pressure is measured.
7. Completely remove the needle and repeat steps 4 to 6 to get a second measurement. A third measurement may be necessary to get two readings in agreement. The needle should be checked between readings for tissue plugs and blood clots.

Stryker 295 Intracompartmental Pressure Monitor System

Equipment

Handheld pressure monitor

Stryker 295 quick-pressure monitor set (disposable pouch)

1 prefilled syringe with saline

1 side-port needle

1 diaphragm chamber

Setup and Procedure

1. The 295 quick-pressure monitor set (disposable) is opened, and contents are removed, maintaining appropriate system sterility.

2. The needle is placed firmly on the tapered chamber stem (Fig. 55-7*A*).
3. The blue cap on the prefilled syringe is removed and screwed onto the remaining chamber stem (see Fig. 55-7*A*). Care must be maintained to not contaminate the fluid pathway.
4. The cover of the monitor is opened. The chamber is placed in the device well (black surface down) and pushed gently until it seats (Fig. 55-7*B*).
5. The cover is snapped closed—*NOT FORCED*. The latch must have "snapped" in place.
6. The clear end cap is pulled off the syringe and the plunger rod is attached to the syringe.
7. The needle is held at approximately 45° up from horizontal while fluid is slowly forced through the disposable system to purge it of air. *Caution:* Saline *MUST NOT* roll down the needle into the transducer well.
8. The unit is turned on and should read between 0 and 9 mm Hg.
9. The intended angle of insertion of the needle into the skin is approximated while the "zero" button is pressed. The display should read "00" (Fig. 55-7*C*). *Note:* The display must read "00" before continuing. If it does not, troubleshooting using that section of the maintenance manual should be undertaken.
10. The needle is inserted into the compartment (see Needle Placement Techniques later in this chapter for details). <0.3 mL of saline should be slowly injected into the compartment for equilibration with interstitial fluids.
11. The pressure is read after the display reaches equilibrium.
12. *For additional measurements:* The unit is turned off and steps 8 to 11 are repeated. The unit must be recalibrated to zero before each measurement.

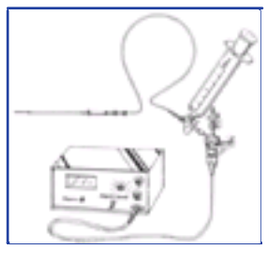
Figure 55-6 The arterial line system for compartmental pressure measurement. *(From Rorabeck CH: Compartment syndromes. In Browner BD, Jupiter JB, Levine AM, Trafton*

PG (eds): Skeletal Trauma: Fractures, Dislocations, Ligamentous Injuries, vol 1, 2nd ed. Philadelphia, WB Saunders, 1992, p 290.)

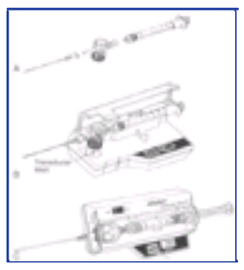

Figure 55-7 *A–C*, The Stryker 295 intracompartmental pressure monitor system assembly. *(Courtesy of Stryker Instruments, Kalamazoo, MI.)*

NEEDLE PLACEMENT TECHNIQUES

General Principles

Once the decision to perform compartment pressure measurement has been made, the approach for needle placement and confirmation of correct placement must be considered. In a prospective study of tibial fractures, Heckman and Whitesides found the highest tissue pressure was at the level, or within 5 centimeters, of the fracture.[40] The requirements for a useful approach include: (1) reliable placement in the compartment to be measured; (2) avoidance of important neurovascular structures; (3) simplicity and reproducibility; and (4) minimal discomfort to the patient. [41] Most compartments are superficial and are easily accessible. Only the deep posterior compartment of the lower leg and the gluteal compartment may require a spinal needle for deeper needle placement. Most approaches allow the needle to enter perpendicular to the skin. Figure 55-8 Figure 55-9 Figure 55-10 Figure 55-11 Figure 55-12 Figure 55-13 Figure 55-14 Figure 55-15 Figure 55-16 Figure 55-17 Figure 55-18 Figure 55-19 in conjunction with the text, provide landmarks for proper needle placement and passage to ensure proper compartment placement and avoidance of neurovascular structures.

Lower Leg

The lower leg traditionally has four compartments [1] : anterior, lateral, deep posterior, and superficial posterior (Fig. 55-8). Occasionally, the tibialis posterior muscle will reside in a separate fascial compartment. [26] The lower leg, especially the anterior compartment, is predisposed to compartment syndrome because of its high vulnerability to injury and the relatively limited compartment compliance. The anterior compartment is often mentioned as the most frequent site of compartment syndrome. [23]

The easiest cross-sectional level for needle placement for all four compartments is approximately 3 cm on either side of a transverse line drawn at the junction of the proximal and middle thirds of the lower leg (Fig. 55-9 Fig. 55-10 Fig. 55-11 Fig. 55-12). When measuring the leg compartment pressures, the patient should be placed in the supine position with the leg at heart level (however, when measuring the superficial posterior compartment, the patient should be prone). Patient preparation should be performed as described previously.

Anterior Compartment

With the patient supine, the anterior border of the tibia is palpated at the level of the junction of the proximal and middle thirds of the lower leg (see Fig. 55-9*A*). The needle entry point is 1 cm *lateral* to the anterior border of the tibia. The needle should be directed perpendicular to the skin to a depth of 1 to 3 cm (see Fig. 55-9*B*). Proper needle placement can be confirmed by seeing a rise in pressure during (1) digital compression of the anterior compartment just proximal or distal to the needle insertion site, (2) plantar flexion of the foot, and (3) dorsiflexion of the foot. These maneuvers should produce a several-fold rise in pressure on the monitor.

Deep Posterior Compartment

With the patient supine, the leg should be slightly elevated off the stretcher if the clinical situation permits. The medial border of the tibia is palpated at the level of the junction of the proximal and middle thirds of the lower leg. The needle entry point is just posterior to the medial border of the tibia (see Fig. 55-10*A*). The posterior border of the fibula should be

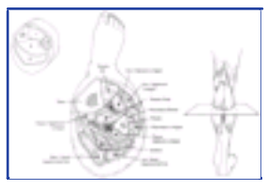

Figure 55-8 Fascial compartments of the lower leg with enclosed muscle groups (insert upper left): (1) anterior; (2) lateral; (3) superficial posterior; and (4) deep posterior compartments.

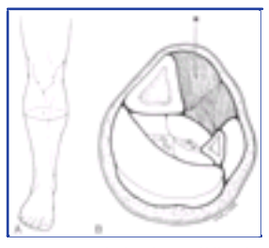

Figure 55-9 Anterior compartment syndrome of the lower leg. Suggested needle entry point is indicated by the small circle *(A)*. The needle should be inserted (*) to a depth of 1 to 3 cm *(B). (Modified with permission from Matsen FA (ed): Compartmental Syndromes. New York, Grune & Stratton, 1980, p 91.)*

palpated on the lateral aspect of the leg at the same level. The needle path should be perpendicular to the skin and directed toward the palpated posterior border of the fibula to a depth of 2 to 4 cm, depending on the amount of subcutaneous fat (see Fig. 55-10*B*). Proper needle placement can be confirmed by seeing a rise in pressure during (1) toe extension and (2) ankle eversion.

Lateral Compartment

With the patient supine, the leg should be slightly elevated off the stretcher if the clinical situation permits. The posterior border of the fibula is palpated at the level of the junction of the proximal and middle thirds of the lower leg. The needle entry point is just anterior to the posterior border of the fibula (see Fig. 55-11*A*). The needle path should be perpendicular to the skin and directed toward the fibula to a depth of 1 to 1.5 cm (see Fig. 55-11*B*). If the needle contacts bone, the needle should be retracted 0.5 cm. Proper needle placement can be confirmed by seeing a rise in pressure during (1) digital compression of the lateral compartment just inferior or superior to the needle entrance site and (2) inversion of the foot and ankle.

Superficial Posterior Compartment

With the patient prone and the leg at heart level, a transverse line at the level of the junction of the proximal and middle

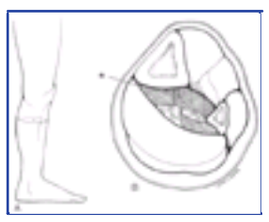

Figure 55-10 Deep posterior compartment syndrome of the lower leg. Suggested needle entry point indicated by the small circle *(A)*. The needle should be inserted (*) to a depth of 2 to 4 cm *(B)*. *(Modified with permission from Matsen FA (ed): Compartmental Syndromes. New York, Grune & Stratton, 1980, p 92.)*

thirds of the lower leg is identified. The needle entry point is at this level and 3 to 5 cm on either side of a vertical line drawn down the middle of the calf (see Fig. 55-12*A*). The needle path should be perpendicular to the skin and directed toward the center of the lower leg to a depth of 2 to 4 cm (see Fig. 55-12*B*). Proper needle placement can be confirmed by seeing a rise in pressure during (1) digital compression of the superficial posterior compartment just inferior or superior to the needle entrance site and (2) foot dorsiflexion.

Forearm

The forearm traditionally has two compartments,[1] volar and dorsal, which are divided by the interosseus membrane. However,

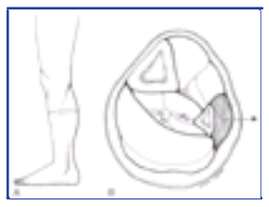

Figure 55-11 Lateral compartment syndrome of the lower leg. Suggested needle entry point indicated by the small circle *(A)*. The needle should be inserted (*) to a depth of 1 to 1.5 cm *(B)*. *(Modified with permission from Matsen FA (ed): Compartmental Syndromes. New York, Grune & Stratton, 1980, p 92.)*

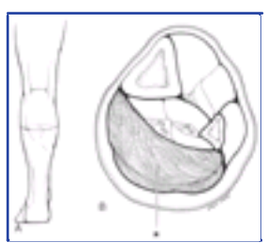

Figure 55-12 Superficial posterior compartment syndrome of the lower leg. Suggested needle entry point indicated by the small circle *(A)*. The needle should be inserted (*) to a depth of 2 to 4 cm *(B)*. *(Modified with permission from Matsen FA (ed): Compartmental Syndromes. New York, Grune & Stratton, 1980, p 92.)*

some authors place the extensor *carpi radialis longus*, extensor *carpi radialis brevis*, and *brachioradialis* muscles into their own compartment, called the "mobile wad."[42] The latter group of muscles is considered a separate compartment in this chapter. The forearm compartments are predisposed to compartment syndrome, especially the volar compartment, because of their use during vigorous exercise, accessibility for drug injection, and vulnerability to burns. [1]

The junction of the proximal and middle thirds of the forearm is the cross-sectional level for needle insertion (Fig. 55-13).[43] When measuring forearm compartment pressures, the patient should be placed in the supine position with the arm at heart level. Patient preparation should be performed as described previously.

Volar Compartment

The forearm should be held in supination. The *palmaris longus* tendon is identified and its course traced proximal to the level of the junction of the proximal and middle thirds of the forearm. The posterior border of the ulna is palpated. The needle entry point is just medial to the *palmaris longus* (Fig. 55-14*A*). The needle path should be perpendicular to the skin and directed toward the palpated posterior border of the ulna to a depth of 1 to 2 cm (Fig. 55-14*B*). Proper needle placement can be confirmed by seeing a rise in pressure during (1) digital compression of the volar compartment just proximal or distal to the needle entry point and (2) extension of the fingers or wrist.

Dorsal Compartment

The forearm should be held in supination with the elbow flexed, allowing the dorsum of the forearm to face downward. The posterior aspect of the ulna is palpated at the level of the junction of the proximal and middle thirds of the forearm. The needle entry point is 1 to 2 cm lateral to the posterior aspect of the ulna (Fig. 55-15*A*). The needle should be directed perpendicular to the skin to a depth of 1 to 2 cm (Fig. 55-15*B*). Proper needle placement can be confirmed by seeing a rise in

Figure 55-13 Level of needle insertion of the forearm *(A)* with cross section through the upper third of the forearm *(B)* demonstrating the three forearm compartments (volar, dorsal, mobile wad). *(With permission from Green DP (ed): Operative Hand Surgery. New York, Churchill Livingstone, 1982.)*

pressure during (1) digital compression of the dorsal compartment just proximal or distal to the needle entry point and (2) flexion of the fingers or wrist.

Mobile Wad

The forearm should be held in supination. The most lateral (radial) portion of the forearm is identified at the level of the junction of its proximal and middle thirds. The needle entry point is the muscle tissue lateral to the radius. The needle should be directed perpendicular to the skin (Fig. 55-16*A*) and inserted to a depth of 1 to 1.5 cm (Fig. 55-16*B*). Proper needle placement can be confirmed by seeing a rise in pressure during (1) digital compression of the mobile wad just proximal and distal to the needle entry point and (2) ulnar deviation of the wrist.

Gluteal Musculature

The gluteal musculature is enclosed by an enveloping fascia that splits into two layers and encases the muscle bellies of the *tensor fascia lata* anteriorly and gluteus maximus posteriorly. This

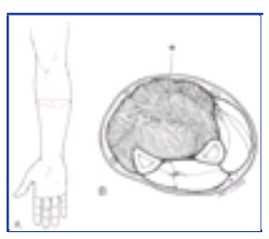

Figure 55-14 Volar compartment syndrome of the forearm. Suggested needle entry point indicated by the small circle on the volar forearm *(A)*. The needle should be inserted (*) to a depth of 1 to 2 cm *(B)*. *(Modified with permission from Matsen FA (ed): Compartmental Syndromes. New York, Grune & Stratton, 1980, p 93.)*

fascia divides the musculature into three distinct compartments: maximus, tensor, and medius/minimus (Fig. 55-17). The sciatic nerve is deep to the fascia but lies between the pelvis-external rotator complex and the gluteus maximus, making it susceptible to injury in compartment syndrome of the gluteus maximus. Most reported cases of gluteal compartment syndrome result from prolonged immobilization and local compression in association with drug or alcohol intoxication. [43] Gluteal

compartment syndrome is rare, making the diagnosis difficult, as local tenderness is often attributed to contusion or hematoma. Rhabdomyolysis should be considered in patients with gluteal compartment syndrome because of the large muscle mass that may be injured.

Gluteal Compartments

The patient should be placed in the prone position with the buttocks at heart level. Patient preparation should be performed as described previously. Cutaneous landmarks for the three compartments are not consistent from patient to patient. In all cases of suspected gluteal compartment syndrome, the needle insertion point can be at the point of maximal

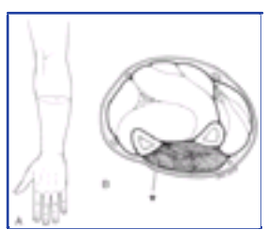

Figure 55-15 Dorsal compartment syndrome of the forearm. Suggested needle entry point indicated by the small circle *(A)*. The needle should be inserted (*) to a depth of 1 to 2 cm *(B)*. *(Modified with permission from Matsen FA (ed): Compartmental Syndromes. New York, Grune & Stratton, 1980, p 93.)*

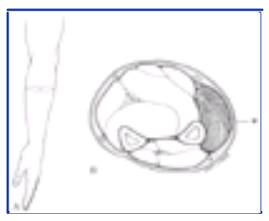

Figure 55-16 Mobile wad compartment syndrome of the forearm. Suggested needle entry point indicated by the small circle on lateral (radial) aspect of proximal fore arm *(A)*. The needle should be inserted (*) to a depth of 1 to 1.5 cm *(B)*. *(Modified with permission from Matsen FA (ed): Compartmental Syndromes. New York, Grune & Stratton, 1980, p 93.)*

tenderness.[43] An 18-ga spinal needle should be used. The needle should be directed perpendicularly to the skin toward the point of maximum tenderness to a depth of 4 to 8 cm. Proper needle placement is confirmed by seeing a rise in pressure during digital compression of the gluteal musculature.

Foot

Compartment syndrome of the foot is rare, but it is being reported with increasing frequency as clinicians become more aware of the disease process. The four compartments of the foot as seen from a coronal section at the base of the first metatarsal are the central, interosseous, medial, and lateral compartments (Fig. 55-18). The foot is vulnerable to isolated

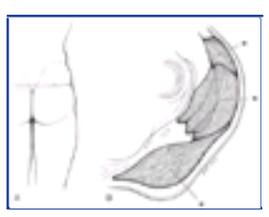

Figure 55-17 Gluteal compartment syndrome. Suggested entry points are indicated by the small circles *(A)*. The needle should be inserted to a depth of 4 to 8 cm depending on which compartment is being measured. Needle tips (*) shown entering muscle compartments *(B)*. *(Modified with permission from Owen CA, Moody PR, Mubarak SJ, et al: Gluteal compartment syndromes. Clin Orthop 132:57, 1978.)*

extremity injury by virtue of its location. Most compartment syndromes in the foot are the result of crushing forces.

When measuring compartment pressures in the foot, the patient should be placed in a supine position with the foot at the level of the heart. [44] Patient preparation should be performed as described previously.

Medial Compartment

The medial compartment contains the *abductor hallucis* and *flexor hallucis brevis* muscles and is bounded medially and inferiorly by the extension of the plantar aponeurosis, laterally by an intermuscular septum, and dorsally by the first metatarsal (Fig. 55-19*A*). The medial aspect of the base of the first metatarsal is palpated. The needle entry point is the medial aspect of the foot just inferior to the base of the first metatarsal and into the *abductor hallucis* muscle.[45] The needle is advanced to a depth of 1 to 1.5 cm. Proper needle placement is confirmed by seeing a rise in pressure during digital compression of the medial compartment of the foot.

Central Compartment

The central compartment contains the *flexor digitorum brevis*, the *quadratus plantae*, the *lumbricals*, and the *abductor hallucis* muscles. Its boundaries are the plantar aponeurosis inferiorly, the osseofascial tarsometatarsal structures dorsally, and the intermuscular septa medially and laterally. The medial aspect of the base of the first metatarsal is palpated. The needle entry point is the medial aspect of the foot just inferior to the base of the first metatarsal and through the *abductor hallucis* muscle. The needle is advanced to a depth of 3 cm.[45] Proper needle placement is confirmed by seeing a rise in pressure during digital compression of the central compartment of the foot.

Lateral Compartment

The lateral compartment contains the abductor, flexor, and opponens muscles of the fifth toe. The boundaries are the fifth metatarsal dorsally, the plantar aponeurosis inferiorly and laterally, and an intermuscular septum medially. [46] The base of the fifth metatarsal is palpated. The needle entry point is just inferior to the base of the fifth metatarsal (Fig. 55-19*B*). The needle should be directed parallel to the plantar aspect of the

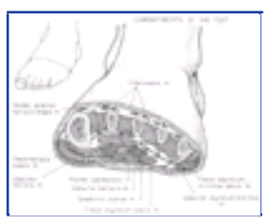

Figure 55-18 The compartments of the foot. *(From Mubarak SJ, Hargens AR: Compartment Syndromes and Volkmann's Contracture. Philadelphia, WB Saunders, 1981.)*

foot to a depth of 1 to 1.5 cm. Proper needle placement is confirmed by seeing a rise in pressure during digital compression of the lateral compartment of the foot.

Interosseous Compartment

The interosseous compartment contains the seven interossei muscles and is bounded by the metatarsals and the interosseous fascia. The dorsums of the bases of the metatarsals are palpated and the second and fourth web spaces are identified. The first web space is avoided to prevent inadvertent puncture of the *dorsalis pedis* or deep peroneal nerve.[45] The needle entry point is the dorsum of the second

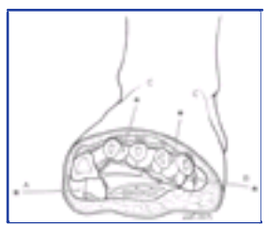

Figure 55-19 Compartment syndromes of the foot. Suggested needle pathways (*) to measure intracompartmental pressures: *A,* Medial. *B,* Lateral. *C,* Interosseous compartments. The central compartment is surrounded by these compartments. *(Modified from Myerson M: Acute compartment syndromes of the foot. Bull Hosp Jt Dis 47:251, 1987.)*

and fourth web spaces at the metatarsal bases (Fig. 55-19*C*). The needle is directed perpendicularly to the skin to a depth of 1 cm. [45] Proper needle placement is confirmed by seeing a rise in pressure during digital compression of the interosseous compartment adjacent to the needle entry point.

COMPLICATIONS

All of the procedures described have a similar risk of infection, both local and systemic; exact risk figures are unavailable. Strict adherence to aseptic technique, careful sterilization of catheters, and use of sterile, disposable components whenever possible help to minimize this risk.

All monitoring procedures cause some pain. The pain associated with the actual insertion of needles and catheters may be reduced by local anesthesia. Caution is advised to avoid injections into the compartment, which might result in inaccurate readings or further increases in tissue pressure. Once inserted and secured, the needle or catheter should produce only minimal discomfort. However, the injection of saline into an already tense compartment may produce further discomfort. Reassurance and systemic analgesia may be required to gain the cooperation necessary for accurate readings. Technically, the injection of fluid may actually exacerbate a compartment syndrome. Whitesides and colleagues found an increase in compartment pressure of 1 mm Hg for each milliliter of saline infused into human anterior leg compartments.[9] It is difficult to assess the relevance of this problem, but recognition of the potential for its occurrence is important.

INTERPRETATION

When properly performed, each method has an acceptable accuracy in the clinical setting. Investigators report standard deviations from 2 to 6 mm Hg with any of the techniques.[1] [11] [12] [13] [14] It is generally agreed that the mercury manometer system is

the least accurate. The arterial line system used with a simple or side-port needle provides a high degree of accuracy for simple, episodic readings. The Stryker intracompartmental pressure monitor system provides consistent, accurate readings for episodic and extended monitoring situations. The electronic transducer-tipped catheter system is a promising new device, but not widely available.

Reports of normal human compartment pressures vary in the literature. In comparing several techniques, Shakespeare and associates found an average pressure of 8.5 mm Hg with slightly higher pressures in individuals who were physically fit. [13] Willy et al. found a mean of 13 mm Hg (+/- 8 mm Hg) using the electronic transducer-tipped probe in healthy volunteers.[17] Other investigators have found similar pressures with a range from 0 to 16 mm Hg. [1] [11] [36] Mubarak and Hargens previously stated that an absolute pressure measurement of 30 mm Hg in the compartment should be the "critical pressure" for recommending fasciotomy. [12] [47] [48] Despite the fact that this tissue pressure is abnormal and corresponds to the onset of pain and paresthesias, [12] it does not necessarily precipitate a compartment syndrome in the absence of other factors.

Some variability seems to exist among patients for tolerance of increased pressures. Matsen found that no patients with pressures of <45 mm Hg had symptoms of compartment syndrome, whereas all patients with pressures >60 mm Hg had symptoms.[1] Whitesides found that fasciotomy was required when the intracompartmental pressure approaches 20 mm Hg *below* the diastolic pressure,[9] whereas McQueen et al. recommend using a differential pressure (diastolic minus the compartment pressure) of <30 mm Hg as a criteria for fasciotomy.[49] Heppenstall concluded that consideration of either criteria will produce similar results. [26] He recommends using a ?P (MAP minus the compartment pressure) of 30 mm Hg or less in nontraumatized muscle as a guide to fasciotomy, whereas in traumatized muscle (in which metabolic demands are greater), the critical ?P is 40 mm Hg.[24] It is clear that factors other than compartment pressure alone may become important. Situations in which the mean arterial pressure is lowered (e.g., hypovolemia) may compromise a patient's ability to tolerate even mildly elevated compartment pressures. Duration of increased pressure is also important. There is continued debate over the effects of early versus late fasciotomy (generally regarded as < or >12 hours) in the management of compartment syndrome, especially in trauma. Authors relate varying outcome with regard to infection rate and limb function. [50] There is agreement that the limb may be salvageable for up to 10 to 12 hours, but with very high pressures, the time period may be as little as 4 to 6 hours. [51]

Compartment pressures must be interpreted within the context of the clinical picture. Inaccurate measurements are far worse than no measurement at all. Care must be taken to identify the area of high pressure and greatest tissue damage. [2] Falsely elevated pressures may be a result of needles placed into tendons or fascia, plugged catheters, or faulty electronic systems. Falsely low readings may result from bubbles in the lines or transducer, plugged catheters, or faulty electronic systems. One must carefully troubleshoot the system before making a decision to treat a presumed compartment syndrome.

Actual treatment consists of improving the perfusion pressure gradient. Support of arterial pressures in hypotensive patients can prevent ischemia in marginal compartments. If external pressure is at fault, removal of pneumatic anti-shock garments, casts, or dressings may be therapeutic. Interstitial edema may be a factor in increased tissue pressure as well. The use of diuretics, [52] mannitol,[53] intracompartmental injections of hyaluronidase, [54] and hyperbaric oxygenation[55] [56] have been described for both primary non-invasive therapy and adjunctive to surgical intervention. The latter remains the treatment of choice. If noninvasive therapies fail, fasciotomy, which involves opening the skin and muscle fascia at key points overlying the involved compartments, should be performed. The escape of enclosed muscles causes a decrease in compartment pressure, thereby improving blood flow to the tissues. This procedure is left to personnel who are experienced in the problem and who will subsequently manage the patient. Details of fasciotomy technique may be found in surgical or orthopedic texts. Escharotomy for release of circumferential burn injury may be required on an emergent basis (see Chapter 39).

CONCLUSION

A compartment syndrome, with its myriad potential causes, is a challenging problem for all clinicians. If untreated, the sequelae are devastating. Appropriate management requires rapid assessment and treatment. In the early stages of the syndrome, clinical examination findings may be equivocal. Pressure readings can be an objective aid to prompt recognition of this problem. All of the techniques outlined in this chapter are clinically acceptable. For more prolonged in-hospital monitoring or research, a technique with reliable serial measurement capability is needed. The complications from all of these procedures are negligible in comparison to delayed recognition of a compartment syndrome. Whether using an arterial line system, a mercury manometer system, or Stryker STIC technique, placement of a needle into the compartment for a pressure reading appears to be the technique best suited to episodic ED evaluation of acute compartment syndromes. It is essential to treat underlying factors like hypotension, coagulopathy, and vascular injury or artificially created external pressure, which will exacerbate the syndrome. The use of adjunctive therapies may be helpful, but should not be routinely used in place of definitive surgical intervention. Maintaining a high index of suspicion, especially in the multitrauma, unresponsive, or uncooperative patient is the best approach to maximize outcome.

Acknowledgment

The authors recognize the contributions of David E. Van Ryn, MD, the author of this chapter in the first two editions of the text, and Lawrence B. Stack, MD, the author of this chapter in the third edition of the text.

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Section IX - Genitourinary Procedures

Chapter 56 - Urologic Procedures

Robert E. Schneider

This chapter addresses urologic conditions that either are initially or eventually associated with an emergency procedure or may need to be performed in the absence of a urologic surgeon.

Paraphimosis and priapism are both urologic emergencies. Treatment options must be instituted as soon as they are encountered. Phimosis by itself is not an emergency unless it results in complete obstruction of the preputial opening (rare) or is transposed into paraphimosis.

Testicular torsion is an emergency that can be difficult to diagnose under the best of clinical conditions. While some argue that surgical exploration, rather than radionuclide scanning or color Doppler ultrasonography, is the diagnostic and therapeutic procedure of choice, most urologists prefer some study prior to surgical exploration. This chapter addresses bedside maneuvers including testicular detorsion for this entity in the setting of scrotal pain.

Access to and the subsequent evaluation of bladder urine is clinically important to every practicing clinician. Various approaches to urine sampling, including the techniques and complications of male and female urethral catheterization in various clinical situations, are addressed.

Finally, a discussion of radiographic imaging of the genitourinary system is provided, with an emphasis on assessing lower urinary tract injury. Although the timing of genitourinary radiologic examinations within the workup of the critically ill multiple trauma patient must be individualized, general guidelines are provided.

PHIMOSIS AND DORSAL SLIT

Background

Phimosis has been recognized since ancient times. Models of phimotic foreskin have been found near the altars of Hygeia and Aesendopius in ancient Greece. Orikosius (A.D. 325 to 403) was the first to describe the dorsal slit as definitive treatment for phimosis.

Following any injury or inflammatory event, the foreskin (prepuce) reacts by forming scar tissue. The normally soft pliable foreskin can develop sufficient distal scarring to make routine retraction of the tissue over the glans penis difficult or impossible. This is especially true if the end of the foreskin is injured, such as in zipper injuries, toilet seat trauma, or other crush injuries (known as the Tristram Shandy syndrome, after the well-known literary character who had a window sash fall on his penis while he was urinating out the window). A chronically irritated and infected foreskin often occurs in diabetic patients. [1] Rarely, a tight phimosis and accompanying poor hygiene may lead to abscesses of the foreskin, which can result in further contracture.

Asymptomatic phimosis does not ordinarily require any treatment. It may prevent or make easy urethral catheterization more difficult. In such situations, the phimotic opening may need to be dilated or crushed and formally incised (dorsal slit), using light sedation and local anesthesia to allow access to the urethral meatus. This minor operative procedure can be performed in the emergency department (ED) by any practicing clinician.

Indications and Contraindications

Dorsal slit of the foreskin is performed in any emergent situation either to gain access to the urethral meatus for urethral catheterization or as definitive treatment following simple foreskin reduction or phimotic ring incision and foreskin reduction in a patient with paraphimosis. Elective circumcision rather than dorsal slit of the foreskin is the definitive procedure of choice in nonemergent situations.

Procedure

The equipment needed to perform dorsal slit of the foreskin is listed in Table 56-1 . After cleansing and draping the penis with sterile towels, one infiltrates 1% plain lidocaine *without* epinephrine into the dorsal midline of the foreskin just beneath the superficial fascia throughout the course of the proposed incision, starting proximally at the level of the coronal sulcus and proceeding distally to the tip of the foreskin (Fig. 56-1). After 3 to 5 minutes, the foreskin is grasped with toothed forceps to test for anesthesia. The operator must be certain that the inner surface of the foreskin is also anesthetized. If this area is not numb, a dorsal nerve block or "ring block" at the base of the penis should be used (Fig. 56-2). [2] [3]

After achieving both adequate local anesthesia and light sedation, the operator takes a straight hemostat and carefully advances both jaws of the hemostat proximally to the area of the coronal sulcus between the inner layer of the foreskin and the smooth glans penis, carefully separating any existing preputial adhesions. Care must be taken that the meatus and urethra are visualized or palpated at all times so they are not inadvertently injured during this maneuver. Once release of adhesions is complete, the hemostat is then opened, and one jaw of the hemostat is placed in the recently developed plane between the glans penis and the superior overlying inner layer of foreskin; the hemostat is advanced to the level of the coronal sulcus and then closed, effectively crushing the interposed anesthetized foreskin (Fig. 56-3). The closed hemostat is left in place for 3 to 5 minutes, after which it is removed, and the resultant serrated, crushed foreskin is cut longitudinally with straight scissors throughout the extent of the crushed tissue. Normally, the incised, anatomically approximated skin edges bleed and ooze. Not infrequently, these skin edges may separate (Fig. 56-4*A* and *B*). Two absorbable chromic or Vicryl running hemostatic sutures may be placed, each beginning proximally at the apex of the dorsal slit and carried distally, reapproximating the two leaves of foreskin.

TABLE 56-1 -- Equipment Needed to Perform Dorsal Slit for the Emergency Treatment of Phimosis

1% lidocaine (Xylocaine) without epinephrine
5-mL syringe
27-ga needle
1 straight Crile clamp
1 straight scissors
1 needle holder
4-0 absorbable suture

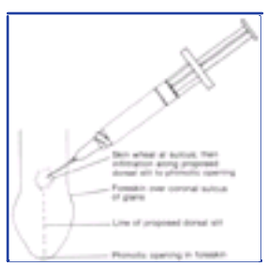

Figure 56-1 Technique for obtaining anesthesia before performing a dorsal slit.

After successful dorsal slit of the foreskin, the prepuce is easily retracted for cleansing of the glans penis or exposure of the urethral meatus. Postprocedural conscientious foreskin reduction to its normal anatomic position must be assured after any distal penile procedure, to avoid iatrogenic paraphimosis.

Ideally, a definitive elective circumcision is recommended following a dorsal slit. Some patients complain about the appearance of their incised foreskin ("dog ears") and the relative inconvenience during urination, whereas others are pleased they no longer have their phimosis and refuse further treatment (Fig. 56-4*C* and *D*).

Complications

Injury to the urethral meatus and the glans penis may occur if the hemostat or straight scissors are blindly and unknowingly introduced into the urethra. Bleeding may occur if the hemostat

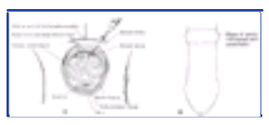

Figure 56-2 *A*, Dorsal nerve block at the base of the penis will provide anesthesia of only the dorsum of the penis. *B,* Circumferential subcutaneous lidocaine infiltration for a ("ring") field block at the base of the penis can provide anesthesia to the entire distal penis. *(A from Soliman MG, Tremblay NA: Nerve block of the penis for postoperative pain relief in children. Anesth*

Analg 57:495, 1978. Reproduced by permission.)

has not adequately crushed the foreskin or the scissor incision is made lateral to the serrated crushed tissue. The latter two problems are easily resolved with the previously described running hemostatic suture.

PARAPHIMOSIS AND FORESKIN REDUCTION

Background

Paraphimosis is a urologic emergency. By definition, it is the inability to reduce the proximally positioned foreskin over the glans penis back to its normal anatomic position. Paraphimosis is most often associated with phimosis. In the obtunded or demented patient, subjective pain may not be perceived or communicated. Today, the most common cause of paraphimosis is iatrogenic: the catheterist or examining health care provider forgets to reduce the foreskin after penile examination or urethral instrumentation. Paraphimosis can be quite subtle and may either be unrecognized or misdiagnosed by those unfamiliar with the condition as an allergic reaction, penile trauma, or an infection (Fig. 56-5*A*).

The coexisting phimotic ring initially interferes with venous and lymphatic drainage, precipitating foreskin swelling. Over time, the degree of swelling prevents manual reduction of the retracted foreskin. When left untreated, eventual arterial embarrassment leads to tissue anoxia, skin ulceration, and, ultimately, to infection or penile gangrene, or both.[4]

Emergent manual or surgical reduction of the edematous foreskin is mandatory to restore proper circulation, relieve discomfort, and permit resolution of potential serious sequelae: skin ulceration and gangrene. It must be done as soon as a paraphimosis is recognized. Once the foreskin is successfully reduced, dorsal slit as previously described is advised in those cases of potential patient noncompliance until definitive circumcision can be performed.

Indications and Contraindications

Emergent reduction of a paraphimotic foreskin is indicated whenever the condition exists. There are no contraindications.

Figure 56-3 Placement of hemostat for treatment of phimosis. The "tenting up" of the foreskin in this manner proves that the tip of the hemostat is *not* in the urethra or under the glans. Once properly placed between the glans and overlying foreskin, the instrument is closed to crush the area of the foreskin to be cut.

Procedure

The equipment needed for reduction of paraphimosis is listed in Table 56-2 .

Manual Reduction[4] [5]

A nonirritating topical anesthetic lubricant is applied to the inner surface of the foreskin (not to the shaft of the penis) and

Figure 56-4 Treatment of phimosis. *A,* Dorsal slit in phimotic foreskin. Exposed glans is shaded. A single (dorsal) lengthwise incision has been made through crushed tissue (a^1, outer layer of foreskin; *a,* inner layer of foreskin). *B*, Cut edges of foreskin drawn back around glans penis. First, a^1 is sutured to *a*, then the remainder of the cut edges are sewn together for hemostasis. *C,* Final "beagle-ear" deformity of ventral transposed foreskin after the dorsal slit procedure has been completed. *D*, Postoperative appearance of dorsal slit.

the glans to reduce friction and decrease the discomfort of the procedure. A previously described penile block may be performed if required. Light sedation is an excellent adjunct. The foreskin is then manually compressed for several minutes to reduce as much edema fluid as possible (Fig. 56-5*B*). Snugly wrapping the distal penis in a 5-cm piece of Elastoplast (or elastic bandage) for 10 minutes has also been described. [6] Injection of hyaluronidase[7] has been reported to reduce edema, but seems unnecessary. If the patient is catheterized, *the catheter may be removed and replaced later, but generally that is not necessary* . Reduction can be completed with the catheter in place.

The index and long fingers of both hands are placed in apposition just proximal to the phimotic ring. Both thumbs are aligned on the urethral meatus. Constant force is applied as the thumbs try to invert the glans penis proximally while the index and long fingers attempt to reduce the phimotic ring distally over the glans penis into its normal anatomic position (Fig. 56-5*C*). Successful reduction results in the appearance of an uncircumcised penis with a phimotic foreskin. Alternatively, the thumb may be used to push the glans through the foreskin that has been encircled by the entire palm—in a maneuver similar to taking off a rubber glove (Fig. 56-5*D* and *E*). The key to success in both of these maneuvers is the application of slow, steady pressure.

Assisted Manual Reduction ("Iced-Glove" Method or Babcock Clamp Method)

If the constricting phimotic ring cannot be brought down over the glans easily, additional measures may be used. In the "iced-glove" method, [8] cold compression is used to reduce foreskin swelling and to induce vasoconstriction in the glans penis. A large latex glove is half filled with crushed ice and water, and the cuff end is securely tied. The thumb of the glove is invaginated by the operator and then is drawn over the lubricated paraphimotic penis. The thumb of the glove is held securely in place over the penis for 5 to 10 minutes. The combination of cooling and compression usually decreases the edema sufficiently to permit manual reduction of the foreskin. If the constricting ring cannot be brought down over the glans after this maneuver, it may be necessary to use Babcock clamps. From six to eight Babcock clamps (*not* Allis clamps, which are serrated and intolerably painful for the patient) are

Figure 56-5 *A,* Paraphimosis, pictured here, may be mistaken for penile trauma, angioedema, or infection. The cause of paraphimosis in this case was failure to replace the foreskin following a catheter change in a demented nursing home patient. Prior to reduction, the catheter is usually removed. When the edema is minimal, the catheter may be left in place during reduction. *B,*

Manual compression of the foreskin, or wrapping the penis with an elastic bandage for a few minutes, may reduce edema before a reduction of the paraphimosis is attempted. *C,* Technique for reduction of paraphimosis. Gentle, steady pressure is placed on the glans with the tips of the thumbs while gentle traction is applied to the foreskin. In this line drawing, the catheter is left in place during reduction. *D,* In a manner reminiscent of removing a rubber glove, the thumb forces the glans through the foreskin that is encircled by the entire palm to achieve final reduction *(E). (C from Neuwirth H, et al: Genitourinary imaging and procedures by the emergency clinician. Emerg Med Clin North Am 7:1, 1989.)*

used to grasp the phimotic ring circumferentially.[9] The clamps are then slowly pulled distally over the glans. With gentle, slow traction, the phimotic ring can hopefully be reduced over the glans penis (Fig. 56-6).

Phimotic Ring Incision

This procedure is indicated when other methods fail or when skin ulceration, infection, or gangrene is present. [10] [11] [12] Although it is usually recommended that this procedure be performed in the operating room, it may be undertaken in the ED using local anesthesia and light sedation.

The penis is cleansed and draped with sterile towels. Using a 1% solution of lidocaine *without epinephrine*, one infiltrates the phimotic ring on the dorsum or 12 o'clock position (when facing the patient), making sure to infiltrate proximally and distally as well as into the constricting ring (Fig. 56-7*A*).

The skin, the edematous subcutaneous tissue, and the constricting ring are then slowly incised perpendicular to the

TABLE 56-2 -- Equipment Needed for Reduction of Paraphimosis

For Nonoperative Emergency Reduction of Paraphimosis
1% lidocaine (Xylocaine) jelly
Crushed ice
Size 8 latex surgical glove
Babcock clamps (6 to 8)
For Operative Reduction of Paraphimosis
Sterile preparation solution
1% lidocaine (Xylocaine) without epinephrine
5-mL syringe
27-ga needle
No. 15 surgical knife with handle
Needle holder
4-0 absorbable suture

phimotic ring. You must incise directly on the ring so as not to injure the penile shaft. When the constricting ring is completely incised, it will spring open, and the foreskin edges will relax laterally and produce a diamond-shaped defect (Fig. 56-7*B*). At this point, successful foreskin reduction can be accomplished quite easily and an immediate dorsal slit incision carried out by extension of the wound, if recurring paraphimosis or patient compliance is a concern. Definitive circumcision, as always, is elective, and should be delayed 7 to 10 days until edema, inflammation, and ulceration have resolved.

Complications

Penile shaft laceration or simple tearing of compromised penile skin may occur during manual or surgical paraphimotic reduction. Simple suturing will resolve most injuries.

PRIAPISM AND PENILE INJECTION/ASPIRATION

Priapism is manifested by a persistent, usually painful, penile erection, unrelated to sexual stimulation and not relieved by ejaculation. Most often priapism is a urologic emergency that is associated with a high incidence of impotence, regardless of treatment. It is characterized clinically by a soft glans penis and spongy urethra in the presence of two erect penile bodies or corpora cavernosa. Although reported in most age groups, the condition is most common between the ages of 30 and 50 years.

The pathophysiology of priapism is complex, and is likely the combination of the sympathetic and parasympathetic

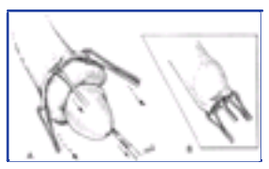

Figure 56-6 *A,* Application of Babcock clamps to reduce paraphimosis. *B,* Foreskin reduced. *(From Skoglund RW, Chapman WH: Reduction of paraphimosis. J Urol 104:137, 1970. Reproduced by permission. © Williams & Wilkins, 1970.)*

nervous system and local factors. The pharmacologic basis for treatment is based on manipulating blood flow via the a and ß receptors. As an overall mechanism, increased arterial inflow of blood into the corpora cavernosa secondary to dilation of the cavernosal arteries with relaxation of the cavernosal tissue and secondary compression of the emissary veins leads to engorgement of both corpora cavernosa during an erection. When the cavernosal pressure approaches the arterial pressure, blood flow is markedly reduced. Ischemic or low-flow priapism results after several hours of continuous painful erection, leading to intracavernosal acidosis and sludging of blood with subsequent thrombosis of the cavernosal arteries, fibrosis of the corporal tissue, and irreversible impotence. This urologic emergency may be treated by the emergency clinician, although in some settings this pathology is best referred.

High-flow priapism is less common than low-flow priapism and usually results from traumatic production of an arteriocavernosal fistula. It is not associated with intracavernosal ischemia or acidosis and therefore is painless and may be treated electively rather than emergently.

In the past, priapism was most often encountered as a complication of a number of medical (e.g., hematologic, neoplastic, and drug-related) conditions (Table 56-3). Today many cases are idiopathic or iatrogenic, resulting from the current practice of using vasoactive substances (e.g., papaverine and phentolamine) to induce penile erections in impotent men. Sickle cell disease is the leading cause of priapism in some settings. Sickle cell patients may experience such a high rate of recurrence that home self-injection of vasoactive drugs

Figure 56-7 *A,* Anesthetizing the penis for surgical treatment of paraphimosis. Line of infiltration of local anesthesia used before performing dorsal slit. *B,* Incision for paraphimosis. Diamond-shaped defect resulting from incision of foreskin. The two apices of the dorsal slit *(a* and *b)* are approximated after the foreskin is reduced.

TABLE 56-3 -- Etiologies of Priapism

Intracavernosal agents
Papaverine
Phentolamine
Prostaglandin E_1
Antihypertensive agents
Ganglion-blocking agents
Arterial vasodilators
a-Antagonist agents
Calcium-channel blocking agents
Psychotropic drugs
Phenothiazines
Butyrophenones
Hypnotics
Trazodone
Selective serotonin-reuptake inhibitors
Anticoagulants
Heparin
Warfarin
Recreational drugs
Cocaine
Marijuana
Ethanol
Performance-enhancing drugs
Hormones
Gonadotropin-releasing hormone (GRH)
Tamoxifen
Testosterone
Miscellaneous

Metoclopramide
Omeprazole
Hydroxyzine
Total parenteral nutrition (TPN)
Hematologic
Anemia
Leukemia
Multiple myeloma
Sickle-cell disease
Thalassemias
Metabolic
Amyloidosis
Fabry disease
Gout
Miscellaneous
Carbon monoxide
Malaria
Black widow spider venom
Spinal stenosis
Asplenism

From Mulhall JP, Honig SC: Priapism: Etiology and management. Acad Emerg Med 3:810, 1996. Reproduced with permission.

into the penis has been advocated. Cocaine use is one etiology that is likely underreported. [13] A drug screen may unravel some discrepancies between clinical findings and history. As an end result, vasoactive drugs promote engorgement of the corpora cavernosa and reduction in venous outflow, which may result in low-flow or ischemic priapism.[14] [15] [16] Prostaglandin E_1 (PGE_1) is currently the only FDA-approved drug for impotence. PGE_1 produces increased penile blood flow by enhancing smooth muscle relaxation, and the incidence of priapism with this medication is quite low. Penile rigidity due to a nondeflating penile prosthesis (pseudopriapism) or malignant replacement of the corpora in patients with bladder or prostate cancer should not be confused with true priapism.

Indications and Contraindications for Priapism Procedures

The emergency clinician should attempt to identify reversible causes for low-flow priapism and, in conjunction with a urologist, initiate specific corrective therapy as soon as possible. Almost 66% of cases of low-flow priapism in children and young adults are due to sickle cell disease, and such cases may respond to noninvasive standard anti-sickling measures (including exchange transfusion).

Regardless of the etiology, this distressing condition is first treated with adequate analgesia, often consisting of parenteral opiates and benzodiazepines. Empiric terbutaline, 0.25 to 0.5 mg given subcutaneously (SQ), is recommended for every patient presenting with low-flow priapism as soon as the diagnosis is made; this treatment may be repeated, when needed, in 15 to 20 minutes. If a patient with previous low-flow priapism calls from home and has the appropriate supplies, he should be instructed to inject himself with terbutaline (as described earlier) or take a 5 mg terbutaline tablet by mouth, if available, before coming to the ED. The pharmacotherapeutic action of terbutaline for priapism is not well elucidated. Oral pseudoephedrine (60 to 120 mg) also has been suggested as a noninvasive initial therapy for low-flow priapism secondary to intracavernosal agents, but its efficacy has not been well studied. [18] While terbutaline and other oral or parenteral medications have been advocated for the treatment of priapism, it is the editors' experience that such interventions are only rarely successful, and many cases will require more aggressive procedures. Table 56-4 lists the reversible causes of low-flow priapism with their respective therapies.

TABLE 56-4 -- Treatment of Low-Flow Priapism Based on Etiology

A. Terbutaline 0.25 to 0.5 mg subcutaneously (SQ) in the deltoid muscle area or thigh for all patients with priapism (may be repeated in 15–20 min); alternatively, 5 mg terbutaline *per os* (PO) may be used (1 dose)
B. Reversible causes
1. Sickle cell anemia
a. Packed red blood cell (RBC) transfusion
b. Hyperbaric oxygenation (investigational)
2. Iatrogenic injection of PGE_1, papaverine, or phentolamine for impotence
a. Corporal aspiration of 30–60 mL of blood, followed by observation
1) Detumescence: no further treatment
2) Persistent erection: inject an equal volume of a-agonist (e.g., phenylephrine, 10 mg in 500 mL of normal saline)
3. Leukemic infiltration
a. Specific chemotherapy
4. Medication (phenothiazines, trazodone)
a. Corporeal aspiration, observation
b. a-Agonist instillation, observation
c. Heparin irrigation of corporeal bodies, observation
d. Corpus cavernosum-spongiosum shunt
C. Nonreversible causes
1. Idiopathic
2. High spinal cord lesion

In the majority of patients, no reversible cause will be disclosed. When detumescence does not occur with SQ terbutaline, the condition necessitates corporal aspiration/injection alone or corporal aspiration and irrigation with an a-agonist (epinephrine or phenylephrine [Neo-Synephrine]). Conservative measures, such as sedation and analgesia, oral estrogens, ice-water enemas, transurethral diathermy, spinal/epidural/general anesthesia, and local anesthetic injections have not

proven to be of value and should not be used in lieu of definitive intracavernosal therapy. [18]

Should initial medical and corporal aspiration and irrigation fail, Lue and associates recommend that further therapy be guided by cavernosal blood gas and pressure measurements.[19] If one elects to use blood gas analysis of aspirated corporal blood to help guide therapy, the initial results will reflect the degree of ischemia present in the priapismic penis. Sequential blood gas analyses that fail to show an improvement in the initial acidosis (pH remaining at =7.10) despite corporal aspiration or irrigation, or both, suggest that a more aggressive definitive corpus cavernosum-spongiosum shunt may be indicated, and urgent urologic intervention should be sought. Although not usually performed, the aspirating or irrigating corporal needle may also be used for measurement of intracorporal pressures similar to the approach outlined for muscular compartmental pressures.[20]

A variety of regimens and protocols have been suggested for the treatment of priapism. No single intervention has known superior efficacy. The mainstay of therapy for advanced priapism is aspiration of the corpora cavernosa combined with saline irrigation, usually coupled with the intracavernosal injection of a-adrenergic agents. When urologic consultation is unavailable or delayed, the emergency clinician can initiate therapeutic corporal aspiration. Because prolonged priapism increases the risk of subsequent erectile dysfunction, an aggressive management strategy is advised. Impotence rates of up to 35% to 60% have been reported when priapism persists for 5 to 10 days, respectively.[18] If present for more than 24 hours, priapism often does not respond to aspiration techniques. Recurrence is not uncommon and some patients require multiple procedures on a frequent basis. Shunting procedures may be required if the measures described earlier are not successful.

Equipment

The equipment needed for aspiration and irrigation of the corpus cavernosum is listed in Table 56-5 .

Procedure

(Simple intracorporal injection): While the aspiration/irrigation technique is often recommended, some clinicians have had success with the simple injection of vasoactive solutions into the corpus cavernosum. This negates the more complicated irrigation procedure and may be attempted as an initial approach to the uncomplicated case. This is the technique used by outpatients as a self-injection technique for recurrent priapism. With this technique a 25- to 27-ga needle is used to inject vasoactive substances into the corpus at the base of the penis, with the aim of pharmacologically reversing the priapism process. Often this can be done without anesthesia. Specific protocols are not established, but one option is to draw up 0.5 mg of phenylephrine into a 3-mL syringe, and add 2 mL of saline diluent. The needle punctures the corpus

TABLE 56-5 -- Equipment Needed for Aspiration of Corpus Cavernosum for Low-Flow Priapism

27-ga needle (for penile block)
12-mL syringe (for local anesthetic)
1% lidocaine without epinephrine (for penile block)
Sterile drapes
Gauze sponges
Povidone-iodine (or alternative) preparation solution
19-ga butterfly needles (for aspiration)
Two 30-mL syringes (for aspiration and injection)
Sterile basin for aspirated blood
Blood gas syringe with cap
Injection fluid (1 of the following vasoactive agents* is diluted with 500 mL of normal saline and up to 20 to 30 mL is administered; 5000 units of heparin added to solution is optional; see text)
Phenylephrine, 10 mg/500 mL of saline
Norepinephrine, 1 mg/500 mL of saline
Epinephrine, 0.5 mg/500 mL of saline

*Systemic absorption of vasoactive agents may occur with adverse cardiovascular effects.

at the 10 o'clock or 2 o'clock position at the base of the penis, blood is aspirated to confirm position, and the solution is injected. If not successful in 30 minutes, a repeat injection can be given, up to a total of 3 injections (Fig. 56-8). In one small

Figure 56-8 As an initial approach to priapism, and as an alternative to aspiration/irrigation, the intracorporeal injection of vasoactive drugs such as phenylephrine may be used. A small gauge needle is used to inject the corpus at 10 o'clock or 2 o'clock, at the base of the penis. Repeat injection may be required.

study, successful detumescence was achieved in eight of nine patients by simple intracorporal injection of phenylephrine with this regimen, with three or fewer injections being required.[21] Alternatively, 0.1 mg of epinephrine (0.1 mL of 1:1000) diluted with 2 mL of saline may be used.

(Aspiration and irrigation): This procedure entails drainage of blood from the erect penis, irrigation of the corpus cavernosum, and the instillation of a vasoactive medication. As an alternative intervention, simple injection is also described. The patient is placed in the supine position (Fig. 56-9*A to D*). Parenteral analgesia and sedation are suggested. Local anesthesia is also recommended for this procedure. An injection of 1% plain lidocaine placed at the base of the penis for a dorsal penile nerve block or placement of a circumferential penile block can be done (see Fig. 56-2). A local anesthetic injection at the site of aspiration is also commonly performed. The penis is sterilely prepared and draped. When standing to the

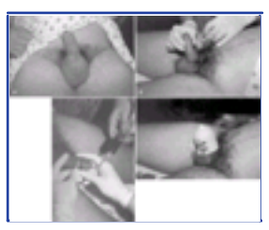

Figure 56-9 *A,* This patient experienced 18 hours of priapism after penile self-injection of papaverine as therapy for impotence. *B,* A penile dorsal nerve block is performed. *C,* A 19-ga butterfly needle is inserted into the corpus via the penile shaft at either the 2 o'clock or 10 o'clock position, and aspiration is performed. Slow steady suction will be most successful, while excessive suction may halt the aspiration. Do not puncture the corpus via the glans (see text). *D,* After detumescence with aspiration or with irrigation and injection of a vasoactive medication (see

text), the penis is wrapped with an elastic bandage to discourage re-engorgement and to compress the puncture site.

right of the patient, the clinician grasps the shaft of the penis with the left hand using the thumb and index finger. An engorged corpus cavernosum is palpated laterally and a 21- to 19-ga butterfly needle is inserted into *one of the corpora cavernosa*.[20] Alternatively, a 20-ga intravenous (IV) catheter may be used, but the butterfly needle catheter is preferred. If palpation failsto demonstrate the corpus, inserting the needle at either 10 o'clock or 2 o'clock will usually readily gain access to this large vascular structure. Since there is communication of blood flow between both sides, the operator needs access to only one of the corpora. Either side may be punctured. The site of needle placement is one of personal preference, and locations from the base to the distal shaft of the penis have been suggested. The editors suggest using the midshaft for irrigation, and the base for simple injection. *The glans should not be used as a puncture site.* The needle is advanced in a 45° angle, using constant suction. Blood is usually readily

aspirated, and once it is obtained, the needle is not advanced. Deep penetration is to be avoided to minimize the risk of injury to the cavernosal artery during this procedure.

An initial 20 to 30 mL of corporal blood is aspirated while the operator milks the corpus with the left hand. *Excessive suction should not be applied since this often halts the aspiration.* A common error is to use too much suction with a 60-mL syringe. Using a 10-mL syringe, and changing it if it fills with blood, is preferable. If a butterfly is used, there is no danger in dislodging the needle when changing syringes. This initial aspiration is continued until the original egress of dark blood ceases and bright red arterial blood is obtained, or complete detumescence is obtained. Because multiple anastomoses exist between the two corpora cavernosa, bilateral aspiration is not required.

If detumescence is achieved after initial aspiration, no further treatment may be required. However, if this is successful, the editors advise injecting an aliquot of vasoactive substance. Simple aspiration is most likely to be successful if the priapism has been present for less than 24 hours. If retumescence occurs, then repeated aspiration, followed by irrigation with an equal exchange of an a-agonist solution for aspirated corporal blood, should be performed. A number of irrigating solutions have been suggested, but none has proven superiority. Some suggest 20 to 30 mL of a phenylephrine/normal saline solution (10 mg of phenylephrine in 500 mL of normal saline) as the exchange for 20 to 30 mL of aspirated corporal blood. Some clinicians add 2500 to 5000 units of heparin to the solution, but the value of heparin is unproven. The use of ß-agonists for corporal injection or irrigation also has been reported, and the advantage of pure a-agonists over ß-agonists is a subject of debate. O'Brien and associates recommend a 1:100,000 solution of epinephrine (1 mg/100 mL of saline), irrigating with 2 to 3 mL at a time to a maximum dose of 0.1 mg (10 mL).[20] Alternatively, 1 mg of epinephrine can be added to 1 L of saline, with irrigation performed using 20 to 30 mL aliquots. A norepinephrine solution also may be used (see Table 56-5). Note that the corpus cavernosum has ready access to systemic circulation and injecting a drug into it is essentially the same as an IV injection. When detumescence occurs, the unmetabolized drug enters the venous system; therefore, vasoactive drug dosages should be monitored. Some clinicians wrap the penis snugly in an elastic wrap with the needle securely in place following aspiration to discourage retumescence and to decrease hematoma formation of the penile shaft.

Complications

Although hematoma and infection can occur with properly performed aspiration, these complications are infrequent. Both phenylephrine and epinephrine can be absorbed systemically, with the potential for toxic effects.[19] Therefore, the intracavernosal use of vasoactive agents is contraindicated in patients with conditions sensitive to these agents (e.g., severe hypertension, dysrhythmias, monoamine oxidase inhibitor use). Blood pressure and cardiac rhythm should be monitored throughout the procedure if the patient is at risk. Supplemental O_2 should be considered in any patient undergoing conscious sedation. Failure to aspirate blood is a potential complication, usually because of a misplaced needle, applying excessive suction, or if blood has clotted. Because impotence is a well-recognized complication following priapism, regardless of the cause or the promptness of therapeutic intervention, the patient must be advised both verbally and in writing of this potential complication.

ADJUNCTIVE TESTING IN ACUTE SCROTAL PAIN

Establishing a diagnosis in the patient with acute scrotal pain creates significant clinician anxiety. The condition most easily confused with torsion of the testicle is acute epididymitis. The prompt diagnosis of testicular torsion and differentiation of this condition from epididymitis can be quite difficult, but it is obviously crucial to the patient's care. The treatment of acute epididymitis requires appropriate antimicrobial and supportive therapy. The treatment of testicular torsion requires emergent operative intervention, not excessive adjunctive testing. [22] Although all clinicians recognize the need for expeditious surgery in the setting of known torsion, not all consultants will agree on surgery in the absence of some adjunctive testing.

Torsion of the testicle is the most frequent cause of acute testicular pain in celibate men younger than 20 years. One pediatric series of patients presenting with a painful scrotum reported a 6% incidence of epididymitis vs a 42% incidence of testicular torsion and a 32% incidence of torsion of the appendix testis and epididymis. [23] It is interesting to note that pyuria was seen in 10% of the patients with testicular torsion and in 80% of the children with epididymitis. In men 20 years or older, testicular torsion is less common, but it must always be considered in the differential diagnosis as it may occur well into the seventh decade of life. [24]

In contrast, epididymitis is an *uncommon* condition in young celibate boys unless a congenital lower urinary tract abnormality is present, promoting urinary tract infection with subsequent epididymitis.

As with all difficult diagnostic dilemmas, the patient's history is of paramount importance in establishing the correct diagnosis. The evaluation of acute scrotal pain is no different. In considering epididymitis, detailed and often probing questions must be asked regarding the patient's work habits and his sexual experiences. The pathophysiology of epididymitis emanates from the generation of increased pressure in the prostatic urethra as a result of either heavy lifting (e.g., in roofers or construction workers), detrusor external sphincter dyssynergia (e.g., in patients with neurogenic bladder dysfunction), or iatrogenic promotion of a Valsalva maneuver for whatever reason. These mechanisms force sterile or infected urine out through the ejaculatory ducts into the vas deferens (urethrovasal reflux), then retrograde down the vas into the tail of the epididymis (globus minor). Initially, this may cause nonspecific lower quadrant abdominal pain or inguinal canal pain (vasitis) before it causes scrotal or testicular pain, and vasitis should be included in the initial differential diagnosis in any male presenting with lower abdominal pain.

Sexually transmitted diseases with subsequent epididymitis are quite common in sexually active young men, usually associated with chlamydial or gonococcal urethritis. Signs and symptoms of infection may be minimal when the patient is first examined. [25] The presence of genitourinary infection, a history of previous sexually transmitted diseases, or a history of multiple sexual partners and the absence of penile protection during intercourse may all be important clues to the diagnosis of epididymitis. In older men, epididymitis is usually due

to urinary tract infection secondary to outlet obstruction or urethral stricture disease. In such cases, enteric gram-negative bacilli and *Pseudomonas* species are the predominant organisms. If one cannot be certain after taking a careful history and examining the patient that epididymitis or torsion of the appendix testis or epididymis is the correct diagnosis, then urologic consultation to exclude testicular torsion is required. A clinical diagnosis of epididymitis should be based on the focal finding of epididymal induration and tenderness (the vas deferens also may be involved) in the proper patient population. The site of tenderness is best discerned when examining the patient in the relaxed supine position. Although testicular tenderness can develop with advanced epididymo-orchitis, the time course is generally gradual and on an order of days. However, marked testicular tenderness in a patient with advanced epididymo-orchitis may reflect secondary testicular ischemia and also warrants urologic consultation. [26]

The "gold standard" for the diagnosis of testicular torsion is scrotal exploration. [27] However, some clinicians incorporate radionuclide testicular scanning or color Doppler ultrasound examination in their diagnostic work-up. [28] Unfortunately, these studies are time-, technician-, and reader-dependent examinations in a clinical situation in which time is essential. The quintessential point in testicular torsion is that no markers, signs, or symptoms will distinguish incomplete from complete testicular ischemia. One minute there may be total absence of blood flow to the involved testis; the next minute sufficient spontaneous detorsion may have occurred to provide temporary testicular perfusion. This is the dilemma. Scrotal exploration, detorsion, and orchiopexy or orchiectomy of the involved testis and orchiopexy of the contralateral testis is a relatively benign, straightforward operative procedure.

Intravaginal testicular torsion is a congenital bilateral abnormality. The ischemic testis must be detorsed and pexed with nonabsorbable (e.g., nylon, polypropylene), rather than absorbable (e.g., chromic, Vicryl), suture. The torsed testis that is pexed with absorbable suture remains at risk for subsequent postoperative torsion. Orchiopexy of the nonischemic contralateral testis is mandatory to ensure prevention of future torsion.

Once the diagnosis of testicular torsion is suspected, a call should be placed immediately to notify a urologist of the suspected diagnosis, the perceived need for surgical exploration, and the fact that you will be attempting testicular detorsion while awaiting patient transport to the operating room. At some point before the patient leaves the ED, meticulous charting to document time, suspected diagnosis, notification of the urologist, and any manipulation of the affected testis must be done. All efforts are then focused on attempting testicular detorsion.

Doppler Assessment of Testicular Perfusion

Indications and Contraindications

The most important nonoperative adjunctive procedure in suspected testicular torsion is testicular *detorsion*. This can be carried out with or without the aid of a Doppler stethoscope. Using the Doppler stethoscope alone to make a diagnosis of testicular torsion or successful detorsion is *not* recommended. Nonetheless, the Doppler device may be used to support one's clinical assessment in cases strongly suggestive of epididymitis or to monitor the response to detorsion efforts.

Equipment

In circumstances in which one wishes to use the Doppler, the directional Doppler operating on a 10-MHz transducer is recommended. See Chapter 69 for further discussion of Doppler ultrasound physics. A pencil transducer is the most appropriate (e.g., the Model 806 directional 10-MHz Doppler, Park Electronics Laboratory, Beaverton, OR). The Doppler response may be transmitted over a loudspeaker, copied on a measuring device, or transmitted through a stethoscope to the clinician. The higher the frequency of the transducer, the narrower the beam and the less the distance of transmission through the tissues. In this respect, a 10-MHz transducer is preferred over a lower-frequency transducer for examination of the testicle.

Technique

Because scrotal tenderness often precludes adequate examination, anesthesia may be needed. A cord block using 1% lidocaine may be applied at the external ring (see discussion of this block under Testicular Detorsion later in this section). The area of maximum testicular swelling is determined. An aqueous transmission gel is then placed over the scrotum. Holding the testicle in one hand and the Doppler probe in the other, one displaces as much of the scrotal wall as possible between the skin and the underlying testicle. The Doppler probe should be placed in the center of the testicle, pointing slightly caudally so that pulsations in the cord are not detected (Fig. 56-10). (Firm probe pressure "focuses" the ultrasound waves deep to the scrotum into the testis.) The pulsation in the tender, ipsilateral testicle is then compared with that in the contralateral testicle. Decreased or absent flow to the ipsilateral testicle is most surely a result of torsion. Increased flow to the ipsilateral testicle may be a result of epididymitis, inflamed scrotal tissue, a false signal from either the cord or the examiner's fingers, or a false comparison with a contralateral partial torsion of the testis. [29]

The *funicular compression test* should be performed to confirm that flow signals are related to perfusion to the testicle. If the increased signal lessens on compression

of the patient's spermatic cord, then the signal is most probably coming from the patient's testicle and not from inflamed scrotal tissues. If there is no change in the signal on adequate cord compression, the increased flow may be originating in inflamed scrotal tissue, and torsion should still be suspected.

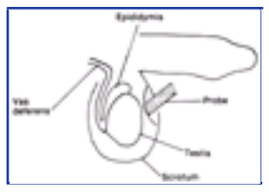

Figure 56-10 Proper position of the Doppler probe in the examination of the acute scrotum. Note caudal orientation of the probe (drawing is a lateral projection).

Complications

Absolute proficiency with the Doppler stethoscope is difficult to achieve, and examination findings are subject to misinterpretation. The greatest risk comes in those cases that represent a later stage of testicular torsion. Although the testes may not be necrotic, a secondary inflammatory response may develop as a result of the underlying ischemia. Any perceived Doppler signal in the hyperemic surrounding scrotum may be misinterpreted by an inexperienced examiner as epididymitis rather than late torsion with an associated inflammatory response, the result of which may be inappropriate treatment. Obviously, the affected testis would remain ischemic, but more important, the contralateral testis would remain at risk for later torsion.

Failure to perform the funicular compression test may lead to the mistaking of scrotal blood flow for testicular blood flow. The Doppler signal should fade and then promptly return as the spermatic cord is first compressed and then released. With an incomplete torsion, one may also hear an attenuated arterial pulse in the testicle. For these reasons, it should be evident that one cannot rely entirely on the Doppler ultrasound examination. [30]

Manual Detorison and Spermatic Cord Anesthesia

Manual detorsion is performed in the following manner. Advise the patient that the procedure will be uncomfortable and painful and offer light sedation if it seems appropriate. The rationale for not using spermatic cord anesthesia with attempted detorsion is that the anesthesia takes away an important subjective end point (i.e., relief of the patient's pain after manipulation of the testis). However, many authors do advocate spermatic cord anesthesia prior to detorsion, and if anesthesia of the spermatic cord is elected, it can be done in the following manner.

Spermatic Cord Anesthesia

Local anesthesia of the spermatic cord using 1% plain lidocaine is usually done at the external inguinal ring. [31] The skin is first prepared with an iodophor solution. The cord can usually be grasped between the thumb and index finger, and 10 mL of 1% plain lidocaine can be directly injected into the cord. If the cord is swollen, as it often is in testicular torsion, or if the testicle is lying very high in the hemiscrotum as a result of spermatic cord torsion (so as to preclude grasping), the cord may be palpated at the pubic tubercle as it passes over the pubis and the lidocaine injected at this landmark. Lee and colleagues [32] were able to perform manual detorsion with local spermatic cord anesthesia in 70% of their adult cases of torsion. Kresling and associates [33] had success in 15 of 16 patients and noted a fair amount of associated cremasteric muscle spasm, which must also be relieved. In their experience, torsion usually resulted from an initial lateral-to-medial rotation (Fig. 56-11), with an occasional caudal-to-cranial component.

Manual Detorsion

The goal of manual detorsion is to reestablish or increase blood flow to a previously ischemic testis. This should be done in conjunction with preparation of the operating suite. *It should never delay operative intervention.*

Before initiating detorsion, one must ensure that the patient is as comfortable as possible in a reclining or supine position. Lithotomy position gives the examiner the most

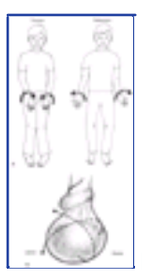
Figure 56-11 *A,* Testicular torsion usually occurs in a medial direction. Detorsion should be attempted initially by rotating the testis outward toward the thigh. This is most successful if attempted within the first few hours of torsion, before the onset of significant scrotal swelling. Intravenous narcotics (such as fentanyl) or a cord block can be administered before attempting detorsion. *B,* Detorsion of the testicle may require testicular rotation through two planes. To release the cremasteric muscle, the testis is rotated in a caudal to cranial direction simultaneously with medial to lateral rotation. The right testis is shown. *(B from Freeman S, et al: Urologic procedures. Emerg Med Clin North Am 4:543, 1986.)*

access to the patient's genitalia and prevents the patient from retreating during the procedure. If light analgesia/sedation are selected, they should be implemented at this time.

Manual detorsion begins with the clinician standing comfortably at the side of the bed or stretcher, preferably on the patient's right side if the clinician is right handed, or vice versa. Detorsion is begun just as one would open a book

(i.e., an initial 180° detorsion of the patient's right testis is done in a counterclockwise fashion. The patient's left testis is detorsed 180° in a clockwise fashion (see Fig. 56-11). Pain relief and an increase in the Doppler signal are the objective end points. If one rotation relieves some but not all of the pain, continue with another rotation. If the initial detorsion is mechanically difficult (which it will be if you are detorsing in the wrong direction) or makes the pain worse, detorse the testis in the opposite direction and observe your result. The objective success or failure of any testicular manipulation can be substantiated by an increase in Doppler signal and the patient's relief of pain.

With successful detorsion, the testicle returns to its normal anatomic position. Resolution of induration and swelling of the spermatic cord, testis, and epididymis will depend on the degree and duration of ischemia. Thus, the more severe the torsion and the longer it has been present, the longer it will take for the edema and induration to resolve. With significant ischemia, the entire epididymis often becomes enlarged like a link sausage (uncommon in epididymitis except in severe cases or those that are initially misdiagnosed or seen late in their clinical course), and the testis becomes quite firm, simulating a testicular tumor. In the author's experience, both of these reversible changes usually resolve over 3 to 4 hours. Occasionally the testis will torse in the opposite direction (medial to lateral) or have multiple twists. This may become apparent as the clinician assesses the results of the detorsion procedure by palpation, relief of edema, and return or increase of the Doppler signal. Even though manual detorsion will save an ischemic testicle, *it should not be substituted for definitive scrotal exploration*.

URETHRAL CATHETERIZATION

Alternatives to Catheterization

The merits of alternative approaches to urine specimen collection over patient catheterization are dependent on the patient's age and clinical setting.

In children, the collection technique reported by Amir and coworkers consists of placing the young child on his or her back. [34] In very young children, collection and analysis of spontaneously voided urine specimens following penile cleansing or suprapubic "finger tap" showed identical urine culture results to those specimens obtained by suprapubic aspiration. [34] The applicability of this to a busy ED is questionable. Often, it is possible to collect a spontaneously voided midstream specimen in a child if ED personnel are prepared in advance. The problem is that the voiding events tend to occur as the child is undergoing venipuncture, spinal tap, or attempted urethral catheterization. And more important, if the first specimen is missed or inadequate, how long are we willing to wait for another chance? Urethral catheterization is quick, definitive, and routinely used.

In adult men without anatomic lesions, first-voided specimens can define the presence or absence of culture-proven bacteriuria. [35] This certainly represents a user-friendly approach to urine collection in a busy ED and needs to be carefully considered.

In adult women, collection of clean-catch midstream specimens has been found to be as reliable bacteriologically as catheterized specimens. [36] A few caveats are worth mentioning: Patients must sit backward on the toilet when collecting the specimen (i.e., facing the wall, which theoretically promotes labial spreading). Of more concern is the fact that these studies excluded patients with vaginitis, urologic abnormalities, pregnancy, and vaginal bleeding. These are the clinical circumstances for which urine is commonly examined in young women visiting the ED. In this "at-risk" population, catheterized urine specimens are preferred.

Urethral catheterization seems a simple task—insertion of one tube into a larger tube. Nonetheless, many difficulties may arise. Patients often remember catheterization as either painful or uneventful and reflective of the operator's expertise, confidence, and gentleness.

Patients are often apprehensive about catheterization. If the clinician shows concern regarding position and exposure, the patient will be reassured. A moment should be spent in making sure a patient is positioned comfortably and appropriately for the procedure. Although adequate exposure may be obtained from a frog-legged position, the use of a table with stirrups (lithotomy position) is ideal, especially for female catheterization.

Anticipation and preparation of all materials necessary for urethral catheterization beforehand is reassuring to the patient. It is frustrating for the health care provider and upsetting to the patient when he or she is told "not to move or touch anything" while a search is made for additional equipment. Most catheterizations are performed using a standard catheterization tray. Often, these trays contain more equipment than is truly needed. This necessitates opening the tray and establishing a sterile field at the bedside, selecting those items that will be needed, and discarding the rest of the equipment. Once the penis or labia has been touched in preparation for the procedure, the touching hand is contaminated and ideally should not be handling any of the sterile equipment. When a standard catheterization tray is not used or is not available, catheterists should go through the anticipated procedure mentally to secure all of the appropriate equipment before actually starting the procedure.

Indications and Contraindications

Urinary catheterization and instrumentation are rarely a primary cause of urinary infection in otherwise healthy patients who urinate normally and carry small amounts of post-void residual urine. As with any procedure, catheterization needs to be limited to those clinical situations in which the benefits outweigh the risks. The following are considered to be indications for urethral catheterization:

1. Acute urinary retention.
2. Urethral or prostatic obstruction leading to compromised renal function.
3. Urine output monitoring in any critically ill or injured patient.
4. Collection of a sterile urine specimen for diagnostic purposes.
5. Intermittent bladder catheterization in patients with neurogenic bladder dysfunction.
6. Urologic study of the lower urinary tract.

Urethral catheterization should be avoided when other less invasive procedures will be as informative. The only absolute contraindication to urethral catheterization is the trauma patient with suspected urethral injury as evidenced by blood at the urethral meatus; an abnormal-feeling or high-riding prostate on rectal examination; or penile, scrotal, or perineal hematoma. These findings dictate the need for

retrograde urethrography to define the integrity of the urethra prior to any attempted urethral catheterization. [37]

Equipment

The equipment listed in Table 56-6 is included in most standard catheterization trays and must be at hand before attempting urethral catheterization. The catheterist should check the list of contents *before* opening the tray, as some trays do not include certain items. For most routine adult in-and-out catheterizations, a 14 Fr red rubber catheter or Foley balloon catheter is adequate. In infants or neonates, a 2 or 5 Fr feeding tube taped in place produces the least amount of urethral trauma. In older boys, a 5 to 12 Fr red rubber catheter or Foley balloon catheter may be used. Table 56-7 lists appropriate-sized catheters and feeding tubes for all ages. A 14 to 18 Fr coudé catheter should be considered after unsuccessful passage of a straight Foley balloon catheter or in any male patient with known enlargement of the prostatic median lobe. If a coudé catheter is not available, a larger 18 to 22 Fr Foley balloon catheter can be tried. In a male patient with a urethral stricture in whom attempts at catheterization with a straight Foley or coudé catheter have failed, passage of filiforms and followers is the next logical step. This requires special

TABLE 56-6 -- Sterile Equipment Required for Urethral and Suprapubic Catheterization

Urethral Catheterization
Foley catheter of appropriate size
Water-soluble lubricant for catheter
10-mL syringe of sterile water for Foley balloon
Sterile drainage bag with tubing
Sterile drapes
Sterile gloves
Povidone-iodine
Sterile gauze pads or cotton balls
Sterile specimen cup with lid

Cloth, paper, or plastic tape (to secure catheter to trunk or leg)
Benzoin (for increasing tape adherence)
Forceps
Suprapubic Catheterization
Foley catheter of appropriate size*
Sterile gloves
Sterile drapes
Sterile drainage bag with tubing
Sterile specimen cup with lid
Cloth, paper, or plastic tape (to secure catheter to trunk or leg)
Benzoin (for increasing tape adherence)
Materials in Cook "Peel-Away" Catheter Set
10-mL syringe
25-ga needle
22-ga needle
1% plain lidocaine
Povidone-iodine swab-sticks
Fenestrated drape
Gauze sponges
Disposable scalpel
Suture with needle
Introducer needle
Curved J-tipped wire guide
Peel-away sheath
Introducer

*Select the sheath size (in catheter set) based on the Foley catheter size. Generally, the sheath is 1 to 2 Fr sizes larger than the catheter (e.g., use an 18 Fr peel-away sheath with a 16 Fr Foley).

TABLE 56-7 -- Pediatric Urethral Catheter Size

Age Group	Pediatric Catheter Size*
Infants	8 Fr feeding tube
1–3 yr	10 Fr feeding tube
4–6 yr	10–12 Fr red rubber catheter (Robinson)
7–12 yr	>12 yr
5–8 Fr feeding tube	14 Fr red rubber catheter (Robinson)

*6 Fr is the smallest balloon catheter and is quite flimsy; would recommend 8 Fr in most cases; 12 Fr is the smallest coudé catheter.

expertise, but it can be learned quickly. If immediate bladder access is required in this circumstance or in any emergency, suprapubic placement of a peel-away sheath and Foley balloon catheter using the Seldinger technique will be needed. [38]

Anatomic Considerations

Female Catheterization

The female urethra is a short (approximately 4 cm), straight tube, usually of wide caliber, lying on top of the vagina. It must be approached between double labia, and the urethral meatus is occasionally hidden and not obvious (in contradistinction to that of most males, except those with hypospadias). If the female patient nervously adducts her legs, successful catheterization is most uncertain.

The female urethral meatus is an anteroposterior slit with rather prominent margins that is situated directly superior to the opening of the vagina and approximately 2.5 cm inferior to the glans clitoris (Fig. 56-12).[39] It is the first of three orifices encountered when examining any female genitalia cephalad to caudad in the lithotomy position. The urethral meatus may be especially difficult to find in the very young infant and the older postmenopausal woman. Anticipation of this and knowledge of the anatomic variances will help to modify any patient discomfort associated with needless catheter tip probing, which is an unsettling experience for both the patient and the catheterist.

Occasionally, the urethral meatus recedes superiorly into the vagina and is not immediately visible, because of either prior surgical procedures or atrophic postmenopausal changes. Anticipation of such cases will allow the examiner to gently advance an index finger into the vagina in the superior midline. The urethral meatus can usually be palpated and often visualized as a soft mound surrounded by a firmer ring of supporting periurethral tissue. Rarely, the meatus will have receded so far superiorly that it cannot be visualized at all, and catheterization must be carried out by palpation alone. From the meatus (if the patient assumes a supine position), the urethra proceeds straight back to slightly downward as it advances into the bladder just behind the symphysis pubis (Fig. 56-13*A*).

In women with a *urethrocele or cystourethrocele,* in whom the urethra or the bladder falls into the vagina, the "normal" urethral course may be more significantly posterior (Fig. 56-13*B* and *C*). The normal anatomic relationships in these situations may be recreated by spreading the index and long fingers and placing them along the superior vaginal wall and gently applying upward support (Fig. 56-13*D* and *E*). This reconstitutes the normal anatomic relationships and permits straight, rapid urethral catheterization.

Because the female urethra is so short, only half the total length of the catheter has to be inserted before it is safe to inflate the Foley balloon.

Figure 56-12 *A,* Anatomy of the female external genitalia. *B,* Uncomplicated catheterization in the female. *(A from Flocks RH, Culp DA: Surgical Urology: A Handbook of Operative Surgery, 4th ed. Chicago, Year Book Medical Publishers, 1975, p 357. B from Brunner LS, Suddarth DS: Lippincott Manual of Nursing Practice. Philadelphia, JB Lippincott, 1974, p 465. Reproduced by permission.)*

Male Catheterization

Because the urethral meatus is usually evident in most males, it may seem a simple matter to insert a urethral catheter. [40] Yet catheterization can be quite difficult. The normal male urethra is approximately 20 cm long from the external urethral meatus to the bladder neck (Fig. 56-14). The posterior prostatic urethra is approximately 3.5 cm long, and the contiguous external sphincter or urogenital diaphragm that encompasses the membranous urethra is 4 cm from the bladder neck. In males, any catheter must be fully inserted to the balloon-inflating side-arm channel before it is safe to inflate the balloon (Fig. 56-15). At the first egress of urine from the catheter, the balloon is just passing through the membranous urethra. The catheter still has greater than or equal to 3 cm to go before clearing the bladder neck. Inflation of the Foley balloon at any point other than full insertion of the catheter may result in iatrogenic urethral injury.

The male urethra is relatively fixed at the level of the urogenital diaphragm and symphysis pubis; traction downward on the penis kinks and promotes urethral folding at the level of the penile suspensory ligament (Fig. 56-16*A*). This creates a level of spurious obstruction. For this reason, the penis should always be held taut and upright during any urethral instrumentation including catheterization (Fig. 56-16*B*). The catheter then needs to make only a single curve rather than a complex S curve as it traverses into the bladder.

General Procedure

As stated previously, urethral catheterization must be done using sterile technique. Both male and female patients have special urethral meatal considerations. In the male, absolute total control of the penile foreskin is paramount to ensuring success. This is true for any male patient, but it is especially important in uncircumcised patients. Prior to establishing a sterile field, the foreskin should be retracted to its fullest extent proximal to the glans penis (Fig. 56-17*A*). A standard 4 × 4 gauze pad is unfolded, then refolded in its longest dimension, and carefully wrapped around the retracted foreskin at the level of the coronal sulcus (Fig. 56-17*B*). This will prevent the tendency for normal anatomic foreskin reduction during catheterization and provide a continuously dry and sterile field. The folded 4 × 4 gauze surrounding the foreskin is secured between the nondominant long and ring fingers when beginning the procedure and is not released until the procedure is completed (Fig. 56-18). This position leaves the nondominant index finger and thumb available for manipulating the catheter. Following catheterization, removal of the 4 × 4 pad and reduction of the penile foreskin to its normal anatomic position will prevent the development of iatrogenic paraphimosis.

In the female patient, the catheterist's nondominant thumb and index finger are used to separate the labia and create exposure of the urethral meatus. Once meatal visualization is achieved, the nondominant hand should not be removed (see Fig. 56-12*B*). This underscores the importance of proper equipment preparation. Access to antiseptic solution, viscous lubricating jelly, the appropriate catheter, and the urinary collection device should be prepared in advance to permit single-handed sterile use.

Following exposure of the urethral meatus in the female and the glans penis and urethral meatus in the male, an antiseptic solution (e.g., povidone-iodine) soaked into cotton balls or oversized cotton-tipped applicators is used to cleanse the exposed meatus and surrounding tissues. This is best done by hand but can also be done using the plastic forceps in the catheterization tray. The cleansing circular motion should begin on the urethral meatus and proceed outward, intentionally moving any debris toward the periphery and thus creating a sterile field.

An appropriately sized catheter (10 Fr is adequate for small children, whereas 14 to 16 Fr is commonly used in adults) that has been lubricated with viscous rather than inspissated lubricating jelly is gently passed by hand, or with the aid of a hemostat or plastic forceps, into the urethra and upward into the bladder. Injection of the male urethra with 5 mL of 2% viscous lidocaine (Anestacon) or a similar anesthetic lubricant administered through a syringe can be helpful for urethral distention and topical anesthesia. Regardless, the

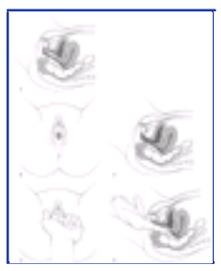

Figure 56-13 Female urethra. *A,* Sagittal view showing normal urethral course into the bladder. *B* and *C,* Cystourethrocele with loss of anterior support (frontal and sagittal views). *D* and *E,* Intravaginal re-creation of normal anatomic relationships with nondominant index and long fingers (frontal and sagittal views).

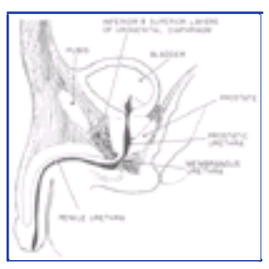

Figure 56-14 Anatomy of the male urethra. *(From Flocks RH, Culp DA: Surgical Urology: A Handbook of Operative Surgery, 4th ed. Chicago, Year Book Medical Publishers, 1975, p 359. Reproduced by permission.)*

patient should be advised of mild urethral discomfort and the potential urge to void. During slow, gentle passage of the catheter, one should be aware of the anatomic considerations discussed previously. A catheter that inadvertently enters the vagina should be discarded.

After passing the catheter "to the hilt" in all male patients, the balloon should be slowly inflated with 10 mL of air or tap water. Sterile water or saline is not required. Most 5-mL balloons will easily accommodate up to 30 to 50 mL of air or water without bursting. Obvious resistance or patient discomfort on balloon inflation should signal potential erroneous urethral positioning and mandates

Figure 56-15 Foley catheter placement. Foley catheter is inserted fully (i.e., to the balloon port) before air or water is injected to inflate the balloon.

reevaluation. If this occurs, the Foley balloon should be immediately deflated and the catheter repositioned or withdrawn slightly, then passed "to the hilt" again prior

to balloon reinflation. If this is unsuccessful a second time, catheter removal and urethral evaluation for a potential obstructive problem or false passage using retrograde urethrography is recommended (see Retrograde Urethrogram later in this chapter).

Following successful catheter passage and Foley balloon inflation, the catheter should be slowly withdrawn until the approximation of the balloon with the bladder neck precludes further withdrawal. The catheter is then connected to either a sterile leg bag or closed-system bedside drainage bag. If the patient will be released with an indwelling Foley catheter, it can be initially connected to a leg bag, which is then comfortably fastened to the lower thigh and upper calf. The patient and family must be instructed regarding proper care of the catheter and drainage device. In most other cases, the catheter may be secured either to the thigh or the lower abdomen (preferred with males) with adhesive tape or simply placed under the knee and left to drain dependently into the bedside drainage bag.

The use of antibiotics before simple catheterization is not warranted. However, patients with known valvular heart disease, suspected urinary tract bacteremia, chronic urinary tract infection, urethral stricture disease, or outlet obstruction associated with infection are considered to be at risk for procedure-induced bacteremia. In these patients, any urinary instrumentation, especially urethral dilation, requires early prophylactic parenteral gram-negative antibiotic coverage before any instrumentation is begun. These patients must also be released with appropriate antibiotic coverage and be scheduled for early urologic follow-up within 1 to 2 days. [41]

Difficulties in Male Catheterization

Phimosis

Physiologic adhesions between the foreskin (prepuce) and glans penis with associated inability to retract the foreskin are normal in children. This condition must be distinguished from phimosis, which is the inability to retract the foreskin proximally over the glans penis due to recurrent inflammation or trauma, which results in gradual scarring of the preputial opening. [42] It is not essential to retract the foreskin in uncircumcised boys at the time of catheterization if the urethral meatus can be visualized. At birth, the foreskin is fully retractable in only 4% of boys. Sufficient foreskin retraction to visualize the urethral meatus is possible in only half of newborn boys. Although the foreskin can be retracted completely in only 20% of 6-month-old boys, 90% of 3-year-old boys have a fully retractable prepuce. By 17 years of age, the foreskin should be physiologically separated and fully retractable in all males unless they have experienced secondary infections or repeated preputial trauma. [43]

The foreskin, especially in diabetics, is susceptible to recurrent infections and inflammation. A scarred, contracted, difficult-to-retract preputial opening may result, leading to phimosis. Phimosis precludes optimal hygiene of the glans penis and coronal sulcus, resulting in an increased risk of bacterial infection, transmission of sexually transmitted diseases (especially human immunodeficiency virus), and the development of penile malignancies. Occasionally the

Figure 56-16 Foley catheter placement. *A,* Holding penis taut and upright prevents urethral folding, lessens external sphincter spasm, and promotes unobstructed catheterization. *B,* Proper uncomplicated male catheterization with upward traction on the penis. *(From Brunner LS, Suddarth DS: Lippincott Manual of Nursing Practice. Philadelphia, JB Lippincott, 1974, p 465. Reproduced by permission.)*

phimotic opening becomes so tight that the meatus cannot be visualized or palpated, even for nonsterile catheterization. If the patient requires catheterization, it will be necessary either to perform a dorsal slit of the foreskin to expose the glans and urethral meatus sufficiently for cleansing and catheterization or to dilate the phimotic opening sufficiently to identify the urethral meatus and blindly pass the catheter. These procedures are discussed at the beginning of the chapter.

Edema of the Foreskin

Patients with penile trauma, paraphimosis, anasarca, or significant lymphatic obstruction from irradiation or cancer may have marked edema of the foreskin, subsequently burying the

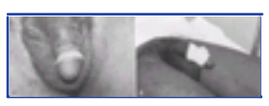

Figure 56-17 Foley catheter placement. It is important to fully retract the foreskin *(A),* especially in the uncircumcised male, so that a folded 4 × 4 gauze sponge can be wrapped around the coronal sulcus *(B)* and prevent foreskin reduction during the procedure. Once the procedure is terminated, the foreskin must be reduced to its normal anatomic position.

urethral meatus and glans penis in several centimeters of boggy foreskin (Fig. 56-19). Because the latter group of patients often require careful fluid monitoring, they may need an indwelling catheter. The clinician's first responsibility is to disclose the proper etiology for the foreskin edema. This will require enough foreskin manipulation to identify the glans penis, coronal sulcus, and urethral meatus, as well as the relationship of the prepuce and preputial opening to these structures, to ensure the absence of foreign body strangulation or paraphimosis (Fig. 56-20).

Two separate methods of visualizing the glans penis and urethral meatus are available to the clinician. [44] The simplest method is to manually compress the swollen foreskin by hand

Figure 56-18 Penile stabilization. To optimize manual dexterity during urethral instrumentation, the prepared penis should be grasped between the nondominant long and ring fingers. This leaves the index finger and thumb available to stabilize the catheter or glans.

or between opposing cold packs in an attempt to reduce the edema fluid (see Fig. 56-5*C* and *D*). Snugly wrapping the distal penis with Elastoplast for 10 minutes may also be helpful. In most mild cases, manual compression is often successful, and no further maneuvers are required. In more severe cases, the foreskin may be 6 cm thick in diameter. In such cases, the least traumatic but unfortunately most cumbersome way to visualize the glans penis and urethral meatus is to use a pediatric-sized vaginal speculum. The outer surfaces of the speculum are lightly lubricated, and the speculum is inserted into the opening of the edematous foreskin. The operator then opens the speculum gently and visualizes the glans penis, similar to viewing the cervix. Commonly, the 2 leaves of the speculum will slip out of the foreskin, making adequate visualization quite difficult. Subsequent meatal cleansing and catheterization performed with plastic instruments is cumbersome and is not recommended. It is much easier to palpate the urethral meatus with the index finger of the nondominant hand, then use the same index finger as a guide to blindly insert the Foley or coudé catheter into the urethra and upward into the bladder.

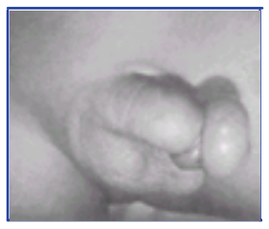

Figure 56-19 Paraphimosis. Tremendous foreskin edema from paraphimosis can make this diagnosis difficult unless the examiner searches diligently for the constricting band.

Meatal Stenosis

The urethral meatus may be either congenitally or secondarily narrowed by scarring, resulting in meatal stenosis. The stenosis may prevent admission of a normal-sized catheter. If the meatus admits a small-caliber tube (i.e., 5 Fr pediatric feeding tube or larger), this may be all that is required for the short term. It should be remembered that the inner diameter of the feeding tube or catheter is the important diameter for urinary drainage. A smaller, single-lumen tube may provide better drainage than a larger, double-lumen Foley balloon catheter.

If a larger-caliber catheter or a Foley balloon catheter is required, meatal dilation or meatotomy may be necessary. Meatal dilation is accomplished by repeatedly inserting larger meatal dilators to a certain end point. This procedure is painful and should be performed with topical meatal anesthesia in conjunction with light IV sedation.

On occasion, a ventral meatotomy may be required in men in whom repeated instrumentation or long-term catheterization has resulted in severe meatal stenosis. Using a 25- or 27-ga needle, infiltration of the ventral midline of the glans from the coronal sulcus to the distal tip of the meatus is performed with 1% plain lidocaine. A straight hemostatic clamp is then gently applied to the anesthetized area, with one jaw inside the meatus and the other on the anesthetized ventral midline of the glans. After the hemostat has been closed and left in place for 3 to 5 minutes, it is opened and removed. The crushed tissue is then cut with straight scissors. Some clinicians place a running hemostatic 4-0 chromic suture through the apex of the meatal incision and carry it out distally on both sides of the incision to prevent restenosis of the meatus. A 16 or 18 Fr Foley catheter left indwelling for 3 to 4 days may serve the same purpose. This procedure is best performed by a urologist.

Urethral Stricture

Urethral obstruction encountered in the anterior or bulbous urethra during catheterization is usually the result of urethral stricture disease. [45] Urethral strictures develop as a result of trauma, infection (especially sexually transmitted diseases), lower urinary tract instrumentation, or long-term indwelling catheter drainage. Strictures may remain asymptomatic for a period of time, but eventually most become symptomatic and cause urethral voiding symptoms, hematuria, or bloody urethral discharge that may or may not be associated with infection. *Manual force should not be used to negotiate or to dilate urethral strictures.* Force merely promotes a vicious cycle of false passages, bleeding, and eventual increased scarring, which makes catheterization more difficult. Inability to negotiate a urethral stricture with a simple straight Foley catheter or coudé catheter leads to consideration of urethral dilation using filiforms and followers.

Filiforms are very narrow, flexible, solid catheters, usually not exceeding 4 Fr in caliber. They are not dilators. Their sole function is to locate and successfully negotiate a strictured urethral segment. Each filiform has a straight or pigtailed (curved) distal end and a proximal female-threaded coupling into which the distal male end of a follower may be threaded. Following generous topical anesthesia with 2% lidocaine (Anestacon), the previously prepared penis is grasped between the nondominant long and ring fingers and stretched upward. The filiform is slowly passed down the urethra in an attempt to negotiate the strictured iris or

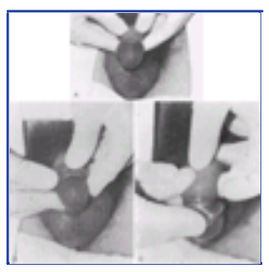

Figure 56-20 Paraphimosis. The appearance of this penis *(A)* could easily be mistaken as being normal. Proximal foreskin retraction reveals a normal coronal sulcus *(B)*. Distal foreskin manipulation *(C)* reveals a phimotic band causing paraphimosis.

narrowed segment of involved urethra (Fig. 56-21*A*). This is all done by "feel" and experience and may require several attempts. It is always best to advance the filiform with the gentlest of pressure.

Filiforms should never be forced through the urethra. Any encountered resistance represents the potential edge of the strictured urethra or fold of urethral mucosa. Undue force applied to the filiform at this point may result in perforation of the urethral mucosa and creation of a false passage along the urethral body or inferiorly into the perineum. If the filiform meets resistance, it should be partially withdrawn, rotated 90°–180°, then gently reinserted. If resistance is met again at the same location, the first filiform may be left in place (to fill up that particular obstructing site) and a second filiform advanced alongside it. A third and fourth filiform may be necessary before one of them successfully navigates the narrowed aperture of the stricture and advances upward through the normal proximal urethra and into the bladder (Fig. 56-21*B–D*). The sine qua non of success is the effortless passage of the filiform through the strictured area without spontaneous discharge of the filiform when it is released. Any amount of filiform return should alert the catheterist that the filiform has not negotiated the stricture and requires replacement. Pigtail filiforms (with a corkscrew-shaped tip) (Fig. 56-21*E*), rather than straight-tipped filiforms, are often easier to advance over an abrupt urethral edge and through the smallest of strictured openings. Once through the stricture, the filiform is advanced until the threaded coupling is near the urethral meatus (Fig. 56-21*F*).

Because filiforms are very pliable, they must be grasped securely. This is ensured by stabilizing the retracted foreskin and penis just proximal to the glans between the long and ring fingers of the nondominant hand as described previously (see Fig. 56-18) and holding the filiform between the nondominant

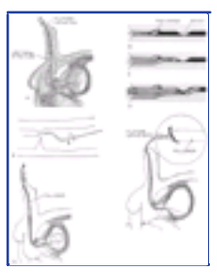

Figure 56-21 *A,* Passage of the filiform past the urethral stricture. *B,* Sequential passage of filiforms, with entry of the first catheter into the false passage. *C,* Advancement of the second filiform to the stricture site. *D,* Passage of the third filiform past the stricture. Following passage of the filiform into the bladder, redundant catheters are removed. *E,* Pigtail filiform passing through the stricture. *F,* Attachment of a follower to the filiform. *G,* Dilation of the stricture with a follower. *(E from Blandy J: Operative Urology. Oxford, Blackwell Scientific Publications, 1978, p 204. B, C, and D from Hill GJ: Outpatient Surgery. Philadelphia, WB Saunders, 1973. Reproduced by permission.)*

index finger and thumb. This allows controlled attachment and subsequent detachment of graduated followers as serial dilation is performed (Fig. 56-21*G*).

A follower of the smallest caliber (usually 8 Fr) should always be selected first; it should be copiously lubricated and threaded onto the filiform. When it has been threaded completely (no threads showing), it is gently advanced without pressure through the upwardly stretched penis and urethra into the bladder (see Fig. 56-21*G*). Stretching the penis upward discourages telescoping of the urethra and subsequent kinking of the filiform and follower (see Fig. 56-16*B*). The follower is advanced into the bladder until there is spontaneous egress of

urine from the follower, guaranteeing successful passage. Note that the side drainage holes of the 8 and 10 Fr followers are quite small and frequently become occluded with lubricating jelly. This may prevent spontaneous urinary drainage from the follower even though it has successfully negotiated the stricture and passed into the bladder. In this circumstance, easy passage of the 8 Fr follower into the bladder and the lack of gross blood or bloody drainage at the filiform-follower coupling when changing follower sizes are indicative of successful passage and dilation rather than creation of a false passage.

Absolute certainty of location of the 8 or 10 Fr follower can be established at any time by irrigating the follower with sterile saline, much like irrigating a Foley catheter. The irrigating fluid will dislodge any obstructing lubricating jelly from the side holes of the follower and will allow egress of urine from the follower. The entire dilating procedure is repeated with sequentially larger followers until one size larger than the proposed retention catheter is successfully introduced. Following completed dilation, the coupled filiform and follower are removed intact, the urethra is relubricated with topical lidocaine (Anestacon) anesthesia, and the previously selected 14 to 16 Fr balloon Foley or coudé catheter is passed to its fullest extent into the bladder. After return of urine from the Foley ensures proper placement, the Foley balloon is inflated and the catheter is withdrawn and left to drain as described previously.

Occasionally, a urethral stricture is so dense and irregular that a filiform and 8 Fr follower may pass successfully, but the density and length of the stricture prevent further dilation. In this circumstance, the indwelling filiform and follower may be taped to the penile shaft for 1 to 2 days to provide adequate bladder drainage and probable softening of the stricture.

The following procedure is used to *tape the follower to the penis*. After wiping excess lubricant from the follower and from the penile glans and shaft, a small amount of tincture of benzoin is applied to the follower and to the unbroken skin of the penile shaft (*not to the glans*). After the benzoin has dried, strips of paper tape 1.25 cm in width are placed longitudinally down the penile shaft without overlapping. The distal ends of each strip of tape are wrapped around the follower much like taping a nasogastric tube to the nose. These longitudinal strips will keep the follower in place. *It is important not to circumferentially wrap these strips around the penis.* If the tape were applied circumferentially, the tape might constrict venous and lymphatic return sufficiently to produce a relative paraphimosis.

Urethral dilation with filiforms and followers should be neither bloody nor excessively uncomfortable for the patient. If the procedure is bloody or uncomfortable or if no urine is returned despite advancement of the follower for at least 24 cm, the clinician should consider that the filiform may not be in the urethra, but instead has created a false passage. In such a situation, retrograde urethrography will define the urethral anatomy. In cases in which urethral instrumentation is unsuccessful for whatever reason, it may be necessary to place a suprapubic peel-away sheath cystostomy tube rather than persist with unsuccessful urethral dilation.

Spasm of the External Urethral Sphincter

The male patient may voluntarily or involuntarily contract the urogenital diaphragm (external sphincter), the striated urethral sphincter at the apex of the prostate. (This is especially true of trauma patients and men with neurogenic bladder dysfunction and pelvic floor spasms.) This produces spurious urethral resistance at approximately 16 cm from the meatus. Because increased abdominal pressure or voluntary perineal contraction causes reflex contraction of the external sphincter, the patient in these situations should be encouraged to lie supine and take slow, deep breaths, consciously trying to relax the perineum and rectum. Plantar flexion of the toes and ankles also aids in relaxation of the pelvic floor. Because the external sphincter is composed of striated muscle and fatigues within a few minutes, gentle but steady pressure exerted on the syringe or the catheter while the previously mentioned maneuvers are undertaken usually results in successful catheterization. If these maneuvers do not result in successful passage of the catheter, the catheterist may be encountering an anatomic abnormality that will require definitive retrograde urethrography before instrumentation.

High Bladder Neck

Occasionally, a patient may have an enlarged intravesical portion of the prostate with a secondary high-riding bladder neck. The tip of the standard Foley catheter may encounter this intravesical portion of the prostate and may not readily pass above it into the bladder. Resistance is usually encountered after the catheter has been passed 16 to 20 cm into the urethra. Slow instillation or injection of 20 to 30 mL of sterile lubricating jelly into the urethra may allow the catheter to slip over the prostate and into the bladder. If this fails, a coudé catheter may be inserted. This catheter has a bend in the tip, and one will almost always be able to maneuver it gently into the bladder (Fig. 56-22*A* and *B*). Passage of the coudé catheter often may be enhanced by having an assistant exert digital compression and flattening of the elongated prostate using a gloved finger in the patient's rectum (Fig. 56-22*C*).

Use of a catheter guide or stylet is *best reserved for the urologist,* as such techniques encourage creation of a urethral false passage (Fig. 56-22*D*). If the coudé catheter cannot be advanced on its own or with the added help of an assistant, a scarred, fixed bladder neck contracture, usually the result of transurethral surgery, may be present. Such a contracture is often very difficult to blindly negotiate. An attempt with filiforms and followers may be warranted. Direct cystoscopic visualization and incision of the bladder neck obstruction by a urologist is usually required.

Catheterization in the Patient with Pelvic Trauma

The patient with pelvic trauma or a straddle injury presents special problems in urinary management. The patient may be in shock from associated injuries. Accurate minute-to-minute monitoring of urinary output requiring bladder catheterization may be required in the initial resuscitation. Furthermore, radiographic evaluation to define the extent of lower urinary tract injury may require a retrograde cystogram, which necessitates urethral catheterization. Prior to urethral catheterization in all trauma patients, the penis should be examined for evidence of blood at the urethral meatus, the absolute marker of urethral injury. [37] Blood at the urethral meatus, a "high-riding" or abnormal feeling prostate on rectal examination, and penile, scrotal, or perineal ecchymoses are all hallmarks of potential urethral injury and represent contraindications to empiric urethral catheterization without an antecedent normal retrograde urethrogram. [37]

The hazard of injudicious urethral catheterization in the pelvic trauma patient is the potential worsening of an already

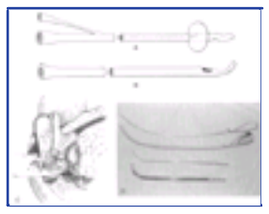

Figure 56-22 *A,* Self-retaining Foley-type catheter. *B,* Coudé catheter (also available in a non-self-retaining model). *C,* Superior pressure on the prostate may facilitate catheter passage. *D*, Catheter guide or stylet should be used only by a urologist.

existing, less serious urethral injury that is often associated with such trauma. Pelvic fractures most often impart injury to the prostatomembranous urethra just above the urogenital diaphragm. The shearing pelvic fracture fragments transect the urethra and puboprostatic ligaments, thereby displacing the prostate superiorly from its normal anatomic attachment to the posterior surface of the public bone. A partial or complete urethral injury may occur. An appropriately recognized and treated partial urethral disruption may heal with little or no scarring. However, a complete urethral disruption requires surgical repair and usually results in some degree of postoperative urethral stricture and, occasionally, urinary incontinence and impotence, all of which portend significant morbidity. The danger of injudicious urethral catheterization in this situation is the potential conversion of a partial injury into a complete urethral injury, with its associated complications. Retrograde urethrography is the diagnostic procedure of choice in any suspected urethral injury (see Retrograde Urethrogram later in this chapter).

In the event that contrast material flows easily from the urethra into the bladder without extravasation, complete urethral integrity is ensured, and an attempt to pass a 14 or 16 Fr Foley catheter should be made. If *any* urethral resistance is encountered other than that normally expected at the voluntary external sphincter (urogenital diaphragm) in a conscious, anxious patient, the catheterization should be immediately aborted and a urologist consulted. If passage is successful, the catheter balloon should be inflated and the catheter withdrawn until it approximates the bladder neck. At this point, it is left to dependent drainage, and very careful attention is

paid to the initial bladder effluent. Any colored urine other than clear or yellow is considered gross hematuria and mandates evaluation of the bladder and upper urinary tract to disclose the source.

If the retrograde urethrogram shows urethral extravasation *AND* passage of contrast into the bladder, a partial urethral injury has been identified. One gentle attempt at passage of a 12 to 14 Fr Foley or coudé catheter may be attempted, depending on the experience and confidence of the catheterist. Once again, any degree of resistance dictates termination of the procedure and urologic consultation.

In the patient without external sphincter spasm in whom the retrograde urethrogram shows evidence of urethral extravasation without any contrast filling the bladder, a complete urethral injury has been identified. Upon identification, immediate urologic consultation is indicated for placement of a suprapubic catheter and subsequent surgical repair of the complete urethral injury. One caveat: if more than 50 to 60 mL of contrast is used at any time for urethrography and more than gentle pressure is exerted during urethral contrast instillation, it is possible to create objective penile venous intravasation of contrast, which is benign. This intravasation of contrast may simulate urethral extravasation and produce a spurious examination result similar to urethral injury. The distinguishing feature with intravasation is that any subsequent film (i.e., post-void film) will disclose immediate clearing of the iatrogenic penile venogram, whereas urethral extravasation will persist indefinitely.

Suprapubic placement of a peel-away sheath and Foley catheter as an alternative to urethral catheterization for the trauma patient is covered elsewhere in this chapter.

Complications of Urethral Catheterization

Although urethral catheterization performed by skilled personnel in appropriate circumstances has an acceptable complication rate, untoward sequelae of catheterization are not unusual.

The frequency of bacteriuria after a single catheterization in a healthy outpatient population is probably less than 1%. [41] However, in hospitalized, elderly, debilitated, or postpartum patients, the rate may be considerably higher. Urinary catheterization is the leading cause of nosocomial urinary tract infections. The mortality in patients with nosocomial urinary tract infection is approximately three times that in patients not acquiring infection. [46] Of patients catheterized with a closed system for 2 to 7 days, 8% to 10% will have significant bacteriuria once the catheter is removed. [47] Patients with catheters indwelling more than 10 days almost always acquire an infection. Infection from the urethra and the bladder may disseminate to cause epididymitis, pyelonephritis, and bacteremia. Although the use of a povidone-iodine lubricating gel has been shown to reduce the inoculation of bacteria into the bladder at the time of catheterization, [48] further study will be needed to determine if this antiseptic lubricant actually reduces infectious sequelae.

With long-term catheterization, bacteriuria is inevitable. Although episodes of high temperature (=38.8°C) due to urinary tract infection in patients with long-term catheterizations are rare (2 per 1000 patient-days), these episodes can be associated with bacteremia and death. [49] Use of a condom catheter, adult diapers, and intermittent self-catheterization [50] represent noninvasive alternatives to long-term Foley catheterization in nonambulatory incontinent men and women. Other rare complications of long-term indwelling urethral catheterization include bladder stones, recurring bladder spasm, periurethral abscesses, bladder perforation, [51] and urethral erosion. [52]

In addition, complications may occur during the act of catheterization. False passages may be established in any area of the urethra when force is exerted on the catheter. In an uncircumcised patient, negligence in reducing the retracted foreskin to its normal anatomic position after urethral catheterization or instrumentation may lead to painful paraphimosis and associated complications.

Leaving a catheter indwelling too long or using a larger catheter than is needed promotes poor drainage of the periurethral glands, urethritis, and periurethral abscesses, all of which may lead to urethral stricture disease. Likewise, concretions may form around the catheter balloon and lead to the formation of bladder stones, many of which require operative removal.

The use of silicone rather than latex catheters for postoperative urinary drainage in adult males undergoing cardiac surgery has been shown to reduce the incidence of subsequent urethral stricture formation. [53] Although patients with indwelling latex catheters were catheterized less than 48 hours, a 2% incidence of urethral stricture was still noted at 1 year and a 5% incidence at 2 years. None of the patients with indwelling silicone catheters developed a stricture.

Hematuria has long been considered to be common immediately following even atraumatic catheterization. Although Sklar and colleagues found a small increase in urinary red blood cell (RBC) count with catheterization, only 1 in 47 patients had an increase of >4 RBCs per high-power field (HPF) attributable to the procedure. [54] They suggest that >4 RBCs per HPF following catheterization is unlikely to be due to the procedure and is, in fact, evidence of preexisting hematuria, which must be explained.

Undesirably retained urethral catheters are an uncommon but frustrating problem. Catheters may be retained because of balloons that do not deflate (see following section) or very rarely because of a knot that has spontaneously developed in the catheter (very rare). Catheter knotting has been associated with the insertion of a highly flexible catheter far into the bladder. [55] A guide wire passed up the catheter may be successful to manipulate some knots free, but urethral dilation with progressively larger catheters adjacent to the retained catheter may be needed to permit urethral passage of the knot.

REMOVING THE NON-DEFLATING CATHETER

The self-retaining Foley balloon-type catheter obviates the need for cumbersome taping or suturing of the catheter to keep it in place. Occasionally, however, an indwelling catheter balloon does not deflate. Needless to say, this problem has challenged and frustrated many clinicians and has produced a number of solutions. The usual cause of the nondeflating catheter balloon is the malfunction of the flap-type valve in the balloon lumen of the catheter, which normally allows fluid to enter the balloon of the catheter but prevents passive egress (Fig. 56-23).[56] The ideal solution is one that resolves the problem—deflating the balloon—without creating another problem (i.e., unnecessary bladder irritation or balloon fragmentation). Of the methods recommended to decompress nondeflating catheter balloons, the only technique that approaches the ideal directly attacks this flap valve deformity. Other methods of deflation are effective but require more creativity and dexterity on the part of the catheterist.

Techniques

One method of balloon deflation consists of simply overstretching the balloon with air or water to the point of rupture. Up to 200 mL of fluid can be injected before a 5-mL balloon will rupture.[56] [57] Adding volume to the empty bladder may not be a problem. Unfortunately, this technique may produce unacceptably painful bladder distention for the patient whose catheter is blocked and whose bladder is either secondarily

Figure 56-23 A flap-like defect in the inflating channel of a balloon catheter that is being raised by a wire stylet passed down the inflating channel to deflate the balloon. *(From Eichenberg HA, Amin M, Clark J: Nondeflating Foley catheters. Int Urol Nephrol 8:171, 1976. Reproduced by permission.)*

contracted due to chronic infection or neurogenic bladder dysfunction or distended to the point of maximum filling. An even more compelling reason not to use this technique is the disconcerting frequency of balloon fragmentation and subsequent foreign-body bladder stone formation. In an experimental study of 100 catheters (50 of which were overdistended with water and 50 of which were overdistended with air), all 100 catheter balloons ruptured into fragments. [56] Cystoscopic inspection of the bladder and removal of any fragments will be required to prevent bladder stone formation if this method of balloon deflation is selected.

A second method of balloon deflation involves injecting an erosive substance into the balloon port. This causes the balloon to deflate after part of the balloon wall has been eroded. Organic compounds that attack the latex polymers are often used. Ether, acetone, mineral oil, and even petrolatum ointment have been used. In general, the more volatile the substance, the more rapidly it ruptures the balloon. Rupture of the balloon may be partly a result of the rapid expansion that some of these volatile substances—especially ether—undergo at body temperature. Ether was reported to rupture 58 of 60 catheter balloons within 2 minutes of injection into the balloon port. Unfortunately, in 56 of the catheters, a free fragment of the balloon was created. Mineral oil, which works more slowly, was associated with fragment production in 95 of 100 catheters tested.[56] When released into the bladder, organic substances often produce a symptomatic chemical cystitis. Use of these substances is discouraged.

A third method of deflating the balloon is to puncture it with a needle. With gentle traction, the balloon is located in the urethra or drawn against the bladder neck and is punctured with a thin 25- or 27-ga spinal needle. This needle may be directed suprapubically (transvesically), transvaginally, transperineally, or transrectally. The procedure may be done either blindly[58] or with the aid of ultrasound. In women, a spinal needle may be gently introduced transurethrally alongside the catheter. Fragmentation during puncture can occur, but it is much rarer than in the two techniques described previously.

The easiest way to deflate a nondeflating balloon is to attack the inflate-deflate channel that normally prevents the passive egress of inflating fluid. Patients may be sent to the ED in the late evening or early morning hours after their catheters have been progressively shortened by ingenious health care providers during the day. Cutting the catheter may result in rapid deflation if the valve-flap defect happens to be present in the part of the catheter that is cut off. This is an uncommon occurrence. A shorter catheter with a more proximal valve-flap defect can often be left for 24 hours with ongoing slow balloon deflation, but this maneuver leaves the problem of managing an unconnected catheter and an incontinent patient. Devising a waterproof and aseptic method of collecting urine from the shortened Foley catheter may require use of a ureteral catheter drainage bag or other ingenious approaches.

When presented with this situation, it is often best to insert a thin, rigid wire into the balloon-port lumen in an effort to deflate the valve-flap defect sufficiently and promote the escape of fluid from the balloon. A stainless steel wire suture of 3-0 or 4-0 gauge is the thinnest suitable material. The wire stylet from an angiographic catheter, guide wires from ureteral catheters, and very small, well-lubricated ureteral catheters themselves have all been reported to be successful. When a ureteral catheter guide wire was used in one series, 34 of 39 balloons were deflated without fragmentation. In the five unsuccessful cases, needle puncture of the balloon was required and was successful.[58]

One approach is to use a stepwise series of maneuvers. If the balloon does not deflate, remove the syringe adapter plug from the balloon-inflating channel. This rules out a malfunction of the adapter. If the balloon water does not escape, next insert an angiographic catheter stylet into the balloon-inflating channel and rotate it. Usually, the water from the balloon flows out along the wire. If it does not, place the catheter on traction and attempt to locate the balloon by palpation either perineally, transvaginally, or transrectally. If this is successful, a 25- to 27-ga spinal needle under local anesthesia is used to blindly puncture the balloon and then remove the catheter. If localization is unsuccessful, multiple blind passes with the 27-ga needle can be attempted; this is usually successful in decompressing the balloon and removing the catheter.[59] If the patient requires a permanent indwelling catheter, one may be replaced immediately. Concomitant inadvertent needle punctures of the rectum are usually of no clinical significance.

Once a malfunctioning balloon has been deflated, it is mandatory to carefully inspect the balloon itself for missing fragments. If a piece of the balloon is missing, it is necessary to arrange for subsequent cystoscopy to look for and remove the fragment. Unfortunately, pretesting Foley catheter balloons by trial inflation and deflation before insertion does not eliminate the potential for a nondeflating Foley catheter balloon.

SUPRAPUBIC ASPIRATION OF THE BLADDER

One problem of interpreting voided urine samples is that the urine from the bladder passes through a progressively more contaminated urethral conduit. In the female, the perineum is a culture medium where bacteria are seemingly eager to be swept along into the sterile collection cup and onto the agar plate. To avoid the dilemma of interpretation, clinicians have devised maneuvers to minimize the presence of contaminating organisms. Male patients are instructed to retract the foreskin, cleanse the meatus, discard the first portion of urine, and catch the midstream part of the voided specimen. Female patients are asked to perform even more difficult maneuvers to avoid bacterial contamination: sit backward on the commode facing the wall, hold the labia apart with one hand, cleanse the periurethral skin blindly with the other, then reach for the cup, initiate voiding, and catch the midstream urine—all while holding the labia apart and maintaining the precarious position on the commode. Some experts[60] have women void in the lithotomy position after an assistant retracts the labia, cleanses the perineum, and then catches the midstream urine.

In standard transurethral bladder catheterization, even under ideal circumstances, the procedure is often uncomfortable. The catheter must traverse the distal contaminated urethra and may infrequently introduce contaminating bacteria into the specimen and into the bladder of the patient, resulting in infection, primarily in patients who don't empty their bladder with normal voiding.

Suprapubic aspiration of the bladder, first reported as a method of collecting urine for bacteriologic study in 1956, [61] offers the clinician a relatively simple means of obtaining uncontaminated bladder urine. Urethral contamination is successfully avoided, and positive results always represent

true bacteriuria. The one caveat is that the bladder must be full to avoid multiple painful needle sticks, a clinical situation that may be difficult to discern in a sick child.

Indications

In the neonate or the young child, suprapubic aspiration or urethral catheterization can provide the clinician with a sample that is reliable for bacteriologic interpretation.[61] [62] [63] Although disconcerting to some parents (they may wish to leave the room or look away during the procedure), suprapubic aspiration is not a dangerous procedure, and the sensitivity of urinalysis of this urine for bacteriuria approaches 100%. However, for children 2 years or older, urine can generally be more easily collected by urethral catheterization.

For adult patients, the indications for suprapubic aspiration are more limited, because these patients usually can cooperate with the clinician. Men with condom catheters or phimosis, however, may require suprapubic aspiration to minimize urethral contamination. Aspirated cultures, rather than catheterized specimens, may help rule out contamination in patients with asymptomatic bacteriuria on routine urine collection. In infections caused by organisms that in other circumstances are often discounted as contaminants (e.g., *Staphylococcus epidermidis* or *Candida albicans*), suprapubic aspiration or a catheterized specimen is required to confirm the presence of such pathogens.

In patients in whom the possibility of infravesical infection is a concern (e.g., patients with chronic infections of the urethra or the periurethral glands), suprapubic aspiration may help localize a bladder from a urethral source.

Figure 56-24 *A,* For a suprapubic bladder tap, the infant is restrained and placed in a frog-legged position. *B,* A 22-ga needle punctures the abdominal wall in the midline approximately 1 to 2 cm cephalad to the superior border of the pubic bone. The syringe is perpendicular to the plane of the abdominal wall (usually 10°–20° from the true vertical). The bladder is an abdominal organ in infants, and placing the needle too close to the pubic bone or angling toward the feet may cause the needle to miss the bladder. Localizing the bladder with bedside ultrasound facilitates this procedure.

Procedure

The clinician must first locate the bladder. A full, palpable, or percussible bladder should be readily apparent, but this can be difficult to discern in all but the thinnest patients. If there is any question about the location or the amount of bladder urine, a quick ultrasound examination is informative. The point of entry in the skin should be 1 to 2 cm above the superior edge of the symphysis pubis. The syringe and needle are passed perpendicular to the abdominal wall toward the bladder, usually a 10° to 20° angle from the true vertical, somewhat *cephalad* in *children* (Fig. 56-24) and somewhat *caudad* in *adults* (Fig. 56-25). Note that the bladder of a newborn is an abdominal organ and that it will be missed if the needle is inserted too close to the pubis or is angled toward the feet.

The child is placed supine and is restrained with the legs in a frog-legged position. Once the prepared skin has been draped and the point of entry has been chosen, a skin wheal of local anesthesia is raised to reduce discomfort. When the skin has been anesthetized, a longer, larger-caliber needle (usually 22-ga, 3.75 to 8.75 cm in length) is advanced in the midline through the skin and quickly into the bladder. The editors prefer to advance the needle attached to a syringe, with active aspiration during advancement. As soon as the bladder is entered, urine appears in the syringe. A short needle is adequate for virtually all pediatric patients. After the urine is collected, the syringe and needle are withdrawn. Microscopic hematuria always follows the procedure but gross hematuria is uncommon. A bandage may be placed over the puncture site. If urine is not obtained, the needle is not removed but withdrawn to a subcutaneous position and redirected at a

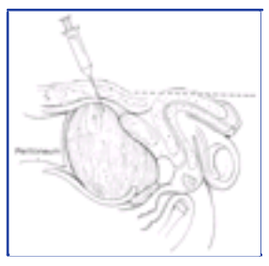

Figure 56-25 The peritoneum is pushed cephalad by the filled bladder during suprapubic aspiration in an adult. The needle is directed slightly caudad.

different angle. Often a child may spontaneously start to void following any type of invasive stimulus (e.g., bladder irritation by a probing needle, venipuncture, or lumbar puncture). Hence, preparation to collect a spontaneously voided specimen is recommended, should that option arise. This should be anticipated before beginning blood or spinal fluid collection during the bacteremic workup of the febrile neonate.

In most patients, an acceptable urine sample can be obtained with the first needle pass. If the needle points too caudad in an effort to avoid entering the peritoneum, it is possible to enter the retropubic space, skimming the bladder muscle and never penetrating the bladder mucosa.

Complications

Stamey has performed several thousand aspirations without complications. [63] Bacteremia does not result from this procedure. [64] Bowel penetration has occurred in children with distended abdomens from gastrointestinal disturbances. [65] The combination of gaseous bowel distention and relative hypovolemia may displace and flatten the relatively empty bladder against the pelvic floor. Even when the large bowel has been penetrated, patients recover uneventfully. Simple penetration of the bowel with a needle is considered an innocuous event and requires no specific treatment.

PERCUTANEOUS SUPRAPUBIC CYSTOSTOMY

Background

Although suprapubic cystostomy was described as early as 4 centuries ago, the safety of the procedure was first demonstrated by Garson and Peterson in 1888. The first modern method was the Campbell trocar set, described in 1951. [66] [67] Campbell used a sharp trocar passing through a sheath. The sheath had one longitudinal portion of its wall missing to permit a balloon-type Foley catheter to be passed into the bladder. The Campbell trocar is a large-diameter instrument, accepting up to a 20 Fr catheter. Newer technologies have made its use obsolete in the ED.

The development of punch thoracostomy tube sets suggested their use as modified cystostomy tubes. This led to the invention of medium-caliber cystostomy tubes, which were easier to insert than the Campbell trocar but provided more satisfactory drainage than adaptations of IV infusion sets. [68] [69] [70] Ingram's trocar catheter is perhaps the best known of these tubes. It has three lumina: one for inflating the retention balloon and the other two for drainage or irrigation. The Ingram catheter is available in a 12 or 16 Fr size. The Stamey suprapubic catheter is another variation of this type, but it uses a four-wing Malecot-type retention device rather than a preferred user-friendly inflatable balloon.

Perhaps the most widely known and frequently used trocar-type cystostomy tube is the Cystocath. [71] It is available in 8 and 12 Fr sizes. The latter is more commonly used for adult patients. The Cystocath is packaged as a self-contained set supplying virtually everything needed for insertion. The device is easy to insert and may be satisfactory for relatively long periods of trouble-free use if the patient is given conscientious nursing care.

The major difficulty with cystostomy tubes of all designs has been securing them to the patient's skin. Those with retention balloons, such as the regular Foley urethral catheter or the Ingram catheter, are most secure and only need tape to secure them to the anterior abdominal wall. Virtually all other systems depend on tape or skin adhesive to hold either the tube or the appliance in place. They become an annoyance to both the patient and the care provider.

The most user-friendly device for suprapubic bladder access is the Cook peel-away sheath unit. [38] It uses the Seldinger (guide wire) technique to gain bladder access and allows suprapubic placement of a Foley balloon catheter for definitive bladder drainage. This device is recommended for ED use over other suprapubic bladder access approaches and is discussed in this section.

Indications

In general, any patient who would require a urethral catheter but in whom a catheter cannot be passed is a candidate for a suprapubic cystostomy tube. In emergency situations, the majority of these patients are men with urethral stricture or complex prostatic disease and trauma patients with urethral disruption. Depending on the experience of the catheterist, dilation can usually be performed in patients with urethral strictures using filiforms and followers. If there is any difficulty with urethral instrumentation, a suprapubic cystostomy tube is prudent and prevents further urethral injury. Complete urethral transection associated with a pelvic fracture is an absolute indication for emergent suprapubic cystostomy. Many affected patients need laparotomy because of associated injuries, and a large suprapubic catheter can be placed intraoperatively. However, if the patient does not require laparotomy, a percutaneously placed Foley catheter allows urologic surgery to be done electively after the patient's condition has stabilized clinically.

Patients with lower genitourinary infection deserve special care before instituting any type of urethral instrumentation. The risk of inciting an episode of gram-negative bacteremia with urethral dilation must be considered, and

appropriate IV gram-negative antibiotic coverage started before the patient is instrumented. Foley catheter drainage is the first choice and suprapubic drainage is an option in patients with acute prostatitis or epididymitis who require bladder drainage. Ideally, a suprapubic catheter allows both bladder drainage and unobstructed drainage of prostatic, seminal vesicle, and urethral secretions but requires an invasive procedure with its associated risks.

Neurologically disabled patients (e.g., quadriplegics or paraplegics) or patients with any type of neurogenic bladder dysfunction who have been successfully maintained on a program of intermittent self-catheterization occasionally have difficulty with urethral catheterization. In these patients, especially those with high spinal cord lesions, suprapubic needle aspiration or suprapubic cystostomy can be a rapidly effective method of relieving autonomic hyperreflexia associated with acute bladder distention. Bladder decompression in the dysreflexic, profusely perspiring, hypertensive quadriplegic in sympathetic crisis provides dramatic symptom resolution, whether by suprapubic bladder decompression or Foley catheter placement.

Suprapubic catheterization is *not* recommended as first-line treatment for the patient who is voiding poorly from lower urinary tract prostatic obstruction. Such patients, although symptomatic, are better off with intermittent self-catheterization or an indwelling Foley catheter if they are in retention or have chronically infected urine. Young women with psychosocial or emotional neurogenic bladder dysfunction are best managed by intermittent self-catheterization. In all such cases, clinical judgment will dictate the most appropriate form of treatment and whether concomitant antibiotic therapy is required.

Contraindications

Because placement of a suprapubic tube involves some risk, patient selection is important. The procedure should not be performed in a patient whose bladder is not definable. Although no absolute reported minimum bladder volume has ever been established, there must be enough urine in the bladder to allow the needle to fully penetrate the bladder dome without immediately exiting through the base. There must also be enough urine in the bladder to displace the bowel away from the anterosuperior surface of the bladder and the entrance of the needle. Ultrasound may be helpful in defining bladder anatomy.

Individuals who have a history of previous lower abdominal surgery, intraperitoneal surgery, or irradiation may have developed adhesions or adherence of the bowel to the anterior bladder wall. They are potentially at greater risk for bowel injury during percutaneous suprapubic cystostomy tube placement than those without previous abdominal surgery. Blind suprapubic cystostomy tube placement in these patients should be avoided. The absence of any of these risk factors does not totally exclude the risks of bowel or intraperitoneal injury, but it reduces them significantly.

Patients with bleeding diatheses are at greater risk for postinsertion bleeding, either into the bladder or into the retropubic space, than their normal counterparts.

Equipment

The items of equipment needed for Cook's peel-away sheath placement are listed in Table 56-6 .

Procedure

The following comments describe the placement of the Cook peel-away sheath. With modifications, these guidelines are adaptable for any type of suprapubic catheter placement.

Preparing the Patient

If necessary, the lower abdomen is shaved. Povidone-iodine skin preparation or another suitable bactericide is used to cleanse the area. The extra liquid is removed, and the skin is allowed to dry. A 6-mL syringe is filled with 1% lidocaine, and a 22-ga, 7.75-cm spinal needle is attached. A skin wheal is raised in the proposed site (approximately 2 to 3 cm above the pubic symphysis), and the subcutaneous tissue and rectus abdominis muscle fascia is infiltrated at a 10°–20° angle toward the pelvis.

The bladder is located by advancing the needle in the prescribed direction while aspirating the syringe. Urine is easily aspirated when the bladder is entered (Fig. 56-26*A*).

Placing the Tube

Once the bladder has been located, the syringe is removed from the needle and a guide wire is advanced through the needle into the bladder (Fig. 56-26*B*). The needle is withdrawn, leaving only the guide wire traversing the anterior abdominal wall and positioned inside the bladder. A No. 15 scalpel blade is used to make a stab incision directly posterior to the wire through the skin, subcutaneous tissue, and superficial anterior abdominal wall fascia. The peel-away sheath and indwelling fascial dilator are then passed together over the wire into the bladder (Fig. 56-26*C*). The guide wire and fascial dilator are removed, leaving only the peel-away sheath inside the bladder (Fig. 56-26*D*). A preselected Foley balloon catheter is then passed through the indwelling intravesical sheath into the bladder (Fig. 56-26*E*). Urine is aspirated to confirm proper placement. The Foley balloon is inflated with a minimum of 10 mL of air, water, or saline (Fig. 56-26*F*). The peel-away sheath is withdrawn from the bladder and anterior abdominal wall and is literally peeled away from the catheter, leaving only the indwelling suprapubic Foley catheter (Fig. 56-26*G*). The catheter is withdrawn slowly until the inflated balloon approximates the cystostomy site (Fig. 56-26*H*). The catheter is connected to a drainage bag, and the wound is dressed with 4 × 4 gauze pads to complete the procedure.

Complications

A wide variety of complications specific to each procedure have been reported, which serve as reminders that suprapubic cystostomy is not innocuous. Occasionally, despite the best intentions, the suprapubic tube or catheter cannot be positioned or maintained successfully without untoward sequelae (Table 56-8).

The most serious complications involve perforation of the peritoneum or the intraperitoneal contents. Any condition that might fix the anterior peritoneum so that the filled bladder cannot lift the peritoneum cephalad may result in either transperitoneal bladder puncture or possible perforation of small or large bowel. [73] [74] [75] Although finding the bladder using a small-gauge scout needle may help reduce bowel injury, even in the most apparently successful of bladder punctures, a complication may result.

Figure 56-26 Suprapubic cystostomy with the Cook peel-away sheath introducer. *A,* Bladder is entered with a syringe and needle. Location is confirmed by the aspiration of urine. *B,* Syringe is removed and the guide wire is passed through the needle into the bladder. *C,* The needle is removed, then the dilator and peel-away sheath are passed over the wire into the bladder. A small stab wound in the anterior abdominal fascia may be required to accommodate the dilator and sheath. *D,* The dilator and wire are removed, leaving only the sheath inside the bladder. *E,* The preselected Foley balloon catheter is passed through the sheath into the bladder. Urine is aspirated to confirm location. *F,* The balloon is inflated with a minimum of 10 mL of air, saline, or water. A 5-mL balloon will accommodate 10 mL easily and make accidental catheter distraction less likely. *G,* The sheath is removed from the bladder, anterior abdominal wall, and cutaneous entry site, and is then literally peeled away from the indwelling catheter. *H,* The catheter is withdrawn until a snug fit is ensured at the cystostomy site.

TABLE 56-8 -- Reported Complications of Suprapubic Cystostomy

Bowel perforation
Intraperitoneal extravasation (without a prior history of surgery)
Extraperitoneal extravasation
Infection of space of Retzius
Ureteral catheterization
Obstruction of tubing by blood, mucus, or kinking
Tubing comes out
Hematuria

The cystostomy tube or catheter that merely traverses the peritoneum may produce a mild ileus, serve as a route for peritoneal infection, or drain the bladder contents into the peritoneal cavity. The last situation would be expected if a Cystocath, rather than a peel-away sheath, were used, and one of the extra holes of the Cystocath tubing opened into the peritoneal cavity. Through-and-through bladder penetration with associated rectal, vaginal, or uterine injury has been reported, although the consistent use of small-gauge bladder locator needles and the judicious advancement of fascial dilators should reduce the incidence.

Occasionally the clinician is tempted to proceed with suprapubic cystostomy when the bladder is not palpable and has not been located with a syringe and needle. Injury of adjacent organs is much more frequent in these circumstances. If clinicians remind themselves that the bladder eventually refills, they will find waiting much more tolerable. If faced with an emergency, *ultrasound guidance may be helpful for determining bladder size and location*.

Infection may occur at the suprapubic cystostomy skin site or anywhere along the course of the catheter. [76] Use of antimicrobial ointment daily after cleaning the catheter entry site may reduce purulence around the tube. However, topical care does not prevent eventual deep space or bladder infection from the presence of a foreign body. Deeper tissue infections may result from extravasated infected urine or from a superficial infection spreading along the tube to a hematoma at the bladder or fascial level. Parenteral antibiotics may be required. Open drainage is rarely needed unless a loculated abscess has formed.

Hematuria is rarely more than a transient problem. [77] After suprapubic Foley catheter insertion, bladder irrigation may occasionally be required to clear the hematuria. Transient Toomey syringe aspiration may be needed to evacuate clots.

EMERGENCY LOWER GENITOURINARY RADIOLOGIC PROCEDURES

Trauma to the urinary tract accounts for about 10% of all injuries seen in EDs. Although the signs of genitourinary trauma in general can be quite subtle, lower urinary tract injury can often be quickly identified and thoroughly evaluated radiographically in the ED. Radiologic imaging of the upper urinary tract is generally a less urgent matter and can usually be done in the radiology suite or, when important for emergency operative decision-making, as a single shot intravenous pyelogram (IVP) in the operating room. Hence, this section does not discuss the role or technique of IVP in detail. Note that the timing of any radiologic evaluation can be challenging to the emergency clinician, especially when faced with a critically ill multiple trauma patient. The priority and extent of such an evaluation, of course, must be determined by the trauma team of clinicians involved in each resuscitation.

Indications for Evaluation

The urinary tract includes the kidneys, ureters, bladder, urethra, and external genitalia. Approximately 8% to 10% of *blunt* abdominal trauma is associated with injuries to the urinary tract.[78] In one large series,[79] 7% of gunshot wounds and 6% of stab wounds to the abdomen resulted in *penetrating* wounds to the kidney. For injury identification purposes, the genitourinary system is best divided into lower urinary tract (i.e., urethra and bladder), upper urinary tract (i.e., kidneys and ureter), and external genitalia (i.e., penis, scrotum, and testes or vagina, labia majora, and labia minora). Each of these subdivisions has its own markers for potential injury. These markers are addressed during the resuscitation phase of trauma care and during secondary injury survey when the abdomen, pelvis, external genitalia, vaginal vault, and rectum are systematically examined.

The markers for lower urinary tract injury are blood at the urethral meatus, abnormal position of the prostate on rectal examination (in men), and gross hematuria. [80] Perineal ecchymosis and scrotal hematoma also represent potential lower urinary tract injury, but these findings are usually seen later in the patient's course rather than acutely in the ED. Gross hematuria or microscopic hematuria (=3 to 5 RBCs per HPF-spun specimen) *in conjunction with any history of shock* (systolic blood pressure =90 mm Hg) *in the field or in the ED following blunt trauma* are markers of potential upper urinary tract injury in any adult. [81] In children, a meta analysis has defined 50 RBC/hpf as the quantity below which imaging may be omitted and no significant injuries missed. [82]

In genitourinary trauma, the lower urinary tract is always evaluated before the upper urinary tract. Retrograde urethrography and retrograde cystography are the diagnostic procedures of choice to evaluate potential injury to the lower urinary tract. These studies must be carried out in the proper sequence and in a retrograde fashion to avoid missing potential injuries. *Retrograde* refers to the technique of instilling contrast retrograde through the urethra or by gravity filling of the bladder. It must be distinguished from antegrade filling, in which IV contrast for IVP or abdominal computed tomography (CT) is excreted from the kidneys and allowed to fill the bladder passively over time.

Contrast-enhanced CT is the diagnostic examination of choice for suspected renal trauma. It provides greater resolution and sensitivity than bolus infusion IVP with nephrotomography and has the advantage of evaluating other intra-abdominal structures as well. [83] However, it is expensive, and in some hospitals it is not readily available on a 24-hour basis. A reasonable course of action under these circumstances would be to initiate the upper urinary tract investigation with bolus-infusion IVP with nephrotomography and to investigate further with contrast-enhanced CT if an ill-defined or poorly visualizing kidney is the result of the initial study. Contrast-enhanced CT should be performed initially if thoracic or intra-abdominal injuries are present or suspected, or if there is concern about renal pedicle injury.

Gross Hematuria

Gross hematuria is indicated by any color of urine other than clear or yellow. It is an *absolute* marker for urinary tract injury and an indication for diagnostic evaluation. The resuscitating clinician must be responsible for observing the initial bladder effluent following Foley catheter insertion. Vigorous fluid resuscitation may quickly clear initial gross hematuria and eliminate the only marker for potential injury. When gross hematuria is encountered as an injury marker, the bladder and kidneys are thought of as potential sources for the hematuria. In most cases, gross hematuria in association with a pelvic fracture will implicate the bladder as the most likely source of injury. In the absence of a pelvic fracture and with a history of upper abdominal or chest trauma, the kidneys are the most likely source of the hematuria. In urologic trauma, the lower urinary tract must always be studied before the upper urinary tract (i.e., study the urethra before the bladder, study the bladder before the kidneys). The specific diagnostic studies must always be done in a retrograde fashion. This allows the responsible clinician to directly control the amount of contrast used to investigate potential urethral or bladder injuries. Whenever any doubt exists about the mechanism of injury, the patient's physical examination, or the source of gross hematuria, the resuscitating clinician is always advised to begin with an evaluation of the lower urinary tract before evaluating the upper urinary tract.

Evidence of Lower Urinary Tract Injury

In the resuscitation of any trauma patient, placement of a Foley catheter has become the standard method of monitoring urinary output. Blood at the urethral meatus, however, indicates a potential partial or complete urethral disruption and dictates the need for a retrograde urethrogram to delineate urethral integrity. This study can be done by the resuscitating clinician in the ED or on the operating room table by the trauma surgeon or urologist if the patient requires immediate surgical intervention for life-threatening injuries.

The male posterior urethra, which includes the membranous and prostatic urethra, is injured more frequently than the anterior urethra. The urogenital diaphragm encloses and fixes the membranous urethra; the prostate and prostatic

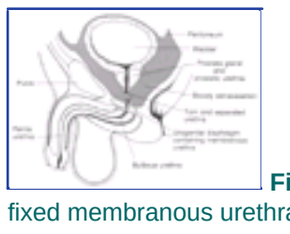

Figure 56-27 A common posterior urethral injury is a disruption of the membranous urethra. In this case, a distended bladder and attached prostate gland are sheared from the fixed membranous urethra. Note the development of a perivesical hematoma and the presence of a "high-riding" prostate gland.

urethra are firmly attached to the posterior surface of the symphysis pubis by the puboprostatic ligaments. Blunt trauma and pelvic fractures, especially in the presence of a full bladder, may result in shearing forces that partially or completely avulse portions of the firmly attached posterior urethra. Usually the bladder and prostate gland are sheared from the membranous urethra, resulting in a complete urethral disruption (Fig. 56-27). The female urethra, in contrast, is short and relatively mobile and generally escapes injury in blunt trauma. Occasionally, a significant pelvic fracture will result in a laceration or avulsion of the female urethra at the bladder neck. Direct injuries to the female urethra may also occur secondary to penetrating trauma to the vagina or perineum. These injuries often are disclosed by blood at the introitus or an abnormal vaginal examination in the female pelvic fracture patient. [84]

Contusions or lacerations of the male anterior urethra occur when the bulbous urethra is compressed against the inferior surface of the symphysis pubis. This happens most commonly as a result of straddle injuries in males but may result from any blunt perineal trauma. Significant trauma to the penile urethra is rare without penetrating injuries or urethral instrumentation. *Anterior urethral injuries* may result in extravasation of blood or urine into the penis, scrotum, or perineum, or along the anterior abdominal wall, depending on whether or not Buck fascia has been violated (Fig. 56-28).[37] This is in contrast to *posterior urethral injuries*, in which blood and urine extravasate into the pelvis.

The rectal examination is highly specific in the evaluation of a posterior urethral disruption. If the prostate is not clearly defined (it should have the consistency of the examiner's thenar eminence), is high-riding rather than in its normal anatomic location, or if a pelvic hematoma can be palpated (see Fig. 56-27), one should be

suspicious of a posterior urethral injury, and a retrograde urethrogram should be performed before attempting urethral catheterization. However, a normal rectal examination, by itself, *should not be considered definitive evidence of an intact urethra* if other clinical signs raise suspicion for urethral injury. Retrograde urethrography is a quick, technically easy study to perform and should be part of every emergency clinician's armamentarium.

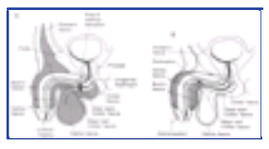

Figure 56-28 *A,* Disruption of the anterior urethra (bulbous urethra) occurs with straddle-type injuries in the male. Extravasation of urine and blood may occur in the perineum or scrotum, or along the anterior abdominal wall. Note that in this diagram, Buck fascia has been penetrated. *B,* Anterior urethral injury in which Buck fascia remains intact. In this situation, extravasation is confirmed and results in a swollen and ecchymotic penis. Such an injury usually results from instrumentation of the anterior urethra.

Pelvic Fracture

Pelvic fractures occur commonly in patients with urethral or bladder injury. The incidence of lower tract injuries in males with pelvic fractures ranges from 7% to 25%. Conversely, approximately 80% of all posterior urethral and bladder injuries are associated with pelvic fractures. [80] Because of the severity of late complications, especially urethral strictures, which most often require difficult surgical repair, it is paramount that these injuries not be missed. Again, in any female patient with a pelvic fracture, it is most important to examine the introitus and vaginal vault for blood, which may be indicative of urethral, bladder neck, or vaginal wall lacerations. In male patients, rectal examination of the prostate to assess its position will be most helpful in assessing the posterior urethra. A pelvic fracture in association with gross hematuria is an absolute indication for retrograde cystography. In a review of 234 patients with traumatic pelvic fractures, no major lower urinary tract injuries were found in the absence of gross hematuria. [80]

Radiographic Contrast Material

Radiographic contrast material is used to fill vessels and other structures to render them diagnostically radiopaque. To evaluate the urethra and bladder, contrast is injected or instilled into these structures in a retrograde manner. To evaluate the kidneys and ureters, a bolus of contrast material is injected into the venous system, opacifying the renal parenchyma and collecting system as it is excreted unchanged in the urine. Three types of contrast material are currently available (Table 56-9). All contain iodine, and all are hyperosmolar with respect to blood. Conventional agents, such as Hypaque and Renografin (diatrizoate), are triiodinated water-soluble agents (ionic monoacetic monomers) that completely dissociate into anion and cation moieties on intravascular injection. Osmolality is quite high, ranging from 1200 to 2000 mOsm/kg. Many of the side effects of contrast agents have been attributed to their osmolarity. Although iodine concentrations do determine the quality of the radiographic image, iodine itself is not thought to play a major role in the typical anaphylactoid side effects. [85]

Two new classes of contrast agents are ioxaglate (Hexabrix), an ionic monoacetic dimer, and nonionic (nondissociating) agents, such as iopamidol (Isovue) and iohexol (Omnipaque). The newer agents have twice as many iodine atoms per particle in solution as conventional agents and therefore provide significantly higher urinary iodine concentration, offering better diagnostic imaging. The osmolality of the newer agents is markedly lower, ranging from 600 to 700 mOsm/kg. The lower osmolality and improved chemical structure may be associated with fewer adverse side effects. [86] [87] Although these new agents are promising for intravascular use, there is still some skepticism that they will truly limit major or clinically significant contrast reactions. [88] The lower-osmolarity nonionic agents have not been associated with a lower incidence of contrast-induced nephropathy. Furthermore, there is no indication for using these more expensive products in the retrograde evaluation of the injured lower urinary tract.

Radiographic Techniques

Kidneys, Ureters, and Bladder

The plain film, scout film, or KUB (for kidneys, ureters, and bladder) film of the abdomen, as this view is variously referenced, includes the kidneys, ureters, bladder, and full pelvis. It is essential as the initial diagnostic film because it serves as a reference for all subsequent films after injection or instillation of contrast material. Incidental nondiagnostic findings on initial KUB that may alert the clinician to the possibility of

TABLE 56-9 -- Clinical Use of Radiographic Contrast Material (RCM) for Intravenous Pyelogram (IVP) and Retrograde Studies

Use of RCM for IVP	Iodine Content (mg/mL of Solution)	Osmolality (mOsm/kg) (H_2O)	Average Volume for IVP
Conventional ionic RMC	288	1511	Adult: 100 mL over 30–60 sec*
Renografin-60 (diatrizoate sodium)			Child: 1.5 mL/kg†
Hypaque (50%) (diatrizoate sodium)	300	1500	Adult: 100 mL over 30–60 sec*
			Child: 1.5 mL/kg†
Conray (methyl glucamine iothalamate)	282	1217	Adult: 100 mL over 30–60 sec*
			Child: 1.5 mL/kg†
New nonionic RMC	300	616	Adult: 50 mL over 30–60 sec‡
Isovue (iopamidol)			Child: 1–1.5 mL/kg†
Omnipaque 300 (iohexol)	300	672	Adult: 50 mL over 30–60 sec‡
			Child: 1–1.5 mL/kg†

Use of RCM for Retrograde Studies	Use	Procedure
Renografin-60 or Hypaque (50%)	Dilute stock solutions with saline 1:10 (10% solution)	Urethrogram: 10–15 mL of dilute solution injected slowly through urethral meatus. Children: 0.2 mL/kg Cystogram: after plain film and with Foley catheter in place, fill bladder of adult with 400 mL of dilute contrast material, introduced under gravity. Children: 5 mL/kg

*Average dose of iodine for IVP with ionic RCM: 350–400 mg/kg or 1.5 mL/kg
Adult: Low dose: 10 g
Intermediate dose: 30 g
High dose: 60 g
†Do not exceed 3 mL/kg total dose.
‡Because the ratio of iodine atoms to dissolved particles is 1.5 with conventional ionic agents and 3.0 with the nonionic agents, less volume is required with the new agents. Average dose is 200–350 mg/kg.

Figure 56-29 Retrograde urethrogram. The foreskin is fully retracted and an unwrapped 4 × 4 gauze sponge is folded in half longitudinally and wrapped around the penis proximal to the coronal sulcus, to prevent foreskin reduction. *A,* The penis is held between the long and ring fingers of the nondominant hand. The thumb and index finger ensure a snug fit of the syringe in the urethra. *B,* Equipment needed for retrograde urethrography and cystography. *C,* Alternative technique for securing the "irrigation-tip" syringe in the urethra.

urinary tract injury include the following:

1. Loss of one or both psoas shadows secondary to blood in the retroperitoneum.
2. Spinal curvature secondary to splinting—usually concave to the side of the injury.
3. Lower rib or transverse process fractures, both of which may be associated with upper urinary tract injury.
4. Pelvic fracture.

The KUB must always precede the injection or instillation of contrast material, because radiopaque shadows seen on the plain film must be differentiated from extravasation on the postevacuation film.

Retrograde Urethrogram

Retrograde urethrography is indicated whenever there is uncertainty about the integrity of the urethra. In cases associated with pelvic fracture, the patient should remain supine throughout the entire radiographic examination. This is important to ensure stability of any possible retropubic hematoma that may result from extensive venous bleeding associated with the initial pelvic fracture. In cases of suspected urethral injury not associated with pelvic fracture, it is acceptable to obtain oblique films during the study that may complement the examination findings. Perpendicular stretching of the penis across the thigh or oblique films may be needed to ensure urethral unfolding and a high-quality urethrogram.

Although several techniques have been promoted for retrograde urethrography, one is emphasized in this section. The choice of technique is not as important as attention to detail. Solutions of either full-strength Hypaque (50%), Cystografin or Renografin-60, or the same agents diluted to a less than 10% solution using sterile saline as the diluent, are frequently used (see Table 56-9). First, a plain film (KUB) of reference must be taken before injection of any contrast material. [81] The penile foreskin must be retracted and secured with a folded 4 × 4 gauze sponge. Second, the penis should be held between the long and ring fingers of the nondominant hand to allow the thumb and index finger of the nondominant

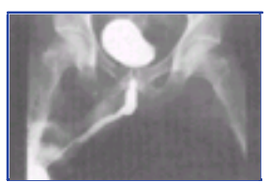

Figure 56-30 Normal retrograde urethrogram. The patient is supine on the examination table. The penis is stretched perpendicularly across the patient's right thigh to allow urethral unfolding and complete urethral visualization.

hand (see Fig. 56-18) a snug fit of the contrast-filled syringe inside the urethra (Fig. 56-29*A*).

After sterile penile preparation, a catheter-tipped Toomey irrigating syringe or a regular 60-mL syringe with an attached Christmas-tree adapter (Fig. 56-29*B*) is gently advanced inside the urethral meatus until a snug fit is ensured (Fig. 56-29*C*). Third, approximately 50 to 60 mL of full- or half-strength contrast material is then injected *slowly* under constant pressure into the urethra. Prior to the injection of contrast, the penis should be stretched perpendicularly across the patient's thigh to prevent urethral folding (i.e., the double image of the proximal penile and bulbous urethra superimposed on one another) (Fig. 56-30). Overly forceful injection of contrast material may cause intravasation of contrast material into the venous plexus of the urethra (Fig. 56-31). Finally, during the injection of the last 10 mL of contrast, a film (the urethrogram) is taken.

Figure 56-31 Venous intravasation (*arrows*) during a forceful retrograde urethrogram. This may mimic urethral extravasation, but it clears immediately, as opposed to actual extravasation, which remains indefinitely. The presence of intravasation is benign. *(From Richter MW, Lytton B, Myerson D, Grnja V: Radiology of genitourinary trauma. Radiol Clin North Am 11(3):626, 1973.)*

The alternative to this technique, which is discussed in most standard textbooks, is to insert a Foley catheter just inside the urethral meatus, inflate the balloon to ensure a snug fit in the fossa navicularis, and then inject contrast through the catheter (Fig. 56-32). If not done carefully, this technique often results in the spillage and deposition of contrast outside the urethra and onto the patient and the examination table, thus yielding a spurious result.

The extravasation of contrast material from a urethral disruption usually appears as a flame-like density outside the urethral contour (Fig. 56-33*A–C*). If any contrast material is seen within the bladder in conjunction with urethral extravasation, a partial rather than complete urethral disruption is more likely. In a complete urethral disruption, urethral extravasation will be present without evidence of contrast within the bladder. The examiner needs to be certain that the lack of bladder contrast is not secondary to voluntary contraction of the external sphincter. Occasionally, as mentioned previously, intravasated contrast material is seen in the periurethral penile venous

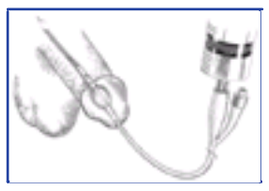

Figure 56-32 Retrograde urethrogram using a Foley catheter (8 Fr). Slowly inflate the balloon with 2 mL of air, tap water, or sterile saline to create a snug fit; then slowly inject 60 mL of a 10% solution of contrast material through the catheter lumen (see text).

Figure 56-33 *A,* Retrograde urethrogram. Urethrogram in case of supramembranous urethral rupture. Contrast extravasation is typical of that seen with this type of injury. *B,* A

rupture at the proximal bulbous urethra into the scrotum (*arrows*). *C,* Residual contrast material within perineum and scrotum. *(A from Morehouse DD, MacKinnon KJ: Posterior urethral injury: Etiology, diagnosis, initial management. Urol Clin North Am 4:74, 1977. C from Richter MW, Lytton B, Myerson D, Grnja V: Radiology of genitourinary trauma. Radiol Clin North Am 11:627, 1973.)*

plexus (see Fig. 56-31). It is of no clinical significance and should not be mistaken for urethral extravasation. As expected, penile venous intravasation (venous plexus opacification) is seen to clear spontaneously on any post-void films, as compared with urethral extravasation, which remains indefinitely.

If a Foley catheter has been successfully placed into the bladder and a partial urethral injury is suspected later, such an injury can be easily demonstrated without removing the catheter. The lubricated end of a pediatric feeding tube is placed into the penile urethra alongside the existing Foley catheter (Fig. 56-34). A seal can be obtained by compressing the glans penis with the nondominant thumb and index finger and gently injecting contrast material via a Luer-Lock syringe with the dominant hand. In this way, extravasation can be demonstrated. It should be noted, however, that successful placement of the Foley catheter obviates the need for further treatment of a partial urethral tear in the emergency setting, because an indwelling catheter alone is appropriate initial management for this type of injury. The finding of an associated urethral injury must be conveyed to a urologist, as it will dictate the duration of definitive Foley catheter drainage.

Retrograde Cystogram

A retrograde cystogram is performed any time a bladder injury is suspected. It assumes the urethra is normal prior to passing the Foley catheter. A preliminary KUB is obtained that will serve as the reference film for the entire examination. Next, the bladder is filled under direct operator supervision by *gravity instillation of contrast material*. After the central piston is removed from a 60-mL catheter-tip syringe, the catheter-tipped end of the syringe is attached to the Foley catheter and held above the level of the patient's bladder. The contrast material is poured into the syringe

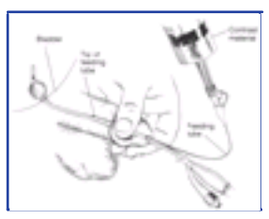

Figure 56-34 Evaluation of a urethral injury with a Foley catheter in place. A lubricated pediatric feeding tube has been advanced into the urethra beside the indwelling Foley catheter.

and allowed to fill the bladder by gravity instillation to 1 of 3 end points: (1) 100 mL with evidence of gross extravasation on fluoroscopy or on plain film, (if the examiner elects to check at this point); (2) 400 mL in an adult or any child 11 years or older. In children younger than 11 years, bladder capacity, and therefore appropriate contrast volumes, are estimated based on the formula "(age in years + 2) × 30"; or (3) to the point of initiating a bladder contraction (see later), then adding an additional 50 mL by hand injection under pressure.

Anteroposterior (AP) and complementary oblique projections are obtained so long as there is no evidence of a

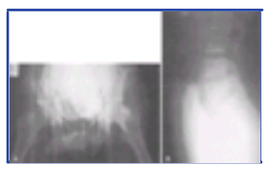

Figure 56-35 Retrograde cystogram. *A,* In patients with pelvic fracture, retrograde cystography should be done with the patient supine throughout the examination. Here, gross extravasation is evident, but its superior extent is not well defined. *B,* A lateral film may help define the extent of any extravasation. This film shows no intraperitoneal extension, so the extravasation must be totally extraperitoneal.

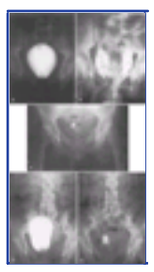

Figure 56-36 Retrograde cystogram. *A,* Anteroposterior (AP) filled-bladder film. *B,* AP postevacuation film of same patient showing extensive extravasation not seen on the AP filled-bladder film. Balloon of catheter is identified by *arrows*. *C,* "KUB" (kidneys, ureters, and bladder) showing bullet in the area of pelvis. *D,* AP filled-bladder film of same patient showing bladder displacement to right, presumably from a pelvic hematoma. No extravasation is visible with the bladder full of contrast. *E,* AP postevacuation film of same patient, showing subtle contrast extravasation in area of bullet that could easily be missed without a high-quality preliminary KUB and post-evacuation film.

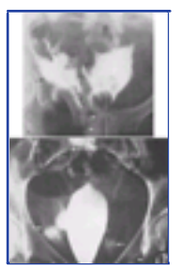

Figure 56-37 Examples of extraperitoneal bladder rupture. *A,* Note the amorphous extravasation of contrast material within the perivesical space (*arrows*) in a patient with a right pelvic fracture (*arrowheads*). *B,* A second patient with a pelvic fracture (*arrowhead*) and perivesical hematoma shows the teardrop shape of a deformed bladder and extraperitoneal extravasation (*arrows*). *(From Richter MW, Lytton B, Myerson D, Grnja V: Radiology of genitourinary trauma. Radiol Clin North Am 11:623, 1973.)*

pelvic fracture. In the presence of a pelvic fracture, all films are obtained with the patient in the supine position for the same reasons that were elucidated for retrograde urethrography. A lateral film may be informative when oblique films are not possible (Fig. 56-35). An AP postevacuation film must be obtained in all cases following bladder drainage. This will disclose posterior perforation in select cases, especially those associated with penetrating trauma (Fig. 56-36*A–E*). Again, a *dilute solution of contrast material* (see Table 56-9) may be used, rather than full-strength contrast. Some authors recommend a dilute solution of contrast material (=10%) because extravasation into periurethral or perivesical tissues may cause considerable inflammatory reaction at higher concentrations. The dilute solutions do not appear to compromise the quality of the study, but this must be a consideration. *Retrograde cystography done by any technique other than hand-poured gravity instillation is subject to inadequate bladder filling or connector tubing-catheter disconnection.* Both conditions will result in spurious examination results, which may adversely impact important patient management decisions.

It must be stressed that in the absence of initial gross extravasation, the bladder must be filled to 400 mL in an

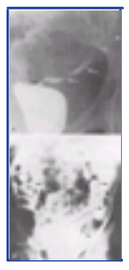

Figure 56-38 Intraperitoneal bladder rupture. *A,* A 22-year-old pedestrian hit by an automobile. Note extravasation of contrast material beginning at the dome and tracking up the left paracolic gutter (*arrows*). *B,* This 57-year-old man had fulguration of a bladder tumor at the bladder dome and sustained perforation. A cystogram dramatically demonstrates the extravasation of contrast material that outlines the bowel loops (*arrows*) and the paracolic gutters. *(Courtesy of Morton A, Bosniak MD, New York.) (From Richter MW, Lytton B, Myerson D, Grnja V: Radiology of genitourinary trauma. Radiol Clin North Am 11:623, 1973.)*

adult, and to an appropriate capacity in a child, and the catheter clamped with a Kelly clamp. Volumes less than 400 mL have been associated with false-negative findings, especially in penetrating bladder injuries. [89] At times, the patient may have difficulty cooperating with bladder filling because of a head injury or associated pain; and in the case of severe injury, the patient may have involuntary bladder contractions, causing contrast material to back up into the Toomey syringe. If this occurs, refill the bladder to the point of initiating a bladder contraction, clamp the Foley, remove the initial syringe, and replace it with a 60-mL contrast-filled syringe, unclamp the catheter, hand-inject the additional 50 mL under pressure, and reclamp the catheter. The goal is to overdistend the bladder. Once the filled-bladder films have been obtained and reviewed, the Foley catheter is unclamped and the contrast material is allowed to drain into a bedside drainage bag. The AP postevacuation film is then obtained to visualize any posterior extravasation that may have been hidden by the distended bladder during the AP filled-bladder film. Once again, care must be taken to ensure that contrast material is not spilled onto the patient or the examination table during the procedure. Spilled contrast can lead to spurious examination results.

Extravasation from an injured bladder may be intraperitoneal, extraperitoneal, or both. Extraperitoneal extravasation is usually seen as flame-like areas of contrast material confined to the pelvis and projecting lateral to the bladder (Fig. 56-37). If the contrast material extravasates intraperitoneally, it tends to fill the paracolic gutters and outline intraperitoneal structures, particularly the bowel, spleen, or liver (Fig. 56-38). It is important to distinguish extraperitoneal from intraperitoneal injury, as the treatment options are totally different (i.e., surgical repair for all intraperitoneal injuries and for extraperitoneal injuries that extend into or primarily involve the bladder neck, especially in women). Most other extraperitoneal injuries can be managed confidently by Foley catheter drainage alone.

Retrograde cystography may be done in conjunction with contrast-enhanced abdominal CT scanning. The bladder must be filled just as if a conventional retrograde cystogram were being obtained. The catheter is clamped, and evidence for contrast ascites is sought on the CT scan (Fig. 56-39). When this is encountered, bladder injury with extravasation must be looked for with selective images of the pelvis.

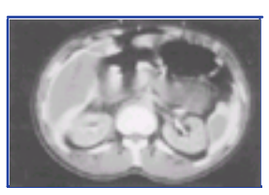

Figure 56-39 Retrograde cystogram and abdominal computed tomography (CT) scan. These two procedures can be done concomitantly. The bladder is filled in the standard retrograde fashion and the catheter is clamped. Intravenous and oral contrast can then be administered and CT scanning performed. This film demonstrates contrast ascites, which is consistent with intraperitoneal bladder rupture and extravasation.

Contrast Medium Reactions and Toxic Effects

In the lower genitourinary tract radiologic procedures that are described in this section, the contrast material is administered within the urinary collecting and drainage system. Hence, the patient is at low risk for systemic absorption and allergic reaction. Even with IV infusion, contrast medium reactions are rare; the incidence of significant reactions (i.e., of sufficient severity to require medical intervention) with IV administration is between 1 in 1000 and 1 in 10,000 uses.

Although IV use of contrast medium is outside the scope of this text, volumes and administration information are outlined in Table 56-9 . The ED use of contrast agents is often necessary and justified despite the small possibility of untoward reactions. At times, the patient's past history may not be known, underlying renal function cannot be rapidly assessed, or alternative imaging techniques (e.g., ultrasonography) are unavailable. In such circumstances, the risks vs the benefits of emergent imaging using IV contrast must be carefully weighed. Often the potential information gain of contrast-enhanced imaging in the unstable patient far outweighs the small associated additional risk.

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Section X - Obstetric and Gynecologic Procedures

Chapter 57 - Emergency Childbirth

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The past century has witnessed a marked improvement in prenatal and obstetric care in the United States and with that a marked reduction in infant and maternal mortality. At the beginning of the 20th century, for every 1000 live births in the United States, six to nine women died of pregnancy-related complications and approximately 100 infants died before 1 year of age. [2] From 1900 through 1997, the maternal mortality rate declined almost 99% to less than 0.1 reported deaths per 1000 live births. Similarly from 1915 to 1997 the infant mortality rate declined 93%, to 7.2 per 1000 live births. [2] Environmental interventions, improvements in nutrition, access to health care, and medical advances (such as better management of pregnancy-related hypertension) and an increase in the number of in-hospital deliveries have all contributed to this remarkable decline. [2] [36]

The degree to which the emergency clinician interacts in the process of labor and delivery varies among institutions, depending on the availability and readiness of inpatient obstetric services. The role of the emergency clinician may be only to determine that the patient is indeed in active labor and to order transport directly to the labor and delivery area. In a hospital with little or no obstetric services, the emergency clinician may alternatively be called on to manage a complicated delivery and neonatal resuscitation until transfer to another hospital is possible.

To this end, the emergency clinician should be able to assess the stage and timing of labor, aid the mother in delivery of the infant, and provide initial stabilization of the newborn.

LABOR

Labor is defined as the coordinated sequence of involuntary uterine contractions that result in progressive effacement and dilation of the cervix. This, coupled with the voluntary bearing-down efforts of the mother, terminates in delivery, the actual expulsion of the products of conception.

Labor is normally divided into three stages. The first stage begins when uterine contractions reach sufficient force to cause cervical effacement and dilation and ends when the cervix is completely dilated. Although the average duration of the first stage of labor is about 4 hours in parous patients and 7 hours in nulliparous patients, there is marked individual variation. [22] The second stage of labor begins when dilation of the cervix is complete and ends with delivery of the infant. The duration of this stage is also highly variable, with a median of 50 minutes in nulliparas and 20 minutes in multiparas. [22] In general, if the second stage lasts more than 2 hours, abnormal labor has developed. [22] The third stage of labor begins after delivery of the infant and ends after delivery of the placenta. Infrequently, a fourth stage of labor is described as the hour immediately following delivery and is the period in which postpartum hemorrhage due to uterine atony is most likely to occur. [22]

IDENTIFICATION OF LABOR

True versus False Labor

Before the establishment of true or effective labor, women may experience so-called false labor. Quite common in late pregnancy, false labor is characterized by irregular, brief contractions of the uterus, usually with discomfort confined to the lower abdomen and groin. These contractions, commonly referred to as *Braxton-Hicks contractions,* are typically irregular in timing and strength, and there is no change in the cervix and no descent of the fetus.

True labor is characterized by a regular sequence of uterine contractions, with progressively increasing intensity and decreasing intervals between contractions. The discomfort produced by the uterine contractions of true labor begins in the fundal region and radiates over the uterus into the lower back. The uterine contractions of true labor are accompanied by effacement and dilation of the cervix, with descent of the presenting part of the fetus.

False labor is most common in late pregnancy and in parous women. Although false labor usually stops spontaneously, it may convert rapidly to the effective contractions of true labor. Therefore, a period of observation may be necessary. The interval between true labor contractions gradually diminishes from 10 minutes at the onset of the first stage of labor to as short as 1 minute or less in the second stage. [22]

Show

A rather common and dependable sign of the approach of labor is the "show" or "bloody show." A rather late sign of labor, show consists of a small amount of blood-tinged mucus discharged from the vagina and indicates that labor is already in progress or will likely occur during the next several hours to few days. Show represents extrusion of the mucus plug that filled the cervical canal during pregnancy and is evidence of cervical effacement and dilation. Normally, only a few drops of blood escape with the mucus plug.[22] More substantial bleeding during labor suggests an abnormal cause such as abruption of placenta or placenta previa, and vaginal examination is generally *contraindicated*. [22]

Rupture of the Membranes

Spontaneous rupture of the membranes usually occurs during the course of active labor. Typically, rupture is evident by a sudden gush of a variable amount of clear or slightly turbid fluid. Rupture of the membranes before the onset of labor at any stage of gestation is referred to as *premature rupture of the membranes* (PROM).

Rupture occurring at term, but before the onset of labor, is called *term premature rupture of the membranes* and complicates approximately 8% of pregnancies.[7] Term PROM is followed by the onset of labor and delivery within 5 hours in approximately 95% of cases. [7] The most significant maternal risk of term PROM is intrauterine infection. Fetal risks associated with PROM include umbilical cord compression and ascending infection. [7]

Membrane rupture occurring before 37 weeks of gestation is called *preterm premature rupture of the membranes* (pPROM). Delivery within 1 week of pPROM occurs in approximately 75% of patients regardless of management or clinical presentation. [7] The most significant maternal risk of pPROM is intrauterine infection, although with appropriate management, serious maternal sequelae are uncommon. [7] The most significant risks to the fetus are complications of prematurity such as respiratory distress, necrotizing enterocolitis, and intraventricular hemorrhage. [7] Definitive treatment of

PROM at any stage of gestation is left to the clinician and is dependent on multiple factors including the presentation, status and gestational age of the fetus, and maternal factors, such as suspected infection and placental location. Treatment options include the induction of labor, operative delivery, the use of prophylactic antibiotics, and the use of antenatal corticosteroids to promote fetal lung maturity. [7] [48] [52]

Although membrane rupture during labor is typically manifested by a sudden gush of fluid, presentation, particularly that of PROM, may be more subtle. Because accurate diagnosis is crucial to management, symptoms suggestive of PROM should be confirmed. Examination should be performed in a manner that minimizes the risk of introducing infection. Therefore digital cervical examinations, which increase this risk, should be avoided unless prompt labor and delivery is anticipated. When obstetric facilities are not immediately available, a sterile speculum examination can be performed. Rupture of the membranes is verified if amniotic fluid is seen extruding from the cervical os or pooling in the posterior fornix. [7] [22]

Differentiation of amniotic fluid from vaginal fluid may be made by testing the pH of a drop of the fluid with Nitrazine paper. Amniotic fluid has a pH of 7.0 to 7.5 and turns the paper blue-green to deep blue. In the presence of vaginal secretions only, with a pH of 4.5 to 5.5, Nitrazine paper remains yellow. [2] [7] [12] [22] Abe[2] found the Nitrazine test to be positive in 98.9% of women with known rupture of the membranes and negative in 96.2% of women with intact membranes. In clinical practice, however, the test is less reliable because it is frequently used in cases of questionable rupture in which the amount of amniotic fluid is small and, therefore, more subject to pH changes from admixed blood and vaginal secretions. False positive tests may occur with blood, semen, or bacterial vaginosis and false-negative tests with minimal fluid.[2] [7] [12] [22]

A less frequently used method to test for amniotic fluid is *fern*ing. A drop of fluid from the cervical os or vaginal fornix is placed on a clean glass slide. Owing to the high sodium chloride content of amniotic fluid, a fern pattern is seen through the microscope as amniotic fluid dries. [7] [29] When the clinical history or physical examination is unclear, ultrasound examination may be useful to document oligohydramnios that, in the absence of fetal abnormalities, is suggestive of membrane rupture. Although outside the scope of the emergency clinician, membrane rupture can be diagnosed unequivocally with ultrasonographically guided transabdominal instillation of indigo carmine dye with subsequent passage of blue fluid from the vagina. [7] If rupture of the membranes is documented in the emergency department (ED), the patient's clinician should be notified and hospital admission of the patient considered.

Evaluation of Labor

When a woman presents in labor, the general condition of the fetus and mother must be quickly ascertained by means of the patient history and physical examination. Inquiry is made as to the onset and frequency of contractions, the presence or absence of bleeding, the possible loss of amniotic fluid, and the prenatal care and condition of the mother and fetus. In the absence of active vaginal bleeding, the position, presentation, and lie of the fetus may be determined by abdominal palpation and sterile vaginal examination. Staging of labor is assessed by vaginal examination. Fetal well-being is monitored by auscultation of fetal heart tones, particularly immediately after a uterine contraction.

Lie, Presentation, and Position

In the latter months of pregnancy, the fetus assumes a characteristic posture within the uterus, usually forming an ovoid mass that corresponds roughly to the shape of the uterine cavity. Typically, the fetus becomes folded or bent on itself in such a way that the back becomes markedly convex, with the head, thighs, and knees sharply flexed. Usually the arms are crossed over the thorax and are parallel to the sides of the body. The umbilical cord lies in the space between the arms and the lower extremities. This characteristic posture is due in part to the mode of growth of the fetus and is also a result of accommodation to the uterine cavity.

Lie refers to the relation of the long axis of the fetus to that of the mother. Lie is either longitudinal or transverse (Fig. 57-1 (Figure Not Available)). Longitudinal lies occur in greater than 99% of pregnancies at term. [22]

The *presentation*, or presenting part, refers to that portion of the body of the fetus that is nearest to, or foremost in, the birth canal. The presenting part is felt through

the cervix on sterile vaginal examination. In longitudinal lies, the presenting part is either the fetal head, the buttocks, or the feet. In *transverse lie*, the shoulder is the presenting part.

Cephalic presentations are classified by the relation of the fetal head to the body of the fetus (Fig. 57-2 (Figure Not Available)). Ordinarily, the head is sharply flexed so that the occipital fontanel is the presenting part. This is referred to as the *vertex* or *occiput presentation*. Less commonly, the neck is fully extended and the face is foremost in the birth canal; this is termed *face presentation*. Occasionally, the fetal head assumes a partially flexed or partially extended position, resulting in *sinciput* and *brow presentations*, respectively. Sinciput and brow presentations, associated with preterm infants, are almost always unstable and convert to either the occiput or face presentation as labor progresses.

Breech presentations are classified as frank, complete, and footling or incomplete (Fig. 57-3 (Figure Not Available)). When the fetus presents with the hips flexed and the legs extended over the anterior surfaces of the body, this is termed *frank breech*. Flexion of the fetal hips and knees results in *complete breech* presentation. When one or both of the feet or knees are lowermost in the canal, an *incomplete* or *footling breech* results.

Figure 57-1 (Figure Not Available) *A,* Transverse lie with shoulder presentation. *B,* Longitudinal lie with vertex presentation. *(From Romney S, Gray MK, Little AB, et al [eds]: Gynecology and Obstetrics: The Health Care of Women. New York, McGraw-Hill, 1975.)*

Figure 57-2 (Figure Not Available) Cephalic presentations-deflexion attitude of fetal head. *A,* Vertex. *B,* Sinciput. *C,* Brow. *D,* Face. Diameter of the presenting fetal head is shown for each of the attitudes. *(From Romney S, Gray MK, Little AB, et al [eds]: Gynecology and Obstetrics: The Health Care of Women. New York, McGraw-Hill, 1975.)*

At or near term, the incidence of the various presentations is approximately 96% for vertex, 3.5% for breech, 0.3% for face, and 0.4% for shoulder. [22]

Position refers to the relation of the presenting part to the birth canal and may be either left or right. The occiput, chin, and sacrum are the determining parts in vertex, face, and breech presentations, respectively. The presentation and position of the fetus are initially determined by abdominal palpation using Leopold maneuvers.

Abdominal palpation (Leopold maneuvers).

Although abdominal ultrasonography has largely replaced abdominal palpation for determination of fetal lie, these maneuvers may be helpful when ultrasound evaluation is unavailable. They can be performed throughout the latter months of pregnancy and during labor in the intervals between contractions. The findings from abdominal palpation provide information about the presentation and position of the fetus and the extent to which the presenting part has descended into the pelvis (Fig. 57-4 (Figure Not Available)). The mother should be placed on a firm bed or examining table with her abdomen bared. For the first three of the four maneuvers, the examiner stands at the side of the bed facing the patient. During the first maneuver (see Fig. 57-4 *A* (Figure Not Available)), the upper abdomen is gently palpated with the fingertips of both hands to determine which fetal pole is present in the uterine fundus. The fetal breech gives the sensation of a large, nodular body, whereas the fetal head is hard, round, and freely movable.

During the second maneuver, the examiner places his or her hands on either side of the abdomen, exerting deep, gentle pressure (see Fig. 57-4 *B* (Figure Not Available)). On one side, the hard, resistant back is felt; on the other side, the fetal extremities or small parts are felt. By noting whether the back is directed anteriorly,

Figure 57-3 (Figure Not Available) Fetal attitude in breech presentations. *A,* Frank. *B,* Complete. *C,* Single footling—incomplete. *D,* Double footling—incomplete. *(From Romney S, Gray MK, Little AB, et al [eds]: Gynecology and Obstetrics: The Health Care of Women. New York, McGraw-Hill, 1975.)*

posteriorly, or transversely, fetal orientation or lie is determined.

The third maneuver is performed by grasping the lower portion of the maternal abdomen just above the symphysis pubis with the thumb and forefinger of one hand (see Fig. 57-4*C* (Figure Not Available)). If the presenting part is not engaged, the position of the head in relation to the back and extremities is ascertained. If the cephalic prominence is palpated on the same side as the small parts, the head must be flexed and therefore a vertex or occiput presentation exists. If the cephalic prominence is on the same side as the back, the head must be extended. If the presenting part is deeply engaged in the pelvis, the findings from this maneuver indicate that the lower pole of the fetus is fixed in the pelvis. The details of presentation and position are then defined by the fourth maneuver.

To perform the fourth maneuver, the examiner changes position and faces the mother's feet. With the tips of the first three fingers of each hand, the examiner exerts deep, gentle pressure in the direction of the axis of the pelvic inlet (see Fig. 57-4 *D* (Figure Not Available)). When the head is the presenting part, one examining hand will be stopped sooner than the other by a rounded body, the cephalic prominence, while the other hand continues more deeply into the pelvis. The cephalic prominence is felt on the same side as the small parts in vertex presentations and on the same side as the back in face presentations. In breech presentations, the information obtained from this maneuver is less precise. [22]

Vaginal examination.

Unless there has been bleeding in excess of a bloody show, a manual (not speculum) vaginal examination should be performed to identify fetal presentation and position and to assess the progress of labor.

Figure 57-4 (Figure Not Available) Abdominal palpation (four maneuvers of Leopold). *A,* Determination of the fetal part occupying the uterine fundus. *B,* Palpation of fetal small parts and back. *C,* Determination of the part occupying the lower uterine segment. *D,* Determination of the cephalic prominence. *(From Romney S, Gray MK, Little AB, et al [eds]: Gynecology and Obstetrics: The Health Care of Women. New York, McGraw-Hill, 1975.)*

The vulva and perineal area are prepared with an antiseptic solution such as povidone-iodine. The woman is placed on a bedpan with her legs widely separated. Scrubbing is directed from anterior to posterior and away from the vaginal introitus; each sponge should be discarded after it passes over the anal region. A dry sponge placed on the introitus prevents contaminated solution from running into the vagina.

After preparation of the vulvar and perineal regions, the examiner uses the thumb and forefinger of a sterile-gloved hand to widely separate the labia to expose the vaginal opening; this prevents the examining fingers from coming into contact with the inner surfaces of the labia. The index and second fingers of the other hand are then introduced into the vagina to perform the examination. Cervical effacement, dilation, and fetal station are assessed. Fetal presentation and position are confirmed.[22]

Cervical effacement refers to the process of cervical thinning that occurs before and during the first stage of labor as the cervical canal shortens from a length of about 2 cm to a circular opening with almost paper-thin edges (Fig. 57-5 (Figure Not Available)). The degree of cervical effacement is assessed by palpation and is determined by the palpated length of the cervical canal compared with that of the uneffaced, or normal, cervical canal. Effacement is expressed as a percentage from 0%, or totally uneffaced, to 100%, or completely effaced.

Cervical dilation is determined by estimating the average diameter of the cervical os. The examining finger is swept

Figure 57-5 (Figure Not Available) Effacement of the cervix. *A,* None. *B,* Partial. *C,* Complete. *(From Romney S, Gray MK, Little AB, et al [eds]: Gynecology and Obstetrics: The Health Care of Women. New York, McGraw-Hill, 1975.)*

from the cervical margin on one side across the cervical os to the opposite margin. The transverse diameter is expressed in centimeters. Ten centimeters constitutes full cervical dilation. A diameter of <6 cm can be measured directly. For a diameter >6 cm, it is frequently easier to determine the width of the remaining cervical rim

and subtract twice that measurement from 10 cm. For example, if a 1 cm rim is felt, dilation is 8 cm.

Station refers to the level of the presenting fetal part in the birth canal relative to the ischial spines which lie halfway between the pelvic inlet and the pelvic outlet (Fig. 57-6). Zero station is used to denote that the presenting part is at the level of the ischial spines. The birth canal above and below the spines is divided into fifths. When the presenting part lies above the spines the distances are stated in negative figures (-5, -4, -3, -2, and -1). Below the ischial spines the presenting fetal part passes +1, +2, +3, +4, and +5 stations to delivery. Determination is made by simple palpation. [22]

Position and presentation of the fetus may be inconclusive before labor, because the presenting parts must be palpated through the lower uterine segment. After dilation and effacement of the cervix, however, further delineation of presentation and position of the fetus may be made by vaginal examination.

After the perineal area has been appropriately prepared, as described previously, 3 maneuvers are used to determine fetal presentation and position. In the first maneuver, 2 fingers of the examiner's gloved hand are introduced into the vagina and advanced to the presenting part, differentiating face, vertex, and breech presentations. In vertex presentations, the examiner's

Figure 57-6 Station of the fetal head. As a reference point, the level of the ischial spines is zero station. *(From Benson RC [ed]: Current Obstetric and Gynecologic Diagnosis and Treatment, 3rd ed. Los Altos, Calif., Lange Medical Publications, 1980.)*

fingers are carried up behind the symphysis pubis and then swept posteriorly over the fetal head toward the maternal sacrum, identifying the course of the sagittal suture. The positions of the two fontanels, located at opposite ends of the sagittal sutures, are then defined by palpation. The anterior fontanel is diamond shaped; the posterior fontanel is triangular.

In face and breech presentations, the various parts are more readily distinguished. In breech presentations, the fetal sacrum is the point of reference; in face presentations, the easily identifiable fetal chin is used.

Fetal Well-Being

Auscultation.

Auscultation of fetal heart tones is necessary to determine fetal well-being. The heart rate of the fetus can be identified with a stethoscope, a fetoscope, or preferably a Doppler ultrasound device placed firmly on the maternal abdominal wall overlying the fetal thorax and repositioned until fetal heart tones are heard. When a Doppler device is used, a conducting gel should be applied to the abdominal wall, interfacing with the Doppler receiver. The region of the abdomen in which fetal heart sounds are heard most clearly varies with fetal presentation and the degree to which the presenting part has descended. In cephalic presentations, fetal heart sounds are heard best midway between the maternal umbilicus and the anterior superior spine of the maternal ilium. [22] To avoid confusion of maternal and fetal heart sounds, the maternal pulse should be palpated as the fetal heart rate is auscultated.

Normal baseline fetal heart rate is 120 to 160 bpm with heart rate varying considerably from beat to beat. [5] [22] Rates above or below this range may indicate fetal distress. Fetal tachycardia occurs when fetal heart rate is >160 beats/min. [22] Brief accelerations in fetal heart rate (i.e., those lasting <20 seconds) occur commonly during labor and are probably a physiologic response to fetal movement. [5] [22] [63] In contrast, persistent fetal tachycardia occurs most commonly in response to maternal fever or amnionitis but may also indicate fetal compromise. [5] [22]

Fetal bradycardia occurs when fetal heart rate falls below 120 beats/min. [22] Like brief accelerations in fetal heart rate, decreases in rate, which reach their nadir with the peak of a contraction and end with or slightly after the end of a contraction (early decelerations), are physiologic and are probably the result of vagal nerve stimulation due to compression of the fetal head. [5] [22] In contrast, decelerations that occur independently of uterine contractions (variable decelerations) and those that persist significantly after a contraction (late decelerations) are ominous and may represent cord compression and uteroplacental insufficiency, respectively. [5] [22] [63]

Changes in the fetal heart rate indicating fetal distress are usually evident immediately after a uterine contraction and, therefore, fetal heart rate is optimally assessed at this time. If prolonged monitoring of labor is necessary in the ED, fetal heart sounds should be assessed at 15-minute intervals during the first stage of labor and at 5-minute intervals during the second stage in the pregnancy at risk. In the absence of risk factors, intervals of 30 minutes and 15 minutes respectively, are probably sufficient.[5] [22] If trained personnel and equipment are available, external tocography provides a noninvasive method for continuous assessment of fetal heart rate and maternal uterine contractions.

Management of fetal distress.

If fetal distress is suspected on the basis of resting fetal heart rate or changes after contractions, changing maternal position, typically into the left lateral decubitus position, may be beneficial. Maternal O_2 should be administered to improve fetal oxygenation. In the absence of bleeding, a vaginal examination should be performed to rule out the possibility of prolapse of the umbilical cord. [5] [29] If immediate obstetric services are not available, consideration should be given to tocolytic therapy to improve fetoplacental blood flow until delivery can be accomplished. By stopping uterine contractions, a more sustained placental blood flow is maintained and, in the case of cord prolapse, intermittent pressure on a compromised umbilical cord may be averted. [5] [63] The definitive therapy for fetal distress is delivery of the infant, either vaginally or by cesarean section. [5] [22]

Cord prolapse usually occurs at the same time as rupture of the membranes and is diagnosed by palpation of the umbilical cord on vaginal examination or by visualization of the cord protruding through the introitus. The incidence of cord prolapse in labor is approximately 0.2% and most often occurs when the fetal presenting part does not completely fill the lower uterine segment during labor or when there is unusual mobility of the cord. [20] [54] [59] Cord prolapse is frequently encountered with breech presentation, multiple pregnancies, and prematurity. [20] [54] [59]

The management of cord prolapse is directed at sustaining fetal life until delivery is accomplished. Unless immediate delivery is feasible or the fetus is known to be dead, preparations should be made for an emergency cesarean section. If immediate obstetric services are not available, tocolytic therapy may be instituted to decrease uterine contractions and improve fetoplacental perfusion. [13] [39] Compression of the umbilical cord should be minimized by exerting manual pressure through the vagina to lift and maintain the presenting part away from the prolapsed cord. The patient should be placed in the knee-chest or deep Trendelenburg position, and this position maintained until delivery is accomplished. [13] [29] [59] Some clinicians recommend that after manual elevation of the presenting part, 500 to 700 mL of saline be instilled into the

bladder to maintain cord decompression. Once the bladder is filled, the vaginal hand may be removed. [13] [39]

With cord prolapse, the fetal prognosis is dependent on presentation, gestation, and the timing of diagnosis and management. Partial cord compression for <5 minutes may not be harmful. Complete occlusion for the same period or partial occlusion for a longer time is likely to cause death or severe central nervous system

damage to the fetus. The perinatal mortality rate from umbilical cord prolapse has fallen and ranges from 3.6 to 16.2%. [55] The cause of death for infants born after cord prolapse now seems to be related more to complications of prematurity and low birth weight than to intrapartum asphyxia per se. [59]

Inhibition of Labor

Tocolytic therapy may be indicated in the ED to prevent the progression of labor when fetal distress is noted, particularly that due to cord compression, and to treat preterm labor.

Preterm Labor

Preterm labor is defined as labor occurring prior to the completion of 37 weeks of gestation. It is a major cause of preterm delivery, a complication that affects approximately 1 in 10 births in the United States and one that continues to be a significant cause of neonatal morbidity and mortality. [6] Although a wide variety of treatments for the inhibition of labor have been advocated, including bedrest, intravenous (IV) fluids, ß-agonist agents such as terbutaline and ritodrine, magnesium sulfate ($MgSO_4$), prostaglandin inhibitors, calcium-channel blockers, and oxytocin receptor antagonists, there has been little improvement in neonatal outcome since the early 1980s.[6] [65] In addition, the efficacy of each of these regimens has been difficult to establish, owing in part to inconsistencies in establishing an accurate diagnosis of preterm labor and the lack of consensus as to what constitutes successful treatment or failure. [6]

The criteria for defining preterm labor vary among investigators. A presumptive diagnosis of premature labor may be made in the woman who is between 20 and 37 weeks of gestation in the presence of regular uterine contractions occurring at intervals of 5 to 8 minutes or less that are accompanied by one or more of the following: (1) progressive cervical effacement and dilation, (2) cervical dilation of >1cm, or (3) cervical effacement of 80% or more. [22] External monitoring devices, when available, are helpful by providing objective evidence of the character of uterine contractions, as well as the condition of the fetus.

Ideally, when preterm labor is suspected, obstetric consultation should be obtained and the patient transferred immediately to the labor and delivery area for monitoring and determination of fetal maturity. When appropriate obstetric facilities are not available, attempts to arrest labor may be initiated in the ED.

Tocolytic Therapy

Basic maneuvers to improve uterine and fetal status should be initiated before instituting pharmacologic tocolytic therapy when either preterm labor or fetal distress is suspected (Table 57-1). Because uterine hypoxia may induce uterine contractions, supplemental O_2, IV infusion of 500 mL of crystalloid, and assumption of the left lateral decubitus position should initially be attempted to improve uterine perfusion. [6] [22] [29] Because uterine, cervical, or urinary tract infections account for 20% to 40% of cases of preterm labor, a specific cause should be sought and treated as appropriate. [29] [52] [68] If contractions persist and cervical changes are documented, despite these basic interventions, pharmacologic therapy may be indicated. [6] [22] [29] Although tocolytic agents are commonly used and have been shown to prolong pregnancy by several days, there are little data to suggest that tocolysis improves long-term perinatal or neonatal outcome. [6] [22] [32] Therefore the principal benefit of pharmacologic therapy may be only to prolong pregnancy to allow maternal transfer to a tertiary care facility or delay delivery sufficiently to improve fetal maturation with corticosteroids.[6] [22] [40] [52] [68] Contraindications to tocolytic therapy include uncorrected fetal distress or fetal death, fetal maturity, chorioamnionitis, and complications requiring early delivery such as severe pregnancy-induced hypertension or antepartum vaginal bleeding. [8] [40] Tocolysis is probably most beneficial to infants between 24 and 28 weeks of gestation when even a short time gained in utero may substantially improve neonatal survival. [22] [40] In contrast, delivery of infants born before 24 weeks of gestation results in serious morbidity or mortality in almost 90%. [15] Similarly, prolonging gestation beyond 34 weeks provides little additional benefit to the nearly mature fetus.[6] [22] [40] Therefore, tocolytic therapy in these latter two groups should be considered carefully.

The most commonly used tocolytic agents in the United States are magnesium sulfate and the $ß_2$-receptor agonists ritodrine and terbutaline. Other agents such as calcium channel blockers, the prostaglandin inhibitor, indomethacin, and the oxytocin receptor antagonist, atosiban, have shown varying efficacy in clinical trials. [6] [22] [32] [40] [52]

TABLE 57-1 -- Drugs for the Emergency Management of Preterm Labor

$ß_2$-Receptor Agonists	
Ritodrine	
Dose	50 to 100 µg/min IV infusion ? 50 µg/min q 10 to 20 min
Endpoint	Cessation of uterine contractions
	Intolerable maternal side effects
	Maximum dose 350 µg/min
Terbutaline	
Dose	0.25 mg SC
	May repeat q 20 to 60 min
Endpoint	Cessation of uterine contractions
	Intolerable maternal side effects
Magnesium sulfate	
Dose	4 to 6 g IV over 20 min ? 2 to 4 g/hr IV infusion
Endpoint	Cessation of uterine contractions
	Signs of magnesium toxicity (e.g., respiratory depression, hypotension, somnolence)
Nifedipine*	
Dose	10 mg PO
	May repeat q 15 to 20 min
Endpoint	Cessation of uterine contractions
	Harmful maternal side effects, e.g., hypotension
	Maximum dose of 40 mg

PO, by mouth; SC, subcutaneously.

Sublinqual dose may cause excessive hypotension. It is best to avoid SL route.

*Variable doses have been used. Data on effects, particularly uteroplacental blood flow, is limited.

$ß_2$-Receptor agonists.

The traditional mainstays of pharmacologic tocolytic agents are the selective $ß_2$-adrenergic agents ritodrine (Yutopar) and terbutaline (Brethine, Bricanyl). Ritodrine

was approved by the U.S. Food and Drug Administration (FDA) for the inhibition of preterm labor in 1980 and is the only agent approved for tocolysis in the United States. Terbutaline has also been used extensively as a tocolytic agent and is frequently more readily available to the emergency clinician. [6] [22]

The ß-adrenergic agents prevent contraction of the myometrium through activation of the enzyme, adenyl cyclase. Adenyl cyclase enhances the conversion of adenosine triphosphate (ATP) to cyclic adenosine monophosphate (AMP), which, in turn, initiates a number of reactions that reduce the intracellular concentration of ionized calcium, thereby preventing activation of contractile proteins. [22] Although both ritodrine and terbutaline stimulate $ß_2$ -receptors primarily, both have some $ß_1$ activity that is responsible for their cardiovascular side effects.

Ritodrine is given as an IV infusion prepared by mixing 150 mg ritodrine in 500 mL fluid, preferably with 5% dextrose solution, yielding a final concentration of 0.3 mg/mL (i.e., 300 µg/mL). The initial infusion rate of ritodrine is 50 µg/min (i.e., 10 mL/hr), which is increased by 50 µg/min (i.e., by 10 mL/hr) every 10 to 20 minutes until uterine contractions cease, intolerable maternal side effects develop, or a maximal dose of 350 µg/min (i.e., 70 mL/hr) is reached. [52] [69] If labor is successfully arrested, IV therapy is continued for 12 to 24 hours after contractions cease. [69] A controlled infusion device is required. Ritodrine is contraindicated in patients with cardiac disease, pulmonary hypertension, hyperthyroidism, or uncontrolled diabetes. It should be used cautiously in patients on other sympathomimetic amines. [69]

The usual clinical side effects of ritodrine are related to its inherent activity as a ß mimetic drug. Maternal cardiovascular effects include a dose-related increase in heart rate, an increase in systolic pressure, and a decrease in diastolic blood pressure. Fetal heart rate also increases slightly. About one third of patients experience palpitations, and up to 15% have tremor, nausea, vomiting, headache, or erythema. Cardiac symptoms including chest pain or tightness, and arrhythmias are reported in less than 3% of patients. [69] Pulmonary edema resulting in maternal death has also been reported. [22] Side effects are usually self-limited and resolve with dosage reduction or discontinuation of the drug. Treatment of the majority of side effects is supportive; severe cardiovascular effects may be treated with ß-blocking agents. [69]

Although not approved for use by the FDA as a tocolytic agent, terbutaline is commonly used in the treatment of preterm labor. [22] [31] [52] When given subcutaneously, terbutaline is administered as a 0.25 mg dose, which may be repeated every 20 to 60 minutes until contractions cease or intolerable maternal side effects occur. [29] [72] Terbutaline is occasionally administered as an IV infusion. Terbutaline should be used with caution in patients with cardiovascular disease, hypertension, hyperthyroidism, diabetes, or seizures and in those on other sympathomimetic amines. [69] Side effects associated with the parenteral use of terbutaline are similar to those of ritodrine. [69]

Magnesium sulfate.

Magnesium sulfate ($MgSO_4$) is not approved in the United States for use as a tocolytic agent. Nevertheless, some perinatal centers prefer $MgSO_4$ over the ß-mimetic agents because of its lower incidence of side effects. [26] [29] [73] Although its mechanism of action is not fully understood, magnesium probably decreases myometrial contractility through its role as a calcium antagonist. [22]

When used as a tocolytic agent, 4 to 6 g of $MgSO_4$ is given IV over 20 to 30 minutes, followed by a maintenance IV infusion beginning at 2 to 4 g/hour. [26] [29] Because studies have questioned the efficacy of magnesium in prolonging labor, some authors have suggested increasing the maintenance infusion to 5 g/hr. [52] [73] Infusion of $MgSO_4$ typically produces sweating, warmth, and flushing. Rapid parenteral administration may cause transient nausea, vomiting, headache, or palpitations. [26] [52] Infrequently, pulmonary edema or chest pain has necessitated stopping the drug. [26] The major side effect of magnesium therapy is related to impairment of the muscles of respiration with subsequent respiratory arrest, an effect usually not seen until the serum magnesium level exceeds 10 mEq/L. The first sign of magnesium toxicity, decrease of the patellar reflex, typically occurs as serum magnesium levels exceed 4 mEq/L, with loss of the reflex as levels approach 10 mEq/L. Therefore, the patellar reflex should be monitored throughout therapy. Respiratory depression typically occurs when serum magnesium levels exceed 10 mEq/L. At levels of 12 mEq/L or greater, respiratory arrest may occur. [69]

Because magnesium is almost totally excreted by the kidney, it is contraindicated in the presence of renal failure and urinary output, and renal function should be monitored throughout therapy. If respiratory depression develops, 10 mL of a 10% solution of calcium gluconate or calcium chloride injected over 3 minutes is an effective antidote. For severe respiratory depression and arrest, prompt endotracheal intubation may be life saving.

Calcium channel blockers.

Smooth muscle activity, including that of the myometrium, is directly related to free calcium within the cytoplasm, and a reduction in calcium concentration inhibits contraction. Therefore, calcium channel blockers have become promising agents in the treatment of preterm labor. Studies comparing oral or sublingual nifedipine (Procardia, Adalat) with ritodrine indicate that nifedipine is more effective in suppressing preterm labor and is associated with significantly fewer neonatal intensive care (NICU) admissions, a lower incidence of neonatal morbidity, especially respiratory distress syndrome, and a lower incidence of maternal side effects. [57] [60] Although dosing regimens vary, nifedipine is frequently given as an initial dose of 10 mg orally or sublingually, repeated every 15 minutes until tocolysis is achieved or a maximum of 40 mg is reached. [57] [60] Oral nicardipine (Cardene), which has been shown to be a more potent inhibitor of muscle contraction in vitro, has also been shown to be an effective and safe tocolytic agent. [45] Because data on these agents are limited, some authors caution that more research is needed to clarify their potential maternal or fetal dangers, particularly the effect of calcium channel blocker-induced decreased vascular resistance, maternal hypotension, and thus decreased uteroplacental perfusion. [22] [52]

Prostaglandin inhibitors.

A number of prostaglandin synthesis inhibitors have been evaluated for their efficacy as tocolytic agents. Indomethacin is the most widely studied and has been demonstrated to offer short- and long-term benefits regarding pregnancy prolongation. Indomethacin is usually given as an initial rectal dose of 100 mg, followed by 25 mg

orally every 6 hours for up to 48 hours. Although it appears that indomethacin is an effective tocolytic agent, its administration is not without significant potential risk to the fetus, including premature closure of the ductus arteriosus, necrotizing enterocolitis, and intracranial hemorrhage. [22] [52]

Oxytocin antagonists.

Oxytocin is a potent stimulator of myometrial contractility. Atosiban is a selective oxytocin-vasopressin receptor antagonist capable of inhibiting oxytocin-induced uterine contractions in both animals and women with preterm labor. Several clinical trials have found atosiban to be an effective tocolytic agent without significant adverse maternal effects. [53] [67] [74] Further study is needed, including that of atosiban's effect on the fetus and newborn, before atosiban can be recommended for ED use. [22]

VAGINAL BLEEDING DURING THE THIRD TRIMESTER

Bleeding during the third trimester should always be considered an emergency. Profound shock secondary to exsanguinating hemorrhage may occur within minutes. Although bleeding may result from local vaginal and cervical lesions, genital lacerations, circumvallate placenta, vasa previa, or rupture of the uterus, the more typical causes are those due to placenta previa and placental abruption. *Placenta previa* refers to implantation of the placenta in the lower uterine segment with varying degrees of encroachment on the cervical os. Complicating approximately 1 in 180 to 1 in 390 deliveries, [22] placenta previa is classically characterized by vaginal bleeding with little or no abdominal or pelvic pain. Premature separation of the placenta, or *abruptio placentae,* refers to separation of the placenta from its site of implantation in the uterus before delivery of the fetus. Placental abruption occurs in about 1 in 200 deliveries and has a perinatal mortality of approximately 25%. [22] Although the clinical signs and symptoms with placental abruption can vary considerably, abruptio placentae is typically associated with varying degrees of abdominal pain and uterine irritability. [22]

As noted previously, third-trimester vaginal bleeding should always be considered an emergency. Stabilization should be initiated with large-bore IV access. Routine blood work should include a complete blood count with platelets and a type and crossmatch. If abruption is suspected, clotting studies including a fibrinogen level, and a toxicology screen for cocaine may be indicated because of the association of abruption with disseminated intravascular coagulation and cocaine abuse, respectively. Until the diagnosis of placenta previa is excluded digital vaginal examination is *contraindicated* because of the possibility of tearing or dislodging a placenta previa, which may result in profuse, potentially fatal hemorrhage. [22] The patient should be immediately transferred to the care of her clinician for further evaluation. The simplest and most precise method of placental localization is by transabdominal ultrasound, which has an accuracy of locating a placenta previa of about 96%.[22] In contrast, ultrasonography has limited sensitivity in detecting abruptio placenta with a reported negative predictive value of between 63% and 88% [22] ; therefore, negative findings on ultrasound should not be used to exclude placental abruption.

DELIVERY

Full dilation of the cervix signifies the second stage of labor, heralding delivery of the infant. Typically, the patient begins to bear down and, with descent of the presenting part, develops the urge to defecate. Uterine contractions may last 1.5 minutes and recur after a myometrial resting phase of less than 1 minute.

The mechanism of labor in vertex and breech presentations consists of engagement of the presenting part, flexion, descent, internal rotation, extension, external rotation or restitution, and expulsion (Fig. 57-7 (Figure Not Available)). The mechanism of labor is determined by the pelvic dimensions and configuration, the size of the fetus, and the strength of uterine contractions. Essentially, the fetus will follow the path of least resistance by adaptation of the smallest achievable diameters of the presenting part to the most favorable dimensions and contours of the birth canal.

The sequence of movements in vertex presentations is as follows:

1. *Engagement.* Usually occurring in the last 2 weeks of pregnancy in the primiparous patient and at the onset of labor in the multiparous patient, engagement refers to the mechanism by which the greatest transverse diameter of the head, the biparietal diameter in occiput presentations, passes through the pelvic inlet.
2. *Flexion.* Flexion of the head is necessary to minimize the presenting cross-sectional diameter of the head during passage through the smallest diameter of the bony pelvis. In most cases, flexion is necessary for both engagement and descent.
3. *Descent.* Descent is gradually progressive and is affected by uterine and abdominal contractions as well as by straightening and extension of the fetal body.
4. *Internal rotation.* Internal rotation occurs with descent and is necessary for the head or presenting part to traverse the ischial spines. This movement is essentially a turning of the head such that the occiput gradually moves from its original, more transverse position, anteriorly toward the symphysis pubis or, less commonly, posteriorly toward the hollow of the sacrum.
5. *Extension.* After internal rotation, the sharply flexed head reaches the anteriorly directed vulvar outlet, undergoing extension. With increasing distention of the perineum and vaginal opening, an increasingly larger portion of the occiput appears gradually. The head is born by further extensions as the occiput, bregma, forehead, nose, mouth, and finally chin pass successively over the anterior margin of the perineum. Immediately after its birth, the head drops downward such that the chin lies over the maternal anal region.
6. *External rotation.* External rotation or restitution follows delivery of the head as it rotates to the transverse position that it occupied at engagement. After this movement, the shoulders descend in a path similar to that traced by the head, rotating anteroposteriorly for delivery. First, the anterior shoulder is delivered beneath the symphysis pubis, followed by the posterior shoulder across the perineum. Expulsion of the remainder of the fetal body occurs with ease.

Figure 57-7 (Figure Not Available) Mechanism of labor for cephalic presentation. *A,* Before labor. *B*, Engagement, flexion, and descent. *C,* Internal rotation and extension. *D,* Extension to delivery of head. *E,* External rotation (restitution). *F,* Delivery of anterior shoulder. *G,* Delivery of posterior shoulder. Note that the head is being supported and guided in *F* and *G*. Traction is to be minimized. *(Adapted from Romney S, Gray MK, Little AB, et al [eds]: Gynecology and Obstetrics: The Health Care of Women. New York, McGraw-Hill, 1975.)*

The mechanism of the labor for breech presentations varies (Fig. 57-8). Usually the hips engage in one of the oblique diameters of the pelvic inlet. As descent occurs, the anterior hip generally descends more rapidly than the posterior hip. Internal rotation occurs as the bitrochanteric diameter assumes the anteroposterior position. Lateral flexion occurs as the anterior hip catches beneath the symphysis pubis, allowing the posterior hip to be born first. The infant's body then rotates, allowing engagement of the shoulders in an oblique orientation. There is gradual descent, with the anterior shoulder rotating to bring the shoulders into the anteroposterior diameter of the outlet. The anterior shoulder follows lateral flexion to appear beneath the symphysis, with the posterior shoulder delivered first as the body is supported. The head tends to engage in the same diameter as the shoulders. Subsequent flexion, descent, and rotation of the head occurs, bringing the posterior portion of the neck under the symphysis pubis. The head is then born in flexion.

Figure 57-8 Mechanism of labor for breech presentation. *A,* Before labor. *B,* Engagement of the buttocks, internal rotation. *C,* Lateral flexion of the trunk, delivery of the buttocks. *D,* External rotation of the buttocks, engagement of the shoulders. *E,* Internal rotation of the shoulders, delivery of the posterior shoulder. *F,* Lateral flexion of the trunk, delivery of the anterior shoulder. *(Adapted from Benson RC [ed]: Current Obstetric and Gynecologic Diagnosis and Treatment, 3rd ed. Los Altos, Calif., Lange Medical Publications, 1980.)*

Delivery of the vertex-presenting infant usually occurs spontaneously. The role of the clinician or attendant is principally to provide control of the birth process, preventing forceful, sudden expulsion or extraction of the infant with resultant fetal and maternal injury.

Equipment for Delivery

Prepackaged "precip trays" are available from several medical supply companies, although the majority of equipment can generally be assembled from existing emergency department stock and may be financially more practical.[50]

Essential equipment includes the following:

1 pair of scissors

2 medium Kelly clamps

1 bulb syringe

Sterile towels and blankets

Gauze sponges

Sterile gloves

Gown, mask, and protective eyewear

Optional equipment includes the following:

1 plastic umbilical cord clamp

1 large basin (for placenta)

1 ring forceps

Syringe and tubes for collection of cord blood

In addition, neonatal resuscitation supplies should be available in the ED as described subsequently.

Technique for Uncomplicated Delivery

Although complete sterility is not a priority, when time permits sterile technique should be used. Sterile gloves, a gown, mask and eye protection should be worn for protection of both the mother and the clinician. The perineum and vulva should be cleansed as described for vaginal examination and may be draped with sterile towels such that only the immediate area about the vulva is exposed. Care should be taken to avoid fecal contamination of the infant. Equipment in the obstetric pack should be sterile.

Ideally the patient should be placed on a delivery table in the dorsal lithotomy position to increase the diameter of the pelvic outlet. Alternatively, the patient may be positioned on a stretcher with her hips and knees partially flexed, her thighs abducted, and the soles of her feet placed firmly on the stretcher. The delivery position may be enhanced by placing the patient's buttocks on the underside of a sterile bedpan, which will provide additional space between the bed and the perineum.

Vertex Delivery

Spontaneous delivery of the vertex-presenting infant is divided into three phases: delivery of the head, delivery of the shoulders, and delivery of the body and legs.

Delivery should be anticipated when the presenting part reaches the pelvic floor. With each contraction, the perineum bulges increasingly and the vulvovaginal opening becomes more and more dilated by the fetal head. Just before delivery, "crowning" occurs; the head is visible at the vaginal introitus, and the widest portion, or biparietal diameter, of the head distends the vulva.

Gentle, gradual, controlled delivery is desirable and explosive delivery of the head should be avoided. Once the fetal head distends the vaginal introitus to 5 cm or more during a contraction, one palm of the clinician's hand is placed over the occipital area, providing gentle pressure to control delivery of the head. The other hand, preferably draped with a sterile towel to protect it from the anus, may exert forward pressure on the chin of the fetus through the perineum just in front of the coccyx in a modified Ritgen maneuver (Fig. 57-9). This maneuver extends the neck at the proper time, such that the smallest diameters of the head pass through the introitus and over the perineum, thereby protecting the maternal perineal musculature.

The head is gently supported during subsequent delivery of the forehead, face, chin, and neck.

After the head has been delivered, the infant's face and mouth should be quickly wiped and the oral cavity and nose suctioned with a bulb syringe. This minimizes the chance of aspiration of amniotic fluid, debris, and blood, which may occur with inspiration during delivery of the thorax. If meconium stained amniotic fluid is detected during delivery, use of wall suction with a large-bore (12 or 14 Fr) suction catheter or wall-mounted DeLee suction trap is preferable to a bulb syringe. [18] [61] [62]

With delivery of the neck, a finger should be passed around the neck to determine whether it is encircled by one or more coils of the umbilical cord (Fig. 57-10). If a cord is felt, it should cautiously be loosened and gently slipped over the infant's head. If this cannot be done easily, the cord should be doubly clamped and cut and the infant delivered promptly. In approximately 25% of deliveries, the umbilical cord is around

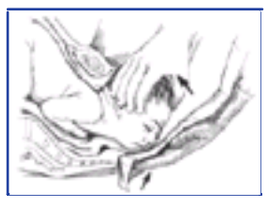

Figure 57-9 Modified Ritgen maneuver. *(From Pritchard JA, MacDonald PC: Williams Obstetrics, 16th ed. New York, Appleton-Century-Crofts, 1980.)*

the infant's neck but is rarely tight enough to cause fetal hypoxia. [22] [23]

Just before external rotation, the head usually falls posteriorly, bringing it almost into contact with the mother's anus. As rotation occurs, the head assumes a transverse position and the transverse diameter of the thorax or bisacromial diameter rotates into the anteroposterior diameter of the pelvis. In most cases the shoulders are born spontaneously. Delivery may be aided by grasping the sides of the head and exerting *gentle* downward (posterior) traction until the anterior shoulder appears beneath the symphysis pubis. The head is then *gently* lifted upward to aid the delivery of the posterior shoulder (see Fig. 57-7 (Figure Not Available)). The remainder of the body usually follows without difficulty.

If delivery of the body is delayed after the shoulders have been freed, delivery may be assisted by *moderate* traction on the fetal head accompanied by *moderate* pressure on the uterine fundus. Hooking the fingers in the axilla during delivery may result in brachial plexus injury and therefore should be avoided. Traction should always be exerted in the direction of the long axis of the infant. If applied obliquely, traction may

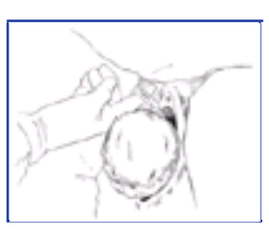

Figure 57-10 Checking for the cord around the infant's neck.

cause bending of the neck and excessive stretching of the brachial plexus. [22] [23]

Immediately after delivery the infant's airway should be thoroughly suctioned. Although some controversy exists as to the optimal timing and position of the infant in relation to the mother during this stage, most authorities recommend that the infant be placed at or slightly below the level of the vaginal introitus during suctioning, that is for approximately 30 seconds, before the cord is clamped. [22] [23] [79]

The umbilical cord is cut with scissors between two Kelly clamps placed 4 to 5 cm from the infant's abdomen. Later, an umbilical cord clamp is applied 2 to 3 cm from the infant's abdomen. Blood samples from the placental end of the cord can be collected for infant serology, including Rh determination. [22] [23]

Immediately after cutting the umbilical cord, the infant should be briefly evaluated and, if necessary, resuscitation should be initiated. Because of the relatively large surface area of the newborn, attention should be directed toward maintaining body temperature by drying the newborn and placing the baby in a heated isolette, under a radiant warmer, or in warm blankets. [18] [22] [23] [61]

Meconium-stained amniotic fluid (MSAF) occurs in approximately 8% to 17% of all deliveries and results in meconium aspiration syndrome in 2% to 6% of these

neonates. Meconium aspiration syndrome is associated with significant neonatal morbidity including respiratory failure. [51] [55] [62] To minimize the risk of aspiration, all infants with MSAF should have the nose, mouth and posterior pharynx thoroughly suctioned after delivery of the head but before delivery of the shoulders and the first thoracic expansion. If possible suctioning should be accomplished using wall suction and a large-bore (12 or 14 Fr) suction catheter or a wall mounted DeLee suction trap.[18] [61] [62] The traditional approaches to MSAF have included direct visualization and suctioning of all infants born through meconium-stained fluid and visualization and suctioning based on the thickness of the meconium. Evidence indicates, however, that tracheal suctioning of the vigorous newborn with MSAF does not improve outcome and may cause complications. [55] [61] [78] Therefore, immediately after delivery the infant should be evaluated for heart rate, respiratory effort, and muscle tone. If the infant has absent or depressed respirations, decreased muscle tone, or a heart rate less than 100 bpm, direct laryngoscopy should be immediately performed. Residual meconium is then suctioned from the hypopharynx under direct vision and intubation and suctioning of the trachea subsequently performed. [55] [61] [78] Initially vigorous neonates exposed to MSAF who later experience apnea or respiratory distress should also receive tracheal suctioning before positive-pressure ventilation is instituted.[55] [61] [78]

Delivery of the Placenta

Placental separation usually occurs within 5 minutes after delivery of the infant and may be recognized by the following signs:

1. The uterus becomes globular and firmer as it contracts.
2. There is a sudden gush of blood.
3. The uterine fundus rises in the abdomen as the separated placenta passes into the lower uterine segment and vagina, pushing the uterus upward.
4. The umbilical cord protrudes farther out of the vagina, indicating placental descent.

The placenta usually delivers spontaneously within 20 to 30 minutes. [29] Intra-abdominal pressure produced by the mother's bearing-down efforts may be enough to effect complete expulsion of the placenta. If maternal force alone is insufficient, delivery of the placenta may be aided by the clinician. After ensuring that the uterus is firmly contracted and placental separation has occurred, the clinician uses one hand to exert gentle pressure through the abdominal wall to lift the uterine fundus cephalad while keeping the umbilical cord slightly taut with the other hand. This is repeated until the placenta reaches the introitus, at which time uterine pressure is stopped and the placenta is gently lifted upward out of the vagina (Fig. 57-11). Membranes that are adherent to the uterine lining should be grasped with a clamp or ring forceps and removed by gentle traction. Placental expression should never be forced before placental separation nor should traction ever be used to pull the placenta out of the uterus. Both traction and premature placental expression may result in uterine inversion with catastrophic hemodynamic consequences. The placenta should be examined for completeness and saved for later evaluation by the clinician. [22] [23] [29]

The vulva, vagina, and cervix should be examined to assess for traumatic lacerations. Cervical lacerations most typically occur at the 9- or 3-o'clock position; vaginal lacerations typically occur at the point of the ischial spines. If found, these injuries are best repaired in the operating room or delivery suite. [29]

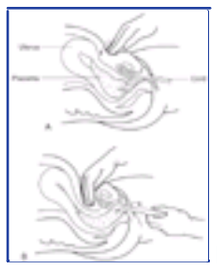

Figure 57-11 Delivery of the placenta. Gentle pressure is exerted through the abdominal wall to lift the uterine fundus upward while keeping the umbilical cord slightly taught. This maneuver is repeated until the placenta reaches the introitus.

After delivery of the placenta, the primary mechanism by which hemostasis is achieved at the placental site is through myometrial contraction. Agents such as oxytocin, methylergonovine and ergonovine may be used to stimulate myometrial contraction. Oxytocin (Pitocin, Syntocinon) is the most commonly used oxytocic drug and is usually given by IV infusion. Twenty units of oxytocin is added to 1 L of normal saline and is given at a rate of 10 mL/min for several minutes until the uterus remains firmly contracted and bleeding is controlled. The infusion rate is then reduced to 1 to 2 mL/min. [22] [23] [29] Alternatively, ergot derivatives such as methylergonovine maleate (Methergine), 0.2 mg, or ergonovine maleate (Ergotrate) 0.2 mg, may be given intramuscularly (IM). [22] [29] Because of their vasoconstrictive properties, ergot preparations are relatively contraindicated in patients with hypertension, including pregnancy-associated hypertension or preeclampsia. [22] Oxytocic agents should not be used before delivery of the placenta because the resultant uterine contraction may entrap the placenta or an undiagnosed twin within the uterus, the latter of which may prove fatal to the entrapped fetus.[22] [23]

Even when oxytocics are administered, the hour after delivery of the placenta is the time during which postpartum hemorrhage owing to uterine atony is the most likely to occur. Therefore, the uterus should be palpated frequently to ensure that it is well contracted. A normally contracted uterus will be firm with its upper margin just below the maternal umbilicus. If bleeding occurs from a flaccid uterus, gentle uterine massage through the abdominal wall should be performed. Occasionally, the placenta may fail to separate completely, resulting in a retained placenta or placental fragments, with persistent uterine bleeding. Manual removal of the placenta, exploration of the uterine cavity for retained products and, occasionally, hysterectomy are indicated. These procedures are beyond the scope of ED care and should be left to the admitting clinician. The patient should be supported with IV fluids and blood transfusion as indicated until definitive therapy is available. Constant firm uterine massage can lessen hemorrhage and may be life saving.

Complex Deliveries

Shoulder Dystocia

The term *shoulder dystocia* refers to impaction of the fetal shoulders in the pelvic outlet occurring after delivery of the head in vertex presentations. The reported incidence of shoulder dystocia ranges from 0.2% to slightly more than 4%. [9] [14] [28] Differences in reported rates of occurrence are due partly to the clinical variation in describing shoulder dystocia as well as the difficulty in retrospective retrieval of information from medical records. [9] [14] Shoulder dystocia is associated with several risk factors including fetal macrosomia, maternal diabetes or obesity, multiparity, and post-term pregnancy. These risk factors, however, are absent in up to 50% of cases, making antenatal identification of fetuses at risk difficult. [14]

Impaction of the fetal shoulders and thorax in the maternal pelvis prohibits adequate respiration, whereas compression of the umbilical cord frequently compromises fetal circulation, making shoulder dystocia a serious, and at times fatal, complication of delivery. [14] [23] Postpartum hemorrhage, usually from uterine atony, is the most common maternal complication. [22] Fetal complications include brachial plexus injuries, clavicular fractures, humeral fractures, and, infrequently, death. [9] [14] [28]

Management.

General anesthesia is desirable but is seldom available in the ED. A wide episiotomy may be used to reduce the incidence of major perineal lacerations and provide additional space for manipulation. Because the techniques used to treat shoulder dystocia frequently require an assistant, and delivery can result in fetal injury or hypoxia, assistance of specialist clinicians should be emergently sought. [14] [22] [64] Although reduction in the interval of time from delivery of the head to delivery of the body is of great importance to infant survival, overly vigorous traction on the fetal head or neck or excessive rotation of the body may seriously damage the infant. [9] [22]

Although a variety of techniques have been described to free the anterior shoulder from its impacted position beneath the symphysis pubis, most cases of shoulder dystocia can be resolved with 1 of 2 simple maneuvers. In the McRoberts maneuver, the mother is placed in the extreme lithotomy position with her hips completely flexed, thereby allowing her knees to rest on her chest. This causes a flattening of the lumbar lordosis and rotation of the maternal pelvis cephalad which frequently frees the impacted anterior fetal shoulder (Fig. 57-12).[9] [14] [22] [27] [30] If the McRoberts maneuver fails to effect delivery, moderate suprapubic pressure applied to the maternal abdomen by an assistant may be added while gentle downward traction is exerted on the fetal head. [14] [28] The "all-fours" or Gaskin maneuver has been described and consists of placing the mother on her hands and knees to effect delivery without the aid of additional maneuvers. [14] [17]

If these simple maneuvers fail to effect delivery, several other techniques exist, the choice of which will depend on clinician preference and experience. In the first maneuver, the clinician places two fingers in the vagina and exerts pressure on the fetal scapula, rotating the posterior shoulder 180° in a corkscrew fashion (reverse Wood's screw or Rubin maneuver). This may cause the impacted anterior shoulder to be released and delivery to progress (Fig. 57-13).[14] [64] Alternatively, delivery of the posterior arm may be attempted. Here, the clinician's hand is inserted along the hollow of the maternal sacrum to the level of the fetus' posterior elbow. By exerting pressure at the antecubital fossa the posterior forearm is flexed, the hand or forearm grasped, and the posterior arm of the fetus then carefully swept across its chest to effect delivery of the posterior arm and shoulder. The shoulder girdle is then rotated into one of the oblique diameters of the pelvis with subsequent delivery of the anterior shoulder (Fig. 57-14).[14] [22] If all of these strategies fail, controlled destructive procedures, such as fracture of the fetal clavicle or cephalic replacement (Zavanelli maneuver) with subsequent cesarean delivery, may be necessary. [22] [23]

Breech Delivery

When compared with cephalic presentations, the breech delivery is associated with a greater incidence of prematurity, prolapsed cord, low implantation of the placenta, uterine and congenital abnormalities, multiple pregnancies, and increased perinatal morbidity and mortality rates. [16] [21] [22] [65] The incidence of breech presentation varies inversely with gestational age and weight. At term, the incidence of breech presentation

Figure 57-12 McRoberts maneuver for shoulder dystocia. The maternal hips are completely flexed, allowing the knees to rest on the abdomen. This causes cephalad rotation of the maternal pelvis and flattening of the lumbar lordosis resulting in an increase in the useful size of the pelvic outlet.

is 3% to 4%; from 28 to 38 weeks' gestation, 17%; and at less than 28 weeks' gestation, 40%. [35]

Increased rates of prematurity and congenital anomalies associated with breech presentation account for much of the perinatal loss. [16] [19] [34] In addition, fetal distress also occurs more frequently. Whereas umbilical cord prolapse occurs in approximately 0.4% of cephalic presentations, the rate increases in breech presentation, particularly when the fetus is small or there is a non-frank breech. Occurring in 0% to 2% of frank breech presentations, the incidence of cord prolapse rises to 5% to 10.5% with complete breech presentation and to 8% to 28.5% with footling presentations. [19]

The increased use of cesarean section has greatly decreased the morbidity and mortality associated with breech delivery. Although cesarean section has been traditionally

Figure 57-13 Rubin or reverse Wood's screw maneuver for shoulder dystocia. *A,* Rotation of the posterior shoulder. *B,* Delivery of the rotated shoulder.

considered the standard of care, vaginal delivery may be the method of choice in carefully selected cases. [16] [25] [34] The emergency clinician seldom is called on to make the decision as to the most appropriate means of delivery, but rather could be faced with the imminent vaginal delivery of the breech infant. Breech delivery is most appropriately performed with both a clinician and an assistant present and preferably with specialist clinicians as well.

Types.

There are three types of vaginal breech delivery. Spontaneous breech is a breech delivery in which the infant is delivered spontaneously without any manipulation or traction other than supporting the infant. Although this form of delivery is rare with term infants, there is little associated traumatic morbidity. Partial breech extraction is when the infant is delivered spontaneously as far as the umbilicus

Figure 57-14 Delivery of the posterior arm for shoulder dystocia. *A,* Flexion of the posterior arm. *B,* delivery of the posterior arm to permit delivery of the anterior shoulder.

and the remainder of the body is extracted. Total breech extraction is when the entire body of the infant is extracted by the clinician.

Delivery is easier and perinatal morbidity and mortality are reduced when the breech is born spontaneously to the level of the umbilicus. [22] Similar to vertex presentations, the role of the clinician is to assist the mother in the birthing process, allowing maternal expulsive efforts to effect delivery of the infant. Premature or aggressive assistance or traction can significantly increase fetal and maternal morbidity.

To perform any vaginal breech delivery, the birth canal must be sufficiently large to allow passage of the fetus without trauma and the cervix must be completely effaced and dilated. If these conditions do not exist, a cesarean section is indicated. To ensure full cervical dilation in the footling or complete breech, it is important that the feet, legs, and buttocks advance through the introitus to the level of the fetal umbilicus before the clinician intervenes and further delivery is attempted. The mere appearance of the feet through the vulva is not in itself an indication to proceed with delivery. This may be a footling presentation through a cervix that is not completely dilated. In this case there may be time to transfer the patient to the labor and delivery suite, preferably in the knee-chest position to minimize the risk of cord compression.[23] Similarly, if the breech is frank, cervical dilatation and outcome is improved if the infant is allowed to deliver to the level of the umbilicus. Prior to this, as with complete and footling presentations, there may be time to effect safe transfer to the labor and delivery area, if available. [23] Tocolytics such as subcutaneous terbutaline may be used to inhibit labor until such patients can be safely transferred. [23]

Technique.

If assisted delivery of the frank breech becomes necessary in the ED, an episiotomy should be performed unless there is considerable perineal relaxation. As the breech progressively distends the perineum, the posterior hip will deliver, usually from the 6-o'clock position. The anterior hip then delivers, followed by external rotation to the sacrum anterior position (see Fig. 57-8). Continued descent of the fetus will allow delivery of the legs, which may be aided by splinting the medial thighs of the fetus with the fingers positioned parallel to the femur and exerting pressure laterally to sweep the legs away from the midline (Fig. 57-15). Following delivery of the legs, the fetal bony pelvis is grasped using both hands with the fingers resting on the anterior superior iliac crests and the thumbs on the sacrum (Fig. 57-16). Because the fetal body is slippery and difficult to hold, it may be wrapped in a towel to assist delivery. Maternal expulsive efforts are used in conjunction with continued gentle downward traction and rotation of the fetal pelvis to bring the fetal sacrum into the transverse position to effect delivery of the scapulae (Fig. 57-17).

Two methods of shoulder delivery are commonly used. In the first method, with the scapulae visible, the trunk is rotated such that the anterior arm and shoulder appear at the vulva and can be easily released and delivered. The body of the fetus is then rotated in the reverse direction to deliver the other shoulder and arm beneath the symphysis pubis. In the second method, if trunk rotation is unsuccessful, the posterior shoulder must be delivered first. The feet are grasped in one hand and drawn upward over the mother's groin. In this manner, leverage is exerted on the posterior shoulder, which slides out over the perineal margin, usually followed

by the arm and hand. The anterior shoulder, arm, and hand are then delivered beneath the symphysis pubis by downward traction on the fetal body (Fig. 57-18).

Occasionally, spontaneous delivery of the arm and hand does not follow delivery of the shoulder. If this occurs, upward traction of the fetal body should be continued after delivery of the posterior shoulder. Two fingers of the clinician are then passed along the fetal humerus until the fetal elbow

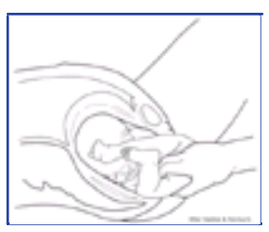

Figure 57-15 Assisted breech delivery. Once the breech has delivered to the level of the umbilicus, delivery of the legs may be aided by placing the fingers parallel to the medial thigh and sweeping the legs laterally away from the midline.

Figure 57-16 Assisted breech delivery. The clinician's hands are placed over the infant's sacrum to deliver the body. *(From Taylor ES: Obstetrics and Fetal Medicine, 2nd ed. Baltimore, Williams & Wilkins, 1977.)*

Figure 57-17 Assisted breech delivery. Rotation occurs as the scapulae emerge. *(From Taylor ES: Obstetrics and Fetal Medicine, 2nd ed. Baltimore, Williams & Wilkins, 1977.)*

is reached. The fingers are used to splint the fetal arm, which is then swept downward and delivered. The anterior arm may then be delivered by depression of the fetal body alone. In some cases it may be necessary to sweep the anterior arm down over the thorax using two fingers as a splint.

After the shoulders appear, the head usually occupies one of the oblique diameters of the pelvis, with the chin directed posteriorly. The head may then be extracted using the Mauriceau maneuver. With the fetal body resting on the clinician's palm and forearm, the index and middle finger of the hand are placed over the infant's maxilla, flexing the fetal head. Two fingers of the other hand are hooked over the fetal neck, and, grasping the shoulders, the clinician applies downward traction until the suboccipital region appears under the symphysis pubis (Fig. 57-19). As the body of the fetus is then elevated toward the mother's abdomen, the fetal mouth, nose, brow, and eventually occiput successively emerge over the perineum. Suprapubic pressure applied by an assistant is helpful in delivery of the head. If delivery of the head is not effected by the Mauriceau maneuver, forceps delivery may be necessary. Forceps application is beyond the scope of this text.

Rarely *breech extraction* of the infant becomes necessary and is indicated only if there is a definite diagnosis of fetal distress unresponsive to routine maneuvers, obstetric services are unavailable, and cesarean section cannot be performed promptly.

If extraction of the incomplete or complete breech is deemed necessary, the clinician's hand is introduced into the vagina and both feet of the fetus are grasped, with the index finger placed between the fetal ankles. Gentle traction is applied until the feet are pulled through the vulva (Fig. 57-20). Gentle downward traction is continued as successively higher portions of both legs and thighs are grasped (Fig. 57-21). When the breech appears at the vulva, gentle traction is applied until the hips are delivered. As the buttocks emerge, the fetal back usually rotates anteriorly. The thumbs

Figure 57-18 Assisted breech delivery. *A,* Delivery of the posterior shoulder by upward traction on the fetal body. *B,* Delivery of the anterior shoulder beneath the symphysis by downward traction. *(From Taylor ES: Obstetrics and Fetal Medicine, 2nd ed. Baltimore, Williams & Wilkins, 1977.)*

are then placed over the sacrum and the fingers over the hips and the remainder of the breech is delivered as described earlier (see Fig. 57-16 Fig. 57-17 Fig. 57-18 Fig. 57-19). At times, delivery of a frank breech may be necessary. Facilitated by an episiotomy, the breech should be allowed to deliver spontaneously as far as possible. Moderate traction may then be exerted by a finger placed in each fetal groin (Fig. 57-22). Once the knees appear outside the birth canal, the legs may be slowly flexed to assist delivery and delivery is accomplished as described earlier.

Complications.

The prognosis for the fetus in breech presentation is considerably worse than that in vertex presentations and is inversely related to the height of the presenting part at the beginning of breech extraction.[22] The major contributors to perinatal loss are preterm delivery, congenital anomalies, and birth trauma, with a high incidence of respiratory distress syndrome, high-grade intraventricular hemorrhage, and neurologic damage.[16] [19] [22] [34] Maternal morbidity is also increased as manipulations within the birth canal increase the risk of maternal infection, lacerations of the cervix, and uterine rupture.[22]

EPISIOTOMY

An episiotomy is an incision of the posterior vaginal wall and a portion of the pudenda, which is made to enlarge the vaginal introitus to permit easier passage of the fetus and, theoretically, to prevent perineal lacerations of the mother, preserving the structure and function of the vaginal introitus. Before the early 1990s, more than 60% of women in the United States received an episiotomy during childbirth, most as a routine procedure. [47] [70] Its use was justified by the theoretic prevention of severe perineal tears, better future sexual function, and a reduced incidence of urinary and fecal incontinence. [33] [47] Studies in the 1990s, however, determined that the routine use of episiotomy resulted in an equal or greater incidence of third- and fourth-degree perineal lacerations, postpartum

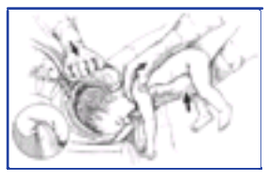

Figure 57-19 Mauriceau maneuver: delivery of the aftercoming head. While suprapubic pressure is applied by an assistant, the head is gently flexed by pressure on the maxilla. *(From Taylor ES: Obstetrics and Fetal Medicine, 2nd ed. Baltimore, Williams & Wilkins, 1977.)*

perineal pain, urinary incontinence, and dyspareunia. [10] [33] [41] [47] Therefore, the routine use of episiotomy has been abandoned for a more selective approach. Indications for selective episiotomy include breech delivery, shoulder dystocia, occiput posterior presentations, and imminent perineal tear. [22] [33]

Two types of episiotomy are used: the median or midline episiotomy and the mediolateral episiotomy (Fig. 57-23). The median incision is the easiest type of episiotomy to perform and repair, results in the least amount of blood loss, heals rapidly with minimal discomfort, and is generally preferred in the United States. [22] [23] The major disadvantage to the median

Figure 57-20 Breech extraction. Traction of the feet and ankles. Note that the index finger is placed between the ankles. *(From Pritchard JA, MacDonald PC: Williams Obstetrics, 16th ed. New York, Appleton-Century-Crofts, 1980.)*

incision is accidental extension of the incision into the anal sphincter or rectum, resulting in third- and fourth-degree lacerations, respectively. [22] [23] [42] [70] Although a mediolateral episiotomy seldom results in extension through the anal sphincter, blood loss is generally greater, repair is more difficult, and painful healing may result. [22] [23]

Figure 57-21 Breech extraction. Traction of the legs and thighs. *(From Taylor ES: Obstetrics and Fetal Medicine, 2nd ed. Baltimore, Williams & Wilkins, 1977.)*

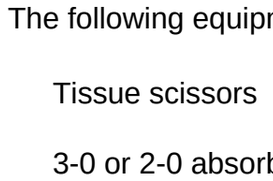

Figure 57-22 Breech extraction. Extraction of a frank breech by moderate traction exerted with a finger in each groin. *(From Pritchard JA, MacDonald PC: Williams Obstetrics, 16th ed. New York, Appleton-Century-Crofts, 1980.)*

Equipment

The following equipment is required for episiotomy and repair:

- Tissue scissors
- 3-0 or 2-0 absorbable suture on atraumatic needle (e.g., chromic catgut, polyglycolic acid)
- Needle holder
- Suture scissors
- Gauze
- Local anesthesia and injection equipment

Technique

The episiotomy should be timed so that it precedes trauma to the maternal tissues and fetus but avoids excessive maternal blood loss before delivery. With vertex presentations, the

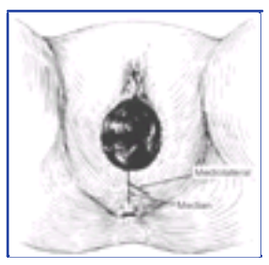

Figure 57-23 Types of episiotomy. The median episiotomy is the easiest to perform and repair.

episiotomy should be performed when the fetal head begins to distend the perineum and the caput becomes visible to a diameter of 3 to 4 cm during a contraction. [22] Anesthesia for episiotomy in the ED is usually limited to local infiltration of the perineum with 1% or 2% lidocaine.

The episiotomy is a simple incision that extends through the skin and subcutaneous tissues, the vaginal mucosa, the urogenital septum, and the superior fascia of the pelvic diaphragm, and if the episiotomy is mediolateral and deep, through the lowermost fibers of the puborectalis portion of the levator ani muscles. As the head crowns the clinician's index and second fingers are placed inside the vaginal introitus to expose the mucosa, posterior fourchette, and perineal body. Scissors are then used to incise the median raphe of the perineum almost to the anal sphincter (Fig. 57-24). For the mediolateral episiotomy, the incision is directed downward and outward in the direction of the lateral margin of the anal sphincter and may be either to the right or to the left (see Fig. 57-23).

After delivery of the infant and placenta, the episiotomy is repaired. The goals of episiotomy repair are to restore anatomy and achieve adequate hemostasis with a minimum of suture material. It is preferable to perform the closure after delivery of the placenta and after inspection and repair of the cervix and upper vaginal canal. The same principles of repair are followed for both the median (Fig. 57-25) and mediolateral episiotomies.

Because there is minimal tension on the closed wound, most authorities recommend the use of 2-0 or preferably 3-0 absorbable suture such as chromic catgut or polyglycolic acid on a large, atraumatic needle. The first step is to close the vaginal mucosa using a continuous suture from just above the apex of the incision to the mucocutaneous junction, reapproximating the margins of the hymenal ring. Burying the closing knot in the incision minimizes the amount of scar tissue and prevents tenderness and dyspareunia. Large actively bleeding vessels may require ligation during closure with separate

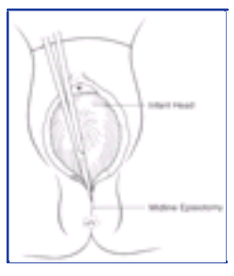

Figure 57-24 Midline (median) episiotomy.

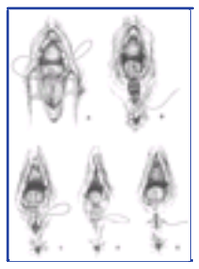

Figure 57-25 Closure of median episiotomy. *A,* Closure of mucosa and hymenal ring with continuous suture. *B,* Approximation of perineal musculature with interrupted sutures. *C,* Continuous suture to unite superficial fascia. *D,* Completion of repair by carrying continuous suture upward as a subcuticular stitch. *E,* Alternatively, closure of the superficial fascia and skin (*C* and *D*) may be accomplished by a series of loosely tied interrupted sutures. *(From Pritchard JA, MacDonald PC: Williams Obstetrics, 16th ed. New York, Appleton-Century-Crofts, 1980.)*

absorbable suture ligatures. Next, the perineal musculature is reapproximated with three or four interrupted sutures. Closure of the superficial layers may be accomplished by 1 of 2 methods. In the first method, a continuous suture is used to close the superficial fascia from the mucocutaneous junction outward and is then continued upward as a subcuticular skin closure, returning to and ending at the mucocutaneous junction. Alternatively, several interrupted sutures may be placed through the skin and subcutaneous fascia and loosely tied. This last method of skin closure avoids burying two layers of suture in the more superficial layers of the perineum.[22] [23]

The most common complication of episiotomy is hematoma formation, owing to inadequate hemostasis. A hematoma is treated by evacuation and drainage. Occasionally, large bleeding vessels will require delayed ligation. Postpartum episiotomy pain can usually be controlled by analgesics and local heat or sitz baths. Infection is an infrequent complication and it usually responds readily to sitz baths, good hygiene, and antibiotic therapy. Although most infections are not serious, examination is important to rule out deeper infection and necrotizing fasciitis. [37]

IMMEDIATE POSTPARTUM HEMORRHAGE

Postpartum hemorrhage is traditionally defined as maternal blood loss greater than 500 mL after delivery. Although this is the classic definition, ample evidence indicates that normal blood loss after vaginal delivery is frequently equal to or greater than 500 mL. [4] [22] [37] [66] Therefore, in clinical practice the diagnosis of postpartum hemorrhage is usually made when the amount of bleeding exceeds the clinician's estimate of "normal." The most common cause of serious obstetric hemorrhage, postpartum hemorrhage accounts for a significant proportion of maternal deaths. [4] Postpartum hemorrhage is conventionally divided into immediate hemorrhage occurring within 24 hours of delivery and delayed hemorrhage occurring nore than 24 hours after delivery.

Postpartum hemorrhage is frequently characterized by steady, moderate bleeding that persists until serious hypovolemia develops, rather than by sudden massive hemorrhage. Because of the relative hypervolemia that occurs during normal pregnancy, these cases of postpartum hemorrhage may remain unrecognized until a large volume loss has occurred.[66] Careful observation for blood loss, including evaluation of uterine size and consistency, is therefore needed during the early postpartum period.

The most common cause of immediate postpartum hemorrhage is uterine atony. Less common causes include lacerations of the vagina and cervix, retained placenta or placental fragments, coagulation disorders, uterine rupture, and uterine inversion. [4] [22] [37] [66]

Management

Management of postpartum hemorrhage consists of replacement of intravascular volume with crystalloid and blood products as needed, as well as therapy directed toward the cause of hemorrhage. The diagnosis of uterine atony, the most common cause of bleeding, is made when uterine palpation reveals a soft boggy uterine corpus. Although the diagnosis may be suspected on the basis of abdominal examination alone, bimanual pelvic examination is frequently necessary to confirm the diagnosis.

Uterine atony is initially managed with firm manual massage of the uterine fundus through the abdominal wall in conjunction with the administration of oxytocic agents (Table 57-2). If bleeding persists, bimanual uterine compression is indicated (Fig. 57-26). One hand of the clinician is used to compress and massage the posterior aspect of the uterus through the abdominal wall while the fist of the other hand is used to gently massage the anterior aspect of the uterus through the vaginal wall. Care must be taken to avoid vigorous downward massage, which can result in acute uterine inversion.

Oxytocics

Oxytocin is ideally administered as an IV infusion, which is prepared by adding 20 to 40 units of oxytocin to 1 L of crystalloid and infused at a rate of 200 to 500 mL/hour, titrated to sustain uterine contractions and control uterine hemorrhage. Oxytocin should not be administered as an IV bolus because profound hypotension and myocardial arrhythmias may occur.[22] If IV access is unavailable, 10 units of oxytocin may be given IM. [69] Because the onset of action of IV oxytocin is rapid, uterine contractions and therefore

TABLE 57-2 -- Drugs for the Management of Immediate Postpartum Hemorrhage

Oxytocin*	
Dose	20 to 40 units in 1 L crystalloid initially infused at 200 to 500 mL/hr ? titrate to sustain uterine contractions and control hemorrhage
Comments	Do not administer as IV bolus; If IV access unavailable, may use 10 U IM
	If IV access unavailable, may use 10 U IM
Methylergonovine maleate or Ergonovine maleate	
Dose	0.2 mg IM
Comments	Avoid in patients with hypertensive disease including preeclampsia
Carboprost tromethamine	
Dose	0.25 mg IM ? repeat q 15 minutes until uterine hemorrhage controlled or maximum dose of 2 mg
Comments	Concurrent use of antiemetics and antidiarrheals recommended to control side effects

IM, intramuscularly.

*Note increased concentration of oxytocin infusion when used for the treatment of postpartum hemorrhage compared with that given to stimulate uterine contractions after uncomplicated delivery.

slowing of hemorrhage should be observed within minutes of administration. If bleeding persists and the uterus remains boggy despite oxytocin therapy, an ergot derivative such as methylergonovine or a prostaglandin may be used. If methylergonovine (Methergine) is chosen to help stimulate uterine contractions, it is given as a 0.2 mg dose IM. Typically, uterine contractions occur within 2 to 5 minutes of administration and last for several hours. [69] Because of their tendency to cause vasoconstriction and severe hypertension in some patients, ergot preparations should be avoided in women with

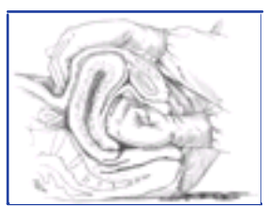

Figure 57-26 Uterine massage. One hand is used to massage the posterior aspect of the uterus through the abdominal wall. The other hand, inserted in the vagina, compresses the anterior uterus. *(From Pritchard JA, MacDonald PC: Williams Obstetrics, 16th ed. New York, Appleton-Century-Crofts, 1980.)*

hypertensive disease, including preeclampsia. [4] [69] Alternatively, 15-methyl prostaglandin F_{2a} (carboprost tromethamine [Hemabate]) may be used to stimulate uterine contractions and is preferred over methylergonovine by some authors. [4] [37] Approved for use in the control of uterine atony by the FDA in the mid-1980s, carboprost is administered in a dose of 0.25 mg IM, which may be repeated at 15 minute intervals as determined by the clinical course. The total dose should not exceed 2 mg. [66] [69] The rate of successful control of uterine hemorrhage with carboprost exceeds 85%. [58] Because nausea, vomiting, and diarrhea occur frequently in patients receiving prostaglandins, concurrent administration of antiemetic and antidiarrheal agents is recommended. [69] Misoprostol (Cytotec), an orally administered prostaglandin E_1 analogue approved for use in the treatment of gastric ulcers due to non-steroidal anti-inflammatory agents, has been found to effectively control postpartum hemorrhage in over 88% of women when administered rectally in a dose of 1000 µg. [1] [56]

Procedures

If vaginal bleeding persists despite uterine massage and a firmly contracted uterus, a cause other than uterine atony should be suspected. The labia, vagina, and cervix should be carefully inspected for lacerations. Bleeding from lacerations may be controlled by direct pressure or, in the case of cervical lacerations, by gentle application of ring forceps to the bleeding point. Absorbable sutures may be used to control bleeding from easily accessible lacerations. Because adequate visualization of the cervix and upper vagina is difficult and repair of extensive lacerations frequently requires general anesthesia, repair of these lacerations is often better left to the obstetrician.

Although rare, occurring in approximately 1 in 1700 to 6400 deliveries, *uterine inversion* is frequently thought to be the result of traction on the umbilical cord during the third stage of labor.[22] [66] [75] Other factors associated with the disorder include manual removal of the placenta, an increase in intra-abdominal pressure, placental abnormalities such as placenta accreta, and congenital weakness of the uterine wall or cervix. [4] [76] The principal signs and symptoms of the disorder are lancinating and, often violent pelvic pain, and excessive postpartum hemorrhage with up to 40% of patients developing shock. [4] [43] [75] [76] Diagnosis is made by visualization or palpation of the soft purplish-red mass of the uterus filling the vaginal vault or protruding through the introitus. On abdominal examination, no mass representing the uterus may be palpated or, when palpable, the uterus may have a cup-like dimpling of the fundus. [4] [43] [66] [76] Treatment is aimed at maintaining cardiovascular stability through the use of IV fluids and immediate repositioning of the uterine corpus. Conscious sedation is indicated and general anesthesia is often necessary, especially if the cervix has contracted. Tocolytic agents such as terbutaline or magnesium sulfate have been used successfully for uterine relaxation and repositioning. [4] [22] [76] Oxytocics should not be given until after the uterus is repositioned. [43] [66] There is controversy over management when the placenta is still attached to the uterus. Some advocate removal of the placenta before replacement of the uterus to decrease uterine bulk and allow for easier repositioning through the cervical ring. Others suggest that early placental removal increases uterine bleeding and may increase the risk of septic complications. [22] [43] [75] [76]

Repositioning of the uterus may be accomplished by inserting one hand into the vagina with the tips of the fingers at the uterocervical junction and the uterine fundus firmly held in the palm of the hand. Gentle pressure exerted with the fingers on the edges of the uterus closest to the cervix in the direction of the umbilicus is followed by gradual replacement of the corpus (Fig. 57-27). Pressure should not initially be exerted centrally on the fundus, because this will cause the uterus to be compressed, forcing more "layers" of the uterus to simultaneously lie within the relatively tight cervical ring. [43] [75] [76] Although replacement of the inverted uterus can usually be accomplished by vaginal manipulation, occasionally a dense cervical contraction ring is present, preventing repositioning. General anesthesia and laparotomy may be necessary for uterine repositioning.

PERIMORTEM CESAREAN SECTION

Perimortem cesarean section, one of the oldest and most dramatic surgical procedures known, has long been used as an attempt to preserve the life of the fetus. [46] Earliest records trace its origin back to 715 BC, when the legendary king of Rome, Numa Pompilious, decreed that the child be excised from the womb of any woman who died late in pregnancy. Under the emperors of Rome, the *caesars*, this law became known as the "lex caesare," and hence the name cesarean operation. [77]

From 1879 until 1986, there were 188 reports of perimortem cesarean section resulting in the delivery of a live infant. [38] Since that time, only isolated cases of long-term fetal survival have been reported. [44] [49] Because most of the literature on perimortem cesarean section involves only small numbers of cases with emphasis on those that are successful, accurate survival statistics are difficult to ascertain. When reported, infant survival rates range from 11% to 70%. [11] [38] [77] Because the potential for survival of a normal infant exists, cesarean delivery must be considered in any woman who suffers a cardiac arrest after 24 weeks' gestation and is unresponsive to brief resuscitation. [38] [44] Although the procedure is rare, the decision and its performance may fall to the emergency clinician.

Indications

Survival of the infant is directly related to the elapsed time from death of the mother to delivery, the maturity of the fetus, the performance of cardiopulmonary resuscitation (CPR) on the mother, and, in certain circumstances, the availability of neonatal intensive care facilities. [38] [44] [77] Although the lower limit of fetal viability varies among institutions, in general, performance of the procedure before the point of fetal viability at approximately 24 weeks is not indicated. [29] If the duration of gestation is not known from the history, fetal maturity may be quickly estimated by calculating gestational age on the basis of the date of the patient's last normal menstrual period or by measuring the height of the uterine fundus. Between 18 and 30 weeks' gestation, the age of the fetus in weeks will correspond to the distance in centimeters from the uterine fundus to the symphysis pubis (e.g., at 28 weeks' gestation the fundus is approximately 28 cm above the symphysis pubis or halfway between the umbilicus and the costal margin). [71] Criteria for intervention should be established prospectively at each institution and be in accordance with the institution's general neonatal policies. [29] [71]

Figure 57-27 Replacement of the inverted uterus.

The potential for infant survival decreases and the chance of neurologic damage increases as the time from maternal death (cessation of circulation) to cesarean section rises (Table 57-3). When cesarean section is performed within 5 minutes of death of the mother, neonatal outcome is generally considered excellent; from 5 to 10 minutes, good; from 10 to 15 minutes, fair; and from 15 to 20 minutes, poor. Because even under optimal conditions CPR results in a cardiac output of 30% to 40% of normal and placental perfusion may be severely compromised, every attempt should be made to begin cesarean delivery within 4 minutes of the cardiopulmonary arrest, completing the procedure within 5 minutes of arrest. [29] [38] [44] [71] Fetal prognosis is generally better after the sudden death of a previously healthy mother than after the death of a mother with a prolonged and debilitating illness. [38] [71] [77]

CPR should, of course, be initiated immediately on cardiac arrest of the mother and be continued until after delivery of the infant. The pregnant state produces certain physiologic

TABLE 57-3 -- Outcome of 61 Infants Who Survived Postmortem Cesarean Section from 1900 to 1985 as a Function of Time from Death to Delivery

Time (min)	No. of Patients (%)	Normal	Neurologic Sequelae
0– 5	42 (69)	42	0
6–10	8 (13)	7	1 (mild)
11–15	7 (11)	6	1 (severe)
16–20	1 (2)	0	1 (severe)
21–25	3 (5)	1	2 (severe)

From Katz VL, Dotters DJ, Droegemueller W: Perimortem cesarean delivery. Obstet Gynecol 68:571, 1986.

changes that adversely affect the adequacy of standard CPR. Vena caval occlusion by the gravid uterus hampers venous return and thus compromises maternal cardiac output. A decreased functional residual capacity of the lungs may impede ventilation. Manual displacement of the uterus away from the inferior vena cava may be attempted by an assistant. However, perimortem cesarean delivery in itself may represent the most important variable for a successful maternal resuscitation. [24] [38] [77]

Legal and Ethical Considerations

No clinician has been found liable for performing a perimortem cesarean section. Indeed, it is thought that the clinician has the legal right and responsibility to provide the fetus with every possible chance of survival when there is no hope of survival of the mother. [38] [71] Verbal permission for the operation should be obtained from the family when possible but not at the expense of delaying the procedure. Failure to obtain permission should not preclude cesarean section. [38] [71]

There is no standard of care relating to emergency clinicians performing a perimortem cesarean delivery. Each case must be individualized. Limited resources often place the clinician in the difficult situation of deciding whether to continue efforts to resuscitate the mother or to attempt to deliver the fetus in a difficult situation under less than ideal conditions. In the absence of obstetric backup immediately at hand, it is reasonable for the emergency clinician to proceed with delivery of the child if the mother cannot be resuscitated. Prolonged attempts to resuscitate the mother are unlikely to benefit either the mother or the fetus.

Equipment

The following equipment is needed for a perimortem cesarean section:

- Scalpel with a no. 10 blade
- Bandage scissors
- Bladder retractor

Large retractors (2)

Forceps

Lap or gauze sponges

Hemostats (curved and straight)

Suction

Obstetric pack (as described earlier for vaginal delivery)

Because of the rarity of this procedure in the ED, it is unlikely that a specific instrument pack will be available. Minimally, a scalpel with a large blade and an obstetric pack are necessary.

Technique

Perimortem cesarean section should be performed by the most experienced person present, preferably an obstetrician. When possible, a neonatologist should be in attendance. Their arrival, however, should not delay onset of the procedure. CPR should be initiated for the mother at the time of cardiac arrest and continued throughout the procedure. Although it is helpful if fetal heart tones are present premortem, time should not be wasted searching for them or attempting to evaluate fetal viability with abdominal ultrasonography.

Rapid extraction of the infant while avoiding fetal and maternal injury is the goal of the procedure. Hence, time should not be wasted preparing a sterile operating field or transporting the patient to an operating suite outside the ED. Using a large (e.g., no. 10) scalpel, a midline vertical incision is made through the abdominal wall extending from the symphysis pubis to the umbilicus and carried through all abdominal layers to the peritoneal cavity (Fig. 57-28). In most gravid women, the hyperpigmented "linea nigra" is apparent and may serve as a guide for the incision. If available, retractors are placed in the abdominal wound and drawn laterally to expose the anterior surface of the uterus. The bladder is reflected inferiorly; if it is full, it may be aspirated to evacuate it and permit better access to the uterus (Fig. 57-29). While avoiding injury to fetal parts, a small (approximately 5 cm) vertical incision is made through the lower uterine segment until amniotic fluid is obtained or until the uterine cavity is clearly entered (Fig. 57-30*A*). The index and long fingers are then inserted into the incision and used to lift the uterine wall away from the fetus. Bandage scissors are used to extend the incision vertically to the fundus until a wide exposure is obtained (Fig. 57-30*B*). The infant is then gently delivered, the mouth and nose suctioned, and the cord clamped and cut (Fig. 57-31). Because the incision is relatively high in the uterus, the infant's head may not be readily accessible to the clinician, in which case the infant's feet are grasped and the infant delivered through maneuvers similar to those of a breech delivery. Neonatal resuscitation should be carried out as necessary.

Because, in rare instances, relief of vena caval compression by the uterus improves maternal hemodynamics such

Figure 57-28 Cesarean delivery. A vertical incision is made through the abdominal wall from the level of the uterine fundus to the symphysis pubis.

Figure 57-29 Cesarean delivery. If available, retractors are used to expose the anterior surface of the uterus, and the bladder is retracted inferiorly.

that survival of the mother is possible, maternal pulses should be checked and CPR continued after delivery of the infant.

Conclusion

Perimortem cesarean section is a rarely performed but potentially life-saving procedure that should be considered in any woman in the third trimester of pregnancy who suffers irreversible cardiopulmonary arrest. Because neonatal survival is enhanced as the time from maternal death to delivery decreases (while the irreversible nature of maternal cardiac arrest becomes more apparent as resuscitative efforts progress), the decision to perform this procedure may be one of the most difficult that the emergency clinician makes. Once the decision is made to perform a cesarean section, it should be done as quickly as possible by the most experienced person present. CPR should be continued until after delivery of the infant. Although rare, maternal survival has been reported after perimortem cesarean section.

THE NEWBORN

Approximately 5% to 10% of newborns require some degree of active resuscitation at birth (e.g., stimulation to breathe) and approximately 1% to 10% of those born in hospitals require assisted ventilation. [61] Evaluation of the newborn begins before delivery with assessment of maternal well-being and gestational age, and with the recognition of fetal distress, as evidenced by meconium staining of the amniotic fluid, fetal bradycardia, or evidence of cord prolapse. Care of the newborn begins with delivery of the head when the nose and mouth are suctioned.

Newborn resuscitation can be divided into four categories of action: (1) basic steps including assessment and initial stabilization, (2) ventilation including bag-valve-mask or bag-tube ventilation, (3) chest compressions, and (4) administration of

Figure 57-30 Cesarean delivery. *A,* Small vertical incision is made with a scalpel through the lower uterine segment. *B,* Bandage scissors are used to extend the incision vertically to the fundus.

medications or fluids. Although most newborns require only basic steps such as suctioning, drying, stimulation, and perhaps supplemental oxygen, some require further intervention, the most crucial action of which is the establishment of adequate ventilation. [61]

Evaluation

Traditionally, the *Apgar scoring system*, applied at 1 and 5 minutes after birth, has been the standard of newborn evaluation (Table 57-4).[18] [22] In general, the higher the score, the better the condition of the infant. Although traditionally the 1-minute Apgar score has been used to indicate the need for immediate resuscitation, the decision to begin resuscitation should not be delayed until an Apgar score is obtained. Rather, resuscitation decisions are based on the clinical triad of respiration, heart rate, and color. [18] [61]

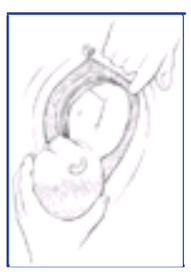

Figure 57-31 Cesarean delivery. The infant is delivered, the nose and mouth suctioned, and the cord clamped and cut.

Respiration.

Normally, the newborn begins to breath and cry almost immediately after birth. [22] After initial respiratory efforts, the newborn should be able to establish regular respirations sufficient to improve color and maintain a heart rate of more than 100 bpm. Gasping and apnea are signs indicating the need for assisted ventilation. [61] If initial respirations are shallow or slow, a brief period of stimulation may be attempted while 100% oxygen is administered. [18] [61] Establishing adequate ventilation and oxygenation will restore vital signs in the vast majority of newborns. [61]

Heart rate.

Heart rate may be determined by auscultation or by palpation of the pulse at the base of the umbilical cord. Heart rate should be consistently >100 bpm in an uncompromised newborn. If heart rate is <100 bpm, positive pressure ventilation with 100% oxygen should be initiated. If the heart rate is <60 bpm, despite adequate ventilation with 100% oxygen for 30 seconds, chest compressions should be initiated. [61] Because chest compressions may diminish the effectiveness of ventilation, they should not

TABLE 57-4 -- Apgar Scoring System

Sign	0	1	2
Heart rate	Absent	Slow (<100)	>100
Respiratory effort	Absent	Slow, irregular	Good, crying
Muscle tone	Flaccid	Some flexion of extremities	Active motion
Reflex irritability	No response	Grimace	Vigorous cry
Color	Blue, pale	Body pink, extremities blue	Completely pink

be initiated until lung inflation and ventilation have been established. [61]

Color.

An uncompromised newborn will be able to maintain a pink color of the mucous membranes without supplemental oxygenation. Central cyanosis is determined by examining the face, trunk, and mucous membranes and, if present, should be treated with supplemental oxygen. Acrocyanosis is usually a normal finding in the newborn and not a reliable indicator of hypoxemia. It may, however, indicate other conditions such as cold stress. [18] [61]

Equipment for Infant Stabilization

The following equipment is recommended for infant stabilization:

Bulb syringe or DeLee suction trap

Suction catheters (size 5 or 6 Fr, 8 Fr, and 10 or 12 Fr)

Endotracheal tubes (size 2.5 mm, 3.0 mm, 3.5 mm, and 4.0 mm)

Endotracheal tube stylet (optional)

Face masks (premature and newborn)

Feeding tubes (8 Fr) with 20 mL syringe

Heat source (radiant warmer or heating lamps)

Interosseous infusion needles

Laryngoscope handle

Laryngoscope blades (straight; sizes 0 and 1)

Laryngeal mask airway (optional)

Neonatal resuscitation bag with pressure-release valve or manometer

Oropharyngeal airways

Oxygen with flow meter

Umbilical vessel catheterization tray

Umbilical catheters (3.5 Fr and 5 Fr)

Wall suction with manometer

Warmed blankets

Stabilization Technique

Following delivery of the infant and cutting of the umbilical cord, the newborn should be placed on his or her side with the neck in a neutral or slightly extended position.[61] A rolled blanket or towel placed under the back and shoulders of the supine infant, thus elevating the torso 2 to 2.5 cm, may help in maintaining head position.[18] [61] Preventing heat loss in the newborn is vital because cold stress can increase oxygen consumption and impede effective resuscitation. [61] Hyperthermia, however, should be avoided because it is associated with perinatal respiratory depression. [55] [61] Placing the infant under a radiant warmer, rapidly drying the skin, and wrapping the infant in warmed blankets will reduce heat loss. [18] [61] Alternatively the mother's body may be used as a heat source.

If initial evaluation indicates the infant is stable, the infant may be dried and placed skin to skin on the mother's chest or abdomen and both covered with blankets. [18] [61]

Healthy vigorous newborns generally do not require suctioning after delivery. If suctioning is necessary secretions should be cleared first from the mouth and then the nose with a bulb syringe or suction catheter (8 Fr or 10 Fr). Because aggressive pharyngeal suctioning can cause laryngeal spasm and vagal bradycardia, mechanical suctioning should be limited in depth and duration, and negative pressures should not exceed 100 mm Hg. [61] Generally drying and suctioning are enough to stimulate effective respirations in the newborn. If effective spontaneous respirations are not established after drying with a towel or gentle rubbing of the back, flicking the soles of the feet may initiate spontaneous respiration. [61] If these maneuvers do not initiate spontaneous respirations, positive-pressure ventilation will be required. [18] [61]

Meconium aspiration is a significant cause of morbidity and mortality in newborns. Careful suctioning of the nose and mouth should be performed in all infants born through meconium-stained amniotic fluid. Intubation and tracheal suctioning is indicated for depressed infants as discussed earlier. [55] [61] [78]

Most newborns who require positive ventilation can be adequately ventilated with a bag and mask. Indications for positive-pressure ventilation include apnea or gasping respirations, heart rate <100 bpm, and persistent central cyanosis despite 100% oxygen. [18] [61] Assisted ventilations are performed at a rate of 40 to 60 per minute (30 breaths per minute if mechanical compressions are being performed). Typically higher inflation pressures (30 to 40 cm H_2O or greater) and longer inflation times are required for the first several breaths than for subsequent breaths. Visible chest expansion is probably a more reliable indicator of appropriate inflation pressures than any specific manometer reading. [18] [61] Because bag-valve-mask ventilation can produce gastric distention, which impedes respiration, an orogastric tube (8 Fr) should be inserted in infants undergoing prolonged positive pressure ventilation. [61]

Endotracheal intubation may be indicated when bag-valve-mask ventilation is ineffective, when tracheal suctioning

TABLE 57-5 -- Medications Commonly Used in Neonatal Resuscitations[61]

Drug	Dose	Indications	Comments
Epinephrine*	0.01–0.03 mg/kg (0.1–0.3 mL/kg of 1:10,000 solution)	Bradycardia, asystole	May be repeated every 3 to 5 minutes as indicated;
			Data regarding high-dose epinephrine in newborns inadequate to support routine use
Naloxone*	0.1 mg/kg (0.25 mL/kg of a 0.4 mg/mL solution or 0.1 mL/kg of a 1 mg/mL solution)	Respiratory depression induced by maternal narcotics during delivery	May be repeated every 2 to 3 minutes as needed
			May be given IM or SC if perfusion adequate
			May induce narcotic withdrawal in the newborn chronically exposed to narcotics and therefore relatively contraindicated
Volume expanders (normal saline or Ringer's solution)	10 mL/kg IV over 5 to 10 minutes	Suspected hypovolemia, shock or blood loss	May be repeated after determination of clinical response;
			Higher initial dose not recommended and may result in volume overload
Bicarbonate	1–2 mEq/kg of a 0.5 mEq/mL solution given over at least 2 minutes	Prolonged arrests unresponsive to other therapy	Not indicated during brief periods of CPR;
			Should be used only after adequate ventilation and perfusion have been established

CPR, cardiopulmonary resuscitation; IV, intravenously; SC, subcutaneously.
*May also be given via endotracheal tube.

for meconium is required, when chest compressions are performed, or when prolonged positive-pressure ventilation is required. [18] [61] Alternatively, the neonatal laryngeal mask airway may provide effective airway management, especially in the case of ineffective bag-valve-mask ventilation or failed endotracheal intubation. [61] Use of the laryngeal mask airway, however, has not been adequately studied in preterm infants and those with deliveries complicated by meconium-stained fluid. Its use, therefore, cannot be recommended in these situations. [61]

The heart rate should be monitored during the course of neonatal evaluation and stabilization with either direct auscultation over the chest or palpation of the pulse at the base of the umbilical cord. A readily discernible heartbeat of greater than or equal to 100 bpm is acceptable. If the heart rate is <60 bpm, despite adequate ventilation with 100% oxygen for 30 seconds, chest compressions should be instituted while ventilation is continued. [61] Chest compressions should be delivered on the lower one third of the sternum and not over the xiphoid to avoid damage to the liver. [18] There are two techniques for performing chest compression in the newborn. In the preferred method, two thumbs of the resuscitator's hands are positioned side by side over the lower one third of the sternum just below the nipple line. If the infant is large or the resuscitator's hands are too small to encircle the chest, two finger compressions using the ring and middle fingers may be used. [18] [61] The depth of compression should be approximately one third to one half of the anterior-posterior diameter of the newborn's chest, such that a palpable pulse is generated. [61] Compressions and ventilations should be coordinated to avoid simultaneous delivery, which may compromise the efficacy of ventilation. The compression to ventilation ratio should be 3:1 with 90 compressions and 30 breaths to achieve approximately 120 respirations per minute. [61] If the heart rate remains <60 bpm, despite these interventions, [61] an umbilical or IV line should be established and appropriate drug therapy initiated. Alternatively, intraosseous access can be used but may not be as effective in the preterm infant. [61] The medications most commonly used in neonatal resuscitation are listed in Table 57-5 .

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Chapter 58 - Culdocentesis

G. Richard Braen

A number of conditions require sampling of the intraperitoneal fluid to confirm a diagnosis or to obtain material for microbial culturing. This fluid can be obtained from the peritoneal cavity in a number of ways. Culdocentesis involves the introduction of a hollow needle through the vaginal wall into the peritoneal space. Culdocentesis is a simple, rapid, and safe procedure. The technique has several indications, but it is used primarily for diagnosing ruptured ectopic pregnancies and ruptured ovarian cysts and for obtaining fluid to aid in the culture diagnosis of pelvic inflammatory disease (PID). As beta subunit human chorionic gonadotropin (ß-hCG) assays have improved, and as the resolution of transvaginal ultrasonography has increased, the use of culdocentesis to diagnose ectopic pregnancies has decreased. In present practice, culdocentesis is used in situations where a ruptured ectopic pregnancy is suspected but a sonographic examination cannot be obtained.[15]

ANATOMY

Before culdocentesis is attempted, the clinician must be familiar with the anatomy of the vaginal and rectouterine pouch (pouch of Douglas). In the adult female, the vagina is approximately 9 cm long. From its inferior to its superior aspect, the posterior wall of the vagina is related to the anal canal by way of the perineal body, the rectum, and the peritoneum of the rectouterine pouch.[10] The rectouterine pouch and the posterior wall of the vagina are adjacent only at the upper quarter (approximately 2 cm) of the posterior vaginal wall. The vaginal wall in this area is <5 mm thick. The uterus lies nearly at a right angle to the vagina.

The blood supply of the upper vagina comes from the uterine and vaginal arteries, which are branches of the internal iliac artery. The area is drained by a vaginal venous plexus that communicates with the uterine and vesical plexuses. The vagina has its greatest sensation near the introitus and little sensation in the area adjacent to the rectouterine pouch.

The rectouterine pouch is formed by reflections of the peritoneum, and it is the most dependent intraperitoneal space in both the upright and the supine positions. Blood, pus, and other free fluids in the peritoneal cavity pool in the pouch because of its dependent location. This pouch separates the upper portion of the rectum from the uterus and the upper part of the vagina. The pouch often contains small intestine and, normally, a small amount of peritoneal fluid.

INDICATIONS

Culdocentesis is indicated in any adult female when fluid aspirated from the rectouterine pouch will help to confirm a clinical diagnosis. Ultrasound examination has largely replaced culdocentesis in the diagnosis of ruptured ectopic pregnancy and ruptured ovarian cysts, but the procedure still has an important role in emergency medicine. If ultrasound examination is not readily available in the emergency department (ED), or if the patient is too hemodynamically unstable to be transported to an off-site location for ultrasound, culdocentesis may be the fastest and most accurate diagnostic technique available to the emergency physician. [23] Analysis of peritoneal fluid is also a reliable method of differentiating inflammatory from hemorrhagic pelvic pathologic conditions. Conditions in which culdocentesis may be of diagnostic value include a ruptured viscus (particularly an ectopic pregnancy or a corpus luteum cyst), PID, and other intra-abdominal infections (particularly appendicitis with rupture or diverticulitis with perforation), intra-abdominal injuries to the liver or the spleen, and ruptured aortic aneurysms. [7]

Ectopic Pregnancy

Ectopic pregnancy is often one of the most difficult gynecologic lesions to diagnose. [14] The incidence of ectopic pregnancy is on the rise, accounting for 1.6% of all pregnancies. Ectopic pregnancy is the most common obstetrical cause of maternal death in the first trimester. [14] In a series of 300 consecutive cases of ectopic pregnancy, 50% of patients received medical consultation at least two times before the correct diagnosis was made. [3] In 11% of the patients in this series, the diagnosis was not made until the third medical visit. Because of the severe consequences of a ruptured ectopic pregnancy, an early, accurate diagnosis is desired.

The clinical picture of ectopic pregnancy may include vascular collapse, pelvic pain, amenorrhea or abnormal menses, shoulder pain, syncope, cervical or adnexal tenderness, adnexal mass, anemia, and leukocytosis. There is often a history of salpingitis, use of an intrauterine contraceptive device, or tubal ligation; however, no combination of these signs, symptoms, or historical data is diagnostic for an ectopic pregnancy. To confuse the diagnosis further, a normal menstrual history is reported in approximately 50% of patients with ectopic pregnancy and the *urine* pregnancy test is occasionally negative. [17] Although rarely seen, the combination of a uterine decidual cast (Fig. 58-1) and a positive pregnancy test are virtually pathognomonic of an ectopic pregnancy. A uterine cast is decidua that has been hormonally stimulated by the ectopic pregnancy but is passed vaginally when the tissue can no longer be supported. The cast is an outline of the uterine cavity, but it can be mistaken for products of conception if not inspected carefully. Therefore, all tissue passed vaginally should be carefully inspected before it is sent to the laboratory for analysis for products of conception.

The greater sensitivity of the serum and urine ß-hCG radioreceptor assay, coupled with the proliferation of trans-vaginal ultrasound and laparoscopy, have greatly increased the chances for early diagnosis of unruptured and ruptured ectopic pregnancy. [6] A sensitive and specific ß-hCG test must be used. The newer urinary ß-hCG tests (enzyme-linked or solid-phase immunoassay) provide sensitivity to 20 to 50 mIU/mL. Quantification of the serum test adds additional information being sensitive to 5 mIU/mL. Therefore, a negative *urine* ß-hCG test rules out pregnancy in greater than 98% of cases, and pregnancy in any site can be ruled out in virtually all patients with a negative *serum* ß-hCG test. [1] A single quantitative ß-hCG is a poor predictor of size of the pregnancy or the risk of ectopic pregnancy, but serial testing is quite helpful. It is expected that the quantitative serum ß-hCG level should double approximately every two days in the first trimester. Serum progesterone determinations

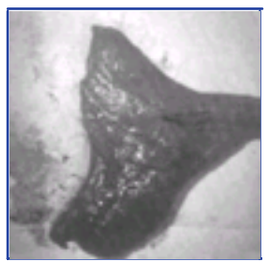

Figure 58-1 This decidual cast, a perfect outline of the uterine cavity, was initially thought to be products of conception when found in the vaginal vault of a pregnant woman treated for abdominal pain and vaginal bleeding. The initial diagnosis was a spontaneous abortion, but this cast is virtually diagnostic of an ectopic pregnancy. The woman later developed hypotension and was found to have a ruptured tubal pregnancy.

may also help to identify a normal or abnormal pregnancy. Although not infallible, a serum progesterone level of <10 ng/mL is usually associated with a nonviable intrauterine pregnancy or ectopic pregnancy, and a level >25 ng/mL is usually associated with a viable intrauterine pregnancy.

To increase the accuracy of diagnosis, it is helpful to combine quantitative ß-hCG testing with ultrasound examination. An empty uterus by transvaginal or abdominal ultrasound combined with certain quantitative serum ß-hCG results can be quite helpful to the clinician. The quantitative range in which the ultrasonographer should detect an intrauterine pregnancy varies, but an intrauterine pregnancy should be detected if the serum ß-hCG level is in the range of 1200 to 1500 mIU/mL when using a transvaginal probe, and if the level is >6500 mIU/mL when using a transabdominal probe. Endovaginal ultrasonic scanning consistently identifies 4-week gestational sacs if the ß-hCG levels =2000 mIU/mL. The presence of a fetal pole and cardiac activity are detectable with endovaginal ultrasound scanning at approximately 6 and 7 weeks, respectively. [8] It is important to note that the absence of an intrauterine pregnancy by ultrasound when the ß-hCG level is below the discriminatory zone is nondiagnostic and could represent an early viable normal pregnancy, a nonviable intrauterine pregnancy, or an ectopic pregnancy. When no intrauterine pregnancy is detected by ultrasound and the serum ß-hCG exceeds the discriminatory zone, the chance of an ectopic pregnancy ranges from 86% to 100%.[2]

Culdocentesis, for some patients, may play an important role in the diagnosis of ectopic pregnancy. The test has an accuracy rate of 85% to 95%. [3] [16] [26] Romero et al reported that an ectopic pregnancy was found in 99% of cases when a positive pregnancy test and a positive culdocentesis were present. [21] Although culdocentesis is most often positive in the presence of a frankly ruptured ectopic pregnancy, it may be diagnostic even in the nonruptured case when bleeding has been slow or intermittent. It is useful to note that many ectopic pregnancies leak varying amounts of blood for days or weeks before rupture. It is important that hemoperitoneum has been noted to occur in 45% to 60% of cases of *unruptured* ectopic pregnancy proved at surgery. [4] [24]

Hence, culdocentesis may be helpful in the stable patient whose ultrasound examination does not demonstrate an intrauterine pregnancy, despite a quantitative serum ß-hCG level in the appropriate range. Although some clinicians opt for outpatient monitoring of serial ß-hCG levels in this setting, patients for whom the clinician has a high suspicion for ectopic pregnancy (e.g., the patient who has or had significant discomfort) or for whom close follow-up cannot be ensured, are candidates for culdocentesis. [23] Although a negative culdocentesis does not rule out an early ectopic pregnancy, patients with a nondiagnostic ultrasound and a negative culdocentesis generally represent patients at lower risk for "rupture" of an ectopic pregnancy during outpatient serum ß-hCG monitoring. Patients with a nondiagnostic ultrasound examination and a serum ß-hCG level below the threshold at which an intrauterine pregnancy should be visible on the ultrasound examination also must be individualized. Those patients with significant pain, an unexplained low hematocrit reading, or postural vital sign changes (or near syncope) are candidates for culdocentesis.

Pelvic Infection

Acute PID has a polymicrobial etiology. [12] [19] [22] It has been common practice to define the etiology of PID by isolation of pathogens from the endocervix. The etiology is probably better defined by examination of the tubal flora. In fact, there is little correlation between cul-de-sac cultures and cervical cultures in PID. [5] Some medical centers routinely use aspirates obtained through culdocentesis to aid in determining the causative microbial agents for PID. [11] As the microbiology of PID becomes more complex and as the causative organisms develop resistance to antimicrobial agents, culdocentesis may evolve as one of the prime methods of obtaining meaningful microbiologic cultures that will dictate appropriate therapy.

Blunt Abdominal Trauma

Although diagnostic peritoneal lavage, computed tomographic (CT) scanning, and ultrasound examination are popular and valuable techniques to detect hemoperitoneum, the use of culdocentesis also has been advocated. [7] [13] Because small amounts of blood tend to collect in the rectouterine pouch, the aspiration of clear peritoneal fluid is of great potential value in *excluding* a diagnosis of hemoperitoneum. The procedure may be more advantageous than peritoneal lavage in some instances because there is less risk of urinary bladder perforation or bowel injury. In addition, previous abdominal surgery is not a relative contraindication to culdocentesis, as it is with peritoneal lavage. [20]

CONTRAINDICATIONS

The contraindications to culdocentesis are relatively few and include an uncooperative patient, a pelvic mass detected on bimanual pelvic examination, a nonmobile retroverted uterus,

TABLE 58-1 -- Correlation Between the Results of Culdocenteses Performed on 77 Patients with Ectopic Gestation and Various Clinical Parameters

Classic Triad	*Bleeding*	*Pain*	*Adnexal Mass*	Peritoneal Signs	Pulse =100/min	Blood Pressure <90/40 mmHg	Mean Hematocrit (%)	Hemoperitoneum =100 ml	Ruptured Tube	Total
Positive	37	54	10	26	19	9	35	52	30	54
Negative	8	8	3	1	1	0	39	0	0	8
Inadequate	13	15	6	5	4	1	38	13	7	15
Total patients	58	77	19	32	24	10	65	37	77	

Note: There is a lack of correlation between positive culdocentesis and peritoneal signs and changes in vital signs. Patients are grouped by culdocentesis result (i.e., positive, negative, or inadequate). Note that only 10 patients were hypotensive and only 24 experienced tachycardia.

From Cartwright PS, Vaughn B, Tuttle D: Culdocentesis and ectopic pregnancy. J Reprod Med 29:88, 1984. Reproduced by permission.

and coagulopathies. Pelvic masses may include tubo-ovarian abscesses, appendiceal abscesses, ovarian masses, and pelvic kidneys. It has been suggested that the only major risk with the procedure is that of rupturing an unsuspected tubo-ovarian abscess into the peritoneal cavity. This can be avoided by careful bimanual pelvic examination to exclude patients with large masses in the cul-de-sac.[5] Although there are no data to guide the age at which culdocentesis may be safely performed, the procedure is generally limited to patients who are beyond puberty. This limitation is suggested on the basis of anatomy and with the consideration that the procedure is difficult to perform through a small, prepubertal vagina.

One should not forgo culdocentesis due to the absence of classic signs anticipated with ectopic pregnancy. It is important to note that a positive culdocentesis does not consistently correlate with peritoneal irritation, blood pressure, pulse rate, or the actual volume of hemoperitoneum.[4] In fact, bradycardia in the presence of significant intraperitoneal bleeding from a ruptured ectopic pregnancy is not unusual (Table 58-1 and Table 58-2).

EQUIPMENT

The equipment required for culdocentesis is listed in Table 58-3 . Either an 18-ga spinal needle or a 19-ga butterfly needle held by ring forceps is acceptable. It may be helpful to anesthetize the posterior vaginal wall at the site of the puncture with 1% to 2% lidocaine with epinephrine administered through a 27- or 25-ga needle. Some physicians use a cocaine-soaked cotton ball to anesthetize the mucosa before infiltration with a local anesthetic. Although local anesthesia is often unnecessary (because *puncture of the posterior vaginal wall at the upper one fourth of the vagina is generally no more painful than a venipuncture*), there is some advantage to use

TABLE 58-2 -- Correlation Between Tubal Status and Hypotension, Tachycardia, Hematocrit, Signs of Peritoneal Irritation, and Hemoperitoneum in 77 Patients with Ectopic Gestation

Culdocentesis	Culdocentesis Positive	Peritoneal Signs	Blood Pressure <90/40 mm Hg	Pulse =100/min	Hemoperitoneum =100 mL	Average Hematocrit (%)
Ruptured (n = 37)	30	25	8	19	37	33.6
Intact (n = 40)	24	7	2	5	28	37.3
Total patients	54	32	10	24	65	

Note: Culdocentesis is frequently positive in the absence of rupture. Patients are grouped by the presence (i.e., "ruptured") or absence (i.e., "intact") of hemoperitoneum. Note that only about one half of patients with "ruptured" status had tachycardia.

From Cartwright PS, Vaughn B, Tuttle D: Culdocentesis and ectopic pregnancy. J Reprod Med 29:88, 1984. Reproduced by permission.

of a local anesthetic if multiple attempts at culdocentesis are required, as is sometimes the case. In addition, the epinephrine may produce vasoconstriction and may reduce bleeding associated with the needle puncture. Culdocentesis is often stressful to the patient, and all attempts should be made to render the procedure as painless as possible. Consideration of parenteral analgesia and sedation (see Chapter 34) should also be made when the patient is uncomfortable or anxious.

TECHNIQUE

Preparation

Culdocentesis is an invasive procedure that, in some hospitals, requires a written, witnessed, and signed consent form from the patient, parent, or guardian when the patient's condition permits. If verbal consent is obtained, this action should be witnessed and a notation in the medical record made documenting that the procedure was described, complications were discussed, and any alternatives (e.g., sonography, immediate laparoscopy) were offered when appropriate.

Once written or verbal consent is obtained, the patient is placed in a lithotomy position with the head of the table slightly elevated (reverse Trendelenburg position) so that intraperitoneal fluid gravitates into the rectouterine pouch. The patient's feet are placed in stirrups. In selected patients, some physicians premedicate with intravenous (IV) opioids or sedatives. The administration of nitrous oxide analgesia also is an accepted practice. When nitrous oxide is used during this procedure, it is important to document the presence of a chaperone, as some patients may develop sexual delusions under this agent. Although pain associated with culdocentesis needle passage is generally minor, the judicious use of analgesia and sedation makes the procedure easier for both physician and patient.

TABLE 58-3 -- Equipment for Culdocentesis

Adjustable examination table with stirrups
Bivalve vaginal speculum
Uterine cervical tenaculum
19-ga butterfly needle or 18-ga spinal needle
27- or 25-ga needle (for local anesthetic infiltration)
Ring sponge forceps
Syringes (20 mL)
Surgical preparation (iodinated, such as povidone-iodine)
Sterile water, cotton balls, 4 × 4 gauze sponges
Cocaine (10% solution) or benzocaine (20% solution)
Lidocaine (1%) with epinephrine
Culture media or test tube without anticoagulant

Radiographs, when indicated in the *stable* patient, are taken *prior* to culdocentesis, to avoid possible confusion if a pneumoperitoneum is detected following the procedure.

Exposure

A bimanual pelvic examination must be performed before culdocentesis to rule out a fixed pelvic mass and to assess the position of the uterus. Palpation of an adnexal mass is possible if the mass exceeds 3 cm in diameter. The examiner then inserts the bivalve vaginal speculum and opens it widely by adjusting both the height and the angle thumbscrews. The *posterior* lip of the cervix is grasped with the toothed uterine cervical tenaculum, and the cervix is elevated (Fig. 58-2). Grasping the cervix may produce minor discomfort, and the patient should be warned in advance of a possible sharp pain during this part of the examination. Bleeding from the tenaculum puncture site or culdocentesis site, or both, may produce postprocedure spotting, and the patient should similarly be informed.

Use of the tenaculum elevates a retroverted uterus from the pouch, exposes the puncture site, and stabilizes the posterior wall during the needle puncture. Some physicians prefer to use longitudinal traction on the cervix to produce the same result. The vaginal wall adjacent to the rectouterine pouch will be tightened somewhat between the inferior blade of the bivalve speculum and the elevated posterior lip of the cervix. This tightening of the vaginal wall exposes the puncture site and keeps it from moving away from the needle when the wall is punctured.

After the tenaculum is applied and the posterior lip of the cervix is elevated or traction is applied, the vaginal wall in the area of the rectouterine pouch should be swabbed with surgical preparation followed by a small amount of sterile water. Local anesthesia (1% lidocaine with epinephrine) may be administered at this point. Anesthesia may be injected with a separate 27- or 25-ga needle or by the spinal needle to be used for the culdocentesis. A cotton ball soaked in cocaine or benzocaine solution can be used for topical anesthesia of the posterior vaginal wall before infiltration with a local anesthetic. The needles used for both the local anesthetic and the puncture should be attached to a 20-mL syringe. A smaller syringe may not be long enough to allow adequate control of the needle, and the physician's hand may block the view of the puncture site if a smaller syringe is used.

Aspiration

Following local anesthesia, the syringe and the spinal needle to be used for the culdocentesis, are advanced parallel to the lower blade of the speculum. It is helpful to fill the syringe with 2 to 3 mL of saline (nonbacteriostatic) before puncture. Following needle puncture, the free flow of the fluid from the syringe expels tissue that may have clogged the needle and confirms that the needle tip is in the proper position and is not lodged in the uterine wall or the intestinal wall. Saline (rather than air) is preferred, because if air is used, one must be careful in interpreting the presence of free peritoneal air on subsequent radiographs. To avoid the need to change the syringe during the procedure, 1% lidocaine may be used for both anesthesia and confirmation of proper needle placement, but the bacteriostatic property of this agent precludes its use if the procedure is performed to obtain fluid for culture.

The vaginal wall should be penetrated in the midline 1 to 1.5 cm posteriorly (inferiorly) to the point at which the vaginal wall joins the cervix (Fig. 58-3).[25] The needle should penetrate a total of 2 to 2.5 cm.[18] [25] Gentle suction is then applied with the syringe while the needle is slowly withdrawn. It is important for the physician to avoid aspirating any blood that has accumulated in the vagina from previous needle punctures or from cervical bleeding because this may give the false impression of a positive tap. Bleeding from the puncture site in the vaginal wall is minimized if epinephrine is added to the local anesthetic.

Blood or fluid may be obtained immediately but may also be obtained just before the needle is withdrawn from the peritoneal cavity. Therefore, it is important to aspirate throughout the gradual withdrawal procedure. If no fluid is aspirated, the needle should be reintroduced and directed only slightly to the left or right of the midline. Directing the needle too far laterally may result in puncture of mesenteric or pelvic vessels. It is important to note that *if no fluid is obtained on the first attempt, the procedure should be repeated*.

Some physicians prefer the use of a 19-ga butterfly needle held with a ring forceps (Fig. 58-4). [25] This technique offers a built-in guide to needle depth and allows for good control of the needle during puncture. An assistant must aspirate the tubing while the physician controls positioning and withdrawal of the needle.

Fluid that is aspirated may be old, nonclotting blood; bright red blood; pus; exudate; or a straw-colored serous liquid. Any fluid that is not blood should be submitted for Gram staining, aerobic and anaerobic culture, and cell counts. Blood should be observed for clotting. Blood should also be sent for a hematocrit determination.

INTERPRETATION OF RESULTS

An interpretation of the results of culdocentesis depends primarily on whether any fluid was obtained. In the absence of a pathologic condition, one will often aspirate 2 to 3 mL of clear, yellowish peritoneal fluid. When there is no return of fluid of any type (a so-called dry tap), the procedure has *no diagnostic value*. Because a dry tap is nondiagnostic, it should not be equated with normal peritoneal fluid. In addition, when less than 2 mL of clotting blood is obtained, this is also considered to be a nondiagnostic tap, because the source of this small amount of blood may be the puncture site on the vaginal wall. Such blood will usually clot. More than 2 mL of *nonclotting blood* is certainly suggestive of hemoperitoneum. However, some researchers interpret as little as 0.3 mL of *nonclotting* blood as a positive tap.[4] There is no particular

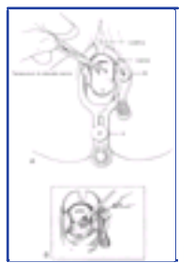

Figure 58-2 *A,* Preparation for culdocentesis. Note that one opens the speculum widely by using both the height *(A)* and the angle *(B)* adjustments. The cervix is grasped on the posterior lip with a toothed tenaculum. X marks the site for puncture of the vaginal wall. *B,* The use of a butterfly needle for culdocentesis. The needle is inserted 1 cm posterior to the point at which the vaginal wall joins the cervix. *(A, From Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. B, From Webb MJ: Culdocentesis. JACEP 7:452, 1978.)*

significance to larger amounts of blood, because absolute volume may be related to the needle position or the rate of bleeding. Brenner and coworkers [3] reported no blood from culdocentesis in 5% of patients with proven ectopic pregnancies, even when rupture had occurred. In the series of 61 patients with surgically proven ectopic pregnancy reported by Cartwright and colleagues, culdocentesis performed within 4 hours of surgery was positive in 70%, negative in 10%, and inadequate in 20%.[4] "Positive" in their series was defined as obtaining at least 0.3 mL of nonclotting blood with a hematocrit of greater than 3%. "Negative" was defined as obtaining 0.3 mL of fluid with a hematocrit of less than 3%. An "inadequate" tap was one in which no fluid was obtained. In the 252 surgically proven ectopic pregnancy patients having culdocentesis reported by Vermesh and others, 83% had a positive tap. [24] They defined a positive tap as nonclotting blood with a hematocrit of > 15%.

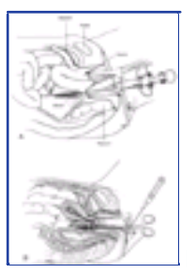

Figure 58-3 The needle is advanced parallel to the lower blade of the speculum. Aspiration is continued throughout the gradual withdrawal of the needle. *A,* The use of a spinal needle. *B,* the use of a butterfly needle and ringed forceps. *(A, from Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. B, from Webb MJ: Culdocentesis. JACEP 7:452, 1978.)*

Because culdocentesis is usually used to diagnose an ectopic pregnancy, a "negative tap" is one that yields pus or clear, straw-colored peritoneal or cystic fluid. A large amount of clear fluid (> 10 mL) indicates a probable ruptured ovarian cyst, aspiration of an intact corpus luteal cyst, ascites, or possibly, carcinoma. The significance of these fluids and the interpretation of results are outlined in Table 58-4 and Table 58-5 . Elliot and associates[9] cautioned that obtaining greater than 10 mL of clear fluid should not automatically rule out an ectopic pregnancy because the latter may coexist with other pathologic conditions.

A "positive tap" is one in which nonclotting blood is obtained, although the presence of nonclotted blood does not confirm a tubal pregnancy. Intraperitoneal blood from any source (ectopic pregnancy, ovarian cyst, ruptured spleen) may remain unclotted after aspiration for days in the syringe as a result of the defibrination activity of the peritoneum. The return of a serosanguineous fluid also suggests a ruptured ovarian cyst. The hematocrit of blood from active intraperitoneal

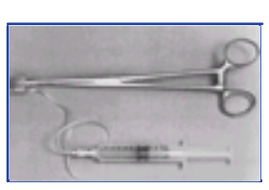

Figure 58-4 Culdocentesis may be performed with a 19-ga butterfly needle held with ring forceps. The ringed forceps grasp the wings of the butterfly needle to direct its placement. An assistant aspirates for fluid through the proximal end of the tubing. *(From Webb MJ: Culdocentesis. JACEP 7:451, 1978.)*

bleeding is greater than 10%. In one series, the hematocrit of blood from a ruptured ectopic pregnancy was =15% in 97% of cases. [3]

It should be emphasized that a positive culdocentesis in the presence of a positive pregnancy test does not always prove an ectopic pregnancy. [24] A ruptured corpus luteum cyst in the presence of an intrauterine pregnancy test is probably the most common cause of a false-positive scenario. When possible, ultrasound may help corroborate the culdocentesis findings.

TABLE 58-4 -- Interpretation of Culdocentesis Fluid

Aspirated Fluid	Condition and Suggested Differential Diagnosis
Clear, serous, straw-colored (usually only a few milliliters)	Normal peritoneal fluid
Large amount of clear fluid	Ruptured or large ovarian cyst (fluid may be serosanguineous); pregnancy may be coexistent
	Ascites
	Carcinoma
Exudate with polymorphonuclear leukocytes	Pelvic inflammatory disease
	Gonococcal salpingitis
	Chronic salpingitis
Purulent fluid	Bacterial infection

	Tubo-ovarian abscess with rupture
	Appendicitis with rupture
	Diverticulitis with perforation
Bright red blood*	Ruptured viscus or vascular injury
	Recently bleeding ectopic pregnancy* (ruptured or unruptured)
	Bleeding corpus luteum
	Intra-abdominal injury
	Liver
	Spleen
	Other organs
	Ruptured aortic aneurysm
Old, brown, nonclotting blood	Ruptured viscus
	Ectopic pregnancy with intraperitoneal bleeding over a few days or weeks
	Old (days) intra-abdominal injury (e.g., delayed splenic rupture)

*Note: The hematocrit of blood from a ruptured ectopic pregnancy is usually =15% (97.5% of cases), but some authors use >3% as positive.

COMPLICATIONS

Culdocentesis is one of the safest procedures performed in the emergency setting, and there are probably fewer complications with this technique than with peripheral venous cannulation. Complications have been reported, the most serious being rupture of an unsuspected tubo-ovarian abscess. [25] Other complications include perforation of the bowel, perforation of a pelvic kidney, and bleeding from the puncture site in patients with clotting disorders. Because the most common

TABLE 58-5 -- Interpretation of Culdocentesis

Positive
>0.5 mL nonclotting, bloody fluid (hematocrit >12%)
Indicates hemoperitoneum
When ß-hCG also positive, ectopic pregnancy found in >95%
Nonspecific—can occur in intrauterine pregnancies and nonpregnant women (e.g., ruptured cyst, retrograde bleeding)
Does not necessarily indicate tubal rupture
50%–62% of ectopic pregnancies with peritoneal blood may be unruptured
Negative
Serous fluid
Excludes hemoperitoneum and tubal rupture
False negative in 10%–15% of ectopic pregnancies (generally unruptured)
Nondiagnostic
Dry tap or clotting blood
Excludes neither ectopic pregnancy nor hemoperitoneum
15% of procedures are nondiagnostic
16% of ectopic pregnancies have nondiagnostic study results

From Brennan DF: Ectopic pregnancy: II. Diagnostic procedures and imaging. Acad Emerg Med 2:1090, 1995.

complications result from the puncture of a pelvic mass, careful bimanual examination of the patient should help prevent this problem. Puncture of the bowel and the uterine wall occurs relatively frequently, but this occurrence does not generally result in serious morbidity. Obviously, penetration of the gravid uterus has greater potential for harm. Occasionally, one will aspirate air or fecal matter, confirming inadvertent puncture of the rectum. Although this may be disconcerting, it is seldom of serious clinical concern and requires no immediate change in therapy.

CONCLUSION

In the emergency setting, culdocentesis can be a helpful diagnostic procedure. In an unstable patient with a strong clinical scenario for a ruptured ectopic pregnancy, the procedure may facilitate life-saving surgery by confirming the diagnosis without the need for time-consuming ultrasound examinations. Although the procedure is mainly used in the evaluation of ectopic pregnancy, culdocentesis should also be considered as a diagnostic aid in the evaluation of PID and in abdominal trauma. This is a safe, simple procedure that every physician who deals with the emergency evaluation of women, *particularly women of childbearing age*, should know and use. It may be the most rapid way to confirm hemoperitoneum in the unstable patient with suspected ectopic pregnancy.

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Chapter 59 - Examination of the Sexual Assault Victim

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Despite a decrease in violent crime overall, sexual assault remains a significant societal problem that affects hundreds of thousands of women and men yearly. [1] Our best estimate of the true incidence of sexual assault in the United States comes from the National Crime Victimization Survey, a national telephone survey that collects data on nonfatal violent crimes against persons older than 12 years. [1] According to this survey that includes data from the year 2000, over 261,000 sexual assaults occurred with females sustaining 20 times the number of assaults as males. [1] Encouraging news found not only a decline in the incidence of sexual assault, but also an increase in reporting of the crime to police. [1] The majority of victims still do not report the assault to anyone, however. It is estimated that over the last decade victims have reported an average of one third of sexual assaults to law enforcement. After reporting to law enforcement, sexual assault victims may be transported to the emergency department (ED) for evaluation, examination, and treatment. Sexual assault victims may also present to the ED for treatment without prior contact with law enforcement. Victims are usually willing to cooperate with police investigation, but they may not be. Most states have laws requiring medical personnel treating sexual assault victims to report the assault to local law enforcement. Clinicians must know their own state laws regarding this.

DEFINITIONS

Although many use the term synonymously with rape, *sexual assault* more accurately refers to any sexual contact of one person with another without appropriate legal consent.[2] Physical force may be used to overcome the victim's lack of consent, but this is not mandatory to prove assault. Lack of consent for sexual contact by intimidation, threats, or fear equals sexual assault. State law differs slightly on the description of exact acts that constitute sexual contact and on which populations are unable to give legal consent. In general those under the influence of drugs or alcohol, those who are minors, and those who are mentally incapacitated are deemed unable to give consent for sexual contact.

Clinicians who treat sexual assault victims have a professional, ethical, and moral responsibility to provide the best medical and psychological care possible. At the same time, they must collect and preserve the proper medicolegal evidence that is unique to the evaluation of sexual assault cases.

Many hospitals and jurisdictions are affiliated with designated sexual assault examination teams that provide specialized evaluation and treatment for victims. The sexual assault response teams (SARTs) provide clear advantages. These are outlined toward the end of the chapter. However, victims may be brought to an ED that does not routinely provide specialized care for sexual assault. This chapter is designed to aid clinicians in such a general care location. Prepared emergency personnel can help attenuate the psychological and physical impact of sexual assault. Through proper care of the victim and careful acquisition of evidence, ED staff can help the victim to recover from the assault and can aid society in improving the prosecution and conviction of sexual predators.

MANAGEMENT OF THE ADULT FEMALE SEXUAL ASSAULT VICTIM

In most places, local jurisdictions or hospitals provide clinicians with detailed forms and instructions for the examination and documentation of sexual assault. This chapter is meant to supplement these instructions and forms. Clinicians should familiarize themselves with local forms and instructions prior to performing a sexual assault exam. Careful step-by-step planning using written protocols to guide the way in which a victim is handled in the ED and in follow-up helps both to ensure the best care for the victim and to aid in the prosecution and conviction of assailants.

Major provisions in the ED protocol include patient privacy and the designation of a separate area for the care of the victims. If medically and logistically possible, the victim should be interviewed in a private room that is separate from the examination room. EDs often have such an area often called the "grieving room" or the "family room." Many legal jurisdictions provide examination kits for the collection of forensic evidence from the victims (Fig. 59-1). These kits should be available in the ED, and the staff should be familiar with them. If such kits are not provided by local jurisdiction, hospital staff may need to assemble their own kits from materials found in most EDs. Alternatively, private companies assemble and sell such kits (The Lynn Peavey Company, PO Box 14100, Lenexa, Kansas 66285-4100). Prepared kits save a tremendous amount of nursing and clinician time when a victim comes to the ED. A checklist for local requirements for sexual assault examinations should be included in the kits and serves as a reminder for all of the medicolegal procedures to be completed.

Although this chapter is primarily devoted to the evaluation of the adult female sexual assault victim, guidelines for the evaluation of the adult male sexual assault victim, the female child victim, the male child victim, and the accused assailant are provided in separate sections of this chapter. The same examiners designated to perform adult female examinations may easily perform male victim and assailant examinations; however, the examination of the child sexual assault victim often requires considerable expertise and training. When possible, medical staff with extra training in the examination of the child sexual assault victim should perform these examinations. If this is not possible, the special section of this chapter should provide emergency medical personnel with a framework to perform an initial examination.

Consent

Consent for the treatment of a sexual assault victim is mandatory. The victim has undergone an experience in which her right to grant or deny consent was taken from her, and obtaining consent for medical treatment and for the gathering of evidence has important psychological and legal implications. The victim has the right to decline medicolegal examination and even

Figure 59-1 Sexual assault examination kit used in Los Angeles County.

medical treatment. Witnessed, written, informed consent must therefore be obtained before beginning evaluation and treatment. In most cases the standard ED "consent to treat" section will suffice. Although most states mandate that medical personnel report sexual assaults to law enforcement, victims may decline to discuss the event with police. However, victims may need to cooperate with police and give consent for evidence collection to have the examination paid for by law enforcement (California and Oregon law). If the victim cannot give consent for a forensic examination owing to a reversible process (e.g., intoxication, an acute psychological reaction), clinicians may wait several hours for the victim's mental status to improve to a reasonable level for consent to be obtained. When victims cannot give consent due to minor status or developmental disability, the person authorized to give medical consent for the patient may consent for the examination unless he or she is a suspect in the assault. Many states allow an adolescent victim of a certain age (e.g., older than 12 to 14 years) to consent to an examination for conditions related to STDs, sexual assault, and pregnancy. State laws also differ in examiners' requirements to make an attempt to contact the legal guardian (unless he or she is a suspected perpetrator). Clearly, emergency personnel must be informed regarding their local laws concerning these requirements.

History

The history of the event should include only those elements necessary for the clinician to complete required forms, to perform a focused physical examination, and to collect evidence. Questions beyond this, such as the details leading up to the assault, should be left to the police investigators. The clinician should avoid the urge to "help" the alleged victim by unduly embellishing or detailing uncorroborated or nonmedical information that is supplied during the examination. Limiting the history not only shortens the evaluation in the ED, but it also helps to prevent discrepancies between the ED history and the official police investigation report, which could weaken the victim's case in court. Documentation of pertinent medical history includes last menstrual period, current contraception, recent anal-genital injuries or surgeries, and pre-existing injuries.

The history of the event required by protocol usually includes the time, date, and place of the alleged assault and a description of the use of force, threats of force, and the type of assault. Elements of force may include the type of violence used, threats of violence, the use of restraints, the number of assailants, the use of alcohol or drugs (forcibly or willingly) by the victim, and any loss of consciousness experienced by the victim. Elements of the type of assault include fondling; vaginal, oral, or anal penetration or attempted penetration; ejaculation on or in the body; the use of a condom; and the insertion of foreign bodies into the vagina or the anus. The use of physical force or violence is partly a police matter, but from a medical standpoint it is desirable to correlate positive findings on the physical examination (abrasions, ecchymosis, scratches) with a description of any force, restraint, or violence.

A gynecologic history should be obtained in preparation for injury documentation and treatment plans. From a medicolegal standpoint examiners must question victims about any recent gynecologic surgical procedures or unintentional genital trauma that might alter the expected normal genital appearance. The history should also include the use of any method of birth control before the attack (with information regarding any missed birth control pills), last normal menstrual period, last voluntary intercourse, gravidity and parity, and recent sexually transmitted diseases.

The medical history should include current medications, tetanus immunization status, and allergies. Documentation of post-assault activity commonly requested by forms includes douching, bathing, urinating, defecating, gargling, or brushing teeth. These can alter the recovery of seminal specimens and other sexual assault evidence. In addition, examiners need to question victims about potential injuries from any preassault bodily trauma.

Elements of the victim history should lead examiners in deciding which potential samples to collect. For example, sperm may be recovered after intercourse from the *cervix* for up to 12 days and from the *vagina* for five days.[3] For example, if the victim had voluntary intercourse 48 hours

before examination and was sexually assaulted 3 hours before examination, the clinician should obtain samples from both the vagina and the cervix, keeping the two specimens separate. By taking a careful history, the clinician is able to perform an appropriate examination given these two separate events. In general, cervical swabs should be collected in addition to the usual vaginal swabs if the time between assault and examination is >48 hours.

While taking the history, the examiner should observe the patient's ability to understand and respond appropriately to questions. Victims of sexual assault may not

possess the capacity to consent to intercourse because of developmental disability, young age, or intoxication with drugs or alcohol. Clinicians must consider obtaining blood or urine, or both, and testing for drugs or alcohol when the history suggests lapses of (or impaired) consciousness. Most often victims who lack consenting capacity owing to developmental disability will have sufficient prior documentation of the condition. In the rare instance when an examiner suspects previously undocumented developmental disability, formal examination of the patient's mental capacity can be assessed at a later time by request of the district attorney.

Physical Examination

The physical examination of the sexual assault victim differs from most other ED examinations because examiners are not only caring for a patient's physical and mental well being, but they are also investigating a crime scene and collecting specific evidence. Examiners must remember to explain every step of the examination to victims. Victims need to be reminded to communicate any discomfort or questions to the examiner and to ask for a break from the examination if needed. Additionally, they need to be reminded of their right to decline any portion of the examination and the ability to stop at any point. Victims should have the opportunity to have a family member, friend, or victim advocate, or both, in the room during all parts of the examination. In some jurisdictions, state law mandates that victims be informed of this right (California Penal Code 679.04, Victim of Sexual Assault; Right to Advocate and Support Person).

Collection of Clothing

If not already collected by law enforcement, the clothes that the victim wore during the assault need to be collected for potential evidence. Victims should disrobe by dropping clothes onto a clean sheet. Using gloved hands examiners can then place each clothing item in a separate paper bag. All collected material needs to be meticulously labeled and its collection noted and described in the chart. The sheet and any material that might have fallen during the victim's disrobing should be bundled and placed in a separate paper bag. When a victim's clothing must be collected, the ED assumes the responsibility of providing suitable dress for the victim to wear after release from the ED.

General Body Examination

After she disrobes, the patient's body should be examined for signs of trauma and foreign material. Important areas for evaluation are the back, the thighs, the breasts, the wrists, and the ankles (particularly if restraints were used). Even in the absence of ecchymosis, tender areas should be noted. Leaves, grass, sand, and other materials can occasionally be found in the hair or on the skin and should be retained as evidence. Areas of trauma should be documented and further evaluated (e.g., with radiographs) as indicated by the type and extent of injury. Approximately 10% to 67% of sexual assault victims display bodily injuries. [4] Documentation of these bodily injuries correlates significantly with successful perpetrator prosecution. [5] Bodily evidence may range from abrasions to major blunt and penetrating trauma. If the victim has not bathed, bodily evidence in the form of dried semen stains may be visible on the hair or the skin of the victim. In a darkened room, dried semen (and many other substances) on skin may fluoresce under examination with short wave light, such as that produced by a Wood's lamp or an alternative light source (ALS) (Fig. 59-2).[6] The ALS may perform better than the Wood's lamp for this function. [7] Furthermore, the possibility of finding seminal fluid with the Wood's lamp is negligible after 24 hours. [8] Potential dried secretions may be collected with moistened swabs; the swabs are then air-dried and preserved as evidence. In addition to the skin, the fingernails can contain hidden bits of evidence. Rape victims may have fragments of the assailant's skin, blood, facial hair, or other foreign material from the rape site beneath their fingernails. Fingernail scrapings are obtained by cleaning under a victim's nails with a toothpick or cutting the nails closely over a clean paper. The toothpick and debris are then folded into the paper, placed in an envelope, and packaged with the other specimens.

Photographs can be a valuable addition to the documentation of bodily injury. Some institutions employ professional quality photographic equipment; others use police photographers. Most institutions require patient consent for photographs taken by hospital personnel. Optimally, institutions should arrange a prior plan to handle film according to a written "chain of custody." Alternatively, self-developing film (Polaroid) that can be permanently labeled (e.g., subject, date, details of pictured injury) may be used. The photographs should be labeled immediately and may be added to the legal evidence. In some jurisdictions, photographs of physical injuries will be taken and retained by an accompanying law officer. These photographs may serve as evidence or may simply refresh the examiner's memory at the time of the trial.

Oral Evaluation

If indicated by history, the oral cavity must be closely inspected for signs of trauma and evidence collected. Potential mouth injuries from forced oral copulation include lacerations of labial or lingual frenulum and mucosal lacerations and abrasions (often produced by the victim's own teeth as her lips are forced inward with the perpetrator's penis). Potential injuries to the posterior pharyngeal wall and soft palate include petechiae, contusions, and lacerations. Clinicians need to document these injuries at the time of initial examination as mucosal injuries heal quickly and may not be present hours or days later. Potential evidence should be collected using swabs rubbed between the teeth and the buccal mucosa on both upper and lower gingival surfaces bilaterally. Spermatozoa have been identified in oral smears up to 6 hours after the attack despite tooth brushing, using mouthwash, or drinking various fluids and may show valuable evidence up to 12 hours after examination. [9] Any foreign material (e.g., hair) should be collected and included as potential evidence. The old practice of collecting saliva for blood group antigen secretor testing as a reference sample has mostly been abandoned in favor of more specific DNA testing by most crime laboratories.

Figure 59-2 Alternative light source.

Genital Examination

Once in the lithotomy position examiners should inspect the victim's thighs and perineum for signs of trauma and for foreign materials, such as seminal stains. An ultraviolet light may again be used to look at suspicious dried secretions. Many jurisdictions recommend routine collection of swabs from the external genital area owing to the high likelihood of evidence being present and the inconsistent fluorescence of seminal fluid with the Wood's lamp.

Pubic Hair Samples

The victim's pubic hair can be combed for foreign material (particularly public hair belonging to the assailant) before the pelvic examination. This is done by placing a clean paper below the victim's buttocks with the victim in the lithotomy position and combing the public hair onto the paper. These hairs and comb are then folded into the paper and placed directly into a large paper envelope to be given to law enforcement. Foreign pubic hairs can often provide enough cellular DNA material from the root to enable the crime laboratory to perform DNA analysis. Additionally, specialized laboratories posses the capability of performing mitochondrial DNA analysis from the hair shaft in many cases.

Unfortunately, significant hair transfer occurs in less than 5% of assaults. [10] In the minority of cases when foreign suspect hairs must be compared with victim hairs, a pulled victim sample may be desired. Although the routine pulling of the patient's hair from the roots may provide the best sample, the act of pulling the victim's hair may be considered insensitive and unnecessary during the initial evaluation. Additionally, these hairs will rarely be needed, as the vast majority of cases do not concern this evidence. A victim can provide the hairs at a later time if needed, and often the victim is willing to pluck the hairs herself at that time.

The genital examination of the sexual assault victim differs considerably from most ED pelvic examinations. Examiners must first perform a careful evaluation of the vulva and the introitus for signs of trauma. The techniques of separation and traction move the tissues most likely to suffer injury into view. If the examiner fails to perform these maneuvers, he or she will miss traumatic genital injuries. Examiners must be familiar with genital anatomy including all terms used to describe these areas (Fig. 59-3 and Fig. 59-4). Although most novice examiners concern themselves with detecting injuries to the hymen, the majority of sexual assault-related

vaginal injuries occur to the posterior fourchette. [11] In fact, hymeneal injuries are rare in sexually active adult women and are more commonly observed in sexually inexperienced adolescents.[12] More uncommon injuries to the vaginal walls and cervix may be discovered during the speculum examination. Reported rates of genital injury among forensically examined victims range from 6% to 53% without colposcopy[4] [13] to 53% to 87% with colposcopy.[11] [13] Most importantly examiners must be cognizant of the fact that a completely normal genital examination is consistent with forced sexual assault. In fact, a study of more than 1000 sexual assault victims found that almost one half of all victims with crime laboratory evidence positive for sperm had no genital injury. [4]

Colposcopy

Teixeira[14] first described the use of colposcopy for documentation in sexual assault in 1981. Although it is not the standard of care, and not readily available in most ED evaluations, the use of colposcopy has revolutionized the documentation of injury. The colposcope provides magnification, a bright light source, and usually permanent documentation of injuries in

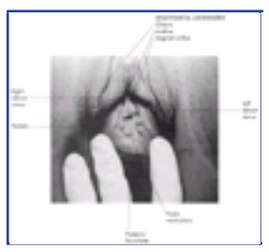

Figure 59-3 Female anatomy. *(From Girardin BW, Faugno DK, Seneski PC, et al: Color Atlas of Sexual Assault. St. Louis, Mosby-Year Book, 1997. Originally from Seidel HM, et al: Mosby's Guide to Physical Examination, 3rd ed. St. Louis, Mosby, 1995.)*

the form of slides, pictures, or video (Fig. 59-5). In one small study the colposcope increased the rate of genital injury detection from 6% to 53%. [13] Colposcopes with photo or video attachments provide excellent photographic documentation for court and allow for expert practitioner review for court testimony without subjecting the victim to re-examination. Although colposcopically visible injuries have also been described in women following consensual intercourse, these so called injuries consist only of increased vascularity and microabrasions, but no lacerations. [15]

Conversely, a totally normal genital examination by colposcopy is often found following sexual assault. Even in sexually inexperienced adolescents, forced penetration can occur without leaving discernible genital injury. [16] Although prior victim sexual experience decreases the likelihood of finding genital injury, experts cannot fully explain the reasons that some rape victims sustain measurable genital injury (Fig. 59-6), when others do not.

Forensic Evidence Collection

Protocols for evidence collection vary in different jurisdictions. Many rape evaluation centers have abandoned the cumbersome rape kits that have been used in the past, substituting simple collection methods that concentrate on important and usable legal evidence. The following discussion is patterned after the model protocol suggested by the state of California and the ACEP manual. [2]

During inspection of the external genitalia, rectum, vagina, and cervix, standard forensic specimens should be obtained. Experts recommend lubrication of the speculum with warm water rather than lubricant owing to the potential spermicidal activity of lubricants; however, the potential for corruption of DNA evidence should be negligible.[17] Generally, the specimens collected will be determined by victim history and local protocol, but they may include any of the items listed in Table 59-1 . Although not a standard examination by the emergency clinician, a wet mount of one swab from the vaginal pool may reveal motile sperm. From 13% to 26% of wet mounts provide evidence of sperm.[4] [18] Because of rapid cell death, studies have shown a negligible chance of finding motile sperm from a vaginal wet mount more than 8 hours after intercourse.[19] Several swabs from the vaginal pool (including the one used to make the wet mount) and the external genitalia are run over clean slides for a dry mount. All swabs and slides must be air dried, labeled, and packaged in

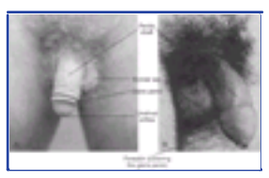

Figure 59-4 Male anatomy. *(From Girardin, et al: Color Atlas of Sexual Assault. St. Louis, Mosby-Year Book, 1997.)*

paper envelopes for the local crime laboratory. Equipment to aid the drying of specimens is shown in Figure 59-7 . Many crime laboratories also request collection of vaginal washing in which 5 mL of sterile (but not bacteriostatic) water or saline is inserted into the vagina and removed. The washing can be placed in a sealed container for later examination for evidence. Additionally, collection of cervical swabs may be indicated if the time from assault to examination (the postcoital interval) is greater than 48 hours, or if there is a history of recent consensual intercourse as well. The crime laboratory may recover sperm from cervical specimens up to 12 days after coitus.[3]

Swabbing the rectum must be performed if anal penetration is stated or suspected. Many experts also recommend routine collection of rectal swabs, because many victims may be unsure or embarrassed about rectal penetration.

Figure 59-5 Video colposcope with real time visualization on monitor shown. Image is eye of teddy bear at 15× magnification.

The CDC guidelines suggest considering a (cervical, rectal, or oral) culture or polymerase chain reaction (PCR) specimen, or both, for chlamydia and for *Neisseria gonorrhoeae*. However, the majority of SART programs in the United States do not routinely perform these tests. [20] Sexually transmitted disease (STD) testing during sexual assault examination can only detect pre-assault infection and provides no meaningful information for the crime laboratory. Additionally, the routine prophylactic treatment with antibiotics effective against *Neisseria gonorrhoeae* and chlamydia makes detection of these pre-existing infections superfluous; however, clinicians may want to consider obtaining cultures on child victims in whom the presence of an STD would be indicative of previous abuse.

Each sample should be labeled separately. The label should include mention of the area from which the specimen was collected and collection should be recorded in the chart.

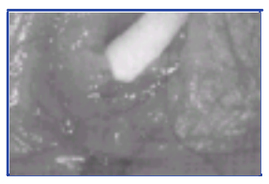

Figure 59-6 Hymenal injury at the 6-o'clock position in adolescent female.

Perineal Toluidine Blue Dye Staining

Toluidine blue dye is a nuclear stain that adheres to areas of injury (subepithelial nucleated cells) but not to intact epithelialized cells. Although it is not a uniform standard of care, the dye can enhance the examiner's ability to visualize genital injuries (Fig. 59-8). Genital lacerations may provide corroborating evidence of non-consensual intercourse. To outline injuries a 1% aqueous solution of toluidine blue dye is applied to the perineum and excess dye is wiped off with a cotton ball moistened with lubricating jelly. Some examiners use acetic acid to remove the excess; however, acetic acid may produce pain when it contacts injured tissues. After the excess dye is removed, any remaining linear areas that retain the stain signify injury. Folds of the area must be separated and carefully examined to avoid missing injuries. Ideally the dye should be applied before speculum examination to eliminate the possibility of iatrogenic injury. The procedure is shown in Figure 59-9 . In one study the use of toluidine blue dye increased the injury detection rate from 16% to 40% and up to 70% in nulliparous women without the use of colposcopy [21] ; however, injuries detected with the aid of toluidine blue dye are not 100% specific for sexual assault

TABLE 59-1 -- Potential Evidence Collected

Clothing (list and describe all clothing collected on chart and checklist form)
Debris
Dried secretions, swabs, and slides
External genital swabs and slides
Pubic hair combings
Oral mucosal swab and slide
Rectal swabs and slides
Vaginal pool swabs and slides
Vaginal lavage fluid in test tube or urine container
Tampon or condom present in vagina (dried or frozen)
Urine or blood toxicology sample (timed collection)
Reference blood, buccal mucosa, and/or hair sample
Collect all evidence using gloved hands to avoid DNA contamination
Change gloves when necessary to avoid inter-location contamination

because injuries have also been found following consensual intercourse, especially in adolescents. [22]

If seminal stains are noted on the perineum, samples should be collected before toluidine blue application. Contrary to earlier thinking, the use of toluidine blue dye does not interfere with recovery of DNA evidence [23] and has proven safe for mucosal application. [24]

Anal Evaluation

The anal exam follows the genital exam in most cases. Because of a reluctance of some victims to admit to anal penetration, some clinicians recommend an anal examination in all cases. Documentation of anal penetration holds significant value as it is a separate crime in addition to vaginal penetration and can add years to the sentence of the alleged perpetrator. Anal swabs are inserted approximately 2 cm into the anus, gently moved in a circular motion, removed, and used to make slides, which are air dried and included in evidence sent to the crime laboratory. In some jurisdictions additional evidence in the form of rectal washings may be requested. This is done by injecting 5 to 10 mL of normal saline with a syringe and a small plastic IV catheter, aspirating and preserving the fluid as evidence. Visualization of injury is increased by the use of toluidine blue dye as described earlier (Fig. 59-10). Anoscopy is not a routine part of the examination but it may be a useful adjunct in the documentation of rectal injuries. Examiners perform this procedure in the same manner as diagnostic anoscopy used to evaluate other anal or rectal emergencies in the ED (Fig. 59-11). In one retrospective observational study of male victims, the use of anoscopy and colposcopy provided superior documentation of injuries over colposcopy alone. [25] The location of anoscopically detected injuries may be recorded geographically.

Reference Samples

Crime laboratories often request reference samples taken from various locations on the victim's body to use in comparison testing with potential perpetrator evidence. These reference samples include blind swabs on the victim's skin in a complementary location to suspicious skin samples. For example, if a suspicious discharge is swabbed from the victim's right shoulder, a control swab may be taken from the victim's left shoulder. Crime laboratories may also request other control samples for victim DNA reference, such as blood, buccal mucosa cells, head hairs, or pubic hairs. The need for such samples will be determined by local protocol.

Blood Tests

Some crime laboratories request blood samples for DNA reference or toxicology analysis, or both. These samples must be included in the materials sent with law enforcement and not to the hospital laboratory unless a firm procedural "chain of custody" has been established for this purpose. When collecting blood for toxicology testing, the exact time of collection must be recorded on the specimen so that the criminologist may estimate dose and timing of substances used to perpetrate the assault.

Urine Tests

Bedside urine ß-hCG testing should be performed on all women victims of childbearing age to exclude pre-existing

Figure 59-7 Device used to hold swabs and slides to speed drying.

pregnancy before consideration of pregnancy and sexually transmitted disease prophylaxis. Additionally, urine may be requested by local crime laboratories for toxicology testing. Crime laboratories request the victim's first available voided urine to optimize recovery of potential evidence.

Spermatozoa, Semen, and DNA Testing

Motile and immotile sperm may be found microscopically in wet mounts of vaginal aspirates and in vaginal, oral, and rectal swabs. Some examiners microscopically

evaluate the slide immediately after the physical examination. These examiners find sperm in 13% to 26% of vaginal wet mount specimens. [4] [16] Early discovery of sperm may be helpful to law enforcement investigation. However, most ED examiners lack formal training in this process, and crime laboratories possess much higher sensitivity for sperm detection, making a negative initial wet mount unhelpful. For these reasons, many examiners do not routinely perform the wet mount examination.[4] After consensual intercourse with normal ejaculate, laboratory testing of vaginal secretions will likely be able to identify

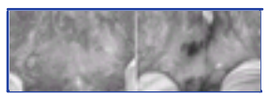
Figure 59-8 Posterior fourchette injuries. *A,* Viewed through the colposcope without the use of toluidine blue dye. *B,* Same posterior fourchette injuries outlined with toluidine blue dye.

sperm after 3 days and in 50% of cases at 4 days.[26] However, despite penile penetration during sexual assault, the crime laboratories may fail to identify sperm. Reasons for this failure include inadequate specimen collection, degradation of ejaculate, azoospermia, failure of the perpetrator to ejaculate, perpetrator vasectomy, or condom use.

A crime laboratory investigator initially looks for semen in a given sample by searching microscopically for sperm on a concentrated specimen and by testing for other components found in semen. Such seminal plasma components include P30 and acid phosphatase. P30 is a glycoprotein specific to the prostate [27] and is regarded as conclusive evidence of semen (i.e., ejaculation within 48 hours), whereas acid phosphatase is only presumptive evidence because it can occur in other body fluids, such as vaginal secretions. Although a main component of crime laboratory investigation in the past, many laboratories have abandoned the acid phosphatase test in favor of the more sensitive P30 test.[27] [28] Despite negative testing for seminal plasma components, laboratories may be able to detect valuable DNA evidence from persistent sperm cells or perpetrator epithelial cells. [29] [30] As DNA testing


Figure 59-9 Toluidine blue application procedure. *(From McCauley J, Guzinski G, Welch R, et al: Toluidine blue in the corroboration of rape in the adult victim. Am J Emerg Med 5:106, 1987.)*

technology rapidly changes, crime laboratory ability to perform a specific forensic test varies by location and over time. Most crime laboratories use PCR (polymerase chain reaction) testing, which requires minimal material.

Chain of Custody

Samples and other evidence must be given to the police, a crime laboratory, or a forensic pathologist. Each sample must be labeled with the patient's name, hospital number, date, time of collection, the area from which the specimen was collected, and the collector's name. These specimens should then be packaged according to local crime laboratory specifications and transferred to the next appropriate official


Figure 59-10 Anal injury is best seen with separation of perianal tissues. *A,* Without toluidine blue dye. *B,* Visualized after toluidine blue dye.

(police officer, pathologist, or other individual) along with a written chain of custody, which includes a list of the specimens, the signature of each person who provided them, and the signature of each person who received them. If this chain is broken, important evidence may be deemed inadmissible in court.

TREATMENT

Sexually Transmitted Disease Prophylaxis

The factors of sexually transmitted disease (STD), pregnancy, psychological distress, and follow-up should be considered in

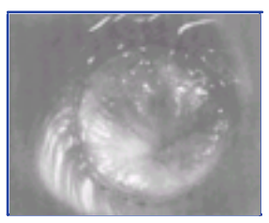

Figure 59-11 Rectal injury visualized with anoscopy.

the treatment of a sexual assault victim. Because pre-assault infection rates are not known, the risk of contracting a sexually transmitted disease (STD) as a consequence of a sexual assault has been difficult to determine, and estimates are tentative or nonexistent. One report of post-assault STDs found a 3% chance of development of gonorrhea and a 0.1% chance of development of syphilis. Other investigations suggest that the risk for these STDs may be as high as 12% and 3%, respectively. [32] Jenny and colleagues[33] found the post-assault incidence of STDs to be 2% for chlamydia and 4% for gonorrhea. The reported rates of 12% for *Trichomonas* and 19% for bacterial vaginosis may reflect a pre-exposure infection since male transmission of these organisms is rare. Glaser and others [34] found similar STD infection rates. The risk of developing herpes, hepatitis B, or human immunodeficiency virus (HIV) infection from being sexually assaulted has not been determined. However, HIV transmission has been noted.[35]

Although the chance of contracting an STD is small, examiners may choose to treat a victim prophylactically for gonorrhea, syphilis, and chlamydia; alternatively, examiners may choose to rely on follow-up cultures. The need for routine prophylaxis against Trichomonas is unclear and many clinicians do not recommend it as a routine intervention. Gonorrhea resulting from a sexual assault may possible be culturable within hours of the attack, but is almost always culturable at a 2-week follow-up visit. Because of the omnipresent fear of contracting a sexually transmitted disease from a sexual assault, it is reasonable to routinely offer prophylaxis to all victims. Because victims tend to have a relatively low compliance with keeping follow-up visits, most examiners offer, at the least, gonorrhea and chlamydia prophylaxis at the time of the initial examination.[20]

With the increasing prevalence of *Neisseria gonorrhoeae* strains resistant to penicillin and tetracycline, ceftriaxone has become the antibiotic of choice in treating gonorrhea. Ceftriaxone also treats incubating syphilis. Spectinomycin (2 g IM) and oral quinolones are single-dose alternatives for penicillin- and cephalosporin-allergic patients, but neither of these has been shown to be effective against incubating syphilis. No single-dose regimen for gonorrhea is effective against coexisting *Chlamydia trachomatis* infection. Therefore, patients also should be given either a single dose of azithromycin (1 g PO) or a seven-day course of doxycycline (100 mg PO BID) or tetracycline (500 mg PO QID). A negative pregnancy test is a prerequisite for using either of the latter two antibiotics. Erythromycin

TABLE 59-2 -- Single-Dose Therapy for Preventing Sexually Transmitted Disease after Sexual Assault

Single-Dose Therapy for the Prevention of Gonorrhea (GC)	
Cefixime	400 mg po
or	
Ofloxicin[1]	400 mg po
or	
Ciprofloxacin*	500 mg po
or	
Gatifloxacin*	400 mg po
or	
Ceftriaxone†	125 mg IM
Single-Dose Oral Therapy for the Prevention of Chlamydia	
Azithromyacin	1000 mg
Single-Dose Oral Therapy for the Prevention of *Trichomonas* and Bacterial Vaginosis	
Metronidazole*	2000 mg

IM, intramuscularly; PO, by mouth.

*Not for use in pregnancy. In pregnant cephalosporin-allergic patients, use Spectinomycin 2 mg IM for prevention of GC.
†Also treats incubating syphilis.

is a second alternative for Chlamydia prophylaxis in the pregnant patient. Some examiners administer prophylaxis for *Trichomonas* with a single 2 g oral dose of metronidazole; although effective and recommended by the CDC[36] this dose of metronidazole may cause significant nausea, vomiting, or diarrhea, or both, which can interfere with the efficacy of pregnancy prophylaxis. Table 59-2 provides one example of a completely oral STD prophylaxis regimen.

Hepatitis B Prevention

Current CDC treatment guidelines in the setting of a high-risk exposure recommend hepatitis B vaccination only, *without* hepatitis B immune globulin (HBIG), at the time of examination, followed by two more vaccines at 1 to 2 months and 4 to 6 months.[36] Interestingly, the same CDC publication recommends the use of HBIG plus vaccination for nonimmune sexual contacts of infected individuals, stating that it is 75% effective in preventing the non-immune individual from contraction of hepatitis B. Clearly, the vaccine alone would be less protective than the vaccine plus HBIG against sexual transmission of hepatitis B. Potential reasons that the CDC may have settled for this less effective prophylactic regimen include the following: HBIG is very expensive, the majority of sexual assaults do not involve an infected perpetrator and a non-immune victim, infant immunization has been standard for the last 7 or 8 years, and the vaccine alone provides some protection. Clinicians may still want to consider administering HBIG to nonimmune victims with a high-risk exposure.

HIV Prevention

In the non-assault scenario, the risk of transmission of HIV from one episode of unprotected consensual receptive vaginal intercourse with an infected individual is approximately 1 in 1000. The incidence with unprotected receptive anal intercourse is significantly higher, at 8 to 32 in 1000. [37] However, sexual assault victims often

sustain tissue injury owing to the violent nature of the act which may increase the transmission rate of the virus. Although treatment of parenteral occupational

exposure to infected body fluids (i.e., needle stick) is believed to be effective based upon case control studies, there is no proof that treatment of human sexual exposure prevents the transmission of the virus.[38] Furthermore, victims of sexual assault frequently present for treatment much later than those who have occupational exposures.

However, 40% of sexual assault victims fear contracting HIV post assault and should, at a minimum, receive counseling and some argue, the option of taking anti-HIV medicines because they may be effective.[39] [40] San Francisco investigators have described a large-scale feasibility trial of AZT/3TC for 28 days post sexual assault, and this regimen seems to be a logical choice in balancing side effects and possible efficacy. [41] In this trial, 32% of those offered prophylaxis accepted the regimen, and one third of those came back in a week to obtain the remainder of the oral prophylactic course. In a Canadian descriptive trial, 28% of sexual assault victims accepted HIV prophylaxis and 40% of those returned after 5 days for the remaining medication. [42] Unfortunately, immediate testing of the perpetrator remains a remote option owing to physical unavailability of the majority of perpetrators and the fact that only a minority of states provide for legal preconviction HIV testing of alleged perpetrators.[39] Given the extreme negative outcome of HIV infection, at least two states have written empiric policies to guide examiners with this complex issue. One such policy is shown in Table 59-3 . However, the antiviral agents are expensive and potentially toxic. Currently, no standard of care based upon objective outcome data exists regarding HIV prophylactic therapy.

Pregnancy Prophylaxis

Pregnancy occurs in up to 4.7% of sexual assault victims.[43] An estimated 22,000 annual rape-related pregnancies could be avoided if all victims sought and received pregnancy prophylaxis within 72 hours.[44] Victims should provide urine for a pregnancy test before administration of postcoital contraception. Modern urine pregnancy tests possess a detection threshold approaching 20 to 25 mIU/mL and usually will be positive 1 to 2 weeks after conception, and often before a menstrual period is missed.

Sexual assault victims must be offered pregnancy prevention using available oral postcoital contraception (Table 59-4). In 1997, the FDA published a list of available oral contraceptives that could be used to provide postcoital contraception.[45] Most of these pills are a combination of ethinyl estradiol and a progestin, known as the Yuzpe regimen. This method prevents approximately 75% of pregnancies that would have otherwise occurred.[46] Alternatively, an FDA-approved progestin regimen used as postcoital contraception prevents 89% of pregnancies that would have otherwise occurred and causes fewer side effects, but may be unavailable. [47] Both regimens involve two doses of the medication taken 12 hours apart within 72 hours of intercourse. Adverse side effects include nausea, vomiting, and breast tenderness. If the patient vomits within 1 hour of taking a dose, it should be repeated. Some routinely offer prophylactic antiemetic therapy; others reserve such treatment for patients who vomit. Two kits containing the needed pills along with an antiemetic (Preven and Plan B) are FDA approved and commercially available. These regimens are also available at pharmacies by prescription. Because of their extreme safety and importance, some states have already made these kits available without a prescription. All available evidence demonstrates no untoward effects on the fetus should pregnancy occur despite postcoital contraception. [48] The common practice of obtaining written patient consent for these medications seems unwarranted.

Unfortunately, religious preferences may deter some hospital EDs from providing postcoital contraception.[49] In these instances, the Web site and number listed in this paragraph provide practitioner referral for easy access to postcoital contraception. Additionally, given the ever-increasing availability of new methods and drugs for postcoital contraception, examiners will want to obtain up-to-date information from these sources (Web site: www.not-2-late.com; telephone: 1-800-not-2-late).

Psychological Support

Sexual assault precipitates a psychological crisis for the patient, and psychological care should begin when the patient first arrives in the ED. [50] [51] Examiners must reassure the victim that she will be in control of the examination, that she may ask questions at any point, and that she should notify the examiner if anything hurts or if she needs a break. Giving the victim control over her body and the examination is the first step toward psychological support. Unfortunately, if this is not made a priority, victims may experience "secondary victimization" and be further traumatized by the forensic examination.[52] Sexual assault victims often develop posttraumatic stress disorder (PTSD), manifested by numbed responsiveness to the external world, sleep disturbances, guilt feelings, memory impairment, avoidance of activities, and other symptoms.[50] *Rape trauma syndrome* is the specific label for PTSD in this population.[50] The victim is particularly vulnerable to this stress disorder because of the following characteristics of sexual assault: (1) it is sudden, and the victim is unable to develop adequate defenses; (2) it involves intentional cruelty or inhumanity; (3) it makes the victim feel trapped and unable to fight back; and (4) it often involves physical injury. Attention to the initial psychological care of the rape victim in the ED is fundamental and can reduce distress during forensic examination.[53] Many areas have a local sexual assault crisis agency that can dispatch an advocate to be with victims during the interview and examination. This same agency may then provide the follow-up psychological support that must be offered to all victims. It is critical that all examiners maintain current contact information with these agencies and use their services when at all possible. The importance of this contact is emphasized in some areas by the fact that state law dictates that medical personnel contact a local sexual assault crisis agency when a victim presents for examination (California penal code 264.2, Notification of a Counseling Center). In the absence of immediate local crisis services, a hospital social worker may fill this role.

Postexamination Follow-Up

Medical and psychological follow-up for sexual assault victims is essential. Unfortunately, less than one third of the victims complete follow-up medical care. [54] Many protocols recommend a 2-week follow-up to re-examine any injuries and to repeat testing for STDs and pregnancy. The timing of this follow-up seems less important given the widespread use of prophylactic medication to prevent STDs and pregnancy. However, given the measurable failure rate of postcoital contraception, repeat pregnancy testing is critical for a victim

TABLE 59-3 -- Empiric Guide to Offering HIV Post-Exposure Prophylaxis

Has less than 72 hours passed since the assault occurred?
If no, do not offer PEP but recommend or refer for baseline and follow-up HIV antibody testing.
If yes, continue risk analysis.
Is survivor 12 years of age or older?
If yes, continue risk analysis.
If no, consult pediatric HIV specialist (To operationalize this guide, this consultant must be identified in advance).
What is the risk of HIV transmission from the assault?
Was the assault one with measurable risk of HIV transmission, such as an assault with anal penetration, vaginal penetration, or injection?
Was the assault one with possible risk of HIV transmission, such as oral penetration with ejaculation, an assault involving other mucous membranes (e.g., eyes), an unknown assault, an assault in which the victim bit the assailant, or the assailant (with a bloody mouth) bit the victim?
Was the assault one with no risk of HIV transmission, such as kissing, object or digital penetration, ejaculation on intact skin, or an assault in which a condom was used?
What other risk factors were present in the assault, including presence of blood, survivor or perpetrator with STD, significant trauma to survivor; ejaculation by assailant, or multiple penetrations of the survivor?
Is the assailant's HIV status known?

If known HIV negative, *do not offer* PEP.

If known HIV positive:

Recommend PEP if assault with measurable risk of HIV transmission has occurred.

Recommend PEP if assault with possible risk of HIV transmission has occurred and at least one additional risk cofactor was present in assault.

Offer PEP if assault with possible risk of HIV transmission has occurred with no additional risk cofactors present.

Do not offer PEP for exposures carrying no risk.

Does the assailant engage in behaviors that put him/her at risk for contracting HIV?

High risk groups include men who have sex with men, past or present injection drug users, commercial sex workers, individuals with multiple sex partners, individuals with prior convictions for sexual assault, and individuals with a history of prison incarceration.

If known or suspected risk factors exist:

Recommend PEP if assault with measurable risk of HIV transmission has occurred.

Recommend PEP if assault with possible risk of HIV transmission has occurred and more than one additional risk cofactor was present in assault.

Recommend or offer PEP if assault with possible risk of HIV has occurred and only one additional risk cofactor was present in assault.

Offer PEP if assault with possible risk of HIV transmission has occurred with no additional risk cofactors present.

Do not offer PEP for exposures carrying no risk.

If assailant is not known and/or if assailant's risk factors are unknown:

Offer PEP if assault with measurable risk of HIV transmission has occurred.

Offer PEP if assault with possible risk of HIV transmission has occurred and more than one additional risk cofactor was present in assault.

Offer PEP if assault with possible risk of HIV transmission has occurred and only one additional risk cofactor was present in assault.

Offer or do not offer PEP if assault with possible risk of HIV transmission has occurred with no additional risk cofactors present.

Do not offer PEP for exposures carrying no risk.

Offering PEP after sexual assault

	Source		
Exposure Risk	***Known HIV†***	***Known or Suspected Risk Factors***	***Unknown Risk Factors or Unknown Assailant***
Measurable risk*	R	R	O
Possible risk + more than 1 cofactor†	R	R	O
Possible risk + 1 cofactor†	R	R/O	O
Possible risk + 0 cofactors	O	O	O/N
No risk‡	N	N	N

Key

R = Recommend

O = Offer

N = Do Not Offer

PEP, post-exposure prophylaxis.

†Acts with possible risk of HIV transmission, including oral penetration with ejaculation, unknown act, contact with other mucous membrane, victim biting assailant, and assailant with bloody mouth biting victim.
*Acts with measurable risk of HIV transmission, including anal penetration, vaginal penetration, and injection with a contaminated needle.
‡Acts with no risk of HIV transmission, including kissing, digital or object penetration of vagina, mouth, or anus, and ejaculation on intact skin.

who does not experience an expected menses. Further follow-up evaluations may be performed at 4 or 6 weeks and 4 to 6 months to repeat serologic tests for HIV, hepatitis B, hepatitis C, and syphilis. In addition, local volunteer support groups can be of immense assistance to a sexual assault victim, and contact with such a group should be offered to each victim.

SPECIFIC POPULATIONS

Male Evidentiary Examinations

Male evidentiary examinations include all of the same forensic evidence collection as female victims except vaginal specimens.

TABLE 59-4 -- Emergency Contraception

Brand	Manufacturer	Pills
Combined Emergency Contraception Oral Therapy		
Preven	Gynétics	2 blue pills immediately and in 12 hours
Ovral	Wyeth-Ayerst	2 white pills immediately and in 12 hours
Ogestrel	Watson	2 white pills immediately and in 12 hours
Alesse	Wyeth-Ayerst	5 pink pills immediately and in 12 hours
Levlite	Berlex	5 pink pills immediately and in 12 hours
Nordette	Wyeth-Ayerst	4 light-orange pills immediately and in 12 hours
Levlen	Berlex	4 light-orange pills immediately and in 12 hours
Levora	Watson	4 white pills immediately and in 12 hours
Lo/Ovral	Wyeth-Ayerst	4 white pills immediately and in 12 hours
Low-Ogestrel	Watson	4 white pills immediately and in 12 hours
Triphasil	Wyeth-Ayerst	4 yellow pills immediately and in 12 hours
Tri-Levlen	Berlex	4 yellow pills immediately and in 12 hours
Trivora	Watson	4 pink pills immediately and in 12 hours
Progestin Only Emergency Contraception Oral Therapy		
Ovrette	Wyeth-Ayerst	2 yellow pills immediately and 1 in 12 hours
Plan B	WCC	1 white pill immediately and 1 in 12 hours

Note: Some regimens cause nausea and an antiemetic may be used. If vomiting occurs, repeat the dose of antiemetic.

The forensic examination is guided by the history of events related by the victim. Most male victims suffer from anal penetration, or sodomy, by their perpetrator. In addition to rape trauma syndrome, heterosexual male victims may suffer psychological trauma, wondering if the assault now means that they "are gay." Examiners should inform such victims that the act of forced sodomy in and of itself does not indicate homosexuality. The increased risk of HIV transmission with anal intercourse is noted (see section on HIV prevention). Because of the extreme emotional reaction men often feel after a sexual assault, they report the crime even more infrequently than do female victims.[55] [56] The male victim deserves the same unhurried, nonjudgmental manner that the female victim deserves. Penile samples from the shaft, glans, corona, and scrotum may be obtained if there is oral or anal contact with the perpetrator.

Child Sexual Assault Examinations

The care and treatment of the pediatric sexual assault patient requires expert knowledge and experience. The presence of obvious genital injury and trauma speaks for itself, based on the history provided. The subtle variations of developmental changes and congenital anomalies leave many clinicians ill-equipped to render an opinion concerning the validity of sexual assault injury. The lives of children and families may be disrupted or severely affected, depending on the practitioner's opinion on the presence of genital penetration-type findings.

A well-known study by Adams et al.[57] demonstrates that the majority of children reporting sexual abuse have normal or nonspecific genital findings. With child sexual assault, it is "normal to be normal." In spite of expert physical examination, the vast majority of sexually abused children cannot be differentiated from nonabused children.[58] The discovery of one of the rare examination markers of injury should be confirmed with experts, and a discussion of these findings, although beyond the scope of this chapter, is covered extensively in other resources. [59] The potential sexual assault history provided by the child or caretaker should, therefore, remain the primary indicator that inappropriate genital contact has occurred. At the very least, the history warrants an investigation of the possibility of sexual abuse.

It cannot be emphasized enough that the examiners' responsibility to the care of the child victim of sexual abuse remains within the realm of experts. However, in EDs without available local experts, careful inspection of the genitalia in a non-hurried, child-friendly manner must be completed. For the very young child with small genital orifices, the aid of the otoscope for magnification may be extremely helpful. Often a parent may assist in the calming, reassurance, and positioning of the child for careful inspection. However, when the parent is a suspect, the practitioner must exclude that parent from the examination. Other than basic lithotomy position for the older, more mature child or adolescent patient, alternative positioning of the pediatric female patient is essential to inspection. The frog-leg position using labial separation and traction is often most beneficial (Fig. 59-12). Care must be taken to gently separate the labia to avoid superficial examiner-induced injuries. Genital findings that are deemed definitive of sexual abuse, penetration, or are nonspecific are included in Table 59-5 . Many normal hymeneal differences exist, however, from one child to the next and the definitive diagnosis of "abnormal" is often difficult for experts. When any doubt exists, ED examiners need merely describe the findings and refer the child for a later examination by experts. The availability of a colposcope or alternative photographic equipment with magnification clearly aids in documentation of any injuries that may heal before a time when an expert examination can be performed.

When disclosure or genital injuries confirm possible penetration of the child, specimens must be collected for the presence of potential evidence. These specimens may be collected without the insertion of a pediatric speculum; in fact, many child abuse experts strongly advocate that no speculum examination be conducted on any prepubescent child.[59] Cases involving severe vaginal trauma or suspected internal genital injury warrant examination under general anesthesia. External anal and vulvar swabs are usually collected without difficulty on a child of any age. Vaginal samples on very young children usually are difficult to obtain

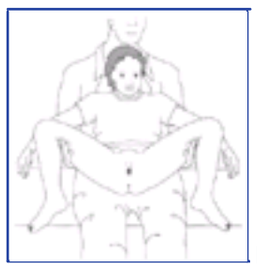

Figure 59-12 Recommended "frog leg" position to examine children.

and should remain the very last evidence collected. Vaginal aspirates using a feeding tube or plastic angiocatheters are alternatives to vaginal swabs.

Specimens for screening of sexually transmitted diseases remains a controversial issue in the realm of child abuse experts. For *Neisseria gonorrheae*, at least, the literature supports the notion that all infected children will display an abnormal discharge. [60] With very young children, the practitioner may have only one opportunity to collect vaginal specimens without subsequently restraining or sedating the child. The practitioner will need to decide at which point specimen collection becomes a second assault on the child. Because the child presents for possible sexual abuse, the primary specimens collected should be for forensic DNA analysis. STD detection and treatment can be performed at a later time. Clinicians must consult local child abuse centers for protocols regarding

TABLE 59-5 -- Genital Findings Possibly Related to Sexual Assault in Prepubertal Children *

Clear Evidence
Local areas of hymenal absence in knee-chest position
Hymenal transection
Anal laceration
Suspicious or Suggestive
Extremely narrow hymen (<1mm)
Acute abrasions or lacerations of labia or vagina external to hymen
Excessive anal dilatation (<15mm without stool in rectum)
Nonspecific
Redness
Increased vascularity
Labial adhesions

**Adapted from Adams JA, Harper K, Knudson S, et al: Examination findings in legally confirmed child sexual abuse cases: It's normal to be normal. Pediatrics 94:310–317, 1994.*

immediate STD specimen collection, referrals, and follow-up services.

Suspect Examinations

As forensic evidence collection in the form of DNA retrieval continues to evolve, EDs may see more requests from local law enforcement for evidence collection from alleged suspects. EDs should be familiar with local and state protocols, especially regarding consent. Some jurisdictions require that no voluntary consent be obtained from the alleged suspect, given imminent degradation of potential biologic evidence. Other jurisdictions require that alleged perpetrators give consent, or at the very least, that police obtain a search warrant from the court. The sooner a suspect is apprehended and brought in for a medical-forensic examination, the better the quality of forensic biologic evidence.

Performing a medical-forensic examination on an alleged suspect can give important corroborating information for the investigation of a crime. It can also help to exonerate the innocent. Law enforcement should always be in attendance during any suspect examination to address safety issues for the examiner. Furthermore, the suspect and victim should never encounter one another in the hospital setting during the examination period and care should be taken to examine the victim and suspect in separate locations within the ED. It is extremely beneficial to conduct the victim examination and history prior to the suspect's examination so that physical findings on the suspect may help to corroborate the victim's history. For example, if, during the victim's history, she relates that she scratched the suspect's left shoulder in defense, the examiner can be certain to examine, document, and preferably photograph the presence (or absence) of the injury on the suspect's left shoulder. However, having the same practitioner examine both victim and suspect can be controversial in some jurisdictions. Concerns have been raised regarding examiner bias against the suspect and potential evidentiary contamination.

The physical and evidentiary examination for the alleged suspect is similar to the victim examination. The primary differences lie in history taking, reference samples, and more "blind" samples (those that are presumed, not evident). During suspect examinations, law enforcement rather than the suspect, provides the history of the event. Reference samples of head and pubic hairs are routinely collected as well as blood, saliva, and urine, if possible. Special attention must also be applied not only to nail scrapings but also to swabbing all of the fingers for possible vaginal epithelial cells from digital penetration. Penile swabs include collecting from the shaft and corona of the glans, then separately collecting swabs from the scrotum for vaginal secretions. From an unwashed penis, swabs almost uniformly show evidence of female cells up to 24 hours after coitus. [29]

Suspect examinations require the same amount of professional sensitivity and respect that any patient receives within the ED. It is not within the realm of the clinician's expertise to determine whether or not the alleged suspect is guilty or innocent.

The Unconscious Victim and "Date Rape"

Alcohol and other drugs play an important role in many sexual assaults. One half of all sexual assaults involve drug or

alcohol ingestion. [61] In many cases it is unclear if a drug was taken voluntarily or if it was surreptitiously given to the assaulted victim.

The media has highlighted increasing awareness of drugs used to facilitate sexual assault under the term *date rape drugs* (Table 59-6). [62] The drugs most commonly associated with drug-facilitated sexual assault are ethanol, marijuana, cocaine, and benzodiazepines. Often more than one drug is found. Although any type of sedative or hypnotic drug, or combination of both, may be used to facilitate sexual assault, the most notable drugs include flunitrazepam (Rohypnol) and gamma hydroxybutyrate (GHB). [63] Despite their reputation in the lay press, flunitrazepam and gamma hydroxybutyrate are associated with drug-facilitated sexual assault in less than 5% of cases. [63] Flunitrazepam is a benzodiazepine unavailable in the United States but available in Mexico. It can be detected in the urine up to 3 weeks post ingestion. [64] Gamma hydroxybutyrate is a federally banned central nervous system depressant not available for legal purchase in the United States but easily manufactured illegally by users. It can be detected in drinking material residue by crime laboratories as well in the victim's urine up to 4 hours post-ingestion.

Drugs similar to GHB are 1,4 butanediol (BD) and gamma butyrolactone (GBL). Often the victim's last memory is of using drugs or alcohol and then passing out. Some may remember short segments of activity that may indicate some type of sexual acts. Some victims may have no memory at all but desire to be "checked" for intercourse. A comprehensive forensic-medical examination should be conducted on

TABLE 59-6 -- Drugs Used to Facilitate Sexual Assault *

Alprazolam
Amphetamines
Barbiturates
1,4 Butanediol (BD)[†]
?-Butyrolactone (GBL)[†]
Cannabis
Cocaine
Chloral hydrate[†]
Clonazepam[†]
Clonidine[†]
Diazepam
Ethanol
Flunitrazepam (Rohypnol)[†]
?-Hydroxybutyrate (GHB)[†]
Ketamine[†]
Lorazepam
Meprobamate[†]
Methamphetamine
Midazolam (Versed)[†]
Oxazepam
Phencyclidine (PCP)
Propoxyphene[†]
Scopolamine[†]
Secobarbital
Temazepam
Triazolam
Zolpidem[†]

After Slaughter L: Involvement of drugs in sexual assault. J Reprod Med 45:425–430, 2000; and Schwartz RH, Milteer R, LeBeau M: Drug-facilitated sexual assault ("date rape"). South Med J 93:558–561, 2000.

*Often more than one drug is found.
†Will not be detected on a routine immunoassay drug screen. A more detailed analysis will be required.

these individuals. All orifices (oral, vaginal, and anal) should have standard sexual assault evidence collected and an evaluation conducted for injuries. Without a history from the victim, samples must be collected from even potential oral or genital contact including neck, breasts, and vulva. Toxicology samples (including ethanol) should be obtained from both blood and urine, if possible, with exact times of collection documented. Because of the wide spectrum of date rape drugs available, the standard hospital's drug screen may be inadequate and a reference laboratory is best consulted. Some forensic laboratories offer a "date rape panel" that tests for a variety of commonly used substances. Obviously a positive drug test does not prove date rape and it may be impossible to distinguish self-administration to clandestine ingestion.

Extreme sensitivity must be used for discussing positive genital findings with a victim who has no memory of any sexual activity. Many times the imagined sexual acts can create just as severe a traumatic response as an actual remembered sexual assault. For the unconscious victim, there is no memory of events to fill in the blanks, only her terrifying imagination of what could have happened.

Legal Issues

It is not uncommon for the examiner to be called to court to testify in cases of sexual assault. A well-documented chart often negates the need for a clinician's appearance in court. When required for this task it is best for the examiner to work with the prosecuting attorney to prepare testimony. As is the case for all ED patients, chart notes should be written with the expectation that the ED evaluation and evidence collection will have to be explained and defended in court. In some jurisdictions it is possible to minimize the time spent away from work by arranging to be called to the courtroom just before the time of testimony or by giving a deposition before the court date. Once on the witness stand, remember that one is called as the examining clinician and not necessarily as an expert witness. The law requires that one testify only to one's best recollection and to what is indicated in the chart. Factual information in answer to questions should be given only if one knows the facts, and assumptions should be avoided. One should not be afraid to acknowledge the limits of one's knowledge or expertise. Statements such as "there were marks on the body that were consistent with bite marks" are preferable to statements such as "there were bite marks." It is the court's decision whether or not a person was sexually assaulted, and the clinician is there to give information about the patient's presentation, statements, what was found, and what was done for treatment.

Sexual Assault Response Team

Traditionally the responsibility of emergency clinicians, since the early 1990s nurses or nurse clinicians have been performing an increasing number of sexual assault examinations. Called SANE (sexual assault nurse examiners), these nurses are the core members of the sexual assault response teams (SARTs). Other members of the SARTs include law enforcement individuals, victim advocates, prosecutors, and forensic laboratory personnel.

Most examinations still take place in the ED but may be done in a space near the ED or an affiliated clinic. To establish SARTs, extra funding by government or charitable organization

is needed because most local police jurisdictions do not reimburse adequately for the evidentiary examination to support a program. Nurse examiners have formed The International Association of Forensic Nurses (IAFN). This group has drafted standards of practice for sexual assault examiners' education and the exams themselves. Advantages of SART using SANEs include

1. The practitioner performing the examination is specifically dedicated to treating the victim, not tending to multiple patients in a busy ED.
2. The clinician has usually completed more extensive training on sexual assault examination (mean of 80 hours) [20] and evidence collection and, as such, may perform a more comprehensive examination with better evidence collection. [65]

3. Many involved feel that designated clinicians consider the emotional needs of the victim more fully because of their extra time in training.

Useful guidelines and resources for establishing SANE programs are currently available. [2] [6]

Acknowledgment

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Chapter 60 - Drugs and Radiation In Pregnancy

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Pregnant women are frequently evaluated in emergency departments (EDs) with varied complaints that may require medication or diagnostic imaging. Their chief complaint may be pregnancy-related, an acute illness/injury, or related to a chronic condition diagnosed before pregnancy. The potential teratogenic effects of some medications are well documented, but adverse effects to the developing fetus from diagnostic radiation and radionuclide procedures are more perceived than real. Since fetal safety is a major concern, it is important for clinicians to have a clear understanding of the actual risk and benefits associated with prescribing medication and radiographic imaging during pregnancy.

RADIATION IN PREGNANCY

Radiological tests are ordered every day in a busy ED. The current actual number is unknown, but approximately 40 million x-ray examinations were done on female patients in the childbearing age group, 15 to 44 years old, in 1980. [1] With such a high volume of imaging studies, there is a high likelihood of caring for a patient who requires an imaging study and has an early unknown or known pregnancy.

In utero radiation exposure of the embryo or fetus generally causes great, but largely unnecessary, anxiety among the parents, their families, and clinician. Much of this anxiety is secondary to a general misconception that any radiation exposure is harmful and will result in an anomalous fetus. More often than not, clinicians themselves add to the confusion and fear by providing exposed women with erroneous information. Many clinicians, nurses, and even radiology technicians are ignorant of the qualitative and quantitative effects of ionizing radiation. [2] This misinformation could lead to inappropriate abortions and litigation. For example, in Greece, following the Chernobyl disaster, 23% of pregnancies were terminated due to unsubstantiated fears of teratogenicity. [3] A better understanding of the true risk estimates will help alleviate this fear.

It is widely held and published that *concerns about possible effects of ionizing radiation exposure should not prevent medically indicated diagnostic procedures from being performed on the mother*. It is not standard of care to withold necessary radiologic studies because of fear of fetal injury from diagnostic studies. According to the American College of Radiology, "No single diagnostic x-ray procedure results in radiation exposure to the degree that would threaten the well being of the preembryo, embryo, or fetus".[4] This remarkable statement helps put the effects of diagnostic radiation exposure on pregnancy in perspective. Standard diagnostic radiological procedures performed in the ED are not associated with significant proven fetal risks. A clear understanding of these risks enables the clinician to make an informed decision and knowingly counsel patients in order to provide more benefit than harm.

Evaluation of radiation exposure on a pregnant patient should involve consideration of the type of radiation, types of examinations performed, gestational age, and radiation dose in order to determine risk estimation. The radiation dose of interest is the dose absorbed by the embryo or fetus, and not by the mother. The main goal of this chapter is to review the basic issues of pregnancy and radiation exposure and provide a practical approach for clinicians in counseling the patient who has or will receive an emergent diagnostic procedure.

Types of Radiation

All imaging techniques involve radiation or transmission of energy from one body or source to another. Imaging modalities used for diagnosis during pregnancy can be subdivided into ionization techniques (x-rays, computed tomography [CT] scans, nuclear imaging) and nonionization techniques (magnetic resonance imaging [MRI] and ultrasonography [US]). The nonionization techniques have insufficient energy to ionize target cells. It is the ionization process and sequelae that induce health-related fetal effects.

Ionizing Radiation

Ionization is the transfer of energy to a medium by either electromagnetic or particulate radiation that is sufficient to overcome the binding energy of an electron. The electron may be ejected from the atom. Both electromagnetic waves consisting of uncharged particles (x-rays and gamma rays) and charged particles (alpha and beta) can produce radiation. Indirect ionization modalities such as x-rays release an electron from a source that interacts with the target. Direct ionization refers to charged particles, alpha and beta, that strike the target directly.

Units of Radiation

The units used to measure the effects of x-rays can be confusing. Descriptive terms include the *rad* and *rem*, along with the modern International System of Units (SI) of Gray and Sievert. In terms of *radiation protection*, the significant radiation quantity is the absorbed dose. The unit of *absorbed radiation* is the rad or the Gray (Gy) (1 Gy = 100 rad). Any risk associated with radiation is related to the amount of energy absorbed.

The dose equivalent expressed in *rem* or Sievert (Sv) is used to quantify the degree of biological effect (1 Sv = 100 rem). This unit reflects the biological response and can be used to compare effects of different types of radiation. The dose equivalent is the product of the absorbed dose times a quality factor. The quality factor is dependent upon the mass and charge of the radiated particle. The quality factor is approximately 20 for an alpha particle, and equal to 1 for x-rays and gamma rays. Therefore, for diagnostic x-rays, CT scans, and Tc^{99} nuclear studies, the absorbed dose is equal to the dose equivalent; that is, an absorbed dose of 1 rad yields a dose equivalent of 1 rem (1 Gy = 1 Sv) (Table 60-1). All reference data was converted into rads for uniformity and comparison throughout the chapter.

Timing of Radiation During Pregnancy and its Effects

The effects of exposure to radiation on the conceptus depend on the gestational age and the amount of absorbed dose.

TABLE 60-1 -- Units of Radiation

Quantity	Unit	SI Unit	Relationship between Units
Absorbed dose	rad	Gray (Gy)	1 Gy = 100 rad
			100 mGy = 10 rad
			10 mGy = 1 rad
			1 mGy = 100 mrad
Equivalent dose	rem	Sievert (Sv)	1 Sv = 100 rem
			100 mSv = 10 rem
			10 mSv = 1 rem
			1 mSv = 100 mrem

Absorbed dose (Gy) × quality factor = equivalent dose (Sv).

The quality factor for x-rays = 1. Therefore, 1 rad = 1 rem, 1 Gy = 1 Sv.

The relationship between radiation-induced effects and stage of pregnancy is shown in Figure 60-1 .[1] The harmful effects of ionizing radiation have the following principal biological effects: intrauterine death, organ malformations, mental impairment, fetal growth retardation, cancer, and genetic mutation. [2]

Radiation-induced health effects are divided into two broad categories, stochastic and nonstochastic effects (Table 60-2). Stochastic effects, such as cancer or genetic mutation, can result from alterations produced in a single cell and are presumed to exist even at low exposure. [2] The probability of such an effect occurring increases with dose and there is no identifiable threshold dose below which the chance is known to be zero. However, at low doses of radiation, the risks are far

below the spontaneous incidence of carcinogenesis [6] or mutagenesis.[7]

The remaining harmful biological effects mentioned earlier are nonstochastic effects. Nonstochastic effects require multicellular injury and have a threshold dose below which deleterious effects do not occur.[2] It is important to emphasize that the vast majority of embryopathologic effects are believed to be threshold phenomenon; therefore, a dose of ionizing radiation below the threshold will not produce these effects.

Stages of Fetal Development

Development of the unborn child is expressed as post-conception age and can be divided approximately into three major phases. These include 1) the preimplantation/implantation phase, (0 to 2 weeks), from conception to implantation, 2) the phase of major organogenesis, which extends from the 3rd to approximately the 8th week post-conception, and 3) the phase of fetal development lasting from 9 weeks until birth.

TABLE 60-2 -- Stochastic and Nonstochastic (Threshold) Comparison

Phenomenon	Pathology	Diseases	Risk	Definition
Stochastic	Damage to a single cell may result in disease	Cancer, germ cell mutation	Some risk exists at all dosages; at low doses, risk may be less than spontaneous risk	The incidence of the disease increases but the severity and nature of the disease remain the same
Nonstochastic (Threshold)	Multicellular injury	Intrauterine death, organ malformations, mental impairment, growth retardation	No increased risk below the threshold dose	Both the severity and incidence of the disease increase with dose

Modified from Brent RL: Utilization of developmental basic science principles: The evaluation of reproductive risks from pre- and postconception environmental radiation exposures. Teratology 54:182, 1999.

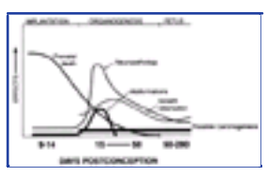

Figure 60-1 Schematic presentation of the various adverse effects associated with radiation and their relative incidence at different stages of gestation. *(Adapted from Mettler FA Jr, Upton A: Medical Effects of Ionizing Radiation, 2nd ed, Chapter 8, WB Saunders, 1995.)*

Preimplantation/Implantation

During the preimplantation and implantation phase the principal radiation-induced health effect is abortion. [8] When the number of cells in the conceptus is small and their nature not yet specialized, the effect of damage to these cells is most likely to take the form of failure to implant or an undeterminable death of the conceptus. [6] The *no effect threshold* of absorbed dose is quite high, estimated at 10 to 15 rad,[8] and not likely to be even distantly approached by diagnostic ED radiographs or radionuclide testing. By the time the pregnancy is at term, the threshold to cause intrauterine mortality has risen to about 100 rad. [8] These estimates have been extrapolated from animal data. This period has been referred to as the "all or none period" since radiation is more likely to kill the embryo than result in a live malformed newborn.

There are few human epidemiological data available for this period of gestation. Since many women are certainly unknowingly exposed to diagnostic radiation during this period, the lack of such data suggests a nonassociation between diagnostic radiation exposure and embryo death. Data from the Japanese atomic bomb experience have been cited for reference, but such a correlation is difficult to justify scientifically. Nonetheless, these data show a decrease in the number of offspring who retrospectively would have been 0 to 3 weeks postconception at the time of this significant radiation exposure, suggesting increased fetal loss caused by irradiation

TABLE 60-3 -- Risks and Threshold Doses of the Main Effects of Prenatal Irradiation

Gestational Age	Stage	Potential Biological Effect	Threshold	Risk
0–2 weeks	Preimplantation/Implantation	Abortion	>10 rad[2] [8]	
3–7 weeks	Organogenesis	Organ Malformation	>5[8] ; >10[2] [9] rad	
		Growth Retardation	>10 rad[2] [9]	
8–25 weeks	Fetal	Growth Retardation	>10 rad[2] [9]	
		Mental Impairment	>10 rad[9] [8]	
Whole Pregnancy		Carcinogenesis	None	6×10^{-4} per rad
		Mutagenesis	None	1×10^{-2} per rad

Modified from Fattibene P, Mazzei F, Nuccetelli C, Risica S: Prenatal exposure to ionizing radiation: Sources, effects, and regulatory aspects. Acta Paediatr 88:693, 1999.

during preimplantation.[9] This decrease in birth rate is likely multifactorial since stress, disease, and malnutrition were coexistent during this traumatic time. Importantly, fetal loss from exposure to an atomic bomb cannot be scientifically extrapolated to exposure to diagnostic radiation.

Any discussion concerning the potential adverse effects from diagnostic radiation must consider the natural incidence of spontaneous abortion. Since the main effect of radiation exposure during the first few weeks postfertilization is abortion due to embryo death, it is paramount to note that the normal incidence of spontaneous abortion in humans not exposed to radiation is in the 30% to 50% range. [2] *Exposure to <10 rad yields no statistical change in the rate of preimplantation/early postimplantation spontaneous abortion from the expected baseline (Table 60-3).*

Organogenesis

Very high-dose radiation exposure, far greater than could be delivered by even aggressive diagnostic radiographs/radionuclide procedures, has been reported to result in teratogenesis. Such information is gleaned from women who received therapeutic radiation (in the range of 250 rad) during early pregnancy for conditions such as pelvic malignancies. Organ malformations are the main consequence of radiation exposure during the organogenesis period (3 to 8 weeks). Abnormalities result from cell killing during the active phase of proliferation and differentiation. Since the embryo is unable to completely replace damaged cells, malformations occur. The most common effects of exposure during organogenesis are malformations caused in the organs under development at the time of exposure and the reduction of skeletal development. *Growth retardation and microcephaly are the predominant effects.* [6] [8] [9] These effects have a reported threshold dose range of 5 to 20 rads or higher, but are not generally observed unless the exposure is several orders of magnitude or higher. [6] [8] This dose range is significantly higher than what is reached in diagnostic radiology or diagnostic nuclear medicine procedures. While ocular abnormalities, developmental facial abnormalities, genital abnormalities, and physical deformities of the extremities have been reported after very high-dose embryonic exposure, such abnormalities are not linked to the amount of radiation that would be delivered from even multiple diagnostic radiographic procedures. Importantly, there are no reports of external radiation inducing morphologic malformations in humans unless offspring also exhibited either growth retardation or a central nervous system (CNS) abnormality. Simply stated, a fetus exposed to radiation will not

develop isolated structural abnormalities. The fear of extra toes, cleft palate, or heart or kidney malformations from diagnostic radiation during pregnancy is simply unfounded, yet often believed by the general public.

Temporary growth retardation is likely with doses in the range of 10 to 25 rad. [9] Infants with low birth weights and lengths may recover fully, attaining normal adult stature. The *natural incidence* of a live birth having a developmental anomaly is 2% to 4% [1] and intrauterine growth retardation is 2% to 3%. [1] Again, it is important to emphasize that exposure to less than 5 rad yields no change in the risk of occurrence of organ malformations or growth retardation [9] (see Table 60-3).

Fetal Period

The predominant observable effects of radiation exposure during this period are growth retardation, microencephaly, and severe mental retardation (SMR). The fetal stage has been subdivided into early fetal (8 to 15 weeks), mid fetal (16 to 25 weeks), and late fetal (26 weeks to term) because of identifiable periods of risk to the developing CNS.

Mental impairment.

From the 8th to the 15th week, there is a rapid increase in the number of neurons that migrate to their ultimate sites and lose their capacity to divide. At 15 to 25 weeks there is more differentiation and architectural definition. [1] These embryological changes make the fetus susceptible to CNS damage during the early and mid fetal periods.

The in utero atomic bomb survivor data indicates that the risk of SMR per rad was higher if exposed during the early fetal versus the mid fetal period. [9] In children receiving greater than 50 rad between 8 and 15 weeks postconception, a drop in IQ score of 0.3 points per rad was estimated. [9] There is no documented increased risk of mental retardation in humans at gestation age less than 8 weeks or greater than 25 weeks evaluated with doses of less than 50 rads. [10] The highest risk for SMR occurs during the early fetal period with fetal doses in the range of 100 rad. All the clinical observations on significant IQ reductions and SMR relate to fetal doses of about 50 rad and higher. [6] This dose range greatly exceeds the dosages utilized for diagnostic imaging.

It is important to relate the magnitude of radiation effects to those abnormalities that occur spontaneously in the population. There are multiple causes of mental retardation that have been identified, including malnutrition, lead poisoning, rubella infections during pregnancy, and maternal alcoholism. Current prevalence figures indicate that the normal incidence of a person with an IQ below 70 is approximately 3%. [6] At fetal doses of 10 rad, the spontaneous incidence of mental retardation is much larger than any potential radiation effect on IQ reduction. [6] Regardless of the time of gestation, IQ

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reduction cannot be clinically identified at fetal doses of <10 rad (see Table 60-3). [6]

Growth retardation.

The human data for Hiroshima and Nagasaki reveal the major congenital anomaly observed was microencephaly. [9] Studies have demonstrated no increased risk for microencephaly in the population exposed to <150 rad in Nagasaki; however, an increased risk in the Hiroshima population exposed to doses as low as 10 to 19 rad has been reported. [11] It is possible that the difference between the two cities is secondary to other causes (trauma, stress, malnutrition) than radiation. In experimental animal data, a dose of 10 to 20 rad does not increase the incidence of microencephaly. [11] A dose threshold for microencephaly, as well as other congenital anomalies, is generally accepted to be in the range of a few rad. Permanent growth retardation isn't typically seen unless doses exceed 50 rad. [9] Irradiation of the human fetus at doses below 10 rad has not been observed to cause congenital malformations or growth retardation (see Table 60-3). [1] [2] [4]

Carcinogenesis

The magnitude of risk for carcinogenesis following low dose radiation exposure and whether the risk changes throughout gestation has been the subject of many publications, [12] [13] [14] yet interpretation of the data remains open to date. Numerous studies [15] [16] [17] [18] indicate a 1.3- to 3.0-fold higher incidence of leukemia in children exposed to diagnostic radiation in utero, although some studies fail to substantiate the association. [11] [19] Excess cancer as a result of in utero exposure has not been clearly demonstrated among Japanese atomic bomb survivor studies even though the population has been followed for about 50 years, but the number exposed is not large. [6] Identification and control of confounding factors make interpretation of radiation carcinogenesis studies difficult, if not impossible, to interpret. Brent notes most investigators agree that low doses of radiation present a carcinogenic risk to the embryo; however, findings of an increased cancer risk among children exposed in utero to low-dose diagnostic radiation must be reconciled with the fact that high-dose animal and human studies have not found marked increase in cancer incidence. [11]

Risk can be expressed in several ways, including as *relative risk* or *absolute risk*. Relative risk indicates the risk as a function of the "background" cancer risk. A relative risk of 1.0 indicates that there is no effect of irradiation, whereas a relative risk of 1.5 for a given dose indicates that the radiation is associated with a 50% increase in cancer above background rates. The absolute risk estimate simply indicates the excess number of cancer cases expected in a population due to a certain radiation dose. [6]

The International Commission on Radiological Protection Publication 84 [6] notes a recent analysis of many of the epidemiologic studies conducted on prenatal x-ray and childhood cancer are consistent with a relative risk of 1.4 (a 40% increase over the background risk) following a fetal dose of about 1 rad. The best methodological studies, however, suggest that the risk is probably lower than this. Even if the relative risk were as high as 1.4, the individual probability of childhood cancer after in utero irradiation would be very low (about 0.3% to 0.4%) since the background incidence of childhood cancer is so low (about 0.2% to 0.3%). Absolute risk estimates for cancer risk from ages 0 to 15 after in utero irradiation have been estimated to be in the range of 600 per 10,000 persons each exposed to 100 rad, or 0.06% per rad. [6] [8] If a fetus is exposed to 0.1 rad, the increased risk for carcinogenesis is 0.006% or 3/50,000, compared to the background incidence of 0.2% to 0.3% or 100 to 150/50,000. The increased carcinogenic risk from 0.1 rad exposure is approximately 50 times smaller than the already low natural incidence of cancer.

Mutagenesis

Investigating possible radiation-induced alteration to human genome is exceedingly difficult. The geneticists who studied the radiated populations in Japan are convinced that there were radiation-induced mutations. However, the calculated and demonstrated risks were so small that the investigators were unable to demonstrate statistically significant genetic effects. [5]

The risk of radiation-induced hereditary disease in humans is reported to be around 1% per 100 rad. [9] [20] If a fetus is exposed to 0.1 rad, then the increased risk is approximately 0.001% or 1/100,000. The natural frequency of genetic disease manifesting at birth is approximately 3% [7] or 3000/100,000. For 0.1 rad, the increased genetic risk is minute compared to the natural incidence of genetic disease.

In order to put all risks into proper perspective, the range of fetal absorbed doses for diagnostic imaging must be reviewed. The vast majority of diagnostic radiographic studies are markedly <5 rad. A comparison of fetal absorbed doses for the more common ED radiographic procedures follows.

Radiation Exposure from Diagnostic Radiographs

Estimated fetal exposure for various diagnostic imaging modalities is presented in Table 60-4 . [21] The number of examinations required to reach a cumulative dose of 5 rads is calculated in the second column to underscore the order-of-magnitude difference between the dose considered to have negligible risk (5 rad) and the actual exposed fetal dose. For example, one would require 5000 x-rays of an upper or lower extremity, 125 pelvic x-rays, or an impressive 70,000 2-view chest x-rays before the 5 rad limit is reached.

Radiation Exposure from Computed Tomography Scans

Many variables affect the calculation of fetal radiation dose from CT scans, especially slice thickness, number of cuts, distance of target organ from fetus, and gestational age. Estimated maximal fetal doses from CT scans are summarized in Table 60-4 . It should be noted that a CT of the lumbar spine delivers radiation to the fetus that approaches the safe cutoff range. A CT scan of the abdomen exposes the fetus to less radiation than the 5 rad cutoff, but alternative methods of investigation such as US should be considered in early pregnancy if the clinical condition warrants.

The most commonly requested CT scan in pregnancy is the head CT. The expected fetal absorbed dose is less than 50 millirad (mrad), which is 100 times less than the dose with negligible risk. The estimated radiation dose to the fetus for CT of the chest is <0.100 rad. Spiral CT is becoming commonplace in radiology departments and is gaining

TABLE 60-4 -- Estimated Fetal Exposure for Various Diagnostic Imaging Methods

Examination Type	Estimated Fetal Dose per Examination (rad)	Number of Examinations Required for Cumulative 5-rad Dose
Plain Films		
Skull	0.004	1250
Dental	0.0001	50,000
Cervical spine	0.002	2500
Upper or lower extremity	0.001	5000
Chest (two views)	0.00007	71,429
Mammogram	0.020	250
Abdominal (multiple views)	0.245	20
Thoracic spine	0.009	555
Lumbosacral spine	0.359	13
Intravenous pyelogram	1.398	3
Pelvis	0.040	125
Hip (single view)	0.213	23
CT Scans (Slice Thickness: 10 mm)		
Head (10 slices)	<0.050	>100
Chest (10 slices)	<0.100	>50
Abdomen (10 slices)	2.600	1–2
Lumbar spine (multiple views)	3.500	1–2
Pelvimetry (1 slice with scout film)	0.250	20
Fluoroscopic Studies	0.056	89
Upper GI series	0.006	833
Barium swallow	3.986	1
Barium enema		
Nuclear Medicine Studies		
Most studies using technetium (^{99m} Tc)	<0.500	>10
Hepatobiliary technetium HIDA scan	0.150	33
Ventilation-perfusion scan (total)	0.215	23
Perfusion portion: technetium	0.175	28
Ventilation portion: xenon (^{133} Xe)	0.040	125
Iodine (^{131} I), at fetal thyroid tissue	590.000	
Environmental Sources (for comparison)		
Environmental background radiation (cumulative dose over nine months)	0.100	N/A

HIDA, hepatobiliary iminodiacetic acid; rad, the unit of absorbed radiation.

Reproduced from Toppenberg KS, Hill DA, Miller DP: Safety of radiographic imaging during pregnancy. Am Fam Physician 59:1813, 1999.

popularity as a diagnostic tool in suspected pulmonary embolism (PE) in the pregnant patient. The dose for a spiral CT of the chest is less because the duration of the procedure is much shorter.[22] CT pelvimetry exposes the fetus to approximately half of the radiation delivered from conventional plain film pelvimetry.

Iodinated contrast material that is injected intravenously for CT scans does not emit radiation and is classified *pregnancy category B*. It is not known to be teratogenic. The product insert for barium sulfate suspension used as oral contrast for an abdominal CT scan (e.g., Redicat) notes no adverse fetal reactions under the heading "Usage in Pregnancy." Barium preparations do not emit radiation.

Nuclear Medicine Studies

The most common nuclear medicine procedure ordered from the ED is the ventilation perfusion (V/Q) scan. The perfusion portion of the scan is performed by injecting a radioisotope intravenously. The isotope emits radiation and is detected by sensitive cameras. This requires that a radioisotope (e.g., Tc^{99}) be tagged to a substrate, most commonly albumin. The albumin-technetium aggregate is temporarily trapped in the arterioles and capillaries in the lung and its distribution can be identified. The principle photon that is useful for detection and imaging with technetium studies is the gamma ray.[23] When the radio-tagged substrate is excreted into the maternal bladder, the fetus will receive additional radiation exposure based on the proximity of the maternal bladder. Patient hydration and frequent voiding or bladder catheterization post V/Q scan will lessen the radiation exposure to the fetus. The measurement of radioactive substances is based on its decay, and the units are the Curie (Ci) or the Becquerel (Bq). Doses are usually expressed in milliCurie (mCi). The usual dose of technetium for the lung perfusion portion of the scan is 1 to 5 mCi of Tc^{99} . Reduced doses, as low as 1 mCi, are often used in pregnancy.

Depending on the radioisotope and substrate employed, the average fetal exposures can be calculated. Commonly used radiopharmaceuticals and estimated fetal doses for V/Q scans, and other radionuclide studies are given in Table 60-5 .[10] A 5 mCi Tc^{99} albumin scan results in 175 mrad fetal exposure. Reducing the dose to 2

mCi results in a 70 mrad fetal exposure. Technetium Tc^{99} albumin is contraindicated in patients with severe pulmonary hypertension and is *pregnancy category C*.

Ten mCi of $Xenon^{133}$ are used for the ventilation portion of the V/Q scan. $Xenon^{133}$ has a short half-life and results in 40 mrad fetal exposure. Normal findings on the perfusion scan may obviate the need for the ventilation scan, and some centers routinely perform only the perfusion portion since most pregnant women have normal ventilation.

Pulmonary Embolism Diagnosis

The reported incidence of PE associated with pregnancy varies widely from 1 in 2700 deliveries to less than 1 in 7000 deliveries. [24] The radiological modalities of choice for definitive diagnosis are V/Q scan, thoracic CT scan with contrast, or angiography. A positive US for deep vein thrombosis of the legs, coupled with a clinical scenario consistent with PE, may suggest a diagnosis and guide therapy. However, since many pulmonary emboli, especially those occurring during pregnancy, are from pelvic veins, this approach clearly has limitations in establishing the presence or absence of PE. [24]

V/Q scans are widely recognized as extremely useful diagnostic tools in the evaluation of PE; however, many clinicians are reluctant to order this test in pregnant patients. The main concern is uncertainty, or essentially misinformation,

TABLE 60-5 -- Radiopharmaceuticals Used in Nuclear Medicine Studies

Examination	Estimated Activity Administered per Examination (mCi)	Dose to Uterus/Embryo per Pharmaceutical (mrad)
Brain	20 mCi ^{99m}Tc DTPA	700
	20 mCi ^{99m}Tc O_4	960
Hepatobiliary	5 mCi ^{99m}Tc sulfur colloid	55
	5 mCi ^{99m}Tc HIDA	150
Bone	20 mCi ^{99m}Tc phosphate	500
Respiratory		
Perfusion	5 mCi ^{99m}Tc-macroaggregated albumin	175
Ventilation	10 mCi ^{133} xenon gas	
		40
Renal	20 mCi ^{99m}Tc DTPA	700
Abscess or tumor	3 mCi ^{67}Ga citrate	840
Cardiovascular	20 mCi ^{99m}Tc labeled red blood cells	120

DTPA, diethylenetriamine pentaacetic acid; Ga, gallium; HIDA, hepatobiliary iminodiacetic acid; mCi, millicurie; mrad, millirad; Tc, technetium.

Reproduced from Cunningham GF: Williams Obstetrics, 21st ed. New York, McGraw-Hill, 2001.

over actual fetal radiation exposure and the long-term effect on the fetus. The mortality rate of untreated acute PE is about 30% compared to 3% in treated patients. [24] Therefore, the potential morbidity of PE and the attendant risk of anticoagulant therapy in pregnant patients necessitate definitive diagnosis. The calculated radiation exposure to the fetus from both V/Q scanning and CT of the chest confers minimal, and essentially only theoretical, fetal risk. [22] Fetal exposure from CT of the chest is less than 0.100 rad. A 5 mCi Tc^{99} perfusion scan and 10 mCi Xe^{133} ventilation scan summates to 0.225 rad. [21] The dose of fetal radiation from the perfusion scan can be altered, and is often lowered in the evaluation of pregnant patients (Table 60-6). Lowering the dose by 60%, a level that will usually produce a suitable study, results in lowering fetal exposure to 0.110 mrad. Pulmonary angiography results in an estimated fetal exposure of 0.22 to 0.37 rad when done via the femoral route but can be lowered to less than 0.05 rad by using the brachial route. [22]

The role of D-dimer levels in the diagnosis of PE is evolving. [25] [26] During pregnancy, D-dimer levels increase and should be considered physiological. D-dimer levels are similar to nonpregnant patients up to around 20 weeks, then noted to increase throughout pregnancy to 3 times higher than the mean of a healthy nonpregnant patient. [27]

Diagnosis of Pregnancy and Consent

If exposure <5 rad does not measurably affect the exposed embryo, then why should the clinician determine the pregnancy status of the patient? Brent reports sound reasoning for diagnosis of pregnancy prior to radiographic study. [2] First, the diagnosis of pregnancy may be in the differential diagnosis for the patient's presenting problem and may change or obviate any further need for imaging. Second, it is beneficial to have the patient informed of pregnancy status prior to the imaging, if possible. An informative discussion about the

TABLE 60-6 -- The Technique of Ventilation/Perfusion (V/Q) Scanning

This test uses both intravenous ([IV] perfusion) and aerosolized (ventilation) agents.
Perfusion
1. Prior to injection, the IV technetium (Tc) must be prepared. Sodium pertechnetate Tc-99m is mixed with macroaggregated human albumin (MAA), forming Tc-99m MAA, the substance that is injected intravenously to investigate blood flow in the lungs. If the preparation is not used within 8 hrs, it is discarded.
2. The usual dose is 1–5 millicurie (mCi). Doses as low as 1 mCi are used in pregnancy.
3. Within 5 min of injection, more than 90% of the Tc albumin aggregate is trapped in the arterioles and capillaries of the lung. The particle size determines where the Tc-99m will be localized in the body.
4. The accumulation in the lung is temporary, and fragile albumin aggregate quickly breaks down, allowing the Tc to enter the general circulation.
5. Once in the body, the half-life of Tc-99m is 6 hrs.
6. The majority of the Tc-99m is excreted in the urine. If it remains in the urinary bladder, it is in close proximity to the fetus.
7. Tc in the bladder also exposes the fetus to small amounts of radiation.
8. Frequent voiding or bladder catheterization after the study will lessen radiation exposure to the fetus.
9. Tc is relatively contraindicated in patients with severe pulmonary hypertension (since the Tc-99m MAA temporarily blocks blood flow in the lungs).
10. Allergic reactions to Tc and human serum albumin are extremely rare.
11. The radiation exposure to the total body from 4 mCi is extremely low: less than 0.1 rad.
Ventilation
1. Some radiologists forgo the ventilation portion of the V/Q scan in pregnancy to limit the total radiation exposure.
2. Most hospitals use xenon 133 for the ventilation portion of the V/Q scan.

3. The fetal radiation from xenon is extremely low, and the 5 rad cutoff is not reached until more than 125 scans have been performed.
4. The estimated dose to the fetus of a standard 10 mCi of xenon used in a V/Q scan is 0.04 rad.
5. If Tc based aerosol is the marker used for the ventilation portion, fetal exposure is higher than with xenon aerosol.

risk/benefit aspects of the test *prior to the study* conveys a concern for the patient and fetus. Discussing the risk/benefit aspects of imaging *after the study* may be misconstrued as "back stepping" and make the patient upset. Many lawsuits are stimulated by the factor of surprise. A frank discussion prior to the imaging may prevent misguided litigation.

Amenorrhea and physical changes in size and shape of the uterus may be consistent with pregnancy. However, menstrual history by itself may not be totally reliable in the determination of pregnancy. A history of recent menstruation, intrauterine device, tubal ligation, no coitus, or the proper use of birth control pills will result in the suggestion of pregnancy more than 90% of the time, but these parameters are not 100% accurate. If the diagnosis of pregnancy is in the differential or imaging is ordered, or both, the definitive determination of

the patient's pregnancy status should be strongly considered if the clinical scenario is reasonable. It is not standard of care to order a pregnancy test on all women of childbearing age prior to obtaining routine diagnostic radiographs. However, a menstrual history and other information should be obtained whenever possible. Urine pregnancy tests to detect early pregnancy are quite sensitive and reliable, and it is not necessary to routinely order a quantitative serum test. Theoretically, there will be a few days' window between fertilization and implantation when no method will exist to confirm the presence or absence of early pregnancy.

The pregnant patient has the right to know the magnitude and type of risks that might result from in utero exposure. The Annals of the International Commission on Radiological Protection Publication 84 [6] summarized the need for informed consent as follows: "The need and degree of disclosure is usually measured by what a reasonable person believes is material to the mother's decision to be exposed to radiation. The level and degree of disclosure should be related to the level of risk. For low-dose procedures, such as chest x-rays, <100 mrad, the only information that may be needed is a verbal assurance that the risk is judged to be extremely low. When fetal doses are >100 mrad, usually a more detailed explanation is given. The information should include potential radiation risks and potential alternative modalities as well as the risk of harm from not having the medical procedure. The degree of documentation of such explanations and consent is variable but many clinicians will include a note of any such counseling or consent in the record of the patient."

Patient Counseling

When a pregnant patient requires an imaging study, one should be prepared to discuss the risk associated with the test. Counseling can be accomplished after attempting to estimate the dose to the conceptus from the procedure and comparing the radiation risk with other risks of pregnancy. It is important to use terminology that is easily understood by the patient. Figure 60-2 depicts three different strategies to inform the patient about the level of exposure from her study compared to established limits. Table 60-7 compares the level of exposure to established background risks.

The main bar graph (*A*) in Figure 60-2 compares the fetal exposure level for various radiographic studies to the maximum accepted fetal dose during pregnancy (5 rad). A patient's particular study may be plotted on this graph, showing the clear margin of safety that exists for all single diagnostic tests.

The upper graph (*B*) equates the exposure from the low-level diagnostic studies to the number of hours needed to accumulate a similar exposure dose from background terrestrial radiation. One of the most commonly ordered studies in pregnancy is a chest x-ray. The potential risk to the fetus can be put into perspective for the patient by comparing the absorbed dose for the chest x-ray to the natural background radiation exposure. The environmental background radiation over 9 months has a cumulative dose of 100 mrad, [28] or 0.015 mrad per hour. The fetal dose exposure for a chest x-ray (2 views) is estimated at 0.07 mrad. Therefore, the exposure dose to the fetus from a chest x-ray is equivalent to the same amount of naturally occurring background radiation to which the patient was exposed in the previous 5 hours.

The lower graph (*C*) depicts the Nuclear Regulatory Commission's (NRC) upper limit for cumulative gestational dose compared to various diagnostic studies. The NRC has established occupational radiation dose limits for pregnancy. Its recommendation is that the dose to the fetus not be allowed to exceed 0.5 rem during gestation. Brent notes that this factor-of-10 lowering of the widely accepted threshold is "extremely conservative." [2] One can explain to the patient that the level of exposure from her x-ray is below the conservative cumulative acceptable dose for a pregnant employee at a nuclear facility in the United States.

Another useful approach is to indicate to the patient the probability of *not* having a child with either a malformation or cancer, and how that probability is affected by radiation. Table 60-7 depicts the probability of bearing healthy children as a function of radiation dose. This discussion should be coupled with the fact that the nonexposed fetus has a baseline incidence of spontaneous abortion, multiple developmental abnormalities, and subsequent childhood cancer.

Numerous organizations have declared fetal exposure to <5 rad as safe. Table 60-8 presents various conclusions from key organizations on use of radiation and pregnancy. *The International Commission on Radiological Protection concludes that fetal doses below 10 rad should not be considered a reason for terminating a pregnancy.* [6] If a patient still has considerable concern or has possibly received >5 rad, referral of the patient to a radiation physicist or genetic specialist for further counseling is reasonable.

Nonionizing Radiation: Magnetic Resonance Imaging and Ultrasonography

The term radiation describes the transmission of energy from one body or source to another. Nonionizing radiation includes the portion of the electromagnetic spectrum where the energy of emitted photons is insufficient to ionize atoms and molecules. MRI and US are forms of nonionizing radiation.

Magnetic Resonance Imaging

MRI is currently not approved by the U.S. Food and Drug Administration (FDA) for use in pregnant patients. [29] However, a body of data in this population is developing. MRI is becoming a valuable complement to US when additional information is needed to make treatment decisions during pregnancy. [30] [31] Recent advances in fast MRI techniques have helped to eliminate prior obstacles of slow imaging times and fetal movement. Possible indications for MRI use in a pregnant patient include further evaluation of adnexal masses, placental evaluation, hydronephrosis, pelvic vein thrombosis, and small bowel obstruction. [32]

The most common reason for emergent MRI scans from the ED is evaluation of neurological emergencies (e.g., spinal cord compression). In light of the serious sequelae of spinal cord compression along with a lack of data supporting adverse fetal effects, a pregnant patient exhibiting symptoms of a cord compression should have a magnetic resonance image taken to establish a diagnosis.

The Safety Committee of the Society for Magnetic Resonance Imaging states that MRI procedures are indicated for use in pregnant women when other nonionizing diagnostic imaging methods are inadequate or when the examination will provide important information that would otherwise require exposure to ionizing radiation. [33] It is required to inform pregnant patients that, although to date there is no

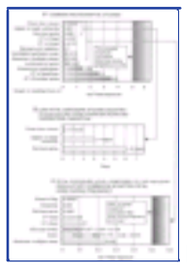

Figure 60-2 *Graph A*: Comparison of common radiographic studies with the accepted 5 rad cumulative fetal exposure limit. *Graph B:* Low fetal exposure studies equated to equivalent dose from background terrestrial radiation. *Graph C*: Fetal exposure level compared to the Nuclear Regulatory Commission's accepted fetal dose during pregnancy.

TABLE 60-7 -- Probability of Bearing Healthy Children as a Function of Radiation Dose

Radiographic Study	Fetal Exposure (rad)	Increased Risk of Abortion, Growth Retardation, Mental Impairment, and Malformation	Probability That Child Will Have *No* Malformation %	Probability That Child Will Have *No* Genetic Disease Present at Birth*,%	Probability That Child Will *Not* Develop Cancer (Age 0–19)†,%
	0	None	97	97	99.7
Chest x-ray 2 views	0.0007	None	97	97	99.7
Extremity	0.001	None	97	97	99.7
Cervical spine	0.002	None	97	97	99.7
CT head	< 0.05	None	97	97	99.7
CT chest	< 0.10	None	97	97	99.7
V/Q scan 3 mCi	0.145	None	97	97	99.7
V/Q scan 5 mCi	0.215	None	97	97	99.7
Multiple studies	1	None	97	97	99.6
Multiple studies	5	None	97	97	99.4

Modified from International Commission on Radiological Protection Publication 84: Pregnancy and medical radiation. Ann ICRP 30:iii, 1, 2000.

*Rounded values. Radiation risk for genetic mutation assumed at 0.01% per rad fetal dose and a linear dose response relationship. Background rate for genetic mutation estimated at 3%. From Osie IK: Fetal doses from radiological examinations. Br J Radiol 72:773, 1999.

†Rounded values. Radiation risk for fatal cancer assumed to be 0.06% per rad fetal dose and a linear dose response relationship. Many epidemiological studies suggest that the risk may be lower than that assumed here. Background risk of childhood cancer calculated from NCI-SEER: Surveillance, Epidemiology and End Results Cancer Statistics Review 1973–1991: Tables and Graphs. Bethesda, MD, National Cancer Institute, 1994.

TABLE 60-8 -- Key Statements on Diagnostic Imaging Modalities During Pregnancy

X-ray Imaging
"No single diagnostic procedure results in a radiation dose that threatens the well-being of the developing embryo and fetus." *(American College of Radiology) From Hall EJ: Scientific view of low-level radiation risks. Radiographics 11:509, 1991.)*
"[Fetal] risk is considered to be negligible at 5 rad or less when compared to the other risks of pregnancy, and the risk of malformations is significantly increased above control levels only at doses above 15 rad." *(National Council on Radiation Protection and Measurements) From NCRPM: Medical Radiation Exposure of Pregnant and Potentially Pregnant Women. NCRPM Report no. 54. Bethesda, MD, NCRPM, 1977.)*
"Women should be counseled that x-ray exposure from a single diagnostic procedure does not result in harmful fetal effects. Specifically, exposure to less than 5 rad has not been associated with an increase in fetal anomalies or pregnancy loss." *(American College of Obstetricians and Gynecologists [ACOG], Committee on Obstetric Practice) From ACOG: Guidelines for Diagnostic Imaging During Pregnancy. ACOG Committee Opinion No. 158. ACOG, Washington, DC, 1995.*
Magnetic Resonance Imaging
"Although there have been no documented adverse fetal effects reported, the National Radiological Protection Board arbitrarily advises against its use in the first trimester." *(American College of Obstetricians and Gynecologists [ACOG], Committee on Obstetric Practice) From ACOG: Guidelines for Diagnostic Imaging During Pregnancy. ACOG Committee Opinion No. 158. ACOG, Washington, DC, 1995.*
Ultrasound Imaging
"There have been no reports of documented adverse fetal effects for diagnostic ultrasound procedures, including duplex Doppler imaging." "There are no contraindications to ultrasound procedures during pregnancy, and this modality has largely replaced x-ray as the primary method of fetal imaging during pregnancy." *(American College of Obstetricians and Gynecologists [ACOG], Committee on Obstetric Practice) From ACOG: Guidelines for Diagnostic Imaging During Pregnancy. ACOG Committee Opinion No. 158. ACOG, Washington, DC, 1995.*
Reproduced from Toppenberg KS, Hill DA, Miller DP: Safety of radiographic imaging during pregnancy. Am Fam Physician 59:1813, 1999.

indication that the use of clinical MRI procedures during pregnancy produces deleterious effects, according to the FDA, the safety of MRI procedures during pregnancy has not been definitively proved. It is advisable to obtain informed consent for MRI of a pregnant patient. Also, because of limited data, most facilities avoid imaging patients in their first trimester. With an inadvertent exposure in a wanted pregnancy, however, the present accumulated data would not warrant an interruption of the pregnancy (Table 60-9).

Gadolinium-based contrast material is not recommended for use in pregnant patients. [32] Gadolinium-based contrast material has been shown to cross the placenta and appear within the fetal bladder moments after intravenous administration. [34] It is then excreted into the amniotic fluid and potentially reabsorbed from the gastrointestinal tract. [34] Because of this reabsorption, the half-life of gadolinium-based contrast material in the fetal circulation is not known.

TABLE 60-9 -- Summary of Magnetic Resonance Imaging (MRI) During Pregnancy

1. MRI involves no ionizing radiation.
2. There are no known biologic risks associated with MRI and no specific fetal abnormalities have been linked with standard low-intensity MRI scanning.
3. Most obstetrical problems can be adequately evaluated with ultrasound.
4. Contrast material is often avoided in conjunction with MRI scanning during pregnancy.
5. Both maternal and fetal anatomy, including the fetal central nervous system, can be evaluated with MRI.
6. The FDA requires documented informed consent and cautions that the full effects of MRI during pregnancy have not yet been determined.
7. MRI is usually eschewed during the first trimester unless there is a clear risk-to-benefit indication, such as possible spinal cord compression.
8. Claustrophobia and the pregnant woman's inability to tolerate prolonged lying on the back can occasionally be problematic.

9. Fetal movement limits information on the fetus unless sedation is provided or ultrafast scans are available.

Ultrasonography

US continues to be the screening modality of choice for the evaluation of the maternal pelvis and the fetus because of its safety profile, relatively low cost, and real-time capability. Obstetric and gynecological US account for more than half of the US imaging volume in the United States. [35] Human data accumulated over 25 years has revealed no consistent adverse effects from prenatal diagnostic US examinations. [36] [37] US in pregnancy is considered a safe procedure.

The American College of Obstetricians and Gynecologists has reviewed the effects of x-rays, US, and MRI exposure during pregnancy and suggested guidelines for radiographic examination during pregnancy (Table 60-10).[4]

Summary

In summary, the threshold dose for the non-stochastic effects throughout the gestational period is >5 rad. Prenatal doses of <5 rad present no measurable increased risk of prenatal death, malformations, growth retardation, or impairment of mental development over the background incidence of these entities. The risk for stochastic effects, carcinogenesis or mutagenesis, is related to the fetal absorbed dose and is very small compared to the natural background incidence of childhood cancer and genetic disease for most diagnostic procedures.

The vast majority of radiographic imaging obtained in the ED exposes the fetus to 100 times less than the threshold for adverse effects. The 5 rad threshold for onset of concern for adverse fetal effects is quite conservative, and any statistically significant change in fetal outcome probably requires at least several times this dose. Utilization of one of the methods put forth in this chapter to counsel pregnant patients in need of diagnostic imaging, and for women inadvertently exposed to radiation prior to the recognition of pregnancy, should help to educate patients and alleviate their fear.

TABLE 60-10 -- Guidelines for Emergency Department Diagnostic Imaging During Pregnancy

1. Women should be counseled that x-ray exposure from a single diagnostic procedure does not result in harmful fetal effects. Specifically, exposure to <5 rad has not been associated with an increase in fetal anomalies or pregnancy loss.
2. Concern about possible effects of high-dose ionizing radiation exposure should not prevent medically indicated diagnostic x-ray procedures from being performed on the mother. During pregnancy, other imaging procedures not associated with ionizing radiation, such as ultrasonography and magnetic resonance imaging, should be considered instead of x-rays when possible.
3. Ultrasonography and magnetic resonance imaging are not associated with known adverse fetal effects. However, until more information is available, magnetic resonance imaging is not recommended during the first trimester.
4. Consultation with a radiologist may be helpful in calculating estimated fetal dose when multiple diagnostic x-rays are performed on a pregnant patient.
5. The use of radioactive isotopes of iodine is contraindicated for therapeutic use during pregnancy.

Reproduced from American College of Obstetricians and Gynecologists (ACOG), Committee on Obstetric Practice: Guidelines for Diagnostic Imaging During Pregnancy. ACOG Committee Opinion No. 158. ACOG, Washington, DC, 1995.

DRUGS IN PREGNANCY

Hundreds of medications are prescribed in EDs across the nation every day. Emergency clinicians routinely inquire about pregnancy status before prescribing medication because of concern about potential adverse fetal effects. Concerns about drug effects on fetal development were heightened in the 1950s and '60s because several prescription medications fueled consumer outrage and provided the impetus for more efficient drug safety profiling. Thalidomide, diethylstilbestrol (DES), and to a lesser extent, Bendectin, sensitized the international health care community to controversy regarding drug use during pregnancy. For decades it was believed that the placenta served as a barrier that protected the fetus from the adverse effects of drugs. The thalidomide disaster drastically changed this perception by demonstrating that fetal exposure to a drug during critical periods of development resulted in fetal anomalies.

Thalidomide was prescribed to pregnant women in the 1950s and '60s because of its antiemetic and sedative effects. Many fetuses of women who took thalidomide developed phocomelia: the absence or malformation of either upper or lower limbs. It is estimated that 7000 deformed infants worldwide resulted from fetal exposure to thalidomide.[36] The majority of the exposed infants occurred in Europe. Less than 1% of Thalidomide-associated embryopathy occurred in the United States. [38] The low incidence of thalidomide babies born in the United States is directly attributable to the FDA, which did not want to distribute the drug without more rigorous safety testing.

DES was promoted as a miracle drug for women thought to be at risk for pregnancy-related problems. Before it was finally banned in 1971, an estimated 3 million pregnant women had been treated with DES-type drugs.[38] Physical consequences of in utero exposure to DES for female offspring include a rare form of vaginal and uterine cancer known as clear cell carcinoma; structural defects of sex organs; dysplasia of cervix; infertility; and adverse pregnancy outcomes. [38] Male offspring exposed to DES in utero have reportedly been at increased risk for anatomical abnormalities of the penis and testicles and increased risk for reproductive dysfunction.[38] The women who ingested the drug are also being followed to detect late adverse consequences, notably an increased risk of breast cancer.

Bendectin, a combination of an antihistamine (doxylamine) and pyridoxine, was the most widely used and only FDA-approved medication for nausea and vomiting associated with pregnancy. During the 1970s, however, many lawsuits claiming that Bendectin was teratogenic were filed against the manufacturer. It is estimated that more than 30 million pregnant women used the drug before it was removed in 1983.[38] The drug was withdrawn despite a substantial body of evidence that the rate of major malformation among the children of women who had received Bendectin during pregnancy did not differ from the rate in the general population. [39] However, comparisons of Bendectin to thalidomide eroded consumer confidence and fueled litigation. Although the potential teratogenicity of Bendectin was never substantiated and was statistically exonerated, the manufacturer withdrew Bendectin because it believed it would be more costly to fight the lawsuits than to settle them.

In 1979, drug safety and fetal well-being concerns prompted the FDA to develop a drug classification "Use in Pregnancy" ratings system (Table 60-11).[39] This system was developed to provide therapeutic guidance to clinicians as they consider the potential maternal/fetal risks and benefits of using a drug during pregnancy.[40] The ratings are written in such a manner that discourages nonessential use of medications during pregnancy. This can be alarming to a clinician or pregnant woman who is concerned about a pregnancy in

TABLE 60-11 -- Use-in-Pregnancy Ratings (U. S. Food and Drug Administration, 1979)

Category	Interpretation
A	Controlled studies show no risk. Adequate, well-controlled studies in pregnant women have failed to demonstrate risk to the fetus.
B	No evidence of risk in humans. Either animal findings show risk (but human findings do not) or, if no adequate human studies have been done, animal findings are negative.
C	Risk cannot be ruled out. Human studies are lacking, and animal studies are either positive for fetal risk or lacking as well. However, potential benefits may justify the potential risk.
D	Positive evidence of risk. Investigational or post-marketing data show risk to the fetus. Nevertheless, potential benefits may outweigh the potential risk.
X	Contraindicated in pregnancy. Studies in animals/humans or investigational/post-marketing reports have shown fetal risk that clearly outweighs any possible benefit to the patient.

Reproduced from FDA: Classification of Drugs for Teratogenic Risk. Teratology 49:446, 1994.

TABLE 60-12 -- Drugs with Proven Teratogenic Effects in Humans*

Drug	Teratogenic Effect
Methotrexate	CNS and limb malformations
Angiotensin-converting-enzyme inhibitors	Prolonged renal failure in neonates, decreased skull ossification, renal tubular dysgenesis
Anticholinergic drugs	Neonatal meconium ileus
Antithyroid drugs (propylthiouracil and Methimazole)	Fetal and neonatal goiter and hypothyroidism, aplasia cutis (with Methimazole)
Carbamazepine	Neural-tube defects
Cyclophosphamide	CNS malformations, secondary cancer
Danazol and other androgenic drugs	Masculinization of female fetuses
Diethylstilbestrol	Vaginal carcinoma and other genitourinary defects in female and male offspring
Hypoglycemic drugs	Neonatal hypoglycemia
Lithium	Ebstein anomaly
Misoprostol	Moebius sequence
Nonsteroidal anti-inflammatory drugs	Constriction of the *ductus arteriosus* necrotizing enterocolitis
Paramethadione	Facial and CNS defects
Phenytoin	Growth retardation, CNS deficits
Psychoactive drugs (e.g., barbiturates, opioids, and benzodiazepines)	Neonatal withdrawal syndrome when drug is taken in late pregnancy
Systemic retinoids (isotretinoin and etretinate)	CNS, craniofacial, cardiovascular, and other defects
Tetracycline	Anomalies of teeth and bone
Thalidomide	Limb-shortening defects, internal-organ defects
Trimethadione	Facial and CNS defects

Valproic acid	Neural-tube defects
Warfarin	Skeletal and CNS defects, Dandy-Walker syndrome

CNS, central nervous system

Reproduced from Koren G, Pastuszak A, Ito S: Drugs in pregnancy. N Engl J Med 338:1128, 1998.

*Only drugs that are teratogenic when used at clinically recommended doses are listed. The list includes all drugs proved to affect neonatal morphology or brain development and some of the toxic manifestations predicted on the basis of the pharmacologic actions of the drugs.

which medication is indicated or use has already occurred. Clinicians often inappropriately use this system as the basis for decisions regarding the continuation or termination of pregnancy in women who have received a medication.[40]

Although very few drugs are recognized as human teratogens, the 1992 Physicians' Desk Reference carried an FDA rating of category X on 7% of 1033 drugs listed (Category A, 0.7%; Category B, 19%; Category C, 66%; Category D, 7%). [40] Use of letters A, B, C, D, and X in the FDA system implies a gradation of risk, but the classification of agents does not consistently follow such a gradation. The vast majority of drugs are in category C. The default assignment of category C is misleading to many clinicians who consider this rating to indicate some degree of risk rather than just a lack of information. The Teratology Society has

TABLE 60-13 -- Common Drugs Initially Thought Teratogenic but Subsequently Proved Safe

Drug	Initial Evidence of Risk	Subsequent Evidence of Safety
Diazepam*	Oral clefts	No increase in risk in large cohort and case-control studies
Oral contraceptives	Birth defects involving the vertebrae, anus, heart, trachea, esophagus kidney, and limbs; masculinizing effects on female fetuses resulting in pseudohermaphroditism	No association between first-trimester exposure to oral contraceptives and malformations in general or external genital malformations in two meta-analyses
Spermicides	Limb defects, tumors, Down syndrome; and hypospadias	No increase in risk in a meta-analysis
Salicylates	Cleft palate and congenital heart disease	No increase in risk in large cohort studies
Bendectin (doxylamine plus pyridoxine)	Cardiac and limb defects	No increase in risk in two meta-analyses

Reproduced from Koren G, Pastuszak A, Ito S: Drugs in pregnancy. N Engl J Med 338:1128, 1998.

*Diazepam taken near term may cause the neonatal withdrawal syndrome or cardiorespiratory instability.

proposed that the FDA abandon the current classification system in favor of more meaningful, evidence-based, narrative statements. [40] This proposal has received public support.[41]

A fetus is subject to the dangers of teratogens carried by the placenta throughout all phases of growth. The organogenesis phase (3 to 8 weeks) is most critical because it encompasses organ development. Surprisingly, the number of drugs or medications that are strongly suspected or proven human teratogens is small. Forty years after the recognition of thalidomide-associated embryopathy, fewer than 30 drugs have been proven to be teratogenic in humans when used in clinically effective doses (Table 60-12). [41] Many other commonly used drugs, including oral contraceptives and diazepam, were once thought to be teratogenic, but have been shown to be safe in subsequent studies (Table 60-13).[41]

TABLE 60-14 -- Select Drugs for Safe Use During Pregnancy, According to Condition

Condition	Drugs of Choice	Alternative Drugs	Comments
Acne	Topical: erythromycin, clindamycin, benzoyl peroxide	Systemic: erythromycin, topical tretinoin (vitamin A acid)	Isotretinoin is contraindicated
Allergic rhinitis	Topical: glucocorticoids, cromolyn, decongestants, xylometazoline, oxymetazoline, naphazoline, phenylephrine; systemic: diphenhydramine, dimenhydrinate, tripelennamine, astemizole		
Constipation	Docusate sodium, calcium, glycerin, sorbitol, lactulose, mineral oil, magnesium hydroxide	Bisacodyl, phenolphthalein	
Cough	Diphenhydramine, codeine, dextromethorphan		
Depression	Tricyclic antidepressant drugs, fluoxetine	Lithium	When lithium is used in first trimester, fetal echocardiography and ultrasonography are recommended because of small risk of cardiovascular defects
Diabetes	Insulin (human)	Insulin (beef or pork)	Hypoglycemic drugs should be avoided
Headache			
Tension	Acetaminophen	Aspirin and nonsteroidal anti-inflammatory drugs, benzodiazepines ß-adrenergic-receptor antagonists and tricyclic antidepressant drugs (for prophylaxis)	Aspirin and nonsteroidal anti-inflammatory drugs should be avoided in third trimester
Migraine	Acetaminophen, codeine, dimenhydrinate		Limited experience with ergotamine has not revealed evidence of teratogenicity, but there is concern about potent vasoconstriction and uterine contraction
Hypertension	Labetalol, methyldopa	ß-adrenergic-receptor antagonists, prazosin, hydralazine	Angiotensin-converting-enzyme inhibitors should be avoided because of risk of severe neonatal renal insufficiency
Hyperthyroidism	Propylthiouracil, methimazole	ß-adrenergic-receptor antagonists (for symptoms)	Surgery may be required; radioactive iodine should be avoided

Mania (and bipolar affective disorder)	Lithium, chlorpromazine, haloperidol	For depressive episodes: tricyclic antidepressant drugs, fluoxetine, valproic acid	If lithium is used in first trimester, fetal echocardio graphy and ultrasonography are recommended because of small risk of cardiac anomalies; valproic acid may be given after neuraltube closure is complete
Nausea, vomiting, motion sickness	Diclectin (doxylamine plus pyridoxine)	Chlorpromazine, metoclopramide (in third trimester), diphenhydramine, dimenhydrinate, meclizine, cyclizine	
Peptic ulcer disease	Antacids, magnesium hydroxide, aluminum hydroxide, calcium carbonate, ranitidine	Sucralfate, bismuth subsalicylate	
Pruritus	Topical: moisturizing creams or lotions, aluminum acetate, zinc oxide cream or ointment, calamine lotion, glucocorticoids; systemic: hydroxyzine, diphenhydramine, glucocorticoids, astemizole	Topical: local anesthetics	
Thrombophlebitis, deep-vein thrombosis	Heparin, antifibrinolytic drugs, streptokinase		Streptokinase is associated with a risk of bleeding; warfarin should be avoided

Reproduced from Koren G, Pastuszak A, Ito S: Drugs in pregnancy. N Engl J Med 338:1128, 1998.

TABLE 60-15 -- Classification of Some Commonly Used Drugs in Emergency Department

ANALGESICS	
Aspirin	D
Acetaminophen	B
NSAIDS	
Celecoxib	C (3rd)
Ibuprofen	B (3rd)
Ketorolac	C (3rd)
Naproxen	B (3rd)
Rofecoxib	C (3rd)
OPIOIDS	
Codeine	C
Fentanyl	C
Hydromorphone	C
Meperidine	C
Morphine	C
Oxycodone	C
OPIOID ANTAGONIST	
Nalmefene	B
Naloxone	B
ANESTHETICS & SEDATIVES	
Etomidate	C
Fentanyl	C
Ketamine	B
Midazolam	D
Propofol	B
Thiopental	C
NEUROMUSCULAR BLOCKERS	
Pancuronium	C
Rocuronium	B
Succinylcholine	C
Vecuronium	C
LOCAL ANESTHETICS	
Bupivacaine	C
Etidocaine	B
Lidocaine	B
Mepivacaine	C
ANTIBIOTICS	
Aminoglycosides	D
Cephalosporins (1st–4th)	B
Penicillins (1st–4th)	B
Quinolones (1st–4th)	C
Tetracyclines	D
Carbapenems	B/C
Macrolides	B/C

Sulfonamides	C
OTHER ANTIMICROBIALS	
Clindamycin	B
Metronidazole	B (1st)
Nitrofurantoin	B
Rifampin	C
Vancomycin	C
ANTIVIRALS	
Anti-Cytomegalovirus	C
Anti-Herpetic	B
Anti-Human immunodeficiency virus	
Non-Nucleoside reverse transcriptase inhibitors	C
Nucleoside reverse transcriptase inhibitors	B/C
Protease Inhibitor	B/C
ANTIFUNGALS	
Amphotericin	B/C
Clotrimazole	C
Fluconazole	C
Griseofulvin	C
Itraconazole	C
Nystatin	B
Terbinafine	B
ANTIPARASITICS	
Mebendazole	C
Pentamidine	C
Pyrimethamine	C
Permethrin	B
RID	C
1st Prohibited usage during first trimester	
3rd Prohibited usage during third trimester	
(1st–4th) First through fourth generations	

Since fetal safety is a major concern, effective drugs that have been in use for long periods are preferable to newer alternatives. If drugs are required for certain conditions, the safest drugs at the lowest effective dose should be used. Table 60-14 lists selected drugs considered to be safe for common conditions afflicting pregnant women. Table 60-15 lists the classification of commonly used drugs in the ED. [42]

To receive up-to-date, evidence-based information on the safety and risk of drugs during pregnancy, clinicians can consult a teratogen information service. Table 60-16 lists

TABLE 60-16 -- Select Teratogen Information Services

Canada
Motherisk Program, Toronto
(416) 813-6780
World Wide Web address: http://www.motherisk.org
United States
Organization of Teratology Information Services
(801) 328-2229 (for referral to nearest service)
World Wide Web address: http://orpheus.ucsd.edu/ctis/
California Teratogen Information Service
(619) 543-2131
(800) 532-3749 (only in California)
District of Columbia Reproductive Toxicology Center
(202) 293-5137
Florida Teratogen Information Service
(352) 392-3050
(800) 392-3050 (only in Florida)
Illinois Teratogen Information Service
(312) 908-7441
(800) 252-4847 (only in Illinois)
Indiana Teratogen Information Service
(317) 274-1071
Massachusetts Teratogen Information Service
(781) 466-8474
(800) 322-5014 (only in Massachusetts)

Nebraska Teratogen Project
(402) 559-5071
New York Teratogen Information Service
(716) 874-4747 (ext. 477)
(800) 724-2454 (ext. 270) (only in New York)
Texas Teratogen Information Service
(800) 733-4727
Utah Pregnancy Riskline
(801) 328-2229
Vermont Pregnancy Risk Information
(802) 658-4310
(800) 531-9800 (only in Vermont)

Reproduced from Koren G, Pastuszak A, Ito S: Drugs in pregnancy. N Engl J Med 338:1128, 1998.

the World Wide Web addresses and telephone numbers of services in the United States and Canada. [41]

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Section XI - Neurologic Procedures

Chapter 61 - Management of Increased Intracranial Pressure and Intracranial Shunts

Khosrow Tabbassi
Cecile G. Silvestre

Headache and head injuries are diagnoses commonly encountered in emergency departments (EDs). If either headache or head injury are accompanied by vomiting, decreased level of consciousness, and abnormal vital signs, the possibility of increased intracranial pressure (ICP) must be considered. Accompanying clinical symptoms can often be vague or subtle, making the diagnosis of possible central nervous system (CNS) pathology difficult. Therefore, the clinician must rely on the physical examination, diagnostic studies, and a high index of suspicion to diagnose increased ICP. Increased ICP is a neurological emergency that must be managed quickly before further brain damage ensues. Therefore, it is important to understand the pathophysiology of ICP. Although many emergency clinicians have knowledge of, and expertis in, the management of intracranial shunts, the shunt-related techniques described here should not be considered standard of care.

PATHOPHYSIOLOGY OF INTRACRANIAL PRESSURE

Alexander Monro, an anatomist in the 18th century, defined the intracranial contents as a fixed volume. The fixed-volume theory was supported by George Kellie a few years later and became known as the Monro-Kellie doctrine. This doctrine has guided over understanding of intracranial dynamics and the principles of autoregulation.

The components of the calvaria are the brain, cerebrospinal fluid (CSF), the venous blood supply, and the arterial blood supply (Fig. 61-1). The CSF and the venous blood supply have the greatest ability to change their volume to compensate for increases due to other reasons.

Figure 61-1 Intracranial contents and their volumes for healthy adults.

These dynamic changes in relative cranial content proportions may not impact the patient if the ICP is not excessive. If a pathologic process overwhelms the compensating mechanisms, the result will be an increase in ICP. Unfortunately, ICP increases nearly exponentially as the brain volume increases, despite compensating mechanisms (Fig. 61-2).

Normal supine ICP usually ranges from 5 to 15 mm Hg relative to foramen of Monro. Transient increases of ICP as high as 80 to 100 mm Hg can be noted with coughing or straining. Other factors that can transiently increase ICP are movement, pain, and fever. A space-occupying lesion such as a tumor, hematoma, pus, or foreign body can also raise ICP. Figure 61-3 demonstrates that the area and the etiology of increased ICP will determine where the brain shift occurs to produce herniation.

Brain.

The brain volume can be increased by edema, benign intracranial hypertension, tumor, or bleeding. There are two types of edema, vasogenic and cytotoxic. Vasogenic edema is due to increased permeability of the capillaries leading to the passage of excess fluid into the extracellular space. Cytotoxic edema is due to brain tissue (neurons and glia) accumulation of intracellular fluid due to dysfunction of the adenosinetriphosphatase (ATPase) pump.

Benign intracranial hypertension (BIH) is also known as pseudotumor cerebri. It is increased CSF pressure that is not due to a tumor, edema, or hydrocephalus. Its exact pathophysiology remains a mystery. Common conditions associated with it are listed in Table 61-1 (Table Not Available) . This is a diagnosis of exclusion. It is usually self-limited without alteration of level of consciousness or severe brain dysfunction. Pseudotumor cerebri can present with clinical symptoms similar to conditions causing increased ICP. In general, patients with BIH have a normal neurological examination except for the occasional papilledema. Severe cases can lead to permanent vision loss (see Chapter 62).

Brain tumors encompass neoplasms that originate in the brain itself (primary brain tumors) or involve the brain as a metastatic site (secondary brain tumors).

Primary brain tumors include tumors of the brain parenchyma, meninges, cranial nerves, and other intracranial structures (the pituitary and pineal glands). Primary CNS lymphoma refers to non-Hodgkin's lymphoma confined to the CNS. The site of origin of this type of tumor remains unknown.

Secondary brain tumors, which originate elsewhere in the body and metastasize to the intracranial compartment, are the most common types of brain tumors.

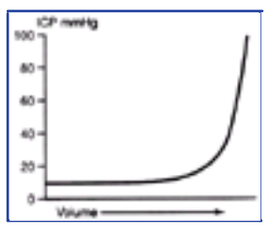

Figure 61-2 Intracranial volume-pressure relationship, demonstrating limits of compensatory mechanisms.

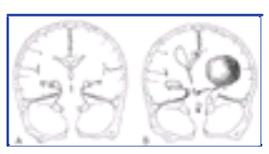

Figure 61-3 Intracranial shifts from supratentorial lesions. *A,* Relationships of the various supratentorial compartments as seen in a coronal section. *B,* Herniation of the cingulate gyrus under the falx (*1*); herniation of the temporal lobe into the tentorial notch (*2*); compression of the opposite cerebral peduncle against the unyielding tentorium, producing Kernohan's notch (*3*); and downward displacement of the brainstem through the tentorial notch (*4*). *(From Plum F, Posner JB: The Diagnosis of Stupor and Coma, 2nd ed. Philadelphia, FA Davis, 1972. Reproduced by permission.)*

Bleeding in the brain can occur spontaneously (as in the case of subarachnoid hemorrhage [SAH]) or can be a result of trauma. Spontaneous SAH is often the result of a ruptured berry aneurysm. Traumatic injury to the brain can often result in increased ICP by causing either epidural hematomas, subdural hematomas, or intraparenchymal hemorrhage. Diffuse

TABLE 61-1 -- Clinical Conditions and Factors Associated with Pseudotumor Cerebri

(Not Available)

Reprinted from McGraw-Hill textbook of Neurosurgery, vol. 1, 2nd ed. by Wilkins & Rengachary.

axonal injury (DAI) may occur in isolation or conjuction with intracerebral bleeding. With DAI, it is believed that the axons are not actually torn, but instead suffer significant injury (shearing effect) that may lead to swelling (shearing effect). [1]

CSF.

The CSF is produced by the choroid plexus at a daily rate of 500 mL. It flows freely from the lateral ventricles into the cisterns and the subarachnoid space and is drained by the arachnoid villi of the dural sinuses to maintain a volume of 100 to 150 mL. Blockage of this flow at any point can cause hydrocephalus proximal to the obstruction. The two types of hydrocephalus are obstructive and communicating. *Obstructive hydrocephalus* occurs when the blockage occurs at the ventricular system by a tumor, colloid cyst, or primary stenosis. Communicating hydrocephalus is due to blockage outside of the ventricular system by either impedance of flow through the basal cisterns or lack of absorption by the arachnoid villi. Communicating hydrocephalus is often due to infection or subarachnoid hemorrhage (Fig. 61-4).

Blood.

Up to a certain range, cerebral blood flow (CBF) is maintained by an autoregulatory mechanism despite fluctuations in cerebral perfusion pressure (CPP) (Fig. 61-5). This autoregulatory zone is CPP of 60 to 160 mm Hg.

Once the CPP is out of the autoregulatory zone, CBF is linearly related to CPP. CPP less than 60 mm Hg can lead to ischemia whereas that above 160 mm Hg can cause hypertensive encephalopathy.

CLINICAL PRESENTATION

The neurological examination can be normal in someone with a mild increase in ICP due to the brain's compensatory mechanisms. Patients who present with a complaint of headache or

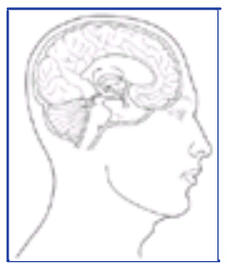

Figure 61-4 Cerebrospinal fluid production and flow *(From Rengachary and Wilkins: Principles of Neurosurgery. Philadelphia, Mosby, 1994).*

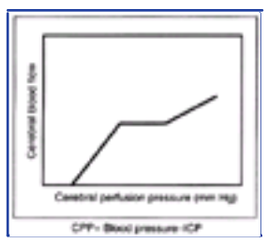

Figure 61-5 Cerebral autoregulation.

head injury may not initially manifest the more dramatic and worrisome symptoms of increased ICP such as vomiting, syncope, altered mentation, or Cushing's triad. (This triad consists of bradycardia, increased blood pressure with wide pulse pressure, and irregular respirations.) ICP correlates poorly with the degree of clinical examination depending on how gradual the increase in ICP is. Early clinical signs are decreased venous pulsations in the funduscopic examination, but this can be difficult to detect in the uncooperative individual. Initially, head computed tomography (CT) scan findings may not correlate well with the patient's neurologic insult. However,

Figure 61-6 (Figure Not Available) The basic tripartite ventricular shunt system is composed of ventricular catheter, valve mechanism, and distal tubing. A slit valve may be used in the far end of the distal tubing instead of a more proximally placed valve as shown in the figure. *(Adapted from Wilkins RH, Rengachary SS: Neurosurgery, 2nd ed. New York, McGraw-Hill, Health Professions Division, 1996, p 3648.)*

as compensatory mechanisms fail, head CT findings as well as clinical symptoms will become more obvious.

Signs and symptoms of severe ICP increases include the following: Glasgow coma scale (GCS) score of 8 or less, decreased level of consciousness, papilledema, cranial nerve findings, or CT scans showing compression of the third ventricle or midline shift. When any of these findings are noted, urgent intervention is warranted, including neurosurgical consultation, because invasive ICP reductionand monitoring may be warranted. The risk of such invasive intervention should be weighed carefully and should be performed by a neurosurgeon. There are less invasive interventions, which are listed as follows.

MEDICAL TREATMENT OF INCREASED ICP

Medical management of increased ICP often can be a useful adjunct pending definitive neurosurgical intervention.

Oxygenation.

In any trauma or medical patient, managing the airway deserves primary attention. If the patient is hypoxic, supportive oxygenation is necessary to prevent further ischemia. Once the patient's GCS is =9 or if any impending sign of inadequate respiratory status is observed, rapid sequence intubation should be considered to protect the airway, control the P_{CO_2} level, and optimize the P_{O_2} level based on the entire clinical scenario.

Sedation and paralytics.

(See also Chapter 5) Care must be given to the sequence and types of medications administered to avoid further increases in ICP while facilitating rapid intubation. Also, rapid assessment of the patient's neurological examination must be done prior to giving sedation and paralytics in order to determine any changes in the patient's status. If time allows, the patient should be premedicated 3 minutes prior to intubation with lidocaine at 1 to 1.5 mg/kg while preoxygenating the patient. Lidocaine has been reported to blunt the rise in ICP associated with laryngoscopy, and may protect against some hypoxia-related dysrhythmias. Atropine (0.02 mg/kg) should also be included in this premedication if the patient is younger than 10 years old since succinylcholine can be associated with bradycardia. This regimen should be followed by sedation with drugs that block the rise in ICP, such as etomidate (at 0.3 mg/kg) or thiopental (50 to 100 mg intravenously [IV] or 3 to 5 mg/kg). Etomidate is preferred over thiopental if the patient has an unstable hemodynamic status. It has two significant benefits—it has a minimal effect on systemic blood pressure and does not appear to increase ICP.[2] [3] A paralytic of choice is succinylcholine at 1.5 mg/kg (2 mg/kg in pediatric patients). Sedation and paralytics should be short-acting in order to follow the patient's neurological examination. Fentanyl (3 µg/kg) is an excellent drug for pain control, which, if untreated, can lead to increased ICP. This should be given during the pretreatment phase if time allows.

Hyperventilation.

Once the patient's airway has been secured, he or she should be maintained on adequate oxygenation, starting at 100%, and titrating to lower levels after transport to ICU settings.[1] In the past, aggressive hyperventilation was widely used to maintain $Paco_2$ at 25 mm Hg in order to reduce ICP. However, aggressive hyperventilation is no longer recommended because P_{CO_2} levels of 25 mm Hg can cause cerebral vasoconstriction resulting in decreased CBF. [1] The most recent recommendations state that in the short term,

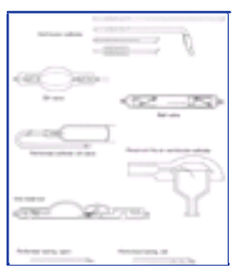

Figure 61-7 Shunt components *(From Rengachary and Wilkins: Principles of Neurosurgery. Philadelphia, Mosby, 1994).*

the target goal for ventilation is now 35 to 40 mm Hg.[4] The long-term recommendation is to avoid hyperventilation because it may cause prolonged vasoconstriction leading to ischemia and worse long-term outcome.[5] The role of hyperventilation in the rapidly deteriorating patient with elevated ICP remains controversial.

Head position.

If shock is not present, there is literature to support head elevation to about 30° in order to decrease ICP. According to Feldman and Kanter, elevation of the head to 30° significantly reduces ICP in a majority of patients without impairing CBF, CPP, and cerebral metabolic rate of oxygenation. [6] This position allows drainage of cerebral veins. Raising the head in a patient with hypotension would further decrease the patient's mean arterial pressure (MAP) and hence lower CPP. If head elevation is to be used, MAP must be maintained above 90 mm Hg.[7] It is also important to avoid neck rotation, flexion, or any intervention that could result in jugular compression. If jugular veins are compressed, venous outflow from the head can be further compromised.

Fluid management.

The goal in fluid management is to maintain euvolemia. Patients with increased ICP are often dehydrated due to profuse vomiting or are in shock due to loss of vasomotor sympathetic tone. The traditional practice of fluid restriction for patients with increased ICP is no longer in favor. Head-injured patients with hypotension have twice the mortality risk. Cardiac pressors, in addition to fluids, are often used to maintain CPP >70 mm Hg and MAP >90 mm Hg.

Diuresis.

Mannitol has been shown to be effective in reducing ICP. If increased ICP causes further deterioration in the patient's status, mannitol can be given at 0.5 to 1.0 g/kg infused every 2 to 6 hours. Current recommendations suggest bolus therapy over continuous infusion. This is a bridging effort while awaiting definitive therapy such as craniotomy.

Mannitol has two properties—it initially acts as a volume expander and then serves as an osmotic agent. Upon administration of mannitol, intravascular volume expands and blood viscosity decreases, resulting in augmentation of CBF. This is the reason the bolus should be administered as rapidly as possible. Once volume expansion has occurred, an osmotic movement of fluid from the cellular compartment to the intravascular compartment begins resulting in a decrease in ICP. The osmotic effect usually occurs within 15 minutes, resulting in decreased ICP. The half-life of mannitol ranges from 90 minutes to 6 hours. Patients who benefit most

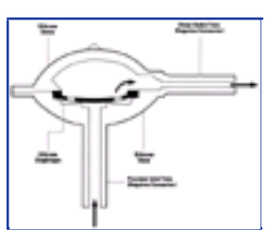

Figure 61-8 Cross section of a Pudenz flushing valve (American Heyer-Schulte, Santa Barbara, CA) illustrating the diaphragm valve. The proximal inlet tube and silicone base are placed in the burr hole so that only the reservoir (silicon dome) protrudes above the skull *(Courtesy of PS Medical, Goleta, CA).*

from the effects of mannitol are those with intact cerebral autoregulation. [8] If the patient's serum osmolality is above 320 mOsm/L, little benefit is obtained with the use of mannitol. Careful monitoring is necessary to maintain the osmolarity between 310 to 320 mOsm/L. [7] Higher levels of osmolality can lead to renal damage. In addition to serum osmolality, electrolytes, pH, and pulmonary status must be monitored closely to avoid the complications associated with these drugs. Hypertonic saline has been suggested as an alternative to mannitol. [9] Mannitol should not be used prophylactically, but instead, reserved for those patients with impending transtentorial herniation.

Seizure prophylaxis.

Controlling seizure activity is important to avoid elevation of ICP and possible herniation. Rapid-acting benzodiazepines such as lorazepam and diazepam are first-line therapy, followed by antiepileptic agents such as pentobarbital or phenytoin. Fosphenytoin has the advantage of rapid administration compared to phenytoin, which can only be given at a maximum rate of 50 mg/minute. Phenytoin can induce hypotension and lower CPP, even at rates <50 mg/minute. Some physicians give patients with severe head injury prophylactic phenytoin or fosphenytoin. The value of this practice for reducing seizure risk is unknown.

Patients who are paralyzed, either chemically or by their neurological disease, are hard to monitor for seizure unless electroencephalogram (EEG) monitoring is available. These patients manifest seizures by elevation in their heart rate or blood pressure or spikes in their ICP monitor. If seizure control is not obtained with benzodiazepines or phenytoin, or both, paralysis and the inducement of a pentobarbital coma should follow. Pentobarbital has been used to treat uncontrolled increased ICP if medical and surgical management have failed. It decreases cerebral blood flow, metabolism, oxygen consumption, and cerebral edema, and it also scavenges free radicals.[10] Pentobarbital is given at a loading dose of 10 mg/kg over 30 minutes followed by infusion of 3 mg/kg per hour with an EEG monitor available for burst suppression. Intensive monitoring of the patient's hemodynamic status is necessary due to pentobarbital's hypotensive effect. Similarly intubation and controlled ventilation is needed for patients receiving this much barbiturate.

Steroids.

Steroids have been shown to be beneficial in patients with vasogenic edema—edema that is associated with brain tumors. Its benefit has been noted to decrease CSF production, stabilize membranes, and restore normal membrane permeability. [11] [12] [13] Dexamethasone is usually given at 10 to 20 mg IV loading dose followed by 4 to 10 mg every 6 hours. No studies support the use of steroids in head-injured patients.

Glucose control.

Head-injured patients tend to be hyperglycemic as a response to stress or steroid administration. Optimization of blood glucose levels is important in order to avoid cellular edema of brain tissue. Hypoglycemic states should be avoided.

Hypothermia.

Lower body temperatures have been associated with a decrease in blood flow, ICP, and metabolism. The prior method of keeping core temperatures to less than 30°C has been abandoned. It fell out of favor due to complications of cardiac arrhythmias and difficulty maintaining this level. One study [14] has shown a statistically significant benefit in terms of survival and decreased ICP with mild

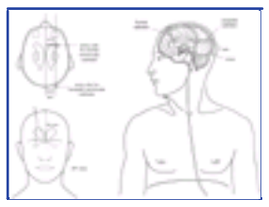

Figure 61-9 Ventriculoperitoneal shunt and alternative occipital placement *(From Rengachary and Wilkins: Principles of Neurosurgery. Philadelphia, Mosby, 1994, Fig. 7-6).*

hypothermia (32° to 34°C). It is important to avoid hyperthermia. A 1° rise in body temperature results in several mm Hg of rise in ICP. Acetaminophen is effective in fever control.

Operative management.

Operative management is the definitive treatment if ICP cannot be adequately managed by the previously discussed measures. This entails craniotomy to relieve the compressive edema, hematoma or tumor, or ventriculostomy with drainage of the increased blood or CSF fluid.

INTRACRANIAL SHUNTS

Intracranial shunts are used for the long-term management of increased ICP. A recurrent elevation in ICP due to shunt malfunction can occur days to years following shunt placement.

Intracranial shunt malfunctions are often diagnosed in the ED. In many cases, it is the patient's significant other or caretaker who provide the subtle clues to diagnose a shunt malfunction. Depending on the age of the patient, recurrent symptoms of increased ICP include changes in behavior, headache, nausea, vomiting, or visual disturbances. In children, lethargy, poor feeding, vomiting, ataxia, decreased or increased activity level, fever, diaphoresis, and fussiness can be indications of shunt malfunction. Failure to gaze upward is another sensitive sign of non-acute shunt malfunction (sunset eyes). Cranial nerves and visual field examination should be routine in the assessment of older, cooperative patients with intracranial shunts.

The essential elements of any shunt system include the proximal and distal catheters, a valve, and a reservoir. A wide array of valves, catheters, and other devices is available for use in shunt systems. Most of them are more or less satisfactory and the selection of a particular one is more subjective than scientific. The valve allows unidirectional flow, incorporates a pumping chamber, and regulates the pressure at which flow will occur by responding to the pressure difference across it. The reservoir is usually located between two valves. The proximal valve allows flow from the ventricles to the reservoir, while the distal valve allows flow from the reservoir to the distal portion of the catheter (Fig. 61-6 (Figure Not Available)). There are many different types of shunt systems incorporating a variety of designs (Fig. 61-7). Some have one valve or unique characteristics such as a double dome, or an absence of valves altogether.

In most cases, the reservoir gives successful access to the system for pressure measurement, patency testing, fluid sampling, and injection of medication or contrast material. In more rare cases, there is other equipment available that can be incorporated into the shunt system under special circumstances and for specific needs, including the on-off switch device, the telemetric pressure sensor, and the anti-siphon device.

In the basic shunt system, the ventricular catheter is usually placed in the right frontal horn of the lateral ventricle

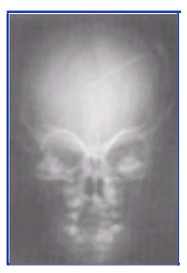

Figure 61-10 A standard shunt series includes an anteroposterior (AP) skull, lateral skull, and chest radiograph and kidney, ureters, bladder (KUB) series. This intact shunt is a Rickham reservoir over the burr hole, with a cylindrical Holter valve a few centimeters distal. This AP skull radiograph reveals the proximal intracranial portion of the shunt and a Rickham reservoir over the burr hole.

(nondominant brain) to connect to a subcutaneous valve (Fig. 61-8) traversing the temporal side. This valve is then connected to a distal catheter threaded subcutaneously into the neck and finally into the peritoneum or some other defined body compartment. Ventricular catheters can either be straight or angled, with the latter having the option of a reservoir component attachment. Valves usually come in four different types (ball, diaphragm, miter, slit), each with its unique flow characteristics. The distal catheter is either a closed or open

Figure 61-11 Lateral skull radiograph of patient in Figure 61-10 . This view better reveals a cylindrical Holter valve situated a few centimeters distal to the Rickham reservoir.

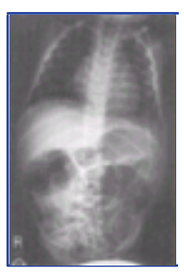

Figure 61-12 Anteroposterior chest and abdominal radiograph of patient in Figure 61-10 . This radiograph reveals the distal portion of the shunt. Note the length of the catheter to allow for patient growth.

Figure 61-13 Lateral chest and radiograph of the patient in Figure 61-10 .

end. Identifying the type of shunt in place is often difficult unless the patient has that information available. The skin overlying the subcutaneous part at the temples can often scar, thus disabling the palpation of the shunt type.

The principle of any extra-cranial shunting technique is to divert CSF into a body cavity from which it can readily be eliminated and drained. Ventricular peritoneal (VP) shunts are presently the mainstay of hydrocephalus treatment in infants and children because of their ease of insertion and reliable long-term function (Fig. 61-9). Other types of extra-cranial shunts include ventriculovenous (VV), ventriculoatrial (VA), ventriculopleural (VPl), and lumboperitoneal. These other shunts are usually reserved for circumstances in which VP shunting has failed because of complications or if there is a history of multiple abdominal surgeries or peritoneal infections.

Shunt Assessment

The usual cause of shunt malfunction is catheter obstruction. Proximal blockage may be due to tissue debris or choroid plexus within the ventricular catheter. Distal obstruction of venous shunts may result from thrombus or venous occlusion (such as in the ventriculoatrial shunt). Peritoneal shunts may be associated with infection (peritonitis) or mechanical obstruction (e.g., omentum blockade). There is also evidence to suggest that delayed hypersensitivity to shunt material is an occasional cause of obstruction. Symptoms of shunt malfunction may be difficult to interpret, particularly if the symptoms are atypical or nonspecific or if the symptoms occur in young children. Likewise, asymptomatic shunt obstruction can take place in children who have developed shunt independence. The clinical evaluation of a child may not always be diagnostic of a shunt malfunction.

The characteristics of valve pumping may be useful in shunt malfunction diagnosis in only about 5% of cases. [15] Partial shunt valve compression should be done since full depression and release may lead to choroid plexus suction. If the CT scan shows narrow (slit) ventricles, indicating overdrainage, further valve compression may cause blockage of the shunt by suction of the choroid plexus. When palpating the shunt, a normal refill time of 15 to 30 seconds should be seen. If the valve fills slowly but can be compressed easily, the obstruction is proximal to the valve. If the valve is not compressible, blockage is either at the valve or distal to it. Proximal obstruction is more common than distal. Other sources of proximal blockage are blood clots or debris related to the surgical procedure. Sources of distal occlusion include malposition, infection, shunt disconnection, and pseudocyst formation. The entire shunt tract and surgical incisions should be examined for signs of wound infection, disruption of the tubing, or CSF leakage around the tract. Definitive treatment usually requires shunt revision.

Infection is often due to skin organisms overlying the valve that may ulcerate and colonize the shunt. [16] The typical organisms are *Staphylococcus Epidermidis* or *Aureus*. Approximately 70% of these infections are seen within 2 months of shunt placement. Other risk factors associated with shunt infection include perioperative infection or any subsequent dental or urological instrumentation. The VA shunt has been largely avoided because of chronic bacteremia associated with it. Two studies have shown that approximately 8% of neurosurgical patients with implanted shunts acquire infections. [17] [18] Treatment is often replacement of the system.

CT scan is likewise an unreliable method of assessing shunt function when there is a question of obstruction, although obliteration of the perimesencephalic cisterns may be a sign of life-threatening shunt failure. The finding of large ventricles on CT reveals little about shunt function *unless serial scans show progressive ventricular expansion*. Shunt devices should include a tapping reservoir proximal to the distal valve to allow percutaneous testing of the proximal shunt patency. Although this is useful in most instances, it can be difficult to assess the rate of inflow or runoff adequately with a small bore needle, particularly if the patient is a child or uncooperative. The potential for infection is a disadvantage of percutaneous tapping. Other invasive techniques include injection of either radionuclide or contrast material into the shunt as a marker of flow. If ventricular fluid pressure is low, there may be little evidence of flow, giving a false indication of shunt malfunction. Various non-invasive techniques have been devised for assessing shunt function. Visual evoked potential (VEP) changes associated with elevated intracranial pressure have been suggested as a means of determining function. Thermographic and Doppler detection of shunt flow is also possible. Using MRI technology, it is possible to assess CSF flow and shunt patency.

Assessing shunt malfunction can be done most efficiently by obtaining radiologic shunt series, which consist of anteroposterior (AP) and lateral skull, AP chest, and AP abdominal x-rays (Fig. 61-10 Fig. 61-11 Fig. 61-12 Fig. 61-13 Fig. 61-14 Fig. 61-15 Fig. 61-16 Fig. 61-17 Fig. 61-18). This may reveal kinkingor brealage of the catheter or its disconnection from the shunt. If the x-ray is nondiagnostic, head CT scan without contrast should follow. As stated earlier, prior CT scans should be obtained for comparison of the ventricular size and for the placement of the catheter (Fig. 61-19 , Fig. 61-20). If distal catheter obstruction is suspected, abdominal ultrasound can be used to determine abnormal fluid collection. Finally, CSF analysis is important whenever an assessment of infection or shunt function is undertaken.

Shunt Tapping

The scalp hairs should be clipped over and around the reservoir, then prepared with a surgical scrub brush for 10 minutes, followed by povidone-iodine solution which is allowed to fully dry. After appropriate draping, the skin is infiltrated using 1% plain lidocaine to a level of adequate local anesthesia. A 25-ga butterfly needle with tubing is used to enter the reservoir percutaneously at a 20° to 30° angle. Lack of CSF flow from the reservoir would indicate a proximal obstruction unless the ventricles are completely deflated (slit ventricles syndrome). Even so, a small amount of CSF should be obtained within the tubing, thus assuring that entry into the lumen of the reservoir has been accomplished. If the ventricles are deflated and only a small amount of CSF is aspirated, the end of the tubing can be held 5 to 10 cm below the level of the reservoir to see if CSF will fill the tubing and eventually begin to drip at the rate of 2 to 3 drops per minute. If the CSF readily aspirates from the reservoir, the tubing is held vertically to give an indication of the intraventricular pressure (Fig. 61-21). This pressure reading, as well as the ease with which CSF is aspirated, will give some indication of proximal obstruction. To assess the runoff or distal end patency, one

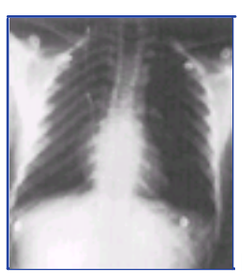

Figure 61-14 Connector discontinuity of a ventricular peritoneal shunt, at the level of the clavicle, is illustrated in this chest radiograph from a shunt series.

should apply pressure to the tubing proximal to the reservoir, and then deflate the reservoir without any resistance. If any resistance is encountered when deflating the reservoir, a distal obstruction should be suspected.

It is imperative to send any aspirated CSF for the relevant studies including cells, differentials, protein, and most all gram stains and cultures in evaluation of possible infection (see Chapter 62).

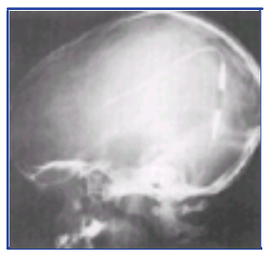

Figure 61-15 Normal lateral skull radiograph demonstrating an intact ventricular peritoneal shunt in a young patient with a Holter valve. This shunt later became disconnected (see Fig. 61-17 and Fig. 61-18).

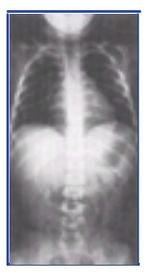

Figure 61-16 Normal anteroposterior chest and abdominal radiograph demonstrating an intact ventricular peritoneal shunt in the patient in Figure 61-15 .

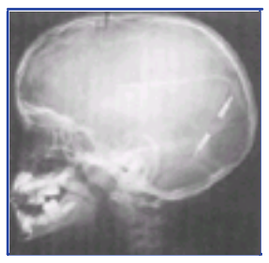

Figure 61-17 Repeat films from the patient in Figure 61-15 and Figure 61-16 illustrating shunt disconnection at the level of the valve and subsequent migration of the distal catheter into the pelvis. This lateral skull radiograph demonstrates the disconnection from the valve.

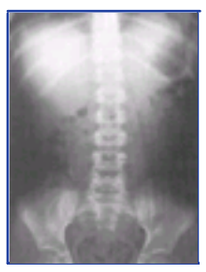

Figure 61-18 Abdominal radiograph of patient in Figure 61-17 demonstrating migration of the catheter into the pelvis.

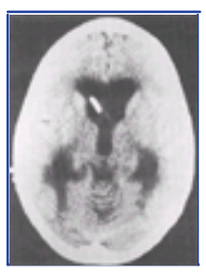

Figure 61-19 Cranial computed tomography scan of the patient with shunt disconnection shown in Figure 61-17 . This CT scan at presentation reveals ventriculomegaly. After revision, the ventricles returned to normal size (see Fig. 61-20).

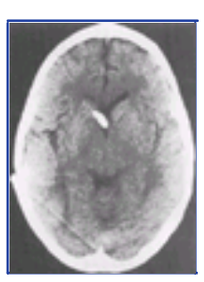

Figure 61-20 Cranial computed tomography scan of patient in Figure 61-19 after shunt revision. The ventricles have returned to normal size.

Special Consideration—Postoperative Complications of Shunts

Major complications after shunting for high-pressure hydrocephalus are infrequent. The most common are shunt malfunction, seizures, and infection.

Hemorrhage: Bleeding can also occur after the placement of the shunt. Both intracerebral and subdural hemorrhages have been noted. Intracerebral hemorrhage is due to trauma to the brain parenchyma as the catheter is passed through the ventricles. Subdural hematoma is due to sudden decompression of the ventricles that leads to tearing of the bridging veins as the brain pulls away from the dura. An acute or chronic subdural bleed is also encountered in shunts with overdrainage problems.

Shunt malfunction: If shunt malfunction is suspected, the first step is to perform head CT scan and shunt series imaging to visualize ventricular size and anatomical alignment of the tubes and devices. The ventricles usually begin to diminish in size within a week post shunt placement in high-pressure hydrocephalus, so continued enlargement suggests shunt malfunction. The shunt series images confirm the continuity of a drainage system from the insertion site through the proximal catheter, reservoir, distal catheter, to the receiving draining cavity, mostly and commonly peritoneum. It is important to know that in normal pressure hydrocephalus, the ventricles may remain large despite good functional shunting. The timing and indications of shunt placement are imperative to know when assessing the urgency of the management, especially when consulting a neurosurgeon.

Seizures: These are an infrequent complication of the shunt placement and occur in about 5% of patients.

Figure 61-21 A 25-ga butterfly needle puncture of a reservoir. To avoid damage to the reservoir, the angle should be approximately 20° to 30°. Note that the dome reservoir is under the skin. Prior to passage of the butterfly needle, the skin is anesthetized, sterilized with povidone-iodine, and nicked with a number 11 scalpal or larger needle. Fluid is not aspirated but is allowed to drain passively.

Technically occipital catheter placement may lessen the incidence of seizures because their location is far from the motor cortex. In patients thought to be at risk for a seizure, antiepileptic medication may be indicated.

Shunt infection—treatment and prevention: Of all the potential complications associated with shunting procedures, infection is the most notorious, occurring in 2% to 10% of the operated cases.[15] Most shunt infections appear within the first 2 months after surgery. The diagnosis may be obvious with variable systematic signs, wound infection, meningitis, peritonitis, or septicemia. It is also possible the shunt may harbor an indolent infection without symptoms or signs. CSF culture obtained from a shunt tap may be negative, even when the shunt is ultimately shown to be infected. [19] [20]

Most shunt infections are caused by otherwise nonvirulent bacteria such as *staphylococcus epidermidis*. In the presence of the shunt, these organisms exhibit unusual virulence. This reflects a variety of factors, including the ability to adhere to shunt surfaces, as well as production of mucoid substances that protect the bacteria from host defenses. The silastic shunt material itself has an adverse effect on the immune system. Specifically, leukocytes cannot adhere to such surfaces as well as bacteria. This situation allows phagocyted bacteria to remain viable within the cell. Controversy exists over the need for shunt removal in the context of treatment of infection. Although the systemic antibiotics alone are of unpredictable benefit, the addition of intraventricular drugs has allowed more success in treating without removal of a functioning shunt. Having said this, many neurosurgeons still recommend ultimately replacing the entire shunt after the infection is eradicated. The most important single factor in preventing shunt infection is obviously aseptic operative and postoperative shunt handling for diagnostic procedures technique. The role of prophylactic preoperative antibiotic treatment remains equivocal. Consultation with neurosurgeons and infectious disease specialists is recommended when deciding to administer antibiotics directly into the CSF either via the shunt or a lumbar puncture. Among the antibiotic options, oxacillin, nafcillin, and vancomycin are good empiric choices. If a shunt infection is suspected, systemic antibiotics may be given, and as with any serious infectious process, the earlier the administration, the better for the patient.

CONCLUSION

ICP is difficult to assess without obvious clinical and radiological findings. Because of compensatory mechanisms, the degree of increased ICP does not correlate with the degree of clinical and radiological abnormalities. It is therefore important to recognize by history, examination, and other ancillary tests how to detect early neurologic findings that may indicate elevations of ICP before further damage occurs. Understanding intracranial shunts and their complications is also necessary to assess patients with possible shunt malfunction.

Acknowledgment

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Chapter 62 - Spinal Puncture and Cerebrospinal Fluid Examination

Brian Euerle

Cerebrospinal fluid (CSF) examination is performed in an emergency department (ED) for the purpose of obtaining information relevant to the diagnosis and treatment of specific disease entities. Many urgent and life-threatening conditions require immediate and accurate knowledge of the nature of the CSF. However, on rare occasion certain harmful consequences may result from a spinal puncture. The procedure should follow a careful neurologic examination with thought given to the risks and merits of the procedure in each given situation.

In 1885, Corning punctured the subarachnoid space to introduce cocaine anesthesia into a living patient. [21] Quincke (1891) first removed CSF in a diagnostic study and introduced the use of a stylet. [96] He studied cellular contents and measured protein and glucose levels. Quincke was also the first to record pressure with a manometer. Subsequently, increasingly sophisticated bacteriologic, biochemical, cytologic, and serologic techniques were introduced. In 1918, Dandy replaced CSF with air to determine normal brain anatomy and changes that would indicate disease. [22] Water-soluble contrast media have been used to delineate the spinal subarachnoid space and cerebral cisterns. Other uses of the spinal puncture include injection of anesthetic agents, chemotherapeutic agents, and antibiotics and drainage of fluids.

ANATOMY OF CSF FORMATION AND CIRCULATION

In the adult, CSF occupies approximately 140 mL of the spinal and cranial cavities, with approximately 30 mL in the spinal canal. This volume is the result of a balance between continuous secretion (primarily by the ventricular choroid plexus) and absorption into the venous system (mainly by way of the arachnoid villi). After formation, the fluid passes out of the ventricles by way of the midline dorsal foramen of Luschka and the lateral ventral foramina of Magendie. The fluid then flows into the spinal subarachnoid space, the basilar cisterns, and the cerebral subarachnoid space. Production is approximately 0.35 mL/min, and CSF ventricular production is such that there is a net flow out of the ventricles of 50 to 100 mL/day. Thus the usual volume of CSF removed at lumbar puncture is commonly regenerated in about 1 hour.

Cerebrospinal fluid may have an embryologic nutritive function; at maturity the CSF most likely acts as a mechanical barrier between the soft brain and the rigid fibro-osseous dura, skull, and vertebral column. It also appears to support the weight of the brain. [130] When buoyed by CSF, the functional brain weight is reduced from 1400 to 50 g. Contraction and expansion of the CSF may accommodate changes in brain volume. Additional functions, including intracerebral transport and maintenance of a stable chemical environment of the central nervous system (CNS), have been reviewed by Fishman. [39]

INDICATIONS FOR SPINAL PUNCTURE

General Indications

The indications for spinal puncture have been reduced with the introduction of noninvasive diagnostic procedures—magnetic resonance imaging (MRI) and computed tomography (CT). A few clinical situations require an early, or even an emergent, spinal puncture. The primary indication for an emergent spinal tap is the possibility of CNS infection, with the exception of suspected brain abscess or a parameningeal process. The need for early detection of meningitis results in performance of many more lumbar punctures than ultimate diagnoses of infection.[4] No other method can be used to completely exclude the diagnosis of meningitis.

The mere presence of a fever does not mandate lumbar puncture. However, CSF generally should be examined for evidence of an infection in patients with a fever of unknown origin, especially if an alteration of consciousness or impaired immune system is present, even in the absence of meningeal irritation. *Meningeal signs may not be present in patients who are old, debilitated, immunosuppressed, or receiving anti-inflammatory drugs or who have had partial treatment with antibiotics.* [82] [103] In a newborn, even a fever is not a dependable sign; temperatures may be normal or even subnormal. For infants under age 1 year, a high index of suspicion is required to make the diagnosis of meningitis. Approximately 25% of infants with meningitis will not have nuchal rigidity, but most will usually appear toxic or moribund. A tense and bulging fontanel is somewhat more reliable, although this sign may be absent in a dehydrated child. Neonatal meningitis occurs in 25% of sepsis cases. In addition, 15% to 20% of infants with meningitis will have negative blood cultures. [8] [137]

In a child between the ages of 1 month and 3 years, fever, irritability, and vomiting are the most common symptoms of meningitis. Typically, handling is painful for the child, and the child cannot be comforted. In addition, the older child may complain of a headache. In all ages, the patient generally looks unusually ill and appears drowsy with a dulled sensorium.

Physical signs become more useful in diagnosing meningitis in children older than age 3 years. [139] These include nuchal rigidity, Kernig's sign (efforts to extend the knee are resisted), and Brudzinski's sign (passive flexion of one hip causes the other leg to rise, and efforts to flex the neck make the knees come up). A useful aid in distinguishing neck rigidity of meningeal origin from that caused by primary pain in the cervical muscles and the soft tissues is the usual preservation of lateral movement in meningeal irritation. A petechial rash in a febrile child should also raise the possibility of *Neisseria* meningitis.[44] Prior use of antimicrobial agents may modify the clinical presentation and alter CSF findings; partially treated children are less likely to be febrile or exhibit an altered mental status. In addition, patients in the early stages of meningitis may lack the classic features associated with advanced disease.

The second indication for an emergent spinal puncture is a suspected spontaneous subarachnoid hemorrhage. The diagnosis will usually be made by head CT scan or by the

finding of blood in the CSF.[53] CT sensitivity drops in patients presenting later than 24 hours after the event to 76% after 48 hours. From 20% to 60% of aneurysmal subarachnoid hemorrhage will have "sentinel thunderclap" or "warning leak" headache before a major hemorrhage. This consists of an unusual sudden headache without nuchal rigidity caused by a "minor" leak of blood from the aneurysm and may precede major rupture by 10 to 320 days. [60] [72] Surgical treatment before a major hemorrhage is the goal of early clinical recognition. After a "warning leak," the head CT scan is usually negative, giving added importance to the performance of a lumbar puncture. Migraine or "vascular" headache is a common misdiagnosis in patients with a subarachnoid hemorrhage who have an initially negative head CT scan.

The usual clinical picture of a subarachnoid hemorrhage is a severe and instantaneous excruciating headache. Patients will usually recall the exact moment the headache occurred. The location of the headache is variable and does not give a clue as to the site of hemorrhage. Nausea, vomiting, and prostration are common symptoms, with approximately one third of patients becoming unconscious at the onset. Examination shows an acutely ill patient with irritability or overt altered mental status. Meningeal signs are commonly present at the time of the initial examination and usually develop in all cases within 2 to 3 days. Meningeal signs may become more severe during the first week after hemorrhage and correspond to the breakdown of blood in the CSF. During the first week, many patients are febrile, reflecting a chemical hemic meningitis.[124] Failure to detect blood radiographically in an awake patient may indicate a small hemorrhage or a predominant basal accumulation of blood. If a patient is seen several days after the hemorrhage, the blood may have become isodense with brain and may no longer be visible on a CT scan. The proper diagnosis would then require spinal puncture. Because 2 to 10% of acute subarachnoid hemorrhages will not be detected by the initial CT scan (even with modern third generation scanners), a lumbar puncture is appropriate to rule out the diagnosis with certainty. [3] [106] [119] [131] In theory, a small subarachnoid hemorrhage may take several hours to reach the lumbar region. Thus, it is possible that such a patient might have normal lumbar CSF if examined soon after rupture. A second, delayed lumbar puncture may occasionally be required for diagnosis in such a situation. If the neurologic picture demonstrates localizing findings, the presence of a large intracranial hematoma should be suspected, and spinal puncture is contraindicated until CT (or arteriography) delineates the nature of the lesion.

The emergency clinician also may be called on to perform a therapeutic lumbar puncture in the patient with known pseudotumor cerebri (also known as benign intracranial hypertension or idiopathic intracranial hypertension). The patient has elevated intracranial pressure (ICP) for unknown reasons and often has headaches and papilledema. The headache worsens with maneuvers that increase the ICP (e.g., Valsalva, squatting, bending, coughing). See the following section for details.

Other nonemergent reasons for CSF examination include evaluation of CNS syphilis, unexplained seizures, instillation of chemotherapy and positive contrast agents, evaluation of suspected demyelinating or inflammatory CNS process, and treatment of headache from subarachnoid hemorrhage or benign intracranial hypertension. [26] Carcinomatous meningitis and suspected spinal cord compression from metastatic disease may require spinal puncture for myelography and cytologic examination. The availability of MRI is a suitable alternative for identifying compressive myelopathies.

Pseudotumor Cerebri

Pseudotumor cerebri, more commonly known as idiopathic intracranial hypertension (IIH) is a rare condition that may present to the ED. [43] IIH is an elevation of intracranial pressure (usually to 250–450 cm H_2O) without hydrocephalus or mass lesions, in the setting of *normal CSF* composition. This is a diagnosis of exclusion. Most patients have headache, papilledema, and minimal or absent focal findings, with a normal CT or MRI scan. Most patients appear otherwise well, but some complain of tinnitus, nausea, or blurred vision. IIH has been related to hypervitaminosis A, tetracycline, estrogen therapy, and a plethora of other conditions, such as sarcoidosis, tuberculosis and carcinomatosis (see Table 61-1 (Table Not Available)). Most cases are idiopathic. IIH is most common in obese adolescent girls and young women, but it can occur in childhood and in men. The mechanism of increased CSF pressure is unclear, but the brain appears normal. One proposed mechanism is decreased CSF outflow.

IIH may regress spontaneously after a few months. The major concern is visual loss, which may be permanent. The diagnosis can only be made by lumbar puncture, performed after neuroimaging. *This condition underscores the need to measure opening pressure during lumbar puncture whenever possible.* Many cases can be controlled with medication, but occasionally lumbar puncture is required to lower ICP, measured in the lateral decubitus position, to 200 mm H_2O. One way to lower ICP in the ED is to drain CSF via lumbar puncture by removing 5–10 mL aliquots of CSF and check ICP after each removal. Herniation does not occur despite elevated pressures. Since spinal fluid is regenerated rapidly, the procedure may need to be performed every few days to keep CSF pressure at this level. Analysis of the CSF is normal in all parameters. Drug therapy, in the form of acetazolamide, glycerol, diuretics, corticosteroids and others have been advocated and may negate the need for repeated lumbar puncture. Resistant cases may require shunting procedures.

CONTRAINDICATIONS FOR SPINAL PUNCTURE

Spinal puncture is absolutely contraindicated in the presence of infection in the tissues near the puncture site. [39] [130] Spinal puncture is relatively contraindicated in the presence of increased ICP from a space-occupying lesion. Caution is particularly advised when lateralizing signs (hemiparesis) or signs of uncal herniation (unilateral third nerve palsy with altered level of consciousness) are present. In such cases, a tentorial or cerebellar pressure cone may be precipitated or aggravated by the spinal puncture. Cardiorespiratory collapse, stupor, seizures, and sudden death may occur when pressure is reduced in the spinal canal. [26]

The risk of herniation seems to be particularly pronounced in patients with brain abscess. [13] [107] Brain abscesses frequently occur as expanding intracranial lesions with headache, mental disturbances, and focal neurologic signs rather than as infectious processes with signs of meningeal irritation. In 75% of cases, a primary source of chronic suppuration is present. Common predisposing causes of brain abscess include

craniofacial trauma, craniocerebral trauma, penetrating injuries with bone fragments in brain, transmitted foreign objects or large animal bites of infant skulls, postneurosurgical procedures, cardiovascular disorders with right to left shunts, bacterial endocarditis, gram-negative sepsis in neonates, dental infections, chronic sinusitis, otitis, mastoiditis, chronic abdominal pulmonary or pelvic infections, bacterial meningitis, and immunosuppression. Infarcted brain tissue may develop abscesses in the presence of sepsis because of a compromised blood-brain barrier. [114] Although the CSF is usually abnormal (elevated pressure, elevated white blood cell count, and elevated protein concentration), spinal puncture in patients with a known, or highly probable, abscess is contraindicated in most cases. In one study by Samson and Clark, 5 of 22 patients exhibited signs of midbrain compression within 2 hours of lumbar puncture. [107] Evidence of herniation markedly reduces the patient's chances for survival.

Brain abscess may spontaneously rupture into the ventricular system producing ventriculitis and meningitis. If the history suggests possible brain abscess, CT can rapidly diagnose and localize the lesion. [144] Because the appearance of brain abscesses on CT is similar to that of neoplastic and vascular lesions, false-positive reports of brain abscess may be encountered. [102]

Lumbar puncture can cause trauma to the dural or arachnoid vessels, which may result in minor hemorrhage into the CSF. This generally is of little consequence. However, the number of patients with hemophilia and human immunodeficiency virus (HIV) infection who require lumbar puncture has increased in the past decade. Spinal epidural hematomas may occur in some subpopulations of patients undergoing lumbar puncture. Individuals most at risk are those with some sort of bleeding diathesis, including those on anticoagulant therapy or those with abnormal clotting mechanisms, especially thrombocytopenia. Edelson and colleagues reviewed more than 100 cases of spinal epidural hematoma; approximately one third were associated with anticoagulant therapy. [29] Most articles describe isolated cases. [29] [71] [104] [112] Spinal subdural hematomas after lumbar puncture are even more rare than epidural hematomas. [25] [30]

When a patient is anticoagulated or has a coagulopathy, the tap should be performed by experienced clinicians, who are less likely to traumatize the dura. The patient should be carefully followed for progressive back pain, lower extremity motor and sensory deficits, and sphincter impairment after the procedure. *Complaints of motor weakness, sensory loss, or incontinence after lumbar puncture should be thoroughly investigated.* Lumbar puncture may be performed in the presence of a coagulation defect if the procedure is expected to provide essential information, such as in the diagnosis of meningitis. In cases of severe thrombocytopenia, the infusion of platelets before the lumbar puncture may be desirable.

The infusion of clotting factors in the hemophiliac patient and normalization of the prothrombin time with fresh frozen plasma in the anticoagulated patient are desirable if the clinical situation permits such delay before performing a lumbar puncture. Because as many as 90% of patients with severe hemophilia are seropositive for HIV, the issue of performing a lumbar puncture in patients with coagulopathies is an increasingly common phenomenon. Silverman and colleagues have demonstrated the safety of lumbar puncture in patients with hemophilia A or B who had their deficit clotting factor replaced before the procedure. In their series of 33 patients (30 with <1% normal factor level) who underwent a total of 52 spinal taps after specific factor replacement, no serious procedure-related complications were identified.[120] Their protocol was to attain an immediate postinfusion factor level between 5% and 100%. Use of additional factor replacement after lumbar puncture is of unknown value.

Howard and colleagues reported on 5223 lumbar punctures done in 958 children with newly diagnosed acute lymphoblastic leukemia. [56] Of these lumbar punctures, 29 were performed with platelet counts of 10×10^9 /L or less, 170 with platelet counts of $11–20 \times 10^9$ /L, and 742 with platelet counts of $21–50 \times 10^9$ /L. No serious complications were reported. The overall rate of traumatic taps was 10.5%, but these were not associated with adverse sequelae. They concluded that in children with acute lymphoblastic leukemia, prophylactic platelet transfusion for lumbar puncture is not required if the platelet count is greater than 10×10^9 /L. The number of patients with platelet counts of 10×10^9 /L or less was too small to reach any conclusion about this group of patients.

If the history and physical examination suggest a treatable illness, such as meningitis or subarachnoid hemorrhage, the clinician may perform a spinal puncture after careful consideration of the entire clinical picture. In all cases, the study should be undertaken after careful thought regarding how the results will assist in patient evaluation and treatment. It is unlikely that the spinal puncture will beneficially alter management in the presence of a neoplasm, a cranial hematoma, an abscess, a completed nonembolic infarction, or cranial trauma.

EQUIPMENT

The standard equipment for a spinal puncture should be assembled prior to beginning the procedure and placed where the operator can easily access it during the procedure. Standard equipment includes several spinal needles; three-way stopcock; manometer; and connecting tubing (optional, to connect needle with stopcock and manometer); at least four specimen tubes; local anesthetic; syringes and needles for local anesthesia (appropriate for both skin and subcutaneous tissue infiltration); sterile drapes, gauze, sponges, and gloves; and an antiseptic solution for skin preparation (e.g., 0.5% chlorhexidine in alcohol, 1% povidone-iodine). [42]

Some operators prefer to use an "atraumatic standard Sprotte needle" (Havel's, Inc, Cincinnati, OH) or Whitacre needle (Becton Dickinson and Co, Rutherford, NJ) to minimize the dural injury associated with needle passage. These styletted needles have a side port for fluid withdrawal and theoretically are more likely to *separate* rather than cut the dural tissue.

Although commercial kits provide most of the items (Fig. 62-1), it is important for the operator to bring additional supplies, including supplemental spinal needles and specimen tubes, gauze and antiseptic solution, additional local anesthesia and needles/syringes, and extra sterile gloves of the appropriate size. Following this advice will minimize procedural interruptions when material in the commercial kit must be supplemented.

TECHNIQUE

Lumbar puncture is commonly carried out with the patient in the lateral recumbent position. A line connecting the

Figure 62-1 Standard lumbar puncture tray. Separate equipment is available for infants, children, and adults. *(Image courtesy of the American Pharmaseal Company, Glendale, CA.)*

posterior-superior iliac crests will intersect the midline at approximately the L4 spinous process (Fig. 62-2 (Figure Not Available)). Spinal needles entering the subarachnoid space at this point are well below the termination of the spinal cord, and the only important neurologic structure is the cauda equina. Generally, the needle will push isolated nerves to the side during advancement. The adjacent interspace above or below may be used, depending on which area appears to be most open to palpation. The space between lumbar vertebrae is relatively wide. In the thoracic region, the spinous processes overlap and are directed caudad, and therefore there is no midline area free of overlying bone. In the adult, the spinal cord extends to the lower level of L1 or the body of L2 in 31% of persons, thus eliminating higher levels as sites for puncture. The puncture in adults and in older children may be performed from the L2 to L3 interspace to the L5 to S1 interspace.

Developmentally, the spinal canal and the spinal cord are of equal length in the fetus. Growth of the cord does not keep pace with longitudinal growth of the spinal canal. At birth, the cord ends at the level of the L3 vertebra. The needle in infants should be placed at the L4 to L5 or L5 to S1 interspaces. The subarachnoid space extends to an S2 vertebral level; however, the overlying bony mass prevents entry into this lowermost portion of the subarachnoid space.

When performed with parenteral sedation and proper local anesthesia, a spinal tap is neither overly distressing nor very painful to most patients. Almost all patients are afraid of a spinal puncture because they have heard stories of severe complications. Explaining the procedure in advance and discussing each step during the course of the test aids in reducing patient anxiety and helps the clinician. The clinician should inquire about history of allergies to local anesthetic agents and topical antiseptics. Use of a written informed consent is optional. However, a detailed procedural note is recommended to document the process of patient/guardian education regarding the indications, procedural techniques, risks/benefits and alternatives to the procedure, and the patient/guardian's assent to the procedure. This step may need to be abridged when the patient is critically ill or eliminated when the patient is mentally incapacitated and no guardian is present. In anxious patients, a benzodiazepine agent given parenterally (e.g., midazolam, 0.05 to 0.075 mg/kg IV) will facilitate the procedure and is recommended.

The next important step is positioning of the patient. Generally the procedure is performed in the lateral decubitus position in older children and adults. The patient is given a pillow to keep the head in the same plane as the vertebral axis. The shoulders and the hips are positioned perpendicular with the table. A firm table or bed is desirable whenever possible. Flexion of the neck does *not* facilitate the procedure to any great extent; and because severe flexion may add to the patient's discomfort, this step may be omitted. The patient's lower back should be arched toward the clinician by having the patient's knees drawn toward the chest.

Some clinicians place the patient in an upright sitting position, because the midline is more easily identified when the patient is sitting. The higher CSF hydrostatic pressure in a sitting, dehydrated patient may aid CSF flow. Caution regarding orthostatic blood pressure changes and airway maintenance must be observed when the patient is sitting for the procedure. Generally the sitting patient is allowed to lean onto a bedside stand using a pillow to rest the head and arms. An assistant also must help support the patient during the procedure. Radiographic studies by Fisher and colleagues [38] have shown the advantages of hip flexion when the sitting position is used. This can be accomplished by having seated patients pull their knees up to their chest, as is generally done when the lateral decubitus position is used. This will increase the

Figure 62-2 (Figure Not Available) When the patient has been correctly positioned for the lumbar tap *(left),* an imaginary line connecting the iliac crests will be exactly perpendicular to the bed. Insertion sites are marked by *x;* the operative field is draped. The needle should be inserted perpendicularly (or nearly so) to the plane of the back, with the forefingers of both hands guiding it in *(right)*. *(From Cole M: Pitfalls in cerebrospinal fluid examination. Hosp Pract 4:47, 1969. Illustration by Carol Donner. Reproduced by permission.)*

lumbar interspinous width, which may increase the success or ease of needle passage.

Although introgenic infection following lumbar puncture is extremely rare, sterile gloves are customary. The use of face masks by medical personnel who are performing a lumbar puncture is controversial. [5] [10] [142] Although definitive supporting data do not exist, some authors recommend use of a face mask while performing a lumbar puncture as an infection control practice. The examiner should wash the patient's back with an antiseptic solution applied in a circular motion. The circumference of the cleansed area should increase with each motion. A povidone-iodine solution will decrease skin flora to a bacterial count of 0 to 2 bacteria/in ^{2} .[59] Friction during skin preparation helps remove loose debris. The patient should be warned that the solution will be cold. The povidone-iodine must be allowed to dry completely. This can be hastened by removing excess fluid with a dry sterile gauze pad. A sterile towel or drape is placed between the patient's hip and the bed. Commercial trays have a second sterile drape with a hole that may be centered over the site selected for the tap.

The skin and deeper subcutaneous tissue are infiltrated with local anesthetic (1% lidocaine). Buffered or warmed lidocaine is preferred (see Chapter 30). The patient should be warned about transient discomfort from the anesthetic. Anesthetizing the deeper subcutaneous tissue significantly reduces the procedural discomfort. *Merely raising a skin wheal is insufficient anesthesia.* Some operators not only anesthetize the interspinous ligament but also apply local anesthesia in a vertically fanning distribution on both sides of the spinous processes near the lamina. This field block on each side of the spinous processes anesthetizes the recurrent spinal nerves that innervate the interspinous ligaments and muscles.

While waiting for the anesthetic to take effect, the clinician should attach the stopcock and manometer and see that the valve is working. Commonly, a 3.5-in, 20-ga needle is used in adults, and a 2.5-in, 22-ga needle is used in children (a 1.5-in, 22-ga needle is available for infants). A needle of this size has enough rigidity to allow the procedure to be accomplished easily but makes less of a dural tear than do larger needles. The patient should be told to report any pain and should be informed that he or she will feel some pressure.

The needle is placed into the skin in the midline parallel to the bed. The needle is held between both thumbs and index fingers. After the subcutaneous tissue has been penetrated, the needle is angled toward the umbilicus. The bevel of the needle should be facing straight up toward the ceiling, assuming that the patient is in the lateral decubitus position. It has been speculated that pointing the bevel parallel to the longitudinal axis of the spine may allow the needle to separate the fibers of the dura rather than cut through them, allowing for less CSF leakage after the needle has been withdrawn.

The supraspinal ligament connects the spinous process; the interspinal ligaments join the inferior and superior borders of adjacent spinous processes. The ligamentum flavum is a strong, elastic, yellow membrane that may reach a thickness of 1 cm in the lumbar region. The ligamentum flavum covers the interlaminar space between the vertebrae and functions to assist the paraspinous muscles in maintaining an upright posture (Fig. 62-3 (Figure Not Available)). The ligaments are stretched in a flexed position and are more easily crossed by the needle. The ligaments offer resistance to the needle, and a "pop" is often felt as they are penetrated.

One should remove the stylet frequently to see if the subarachnoid space has been reached. The pop is occasionally not felt with the very sharp needles contained in disposable trays.

If bone is encountered, the needle must be partially withdrawn to the subcutaneous tissue. The clinician should repalpate the back and ascertain that the needle is in the midline. Directing the needle tip toward the navel often enhances navigation of the interspinal space. If bone is again encountered, the needle should be slightly withdrawn and re-angled, with the point placed so that it angles more sharply cephalad. This should avoid hitting the inferior spinous process.

Clear fluid will flow from the needle when the subarachnoid space has been penetrated. In normal patients the dura will be penetrated when the needle is advanced about ½ to ¾ of its length. In obese patients the entire needle length may required to reach the subdural space (Fig. 62-4).

The clinician should attach the manometer and record the opening pressure. Although commonly omitted in critical patients, whenever feasible, this step should be preformed. In the struggling patient, pressure readings may be inaccurate. Pressure readings are only valid if taken with the patient in the lateral decubitus position and not the sitting position. A three-way stopcock is supplied in disposable trays; this allows both collection and pressure to be measured by a single needle (Fig. 62-5 (Figure Not Available)). Positioning of the manometer is often more convenient if an extension tube (provided with most disposable trays) connects the needle hub to the stopcock, which is in

Figure 62-3 (Figure Not Available) Midsagittal section through lumbar spinal column with spinal puncture needle in place between spinous processes of L3 and L4. Note the slightly ascending direction of the needle. The needle has pierced three ligaments and the dura/arachnoid and is in the subarachnoid space. *(From Lachman E: Anatomy as applied to clinical medicine. New Physician 17:145, 1968.)*

turn attached to the manometer. The manometer should be positioned so that the "zero" mark is at the level of the spinal needle. The patient is then asked to relax. Extending the legs after needle placement does not meaningfully decreased the opening CSF pressure. [2] The fluid column is observed for phasic changes with respirations and arterial pulsations. This ensures needle placement in the subarachnoid space. If the needle is against a nerve root or is only partially within the dura, the pressure may be falsely low, and respiratory excursions will not be seen in the manometer. Minor rotation of the needle may solve these problems. Hyperventilation to relax the patient should not be attempted, because this will reduce the pressure readings owing to hypocapnia and resultant cerebral vasoconstriction.

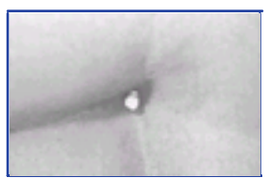

Figure 62-4 The spinal needle is usually advanced ½ to ¾ of its length before the spinal canal is reached. In this obese patient the needle was advanced all the way to the hub of the needle before spinal fluid was returned.

After measuring the pressure, the clinician should turn the stopcock and collect enough fluid to perform all desired studies. Even if the pressure is elevated, sufficient fluid should be removed for performance of all indicated studies, because the risk of the procedure involves the dural rent and not the amount of fluid initially removed. Presumably, more fluid will be lost subsequently through the hole in the dura. After cleaning off the povodine-iodine from the patient's back using a wash cloth moistened with warm tap water, a clean dressing is placed over the puncture site. Commercial trays supply four specimen tubes. Generally, tube 1 is used for determining protein and glucose levels and for electrophoretic studies, tube 2 is used for microbiologic and cytologic studies, and tube 3 is for cell counts and serologic tests for syphilis. In the presence of bloody CSF, cell counts should be performed in tubes 1 and 3 to help differentiate traumatic taps. One may compare water placed in tube 4 with CSF in tube 3 to detect cloudiness or discoloration. Alternatively, tube 4 can be stored under refrigeration in the laboratory for subsequent studies.

Traumatic taps are common and usually clinically inconsequential but they can be minimized by proper patient and needle positioning. A traumatic tap most commonly occurs when the subarachnoid space is transfixed at the entrance of the ventral epidural space, where the venous plexus is heavier. A plexus of veins forms a ring around the cord, and these veins may be entered if the needle is advanced too far ventrally or is directed laterally (Fig. 62-6 (Figure Not Available)). If blood is encountered and the fluid does not clear, the procedure should be repeated at a higher interspace with a fresh needle. A traumatic tap, per se, is not a particularly dangerous problem in the patient with normal coagulation, and no specific precautions are needed if blood-tinged fluid is obtained. However, observation for signs of cord or spinal nerve compression from a developing hematoma within the first several hours should be routine for these patients.

Figure 62-5 (Figure Not Available) Whenever feasible, opening pressure should be measured, not estimated, by means of an air-water manometer affixed to the needle by a three-way stopcock, thus allowing pressure to be taken and fluid withdrawn through a single needle, normal pressure is 70 to 180 mm H_2O. *(From Cole M: Pitfalls in cerebrospinal fluid examination. Hosp Pract 4:47, 1969. Illustration by Carol Donner. Reproduced by permission.)*

Lateral Approach in Lumbar Puncture

The supraspinal ligament may be calcified in older persons, making a midline perforation difficult. A calcified ligament may deflect the needle. In this case, a slightly lateral approach may be used. As the lower lamina rises upward from the midline, the needle is directed slightly cephalad to miss the lamina and slightly medially to compensate for the lateral approach. The needle passes through the skin, superficial fascia, fat, the dense posterior layer of thoracolumbar fascia, and the erector spinae muscles. The needle then penetrates the ligamentum flavum (bypassing the supraspinal and interspinal ligaments), the epidural space, and the dura before CSF is obtained (Fig. 62-7 (Figure Not Available)).

Cisternal Puncture

In situations in which lumbar puncture is contraindicated (such as local infection or acute trauma to the lumbar spine),

Figure 62-6 (Figure Not Available) Spinal contents at L4 and L5 show the relationship of a lumbar puncture needle to the major vessels at this level. The major radiculomedullary vein, shown accompanying the L5 nerve root, is situated far laterally to a needle correctly positioned in the midline of the dural sac. Note the avascular subdural space. *(From Edelson RN, Chernik IVL, Rosner JB: Spinal subdural hematomas. Arch Neurol 31:134, 1974. Illustration by Lynn McDowell. Reproduced by permission. Copyright 1974, American Medical Association.)*

cisternal, or suboccipital, puncture is the usual alternative. Technical problems, such as morbid obesity, cord tumor, arachnoiditis, bony deformities, or prior spinal surgery (fusion), may make lumbar puncture impossible. Contrast material may be injected into the cisterna magna to identify the rostral extent of an obstructing lesion identified by lumbar myelography.

The patient is cleaned and anesthetized in a manner similar to that for a lumbar puncture after the neck has been shaved from the external occipital protuberance to the mastoid process laterally. The patient is preferably placed in a lateral decubitus position, but a sitting position can be used. A pillow is placed under the head to keep the neck and the vertebral axis in the same plane. The patient's neck is flexed to the chest. The spinal needle is placed in the midline halfway between the spinous process of C2 and the inferior occiput. The needle is angled cephalad through the subcutaneous tissue until it comes in contact with the bony occiput. The needle is then withdrawn and subsequently advanced at a less acute angle with the horizontal plane of the cervical

Figure 62-7 (Figure Not Available) Horizontal section through the body of L3. Note the two puncture needles in the subarachnoid space. The medial one is in the midline, corresponding to the position

in Figure 62-3 (Figure Not Available) . The lateral one exemplifies the lateral approach, which avoids the occasionally calcified supraspinal ligament. Note the lateral needle piercing the intrinsic musculature of the back and only one ligament, the ligamentum flavum. *(From Lachman E: Anatomy as applied to clinical medicine. New Physician 17:145, 1968.)*

spine. This is repeated until the dural pop is felt. As in the lumbar region, the stylet should be removed frequently so that the dura is not punctured unknowingly. Fluid is removed in the usual manner. Dural veins are less extensive, and bloody taps are less common. Low-pressure headaches are less common, presumably because the subarachnoid pressure is lower and the dural tear can heal faster.

Lateral Cervical Puncture

The patient is placed in a supine position and fully sterilized and anesthetized. A 20-ga lumbar puncture needle is inserted perpendicular to the neck and parallel to the bed. The landmark for insertion is a point 1 cm inferior and 1 cm dorsal to the mastoid process. The clinician frequently removes the stylet to check for fluid return and, as at other sites, advances the needle slowly. If the needle goes too deeply and encounters paraspinous muscles, it is probably too deep posteriorly and should be repositioned more anteriorly. If bone is encountered, more dorsal placement is needed. Pressure and fluid samples are collected, as in other sites. [146]

The contraindications to cisternal and cervical punctures are the same as those to lumbar puncture. Both techniques are easily mastered, but prior demonstration of the procedure by an experienced operator is advised. When lumbar puncture cannot be performed for technical reasons and meningitis is suspected, placement of the needle under fluoroscopy may help obtain CSF.

Lumbar Puncture in Infants

Lumbar puncture in infants is usually performed to exclude meningitis. The sitting position may allow the midline to be more easily identified. Some authors use a nonstyleted needle in small infants, because this device allows the pressure to be estimated as the needle punctures the dura. [50] However, the failure to use a stylet may be the source of later development of an intraspinal epidermoid tumor. [83] A technique of lumbar puncture in the neonate using a butterfly infusion set needle has been described as a simplified procedure that may be useful in the squirming or hyperactive patient. [50] In general, the stylet is recommended primarily at the time of skin penetration and upon needle withdrawal. Although many operators use the stylet during any advancement of the needle, frequent stylet placement and withdrawal after skin penetration has occurred is optional.

If the child's neck is very tightly flexed, CSF may not be obtained. However, if the head is held in midflexion, CSF usually flows briskly. Prolonged severe flexion of the neck in an infant should be avoided because it may produce dangerous airway obstruction; the airway should be checked if the infant suddenly stops crying. [37] Incorrect positioning usually results in multiple punctures and a bloody tap. If CSF fails to flow, gentle suction with a 1.0-mL syringe may be used to exclude a low-pressure syndrome. Pressure readings are inaccurate in the struggling child and hence not commonly measured in infants or young children. Positioning is very important in the infant and is best accomplished by an assistant, who maintains the spine maximally flexed by partially overlying the child using the holder's chest and body weight to immobilize the thorax and hips, while holding the child behind the shoulders and the knees. The infant has poor neck control; hence, the assistant must also ensure that the child maintains an open airway.

Newborn and preterm infants may experience significant hypoxia during lumbar puncture with clinical deterioration; a sitting position appears preferable. [140] Lumbar puncture in infants with respiratory distress syndrome may cause greater risk than benefit. [31] This is a problem primarily in neonates but may occur in younger infants with sepsis. Gleason and coworkers[45] demonstrated that performing a lumbar puncture in the ill preterm infant using the traditional lateral recumbent position with full neck flexion resulted in significant respiratory abnormalities, presumably owing to a ventilation-perfusion mismatch. All infants with serious cardiopulmonary disease should be closely monitored during the procedure. Preoxygenation with or without oxygen saturation monitoring may be used as a precaution. Particular attention should be given to avoiding marked neck and trunk flexion.

Although local anesthesia or sedation has not been a routine practice during lumbar puncture in the infant or child, it is being reconsidered. Pain is perceived in the neonate, and local anesthesia neither produces physiologic instability nor makes the procedure more difficult. [91] [93] Sedation in the anxious child may be considered as an adjunct as for the adult, although these agents are relatively contraindicated in the obtunded infant or child without a protected airway or in the setting of hemodynamic instability.

Coley and colleagues advocate ultrasound evaluation in cases of failed lumbar puncture in neonates and infants. [20] Ultrasound was able to provide information concerning the presence or absence of CSF, the cause of the failed lumbar puncture, and whether to proceed with further attempts.

COMPLICATIONS

Implantation of Epidermoid Tumors

An epidermoid tumor or cyst is a mass of desquamated cells containing keratin within a capsule of well-differentiated stratified squamous epithelium. Congenital lesions are rare and arise from epithelial tissue that becomes sequestered at the time of closure of the neural groove between the third and fifth weeks of embryonic life. Acquired intraspinal epidermoid tumors result from implantation of epidermoid tissue into the spinal canal at the time of lumbar puncture performed with needles without stylets or with ill-fitting stylets. [83] The clinical syndrome consists of pain in the back and the lower extremities developing years after spinal puncture. Failure to use a stylet upon needle withdrawal may also result in aspiration of a nerve root into the epidural space.

Headache after Lumbar Puncture

A number of complications from lumbar puncture have been reported. By far the most common is the post-lumbar puncture headache. This occurs after 5% to 40% of spinal taps.[35] [39] [74] [133] The syndrome starts up to 48 hours after the procedure and usually lasts for 1 to 2 days and occasionally up to 14 days. Exceptional cases lasting months have been described. The headache usually begins within minutes after the patient arises and characteristically ceases as soon as he or she assumes a recumbent position. The pain is mild to incapacitating and is usually cervical and suboccipital but may involve the shoulders and the entire cranium. Exceptional cases include nausea, vomiting, vertigo, blurred vision, ear pressure, tinnitus, and stiff neck. The headache may improve and change to a positional backache or neckache. The headache appears in the first 1 to 3 days and may last for weeks or rarely months to years. The syndrome is thought to be caused by leakage of fluid through the dural puncture site. This results in an absolute reduction of CSF volume below the cisterna magna and a downward movement of brain tissue with displacement and stretching of pain-sensitive structures, such as meninges and vessels, which, in turn, causes a traction headache. Past puncture cerebral vasodilation is another proposed mechanism. In the recumbent position there is relief, because the weight of the brain is shifted cephalad. Dural leakage has been confirmed at surgery for disk disease and by at least one isotope myelographic study. [77] [129]

The incidence of headache in normal volunteers has been found to be one case per nine subjects with the use of a 26-ga needle and one case per three subjects with a 22-ga needle.[129] The headache was reported to be milder when the smaller needle was used. Technically, a 26-ga needle is difficult to place and to manipulate into a position in which it does not become intermittently obstructed by nerve roots. In addition, a syringe is needed to withdraw fluid, and pressure cannot be easily recorded. Theoretically, the incidence of headache is greater with an 18-ga needle.

Other factors claimed to influence the incidence of postspinal headache have been reviewed by Fishman and Lin. [79] The incidence is higher in young patients than in older patients and is also increased in females and individuals with a prior headache history. *Psychologic factors, quantity and rapidity of CSF removal, forced bedrest, immediate ambutation, postlumbar puncture hydration, and position during lumbar puncture have not been found to be relevant in the incidence of headache.*[70] Such common and strictly applied rituals have been termed mythical by some authors. [79] Some reports suggest a lower incidence with the lateral approach resulting from the production of holes in the dura and the arachnoid that do not overlap.

The influence of activity on postspinal puncture headache has been studied with contradictory results, including worsening of, improvement in, and no effect on the incidence of headaches when patients were mobilized. [14] Brocker studied 1094 patients and reported a reduction of headache from 36.5% to 0.5% by having the patients lie prone instead of supine for 3 hours after puncture with an 18-ga needle. He concluded that the prone position caused hyperextension of the spine and disrupted alignment of the holes in the dura and the arachnoid, making a leak less likely. Others have failed to show a decrease in duration, severity, or incidence of spinal headache with 24 hours of bedrest. [16] Interestingly, Vilming and colleagues [135] noted a slight reduction in postlumbar puncture headache if immediate mobilization was undertaken.

Much medical literature has commented on the incidence and severity of postdural puncture headache as related to spinal needle bevel orientation [41] [143] and type/size.[1] [17] [33] [40] [65] [78] [113] [127] [134] Concerning bevel orientation, it is clear that the incidence of postdural puncture headache is lower when the bevel of the needle is oriented parallel to the longitudinal axis of the spine. Until relatively recently, the explanation for this was that the parallel oriented bevel would separate rather than cut the longitudinally oriented dural fibers and thus result in a smaller dural hole and less CSF leakage. However, it is now known that the dural fibers are not oriented longitudinally but rather randomly. [99] Even with random fiber orientation, it is more likely that a longitudinal dural hole will be pulled closed during back flexion than a horizontal dural hole.

There are two basic types of spinal needles: cutting [96] and non-cutting or pencil-point (Sprotte and Whitacre). The non-cutting needles cause a lower incidence of postdural puncture headache. This may be because the tip of the non-cutting needle tends to separate rather than cut dural fibers. It has traditionally been thought that the dural hole resulting from non-cutting needle puncture is smaller than with cutting needle puncture; however, this may not be true. The important difference may be that the cutting needle causes a clean-cut opening in the dura while the non-cutting needle produces a jagged opening with rough edges. [98] The jagged opening may produce more intense inflammatory response, resulting in edema and more complete hole closure. The use of atraumatic spinal needles is not routine amongst all specialists. Perhaps as more spinal kit manufacturers include them in their kits their use will increase. [9] In thick-skinned individuals, passage of a thin, non-cutting needle may be technically difficult. The initial pass of a thicker cutting needle to the level of the interspinal ligament, followed by removal and advancement of the noncutting needle in the same soft tissue tract, can be helpful.

A smaller diameter needle will cause a smaller incidence of postdural puncture headache because it causes a smaller dural hole. However, a needle must have adequate CSF flow rates in order to allow timely CSF collection and pressure measurement. When these characteristics are considered, the 20-ga atraumatic needle may be the best overall choice for diagnostic lumbar puncture. [17]

Many medications have been advocated for treatment of post-lumbar puncture headache: barbiturates, codeine,

neostigmine, ergots, diphenhydramine (Benadryl), dimenhydrinate (Dramamine), amphetamine sulfate (Benzedrine), ephedrine, intravenous fluids (normal saline, Ringers lactate), magnesium sulfate, and vitamins.[19] [35] [136] Sechzer and Abel[111] in a double-blind demand method, found caffeine sodium benzoate (500 mg in 2 mL normal saline given by IV push) to be effective in 75% of patients. Additional patients responded to a second injection 1 to 2 hours later. Other clinicians prefer to put 500 mg of caffeine sodium benzoate in 1 L of saline and infuse the fluid over 1 hour. [58] This approach is contraindicated in the patient at risk for xanthine toxicity (e.g., supratherapeutic theophylline level, history of cardiac dysrhythmias). Aminophylline, 5 to 6 mg/kg by IV infusion over 20 minutes, is also reported to be useful.

Most clinicians generally follow the practice of using a styleted needle that is as small as possible. A 20- to 22-ga needle is often used for adults because of its stiffness and ease of fluid flow. Multiple punctures should be avoided. There is no certainty about activity and position immediately after the procedure. Most postspinal puncture headaches can be managed with bedrest with the head in the horizontal position. Dehydration should be avoided, because it lowers CSF pressure and might aggravate the headache. Although simple analgesics are commonly prescribed, they have no apparent advantage over bedrest and fluid intake. In the absence of a postural headache it is not necessary to stress immobilization or bedrest after lumbar puncture. [24] A patient with a prolonged headache after spinal puncture should be reassessed to rule out other structural causes of headaches. If the headache is not postural, other causes should be sought.

In cases in which a prolonged low-pressure headache exists, the placing of an epidural blood patch by experienced operators is highly successful. [15] [64] [88] [132] An epidural tap is performed at the level of the prior lumbar puncture. The loss-of-resistance technique using sterile saline can be used to locate the epidural space. [105] Ten to 20 mL of autologous blood is then drawn aseptically into a syringe and slowly injected (1 to 2 mL every 10 seconds) into the epidural space at the site of the

dural puncture. The injection is slowed or discontinued if back pain or paresthesias develop. The patient is kept supine for 1 hour while receiving intravenous hydration. Relief usually occurs within 20 to 30 minutes of the procedure. However, epidural patches are less likely to be effective if symptoms have been present for more than 2 weeks. Pain relief is due to the blood patch forming a gelatinous tamponade, hence stopping the CSF leak and providing an immediate elevation of CSF pressure. Patch failures (15% to 20%) are believed to be due to improper needle placement, injection of an inadequate quantity of blood, or an incorrect diagnosis. A second patch is often successful.

Complications reported after an epidural patch include back stiffness (15%), paresthesias, radicular pain, subdural hematoma, adhesive arachnoiditis, and bacterial meningitis.[15] [88] The procedure should be used in patients with refractory headaches who fail to respond to conservative therapy and should be performed by clinicians trained in the procedure.

Infection

Spinal puncture is absolutely contraindicated in the presence of local infection at the puncture site (cellulitis, suspected epidural abscess, or furunculosis) because of the danger of inducing meningitis.

Several laboratory and clinical investigators have examined the relationship between performance of a lumbar puncture during bacteremia and the later development of meningitis.[90] [126] A large concentration of bacteria in the bloodstream at the time of CSF examination is associated with meningitis. The meningitis could be coincidental ("spontaneous meningitis") or could result from leakage of blood containing bacteria into the subarachnoid space after lumbar puncture ("lumbar puncture-induced meningitis"). Eng and Seligman [32] reported that 14% of 165 cases of bacteremia caused by *Streptococcus pneumoniae, Haemophilus influenzae,* and *Neisseria meningitidis* had evidence of meningitis at the time of initial lumbar puncture. They argue that one cannot easily differentiate spontaneous meningitis from lumbar puncture-induced meningitis with these organisms because of their ability to invade the meninges spontaneously. It is likely that many of these rare cases of lumbar puncture-induced meningitis occur when the cautious clinician performs a lumbar puncture early in the course of meningitis, that is, before the infection has had time to be reflected in the CSF.

Teele and coworkers,[126] however, reported that 7 of 46 children developed meningitis after an initial normal lumbar puncture in the presence of bacteremia. *S. pneumoniae, H. influenzae,* and *N. meningitidis* were recovered in all cases. All cases of lumbar puncture-induced meningitis occurred in children younger than 1 year who received no antimicrobial therapy at the time of initial cultures. Empirical treatment for bacteremia despite a clear CSF is recommended by Teele and associates [126] in the child younger than 1 year if a high fever and leukocytosis are present.

Other studies suggest that the development of meningitis in children with occult bacteremia is more strongly associated with *S. pneumoniae, H. influenzae* type B, or *N. meningitidis* species of bacteria and *not* with the performance of lumbar puncture.[115] Suspected bacteremia is not a contraindication for performing a lumbar puncture; delay in diagnosis because of concern regarding the risks of a lumbar puncture is more serious than the risk of causing meningitis with the procedure. [36] [67]

Herniation Syndromes after Lumbar Puncture

Lumbar puncture is of value in confirming a diagnosis of meningitis and subarachnoid hemorrhage. Generally, when the patient has symptoms consistent with bacterial meningitis, lumbar puncture is performed before a head CT scan. Some have suggested that a lumbar puncture may be the best initial procedure to diagnose sybarachnoid hemorrhage, thus reducing the need for routine CT scanning in certain low risk patients with acute sudden headache. [110] However, in patients with a suspected intracranial mass lesion, a CT scan is generally performed before the lumbar puncture. Antibiotics are best administered *after* blood cultures are obtained and *before* the CT scan when meningitis remains on the differential diagnosis list.

Particularly with supratentorial mass lesions, there may be large pressure gradients between the cranial and lumbar compartments. When brain volume is increased because of a mass lesion or edema, rostrocaudal displacement may occur after lumbar puncture if the skull is intact. Techniques for management of increased ICP are discussed elsewhere (see Chapter 61).

The specific question whether or not lumbar puncture causes brain herniation to occur, when otherwise herniation

would not have occurred spontaneously, cannot be answered with certainty. Lowering the lumbar spinal canal pressure by removing CSF may increase the gradient between cranial and lumbar compartments, theoretically promoting both transtentorial and foramen magnum herniation. The frequency with which a lumbar puncture causes or accelerates transtentorial herniation is unknown, because a seriously ill patient might have developed herniation spontaneously without the procedure. Duffy reported experience with 30 patients referred to a neurosurgical service with post-lumbar puncture herniation syndromes; in this selected population of adverse outcomes, he noted that complications occurred in the first 12 hours after the procedure. [26] Half of these referred patients lost consciousness immediately after the puncture. In half of these patients, the lumbar pressure was normal. Only one third had papilledema.

With the availability of CT scanning, patients at risk of herniation should be less likely to undergo lumbar puncture. However, the literature from both the pre- and post-CT eras suggests a risk of herniation after lumbar puncture to be in the range of 2% to 3%. [66] Baker and colleagues noted that CT scans at their institution were poorly correlated with CSF pressures. [6] Among 13 patients with a CSF pressure of 210 mm H_2O or higher, 6 had normal CT scans and 3 had generalized atrophy. Only four had evidence of an intracranial mass, midline shift, or hydrocephalus. Of note, no patient demonstrated herniation over the subsequent 48 hours. Similarly, Zisfein and Tuchman[145] observed 38 patients with CT scan-documented intracranial mass lesions who underwent lumbar puncture. They noted that only one patient (who had fixed, dilated pupils and absent caloric reflexes before the lumbar puncture) died. All other patients were neurologically stable or had improved within 48 hours. Hence, with the current use of small-caliber spinal needles and aggressive concurrent use of ICP-lowering agents, herniation appears extremely rare and is not fully predictable by CT scan or opening CSF pressure readings.

Horowitz and coworkers[54] reported herniation in 18 of 302 infants with bacterial meningitis, all of whom experienced deterioration within 8 hours of lumbar puncture. Patients with meningitis who have localizing neurologic signs may have a coexisting brain abscess or infarction from arterial or venous thrombosis. Obstructive hydrocephalus and generalized cerebral edema may complicate meningitis with a secondary increase in ICP.

A careful neurologic examination should precede all spinal punctures. When there is a history of headache with progressive mental changes and the development of *localizing* neurologic signs, then spinal puncture should *not* be performed as the initial diagnostic procedure. A study by Gopal and colleagues [46] identified three statistically significant predictors of new intracranial masses: papilledema, focal neurologic examination, and altered mental status. Hasbun et al [51] demonstrated that in adults with suspected meningitis, clinical features could identify patients who were *likely* to have abnormal CT findings. Thus, the *absence* of the findings would suggest that the patient is a good candidate for immediate lumbar puncture since they have a low risk of brain herniation as a result of the procedure (Table 62-1). When these findings are present and meningitis is still clinically suspected, an initial dose of antibiotic should be administered empirically while awaiting results of CT or MRI to help assess the safety of a subsequent lumbar puncture. Appropriate cultures of blood and other body fluid should be obtained before

TABLE 62-1 -- Clinical Characteristics That Are Associated with Abnormal Findings on CT of the Head In Adults with Suspected Meningitis

Characteristic
Age =60yr
Immunocompromised state*
History of CNS disease†
Seizure within 1 wk before presentation
Neurologic findings
Abnormal level of consciousness
Inability to answer two questions correctly

Inability to follow two commands correctly
Gaze palsy
Abnormal visual fields
Facial palsy
Arm drift
Leg drift
Abnormal language‡

Author: Hasburn R, et al Computed Tomography Of The Head Before Lumbar Puncture In Adults With Suspected Meningitis. N Engl J Med 2001; 345:1727

*This category includes patients with human immunodeficiency virus infection or acquired immunodeficiency syndrome, patients who were receiving immunosuppressive therapy, and patients who had undergone transplantation.
†The CNS disease consisted of mass lesion, stroke, and focal infection.
‡The abnormalities consisted of aphasia, dysarthria, and extinction.

antibiotic administration. Blood cultures are positive in 80% of infants with meningitis. Measures to lower ICP before spinal puncture can also be instituted.

A CT scan should identify hemorrhagic lesions and most neoplasms. The CT scan should aid in the decision regarding the need for and the risk involved with spinal puncture. Head CT may help identify patients with unequal pressures between intracranial compartments who are at greater risk for cerebral herniation. CT findings that suggest unequal pressure between intracranial compartments include (1) lateral shift of midline structures, (2) loss of suprachiasmatic and circummesencephalic cisterns, (3) shift or obliteration of the fourth ventricle, and (4) failure to visualize the superior cerebellar and quadrigeminal plate cisterns with sparing of the ambient cisterns.[47] The presence of a posterior fossa mass would be a strong contraindication to a lumbar puncture. Because of bone and motion artifact, the posterior fossa unfortunately may be a difficult area to visualize on CT.

In summary, with regard to CT scan prior to lumbar puncture, there is no totally agreed upon standard of care that mandates when a lumber puncture should, or should not, be performed. A reasonable and logical approach is to avoid initial lumbar puncture, and first perform a CT scan when a mass lesion is suspected or if the patient has signs and symptoms of increased intracranial pressure as outlined in Table 62-1 . This approach correlates with the 2002 clinical policy promulgated by the American College of Emergency Physicians.[121]

Recognition of Herniation Syndromes

Herniation syndromes are the result of downward displacement of the hemispheres and the basal ganglia, which compress and displace the diencephalon and the midbrain rostrocaudally through the tentorial notch. Etiologic features and pathogenesis are detailed in the monograph by Plum and Posner. [92] Herniations have the potential to initiate vascular and obstructive complications that aggravate the original

expanding lesion and can create an irreversible pathologic process. The anterior cerebral artery may be compressed against the falx and may increase ischemia and edema of the herniating hemisphere. Midline displacement posteriorly compresses the deep great cerebral vein and raises pressure in its area of drainage. Compression of the posterior cerebral artery at the tentorial notch can produce occipital infarction and swelling. In addition, kinking of the aqueduct may interfere with CSF circulation. This blockage may produce a normal spinal CSF pressure. Transtentorial herniation displaces the brainstem downward, stretching medial perforating branches of the basilar artery, as the artery is tethered to the circle of Willis. This produces brainstem ischemia and hemorrhages.

In general, pathologic changes with supratentorial mass lesions spread through the hemisphere and move rostrad and caudad in a progressive manner, with progressive dysfunction of the hemisphere and, subsequently, succeeding levels of the brainstem. The infrequent exceptions are seen in patients with acute cerebral-intraventricular hemorrhage and in patients with hemispheric mass lesions with incipient herniation who undergo lumbar puncture. Such conditions may rapidly progress from hemispheric dysfunction to sudden medullary failure.

Central (Transtentorial) Herniation

This condition occurs in response to lesions of the frontal, parietal, and occipital lobes and the extracerebral lesions lying toward the vertex or the fronto-occipital lobes (Fig. 62-8 (Figure Not Available)). Frequently, patients are subacutely or chronically ill with bilateral disease, and the diagnosis may be uncertain. Initially, subjects exhibit a change in alertness or behavior. If the supratentorial lesion enlarges, compressing the diencephalon, stupor and then coma develop. At this point, monitoring of respiratory, ocular, and motor signs helps in diagnosing a supratentorial lesion and in determining the rostrocaudal direction of the disease process. Respirations at this time may be interrupted by deep sighs or yawns and periodic breathing of the Cheyne-Stokes type (periods of hyperpnea regularly alternating with apnea). Pupils are small but react briskly. Eye movements may be conjugate or slightly divergent with roving eye movements. Caloric testing with cold water produces a conjugate slow tonic movement to the side of irrigation. Many individuals have a hemiparesis before herniation. As the diencephalic stage of the central syndrome evolves, the contralateral hemiplegia may worsen, with the homolateral limbs developing a paratonic resistance to movement, but the individual continues to respond to noxious stimuli appropriately. At this stage both plantar responses are extensor. Decorticate responses appear and consist of flexor muscle hypertonus in the upper extremity with predominantly extensor hypertonus in the leg. Recognition of a diencephalic stage of herniation is important in that it gives warning that a potentially reversible lesion may become irreversible.

Once midbrain signs develop, infarction has likely occurred rather than reversible ischemia and compression. The chances of successfully removing or alleviating a supratentorial mass are small once the midbrain stage is reached.

Patients who develop midbrain and upper pons failure exhibit a sustained tachypnea; pupils dilate to a fixed midposition (3 to 5 mm), and oculovestibular reflexes become difficult to obtain, requiring side-to-side head movements and cold caloric irrigation. Disconjugate eye movements appear with failure to adduct (internuclear ophthalmoplegia). Motor responses give way to extensor hypertonus in all limbs (decerebrate rigidity). Midbrain damage results from ischemia and infarction, and few patients recover.

As the brainstem becomes more ischemic, the pupils maintain a fixed position, eye movements are lost, and decerebration gives way to flaccidity. The medullary stage consists of irregular respirations with long periods of apnea. The pupils dilate and the blood pressure falls, with death being inevitable.

Uncal Herniation

Uncal herniation occurs when expanding lesions in the temporal fossa shift the medial temporal lobe (uncus) and the hippocampal gyrus medially over the incisural edge of the tentorium. This flattens the midbrain, pushing it against the contralateral incisura. The third nerve and the posterior cerebral artery on the side of the lesion are caught between the swollen uncus and the free edge of the tentorium (Fig. 62-9 (Figure Not Available) and Fig. 62-10 (Figure Not Available)). The earliest sign is a unilaterally sluggish or slightly dilated pupil. Because the diencephalon may not be the first structure encroached on, impaired consciousness is not consistently present as an early sign of uncal herniation. Other respiratory, ocular, and motor findings may not be appreciably changed from earlier examinations. Pupillary dilation may persist for several hours, but once the patient progresses beyond this stage there is a tendency for midbrain dysfunction to occur rapidly. Ipsilateral external ophthalmoplegia soon follows pupillary dilation along with stupor and coma. Oculovestibular reflexes disappear as ischemia spreads to the midbrain. As the opposite cerebral peduncle is compressed against the tentorial edge, hemiplegia may appear ipsilaterally to the expanding supratentorial lesion (Kernohan's notch). Decerebrate posturing develops, with the opposite pupil becoming dilated and fixed. Progression then proceeds as described for the central syndrome. As with transtentorial herniation, once midbrain failure occurs, survival is much less likely.

Backache and Radicular Symptoms

Minor backache occurs with a frequency of 90% from the trauma of the spinal needle. Frank disk herniation has been reported from the passing of the needle beyond the subarachnoid space into the annulus fibrosis. Transient sensory symptoms from irritation of the cauda equina are also common.

Other reported complications include transient unilateral or bilateral sixth nerve palsies caused by stretching or displacement of the abducens nerve as it crosses the petrous ridge of the temporal bone, subarachnoid hemorrhage, subdural and epidural hematoma, anaphylactoid reactions to local anesthetics, settling of cord tumors, and retroperitoneal abscess produced by dural laceration in patients with meningitis. [75] [128] Most of these are rare and seldom encountered.

Most of the complications of lateral cervical and cisternal puncture are similar to those encountered with a lumbar puncture. In addition, perforation of a large vessel with resultant cisterna magna hematoma or obstruction of vertebral artery flow has been described. Puncture of the medulla oblongata may cause vomiting or apnea, and puncture of the cord may be associated with pain. Long-lasting side effects of cord puncture are probably minor. [146] In addition, traumatic tap and post-spinal puncture headache may occur with lateral cervical and cisternal puncture.

Figure 62-8 (Figure Not Available) Intracranial shifts from supratentorial lesions. *A,* The relationships of the various supratentorial and infratentorial compartments are seen in a coronal section. *B,* Central transtentorial herniation. A patient with carcinoma of the lung and multiple cerebral metastases (none is apparent in this section) died after developing signs and symptoms of the central syndrome of rostral-caudal deterioration. The brain is swollen, the diencephalon is compressed and elongated, and the mammillary bodies lie far caudad to those in the normal brain. Neither the cingulated gyrus nor the uncus is herniated. *C,* Uncal and transtentorial herniation. A patient developed a massive hemorrhagic infarction and died after developing the syndrome of uncal herniation. The cingulated gyrus is herniated after the falx; there is hemorrhagic infarction of the opposite cerebral peduncle and marked swelling and grooving of the uncus on the side of the lesion. Central downward displacement is also present but is less marked than in *B*. *(From Plum F, Posner J: The Diagnosis of Stupor and Coma. Philadelphia, FA Davis, 1980.)*

INTERPRETATION

Pressure

The pressure of the CSF is of great clinical importance. Although, it will occasionally be logistically impossible to obtain, it should be accurately measured whenever feasible. Accurate measurement is dependent on patient cooperation and in the struggling or agitated patient, measurements may be inaccurate. In such cases, sedation may provide more accurate readings. Elevated pressures are abnormal. Opening pressure is taken promptly, avoiding falsely low values due to leakage through and around the needle. Normal pressure is between 70 and 180 mm H_2O. Herniating cerebellar tonsils may occlude the foramen magnum and prevent increased ICP from being reflected in the lumbar pressure reading. Increased ICP can result from expansion of the brain (edema, hemorrhage, or neoplasm), overproduction of CSF (choroid plexus papilloma), a defect in absorption, or obstruction of flow of CSF through the ventricles. Cerebral edema may be associated with meningitis, CO_2 retention, subarachnoid hemorrhage, anoxia, congestive heart failure, or superior vena cava obstruction. Pressure may be falsely elevated in a tense patient, when the head is elevated above the plane of the needle, and, possibly, with marked obesity or muscle contraction. [39] Pressure is not usually measured in the neonate, because a struggling or crying child will have a falsely elevated pressure. The upper limits of normal are 50 mm H_2O in neonates and 85 mm H_2O in infants and young children. [80] Although the manometer reading is falsely elevated in the sitting position, the level should not rise above the foramen magnum in the child.

Low pressure should suggest obstruction of the needle by meninges. Low pressure can also be seen with spinal block. Rarely, a primary low-pressure syndrome occurs in a setting

Figure 62-9 (Figure Not Available) The floor of the anterior and middle fossae, illustrating the tentorial notch and the way in which the third nerve (Oculomoter N.) passes between the posterior cerebral and superior cerebellar arteries over the petroclinoid ligaments. *(From Plum F, Posner J: The Diagnosis of Stupor and Coma. Philadelphia, FA Davis, 1980.)*

of trauma, following neurosurgical procedures, secondary to subdural hematomas in elderly patients, with barbiturate intoxication, and in cases of CSF leakage through holes in the arachnoid. [97] [116]

The Queckenstedt test is useful for demonstrating the presence of obstruction in the spinal subarachnoid space. [39] [130] The test is seldom performed today, because myelographic techniques have been refined and the availability of MRI has reduced the number of myelograms and associated lumbar puncture studies. With the patient in the lateral recumbent position, jugular vein compression causes decreased venous return to the heart. This distends cerebral veins and causes a rise of ICP, which is transmitted throughout the system and is measured in the manometer. After 10 seconds of bilateral compression, CSF pressure usually rises to 150 mm H_2O over the initial reading and returns to the baseline in 10 to 20 seconds after release. If there is no change in the lumbar pressure or if the rise and fall are delayed, it should be concluded that the spinal subarachnoid space does not communicate with the cranial subarachnoid space. In this situation, Pantopaque should be injected before needle removal to facilitate subsequent performance of a myelogram. This is necessary because the lumbar dural sac may collapse, making it impossible to reenter the canal. If cervical cord disease is suspected, the test should be repeated with the neck in the neutral position, hyperextended, and flexed. When lateral sinus obstruction is suspected, unilateral jugular venous compression may be used (Tobey-Ayer test).

Appearance

If the CSF is not crystal clear, a pathologic condition of the CNS should be suspected. The examiner should compare the fluid with water, viewing down the long axis of the tube or holding both tubes against a white background. A glass tube is preferred, because plastic tubes are frequently not clear. The fluid may be clear with as many as 400 RBCs/µL and 200 WBCs/µL. [39]

Xanthochromia is a yellow-orange discoloration of the supernate of centrifuged CSF. Xanthochromia is generally considered to be the result of subarachnoid hemorrhage of at least a few hours duration and has been used to differentiate prior bleeding from a traumatic tap. A traumatic tap usually does not exhibit xanthochromia (see traumatic tap). Xanthochromia is produced by red cell lysis and is caused by one or more of the following pigments: oxyhemoglobin, bilirubin, and methemoglobin. Oxyhemoglobin causes red coloration; bilirubin, yellow; and methemoglobin, brown. Oxyhemoglobin is seen within 2 hours of subarachnoid bleeding and red cell lysis. Formation reaches a maximum in 24 to 48 hours after hemorrhage and disappears in 3 to 30 days. [130] The appearance of bilirubin in the CSF involves the conversion of oxyhemoglobin by the enzyme heme oxygenase. The enzyme is found in the choroid plexus, the arachnoid, and the meninges. Enzyme activity appears approximately 12 hours after the hemorrhage. [130] Bilirubin may persist for 2 to 4 weeks. Bilirubin in CSF caused by hepatic or hemolytic disease does not appear until a serum level of 10 to 15 mg total bilirubin per 100 mL is reached, unless underlying disease associated with a high CSF protein is present. Xanthochromia may be seen with CSF protein values above 150 mg/dL. CSF may clot with complete spinal block and very high CSF protein.

Oxyhemoglobin and bilirubin may be measured chemically or by spectrophotometric analysis. Demonstration of these compounds in CSF may help in the distinction between recent intracranial hemorrhage and a traumatic tap. Oxyhemoglobin may form as a result of red cell lysis if the tube has been allowed to stand for more than 1 hour before testing. Groves and Sidman noted that an RBC concentration of 5,000/µL created by addition of RBCs to a cellular CSF fluid will produce spectrophotometrically evident xanthochromia by 2 hours. [48] The addition of RBCs to form RBC concentrations of 20,000/µL and 30,000/µL will produce xanthochromia by 1 hour or immediately, respectively.

Figure 62-10 (Figure Not Available) The undersurface of the forebrain in transtentorial herniation. Compare the positioning of cranial nerve *III* and the adjacent posterior cerebral artery as well as the shape of the midbrain with those in Figure 62-8 (Figure Not Available) . Large arrows point to the edge where the hippocampus herniated through the tentorium. *Small arrows* and the *dotted line* indicate the lateral shift of the mesencephalon, which produces a hemorrhagic Kernohan's notch at point *K*. *(From Plum F, Posner J: The Diagnosis of Stupor and Coma. Philadelphia, FA Davis, 1980. Reproduced by permission.)*

Methemoglobin is a reduction product of oxyhemoglobin characteristically found in encapsulated subdural hematomas or in old intracerebral hematomas.

Cells

The technique involved in cell counts is reviewed elsewhere. [39] WBC counts over 5 cells/µL should be taken to indicate the presence of a pathologic condition. In a study of 135 normal university students, Tourtellotte and Shorr reported almost exclusively lymphocytes and monocytes, and the presence of one to five lymphocytes in the CSF may be normal. [130] On the other hand, neonatal CSF may show up to 32 WBCs/µL with prominent neutrophils, while infants 4 to 8 weeks old may have 22 WBCs/µL. [12] It has been stated that polymorphonuclear leukocytes are never seen in normal adults. However, with the use of the cytocentrifuge, an occasional specimen may show a neutrophil in an otherwise normal individual. [52] Such a finding should routinely prompt culture of the CSF, because the presence of a neutrophilic pleocytosis is commonly associated with bacterial infections or the early stages of viral infections, tuberculosis, meningitis, hematogenous meningitis, and chemical meningitis due to foreign bodies.

As many as 30% of patients may exhibit CSF pleocytosis after a generalized or focal seizure. In a review of 102 patients without CNS infection who had lumbar puncture within 48 hours of a seizure, Prokesch et al [94] reported an average of 72 cells/mm^3 , noting the presence of both polymorphonuclear and mononuclear cells. Although these patients did not have CNS infection, many did have serious intracranial pathologic processes (subdural hematoma, subarachnoid hemorrhage, or stroke). This finding should prompt a search for infection and assessment of the seizure as being possibly secondary to an underlying neurologic disorder.

Small lymphocytes may be seen in normal individuals. Small and large immunocompetent cells are found with a variety of bacterial, fungal, viral, granulomatous, and spirochetal diseases as well as with the presence of foreign substances.

Eosinophils are always abnormal and most commonly represent a parasitic infestation of the CNS. They may also be seen after myelography and pneumoencephalography and, to a minor degree, in other inflammatory diseases, including tuberculous meningitis and neurosyphilis. CSF eosinophilia has also been reported in cases of subarachnoid hemorrhage, lymphoma, Hodgkin's disease, brucellosis, fungal meningitis, *Mycoplasma pneumoniae* infection, measles, lymphocytic choriomeningitis, rickettsial infections, leukemia, demyelinating diseases, sarcoidosis, acute inflammatory demyelinating polyneuropathy, allergic reactions, and idiopathic eosinophilic meningitis. [69]

Normal CSF RBCs are <10/µL. Counts up to 100/µL that are otherwise unexplained may be due to a traumatic tap. Herpes simplex virus encephalitis may elevate the CSF RBC count in many patients. Myeloid and RBC precursors may contaminate CSF with bone marrow cells from an adjacent vertebral body. [68]

Glucose

Glucose enters the CSF by way of the choroid plexus as well as by transcapillary movement into the extracellular space of the brain and the cord by carrier-mediated transport. It then equilibrates freely with the CSF subarachnoid space. Fishman [39] concluded that a low CSF glucose concentration indicates increased glucose use in the brain and the spinal cord and, to a lesser degree, by polymorphonuclear leukocytes, bacteria, and inhibition of membrane carrier systems. Once in the CSF, glucose undergoes glycolysis and there is an invariable rise in CSF lactate levels. Glucose levels remain subnormal for 1 to 2 weeks after the effective treatment of meningitis.

The normal range of CSF glucose is between 50 and 80 mg/dL, which is 60% to 70% of the glucose concentration in the blood. Ventricular fluid glucose levels are 6 to 8 mg/dL higher than in lumbar fluid. A ratio of CSF to blood of <0.5 or CSF levels <40 mg/dL are invariably abnormal. The ratio is higher in infants for whom a ratio of <0.6 is considered abnormal. Hyperglycemia may mask a depressed CSF glucose level, and the CSF to blood glucose ratio should be measured routinely. With extreme hyperglycemia a ratio of 0.3 has been suggested as being abnormal. [49] Between 90 and 120 minutes is required before the CSF glucose reaches a steady state with blood glucose changes (e.g., after the intravenous injection of glucose). When CSF glucose is of diagnostic importance, CSF and blood samples ideally should be obtained after a 4-hour fast.

Low CSF glucose levels may be found in several diseases of the nervous system (Table 62-2). Only low concentrations of glucose are of diagnostic value, and elevated CSF

TABLE 62-2 -- Low CSF Glucose Syndromes

Bacterial meningitis
Tuberculous meningitis
Fungal meningitis
Sarcoidosis
Meningeal carcinomatosis
Amebic meningitis
Cysticercosis
Trichinosis
Syphilis
Chemical meningitis
Subarachnoid hemorrhage
Mumps meningitis
Herpes simplex encephalitis
Hypoglycemia

glucose levels generally have no significance; elevation usually reflects hyperglycemia. A rapid estimate of the CSF glucose level can be obtained by using bedside reagent strip testing with a commercial autoanalyzer. However, formal laboratory testing is recommended for confirmation of bedside levels.

Protein

The normal range of the lumbar CSF protein level is 15 to 45 mg/dL. Infants normally have a lower level than adults, and protein levels may drop after a lumbar puncture. The concentration is lower in the ventricles (5 to 15 mg/dL) and the basilar cisterns (10 to 25 mg/dL), reflecting a gradient in the permeability of capillary endothelial cells to proteins in the blood. Levels of CSF protein in premature infants and full-term neonates is higher than in adults with a mean of 90 mg/dL; protein levels decline by age 8 weeks, reflecting maturation of the blood-brain barrier. Most of the proteins in CSF normally come from the blood, which normally has a protein concentration of up to 8000 mg/dL. Protein entry is determined by its molecular size and the relative impermeability of the blood-CSF barrier. A full range of serum proteins are found in CSF at several hundred-fold dilution. Faulty reabsorption of protein by arachnoid villi may also elevate protein levels. Increases in CSF total protein levels are a nonspecific abnormality associated with many disease states. Levels higher than 500 mg/dL are uncommon and are seen mainly in meningitis, in subarachnoid bleeding, and with spinal tumors. The high levels seen with cord tumors result from an increase in local capillary permeability. With high levels (generally 1000 mg/dL), CSF may clot (Froin's syndrome).

Hemorrhage into the CSF or the introduction of blood by a traumatic tap increases CSF protein levels. If the serum protein is normal, the CSF protein should theoretically rise by 1 mg/dL for every 1000 red cells, but this is variable. The inflammatory effect of hemolyzed RBCs may also significantly increase CSF protein.

Selective measurement of immunoglobulin fractions in CSF has proved to be of diagnostic value in suspected cases of multiple sclerosis. Elevated CSF immunoglobulin levels may reflect blood-brain barrier disruption or local antibody response to a CNS immune response. [141] Stimuli may be infectious or antigenic, producing an inflammatory response. Immunoglobulin elevation has been found in many chronic inflammatory conditions, including syphilis, viral encephalitis including HIV infection, subacute sclerosing panencephalitis, progressive rubella encephalitis, tuberculous meningitis, sarcoidosis, cysticercosis, and acute inflammatory demyelinating polyneuropathy (Guillain-Barré syndrome).

The Traumatic Tap

The incidence of a traumatic tap is 10–15%, depending on the cut off of RBCs used to define the condition. A cutoff of 400 RBCs/µL is commonly used. It is often possible to distinguish between subarachnoid bleeding and bloodshed by the spinal needle if certain steps are taken *at the time of the initial puncture*. In traumatic punctures the fluid generally clears between the first and third tubes as the needle is washed by CSF. Decreasing cell counts on the first and third tubes help confirm this. In a recent hemorrhage, however, a declining cell count may represent layering of cells in a recumbent patient. The fluid should then be centrifuged. With moderately blood-stained fluid, the supernatant should be clear if the RBCs have been present for =2 hours (traumatic tap). Red cells undergo hemolysis in the CSF after a few hours to produce xanthochromia. Xanthochromia persists for up to 4 weeks, depending on the number of RBCs that were originally present. An early CSF examination may show clear fluid before the development of hemolysis, even after spontaneous subarachnoid bleeding. However, xanthochromia may be measured

immediately after a traumatic tap if the RBC count exceeds 30,000/µL. [48] The presence of a clot in one of the tubes strongly favors a traumatic tap.

In subarachnoid hemorrhage, clotting does not occur because blood is defibrinated at the site of the hemorrhage. Any lumbar puncture performed several days after a traumatic tap may yield stained fluid. An immediate repeat puncture at a higher interspace yielding clear CSF may also help distinguish a traumatic tap. The fluid from a traumatic tap should contain about 1 WBC per 700 RBCs if the complete blood cell count is normal, but this ratio is highly variable. Formulas to assess the contribution of total CSF cell count and protein values from traumatic taps have been proposed but do not have uniform support. [85] [86] Indeed, Bonadio and colleagues[11] suggested that children with meningitis and traumatic taps (i.e., RBC count =1000/µL) generally have other features of meningitis besides an observed-to-predicted WBC ratio of 10 or more (e.g., predominance of neutrophils, low CSF-to-serum-glucose ratio, positive Gram stain). All blood-contaminated CSF should be cultured, especially from uncooperative infants and children being evaluated for sepsis.

Lang and colleagues suggest the use of a D-dimer test on CSF to identify subarachnoid hemorrhage. [73] Although identifying local fibrinolysis, other conditions such as disseminated intravascular coagulation, a previous traumatic tap, or prior thrombolytic therapy may produce false-positive test results. Eskey and Ogilvy [34] have suggested that the routine use of fluoroscopy-guided lumbar puncture in patients with suspected subarachnoid hemorrhage and negative CT scan would reduce the frequency of traumatic punctures.

CSF Analysis with Infections

Bacterial Infections

The CSF findings are essential in establishing a provisional diagnosis of acute bacterial meningitis. CSF analysis establishes the diagnosis, the causative organism, and the choice of antibiotics. CSF must be transported to the laboratory immediately and examined at once. CSF cells begin to lyse within 1 hour of collection, but this may be slowed by refrigeration. In cases of meningococcal infection, a delay in processing may cause the diagnosis to be missed, because the organism

tends to autolyze rapidly. Speed is only slightly less important with other organisms, because early initiation of antibiotic therapy is crucial.

The Gram stain is of great importance, because this often dictates the initial choice of antibiotic therapy. Gram-negative intracellular or extracellular diplococci are indicative of *Neisseria meningitidis*. Small gram-negative bacilli may indicate *Haemophilus influenzae*, especially in children. The presence of gram-positive cocci indicates *Streptococcus pneumoniae*, other *Streptococcus* species, or *Staphylococcus*. Twenty percent of Gram stains may be falsely negative because too few organisms are present. The Gram stain smear is more likely to be positive in patients who have not received prior antibiotic therapy. Acridine orange stain may improve the yield in bacterial infections with negative Gram stains. [62]

For culture, blood and chocolate agar are required. *N. meningitidis* and *H. influenzae* grow best on chocolate agar. The plates are incubated under 10% CO_2. Thioglycolate medium is used for possible anaerobic organisms. Cultures are examined at 24 and 48 hours, but plates should be kept for at least 7 days. Large volumes of CSF may improve yields.[117]

While the culture is pending, one may suspect a bacterial infection in the presence of an elevated opening pressure and a marked pleocytosis ranging between 500 and 20,000 WBCs/µL.[22] The differential count with bacterial infections is usually composed chiefly of neutrophils. A count of more than 1000 cells/µL seldom occurs in viral infections. Occasionally, acellular fluid may be found in the severely immunosuppressed patient or others with appropriate presentations. Repeat lumbar puncture may be required in febrile patients in whom the clinical features remain compatible with meningitis. [49] [100] [138]

CSF glucose levels =40 mg/dL or less than 50% of a simultaneous blood glucose level should raise the question of bacterial meningitis, even in the presence of a negative Gram stain and a low cell count. Glucose levels with bacterial meningitis are occasionally below 10 mg/dL; levels are normal in 9% of bacterial meningitis. [49]

The CSF protein content in bacterial meningitis ranges from 500 to 1500 mg/dL and usually returns to normal by the end of therapy. CSF protein values above 220 mg/dL virtually ensure an acute meningitis of being bacterial. Of note, prior antibiotic therapy may adversely affect the sensitivity of cultures and Gram stains for bacterial meningitis but does not significantly affect WBC counts, CSF to blood glucose ratios, or CSF protein values. [118] Spanos and colleagues[123] have developed a useful nomogram to help distinguish bacterial from viral infections (Fig. 62-11 (Figure Not Available)).

A useful test that can be performed in nearly all laboratories is the measurement of CSF lactate dehydrogenase (LDH) levels. In bacterial and fungal meningitis, LDH levels are increased.[7] The LDH level may be elevated even in patients who have received antibiotics for 1 or 2 days. The LDH levels in viral infections tend to be normal, whereas values in bacterial meningitis are usually two to four times greater than the normal concentration of approximately 1.6 mEq/L.

A study by Durack and Spanos questions the use of repeat spinal taps as a test of meningitis cure. [27] Their review of 165 meningitis cases revealed 13 instances in which the repeat tap led to unnecessary intervention and 2 others in which treatment failure was *not* detected by the repeat spinal tap.

Microbial Antigens and Polymerase Chain Reaction

Several tests other than CSF culture and Gram stain are available to establish a bacterial etiology of meningitis. These include blood cultures, CSF counterimmunoelectrophoresis (CIE), CSF latex agglutination (LA), and coagglutination counterimmunoelectrophoresis. [82] All of the ancillary tests have a low sensitivity for bacterial meningitis, thus limiting their use. In 50 to 80% of bacterial meningitis cases, blood cultures are positive for the etiologic agent. [125]

CIE uses wells in two rows of agarose gel. A different antiserum is placed in each well. A current is passed through the gel with the reactants then moving toward each other by electrophoretic mobilization of the antigen. A line of precipitation visualized in 1 to 4 hours represents positive reaction between antiserum and antigen.[28] Commercial kits are available to detect *S. pneumoniae; Listeria monocytogenes; H. influenzae; N. meningitidis* A, B, and C and W135; group B *Streptococcus*; K1 strains of *Escherichia coli; Klebsiella*; and *Pseudomonas* species.[63]

Particle agglutination involves staphylococcal coagglutination and latex agglutination. Antibody on the surface of a colloid combines with antigen-binding sites to cross-link the colloid-forming antigen bridges. A matrix forms and appears as a macroscopic agglutination. Agglutination tests can detect approximately 10 times less antigen than CIE. False-positive tests can occur in the presence of rheumatoid factor, serum complement components, and possibly other serum proteins. The technique may be used in infections due to *H. influenzae, S. pneumoniae, N. meningitidis*, and group B *Streptococcus*.

Another technique having some potential use is the enzyme-linked immunosorbent assay. This technique may detect 100 to 1000 times less antigen than agglutination tests but is technically more difficult and requires 4 hours to perform.

A positive CSF antigen test may be expected in 70% to 90% of patients with *Neisseria* meningitis. This compares with a positive Gram stain in approximately 70% of patients. Positive latex antigen tests have been reported in approximately 60% of *S. pneumoniae* meningitis cases, with a positive Gram stain in 80%. A positive latex test and Gram stain are reported in approximately 85% of *H. influenzae* meningitis cases.[28] [125] Group B *Streptococcus* can be detected with a 60 to 90% sensitivity. The Gram stain may be difficult to assess after antibiotic therapy has been initiated. Bacterial antigens may persist in the CSF for several days after antibiotic therapy. Twenty-five percent to 33% of positive tests are lost per day in the setting of appropriate antimicrobial therapy. A negative test, however, does not rule out bacterial meningitis.[63] In addition, blood and urine should be examined for antigen. Often, antigen may be found only in the urine. Urine needs to be concentrated and may have the disadvantage of reflecting urinary tract infections. The particle agglutination test for *H. influenzae* type B may be positive for up to 10 days after children have received *H. influenzae* polysaccharide vaccine. Antigen tests are not useful in diagnosing gram-negative bacillary, staphylococcal, and *Listeria* meningitis. In addition, although antigen tests may identify the bacterial pathogen, they do not provide information about the antibiotic susceptibility of the organism.

Future use of polymerase chain reaction (PCR) will aid the rapid diagnosis of CNS infection when results of current techniques are suboptimal. PCR is a method for amplifying target nucleic acid in CSF by use of repeated cycles of DNA

Figure 62-11 (Figure Not Available) Nomogram for estimating the probability of bacterial (ABM) versus viral (AVM) meningitis in immunocompetent host. Step 1: Place the ruler on reading lines for the patient's age and month of presentation, and mark the intersection with line A. Step 2: Place the ruler on the values for the glucose ratio and total polymorphonuclear leukocyte (PMN) count in cerebrospinal fluid, and mark the intersection with line B. Step 3: Use the ruler to join the marks on lines A and B, and then read off the probability of ABM versus AVM. *(From Spanos A, Harrell FE Jr, Durack DT: Diagnosis of acute meningitis: An analysis of the predictive value of initial observations. JAMA 262:2700, 1989. Copyright 1989, American Medical Association. Reprinted by permission.)*

synthesis. PCR requires use of flanking DNA sequences at the opposite ends of the target DNA. Synthetic primers anneal to their respective recognition sequences at the opposite end of the target sequence; they serve as primers for new DNA synthesis. PCR allows detection of and quantification of organisms whose genetic material is DNA or messenger RNA. PCR permits the diagnosis of infectious disease with a high degree of sensitivity and specificity and allows rapid reliable detection of microbes present in small numbers. Laboratory false-negative and false-positive results may occur. PCR is only available at limited centers, although this seems likely to change in the near future.[89]

Empirical Antibiotic Use Before Lumbar Puncture

Many patients are transported to a referral center or within a facility for a CT scan to rule out an intracranial mass after a clinical concern for meningitis is raised. In such instances, CSF examination may not be performed before transport. This may be due to technical problems (uncooperative or large patient) or concerns regarding safety of lumbar puncture in an obtunded patient with possible increased ICP. [49] [80] The initial clinician may have to decide whether to initiate empirical antibiotic therapy. Prior antibiotic administration could obscure the bacterial etiology, whereas a delay in initiating therapy may increase the morbidity and mortality of the illness. It may be difficult to identify individuals at risk for a fulminant course, and the diagnosis of bacterial meningitis cannot always be made with confidence; some cases may be misdiagnosed as subarachnoid hemorrhage or metabolic encephalopathy.

Talan and associates[125] noted that parenteral antibiotic treatment decreases the percentage of positive CSF cultures by 4 to 33% and of positive CSF Gram stains by 7 to 41%. A minimum window of 2 to 3 hours exists after giving parenteral antibiotics when CSF cultures are not adversely affected. After 24 hours post treatment, as many as 38% of meningitis patients may still have positive CSF cultures. Kanegaye et al [61] demonstrated that CSF sterilization may be dependent on the infecting organism. They reported CSF sterilization post antibiotics within the following time frame for various organisms: Meningococcal (<2 hrs), pneumococcal (<4.3 hrs) and Group B streptococcal (>8 hrs).

When CSF is cultured more than several hours after parenteral antibiotics are administered, antigen tests may be of help. Of course, blood cultures should be obtained immediately before antibiotic administrations whenever possible. Occasionally, patients may develop a lymphocytic pleocytosis with antibiotic therapy; but in most cases, cell count, differential, glucose, and protein concentrations are not changed in the first 2 to 3 days of antibiotic therapy. [49] Although there are no data to confidently address the potential advantages or disadvantages of antibiotic therapy before lumbar puncture, it is reasonable to initiate therapy on the premise that a delay may be deleterious. If a lumbar puncture cannot be done, consultation with clinicians at the referral center would seem appropriate. If a lumbar puncture is performed before a transfer, a portion of the CSF (chilled on ice) should be sent with the patient or held in the referring hospital laboratory.

Bacterial meningitis occurring in children younger than 10 years of age historically has been often due to *H. influenzae*. Fortunately, *H. influenzae* appears to be easier

to grow from early postantibiotic cultures and is more likely to be associated with positive blood cultures and antigen tests. In the pediatric population a single dose of antibiotic before transport is unlikely to prevent bacterial identification. In neonates, adults, and immunosuppressed patients, the sensitivity of blood cultures and immunologic tests is less reliable. CSF examination before antibiotic administration or *early* in the course of treatment is preferred.

For suspected or confirmed cases of acute bacterial meningitis, antimicrobial therapy can be started based on the most likely causative organism based on age of the subject, associated diseases, and renal function.

For immunocompromised and postneurosurgical patients, a third-generation cephalosporin (cefotaxime, ceftizoxime, ceftazidime, or ceftriaxone) plus ampicillin and vancomycin should be used for coverage against staphylococci, *L. monocytogenes*, and gram-negative organisms.[82] Table 62-3 Table 62-4 Table 62-5 offers guidelines for emergency antibiotic therapy. The third-generation cephalosporins are efficacious in many empirical regimens or situations where the organism is known. To rely on third-generation cephalosporins alone for all cases of bacterial meningitis would result in treatment failures of all *Listeria* species and increasing numbers of *Enterobacter, Serratia*, and *Pseudomonas* groups. Treatment with older agents may be as or more effective in bacteria demonstrating appropriate sensitivity.

Dexamethasone Therapy in Bacterial Meningitis

Studies have suggested benefit from adjunctive dexamethasone therapy in reducing neurologic sequelae, especially hearing loss, in children with *H. influenzae* meningitis and for a lower mortality and a better overall outcome in adults with acute bacterial meningitis. [23] There appears to be no adverse sequelae from steroid therapy. The adjunctive benefit of corticosteroids during treatment of meningitis caused by other organisms is unknown.

In acute bacterial meningitis, bacterial cell wall components including lipopolysaccharides and teichoic acid initiate

TABLE 62-3 -- Empirical Therapy for Purulent Meningitis*

Predisposing Factor	Antimicrobial Therapy‡
Age	
0–4 wk	Ampicillin plus cefotaxime; or ampicillin plus an aminoglycoside
4–12 wk	Ampicillin plus a third-generation cephalosporin†
3 mo to 18 yr	Third-generation cephalosporin† ; or ampicillin plus chloramphenicol
18–50 yr	Third-generation cephalosporin† §
> 50 yr	Ampicillin plus a third-generation cephalosporin†
Immunocompromised state	Vancomycin plus ampicillin plus ceftazidime
Basilar skull fracture	Third-generation cephalosporin†
Head trauma; postneurosurgery	Vancomycin plus ceftazidime
Cerebrospinal fluid shunt	Vancomycin plus ceftazidime

*Consider corticosteroids prior to antibiotic administration (see text).
‡Vancomycin should be added to all empirical therapeutic regimens when highly penicillin- or cephalosporin-resistant strains of *Streptococcus pneumoniae* are suspected.
†Cefotaxime or Ceftriaxone.
§Add ampicillin if meningitis caused by *Listeria monocytogenes* is suspected.

TABLE 62-4 -- Recommended Dosages of Antimicrobial Agents for Meningitis in Adults with Normal Renal and Hepatic Function

Antimicrobial Agent	Total Daily Dose (IV)	Dosing Interval (hours)
Ampicillin	12 g	4
Cefotaxime	8–12 g	4–6
Ceftazidime	6 g	8
Ceftriaxone	4 g	12–24
Chloramphenicol*	4–6 g	6
Gentamicin†	3–5 mg/kg	8
Tobramycin†	3–5 mg/kg	8
Vancomycin†	2–3 g	8–12

*High dose recommended for pneumococcal meningitis.
†Need to monitor peak and trough serum concentrations.

and exacerbate the host response. These stimulate the production of cytokines, including interleukin-1 and tumor necrosis factor, from macrophages and monocytes. Cytokines may injure vessels, diminish cerebral perfusion, and stimulate cerebral swelling. [80] The dosage of dexamethasone is 0.15 mg/kg (10 mg IV in adults) every 6 hours for 4 days. When used, it is recommended that the corticosteroid *be given before antibiotic use*. Benefit in adults is less certain but experimental data in children would seem to support corticosteroid use, with caution being exercised in immunocompromised or leukopenic patients. A moderate inflammatory response in the meninges is required for penetration of the CNS by many antibiotics. Reducing meningeal inflammation will reduce the concentration of antibiotics in the CSF. Corticosteroids should be discontinued after approximately 4 days of treatment, at which time meningeal inflammation has been reduced by antibiotics. [87] [95]

Although an intriguing concept, the actual benefit from adjunctive corticosteroids is controversial. Current data do not allow for definitive recommendations regarding the use of corticosteroids in bacterial meningitis. With the increasing use of *H. influenzae* and *S. pneumoniae* vaccines, the role of corticosteroids in bacterial meningitis becomes less clear.

Neurosyphilis

The true incidence of this disease is unknown. Approximately 5000 new cases of neurosyphilis are estimated to occur in the United States each year. [55] The natural history and clinical manifestations have been modified in the antibiotic era. The widespread use of oral antibiotics has changed neurosyphilis into a chronic partially treated meningitis. Partial therapy may clear peripheral infection and attenuate the immune response. Therapy may be sufficient to mininimize symptoms but insufficient to eradicate organisms in the CNS and eye, which may then multiply. In the preantibiotic era, seizures were considered to complicate general paresis only late in the course of the illness. Previously, seizures were reported as an early manifestation in less than 5% of symptomatic neurosyphilis cases. More current reports indicate seizures occur in 25% of symptomatic cases and may, on occasion, be the sole manifestation of neurosyphilis. [55] Seizures are usually partial (focal), with one third of patients having no interictal clinical findings. A treponemal serologic test should be obtained in every adult with acquired partial seizures.

Ophthalmologic findings are frequently present in syphilis. These include a slowly progressive optic atrophy, an acute optic neuritis, cranial oculomotor neuropathy, and a

TABLE 62-5 -- Recommended Dosages of Antimicrobial Agents for Meningitis in Neonates, Infants, and Children with Normal Renal and Hepatic Function

	Total Daily Dose IV (Dosing Interval in Hours)		
Antimicrobial Agent	***Neonates (0–7 d)****	***Neonates (8–28 d)****	***Infants and Children***
Amikacin†	15–20 mg/kg (12)	20–30 mg/kg (8)	20–30 mg/kg (8)
Ampicillin	100–150 mg/kg (8–12)	150–200 mg/kg (6–8)	200–300 mg/kg (6)
Cefotaxime	100 mg/kg (12)	150–200 mg/kg (6–8)	200 mg/kg (6)
Ceftazidime	60 mg/kg (12)	90 mg/kg (8)	125–150 mg/kg (8)
Ceftriaxone	—	—	80–100 mg/kg (12–24)
Chloramphenicol	25 mg/kg (24)	50 mg/kg (12–24)	75–100 mg/kg (6)
Gentamicin†	5 mg/kg (12)	7.5 mg/kg (8)	7.5 mg/kg (8)
Tobramycin†	5 mg/kg (12)	7.5 mg/kg (8)	7.5 mg/kg (8)

*Smaller dosages and longer intervals of administration may be advisable for very low birth weight neonates (<2000 g).
†Need to monitor peak and trough serum concentrations.

chorioretinitis. A more specific sign is a dissociated pupillary response to light and convergence with loss of the light reflex (Argyll Robertson pupil).

Meningovascular neurosyphilis involves infection of both the meninges and the cerebral vasculature. The clinical picture is that of a cerebral infarction or an acute meningoencephalitis. Meningovascular syphilis also may present as headache, nausea, vomiting, confusion, stiff neck, and cranial nerve palsies. Arteritis may affect small, medium, or large vessels, producing infarction or ischemia of brain or spinal cord. The peak incidence is 5 to 7 years after initial infection. Tabes dorsalis, general paresis, and gummas occur 10 to 20 years after initial infection.

CSF findings suggestive of neurosyphilis include >5 WBCs/µL, elevated protein concentration, elevated ?-globulin concentration, and a positive serologic test for syphilis. The glucose concentration is usually normal. Higher cell and protein values are seen in early as opposed to late neurosyphilis.

Diagnostic certainty remains difficult. The diagnostic criterion standard is darkfield microscopy to identify morphology and flexing "corkscrew" motility of spirochetes. Serologic tests for syphilis are either treponemal or nontreponemal. Nontreponemal tests detect a nonspecific globulin complex called reagin. Reagin tests, such as the Venereal Disease Research Laboratory (VDRL) flocculation test, lack sensitivity and should not be used to exclude the diagnosis of neurosyphilis. One third to one half of patients with neurosyphilis have a negative VDRL test in the serum, and more than one third have a negative VDRL test in the CSF. [55] CSF VDRL is quite specific with false-positive results seen primarily with traumatic taps.

Treponemal tests provide evidence of a specific immune response to *Treponema pallidum*. These include the serum fluorescent treponemal antibody adsorption (FTA-ABS), microhemagglutination tests for *T. pallidum* (MHA-TP), and the *T. pallidum* hemagglutination assay (TPHA). These assays are believed to remain positive for life. The FTA-ABS and MHA-TP tests may revert to negative in 13 to 24% of patients after therapy for early (primary) syphilis. There is no evidence that these tests revert to negative in untreated disease. [101] The serum FTA-ABS test is the test of choice to confirm the diagnosis of neurosyphilis. The serum FTA-ABS test is reactive in 95 to 100% of cases of neurosyphilis. A negative serum FTA-ABS test makes CSF examination unnecessary. Its false-positive rate is less than 1%, but the incidence of biologic false-positive tests may increase in the presence of collagen vascular disorders.

A positive serum treponemal test indicates past infection with syphilis and may be reactive indefinitely, even after treatment. Therefore, CSF is used as a guide to the

presence and the activity of neurosyphilis. The VDRL test is the test of choice in CSF and, when positive, is strong evidence for neurosyphilis. False-positive CSF VDRL tests are rare. The FTA-ABS test is not used in CSF; the false-positive rate is between 4 and 6% and is believed possibly to represent antibodies that have passively entered from serum.[57] The FTA-ABS test measures IgG antibody and cannot differentiate active from past infection. The CSF VDRL either may be reactive by contamination with seropositive blood (traumatic tap, subarachnoid hemorrhage) or may occur with entry of serum reagin into CSF during meningitis.

There is some concern that today many patients with parenchymal neurosyphilis have normal CSF. This finding leads to the recommendation that a patient with signs of progressive neurosyphilis and a positive treponemal serologic test be treated with antibiotics regardless of the CSF findings. A CSF pleocytosis may be provoked after 1 week of therapy and may supply supportive evidence for a diagnosis of neurosyphilis. PCR may have future applications for diagnosis of neurosyphilis (see the section on neurosyphilis and HIV infection later in this chapter).

Viral Meningitis

The organisms most commonly isolated in viral meningitis are the enteroviruses (coxsackieviruses, echoviruses) and mumps virus. Enteroviruses are most commonly seen in the summer and fall, and mumps appears most frequently in the winter and spring. Viral cultures in most hospitals are not available and play little role in acute decisions regarding diagnosis and treatment. A serial rise in CSF antibody titers may be helpful but may be difficult to obtain in patients who have clinically recovered. Intrathecal production of organ-specific antibodies (IgM, IgG, and IgA isotopes) may be diagnostic of neurologic infection if there is no prior history of infections. This measurement of both serum and CSF antibody titers must be done in a specialized laboratory. Viral meningitis is diagnosed when bacterial culture and Gram stain are negative. A tentative diagnosis may be based on analysis of the CSF.

The WBC count in viral meningitis and encephalitis characteristically shows 10 to 1000 cells/µL. The differential cell count is predominantly lymphocytic and mononuclear in type. In the early stages of meningoencephalitis, however, polymorphonuclear cells may predominate, making the distinction between viral and bacterial infections difficult. In such cases, a repeat tap in 12 to 24 hours will assist in

clarifying the diagnosis. Protein levels are usually mildly elevated, but normal levels may be seen. Antibiotic coverage pending culture results may be reasonably initiated if the diagnosis of viral meningitis is in doubt. [49] The CSF glucose is characteristically normal; however, notable exceptions include some cases of mumps meningoencephalitis and herpes simplex encephalitis. CSF pleocytosis and elevated protein levels have also been found in asymptomatic HIV-seropositive individuals.[18]

If the CSF cannot be delivered to the viral laboratory in 24 to 48 hours, it should be refrigerated at 4°C. Members of the enterovirus group are occasionally isolated from CSF. Herpes viruses and arboviruses are rarely found in CSF. In known viral CNS disease, the stool is more rewarding (85% positive) than CSF (10% positive).[108] Because CSF is normally sterile, any isolate is significant, whereas a stool isolate does not necessarily indicate that the agent is responsible for CNS disease.[117] The PCR is the diagnostic test of choice for herpes simplex meningoencephalitis and PCR will be increasingly applied to the diagnosis of other CNS viral infections.

CSF Analysis in Immunocompromised Patients

The number of immunocompromised individuals is increasing because of the HIV epidemic and the increased survival of patients with cancer and autoimmune disorders. The nervous system is a major target of the HIV virus, with 40 to 60% of infected individuals developing neurologic disease during their lifetime. One third of HIV infected patients will present with neurologic complaints as the initial manifestations of the acquired immune deficiency syndrome (AIDS), and an even higher incidence of nervous system involvement is found at autopsy. [76] Risk of CNS infection depends on the underlying disease, treatment, duration, and type of immune abnormality. Abnormalities include defects in T-lymphocyte and macrophage cellular immune function, defects in humoral immunity, defects in number and function of neutrophils, and loss of splenic function with inability to remove encapsulated bacteria. [109] A summary of the major neurologic manifestations of HIV infection including their clinical characteristics is presented in Table 62-6 .

Patients with defects of their cell-mediated immunity include those with lymphoma, organ transplant recipients, daily corticosteroid therapy, and patients with AIDS. These individuals are vulnerable to infections with microorganisms that are intracellular parasites. The most common source of acute bacterial meningitis in such patients is *L. monocytogenes*. The clinical presentation includes fever, headache, seizures, focal neurologic deficits, and a brainstem encephalitis.

Patients with defective humoral immunity include those with chronic lymphocytic leukemia, multiple myeloma, and Hodgkin's disease after radiotherapy or chemotherapy. These patients have difficulty controlling infection by encapsulated bacteria. They may develop a fulminant meningitis due to *S. pneumoniae, H. influenzae* type b, and *N. meningitidis*. Patients after splenectomy are at risk for the development of meningitis for the same reason. Neutropenic patients are at risk for meningitis due to *P. aeruginosa* and the Enterobacteriaceae.

Establishing a specific diagnosis in HIV and organ transplant patients may be difficult to impossible because of overlapping clinical and radiographic presentations, the presence of simultaneous infections with more than one organism, and CSF changes that may be nonspecific. The immune response may be altered with the absence of signs of meningeal irritation; patients may present with a diffuse encephalopathy or with focal neurologic deficits. CSF is abnormal in 60% of asymptomatic HIV-infected individuals, complicating CSF/clinical correlation. [76]

Neurosyphilis in Patients with HIV Infection

Both syphilis and HIV infection are sexually transmitted diseases, and patients with syphilis are at increased risk of HIV infection. CNS invasion is probably no more common in HIV-infected and HIV-noninfected patients.[81] Both diseases may have elevations in CSF WBC counts, protein levels, and ?-globulin levels. Early CNS infection may be more common in patients with HIV infection. Reports also have noted an increased incidence of syphilitic meningitis, meningovascular syphilis, and ocular syphilis, which has been uncommon in the past. Studies suggest HIV patients treated for syphilis may have viable organisms in CSF after therapy or have persistent CSF VDRL titers. Treatment failures are more likely to occur with single-dose benzathine penicillin therapy.

Diagnosis of neurosyphilis may also be more difficult in HIV-infected patients. A small number of patients with secondary or ocular syphilis have been described with negative serum reagin tests. Positive treponemal tests may revert to nonreactivity after treatment, particularly in individuals with advanced symptomatic HIV disease and a low VDRL titer at the time of diagnosis. CSF pleocytosis and increased ?-globulin levels may not help distinguish between HIV infection and CNS syphilis.

HIV-infected patients should have serum treponemal and nontreponemal tests early in their illness to minimize a false-negative result. [81] Infected patients should undergo a CSF examination. Previously treated patients without a CSF examination at the time of initial syphilis treatment should have a lumbar puncture because of a probable increased risk of neurosyphilis even with a decline in serum nontreponemal titers. Asymptomatic patients with neurosyphilis should be treated to prevent CNS relapse.

Cryptococcal Meningitis

The most common CNS fungal infection is due to *Cryptococcus neoformans*. Approximately 5% of AIDS patients will develop infection with this common fungus. Clinical presentation in AIDS and non-AIDS patients include nonspecific symptoms of headache and altered mental status with or without meningeal signs. Most patients have increased ICP. Immunocompetent patients show a lymphocytic pleocytosis with CSF WBC counts under 500/µL. Glucose levels are depressed with elevation of CSF protein. India ink preparations are positive in 50% of cases. The CSF is less likely to have abnormal cell counts and chemistries in HIV infection. However, CSF cultures and cryptococcal polysaccharide capsular antigens are almost always positive. False-positive antigen tests are rare but may be seen in the presence of rheumatoid factor. Blood cultures are frequently positive in AIDS patients. Cisternal puncture for fluid analysis may be helpful in undiagnosed cases of lymphocytic meningitis in which multiple lumbar punctures have not established a diagnosis. [76]

Toxoplasmosis

Toxoplasma gondii, an intracellular protozoan, is associated with CNS infection in up to 30% of AIDS patients who have

TABLE 62-6 -- Major Neurologic Manifestations of HIV Infection

Diagnosis	CD4+ Count (cells/mm^3)	Presenting Symptoms	Neurologic Signs	Diagnostic Studies	Therapy
HIV dementia	<200	Memory loss, gait disorder, behavioral change	Dementia, spasticity, psychosis	CT/MRI: brain atrophy, white matter abnormalities; CSF: increased $ß_2$ microglobulin	High-dose zidovudine, clinical trial
Toxoplasma encephalitis	<200	Headache, fever, confusion, lethargy, seizures	Dementia, ataxia, hemiparesis	Serum *Toxoplasma* antibodies; CT/MRI: multiple enhancing lesions, edema	Pyrimethamine, sulfadiazine, clindamycin
CNS lymphoma	<100	Headache, confusion, lethargy, memory loss, seizures	Dementia, hemiparesis, aphasia	CT/MRI: enhancing lesions (especially if single); stereotactic biopsy	Radiotherapy
Progressive multifocal leukoencephalopathy	<100	Lethargy, confusion, weakness	Hemiparesis, ataxia, visual disturbance	CT/MRI: multiple hypodense, nonenhancing white matter lesions, stereotactic biopsy	High-dose zidovudine, Ara-C (IV, IT); clinical trial
CMV encephalitis	<50	Rapidly progressive confusion, apathy, weakness	Dementia, cranial neuropathies, spasticity	CT/MRI: periventricular and meningeal abnormalities; CSF: CMV culture, PCR; electrolyte abnormalities	Ganciclovir, foscarnet
Vacuolar myelopathy	<200	Gait dysfunction, lower extremity weakness and stiffness, urinary dysfunction	Spastic paraparesis, Babinski signs, sensory abnormalities	MRI/CSF: normal or nonspecific abnormalities	Baclofen, physical therapy
Cryptococcus neoformans meningitis	<200	Fever, headache, neck stiffness, memory loss	Lethargy, confusion, meningeal signs, cranial nerve palsies	CSF India ink, serum and CSF *Cryptococcus neoformans* antigen and culture	Amphotericin B (plus or minus flucytosine), fluconazole
Neurosyphilis	Any	Headache, memory loss, visual disturbances	Dementia, stroke, meningeal or myelopathic signs, cranial nerve palsies	CSF: increased leukocyte count, increased protein, serum and CSF VDRL	IV penicillin (plus probenecid)
Distal symmetrical polyneuropathy	<200	Distal numbness, parasthesias, pain	Stocking-glove sensory loss, decreased ankle reflexes	EMG: distal axonopathy	Neurotoxin withdrawal, analgesics, tricyclic antidepressants, anticonvulsants, capsaicin
Inflammatory demyelinating polyneuropathy	>500, =50	Progressive weakness, paresthesias	Weakness, areflexia, mild sensory loss	CSF: increased leukocyte count, elevated protein; EMG: demyelination	Early (increased CD4 count): plasmapheresis, IV Ig, steroids. Late (decreased CD4 count): ganciclovir
Manoneuropathy multiplex	>500, =50	Facial weakness, footdrop, wristdrop	Multifocal cranial and peripheral neuropathies	EMG: multifocal axonal neuropathy; nerve biopsy: inflammation, vasculitis, CMV inclusions	Early: none Late: ganciclovir
Progressive polyradiculopathy	<50	Lower extremity weakness, paresthesias, urinary dysfunction	Flaccid paraparesis, saddle anesthesia, decreased reflexes, urinary retention	CSF: increased leukocytes (PMNs), CMV culture, PCR; EMG: polyradiculopathy	Ganciclovir, foscarnet
Myopathy	Any	Muscle weakness, myalgia, weight loss	Proximal muscle weakness	Increased creatine kinase; EMG: irritative myopathy; muscle biopsy: myofiber degeneration, inflammation, inclusions	Zidovudine reduction or withdrawal, corticosteroids

CMV, cytomegalovirus; CNS, central nervous system; CSF, cerebrospinal fluid; CT, computed tomography; EMG, electromyography; IT, intrathecal; IV, intravenous; IV Ig, intravenous immunoglobulin; MRI, magnetic resonance imaging; PCR, polymerase chain reaction; PMN, polymorphonuclear leukocytes.

From Simpson DM, Tagliati M: Neurologic manifestations of HIV infection. Ann Intern Med 121:770, 1994.

antibodies to *T. gondii*. Most adults have antibodies; infection is believed to represent reactivation of latent primary infection. *Toxoplasma* encephalitis usually develops within the first 2 years after the diagnosis of AIDS. Cerebral toxoplasmosis usually presents with the acute/subacute appearance of focal disease including seizures. MRI or CT scanning often reveals multiple abscesses that may represent multiple pathogens. The distinction between toxoplasmosis and lymphoma may be difficult clinically. Less commonly, the presentation is of a chronic meningitis with confusion, memory loss, and lethargy similar to the AIDS-dementia complex. CSF in these patients is nonspecific with increased protein, mononuclear pleocytosis (<100 cells/µL) and, rarely, reduced glucose concentration. Serum and CSF serologies may be either positive or negative and do not help make a diagnosis, although most patients with encephalitis have detectable IgG antibodies. Diagnosis is usually made based on clinical and imaging responses to antibiotics (pyrimethamine or sulfadiazine) or brain biopsy. Treatment failures with relapse occur in 50% of AIDS patients and 15% to 25% of non-AIDS patients, necessitating lifelong treatment.

Mycobacterial Tuberculosis

CNS mycobacteria infection is almost always the result of infection with *Mycobacterium tuberculosis*. Infection occurs in the setting of disseminated tuberculosis. Atypical *Mycobacterium* infection occurring in HIV-infected individuals uncommonly affects the CNS. Clinical presentation is that of meningitis (particularly involving the basal cistern), encephalitis, or abscess formation. If tuberculosis is suspected, a large volume of CSF (10 mL) is required for adequate culture. The cell count varies from 100 to 400 cells/µL, with a lymphocytic predominance; 30% may show predominantly neutrophils early in the course of infection. Protein levels are elevated (100–500 mg/dL); CSF glucose level may be depressed. Acid-fast stains should be examined by experienced technicians. Fluid is inoculated onto Löwenstein-Jensen medium, and the absence of visible growth on the medium should not be considered negative for 8 weeks. CSF cultures are more sensitive than stains. Many of the current tests for mycobacteria are likely to be replaced by PCR tests. [109]

Primary CNS Lymphoma

The risk of developing CNS lymphoma in an AIDS patient is 2%. Presentation is that of a diffuse encephalopathy, although occasionally focal neurologic deficits occur. Leptomeningeal spread of malignancy is reflected in a modest lymphocytic pleocytosis with slightly elevated protein and decreased glucose. Cytologic yield is

improved by repeat lumbar punctures and submitting large quantities of CSF or sampling fluid by cisternal puncture. [76]

Progressive Multifocal Leukoencephalopathy

Progressive multifocal leukoencephalopathy is an uncommon disorder in individuals with impaired cell-mediated immunity and is caused by reactivation of the JC papovavirus in the kidney. Progressive demyelination presents as combinations of dementia, blindness, aphasia, hemiparesis, and seizures that progress until death. MRI and CT demonstrate nonenhancing white matter lesions without mass effect. Definitive diagnosis is made by brain biopsy, but CSF may show the presence of myelin basic protein, increased IgG, with acellular or a mild CSF pleocytosis (<50 WBCs/µL). Average survival is 4 months. [109]

Brain Abscess

Brain abscesses also occur in the immune-deficient host. Diverse causes have been described as well as multiple pathogens. Lesions may be difficult to detect by CT with contrast enhancement, particularly if a patient is undergoing corticosteroid therapy and has a diminished host inflammatory response. Organisms reported include *T. gondii, Nocardia asteroides, Cryptococcus neoformans, Mycobacterium* species, aerobic gram-negative bacteria, *Aspergillus* species, *Candida* species, and Zygomycetes.

Cytomegalovirus Infection

Cytomegalovirus may be detected in 30% of brains of HIV-infected persons who come to autopsy. A distinct CNS disorder has not been defined. CSF pleocytosis may be minimal. Retinitis and painful polyradiculopathies are recognized with a prominent CSF pleocytosis in the later condition. [76]

CONCLUSION

A spinal tap should only be performed when the treating clinician believes the CSF specimen or specimens will be of diagnostic or therapeutic value, as in the case of patients with symptomatic pseudotumor cerebri. The procedure is often indicated in the diagnosis of meningitis or subarachnoid hemorrhage. Complications are uncommon, and risks are usually outweighed by the benefit of the procedure. Most contraindications are relative and not absolute, particularly if infection is an overriding consideration.

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Chapter 63 - Special Neurologic Tests and Procedures

J. Stephen Huff

Neuro-otologic tests and procedures are used in a variety of clinical scenarios ranging from evaluation of the dizzy patient to diagnosing brain death. Many of these have been replaced by neuroimaging or other tests but there is still utility in bedside testing in selected patients. In addition to caloric testing, the Dix-Hallpike maneuver for diagnosing benign paroxysmal positional vertigo is described and techniques of canalith repositioning are summarized. A new discussion of brain death aimed at the emergency clinician follows, and a brief synopsis of emergency department (ED) testing for myasthenia gravis completes the section.

CALORIC TESTING

An accurate assessment of the comatose patient requires a thorough neurologic examination including careful evaluation of the patient's responses to a variety of external stimuli. In a comatose individual with normal brainstem and cranial nerve function, stimulation of the vestibular labyrinth results in well-described and reproducible extraocular movements. This response is known as *the vestibulo-ocular reflex* and forms the physiologic basis for caloric testing. Caloric testing involves delivering a thermal stimulus to the external auditory canal to activate the labyrinth. Pathologic conditions involving either the vestibular or oculomotor reflex pathways will alter or abolish the usual response to caloric stimulation.

Caloric tests may be performed in both conscious and unconscious patients, depending on the diagnostic circumstances. Quantitative caloric examination is conducted in the awake ambulatory patient for evaluation of possible vestibular dysfunction. This type of testing requires precisely controlled irrigation temperatures and specialized recording devices and is best undertaken in a properly equipped laboratory under the supervision of an experienced neuro-otologist. However, the neurologist, neurosurgeon, or emergency clinician may perform *qualitative* caloric testing in the comatose patient to detect gross disruption of vestibulo-ocular reflex pathways that may indicate structural lesions or metabolic abnormalities affecting the labyrinth, vestibulocochlear nerve, or brainstem. In this clinical setting, ice water is used to provide maximal stimulation of the vestibular system. Such testing needs no special expertise and can be done at the bedside using equipment readily available in the ED. This simple procedure can provide valuable diagnostic and prognostic information necessary for management of the comatose patient.

Background

Brown-Séquard first described the effects of introducing cold water into the ear canal in the mid-19th century. [11] The clinical importance of the phenomenon was first realized in 1906 by Bárány, [3] who developed a caloric procedure using an ice water stimulus. He postulated, correctly, that caloric stimulation of the auditory canal induced the formation of convection currents within the semicircular canals of the vestibular labyrinth.

Standardization of the procedure awaited the introduction of the Fitzgerald-Hallpike technique in 1942. This technique used both warm and cool water stimuli under rigidly specified conditions permitting quantification of normal and abnormal caloric responses. Today, most formal caloric testing of conscious patients is based on variations of the original Fitzgerald-Hallpike procedure. [27]

The value of caloric testing in the assessment of the comatose patient was emphasized by the work of Klingon [49] and Bender and associates.[5] Vaernet[91] and Ethelberg[23] studied the changes in caloric reactions during transtentorial herniation of the brainstem. Blegvad [7] reported the effects of barbiturate intoxication on the vestibulo-ocular reflex.

More recent advances include the development of electronystagmography (ENG), which provides a graphic record of reflex eye movements and permits precise determination of the intensity of the caloric response. [4] Some authorities now advocate performing caloric testing at a single temperature (monothermal calorics) as an equally sensitive and less time-consuming alternative to standard bithermal calorics. [44] Others have investigated the use of heated air as an alternative to traditional water caloric tests.[98]

Physiology and Functional Anatomy

Proper performance and interpretation of the caloric test require a basic understanding of the structure and function of both the vestibular and oculomotor systems. The anatomic pathways underlying the vestibulo-ocular reflex begin in the posterior portion of the labyrinth of the inner ear. The peripheral vestibular apparatus is located within the temporal bone and consists of the utricle, the saccule, and the lateral, anterior, and posterior semicircular canals. Because of its proximity to the external ear canal, the lateral or horizontal canal is of principal interest in caloric testing. The lateral canal is oriented at a 30° angle to the horizontal. Deflections of the cupula due to movement of endolymphatic fluid within the canal result in polarization changes in the underlying hair cell, which in turn are relayed to the afferent limb of the primary vestibular neuron.

Impulses of the primary neuron travel via Scarpa's ganglion and cranial nerve (CN) VIII to the brainstem to synapse with secondary vestibular neurons in the superior and medial vestibular nuclei of the upper medulla and lower pons (Fig. 63-1). Although the connections between the vestibular and oculomotor nuclei in the brainstem are quite complex, two main pathways exist. The direct projection runs from the vestibular complex to the nuclei of CN III and VI via the medial longitudinal fasciculus (MLF) and involves only three neurons: (1) primary vestibular, (2) secondary vestibular, and (3) oculomotor. [89] The indirect projection between these nuclei occurs over multisynaptic circuits in the tegmental reticular formation. [64] Another brainstem structure that contributes to the vestibulo-ocular interaction is the parapontine reticular formation (PPRF), a poorly characterized group of pontine neurons that coordinates both voluntary and involuntary lateral gaze. The PPRF receives multiple inputs, including projections from the vestibular system and the contralateral frontal cortex, and sends output to oculomotor neurons through both

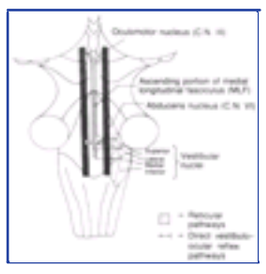

Figure 63-1 Schematic drawing of the brainstem, showing the major elements of the vestibulo-ocular reflex (VOR) pathway. The *solid line* indicates the direct projection between the vestibular nuclei and the third and sixth nuclei. The *stippled area* represents the indirect projections between the nuclei. *(Modified from Barr ML, Kiernan JA: The Human Nervous System—An Anatomical Viewpoint, 5th ed. Philadelphia, JB Lippincott, 1988, p 330.)*

the direct and indirect pathways. Excitatory impulses originating in the lateral canal finally travel via the oculomotor and abducens nerves to the ipsilateral medial rectus and contralateral lateral rectus muscles.

Rotation of the head generates flow of endolymphatic fluid within the semicircular canals. The firing rate of the primary vestibular neuron is dependent on the direction of flow. For example, in the lateral canal, flow toward the ampulla (ampullopetal) increases the firing rate, whereas flow away from the ampulla (ampullofugal) decreases the firing rate.[2] Increased firing on one side results in conjugate deviation of the eyes toward the opposite side, whereas decreased firing causes deviation to the same side. This principle forms the physiologic basis of caloric testing. When the lateral canal is placed in the vertical position and ice water is infused into the ear, the endolymph nearest the canal cools and sinks, resulting in ampullofugal flow (Fig. 63-2 (Figure Not Available)). As the firing rate decreases, the eyes deviate conjugately toward the side of irrigation. If warm water were used in the same position or if the canal were inverted 180° the opposite would occur.

Eye movements induced by caloric stimulation in conscious, neurologically normal individuals are more complex. Ice water infusions induce a rhythmic jerking of the eyes that includes a slow deviation toward the irrigated side followed by a quick compensatory saccade toward the midline. This is known as *caloric nystagmus*. By convention, *caloric nystagmus is named for the fast component,* thus the popular mnemonic "fast COWS" (*C*old irrigation—*O*pposite beating nystagmus; *W*arm irrigation—*S*ame-sided beating nystagmus). Most sources attribute the slow phase of nystagmus to vestibular activity transmitted over the direct pathway, whereas the fast phase is believed to be generated by the PPRF in conjunction with cortical activity and carried over indirect pathways within the reticular formation. Numerous factors, including physiologic, pharmacologic, and pathologic factors, can alter caloric-induced eye movements.

Indications and Contraindications

Caloric testing of the comatose patient is indicated when the clinician needs information regarding the functional integrity of the brainstem. When the cause of the coma is initially unknown, qualitative caloric testing at the bedside may assist in differentiation among structural, metabolic, and psychogenic causes for unresponsiveness. Even when the cause is clear, caloric testing may provide an indication of the depth of coma and the prognosis for eventual recovery.

In conscious patients who complain of vertigo, quantitative caloric testing may be indicated for the nonemergency evaluation of possible vestibular disorders. Ice water produces maximal stimulation of the labyrinth. This can be quite uncomfortable and may induce nausea and vomiting in awake, susceptible individuals. These patients are best referred to a qualified neuro-otologist or audiologist, who may conduct more accurate testing in the ENG laboratory with less discomfort to the patient.

There are few contraindications to caloric testing in the unresponsive patient. An absolute contraindication is the presence of a basilar skull fracture, either documented radiologically or suspected by clinical signs, because of the risk of introducing infection into the central nervous system (CNS) through an associated dural tear. If a basilar skull fracture is discovered to be unilateral, testing of the intact ear with warm then cold water will yield results similar to those of the standard bilateral examination with cold water stimulation.

Relative contraindications to water caloric testing include perforations of the tympanic membrane (those not due to temporal fractures), otitis media and externa, and the presence of previous otologic surgery (e.g., mastoidectomy). Although the risk of causing otitis media is probably small, carrying out the caloric test in the comatose patient under these conditions remains a matter of clinical judgment. When available, auditory evoked potentials may provide an alternative source of information when caloric testing is contraindicated.

Equipment

The equipment needed for performance of the caloric test is minimal and is readily available in the ED or hospital ward. Although almost any size syringe will suffice, a 30- or 50-mL plastic syringe is ideal for irrigation. The syringe may be used as is, or a short length of soft plastic tubing may be attached. A good source of tubing is a butterfly catheter with the needle cut or pulled off. At least 100 mL of ice water should be available, although larger quantities of cool tap water (<25°C) can be used with similar results if ice is unavailable. Sterile or bacteriostatic saline may be used, although no advantage over tap water has been demonstrated. A small, curved, plastic emesis basin is useful to collect water as it drains from the ear canal. Additional required equipment includes an otoscope, several sizes of ear speculums, and equipment for removal of cerumen. Towels and a thermometer that reads from 0° to 50°C may be helpful.

Figure 63-2 (Figure Not Available) The effects of ice water irrigation on the lateral semicircular canal. The cooler endolymph sinks, resulting in ampullofugal flow and decreased neural firing of the primary vestibular neuron. Actual temperature changes within the endolymphatic fluid are on the order of 1° to 2°C. *(Modified from Baloh RW, Honrubia V: Clinical Neurophysiology of the Vestibular System. Philadelphia, FA Davis, 1979, p 133.)*

Procedure

Vestibulo-ocular reflex testing should be deferred until the patient's condition has been stabilized with attention to the airway and evaluation of the cervical spine in trauma patients. A thorough neurologic assessment should be performed prior to caloric testing, with special attention given to the ocular examination. Pupillary responses, spontaneous ocular movements, and resting eye position should be accurately recorded. The ears should be inspected before insertion of the otoscope. If active bleeding or CSF otorrhea or rhinorrhea is noted in the trauma victim, caloric testing and further otoscopic examination should be deferred and the patient should be treated for a probable basilar skull fracture. If the external ear canal appears normal, the otoscopic examination should be completed. Signs of active ear infection or perforation of the tympanic membrane are contraindications to caloric testing. Tympanic rupture, hemotympanum, and step deformities of the canal may indicate fracture of the temporal bone; caloric testing in this situation is contraindicated. Excess cerumen and foreign material must be removed, and the tympanic membrane should be clearly visualized. The ear speculum may be left in the canal as a guide for irrigation.

Though the test is commonly performed with the patient supine, ideally the head or upper body should be raised 30° (two pillows will provide the appropriate angle). The elevation places the lateral canal in the vertical position and ensures a maximal response. The patient should be draped with a towel, and a small basin should be positioned below the ear to collect the water outflow. A container should be filled with ice water and placed near the bedside.

The syringe and catheter system (*minus the needle*) should be filled with 10 mL of ice water, and the irrigation stream should be directed at the tympanic membrane. Because the goal of the qualitative caloric test is to induce a maximum response, the amount and rate of infusion are not critical. As a general guide, 5 to 10 mL of ice water should be initially infused over a period of 5 to 10 seconds; amounts less than 5 mL may be advisable in suspected cases of light coma or psychogenic unresponsiveness. If no response is noted within a moment, at least 100 mL should be infused before declaring that there is no response. Testing of the contralateral ear may begin 5 or 10 minutes after the eyes have returned to their original position. At the conclusion of the testing, the otoscopic examination may be repeated to check for blanching of the tympanic membrane, a sign that the irrigation stream was properly directed.

Observation of eye deviation is easier when an assistant holds the patient's eyelids open. Movement usually occurs after a latency of 10 to 40 seconds, with persistence of the response for as long as 4 to 5 minutes. Small deviations may be detected by focusing on a small scleral vessel. Alternatively, a dermographic pencil may be used to mark the initial position of the pupil with respect to the eyelid.

Variations of the caloric technique may be useful in certain situations. Warm water caloric testing may be performed if no response to bilateral ice water caloric testing is obtained or in cases in which only one ear can be tested. Water temperature should be kept below 50°C. The response elicited will be the opposite of that obtained with ice water. In patients who fail to respond to ice water caloric testing alone, additional stimulation may be obtained by combining irrigation with repeated head turning away from the irrigated side *(doll's-eye maneuver). Obviously, cervical injury must be excluded before using this technique*. This combination of techniques may produce eye movements in patients who do not respond to caloric testing alone.[26] Eviatar and Goodhill[24] have described a technique for caloric testing in tympanic perforations using a small latex finger cot placed in the ear canal to prevent water from entering the middle ear.

Complications

The few complications that are possible with caloric testing can be avoided by carefully selecting both the patients to be tested and the equipment and technique to be used. Using needles or other sharp objects to irrigate the ear may result in laceration or perforation of the tympanic membrane or canal wall if the patient moves unexpectedly. The use of plastic syringes and soft catheter tubing aids in reducing such occurrences.

Other potential complications of caloric testing include otitis media, meningitis, and the induction of vomiting and subsequent aspiration. Ice water irrigation in the presence of tympanic membrane perforation increases the chance of middle ear infection; however, the incidence of this complication following caloric testing has not been reported. Meningitis may follow basilar skull fractures with meningeal tears; the additional risk of calorics in such situations is not known. Therefore, caloric testing should be omitted in the head-injured patient if there is any suspicion of temporal fracture. Although ice water irrigation may produce nausea and even emesis in a few awake patients, vomiting with aspiration has not been reported as a complication of caloric testing in the comatose patient. Nevertheless, some operators prefer to delay testing until the patient's airway is protected.

Interpretation

First phase of interpretation.

Analysis of initial eye position and spontaneous movements should be made before irrigation. The eyes of comatose patients with intact oculomotor pathways are usually directed straight ahead or are slightly divergent. Unilateral destructive lesions of the cerebral hemisphere can cause conjugate deviation of the eyes toward the side of the lesion, whereas irritative foci, as might be seen in status epilepticus, can cause conjugate deviation away from the affected side. Deviations of this type can usually be overcome by caloric stimulation, although combined irrigation and head turning may be required in the first hours following the insult. Lesions in or near the PPRF in the brainstem cause conjugate deviation away from the side of the lesion that usually cannot be overcome by calorics. Conjugate downward deviation can be seen with structural lesions of the brainstem or in the deeper phases of metabolic coma. Dysconjugate gaze may indicate damage at the level of the oculomotor nuclei or below or reflect disruption of the ocular muscles themselves. [61] Dysconjugate gaze is also seen in drug-induced coma in the presence of a structurally intact brainstem pathway. In the very late stages of brainstem dysfunction, the eyes usually return to the central position. Spontaneous roving movements of the eyes, either conjugate or dysconjugate, may be seen in supratentorial insults, but these, too, disappear with brainstem involvement. [26] Ocular "bobbing" is an intermittent, spontaneous downward jerking of the eyes that may occur with massive pontine lesions. Paralysis of both voluntary and reflex lateral gaze occurs, and caloric stimulation may increase the rate of bobbing without causing lateral deviation of the eyes. [87] Ocular "dipping" is a more prolonged, downward conjugate deviation of the eyes and has been reported in cases of severe anoxic encephalopathy (e.g., carbon monoxide poisoning). [68] The pathophysiologic basis of these eye movements is poorly understood.[69]

The second phase of interpretation.

After irrigation, the ocular movements should be observed for any response to the stimulus. Again, typically there is a latency of response of 10 to 40 seconds. Reactions to ice water irrigation may be divided into four categories: (1) caloric nystagmus, (2) conjugate deviation, (3) dysconjugate deviation, and (4) absent responses (Fig. 63-3*A, B* (Figure Not Available)). The first reaction, caloric nystagmus with the fast component beating away from the side of ice water irrigation, is seen in normal, alert individuals; in cases of psychogenic unresponsiveness; and in those who have very mild organic disturbances of consciousness. The intensity of nystagmus is highly variable in conscious subjects and depends on the degree of visual fixation and the level of mental alertness. The response is present in more than 90% of children by age 6 months and declines in magnitude only after the seventh decade of life. [12] Corneal reflexes and responses to facial pinprick may be abolished by hypnotic suggestion in susceptible individuals; however, caloric nystagmus appears to be immune to similar manipulation. [13]

Caloric-induced nystagmus following cold water irritation in the apparently comatose individual usually results from testing individuals with psychogenic unresponsiveness due to catatonia, conversion reactions, schizophrenia, or feigned coma. Hyperactive caloric responses may result from testing in the presence of tympanic perforation or mastoid disease. Hypoactive caloric responses are recorded in a wide variety of vestibular and neurologic disorders.

Hypoactive or abnormal caloric responses may be further evaluated with quantitative caloric testing and ENG. Caloric nystagmus may be *inverted* (beating to the wrong side) or *perverted* (beating in the wrong plane); both responses are seen in brainstem lesions. *Pseudocaloric nystagmus* is a pre-existing latent nystagmus that is brought out by the general arousal of ice water irrigation; it can be distinguished from true nystagmus by its failure to reverse direction with warm water irrigation.

As the level of coma deepens, the fast phase of nystagmus becomes intermittent and then disappears, probably as a result of decreased activity in the cerebral cortex.

In the second type of response to cold caloric stimulation, the eyes deviate conjugately toward the side of ice water stimulation (they "look" toward the source of irritation). When present, this reaction indicates intact brainstem function as well as intact afferent and efferent limbs of the reflex. This is seen during general anesthesia, in supratentorial lesions *without brainstem compression*, and in many metabolic and drug-induced comas. In such situations, bilateral simultaneous irrigation with ice water may result in conjugate downward deviation, implying that brainstem centers for vertical gaze are functional.

Dysconjugate reactions constitute the third type of caloric response to ice water stimuli. The most common dysconjugate reaction is internuclear ophthalmoplegia, in which a lesion of the MLF causes weakness or paralysis of the *adducting* eye following caloric irrigation. Internuclear ophthalmoplegia may be due to acute damage to the rostral pons or may be seen as a manifestation of multiple sclerosis or stroke.

In acute supratentorial lesions, the development of dysconjugate caloric responses is a significant sign that may indicate compression of the brainstem and impending herniation. Caloric responses of this type are less common with metabolic and drug-induced coma, and when present in metabolic coma, their significance is less ominous. Reversible internuclear ophthalmoplegia has been reported in hepatic coma and may occur during toxic response to phenytoin, barbiturate, or amitriptyline. Forced downward deviation of the eyes, either conjugate or dysconjugate, may be seen in sedative-hypnotic drug-induced coma when unilateral caloric testing is performed. [81]

Palsies of the oculomotor nerves are another cause of dysconjugate reactions, although most should be apparent before irrigation. Causes include diabetic neuropathy (especially CN VI), increased intracranial pressure, and Wernicke's

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Figure 63-3 (Figure Not Available) *A,* The four types of caloric responses seen with unilateral and bilateral irrigations: *1,* Normal nystagmus; *2,* Conjugate deviation; *3,* Dysconjugate deviation. The most common type, internuclear ophthalmoplegia, is shown here. Vertical eye movements usually remain intact in this lesion; *4,* Absent caloric responses. MLF, medial longitudinal fasciculus. *B,* Oculocephalic and oculovestibular testing in selected clinical conditions. *(A, Modified from Plum F, Posner JB: The Diagnosis of Stupor and Coma, 3rd ed. Philadelphia, FA Davis, 1980, p 55. B, From Smith M, Bleck T: Techniques for evaluating the cause of coma. J Crit Illness 2:51, 1987.)*

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encephalopathy. Finally, Plum and Posner[61] reported that unusual and poorly characterized caloric responses may be obtained from the testing of comatose patients with long-standing severe brain injury.

Absent caloric response is the fourth category of reactions to ice water stimuli. As a general rule, the oculovestibular response is preserved longer than other brainstem reflexes; however, the oculocephalic or doll's eye response may persist in the absence of caloric responses due to bilateral labyrinthine disease because of additional input from proprioceptive receptors in the neck. Loss of caloric responses in comatose patients with structural lesions is usually a sign of brainstem damage. In supratentorial lesions, progressive loss of caloric responses may be seen in the final stages of transtentorial herniation. The oculovestibular reflex may also be transiently absent or decreased on the side opposite massive supratentorial damage during the first hours following injury. [62] Absent caloric responses may occur in any subtentorial lesion that affects vestibular reflex pathways, including pontine hemorrhage, basilar artery occlusion, cerebellar hemorrhage, or infarction with encroachment on the brainstem, and in any expanding mass lesion within the posterior fossa. Calorics may disappear in deep coma resulting from subarachnoid hemorrhage, perhaps owing to pressure on the brainstem.

The vestibulo-ocular reflex is usually retained until the late stages of metabolic coma. Disappearance of the reflex, as in the case of hepatic coma, is frequently a preterminal event.[32] Nevertheless, caloric responses may be transiently absent in certain types of drug-induced coma, with the *eventual complete recovery of the patient*. The vestibulo-ocular reflex seems particularly sensitive to the effects of sedative-hypnotic drugs (e.g., barbiturates, glutethimide), antidepressants (e.g., amitriptyline, doxepin), and anticonvulsants (e.g., phenytoin, carbamazepine). [7] [84] [85] Obviously, neuromuscular blocking agents (e.g., succinylcholine) will abolish caloric-induced ocular movements.

Finally, the caloric response may be absent for reasons other than the neurologic causes responsible for the coma. Inadequate irrigation owing to excess cerumen or poor technique and unilateral or bilateral dysfunction of the peripheral vestibular apparatus must be considered. Bilateral loss of caloric response (areflexia vestibularis) is uncommon in conscious patients, constituting 1.7% and 0.2%, respectively, of the ENG clinical population in two large series of patients. [85] [86] Some of the causes of unilateral and bilateral loss of oculovestibular reflexes in conscious patients are listed in Table 63-1 .

The vestibulo-ocular reflex has prognostic as well as diagnostic significance in the comatose patient. In a study of 100 patients who were comatose from head trauma, absence of calorics at 1 to 3 days after injury was associated with extremely high mortality. [63] Testing in the immediate posttraumatic period may yield inconsistent responses and is of considerably less prognostic value. Levy and coworkers studied 500 cases of nontraumatic, non-drug-induced coma in a large multicenter effort. Absence of the vestibulo-ocular reflex correlated with less than a 5% chance of achieving functional recovery within 1 year when tested within 6 to 24 hours of coma onset. [43] In one study of comatose patients, the combination of absent vestibulo-ocular reflex and absent pupillary light reflex at 24 hours was associated with 100% mortality. [51] Complete loss of caloric responses is part of the

TABLE 63-1 -- Possible Causes of Absent Vestibulo-ocular Reflex in Conscious Patients

Inadequate Irrigation
Cerumen Impaction
Postinfection
Meningitis
Encephalitis
Syphilis
Neoplastic
Acoustic neuroma
Other cerebellopontine-angle tumors
Posterior fossa tumors
Inflammatory
Systemic lupus erythematosus
Cogan's syndrome
Traumatic
Previous temporal fracture
Previous head injury
Postlabrinthectomy
Labyrinthine
Vestibular neuronitis
Suppurative labyrinthitis
Congenital
Congenital hydrocephalus
Hereditary spinocerebellar degeneration
Idiopathic
Drugs
Aminoglycoside antibiotics
Neuromuscular blocking agents
Anticonvulsants*

*Reported rarely in conscious patients who have taken more than normal therapeutic dosage.

criteria for the diagnosis of brain death and correlates with the irreversible cessation of cerebral function at least as well as an isoelectric electroencephalogram (EEG). [34] Excessive reliance on a single clinical sign must be avoided in the consideration of brain death, and decisions regarding neurologic prognosis and future therapy should be based on complete consideration of all evidence available. The topic clinical diagnosis of brain death is discussed in detail subsequently.

Summary

Caloric testing is a simple, easily performed bedside procedure that may enhance the neurologic assessment of the comatose patient. When reliably interpreted, caloric testing may furnish valuable diagnostic and prognostic information. Even if the cause of the coma is known, the test may provide a baseline for the evaluation of changes in the patient's status. In the emergency patient, this test should be reserved for the stable patient undergoing secondary assessment. The examination requires minimal equipment and can be conducted in a few minutes while awaiting laboratory results or during preparation for computed tomographic scanning. Complications are few if patients are properly selected and correct technique is used.

DIX-HALLPIKE TEST IN DIAGNOSIS OF POSITIONAL VERTIGO

Vertigo occurring only and repeatedly with position change is probably benign paroxysmal positional vertigo (BPPV); the head-hanging positioning maneuver (Dix-Hallpike test, sometimes referred to as the Nylèn-Bárány maneuver) is useful in confirming the clinical suspicion of BPPV since the provoked abnormal nystagmus is characteristic of the disorder. [18] [28] Evolving pathophysiologic theory is that calcium crystal material displaced from the vestibule floats within the endolymph of the posterior semicircular canal and head movement induces bidirectional forces in the fluid acting on the cupula, triggering the BPPV attack. [9] [10] [21] [59] [73] The

posterior semicircular canal is most commonly affected, [28] [59] but at times the horizontal canal is thought to be involved, leading to variants of typical BPPV. [15] [49] [93]

Background

BPPV is a common mechanical disorder of the inner ear in which vertigo is precipitated by certain head movements; nystagmus and autonomic symptoms such as nausea commonly accompany the vertigo. Syndromes of positional vertigo and provocative maneuvers have been described by clinicians for more than 100 years; the description of BPPV has been attributed to Adler, Bárány, Nylèn, Bruns, Borries, Dix, and Hallpike. [42] Current consensus is that Dix and Hallpike most fully defined the disorder and that the provocative technique they described is superior and most accurately should bear the eponymic Dix-Hallpike positioning test or the Dix-Hallpike test. [28] [42]

Indications and Contraindications

The Dix-Hallpike test may be a useful diagnostic test in confirming BPPV by provoking a specific type of nystagmus and in selecting patients suitable for positional therapy (discussed later). The maneuver should not be performed in patients with severe cervical spine disease, unstable spinal injury, high-grade carotid stenosis, or unstable heart disease. [25] Patient discomfort or physical infirmity are relative contraindications; some elderly patients or patients with ongoing nausea or vertigo may not tolerate the body position changes necessary for performance of the procedure. If nystagmus is present at rest without any provocation or if associated neurologic signs or symptoms exist, the diagnosis of BPPV is likely excluded and the maneuver is not clinically indicated.

Procedure

The Dix-Hallpike maneuver is illustrated in Figure 63-4 (Figure Not Available) . The seated patient, whose head is turned 45° to one side, is quickly laid back to a head-hanging position and the eyes are observed for any induced nystagmus. [28]

Interpretation

The Dix-Hallpike head-hanging positioning maneuver produces vertigo only in patients with positional vertigo that is most commonly characterized as BPPV. The stereotypic positive response in BPPV is provoked vertigo developing after a brief delay (1 to 10 seconds), lasting less than a minute, and with direction-fixed rotary or vertical nystagmus. Nausea or other systemic symptoms are often present. [18] [28] The eye movements are mixed torsional and vertical nystagmus of both eyes with the upper pole of the eye beating toward the dependent ear and the vertical nystagmus beating toward the forehead. The side of the ear in the downward position during the Dix-Hallpike test that elicits the greater nystagmus likely identifies the affected ear. After the patient is returned to the sitting position, the nystagmus may again be transiently observed in a reversed direction. If the positional nystagmus is atypical or if the maneuver fails to elicit nystagmus in a patient with ongoing symptoms of vertigo, another diagnostic possibility should be considered. [25] [28]

Complications

The maneuver may precipitate brief vertigo and nausea in patients with BPPV.

Summary

The Dix-Hallpike test may be useful in confirming the diagnosis of BPPV in patients when there is doubt and in localizing the abnormality to one ear. Accurate identification of BPPV is of interest because of the possibility of interventional therapies as described subsequently.

CANALITH-REPOSITIONING MANEUVERS

If the clinical evaluation of the patient with vertigo is consistent with the diagnosis of BPPV and the Dix-Hallpike test is supportive of the diagnosis and lateralizes to one ear, the patient may be a candidate for attempted canalith repositioning maneuvers. These techniques have largely been described in the otologic and neurologic literature and have not been studied in the emergency patient population or as performed by emergency clinicians. Success rates of 44% to 100% are reported. [28] The success in the ED population as performed by emergency clinicians is unknown.

Background

With theory suggesting that stray material in the posterior semicircular canal is causative of the symptoms of BPPV, maneuvers were designed using sequential head movements to reposition the debris to the vestibule. [8] [21] [28] [75] The manipulation of head position theoretically allows the debris ("canaliths") to sequentially fall from the problematic location in the semicircular canal to the vestibule of the labyrinth where they presumably adhere. The currently recommended maneuver was introduced by Epley. [21] Other maneuvers are described but require repeated trials or are more difficult to perform and perhaps more uncomfortable for the patient. [8] [28] [41] [75] The Semont maneuver will also be described because it may be performed at the bedside. In theory, the Semont maneuver would be effective primarily in patients in whom the displaced canaliths were adhering to the cupola of the posterior semicircular canal (Schuknecht cupolith theory) though it might be effective as well when the debris are free floating in the long arm of the semicircular canal; the Epley maneuver would be effective only in patients with debris floating in the long arm of the posterior semicircular canal (canalith theory). [33] In one small trial comparing the efficacy of either the Semont or Epley maneuvers in a carefully selected outpatient otolaryngology population, the maneuvers were found to be of roughly the same effectiveness (90%) in relieving or improving symptoms of BPPV. [33]

Indications and Contraindications

The indication for any canal-clearing or "liberatory" procedure is the clinical diagnosis of BPPV as confirmed by history and physical examination including the Dix-Hallpike maneuver. Careful patient selection is likely a key point in success of any of these maneuvers as well as correct identification of the impaired ear, which will govern the initial position and

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Figure 63-4 (Figure Not Available) The Dix-Hallpike test of a patient with benign paroxysmal positional vertigo (BPPV) affecting the right ear. *A,* The examiner stands at the patient's head 45° to the right to align the right posterior semicircular canal with the sagittal plane of the body. *B,* The examiner moves the patient, whose eyes are open, from the seated to the supine right ear-down position and then extends the patient's neck slightly so that the chin is pointed slightly upward. The latency, duration, and direction of the nystagmus, if present, and the latency and duration of vertigo, if present, should be noted. The arrows over the eyes in the insert depict the direction of nystagmus in patients with typical BPPV. The presumed location in the labyrinth of the free-floating debris thought to cause the disorder is also shown. *(From Furman C: Benign paroxysmal positional vertigo. New Engl J Med 341:1590, 1999.)*

movement of the patient. Contraindications are the same as for the Dix-Hallpike maneuver as noted earlier.

Procedure

The Epley procedure has several variations. Please refer to Figure 63-5 (Figure Not Available) and the caption for details. Briefly, with the patient sitting, the head is turned 45° toward the affected side; the patient is then moved to the head-hanging orientation as in the Dix-Hallpike procedure. After 20 seconds or after any symptoms subside, the patient's head is then rotated to face the opposite shoulder remaining in a head-hanging orientation. Then the patient is rolled further onto his or her side while the head is rotated further into a face-down position. Again, after 20 seconds or after any symptoms subside, the patient is returned to a seated position. [10] [25] [41] Some authors suggest keeping the patient's head in each position long enough for any provoked symptoms of nystagmus or vertigo to resolve. [28] Others suggest a period of 4 minutes after the head is placed in the hanging position and again after rotation of the head

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Figure 63-5 (Figure Not Available) Bedside maneuver for the treatment of a patient with benign paroxysmal positional vertigo affecting the right ear. The presumed position of the debris within the labyrinth during the maneuver is shown in each panel. The maneuver is a three-step procedure. First, a Dix-Hallpike test is performed with the patient's head rotated 45° toward the right ear and the neck slightly extended with the chin pointed slightly upward. This position results in the patient's head hanging to the right *(A)*. Once the vertigo and nystagmus provoked by the Dix-Hallpike test cease, the patient's head is rotated about the rostral-caudal body axis until the left ear is down *(B)*. Then the head and body are further rotated until the head is face down *(C)*. The vertex of the head is kept tilted downward throughout rotation. The maneuver usually provokes brief vertigo. The patient should be kept in the final, face-down position for about 10 to 15 seconds. With the head kept turned toward the left shoulder, the patient is brought into the seated position *(D)*. Once the patient is upright, the head is tilted so that the chin is pointed slightly downward. *(From Furman C: Benign paroxysmal positional vertigo. New Engl J Med 341:1590, 1999.)*

("modified Epley maneuver"). [33] The maneuver may be repeated after the first sequence to determine if positional symptoms have subsided. The maneuver may be repeated a few times until some improvement in symptoms occurs though this is not typically described in the literature. [33] Usually it is recommended after a successful procedure that the patient remain in a head-upright position for 24 hours.

The Semont maneuver involves larger and more abrupt body movements. The affected ear is identified by the Dix-Hallpike maneuver. With the patient seated on the side of an examination table or bed, the head is turned toward the unaffected side. The patient is then quickly moved to a sidelying position (Fig. 63-6 (Figure Not Available)) and kept there until symptoms subside. The patient is then moved abruptly through the sitting position to the opposite sidelying position and kept there until symptoms subside. He or she is then returned to the upright position. [33] [41] Again, typically it is recommended after a successful procedure that the patient remain in a head-upright position for 24 hours.

Complications

Exacerbation of vertigo occurs occasionally and is thought to result from displacement or the dislodgment of the canal debris. Repeating the procedure is recommended for relief. Similar symptoms may occur in up to 50% of patients with up to 20% in the first 2 weeks. [10]

Summary

In most patients with correctly identified BPPV, canalith-repositioning techniques may be useful and may bring immediate relief or improvement of symptoms. Experience in an emergency patient population with treatment by emergency clinicians has not been reported, though with careful patient selection some success should be achieved.

BRAIN DEATH TESTING

In a textbook of emergency medicine from 1988, the author wrote that because emergency medicine was a life-supporting-moriented specialty, the determination of brain death was outside

Figure 63-6 (Figure Not Available) Semont's maneuver. The patient is moved quickly into the position that provokes the vertigo and remains in that position for 4 minutes. The patient is then turned rapidly to the opposite side, ear down, and remains in this second position for 4 minutes before slowly sitting up (for detailed description of the technique, see text). The insets represent the right labyrinth as it would be viewed from the front of the patient. The orientation of the labyrinth is shown for when the patient is sitting *(A),* lying on the affected side *(B),* and lying on the opposite side *(C)*. The solid black arrow indicates the location of the relative position of free-floating debris with the canal during the different stages of this maneuver. *(From Herdman SJ, et al: Single treatment approaches to benign paroxysmal positional vertigo. Arch Otolaryngol Head Neck Surg 119:450, 1993.)*

the practice of the emergency clinician.[35] The date of publication reflects practice at that time; neuroimaging was not often performed while patients were in the ED and delays for inpatient bed availability were unusual. With increased use of neuroimaging in the ED and general trends to increased ED length of stay, the occasion may arise for emergency clinicians to be involved in assessment of brain death. The duty to identify potential organ and tissue donors is another consideration in identifying patients with irreversible loss of brain function. Local practices, policies, regulations, and laws differ and these remarks are general; the clinician is urged to be familiar with local practices and administrative policies regarding the issue of brain death.

For purposes of this discussion, brain death is defined as irreversible loss of functioning of the cerebral hemispheres and brainstem consistent with definitions in the literature.[1] [6] [16] [94] [95] The cause of the brain injury should be known or identified since some toxicologic syndromes, notably barbiturate toxicity, exactly duplicate the clinical syndrome of brain death. In keeping with the theme of this book, this chapter focuses on clinical procedures commonly used to delineate brain death. Radiologic and other tests will be mentioned only in passing. This chapter is not a historical summary of the complex legal and ethical issues surrounding this topic or a guide to approaching the family of a severely injured or impaired patient. Frequently in current practice, if a cause for coma or neurologic injury is identified and prognosis is shared with family, the option of withholding or withdrawing advanced life support is considered and implemented. This is entirely within the realm of patient, family, and clinician interactions and any discussion of brain death does not surface in those circumstances.

Background

With the advancement of intensive care techniques, patients were identified with continuing spontaneous cardiovascular function while on ventilatory support but without evidence of central nervous system activity. Observation of these patients showed that cardiovascular function would eventually fail though rare prolonged survival has been reported.[14] [75] Efforts to reliably identify these patients were sought for improved resource allocation, optimal family counseling, and also to identify possible organ donors. Part of the impetus for reliably establishing clinical brain death was to allow the medical and legal pronouncement of death and allow discontinuation of advanced life support without fear of legal consequences. Death had long been defined as cessation of cardiac function; these deeply comatose patients with persistent cardiac activity but without demonstrable brain function challenged the traditional definition of death. The concept of brain death has been entangled in ethical, legal, and policy discussions roughly since the advent of clinical application of mechanical ventilation in the 1960s. [1] [88] [96] Current medical practice in the United States allows withdrawal of life-sustaining therapies when they are considered futile. In most patients, formal declaration of brain death is not necessary before withdrawing support.

Indications and Contraindications

Assessment of brain function is part of the ongoing care of every patient. Evaluation for brain death implies that severe CNS dysfunction has been identified, that the cause of CNS dysfunction is known, and that reversible causes of profound coma have been confidently excluded. [6] [16] [95] Formal brain death assessment is in preparation for pronouncement of death to allow organ harvest or in uncommon cases of disparate family or caregiver convictions regarding patient prognosis. Complex medical issues that may confound the assessment

and should be considered and ruled out include: severe electrolyte disturbances, hypothermia (defined as core temperature <32°C), hypotension, drug intoxication or poisoning, and pharmacologic neuromuscular blockade.[95] Any neuroimaging studies should be reviewed.

Procedure

By definition, the patient under evaluation will be in coma without spontaneous respirations. Certain examination techniques are used to establish loss of function of the cerebral cortex and brainstem; the clinical neurologic examination remains the standard for determination of brain death. [95] These typically involve assessment for cortical function and brainstem reflexes including respiratory drive. Should any cerebral or brainstem function be discovered, the patient is not brain dead by definition in spite of what may be severe brain injury. Some institutions may require evaluation by two clinicians of particular specialty training, or local practice patterns may require ancillary testing with EEG, nuclear angiography, or other techniques. Again, the clinician must be familiar with local practices and policies. Brain death in the pediatric population is more complex with varying recommendations of repeat examinations and ancillary tests; that discussion is outside the scope of this chapter but is summarized elsewhere. [95]

Establishment of coma and cortical assessment.

By definition, patients undergoing assessment for possible brain death will be in an unresponsive state. However, loud verbal commands such as "Look up!" are given while holding the patient's eyes open to assess for any voluntary eye movements as has been described in the patient with locked-in syndrome. Additionally, a strong painful stimulus should be delivered by forcefully pressing on brow, sternum, or nail bed; any response including abnormal posturing excludes the diagnosis of brain death.

Brainstem Reflex Testing

Pupillary response.

The pupils in brain dead patients are unreactive and mid-position to dilated. Brain death assessment involves shining a bright light stimulus into the pupil and observing the reaction; there will be none in the brain dead patient. Should any reactivity be noted, the patient is not brain dead.

Auditory reflex.

A loud handclap is delivered in each ear; any observed eye blink or other reaction establishes that some brainstem function remains and excludes brain death.

Caloric testing.

Cold-water irrigation of the external auditory canals should be performed with large volumes (100 mL or more) to elicit any eye movements through the oculovestibular

reflexes (described in detail earlier in this chapter). In the brain dead patient, there will be no movement of the eyes in response to irrigation. Any eye movement excludes brain death.

Corneal reflex.

Stimulation of the cornea is performed with a cotton wisp or applicator; any observed eye closure indicates that the V–VII reflex arc remains intact and excludes the diagnosis of brain death.

Cough reflex.

Suctioning with stimulation of the trachea or mainstem bronchi is performed to assess for cough response; there should be no cough or other reaction.

Apneic oxygenation test.

CNS control of respiratory drive resides in the medulla. Establishing apnea is necessary to confirm medullary failure. Mechanical hyperventilation may artificially depress respiratory effort. Recall that it is hypercapnia, not hypoxia, that triggers respiratory effort. Simply disconnecting the ventilator to allow development of hypercapnia for apnea testing may lead to hypoxia.[46] A variety of techniques have been described that allow adequate hypercapnia for medullary stimulation to develop but ensure that oxygenation is adequate during the test. The most commonly described technique is to disconnect the patient from the ventilator and deliver oxygen at 10 to 15 L/min through a catheter inserted into the trachea and then observe for respiratory efforts for 8 minutes. Any observed excursion of the abdomen or chest sufficient to produce a tidal volume suggests that brain death is not present. If there are no observed respiratory excursions, arterial blood gas analysis is obtained and the patient is reconnected to the ventilator pending results. An arterial pCO_2 =60 mm Hg and the absence of respiratory excursions are the criteria for a positive apnea test.[94] Others suggest that a possibly safer technique is to simply set the ventilator rate to zero while allowing continuous oxygen flow to continue and maintaining any necessary continuous positive pressure through the ventilator.[66]

Declaration of Death

If the criteria for brain death are satisfied, the family and all clinicians involved in patient care should be informed to allow further management decisions. At some institutions, the patient is declared dead at the time criteria are met and further care is assumed by transplant services if that is the anticipated course. Per institutional protocol a two-person confirmation of brain death may be required. Families are generally given the option of being present at the bedside while mechanical ventilation is discontinued, though some advise against this because spontaneous reflex movements such as the Lazarus sign (discussed later) may occur and disturb the family.[66] Again, for patients age 18 years or younger, particularly infants, repeated examinations and confirmatory tests are generally recommended.[95] [96]

Complications

Carefully following the physical examination protocol reliably identifies the majority of patients with irreversible loss of brain function. Two basic areas of error are possible; either erroneously declaring a patient brain dead when in fact some CNS function is retained, or failing to correctly identify brain death.

Profound barbiturate intoxication may simulate the picture of brain death at times. To guard against the error of failing to detect surreptitious pharmacologic coma, some protocols include toxicologic screening tests for barbiturates as part of the process of assessment for brain death. Ancillary techniques for assessing intracranial blood flow would also prevent this error. Two examinations both confirming brain death performed several hours apart have been part of some protocols with the idea that toxicologic coma might improve during this period of observation. Again, familiarity with local practices is necessary.

A variety of movements have been observed in brain dead patients; at times these spinal-level-mediated reflexes may be dramatic and erroneously lead observers to believe that the brain dead patient is demonstrating voluntary movements or brain-mediated reflex movements. Finger jerks and facial myokymia (spontaneous, fine fascicular muscular

contractions) have been reported to be spontaneously present in brain dead patients. Decerebrate-like extensor posturing, the Lazarus sign (flexion of the arms at the elbow, shoulder adduction, lifting arms, dystonic posturing of the hands with crossing of the hands), undulating toe flexion, triple flexion response in the lower extremities, and flexion of the trunk (giving the appearance of sitting up) are among the signs described during apnea testing and following the termination of ventilation, or triggered by tactile stimuli.[37] [38] [45] [47] [68] [72]

Summary

The emergency clinician may become involved in assessment of patients for brain death in the course of current practice. The screening physical examination for assessing brain death is described earlier. Again, the cause of irreversible coma should be known. The clinician is urged to be familiar with local practices and regulations in this sometimes complex medicolegal process.

MYASTHENIA GRAVIS TESTING

Background

Myasthenia gravis is the most common disease of neuromuscular transmission but still the incidence is only 1 in 20,000 in the general population. Patients with myasthenia may be grouped into two major categories. The first group shows weakness in proximal muscles, which increases with activity and improves with rest. The other group presents with ocular complaints of diplopia or ptosis. Patients in the ocular weakness group may or may not have generalized symptoms as well. Fatigue is the hallmark of the disease; symptoms typically wax and wane. Patients often see a number of clinicians before a correct diagnosis is made. [20] [78]

Myasthenia gravis results from an immune-mediated destruction of postsynaptic acetylcholine (ACh) receptors, with variable failure of neuromuscular transmission. [19] [20] [52] [78] ACh is the transmitter at the neuromuscular junction. When the nerve terminal is stimulated, ACh is released in a quantity far in excess of that needed for effective activation of the ACh receptor. ACh diffuses across the synaptic cleft to transiently interact with the ACh receptor, and an electrical potential is generated at the myoneural end plate. If it is of sufficient magnitude, the end plate potential initiates an action potential that is propagated along the muscle membrane and muscle fiber contraction follows. The ACh is rapidly hydrolized by acetylcholinesterase in the synaptic cleft. Figure 63-7 summarizes neurotransmitter action at the neuromuscular junction. Of the millions of receptors at each myoneural junction, only a fraction must depolarize to stimulate muscle fiber contraction. Any factor that decreases interactions of ACh with ACh receptors decreases the probability of an action potential being generated and may lead to failure of neuromuscular transmission with resulting weakness. Acetylcholinesterase inhibitors (anticholinesterases) have been the mainstay of therapy for myasthenia gravis for years, but they have been supplemented by immunosuppressive regimens and thymectomy. Failure of neuromuscular transmission may also occur with excessive acetylcholinesterase inhibition; the persistence of ACh in the synaptic cleft leads to continuous depolarization of the receptor.

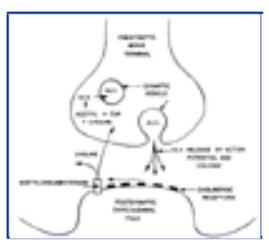

Figure 63-7 Neurotransmitter action at a neuromuscular junction. Acetylcholine *(Ach)* is formed and stored in the nerve terminal. It is released by nerve terminal depolarization in the presence of calcium and binds with receptors on the postsynaptic membrane. After producing an ionic conductance charge, it is hydrolyzed by acetylcholinesterase. *(From Daube JR, Reagen TJ, Sandok BA, Westmoreland BF: Medical Neurosciences: An Approach to Anatomy, Pathology and Physiology, and Physiology by Systems and Levels. Boston, Little, Brown, 1986, p 278.)*

Bedside diagnostic testing for myasthenia gravis has been described in patients presenting to the ED with two different clinical pictures. The first presentation involves a known patient with myasthenia gravis on cholinesterase inhibitor therapy with increased weakness; pharmacologic testing in this setting is controversial. The second presentation involves a previously undiagnosed patient in whom the diagnosis of myasthenia gravis is suspected with ptosis, diplopia, or fluctuating muscular weakness; bedside testing may be helpful in select patients in this group.

A battery of tests can be used in the assessment of the patient with suspected myasthenia gravis, but only a few are available to the emergency clinician. ACh receptor antibody assay is positive in more than 80% of patients with myasthenia but has a turnaround time of several days. [35] [83] Repetitive nerve stimulation and single-fiber electromyography tests are available in the electrophysiology laboratory. [60] Several pharmacologic tests have been used to aid in the diagnosis of suspected myasthenia gravis including parenteral administration of edrophonium chloride (Tensilon), neostigmine, or curare. Edrophonium chloride administration for diagnosis of myasthenia gravis (Tensilon test) is described in detail because of the drug's rapid onset, short duration of action, and widespread acceptance for this diagnostic challenge; the emergency clinician may find occasion to use this test. The ice-pack test is discussed because of favorable reports of its utility and the noninvasive nature of the test.[22] [76] The use of curare has been described to aid in diagnosis of myasthenia gravis by both systemic and regional administration. A technique is described for administration into an ischemic arm; this modification is known as the *regional curare test*.[60] The

use of curare in this setting is outside the realm of practice of the emergency clinician.

Edrophonium (Tensilon) Test

Background

Edrophonium chloride (Tensilon) is an acetylcholinesterase inhibitor that has been used in the diagnosis of myasthenia gravis since the 1960s. The short duration of action of edrophonium that made it unsatisfactory as a therapeutic agent for myasthenia gravis makes it useful as a diagnostic agent. The drug's onset of action is rapid, and the duration of maximal effect is short, usually <2 minutes. Any effect resolves within 5 to 10 minutes. [56]

Edrophonium administration has also been recommended in the past to monitor acetylcholinesterase inhibitor therapy. [57] [58] However, edrophonium administration is not sufficiently reliable for titrating the effect of anticholinesterase medication. [71] The use of the Tensilon test in a patient with known myasthenia gravis is controversial and should be done only in consultation or in the presence of the treating neurologist, if at all.

Myasthenic crisis may be loosely defined as respiratory distress in a patient with myasthenia gravis. Earlier concepts included "myasthenic" crisis from insufficient drug administration, "cholinergic" crisis from cholinesterase inhibitor overdosage, and the "brittle" patient with rapidly changing drug requirements, [30] [90] but terminology in this setting remains controversial. [31] Even the clinical existence of the different types of crisis has been debated. [71] Certainly, in the myasthenic patient with respiratory distress, airway management and assisted ventilation are top priorities. Edrophonium administration should not be viewed as a possible alternative to intubation and mechanical ventilation. Several authorities advocate withdrawing all cholinesterase inhibitors in this setting because most patients show increased responsiveness to cholinesterase inhibitors after several days without taking the drug. [31] [71] Others advocate a trial of edrophonium chloride at reduced dosage (1 to 2 mg) only after respiratory support is achieved. [30] [74]

Indications and Contraindications

The bedside Tensilon test is indicated for diagnosis of patients with suspected myasthenia gravis when there is clinical need to make that diagnosis immediately. If other bedside tests such as the ice pack test (discussed later) are conclusive, or if eyelid fatigue on prolonged upgaze can be demonstrated, there is no need to administer Tensilon.[55] If the tempo of the clinical scenario is such that consultation or admission and obtaining specialized EMG or serologies is feasible, then deferring the Tensilon test in the ED may be the preferred strategy, realizing that serologies and neurophysiologic testing will not be completed for some days.

Administration of edrophonium is commonly performed in office settings by neuro-ophthalmologists with occasional complications of bradycardia that at times may be symptomatic with hypotension and loss of consciousness.[36] [92] One study reports the complication rate in office settings as 0.16%. [36] A history of asthma or cardiac dysrhythmias is a relative contraindication to administration of cholinesterase inhibitors. Complications reported in the literature from Tensilon testing largely date from the era when it was used in cardiology to differentiate supraventricular rhythms. [55] Reported complications include cardiac rhythm disturbances and even death; several of these patients were taking digoxin or beta-blockers. [29] [48] [70] [97] One report notes transient asystole following Tensilon administration in a patient with suspected myasthenia; the patient was critically ill and had been receiving intravenous labetalol. [55] Hence, caution is advised in Tensilon administration in patients receiving beta-blocking agents, digoxin, or other drugs with atrioventricular-blocking properties.

Administration of edrophonium to a patient with myasthenia gravis being treated with cholinesterase inhibitors is controversial. Many investigators consider myasthenic crisis to be a contraindication to edrophonium administration, though a series of patients with myasthenic crisis continue to report its use in the clinical setting.[30] [74]

A muscle that is clearly weak must be identified to monitor during Tensilon testing; ptosis is a commonly monitored sign. If a specific muscle cannot be isolated for objective testing, edrophonium administration should be deferred and other approaches to diagnosis pursued.

Equipment

The following materials are needed for testing an adult. Intravenous access should be secured with D_5W at a keep-open rate or with a saline lock. Ten milligrams of edrophonium chloride should be drawn up in a tuberculin syringe. Edrophonium chloride is supplied in 1- and 10-mL vials at a concentration of 10 mg/mL. A second syringe of normal saline should be available to administer as a placebo, although some clinicians have recommended nicotine, calcium chloride, or atropine for this purpose.[58] Atropine and other cardiovascular drugs and resuscitative equipment should be readily available. Cardiac monitoring is generally recommended. Photographic recording equipment is desirable to objectively document any improvement in motor function.

Procedure

Again, a muscle that is clearly weak must be identified. A clinically evident extraocular muscle weakness or the presence of ptosis allows direct observation of a single weak muscle becoming stronger in response to the drug. Simple grip dynamometry does not aid in evaluation; a repetitive measure of grip strength (ergogram) is necessary. Ideally, one person is available to administer the edrophonium or placebo and a second person is free to observe the effect of medication on the patient. It is best if both the observer and the patient do not know which syringe contains edrophonium and which contains saline, thus creating a double-blind testing situation.

The paretic muscle to be tested is identified. Again, ptosis is an easily testable sign and is generally used if present. The principles involved with assessing the effect of edrophonium on ptosis may be extended to testing other muscles. The patient is instructed to look upward for several moments in an attempt to fatigue the levator muscles. The degree of ptosis is noted and documented by measurements or photographs. After a moment's rest, with the patient looking straight ahead, 0.2 mL of the test substance in one syringe is injected (2 mg of edrophonium or saline). If there is no response within 1 minute, another 0.3 mL is injected (3 mg of edrophonium or saline). If there is still no response, the remainder of the syringe is injected 3 minutes later. Any increase in strength reflected by an increase in palpebral fissure size is noted. The procedure may then be repeated with the other test substance.

Complications

A small percentage of individuals are hypersensitive to even the initial small dose of edrophonium and show cholinergic side effects of salivation, lacrimation, and miosis. These effects are transient. Atropine, 0.5 mg, may be given IV if necessary to counteract these symptoms. A smaller number of patients may experience symptomatic bradycardia that responds to atropine. As described earlier, rare cardiac arrhythmias and death have been reported, usually in patients taking digoxin or beta-blockers.

Interpretation

The key to the procedure is its interpretation. If a clearly paretic muscle has been identified, objective signs of improvement in the strength of that muscle within a moment of administration of edrophonium and the fading of that improvement over the next 5 minutes are criteria for a positive test result. Up to 90% of patients with myasthenia have a positive test result under ideal circumstances. [57] [58] False-negative results do occur consistently.

For evaluating the effect of edrophonium on ptosis, a positive test consists of the patient having increased ability to elevate the eyelids after administration of 5 to 10 mg of edrophonium. The ptosis returns within 5 minutes. Subjective increases in general strength or relief of fatigue do not constitute a positive test. Fasciculations, brief twitches of muscles, are not usually observed in the patient with myasthenia who has received edrophonium, in contrast to normal subjects. The Tensilon test may be repeated in 30 minutes if desired.

Normal subjects have no change in muscle strength. They may transiently experience the side effects of salivation, lacrimation, and diaphoresis. Perioral, periocular, or lingual fasciculations are almost always noted in the normal patient following edrophonium administration.

The reproducible and unequivocal reversal of weakness in a specific muscle is extremely specific for myasthenia. [53] [54] False-positive test results have been reported in patients with Eaton-Lambert syndrome and rarely in patients with intracranial lesions. [17] [50] [65] Other rare reports of positive test results involve patients with amyotrophic lateral sclerosis. A "perverse" reaction has been noted rarely, in which a paretic extraocular muscle weak from other causes becomes even weaker with edrophonium administration.[77]

Ice Pack Test

Background

It has been observed clinically that myasthenic patients have exacerbations of weakness with environmental heat and improvement in strength with cold temperatures. A simple bedside test uses these observations to evaluate ptosis. [22] [76] Ice placed in a surgical glove or wrapped in a towel is placed lightly over the eyelid of a patient. Cooling of the eyelid below 29°C is accomplished within 2 minutes. The ptosis has been noted to improve in 80% or more of patients tested and may be more sensitive than the edrophonium test in detecting ocular myasthenia gravis. Although the reported number of patients evaluated by this method continues to be small, the test is included here because of its potential application in the ED, its lack of side effects, and its noninvasive nature.

Indications

Unilateral or bilateral ptosis of uncertain etiology in which myasthenia is a diagnostic possibility is the sole indication for this test.

Procedure

Ice and a surgical glove or towel are the only materials required. A camera to record any response is optional. The degree of ptosis of the patient is noted and, ideally, photographed. Momentary upward gaze may be used to provoke the ptosis. If bilateral ptosis is present, the more affected eye should be tested. Eyelid cooling is accomplished by lightly holding the wrapped ice to the patient's eyelid for 2 minutes or until patient discomfort limits the application. The width of the palpebral fissure is immediately compared with the pretest width (Fig. 63-8).

Complications

Patient discomfort from the ice pack application may limit the cold exposure time to less than 2 minutes but may still allow a successful test.

Interpretation

A clear improvement of ptosis in the cooled eye is the criterion for a positive test. The effect should be reproducible. In small clinical studies, the ice pack test is at least as sensitive as edrophonium administration in improving ptosis in patients with ocular myasthenia. False-negative results do occur, probably at about the same frequency as those in Tensilon testing. One individual has been reported to have had a negative ice pack test result with a positive Tensilon test result. Negative or

equivocal Tensilon tests have been reported in other individuals who had clearly positive ice pack test results. Normal individuals showed no change in palpebral fissure width after the cold exposure. There are no reports of false-positive results. [22] [76]

Summary

The bedside Tensilon test has a long history of utility in diagnosing myasthenia gravis, but it has been largely replaced by

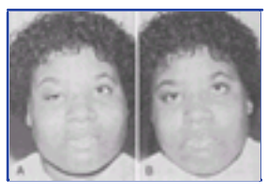

Figure 63-8 *A,* Before ice pack placement. *B,* After ice pack placement, improvement is noted in ptosis of the right eye. *(From Sethi KD, Rivner MH, Swift TR: Ice pack test for myasthenia gravis. Neurology 37:1383, 1987.)*

acetylcholinesterase receptor assay and electrodiagnostic studies in the ambulatory setting. [39] [54] [55] [60] On occasion when rapid diagnosis is desired or myasthenia gravis is suspected in the presence of a normal ACh receptor titer, a carefully performed Tensilon test is still clinically valuable. The use of the Tensilon test in the setting of myasthenic crisis is controversial and is discouraged.

The ice pack test is so simple and noninvasive that it should become the initial procedure of choice in the ED for evaluating the possibility of ocular myasthenia. A positive ice pack test result strongly suggests ocular myasthenia gravis and alleviates any need for the Tensilon test. False-negative results do occur, and additional testing should be performed if the clinical suspicion of myasthenia gravis is strong.

It is often the case with neuromuscular diseases, given the broad diagnostic possibilities, that the emergency clinician is unable to establish a confident diagnosis at a single patient encounter. [82] Appropriate consultation and referral are necessary.

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Section XII - Ophthalmologic, Otolarynologic, and Dental Procedures

Chapter 64 - Ophthalmologic Procedures

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The following discussion focuses on procedures performed by emergency clinicians during the evaluation and treatment of injuries and diseases of the eye. The emphasis is on the practical application of the techniques, and cautions to be heeded by the emergency clinician are included.

VISUAL ACUITY ASSESSMENT

Visual acuity may initially be eschewed in simple, obvious, or straightforward cases, such as a stye, periorbital laceration, or minor eye irritation; however, visual acuity assessment should be the first procedure performed in the majority of patients who present to the emergency department (ED) with an eye complaint. While it may be initially deferred in the triage or trauma room setting, or under other relevant scenarios, it is incumbent upon the emergency clinician to ensure that visual acuity or function is ultimately adequately assessed.

Indications

Visual acuity should be done as soon as practicable and before the patient is examined with bright lights. In the event of blepharospasm from an injury (e.g. abrasion, chemical exposure), a topical anesthetic may facilitate the examination. Patients often present in the context of an eye complaint saying that they "can't see." In these instances, emergent visual acuity assessment should first be performed beginning with the assessment of light perception, then hand motion, and finally counting fingers at 3 feet (Fig. 64-1). If the patient succeeds in performing these assessments, a near vision card may then be used, or distant visual acuity assessed. Under emergent circumstances, detailed formal vision testing is not essential; however, some form of visual acuity assessment is needed. In this situation, the ability to count fingers or read newsprint gives some indication of gross visual function. Formal visual acuity testing should never delay important therapeutic interventions such as eye irrigation.[1]

Distant Visual Acuity Procedure

For formal vision testing, the patient faces a well-lit standard Snellen or similar eye chart from a premeasured distance of 20 feet. A card or the palm of the hand is used to occlude one eye at a time. If possible, all patients should be examined while wearing their current lens correction in order to obtain the best corrected distant visual acuity. If this is not available, measure visual acuity first without correction, then with a pinhole device and note any improvement in visual acuity. This device functions as a corrective lens by reducing corneal refractive error. In general, visual acuity should be improved with the pinhole device. Decreased visual acuity that is not improved with this device suggests that corneal refractive error is not the cause. A pinhole device can be made by punching several holes in the center of a card (3" × 5" index card) with an 18-gauge (ga) needle. Devices with one or more pinholes drilled into an eye cover are available commercially (Fig. 64-2). A visual activity chart for visual acuity testing while the patient is on a stretcher or in a chair is depicted in Figure 64-2*C* . The chart can be used directly from this text if it is held 14 inches from the eye. Begin by testing the affected eye or the one presumed to have the worst visual acuity. First, instruct the patient to read the smallest letters on the chart that can easily be seen. Then, ask the patient to read letters that can just barely be made out (i.e., they do not have to be clear). If the patient is unable to read the largest letter on the chart, move the patient to one half the distance from the chart (10 feet) or move the figure 7 inches closer to the edge and repeat the procedure. Record the results reflecting the change in distance (e.g., 10/200). The numerator in the vision ratio is the distance of the patient from the chart and the denominator is the distance at which a patient with normal vision can read the line of letters. For the patient who still cannot read the letters on the chart, test vision progressively as follows: ability to count fingers, detect hand motion, perceive light (with or without projection and ability to perceive the direction of light), and finally note the inability to perceive light. [2]

Near Visual Acuity Procedure

Near visual acuity is performed in the ED at the bedside or at triage. A pocket near vision card (Figure 54-2*C*) or any printed material is held at a distance of approximately 14" in good light in front of the patient as each eye is alternately occluded as described earlier. When using available printed material in lieu of a near vision card, the size of the letters discerned by the patient should be measured. These can be compared with the letter size on the near vision card to deduce the actual visual acuity at a later time. When near vision is decreased, it is usually from either loss of visual function or as a result of poor accommodation from advancing age

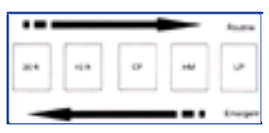

Figure 64-1 The "routine" progression of visual acuity assessment is reversed in the emergent presentation. Assessing an intact visual pathway begins with quickly discerning if the patient has light perception (LP), can see hand motion (HM), and count fingers at 3 feet (CF). Subsequent progression to assess vision at 10, then 20, feet from a standard eye chart ensues.

*This article represents the views of the authors and is not to be interpreted as official, or representing the U.S. Navy or the Bureau of Medicine.

Figure 64-2 A commercial pinhole device reveals refractive error caused by corneal aberration (excess tearing or nearsightedness). *A*, First measure visual acuity without the device. *B*, Then measure with the pinhole cover lowered. Acuity with and without the pinhole device should be documented. *C*, If the patient cannot stand or a formal eye chart is not available, ask the patient to read this "distance equivalent" chart by holding this figure 14 inches away from the patient.

(presbyopia). Less commonly it is caused by traumatic mydriasis. Thus, patients with presbyopia should be examined with their reading correction in place to obtain the best corrected near visual acuity.

For patients who cannot communicate or in whom factitious blindness or malingering is suspected, checking for optokinetic nystagmus (OKN) will determine if there is an intact visual pathway. OKN is induced by the passage of a regularly sequenced pattern in front of the eyes. This is usually tested with the rotation of a striped drum in front of the patient (Fig. 64-3). This is not available in many EDs, though. A printed piece of paper such as newsprint (without photographs or large areas with no print) or a standard tape measure can be substituted. It should be passed in front of the eye at reading distance while instructing the patient to look at it as it moves by rapidly. Tracking is demonstrated by nystagmus-like eye movements seen when the test object is moved from side to side in front of the patient and indicates an intact visual pathway. Finally, another effective method is to hold a mirror in front of the patient and slowly rotate the mirror to either side of the patient. The patient with an intact visual pathway will maintain eye contact with himself or herself as demonstrated by eye movement as the mirror is moved. A large mirror that reflects the patient's entire face is most effective for this purpose.

All patients with decreased visual acuity from their baseline require routine referral for further ophthalmologic follow-up; however, those patients with moderately or severely decreased visual acuity not explained by refractive error require ophthalmologic consultation in the ED.

Figure 64-3 Optokinetic Nystagmus (OKN) testing will determine if there is an intact visual pathway. OKN is induced by the passage of a regularly sequenced pattern in front of the eye such as this commercially available drum. The patient is directed to look at the drum that is held in front of the patient and rotated slowly. Alternatively, a tape measure can be drawn across the line of sight while asking the patient to look directly at it as it passes.

DILATING THE EYE

Dilating the eye is useful for both diagnostic and therapeutic purposes. Clinicians should be advised, however, that on various occasions, an attack of narrow-angle (angle-closure) glaucoma may be precipitated by dilating the pupil. The most common form of glaucoma, however, is open-angle glaucoma and this type is not precipitated by dilating the pupil. Some patients may have a "mixed-mechanism" glaucoma with both open-angle and narrow-angle components. Systemic reactions, such as bradycardia from beta blocker eye drops, can be produced by mucosal absorption of dilating medications. To minimize complications, the correct agent must be chosen and the proper technique used.

There are two types of dilators: sympathomimetic agents, which stimulate the dilator muscle of the iris, and cycloplegic agents, which block the parasympathetic stimulus that constricts the iris sphincter. Cycloplegic agents also block the contraction of the ciliary muscles, which control the focusing of the lens of the eye. This second effect of cycloplegic agents is of great importance in the therapeutic use of dilators for iritis.

Cycloplegic agents were used cosmetically as early as Galen's time. Beginning in the early 1800s, extracts from the plants *Hyoscyamus* and belladonna were used in ophthalmology. Atropine was first isolated in 1833. Epinephrine was used on eyes in 1900 as the first sympathomimetic agent. [3]

Indications and Contraindications

There are diagnostic and therapeutic indications for dilating the pupil. Dilation is indicated for *diagnosis* when the fundus cannot be examined adequately through an undilated pupil. The elderly patient with miotic pupils and cataracts is an example of a patient in whom dilation may facilitate fundoscopic examination. Dilation is *therapeutically* useful for many ophthalmic conditions, including inflammation in the eye. In the emergency setting, corneal injury with a secondary traumatic iritis is a common example. Dilation helps the inflamed eye in two ways. First, it may hinder adhesions (synechiae) from forming between the iris and other ocular structures. Such adhesions eventually limit the movement of the pupil and may precipitate glaucoma. Second, cycloplegic dilating agents relax the ciliary muscle spasm that often accompanies an inflamed eye and thus may reduce the pain associated with inflammation. While traditionally used for these purposes, both benefits are largely theoretical, with little formal evidence to support or refute their use in the ED.

Dilation is discouraged in the patient with head injury at risk for herriation, when monitoring pupil findings can be helpful prior to obtaining a head computed tomography (CT) scan. Dilation is contraindicated in the presence of narrow anterior chamber angles. Patients who are predisposed to having narrow angles may be unaware of this condition. The clinician should evaluate the depth of the anterior chamber before this procedure and the pupil should not be dilated if there is any question of a narrow angle. The depth of the anterior chamber can be estimated by shining a penlight in tangentially from the lateral side of the eye. When the depth of the anterior chamber is normal, a uniform illumination of the iris is seen. However, when there is a forward convexity of the iris in the case of a narrow anterior chamber, only a sector of iris is illuminated and there will be a shadow on the medial (nasal) side of the iris (Fig. 64-4). With a slit lamp, the depth of the anterior chamber angle can be assessed directly. The definitive test for assessing the anterior chamber angle is gonioscopy, in which the anterior chamber angle structures are viewed directly by means of a special mirrored contact lens and the slit lamp. Gonioscopy is not a technique normally performed by emergency clinicians.

Systemic effects can develop after the application of eyedrops. [4] [5] [6] [7] [8] [9] [10] [11] [12] [13] [14] [15] [16] The reader should review the following sections on agents and complications before using these drugs in patients with compromised cardiovascular function.

Agents

Only two dilating agents are really needed in the ED. Phenylephrine (Neo-Synephrine) 2.5% is used for diagnostic dilation of the pupil for visualization of the fundus. The drug is short-acting, and because accommodation is not affected, the patient's vision is not altered. Phenylephrine 10% should not be used routinely because it can be absorbed systemically and in rare cases has caused hypertensive crisis, myocardial infarction, and death. [17] [18]

For therapeutic cycloplegia in iritis, homatropine 5% works well. Although Table 64-1 indicates a maximum duration of 3 days, 24 hours is a more common duration. Therefore, homatropine 5% is a useful therapeutic agent for traumatic iritis. Atropine should not be used for traumatic iritis because the undesirable effects of pupillary dilation and blurred vision persist for a week or longer after healing of associated corneal abrasions. Atropine drops may be prescribed as part of the therapy for nontraumatic iritis after appropriate ophthalmologic consultation. Individuals with lightly pigmented irides tend to have a greater sensitivity to the cycloplegic agents than do individuals with greater pigmentation; the cycloplegic effect may therefore be more prolonged in people with light eyes. It may be difficult to dilate some patients with deeply pigmented irides, and numerous applications of drops may be required.

Figure 64-4 Anterior chamber depth and transillumination test. *A,* Normal anterior chamber with negative transillumination test. Note that the entire iris is illuminated. *B,* Shallow anterior chamber with positive transillumination test. Note the shadow on the outer half of the iris. *C,* Shallow anterior chamber with correctly placed light source yielding a true-positive test result in the presence of a shallow anterior chamber. *D,* Shallow anterior chamber with incorrectly placed light source giving a false-negative test result. *E,* Clinical use of the penlight examination to assess the depth of the anterior chamber. The examiner sits face-to-face with the patient to ensure that the light source is perfectly perpendicular to the line of vision. *F*, Note the shadow cast on the nasal portion of the iris, indicating a very narrow anterior chamber in this patient with acute angle closure glaucoma. *(A–D from Bresler MJ, Hoffman RS: Prevention of iatrogenic acute narrow-angle glaucoma. Ann Emerg Med 10:535, 1981. Reproduced by permission.)*

Malingerers may use mydriatic agents to dilate a pupil unilaterally for the purpose of feigning neurologic disease. Normally, a pupillary dilation caused by intracranial third cranial nerve compression will constrict with 2% pilocarpine eyedrops. The mydriatic-treated eye can be identified by full motor function of the third cranial nerve and the absence of

TABLE 64-1 -- Mydriatic Agents

Agent	Maximum Mydriasis	Duration of Mydriasis[§]	Common Trade Name
Sympathomimetics			
Phenylephrine[*] , 2.5%[†]	20 min	3 hr	Neo-Synephrine
Cocaine, 5% or 4%	20 min	2 hr	—
Parasympatholytics (Cycloplegics)			
Atropine, 1%	40 min	12 days	—
Scopolamine, 0.25%	30 min	7 days	—
Homatropine, 5%[‡]	30 min	1–3 days	—
Cyclopentolate, 1%	30 min	6–24 hr	Cyclogyl
Tropicamide, 1%	30 min	4 hr	Mydriacyl

§The duration of effect shows considerable individual variation. These are general estimates.
*Preferred for funduscopic examination.
†A 10% solution may produce cardiovascular reaction and hence should not be used.
‡Preferred for iritis or corneal abrasion therapy.

miosis after pilocarpine instillation. A fixed and dilated pupil in an awake and alert patient cannot be secondary to brain herniation. Although other neurologic problems may be present, in the normal-appearing patient with a fixed and dilated pupil, a pharmacologic cause is highly likely. It should be noted that legitimate patients may not recall the name of an

Figure 64-5 *A*, After neosynephrine drops were instilled in the nose to facilitate tube passage, this comatose patient was nasotracheally intubated for his drug overdose. *B*, On a subsequent examination a unilateral fixed and dilated pupil was noted. The pupil dilation was from neosynephrine nose drops that were snorted from the nose into the eye during intubation, simulating cerebral herniation. Other unusual causes of a fixed and dilated pupil are endotracheal epinephrine expelled from the lungs and splashed in the eye during cardiopulmonary resuscitation and inadvertant contamination of the eye following application of a scopolamine patch behind the ear.

eye medicine that they used but will usually recall whether the bottle had a red cap, as is found on all cycloplegic solutions. An unexpected mydriasis in a trusted patient may be the result of such an agent. Medications that constrict the pupil, such as pilocarpine, have a green cap. Pressure-lowering drops for glaucoma may be yellow- or blue-topped (ß-blockers), purple-topped (adrenergic agents) or orange-topped (topical carbonic anhydrase inhibitors).

A fixed and dilated pupil from a pharmacologic cause may be encountered following both nasotracheal and orotracheal intubation (Fig. 64-5). In such ill or injured patients, cerebral herniation must be considered. When neosynephrine is used to constrict the nasal mucosa prior to nasal intubation (endotrachial tube, nasogastric tube), the inadvertant contamination of the eye will cause a fixed and dilated pupil. The same scenario may occur after endotracheal epinephrine has been instilled into the lungs during resuscitation, and cardiopulmonary resuscitation has expelled epinephrine into the eye. Under such scenarios, the affected pupil will not constrict following intraocular pilocarpine administration. Finally, a fixed and dilated pupil may occur from inadvertent contamination of the eye with scopolamine following application of a scopolamine patch.

Procedure

The instillation of mydriatic agents is similar to the administration of other eye solutions. For medicolegal purposes, visual acuity should be noted before the instillation of the medicine. This documents that any decreased vision is not the result of the mydriatic agent. Whenever dilation is performed, it is important to note on the patient's chart the dose and time that agents have been given to avoid confusion during subsequent neurologic evaluation.

The patient is placed in a supine or a comfortable semirecumbent position. The patient is then instructed to gaze at an object (such as a fixture on the ceiling) in the upper visual field. The clinician gently depresses the lower lid using a finger on the epidermis (Fig. 64-6). A *single* drop of the solution is instilled into the lower lid fornix, and the patient is permitted to blink and to spread the medication. More than a single drop is not recommended because it produces reflex tearing and reduces the concentration in contact with the conjunctiva. The patient should be forewarned that the medication is uncomfortable and that the closed eye can be blotted but *not*

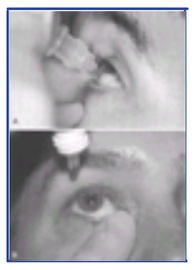

Figure 64-6 *A*, Administration of eyedrops. The patient should lie in a supine position or with the head tilted back. The patient's gaze should be directed upward. The lower lid is pulled downward and a single drop of medicine is instilled in the lower conjunctival fornix. The patient should be instructed to close the eyelids for 1 minute to increase the contact of the medicine with the globe and to decrease the medication outflow down the tear duct and over the lid margin. *B*, If one is administering large amounts of eye drops that have systemic effects, such as beta blocker drops, the operator's index finger is placed under the inferior eyelid along the nasal borders of the eye, firmly compressing the nasolacrimal duct against the globe for a few minutes, thereby preventing migration of the drops into the nose and reducing systemic absorption.

rubbed with a tissue after the medicine is used. If the desired effect is not noted in 15 to 20 minutes, a repeat dose may be used, but this is seldom required.

Complications

As mentioned in the section on contraindications, any dilator can precipitate an attack of angle-closure glaucoma in susceptible patients. [19] In a case of angle-closure glaucoma, it may take several hours before symptoms become evident. The patient often complains of smoky vision with "halos" around lights as well as an aching pain that is sometimes severe. There may be nausea and vomiting. The affected eye becomes injected in association with a hazy cornea, elevated pressure on tonometry, and an oval, fixed pupil. Immediate consultation with an ophthalmologist should be obtained. The treatment usually includes osmotic agents, carbonic anhydrase inhibitors, beta-blocker drugs, pilocarpine, and, later, definitive laser or surgical procedures (Table 64-2).

The practitioner should be aware that the use of eye medications may introduce infections. Most solutions contain bactericidal ingredients, although contamination of the tips of the droppers can still occur. [20] Only newly opened bottles of eye medication should be used if the practitioner suspects a deep corneal injury or if the patient has recently had eye surgery. Out-of-date drops and drops in which crust or other material is found around the nozzle should be discarded promptly.

Any cycloplegic (in contrast to a sympathomimetic) blurs a patient's near vision. Patients should be forewarned of this effect. Vision will be less blurred in adults older than 45 years of age, who generally have a reduced ability to focus for near vision. Although most adults will be able to drive safely, even with both eyes affected, it is advisable to have someone else drive whenever feasible. Light sensitivity caused by pupillary dilation may also be bothersome; sunglasses are sufficient for this problem.

Systemic reactions can be produced by sympathomimetic and cycloplegic eyedrops. [4] [5] [6] [7] [8] [9] [10] [11] [12] [13] [14] [15] [16] In one report of 33 cases of adverse reactions associated with 10% phenylephrine, there were 15 myocardial infarctions (11 deaths), 7 cases of precipitation of angle-closure glaucoma, and a variety of systemic cardiovascular or neurologic reactions. [18]

After instillation of eyedrops into the conjunctival sac, systemic absorption can occur through the conjunctival capillaries as well as by way of the nasal mucosa, the oral pharynx, and the gastrointestinal tract after passage through the lacrimal drainage system. Mucosal hyperemia enhances absorption. Symptoms can often be avoided by maintaining digital pressure on the nasal canthus, thus occluding the puncta, for several minutes after administration. [4]

TABLE 64-2 -- Treatment Options for Acute Angle-Closure Glaucoma

1. Pilocarpine 4%: 2 drops every 15 min for 1–2 hr*
2. Glycerol: 1 mL/kg by mouth (as 50% solution in citrus juice)

3. Mannitol: 1.5–3 g/kg intravenously over 20 min (as 20% solution)[*]
4. Acetazolamide: 500 mg intravenously[*]
5. Beta-blocker drugs (e.g., timolol 0.5%) 1 drop every 30 minutes for 2 doses[*, †]
6. Alpha-2-agonist (apraclonidine [Iopidine] 0.5%) 1 drop[†]

*First line therapy
†May cause cardiovascular effects

THE FLUORESCEIN EXAMINATION

Fluorescein staining of the eye should be part of the evaluation of all cases of eye trauma and infection. It is a quick and easy technique that is crucial for the proper diagnosis and management of common eye emergencies. The fluorescein-stained cornea and conjunctiva should be viewed under a "blue" light and ideally in conjunction with slit lamp magnification (see Slit Lamp Examination later in this chapter).

Sodium fluorescein is a water-soluble chemical that fluoresces—it absorbs light in the blue wavelengths and emits the energy in the longer green wavelengths (Fig. 64-7). It fluoresces in an alkaline environment (such as in the Bowman membrane, which is located below the corneal epithelium) but not in an acidic environment (such as in the tear film over an intact corneal epithelium). [21] Thus, it is useful in revealing even minute abrasions on the cornea.

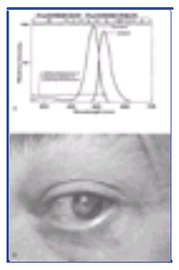

Figure 64-7 *A*, Sodium fluorescein absorbs light in the blue wavelengths and emits the energy in the longer green wavelengths. *B*, This large corneal abrasion is readily seen without the slit lamp when fluorescein is instilled into the eye. Smaller abrasions, or corneal injuries produced by keratitis or welder's arc flash injuries, require slit lamp evaluation to identify minor corneal defects. Even minor traumatic abrasions will escape detection with only a blue light examination by the naked eye.

Fluorescein was first used in ophthalmology in the 1880s. [22] It was first used as a drop, but when the danger of contamination by bacteria (especially *Pseudomonas*) was recognized in the 1950s, [23] paper strips impregnated with fluorescein were developed. These are now supplied in individual sterile wrappers and should be used instead of the premixed solution.

Indications and Contraindications

Fluorescein staining is indicated for evaluation of suspected abrasions, foreign bodies, and infections of the eye. [24] This includes "simple" cases of conjunctivitis, which may actually be herpetic keratitis. Pepper spray exposure to the face has been associated with corneal abrasions and these patients should be stained with fluorescein and evaluated with slit lamp or Wood's lamp. [25] Corneal defects may be seen after unprotected viewing of a welder's arc flame.

Fluorescein permanently stains soft contact lenses. Therefore, when fluorescein is used, soft contact lenses should first be removed and the patient cautioned not to put the lenses back into the eye for several hours. Topically administered fluorescein is considered nontoxic, [26] although reactions to a fluorescein-containing *solution* (not impregnated strips) have been described. These reports consisting of vagal reactions [27] and generalized convulsions [28] are rare, not rigorously supported, and are believed to be caused by agents other than fluorescein in the solution. The practitioner who chooses to use one of the fluorescein-containing solutions rather than the fluorescein-impregnated strips should be aware of these potential, yet scientifically suspect, idiosyncratic reactions.

The clinician should also be aware that fluorescein dye may enter the anterior chamber of the eye in the presence of deep corneal defects. This form of intraocular fluorescein accumulation is also nontoxic. When the anterior chamber is viewed under the blue filter of the slit lamp, a fluorescein "flare" is visible and should not be confused with the flare reaction noted with iritis.

Procedure

Theoretically, one should not use topical anesthetics before fluorescein staining, because some patients may develop a superficial punctate keratitis from the anesthetic, [21] which can confuse the diagnosis. However, with patients who are tearing profusely and who are squeezing their eyes shut from an abrasion or a foreign body (FB), the examination often is impossible if a topical anesthetic is not first used. Theoretical downsides notwithstanding, it is common practice to instill fluorescein after the application of a local anesthetic.

The fluorescein strip is grasped by the non-orange end, and the orange end is wetted with 1 drop of saline (most conveniently available as a small bottle of artificial tears, or a 5 mL "bullet" or "fish" of normal saline usually used for nebulizer treatments) or dextrose solution. Some clinicians will wet the strip with tap water or the recently used local anesthetic drops. The moistened strip is then placed gently onto the inside of the patient's lower lid (Fig. 64-8). The strip is withdrawn, and the patient is instructed to blink. The patient's blinking spreads the fluorescein over the surface of the eye. The key to a good examination is a *thin* layer of fluorescein over the corneal and conjunctival surfaces. If the strip is heavily moistened before application in the lower fornix, the eye may become flooded with the solution, thus making evaluation

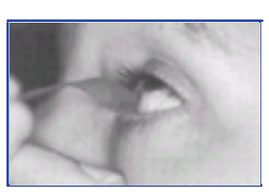

Figure 64-8 The fluorescein strip has been moistened with 1 drop of saline or tap water. The lower lid is depressed and a wetted strip is then placed gently onto the inside of the patient's lower lid so that only the smallest amount is instilled. Excess fluorescein may obscure subtle findings and thus should be avoided. Fluorescein will permanently stain contact lenses not removed (as seen in this model).

difficult. On the other hand, use of a dry strip in the unanesthetized eye may be irritating. If too much dye accumulates, the patient can remove the excess by blotting the closed eye with a tissue. The clinician uses a Wood's lamp, the blue filter of a slit lamp, or simply a penlight with a blue filter to examine the eye in a darkened room, checking for areas of bright green fluorescence on the corneal and conjunctival surfaces. Small defects may not be seen with the naked eye; ideally, a slit lamp should be used to examine the stained cornea before ruling out a pathologic process. After completion of the fluorescein examination, excess dye should be irrigated from the eye to minimize damage to the patient's clothing from dye-stained tears.

The *Seidel* test [29] uses fluorescein to detect perforation of the eye. To perform this test, the clinician instills a large amount of fluorescein onto the eye by profusely wetting the strip. The eye is then examined for a small stream of fluid leaking from the globe (Fig. 64-9 (Figure Not Available)). This stream will fluoresce blue or green in contrast to the orange appearance of the rest of the globe flooded with fluorescein. [21]

Interpretation

Fluorescein is mainly used for evaluation of corneal injuries. Although conjunctival abrasions pick up the stain, most of the staining on the conjunctiva represents patches of mucus rather than a real pathologic condition. Corneal staining is more specific for injury, and the pattern of injury often reflects the original insult.

Corneal staining patterns are illustrated in Figure 64-10 . Abrasions usually occur in the central cornea because of the limited protection of the patient's closing eyelids. The margins of the abrasions are usually sharp and linear if seen in the first 24 hours. Circular defects are seen about embedded FBs and may persist for up

to 48 hours after removal of a superficial foreign object. Deeply embedded objects may be associated with defects persisting for longer than 48 hours. Objects under the upper lid (including some chalazia) often produce *vertical linear lesions* on the upper surface of the cornea. When vertical lesions are noted, a *diligent search for a retained FB under the upper lid should be made*. Hard contact

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Figure 64-9 (Figure Not Available) Positive Seidel test showing aqueous leaking through a full-thickness corneal wound. Aqueous will turn fluorescein lime-green under cobalt-blue light as it oozes through the wound while being observed at the slit lamp. *(From Mitchell JD: Ocular Emergencies. In Tintinalli JE, Kelen GD, Stapcynski JS (eds): Emergency Medicine—A Comprehensive Study Guide, 5th ed. New York, McGraw-Hill, 2000. Reproduced by permission.)*

lens overuse diminishes the nutrient supply to the cornea. The central cornea receives the most injury and thus fluoresces brightly when stained. Ultraviolet light exposure from sunlamp abuse, snow blindness, or welding flash produces a superficial punctate keratitis, which in its mildest form may not be visible without a slit lamp. The central cornea is the least protected by the lids, and a central horizontal band-like keratitis can result. Herpetic lesions may develop anywhere on the cornea. Classically, these lesions are dendritic, although ulcers may also be punctate or stellate. [30] [31]

Any area of corneal staining with an infiltrate or opacification beneath or around the lesion should alert the practitioner to the possibility of a viral, [30] [31] bacterial,[32] or fungal[33] *keratitis*. Urgent ophthalmologic consultation should be obtained so that cultures of the possible etiologic agents can be procured and appropriate treatment initiated.

Many *Pseudomonas* organisms fluoresce when exposed to ultraviolet light [34] ; therefore, presence of fluorescence before the instillation of fluorescein in the red eye should suggest the possibility of a pseudomonal infection.

Summary

Fluorescein staining is a quick, easy diagnostic procedure that should be part of every eye evaluation. The extra minute that the examination takes provides a wealth of diagnostic information for patients with eye trauma or infection. With the exception of the reactions noted with fluorescein solution, the potential discoloration of soft contact lenses, and the potential for infection when premixed solutions rather than fluorescein-impregnated paper strips are used, no complications are associated with the procedure.

EYE IRRIGATION

Eye irrigation is the crucial first step in the treatment of chemical injuries to the eye. Irrigation should be clinically appropriate to the exposure and severity of the injury. Serious chemical injury to the eye requires irrigation at the site of injury, *before* the patient is brought to the ED.[24] Corneal injury can occur within seconds of contact with an alkaline substance. Eye irrigation must often be continued in the ED.

This section discusses methods of irrigation. Although it is best to irrigate liberally, copious irrigation is not needed when the patient has gotten a small amount of a noncaustic, nonalkaline compound in the eye.

Indications and Contraindications

Irrigation is indicated for all acute chemical injuries to the eyes. Irrigation may also be therapeutic for patients having an FB sensation with no visible FB. Small, unseen foreign material in the conjunctival tissues may be flushed out with irrigation. There is no contraindication to eye irrigation, but if there is also a possible perforating injury to the eye, the irrigation must be performed especially gently and carefully.

Equipment

The following equipment is necessary for eye irrigation:

- Topical anesthetic, such as proparacaine 0.5%
- Sterile irrigating solution (warmed intravenous saline or lactated Ringer's [LR] solution in a bag with tubing) *
- A basin to catch the fluid
- Cotton-tipped applicators
- Gauze pads to help hold the patient's lids open
- Lid retractors
- Irrigating device (e.g., Morgan Therapeutic Lens, modified central venous catheter, or Eye Irrigator) for prolonged irrigation

Procedure

Basic Technique

Topical anesthetic is first instilled in the eye. Any particulate matter should be swept out of the conjunctival fornices with

*A balanced salt solution designed for eye irrigation is preferred by some (when available) and may produce less corneal edema with chemical injuries.

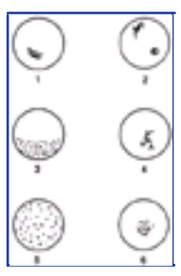

Figure 64-10 Patterns of acute corneal injury.

1. Traumatic abrasion: usually with linear features and sharp borders when seen early (< 24 hours). Occurs more in the central cornea.
2. Abrasion from foreign body (FB): vertical abrasions on the upper cornea seen when an FB is embedded in the upper lid. Also shown is a rust ring with a metallic FB.
3. Exposure pattern: seen with prolonged exposure to ultraviolet (e.g., welding flash, sunlamp exposure); produces bandlike keratitis over lower half of the cornea. Squinting in the setting of the bright light protected the upper corneal surface.
4. Herpes simplex keratitis: classic dendritic pattern.
5. Adenovirus keratitis: Diffuse minute corneal staining seen in epidemic keratoconjunctivitis about 7 days after symptoms.
6. Contact lens overuse: Central punctate staining.

(From Knopp, K, Trott A: Ophthalmologic procedures in the emergency department—Part III: Slit lamp use and foreign bodies. Acad Emerg Med 2:227, 1995. Reproduced by permission.)

moistened cotton-tipped applicators.[24] This requires eversion of the upper lid (see Ocular Foreign Body Removal later in this chapter and Fig. 64-11). During actual irrigation, the lids must be held open. It is easiest to use the gauze pads to get a

Figure 64-11 Double eversion of upper lid using a lid retractor. From Fogle JA, Spyker DA: Management of chemical and drug injury to the eye. *(In Haddad LM, Winchester JF (eds): Clinical Management of Poisoning and Drug Overdose, 2nd ed. Philadelphia, WB Saunders, 1990 Reproduced by permission.)*

grip on the wet, slippery lids. At times the patient has blepharospasm to such a degree that lid retractors (Desmarres or paper clip retractors; Fig. 64-12) may be necessary (Fig. 64-13). When lid retractors are used, the practitioner must be certain that the eye is well anesthetized, that the retractors do not injure the globe or the lids, and that chemicals are not harbored under the retractors. Retractors fashioned from metal paper clips (especially those that are nickel-plated and shiny) may have surface chipping, which can create an ocular FB.[35] Caution is needed to avert ocular injury when using such a makeshift retractor.

Deutsch and Feller recommend an ipsilateral facial nerve block for severe blepharospasm (Fig. 64-14).[24] To avoid swelling of the periorbital tissue, the facial nerve is blocked just anterior to the condyloid process of the ipsilateral mandible. A line of anesthesia (2% lidocaine) is placed subcutaneously to temporarily paralyze the *orbicularis* muscle.

The saline or LR solution exiting from the intravenous tubing is directed over the globe and into the upper and lower fornices. The choice of fluid initially is less important than the rapidity of irrigation initiation. Tap water should be readily available at the scene of the injury, and copious immediate irrigation should be encouraged before patient transport to the hospital. Out-of-hospital care providers should be taught to irrigate all acid injuries of the eye for at least 5 minutes at the scene and to irrigate all alkali injuries for at least 15 minutes.[36] [37] LR solution or normal saline are preferred over tap water or D_5W for eye irrigation, because they are isotonic and do not contain dextrose. Dextrose can be quite sticky if spilled and may serve as a nutrient for an opportunistic bacterial infection. Although one

clinical trial found a balanced salt solution less painful in patients with a chemical eye injury, [38A] another volunteer study on *uninjured* eyes found LR solution is better tolerated than normal saline and balanced saline solution when used with a morgan lens. [38B] Warmed fluids are also better tolerated than fluids at room temperature.[39] Warmed LR solution should be considered when both it and normal saline are available for eye irrigation.

Care should be taken to direct the irrigating stream onto the conjunctiva and then across the cornea without letting the stream splash directly onto the cornea, because the mechanical injury of the solution striking the eye can of itself be harmful. Direct irrigation of the cornea can result in the development of a superficial punctate epithelial keratopathy.

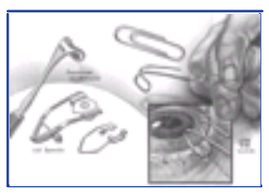

Figure 64-12 Devices for separating eyelids. A Desmarres retractor and a retractor improvised from a paper clip allow active manipulation of lids. Freestanding specula may require a seventh nerve block to reduce blepharospasm. *(From Fogle JA, Spyker DA: Management of chemical and drug injury to the eye. In Haddad LM, Winchester JF (eds): Clinical Management of Poisoning and Drug Overdose, 2nd ed. Philadelphia, WB Saunders, 1990. Reproduced by permission.)*

Duration of Irrigation

Although Deutsch and Feller recommend that a full liter of irrigating solution be used in every case of caustic injury, [24] the duration of the irrigation is best determined by the extent of exposure and the causative agent. Acids are quickly neutralized by the proteins of the eye surface tissues and, once irrigated out, cause no further damage. The only exceptions are hydrofluoric and heavy metal acids, which can penetrate through the cornea. Alkalis can penetrate rapidly and, if not removed (because of the slow dissociation of the cation from combination with proteins), will continue to produce damage for days. [24] Therefore, prolonged irrigation is indicated; at least 2 L of solution should be used. Although rapid flushing with the first 500 mL is prudent, slow continuous irrigation as discussed below at a rate sufficient to generate a continuous trickle is often more effective, and better tolerated than continued high volume flushing. If the nature of the offending agent is in doubt, prolonged irrigation should be used.

Ophthalmologic consultation should be obtained for all alkaline, hydrofluoric acid, and heavy metal acid injuries. Irrigation on an inpatient basis may be required for a period of 24 hours or more. This is especially likely when the cornea is hazy or obviously thickened. It should be noted that the magnesium contained in sparklers combines with water from tears to produce magnesium hydroxide. [40] Such fireworks injuries should be treated as alkaline injuries rather than thermal injuries. Eye damage from hair straighteners, [41] phosphate-free detergents,[42] and automobile airbags[43] must also be treated as alkaline injuries.

A good method of checking the effectiveness of irrigation is to measure the pH of the conjunctival fornices with a pH paper strip. The pH indicator on urine multi-indicator sticks can also be used. The pH indicator on urine dipsticks is conveniently closest to the handle; all the distal indicator squares can be cut off with scissors. The normal tear film pH is 7.4. The non-injured eye may be used as a control if the results are equivocal. If the pH measured in the conjunctival fornices after the initial irrigation is still abnormal, irrigation is to be continued. If the pH is normal after irrigation, *one should check it again in 20 minutes to make sure that it remains normal, especially in the presence of an alkaline contamination*.

Delayed pH changes are usually the result of incomplete irrigation and inadequate swabbing of the fornices. In anticipation of this deficiency, one should measure the pH deep in the fornices. Often, double-lid eversion with a lid elevator is required to expose the upper fornix for swabbing, irrigation, and pH testing (see Fig. 64-11).

Prolonged Irrigation

Prolonged irrigation may be required with alkaline burns. Ophthalmologic consultation is essential in such situations. One technique for prolonged irrigation uses a contact lens-type irrigation device (e.g., Morgan therapeutic lens), which allows for slow continuous irrigation once the more vigorous irrigation described earlier has been used. During use, the device is set on the anesthetized eye, and the lids are allowed to close around the intravenous tubing adaptor (Fig. 64-15). Continuous flow through the device onto the cornea and into

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Figure 64-13 Irrigation technique using a Desmarres retractor for lid separation Irrigation is performed with saline or lactated Ringer's solution. For alkaline injuries the pH of the eye should be checked with pH paper to assess the adequacy of irrigation (normal pH = 7.4). Since a rebound may occur with alkaline products, the pH should be rechecked 20 to 30 minutes *after irrigation* to assure complete removal. *(From Fogle JA, Spyker DA: Management of chemical and drug injury to the eye. In Haddad LM, Winchester JF: Clinical Management of Poisoning and Drug Overdose, 2nd ed. Philadelphia, WB Saunders, 1990. Reproduced by permission.)*

the fornices occurs. This contact lens device can become uncomfortable as local anesthetic agents are washed out during the irrigation process; the anesthetic agent must be reapplied frequently during irrigation for patient comfort. Such short-term use of local anesthetics will not inhibit healing of the cornea.

Another irrigation device can be made for the prolonged irrigation of the fornices by modification of a small-caliber central venous catheter. Multiple perforations are made with a scissors or a scalpel in the distal portion of the catheter. This perforated section is placed in the inferior fornix of the anesthetized eye beginning at the lateral canthus and is then looped back in the upper lid fornix (Fig. 64-16). The patient is instructed to tilt the head toward the side of the tubing to permit drainage into a laterally placed basin. After loosely applying an eye patch, one attaches the catheter to an intravenous setup for continuous irrigation. An anesthetic agent is also needed with this technique, albeit less frequently than with the contact lens-type device.

Yet another irrigation device, the Eye Irrigator, is designed to slide up easily under the upper lid (Fig. 64-17). This device is marketed for use at the injury site as part of the initial first aid to supplement flushing at the eyewash station.

Lippas described the successful treatment of two alkaline burns using a similar but more invasive continuous irrigation technique for prolonged irrigation after immediate copious irrigation.[44] Continuous irrigation of an alkaline injury with a 10% or 20% acetylcysteine (Mucomyst) solution to inhibit collagenase [45] and topical (1%) calcium gluconate eye drops[46] have been recommended, although these recommendations are controversial and poorly researched, and one should await ophthalmologic consultation before using these methods.

Complications

The only complication from irrigation is abrasion of the cornea or the conjunctiva. This can be a mechanical injury from trying to keep the lids open in an uncooperative patient, a small corneal epithelial defect from a Morgan irrigating lens or other irrigation device, or a fine punctate keratitis from the irrigation itself. [47] It is for this reason that the stream should not be directed onto the cornea. If a superficial corneal defect occurs, it is treated in the usual manner. Deep or penetrating corneal injuries are likely to be the result of the caustic chemical and require emergency ophthalmologic consultation. They should receive slow continuous irrigation pending ophthalmologic consultation. There is some experimental evidence that massive parenteral or oral ascorbic acid supplementation may prevent the development of deep corneal injury, [48] although this treatment has not gained universal acceptance.

Summary

Eye irrigation is easy, and complications associated with the technique are minimal. At times, the clinician may be unsure whether a chemical injury is toxic enough to warrant

Figure 64-14 Injection points for facial and orbital anesthesia and akinesia. *A,* Van Lint technique of orbicularis infiltration. *B,* Retrobulbar injection site. *C,* O'Brien facial nerve block. *C',* Alternative facial nerve block by tympanomastoid fissure injection. *D,* Infraorbital sensory block. *E,* Supraorbital sensory block. Injection of orbicularis *(A)* or facial nerve *(C* or *C')* permits examination and treatment of the eye in the setting of severe blepharospasm. Anesthesia is placed within several mm of the nerves. *(From Deutsch TA, Feller DB: Paton and Goldberg's Management of Ocular Injuries, 2nd ed. Philadelphia, WB Saunders, 1985, p 17.)*

Figure 64-15 *A,* The Morgan therapeutic lens attached to intravenous tubing. *B,* Placement of the device into the anesthetized eye for irrigation. *(Courtesy of MorTan, Inc, Missoula, MT.)*

irrigation. One should irrigate the eye if any doubt exists rather than omit this vital procedure and permit the progression of eye injury.

OCULAR FOREIGN BODY REMOVAL

Patients with an external FB in the eye are frequently seen in EDs. They are often in pain and desperate for help. A high degree of suspicion for FB injuries and perforations of the eye is necessary, because such injuries may be occult and not readily detected. Not all FB injuries are associated with pain; glass embedded in the cornea may be particularly difficult to detect. This section is a review of the procedures for locating and removing extraocular FBs. A brief discussion covering evaluation for perforation of the globe and for an intraocular FB is provided. Care of the patient after removal of an extraocular FB is presented.

Indications and Contraindications

Removal of extraocular FBs is always indicated. The timing of removal and the technique that is required vary according to the patient's clinical status and the type of injury. For the most part, the emergency clinician can proceed directly to removal of the object using the techniques described in this section. When the patient is extremely uncooperative (e.g., intoxicated, a mentally deficient individual or a young child) or when the injury is complicated (e.g., deeply embedded object, multiple foreign objects from a blast injury, or possible globe penetration), immediate ophthalmologic consultation is

Figure 64-16 Continuous irrigation device. A central venous catheter (after perforation by the clinician) is looped in the fornices of the anesthetized eye, beginning from the temporal border of the eyelids. The eye is lightly patched after catheter placement.

indicated. *Any patient presenting with a suspected FB or abrasion after exposure to a projectile (grinding wheel, hammering, metal objects colliding) should be rigorously evaluated for the presence of a deep intraocular FB.* See further discussion later in this chapter. A penetrating injury to the cornea is particularly troublesome in that iris tissue may prolapse and may appear to represent a corneal FB (Fig. 64-18). Hence, in addition to the history of projectile exposure, an irregular pupil, especially a pear-shaped pupil, should alert the clinician that a penetrating injury may have occurred.

Globe Protection

In the evaluation of a patient where a penetrating injury to the globe is suspected, a careful expeditious examination of the eye is indicated, preferably with a slit lamp. Care should be taken to avoid any pressure on the eye or rapid eye movements. If perforation is obvious (teardrop pupil, flaccid globe, flat anterior chamber, prolapsed iris) or confirmed by slit lamp (Seidel test positive—see Fig. 64-9 (Figure Not Available)), no procedures (save perhaps irrigation) should be attempted, and early ophthalmologic consultation is warranted for definitive diagnosis and care. Pending evaluation by the consultant, the eye should be protected from further harm by keeping the patient quiet,

Figure 64-17 The Eye Irrigator is designed to slide up under the upper lid as shown. Irrigation is accomplished by connecting the device to an intravenous bag containing the desired irrigation fluid. It is used primarily at the injury site to supplement flushing at the eyewash station. *(Courtesy of the Eye Irrigator, American Health and Safety, Inc.)*

elevating the head of the bed, and placing a protective shield over the eye. Commercial shields are available for this purpose. When a metal shield is not available, a makeshift shield can be fashioned with available materials (paper, plastic, or Styrofoam cups) (Fig. 64-19). The protective shield functions to avoid pressure on the globe and overlying tissues to avoid extrusion of vitreous and other ocular contents. The shield edges should extend to or beyond the bony orbital rim for this purpose. Adhesive tape is applied from forehead to cheek over the shield to secure the shield in position.

Patients with globe perforation should be treated with systemic antibiotics (a combination of cefazolin and gentamycin is a good initial choice), texnus toxoid, and antiemetics in doses aggressive enough to halt vomiting.

Equipment

The following equipment is necessary for extraocular FB removal:

- Topical anesthetic, such as proparacaine 0.5%
- Sterile cotton-tipped applicators
- Fluorescein strips
- Magnification: loupes plus a Wood's lamp or a slit lamp
- Eye spud or 25-ga needle attached to a 1- or 3-mL syringe or to the tip of a cotton-tipped applicator
- Dilator drops, such as homatropine 5%
- Antibiotic ointment, such as erythromycin

Consideration of Intraocular Foreign Body

The emergency clinician should always remain cognizant of the potential for an *intraorbital* or *intraocular* FB when examining the patient with a "FB" sensation. Penetrating injuries represent a greater threat of visual loss than extraocular FBs and can be disastrous if overlooked. *Intraocular FB, can be deceptively subtle on intial presentation.*

The clinical presentation is most helpful in the determination of which patients are at risk for a penetrating injury to the globe. An individual who complains of an FB sensation in the *absence* of trauma or one whose history is simply that something "fell" or "blew" into the eye is at *low* risk for a globe perforation. On the other hand, there is a greater probability of globe penetration in the individual who has sustained a high-velocity wound to the eye (e.g., drilling, hammering, grinding metal, blasting rock). The presence of any of the following physical findings should alert the clinician to a probable *intra*ocular FB: irregular pupil, shallow anterior chamber on slit lamp examination, prolapsed iris, positive Seidel test (see Fluorescein Examination), focal conjunctival swelling and hemorrhage, hyphema, lens opacification, and reduced intraocular pressure (IOP). Tonometry should not be performed in the presence of other physical findings suggesting penetration of the globe. One should be aware that a penetrating injury may *not* be associated with eye pain. Strong historical evidence and physical findings supporting a diagnosis of penetration

of the globe should prompt emergent ophthalmologic consultation.

Often, *intraocular* FBs are not visible on direct ophthalmoscopy. Although orbital radiography for radiopaque objects and ultrasonography of the globe have been used for indirect FB localization, [24] computed tomography of the orbit is now considered the most useful technique. [49] [50] When plain orbital radiography is performed looking for intraocular FBs,

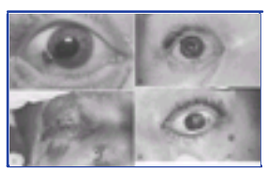

Figure 64-18 *A*, Corneal laceration with prolapse of the iris. The extruded iris is dark, mimicking a corneal foreign body. Often the only clue is an abnormal pupil and the extruded iris may not be appreciated as intraocular tissue. The pupil is irregular (often pear- or teardrop-shaped), pointing toward the laceration (photo courtesy of Lawrence B. Stack, MD). *B*, A pear-shaped pupil without protrusion of the lens is a more subtle, yet characteristic, indication of a perforated globe. *C*, Another indication of a penetrating globe injury is periorbital fat protruding from an upper eyelid laceration. This patient was stabbed by a knife. *D*, This patient has an obviously cloudy lens soon after trauma. A projectile entered the temporal portion of the globe and produced a seemingly minor scleral hemorrhage. Patients with penetrating injuries to the globe should be treated with systemic antibiotics (such as cefazolin/gentamycin combination), tetanus toxoid if indicated, and antiemetics to control vomiting (which raises intraocular pressure).

one must be aware that an eyelid FB may mimic an intraocular FB.[51] Patients with a suspected metallic FB should *not* undergo magnetic resonance imaging if the FB may be *intra*ocular. Therapy for intraocular and intraorbital FBs must be individualized. Often, an ophthalmologist can localize an intraocular FB (if the vitreous is clear) using indirect ophthalmoscopy. The role of the emergency clinician is to suspect the diagnosis, to protect the eye from further harm, and to obtain ophthalmologic consultation. The remainder of this section addresses the problem of extraocular FBs.

Procedure

Foreign Body Location

The first step is to locate the FB. A drop of both topical anesthetic and fluorescein is applied to the inside of the lower lid (see Fluorescein Examination Figure 64-8 , Figure 64-17 earlier in this chapter). Vertical corneal abrasions from FBs under the lids are helpful for localizing these hidden foreign objects (see Fig. 64-10*C*). One should use a penlight and loupes or a slit lamp to examine the bulbar conjunctiva by having the patient look in all directions. The clinician examines the inside of the lower lid by pulling it down with the thumb while the patient looks up. One everts the upper lid by having the patient look down as the end of an applicator stick is pressed against the superior edge of the tarsal plate of the upper lid. Meanwhile, the clinician grasps the lashes and pulls down, out, and then up to flip the lid over (Fig. 64-20).

Minute FBs under the lid will be missed with simple visual inspection. Ideally, the everted lid should be examined under magnification with loupes or a slit lamp. With simple lid eversion it is still not possible to see the far recesses of the upper conjunctival fornix. Although double eversion of the upper lid (see Fig. 64-11) is helpful, the best way to rule out an FB in the upper fornix is to sweep the anesthetized fornix with a moistened applicator as the upper lid is held everted. The applicator tip should be examined for removed foreign material. Small conjunctival FBs not hidden by the lids are often best removed with a moistened nasopharyngeal swab (e.g., nasopharyngeal Calgiswab).

The cornea is then reexamined. Most corneal FBs have an area of fluorescein staining around them. A slit lamp makes the examination easier. If the clinician is limited to loupes and a penlight, the light is shined diagonally on the cornea. One then finds an FB directly or indirectly by noting a shadow on the cornea or the iris (Fig. 64-21).

With a history of a high-speed projectile hitting the eye, one must rule out an intraocular FB. In the case of a blast injury, multiple FBs may penetrate the eye. If an FB cannot be found

Figure 64-19 When a penetrating globe injury is suspected and a metal shield is not available in the emergency department or prehospital setting, a makeshift shield can be fashioned with available materials. A paper cup was used to fashion this shield.

on the surface despite a suggestive history, the eye should be examined for physical evidence of penetration as discussed earlier. The pupil is dilated, and the fundus is examined. If in doubt regarding an intraocular FB, one should consider computed tomography and ophthalmologic consultation.

Foreign Body Removal

Once an extraocular FB is located, the technique of removal depends on whether it is embedded. If the FB is lying on the

Figure 64-20 Single upper lid eversion. *A,* The patient is instructed to look downward, and the end of a cotton-tipped applicator is placed above the tarsal plate while the lashes and the lid margin are pulled down, out and up. *B,* One everts the lid and holds it by pressing the lashes against the superior orbital rim. If the foreign body is seen on the lid, it can usually be removed by touching it with a moistened cotton-tipped applicator. *(From Pavan-Langston D: Manual of Ocular Diagnosis and Therapy, 3rd ed. Boston, Little, Brown & Co, 1991. Reproduced by permission.)* *C*, This patient presented with a swollen and tender upper eyelid thought to be secondary to a stye. *D*, With lid eversion a small pustule was found under the upper eyelid. *E*, With a 27-gauge needle the pustule was incised and a drop of pus was expressed, and she made a rapid and uneventful recovery.

surface, a stream of water ejected from a syringe through a plastic catheter usually washes the object onto the bulbar conjunctiva. Once the FB is on the inner lid or bulbar conjunctiva, a wetted cotton-tipped applicator can be gently touched to the conjunctiva and the object will adhere to the applicator tip. Overzealous use of an applicator for corneal FB removal can lead to extensive corneal epithelial injury. A spud device is required for removal of objects that cannot be irrigated off the cornea.

Embedded corneal FBs (Fig. 64-22) are best removed with a commercial spud device, a Burr drill, a short 25- or 27-ga needle on a small-diameter syringe (e.g., insulin or "TB" syringe), or a cotton-tipped applicator (Fig. 64-23*A* and *B*). The applicator or the syringe serves as a handle for the attached needle. Contrary to what one might expect, it is difficult to penetrate the sclera or the cornea with a needle, especially when it is applied tangentially to the cornea. [39] As with removal of conjunctival FBs, the eye must be well anesthetized. The patient should be positioned so that the head is well secured (preferably in a slit lamp frame). At this point a simple explanation of the procedure usually ensures excellent compliance on the part of the patient. The examiner's hand rests on the patient's cheek so that unexpected movements on the part of the patient will not result in large movements of the removal device. The patient must be instructed to gaze at an object in the distance (e.g., the practitioner's ear when a slit lamp is used) to stabilize the eye further. The removal device is brought close to the eye *under direct vision*; then, while it is in focus, the device is manipulated under the slit lamp to remove the FB. The device is held *tangentially* to the globe, and the foreign object is picked or

scooped out. If a Burr drill

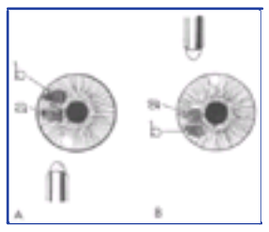

Figure 64-21 An "invisible" corneal abrasion casts an obvious shadow on the iris. The relative positions of *(a)* the corneal abrasion and *(b)* the iris shadow depend on the direction of the incident light (as in *A* and *B*). *(From Paton D, Goldberg MF: Management of Ocular Injuries. Philadelphia, WB Saunders, 1976. Reproduced by permission.)*

is used, the side of the drill is pressed against the FB until removal is accomplished (Fig. 64-23*C*).

During removal, the clinician should brace his or her hand against the patient's face. It also may be helpful to brace the elbow with a pad or half-full tissue box to provide further support to the arm as the FB is removed. The right-handed clinician should place the lower hand against the left maxillary bone when removing a foreign object from the patient's left eye and against the bridge of the patient's nose or infranasal area when removing an object from the right eye. These positions should be reversed by the left-handed clinician. The use of loupes or a slit lamp for magnification is highly recommended to minimize further injury during removal. In particular, corneal contact with the spud device is more readily discerned when magnification is used. Only topical anesthesia is required to remove FBs from the cornea. Although the patient may feel pressure during FB removal, pain should not be felt after the eye is anesthetized.

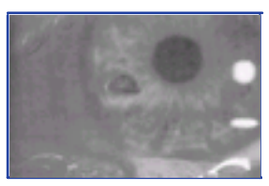

Figure 64-22 This embedded corneal foreign body is readily seen under slit lamp examination. A removal device (needle, spud, or burr drill) should be used for careful removal.

Rust Rings

A common problem with metallic FBs is the development of rust rings (Fig. 64-24). These can develop within hours because of oxidation of the iron in the FB. There are two preferred techniques for removal of a rust ring. The most direct method is to remove it at the same time as the FB, either with repeated picking away with a spud device or with a rotating burr. The second approach is to let the iron of the rust ring oxidize and kill the surrounding epithelial cells during a 24- to 48-hour patching period. At that time, the rust ring will be soft and often comes out in one solid plug. [52] Generally, a small rust ring produces little visual difficulty unless it is directly in the line of sight. The rust ring, if large, may delay corneal healing. Close follow-up is important to ensure healing of the cornea and total removal of the rust ring and FB.

Multiple Foreign Bodies

The patient with multiple FBs in the eye, such as from an explosion, should be referred to an ophthalmologist. A technique that may be chosen by the ophthalmologist is to denude the entire epithelium with alcohol and remove the superficial FBs. The deeper ones gradually work their way to the surface, sometimes years later.

Aftercare

After removing the FB, an antibiotic ointment is frequently instilled. Although commonly used, the value of the ointment for *superficial* corneal defects after FB removal is unproven, and no specific standard of care is supported by scientific evidence. *Conjunctival* and corneal abrasions do not need patching, but if the eye is patched, the patient should be reexamined in 24 hours. Data suggest that eye patching offers no benefit in healing small, uncomplicated corneal abrasions secondary to FBs.[53] If the patient sustains a *superficial* injury from the FB, the clinician may wish to instruct him or her to return only if the eye does *not* feel completely normal *or* if there is any blurred vision. The majority of superficial injuries heal without difficulty. The patient should be warned that the FB sensation may return temporarily when the anesthetic agent has worn off. One animal study with direct ocular exposure to *Clostridium tetani* organisms suggests that nonpenetrating ocular injuries are unlikely to lead to tetanus.[54] Tetanus prophylaxis after a corneal FB removal is not standard, but it may be considered. However, tetanus prophylaxis appears essential for injuries that penetrate through the cornea or sclera.

Use of Ophthalmic Anesthetic Agents

Application of topical anesthetic agents can be both diagnostic and therapeutic. Relief of discomfort with topical anesthetic use often suggests, but does not ensure, a conjunctival or corneal injury. An ocular irritant may also be masked by the use of these agents. Classic teaching is that the anesthetic preparations should not be self-administered by patients. The absence of protective reflexes while the patient is under the effect of the medicine may encourage the patient to use the eye while an FB or a corneal infection inflicts further corneal injury.

Bartfield and coworkers found that the pain of instillation of 0.5% proparacaine was significantly less than that of 0.5% tetracaine. [55] As evident from Table 64-3 , the anesthetic

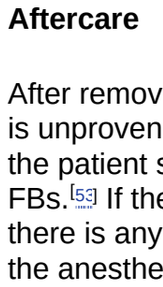

Figure 64-23 It is preferable to remove the corneal foreign body (FB) under the slit lamp. A topical anesthetic is applied and the operator uses a small syringe with a short 25- to 27-ga needle (such as a tuberculin syringe). Be certain that the needle is firmly attached to the syringe. *A*, *Under direct vision*, the syringe is brought close to the eye and the operator rests the hand on the patient's cheek. Be sure that the patient's forehead maintains continual contact with the cross bar on the slit lamp. *B,* While looking through the slit lamp, the needle is brought to the cornea and the FB is removed. *C,* The side of the instrument (drill bit or beveled edge of the needle) should be held tangential to the cornea. Arrows indicate the direction of device in order to remove the FB. *(From Knoop K, Trott A: Ophthalmologic procedures in the emergency department—Part III: Slit lamp use and foreign bodies. Acad Emerg Med 2:224, 1995. Reproduced by permission.)*

solutions that are commonly used have a duration of action of <20 minutes. The patient with a large corneal lesion may need a more extended period of pain relief. The discomfort associated with a large healing corneal lesion is usually made tolerable by bedrest, opioid analgesics, and appropriate sedatives. Even in the absence of infection or a retained FB, the long-term *repeated* use of ophthalmic anesthetic ointments may be detrimental to corneal healing. [56]

A final word of caution should be added regarding the use of ophthalmic solutions. Guaiac solutions are commonly supplied in dropper bottles similar in size and appearance to those containing ophthalmic solutions. Well-intentioned ED personnel may store the guaiac reagent bottles with the ophthalmic bottles. One should encourage both color-coding of the bottles and examination of them and their labels before each use to avoid corneal injury from inadvertently instilling guaiac

reagent in the eye.

Complications

Complications associated with ocular FB removal are rare. The most frequent problem is incomplete removal of the FB. In such cases the epithelium has difficulty healing over the affected area, and thus the eye stays inflamed. Eventually, the diseased epithelium either sloughs off and heals, or heals over the FB remnants, which are gradually absorbed. In either case, the adverse effects on the eye are minimal; a minute scar on the cornea, even directly in the center, will rarely affect the vision. Nonetheless, incomplete removal of a corneal foreign object warrants ophthalmologic follow-up.

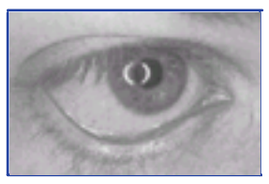

Figure 64-24 A typical rust ring is seen at 8 o'clock on the cornea. A burr drill can be used for attempted removal, which if unsuccessful, can be reattempted in 24 hours. *(From Knoop KJ, Stack LB, Storrow AB (eds). Atlas of Emergency Medicine, 2nd ed. New York, McGraw-Hill, 2002. Reproduced by permission.)*

Conjunctivitis may develop after removal of an extraocular FB. In most cases, the bacteria producing the infection are introduced by the patient through rubbing of the irritated eye.

Although perforation of the globe by the clinician's spud device is theoretically possible, this complication is exceedingly rare. Treatment of this type of corneal puncture wound consists of antibiotics, eye shield placement, and ophthalmologic consultation. In the absence of resultant endophthalmitis, permanent sequelae are unlikely to develop.

Epithelial injury can occur when cotton-tipped applicators are vigorously used to remove corneal FBs. Indeed, the use of cotton-tipped applicators for embedded *corneal* FB removal is condemned.

Summary

Ocular FBs are one of the most common eye emergencies. Searching for and removing the FB is usually straightforward. The only real trap is missing an intraocular FB. This must be ruled out if there is a history of a high-speed projectile hitting the eye or if physical findings suggestive of globe penetration are present.

Use of Ophthalmic Nonsteroidal Anti-Inflammatory Drugs

Ophthalmic nonsteroidal anti-inflammatory drugs (NSAIDs) have been evaluated for their effectiveness in the treatment of

TABLE 64-3 -- Ophthalmic Anesthetic Agents

Generic name	Tetracaine	Proparacaine	Benoxinate
Trade name	Pontocaine	Ophthaine, Ophthetic	Dorsacaine
Concentration	0.5 to 1.0%	0.5%	0.4%
Onset of anesthesia	< 1 min	< 20 sec	1–2 min
Duration of anesthesia	15–20 min	10–15 min	10–15 min
Comments	Marked stinging; also available in ointment	Least irritating; no cross-sensitization with other agents	Only anesthetic compatible with fluorescein in solution

traumatic corneal abrasions. Examples include ketorolac tromethamine, diclofenac, and flurbiprofen. These agents are safe to use and effective for the relief of pain associated with corneal abrasions. [57] [58] [59] [60]

EYE PATCHING

Patching the lids shut has traditionally been the last step in treatment of a number of common eye emergencies; however, multiple studies have shown that eye patching may offer no benefit in pain relief or healing rates with conjuctival or corneal abrasions. [53] [61] [62] [63] Many clinicians have only a vague idea of what the purpose of patching is, how to do it, and how to follow up. Even this simple procedure can be performed incorrectly.

Patching may be used for several goals. A simple patch may be used to protect the dilated eye from bright light. Pressure patching as discussed in this section is the use of a patch to hold the eyelid closed to possibly facilitate healing of a large corneal defect by limiting eyelid movement over an injured area.

It has been suggested in some studies that small, simple corneal abrasions do not require patching to aid healing or for reduction of pain. [64] [65] [66] A recent meta-analysis has elaborated on whether patching or not patching influences healing times or pain perception in corneal abrasions. The conclusion was that "eye patching was not found to improve healing rates or reduce pain in patients with corneal abrasions." [63] Collectively these studies support the treatment of small corneal defects without a pressure patch. The value of pressure patch application for large defects remains unclear. The patch may be of value until a bandage, soft-contact lens, or collagen corneal shield can be obtained at follow-up (see later sections on recurrent corneal erosions).

Indications and Contraindications

Patching was indicated in the recent past whenever the surface of the cornea had been injured; however, as mentioned earlier, there is little evidence to support its routine use. Nonetheless, practitioners may elect to patch an injured eye since the procedure has little downside. Injury can occur after a mechanical abrasion, such as a fingernail scratch, or after the removal of an FB. Chemical damage, damage from prolonged *hard* contact lens use, and ultraviolet light injuries are also commonly seen in the ED. With each of these forms of injury, if a patch is used, the purpose is to keep the lids from moving over the cornea and to keep light out. After patching, the patient may immediately experience less pain and tearing.

Patching is contraindicated when the corneal epithelial loss results from an active infection—such as a corneal ulcer—rather than from an abrasion. The first consideration in the differentiation between ulcer and abrasion is the history of the injury (how recently it occurred and how clean the offending object was). The second determining factor is the appearance of the cornea (i.e., an ulcer will have an infiltrate of white cells beneath the area of epithelial loss and will be accompanied by a purulent discharge). Generally, abrasions associated with *soft* contact lens use should be considered infected and patching avoided until ophthalmologic evaluation. [67] Of particular concern is the rapid development of *Pseudomonas* keratitis, an unusual but serious infection associated with corneal abrasions from soft contact lenses. A pressure patch should never be applied to an eye with a penetrating injury. A protective cup is the preferred covering in such cases pending ophthalmologic consultation.

Equipment

The following equipment is necessary for patching the eye:

- Gauze eye patches (2)
- Tape (e.g., 1-in. paper tape, preferably nonallergenic)
- Antibiotic ointment (e.g., sulfacetamide 10%)
- Dilator eyedrops (cyclopentolate [Cyclogyl] 1% or homatropine 5%)

The tape should be precut into 4- to 6-in. strips and should be kept within reach. Commercial patching products are also available and are discussed later.

Procedure

Before patching a corneal abrasion, one may apply both a dilator drop and an antibiotic ointment. While commonly used, there are no data to support or refute efficacy of routine pupillary dilation. The dilator must be a cycloplegic to relax the ciliary muscle spasm that accompanies corneal abrasions. Both cyclopentolate 1% and homatropine 5% last approximately 24 hours. The patient should be checked for a narrow

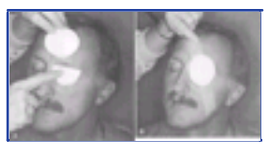

Figure 64-25 Application of eye patch. *A,* Vertically folded first patch in orbital recess. *B,* Horizontally oriented second patch with forehead to cheek taping. The contralateral eye should be kept closed throughout patching.

anterior chamber before the drop is instilled (see Fig. 64-4). Antibiotic ointment may be used prophylactically, although there are no data to support or refute efficacy. It is rare for abrasions to become infected. [68] In the past it was thought that the ointment vehicle would slow epithelial healing, but the vehicles that are currently used do not have this effect. [69] [70]

An effective patch must be put on tightly enough to keep the lids shut. The clinician should have the patient shut both eyes and should remind him or her to keep them shut throughout the entire procedure. Two patches are used. The vertically positioned first patch is doubly folded and placed over the closed lids. The unfolded second patch is then put horizontally over the first (Fig. 64-25). The tape strips are stretched diagonally from the center of the forehead to the cheekbone. The clinician can pull up the skin of the cheek; when he or she lets go, the tape will be even tighter. If the tape completely covers the patch, slippage of the patch and resultant eye movement are avoided (Fig. 64-26). The tape should not extend onto the angle of the mandible, because mastication loosens the tape in such a situation. Some clinicians paint the skin about the eye with tincture of benzoin to help secure the tape. Care must be taken not to introduce the benzoin into the eye. The presence of extensive facial hairs may prevent tight taping of the patch. An adjustable elastic strap pressure patch (e.g., Presspatch II, Precision Therapeutics, Inc, Las Vegas, NV) may be useful in this setting (Fig. 64-27). The patient must be carefully instructed in the use of this commercial patch, and avoidance of excessive globe pressure from the elastic straps must be confirmed.

It is imprudent to leave a pressure patch on for >24 hours. When repatching is required, a clean patch should be applied within each 24 hours. Repatching prevents a patch from becoming moist and serving as a nidus for the development of infection.

The Donaldson Eyepatch (Keeler Instruments, Inc, Broomall, PA) is a commercial product used for enforced eyelid closure that avoids the bulkiness of pressure patching (Fig. 64-28). The device has two components: one adheres to the upper lid, and the other is a circle that adheres to the


Figure 64-26 Final appearance of pressure eye patch.

ipsilateral cheek. A tab on the upper component connects by Velcro to the lower component. This patch may be advantageous in some settings, but its design may encourage some individuals to release the lid closure prematurely.

Because of conjugate eye movement, patching only one eye does not totally immobilize the globe. When all movement must be stopped, bilateral eye patches should be applied. The use of bilateral patches may not be accepted by all patients, and one must seek both patient understanding and family support for patient home activities if dual eye patching is to be successful.

Complications

There are few complications involved in patching. It is possible to patch the patient's lashes in between the lids so that they abrade the cornea. This can occur if the patient partially


Figure 64-27 Use of the adjustable elastic strap pressure patch, Presspatch II. The clinician should ensure that the elastic straps for the patch are adjusted to avoid excessive globe pressure. *(Courtesy of Precision Therapeutics, Inc, Las Vegas, NV.)*


Figure 64-28 Donaldson eye patch (Keeler Instruments, Inc, Broomall, PA). *Inset* shows the means by which the patch can be released for eye inspection or medication administration.

opens the eye during the procedure. This can be avoided by insisting that both eyes stay closed during the entire patching.

Most problems develop when the eye is not securely patched and excessive lid motion occurs. In this situation the corneal epithelial cells are not permitted to migrate over and close the epithelial defect. This leads to increased pain and delayed healing. Some corneal defects are extensive and may require 3 to 5 days for healing. Patients with extensive injuries should be observed frequently and treated with cycloplegic agents, pain medication, and (if clinically indicated) sedatives for sleep. The practitioner should document the size of the corneal defect at each visit. If healing does not occur in a progressive fashion, ophthalmologic consultation should be obtained. As mentioned earlier, patching is contraindicated when a corneal ulcer is present, the possibility of infection is high, or there has been soft contact use. A corneal "abrasion" that does not heal could very well be a herpetic ulcer.

Recurrent Corneal Erosions

Patients whose eyes have been patched for a corneal abrasion may experience *recurrent corneal erosions* in the future as a complication of the original injury. The original abrasion may appear to have healed perfectly, but days, weeks, or even months later a small area of corneal epithelium can come off, re-creating the symptoms of the original abrasion in the absence of new trauma. This usually occurs in the mornings as the patient opens the eyes, presumably due to adherence of the weaker, recently healed corneal epithelium to the upper lid during sleep. These erosions may heal before the patient is reexamined and can be very puzzling. The cause is

a failure of bonding of the corneal epithelium to its basement membrane. [71] Patients who develop this syndrome are given 5% sodium chloride ointment to use nightly to prevent the erosions; some require bandage soft (collagen) contact lenses. [72]

Summary

Patching is generally not necessary in the treatment of corneal epithelial defects, but if performed, should be an easy, straightforward procedure. A common problem is the lack of follow-up instructions given to the patient after the patching. Too often, the patient is given a bottle of antibiotic drops to be used every 4 hours and only vague recommendations for a follow-up check. When patients remove the patch to put in an antibiotic drop, they are never able to replace the patch properly again and may actually deter any healing that has already taken place. Instead, they should be told to keep the patch on for 24 hours and to return to have their abrasion checked by a clinician after that time. Generally a large abrasion will take several days to heal. Serial assessment is valuable for patient reassurance and assuring that complications do not develop.

The patient with an eye patch should be instructed to rest the uninjured eye. Reading should be discouraged, because involuntary movement of the patched eye will result. Watching television from a distance of 10 feet or more promotes eye fixation and is acceptable. Distant vision is unaffected by patching, although a small degree of peripheral vision is lost on the patched side. Although the visual field is minimally affected, driving after a patch is placed is not advisable. Preferably, the patient should be driven home from the hospital to minimize the potential for patient injury. An elderly patient may require assistance with routine ambulation after eye patching. Use of collagen shields applied directly to the cornea may offer an attractive alternative to eye patches. [72] These devices have yet to be introduced into ED practice.

CONTACT LENS PROCEDURES

An estimated 24 million Americans wear a form of contact lenses. [73] Removal of these lenses in the ED may be required to permit further evaluation of the eye or to prevent injury from prolonged wear. Emergency clinicians also evaluate patients for "lost" contact lenses, which may be trapped under the upper lid. At times, the patient may request that the clinician remove a lens that he or she has failed to extract from the cornea. Corneal ulcers may occur in patients who wear contact lenses and may require prompt treatment. This section on contact lens procedures addresses these concerns and discusses injuries associated with removal attempts, the mechanism of injury from prolonged wear, and instructions to be given to patients at discharge.

The first contact lenses were scleral lenses made of glass. These lenses, covering the cornea as well as much of the surrounding sclera, are reported to have been in use from 1888 to 1948.[74] Glass corneal lenses (sitting entirely on the cornea) made by the Carl Zeiss Optical Works of Jena were first described in 1912. A practical synthetic scleral lens using methyl methacrylate rather than glass was discussed by Obrig and Mullen in 1938. [75] [76] In 1947, Tuohy redeveloped the corneal lens using methyl methacrylate. This was the forerunner of the current hard contact lens. [77] The development in Czechoslovakia of lenses made of soft gas-permeable polymers was reported in 1960.[78] These hydrogel (hydrophilic gelatinous-like) lenses have evolved into today's soft contact lenses. Soft contact lenses now come in a variety of types including extended and daily wear. Some extended-wear lenses are disposable. All lenses should be removed at least once a week.

Mechanism of Corneal Injury from Contact Lens Wear

Hard Contact Lenses

The oxygenation of the cornea is dependent on movement of oxygen-rich tears under the hard contact lens during blinking. During the "adaptation" phase of early wear, the wearer of hard contact lenses produces hypotonic tears as a result of mechanical irritation from the lens. [74] This results in corneal edema, which reduces subsequent tear flow under the lens during blinking. Overwearing a lens at this time leads to corneal ischemia, with superficial epithelial defects predominantly in the central corneal area (see Fig. 64-10*D*), where the least tear flow occurs. With adaptation, the tears become isotonic and the blinking rate normalizes, permitting increased wear time. During early adaptation blinking is more rapid than normal and then slows to a subnormal rate during late adaptation. Mucus delivery to the cornea in the tear film may also play an important role in maintaining corneal lubrication. Tight-fitting contact lenses may never permit good tear flow despite an adaptation phase; individuals with tightly fitted lenses may never be able to wear their original contact lenses for longer than 6 to 8 hours. Lenses that are excessively loose can also cause irritation by moving during blinking. Rough or cracked edges can cause corneal abrasions.

In the ED, the patient who presents with irritation caused by prolonged wear may be either a new or an adapted wearer. The adapted wearer may have been exposed to chemical irritants (e.g., smoke), which reduce the tonicity of tears and lead to corneal edema and decreased tear flow. Alternatively, the adapted wearer with irritation may have ingested sedatives (e.g., alcohol) or may have fallen asleep wearing the contact lenses, thus decreasing blinking and tear flow. Another possibility is that the patient may actually be wearing tight-fitting contact lenses that have never allowed true adaptation despite many months of wear.

The patient with the overwear syndrome usually awakens a few hours after removing the lenses. The patient experiences intense pain and tearing similar to that caused by an FB. The delay in the onset of symptoms until after removal of the lenses is caused by a temporary corneal anesthesia produced by the anoxic metabolic by-products that build up during extended lens wear. [79] A second factor is the slow passage of microcysts of edema, which are pushed up to the corneal surface by mitosis of the underlying cells. When the cysts break open on the surface, the corneal nerve endings are exposed. [80]

Most patients with the overwear syndrome can be managed with reassurance, frequent administration of artificial tears, oral analgesics, and advice to "wait it out" in a darkened room. Some patients require patching for comfort. A patient who has experienced no problems with contact lenses before an overwear episode can return to using the lenses after 2 or 3 days of wearing glasses but should be

advised to build up wearing time gradually. A patient who was having chronic problems with lens comfort before the episode should check with an ophthalmologist before using the contact lenses again.

Soft Contact Lenses

Although there is also oxygenation of the cornea by way of the tear film with soft contact lenses, only approximately one tenth of the flow behind the lens that occurs with a hard lens is present during soft contact wear. [74] The high degree of lens gas permeability permits the majority of oxygenation to occur directly through the lens. The hydrogel lens is more comfortable than the hard contact lens because lid motion over the lens is smooth. The minimization of lid and corneal irritation allows a more rapid adaptation phase because the initial reflex-induced tearing and blinking changes are reduced. Nonetheless, the lenses may still lead to corneal edema and secondary hypoxic epithelial changes if worn for an excessive period when blinking is inhibited. Some individuals can tolerate the lenses for extended periods and may on occasion sleep with the contact lenses in place, although this practice is not encouraged. Newer extended-wear hydrogel lenses (e.g., Permalens) permit wear for several days without injury. These lenses are not discernible from standard soft lenses on examination.

Although the acute overwear syndrome that occurs with hard contact lenses can also occur with soft lenses, it is infrequent. More commonly, ocular damage from soft contact lenses falls into one of the three following categories:

1. *Corneal neovascularization.* Often the patient is asymptomatic, but on slit lamp examination fine vessels are seen invading the peripheral cornea. The treatment is to have an ophthalmologist refit the patient with looser or thinner lenses or with contact lenses that are more gas permeable.
2. *Giant papillary conjunctivitis.*[81] The patient notes decreased lens tolerance and increased mucus production. On examination of the tarsal conjunctiva (best seen on eversion of the upper lid), large papillae are seen. These grossly appear as a cobblestoned surface. The treatment is to discontinue wearing the lenses until the process reverses and then have the lenses refitted.
3. *A sensitivity reaction to the contact lens solutions* (usually thimerosol or chlorhexidine). [82] [83] There is diffuse conjunctival injection and sometimes a superficial keratitis. The treatment is to switch to preservative-free saline with the use of heat sterilization. Often, the contact lenses will need to be replaced before lens wear can be resumed.

All of these problems with soft lenses have bilateral, subacute onsets and do not require emergency treatment. The only form of ocular damage associated with soft contact lenses that is a true emergency is a bacterial (often *Pseudomonas* or *Acanthamoeba* with soft contact lenses) or fungal corneal ulcer. [84] [85] [86] Because the nature of soft contact lenses is to absorb water, they can also absorb pathogens, which then can invade the cornea. This is especially true if the soft lens is worn continuously for extended periods of time. The patient presents with a painful, red eye with associated discharge and a white infiltrate on the cornea. Immediate ophthalmologic consultation is required for appropriate culturing and antimicrobial treatment. These infections can permanently affect the patient's visual acuity.

Indications for Removal

Removal of a contact lens is recommended in the following situations:

1. *Contact lens wearer with an altered state of consciousness.* The emergency clinician should always be aware that the patient with a depressed or acutely agitated sensorium may be unable to express the need to have his or her contact lenses removed. Furthermore, it is likely that patients with a depressed sensorium will have decreased lid motion. During the secondary survey of these patients, the emergency clinician should identify the presence of the lenses and should arrange for their removal and storage to prevent harm from excessive wear or possible accidental dislodgment at a later time. Without magnification, soft

contact lenses may be difficult to see. Examination with an obliquely directed penlight should reveal the edge of the soft lens 1 to 2 mm from the limbus on the bulbar conjunctiva.

2. *Eye trauma with lens in place.* After measurement of visual acuity with the patient's lenses in place, the lenses should be removed to permit more detailed examination of the cornea. Fluorescein may discolor hydrogel lenses; when possible, extended-wear lenses should be removed before the use of this chemical. After the dye is instilled, the eyes should be flushed with normal saline; at least 1 hour should pass before reinsertion. [74] The availability of single-use droppers of 0.35% fluorexon (Fluresoft) has permitted the safe staining of eyes when soft lenses are to be worn immediately after the examination. A limited eye irrigation after the use of fluorexon drops is still recommended before the reinsertion of soft contact lenses.
3. *Inability of the patient to remove the contact lens.* A patient may present with a hard contact lens that cannot be removed because of corneal edema from prolonged wear. Alternatively, the patient may present with a "lost" contact lens that is believed to be behind the upper lid. There is no urgency for contact removal in the out-of-hospital setting; hence, removal can wait until the patient has been evaluated by a clinician.

Contraindication to Removal

The only major problem with contact lens removal occurs when the cornea may have been perforated. In this case, the suction cup technique of removal described later is preferred.

Procedure

Hard Contact Lens Removal

A number of maneuvers have been devised for removal of the corneal lens. One technique is to first lean the patient's face over a table or a collecting cloth. The clinician pulls the lids temporally from the lateral palpebral margin to lock the lids against the contact lens edges. The patient should look toward the nose and then downward toward the chin. This movement works the lower eyelid under the lower lens edge and flips the lens off the eye. The technique requires a cooperative patient because the clinician must pull the patient's lids tightly against the edge of the contact lens. The movement of the patient's eye then flips the contact free.

In the unresponsive patient, a modification of the technique can be used while the patient is supine. The clinician

takes a more active role in lid movement using the following procedure: one thumb is placed on the upper eyelid and the other on the lower eyelid near the margin of each lid. With the lens centered over the cornea, the eyelids are opened until the lid margins are beyond the edges of the lens (Fig. 64-29*A*). The clinician then presses both eyelids gently but firmly on the globe of the eye and moves the lids so that they are barely touching the edges of the lens (Fig. 64-29*B*). One presses slightly harder on the lower lid to move it under the bottom edge of the lens. As the lower edge of the lens begins to tip away from the eye, the lids are moved together, allowing the lens to slide out to where it can be grasped (Fig. 64-29*C*). The clinician should remember to use clean hands (and preferably wear examination gloves that have been rinsed in tap water or saline) when removing the lens.

Alternatively, one can move the lens gently off the cornea using a cotton-tipped applicator to guide the lens onto the sclera, where the applicator tip can be forced under an edge of the lens to flip the contact loose. Topical anesthesia is indicated when using an applicator and the patient is awake. Care must be taken with this technique to avoid contact of the applicator with the cornea when the lens is moved off the eye. Perhaps the easiest technique is to use a moistened suction-tipped device and simply lift the lens off the cornea (Fig. 64-30).

Several lenses (those hard contact lenses that cover both the cornea and an amount of the sclera) can be removed by an exaggeration of the manual technique described earlier (Fig. 64-31). Elevation of the lens with a cotton-tipped applicator or a suction-tipped device is also an effective technique. Soft

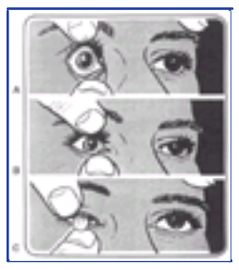

Figure 64-29 Manual technique for removing a hard contact lens. *A,* Separation of lids. *B,* Entrapment of lens edges with lids. *C,* Expulsion of lens by forcing of lower lid under inferior edge of lens. *(From Grant HD, Murray RH, Bergeron JF: Brady Emergency Care, 5th ed. Englewood Cliffs, NJ, Prentice Hall, 1990, p 338. Reproduced by permission.)*

Figure 64-30 Use of a moistened suction cup to remove a hard contact lens. *(From Grant HD, Murray RH, Bergeron JF: Brady Emergency Care, 5th ed. Englewood Cliffs, NJ, Prentice Hall, 1990, p 338. Reproduced by permission.)*

contact lenses should not be removed with a suction-tipped device because tearing or splitting of the lens may occur.

Soft Contact Lens Removal

With clean hands (preferably using gloves rinsed in saline or tap water), the clinician pulls down the lower eyelid using the middle finger. The tip of the index finger is placed on the lower edge of the lens. The lens is slid down onto the sclera and is compressed slightly between the thumb and the index finger. This pinching motion folds the lens and allows its removal from the eye (Fig. 64-32).

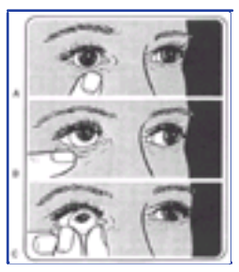

Figure 64-31 Removal of a hard scleral lens. *A,* Separation of lids. *B,* Forcing of lower lid beneath edge of scleral lens by temporal traction on lower lid. *C,* Lifting of lens off eye. *(From Grant HD, Murray RH, Bergeron JF: Brady Emergency Care, 5th ed. Englewood Cliffs, NJ, Prentice Hall, 1990, p 338. Reproduced by permission.)*

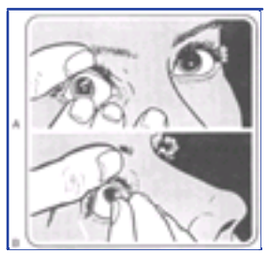

Figure 64-32 Removal of a soft contact lens. *A,* Separation of lids and movement of contact onto sclera using index finger. *B,* Pinching of lens between thumb and index finger. *(From Grant HD, Murray RH, Bergeron JF: Brady Emergency Care, 5th ed. Englewood Cliffs, NJ, Prentice Hall, 1990, p 338. Reproduced by permission.)*

Lens Storage

After a contact lens is removed, it should be stored in sterile normal saline solution. It is best to use the patient's own storage container and, if available, the patient's lens solution. A variety of alternative sterile containers are available for use in the ED. One should be certain that right and left lenses are kept separate and in appropriately labeled containers. The containers should be kept with the patient until a friend or family member can procure them, or they should be locked with the patient's valuables.

Evaluation of the "Lost Contact"

A patient may present with a request to be examined for a "lost" contact lens. The patient may be unsure if the lens is hidden under a lid, remains on the cornea, or is truly outside the eye. The evaluation of the patient with a "lost" contact should begin, as should all eye examinations, with the measurement of visual acuity. Visual acuity is preferably measured using a 20-foot eye chart. A diminished visual acuity in the eye in which a patient just cannot seem to "take out" a soft contact lens may be the most convincing evidence that the lens is missing. Although transparent, soft contact lenses in proper position are usually seen easily when viewed closely with loupes or on slit lamp examination. The lens forms a fine line where it ends on the sclera several millimeters peripherally to the limbus. Hard contact lenses are even more evident as they change in position on the cornea.

If the contact is not evident on initial inspection, the lids are everted as discussed in the section on FB removal (double eversion of the upper lid). If the lens is still not visible, a drop of topical anesthetic is placed in the eye. The upper fornix is gently swept with a moistened cotton-tipped applicator while the patient looks toward the chin. If the lens is still not evident even though the patient remains insistent that it is in the eye, one may perform a fluorescein examination after explaining that the dye will color the lens (permanently). The upper lid should again be doubly everted and visualized using an ultraviolet light source.

If the lens remains elusive, the patient should be reassured that a thorough examination was performed and that no object was located under the eyelids or on the cornea. The cornea should then be examined for defects that warrant antibiotic ointment and a pressure patch placed over the eye (as discussed in the section on patching). Follow-up with the patient's eye specialist for a replacement lens and further reassurance is encouraged. One also should ask the patient to retrace movements at the time the contact began to give trouble or was missed and to check the clothing being worn for the presence of the lens. A final possibility is that the patient may have accidentally placed the two lenses together in the same side of the carrying case, causing them to stick together. In fact, patients have inadvertently placed one contact lens over the other—both in the same eye. One should note that hard contact lenses have been found embedded in conjunctival tissue under the upper lid (Fig. 64-33) after more than a year.[87] [88] Hence, a methodical approach, as outlined earlier, should be taken to ensure that no lens remains hidden in the eye.

Complications of Lens Removal

Unless care is used during lens removal, a corneal abrasion can occur. It is difficult at times to determine whether the injury was produced by the patient or was a result of the clinician's technique. Fortunately, the corneal injury is usually of a superficial nature and responds well to eye patching, or other symptomatic care.

Summary

Contact lens removal is seldom a difficult task. More challenging situations are the identification of emergency patients at risk for corneal injury due to overuse, the evaluation of patients who cannot locate a soft lens, and the instruction of patients with contact lens-related problems concerning aftercare.

Figure 64-33 Hard contact lens embedded in conjunctival tissue of upper lid. *(From Mandell RB: Contact Lens Practice, 3rd ed. Springfield, IL, Charles C Thomas, 1981. Reproduced by permission.)*

INFECTIOUS KERATITIS

Infectious keratitis with corneal ulceration can have a variety of causes, including the overwear of contact lenses. Diagnosis of a corneal ulcer requires the use of a slit lamp and an accurate determination of the patient's history. Infectious keratitis is a frequent problem in ophthalmic practice. Herpes simplex is a common corneal pathogen. *Acanthamoeba* is another pathogen that is particularly associated with contact lens use and exposure to organism-tainted environments. Patients presenting with a corneal ulcer require prompt referral to an ophthalmologist. When immediate referral to an ophthalmologist is not possible, telephone guidance from the ophthalmologist and therapy needs to be initiated with ophthalmology follow-up in 24 hours or less.

Patients with herpes simplex keratitis often give a history of prior episodes of the disease. Patients who undergo almost any form of corneal stress may sustain an activation of preexisting corneal disease. Herpes simplex keratitis is classically recognized by its dendritic pattern on fluorescein staining.

Acanthamoeba keratitis is a disease with potentially devastating consequence. Its frequency seems to be increasing, particularly in contact lens wearers, and its pathophysiology is not completely understood. Patients often present with a red eye in which initial bacterial culture results are negative.

Bacterial keratitis occurs in a variety of settings. Organisms range from the relatively common *Staphylococcus* or *Streptococcus* to *Mycobacterium,* which can be difficult to identify. A variety of antibiotics are used against bacterial agents. Ciprofloxacin is a quinolone that has demonstrated efficacy against most of the common causative agents. Bacterial organisms in the cornea can develop resistance to any antibiotic and resistance to fluoroquinolones has been observed. [85] Ideally, treatment follows culturing of the ulcer.

In instances in which a cellular infiltrate is seen on slit lamp examination and in which there will be a delay of hours before an ophthalmologic consultant can culture the patient, it is prudent to initiate therapy with topical ciprofloxacin. In such circumstances, the emergency clinician may consider corneal cultures if suggested under the telephone guidance of the consultant before administering the antibiotic. One approach is to lightly touch a culture-moistened cotton-tipped swab against the ulcer and then streak standard culture media. If the ulcer is chronic or the patient is immunocompromised, fungal organisms may be culprit. Finally, a saline-moistened cotton-tipped swab may be used to obtain a Gram stain of the ulcer. Initiation of therapy before obtaining specimens for culture makes the subsequent identification of an organism difficult. For this reason the immediate initiation of treatment depends on the circumstances of the individual case.

TONOMETRY

Tonometry is the estimation of IOP obtained by measurement of the resistance of the eyeball to indentation of an applied force. Prolonged elevated IOP is associated with visual field loss and blindness. Sudden elevation of IOP can follow trauma or occur with primary angle-closure glaucoma. Often, patients with primary angle-closure glaucoma come to the ED with systemic complaints that include nausea, vomiting, and headache. The emergency clinician must determine the IOP and its relationship to the systemic symptoms. Occasionally, such patients are surprisingly free of pain in or about the eye.

Ophthalmologists depended on tactile estimation of eye pressure until the 1860s when von Graefe developed the first mechanical tonometer. [3] [22] Applanation tonometry was introduced in 1885 by Maklakoff[90] but was not popularized until Goldmann [91] improved the instrument in the 1930s. Schiotz developed an impression tonometer in 1905 and modified it in the 1920s; this form is still in use today. [92] Aside from modifications in configuration, current tonometers closely resemble the devices popularized by Schiotz and Goldmann. The most dramatic variations are the Mackay-Marg tonometer, [93] which permits a continuous tonographic recording, and the noncontact tonometer, which is a pneumatic applanation tonometer.[94] Pocket-sized tonometers using the MacKay-Marg tonometer principle are available. One such device is the Tono-Pen XL (Mentor O & O, Inc, Norwell, MA).[95] These devices are portable, lightweight, and relatively accurate, with built-in provisions for calibration. They have the advantage of a one-time-use replaceable cover that eliminates concern about the possible transmission of an infectious agent. While numerous devices are available, the Schiotz tonometer is the standard way for emergency clinicians to measure IOP.

Tonometric Techniques

Three tonometric techniques are reliable and clinically useful for estimating IOP:

1. The impression method uses a plunger (3 mm in diameter) to deform the cornea and the "indentation" is then measured. This technique was popularized by Schiotz and commonly bears his name.
2. The MacKay-Marg method is a refined version of the impression technique in which smaller amounts of cornea are indented.
3. In the applanation method a planar surface is pressed against the cornea.

One can either measure the pressure necessary to flatten a defined area or measure the size of a flattened area produced by the defined pressure. These tonometric techniques are based on the Imbert-Fick law, which states that if a plane surface is applied with force (F) to a thin, spheric membrane within which a pressure (P_t) exists, at equilibrium the expression $P_t = F/A$ is valid if A is the area of the applied surface (Fig. 64-34). The Schiotz tonometer (Fig. 64-35) actually measures the total IOP (initial pressure plus the pressure added by the weight of the tonometer and the plunger). Friedenwald empirically found that a "rigidity coefficient" could be introduced to allow an estimation of the true intraocular eye pressure. [96] One must be aware, however, that calculated conversion tables for Schiotz tonometers use an average estimate of the rigidity coefficient and hence are not accurate when eye rigidity is altered (e.g., after scleral buckle procedures for retinal detachment or with extreme myopia). Although the applanation tonometer (Fig. 64-36) also increases the IOP during measurement, the applied pressure is much smaller and is partially countered by the surface tension of the eye tear film. Emergency clinicians usually do not have experience or availability of this technique, but studies have shown applanation tonometer measurements to be within 2% of the true IOP.[97]

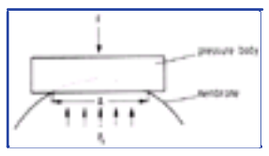

Figure 64-34 Principle of tonometry. At equilibrium: $P_t = F/A$. *(From Draeger J, Jessen K: Tonometry and tonography. In Bellows JG (ed): Glaucoma: Contemporary International Concepts. New York, Masson Publishing USA, 1979. Reproduced by permission.)*

The noncontact tonometer is a pneumatic applanation tonometer that permits IOP measurement without eye contact. A pulsed air jet is used to deform the cornea. The technique is also dependent on ocular rigidity. Although readings taken by different examiners correlate well, the measurements are altered by the use of local anesthetics and show a wide standard deviation of measurement in patients with pathologic elevation of ocular pressure (when standard applanation tonometry is used as a reference).[98] Furthermore, the technique is not useful with corneal surface irregularities (e.g., corneal edema, keratoconus, corneal perforation) or when medications in viscous preparations have been used. The use of this type of tonometer is not recommended when accurate determination of the IOP is required. This type of device is primarily useful for glaucoma screening.

Indications for Tonometry

Measurement of IOP in the ED by tonometry is a technique available to most emergency clinicians. Tonometry is not a standard procedure for many eye-related complaints, but special situations in which tonometry may be particularly helpful are as follows:

Confirmation of a clinical diagnosis of acute angle-closure glaucoma. The middle-aged or elderly patient who presents with acute aching pain in one eye, blurred vision (including "halos" around lights), and a red eye with a smoky cornea and a fixed midposition pupil obviously needs a pressure reading. Sometimes the findings are less dramatic, and

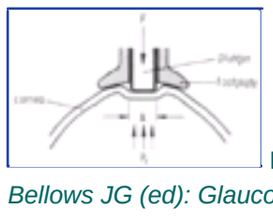

Figure 64-35 Principle of impression tonometry. In reality, P_t is increased slightly by the weight of the instrument. *(From Draeger J, Jessen K: Tonometry and tonography. In Bellows JG (ed): Glaucoma: Contemporary International Concepts. New York, Masson Publishing USA, 1979. Reproduced by permission.)*

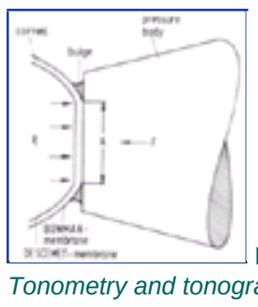

Figure 64-36 Principle of applanation tonometry. The effect of surface tension counters the pressure rise produced by application of the instrument. *(From Draeger J, Jessen K: Tonometry and tonography. In Bellows JG (ed): Glaucoma: Contemporary International Concepts. New York, Masson Publishing USA, 1979. Reproduced by permission.)*

sometimes the patient complains mostly of nausea and vomiting that suggest a "flu" rather than an eye disorder.

Determination of a baseline ocular pressure after blunt ocular injury. Patients with hyphema often have acute rises in IOP because of blood obstructing the trabecular meshwork.[99] Later, angle recession can cause a permanent form of open-angle glaucoma. Arts and coworkers suggest that an IOP >22 mm Hg or a

difference of 3 mm Hg or greater between eyes is a good marker for "ocular injury" in the setting of an orbital fracture. [100]

Tonometry may also be considered under the following scenarios:

Determination of a baseline ocular pressure in a patient with iritis. Patients with iritis can develop both open- and closed-angle glaucoma as well as corticosteroid-induced glaucoma. Since most cases of iritis are referred, tonometry may also be deferred unless there are signs of increased IOP.

Documentation of ocular pressure in the patient at risk for open-angle glaucoma. All patients older than 40 years with a familial history of open-angle glaucoma, optic disc changes, visual field defects, and pressures =21 mm Hg should be referred to an ophthalmologist for further work-up. Referral should also be made for those patients with suspiciously cupped discs who have normal pressures; some of these patients may have "low pressure" glaucoma associated with visual field defects. This is usually part of an ophthalmologist's examination.

Contraindications to Tonometry

Tonometry is relatively contraindicated in eyes that are infected unless one is using a device such as the Tono-Pen XL, which uses a sterilized cover. [2] One should sterilize a tonometer before and after applying it to a potentially infected eye. Infected eyes are preferably measured with either a noncontact tonometer or a device with a covered tip (e.g., Tono-Pen). The contact portions of any device should be swabbed with alcohol and allowed to dry before use on another eye. Not all viruses are destroyed by alcohol cleansing. Hydrogen peroxide is effective for deactivating the human immunodeficiency virus responsible for the acquired immunodeficiency syndrome (AIDS). Ultraviolet sterilization, cold-sterilizer

bathing of the footplate and plunger, and ethylene oxide sterilization have been advocated as alternatives to sterilize the Schiötz tonometer tip. The Schiötz tonometer is also used with sterile disposable coverings (marketed as Tonofilm). Nonetheless, measurement of IOP in an obviously infected eye can be deferred until a subsequent visit to the ED or private clinician unless the red eye demands an immediate determination of IOP.

Examples of indications for immediate tonometry in the setting of a red eye are suspected angle-closure glaucoma (acute onset of redness and pain in the eye with smoky vision, a cloudy cornea, and a fixed pupil in mid-dilation) and iritis (ciliary injection with photophobia), in which secondary angle-closure or corticosteroid-induced pressure changes may occur. Reported cases of conjunctivitis spread by tonometry predominantly tend to be viral infections. Particular efforts should be made to avoid use of the instrument on patients with active facial or ocular herpetic lesions or on patients who may have AIDS.

The presence of corneal defects also represents a relative contraindication to tonometry. [3] [22] The use of a tonometer on an abraded cornea may lead to further injury and is commonly deferred until a subsequent visit. Patients who cannot maintain a relaxed position (e.g., because of significant apprehension, blepharospasm, uncontrolled coughing, nystagmus, or uncontrolled singultus) are unlikely to permit an adequate examination and can receive corneal injury when sudden movements occur during an examination. Furthermore, tonometric examination, with the exception of the palpation technique (through the lids) and the noncontact method, should not be performed on a cornea without complete anesthesia.

Tonometry should *not* be performed with a suspected penetrating ocular injury. [2] Globe perforation may be exacerbated by pressure on the globe with resultant extrusion of intraocular contents. Slit lamp examination can be used for detection of a possible perforation.

Procedure

Palpation Technique

All forms of tonometry are essentially ways of determining the ease of deforming the eye; an eye that is easily deformed has a low pressure. The most direct way to do this is simply to press on the sclera through the lids and grossly compare one eye with the other. One can easily distinguish the rock-hard eye of acute glaucoma from the normal opposite eye by this method. The patient is directed to look down but without closing the lids. The examiner rests both hands upon the patient's forehead and alternately applies just enough digital pressure on the globe to indent it slightly with one index finger while feeling the compliance of the globe with the other (Fig. 64-37). [101] An experienced examiner is able to estimate the IOP within 3 to 5 mm Hg of the actual IOP with the palpation technique, but most emergency clinicians do not have enough experience to trust this method. [39]

Another method is to anesthetize the eyes topically and press a wetted applicator on the sclera of each eye. Again, eye deformation is inversely related to ocular pressure. Rigidity of the globe also is a factor in this crude method of tonometry.

Impression (Schiötz) Technique

Use of the Schiotz tonometer requires relaxation on the part of the patient and steadiness on the part of the clinician. The

Figure 64-37 The relatively unskilled examiner can detect very high intraocular pressure of acute angle closure glaucoma with tactile tonometry. The examiner rests both hands upon the patient's forehead and alternately applies just enough digital pressure on the globe to indent it slightly with one index finger while feeling the compliance of the globe with the other.

patient is placed in either a supine or a semirecumbent position and is instructed to gaze at a spot directly above the eyes. A spot on the ceiling should suffice; alternatively, the patient can stretch the arm up over the head and gaze at the thumb. A drop of topical anesthetic is placed in each eye. After the irritation of the drop passes, the patient is allowed to blink while the clinician blots the tears away with a tissue. Rubbing the eyes lowers IOP. The patient is reassured that further discomfort during the procedure will not occur.

The patient *keeps both eyes wide open and fixed on an object*, and the clinician separates the eyelids on the side to which he or she is standing. Care must be taken to direct pressure onto the orbital rims rather than into the orbit, because pressure directed into the orbit falsely raises the reading (Fig. 64-38). The tonometer is momentarily held over the open eye, and the patient is informed that the instrument will block vision in the one eye. The patient is instructed to continue to gaze at the fixation point as though the instrument were not there. After the patient relaxes the involuntary muscle contraction that occurs when the instrument is first placed in the line of sight, the instrument is gently lowered onto the middle portion of the cornea. This is a painless experience for the patient with an anesthetized cornea. The instrument is vertically aligned with the footplate resting on the cornea; the reading should be in mid-scale. Should the reading be on the low end of the scale (<5 units), additional weight should be added to the plunger after the instrument has been removed. The process is repeated as before with the additional weight.

The opposite eye is measured in the same fashion. A converted scale reading giving an IOP of =21 mm Hg requires ophthalmologic referral (Table 64-4). Patients with elevations of IOP =30 mm Hg require more urgent consultation and initiation of therapy. Associated symptoms or signs of angle-closure glaucoma (primary or secondary) represent an ophthalmologic emergency. [102]

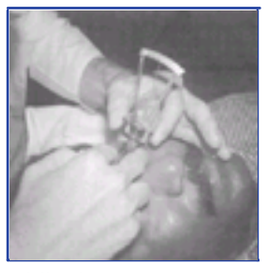

Figure 64-38 One technique of lid separation and Schiotz tonometer placement. Lid separation pressure is applied to the bony orbital rims. An assistant may separate the lids while the operator concentrates on proper placement of the tonometer. The tonometer is held vertically during use, and the clinician's hand is established against the patient's facial bones. After instillation of anesthetic drops, the patient will not experience any pain from this procedure. It is important to have a relaxed patient, since squinting and blepharospasm may interfere with the reading. *(From Keeney AH: Ocular Examination, 2nd ed. St Louis, CV Mosby, 1976. Reproduced by permission.)*

Errors with impression tonometry.

Inaccurate readings occur with the Schiötz tonometer for a variety of reasons. If the plunger is sticky, falsely low readings may be obtained. Plunger motion and the zero point of the tonometer should be checked on a firm test button before use. A sticky plunger can be cleaned with isopropyl alcohol and dried with a tissue. When

TABLE 64-4 -- Schiötz Tonometry

	Tonometer Weights (g)		
Tonometer Scale Reading (Units)	***5.5 (mm Hg)***	***7.5 (mm Hg)***	***10 (mm Hg)***
2.50	27	39	55
3.00	24	36	51
3.50	22	33	47
4.00	21	30	43
4.50	19	28	40
5.00	17	26	37
5.50	16	24	34
6.00	15	22	32
6.50	13	20	29
7.00	12	18	27
7.50	11	17	25
8.00	10	16	23
8.50	9	14	21
9.00	8	13	20
9.50	8	12	18
10.00	7	11	16

The table provides estimates of the intraocular pressure to the nearest mm Hg for the different weight of the Schiötz tonometer. Accuracy is most dependable with scale readings larger than 5. If the scale reading is less than 5, use the next highest weight that will give a reading of 5 or more.

the lids are held open, pressure directed into the orbit elevates the IOP and provides a falsely elevated reading. The following eye movements have been found to elevate the IOP: closure of the lids (increase by 5 mm Hg), blinking (increase by 5 to 10 mm Hg), accommodation (increase by 2 mm Hg), and looking toward the nose (increase by 5 to 10 mm Hg).[103] Repeated measurements or prolonged measurements have been found to lower the IOP approximately 2 mm Hg and may also lower the pressure in the opposite eye.[104] As mentioned in the introduction to this section, the calibration of the Schiötz tonometer is based on a mean rigidity coefficient. Factors that produce a reduction in ocular rigidity falsely lower the measured pressure. These factors include high myopia, anticholinesterase drugs, overhydration (e.g., four large cups of coffee or six cans of beer), and scleral buckle operations. [103] [105]

Ocular pressure measurements can vary with ocular perfusion. When measured after a premature ventricular contraction, the IOP may be reduced as much as 8 mm Hg.[106] Similarly, decreased venous return as produced by breath holding, the Valsalva maneuver, or a tight collar can increase the IOP. [103]

Impression (Tono-Pen XL) Technique

When using this device (Fig. 64-39), the preparations for testing are similar to those for the Schiötz device. The patient is encouraged to relax, and a topical anesthetic is used to numb the cornea. The patient should be encouraged to stare with both eyes at a distant object during testing. As noted previously, the operator can help separate the eyelids, but any pressure on the globe itself should be avoided. One major advantage to the use of the Tono-Pen XL is that the patient may be evaluated in any position as long as the device is applied perpendicular to the corneal surface. Another advantage is that the device can be used in cases of irregular or high corneal astigmatism.

Although ideally the complete instructions that are provided with the device kit should be consulted prior to each use, the following synoposis is provided to help in circumstances in which instructions are unavailable. It is assumed that the previously mentioned preparations for testing have been made before the device is used.

The probe tip is first sprayed with compressed gas to clean the mechanism and ensure its free movement. An Ocu-Film (latex) cover is placed snugly (but without tension) over the probe tip.

Calibration is performed before use at least once each day. The *activation* switch is momentarily depressed and released. The liquid crystal display (LCD) should show "—." If the device beeps and "====" appears on the LCD, the activation switch should be pushed again so that the

Figure 64-39 Bedside tonometry is easily accomplished with the Tono-Pen XL. The battery-powered device averages four consecutive readings and reports statistical reliability.

"—" reappears. If the prior calibration was "bAd" (on LCD), a long beep sounds, followed by "CAL" (on LCD). A short beep follows and then the desired "—" is displayed.

Once the "—" is displayed, the probe is held vertically with the tip pointing straight down. The activation switch is then pressed and released twice in rapid succession. Two beeps will then sound, and "CAL" will appear (on LCD). The probe is held in this position (up to 20 seconds) until a beep sounds and "-UP-" appears (on LCD). The probe is then immediately turned 180° so that the tip points straight up. In a few seconds, another beep occurs, and the LCD changes. If the LCD reads "Good,"

the calibration was successful. If the LCD reads "bAd," the calibration was unsuccessful.

With an unsuccessful calibration, repeat the calibration steps described earlier until two consecutive "Good" readings are obtained. If further attempts are unsuccessful, loosen the Ocu-Film tip cover and repeat the calibration process. If attempts are still unsuccessful, press the RESET button and repeat the process. If still unsuccessful, use compressed air to clean the probe tip and repeat the process. If still unsuccessful, the battery should be replaced and the process repeated. Continued failure warrants a call to the MENTOR Technical Service Group at 800-992-7557 or 617-871-6950.

Measurement occurs once the device is calibrated and the patient is prepared as outlined earlier. The activation switch is again depressed and released to obtain "====" (on LCD). A beep will occur with this change. If the switch is not depressed long enough, the LCD will be blank. If a blank screen is seen, again use the activation switch to obtain "====" (on LCD). The probe is held like a pen and briefly and lightly touched to the cornea (Fig. 64-40). This is done four times. A click will sound and a reading will appear on the LCD each time a valid reading is obtained. After four valid readings, a final beep will sound and the averaged measurement will appear on the LCD. The number represents the IOP in millimeters of mercury. The associated bar reflects the statistical reliability (a reading of > 20% reflects an unreliable measurement, which should be repeated).

If four dashes ("----") appear on the LCD after the final beep, too few valid readings were obtained. In such a case, the probe can be reactivated (without recalibration) and the


Figure 64-40 After topical anesthesia, the Tono-Pen XL is touched lightly and briefly to the cornea with a tapping motion, then withdrawn. Disposable tip covers are used to minimize any cross contamination.

measurement procedure repeated. If the probe is not reactivated within 20 seconds, the LCD will clear, but the device can be activated as noted previously without recalibration.

The values are interpreted as outlined earlier for the Schiötz device. Readings may be affected by the same features noted as causes of errors with impression tonometry via the Schiötz device. The device should be stored with an unused Ocu-Film cover protecting the probe tip.

Applanation Technique

One can perform this technique using a slit lamp attachment for an applanation tonometer with the patient's head stabilized in the headrest of the slit lamp (see the following section on the slit lamp examination) (Fig. 64-41).[107] [108] A portable device also is available and is similar in principle. The portable device is not discussed specifically.

The patient must be comfortable and relaxed. The clinician should anesthetize the eye as discussed previously, avoiding ocular pressure, which can lower the subsequent measurements. Fluorescein should be applied to each eye. Excess fluorescein should be blotted from the eye. The patient's head should be in the slit lamp with the forehead firmly against the headrest, and the clinician should direct the patient to gaze straight ahead. One can use a light for fixation or can ask the patient to focus on the clinician's ear on the side opposite the eye being examined.

The cobalt blue light filter is placed in the light beam, and the slit diaphragm is opened fully. The light arm is angulated to shine on the applanation prism in the region of the encircling black line near the anterior prism tip at an angle of 45°–60° to the line of observation. The voltage is turned to the maximum setting, and the low-power microscopic system is focused through the plastic prism so that the front face is


Figure 64-41 Goldmann applanation tonometer with the biprism aligned with the patient's right cornea.

clearly seen through the chosen eyepiece. The pressure knob of the tonometer is turned to 1 g (10 mm Hg), bringing the prism arm to its forward stop. Thus, when corneal contact is made, the prism will be exerting only light pressure. The room lights are dimmed.

The patient's eye that is being examined and the applanation prism are watched from the side (or with the operator's eye not sighting through the microscope) as the instrument is brought forward by the joystick control until gentle contact is made between the prism face and the corneal center. Contact is evidenced by an immediate bluish glow throughout the limbus. The patient's lids must be wide open and unblinking. Contact with the lid margins produces reflex blinking, and the lids may require separation by the clinician's fingers. Pressure during lid separation must be exerted only against the orbital rims. Through the microscope, the clinician sees two blue semicircles (surrounding the flattened area of cornea). Each semicircle is bordered by an arc of green light and pulses synchronously with the cardiac rate (Fig. 64-42).

The semicircles should be of equal size; their width should be approximately one tenth the diameter of the flattened surface contained within each arc. If the semicircles are grossly widened, either excessive tears are present or the prism was probably wet before contact. A wet prism must be withdrawn, dried, and reapplied. If the semicircles are grossly narrowed, the tear film has dried excessively. In this case, the prism must be withdrawn and the patient instructed to blink several times before contact with the cornea is attempted again. If the semicircles are so broad that they extend beyond the illuminate field, there is excessive flattening and the slit lamp must be drawn back. If the semicircles suddenly shrink, either the patient has moved back or the instrument has been backed away from the eye. The semicircles should be of equal extent above and below a horizontal dividing line. If the dividing line is not horizontal, the applanation prism assembly should be rotated on its holder until the line is horizontal. If the semicircles are not equally divided above and below the line, vertical adjustments of the slit lamp should be made.

Readings should be taken at approximately the midpoint between systole and diastole, when the inner (concave) boundaries of each semicircle rhythmically glide past each


Figure 64-42 Schematic representation of semicircles seen through the contact applanation prism of the Goldmann tonometer. *A,* Semicircles are too wide, suggesting excessive moistening of the prism or cornea. The prism must be withdrawn and dried. *B,* Semicircles are too narrow, suggesting that the lacrimal fluid has dried out, as during a prolonged measurement. The prism must be withdrawn so that the patient may blink a few times. The measurement is then repeated. *C,* Semicircles are of appropriate width, and their inner borders just touch. Cardiac pulsations transmitted through the globe cause rhythmic or pulsating movement of the semicircles over each other through a small amplitude. *D,* Semicircles are slightly separated, indicating applied pressure below that of the eye. The measuring drum must be turned to increase applanation pressure until the end point is reached. *(From Keeney AH: Ocular Examination, 2nd ed. St Louis, CV Mosby, 1976. Reproduced by permission.)*

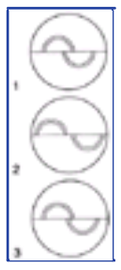

Figure 64-43 Appearance of the semicircles in applanation tonometry. Rotate the pressure knob to align the innermost concave margins of the two semicircles. IOP = intraocular pressure. (1) Pressure on the tonometer head is too high—rotate the knob to decrease reading. (2) Pressure on the tonometer head is too low—rotate the knob to increase reading. (3) Pressure on the tonometer head is equal to IOP—the dial reading equals IOP. *(From Knoop K, Trott A: Ophthalmologic procedures in the emergency department—Part III: Slit lamp use and foreign bodies. Acad Emerg Med 2:224, 1995. Reproduced by permission.)*

other through excursions of equal distance (see Fig. 64-42*C*). One finalizes adjustments to the end point of properly located and sized semicircles by rotating the pressure knob back and forth. When applanation pressure exceeds IOP, the semicircles are too small to intersect (Fig. 64-43).

At the end point is a flattened disc area 3.06 mm in diameter within the 7-mm diameter of the prism face. Here the attractive surface tension of the tears toward the prism is counterbalanced by the elasticity, or springiness, of the cornea; at this point the grams of force applied through the prism (indicated on the pressure knob) are directly convertible (when multiplied by 10 into mmHg) to express IOP. With an

applanation tonometer, the average IOP in a seated adult is 14 to 17 mm Hg.

After use, the tonometer should be wiped dry and removed for storage if used infrequently in the ED. One should verify the pressure adjustment periodically using the test weight or metal balance bar supplied with the instrument.

Potential sources of error with the applanation tonometer are similar to those mentioned for the impression tonometer, with the exception that ocular rigidity is not a factor. Inaccuracies primarily result from ocular motion or tensing of the lids.

Complications

When tonometric instruments are used properly and reasonable precautions are taken, complications are unusual. The eye with preexisting corneal injury should be spared the additional trauma of tonometer placement. Corneal abrasions can be produced by ocular movement during testing. In particular, patients with uncontrollable nystagmus, singultus, or coughing or those who are extremely apprehensive should not be subjected to tonometry. Infection can be transmitted by the use of the instrument. Careful cleansing of the device and avoidance of tonometry in patients with obvious conjunctivitis, corneal ulcers, or active herpetic lesions should minimize the risk of spreading the infection to the unaffected eye or to subsequent patients. Although protective coverings can be placed over the tonometer contact, tonometry can usually be postponed in the aforementioned individuals until the risk of infection is minimal. Extrusion of ocular contents with penetrating injuries is a potential but rare complication.

Summary

Tonometry is an easily learned technique that should be used by the emergency clinician for the detection of elevated IOP. An elevated IOP in conjunction with physical findings suggestive of acute angle-closure glaucoma is an indication for therapy and consultation with an ophthalmologist. The baseline measurement of IOP will aid the ophthalmologist in subsequent evaluation of a referred patient. In addition, the emergency clinician can serve as a referral source for patients with elevated IOP who are suspected of having open-angle glaucoma. In particular, future drug therapy for systemic hypertension may be altered by the presence of concomitant intraocular hypertension. The emergency clinician who aggressively manages patients with hypertensive crises must also be aware of potential visual field defects when systemic blood pressure is vigorously lowered without concurrent lowering of IOP.

SLIT LAMP EXAMINATION

The slit lamp is an extremely useful instrument; it makes the examination of the anterior segment of the eye a pleasure. The instrument can reveal pathologic conditions that would otherwise be invisible. The slit lamp permits detailed evaluation of external eye injury and is the definitive tool for diagnosing anterior chamber hemorrhage and inflammation.

Since the 1800s, clinicians have searched for a better way both to magnify and to illuminate the anterior segment of the eye. In 1891, Aubert developed the first true binocular stereoscopic microscope. Then, in 1911, Gullstrand introduced a slit illuminator device. The microscope and the illuminator were combined by Henker in 1916; the result was the first true slit lamp. Goldmann improved the mechanical supports for the microscope and the illuminator and in 1937 marketed a slit lamp that closely resembles the device used today.[109]

Indications and Contraindications

The slit lamp can be used in the majority of eye examinations. It is especially useful in the ED for the diagnosis of corneal abrasions, FBs, and iritis. [39] The slit lamp facilitates FB removal and is also used in conjunction with most applanation tonometers. Although portable slit lamp instruments exist, emergency clinicians generally have access only to a stationary, upright device. Therefore, in the absence of a portable device, a slit lamp examination is contraindicated in patients who cannot tolerate an upright sitting position (e.g., those with orthostatic syncope).

Equipment

The slit lamp has three essential components: a binocular microscope mounted horizontally, a light source that can create a beam of variable width, and a mechanical assembly to immobilize the patient's head and to manipulate the microscope and the light source. The location and arrangement of the knobs that control these components vary in devices made by different manufacturers. Usually, by simply turning each knob and watching the results, one can quickly master a new machine. Figure 64-44 illustrates the location of the functional controls on one particular instrument.

The first knob that one should locate is the on/off switch for the entire machine. Often this switch incorporates or is adjacent to a rheostat that provides two or three different power settings. The lowest setting is adequate for routine examination and will preserve bulb life. One can use a high-intensity setting when examining the anterior chamber with a narrow slit beam. Often, these controls are located on a transformer placed beneath the table to which the slit lamp has been attached. The second knob that one should find is the locking nut for the mechanical assembly. This must be loosened for the assembly to be moved.

The patient should be comfortable while sitting with the head in the device. The patient's forehead should be firmly against the headrest, and the chin should be in the chinrest. By varying the table height and height of the chinrest, one should be able to maximize the comfort of the patient's neck and back. The chinrest should be adjusted to align the patient's eye level with the mark on the headrest support rods.

The binocular microscope has a control for varying the magnification. Usually low powers, such as 10× or 16×, are the most useful. A higher power is helpful when the anterior chamber is examined for cells and flare and when the cornea is examined in minute detail. The binocular interpupillary distance should be adjusted to match that of the examiner. One can focus the eyepieces by moving the instrument forward and backward until the narrowed vertical beam is sharpest on the patient's cornea when viewed with the unaided eye. Then, while viewing through each eyepiece individually, the clinician adjusts the focus of each to produce a sharp image of the anterior cornea.

Figure 64-44 Slit lamp controls. *(From Operating Instructions for Slit Lamp Microscopes. Marco Equipment, Inc., Jacksonville, FL.)*

The light source is mounted on a swinging arm. There are knobs to vary the width and the height of the light beam. There are also filters that can be "clicked" in; only white and blue filters are usually needed. The angle of the slit beam can be varied from vertical to horizontal. The vertical alignment is preferred for routine examinations in the ED.

Both the microscope and the light source are mounted on swivel arms, linked at their base to a movable table. One can change the position of this table by pushing on any part of it. For finer movements, the clinician uses a joystick. One can vary the height of the microscope and the light source by twisting either the joystick or a separate knob at the base, depending on the design of the instrument.

Procedure

There are 3 setups that every slit lamp operator must know.[107] [110] The first is for an overall screening of the anterior segment of the eye. For examination of the patient's right eye, the light source is swung to the examiner's left at a 45° angle while the microscope is directly in front of the eye. The slit beam is set at the maximum height and the minimum width using the white light. To scan across the patient's cornea, one first focuses the beam on the cornea by moving the entire base of the slit lamp forward and backward. One then moves the whole base left and right to scan across. The 45° angle between the microscope and the light source should be the default position. The most common mistake is to try to scan by swinging the arm of the light source in an arc; this does not work because the light beam will remain centered on the same point of the patient's eye. The examiner scans across at the level of the conjunctiva and the cornea and then pushes slightly forward on the base or joystick and scans at the level of the iris. The depth of the anterior chamber is easily appreciated with this low magnification setup (Fig. 64-45). When the depth of the anterior chamber is reduced, one should suspect a corneal perforation or a predisposition to angle-closure glaucoma.

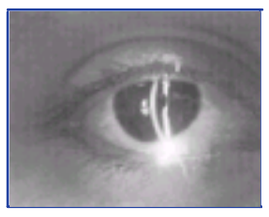

Figure 64-45 Slit lamp photograph of a normal right eye under low power. The curved slit of light on the left is reflected off the cornea while the slit on the right is reflected off the iris. The depth of the anterior chamber can easily be appreciated under this low magnification setup. *(Courtesy of D. Price.)*

This basic setup can also be used to examine the conjunctiva for traumatic lesions, inflammation, and FBs. The lids can be examined for hordeolum, blepharitis, or trichiasis. Complete lid eversion (as described earlier in the section on FB removal) can be performed in conjunction with the slit lamp examination to permit evaluation of the undersurface of the upper lid for FB retention.

Corneal FB removal can be enhanced by use of the slit lamp. In particular, the instrument allows stabilization of the patient's head. Magnification also minimizes corneal injury during FB or rust ring removal. The upper eyelid may be immobilized by a cotton-tipped applicator, as discussed previously. The clinician's hand can be steadied against the patient's nose, cheek, or forehead or against the support rods of the headrest. The patient should be instructed to stare straight ahead at a fixed light or at the clinician's ear during removal of the FB.

The second setup is essentially the same as the first but uses the blue filter. The purpose is to identify any areas of fluorescein staining. After fluorescein is applied, the blue filter is "clicked" into position, and the beam is widened to 3 or 4 mm. A patient can tolerate a wider beam without photophobia if it is blue. Corneal defects (as discussed earlier in the section on the fluorescein examination) are sought with this setup. The blue filter is also used with applanation tonometry, as discussed earlier in the section on tonometry.

The purpose of the third setup is to search for cells in the anterior chamber—either the white cells of iritis or the red cells of a microscopic hyphema. The height of the beam should be shortened to 3 or 4 mm and should be as narrow as possible. The microscope should be switched to high power. The beam is first focused on the center of the cornea and is then pushed forward slightly so that it is focused on the anterior surface of the lens. When the joystick is again pulled back to a focus point midway between the cornea and the lens, it will be focused on the anterior chamber (Fig. 64-46). One should keep the beam centered over the pupil so that there is a black background. Normally, the aqueous humor of the anterior chamber is totally clear. If small particles are visible floating up or down through the beam, these are usually circulating cells. If the beam lights up the aqueous like a searchlight in the fog, then the examiner has found the protein flare that accompanies iritis. Note should be made of the fact that

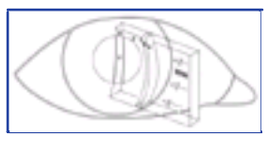

Figure 64-46 Appearance of the left eye during anterior chamber examination under low power: *a,* corneal epithelium; *b,* corneal stroma; *c,* corneal endothelium; *d,* anterior chamber (potential location of cells or flare); *e,* iris; *f,* lens reflection. The slit of light shines in the temporal to nasal direction at 45° to the anterior surface of the cornea. The depth of the cornea and anterior chamber examination are best done under high power in a dark room.

fluorescein can penetrate an abraded cornea, producing a fluorescein flare on slit-lamp evaluation. To avoid confusion, some clinicians prefer to examine for anterior chamber flare before the stain is used.

Summary

In practice, the 3 setups described here take only 1 minute per eye. Experience with the instrument enhances the ability of the user. The device is helpful for the evaluation of ocular infections and corneal lesions, the removal of corneal FBs, the measurement of IOP by applanation tonometry, and the diagnosis of iritis.

UNILATERAL LOSS OF VISION

There are a variety of reasons that an individual may sustain a complete loss of vision in one eye, but most commonly such loss may be related to occlusion of the central retinal vein or the central retinal artery or to optic nerve damage. Less commonly, pressure in the orbit from a retro-orbital hemorrhage may compromise the ophthalmic artery.

Although discussion of all the potential causes of unilateral loss of vision is beyond the scope of this text, amaurosis fugax deserves special mention. Amaurosis fugax is a transient loss of vision that is most commonly due to cholesterol or platelet emboli from atherosclerotic carotid occlusive disease. When plaques are visualized in the retinal vasculature, it is prudent to auscultate for carotid bruits and to refer the patient for ultrasound examination of the carotid artery. [111] [112]

Central Retinal Artery Occlusion

The patient with central retinal artery occlusion generally presents with a recent sudden (complete or nearly complete) unilateral vision loss. On examination there is an afferent pupillary defect (i.e., sluggish or nonreactive pupil in the affected eye with direct illumination with a normal consensual response) and reduced visual acuity. Immediately after the event, the fundus may appear nearly normal; however, it soon becomes pale and a classic "cherry-red spot" in the macula may be evident as a result of patent choroidal vessels showing through the transparent fovea.

Therapy

Visual recovery has been noted to occur up to 3 days after central retinal arterial obstruction. It has been recommended that treatment be started if the patient is seen within 24 hours after onset of symptoms.[113] Ophthalmologic consultation should be made while initiating therapy.

Most of the emergency techniques suggested to treat vascular insults to the eye in the ED are theoretically sound but are not supported or refuted by rigorous scientific data. No specific standard of care has been promulgated for these interventions by emergency clinicians. Techniques discussed below are likely safe and possibly useful, and may be attempted in an emergency situation. It is unknown whether or not these interventions will be vision saving.

Although of unknown value, slow *rebreathing* into a paper bag is believed to increase the arterial CO_2 level, thus aiding vasodilation and permitting the occlusion to move more peripherally, possibly reducing the ischemic area. The clinician should concurrently initiate *digital globe*

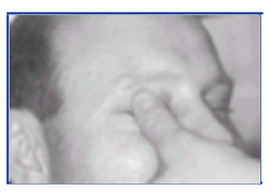

Figure 64-47 Digital globe massage is performed by applying firm steady pressure on the globe with the examiner's thumb for approximately 5 seconds, followed by abruptly releasing the pressure for 5 to 10 seconds. The process is repeated for up to 20 minutes or until improvement of vision is observed.

massage. With the patient lying supine, firm steady pressure is applied to the affected globe through the patient's closed lids using the clinician's thumb. The pressure is applied for 5 seconds and then abruptly released (Fig. 64-47). The procedure is immediately repeated several more times for up to 20 minutes. The technique is intended to help break up the occlusion and to encourage its movement more peripherally.

A more aggressive therapy, generally performed only by ophthalmologists, is *anterior chamber paracentesis*. In the absence of available consultation, this technique may be considered when central retinal occlusion is recent and unresponsive to the previously described therapeutic approaches. For this procedure, the patient is kept supine with the head and eyelids secured. The cornea is anesthetized with topical anesthetic drops (e.g., 0.5% proparacaine drops) and the conjunctiva is anesthetized. The conjunctiva is injected adjacent to the limbus using a 27- or 30-ga needle until the entire perilimbal area is infiltrated, giving the appearance of chemosis in all quadrants. During the remainder of the procedure, an assistant must firmly grasp the conjunctiva with toothless forceps at the 3 and 9 o'clock positions to stabilize the eye. A 30-ga needle on a tuberculin syringe is then inserted obliquely just adjacent to the limbus, at either the 4:30 or 7:30 o'clock position and directed toward the 6 o'clock position to avoid the lens (Fig. 64-48). After 1 to 2 drops of aqueous are expressed with gentle pressure on the globe, the needle is withdrawn.[114] [115]

One study describes a systematic approach in which ocular massage, sublingual isosorbide dinitrate 10 mg, acetazolamide 500 mg intravenously (IV), mannitol 20% (1 mg/kg) or oral glycerol 50% (1 mg/kg), anterior chamber paracentesis, methylprednisolone 500 mg IV, streptokinase 750 kIU, and retrobulbar tolazoline 50 mg were given until visual symptoms improved or until all steps were complete.[116] Of the 11 patients in this arm of the study, 8 had improved visual acuity. In those who improved, all had symptoms =12 hours. The presumed cause was either platelet-derived or cholesterol embolus from atheroma, or glaucoma. [115] Although this study is small, it supports emergent ophthalmology consultation and aggressive treatment of patients who present within 12 to 24 hours of symptom onset.

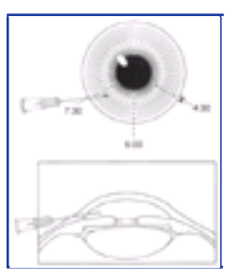

Figure 64-48 Anterior chamber paracentesis. After topical and subconjunctival anesthesia (see text), a 30-ga needle is directed obliquely from the 4:30 or 7:30 o'clock position toward the 6 o'clock position to avoid the lens. An assistant stabilizes the globe with forceps, grasping the conjunctiva (see text). *Top*, Anteroposterior projection. *Bottom*, Tangential projection. *(From Knoop K, Trott A: Ophthalmologic procedures in the emergency department: I. Immediate sight-saving procedures. Acad Emerg Med 1:408, 1994.)*

Complications

Overzealous globe massage has the potential to produce intraocular trauma including retinal detachment and intraocular hemorrhage. Anterior chamber paracentesis may produce hemorrhage, infection, or mechanical injury to the cornea, iris, or lens. [117] Although these complications are rare, ophthalmologic consultation for assistance with the underlying central retinal artery occlusion and surveillance for these potential complications should be initiated on an emergent basis.

Orbital Compartment Syndrome

Acute facial trauma or recent retrobulbar anesthesia may produce retrobulbar hemorrhage with sufficient pressure to compromise the ophthalmic artery, resulting in an orbital compartment syndrome. A form of post-traumatic glaucoma may also occur when the retrobulbar hematoma forces the globe against the eyelids. In this case IOP rises precipitously because the globe is in a relatively closed space due to the firm attachment of the eyelids to the orbital rim by the medial and lateral canthal ligaments. The optic nerve and its vascular supply, and the central retinal artery are compressed, resulting in ischemia and subsequent visual loss. In this situation an emergency canthotomy may be considered for relief of the pressure on the eye. It would not be considered a standard of care for most emergency

clinicians to possess the skills for this procedure, but under the proper scenario, it may be a prudent intervention.

Ophthalmoscopic evaluation reveals a blanched ophthalmic artery in the presence of obvious retrobulbar pressure and ecchymosis around the eye. The patient exhibits *decreased visual acuity,* and an afferent pupil defect is often seen. The IOP is markedly elevated but may be relieved by an emergency lateral canthotomy and cantholysis. Such a procedure needs to be performed quickly because the ischemic retina will not retain function if it is deprived of blood for a long period of time.

Technique: Lateral Canthotomy and Cantholysis

The goals of the procedure are to release pressure on the globe and to decrease IOP enough to reinstitute retinal artery blood flow. Because retinal recovery is unlikely to occur if rapid relief of ischemia is not accomplished, taking time to clean the eye beyond simple saline cleansing of the lids and lateral canthus is ill-advised. While the patient's head and lids are stabilized, the lateral canthus is first anesthetized with injectable 1% to 2% lidocaine with epinephrine. A small hemostat is used to crush the lateral canthus for 1 to 2 minutes to minimize bleeding before incising the lateral canthus. The canthus is incised using iris or Steven's scissors, with precautions taken to avoid injury to the protruding globe (Fig. 64-49). The incision begins at the lateral canthus and extends toward the orbital rim. The superior and inferior crus of the lateral canthal tendon are found and released from the orbital rim. Some operators prefer to release the inferior crus and reassess the IOP before considering release of the superior crus. An instructional video of the procedure can be found on the World Wide Web at *www.brown.edu/Administration/Emergency_Medicine/eye.htm*. [118]

Complications

Although hemorrhage, infection, and mechanical injury may result from the procedure, these complications generally respond to therapy better than retinal injury from prolonged ischemia. Emergent ophthalmologic consultation should be obtained, although when the procedure is indicated, it may be considered by the emergency clinician. Lateral canthotomy incisions generally heal without suturing or significant scarring.

REDUCTION OF GLOBE LUXATION

Although luxation of the globe is uncommon, the emergency clinician should be aware of the condition and its mechanisms, know how to reduce the globe, and know when to prioritize ophthalmologic consultation. With luxation of the globe there is extreme proptosis, which permits the lids to slip behind the globe equator (Fig. 64-50). Subsequent spasm of the *orbicularis oculi* muscles sustains the luxation and limits extraocular movements. Traction on the optic nerve and retinal vessels may produce direct or indirect injury to the optic nerve and retina.

Luxation may be spontaneous, voluntary, or traumatic. A variety of conditions (e.g., orbital neoplasms, Graves disease, histiocytosis X, cerebral gumma, and craniofacial dysostoses) may predispose the patient to luxation. Triggering events include maneuvers increasing intraorbital pressure (e.g., the Valsalva maneuver), trauma to the orbit or forehead, or eyelid manipulation.

Indications and Contraindications

Early globe reduction is indicated to relieve symptoms and to minimize visual impairment. Attempts at reduction in the ED

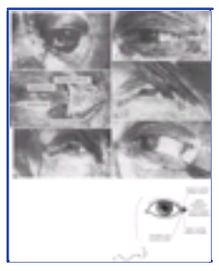

Figure 64-49 *A,* Severe proptosis secondary to acute traumatic retrobulbar hemorrhage. *B,* Anatomy of orbital structures demonstrating the inferior and superior crura of the lateral canthal tendon beneath the lateral canthus. The crura join and as a common tendon are attached to the inner aspect of the lateral orbital wall, forming Whitnall's tubercle. The lateral canthus, formed by the upper and lower eyelid, has been removed. *C, D,* A clamp crushes the lateral canthus to reduce bleeding when it is incised. The canthotomy allows the inferior crus to be exposed and cut to decompress the eyeball. *E,* A 1 centimeter horizontal incision is made in the lateral canthus, through the tissue that was compressed. *F,* The lower lid is pulled down and away from the lateral orbital rim, separating the skin and conjunctiva. If bleeding hinders identification of the inferior crus, it may be palpated. *G,* Only the inferior crus need be lysed initially. If intraocular pressure is not reduced, the superior crus is lysed. *(From S. Vassallo et al: Traumatic retrotubular hemorrhage: Emergent decompression by lateral canthotomy and cantholysis. J Emerg Med 22:21, 2002.)*

Figure 64-50 Appearance of bilateral luxated globe; superior (*top)* and lateral (*bottom*) projections. *(From Love JN, Bertram-Love JE: Luxation of the globe. Am J Emerg Med 11:61, 1993; original photographs courtesy of WR Green, MD, Wilmer Eye Institute, Baltimore.)*

are relatively contraindicated when there is obvious rupture of the globe.

Technique

Before globe reduction, it is valuable to perform a rapid eye examination to document visual acuity, range of eye motion, pupillary reactivity, and any evidence of globe rupture (see earlier discussion). [119] The patient is made comfortable in a recumbent position, and a topical ocular anesthetic agent (e.g., 0.5% proparacaine) is administered. When the lashes are visible, an assistant should apply steady outward and upward traction while the globe is gently pushed behind the lids. The globe is manipulated back into the orbit using gloved fingers to apply steady scleral pressure. When the lashes cannot be grasped, a lid retractor may be introduced behind the lid to provide countertraction. Others recommend placement of a suture through the anesthetized skin of each lid to provide countertraction.

After the procedure, a repeat eye examination documenting visual acuity and extraocular movement is warranted. It is not uncommon for return of full visual function to be delayed for several days, occasionally longer.

Complications

It is common with this procedure for lashes to be retained in the conjunctival fornices. It is important to evaluate for and remove any free lashes to prevent corneal injury. Edema, retrobulbar hemorrhage, or orbital deformity may prevent outpatient reduction. When reduction is not possible in the ED, saline drops should be applied to the globe and a noncontact eye shield applied.

Aftercare

Patients with spontaneous luxation and no visual impairment in whom the globe is easily reduced warrant follow-up within 24 to 48 hours. Instructions to avoid potential triggering maneuvers should be given. Recurrent luxation may warrant lateral tarsorrhaphy. Further evaluation of potential precipitating illness can be pursued on an outpatient basis.

Patients with traumatic luxation are at greater risk for underlying ophthalmic injury and warrant emergent consultation. A computed tomographic scan of the orbit is helpful for

evaluating both the soft tissue and bony structures about the globe.

Acknowledgment

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Chapter 65 - Otolaryngologic Procedures

Ralph J. Riviello

Examination of the oropharynx, larynx, ear canals, and nasal passages and the management of related acute disorders are most effectively performed using special equipment and techniques. This chapter addresses these techniques from the perspective of the emergency clinician, who often must assess injuries or illnesses of potential compromise to the airway and to auditory function. Decisions related to definitive treatment in the emergency department (ED) vs timely referral are addressed. Related topics, including airway management, esophageal and laryngeal foreign bodies (FBs), and assessment of caloric testing, are discussed elsewhere in the book.

PHARYNX/LARYNX

Examination of the Larynx

Several techniques to visualize the larynx are discussed. The clinician should become adept at examining the hypopharynx and larynx with more than one method (Fig. 65-1). Laryngoscopy is indicated for the evaluation of unexplained hoarseness, dysphagia, odynophagia, or FB sensation. The majority of patients will require a repeat examination by an otolaryngologist for verification, but laryngoscopy in the ED may identify pathologic conditions requiring more urgent consultation. Clinicians should follow guidelines for "universal precautions" (as described in Chapter 71) to protect themselves and their patients from infection transmitted by blood and body fluids.

Laryngoscopy has traditionally been discouraged in the patient with a high potential for epiglottitis, as oropharyngeal manipulation may in rare cases precipitate laryngospasm and acute respiratory arrest. However, some authors report that careful laryngoscopy may be performed in stridulous patients with the presumed diagnosis of croup to rule out epiglottitis when the suspicion for the latter condition is low. [1]

When impending airway obstruction from epiglottitis is suspected, the first priority is to quickly assemble a predesignated team (usually consisting of an anesthesiologist and an otolaryngologist) in the operating room. Any attempt at laryngoscopy should follow full preparation for rigid bronchoscopy or a surgical airway. Patients with severe laryngeal trauma or partially obstructing hypopharyngeal FBs should be approached in a similar manner.

Illumination

The reflected light from a head mirror or direct illumination from a head lamp can be used not only for indirect laryngoscopy, but also for inspection of, and procedures in, the oropharynx, nares, and auditory canal. For example, a peritonsillar abscess is afforded excellent illumination for drainage with such devices. The advantages of a head mirror are the high degree of brightness it provides into deep recesses and its simplicity of design. Generally, the beam of light from an electric head lamp is easier to focus than is the head mirror.

Procedure

Head mirror/Light source.

The head mirror is convex with a central hole that allows the examiner to see directly along the reflected light beam. Begin by swinging the mirror down over the dominant eye just touching the skin or glasses. Position the light source (a 150-watt bulb works well) over the patient's shoulder on the same side as the head mirror. Keeping your eyes open, adjust the focus of light by adjusting your distance from the patient. Change the direction of the beam by tilting the mirror or turning your head slightly.

Head lamp.

The electric head lamp also attaches by a forehead strap, and the light should be placed nearly between the examiner's eyes to be maximally effective. The intensity of light in this position is nearly as bright as the head mirror. After securing the head lamp, hold your hands in front of you at a distance that is comfortable for working. Focus the beam of light at this point by adjusting the head lamp into position without moving your head or eyes. This allows for the beam to shine on and follow the area on which your eyes are focused.

Indirect Laryngoscopy

This traditional method is most commonly used by the otolaryngologist, but it has some application in the emergency setting if the necessary equipment is readily available.

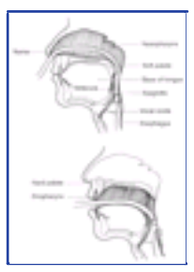

Figure 65-1 Anatomy of the oropharynx. Sagittal section of the neck. Also depicted is the use of the nasopharyngeal scope through the nasopharynx and the oropharynx.

The clinician who is unfamiliar with this method should practice frequently, as it requires significant eye-to-hand coordination to reflect the light beam off the angulated mirror onto the larynx. When this procedure is properly performed, most patients are able to tolerate it without anesthesia of the oropharynx. Fiberoptic nasopharyngoscopy has largely replaced indirect laryngoscopy in the ED when the equipment is available.

Begin by establishing rapport with the patient by explaining how the examination will be performed. Have the patient sit erect in the "sniffing position," with the feet flat on the floor and leaning slightly forward. Warm the mirror with warm water or in a flame to prevent fogging, but check the temperature of the mirror with your hand before placing it into the oropharynx so as not to burn the patient. Alternatively, anti-fogging solutions can be applied to the mirrored side. Wrap the patient's tongue with gauze to prevent it from slipping or being injured by the lower incisors and then grasp it with the nondominant hand (Fig. 65-2). Apply gentle traction to the tongue with your thumb and index finger while lifting the patient's upper lip with your middle finger. Slide the mirror into the oropharynx with the glass surface parallel to the tongue but not touching it. Place the back of the mirror against the uvula and soft palate, smoothly lifting until the larynx is visualized. While this should not induce gagging, try to make only slight changes in mirror position to inspect the appropriate structures.

In patients who cannot tolerate this procedure without gagging, apply topical anesthetic to aid in the examination. Benzocaine (Hurricaine spray or Cetacaine gargle) or aerosolized tetracaine or lidocaine may be used. One or two quick sprays of benzocaine into the posterior oropharynx are sufficient. Prolonged or repeated spraying may rarely result in methemoglobinemia. Reassure the patient beforehand that although this may make the throat feel as if it is swelling or paralyzed, in actuality it is just the numbness that accounts for

Figure 65-2 Indirect mirror evaluation of oropharynx. Grasp the patient's tongue between the thumb and first finger, using a gauze pad to provide traction. Elevate the upper lip with the middle finger. Advance the warmed laryngeal mirror into the posterior oropharynx, taking care not to stimulate the posterior tongue or pharynx. Remember that the structures in the mirror will

be reversed. Always use universal precautions.

the sensation. The tendency to gag also can be minimized by having the patient concentrate on his or her breathing efforts and keep the eyes open, with vision fixed on an object in the distance.

Once the patient is anesthetized, repeat the steps described earlier and position the mirror against the soft palate. Rotate the angle of the mirror and systematically inspect the base of the tongue, valleculae, epiglottis, pyriform recess, arytenoids, false and true vocal cords, and, if possible, the superior aspect of the trachea (Fig. 65-3). Observe for masses, evidence of infection, asymmetry, or FBs. Further evaluate the anterior structure of the larynx and function of the vocal cords by having the patient say "eeee" in a high-pitched voice. This should move the epiglottis away from blocking the view of the larynx and bring the true cords together at the midline.

Laryngoscopy by Angled Telescopes

A less frequently used method of examining the larynx in the emergency setting is by rigid angled laryngoscopy (e.g., LarynxVue II, Astralite Corp., Anaheim, CA). This provides a clear and more continuous view of the larynx during respiration, but its use is limited in patients whose epiglottis blocks the view. The light source is powered by batteries or a wall outlet. The degree of mirror angulation is fixed, but it may vary between instruments (usually 70° to 90°). At 70° of mirror angulation, the scope does not need to be placed as far posteriorly into the oropharynx to visualize the structures.

Position the patient as for indirect laryngoscopy and anesthetize the soft palate if necessary, as previously described. Gently grasp the tongue and slide the scope into the oropharynx. Stabilize the scope on the fingers that are holding the tongue, taking care not to touch the sensitive base of the tongue. Once the scope is touching the soft palate and is near proper position, look into the eyepiece and make final adjustments to bring the laryngeal structures into focus. Observe the anatomy and function as previously described in a systematic fashion.

Flexible Fiberoptic Laryngoscopy

Fiberoptic examination of the nasopharynx and larynx can be accomplished with either a flexible nasopharyngoscope or a bronchoscope. The nasopharyngoscope is thinner, shorter, and easier to manipulate (Fig. 65-4). Fiberoptic visualization is especially useful in patients who are difficult to examine because of persistent gagging or unusual anatomy.

Figure 65-3 View of the larynx from above. The true and false vocal cords are sketched, with the arytenoid eminences behind them on each side. The epiglottis, piriform fossae, and valleculae are also labeled.

Figure 65-4 Fiberoptic scope with light source.

Attach the endoscope to its light source, attach the suction to its port (if available), and ensure that both are functioning properly before beginning. Before inserting the scope, adjust the eyepiece to the operator's visual acuity; it is helpful to check the scope focus on newsprint or a small object at this time. Review of the scope's directional controls is also recommended. Examine the nares and choose the more patent one to enter. Anesthetize and vasoconstrict the naris with lidocaine and epinephrine (as described in Epistaxis, later in the chapter). Some clinicians also anesthetize the pharynx to minimize gagging. Warm the end of the scope in warm water to help prevent fogging. The patient should be seated, with the head placed against a headrest, in the "sniffing" position. Insert the tip of the lubricated scope just inside the naris. A series of soft nasal trumpets may be used to dilate the nasal cavity, allowing easier passage of the scope. The movement of the scope against the inside of the nasal passage may be irritating; this discomfort can be minimized by resting the fourth and fifth fingers on the bridge of the patient's nose while stabilizing and guiding the passage of the scope between the thumb and index finger (Fig. 65-5).

While looking through the eyepiece, slowly advance the endoscope past the inferior turbinate into the nasopharynx or through the lumen of the trumpet. To clear fogging or mucus off the lens, have the patient swallow, wipe the lens against the pharyngeal mucosa, or use the suction. Once it is in the nasopharynx, direct the tip inferiorly, using the thumb control near the eyepiece. *Up-and-down movements of the scope may be accomplished with the thumb control, whereas rotating the scope about its axis and then applying the thumb control provides for lateral movement and visualization.* At this point, the base of the tongue and tonsils will come into view. Slide the scope further caudad to bring the larynx into focus. Once again, systematically view the anatomy and function during both respiration and phonation.

If the nasopharyngeal scope will not pass through either naris, pass it through the oropharynx. Properly anesthetize the oropharynx and avoid the posterior tongue to prevent gagging. Cut a 10-mL syringe (without the plunger) in half. Have the patient hold this in the mouth between the incisors. Pass the fragile endoscope through this tube to prevent accidental biting of the scope.

Complications include traumatic abrasions and bleeding anywhere along the path of the laryngoscope. In patients with

Figure 65-5 Fiberoptic nasopharyngoscope in use. Prepare the patient's throat and nares with topical anesthetic. Topical vasoconstrictors may also be used in the nares. The use of a nasal trumpet is optional. Advance the scope slowly into the naris with the hand stabilized on the patient's nose; guide the scope using the thumb and index finger. Visualize the passage of the scope through the naris into the posterior nasopharynx. Always use universal precautions.

head injury, there is always the slight risk of passing the scope intracranially if a basilar skull fracture exists, but use of a soft nasal trumpet significantly reduces this risk. The induction of laryngospasm and acute airway compromise is possible in patients with paraglottic infections.

Peritonsillar Abscess

Anatomy

Peritonsillar abscess (PTA), also known as *quinsy*, is most common during the second and third decades. It is rarely seen in children younger than 6 years of age, making it diagnostically challenging in younger children and infants. It remains the most common head and neck abscess in children and adults. The incidence for PTA is about 45,000 cases annually in the United States. The relative anatomy must be understood to treat peritonsillar abscesses (Fig. 65-6). The palatine tonsils are located between the anterior and posterior pillars of the throat, bound in a capsule and covered by mucosa. The lateral wall of the tonsil is defined by the superior pharyngeal constrictor muscle. Of great importance is the *internal carotid artery,* which lies approximately 2.5 cm posterolateral to the tonsil.

Pathophysiology and Presentation

Peritonsillar abscesses occur in patients with inadequately treated tonsillitis and in recurrent tonsillitis. The abscess is usually unilateral and is defined as a collection of pus between the tonsillar capsule, the superior constrictor muscle, and the palatopharyngeus muscle. It is believed to arise from the spread of infection from the tonsil or from the mucous glands of Weber, located in the superior tonsillar pole. [2] The abscess is most commonly initiated from the upper pole of the tonsil. However, it can also spread from the middle or inferior poles. Complications may include pharyngeal obstruction or extension into the closely approximated neurovascular bundles and parapharyngeal space.

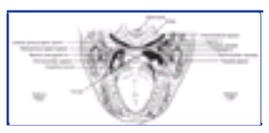

Figure 65-6 Anatomy of a peritonsillar abscess. The palatine tonsil and peritonsillar space are identified on the patient's right. A peritonsillar abscess is shown on the patient's left. Note that the abscess can extend medially, displacing the uvula. The carotid artery and jugular vein are posterior and lateral to the abscess. Avoid lateral angulation of the aspirating needle and use a needle guard to prevent injury.

Most patients present primarily with a peritonsillar abscess, but some have been previously treated for tonsillitis. There are no data proving that antibiotics, even the correct ones in proper doses, invariably prevent the progression of tonsillitis to abscess formation. Inadequately treated tonsillitis can progress to abscess when a patient fails to follow the prescribed regimen or when the regimen is inadequate. The latter may occur as the result of an improperly chosen antibiotic or because of increasing antibiotic resistance. Although penicillin still remains the best initial treatment for tonsillitis, anecdotal cases of failure after intramuscular long-acting penicillin have also been noted. Although Group A *Streptococcus* remains the leading cause of peritonsillar abscess, *Staphylococcus aureus*, *Haemophilus influenzae*, *Bacteroides, Peptostreptococcus,* and mixed anaerobic infections are common. ß-lactamase-producing organisms are present in about 50% of cases. [3] Fine-needle aspiration of peritonsillar abscesses may allow identification of organisms and appropriate modification in antibiotic therapy, thus avoiding the need for tonsillectomy.

Patients with peritonsillar abscesses present with sore throat, odynophagia, low-grade fever, and a variable degree of trismus. The trismus develops secondary to pterygoid muscle irritation. The patient may also complain of ipsilateral otalgia. As the abscess expands, the patient may experience dysphagia with drooling. Patients are often dehydrated secondary to poor oral intake. Voice changes are common (hot potato voice) and are caused by transient velopharyngeal insufficiency and muffled oral resonance. Rancid breath is also common. Tender ipsilateral anterior cervical lymphadenopathy is frequently present. Fever >39.4°C has been associated with parapharyngeal extension and sepsis. [4]

Examination of the oropharynx may be difficult because of associated trismus. Have the patient sit with the head in the "sniffing" position. Encourage the patient to open the mouth as wide as possible, and depress the tongue to obtain a better view of the oropharynx. Use a head lamp or head mirror/light source to ensure adequate illumination. Digital palpation for a fluctuant site can be useful, and may be the best way to differential abscess from cellulitis. The clinician places the gloved index finger into the mouth and feels for hardness or fluctuance in the peritonsillar region (Fig. 65-7). If no abscess can be appreciated, the diagnosis is unlikely. This technique is usually well tolerated but the patient may gag or bite the examiner by reflex. Physical findings are often diagnostic of a peritonsillar abscess, but in about 20% of cases the diagnosis is in doubt until needle aspiration is performed. Inferior and medial displacement of the tonsil and uvula are noted along with a fluctuant mass involving the tonsillar pillar. Swelling obliterates the normally sharply delineated pillar-like

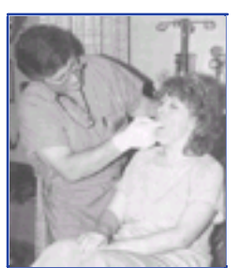

Figure 65-7 Clinical symptoms and visual inspection may not be sufficient to differentiate peritonsillar abscess from cellulitis. The clinician's gloved index finger is used to palpate the peritonsillar area to search for fluctuance and localized swelling.

structure. The tonsil looks edematous and erythematous, and may be covered with a whitish exudate.

The differential diagnosis for this acute process includes unilateral tonsillitis, peritonsillar cellulitis, retropharyngeal abscess, infectious mononucleosis, *Herpes simplex* tonsillitis, retromolar abscess, neoplasm, FB, and, possibly, internal carotid artery aneurysm. Chronic conditions include leukemia, carcinoma, and parapharyngeal space tumor.

Differentiation of a peritonsillar abscess from peritonsillar cellulitis may be difficult, especially in the early stages of an abscess. The history and time course for the two disease processes are quite similar. Trismus and uvular deviation are uncommon in peritonsillar cellulitis. [5] Needle aspiration will be diagnostic if purulent material is removed. However, a negative test does not rule out an abscess. The abscess may be located posteriorly, and not accessible by the aspiration needle. Intraoral sonography has a sensitivity and specificity of 91% and 80%, respectively, in detecting peritonsillar abscesses [6] and may therefore augment diagnostic accuracy. Also, if there is any question as to the diagnosis or actual location of the abscess, CT scanning may be utilized.

General Treatment

The treatment of peritonsillar abscess has undergone significant change in the past 100 years and continues to do so at this writing. A myriad of opinions exists on the appropriate treatment method, although most agree that some form of drainage procedure should be performed in conjunction with antibiotics and pain control. Three options for surgical drainage include: needle aspiration, incision and drainage, and immediate (quinsy) tonsillectomy. Each method will be discussed.

Needle aspiration is relatively simple, can be performed by clinicians who are not head and neck specialists, does not require special equipment, and is relatively inexpensive. Other benefits of needle aspiration over incision and drainage include decreased pain and trauma. Many feel that this should be the initial surgical drainage procedure for adults and children. The recurrence rate following needle aspiration is 10% [7] and its cure rate is about 94%. [2] About 4% to 10% of patients require repeat aspiration. [2] [7] One drawback is that needle aspiration may miss the peritonsillar abscess and therefore allow misdiagnosis as peritonsillar cellulitis. Up to 12% to 24% of abscesses have been missed on initial aspiration. [8] [9] Therefore, some authors propose admission of patients with negative aspirations with the presumed diagnosis of peritonsillar cellulitis for intravenous (IV) antibiotics and observation to prevent further morbidity. [10] Although most studies were performed with hospitalization and IV antibiotics, selected outpatient treatment with oral antibiotics has been successful. [10] [11] Most patients with a successfully drained peritonsillar abscess can be safely and effectively managed with needle aspiration and outpatient antibiotics. [12]

Incision and drainage is commonly done as an outpatient procedure under local anesthesia. This procedure is usually performed after pus is obtained by needle aspiration, but occasionally it is the primary procedure. It seems most logical to first attempt aspiration and only follow with incision and drainage if additional pus is suspected or there are other extenuating circumstances. The recurrence rate following incision and drainage is 5.9% to 22.7%. [13] Despite these shortcomings, incision and drainage is the initial surgical treatment used by an estimated 54% of U.S. otolaryngologists. [2]

Immediate (quinsy) tonsillectomy is thought by some to be the only way to completely drain the abscess and completely eliminate the risk of recurrence. They also feel that hospital time and the patient's disability are shortened. Arguments against this rationale include:

1. An initial PTA is no longer an indication for tonsillectomy.
2. Needle aspiration/incision and drainage have high success rates.

3. Studies have shown longer recovery times for patients following immediate tonsillectomy compared to the other drainage procedures.
4. There is a time delay (6 to 70 hours) in assembling equipment and personnel for the tonsillectomy. Patients successfully treated with needle aspiration are often well on the way to recovery in that amount of time. [7]

Treatment guidelines based on a review of the literature [2] [7] suggest that pediatric patients with peritonsillar abscess and adults without history of recurrent tonsillitis be initially treated with needle aspiration. Incision and drainage and immediate tonsillectomy should be reserved for treatment failures or recurrences. Adult patients with *recurrent* tonsillitis/peritonsillar abscess should be treated with either needle aspiration followed by delayed tonsillectomy or with abscess tonsillectomy alone. These procedures can be done in combination with hospital admission and administration of IV antibiotics or as an outpatient with oral antibiotics. The approach depends on the patient's clinical status and medical history. Decisions about the treatment of a peritonsillar abscess in the ED are often made by the emergency clinician, but as local protocols dictate, consultation with an otolaryngologist is often appropriate.

Needle Aspiration/Incision and Drainage

The two procedures described here include needle aspiration and incision and drainage. They should only be performed in the cooperative patient without severe trismus. With the carotid artery located 2.5 cm behind and lateral to the tonsil, there is minimal room for error, patient movement, or poor anesthesia.

Have the patient sit upright, with a support behind the head. This is best done as a two-person procedure. An assistant can retract the cheek laterally to maximize visibility. A head lamp provides optimal lighting; a double tongue-blade setup aids visualization of the operative area (Fig. 65-8). It is

Figure 65-8 Needle aspiration of a peritonsillar abscess. The patient sits upright with the head supported by the back of the stretcher (or a dental chair head rest). A head lamp provides light and an assistant retracts the cheek laterally to maximize visibility.

Figure 65-9 *A*, As a safeguard to prevent deep penetration of a needle used to drain a peritonsillar abscess, select a long 18- to 20-gauge needle. Remove the plastic needle guard and cut off the distal 1 cm. *B*, Replace the guard on the needle and tape it to the hub.

suggested that parenteral narcotic analgesia or mild sedation, or both, be administered before attempts at aspiration. Fentanly, 2 to 3 mcg/kg IV a few minutes before the procedure, is often ideal. Midazolam may be judiciously used, but the patient should not be overly sedated. The combination of midazolam, ketamine, and glycopyrrolate has been reported as being safe and effective for the outpatient peritonsillar drainage in children. [14]

Anesthetize the area topically with Cetacaine spray, or 4% to 10% lidocaine. Determine the fluctuant area of the abscess by manual palpation. Additionally anesthetize this area with local infiltration of 1 to 2 mL of 1% lidocaine with 1:100,000 epinephrine via a 27-gauge (ga) needle. Using a 5-mL syringe with a long needle allows visualization of the area to be injected, while a small syringe often causes the clinician's hand to block the view. Infiltrate the lidocaine intramucosally for the best results, and *seek a blanching area*

Figure 65-10 *A*, Needle aspiration of a peritonsillar abscess. Anesthetize the posterior pharynx with topical lidocaine spray. Blanch the mucosa with lidocaine/epinephrine with a 27-gauge (ga) needle on a long syringe (to allow visualization of the site) in the area to be aspirated. Advance an 18- or 20-ga needle with needle guard into the area of greatest fluctuance, usually the superior pole. Aspirate as you advance the needle. Advance the needle in the sagittal plane. Do *not* direct the needle laterally toward the carotid artery or jugular vein. *B,* The superior pole is aspirated first, but the middle and inferior poles should be aspirated if pus is not obtained initially. Note that the tonsil itself is not aspirated. The *peritonsillar space* contains the abscess.

on the mucosal surface. During the injection, be careful not to increase the abscess size by direct injection into the abscess cavity. With proper local infiltration the patient will not feel the penetration of the aspirating needle. If the trismus is so pronounced as to prevent adequate anesthesia administration, it will probably be too difficult to aspirate or incise the abscess properly.

For *aspiration*, prepare a long 18- to 20-ga needle on a 10- to 20-mL syringe. Fashion a needle guard by cutting off the distal 1 cm of the plastic needle cover, replace the cover on the needle, and securely attach this guard to the needle and syringe with tape to prevent inadvertent displacement (Figs. 65-9 A & B). Ensure that the needle protrudes only 1 cm beyond the cover. This procedure will limit the depth of needle penetration and lessen the risk of entering any major vascular structure. If pus is not obtained at a 1-cm depth, deeper penetration is discouraged. Insert the needle into the

most fluctuant (or prominent) area as previously determined, which is most commonly the superior pole of the tonsil. Note that the tonsil itself is not aspirated because the abscess develops in the peritonsillar space surrounding the tonsil. *The needle is advanced only in the sagittal plane and is not directed laterally, where it may injure the carotid artery.* If the aspirate is positive, remove as much purulent material as possible. If the aspirate is negative, attempt aspiration again in the middle pole of the tonsil approximately 1 cm caudal to the first aspiration. A third and final attempt should be performed at the inferior pole. Up to 30% of abscesses will be missed if only the superior pole is aspirated. It must be stressed that a negative aspirate does *NOT* rule out a peritonsillar abscess.

Usually 2 to 6 mL of pus is obtained. It is unusual to recover more than 8 to 10 mL (Fig. 65-11). There is no specific advantage of sending the aspirate to the laboratory for culture. Many clinicians forgo culturing since it has not been demonstrated that culture results influence treatment. [10] When significant amounts of pus are aspirated, the patient usually feels immediate improvement in pain and dysphagia. After the needle is removed, some bleeding will be noted. A slight ooze may be noted for a few hours, especially if warm water rinses are used. There may be continued drainage of pus, often sensed as a foul taste by the patient. If significant additional pus drains, this may be an indication for a repeat aspiration, incision and drainage or hospital admission.

Some clinicians advise a formal incision and drainage (see later) if frank pus is obtained, whereas others now accept needle aspiration (with close follow-up) as the definitive *initial* treatment. Theoretical reasons to combine aspiration and formal drainage in the same visit are if large amounts of pus are obtained (>5 to 6 mL) or if pus continues to drain from the aspiration site. There are no agreed-upon standards regarding best practice for this issue.

To *incise* a peritonsillar abscess, anesthetize the area as described earlier. Prepare a No. 11 or 15 scalpel blade by taping over all but the distal 0.5 cm of the blade to prevent deeper penetration (Fig. 65-12). Incise the area of maximal fluctuance or where a preceding aspiration, if one was performed, located pus. Incise the mucosa in an area 0.5 cm long in a posterior to anterior direction. A stab incision with

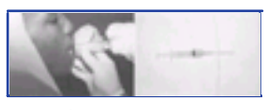

Figure 65-11 Needle aspiration usually yields 2 to 6 mL of thick pus. Greater volumes are unusual. Removing only a small amount will produce a marked reduction in symptoms.

a No. 11 blade usually suffices. Warn the patient that the pus will flow posteriorly and he or she must expectorate this fluid. Expect bleeding, as this is a vascular

area. Suction the incised area with a No. 9 or 10 Frazier suction tip or a tonsil suction tip to aid in removal of the purulent material. Place a closed Kelly clamp into the opening and gently open it to break up the loculations. Allow the patient to rinse and gargle with a saline or dilute peroxide/saline solution. Packing is *not* used in the drainage of this abscess. *Following aspiration or incision, it is prudent to observe the patient for about an hour to watch for complications (e.g., bleeding) and to ensure the ability to tolerate oral fluids.* Most patients can be discharged with 24 hour follow-up. Toxic patients, those with excessive volumes of aspirate, those with persistent bleeding, or those unable to take oral antibiotics are candidates for admission or more prolonged observation. Frequent rinses with warm saline are quite helpful in relieving postaspiration symptoms.

Following either needle aspiration or incision and drainage, antibiotics are recommended to eradicate the offending organisms. Penicillin, clindamycin, or cephalosporins are a reasonable first choice. Resistance rates to penicillin range from 0 to 56% but laboratory sensitivity testing is not always reflective of a clinical response.[7] Alternatives include ampicillin/sulbactam and amoxicillin/clavulanate. Reasonable cure rates have been obtained with oral penicillin in modest doses (500 mg per os [PO] four times a day [QID]). Many clinicians prefer to administer an IV loading dose of penicillin (5 million units) or cefazolin (1 g) before releasing the patient. While the benefit is not well established, many clinicians empirically also administer a single parenteral dose of dexamethasone (10 mg) and this may further ameliorate symptoms. Any patient who appears to have a toxic response, whose immune system is compromised, is unable to take oral antibiotics, or is dehydrated should be admitted for IV fluid hydration and antibiotic administration. Reevaluation of all patients treated with needle aspiration should be performed in 24 hours to assess the need for repeat aspirations or formal incision and drainage. At 24 hours most patients are markedly improved, and failure to see this response requires further evaluation. Warm saline gargles and mild opioid analgesics also are recommended with outpatient care. All patients should

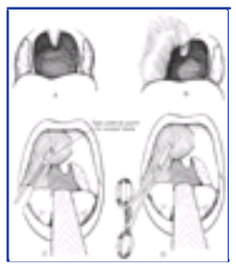

Figure 65-12 Aspiration is often the only procedure required to successfully treat a peritonsillar abscess, but it has a 10% failure rate. In some instances the clinician will opt for incision and drainage of a peritonsillar abscess. This procedure may used initially, or after aspiration if copious pus is aspirated, or if pus continues to drain or reaccumulates. *A,* Normal-appearing oropharynx. *B,* Peritonsillar abscess on the right side of the throat. *C,* Incision of the abscess at the area of greatest fluctuance. Notice that the scalpel is taped to prevent deep penetration. *D,* Loculations are removed by gentle probing with hemostats.

immediately return for recurrence of symptoms, fevers, or continued bleeding from the incision.

Complications

Needle aspiration is an accepted, safe, and effective technique for the ED treatment of peritonsillar abscess. There is an approximate 10% failure rate or need for subsequent drainage. Aspiration or incision of the carotid artery or a misdiagnosed carotid artery aneurysm may have devastating results. The potential for penetrating a deep vascular structure is largely theoretical if proper technique is followed—there are no documented cases in recent literature. If the patient has cellulitis, the aspiration will be of no help, but it will not worsen morbidity. Failure to obtain pus should prompt highdose antibiotics and a recheck in 24 hours. Many clinicians will opt for admission in such instances. A too-large or too-small incision may lead to poor healing or inability to completely evacuate the abscess, respectively.

EAR

Anatomy of the External Auditory Canal

The external auditory canal (EAC) extends from the tympanic membrane to the concha and measures approximately 2.5 cm in the adult. It is relatively short and straight in early infancy but begins to take on its adult S-shape and overall anterocaudal orientation beginning at age 2 years. Initially, the EAC is almost entirely cartilaginous, but by adulthood its medial two-thirds is comprised of bony support with an overlying thin, stratified, squamous epithelium. The lateral third has a less sensitive, thicker, hairy epithelium that produces cerumen and retains its cartilage as support. The arterial supply to the EAC originates from the external carotid artery via the posterior auricular, maxillary, and superficial temporal branches. The mandibular branch of the fifth cranial nerve (V_3) and the vagus nerve innervate the ear.

Other important anatomic considerations include the following: (1) Two natural narrowings of the EAC exist, which are important when considering FBs. One is located at the junction of bone and cartilage and the other lies just lateral to the tympanic membrane. (2) A blind spot may occur in the tympanic sulcus (inferior and anterior to the tympanic membrane) due to the oblique orientation of the tympanic membrane. An examiner using a simple otoscope may not visualize an FB in this sulcus.

Anesthesia of the Ear

External Ear/Auricle

Indications for local anesthesia of the auricle include closure of extensive lacerations or other painful procedures, such as

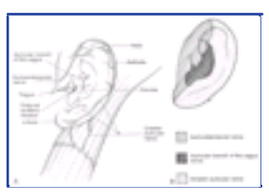

Figure 65-13 *A* and *B,* External anatomy of the ear and innervation of the auricle.

hematoma incision and drainage. Four nerve branches supply the external ear, and knowledge of their anatomy is required to understand the location for anesthesia injection (Fig. 65-13*A* and *B*). The *greater auricular nerve* (branch of the cervical plexus) innervates most of the posteromedial, posterolateral, and inferior auricle. A few branches of the *lesser occipital nerve* may contribute to this area. The *auricular branch of the vagus* supplies the concha and most of the area around the auditory meatus. The *auriculotemporal nerve* (from the mandibular branch of the trigeminal nerve) supplies the anterosuperior and anteromedial aspect of the auricle.

Procedure

Fill a 10-mL syringe with either 1% lidocaine or 0.25% bupivacaine (both with epinephrine if a regional block is planned in an area *without* evidence of traumatized vascularity) and attach the syringe to a 25- or 27-ga needle (5 to 7 cm in length). One of several methods may be used to accomplish partial or complete anesthesia, depending on the area of concern. The greater auricular and lesser occipital nerve branches may be anesthetized by injecting between 3 and 4 mL of anesthetic in the posterior sulcus (Fig. 65-14*A*). Insert the needle behind the inferior pole of the auricle and gradually aspirate and inject toward the superior pole, following the crescent-shaped contour of the posterior auricle. Anteriorly, the auriculotemporal nerve may be anesthetized by placing 3 to 4 mL of anesthetic just superior and anterior to the cartilaginous tragus. Use the technique shown in Figure 65-15 and Figure 65-16 to provide anesthesia of the auricular branch of the vagus to include more central areas of the auricle.

Another and possibly more effective option is the regional block shown in Figure 65-14*B* . Insert the needle subcutaneously (SQ) at a point approximately 1 cm above the superior pole of the auricle and direct it to a point just anterior to the tragus. Be sure to inject the skin of the scalp while avoiding the auricular cartilage. Aspirate, then slowly withdraw the needle, injecting anesthetic until the needle is almost to the puncture site. Redirect the needle posteriorly and repeat the process while aiming at the skin just behind the mid-auricle. Remove the needle and perform the same procedure, but

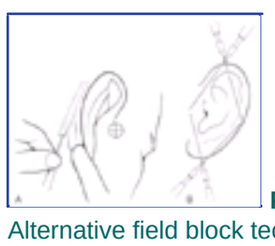

Figure 65-14 Field blocks of the auricle. *A,* One method uses approximately 3 to 4 mL of anesthetic, both in the posterior sulcus and at a point just anterior to the tragus. *B,* Alternative field block technique that deposits 2 to 3 mL of anesthetic for each needle pass. See text for more details.

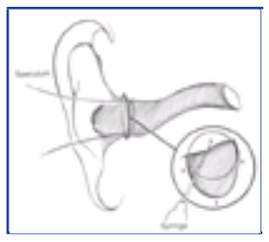

Figure 65-15 Four-quadrant field block anesthesia of the external auditory canal. Local anesthetic is injected subcutaneously in the four quadrants of the lateral portion of the ear canal. The largest speculum that will fit is used to guide the injections. The speculum is withdrawn slightly, tilted toward each of the four quadrants, and the needle is inserted subcutaneously *(x)*. A very small amount of anesthetic (0.25 to 0.50 mL) is injected to produce a slight bulge in the soft tissue. A total of 1.5 to 2.0 mL of anesthetic is usually sufficient to anesthetize the ear canal and permit painless removal of a foreign body.

insert the needle just inferior to the insertion of the ear lobule and anesthetize in a superior direction. Again, block the auricular branch of the vagus as described in Figure 65-17 if additional anesthesia of the concha is required.

Use caution if adding epinephrine to the anesthetic solution when placing regional blocks of the ear, especially if

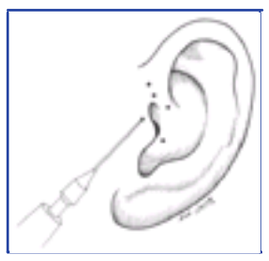

Figure 65-16 Diagram of injection sites for an alternative technique to anesthetize the ear canal and central concha. Each site should be injected with approximately 0.5 mL of 1% lidocaine. Do not inject if external signs of infection are present.

Figure 65-17 Examination of ear canal. The pinna is retracted in a superior and posterior direction to straighten out the ear canal. The scope is held in the other hand and stabilized against the patient's head. This prevents inadvertent injury if the patient moves unexpectedly.

the blood supply has already been traumatically reduced. Do not include epinephrine when directly infiltrating wounds of the auricle, as restriction of blood flow through end arteries here may result in tissue necrosis. Other complications related to local anesthetics and regional blocks of the head and neck may be reviewed in Chapter 30 and Chapter 31 .

External Auditory Canal and Tympanic Membrane

The EAC is innervated by the auricular branch of the vagus (inferiorly and posteriorly) and by the auriculotemporal nerve (superiorly, anteriorly, and inferiorly). The primary indication for local anesthesia of the auditory canal is for FB removal, including debridement of otitis externa or removal of significant cerumen impaction. It is very difficult to obtain adequate anesthesia of the inner ear and tympanic membrane for painful procedures. Simply stated, no easy and completely effective procedure consistently works well. If total anesthesia is required, general anesthesia, especially in children, is often the only alternative. Topical anesthetics are inadequate due to their poor absorption through the rather impermeable and keratinized epithelial surface of the EAC. Although effective for some procedures, injecting local anesthetics in and around the auditory meatus is quite painful and is often difficult to perform in a struggling and uncooperative patient. Certain instances warrant adjunctive use of conscious sedation (see later discussion under Foreign Body Removal).

It is quite difficult to obtain anesthesia of the tympanic membrane. The membrane is sensitive and can be stimulated during attempts at removing a FB from the ear canal. Topical anesthetics have limited value, but Moller and Grontved demonstrated that 10% aerosolized lidocaine (first sprayed into a syringe and shaken to evaporate the propellant) and 4% lidocaine suspension, when dripped into the ear canal, provided good anesthesia of the membrane. [15] However, it required 30 minutes for this anesthesia to take effect. Both solutions were alkaline and it was noted that lidocaine

hydrochloride, the form usually used for wound infiltration, is acidic and provided no anesthesia when applied topically. Auralgan, a combination of benzocaine and other ingredients, may provide analgesia for painful earaches due to otitis, but it has little benefit for painful procedures.

Procedure

Local anesthesia is performed with a 25- or 27-ga needle (3 to 5 cm in length) attached to a syringe of 1% lidocaine with epinephrine (1:100,000). A 1:10 mixture of 8.4% sodium bicarbonate to lidocaine helps to reduce pain with injection in this sensitive area. Place a speculum just inside the auditory meatus and inject 0.3 to 0.5 mL of the anesthetic into the SQ tissue, stopping after a small bulge in the skin is raised. Inject in this manner in all four quadrants by moving the speculum after each injection (see Fig. 65-15). If additional anesthesia is necessary, give two more small injections. Inject the same amount slightly farther into the canal, once along the anterior wall and again at the posterior wall at the bone-cartilage junction.

Another similar technique involves depositing the anesthetic just lateral or exterior to the external auditory meatus. Using the same size needle and type of anesthetic solution as just described, inject approximately 0.5 to 1.0 mL into each of 5 points around the auditory meatus and tragus (see Fig. 65-16).

Examination

Several methods are available to examine the EAC and tympanic membrane. In all methods, the superior pinna should be grasped and pulled cephalad and posterior to straighten the slightly tortuous EAC. The most common manner of examination is with a *fiberoptic otoscope* (see Fig. 65-17). The clinician may insufflate the tympanic membrane as well as examine the EAC with the diagnostic head, while the operating head allows for instruments to be passed into the EAC and maneuvered more easily. A plastic or metal speculum may be placed in the auditory meatus for examination, using a head lamp or head mirror/light bulb as a light source. Although this provides excellent illumination, magnifying loupes are generally needed for adequate visualization during procedures. The ideal setup for cerumen or FB removal consists of an operating microscope and a speculum. This provides binocular vision and frees the examiner's hands for instrumentation (unfortunately this equipment is seldom found outside of the otolaryngology clinic setting). The hand holding the otoscope is stabilized against the patient's temporal skull to prevent inadvertent canal injury due to unexpected patient movement.

Cerumen Impaction

The excretion of the ceruminous or apocrine and sebaceous glands together with cells exfoliated from the external auditory canal combine to form cerumen. One study[16] found that cerumen is composed of lipids, complex proteins, and simple sugars. Cerumen repels water, has documented antimicrobial activity, and forms a protective barrier against infection. Cerumen often becomes impacted, causing complaints of a "blocked" ear, hearing impairment, or dizziness. Symptomatic impaction is an indication for removal, although symptoms are rare until complete obstruction is present. The sudden loss of hearing is a common complaint in patients with totally occluding impacted cerumen. Cerumen obstructs visualization of the tympanic membrane and can be evacuated as a part of the evaluation of a febrile child or the patient complaining of ear pain. However, cerumen removal in a child is rarely indicated in the ED simply to visualize the tympanic membrane.

Cerumen impacted for prolonged periods, and vigorous attempts to remove it, may precipitate otitis externa. It is reasonable to instill antiseptics (Vol Sol and others) or antibiotics for a few days post cerumen removal to prevent this. No standard exists and practices vary widely.

Cerumen Removal

Irrigation is an effective approach for cerumen removal and has the advantage of being painless and simple to perform. The patient does not have to remain completely still; thus, it is ideal for the pediatric population. It is estimated that 150,000 ears are irrigated in the United States each week. [17] Although usually more time-consuming than manual extraction, irrigation is an appropriate initial method to attempt and can be performed by technicians with guidance from the clinician. One contraindication is known or suspected tympanic membrane perforation. Use irrigation judiciously in elderly and immunocompromised patients, as malignant otitis externa is frequently preceded by irrigation of the EAC. [18] Generally, the procedures used to remove cerumen are safe; however, otologic injury has resulted from this "minor" procedure and has even resulted in litigation. [17] Whichever of the following techniques are used, some tips for successful cerumen removal include: use proper lighting, pay attention to patient comfort, and never continue beyond the patient's comfort level.

Ceruminolytics

These products may soften obviously hardened or impacted cerumen. They are used as adjuncts to other procedures—simply instilling ceruminolytics into the canal will not remove enough cerumen to aid the emergency clinician. If irrigation fails, the continued outpatient use of ceruminolytics is often prescribed, often combined

with home irrigation using a bulb syringe. Although many products are available as ceruminolytics, a 5% or 10% solution of sodium bicarbonate disintegrates cerumen much more quickly and efficiently compared with commercially prepared ceruminolytics and other products. Cerumenex, Cerumol, Auralgan, Buro-Sol, alcohol, and oils were all tested and took more than 18 hours to disintegrate cerumen vs ~90 minutes for the sodium bicarbonate solutions. [19] Hydrogen peroxide is another commonly used ceruminolytic, but its use has not been systemically studied. One study [20] found the liquid preparation of the stool softener docusate sodium (Colace) was much more effective as a ceruminolytic than Cerumenex. Place the patient in the supine position with the affected ear up and instill the solution at least 15 minutes before attempts at removal. Instillation can be repeated between attempts at manual extraction or irrigation.

Irrigation (Ear Syringing)

This procedure is best achieved by having the patient sit upright and holding an emesis or ear irrigation basin flush against the skin just below the ear lobule (Fig. 65-18). Insert the irrigation tip into the EAC only as far as the cartilage-bone junction, and direct the stream of water superiorly to wash the impacted cerumen away from the tympanic membrane. Water should be near body temperature to prevent caloric

Figure 65-18 Irrigation is a simple, painless, and usually successful way to remove cerumen. After a wax-softening agent has been instilled for 15 to 20 minutes, an assistant applies traction on the ear to straighten the canal, and the plastic tubing of a 19-ga butterfly device (needle and wings removed) is inserted 1 cm into the canal. A basin is held by the patient, and warm water is introduced with a 20-mL syringe. A number of irrigations may be required, and the procedure may be supplemented with careful removal of large cerumen pieces with a curette.

stimulation. Multiple attempts may be necessary, and intermittent attempts at manual removal of loosened cerumen may help hasten the process. During the irrigation, the operator or an assistant should apply traction to the pinna to straighten the canal for more efficient irrigation.

Irrigation techniques using manual pressure to discharge the water include metal ear syringes and bulb syringes. The metal ear syringe (*Reiner-Alexander Ear Syringe*) is inexpensive and readily available. Some disadvantages include: slow operation, it is poorly balanced, the tip tends to wobble, and minor canal trauma is common.[17] [21] Improvisational irrigation systems can be assembled with equipment found in the ED. Attach a 20- or 30-mL syringe to a 19-ga or larger butterfly device, cutting off the needle and wings and leaving the resultant tubing for irrigation. A plastic or Teflon IV catheter (16- or 18-ga with the needle removed) can similarly be affixed to a syringe. Contraindications to ear syringing include [22] :

Patient aversion to or history of injury from syringing

History of middle ear disease

History of ear surgery

Perforated tympanic membrane

Severe otitis externa

Narrow ear canals

FBs, especially sharp objects and vegetable matter

Uncooperative patient

Occluding aural exostoses

Known inner ear disturbance, especially if patient has severe vertigo

History of radiation therapy to the external or middle ear, skull base, or mastoid

The most common way to irrigate an ear is with a syringe and catheter (Fig. 65-19). Although most commonly found in an otolaryngologic clinic, automated pressure devices may also be available. The *DeVilbiss irrigator* (Fig. 65-20) uses a pressurized air source to propel the irrigating solution into the ear. It allows the clinician to control the water pressure and direction, making it pleasant for the patient. Unfortunately, it is rarely available in hospitals, requires a pressure source, and clinician training is needed. Use of *oral jet irrigators* (Water Pik) is another accepted method. They are fast, portable, and inexpensive. Disadvantages to the oral jet irrigator include: trauma to the stapes and cochlea, off-label use may expose the clinician to litigation, there is splash back, and many patients find it uncomfortable. The Hydro Med Ear Irrigator Tip (Hydro Med, Sherman Oaks, CA) can be combined with the Water Pik to improve safety and comfort. The tip keeps the stream of water from hitting the eardrum. The tip is smooth and convex, reducing the potential for injury. [17] Although the instances have been rare, tympanic membrane rupture has

Figure 65-19 The most readily available device for ear irrigation is an 18-gauge flexible catheter attached to a 60-mL syringe. Since multiple irrigation may be required, small syringes are counterproductive.

Figure 65-20 This DeVilbiss irrigator uses compressed air to eject the irrigating solution, which should be near body temperature to avoid caloric stimulation of the inner ear. The more conventional metal ear syringe can be equally effective and does not require a source of compressed air to function.

been documented with these apparatuses.[23] Therefore, use the lowest-power setting and guide the stream of the water against the EAC wall, not directly toward the tympanic membrane.

After irrigation of the EAC, application of several drops of isopropanol in the EAC will facilitate evaporation of residual moisture. The tympanic membrane must be intact if isopropanol is to be used. Further, topical Cortisporin Otic suspension drops may be soothing after prolonged irrigation. Since diabetics can develop severe otitis external following irrigation, some clinicians routinely prescribe antibiotic ear drops (fluoroquinolones and others) for a few days post irrigation in high-risk patients.

Although more common with jet irrigators, complications may occur with any method of ear irrigation. These include otitis externa, tympanic membrane perforation, or

middle ear injury from a preexisting defect in the tympanic membrane. Irrigation should be stopped and the tympanic membrane examined in any patient experiencing sudden pain, tinnitus, hearing loss, nausea, or vertigo. If the membrane is ruptured, prophylactic antibiotics for otitis media should be given along with a referral to an otolaryngologist.

Manual Instrumentation

This procedure is more advantageous as it is usually quicker, and the examiner may more easily remove hardened or larger concretions of cerumen under direct visualization. However, it is difficult to manually remove cerumen without causing significant pain. Either the diagnostic or operating head of the fiberoptic otoscope or a speculum may be placed in the auditory meatus to serve as a protective port through which instruments are passed and manipulated (Fig. 65-21). An operating microscope works best in this situation, but, again, is usually not available. To prevent startling or agitating an already anxious patient, allow the patient to experience the sensation of an instrument in the canal by first placing the instrument softly against the ear canal wall.

Instruments used for cerumen removal include *flexible plastic or wire loops, right-angle hooks, suction-tip catheters, or plastic scoops* (Fig. 65-22). The spoonlike instruments and irrigation are both more effective in removing softer cerumen. Firm cerumen ordinarily is more easily withdrawn with loops or right-angle hooks. Gently tease the cerumen off of the

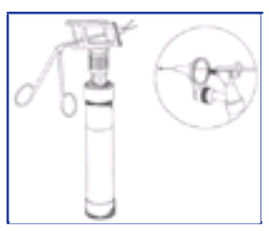

Figure 65-21 Technique for direct visualization and mechanical removal. Use of alligator forceps through a diagnostic otoscope. Note that the magnification device has been slid laterally and that no ear speculum has been attached. *Inset,* Use of ear curettage through operating otoscope. *(From Fritz S, Kelen GD, Sivertson KT: Foreign bodies of the external auditory canal. Emerg Med Clin North Am 5:184, 1987.)*

canal wall using loops and then pass hooks or loops around the cerumen and withdraw the cerumen slowly (Fig. 65-23). Care should be taken to keep both hands in contact with the patient's head, as any sudden movement may cause trauma to the canal or the tympanic membrane. Complications most commonly occur when inadvertent contact is made with the thin, friable skin of the bony canal. Trauma may cause EAC laceration, hematomas, otitis externa, or tympanic membrane perforations.

Otitis Externa

Otitis externa, or "swimmer's ear," is an inflammation of the skin of the external auditory canal. This is essentially a cellulitis of the ear canal. Otitis externa can be disabling enough to cause 36% of patients to interrupt their daily activities for a median duration of 4 days. [24] Precipitants of otitis externa include water exposure and trauma. Excessive moisture in the canal raises the pH and removes the cerumen. Keratin can now absorb water, creating a medium for bacterial growth. Trauma, especially self-manipulation with FBs (cotton swabs, fingernails, etc.), causes abrasions to the ear canal and introduces infection. Removal of cerumen by water irrigation is a well-recognized risk factor for the development of otitis externa. [24] [25]

The disease process involves a continuum of gradually worsening inflammatory changes. The patient may present with symptoms ranging from slight itching and discomfort to severe pain, purulent discharge, or systemic toxicity. Pain with manipulation of the pinna is the hallmark for otitis externa. Otoscopy of the external auditory canal may initially reveal minimal debris and erythema, but as the infection progresses, more edema, exudate, erythema, and possibly even a surrounding cellulitis may become apparent. In severe stages the edema may obstruct the canal, preventing instillation of eardrops.

Figure 65-22 Instruments used for foreign body extraction. From left to right: alligator forceps, bayonet forceps, right-angle hook, wire loop, soft-tipped suction, Frazier suction.

Common bacteria cultured from patients with otitis externa include *Pseudomonas aeruginosa* and *Staphylococcus aureus*. Other bacteria include *Acinetobacter*, *Proteus*, *Enterococcus*, and *Bacteroides*. Approximately 50% of patients have polymicrobial infection and 8% have anaerobic isolates. [26] Of concern, one study[27] found that 6% of staphylococcus isolates were methicillin resistant. Fungi are identified in about 10% of otitis externa cases and are often coexistent with bacterial infections. Aspergillus is responsible for 80% to 90% of cases followed by Candida. It characteristically presents as a furry lining of the ear canal with a fluffy white discharge. Herpes zoster affecting the geniculate ganglion may appear as grouped vesicles on an erythematous base within the canal. This condition, known as Ramsay Hunt syndrome, is associated with facial nerve palsies, hearing loss, and other cranial nerve impairment.

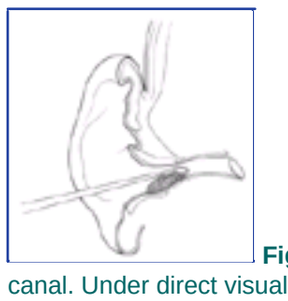

Figure 65-23 Removal of impacted cerumen. Pass the tip of the wire loop beyond the wax and gently tease the wax off the ear canal wall. Extract the wax slowly from the canal. Under direct visualization, avoid contact with the skin of the ear canal to prevent pain and excoriation.

Diabetics and other immunocompromised patients, especially HIV-positive patients, are susceptible to *malignant (necrotizing) otitis externa*, a life-threatening form of otitis externa caused by *Pseudomonas*. Deep tissue necrosis, osteomyelitis, intracranial extension, and systemic toxicity are hallmark features. Malignant otitis externa is difficult to treat and mortality rate can be as high as 53%. [24] The diagnosis of malignant otitis externa should be considered in the diabetic or immunocompromised patient with significant symptoms who fails to respond to initial outpatient treatment.

Canal Debridement/Wick Placement

It has been touted that key to successful treatment is adequate removal of canal debris. However, vigorous attempts to remove debris on the first visit are very painful, of unproven value, and often eschewed. Gentle attempts to remove debris with small swabs (e.g., urethral swab) (Fig. 65-24) are reasonable, but reserving this procedure for treatment failure or at a follow-up visit is also practiced. Gentle irrigation is one initial approach, but many patients will be cured without extensive debridement. Because the inflamed canal is susceptible to trauma, debris removal may also be accomplished by suctioning under direct visualization using the open or operating otoscope head and a 5 or 7 French (Fr) Frazier tip suction. Irrigation of the canal can be performed only if the clinician is assured there is no tympanic membrane perforation, which may be difficult to confirm due to edema and patient discomfort. [24] [25] [26] For more advanced cases presenting with significant exudate and edema, debris removal remains necessary but is intensely painful. The author recommends using a local block of the auditory canal (see Fig. 65-16) as long as the cellulitis has not extended out to the tragus or concha. Administer parenteral analgesics if additional pain control is required.

Ear wicks may be used when edema, debris, and exudate are marked enough to impede antibiotic drops from contacting the canal skin. The wick is used as a conduit to deliver the antibiotic solutions to the ear canal. After debridement, use one of several methods to accomplish this. The true benefit of wick implantation is unknown. One approach is to place a 0.25-in. strip of Nu-Gauze dressing covered with an antibiotic and steroid cream (Cortisporin Otic cream) into the external acoustic canal

in a fashion

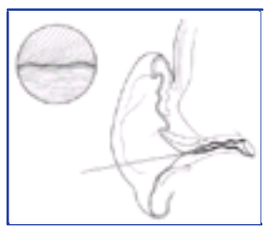

Figure 65-24 Debridement of external otitis. A cotton-tipped applicator is inserted into the canal and debris is gently removed from the canal. Irrigation of the ear may also be helpful if the tympanic membrane is not perforated. *Inset,* the edema has almost closed the canal and will not allow medication to be instilled into the inner canal. An ear wick will prove useful in this situation.

similar to the technique used for anterior nasal packing. Using an otoscope and alligator forceps, place the leading edge of the gauze deeply in the canal until it is fully packed. Withdraw the otoscope and finish by packing the lateral aspect of the canal as well.

Another choice is to place commercially available ear wicks, such as the Pope Merocel ear wick. Place this wick into an edematous canal and apply antibiotic/hydrocortisone drops onto it. The wick swells and helps to reduce edema by the antimicrobial and anti-inflammatory effects of the solution and through pressure exerted against the walls as it expands. Leave wicks in place until the patient is followed up in 24 to 48 hours for removal and further debridement. Although relatively safe to use, the ear wick is designed for short-term use. Generally these wicks will fall out of the canal as edema subsides. However, the unusual retention of these wicks can harbor bacteria and cause tissue ingrowth, resulting in long-term problems for the patient. [28]

Antibiotic Therapy and Follow-up

Most cases of otitis externa can be effectively treated with debridement and topical antibiotic drops. A study by Halpern [29] showed that <20% of patients have a concomitant diagnosis treatable by oral medications, yet 40% of patients receive topical and oral medications and many of the oral antibiotics prescribed are not active against Pseudomonas and Staphylococcus. They also found that only 7% of adult doctor visits and 2% of pediatric clinician visits reported ear irrigation. Antibiotic eardrops most frequently consist of some combination of neomycin and polymyxin, but an acetic acid solution is another acceptable first-line therapy. Eardrops are instilled as 2 to 4 drops QID for 7 to 10 days. Hydrocortisone may be added to either the antibiotics (Cortisporin suspension or solution) or the acetic acid (VoSol HC). Cortisporin Otic *solution* (clear appearing) is harmful to the middle ear if it passes through the tympanic membrane. At times it is difficult to distinguish between a ruptured tympanic membrane secondary to otitis media and severe otitis externa in a child. Therefore, the cloudy-appearing Cortisporin *suspension* is recommended in any case of suspected or known tympanic membrane perforation. Topical antibiotic preparations have been recommended for chronic suppurative otitis media, tympanostomy tube otorrhea, and acute otitis media. [30] Antibiotic preparations entering the middle ear are rarely problematic. There is no compelling reason to withhold drops if the tympanic membrane has been ruptured, although in that circumstance, gentamycin preparation and corticosporin solution (not suspension) are discouraged.

Fluoroquinolones have good activity against Pseudomonas and Staphylococcus. Two topical fluoroquinolone preparations are available, ofloxacin 0.3% (Floxin Otic) and Ciprofloxacin 0.2% with hydrocortisone 1% (Cipro HC Otic). Ofloxacin solution is approved for treatment of otitis externa and otitis media with perforated or ventilated tympanic membrane. Ciprofloxacin suspension is approved for the treatment of otitis externa only. Both may be used in patients one year and older. The topical fluoroquinolones show equal efficacy when compared to polymyxin B-neomycin-hydrocortisone suspension. Side effects are minimal and there is no reported ototoxicity.[31] Some practitioners consider these agents first-line therapy.

Holten and Gick[32] provide an evidence-based review of otitis externa treatment. The best evidence demonstrated equivalent results with ear cleaning, ear wick, and topical agents. Treatment with 1 of 3 regimens for 4 days was recommended. The regimens are: (1) ear cleaning + ear wick + acidifying agent dosed QID, or (2) ear cleaning + ear wick + topical antibiotic dosed QID (twice daily if fluoroquinolone), or (3) ear cleaning + ear wick + topical antibiotic/steroid combination dosed QID (twice if fluoroquinolone). There was less evidence for the use of single topical treatment or oral antibiotics. This paper also provides evidence-based treatment options for malignant otitis externa as well.

Inform patients to avoid getting water in the ear for the full course of treatment and to apply their eardrops immediately if water does contact the ear canal. Follow up severe cases in 24 to 36 hours and repeat debridement if needed. Generously administer oral opioids for the first 24 to 48 hours, as this condition can be quite painful. Nonsteroidal anti-inflammatory drugs are also effective for pain control. To prevent the recurrence of otitis externa, patients should avoid its many precipitants. This is especially true for patients who perspire excessively, regularly participate in water sports, have unusually viscous cerumen, have a narrowed external auditory canal, or systemic allergies. Measures include: drying the external auditory canal with a hair dryer on the lowest heat setting after bathing or swimming, using prophylactic acidifying drops with alcohol (Swim Ear) or without (Burow's solution, Star-Otic), avoiding scratching or overzealous cleaning, and using protective barriers while swimming (tight-fitting bathing cap or well-fitting ear plugs).

Broad-spectrum oral antibiotics should be used in cases of persistent otitis externa, concomitant otitis externa, systemic symptoms, and local cellulitis. A cautious approach to treating the mild to moderate case of otitis externa in the immunocompromised patient without toxic reaction is to add oral ciprofloxacin for greater coverage of *Pseudomonas*. Promptly admit suspected cases of malignant otitis externa and begin an IV anti-pseudomonal antibiotic. In addition, immediately consult an otolaryngologist for possible surgical debridement.

Patients with Ramsay Hunt syndrome should be treated with antivirals. Some clinicians suggest admission for administration of IV acyclovir. Treat otomycosis by swabbing the canal with a cotton-tipped applicator saturated with an antifungal solution (such as clotrimazole or boric acid/alcohol) in an effort to remove debris. Repeat this if necessary on follow-up in 3 to 7 days. If the infection is not responding, over-the-counter clotrimazole 1% solution (Lotrimin) can be used. If the tympanic membrane is perforated, then use tolnaftate 1% solution (Tinactin). Topical solutions of thimerosol and M-cresyl acetate are also effective. All of these topical agents are prescribed as 3 or 4 drops twice daily for 7 days. Because Aspergillus may be resistant to clotrimazole, oral itraconazole (Sporanox) can be used. [24] Do *not* administer corticosteroids in cases of known fungal otitis externa.

Foreign Bodies of the Ear Canal

Despite its small size, the EAC may play host to numerous types of FBs.[33] [34] Living insects account for most FBs found in adults. Children frequently place food (e.g., peas, beans), organic matter (e.g., grass, leaves, flowers), and inorganic objects (e.g., beads, rocks, dirt) into the ear canals during play, and they often fail to admit this to parents. Button batteries may cause significant tissue destruction in a matter of hours, and it is vital to immediately obtain otolaryngologic consultation for removal if the button battery is not easily extracted. Symptoms of FB retention are usually ear pain, fullness, or impaired hearing in the adult, whereas the pediatric patient may not present until an associated otitis externa with a purulent discharge has developed. Tinnitus, vertigo, significant hearing loss, or bleeding from behind the object should raise a high suspicion for an associated tympanic membrane rupture.

As previously described, the anatomy of the external auditory canal predisposes to the entrapment of FBs in either a lateral or deeper position. Removal of more medial objects can be much more painful and anesthesia is usually required. Even the most cooperative patient may become difficult after feeling pain during manipulation of the ear canal. It is probably impossible to adequately immobilize the head of an uncooperative awake child to delicately extract a FB. Some authorities claim local anesthesia makes extracting FBs even more difficult because of soft tissue distortion, although swelling should be minimal if proper amounts of anesthetic are used. Anesthesia of the auditory canal may be difficult to achieve. Topical anesthetics have a partial effect and a four-quadrant technique may not produce complete anesthesia, especially of the tympanic membrane.[35] A more realistic concern is that if the FB is deeply or firmly embedded, the patient should be referred early for removal under an operating microscope, before canal trauma and swelling mandate admission for removal under general anesthesia. Conscious sedation (preferably an analgesic-sedative combination or dissociative anesthetic) can aid in the removal of FBs in the distraught child by preventing further struggling and potential canal trauma. Ketamine is an excellent anesthetic for simple FB removal in the outpatient setting. The care provider must weigh the inherent risks of

conscious sedation against those of general anesthesia and the cost of hospital admission.

Before initiating removal, the clinician should set realistic limits on the number of attempts to be made. Even the best clinician can become too aggressive as frustration builds with failed attempts to extract the object. Early consultation with an otolaryngologist should not be considered a failure in cases of difficult FBs. Indeed, with the proper equipment and experience, most objects can be atraumatically removed.

Adequate visualization of the object is needed for successful removal. One study showed that canal lacerations occurred in 48% of patients where removal was attempted without a microscope and 4% when it was used.[35] The otoscope is the traditional ED instrument for viewing FBs of the ear canal. The otoscope is less likely to be useful in retrieval because it is difficult to instrument through or around the small speculum end. A specialized ear, nose, and throat (ENT) speculum allows more space for instrumentation. A head lamp provides a good light source and leaves both hands free. Magnifying loupes also provide hands-free magnification. Table 65-1 lists the equipment helpful for FB removal in the ED. [35]

Procedures

It is reasonable to make a judicious attempt to remove ear FBs in the ED setting. Prolonged traumatic attempts should be avoided since this often terrifies the patient, further complicates subsequent attempts, and can cause bleeding and swelling. These factors make subsequent efforts difficult. Most approaches to FB removal are anecdotal and are found in the literature as case reports or case series rather than as prospective clinical trials. The clinician should be familiar with several techniques, as the most appropriate choice can vary depending on depth of impaction or the size, shape, and consistency of the object. A summary of these techniques is provided in Table 65-2 . [35]

Irrigation is the least invasive option and the techniques and related complications are explained in detail earlier in this chapter (see Cerumen Removal). Deeply embedded vegetable matter (such as beans, peas, or seeds) should not be irrigated, however, as swelling may occur and make extraction more difficult. Irrigation works particularly well with small rocks, dirt, or sand that lie deep in the canal next to the tympanic membrane.

Suction-tip catheters.

This technique works well with objects that are round and difficult to grasp. Suction is readily available in the ED and needs to provide 100 to 140 mm Hg of negative pressure to be useful. To prevent iatrogenic injury, the patient must be informed of the impending noise to prevent sudden movements from a startle reflex. Place either the blunt or soft plastic tip against the object and slowly withdraw. If using a suction instrument with a thumb-controlled release valve (as with the Frazier suction) (see Fig. 65-22), remember to cover the port to activate the suction.

The Hognose (IQDr, Inc.), a commercially available device designed by an emergency clinician, aids in the removal of auditory canal FBs. It is used in combination with an otoscope and suction setup. It is essentially an otoscope speculum with suction attachment and a soft self-molding tip that can attach to objects. The flange comes in three color-coded sizes: 4mm, 5mm, and 6mm. To use, first attach the Hognose to the otoscope and standard wall suction set at low to medium vacuum setting (Fig. 65-25*A*). Next, under direct visualization, approach the FB with the otoscope. Finally, engage suction by applying finger pressure to the open insufflation port and withdraw (Fig. 65-25*B*).

Manual instrumentation.

This approach may be attempted with various instruments (Fig. 65-26). Illumination

TABLE 65-1 -- Removal Techniques for Aural and Nasal Foreign Bodies

Technique	Orifice	Advantages	Disadvantages
Irrigation	Ear	Ease of use	Contraindicated in TM perforation and vegetable matter
	Nose (possibly)		
Positive pressure	Nose	Nontraumatic	Risk of barotrauma
		Parent participation	
Negative pressure	Both	Easy to use	Need solid seal
		Good for round, small objects	Posterior FBs difficult
		Anterior FBs	Noise may frighten children
Glue	Both	Easy to use	Adhesion of glue to patient
		Nontraumatic	Must visualize FB
		Good for round, smooth objects	
Catheter techniques	Both	Good for awkward and posterior FBs	Cost and catheter availability
			Possible trauma
			Needs good anesthesia
Surgical instruments	Both	Availability	Trauma
			Posterior displacement of FBs
Manufactured instruments	Both	Instrument can be designed to fit the FB shape	Trauma
			Posterior displacement of FBs

FBs, foreign bodies; TM, tympanic membrane.

Adapted from Davies PH, Benger JR: Foreign bodies in the nose and ear: A review of techniques for removal in the emergency department. J Accid Emerg Med 17:91, 2000.

and magnification are accomplished by either the diagnostic or operating head of a fiberoptic otoscope. An assistant should hold the pinna back and out so that the examiner may hold the otoscope with one hand and manipulate the instrument with the other. A speculum and either a head lamp or head mirror/light source can also provide illumination, but magnifying loupes are usually required for adequate visualization. Use small alligator forceps to remove objects with edges that can be grasped, but avoid trying to encircle an impacted round FB, as this may cause trauma to the canal wall. A small right-angle hook is another choice. Place the tip past the object, rotate it 90°, and then pull the object from the canal. Fine-tissue or Adson forceps, curettes, and skin hooks are other instruments

TABLE 65-2 -- Recommended Equipment for Ear and Nose Foreign Body Removal

Local anesthesia
Vasoconstrictors
Sedation (as per hospital/department protocols)
Visualization equipment
Otoscope

Nasal speculum
Ear, nose, and throat speculum
Headlight
Loupes
Illuminating magnifying glass
Specific instruments
Wire loop
Right angle hook
Cerumen curettes
Alligator forceps
Curved hook
Bayonet forceps
Suction and catheters of various sizes and shapes (Hognose catheter)
Irrigation equipment
Hartman's forceps
Foley and Fogarty catheters

that are used occasionally. Use of these instruments is commonly associated with abrasions and bleeding of the ear canal. [35] They should be used only on compliant, cooperative patients and direct visualization of the object is essential.

Small fogarty catheters.

Small Fogarty catheters (biliary or vascular) may be used in a manner similar to that described later in the chapter for nasal FB removal. [36] Attach the catheter tip to a 2-mL syringe and gradually inflate the balloon once the tip is past the object; then drag the FB out along with the balloon. Immediately deflate the balloon if sudden pain occurs because tympanic membrane rupture is a potential complication.

Cyanoacrylate (Superglue).

The use of glue in FB removal was first reported in India in 1977. [35] [37] Glue is most effective in removing smooth, round objects that are difficult to grasp. The FB should be dry and easily visualized. Apply a small amount of glue to the tip of a thin paintbrush, a straightened paper clip, or the blunt end of a wooden cotton-tipped applicator.[36] Allow the glue to become tacky. Place the tip against the object, allow it to dry, then carefully withdraw the FB. Minor complications are possible if the tip dries against the canal wall (abrasion, excoriation) or if the glue spills or drips onto the wall (creating a new FB). This technique may be more useful in adults as cooperation is required. [37]

Dissolution of styrofoam.

Styrofoam impaction in the ear canal can be problematic. Application of acetone in a very small amount (0.1 mL) was reported to effectively dissolve deep-seated Styrofoam in a child's ear canal.[39] The ear was irrigated immediately after carefully dripping the acetone directly onto the Styrofoam, and no significant complications of the canal or tympanic membrane were noted. The potential ototoxic effects of acetone in the middle ear have not been studied; therefore, this technique should not be performed if there is any suggestion or history of tympanic membrane perforation. Ethyl chloride has also been effective in dissolving Styrofoam beads. [35] Once again, this technique should not be performed if there is any suggestion or history of tympanic membrane perforation.

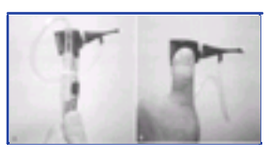

Figure 65-25 The Hognose device for foreign body removal. *A*, The Hognose attached to the otoscope and to wall suction. *B*, Occlusion of the open insufflation port to engage suction and remove the foreign body (attached).

Removal of Insects.

Cockroaches are the most commonly found live insect as an intra-aural FB; treatment consists of instillation of various substances into the ear canal to first immobilize or kill the bug before removing it. This helps retrieval by allowing for a stationary target and also halts the disturbing and painful movement of the insect. Controversy exists about which agent most effectively accomplishes this. Mineral oil has traditionally been used, although lidocaine has been reported to paralyze roaches and to allow for easier extraction than the more viscous mineral oil. An in vitro comparative study showed that immersion in mineral oil and 2% or

Figure 65-26 Application of various methods of foreign body removal from the ear canal. *A*, Right-angle hook. *B*, Irrigation. *C*, Alligator forceps. *D*, Soft-tipped suction.

4% lidocaine solution killed roaches in <60 seconds (~27 and 41 seconds, respectively). [40] The roaches struggled less in the viscous oil than in the lidocaine, which did not appear to cause paralysis. Other substances (Auralgan, isopropanol, water, succinylcholine, hydrogen peroxide) were shown to be ineffective in killing the roaches in a reasonable amount of time. Once disabled, insects may be removed with mechanical extraction as previously described, and pieces can be suctioned out if fragmentation occurs. Mineral oil is safe, but lidocaine has been reported to cause vertigo and middle ear symptoms in patients with tympanic membrane perforations.[41]

Figure 65-27 *A,* Subperichondrial hematoma within the concha of the ear. *B,* Needle aspiration of an auricular hematoma. A topical antiseptic is used to clean the ear, but local anesthesia is seldom required. While stabilizing the pinna with the thumb and fingers, the most fluctuant part of the hematoma is punctured with a 20-ga needle. The thumb "milks" the hematoma into the syringe until the entire hematoma has been evacuated. The thumb maintains continued pressure on the ear for 3 minutes after the needle has been withdrawn. A pressure dressing is then applied, and the ear is checked for reaccumulation of blood in 24 hours. Reaspiration may be required, and persistent accumulations require incision and drainage. *(B redrawn with permission from Fleisher GR, Ludwig S, Henretig FM, et al: Textbook of Pediatric Emergency Medicine. Baltimore, Williams & Wilkins, 1983.)*

Follow-up/Complications

Hearing should be evaluated before and after FB removal, especially in patients with suspected tympanic membrane or middle ear injuries. Examine both the opposite ear and nose of children to search for the rare but possible second FB. Minor lacerations or excoriations of the canal usually heal quickly with or without antibiotic eardrops, so long as the canal is kept clean and dry. Document preexisting canal trauma or suspected tympanic membrane rupture before attempts at removal; otherwise, this may falsely be attributed

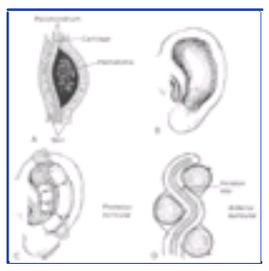

Figure 65-28 Auricular hematoma. *A*, Hematoma separating the perichondrium from the cartilage. *B*, Incision *(arrows)* made along the skin curvature at the posterior edge of the hematoma. Hematoma is evacuated and area irrigated. *C*, Two anterior dental rolls are secured with sutures to a posterior dental roll to maintain normal anatomy of the pinna. *D*, Side view illustrates position of sutures and dental rolls in relation to incision site. Note that the perichondrium is apposed to the cartilage. *(From Clemons JE, Seveneid LR: Otohematoma. In Cummings CW (ed): Otolaryngology—Head and Neck Surgery, 2nd ed. St Louis, Mosby-Year Book, 1993, p 2866.)*

to iatrogenic causes at a later date. Most patients with FBs that cannot be removed in the emergency setting can be safely referred to an otolaryngologist the next day. Exceptions that require immediate consultation include severe pain, suspected tympanic membrane rupture, embedded button batteries, or a concomitant canal infection. Generally, no routine follow-up is necessary except in cases of infection, severe trauma, or tympanic membrane perforation. Parents should be educated to reduce the exposure of children to potential FBs.

Auricular Hematoma

Auricular hematomas occur after a shearing force to the ear, most commonly in wrestlers. A subperichondrial hematoma forms, separating the perichondrium from the cartilage. Recurrent or untreated injuries allow the development of new cartilage, which subsequently deforms the auricle (cauliflower ear).

Procedure

The treatment of an auricular hematoma involves complete evacuation of the subperichondrial hematoma and reapproximation of the perichondrium to the cartilage.

Needle aspiration.

Aspiration of an auricular hematoma is performed by perforating the hematoma with a 20-ga needle (Fig. 65-27). The hematoma is "milked" between the thumb and forefinger until the entire hematoma is evacuated. A pressure dressing is subsequently applied. Frequently reexamine

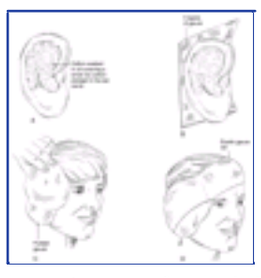

Figure 65-29 Compression dressing of the ear. Following successful aspiration of an auricular hematoma, a compression dressing is used to prevent reaccumulation of the hematoma or fluid. *A,* Dry cotton is first placed into the ear canal. A conforming material is then carefully molded into all the convolutions of the auricle. One may use Vaseline gauze or cotton soaked in mineral oil or saline. *B,* When the convolutions are fully packed, a posterior gauze pack is placed behind the ear. A V-shaped section has been cut from the gauze to allow it to fit easily behind the ear. *C,* Multiple layers of fluffed gauze are placed over the packed ear, and the entire dressing is held in place with Kling gauze or an elastic gauze roll. *D,* The ear is thus compressed between two layers of gauze, and the packing ensures even distribution of pressure to all parts of the auricle.

the ear for reaccumulation of the hematoma. Reaccumulation of blood requires reaspiration. Because of the possibility of inadequate evacuation and recurrent hematomas, the author does not recommend needle aspiration.

Incision.

The auricular hematoma, if <7 to 10 days old, may be incised along the natural skin folds. Anesthetize the pinna using local infiltration of 1% lidocaine (without epinephrine) or by an auricular block (described earlier). Incise the skin with a No. 15 blade at the edge of the hematoma, following the curvature of the pinna (Fig. 65-28). Gently peel the skin and perichondrium off the hematoma and underlying cartilage. Completely evacuate the hematoma and irrigate the remaining pocket with normal saline.

Aftercare.

After removal of the hematoma, apply antibiotic ointment and reapproximate the perichondrium to the cartilage via a pressure dressing. A compression dressing may be applied to the ear as shown in Figure 65-29 . An alternative

technique is to suture dental rolls over the area (see Fig. 65-28).[42] To accomplish this, pass a 4-0 nylon suture through the entire thickness of the ear over the hematoma. Wrap the suture around a dental roll on the posterior aspect of the ear and then pass the needle back through the pinna. Wrap and tie the suture around a second dental roll on the anterior aspect of the pinna. A second suture may be placed to secure a third dental roll. The dressing should firmly re-approximate the perichondrium to the cartilage without vasculature compromise. Remove the dressing in 1 week.

Prescribe anti-staphylococcal antibiotics and instruct the patient to inspect the wound frequently for evidence of vascular compromise or infection, or both. Reevaluate the wound in 24 hours for recurrence of the hematoma. Infection is treated with bandage removal, surgical drainage, and IV antibiotics. Refer patients with auricular hematomas of >7 days' duration to a surgeon, as the new perichondrial growth must be debrided to prevent auricular deformation.

NOSE

Anatomy

The nose consists of the vestibule, nasal septum, lateral wall, and the nasopharynx. The vestibule is the anterior most portion of the nares, which is composed of skin and contains the hair follicles. The nasal septum is the midline structure, which is composed of cartilage anteriorly and of bone posteriorly. The lateral wall of the nose contains the superior, middle, and inferior turbinates as well as the auditory tube opening.

Three major arteries supply the nose and conjoin via anastomoses. The sphenopalatine artery emerges from the sphenopalatine foramen, which is located at the posterior aspect of the middle turbinate (Fig. 65-30). This is the most common source of posterior epistaxis. This artery supplies the turbinates laterally and the posterior septum. The anterior and posterior ethmoidal arteries branch off the ophthalmic artery and penetrate the cribriform plate to supply the superior nasal mucosa. The superior labial branch of the facial artery completes the triad, supplying the nasal septum and vestibule. The

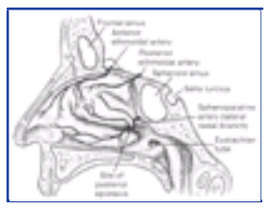

Figure 65-30 Vascular supply to the lateral wall. The most common site of posterior epistaxis is the sphenopalatine artery as it emerges posterior to the middle turbinate. *(From Maceri DR: Epistaxis and nasal trauma. In Cummings CW (ed): Otolaryngology—Head and Neck Surgery, 2nd ed. St Louis, Mosby-Year Book, 1993, p 728.)*

watershed area on the anterior septum, also known as Kiesselbach plexus, is the most common source of anterior epistaxis (Fig. 65-31).

Anesthesia

Apply local anesthetic and vasoconstrictors on cotton swabs. Placement of these swabs is illustrated in Figure 65-32 . If a larger area of anesthetic is required, use cotton pledgets. Figure 65-33 describes the procedure of making pledgets. Soak each pledget in anesthetic or a vasoconstricting substance and then squeeze the excess fluid out of the pledget. Be aware of the total amount of drug being administered and stay within recommendations for the maximum safe dosage. Place each pledget horizontally on the floor of the nasal cavity,

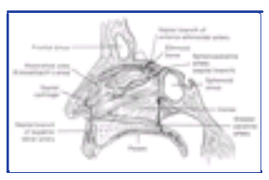

Figure 65-31 Vascular supply to the septum. The most common site of anterior epistaxis is within the area labeled Kiesselbach's plexus. *(From Maceri DR: Epistaxis and nasal trauma. In Cummings CW (ed): Otolaryngology—Head and Neck Surgery, 2nd ed. St Louis, Mosby-Year Book, 1993, p 728.)*

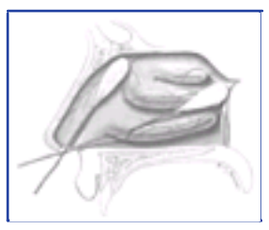

Figure 65-32 Placement of local anesthetic in the nose for anesthesia before reduction of nasal fracture by blockage of the anterior ethmoidal nerve superiorly and the sphenopalatine ganglion at the posterior end of the middle turbinate. *(From DeWeese DD, Saunders WH, Schuller DE, Schleuning AJ II (eds): Otolaryngology—Head and Neck Surgery, 7th ed. St Louis, CV Mosby, 1988.)*

stacking the next pledget on top. Three pledgets are usually required to pack the nasal cavity. These can be replaced with new pledgets in 5 minutes if the desired anesthetic effect is not achieved. Hurricaine spray may also be used as a topical

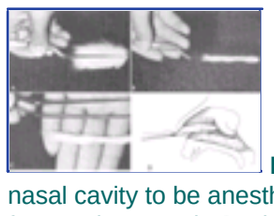

Figure 65-33 Topical anesthetic and vasoconstrictors are applied on individually made cotton pledgets. The size of the pledget may be changed according to the extent of the nasal cavity to be anesthetized and the size of the patient. *A,* An appropriately sized cotton pledget is grasped in a bayonet forceps. *B,* The cotton is then grasped with the opposite hand, and the forceps is rotated. *C,* The pledget is removed and is ready for insertion. *D,* To completely anesthetize the nasal cavity, three pledgets are necessary. The first is placed on the floor of the nose, the second in the middle meatus between the inferior and middle turbinates, and the third in the roof of the nasal cavity and the anterior nasal vestibule. *Note:* This pledget technique can be used to make a cotton wick for the treatment of otitis externa.

anesthetic. Remind the patient that excess anesthetic may numb the throat but will not inhibit swallowing.

As with all drugs, review the maximum dose allowed before administering the anesthetic (see Chapter 30). Always use the smallest amount of anesthetic required to obtain adequate analgesia. The maximum dose of lidocaine without epinephrine is 5 mg/kg, up to 300 mg. The maximum dose of lidocaine with epinephrine is 7 mg/kg, up to 400 mg. The maximum dose for cocaine is 3 mg/kg for mucosal application, up to 200 mg. Remember that these are guidelines and that any patient can have a severe reaction with less than the total dose. For a 60-kg person, the maximum total dose of lidocaine would equal 300 mg (60 kg × 5 mg/kg = 300 mg). A 1% solution (10 mg/mL) would allow for a total of 30 mL to be administered (30 mL × 10 mg/mL = 300 mg). A 2% solution (20 mg/mL) would allow for only 15 mL to be administered (15 mL × 20 mg/mL = 300 mg). Cocaine is packaged in a 4% solution (40 mg/mL), allowing only 5 mL for a mucosal application to a 70-kg patient (3 mg/kg × 70 kg = 210 mg; 5 mL × 40 mg/mL = 200 mg, which is the maximum total dose). Clinicians should be familiar with the side effects and potential complications of the drugs being administered. Cocaine can induce tachycardia, hypertension, irregular respirations, seizures, and hyperpyrexia (especially in children). Lidocaine will manifest its antiarrhythmic and membrane stabilization effects.

Examination

Examination of the nares is relatively straightforward. When using a nasal speculum, insert it into the naris with the handle parallel to the floor and slowly open the blades in the superior-to-inferior direction. Stabilize your hand on the patient's nose to prevent damage to the mucosa due to unexpected movement (Fig. 65-34).

When attempting to visualize the nasal passageway, remember to have the patient keep the floor of the nose parallel to the ground. Tilting of the head only allows for a view of the anterosuperior area (Fig. 65-35). A nasopharyngoscope may be used to view the nasal passageway as well, and its use is described in the previous section on examination of the pharynx.

Epistaxis

Nasal hemorrhage commonly presents to the ED. Identification of the source of bleeding and subsequent control are paramount to the treatment of epistaxis. Although this can be frightening to both clinician and patient, a systematic approach with the proper equipment will lessen the anxiety of the situation. The purpose of the procedure is to tamponade the bleeding. If the source is anterior, this may be the final treatment. For posterior bleeds, these are generally temporizing maneuvers until a consultant can complete a definitive hemostatic procedure. The procedures can be performed in the ED with proper lighting and the equipment listed later in this section. Controlling epistaxis may be a time-consuming process without proper equipment or patient cooperation.

In preparation for any procedure to treat epistaxis, evaluate the patient's hemodynamic status by assessing vital signs and orthostatic symptoms and quantifying blood loss. Also determine if the patient has any underlying medical problem, such as angina or chronic obstructive lung disease, which may be exacerbated due to hypovolemia or anemia. If the patient is symptomatic in any of these areas, or if the blood loss is deemed significant, start a large-bore IV line and administer fluid boluses. Hematologic testing is rarely useful and not required for most patients, but if there are extenuating circumstances, obtain a complete blood count

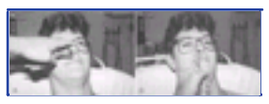

Figure 65-34 To examine a nose properly, a nasal speculum must be used. *A,* The clinician's index finger rests on the bridge of the nose, and the speculum is spread in an inferior-to-superior direction. *B,* It is *incorrect* to spread the speculum laterally or to use the instrument in an unsupported manner.

and consider a type and screen. Coagulation studies are not routinely indicated but should be undertaken in patients taking anticoagulant therapy, those with underlying hematologic abnormalities, or those with recurrent or prolonged epistaxis. [43]

Many patients with epistaxis are hypertensive as well. No direct correlation has been proven between hypertension and epistaxis. Most authors now consider hypertension to be a stress response instead of an inciting event. [44] Therefore, hypertension does not require treatment until the bleeding is controlled and the anxiety of the situation has resolved. However, any patient exhibiting other signs of a hypertensive emergency needs immediate treatment in addition to control of the epistaxis.

Indications/Contraindications

Any continuing episode of epistaxis can be treated with the following techniques. Massive facial trauma with the possibility of a basilar skull fracture would preclude the use of an intranasal balloon, as it may travel into the skull cavity.

Equipment

The key to successful management of a patient with epistaxis is preparedness. The following list of equipment should be readily available to the emergency clinician.

- Chair with headrest or gurney with inclinable back
- Headlight with light source, head mirror
- Wall suction with multiple suction catheters
- Gloves, mask, and gown for clinician
- Gown or drapes for patient
- Topical anesthetic
- Topical vasoconstrictor
- Nasal speculum
- Tongue depressors
- Small red rubber catheters
- Bayonet forceps
- Scissors
- Kidney basin
- Gauze (4 × 4 in., 2 × 2 in.)
- Dental rolls or cotton
- No. 2 surgical silk ties

- 1.2-cm wide Vaseline gauze or 0.5-in. wide Nu-Gauze packing
- Antibiotic ointment
- Silver nitrate sticks or electrocautery
- Pediatric Foley catheters (12 Fr)
- Nasal tampons
- Dual balloon pack

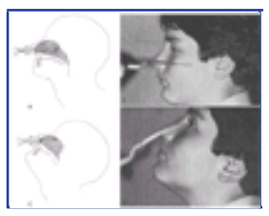

Figure 65-35 *A* and *B,* Correct position for examining and treating diseases of the nasal cavity. The patient is in the "sniffing" position, sitting upright or leaning slightly forward with the head only slightly extended. When the nasal tip is raised with the nasal speculum, the view is parallel to the floor of the nose and allows visualization of the entire nasal cavity. *C* and *D,* When told that their nose will be examined, most patients extend the neck and look toward the ceiling. In this position, only the most anterior portions of the nasal cavity are visible.

Epistaxis Examination

As most patients are frightened by continued epistaxis, reassure the patient that you will control the bleeding. Drape the patient with a gown to protect his or her clothing from the bleeding. Have the patient hold an emesis basin to collect any continued bleeding and as a precaution to emesis of swallowed blood. Minor anterior bleeds are usually easily controlled with minimal techniques, patients with significant epistaxis can be quite anxious, and manipulation of the nose can be quite painful. Therefore, parenteral sedation/analgesia with appropriate physiologic monitoring should be considered when extensive manipulation is anticipated.

Have the patient sit upright in the "sniffing" position with the neck flexed and head extended. The base of the nose should remain parallel with the floor. Position yourself in front of the patient, level with the patient's nose after putting on a face shield, gown, and protective gloves. Allow the patient to clear the nose of any blood clots by forceful blowing or, alternatively, you may suction the passageway until it is free of clots. Suctioning should be done from front to back along the nasal septum, then laterally. If the bleeding is minimal, attempt to identify the bleeding source. If the bleeding is too profuse for visualization, administer a topical anesthetic and vasoconstrictor. The easiest way to apply the preparations is to simply soak a cotton pledget in the solution and place it in the nose for 4 to 5 minutes. The patient can clamp the nostrils to limit bleeding and promote contact with the mucosa. Numerous preparations are available. A 4% cocaine solution would be ideal, but it is not commonly stocked. The 1% to 2% lidocaine used for laceration repair does little to provide topical anesthesia or vasoconstriction. A solution of 1% tetracaine and 0.05% oxymetazoline (Afrin) is an effective

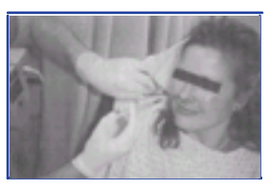

Figure 65-36 If an arterial bleeding site is found in the nasal septum, both hemostasis and anesthesia for cautery can be accomplished by injecting the mucosa at the base of the bleeder with a small amount of lidocaine with epinephrine via a tuberculin or insulin syringe. This procedure best follows an initial application of topical anesthesia.

topical anesthetic and vasoconstrictor.[45] If a discrete bleeding site is initially identified, an effective way to provide hemostasis and anesthesia before cautery is to inject the mucosa at the base of the bleeder with 2% lidocaine with 1:100,000 epinephrine via a tuberculin syringe (Fig. 65-36).

Insert the nasal speculum into the naris and use the suction catheter to evacuate any blood. Do not hesitate to reapply anesthetic or vasoconstrictor if the desired results have not been achieved. As most cases of anterior epistaxis occur in Kiesselbach plexus, inspect this area closely for areas of bleeding, ulceration, or erosion. If the patient presents with a history of epistaxis but is no longer bleeding, gentle stroking of the anterior nasal mucosa with a cotton swab or having the patient forcefully blow the nose may reveal the bleeding source. If no source is found and the bleeding has ceased, pack the nose only if the epistaxis is recurrent. If no anterior source is found and bleeding continues down the posterior pharynx, assume a posterior source and pack the nose with an anterior and posterior pack.

Figure 65-37 Equipment used for treatment of epistaxis. *Top:* Electrocautery device and silver nitrate sticks. Left to right, bottom row: Merocel nasal tampon, posterior packing ball, Merocel nasal tampon with catheter included (Doyle Pack), anterior packing gauze with bayonet forceps, Foley catheter, Epistat dual balloon catheter.

Cautery

After identifying an anterior source of bleeding, there are several options to obtain hemostasis (Fig. 65-37). *Silver nitrate sticks* may be used to cauterize the area. Remember that silver nitrate sticks will not cauterize an actively bleeding source; therefore, hemostasis must be achieved first. If a bleeding site is seen, hold the silver nitrate stick tip gently against the site for 4 to 5 seconds. If needed, a second application can be applied. If the bleeding site is not seen, but a blood vessel is identified, the lightly moistened cautery stick can be gently stroked along the vessel until the desired burn depth is achieved. Wipe away any excess silver nitrate to prevent inadvertent cautery of other areas of the nose. If this restarts the bleeding, the initial cautery was insufficient. *Electrocautery* will work in the same manner, but the cauterization will penetrate more quickly than the silver nitrate sticks. In either cautery technique, care must be taken to prevent inadvertent septal perforation due to overaggressive or repeated cautery. If cautery has not been successful after two attempts (either twice at the first visit or on the second visit), another technique is indicated. *Multiple cautery attempts can significantly injure the nasal septum, and bilateral cautery should not be performed.* Cautery works well for a small, circumscribed area of bleeding.

If this is the initial bleed and hemostasis is achieved, no packing is necessary. If this is a recurrent bleed within 72 hours of another, or if cautery does not provide hemostasis, the anterior cavity should be packed. If hemostasis is accomplished, apply Vaseline or antibiotic ointment to the area to prevent desiccation. Do not administer aspirin or nonsteroidal anti-inflammatory drugs for 4 days after epistaxis. If bleeding recurs at home, instruct the patient to pinch the nostrils closed for 20 minutes. Instruct the patient to return to the ED if this maneuver is unsuccessful or the bleeding is profuse.

Anterior Nasal Packing

Anterior gauze packing is placed to complete hemostasis, prevent desiccation, and protect the area from trauma. However, improperly placed packing may further abrade the area, dislodge prematurely, or migrate into the posterior pharynx. Anterior packing must be placed with adequate analgesia,

proper visualization, and deliberate movements. Traditional petrolatum gauze packing is applied in an "accordion" fashion so that each layer extends the entire length of the nasal cavity. However, this type of packing has been almost entirely replaced with specialized compression devices that are easier to place, better tolerated, and very successful.

Coating any packing material with antibiotic ointment aids in its placement and theoretically prevents infection and toxic shock syndrome secondary to nasal packing. Proper placement of the speculum allows visualization of the floor of the nasal cavity. Lay a strip of 1.2-cm (0.25- or 0.5-in. Nu-Gauze) gauze across the nasal floor, with the starting end of the gauze at the naris (Fig. 65-38). Replace the speculum over the layer of gauze, gently packing it to the floor of the nose. Measure the gauze to twice the length of the nasal cavity. Grasp the gauze at the midpoint and insert this point to the posterior aspect of the nasal cavity. This allows placement of the gauze without movement of the underlying layer. Continue this pattern, replacing the speculum after each layer until the cavity is filled.

Preformed nasal packing products are convenient alternatives to anterior nasal packing (e.g., Merocel *nasal tampon*

Figure 65-38 The key to placement of an anterior nasal pack that will control epistaxis adequately and stay in place is to lay the packing into the nasal cavity in an "accordion" manner, so that part of each layer of packing lies anteriorly, preventing the gauze from falling posteriorly into the nasopharynx. *A,* The first layer of 0.25-in. Vaseline gauze strip is grasped approximately 2 to 3 cm from its end. *B,* The first layer is then placed on the floor of the nose through the nasal speculum (not pictured here). The bayonet forceps and nasal speculum are then withdrawn. *C,* The nasal speculum is reintroduced on top of the first layer of packing, and a second layer is placed in an identical manner. After several layers have been placed, it is often useful to reintroduce the bayonet forceps to push the previously placed packing down onto the floor of the nose, making it tighter and more secure. *D,* A complete anterior nasal pack can tamponade a bleeding point anywhere in the anterior nasal cavity and will stay in place until removed by the clinician or patient.

or Epistat II nasal catheter [Xomed Surgical Products, Jacksonville, FL]) (Fig. 65-39*A* and *B*). The Merocel packing consists of compressed polyvinyl acetate with or without a drawstring that expands on contact with fluid. The pack can be trimmed with scissors or a scalpel before insertion. The Merocel Doyle nasal pack (see Fig. 65-37) has an airway tube in the center of the compressed material and is a more anatomic shape. Each are available in various sizes, but usually an 8 × 1.5 × 2-cm standard Merocel or 8 × 1.5 × 3-cm Doyle will suffice. The Rapid Rhino Stat Pac (Applied Therapeutics, Tampa, FL, rapidrhino.com) is a high-volume, low-pressure balloon device with an open lumen air passage, a pilot cuff to check pressure, and a gel knit covering designed to promote platelet aggregation. Numerous variations for anterior, posterior, and combination packs are available (Fig. 65-40).

The easily applied nasal tampon is a reasonable first choice for most anterior bleeds. The tampon is lubricated with antibiotic ointment and inserted into the nasal cavity with a bayonet forceps. The insertion may be painful so a rapid single motion is suggested. Generous ointment and careful trimming can minimize the trauma of insertion. The packing is

Figure 65-39 Nasal packing alternatives. *A,* Merocel nasal pack. (1) Merocel sponge compressed and expanded. To insert the sponge packing, lubricate the device with an antibiotic ointment and place it in the nasal cavity. (2) Some trimming of the product may be needed and prior use of topical anesthesia is advised. When the sponge is in place, hydrate it with saline. Expansion of the moist sponge will compress the bleeding site. (3) It may remain in place for up to 3 days. Rehydrate the sponge before removal. *B,* Epistat II nasal catheter. The catheter is usually left in place up to 3 days and can be used in outpatients. A hollow inner airway tube allows the patient to breathe.

advanced along the floor of the nose. Once the packing is in the nasal cavity, it is expanded with 5 to 10 mL of saline, although, oftentimes, contact with moisture will cause it to swell (Fig. 65-41). It is sometimes necessary to place two tampons side by side before inserting them, to fill the nasal cavity. [44] [46] Advantages to the Merocel tampon include rapid insertion, little discomfort, ease of use even in inexperienced hands, and possible inhibition of bacterial growth. [47] Corbridge[48] found that there was no significant difference in efficacy, patient tolerance, or complications when compared to gauze packing.

Anterior packs are usually left in for 2 to 5 days. Premature removal may result in rebleeding. During use and before removal, the commercial nasal tampon should be hydrated with saline. If it contains an airway tube, first remove the tube, then irrigate the space once occupied by the tube.

Complications

Any packing in the anterior nasal cavity may obstruct drainage of the paranasal sinuses or block the nasolacrimal ducts. In addition, because it is a FB, it will stimulate mucus production and act as an impetus for infection. Antibiotics (amoxicillin, trimethoprim/sulfamethoxazole, and others) are often prescribed with any packing left in the nose after emergency treatment because of the risk of sinusitis and toxic shock syndrome. The necessity of antibiotics for short-term anterior packing is unproven. Decongestants are also often concomitantly prescribed to decrease secretions. Practices vary and no common standard exists. Hollis [47] reported massive pneumocephalus following Merocel nasal tampon insertion in an elderly woman, presumably from fracture of the ethmoid plate. There have been case reports of ethmoid

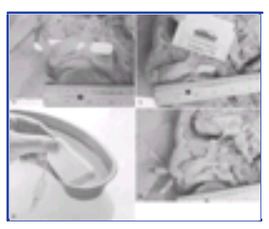

Figure 65-40 Examples of various packs for epistaxis in a cadaver model. *A*, The posterior Merocel sponge, not inflated, will stop most nosebleeds and is more comfortable than some balloon devices. *B*, Wetting the Rapid Rhino before placement activates the slippery covering for easier insertion. *C*, Anterior/posterior Rapid Rhino in place. *D*, Posterior pack with balloon inflated.

fracture following anterior nasal gauze packing and with the use of an intranasal balloon.

Posterior Nasal Gauze Packing

The patient has posterior epistaxis if no source of bleeding is found anteriorly and the patient continues to hemorrhage down the posterior pharynx. Posterior epistaxis may respond to topical vasoconstrictors. However, anterior nasal packing will not provide hemostasis for a posterior bleed, as it will not cover the source of bleeding. A posterior pack directly compresses the sphenopalatine artery and prevents the passage of blood or anterior packing into the nasopharynx.

The *posterior nasal gauze pack* is the classic method of treating posterior epistaxis. However, because balloon devices are easier to use and are less distressing to the patient, posterior nasal packing is less commonly used. To place a posterior nasal gauze pack, anesthetize the patient's nares and posterior pharynx with topical anesthetic. Prepare a roll of gauze with two No. 2 silk ties secured around the middle and extending in opposite directions. One set of ends will be used to place the posterior pack and the second will remain extruding from the oral cavity to remove the pack. Place a No. 10 red rubber catheter through the bleeding nostril (Fig. 65-42 Fig. 65-42). When it is seen in the posterior pharynx, grasp it with forceps, and guide it out of the mouth. Attach it to one set of ends of the silk ties secured to the gauze pack. Retract the red rubber catheter, thus carrying the No. 2 silk tie through the nasopharynx and out of the nose. Grasp the suture and pull the pack into the nasopharynx. Guide the pack swiftly into the oral cavity and nasopharynx with the other hand. Attach the silk tie that remains in the oropharynx to the patient's cheek to aid in removal or rescue of the posterior pack. Use the silk ties exiting the nostril to maintain the position of the posterior pack. Pack the anterior passage as described for anterior epistaxis. Secure the silk ties over a gauze pad or dental roll. Patients with this type of posterior pack are admitted to the hospital.

Inflatable Balloon Packs

Inflatable balloons come in two varieties. The *Foley catheter* is often used as a posterior pack because of its availability,


Figure 65-41 An expanding Merocel tampon is ideal for minor anterior bleeds. The dehydrated pack is trimmed to fit the nose and generously lubricated with antibiotic ointment. A swift single motion is used to insert the pack to its full length. The pack expands when hydrated with saline. The patient continues to keep the pack moist at home and it is again well hydrated just before removal.

ease of use, and successful tamponading effect.[49] Insert a 12 Fr Foley catheter through the bleeding naris into the posterior pharynx (Fig. 65-43). Inflate the balloon halfway with about 5 to 7 mL of normal saline or water. Slowly pull the Foley into the posterior nasopharynx and secure it against the posterior aspect of the middle turbinate. Finish inflating the balloon with another 5 to 7 mL of normal saline or water. If there is pain or inferior displacement of the soft palate, deflate the balloon until the pain resolves. Complete inflation of the balloon before proper placement causes the balloon to remain too posterior in the nasopharynx, thus failing to achieve hemostasis.[50] While maintaining constant gentle anterior tension on the Foley catheter, an anterior nasal packing is placed using layered petroleum-impregnated gauze. The opposite nasal cavity is packed to counteract septal deviation. Then, place a short section of plastic tubing over the catheter and secure it with a nasogastric tube clamp or umbilical clamp. Be careful not to exert undue pressure on the nasal alar, as this may cause necrosis.

The second type of inflatable balloon pack is the premade *dual balloon tamponading system*. Goitschach Nasostat (Sparta Surgical Corp, Hayward, CA) and Xomed Epistat (Xomed Inc, Jacksonville, FL) are examples of available devices. The *dual balloon pack* has a posterior balloon that inflates with about 10 mL of air and an anterior balloon that inflates with about 30 mL of air (Fig. 65-44). Each device may vary slightly (Fig. 65-45). After appropriately anesthetizing the naris, place the lubricated pack along the floor of the affected naris as far back as possible. Inflate the posterior balloon about halfway with air and then pull the balloon into place up against the posterior aspect of the middle turbinate. Complete the inflation of the posterior balloon with air. Some clinicians prefer to inflate all balloons with saline instead of air since air may deflate slowly. If the patient complains of pain or if the posterior soft palate deviates downward, deflate the balloon until the symptoms are relieved. Maintain the position of the balloon and inflate the anterior balloon with up to 30 mL of air. Again, halt the inflation if the patient experiences increasing pain or deviation of the nasal septum. Some authors suggest packing the opposite naris to prevent this lateral deviation. A small piece of gauze is placed between the nose and external catheter hub to decrease skin irritation.

Other Techniques

ENT consultation may be required for posterior nosebleeds that do not respond to the previously described posterior packing techniques. Other treatment options include ligation or embolization of the internal maxillary artery and posterior endoscopic cautery.

Complications

Care of the posterior nasal packing is of some concern. Posterior nasal packing is uncomfortable and often painful. These bleeds are more complicated than simple anterior septal bleeds. Complications associated with posterior packing include infection, dysphagia, eustachian tube dysfunction, tissue necrosis, and dislodgment. Other serious complications associated with posterior packing are hypoxia, hypercarbia, aspiration, hypertension, bradycardia, arrhythmias, myocardial infarction, and death. [51] Therefore, most patients, with a posterior pack, especially the elderly and those with pulmonary and cardiovascular diseases, should be admitted to the hospital for sedation and monitoring. This recommendation was common for formal posterior packs, but the ease and safety of the balloon devices now allow select patients to be treated as outpatients despite the presence of posterior packing. Rebleeding may also be seen with early pack removal; one series found pack removal within 48 hours to increase the risk of rebleeding. [52] Most posterior packs are left in place 72 to 96 hours.

Infection risk with posterior packing includes toxic shock syndrome, nasopharyngitis, and sinusitis. The packing blocks the sinus ostia, preventing proper drainage of the sinuses. In addition to coating the packing with antibiotic ointment, broad-spectrum antibiotics should be administered. *Dysphagia* due to the packing can lead to poor oral intake, and IV fluid hydration may be required.

A significant decrease in PaO_2 (7.5 to 11 torr) and increase in $PaCO_2$ (7 to 13 torr) is seen in patients with nasal packing who are treated with sedation. [53] A posterior pack will cause vagal stimulation, resulting in varying degrees of bradycardia and bronchoconstriction. This physiologic adaptation is even more worrisome in patients with underlying lung or heart disease. With the risk of *hypercarbia* and *hypoxia*, patients with lung disease with posterior nasal packs should be closely monitored in an intensive care setting.

Tissue necrosis of the nasal ala, nasal mucosa, and soft palate has been described secondary to improper placement or padding. Protecting the skin with gauze placed under the device will reduce skin maceration. The risk of necrosis increases with the duration of the packing, so all packing should be removed in 3 to 5 days. Bleeding from the nasolacrimal duct is a common benign problem with nasal packing (Fig. 65-44*D*)

If the *posterior pack becomes dislodged*, it will fall into the oropharynx with the risk of asphyxiation, vomiting, and aspiration. The patient and nursing personnel need to be familiar with the technique for removing the pack. To accomplish this, cut the anterior sutures that exit the naris from the gauze roll if they have not already broken. Grasp the sutures exiting the mouth and guide the packing out of the nasopharynx. It may be necessary to manually extract the packing with forceps or digits.


Figure 65-42a Posterior nasal pack. *A,* Following topical anesthesia, a red rubber catheter is passed through the nose and carefully grasped in the oropharynx with ringed forceps and brought out through the mouth. *B (upper right),* A posterior nasal pack made by wrapping a cotton ball in a 4 × 4-in. gauze pad and tying 2 long silk sutures or umbilical tapes around the neck of the pack. One tie must be left long so that it can be taped to the cheek until needed for removal of the pack. *C (*center), Alternatively, a gauze pad can be folded and rolled into a cylinder and tied with two strings. Two of the long strings are used to tie the pack to the tip of the catheter and the other two will be used to remove the pack. *D,* As an option, a second catheter, which has been passed through the nonbleeding side and brought out the mouth, can be used to retract the palate forward to aid in the placement of the pack (not shown). The optional "retraction" catheter is removed after the pack is in the proper position.


Figure 65-42b *E,* The pack is digitally guided into the nasopharynx. *F,* A gauze roll secures the pack to the nose and the rescue ties are taped to the cheek.

Toxic shock syndrome has been rarely described with nasal packing. [46] [54] The syndrome is caused by a toxin released by *Staphylococcus aureus* infection of the packing. Sudden onset of vomiting and diarrhea with high fever, as well as development of an erythrodermic rash, heralds the onset of the disease. Untreated, the disease can advance into shock and multi-system organ dysfunction. Therefore, in addition to coating the gauze with antibiotics to decrease the local bacterial concentration, broad-spectrum antibiotics should be given to any patient with nasal packing. No single antibiotic has been traditionally recommended, but a cephalosporin (such as cephalothin), ampicillin/sulbactrim (Unasyn), or ampicillin/clavulanate (Augmentin) appear reasonable. Any symptoms of toxic shock syndrome

should prompt the removal of the packing and admission to the hospital for fluid hydration and IV nafcillin or vancomycin therapy.


Figure 65-43 Foley catheter placed into the nasopharynx, inflated with water, and retracted into position. The distal tip of the catheter has been cut off. An anterior pack (not shown) is then placed around the catheter. The ala and columella are protected with gauze padding, and a plastic umbilical clamp or nasogastric clamp is applied to the catheter to maintain slight tension on the balloon.

Septal Hematoma

Trauma to the anterior portion of the nasal septum may cause a hematoma to form. A buckling stress tears the submucosal blood vessels. If the mucosa remains intact, the blood will accumulate between the mucoperichondrium and the septal cartilage. Stagnant blood is an excellent medium for growing bacteria and forming an abscess. Common bacteria include Staphylococcus aureus, Streptococcus pneumoniae, and Group A ß-hemoltyic streptococcus. Other complications of an untreated hematoma include septal perforation and cartilage destruction, with resultant saddle-nose deformity. Septal hematomas may present immediately following the trauma or, more commonly, may occur in the first 24 to 72 hours following the injury.[55] Septal abscess formation may occur in a matter of days and significant destruction of the nasal cartilage and a cosmetic deformity may be seen.

It is usually possible to diagnose a septal hematoma by inspecting the nasal septum *with a speculum* for swelling, pain, and a fluctuant area. The presence of septal asymmetry with a bluish or reddish hue of the mucosa is suggestive of a septal hematoma. Direct palpation may be necessary, as newly formed hematomas may not yet be ecchymotic. Inspect both sides, as bilateral hematomas are possible. The best way to palpate for a septal hematoma is to insert the gloved small fingers in each side of the nose and palpate the entire septum, feeling for swelling, fluctuance, or widening of the septal space (Fig. 65-46). Most common symptoms of a septal hematoma are nasal obstruction, pain, rhinorrhea, and fever. Most patients will complain of inability to breathe through the affected side, but the absence of nasal obstruction does not rule out a septal hematoma.

Drainage

Treatment of a septal hematoma consists of evacuation of the clot with subsequent reapproximation of the perichondrium to the cartilage. To drain the hematoma, *incise* the mucosa horizontally over the hematoma after adequate anesthesia is achieved (Fig. 65-47). Suction out all of the clot and then irrigate with normal saline. Excise a small amount of mucosa to prevent premature closure of the incision and place a section


Figure 65-44 *A* and *B,* The balloon tamponade device serves as both an anterior and a posterior pack. It is easily inserted and is often successful for the temporary control of posterior epistaxis in the emergency department. *C,* Although often effective for hemorrhage control, balloon inflation within the nasal cavity can be quite painful and prolonged pressure on the nasal alae can produce tissue injury. Gauze should be placed between the external balloon and the skin of the nose. *(Courtesy of Xomed Inc, Jacksonville, FL) D*, Although alarming to the patient, blood in the eye from back bleeding via the nasolacrimal duct from a balloon pack is benign.

of a sterile rubber band to act as a drain. The nostril should then be packed, as in anterior epistaxis, to reapproximate the perichondrium to the cartilage.

Give the patient broad-spectrum antibiotic therapy. Inspect the septum daily for signs of infection, recurrent hematoma, or necrosis. Recurrent hematomas should be evacuated. When there is no further hematoma for a 24-hour period, remove the drain. Pack the affected naris for 1 more day to complete the apposition of the perichondrium to cartilage where the drain has been. Any evidence of infection should prompt admission for IV antibiotics and surgical debridement.

Nasal Fracture

Nasal fracture is the most common facial fracture. Nasal fractures present with symptoms ranging from mild swelling to epistaxis to periorbital ecchymosis with obvious deformity. As with any trauma to the head, the patient must be evaluated for coexistent intracranial injury or neck injury. In the evaluation of nasal trauma, rule out the existence of a septal hematoma or cerebrospinal fluid rhinorrhea. In most cases, swelling and soft tissue deformity prevent adequate evaluation or treatment, or both. Evaluation of the patient with a suspected nasal fracture should include a thorough history, external nasal examination, and internal nasal examination using a nasal speculum with or without the use of a rigid nasal endoscope. Nasal radiographs are not routinely recommended, as they will not alter the course of treatment or injury.[56] [57] [58] Have the patient apply ice to the area and keep the head elevated to treat the soft tissue swelling. Refer the patient to an otolaryngologist or plastic surgeon for reexamination and definitive treatment in 3 to 5 days. Stress to the patient the importance of being evaluated again within 10 days so that the bones do not set in a malaligned state.

Nasal Fracture Reduction

Simple fractures with minimal local swelling can be treated with closed reduction. To minimize potential litigation, obtain written consent and take prereduction and postreduction photographs. Inform the patient that the outcome is not guaranteed, as impacted fractures may not reduce and greenstick fractures may deform again after reduction. Any swelling may obscure the extent of the deformity or the degree of reduction.

Anesthetize the mucosa as described earlier (see Fig. 65-32 and Fig. 65-33). Topical anesthetic (cocaine 4% to 10%) is usually adequate; however, infiltration with 1% lidocaine without epinephrine may be used adjunctively. For infiltrative anesthesia, inject the lidocaine either deep to the nasal fracture by entering intranasally or externally into the fracture site for a hematoma block. The latter approach may be quite uncomfortable for the patient. In the external technique, the needle enters bilaterally at the caudal edge of the nasal bone, midway between the nasal bridge and the maxilla. Bilateral infraorbital blocks may also be used. Intravenous sedation may be required. There is debate in the literature as to which method provides adequate anesthesia with minimal patient discomfort. [58] [59]

Use Asch forceps, Walsham forceps, or a scalpel handle to advance into the naris with the dominant hand (Fig. 65-48). Apply pressure in an anterosuperior direction at a right angle to the ridge of the nasal bone. The other hand manipulates the nasal fracture in an anteroinferior direction. Firm, constant


Figure 65-45 Xomed Rhinology Products. *A,* Xomed Epistat nasal catheter. *B,* Pope Flex-Pak nasal packing (shown expanded and compressed). *C,* Xomed Epistat II nasal catheter. *D,* Staxi-Stat pack without drawstring. *E,* Large Fast-Pak nasal pack with applicator. *F,* Weimert epistaxis packing. *G,* Small Fast-Pak nasal pack with applicator.

pressure is maintained until the bone shifts back into its original position. Alternatively, a Boies elevator may be used to lessen the risk of nasal mucosa damage.

Care must be taken not to perforate the cribiform plate when using these surgical instruments. Assess the reduction for malalignment or subsequent displacement secondary to a greenstick fracture. If either occurs, refer the patient to an otolaryngologist for the possibility of open reduction.

Exterior splint dressings can be applied to maintain reduction. Some authors believe this will mask an incomplete reduction or adversely manipulate the reduction during placement. Remove the splint in 7 to 14 days.[60] As in most closed fractures, antibiotics are not indicated. Epistaxis or direct evidence of an open fracture should prompt referral to an otolaryngologist and initiation of broad-spectrum antibiotics.

Figure 65-46 If visual inspection of the nose with a speculum does not rule out a septal hematoma, the clinician's gloved fingers, passed posteriorly along both sides of the septum, may reveal bulging or fluctuance.

Nasal Foreign Bodies

Nasal FBs frequently present in the pediatric patient, but it is not uncommon to find them in psychiatric or mentally retarded populations as well. Usually a family member has witnessed the event or the patient actually complains of

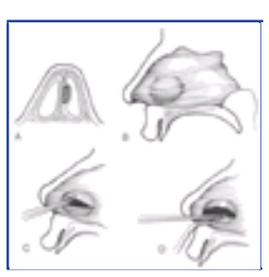

Figure 65-47 *A*, A small left-sided septal hematoma. *B*, After applying appropriate topical anesthesia, supplemented with local infiltration, if necessary, a *horizontal* incision is made through the mucosa and the perichondrium covering the hematoma. *C,* A small cup forceps or scissors is used to remove enough mucosa to prevent premature closure of the wound and reaccumulation of hematoma. *D,* A sterile rubber band is then placed as a drain, and the naris is packed.

Figure 65-48 Reduction of a depressed and dislocated nasal bone fracture. This reduction is accomplished in two steps following anesthesia by first elevating the depressed nasal bone as illustrated and then manually displacing the pyramid to the midline. The handle of a scalpel may be used if an elevator is unavailable. *(From Adams GL, Boies LR Jr, Hilger PA (eds): Boies Fundamentals of Otolaryngology, 6th ed. Philadelphia, WB Saunders, 1989.)*

discomfort from the FB. However, the lack of a history of FB insertion is of little value as most children will not admit to doing it. Therefore, emergency clinicians need to maintain a high level of suspicion for nasal FB, especially in patients who present with unilateral purulent nasal discharge (which may be bloody), unilateral sinusitis, or recurrent unilateral epistaxis. Retained FBs, especially plastic ones, can often initially fail to cause pain or other symptoms. [61] [62]

Types of nasal FBs vary widely and often include food (e.g., meat, nuts, beans), rubber erasers, paper wads, pebbles, marbles, sponges, beads, jewelry, hardware (e.g., nuts, screws), and even certain living larvae or worms.[62] Alkaline button batteries pose a unique problem because they may cause significant nasal injury within hours to days. They are composed of heavy metals like mercury, zinc, silver, nickel, cadmium, and lithium. Injuries reported include mucosal burns, ulcerations, liquefaction necrosis, and even septal perforation, synechiae, and stenosis of the nasal cavity. [62] [63] [64] It is imperative to remove these batteries promptly before tissue damage occurs from leakage of battery contents, electrical currents, or direct pressure. A relatively new and interesting nasal FB is the magnetic nose ring. These are small, commercially available earth magnets usually worn on either side of the alar cartilage, giving the appearance of a pierced nasal stud. The magnets can displace and become polarized across the nasal septum. The magnetic attraction can be quite strong and can lead to pressure necrosis of the nasal mucosa and possible septal perforation. This attraction can also make removal difficult as well as painful for the patient. [65] [66]

Nasal FBs often come to rest on the floor of the anterior or middle third of the nose. Metallic or calcified objects may show up on x-ray, but physical examination remains the most reliable means for diagnosis. Maxillary, ethmoid, or sphenoid sinusitis may also accompany FB retention. Plain radiographs or facial computed tomographic scanning may be of value in detecting sinusitis, although these studies are usually not necessary in the acutely retained object.

Failure to remove nasal FBs results in admission for removal under anesthesia; therefore, it behooves emergency medicine clinicians to be skilled in this procedure. Admission incurs increased cost, inherent procedural risks, and psychological stressors for parents and patients. Like the removal of auricular FBs, the removal of nasal FBs can be both frustrating and time consuming.

Nasal Foreign Body Removal

A cooperative patient is essential; therefore, young children often require general anesthesia for more posterior FBs. While conscious sedation may aid in removal and preclude the need for admission and general anesthesia, the possible increase in aspiration risk must be considered with use of agents that blunt protective airway reflexes. Before attempting removal, anesthetize and vasoconstrict the affected naris, as previously described in the chapter. Obtain assistance to stabilize the patient's head, and immobilize younger patients as required. The more cooperative patients should be placed in the "sniffing" position, and a head lamp should be used for proper illumination. Several of the techniques previously mentioned for EAC FB removal, including the use of cyanoacrylate glue, can be used in the management of a nasal FB.

Use *alligator forceps* or *bayonet forceps* to retrieve more anteriorly lodged FBs with edges that are amenable to grasping. For hard, larger objects, *wire loops, right-angle hooks,* or even a properly bent *paper clip* may be carefully passed beyond the object and rotated, allowing it to be pulled from the naris (see Fig. 65-22). Direct mucosal trauma and epistaxis may occur with any of these methods. FBs with smooth, rounded edges that are difficult to grasp or to pass instruments around may be extracted with a *suction-tip catheter* in a similar manner as that described earlier for EAC FBs. The Hognose catheter described earlier also works well for nasal FBs.

Objects that cannot be removed with anterior instrumentation may be removed with *balloon catheters*. Several case series[67] [68] have depicted Fogarty catheters as being highly effective in removing nasal FBs (Fig. 65-49). A No. 4 or 5 vascular Fogarty catheter, a 12 Fr Foley catheter, and a No. 6 biliary Fogarty catheter have all been described in the literature for this use. The biliary catheter reportedly is less apt to rupture. The patient lies supine after proper vasoconstriction and anesthesia of the affected naris. With a 5-mL syringe attached, and the catheter lubricated with lidocaine gel, the tip is passed above the object and into the nasopharynx. The balloon is then inflated with air or water (approximately 2 mL in small children and 3 mL in older children) with the thumb controlling the syringe plunger and, thus, balloon size. The catheter is slowly withdrawn until resistance is felt, and then the object is removed. The Katz Extractor oto-rhino FB remover (InHealth Technologies, CA) (Fig. 65-50) is a disposable, single unit, composed of a syringe with attached flexible catheter with balloon tip. The procedure for its use is similar to that of a Fogarty catheter. Complications mentioned in the literature are limited to occasional mild posttraumatic bleeding only, but the theoretical risk of airway obstruction by the balloon or aspiration from further displacement of the object should be considered.

A newly reported yet controversial method is the nasal wash technique. [69] [70] In this method, a bulb syringe filled with approximately 7 mL of sterile normal saline is

placed in the opposite nostril from the one that contains the FB. The bulb is forcibly squeezed and the object is expelled through the

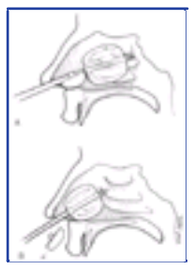

Figure 65-49 *A* and *B,* Fogarty balloon extraction of nasal foreign body. Insert the lubricated catheter tip above the foreign body, then gradually inflate the balloon. Slowly withdraw until resistance is met, then pull the object out of the naris.

nostril containing the object by the flow of saline. This technique may be difficult in uncooperative, younger patients. There is risk of injury to the nasal mucosa from the syringe tip and the flow of saline into the nasopharynx may cause coughing, choking, and discomfort. Other complications include reflux of saline and nasal contents into the eustachian tubes and FB aspiration. Because this technique has not been extensively studied, manual removal of the FB under direct visualization is still the preferred method.

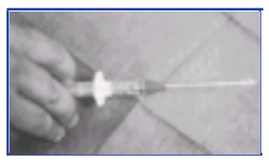

Figure 65-50 The Katz Extractor (InHealth Technologies, Carpinteria, CA) for oto-rhino foreign body removal shown with balloon inflated.

Another approach to the posteriorly placed nasal FB is blowing the object out with *positive air pressure*. The simplest way is to ask the child to blow his or her nose while occluding the unaffected nostril. This is really only effective in the older child. Alternatively, a bag-valve mask [71] can be placed over the child's *mouth* to provide the positive pressure. The opposite nostril should be occluded and the Sellick maneuver applied to prevent air passage into the esophagus. This technique often requires restraint and can also be threatening to a young child. Another technique [72] [73] requiring no instrumentation is performed by having the child lie supine and occluding the unaffected nostril (Fig. 65-51). The patient then exhales air until resistance (from the patient's epiglottis shutting) is felt. Next, as in mouth-to-mouth resuscitation, air is briskly blown into the mouth which produces an outward pressure behind the object. This either moves it within grasping reach or pops it completely out of the naris, usually onto the clinician's cheek. Once again, this can be a frightening experience for the child, it requires restraint, and there is a theoretical risk of disease transmission. An effective compromise is using "a parent's kiss." In this technique, the child is allowed to sit or stand and no restraint is required. The parent is asked to make a firm seal with his or her mouth over the child's open mouth and then to give a short, sharp puff of air into the child's mouth. The opposite nostril is occluded throughout the procedure. To gain the child's cooperation, tell them that the parent is going to "give them a big kiss." If it fails, the technique can be repeated. A study of the "parent's kiss" by Botma et al. [74] found a success rate of 79%, and all parents thought the technique was acceptable and preferable

Figure 65-51 Bag-valve-mask technique to blow foreign body out of the naris. Ensure that the face mask forms a tight seal around the patient's mouth and that the unaffected nostril is completely occluded. Attempt to firmly compress the bag as the patient exhales (an assistant is helpful to hold the mask snugly and to occlude the other nostril). This technique works best with objects that completely occlude the nostril.

to instrumentation or restraint. Although theoretical complications include possible barotrauma to the tympanic membrane or other complications seen with positive-pressure ventilation (pneumothorax, mediastinal emphysema), no reported complications have been published in regard to these three positive-pressure techniques.

CONCLUSION

A number of procedures are relevant to the examination of the ears, nose, and throat. These procedures generally require a calm and cooperative patient for success and minimization of complication risk. When patients are uncooperative or their anxiety cannot be addressed by mild sedation, specialist referral and examination under general anesthesia should be considered.

Acknowledgment

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Chapter 66 - Emergency Dental Procedures

Kip Benko

Complaints pertaining to the teeth and supporting maxillofacial structures are common, and patients frequently present to the emergency department (ED) for initial evaluation. Complaints may range in scope from a simple chipped tooth to a severe odontogenic deep space infection or maxillofacial injury. Treating these patients can be challenging and frustrating for the busy emergency clinician. Many emergency clinicians do not receive specific training in dental emergencies during their residencies, yet it is important for them to be able to recognize and treat a wide range of dental problems. Some dental emergencies can lead to life-threatening airway compromise while others lead to morbidities such as tooth loss, pain, infection, and craniofacial abnormality.

Management of specific dental emergencies requires a thorough understanding of adult and pediatric dentition. The relevant anatomy of both populations will be outlined. The techniques described for management of the various traumatic and infectious problems will, in most cases, be temporizing until definitive dental or oral/maxillofacial surgery referral can be obtained. Those conditions requiring emergent consultation will be discussed. Topical, local, and regional anesthesia are of particular importance and utility in the management of odontogenic emergencies and the clinician should be very familiar with these techniques (see Chapter 30 and Chapter 31). Trauma to the facial bones and other facial structures is covered in another chapter.

Although this chapter describes the diagnosis and treatment of dental injuries that may confront the emergency clinician, there is no standard of care mandating that dental problems be definitively handled in the ED setting. It is appropriate to refer all significant dental pathology to a dentist or oral surgeon, and failure to stabilize teeth or provide invasive drainage procedures in the ED should not be considered substandard care.

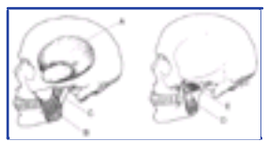

Figure 66-1 Muscles responsible for closing and excursive mandibular movements. Sagittal skull views illustrating the anatomical positions of the following muscles: *A,* Temporalis. *B,* Superficial masseter. *C,* Deep masseter. *D,* Medial pterygoid. *E,* Lateral pterygoid. *(From King R: Oral-Facial Emergencies-Diagnosis and Management, 1st ed. Portland, OR, JBK Publishing, 1994.)*

ANATOMICAL REVIEW

Stomatognathic System

The muscles of mastication are responsible for opening and closing the mouth and are those most frequently associated with temporomandibular disorders (TMDs). The clinician must be knowledgeable about the position of the muscles in order to perform an examination properly and to recognize the origin of certain painful conditions. The muscles that close the mouth are those most often associated with TMDs and include the masseters, the temporalis, and the medial pterygoids (Fig. 66-1). The contraction of this group of muscles bilaterally serves to move the condyle superiorly and posteriorly, causing the mouth to close. The opening muscles include the anterior digastric, posterior digastric, mylohyoid, geniohyoid, and infrahyoid muscles (Fig. 66-2). They are not often affected in TMDs. The lateral pterygoids are responsible for anterior translation and lateral movement of the mandible (Fig. 66-3). Unilateral contraction causes lateral movement away from the side of the muscle contraction while bilateral contraction causes protrusion of the mandible.

The mandible consists bilaterally of the horizontal body and the ascending ramus, which are connected by the angle. The bodies of the mandible are connected by the symphysis in the midline. The ascending ramus gives rise superiorly to two processes, the condylar process and the coronoid process (Fig. 66-4). The mandibular condyle, along with the mandibular fossa and the articular eminence of the temporal bone, make up the temporomandibular joint (TMJ). The TMJ provides for both hinge and gliding actions. Between the mandibular condyle and the articular eminence is a fibrous collagen disk, also known as the meniscus. A ligamentous joint capsule surrounds the TMJ and serves to limit condylar movement. TMJ pain may be caused by a number of conditions, both traumatic and nontraumatic. The main distinction to be made is whether the pathology is intracapsular or extracapsular. This is often difficult to determine and the focus in the emergent setting is to rule out fracture as a cause of pain.

Teeth

The adult dentition normally consists of 32 teeth, which comprise four types: 8 incisors, 4 canines, 8 premolars, and 12 molars. From the midline to the back of the mouth there is a central incisor, a lateral incisor, a canine, two premolars (bicuspids), and three molars, the last of which is the wisdom

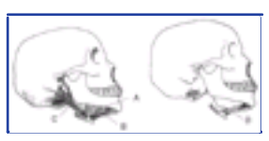

Figure 66-2 Muscles responsible for mandibular opening. Oblique skull views illustrating the anatomical positions of the following muscles: *A,* Mylohyoid. *B,* Anterior belly of the digastric. *C,* Posterior belly of the digastric. *D,* Geniohyoid. *(From King R: Oral-Facial Emergencies-Diagnosis and Management, 1st ed, Porland, Or: JBK Publishing, 1994.)*

tooth (Fig. 66-5). The 20 primary or deciduous (baby) teeth consist of eight incisors, four canines, and eight molars. From the midline to the back of the mouth there is a central incisor, a lateral incisor, a canine, and two molars (Fig. 66-6 (Figure Not Available)). Agenesis, or the lack of proper formation of a tooth or teeth, is not uncommon, especially in the maxilla. Likewise, supernumerary, or extra, teeth also occur. The adult teeth are numbered from 1 to 32, with the first tooth being the right upper third molar and the 16th tooth being the left upper third molar. The left lower third molar is 17th, and the 32nd tooth is the right lower third molar (see Fig. 66-6 (Figure Not Available)). There are numerous classification and numbering systems of the teeth; however, it is probably best for clinicians to simply describe the location and type of tooth in question (e.g., upper left second premolar, lower right canine). This removes any question when you are discussing a case with a consultant.

A tooth consists of the central pulp, the dentin, and the enamel (Fig. 66-7). The pulp contains the neurovascular supply of the tooth, which is responsible for carrying nutrients to the dentin, a microporous substance that consists of a system of microtubules. The dentin makes up the majority of the tooth and cushions the tooth during mastication. The enamel is the white, visible portion of the tooth and is the hardest part of the body. The tooth may also be described in

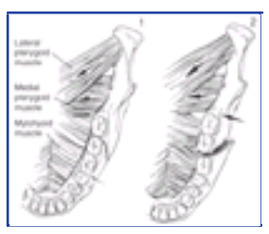

Figure 66-3 Axial view of the floor of the mandible. The arrows indicate the direction of pull of the lateral pterygoid, medial pterygoid, and mylohyoid muscles. *(From Eisele D and McQuone S: Emergencies of the Head and Neck, 1st ed. St. Louis, MO, Mosby, 2000.)*

terms of the crown (coronal portion) or the root. The crown is that portion covered in enamel, and the root is the part which serves to anchor the tooth in the alveolar bone.

The following descriptive terminology is used for the different anatomic surfaces of the tooth. These terms are useful when describing the specific tooth injury to a consultant or colleague:

- *Facial:* that part of the tooth that faces the mouth. This is the part that you see when somebody smiles. This is a general term applicable to all teeth.
- *Labial:* refers to the facial surface of the incisors and canines.
- *Buccal:* refers to the facial surface of the premolars and molars.
- *Oral:* that part of the tooth that faces the tongue or the palate. This is a general term applicable to all teeth.
- *Lingual:* toward the tongue, the oral surface of the mandibular teeth.
- *Palatal:* toward the palate, the oral surface of the maxillary teeth.
- *Approximal/Interproximal:* the contacting surfaces between two adjacent teeth.
- *Mesial:* the interproximal surface facing anteriorly or closest to the midline.
- *Distal:* the interproximal surface facing posteriorly or away from the midline.
- *Occlusal:* biting or chewing surface of the premolars and molars.
- *Incisal:* biting or chewing surface of the incisors and canines.
- *Apical:* toward the tip of the root of the tooth.
- *Coronal:* toward the crown or the biting surface of the tooth.

The Periodontium

The periodontium, also known as the attachment apparatus, consists of two major subunits and is necessary for maintaining the integrity of the normal dentoalveolar unit.

The *gingival* subunit consists of the junctional epithelium and the gingival tissue. The gingival tissue is keratinized, stratified, squamous epithelium and it can be divided into the *free gingival margin* and the *attached gingiva*. The free gingiva is the cuff of tissue that is formed around the neck of the tooth. The gingival sulcus is that space between the free gingiva and the tooth. It is rarely greater than 2 to 3 mm in depth in normal healthy dentition. The attached gingiva is the portion of gingiva that is attached to the alveolar bone and extends apically (away from the tooth) to the mucogingival junction (or the mucobuccal fold). At this point the tissue, loose

and nonkeratinized, is called the alveolar mucosa (or buccal mucosa).

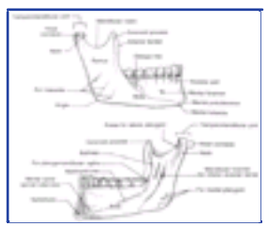

Figure 66-4 Anatomy of the mandible. *Top,* View from lateral (buccal) perspective. *Bottom,* View from medial (lingual) perspective. *(Redrawn from Grant JC: Grant's Atlas of Anatomy, 5th ed. Baltimore, Williams & Wilkins, 1962.)*

The *periodontal subunit* includes the periodontal ligament, alveolar bone, and the cementum of the root of the tooth. The periodontal ligament consists of collagen that extends from the alveolar bone to the root of the tooth. One end of the periodontal ligament inserts into the alveolar bone, the other end into the cementum (see Fig. 66-7).

The gingival subunit is primarily responsible for maintaining the integrity of the periodontal subunit. Certain disease states such as gingivitis weaken the attachment apparatus and can result in tooth loss.

DENTOALVEOLAR TRAUMA

Dental Fractures

Dentoalveolar trauma is a common reason for ED visits. Injury to the maxillary central incisors accounts for between 70–80% of all fractured teeth. [5] [6] [16] Trauma to the teeth is usually not life-threatening; however, the morbidity associated with dental fractures can be significant and includes the following: failure to complete eruption, color change of the tooth, abscess, loss of space in the dental arch, ankylosis, abnormal exfoliation, and root resorption. Dental injuries are often associated with intraoral lacerations. When a tooth is chipped or missing, and there is a concomitant intraoral laceration, it should be noted that the missing portion of the tooth may be imbedded in the depths of the laceration.

Some general principles apply to the evaluation and management of dental trauma. First, identify all fracture fragments and mobile teeth. Each tooth surface should be percussed for mobility and sensitivity. If a tooth is missing, it cannot always be assumed that it has been avulsed. Teeth are aspirated into the respiratory tract, or they can be fully intruded into the maxillary sinus, alveolar bone, or nasal cavity. Radiographs should be taken if there is any suspicion of aspiration of tooth fragments or intrusion of fragments into the gingiva or alveolar bone. Second, the dentition is much more easily manipulated if the patient is not in significant discomfort. Tooth infiltration and common dental blocks should be part of the emergency clinician's armamentarium. Third, topical tooth remedies and analgesics, both over-the-counter and prescribed, should be discouraged as their use can lead to the development of sterile abscesses and soft tissue irritation. Fourth, tetanus vaccination should be administered if needed.

The management of fractured teeth depends upon the extent of fracture with regard to the pulp, the degree of development of the apex of the tooth, and the age of the patient. There are many ways to classify dentoalveolar injuries and, in particular, tooth fractures. [7] The Ellis classification is one system that is often cited in the emergency medicine literature; however, many dentists and maxillofacial surgeons do not use this nomenclature, making it less than ideal when discussing these types of injuries (Fig. 66-8).[6] The most easily understood method of classification is, perhaps, one based on injury description.

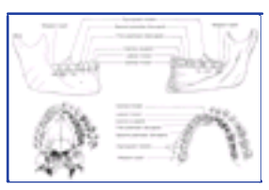

Figure 66-5 Classification of teeth. *Top left,* Lateral view. *Top right,* Medial view. *(Redrawn from Grant J, Basmajian J: Grant's Method of Anatomy, 7th ed. Baltimore, Williams & Wilkins, 1965.)*

Crown fractures may be divided into uncomplicated and complicated categories. Uncomplicated crown fractures result from injuries to the enamel alone or to a combination of the enamel and the dentin.

Ellis class I fractures.

Uncomplicated crown fractures through the enamel only are called Ellis class I fractures. They are usually not sensitive to either temperature or forced air. These fractures usually pose no threat to the health of the dental pulp. They may feel sharp to the patient's tongue, lips, or buccal mucosa. Immediate treatment is not necessary but may consist of smoothing the sharp edge of the tooth with an emery board or rotary disk sander. The patient should be reassured that the tooth can be restored by a dentist to its normal appearance with composite resins and bonding materials. Follow-up is important with these injuries as pulp necrosis and color change can rarely occur (in less than 1%) (Fig. 66-9*A, B*). [2] [6]

Ellis class II fractures.

Uncomplicated fractures through the enamel and dentin are called Ellis class II fractures. Fractures that extend into the dentin are at higher risk of pulp necrosis and, therefore, need more aggressive treatment by the emergency clinician (Fig. 66-10). The risk of pulp necrosis in these patients is 1–7%, but this increases as treatment time extends beyond 24 hours.[6] These patients often complain of sensitivity to heat, cold, or forced air. The physical exam reveals the yellow tint of the dentin in contrast to the white hue of the enamel. Fractures that are closer to the pulp cavity will reveal a pink tinge to the dentin. The tooth is usually sensitive to percussion with a tongue blade. The porous nature of the dentin allows passage of bacteria from the oral cavity to the pulp that may result in inflammation and infection of the pulp chamber. This is more likely to occur after 24 hours of dentin exposure but occurs sooner if the fracture site is closer to the pulp. Likewise, patients younger than 12 years have a pulp/dentin ratio larger than in the mature adult and are at increased risk for pulp contamination. For this reason, younger patients should be treated aggressively and should be seen by their dentist within 24 hours. [1] [11]

The goal of treating dentin fractures is twofold: to cover the exposed dentin, thus preventing secondary contamination or infection, and to provide pain relief. After the tooth is covered, the dentist can rebuild it with modern composites in many cases. A supraperiosteal infiltration or a regional tooth block performed prior to any tooth manipulation allows for easier application of the dressing since the procedure then becomes relatively painless.

Dressings that may be applied to the surface of the tooth include calcium hydroxide, zinc oxide, and glass ionomer composites. Recent literature suggests that glass ionomers may be superior to other coverings; however, the difference is probably slight and the increased cost of the glass ionomers is not justified for routine ED use at this time.[4] [5] Certain composites may be cured with a bonding light. This is routinely done in the dentist's office but is beyond the scope of most emergency practice. Bone wax and skin glues such as the cyanoacrylates are not recommended as dressings. Most dressings come as a base and a catalyst, which require mixing. This is easily accomplished with a dental spatula and a mixing pad, which are obtainable from any dental supply house. A commonly used ED dressing is Dycal (calcium

Figure 66-6 (Figure Not Available) Identification of teeth. *(From Beaudreau R: Oral and Dental Emergencies. In Tintinalli J, Kelen G, Stapczynki S: Emergency Medicine-A Comprehensive Study Guide, 5th ed. New York, McGraw-Hill, 2000.)*

hydroxide). The catalyst and the base are mixed in equal portions, and a small amount is placed on the exposed area with an applicator such as a dental spatula or another appropriate instrument (Fig. 66-11).

The tooth surface should be dry prior to application to assure adherence of the CaOH. This can be accomplished by having the patient bite into gauze pads. Dycal will dry within minutes after being exposed to the moist environment of the mouth. Although placing dental foil over the CaOH dressing is recommended, it is not usually necessary if the patient plans to follow up with a dentist within 24 to 36 hours. To prevent dislodging the dressing, the patient should be

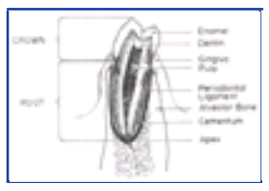

Figure 66-7 The dental anatomic unit.

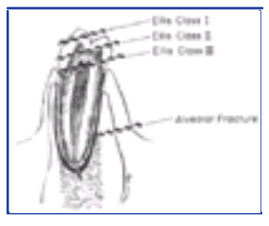

Figure 66-8 The Ellis classification for fractured alveolar teeth. The easiest method to classify fractured teeth is by description (e.g., fracture through the dentin of the first upper right molar).

instructed to eat soft foods only until seen by the dentist. Although unproven, it is reasonable to begin antibiotic treatment with penicillin or clindamycin until definitive dental treatment can be obtained.[10] [11]

Many patients who sustain a fracture through the dentin will require a root canal or other definitive endodontic treatment. The timely application of an appropriate dressing in the ED, however, may prevent contamination of the pulp and make root canal therapy unnecessary. As with any trauma to the anterior teeth, it is advisable to explain to the patient that disruption of the neurovascular supply is possible and that long-term complications such as pulp necrosis, color change, and resorption of the root may occur.

Ellis class III fractures.

Complicated fractures of the crown involving the pulp are called Ellis class III fractures. Complicated fractures of the crown that extend into the pulp of the tooth are true dental emergencies. These fractures result in pulp necrosis in 10% to 30% of cases even with appropriate treatment.[6] They may be distinguished from fractures of the dentin by the pink color of the pulp. The fractured surface of the tooth should be wiped off with gauze and observed for frank bleeding or a pink blush, which indicates exposure of the pulp. Fractures through the pulp are often excruciatingly painful but occasionally there is a lack of sensitivity secondary to a disruption of the neurovascular supply of the tooth.

Immediate management includes referral to a dentist, oral surgeon, or endodontist. The patient often requires a pulpectomy (complete removal of the pulp) or, in the case of primary teeth, a pulpotomy (partial removal of the pulp) as definitive treatment.[2] [5] The longer the pulp is exposed, the greater the likelihood of contamination and abscess formation. If a dentist cannot see the patient immediately, the emergency clinician should attempt to relieve the pain and cover the exposed pulp. If significant pain is present a dental block should be performed. Subsequently, the tooth should be covered with one of the dressings described earlier (Fig. 66-12). Sometimes bleeding is brisk and needs to be controlled before applying a dressing. This can usually be accomplished by having the patient bite into a gauze pad that has been soaked with a topical anesthetic containing a vasoconstrictor such as epinephrine. Alternatively, a small amount of the anesthetic/vasoconstrictor may be injected into the pulp to control bleeding. After the covering is applied, the patient is instructed to follow up as soon as possible with a dentist. Antibiotics with coverage directed at oral flora

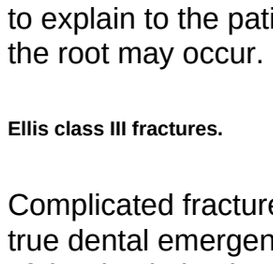

Figure 66-9 *A,* A common injury from a fist to the mouth results in a chipped tooth (here, fractured through the enamel only) and a laceration of the lip. One should always explore the mucosal laceration, looking for the missing piece of tooth. *B,* When this lip laceration was explored, a piece of tooth was found embedded within the laceration. If this foreign body is not removed, an infection is certain a few days later. *C,* Smoothing the enamel with a disc sander or an emory board will ameliorate the cosmetic deformity of a chipped tooth.

(e.g., penicillin, clindamycin) should be prescribed and only soft foods should be eaten. Removal of the pulp with specialized instruments by the emergency clinician is not recommended, although some authors have advocated this in

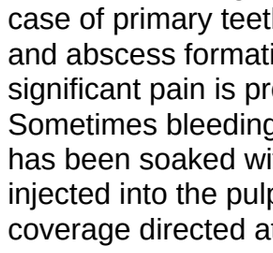

Figure 66-10 A fracture through the dentin.

the past. This procedure is the realm of the dental professional and is likely to result in complications if not done properly.

Luxation, Subluxation, Intrusion, and Avulsion

Luxation and subluxation.

Subluxation refers to teeth that are mobile but not displaced. Luxation refers to teeth that are displaced, either partially or completely, from their sockets. Luxation injuries are divided into four types:

1. Extrusive luxation is an injury in which the tooth is forced partially out of the socket in an axial direction (Fig. 66-13).
2. Intrusive luxation, or intrusion, occurs when the tooth is forced apically and may be accompanied by crushing or fracture of the tooth apex (Fig. 66-14 and Fig. 66-15).
3. Lateral luxation occurs when the tooth is displaced either facially, lingually, mesially, or distally (Fig. 66-16). This injury is often associated with injuries to the alveolar wall.
4. Complete luxation, also known as complete avulsion, results in loss of the entire tooth from the socket.

Even minor trauma to the oral cavity requires meticulous examination for loose or missing teeth. Each tooth surface should be examined for mobility by using a

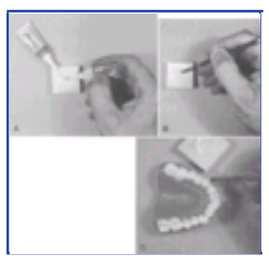

Figure 66-11 *A,* Calcium hydroxide paste is one acceptable material to cover dentin or pulp fractures. *B,* Mixing the calcium hydroxide paste with a spatula on a mixing pad. *C,* Application of the periodontal paste to the fractured surface of the tooth.

back and forth motion on each side of the tooth surface with the fingertips or two tongue blades. Any blood in the gingival crevice (area where the gingiva touches the tooth) is suggestive of a traumatized tooth.

Teeth that are minimally mobile and are not displaced do very well with conservative treatment only. The tooth will tighten up in the socket if not retraumatized. Patients should be instructed to eat only a soft diet for 1 to 2 weeks and follow up with their dentist as soon as possible.

Grossly mobile teeth require some form of stabilization as soon as possible. It is important to note that in certain patients with poor gingival health, luxated teeth may not be

Figure 66-12 *A,* Covering the pulp fracture with calcium hydroxide paste. *B,* The calcium hydroxide paste hardens quickly in the moist environment of the mouth.

salvageable due to disease of the attachment apparatus. Fixation is best performed by the dental specialist with enamel bonding materials or wire ligation (Fig. 66-17). Although many different "home remedies" exist for splinting loosened teeth in the ED, one must be aware of the concern for aspiration of teeth if the splint fails. The clinician should also avoid the use of nonapproved medications in the mouth. An example is the use of skin adhesives, which, to date, have not been approved for intraoral use.

Splinting techniques are suitable for the emergency clinician to perform as temporizing measures until definitive care can be arranged. One simple technique for emergency

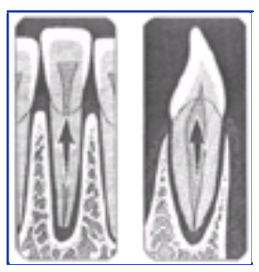

Figure 66-13 Extrusive luxation occurs when the tooth is forced partially out of the socket in an axial direction. *(From King R: Oral-Facial Emergencies-Diagnosis and Management, 1st ed. Portland, OR, JBK Publishing, 1994.)*

use is to apply periodontal paste commercially available as Coe-pak. Coe-pak consists of a base and a catalyst that, when mixed, form a moderately sticky claylike dressing. This dressing becomes firm after application. It is applied over the enamel and gingival, as well as the adjacent teeth, to splint the subluxated tooth into place. Although the splint performs best if placed on the facial and buccal surfaces of the teeth, it is usually sufficient to apply the paste only to the front (facial) surface of the teeth (Fig. 66-18 and Fig. 66-19). The dressing is most easily applied when the clinician's gloves are lubricated with water or lubricating jelly and the gingiva and enamel are completely dry. It is important to apply the dressing into the grooves between the teeth as well as to the adjacent teeth. The

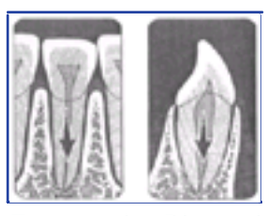

Figure 66-14 Intrusive luxation of a tooth compresses the periodontal ligament and vascular supply of the pulp. It may even crush the apical bone. *(From King R: Oral-Facial Emergencies-Diagnosis and Management, 1st ed. Portland, OR, JBK Publishing, 1994.)*

patient is reminded to eat a soft diet until seen in follow up within 24 hours.

Self-cure composite is another splinting option in the ED. Self-cure composite is a compound that does not need a curing light to harden. It is formed by mixing a base and a catalyst, which harden when combined. This product is only applied to the enamel of the involved tooth, not the gingiva, and to the nonmobile adjacent teeth (Fig. 66-20). Both Coe-pak and self-cure composite are fairly simple for the dentist to remove during formal restoration.

Teeth that are luxated in either the horizontal or axial plane or are slightly extruded can also be splinted with the techniques described earlier. It is important that the loosened tooth be in perfect alignment when the final adjustments are made at the dentist's office; however, the alignment does not need to be precise when the tooth is splinted in the ED. The important point is that the tooth is splinted adequately and follow up is ensured.

Intrusion and avulsion.

Intruded teeth are those that have been forced apically into the alveolar bone. This often results in disruption of the attachment apparatus or fracture of the supporting alveolar bone, especially in permanent teeth with mature roots. [1] [3] These teeth are usually immobile and do not require stabilization in the ED. Intruded teeth often require endodontic treatment because of pulp necrosis. It is important to consider the possibility of an intruded tooth anytime there is a space in the dentition. Undiagnosed intrusion of the teeth can lead to infection and craniofacial abnormalities. X-rays should be obtained anytime there is uncertainty as to whether a tooth is intruded or simply avulsed. Intruded teeth are best managed by the dentist or dental specialist and referral should take place within 24 hours. Permanent teeth often require repositioning and immobilization, but primary teeth are usually given a trial period to erupt on their own before any intervention is taken.

Avulsed teeth are those that have been completely removed from their ligamentous attachments and are true dental emergencies. The first consideration in treating dental avulsions is to ask, "Where is the tooth?" Missing teeth may have been intruded, fractured, aspirated, swallowed, or embedded into the soft tissues of the oral mucosa; therefore, x-rays should be considered anytime an avulsed tooth cannot be located. Management of the avulsed tooth in the ED is dependent upon a number of factors. These include the age of the patient, the amount of time that has elapsed since the tooth was avulsed, associated trauma to the oral cavity such as alveolar ridge fractures and the overall health of the periodontium. [3] Primary teeth are not replaced after avulsion because they can fuse to the alveolar bone and potentially cause craniofacial abnormalities or infection. Reimplanted primary teeth may also interfere with the eruption of the secondary teeth. The parents of these patients need to be reassured that a prosthetic replacement for the avulsed teeth can easily be made and worn until the permanent teeth erupt.

Time is the other important consideration when deciding whether to replace an avulsed tooth. In general, the longer the tooth is out of the socket, the higher the incidence of periodontal ligament necrosis and subsequent failure of reimplantation. The periodontal ligament cells generally die within 60 minutes outside of the oral cavity if they are not placed in appropriate transport media. [13] A significant amount of research has been conducted on different media used to keep the cells of the periodontal ligament alive.

Figure 66-15 Intruded tooth secondary to trauma. On superficial examination it appears that the tooth was simply knocked out. A dental radiograph or CT scan is necessary to determine intrusion or avulsion. Intruded teeth create the potential for infection or cosmetic deformities. Intrusion of an upper tooth into the maxillary sinus can cause recurrent sinusitis. Teeth can also intrude into the nasal cavity and cause infection or bleeding, or they can be aspirated into the airway. The incisors are the most commonly intruded teeth. *(From Johnson R: The treatment of the traumatized incisor in the child patient. In University of Pennsylvania School of Dental Medicine: Continuing Dental Education, vol 2. Philadelphia, University of Pennsylvania, 1978.)*

Various transport media that have been studied include milk, Hank's Balanced Salt Solution, Save-A-Tooth, saliva, cell culture media, and water. Although certain cell culture media have been developed to stimulate the periodontal ligament cells to proliferate and remain viable, milk and the commercially available Save-A-Tooth are best for both prehospital care and ED storage (Fig. 66-21).[13] [15] Both will preserve the periodontal ligament for up to 8 to 12 hours; however, reimplantation should take place at the earliest possible opportunity. The key is to get the tooth into the transport media immediately because even 10 minutes outside of some type of storage media can cause desiccation and death of the periodontal ligament cells. Saliva should be used at the scene if milk or Save-A-Tooth is not available. The tooth should be reimplanted by the patient in the prehospital setting, if possible. The following principles should be

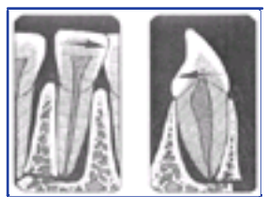

Figure 66-16 Lateral luxation occurs when the tooth is displaced in either a lingual, mesial, distal, or facial direction. Fractures of the alveolus frequently accompany lateral luxation injuries. *(From King R: Oral-Facial Emergencies-Diagnosis and Management, 1st ed. Portland, OR, JBK Publishing, 1994.)*

followed when providing instructions to prehospital providers or to a patient who calls for advice:

- The tooth should be handled by the crown only. Handling the tooth by the root can damage the alveolar ligament.
- The tooth should not be replaced if it is fractured or if there is significant maxillofacial trauma such as an alveolar ridge fracture.
- If the tooth can be replaced in the prehospital setting, the root should be gently rinsed off first to remove any debris. The root should not be wiped off as this removes the periodontal ligament.
- If the tooth cannot be reimplanted successfully in the field, it should be placed in a transport media as described earlier. Transporting the tooth in the oral cavity such as a cheek is risking aspiration. This location is also not ideal for keeping the periodontal ligament alive because of the bacterial flora and low osmolality of the saliva.
- Once the patient arrives in the ED, the treating clinician should confirm proper placement and alignment. It is not important that the tooth is in perfect position, as final adjustments can be made by the dentist. Splinting of the repositioned tooth with periodontal paste or composite as outlined above may be necessary if mobility is present.

Should reimplantation not be successful in the prehospital setting, it must be done in the ED using the following guidelines:

- The tooth should be stored in appropriate media if reimplantation is delayed for any reason.
- It is extremely helpful to perform a supraperiosteal dental infiltration before the manipulation or replacement of teeth. This makes the procedure more comfortable for the patient and easier for the clinician to perform.
- The oral cavity should be checked for trauma. If an alveolar ridge fracture is present or the socket is significantly damaged, the tooth should not be reimplanted.
- The socket first must be suctioned with a Frasier suction tip to remove the accumulated clot. Be careful not to damage the walls of the socket as this can further damage periodontal ligament fibers. Gentle irrigation should follow suctioning. If the clot is not removed, reimplantation and

realignment will be difficult. Any debris on the tooth should be rinsed, not scrubbed, with saline. The tooth is then implanted into the socket using firm, but gentle, pressure. Remember to handle the tooth only by the crown.
- Having the patient gently bite down on gauze may help to align the tooth. The tooth may require splinting after replacement. If significant mobility is present such that temporizing splints are not adequate, wiring or arch bars may need to be placed by the dental consultant.
- Tetanus should be updated as necessary. A liquid diet should be prescribed until seen in follow up.

Antibiotics are controversial in the treatment of fractured and avulsed teeth. Although the American Association of Endodontics does not recommend the routine use of antibiotics for fractures or avulsions, other authors recommend the use of antibiotics that cover mouth flora (e.g., penicillin, clindamycin) to decrease the inflammatory resorption of the root.[6] [18] It is probably prudent to use antibiotics if the root or socket are heavily soiled; otherwise, treatment should be tailored to the individual patient and discussed with the consultant.

Prognosis.

The prognosis of a reimplanted tooth depends on many things. As discussed earlier, the time to

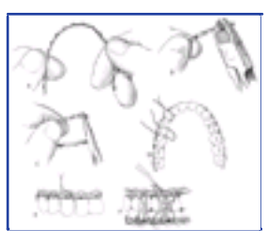

Figure 66-17 Application of Erich arch bars. *A through C,* A commercially available arch bar is shaped to fit the maxillary and mandibular arches. *D and E,* Stainless steel wire secures the bar to the necks of the teeth. *F,* The maxilla and the mandible are brought into occlusion and held in place with rubber bands. This technique is generally not used by the emergency clinician, but this equipment should be available for the oral or maxillofacial surgeon. *(From Converse JM: Reconstructive Plastic Surgery, 2nd ed, vol 2. Philadelphia, WB Saunders, 1977.)*

reimplantation is critical. Likewise, the age of the patient, the stage of development of the root (younger is better), and the overall health of the gingiva are also very important. An individual with gingival disease is more likely to have an unsuccessful reimplantation.

The goal in any tooth avulsion or fracture is to keep the native tooth if at all possible. A tooth that has been avulsed and reimplanted usually loses the majority of its neurovascular supply and undergoes pulp necrosis. However, if the periodontal ligament remains intact, there is a greater chance of a functional tooth. It is important that the patient be aware that some root resorption is always going to occur after reimplantation, and loss of the tooth may occur.

Alveolar Bone Fractures

Trauma involving the anterior teeth may be associated with fractures of the alveolus, which is the tooth-bearing portion of the maxilla or mandible. Alveolus or alveolar ridge fractures often occur in multi-tooth segments and will vary in the number of teeth involved, the amount of displacement, and the mobility of the affected segment. The patient usually complains of pain as well as malocclusion. The diagnosis is usually clinically


Figure 66-18 *A,* Squeeze out equal-sized ribbons of the periodontal paste. *B,* Mix the base and catalyst together with a tongue blade. *C,* Using moistened gloves, apply the paste to the dry enamel and gingiva.

apparent and is notable for a section of teeth that are misaligned and variable in mobility. Avulsed teeth, fractured teeth or displaced teeth may be present within the alveolar segment itself. Dental bite wing x-rays confirm the diagnosis. In the ED, Panorex or facial films may show the fracture line just apical to the root of the involved teeth; however, these films are often inconclusive or normal.

Treatment of alveolar ridge fractures involves rigid splinting after repositioning of the involved segment. This is usually beyond the scope of the emergency clinician, and urgent consultation with an oral surgeon or dentist is necessary. The role of the emergency clinician is to identify the injury as well as any avulsed or fractured teeth and preserve as much of the alveolar bone and surrounding mucosa as possible. Alveolar bone that is lost, debrided, or missing is difficult for the specialist to restore properly.[4]

Lacerations and Dentoalveolar Soft Tissue Trauma

Trauma to the face and perioral region is often associated with soft tissue injuries such as abrasions or lacerations. Before any repair can take place, it is imperative to do a thorough inspection of all wounds and abrasions to determine the extent of the wound and whether or not foreign bodies are present. Through-and-through lacerations are easily overlooked as are small foreign bodies and debris such as tooth fragments. Radiographs may be helpful if there is any question of tooth fragments. Potential airway compromise should also be identified at this time.

As a general rule, injured teeth should be repaired before any soft tissue management is undertaken because manipulation of the soft tissue, as would occur when repairing teeth, will likely cause further damage to sutures already placed in the soft mucosa. The repair of lacerations in the perioral region begins with standard wound care. After appropriate local or regional anesthesia, devitalized, crushed, or macerated tissue should be debrided. Profuse irrigation should follow. The role of antibiotics in mucosal trauma has not been definitively established, and there is no definitive standard of care promulgated. Several studies suggest a minimal benefit; however, this remains to be completely proven.[17] A reasonable guideline to follow is to use antibiotics if a significant amount of devitalized or crushed tissue is present or if the wound is through-and-through. Coverage of oral flora (e.g., penicillin, clindamycin) is fine for mouth lacerations and additional skin coverage should be considered for through-and-through lacerations (clindamycin, dicloxacillin). Dentoalveolar trauma may present the emergency clinician with several different situations that should generally be approached as follows.

Buccal mucosa.

Most lacerations and abrasions of the buccal mucosa heal quickly and rapidly without repair. If only the mucosa is lacerated, even seemingly large lacerations heal well and quickly. However, large lacerations (>1–2 cm) should generally be repaired. Many clinicians still prefer using chromic gut in the mouth, but any absorbable suture, such as vicryl in the 4-0 to 5-0 size, may be used. Absorbable sutures are preferred and they should be placed so that the knots are buried. Silk can be used as an alternative, but it has


Figure 66-19 Application of periodontal paste. *A,* Equal-length ribbons of base and catalyst are squeezed onto the mixing pad. *B,* The base and catalyst are mixed together using a moistened spatula or tongue blade. *C and D,* Using moistened gloves, roll the mixture into a ribbon. *E through H,* Apply to the enamel and gingiva, extending the paste two teeth on each side of the loosened tooth. The paste adheres best when the surface of the enamel and gingiva are as dry as possible.

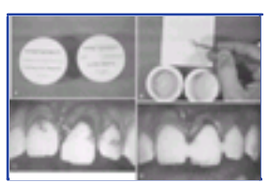
Figure 66-20 *A and B,* Equal amounts of the self-cure composite are placed on the mixing pad and mixed together. *C,* Subluxed teeth should be repositioned prior to application of the composite. *D,* The mixture is applied only to the dry enamel of the teeth. The composite should extend to one tooth on each side of the loosened tooth.

higher reactivity and is nonabsorbable. Nylon should be avoided because it is sharp and irritating to the tissues.

Through-and-through lacerations of the oral cavity present a special situation. The integrity of the salivary ducts (Wharton's duct and Stensen's duct) and the facial nerve needs to be established. If intact, repair can proceed. Guidelines

Figure 66-21 Using the "Save A Tooth" system, a tooth is placed into the container and closed. The preservative will increase the life span of traumatized periodontal ligament cells.

for closure are controversial, but generally, larger lacerations (>1–2 cm) should be closed. The mucosa should be closed as noted earlier and the skin closed aesthetically with 6-0 nylon, prolene, or a rapidly absorbable suture. The mucosa should be closed first so as not to disturb the skin repair. If the mucosal wound is small or is a puncture wound, it is reasonable to close only the skin layer. Very large, gaping, or complicated lacerations should be referred to an oral surgeon.

Large lacerations or through-and-through lacerations of the oral cavity should be rechecked in 2 to 3 days. Sutures should be removed (if nonabsorbable) in 7 to 10 days. Saline rinses 4 to 6 times a day and a soft diet may be prescribed. A topical skin antibiotic may be applied for 24 to 48 hours and intraoral antibiotic ointment (Ora-5) (Fig. 66-22) can be considered, as well, for the first several days.

Gingiva.

Small lacerations of the hard gingiva overlying the maxillary or mandibular alveolus usually heal uneventfully without repair. If the laceration is large, if there is a flap present, or bone is exposed, the gingiva should be approximated with a 4-0 or 5-0 vicryl or dexon suture. As earlier, silk is an option. Difficulty sometimes arises when suturing gingiva as there is little supporting soft tissue underneath. A helpful technique is to circumferentially wrap the suture around the teeth and use the teeth as anchors (Fig. 66-23 and Fig. 66-24). Suture and wound care is as described earlier.

Frenulum.

The maxillary frenulum rarely requires sutures for simple lacerations. If the laceration is extensive

Figure 66-22 The Ora 5 Topical Bactericidal Agent can be applied to intraoral lacerations or injuries to help prevent infection.

or extends significantly into the surrounding mucosa or gingiva, it should be approximated with a chromic vicryl or dexon suture. These wounds are often significantly painful and analgesia should be prescribed, even if the wound does not require suturing. The lingual frenulum is very vascular in nature and often will need a suture or two to control hemostasis. A local anesthetic with a vasoconstrictor aids in hemostasis while the wound is repaired.

The tongue.

Tongue lacerations are challenging (see Chapter 36). Although they may be tempting to suture, any

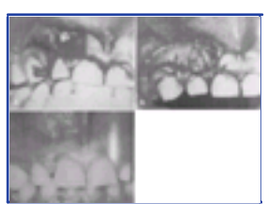

Figure 66-23 *A,* Gingival lacerations sometimes leave little tissue for approximation. *B,* The teeth can be used as anchors for sutures and help approximate the lacerated tissue. *C,* Gingival lacerations usually heal rapidly. (See also Fig. 66-24 .)

large lacerations of the body of the tongue, such as those that occur from a seizure, will heal well without suturing. Tongue lacerations that have the wound edges approximated do not need to be sutured, but larger lacerations that gape need repair because the cleft left by the wound will epithelialize leaving a grooved or bifid appearance. Likewise, wounds that are bleeding profusely, are flap-shaped, involve muscle, or are on the edge of the tongue should be approximated. Small avulsions or those in the center of the tongue usually heal without intervention.

Explaining the procedure in detail to the patient goes a long way in the repair of these wounds. The tongue usually can be secured by an assistant holding it with gauze. If the tongue cannot be secured in this manner, a towel clip applied to the end of the anesthetized tongue can be used. Children with tongue lacerations that need repair usually require sedation or repair by a specialist in the surgical suite, but many of these lacerations are small and heal uneventfully on their own.

Repair should be initiated with either a local infiltration of anesthetic or a lingual block. Lidocaine with epinephrine may be infiltrated locally to promote hemostasis. Repair should take place using absorbable sutures such as 4-0 chromic, vicryl or dexon. Silk can be used, but it must be removed in 7 to 10 days. Nylon is not used because it is very irritating to the surrounding tissues. Lacerations extending through muscle should be closed with one deep stitch penetrating both the mucosa and muscle. When possible, bury the knots of absorbable sutures as they often work their way loose. Full thickness lacerations can be closed in a number of ways. A suture that is placed through all three layers is acceptable as is the technique of closing the top mucosa and muscle together and, subsequently, doing the same thing on the underside of the tongue. Bleeding from large lacerations is almost always controlled with primary repair.

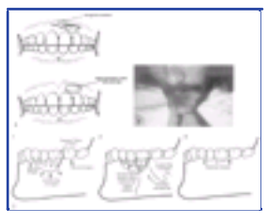

Figure 66-24 Repair of avulsed or lacerated gingiva. *A,* simple flap lap lacerations are approximated and closed with interrupted soft sutures, such as dexon or vicryl. *B,* Large gingival avulsions should be approximated to an anatomical position with interrupted sutures. *C1,* The exposed roots of the teeth should be covered. The thin and friable avulsed gingiva can not be sutured to the remaining gingiva or submucosal tissue. *C2,* The suture begins on the outer surface of the avulsed flap and is passed behind an anchoring tooth, like dental floss. The underside of the avulsed segment is then entered by the suture needle so the needle exits on the gingival surface. *C3,* Sutures pull the gingival to an anatomical position to cover the roots of the teeth and are tied on the outer surface. Sutures are removed in 5–7 days. See also Figure 66-23 .

In some instances hemostasis can be achieved without the use of sutures with the use of Gelfoam impregnated with topical thrombin (Fig. 66-29).

ORAL HEMORRHAGE

Bleeding from the oral cavity is not unusual and is most commonly associated with dental procedures. It is important to ascertain whether any recent dental work has been performed and of what nature it was. Spontaneous bleeding of the gingiva or oral cavity not associated with dental manipulation or trauma is suggestive of advanced periodontal disease or an underlying systemic process. It is also important to ask about other medical conditions that predispose to bleeding (liver disease, platelet abnormalities, etc.) as well historical factors, which may suggest a bleeding abnormality or clotting factor deficiency. It should be determined whether the patient is taking aspirin or other anticoagulants. Laboratory testing should be considered if there is significant suspicion for a pathologic coagulopathy, but not routinely in the patient presenting after dental manipulation.

Gingival bleeding after scaling or minor dental procedures is usually controllable with direct pressure and saline/hydrogen peroxide rinses. Bleeding that persists from the gingival areas despite pressure and rinses should raise suspicion for a bleeding abnormality. A much more common cause of oral hemorrhage that presents to the ED is postextraction bleeding. Minor oozing from dental extractions, such as wisdom teeth extraction, is normal for 2 to 4 days post surgery, but many patients get concerned when bleeding persists, despite warning from the oral surgeon. These patients usually present when their dentist cannot be contacted and after many hours of futile attempts at home to stop the bleeding. There are a number of options that the emergency

clinician can use to obtain hemostasis from postextraction bleeding.

Direct pressure.

Although the patient may have been using this technique at home, a few simple procedures may make it more effective. Any excessive clot that has built up around the oozing site should be removed with a suction catheter and the area gently irrigated. The clot that is in the socket (if any is present) should be left intact. Often, this clot is missing or partially missing. Once the clot is removed, gauze should be inserted as firmly as possible directly onto the bleeding site. This is best accomplished by using dental roll gauze (see section on dental materials later in this chapter) inserted directly over the bleeding site and then covered with 2 × 2 gauze. Dental roll gauze has the advantage of fitting more precisely between the teeth and, therefore, affords more pressure; however, 2 × 2s can be substituted. Most EDs have a topical vasoconstrictor such as epinephrine on the ENT cart for nosebleeds. It helps to moisten the roll gauze with topical vasoconstrictor before placement over the bleeding site. The patient should then be instructed to bite down and hold pressure for 15 minutes or so.

If active bleeding persists after 15 minutes, infiltration with a vasoconstrictor should be performed. Infiltrate the bleeding area and the gingiva surrounding the socket with lidocaine and epinephrine (1:100,000) until blanching occurs. Reapply the gauze over the site and instruct the patient to bite down for 15 more minutes. The injection serves two purposes: It causes vasoconstriction, and it anesthetizes the area so that adequate pressure can be generated during biting.

If bleeding persists, insert coagulating agents (Gelfoam, Surgicel, Avitene, Instat) into the socket and then loosely close the gingiva surrounding the socket with silk suture. Instruct the patient to bite down on gauze placed over the sutures. Soaking Gelfoam with topical thrombin is a good way to halt minor persistent bleeding (see Fig. 66-29).

If these measures fail to control the bleeding, consult a specialist. It is also reasonable to check blood counts and coagulation profiles at this time.

Patients whose bleeding is controlled can be discharged and instructed not to take anything by mouth for four hours and then only liquids and soft foods. Silk sutures require removal in seven days.

ALVEOLAR OSTEITIS (DRY SOCKET)

The pain associated with an extracted tooth is significant but usually manageable with current pharmacologic modalities. The pain associated with dry socket, however, can be very severe and often requires more definitive treatment. Alveolar osteitis, or dry socket, is a localized osteomyelitis that occurs when the alveolar bone becomes inflamed. This condition usually occurs when the clot that is normally present in the socket after a tooth extraction becomes dislodged or dissolves and is most common in the 2- to 4-day period after a tooth extraction. The exam is essentially unremarkable with the exception of a missing clot where the tooth was extracted. Anything that increases negative intraoral pressure in the mouth (e.g., smoking, excessive rinsing, spitting, drinking from a straw), as well as hormone replacement and periodontal disease, will predispose a patient to a dry socket. Only a small percentage of patients will develop a dry socket (2%–5%); however, this number increases with traumatic extractions or impacted third molars. [10] [14]

Figure 66-25 Dry Socket Paste. Apply alone or as a slurry mixed with Gelfoam into the socket to relieve alveolar osteitis pain.

The pain associated with a dry socket is extremely severe and if a patient presents several days after an extraction with a relatively normal exam and severe pain, it is likely a dry socket. The pain will not be relieved with traditional pain medications, but a dental block usually provides instant relief. Once the block is performed, the alveolar osteitis can be treated. The socket should be irrigated and gently suctioned to remove any accumulated debris. Next, the socket may be filled using a variety of techniques, which will prevent recurrence of pain and allow healing to begin.

The socket may be packed with ¼ in. gauze that is impregnated with eugenol (oil of cloves) or a local anesthetic. These can be obtained from the hospital pharmacy. The gauze tends to dry out and loosen and, therefore, should be replaced in 24 to 36 hours. Patients should be seen by a dentist the next day if at all possible. The socket may also be packed with a slurry of Gelfoam and eugenol. The Gelfoam acts as a matrix to hold the eugenol in the socket. A commercial product known as Dry Socket Paste (Fig. 66-25) can be applied by itself into the socket or it can be mixed with Gelfoam and placed into the socket. Dry Socket Paste is a very sticky thick paste containing eugenol. It often stays in place longer than gauze and does not dry out. (See section on dental materials later in this chapter.)

Although antibiotics may be given to prevent alveolar osteitis, they are not usually necessary once the socket has been packed and should be prescribed at the discretion of the patient's oral surgeon or dentist. [1] [4] [10]

DENTOALVEOLAR INFECTIONS

Infections of the oral cavity run the spectrum from minor, easily managed abscesses to severe, life-threatening, deep-space infections that require airway management and operative drainage. Although dental infections of all severity present to the ED, the most common are those related to pulp disease. Others are associated with the attachment structures of the teeth such as the gingiva, periodontal ligament, and the alveolar bone. These infections are often chronic conditions, but they can progress to the point where periodontal abscesses form and emergency treatment is required.

Emergency clinicians will be called on to drain abscesses of dental origin that do not extend into the deep spaces and that have well-defined boundaries that are easily accessible by intraoral or external drainage.

Disease of the pulp.

Disease of the pulp can occur from trauma, operations, or other unknown causes, but the most frequent cause is invasion of microorganisms after carious destruction of the enamel. As the enamel is destroyed, caries development progresses more rapidly through the dentin and into the pulp chamber causing an inflammatory response, referred to as pulpitis. If the path of carious destruction through the tooth is adequate for drainage of the developing inflammation, the patient may be asymptomatic for a long time. If drainage is blocked, however, the process progresses forward to rapidly involve the entire pulp cavity and the periapical space. The tooth is usually exquisitely tender at this point. Abscesses in the periapical region are usually picked up on dental x-rays and less commonly on a Panorex; however, unless extension through the cortex exists, it is not important for the emergency clinician to make the distinction between pulpitis and a periapical abscess. Examination often reveals gross decay of one or many teeth and percussion tenderness of the abscessed tooth. A periapical abscess will follow the path of least tissue resistance if not treated. This may be through the alveolar bone and gingiva and into the mouth or into the deep structures of the neck. If the infection has progressed apically through the alveolar bone and localized swelling and tenderness exists, incision and drainage should be performed (discussed subsequently).

In the ED setting, it is uncertain as to whether a periapical abscess or simple pulpitis exists. Dental x-rays are usually not available. In the absence of trauma or recent instrumentation, it is prudent to begin antibiotic coverage for the typical oral flora. Penicillin and clindamycin are good choices. Analgesia should be provided as well. A supraperiosteal infiltration (tooth block) using a long-acting anesthetic should be performed in most cases as this not only provides immediate and long lasting relief, but it also decreases the requirement for narcotic analgesics once the anesthetic effect has dissipated. A supraperiosteal injection should not be performed if the abscess has extended through the gingival tissue and is present near the injection site.

DISEASE OF THE PERIODONTIUM

Periodontal disease is also a very common disease and affects practically all adults to some degree. Periodontal disease refers to infection of the attachment apparatus of the teeth: the gingiva, the periodontal ligament, and the alveolar bone. Unlike pulpal disease, periodontal disease is not usually symptomatic and, therefore, rarely is a primary reason to come to the ED. *Gingivitis* is an inflammation of the gingiva caused by bacterial plaque. In advanced disease the gingiva becomes red and inflamed and tends to bleed easily. With chronic periodontal disease, an abscess can form when organisms become trapped in the periodontal pocket. The purulent material usually escapes through the gingival sulcus; however, occasionally it invades the supporting tissues, the alveolar bone, and the periodontal ligament (periodontitis). Periodontal abscesses that are not draining spontaneously through the sulcus can be drained in the ED. Saline rinses are encouraged to promote drainage. Antibiotics should be reserved for severe cases or for abscesses that cannot be drained. If it is uncertain whether the abscess is from the pulp or the periodontium, antibiotics should be prescribed even if the abscess is drained. [10] [14]

Pericoronitis is a localized inflammation that occurs when gingiva overlying erupting teeth becomes traumatized and inflamed. Third molars are especially susceptible; however, any tooth can be affected. The gingiva overlying the crown may entrap bacteria and debris. Subsequent infection may develop. Typical signs of inflammation and infection may develop including erythema, edema, pus, and foul breath. Examination of the overlying gingiva with a tongue blade or finger will elicit tenderness and may produce drainage from the infection underlying the tissue flap. Pain may be moderate to severe and referral to the ear region is common. The localized infection occasionally spreads to deeper spaces such as the pterygomandibular or submasseteric spaces. Clinically, patients with significant spread of their pericoronal infection will present with trismus secondary to irritation of the masseter and pterygoid muscles.

ED treatment of pericoronitis is directed at detecting regional spread to the deeper spaces. Trismus or other systemic signs of advanced infection require IV antibiotics and urgent consultation for drainage procedures that usually require extraction of the offending tooth. If pericoronal infection is localized, local or nerve block anesthesia is followed by removal of submucosal debris. Saline rinses and oral antibiotics are prescribed with dental follow-up in 24 to 48 hours.

DRAINAGE OF DENTOALVEOLAR INFECTIONS

The important determination for the emergency clinician to make is whether or not the odontogenic infection is localized, confined, and easily accessible or whether it is complex and involves several potential spaces. Likewise, the patient must not appear toxic or exhibit any signs of airway compromise or trismus. Patients not meeting these criteria require specialist referral.

Several anesthetic techniques can make the drainage process more comfortable. Topical anesthetics work well, but the best performing of these agents are often not present in the ED. Benzocaine 20% gel or lidocaine 5% gel generally provide good topical anesthesia. Application of these to the mucosa before injection of the local anesthetic decreases the pain of injection. After application of the topical anesthetic, local anesthetic with a vasoconstrictor should be infiltrated slowly until the tissue blanches. Either a short-acting anesthetic (2% lidocaine) or a longer-acting anesthetic (0.5% bupivacaine) may be used depending upon the clinical circumstances. Regional or dental blocks may be performed instead of local infiltration if needle placement will not track already infected tissues into healthy areas. Otherwise, local infiltration over the site of the abscess should be considered. Instruments necessary for drainage of dentoalveolar abscesses are those usually found on a standard incision and drainage tray and include hemostats, scalpel (#15 or #11), packing material (¼ in. gauze), and a fenestrated penrose drain (Fig. 66-26).

Intraoral Technique

Intraoral abscesses don't routinely require any antiseptic mucosa preparation prior to drainage. After anesthetizing the

Figure 66-26 Equipment for incision and drainage of dental infection. From left to right on lower row. Cotton-tipped applicators, dental (aspirating) syringe with local anesthesia, topical anesthetic and 2 × 2 gauze, scalpel with No. 11 blade, hemostat, iris scissors, gauze (Nu-gauze) packing, and needle holder.

region, a small incision (0.5–1.0 cm) is made over the area of fluctuance, keeping the point of the blade toward the alveolar bone. The hemostat is then used to bluntly dissect the abscess and break up any loculations. Cultures are not necessary unless the patient is immunocompromised, etc. The wound should then be irrigated profusely with normal saline. If the wound is large enough to place a drain or gauze, one end should be tacked to the mucosa with a silk suture to prevent aspiration. Discharge instructions include hourly salt water rinses and follow-up in 24 to 48 hours by the dentist or oral surgeon to remove the drain and provide continued management. The source of the abscess is not always known to the emergency clinician and, therefore, antibiotics are usually prescribed (Fig. 66-27).

Extraoral Technique

Most simple dental infections can be drained intraorally, but occasionally, an abscess may spread to the face and require drainage through the skin. It is important to realize that most dental infections should be drained through the mouth, if possible, as any extraoral drainage will cause some scarring. Abscesses on the face, which should be drained in the ED, are usually very localized and fluctuant and have not spread to any of the deep spaces of the head or neck. It is also important that any incisions made on the face are not in direct proximity to important structures such as the facial nerve or the parotid gland and duct.

The patient is prepared in the usual manner for incision and drainage with a skin scrub and povidone-iodine (Betadine) preparation. The face is draped and the skin is infiltrated with local anesthetic containing a vasoconstrictor (1% or 2% lidocaine with epinephrine). Any incision should be made in the healthy skin slightly below the area of fluctuance and should follow the dynamic skin tension lines. After the incision is made, blunt dissection is made in a direction toward the most fluctuant area until adequate drainage is achieved. The abscess cavity is then profusely irrigated and a drain or packing is placed. The abscess cavity should not be packed tightly, but just enough to keep the incision open and draining. Unlike intraoral drainage, suturing the drain in place is not necessary (Fig. 66-28). The patient should be instructed to take antibiotics and follow up with the oral surgeon in 24 to 48 hours for packing change and definitive management.

Not all infections about the mouth are the result of a simple dental infection. Osteomyelitis of the mandible, various tumors, and other exotic diseases can present as a dental infection.

DEEP-SPACE INFECTIONS OF THE HEAD AND NECK

It is not unusual for odontogenic infections to spread into the various potential spaces of the face and neck. Presenting signs and symptoms consist of fever, chills, pain, difficulty with speech or swallowing and trismus. Although infections of certain teeth usually spread to particular contiguous spaces, the rapid spread of these infections often makes localizing the exact space involved difficult. Any space including the buccal, temporal, submasseteric, sublingual, submandibular, parapharyngeal, and others, may be involved (Fig. 66-30 and Fig. 66-31).

Maxillary extension of periapical abscesses can spread into the infraorbital space and, subsequently, to the cavernous sinus through the ophthalmic veins, resulting in cavernous sinus thrombosis. Cavernous sinus involvement is associated with periorbital cellulitis as well as meningeal signs or a decreased level of consciousness. Periapical infections of the anterior mandibular teeth often spread to the buccinator space or the sublingual space, while those of the mandibular molars spread into the submandibular space. The submandibular space connects with the sublingual space. Infection involving both of these spaces is known as Ludwig's angina, which can be life-threatening (Fig. 66-32). As infection progresses, the submandibular, submental, and sublingual spaces all become edematous and there may be elevation of the tongue and the soft tissues of the mouth. The soft tissues of the posterior

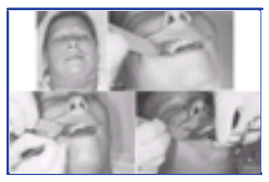

Figure 66-27 *A,* This patient presented with facial swelling up to the right eye. She had minor tooth pain but mostly complained of an ache in the face. X-rays revealed maxillary sinusitis with an air-fluid level. *B,* Intraoral examination revealed a pea-sized pointing abscess at the base of an upper tooth, the cause of sinusitis. *C,* Following local anesthesia, a blade incised the abscess, with drainage of copious pus. *D,* A hemostat was inserted into the abscess cavity to assure drainage, and a small piece of gauze was placed into the cavity. IV antibiotics (clindamycin) followed by oral antibiotics were given, the patient saw her dentist the next day, and plans to extract the tooth were made. She recovered fully.

pharynx can also become involved. Securing the airway becomes of paramount importance. The suprahyoid region of the neck appears tense and indurated, and landmarks may be obscured.

The management of complicated odontogenic head and neck infections centers on airway management, surgical drainage, and antibiotics. If there is uncertainty as to whether the deep spaces are involved, CT scanning can delineate the extension of the infectious process. Airway intervention should be performed early if there is any question of compromise, and tracheostomy may need to be considered. Surgical consultation with an oral maxillofacial surgeon is mandatory as drainage and removal of necrotic tissue may be necessary. Antibiotics should be administered to slow down the spread of the infection and decrease hematogenous dissemination. The bacteria involved are typically a streptococcus/staphylococcus combination but a mixed aerobic/anaerobic infection is also possible. There has recently been an emergence of ß-lactamase-producing organisms in upwards of 40% of isolates from odontogenic neck abscesses. [14] Antimicrobials of choice in complicated odontogenic infections are usually the penicillins. The expanded spectrum penicillins (Amp/Sulb, Tic/Clav, Pip/Tazo) are effective against ß-lactamase-producing bacteria and also cover the anaerobe B. fragilis. Clindamycin is an effective choice when patients are allergic to penicillin. It should be used in combination with a cephalosporin, such as cefotetan, or cefoxitin, in order to cover recently emerging resistant organisms. It is important to realize that in many of these infections, antibiotics are adjunctive therapy and not a substitute for surgical intervention.

DENTAL MATERIALS

As a general rule, EDs should have a well-stocked supply of basic dental materials. Many commercially available products can be used interchangeably with many of the items listed below. These can often be kept with the ENT cart or in another appropriate location. The following is a basic list to consider:

1. Packing gauze
2. Dental roll gauze
3. Calcium hydroxide paste, glass ionomer cement, or zinc oxide cement
4. Dry Socket Paste or eugenol
5. Topical anesthetic gel (20% benzocaine or 5% lidocaine)

6. Topical bactericidal intraoral solution (Ora-5)
7. Periodontal paste (Coe-pak) or self-cure composite
8. Bupivacaine cartridges with epinephrine
9. Save-A-Tooth Tooth Preservation System or fresh milk
10. Zinc oxide/eugenol temporary cement (Temrex)
11. Ringed injection syringe
12. Stainless steel spatula and mixing pads
13. Oral surgery tray with arch bars and ligature wires
14. Tongue blades and cotton-tipped applicators

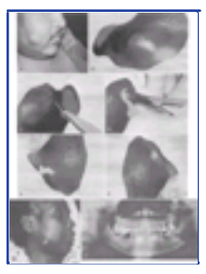

Figure 66-28 *A,* Most dental infections associated with facial swelling should be drained intraorally. Illustration of a facial abscess that points extraorally. *B through F,* Technique of cutaneous drainage in a second patient. *B,* Anesthetic infiltrated below abscessed area. *C,* Incision. *D,* Drainage with blunt dissection. *E,* Gauze packing. *F,* Gauze sutured in place. This step may be omitted if desired. *G and H,* Osteomyelitis of the jaw presenting as a dental problem. This patient had a toothache for weeks that had turned into an abscess pointing on the side of the face. She had been to several emergency departments and was given prescriptions for antibiotics. No history of trauma was forthcoming. *G,* An obvious pointing abscess over the mandible. *H,* A panorex x-ray revealed an old fractured mandible with osteomyelitis draining into the soft tissues.

Figure 66-29 *A*, For persistent bleeding of a tongue, mucosa, or dental extraction site, topical thrombin can be sprayed onto gelfoam. *B*, A tongue laceration like this will heal well without sutures once bleeding is controlled. Topical thrombin may be an option if hemostasis is problematic.

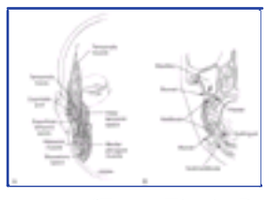

Figure 66-30 *A,* Location of temporal space abscesses. *B,* Route of infection into the buccal space, vestibular spaces, submandibular space, sublingual space, and palatal space. *(From Eisele D, McQuone S: Emergencies of the Head and Neck, 1st ed. St. Louis, MO, Mosby, 2000.)*

Regional dental supply houses are good sources for these items. The Dental Box is a commercially available kit that contains many of the items and is designed for ED use. It is

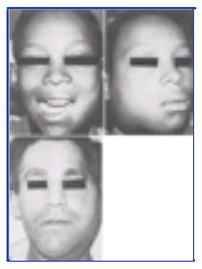

Figure 66-31 *A and B,* Masticator space infection with trismus. *C,* Combined fascial space infections involving the masticator, parapharyngeal, and temporal spaces.

available at The Dental Box Company, PO Box 101430, Pittsburgh, PA 15237; telephone: (412) 364-8712; e-mail: Dentalbox@aol.com.

MANDIBULAR DISLOCATION

In acute dislocation of the mandible, the condyle moves too far anteriorly in relation to the eminence and becomes locked. Subsequent muscular trismus prevents the condyle from moving back into the temporal fossa. Spasm of the external pterygoid, masseter, and internal pterygoid muscles, as well as the associated edema, results in extreme discomfort and anxiety for the patient. It is difficult for the patient to verbalize a complaint because he or she cannot close the mouth. Predisposing factors include anatomic disharmonies between the fossa and the interior articular eminence, weakness of the capsule forming the temporomandibular ligaments, and torn ligaments. Dislocation is likely to occur during maximum opening, such as during yawning, laughing, or "popping" of the mandible in an open position. Although the TMJ is a double joint, dislocation may occur bilaterally or unilaterally. The jaw may be locked open symmetrically or may deviate to the side opposite the side of dislocation. Palpation of the TMJs may reveal them to be anterior to the articular eminence. In the face of trauma, radiographs should be taken to rule out

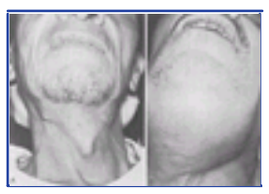

Figure 66-32 *A,* Ludwig angina may initially appear benign. *B,* In Ludwig angina, rapid progression may compromise the airway in a few hours.

fracture, since the clinical picture of both of these conditions is similar and similar occlusal disturbances are produced. Radiographs may not be necessary on an emergency basis if there is no history of trauma or if the condition is recurrent. [14]

Technique for Reduction

If one appreciates the anatomy of the TMJ, the proper sequence for manual manipulation to reduce the dislocation is clear. Following dislocation, the powerful masseter muscles may be in tremendous spasm. For a smooth reduction, it is mandatory to relieve the patient's pain and muscle spasm with slow IV injection of a benzodiazepine, with or without an opioid (see Chapter 35). Some clinicians advocate direct injection of the condylar area with a local anesthetic. When the patient is sufficiently relaxed and analgesia is obtained, the physician faces the patient and grasps the mandible with both hands—one on each side—with the thumbs (which have been wrapped with gauze alone or tongue blades wrapped in gauze) facing the occlusal surfaces of the posterior teeth. The fingertips are placed around the interior border of the mandible in the region of the angles. Some prefer to have the patient seated in a chair or on the floor with the patient's back against the wall. Alternatively, the patient may be in a recumbent position. Downward pressure is slowly and steadily applied to free the condyles from their anterior position to the eminence. The chin is then pressed backward after the jaw has been forced downward, and the mouth is closed while the condyle returns to its position in the fossa. In cases of severe muscle spasm, the jaw may snap back quickly. Therefore, protection of the thumbs is essential.

Following the procedure, the patient should be instructed to stay on a soft diet for 1 week, to avoid wide opening of the mandible, and to take analgesics and muscle relaxants. Local heat may also provide relief. Chronic dislocations or patients who suffer acute recurrences may be helped by a bandage applied around the head holding the mandible to the maxilla for 2 weeks to prevent maximum opening, but this is rarely used. Very severe cases may require intermaxillary wiring and fixation for complete control with the use of Erich arch bars (described in Dental Alveolar Trauma). Patients who have suffered dislocation of the TMJ should be referred to an oral or maxillofacial surgeon for follow-up, since chronic dislocation may require a surgical alteration of the eminence for relief.

CONCLUSION

Dental emergencies presenting to the ED are rarely life-threatening; however, they are often very painful and of great cosmetic concern to patients. They are also common. Many emergencies covered in this chapter are definitively best handled by specialists in the dental or maxillofacial field, but often these patients present to the ED first. The emergency clinician must have a basic working knowledge of the stomatognathic anatomy and be prepared to intervene in order to save a tooth, repair soft tissue, treat severe infection, and relieve pain. The procedures discussed in this chapter will avail themselves to every emergency clinician who practices clinical medicine. The medications, medicaments, glues, and pastes will improve over time and ED personnel should feel free to discuss these advances with their dental colleagues. Likewise, the treatment of any dental or maxillofacial emergency should involve specialty consultation when the emergency is beyond the scope of the emergency provider's practice. Despite the fact that many patients present to the ED with dental problems or postsurgical complications, it is not standard for emergency clinicians to be familiar with complex dental problems.

Acknowledgments

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Section XIII - Special Procedures

Chapter 67 - Procedures Pertaining to Hypothermia

Timothy Erickson
Heather Prendergast

With an increase in outdoor activities, changing weather patterns, and the growing epidemic of homelessness in our country, issues pertaining to hypothermia remain in the forefront. Hypothermia not only is a common diagnosis in rural areas, but also has become even more commonplace in urban centers across the nation secondary to inadequate housing or lack of preparation for cold weather changes. [1] Every year many recreational and elite athletes participate in outdoor sporting events. The higher the environmental stress, the greater the potential for performance failure and development of hypothermia. [2] Optimal treatment for hypothermia remains controversial. It is, however, a well-accepted practice that resuscitation of these individuals is carried out for extended periods of time. The medical literature contains numerous anecdotal reports of profoundly hypothermic individuals who are successfully resuscitated and are neurologically intact at the time of discharge. [3] [4] [5] Despite these spectacular reports of survival, there continues to be both morbidity and mortality from hypothermia. The annual mortality rate is estimated at 757 lives.[6]

This chapter critically reviews approaches and procedures appropriate to the management of several categories of hypothermic patients. The recommendations combine treatment efficacy with safety. Before describing procedures and making recommendations, essential terms are defined and the pathophysiology of hypothermia is briefly reviewed.

DEFINITIONS

Accidental hypothermia (AH) has been defined as an unintentionally induced decrease in the core (vital organ) temperature below 35°C (95°F). [3] [6] Victims of hypothermia can be separated into the following categories: *mild hypothermia*, 35° to 32°C (95.0°–90.0°F); *moderate hypothermia*, <32° to 30°C (<90.0°–86.0°F); and *severe hypothermia*, <30°C (<86.0°F). Other factors that may be useful in separating groups of patients with AH include the presence of underlying illness, [7] [8] [9] [10] altered neurologic state on arrival, hypotension, and the need for prehospital cardiopulmonary resuscitation (CPR). A hypothermia outcome score that incorporates some of these factors and may permit comparison of outcomes for patient groups treated with different modalities has been developed. [11]

Risk factors for the development of AH include: burn injuries, extremes of age, ethanol intoxication, dehydration, major psychiatric illness, trauma, use of intoxicants, significant blood loss, sleep deprivation, malnutrition, and concomitant medical illnesses. [12] [13] Risk factors for development of indoor hypothermia include: advanced age, coexisting medical conditions, being alone at time of illness, being found on the floor, and abnormal temperature perception or regulation. [9] [14]

Because signs and symptoms may be vague and nonspecific, mild to moderate hypothermia may easily be overlooked in the emergency department (ED). A common error is failure to routinely obtain an accurate core temperature on all patients at risk. Often the diagnosis is delayed because of false reliance on standard oral temperatures. Presenting symptoms such as confusion in the elderly and combativeness in the intoxicated patient may not initially be recognized as symptoms of hypothermia. *Hypothermic patients frequently will not feel cold or shiver.* This is particular true in the elderly populations who have impaired thermoregulatory responses because of their advanced age. [15] [16] Paradoxical undressing, a cold-induced psychiatric dysfunction, has been described in confused patients who develop the sensation of heat at lowered body temperatures as a result of constricted blood vessels near the body surface, which suddenly dilate. In many cases, these patients are mislabeled as psychotic, leading to further delays in appropriate treatment. [17]

CORE TEMPERATURE MEASUREMENT

Because of the nonspecific nature of symptoms of hypothermia, an accurate temperature assessment is a necessity in any clinical setting where the diagnosis may be suspected. It is of paramount importance not only for confirmation of the diagnosis but also for guidance in further diagnostic and therapeutic decisions. Any thermometer that does not record temperatures in the hypothermic range is *inappropriate* for evaluating significant hypothermia. Standard glass/mercury thermometers generally cannot record temperatures of <34°C (<93.2°F), although some models are available to record temperatures as low as 24°C (75.2°F) (Dynamed, Inc, Carlsbad, CA). An electronic probe and accompanying calibrated thermometer (Fig. 67-1) is recommended when monitoring this vital sign.

Figure 67-1 Electric thermometer (without probe). This is model 43TA with Fahrenheit and Celsius scales. Infrared tympanic membrane thermometers are acceptable alternatives, but the rectal probe has the advantage of providing a continuous readout. *(Image courtesy of Yellow Springs Instrument Company, Yellow Springs, OH.)*

Core temperature is traditionally estimated with a rectal probe. The rectal temperature, however, often lags behind the core temperature because of large gradients within the body.[18] Esophageal probes may be used, although they may be affected by warm humidified air therapy, which is commonly used in severe hypothermia. Other possible sites for temperature measurement include the tympanic membrane, nasopharyngeal passage, and the urinary bladder. [18] [19] [20] Fresh urine temperature can closely approximate core temperature.[21] "Deep forehead" temperatures measured with a Coretemp thermometer (Teramo, Tokyo, Japan) have also demonstrated excellent accuracy and approximation of core temperatures.[22] For continuous monitoring purposes, rectal or bladder probes are preferred. Infrared tympanic temperatures have demonstrated excellent correlation with core temperatures. Studies show that although easier to use and faster, infrared tympanic temperatures can be inaccurate with extremes of temperature by underestimating higher temperatures and overestimating lower temperatures.[23] When a rectal probe is used, it should be inserted at least 10 cm beyond the anal sphincter and its position frequently verified. One should remember that temperature gradients exist in the human body and consistency of monitoring at one or more sites is mandatory. A chart and formula that convert centigrade to Fahrenheit temperatures will assist the clinician in assessing the severity of hypothermia (Fig. 67-2).

PATHOPHYSIOLOGY

AH results from the failure of the body's thermoregulatory responses to generate enough heat to compensate for heat losses. These thermoregulatory responses include shivering, tachycardia, tachypnea, increased gluconeogenesis, peripheral vasoconstriction, and shunting of blood to central organs. [24] As

Figure 67-2 Temperature conversion scale. To change Celsius (centigrade) to Fahrenheit, multiply the Celsius temperature by 9/5 and add 32. To change Fahrenheit to Celsius, subtract 32 from the Fahrenheit number and multiply by 5/9.

the core temperature drops despite these compensatory mechanisms, the patient becomes poikilothermic and cools to the ambient temperature.

Four methods of heat loss affect the body: radiation, conduction, convection, and evaporation. Radiation involves the transfer of heat from a warmer body to a cooler environment and accounts for approximately 60% of heat loss in a normothermic individual. Conduction refers to heat loss from direct contact with a cooler surface. These losses are most profound with immersion hypothermia. Convection occurs when cool air currents pass by the body and accounts for 15% of heat loss, especially with a wind chill factor. Evaporation refers to significant heat loss through sweating and insensible water losses. [16] [24] In hypothermia, the enzymatic rate of metabolism itself decreases two to three times with each 10°C (18°F) drop and cerebral blood flow decreases 6 to 7% per 1°C (1.8°F) drop.

Signs and symptoms of hypothermia vary according to the core temperature. The overall functioning of all organ systems are impaired by the cold. [25] The greatest effects, however, are seen with the cardiovascular, neurologic, and respiratory systems (Table 67-1). As the core body temperature drops below 33°C (91.4°F), the patient becomes confused and ataxic.[26] The initiation of involuntary motor activity (shivering) prevents the reduction in core temperature. [27] Shivering thermogenesis in skeletal muscle operates on acute cold stress. In the malnourished patient, the mechanism may be rendered ineffective secondary to reduced muscle mass. [28] Shivering stops at about 32°C (89.6°F); however, shivering artifact on an electrocardiogram has been associated with increased survival in severe hypothermia. [29] Atrial fibrillation occurs frequently as the temperature continues to drop and the patient loses consciousness. A J wave in the electrocardiogram often appears before ventricular fibrillation (Fig. 67-3). Although classically considered pathognomonic for hypothermia, the J or "Osborne" wave has no prognostic or predictive value in cases of hypothermia. A recent study found that Osborne waves occurred in 36% of AH survivors and in 38% of nonsurvivors. [29] [30] Ventricular fibrillation may occur below 29°C (84.2°F) and becomes common as the core drops to 25°C (77°F).[31] The electroencephalogram flattens at 19° to 20°C (66.2°F–68°F), [32] and asystole commonly occurs at 18°C (64.4°F) but has been seen at higher temperatures. Initial core temperature does not necessarily correlate with vital outcome. [33] The lowest recorded temperature for a survivor of AH is 9.0°C (43.7°F). [24]

INITIAL EVALUATION AND STABILIZATION OF THE HYPOTHERMIC PATIENT

The treatment of hypothermia can be divided into prehospital care and ED management.

Prehospital Care

In the prehospital setting, the primary focus is removal of the individual from the current environment to prevent further decreases in core temperature. Studies have shown that oral temperatures are sufficiently accurate for field use. [34] In addition, infrared tympanic thermometers may not be reliable in the prehospital setting. [35] Special care must be taken in handling these patients, as aggressive measures can inadvertently trigger

TABLE 67-1 -- Signs and Symptoms of Hypothermia

Core Temperature	Cardiovascular System	Respiratory System	Central Nervous System
34–35°C	Tachycardia	Tachypnea	Lethargy
	Increased afterload	Increased minute ventilation	Mild confusion
	Increased systemic blood pressure		Loss of fine motor coordination
30–34°C	Progressive bradycardia	Increased bronchial secretions	Delirium
	Decreased cardiac output	Diminished gag reflex	Slowed reflexes
	Hypotension	Depressed cough response	Muscle rigidity
	Lengthening of cardiac conduction		Abnormal EEG
	Atrial/ventricular dysrhythmias		
<30°C	Spontaneous ventricular fibrillation	Respiratory rate decreased to 5 breaths/minute	Aflexia
	Osborne waves at 25°C		Coma
			Fixed pupils
			Rigidity
			EEG silent at 19°C

cardiac dysrhythmias. Prolonged transport times should be avoided. [36] [37] Patients should be wrapped in dry blankets after removal of wet clothing. Studies have shown that for mild hypothermia, resistive heating (e.g., warming blankets) can be safely used in the prehospital setting. Resistive heating augments thermal comfort, increases core temperature by approximately 0.8°C/hr, and reduces patient pain and anxiety during transport. [34] In one study, resistive heating more than doubled the rewarming rate compared to passive insulation without producing an afterdrop. [38]

Potential traumatic injuries to the spine or extremities should be immobilized before transport. Continuous attention to airway maintenance should be a priority. Fluid resuscitation can be initiated with intravenous crystalloid, preferably with D5NS.

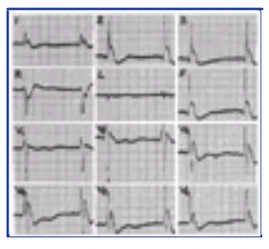

Figure 67-3 In severe hypothermia, the electrocardiogram (ECG) exhibits marked elevation of the J deflection, so-called Osborne waves. The height of the J wave is proportionate to the degree of hypothermia, and this finding is usually most marked in the midprecordial leads. The ECG is of a patient with sinus bradycardia, but approximately half of patients with a temperature below 32°C (89.6°F) develop slow atrial fibrillation, a rhythm that usually converts spontaneously with rewarming. *(From Marriott HJ: Practical Electrocardiology, 8th ed. Baltimore, Williams & Wilkins, 1988.)*

Most hypothermic patients are *dehydrated* because fluid intake is reduced and cold causes a diuresis. Theoretically, it is best to avoid using lactated Ringer's solution because of the decreased metabolism of lactate by cold-induced hepatic dysfunction. If possible, warmed intravenous fluids can be administered to patients and are generally well tolerated. [39] [40] Flameless heaters currently used by military medical units provide easy field-expedient means of warming fluids in the prehospital setting.[41] Unresponsive patients should be intubated, although there is no universal agreement on when to intubate a hypothermic patient who has detectable vital signs. Pulse oximetry is usually not helpful since vasoconstriction limits blood flow to the periphery and readings may be inaccurate or not possible.

Some authors suggest that pulseless victims with core temperatures below 32°C should be transported with continuous CPR. [39] Other authors believe that it is unnecessary to perform CPR on a patient who has any perfusing cardiac rhythm since it may precipitate ventricular fibrillation. [5] At this juncture there is no universally accepted standard for intubation or CPR in hypothermic patients with detectable vital signs.

Emergency Department Management

Treatment priorities in the ED setting are preventing further decreases in core body temperature; establishing a steady, safe rewarming rate while maintaining the stability of the cardiopulmonary system; and providing sufficient physiologic support. The rate of rewarming and the techniques used depend upon the degree of hypothermia and the severity of the patient's clinical condition (Table 67-2). Inherent in the goals of management of these patients is the anticipation and prevention of complications.

In the pulseless, apneic patient, CPR should be initiated and continued until the core temperature is above 34°C. Profound hypothermia results in coma, hyporeflexia, fixed and dilated pupils, severe bradycardia, and often unobtainable blood pressure. In severe hypothermia, a pulse may not be palpable and blood pressure measurement may require the use of a Doppler device. Electrocardiographic monitoring provides rate and rhythm status. All patients who have more than minimal impairment require frequent determination of their oxygenation, ventilation, and acid-base status by means of arterial blood analysis. All patients should have large-bore IV lines established, if feasible. Maintenance IV fluids should be given routinely. Warming of all IV fluids to 40° to 42°C (104°–107.6°F) is reasonable, but the usual volumes administered will not contribute significant calories of heat. In addition with long standard IV tubing, the heated IV fluids may actually cool to room temperature before entering the patient's IV site.

TABLE 67-2 -- Rewarming Techniques

Core Temperature	Method and Techniques
>32°C	Passive external rewarming (PER)
	• Dry blankets, clothing
	• Heated IV solutions (43°C) D5NS
	• Warm fluids if fully alert
=32°C	Active external rewarming (AER)
	• Heated blankets, heating pads, warm air convection
	• Radiant heat sources
	• Alcohol-circulating blankets
	Active core rewarming (ACR)
	• Peritoneal dialysis
	• Bladder, gastric or colonic lavage with warm fluids (43°C)
	• Heated IV fluids
	• Heated humidified oxygen
	• Thoracic cavity lavage (43°C)
	• Extracorporeal blood rewarming
	• Hemodialysis
	• Ultrasonic and low frequency microwave diathermy
	• Arteriovenous anastomoses rewarming

With a mild to moderate reduction in core temperature, the level of mentation correlates with the severity of the AH, associated illness, or both. Noteworthy exceptions are alcoholics and diabetics who can present in comas at higher core temperatures because of concomitant hypoglycemia. These patients should routinely have bedside glucose measurements performed upon arrival to the ED. There is a high correlation between alcohol consumption and development of hypothermia especially in colder climates. [1] A review of 68 cases of hypothermic deaths in Jefferson County, AL found that a significant number of cases involved middle-aged males who had consumed alcohol. [42] In the 22 cases of AH reviewed by Fitzgerald, [43] all except two patients were alcoholics. The serum glucose level was <50 mg/dL in 41% (nine patients). This study noted glycosuria in two patients, even when low serum glucose values were evident, and described a renal tubular glycosuria in AH. Such glycosuria may worsen or cause hypoglycemia; *hence, glycosuria in AH is no guarantee of an adequate serum glucose concentration*. This supports the routine use of supplemental IV glucose unless a normal serum glucose value can be quickly ensured. Also, IV thiamine (100 mg) and a trial dose of 0.4 to 2 mg IV naloxone (Narcan) may be considered. Although failure to rewarm spontaneously has been noted in victims with hypothyroidism and other endocrine deficiencies, the use of thyroid hormones and corticosteroids is reserved for those patients with suspected thyroid and adrenal insufficiency, respectively.

The thermoregulatory vasoconstriction caused by hypothermia significantly decreases the subcutaneous oxygen tension. [16] A good correlation between the incidence of wound infections and the subcutaneous oxygen tension exists. As core temperatures decrease from 41°C to 26°C, neutrophil function is significantly impaired. [16] Conversely, in animal models, hypothermia appears to decrease leukocyte sequestration within brain parenchyma, offering some resistance to meningitis. [44] Although antibiotics are not routinely indicated in uncomplicated mild hypothermia, some authors advocate the routine empirical initiation of broad-spectrum antibiotic therapy on admission of severely hypothermic patients. No specific standard exists.

MANAGEMENT GUIDELINES

Hypothermia affects virtually every organ system owing to the generalized slowing of the body. Management goals depend upon the severity of hypothermia, but in all cases the primary goal is to increase the core temperature and prevent further losses. In the patient with mild hypothermia, a conservative approach to rewarming is generally advocated. Overly aggressive methods may be more harmful to the patient by causing a worsening hypotension, a paradoxical decrease in core temperature, and cardiac dysrhythmias. Other complications may include bleeding [16] and infection of surgical incisions. The optimal rewarming rate remains unclear and varies with each case. Standard rewarming rates are a 0.5°C/hr to 2.0°C/hr (0.9°F/hr–3.6°F/hr) rise in temperature *in the otherwise stable patient* (Table 67-3). Invasive therapy should be carefully considered and individualized to the severity of the hypothermia and the condition of the patient, and one should avoid the temptation to *overtreat and overmonitor with invasive techniques the otherwise stable hypothermic patient*.

TABLE 67-3 -- Warming Rates (°C/hr)

	Passive External	Active External	Inhalation of Warm Air	Peritoneal Lavage	Colon/Gastric/Bladder Lavage
1st hr	1.4	1.5	1.5	1.5	1.3
2nd hr	1.4	2.4	2.0	2.5	1.7
3rd hr	1.8	2.0	1.9	3.2	1.8

From Danzl D, Pozos RS: Multicenter hypothermia study. Ann Emerg Med 16:1042, 1987.

The patient with severe underlying problems such as hypoglycemia, hyperglycemia, sepsis, adrenal crisis, drug overdose, or hypothyroidism should be treated appropriately for those conditions as well as aggressively for hypothermia because long-term outcome may depend more on treatment of the underlying illness than the hypothermia.[45]

Passive External Rewarming

The cornerstone of effectiveness for this method relies on the body's ability to restore normal body temperature through its own mechanisms for heat production. Initial management begins with the removal of wet clothing followed by placement of blankets. Warm fluids containing glucose may be given orally to the patient who is fully alert. For patients with mild AH, removal of wet clothing followed by *passive external rewarming* with blankets generally suffices (Fig. 67-4). The technique is simple; however, the patient must be capable of generating enough body heat for this method to be successful. Patients who cannot shiver, patients who are hypotensive, or patients who are intoxicated or malnourished may not have this capability. Survival rates using passive external rewarming have ranged from 55–100%.[46] [47] [48]

Figure 67-4 Passive external rewarming features. Further heat loss must be stopped through insulation and environmental manipulation. Damp clothing is removed. Dry, warmed blankets are applied. Warmed intravenous fluids are given to counteract the cold-induced diuresis. Internal heat generation is required for rewarming. Rewarming rates are relatively slow. Aggressive intervention with drugs and invasive monitoring may be more harmful than helpful in the otherwise stable hypothermic patient. Note the absence of tracheal intubation, central lines, and Foley catheter in the mild to moderately hypothermic patient.

For patients in the moderate or severe category of hypothermia, a more aggressive approach may be warranted. The options available are active external rewarming and active core rewarming. Active core rewarming techniques can be further divided into less invasive and more invasive techniques. Generally, the aggressiveness of therapy depends more on the patient's underlying health, hemodynamic status, and response to initial therapy than the initial temperature.

Active External Rewarming

The application of heat to the skin of the hypothermic patient has been termed *active external rewarming*.

Indications

Although there is some suggestion that active external rewarming of profoundly hypothermic patients by immersion may be associated with an increase in mortality over other treatments,[11] [49] more recent studies have suggested that this technique is highly effective for mild hypothermia. [26] [50] It should be used selectively and limited to the trunk. Other forms of active external rewarming are increasingly used in the ED as adjunctive care of moderately hypothermic, otherwise healthy individuals. Vasoconstriction limits the ability to increase core temperature using techniques that primarily warm the skin. [51]

Active external rewarming is most beneficial in cases where heat supplied by the external source is greater than the loss of rewarming heat incurred by the cessation of shivering. Finally, in settings where more aggressive warming techniques are precluded owing to lack of equipment or personnel, active external rewarming with body-to-body contact may be the only option available to the rescuer. However, the rewarming contribution of body-to-body contact appears limited. [52]

Equipment

Traditionally, immersion therapy has used a heated (40° to 42°C) water tank of the type present in most burn units. Generally, the hypothermic patient is immersed entirely except for the extremities and head (Fig. 67-5), although immersion of the extremities may hasten rewarming.[18] [53] [54] Alternatively, conduction warming is performed using warm water-filled heat exchange blankets (e.g., Blanketrol; Cincinnati Sub-Zero Products, Cincinnati, OH). Intraoperative studies have demonstrated excellent results.[53] A forced warm air convection system (Bair Hugger; Augustine Medical, Eden Prairie, MN; Snuggle Warm Convective Warming System; Sins Level 1, Inc., Rockland, MA) has been used for postsurgical rewarming.[53] [55] This approach also has been used successfully for ED-based AH therapy. Warm air convection rewarming permits continued monitoring in the ED and is better tolerated than immersion due to the less rapid development of vasodilation in peripheral tissues.

Technique

Because profound fluid shifts can occur with conduction warming, the patient should receive supplemental IV fluid that has been warmed to 40°C (Hotline Fluid Warmer; Sims Level 1, Inc., Rockland, MA), given at a rate sufficient to generate a urinary output of 0.5 to 1.0 mL/kg per hour. An initial fluid bolus of 500 mL of 5% dextrose and normal saline may be beneficial. Note that blood pressure is not an accurate way to gauge fluid resuscitation because serious hypothermia is always accompanied by "physiologic" hypotension. Because

Figure 67-5 Active external rewarming features. A heat source warms the skin. Traditionally, warm water immersion has been used (as shown). Rates of rewarming are very rapid in some series. There have been suggestions of increased mortality with this method when used for patients with moderate to severe hypothermia, but certain conditions may warrant its use. A major drawback is the inability to closely monitor the patient undergoing immersion. Use of warm air convection (e.g., use of Bair-Hugger device, Eden Prairie, MN), however, allows rewarming in the ED with full patient monitoring and less rapid vasodilation during rewarming.

patients requiring mechanical ventilation have rarely been subjected to tank immersion, it cannot be recommended for hypothermic patients who require intubation. Rewarming rates ranging from 0.9° to 8.8°C (1.6° to 15.8°F) per hour have been reported with immersion therapy. [4] [56]

A heat exchange blanket allows the patient to receive other treatments that may be difficult or impossible to carry out in a tub, such as defibrillation, CPR, or more invasive warming techniques. With this latter form of conduction heating, the heating blanket and overlying cloth sheet is placed under the patient. The blanket temperature is set at 40° to 42°C, and the measures described under passive rewarming techniques (see Fig. 67-4) are initiated.

Forced-air rewarming (convection) uses a blanket cradle to create an environment through which heated air is blown. Access to the patient is quite good with this system in that the overlying blankets can be raised temporarily to evaluate the patient or perform procedures. One study found this approach superior to a heating blanket.[57] Experience with mild immersion-induced hypothermia in volunteers suggests that the forced-air technique warms at a rate comparable to vigorous shivering, but with less metabolic stress and less afterdrop. [58]

Arteriovenous Anastomoses Rewarming

Ateriovenous ananstomoses (AVA) rewarming involves the immersion of the distal extremities (hand, forearms, feet, and lower legs). Advantages include rapid rewarming rates. A study in healthy volunteers using AVR immersion in temperatures of 45°C and 42°C, respectively, demonstrated rewarming rates of 9.9°C/hr (±3.2°C/hr) for the former and rewarming rates of 6.1°C/hr (±1.2° C/hr) for the latter. There was also a decrease in postcooling afterdrop. AVR is well tolerated by patients because of the rapid rise in core temperature and the shortened period of shivering. [18]

Complications

There is concern that surface warming with accompanying vasodilation may produce a relative hypovolemia in the hypothermic patient. Other complications described with the active external rewarming method include core temperature afterdrop and rewarming acidosis. In core temperature afterdrop, colder peripheral blood is transported to the warmer core organs, further reducing the core temperature. In rewarming acidosis, there is a return of colder blood and lactic acid to the core organs, worsening the acidosis. To limit these complications in moderate hypothermia, some authors advocate using active external warming only after active internal techniques have been initiated. [24]

Importantly, CPR and other advanced cardiac therapy and monitoring are impossible with immersion rewarming. Until studied further, active external rewarming should only be considered in a clinically monitored setting for mildly hypothermic patients who can protect their airways. When using a heating device, the potential for burns to the areas in greatest contact with the heating source should also be monitored.

Active Core Rewarming

There is evidence that active core rewarming may decrease mortality from *severe* hypothermia exposure compared with other techniques. In the face of circulatory failure, often the best chance of survival is treatment with extracorpeal circulation (ECC) and warming of the blood. [59] Several methods have been described, including the use of warm humidified air through an endotracheal tube or mask (Fig. 67-6), peritoneal lavage, gastric or bladder lavage with warm fluid, thoracic tube lavage, cardiopulmonary bypass (Fig. 67-7), arteriovenous anastomoses, peripheral vascular extracorporeal warming, hemodialysis, and thoracotomy with mediastinal lavage (Fig. 67-8). These techniques transfer heat actively to the body core, achieving varying rewarming rates. The specific techniques as well as some of the advantages and disadvantages for each procedure follow.

Figure 67-6 Active core rewarming methods. Inhalation of warm, humidified air or oxygen causes gradual core rewarming and should be a *mainstay of rewarming therapy*. This method can be combined with other methods such as heated intravenous fluids. Peritoneal lavage is a more rapid method of core rewarming that requires placement of one or more intraperitoneal catheters. Other techniques are described in the text.

Figure 67-7 Active core rewarming cardiac bypass features. Bypass technology and a surgical team are needed. The rewarming rates are rapid. This method is useful in a patient with cardiac arrest; the technique is invasive, expensive, and technically difficult.

Emergency Warming of Saline in a Microwave

Under ideal circumstances saline is kept warm in a standard warming device. When large amounts of saline are required for such procedures as peritoneal lavage, saline bags may be rapidly warmed in a standard microwave oven.[59A] Although devices will vary, a 650-watt microwave has been demonstrated to warm a liter of room temperature nondextrose containing saline from 21.1°C to 38.3°C (70°F to 101°F) in 120 seconds on the high setting. A suggested protocol is midcycle (i.e., after 60 seconds) interruption with agitation, and end-cycle agitation before infusion.

Inhalation of Heated Humidified O_2 or Air

The use of warm humidified oxygen to treat hypothermia was described by Lloyd in 1972. [60] Average rates of rewarming of 1°C/hr via mask and 1.5°C to 2.0°C/hr via endotracheal tube with heated aerosol at 40°C can be obtained. [4] [26] Faster rewarming rates may be accomplished using a maximum safe aerosol temperature of 45°C (113°F).

Figure 67-8 Active core rewarming open thoracic lavage features. The heart is warmed directly. This method requires surgical backup and is relatively invasive. The rewarming rates are rapid. An alternative that is more practical for the ED is pleural rewarming repeatedly using 200 to 300 mL warmed saline intermittently placed, then withdrawn through a chest tube (see text).

Core rewarming with the technique occurs through the following mechanisms. The warmed alveolar blood returns to the heart, thereby warming the myocardium. The warmed humidified air delivered to the alveoli also warms contiguous structures in the mediastinum by conduction. Finally, warming the inhaled air or oxygen eliminates a major source of heat loss.

Indications and contraindications.

The use of heated humidified air or oxygen is a simple technique that *should be used routinely*, either by itself or in combination with other methods, in all patients with hypothermia, regardless of severity. If the correct equipment is available, it can be used in the field as well as the hospital. [36] [37] However, one must address the risk of burns during inhalation of warm air in the field environment. [61] Mouth-to-tube ventilation in the intubated hypothermic prehospital patient has the theoretical advantage of providing warm humidified air without special equipment. The ventilating rescuer can inhale oxygen before expiring into the patient's endotracheal tube to provide air with increased oxygen content. There are no contraindications for or reported complications from the use of warm humidified air for hypothermia, and there is no afterdrop. [62]

Technique.

A heated cascade nebulizer can be used with a mask for patients with spontaneous respirations or with a volume ventilator for intubated patients. It is important to monitor the inspired air to maintain a temperature of approximately 45°C. [63] Temperatures higher than 50°C (122°F) may burn the mucosa, and temperatures lower than 45°C (113°F) do not deliver maximum heat. The air or oxygen must be humidified, and the heater module may need modification because many units have feedback mechanisms that shut off at a given temperature. As a practical issue, it may be difficult to deliver oxygen at the recommended temperature because of equipment limitations. In many cases the air temperature is only 30°C (86°F).

Summary.

Studies have suggested that the rewarming rate of inhalation therapy is inferior to that of peritoneal lavage, thoracic lavage, and bath rewarming. [4] However, because inhalation therapy can be combined with any and all other methods of rewarming and because it is relatively noninvasive and inexpensive, it should be considered as the initial treatment of choice for hypothermic patients.

Peritoneal Dialysis (Lavage)

Peritoneal dialysis (lavage) is an attractive treatment for severe hypothermia because it is available in most hospitals and does not require any unusual equipment or training. Rewarming rates of 2°C to 3°C (3.6° to 5.4°F)/hr, depending on the dialysis rate, can be achieved without sophisticated equipment that may delay therapy or require transfer of the patient to a tertiary care facility. [64] This technique can also be used to help correct electrolyte imbalances.

Peritoneal dialysis rewarming was first used successfully in a patient in ventricular fibrillation with a temperature of 21°C (69.8°F). [65] Since that time, there have been reports of successful rewarming with peritoneal lavage in stable, severely hypothermic patients and unstable hypothermic patients in cardiac arrest. [66] [67] Peritoneal lavage works through heat transfer from lavage fluid to the peritoneal cavity. The peritoneal great vessels and abdominal organs provide a large surface area for heat exchange. The use of warmed peritoneal lavage fluid is an effective approach to rewarming. [68] There have been reports of success in the literature

using rapid high-volume peritoneal lavage (RHVP) in pediatric patients. The technique involves use of an infra-umbilical "mini-laparotomy" incision followed by placement of a large silicone peritoneal dialysis catheter. The catheter is connected to a rapid infusion device with the delivery of 1 liter of warmed normal saline every 90 seconds. [68]

Indications and contraindications.

Peritoneal dialysis is appropriate therapy in any severely hypothermic patient. In practice, however, it is often omitted if other measures appear to be successful. There are no universally agreed upon criteria for performing peritoneal lavage in hypothermic patients who have detectable vital signs. Although theoretically less effective than other techniques that directly warm the thorax in the setting of cardiac arrest, it has been used successfully in that situation. It is theoretically useful in hypothermic patients who have overdosed with a dialyzable toxin. Other less invasive methods, such as gastric or bladder lavage, or warm nebulized air or oxygen inhalation, may be preferred in stable patients with temperatures higher than 26°C to 28°C (78.7°F to 82.4°F). Peritoneal dialysis should not be performed on patients with previous abdominal surgery. It should be used with extreme caution in patients with a coagulopathy after the risks and benefits have been considered.

Equipment.

The equipment described in Chapter 44 is needed. We recommend using the Seldinger technique with a commercially available disposable kit (e.g., Arrow Peritoneal Lavage Kit, product no. AK-09000, Arrow International, Inc., Reading, PA) because of the ease of performance and minimal morbidity associated with this procedure.

Technique.

In the noncritical patient, a coagulation profile should be obtained before the procedure, but in life-threatening situations immediate initiation of the procedure is warranted before laboratory studies. The patient should be supine with a Foley catheter and nasogastric tube in place. After infiltration with lidocaine, an infraumbilical stab incision is made with a No. 11 blade, and an 18-ga needle is placed into the peritoneal cavity directed toward the pelvis at a 45° angle. A standard flexible J-wire is inserted through the needle, and the needle is removed. The 8-Fr dialysis catheter is passed over the wire with a twisting motion, and the wire is removed.

Lavage rates of 4 to 12 L/hr can be achieved with two catheters. Fluid is warmed with a standard blood warmer to 40°C to 45°C. A standard 1.5% dextrose dialysate solution is used; adding potassium (4 mmol/L) is recommended if the patient becomes hypokalemic. Ringer's solution and normal saline have also been used successfully. The rate should be at least 6 L/hr and preferably 10 L/hr. [67]

Complications.

The Seldinger method has a complication rate of <1%. [69] [70] A "mini-lap" using direct dissection may also be used but may have a higher complication rate. [69] Further discussion of potential complications is provided in Chapter 44 .

Summary.

Peritoneal dialysis is a useful method because it uses readily available fluid and can be done with a self-contained disposable kit. [71] If a hospital also treats trauma victims, the same lavage kit can be used for evaluation of abdominal trauma. If this technique is combined with warm nebulized inhalation, warming rates of 4°C

(7.2°F) per hour can be achieved.[72]

Gastrointestinal and Bladder Rewarming

Gastric or bladder irrigation offers some of the same advantages as peritoneal dialysis without invading the peritoneal cavity. Heat is delivered to structures in close proximity to the core. In the Multicenter Hypothermia Study, gastric/bladder/colon lavage had a first hour rewarming rate of 1.0° to 1.5°C/hr and a second hour rewarming rate of 1.5° to 2.0°C/hr for severe hypothermia. [71] In a multifactorial analysis of the Multicenter Hypothermia Study, there was a trend toward improved survival in patients on whom this method was used.[11]

Although the amount of heat delivered with gastric lavage appears less than that delivered with peritoneal dialysis, it is somewhat easier to use and less invasive. When combined with other methods, gastric or bladder lavage provides significant warming. [71] [72] Serum electrolyte levels should be monitored if large volumes of tap water are used, because dilutional electrolyte disturbances may occur. Children and geriatric patients may be more susceptible to electrolyte changes with tap water irrigation.[73]

Indications and contraindications.

Warmed gastric or bladder lavage may be used as adjunctive therapy in moderate or severe hypothermia. It can be combined with other warming techniques when rapid rewarming is needed. Patients who are obtunded and lack protective airway reflexes should have endotracheal intubation before gastric lavage to prevent aspiration of gastric contents. Refer to the appropriate chapters concerning nasogastric tube placement (see Chapter 41), gastric lavage (see Chapter 43), and urethral catheterization (see Chapter 56) for specific contraindications to these procedures.

Equipment.

Use a large-diameter 32- to 40-Fr lavage tube with normal saline or lactated Ringer's solution warmed to 40° to 45°C (in a microwave or blood warmer with verification of temperature before use) and a Y connector and clamp as described in Chapter 43 . Although smaller tubes are easily passed nasally, oral placement of the large lavage tubes is recommended. A modified Sengstaken tube with gastric and esophageal balloons may also be used.

Technique.

Generally, 200- to 300-mL aliquots of fluid are instilled into the stomach before removal by gravity drainage. For bladder irrigation, the optimal volume is not known but bladder distension should be avoided (100- to 200-mL aliquots should be sufficient). The amount of time that the irrigant should be left before removal is not known, but rapid exchanges with a dwell time of 1 to 2 minutes is suggested.

Complications.

Lavage complications include trauma to the nasal turbinates (especially if a large tube is passed nasally), gastric and esophageal perforation, dilutional hyponatremia, inadvertent placement of the tube in the lungs, and pulmonary aspiration. All of these can be minimized by careful, proper technique. Fluid overload or electrolyte disturbances when using tap water are potential complications in pediatric and geriatric patients.

Summary.

Gastrointestinal and bladder lavage with heated fluids is easily performed using equipment and solutions available in any hospital. Because of its ease and availability, it can be started early in the resuscitation and combined with any other rewarming method to significantly add heat, [58] although the specific effect on morbidity and mortality is not known.

Thoracic Cavity Lavage

Thoracic cavity lavage can be performed either *closed*, through chest tubes placed in the one hemithorax,[74] [75] or *open*, after resuscitative thoracotomy.[76] The former approach offers the advantages of being less invasive and an effective form of treatment in hospitals not equipped for cardiopulmonary

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bypass.[75] Furthermore, closed-chest CPR can be continued while this technique is used. The open thorax approach offers the theoretical advantage of direct heart warming and the option of open-chest cardiac massage. Rapid warming rates of 6° to 7°C in 20 minutes have been described. [74] [75]

Indications and contraindications.

Thoracic cavity lavage should be considered for patients requiring rapid core rewarming in the setting of cardiac arrest or inadequate perfusion (e.g., shock, during CPR) when cardiac bypass is not available. Open thoracic lavage should be considered in those patients who will receive open chest massage or thoracotomy for other reasons (e.g., hypothermic arrest with penetrating trauma). Thoracic lavage is not necessary for patients with mild or moderate hypothermia who can be rewarmed by other, less invasive methods. The technique should be avoided in the patient with a coagulopathy unless needed as a life-saving measure.

Closed thoracic lavage.

Two large-bore thoracostomy tubes (e.g., 36- to 38-Fr in 70-kg adults) are placed in one hemithorax (see Chapter 10). One chest tube is infused with 3-L bags of heated normal saline (40° to 41°C [104° to 105.8°F]) using a high-flow fluid infuser (e.g., Level-1 Fluid Warmer, Technologies, Inc, Marshfield, MA). The effluent is collected with an autotransfusion thoracostomy drainage set (e.g., Pleur-evac, Deknatel A-5000-ATS, Fall River, MA), and the removable reservoir is repeatedly emptied as needed. The use of a single chest tube system using a Y-connector arrangement similar to gastric lavage is also effective. Aliquots of 200 to 300 mL with a 2-minute dwell time followed by suction drainage (at 20 cm H_2O) are recommended.

Closed-chest massage is used until adequate spontaneous perfusion occurs. Closed-chest defibrillation may be required in the patient warmed to 30°C (86°F) with persistent ventricular fibrillation. Thoracic lavage is generally continued until the patient's temperature approaches 35°C (95°F).

Open thoracic lavage.

A left thoracotomy is performed as described in Chapter 18 . Saline warmed to 40° to 41°C can then be continuously poured into the thoracic cavity, bathing the heart while an assistant suctions the excess fluid from the lateral edge of the thoracotomy. Alternatively, fluid may be added to the thorax and mediastinum intermittently and suctioned after several minutes, and more warm saline may be added (see Fig. 67-8). This technique also allows for direct myocardial temperature monitoring. Direct cardiac massage is used until adequate spontaneous perfusion occurs. Direct cardiac defibrillation may be required in the patient warmed to 30°C with persistent ventricular fibrillation. When defibrillation is successful, direct myocardial warming should continue until the patient's temperature approaches 35°C. If defibrillation is unsuccessful at a core temperature of 30°C, further warming is warranted while oxygenation, perfusion, and other physiologic parameters are optimized before further defibrillation attempts.

Summary.

Thoracic lavage is an effective form of active core rewarming that is usually reserved for hypothermic arrest patients. [74] [75] Thoracic lavage may be considered when

vital signs are inadequate or unstable enough to severely limit perfusion. However, precise indications are not clarified beyond the patient in cardiac arrest.

Cardiac Bypass

The use of cardiac bypass or an extracorporeal shunt through either the femoral artery-femoral vein or aorta-caval procedure can result in rapid rewarming but requires surgical expertise, availability of appropriate equipment, and technical support. [3] [5] [7] This procedure has not been compared with other rewarming methods in a controlled fashion and few centers have this modality available in a time frame that would impact survival rates. Its main advantages appear to be the rapid rate of warming it produces and optimal patient oxygenation and perfusion. Femoral flow rates of 2 to 3 L/min with the warmer set at 38° to 40°C (100.4° to 104°F) will raise the core temperature 1 to 2°C every 3 to 5 minutes. [4] Drawbacks include potential delays in assembling the appropriate team and equipment, delays owing to the time necessary to complete the operation, complications from the operation, the expense of the procedure and bypass equipment, and the potential for infection. Its use in extreme situations that may include cardiac arrest should be based on individual characteristics of the patient, physician team, and hospital resources. If rapidly available, it should be strongly considered in hypothermic patients with asystole or ventricular fibrillation. [33] If oxygenation is not a consideration, venovenous rewarming with an extracorporeal venovenous rewarmer can achieve rapid rewarming rates (2 to 3°C [3.6 to 5.4°F]/hr), although they are slower than cardiopulmonary bypass.[77] Such a device is relatively easy to use, uses readily available technology, and probably does not require heparin. However, it needs to be assembled before patients present with hypothermia.

Hemodialysis

Hemodialysis was first described in the management of AH in 1965. [6] It is a rapid and efficient modality of rapid internal rewarming for moderate-to-severe AH, but it is rarely used in clinical practice. Core rewarming by hemodialysis has been achieved after placement of a dialysis catheter or with use of an existing shunt. Some of the potential advantages and drawbacks of cardiac bypass also apply to this procedure, although slower warming rates have been reported. A range from 0.6°C/hr to rates as high as 4.5°C/hr have been achieved with fluid warmed to 40°C. [6] For patients who have ingested a dialyzable toxin (such as barbiturates and toxic alcohols), hemodialysis can be used to both remove the toxin and rewarm the blood. In such cases its use may be appropriate.

Experimental Techniques

Ultrasonic, radiowave, and low-frequency microwave diathermy rewarming appears to be a rapid, safe, noninvasive technique with promise in animal studies. [56] [78] Frequencies of 13.6 to 40.7 MHz are typically used. However, the technique seems less effective than immersion therapy and equivalent to passive rewarming techniques in a volunteer study.[78]

Total liquid ventilation is currently being studied in animals as a method to rapidly rewarm the core using warmed oxygenated perflurocarbon. Benefits include shorter rewarming times when compared with warm humidified oxygen (1.98 ± 0.5 versus 8.61 ± 1.6 hours; $p < 0.0001$), no afterdrop phenomenon, and no increase in lactate dehydrogenase and aspartate aminotransferase.[62]

Very hot intravenous fluids (65°C [149°F]) have been used in animals with little vascular damage or hemolysis. Trials in humans undergoing burn debridement have been very successful in preventing hypothermia during operative procedures. Patients had saline heated to 60°C using modified fluid warmers infused through central venous access. Patients had no evidence of intravascular hemolysis or coagulopathy

following infusions.[79] The role of hot intravenous fluids in the management of accidental hypothermia is currently undefined.

SPECIAL SITUATIONS

Cardiac Arrest

Cardiac arrest from AH requires immediate treatment for the best chance of a successful outcome. Rapid rewarming and restoration of cardiac rhythm is essential for patients in cardiopulmonary arrest and can best be achieved by a combination of passive and multiple active core rewarming techniques. Because numerous cases of survival from hypothermic cardiac arrest with prolonged external cardiac compression exist, [3] [33] [80] thoracotomy is not mandatory but does offer some theoretical advantages, such as increased cardiac output with open-chest massage, [76] direct observation of cardiac activity, and direct warming of cardiac tissue with thoracic cavity lavage of warm fluid. Cardiopulmonary bypass is an effective technique for rapid rewarming. Blunt trauma and head trauma victims were previously not ideal candidates for cardiac bypass because of the anticoagulation requirement. However, some authors have advocated this technique using heparin-bonded tubing even in the setting of known traumatic injury.[3] A review of outcome from hypothermic cardiac arrest from one institution found that the average time from thoractomy to development of a perfusing rhythm was 38 minutes (range, 10 to 90 minutes).[3] The optimal rate of cardiac compression in hypothermia is not known, but because of decreased oxygen consumption of vital organs, the rate required in hypothermic cardiac arrest is less than that recommended in normothermic cardiac arrest. Cardiac compressions should be initiated at half the normal rate in profoundly hypothermic patients. Guidelines developed by the American Heart Association and the Wilderness Medical Society recommend that CPR should be initiated in accidental hypothermia unless any of the following conditions exist: a "do not resuscitate" (DNR) status is documented and verified, obvious lethal injuries are present, chest wall depression is impossible, any signs of life are present, or rescuers are endangered by evacuation delays and altered triage conditions. [4] [81]

The duration of CPR depends on the time required to raise the core temperature to a level at which defibrillation should be successful (i.e., >30°C [86°F]). Previously, it was recommended that patients should not receive a set of three countershocks until a core temperature above 30°C can be reached. However, there have been reports of successful defibrillation in patients with profound hypothermia with core temperatures of 25.6°C. [82] The decision to terminate resuscitative efforts remains a clinical decision. However, there are certain poor prognostic factors. Certainly, survival is unlikely in patients who persist in asystole or go from ventricular fibrillation to asystole as they are warmed past 32°C (89.6°F). Prognostic markers for patients with severe hypothermia and cardiac arrest have been proposed as contraindications to ED thoractomy and/or cardiac bypass by some authors. [3] These include elevated potassium levels above 10 mmol/L (meq/L) and pH levels below 6.5. Nonetheless, there are survival reports for patients with higher potassium levels and a pH as low as 6.51. [3] Therefore, the decision to continue resuscitative efforts should not be based solely on specific laboratory values or presenting core temperature.

Isolated reports of survival with prolonged CPR in hypothermic patients make extended efforts to resuscitate such patients reasonable. Children may be the best candidates for heroic measures.[5] Under ideal conditions, hypothermic cardiac arrest patients may reasonably be admitted to an intensive care unit for a 4- to 5-hour trial of rewarming with CPR in progress. Manual CPR should be replaced by mechanical methods if equipment is available (see Chapter 17). The oxygen-powered "thumper" has been successful during prolonged hypothermic resuscitations. Absence of responsiveness to treatment in conjunction with a highly elevated potassium level is an indication for termination of resuscitative efforts.

Airway Management

A secure functioning airway must be maintained for the hypothermic patient, just as in any critically ill patient. In mild hypothermia, heated humidified oxygen can be delivered effectively by a face mask. The hypothermic patient can be combative and uncooperative and may require arm restraints if a mask is used. For the patient with decreased sensorium who cannot reliably maintain his or her airway or the hypothermic patient who may be hypoxic, endotracheal intubation may be performed safely without the added risk of ventricular dysrhythmias.[10] The technique for endotracheal intubation depends on the specific presenting circumstances and the expertise of the operator. Once an endotracheal tube has been placed and secured, it may be used for treatment of the patient with warm humidified oxygen. There is no evidence that tracheal intubation is detrimental in the severely hypothermic patient, and should be considered if indicated for ventilation, oxygenation, or airway protection.

Acid-Base Disturbances

Acid -base disturbances are variable and can lead to metabolic acidosis from carbon dioxide retention and lactic acidosis or metabolic alkalosis resulting from decreased carbon dioxide production or hyperventilation. The interpretation of arterial blood gases in the hypothermic patient has been the cause of some confusion. Previously it was suggested that all blood gases be corrected for temperature with correlation factors. With a decrease in temperature of 1°C, the pH rises 0.015, the PCO_2 drops by 4.4%, and the PO_2 drops 7.2% compared to values that would be obtained on blood analyzed under normal conditions. Despite the conversion guide, optimal or normal values in hypothermia have not been well documented. [26] The most recent literature supports the use of uncorrected arterial blood gases to guide therapy with bicarbonate or hyperventilation. [24] [26] This approach appears appropriate to support optimal enzymatic function. A gradual correction of acid-base imbalance will allow for the increased efficiency of the bicarbonate buffering system as the body warms. Arterial pH did not correlate with patient death in the Multicenter Hypothermia Study[72] and should not be used as a prognostic guide to resuscitation.

Coagulopathies

Abnormal clotting frequently occurs in hypothermia, probably because cold inhibits the enzymatic coagulation cascade. [83] Hypothermia-induced coagulopathy does not result from

excessive clot lysis, but rather from impaired clot formation.[16] Platelet function is also impaired during hypothermia because the production of thromboxane B_2 is inhibited. Hypothermia-induced platelet aggregation (HIPA) with or without neutrophil involvement has been associated with neurological dysfunction in patients undergoing surgical procedures.[12] Hypercoagulability with risks of thromboembolism may also occur, but the main importance of cold-induced coagulopathy is in patients with coincidental trauma. Such victims often have bleeding that is difficult to control. Replacement of appropriate clotting factors and use of warm blood may limit further blood loss and worsening of hypothermia.

Trauma and Hypothermia

Clearly, there is an increased mortality in trauma patients with temperatures below 32°C (89.6°F). It is not clear if this increased mortality is actually a result of hypothermia or whether hypothermia is merely an indicator of severe injury and response to a massive transfusion of cold fluid. [16] [84] [85] Patients with severe trauma are prone to hypothermia because their injuries often expose them to environmental heat loss. Concurrent alcohol intoxication may add to the heat loss owing to the vasodilatory effects on cutaneous vasculature and prolonged cold exposure secondary to altered mental status. Severe injury victims also lose heat because of exposure during resuscitation and rapid administration of cold fluids.

The degree to which correcting the hypothermia improves outcome is unknown. Nevertheless, devices to rapidly infuse warm fluids such as the Level 1 fluid warmer (Level 1 Technologies, Rockland, MA) and the Thermostat 900 (Arrow International, Reading, PA) are frequently used to warm large volume fluid transfusions. These devices seem reasonable to prevent the hypothermia associated with massive transfusions (see Chapter 24). Their use in hypothermia not associated with severe trauma is limited by the relatively low fluid requirements of environmental exposure hypothermia.

PHARMACOTHERAPY AND MONITORING

Hypothermia alters the pharmacodynamics of various drugs. It markedly alters drug kinetics, but not enough is known about this phenomenon to define specific therapeutic guidelines. Drug administration in the hypothermic patient must be done with caution (Table 67-4). Because of the negative effects of hypothermia on both hepatic and renal metabolism, toxic levels of medications can accumulate rapidly after repeated use. Certain drugs, such as digitalis, should be avoided entirely. Sinus bradycardia and most atrial arrhythmias do not require pharmacological treatment as most resolve with rewarming. Transient ventricular dysrhythmias also do not require treatment. For those patients requiring medication for ventricular dysrhythmias, bretylium is the preferred agent, although lidocaine, magnesium, and propranolol have been used safely.[24] For severe acidosis (pH < 7.1), IV sodium bicarbonate can be used with extreme caution. Vasopressors should be used with caution, perhaps in much smaller doses than usual, because of the arrhythmogenic potential and the delayed metabolism. A review of ICU admissions for hypothermic patients found that treatment with vasoactive drugs was an independent risk factor for mortality, but this phenomenon remains poorly understood. [33] In

TABLE 67-4 -- Commonly Used Medications in Hypothermia

Clinical Situation	Medication	Dosage
Hypoglycemia	D50 W	1 mg/kg IV
Alcoholic/Malnourished	Thiamine	100 mg IV
Altered mental status	Naloxone	0.4 to 2 mg IV
Ventricular fibrillation	Bretylium*	5 mg/kg IV
	Magnesium sulfate	100 mg/kg IV

*The role of more available antidysrhythmics such as amiodarone in hypothermia remains to be determined.

animal studies, use of epinephrine impaired myocardial efficiency in cases of moderate hypothermia. [86] There also was no advantage to repeated doses of epinephrine or high-dose epinephrine in the hypothermic cardiac arrest animal models. [87] The use of inamrinone (formerly known as amrinone) has been investigated in cases of deliberate mild hypothermia. Initial results indicate that amrinone accelerates the cooling rate of core temperature potentially limiting the usefulness in management of accidental hypothermia.[88] Intravenous fluids should be slowly administered to prevent fluid overload as a result of the decreased cardiac output. In addition, fluids should be started early because most hypothermic patients have intravascular volume depletion. Dextrose 5% with normal saline has been advocated as the ideal initial resuscitation fluid. [50] [56] Potassium should be avoided until electrolytes are measured and normal renal function is confirmed.

Placement of a Swan-Ganz catheter and close monitoring of urinary output may assist in the fluid management of severely hypothermic patients. The risks of precipitating ventricular fibrillation should be weighed against the potential benefits of the Swan-Ganz catheter.

Elevation of creatine phosphokinase in hypothermic patients may indicate rhabdomyolysis, and careful monitoring of renal function is essential. Aggressive fluid replacement may prevent the development of renal failure.

Finally it should be emphasized that hypothermic patients exhibit a "physiologic" (and probably somewhat protective) hypotension, hypoventilation, depressed mental status, and bradycardia, the extent of which depends on the core temperature. This observation prohibits precise recommendation on the indications and use of medications, intubation, CPR, and other resuscitative interventions that are better defined in the normothermic patient. Hypothermic patients who present with a blood pressure, respiratory rate, or mental status that would prognosticate certain morbidity in normothermic patients may recover with minimal intervention to their normal pre-hypothermic state. *The clinician should avoid aggressive therapies or medications that are aimed at providing the hypothermic patient with vital signs that would be desirable in the normothermic patient but which may be supraphysiologic in the hypothermic patient.*

FROSTBITE

Hypothermic patients frequently suffer other forms of cold-related injuries in addition to their systemic hypothermia. The mildest form of frostbite is termed *frostnip*, a condition that involves only the skin, sparing the subcutaneous tissues. The skin is blanched and numb, but the injury is immediately reversible with no permanent sequelae if the area is quickly

rewarmed. Rapid rewarming should be done in a water bath at 40° to 42°C. Frostnip occurs most frequently on the distal extremities, the nose and ears. Nonfreezing temperatures also produce *trenchfoot*, an intermediate step in the progression to true frostbite. Trenchfoot is the result of prolonged immersion. Treatment involves rewarming followed by dry dressings.[89] [90]

In frostbite, the body parts most susceptible are those farthest away from the body's core: the hands, feet, earlobes, and nose. Exposure of the fingers to severe cold leads to cold-induced vasodilation (CIVD). [91] [92] Apical structures rich with AVAs can shunt blood flow away from tissues.

The pathophysiology of frostbite includes three pathways of tissue freezing: (1) through the extracellular formation of ice crystals, (2) hypoxia as a result of cold-induced local vasoconstriction, and (3) the release of inflammatory mediators. These pathways can and often do occur simultaneously, intensifying tissue damage. At the early stages of frostbite, the "hunting reaction" is observed whereby the body alternates between periods of vasoconstriction and vasodilation. As the temperature continues to decrease the reaction stops and vasoconstriction persists. [31] [90] Cold also increases blood viscosity, promotes vasospasm, and precipitates microthrombus formation. The release of inflammatory mediators prostaglandins PGF2 and thromboxane A2 that are found in blister fluid cause further vasoconstriction leading to cell death. The release of these mediators peaks during rewarming, and cycles of recurrent freezing and rewarming only increase their tissue levels. *Rewarming must be avoided until refreezing can be prevented.*

The clinical signs and symptoms of frostbite vary according to the degree of injury. Although useful clinically, the degree classification does not predict the extent of further tissue damage.[31] [83] The appearance of the affected extremity will depend on the extent of the frostbite. In superficial frostbite, the affected extremity appears pale, waxy, and numb; has poor capillary refill; and is very painful on rewarming. In deeper frostbite, the affected extremity is hard, solid, and blanched. Hemorrhagic blisters may be present. Initially there is no pain or feeling in the frostbitten extremity. After rewarming, the affected area develops severe edema and blistering, eventually exhibiting dry gangrene and mummification, leading to tissue sloughing.

Favorable prognostic signs for frostbite include intact sensation, normal color, warm tissues, early appearance of clear blisters, and edema. Unfavorable prognostic signs include no sensation, cold, cyanotic appearance, white "frozen" appearance, late appearance of hemorrhagic or dark blisters, and absence of edema. [93] Based on early bone scans and retrospective studies, researchers from France have proposed a new classification for predicting frostbite outcomes on day 0. [94] Four degrees of severity are defined. With first degree, there is complete recovery. Second degree often leads to soft tissue amputation. With third degree there is the need for bone amputation, and with fourth degree, there are systemic effects.[94]

Rapid rewarming is the treatment of choice for frostbite.[89] [90] The aim is to limit the length of time the tissue remains in the frozen state. The most practical way to rewarm an extremity is to totally immerse the area in warm water at 40° to 42°C for 15 to 30 minutes. The affected area should be carefully protected to ensure that the tissue is not additionally injured through contact with the sides or rim of the container. After thawing, the area should be meticulously protected from injury. An extremity should be elevated and cotton or gauze placed between the toes/fingers to limit maceration. White or clear blisters should be debrided. Hemorrhagic or dark blisters should be left intact as disruption may cause damage to the vascular supply and viable tissue.

The use of topical aloe vera (a thromboxane inhibitor) and systemic antiprostaglandins (such as ibuprofen) may be helpful. The use of semiocclusive dressings has shown promising results for management of deep frostbite injuries of the fingertips.[95] Tetanus prophylaxis should be provided. Adjuvant therapies involving the use of heparin or low-molecular-weight heparin, warfarin, vasodilators, corticosteroids, or immediate surgical sympathectomy have failed to improve outcomes. There has been mixed success with the use of hyperbaric oxygen and thrombolytics.[96]

Agents that can inhibit the formation of free radicals are promising. These agents include superoxide dismutase, PGE1 analogues, and drugs containing antiplatelet activity such as pentoxifylline.[31] [90] The use of antibiotics is controversial, although some authors advocate agents with staphylococcus/streptococcus coverage (e.g., cephalosporins, pencillins). Debridement of tissue should be avoided in the ED. Patients should be given analgesics (IV opioids) as needed.

COLD WATER IMMERSION/SUBMERSION

One of the leading causes of hypothermia remains cold water immersion/submersion. [97] In one retrospective review of accidental hypothermia cases in a three-year period, submersion hypothermia accounted for the greatest number of cases. [98] Unlike in cases of AH secondary to cold exposure, risk factors (both internal and external) are harder to identify secondary to the high mortality from drowning. [83] Studies have shown that at cold water temperatures (8°C), core cooling occurs at slower rates in persons with increased body mass and subcutaneous fat, and at faster rates when there is increased voluntary activity (e.g., treading water). Risk factors for submersion hypothermia include impaired performance and initial cardiorespiratory response to immersion. A study in healthy volunteers found that swimming efficiency and length of stroke decreases while rate of stroke and swim angle increases as the water temperature drops. [99]

The body's response to cold-water immersion (head-out) has been previously described as occurring in three phases. [55] The initial phase involves the "cold-shock response," which typically occurs within the first 4 to 6 minutes. Signs include peripheral vasoconstriction, gasp reflex, hyperventilation, and tachycardia. At this stage, there is a higher incidence of sudden death resulting from hypocapnia, inability to breathhold, and increased cardiac output. [55] Following the initial cold shock response, the body undergoes profound cooling of the peripheral tissues. The peripheral cooling tends to be the greatest in the hands leading to incoordination and grasping difficulties. [55] In prolonged immersion in cold water, heat is lost from the body quicker than it is produced, thus predisposing to hypothermia. [100]

In cases of cold-water submersion, researchers have found that rapid cooling is protective against neurological impairment and increases chances of survival. There are numerous reports in the literature of survival following cold-water submersion in children, but very few reports in adults. There are reports of survival following up to 66 minutes

of cold water submersion. [101] Recently, there was a case report of survival in an elderly male following 22 minutes of submersion. [102] Overall, children tend to have a better prognosis because of the presence of the mammalian dive reflex, and a greater body surface area to mass ratio that allows for more rapid cooling. Orlowski identified five poor prognostic factors for near drowning in pediatric patients: (1) maximum submersion time >5 minutes, (2) comatose upon arrival to ED, (3) arterial blood gas pH <7.10, (4) age younger than 3 years, and (5) resuscitation not attempted for at least 10 minutes following rescue. [103] [104] Adults tend to have higher mortality rates because of the following: (1) the lack of the mammalian dive reflex and (2) slower rates of temperature cooling secondary to lower body surface area to mass ratios in adults as compared to children.

Various mechanisms for brain and body cooling during submersion hypothermia have been described including the mammalian dive reflex, cold-induced changes in neurotransmitter release, and water ventilation. [103] The mammalian dive reflex prevents or delays aspiration or ventilation until the body has cooled to a point where hypothermia protection occurs. Much attention has focused on the theory of water ventilation as a key component of accelerated brain cooling. Animal studies comparing immersed (head-out) and submersed dogs found that cooling rates were faster in the submersed dogs than in the immersed dogs. The submersed dogs cooled by convective heat exchange in the lungs whereas the immersed dogs cooled by surface conduction only. Laboratory data obtained following the submersion indicated that there was indeed ventilation exchange in the water. [101] The body also undergoes a relative bradycardia as another protective measure. Bradycardia is inversely proportional to the water temperature with heart rates reaching 18 bpm in 10°C water. [103]

Many authors advocate therapies aimed at symptoms resulting from near drowning rather than severe hypothermia because in fatal cases of submersion, death occurs too rapidly for hypothermia to be a significant contributor. Complications of near drowning include pneumonia, lung edema, hemorrhagic pancreatitis, and skin edema. [83]

CONCLUSION

Mortality rates from AH are decreasing due to increased recognition and advanced therapy. Caution should be used when extrapolating published data obtained in adults to children.[46] With the exception of severe hypothermia, the prognosis mostly correlates with the presence or absence of underlying disease states. Studies have shown that the prognosis is excellent in patients in whom no hypoxic event precedes hypothermia and no serious underlying disease states exist. [105] Previously healthy individuals usually have a full recovery with mortality rates <5%, whereas patients with coexisting medical illnesses have reported mortality rates of >50%. [45]

As a general guideline, one should take a conservative approach to rewarming the stable hypothermic patient, with avoidance of overtreatment and the selective and careful use of invasive monitoring. The patient's "physiologic" hypotension, hypoventilation, and bradycardia should be evaluated with regard to that which is expected for the core temperature.

In moderate hypothermia, underlying problems should be sought, passive rewarming and basic support started, and less invasive core rewarming begun. This approach should include mask ventilation with warm humidified air or oxygen in the conscious patient and intubation and ventilation in the unconscious patient. In selected patients, gastric or peritoneal lavage with warm fluid may be considered.

For severely hypothermic, *unstable* patients, cardiac bypass and thoracic lavage may offer additional benefits, including rapid warming rates and direct heart warming. The benefits should be weighed against the institutional capabilities, time, expense, and the danger of complications that these procedures entail.

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Chapter 68 - Procedures Pertaining to Hyperthermia

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Human epidemics of heat-related illness have been well documented historically. [1] [2] A Roman army was decimated by heat in 24 BC, and King Edward's heavily armored crusaders were defeated by "heat and fever" during the final battle of the Holy Land. In Peking, 11,000 residents died during a heat wave in 1743. More than 1000 heat-related deaths occurred during a pilgrimage to Mecca in the early 1960s. In the United States in 1980, 1983, and 1988 (years with prolonged heat waves), 1700, 556, and 454 deaths, respectively, were attributed to heat exposure. [3] In the 1990s, there were a significant number of hyperthermia-related fatalities in major metropolitan cities across the country, including Chicago and Los Angeles. [1] [4] [5] [6] [7]

In contrast, malignant hyperthermia and neuroleptic malignant syndrome (NMS) have been recognized and described only since the 1960s. [8] [9] [10] These conditions are largely iatrogenic and are most commonly triggered by modern pharmacologic therapy. In addition, the incidence of severe hyperthermic conditions induced by psychostimulant drugs of abuse, such as cocaine and amphetamine derivatives, is on the rise. [11]

Heatstroke remains a common clinical problem with significant morbidity and mortality. Each year, 175 to 200 people on average die from heat-related illnesses. [12] As the weather patterns continue to change with a trend toward hot and humid summers, the number of individuals adversely affected by the heat continues to rise. A variety of cooling techniques have been advocated since World War II. Although some cooling techniques have been compared in controlled human and animal models of heatstroke, practice decisions are not solely based on the theoretical rate of cooling. Other important factors include the ease of use, rapidity of initiation, and safety.

Before considering the various cooling techniques, it is essential that the underlying disorder of hyperthermia be clearly understood. Heat illness presents a spectrum of disease ranging from mild heat exhaustion (primarily a volume loss disorder) to severe heatstroke (with the thermal-related multiorgan-dysfunction syndrome). The latter includes disorders such as malignant hyperthermia and NMS. Treatment of this spectrum of disease requires a discriminating approach, including supportive care only for heat exhaustion and rapid cooling for heatstroke. Malignant hyperthermia requires specific pharmacologic therapy (e.g., dantrolene) in addition to cooling measures. A brief discussion of hyperthermic disorders is therefore necessary before describing cooling techniques.

NORMAL THERMOREGULATION

Body temperature typically follows a diurnal pattern, increasing from about 36°C in the early morning to 37.5°C in the late afternoon, and reflects the balance between heat production and heat dissipation. [13] [14] Heat is produced as a by product of all metabolic processes and when ambient temperatures exceed the body temperature. The body temperature increases when the rate of heat production exceeds the rate of heat dissipation. In response to rising core temperature, the thermal center (located in the preoptic nucleus of the anterior hypothalamus) activates efferent fibers of the autonomic nervous system to produce vasodilation and increase the rate of sweating. Vasodilation dissipates heat by convection, and sweat dissipates heat by evaporation.

Hyperthermia occurs when thermoregulatory mechanisms are overwhelmed by excessive metabolic production of heat, excessive environmental heat, or impaired heat dissipation. In contrast, fever occurs when the hypothalamic setpoint is increased by the action of circulating pyrogenic cytokines, causing peripheral mechanisms to conserve and generate heat until the body temperature rises to the elevated setpoint. Hyperthermia and fever cannot be differentiated clinically on the basis of the magnitude of temperature or on the pattern of its changes. [14] [15] [16]

In the ensuing discussion, temperatures are given in degrees Celsius (centigrade). A temperature conversion scale is provided in the previous chapter (see Fig. 67-2).

TYPES OF HYPERTHERMIA

Mild Heat Illness

Heat cramps and heat exhaustion are induced by a hot environment. [17] [18] [19] In general, the body's heat dissipation mechanisms are able to keep up with heat production and absorption in these disorders. Symptoms are largely due to the mechanisms used by the body to dissipate heat, and body temperatures remain at or near normal. Rapid cooling techniques are *not* required, and supportive care and hydration in a cool environment are usually adequate therapy.

Heat cramps are intensely painful but generally benign, involuntary skeletal muscle spasms (most often in the calves, hamstring, or quadriceps muscles). The pain may also be in the arms and back. The cramps may be severe and prolonged and rarely may lead to rhabdomyolysis. Heat cramps occur after strenuous exercise or heavy labor in a hot environment. Previously heat cramps were considered the result of dehydration associated with significant sodium chloride losses; however, recent clinical observations have not proven this to be accurate. In fact, cramps can occur at rest or during exercise under any environmental conditions. Cramps appear to be specific neither to exercise nor to exercise in heat. [20] The most recent theory suggests that cramps may be the result of alterations in the spinal neural reflex activity triggered by fatigue in susceptible individuals, underscoring the importance of adequate stretching before and after exercise. There are no controlled clinical trials evaluating the efficacy of electrolyte replacement, quinine, or other medications. [20] Nonetheless, commonly followed practices of rest in a cool environment and vigorous oral fluid replacement with isotonic solutions are usually adequate therapy, but in some cases IV saline is required. A common mistake is to rely on thirst to indicate dehydration. Severe cramping may be surprisingly resistant to narcotics in the absence of adequate fluid replacement.

Heat exhaustion, commonly referred to as heat syncope, is a poorly defined syndrome with a spectrum of nonspecific symptoms that *occur after heat exposure*. Malaise, influenza-like symptoms, orthostasis, dehydration, nausea, headache,

and collapse may all occur. Heat exhaustion is considered the result of dehydration-induced heat retention that is not severe enough to cause heatstroke. [21] As distinct from the more severe heat disorders, mental status is normal and body temperature is normal or mildly elevated. There does not appear to be any thermoregulatory failure in heat exhaustion. Rehydration (usually IV), rest, and supportive care in a cool environment are adequate therapy for heat exhaustion. [22] Theories as to the etiology of heat exhaustion include sudden onset of postural hypotension after cessation of exercise (owing to a precipitous fall in central rather than circulating blood volume). Some authors advocate cooling and elevation of the patients' legs or by placing them in the head-down position between the knees in order to decrease skin blood flow and increase venous blood flow to the heart. [20] [21] Recovery is usually evident within a few minutes to hours. Occasionally heat exhaustion is accompanied by heat cramps, presenting a confusing scenario if the diagnosis is not suspected. Rapid cooling techniques are not usually required, but patients should be observed for progression to heatstroke as heat exhaustion and heatstroke are a continuum of one disease process. [23]

Heatstroke

When the body's normal heat dissipation mechanisms are overwhelmed, core temperature elevation and heatstroke rapidly ensue. Heatstroke represents a true state of thermoregulatory failure. [12] [16] [24] [25] Two forms of heatstroke are described in the literature. *Classic (nonexertional) heatstroke* usually occurs during summer heat waves. Poor people, urban elderly people, infants, and people with impaired mobility are at greatest risk. [18] [21] [26] [27] Dehydration, lack of air conditioning, obesity, neurologic disorders, hyperthyroidism, marginal cardiovascular status, impaired mentation, and medications that interfere with heat dissipation (e.g., phenothiazines, diuretics, anticholinergics) predispose this population to heatstroke. *Exertional heatstroke,* a consequence of strenuous physical activity, usually afflicts a younger segment of the population. Highly motivated, poorly acclimatized, or unconditioned athletes and military recruits are common victims, as are individuals who perform heavy physical labor in hot, humid conditions. [28] [29] In exertional heatstroke, the risk appears to be greatest in those individuals performing high-intensity exercise for relatively short durations. [20] A retrospective review of long-distance cyclists participating in the California AIDS Ride found that as the number of chronic medical illnesses increased so did the risk of developing an exertional heat-related illness. Human immunodeficiency virus seropositivity alone was not associated with an increased risk of exertional heat-related illness. [30]

The degree of hyperthermia necessary to produce heatstroke in humans is unknown. In tissue culture cells, thermal injury is observed in the range of 40 to 45°C. Studies of hyperthermia in cancer therapy reveal that tissue sensitivity to heat is increased by relative hypoxia, ischemia, and acidosis. [31] [32] [33] Studies suggest that the critical thermal maximum in humans is a temperature of 41.6°C to 42°C for 45 minutes to 8 hours. Cellular necrosis and the destruction of all cellular structures occurs in <5 minutes with body temperatures >49°C. [25]

The specific tissue pathophysiology of heat stroke is complex and multifactorial. One current theory is that thermoregulatory failure is coupled with an exaggerated acute phase response that involves direct cytotoxity of heat, and inflammatory and coagulation responses of the host. This process involves the interplay and modulation of inflammatory cytokines, such as tumor necrosis factor, numerous interleukins, and interferons. The initiating event may be splanchnic hypoperfusion resulting in alterations in immunologic and barrier functions of the intestines. The end result is multiorgan dysfunction, including disseminated intravascular coagulation. [25]

The key clinical findings in the diagnosis of heatstroke are (1) a history of heat stress or exposure, (2) a rectal temperature higher than 40°C, and (3) central nervous system (CNS) dysfunction (e.g., altered mental status, disorientation, stupor, seizures, coma). The cerebellum is most sensitive to heat, and ataxia may be an early clue. Although anhidrosis is traditionally described as a classic sign of heatstroke, investigations have demonstrated that cessation of sweating is only a latent sign. Failure to consider the diagnosis of heatstroke in a diaphoretic patient with mental status changes could prove disastrous. [21] [34] All patients with heatstroke exhibit hyperventilation and tachycardia. Hypotension is common. In *nonexertional* heatstroke, common laboratory abnormalities include hypophosphatemia, hypercalcemia, hypokalemia, and a respiratory alkalosis. *Exertional* heatstroke manifests hyperphosphatemia, hypocalcemia, and hypokalemia, usually seen after cooling. The acid-base disturbance is both a respiratory alkalosis and lactic acidosis.

The sequelae of heatstroke are caused by thermal damage to multiple organ systems. [12] After a hyperthermic event, tissue injury continues. [35] Delirium, seizures, and coma can result from direct effects of heat on the CNS. Cardiovascular collapse results from dehydration, maximal cutaneous vasodilation, and direct heat-induced myocardial depression. Coagulopathies and liver dysfunction (elevated levels of bilirubin and transaminases) occur as consequences of thermal breakdown and consumption of serum proteins, as well as direct heat damage to hepatic cells. Children often demonstrate diarrhea. Renal failure can result from myoglobinuria (related to rhabdomyolysis) and acute tubular necrosis. [24] [27] In addition to encephalopathy, rhabdomyolysis, acute renal failure, and DIC, other manifestations of multi-organ dysfunction include acute respiratory distress syndrome, intestinal ischemia or infarction, and pancreatic injury.

Rodent studies of hyperthermic events demonstrated significant gastrointestinal changes. In rats successfully cooled to normothermia, delayed secondary deterioration occurred with massive GI bleeding. [35] Metabolic acidosis is the primary acid-base alteration observed in heatstroke with the prevalence increasing with the degree of hyperthermia. [36]

The treatment of these sequelae in acute heatstroke does not differ from that in other disorders, with the sole exception that rapid cooling is necessary to prevent further damage and reverse heat stress. The more rapidly the rectal temperature is reduced to 38°C, the better the prognosis. [16] [18] [24] Importantly, the human body tolerates hyperthermia poorly. Unlike patients with hypothermia, in whom slow, gentle rewarming and supportive care often result in a favorable outcome, victims of severe heatstroke must be aggressively treated with measures designed to rapidly lower the core temperature. Studies investigating precooling techniques before exertion to avoid heatstroke have been relatively unsuccessful in attenuating increases in core body temperature and are not recommended. [37] In contrast, whole body precooling is shown to increase overall exercise endurance. [38]

Malignant Hyperthermia

Malignant hyperthermia (MH) is a pharmacogenetic disease, which predisposes the patient to the triggering of a life-threatening, hypermetabolic syndrome. [39] MH results from a rare inherited autosomal dominant abnormality of the skeletal muscle membrane with an incidence of 1:50,000 in adults. [8] [10] [40] In response to certain stresses or drugs (Table 68-1), patients with this disorder sustain a massive efflux of calcium from skeletal muscle sarcoplasmic reticulum, resulting in contraction of the sarcomeres; skeletal muscle rigidity; increased skeletal muscle metabolism, elevated serum creatine kinase levels, and heat production; and, finally, systemic hyperthermia.[41] [42] Hyperthermia is a late development occurring after rigidity has been present for some time and the body's normal heat dissipation mechanisms have been overwhelmed. The earliest signs of malignant hyperthermia are increased CO_2 production, muscle rigidity, and tachycardia. [42] [43] Cardiac output and cutaneous blood flow also increase to maximize heat loss. Malignant hyperthermia is diagnosed based on the clinical triad of (1) exposure to an agent or stress known to trigger the condition, (2) skeletal muscle rigidity, and (3) hyperthermia.

Malignant hyperthermia is usually encountered in the operating room while patients are undergoing general anesthesia, particularly with halogenated inhalation agents and depolarizing muscle relaxants. Heat production in the anesthetized patient can be profound with as much as a fivefold increase in oxygen consumption. [39] However, cases of malignant hyperthermia may be encountered anywhere general anesthetics or neuromuscular blocking agents are used. [44]

As with heatstroke, treatment of malignant hyperthermia requires rapid cooling and supportive care for the sequelae described previously. Unlike heatstroke, which does not respond to pharmacologic agents, malignant hyperthermia requires specific pharmacologic therapy to stop excess heat production by skeletal muscle. Dantrolene sodium induces muscle relaxation in malignant hyperthermia by blocking calcium release from muscle cell sarcoplasmic reticulum. [42] [43] In all cases of malignant hyperthermia, the inciting stimulus (see Table 68-1) should be discontinued immediately and dantrolene therapy administered. Although different treatment regimens exist, the editors recommend an aggressive dosing (3 mg/kg IV every 10 minutes for three doses) until the episode resolves. Procainamide has been used successfully when dantrolene is unavailable. [45]

TABLE 68-1 -- Triggers for Malignant Hyperthermia

Drugs	Conditions
Halothane	Heat stress
Methoxyflurane	Vigorous exercise
Enflurane	(?)Emotional stress
Diethyl ether	
Cyclopropane	
Succinylcholine	
Tubocurarine	
Lidocaine	
Mepivacaine	
Isoflurane	
Ketamine	
Trichloroethylene	
Chloroform	
(?)Gallamine	
(?)Nitrous oxide	

It has been suggested that dantrolene administration speeds cooling of heatstroke victims by reducing skeletal muscle heat production. [46] However, in a porcine model of heatstroke, dantrolene administration resulted in faster cooling rates than those seen with conventional cooling methods but did not appear to be superior. [47] Although one small study of exercise-induced heatstroke in humans during a religious pilgrimage suggested that dantrolene may speed cooling rates in exertional heatstroke,[48] a larger controlled trial in a similar setting found no benefit. [49] Dantrolene administration is best reserved for patients with clinical muscle rigidity or suspected malignant hyperthermia. Routine use of this drug in heatstroke patients is not recommended. [50]

New and promising treatments for malignant hyperthermia are being investigated. Researchers have discovered mutations in the gene coding for the ryanodine receptor calcium release channel (RyRI) in families with MH, which may be the functional basis for MH. Recent studies have examined the effects of MH mutations on the sensitivity of the RyRI to drugs and endogenous channel effectors including Ca^{2+} and calmodulin.[39] [51]

Neuroleptic Malignant Syndrome

First described in the late 1960s, NMS is characterized by fever, muscle rigidity, altered level of consciousness, and autonomic instability. [9] Mortality from NMS is estimated at 20% of patients who develop the condition.[52] This idiosyncratic disorder follows the therapeutic use of neuroleptic drugs, including phenothiazines, butyrophenones, thioxanthenes, lithium, and tricyclic antidepressants. The reaction is triggered by blockade of dopaminergic receptors, resulting in spasticity of skeletal muscle, which generates excessive heat and impairs hypothalamic thermoregulation and heat dissipation. [53] Muscle rigidity, described as "lead-pipe" rigidity in its most severe form,[54] can manifest as oculogyric crisis, dyskinesia, akinesia, dysphagia, dysarthria, or opisthotonos. NMS occurs in 0.2% of patients who receive neuroleptic agents either chronically or acutely. Haloperidol appears to be the most common offending agent. Temperatures can exceed 42°C. Initial agitation often progresses to stupor and coma. Catatonia and mutism may also be present. Autonomic instability manifests as tachycardia, labile blood pressures, sweating, and incontinence. Ventilations are impaired by chest wall rigidity.

This syndrome is not secondary to a drug overdose per se, but more likely to occur at the initiation of or after an increase in neuroleptic dosage. It may also occur if antiparkinsonian drugs are suddenly discontinued. [52] NMS resembles malignant hyperthermia but usually takes considerably longer to develop (2 to 3 days) and lasts longer (5 to 10 days) after the inciting drug is discontinued. The syndrome may be misinterpreted as a worsening of the underlying psychiatric disorder, drug intoxication (e.g., cocaine, amphetamines), a severe dystonic reaction, tetanus, or a variety of CNS infections. In addition to agents with increased dopaminergic blocking activity, other risk factors for NMS include: dehydration, prior history of dystonia, catatonia, or agitation, and iron deficiency. [45] The mortality rate is high, and respiratory failure, renal failure, cardiovascular collapse, or thromboembolic disease usually cause death. Unlike malignant hyperthermia, NMS is thought to be caused by a central disorder of thermoregulation.

Treatment of severe NMS (i.e., hypotension, hyperthermia, marked rigidity) closely follows that of malignant hyperthermia, except that therapy must be maintained for several days until symptoms resolve. Discontinuation of the triggering agent, rapid cooling, benzodiazepines, and supportive treatment for ensuing organ failure remain the cornerstone of therapy.[53] Although the effects are not immediate, pharmaceutical therapy is directed at overriding the dopaminergic blockade caused by offending neuroleptic agents or dopamine depletion resulting from the cessation of antiparkinsonian medications. There are reports of successful treatment of NMS using subcutaneous apomorphine monotherapy.[55] As with malignant hyperthermia, dantrolene (2 to 3 mg/kg IV every 10 minutes for 3 doses) can be given to treat NMS-induced muscle rigidity. The beneficial response stems not only from the effects at the sarcoplasmic reticulum of skeletal muscles, but also from central dopamine metabolism of calcium in the CNS. In addition, bromocriptine, a central dopamine agonist, is reported to be efficacious in NMS therapy at doses of 2.5 to 10

mg 3 times a day.[53] Although both agents have been noted to reduce the duration of hyperthermia, there have been mixed results of success using bromocriptine and dantrolene.[53] [58] [59]

A more recently described disorder that is often confused with NMS is the *serotonin syndrome*.[60] This syndrome involves the newer antidepressants (fluoxetine, paroxetine, citalopram, fluvoxamine, venlafaxine, and sertraline), [45] [58] [59] [60] [61] [62] which are selective serotonin reuptake inhibitors (SSRIs). These drugs can adversely react with other stronger serotonin receptor agents such as monoamine oxidase inhibitors (MAOIs) and non-selective serotonin reuptake inhibitors (clomipramine and tricyclic antidepressants) to induce a clinical presentation similar to that of NMS, only milder. Serotonin syndrome classically occurs when two or more drugs that interfere with serotonin metabolism act synergistically on the 5-HT $_{1A}$ receptor leading to overstimulation.[45] [60] Drugs that act at any of the other serotonin receptors are not likely to produce the syndrome.[45] The range of symptoms varies from mild gastrointestinal upset, insomnia, and agitation to the most severe symptoms including: muscle spasms, seizures, ataxia, rhabdomyolysis, and autonomic instability. In a review, Mills [62] found that muscle rigidity, more commonly involving the lower extremities, was present in 50% of cases. Treatment is primarily supportive in milder cases and consists of prompt recognition and withdrawal of the offending agent. Most cases resolve spontaneously within 24 hours. For the more severe cases, aggressive ICU management is warranted to prevent renal failure and death. The drug cyproheptadine (Periactin) in doses of 4 to 8 mg PO has shown promise in managing the agitation often seen with severe cases.[45] Cyproheptadine is an antihistamine with antiserotonergic properties. There has been limited success using benzodiazepines and ß-blockers in these patients, but various benzodiazepines are commonly given for agitation.[45]

Hyperthermia and Psychostimulant Overdose

As mentioned earlier, the recognized incidence of hyperthermia induced by sympathomimetic psychostimulant drugs of abuse is on the rise. The offending agents most commonly described are cocaine, phencyclidine, amphetamine, and the amphetamine derivatives such as 3,4-methylenedioxymethamphetamine (MDMA; "Ecstasy") and 3,4-methylenedioxyethamphetamine (MDEA; "Eve").[63] [64] A number of studies have looked specifically at the club drug MDMA and its impairment of heat dissipation.[11] Animal studies in rats have suggested that MDMA-induced hyperthermia results not from MDMA-induced 5-HT release, but from an increased release of dopamine acting at D1 receptors suggesting a future role for use of dopamine antagonists in clinical treatment.[65]

Hyperthermia is a common feature of these potentially severe to lethal poisonings with sympathomimetic psychostimulant drugs and may be the primary cause of fatality in many cases.[63] Because of the nonlinear pharmokinetics of MDMA and GHB, it is difficult to estimate a dose-response relationship.[63] Some have applied a pathophysiologic model of exertional heatstroke or NMS to profound cocaine intoxication.[66] [67] In addition to profound hyperthermia (>42°C), acute rhabdomyolysis, severe metabolic acidosis, disseminated intravascular coagulation, psychiatric and cognitive sequelae, renal failure, coma, seizures, and death have been described in these patients.[63] [64] [65] [66] [67] As demonstrated by Roberts and colleagues, even a patient with a core temperature of 114°F owing to acute cocaine intoxication may survive with aggressive cooling methods.[68] Treatment requires prompt recognition, maintenance of adequate hydration, rapid cooling (as outlined later), correction of metabolic acidosis, and the *aggressive use of sedative or paralyzing agents, or both, to control agitation*. Importantly, the longer that psychostimulant-overdosed patients remain hyperthermic, the higher their morbidity and mortality rates. Sudden unexpected death in a previously healthy individual is not uncommon if this syndrome is not aggressively treated. Agitation and seizures must be chemically controlled, as they lead to continued generation of heat and muscle injury. Physical restraint, without the use of chemical restraint, has been associated with increased mortality. Therefore, very liberal doses of benzodiazepines are recommended.[69] [70] [71] There is no maximum dose of benzodiazepines. Standard doses are generally ineffective and as much as 500 to 2000 mg of diazepam may be required to gain control of the patient. By the time such doses are required, however, muscular paralysis should have been instituted. Some have advocated the use of bromocriptine[71] and dantrolene[72] as for malignant hyperthermia and NMS, but their efficacy in the setting of drug-associated hyperthermia remains controversial.

Hemorrhagic Shock and Encephalopathy Syndrome

The condition of hemorrhagic shock and encephalopathy (HSE) in children (mainly infants, but some older children) resembles heatstroke in adults. The full-blown syndrome includes hyperthermia, coagulopathy, encephalopathy, and renal and hepatic dysfunction.[73] [74] [75] Although there may be an association with concurrent viral illness, the condition generally follows a temperature elevation, which may be triggered by the "bundling" of a child with a low-grade fever. Therapy is largely supportive and includes volume replacement and rapid cooling of the hyperthermic child while sources of bacterial infection are sought and treated.

COOLING TECHNIQUES

The treatment of heat cramps, heat exhaustion, and other forms of hyperthermia are discussed earlier. The therapeutic

objectives in patients with heatstroke are immediate cooling, the maintenance of adequate cardiorespiratory function, and support of all organ system functions. The basic function of all cooling measures is to transfer heat from the core, to the skin, to the environment, without compromising other physiologic functions. Cooling occurs via internal and external conduction techniques, and by convection and evaporation techniques. There have been no controlled studies comparing the effects of various commonly used techniques on cooling times or outcome in humans with heatstroke. Therefore, there are no totally agreed upon standards of care for empiric therapies that intuitively seem reasonable. Various techniques are discussed later, but the clinician should use the easiest and most logical technique as dictated by the specific clinical scenario and available logistical support.

General Considerations

Heatstroke mortality is proportional to the magnitude and duration of thermal stress measured in degree-minutes. [76] *Delay in cooling may represent the single most important factor leading to death or residual disability in those who survive.*[24] In addition, advanced age and underlying disease states are significant contributing factors.[12] [18] [19] Residual brain damage may occur in as many as 20% of survivors despite optimal treatment. The overall mortality rate may approach 50%.

Many exertional heatstroke victims are volume-depleted and may present with hypotension. As a result, initial stabilization with cooled (room temperature) IV fluids and correction of electrolyte abnormalities are valuable in the hypotensive patient. Traditional sources recommend a rate of 1200 mL over the first 4 hours, [77] [78] whereas others advise a 2 L bolus over the first hour, with an additional 1 L/hour for the following 3 hours. [78] Seraj and colleagues[79] have challenged this more aggressive recommendation. In their study of pilgrims who suffered heatstroke, 65% had a normal or above normal central venous pressure (CVP) measurement on arrival. They found that an average of 1 L of saline was sufficient to normalize the CVP during the cooling period in their patients, who had a mean age of 55 years (range, 31 to 80 years). Hence, fluid resuscitation should be monitored carefully in older patients to avoid pulmonary edema.

For heatstroke, there are no pharmacologic agents to accelerate cooling that are effective or that are associated with decreased morbidity or mortality. Specifically, dantrolene sodium is ineffective for heatstroke unassociated with malignant hyperthermia. Even in the latter setting, dantrolene sodium is an *adjunct* to direct cooling techniques.

Regarding antipyretics, *there is no known benefit for either salicylates or acetaminophen in the setting of heatstroke,* as their efficacy depends on a normally functioning hypothalamus. In addition, overzealous use of acetaminophen could theoretically potentiate hepatic damage, and salicylates may promote bleeding tendencies.[15] [80] A study comparing acetaminophen and physical cooling methods found that in patients treated with antipyretics only, the mean body temperature increased by 0.2°C on average.[81] The role of immunomodulators, such as interleukin receptor antagonists, antibodies to endotoxin, or corticosteroids is unproven.

Given that rapid cooling is accepted as the cornerstone of effective heatstroke therapy, the clinician must choose which cooling technique to use. Studies in animal models are based on the assumption that the fastest cooling technique is the best. In clinical patient care, other factors will also influence the choice of technique. Patient access, monitoring, safety, ease of use, and availability are all considerations, in addition to speed of cooling. A technique that may not be the most rapid but allows easy patient access and is readily available may be preferable to more cumbersome (albeit more rapid, once established) cooling techniques in some clinical settings.

The cooling rates achieved in various human and animal studies of heatstroke are summarized in Table 68-2 . As experimental subjects and techniques vary, it is not surprising that reported cooling rates show considerable variation. The relative advantages and disadvantages of various cooling techniques are outlined in Table 68-3 .

In addition to the cooling procedures outlined later, it is imperative that the clinician use judicious sedation and/or muscle paralysis to control agitation, suppress shivering, reduce energy expenditures, and to make the patient receptive to sometimes unpleasant therapies. [15] [21] [24] In general, IV benzodiazepines are the easiest and safest first-line drugs used for sedation.

Indications for Rapid Cooling

Rapid cooling should be instituted as soon as the diagnosis of heatstroke (rectal temperature >40°C, altered mental status, history of heat stress or exposure) is made. Rapid cooling is also indicated for the treatment of malignant hyperthermia and NMS but should be instituted concurrently with discontinuation of the triggering agent or drug and administration of dantrolene. Because studies show that the degree of organ damage correlates with the degree and duration of temperature elevation above 40°C, a reasonable clinical goal is to reduce the temperature to below 40°C within 30 minutes to an hour of the start of therapy. [12] [16] [24] There is no evidence to support a specific temperature end point at which cooling should be halted, but most series have halted aggressive cooling when the rectal temperature has dropped below 39.5°C.[25]

Contraindications for Rapid Cooling

Rapid cooling, per se, is never contraindicated in the presence of heatstroke. Immersion cooling is relatively contraindicated when cardiac monitoring of an unstable patient is required or when limited personnel make constant patient supervision impossible. Iced gastric lavage is contraindicated in patients with depressed airway reflexes unless the airway is protected by endotracheal intubation. Gastric lavage is also contraindicated by conditions that preclude placement of an orogastric or nasogastric tube. Cold peritoneal lavage is relatively contraindicated when multiple previous abdominal surgeries make placement of a lavage catheter risky owing to potential bowel perforation (see Chapter 44).

Evaporative Cooling

Evaporating water is thermodynamically a much more effective cooling medium than melting ice. Evaporating 1 g of water requires 540 kcal. Melting 1 g of ice requires only 80 kcal. In theory, therefore, evaporative cooling should be approximately seven times more efficient than ice packing. In practice, evaporative cooling is more efficient.[15] [82] In separate human studies, Wyndham et al.[83] and Weiner and Khogali[84]

TABLE 68-2 -- Cooling Rates Achieved with Various Cooling Techniques

Technique	Author(s), Year	Model	Rate (°C/min)
Evaporative	Weiner & Khogali, 1980	Human	0.31
	Kielblock et al, 1986	Human	0.09
	Wyndham et al, 1959	Human	0.034

	Daily & Harrison, 1948	Rat	0.93
Immersion (ice water)	Weiner & Khogali, 1980	Human	0.14
	Wyndham et al, 1959	Human	0.14
	Magazanik et al, 1980	Dog	0.27
	Daily & Harrison, 1948	Rat	1.86
	Costrini, 1990	Human	0.15
Ice packing (whole body)	Kielblock et al, 1986	Human	0.034
Strategic ice packs	Kielblock et al, 1986	Human	0.028
Evaporative strategic ice packs	Kielblock et al, 1986	Human	0.036
Cold gastric lavage	Syverud et al, 1985	Dog	0.15
	White et al, 1987	Dog	0.06
Cold peritoneal lavage	Bynum et al, 1978	Dog	0.56
	White, 1993	Dog	0.14
Cyclic lung lavage	Harris et al, 2001	Dog	0.5

found evaporative cooling to be 1.5 to 2.2 times faster than ice water immersion. Studies in primate models demonstrated faster cooling rates using evaporative cooling as an adjunct to ice bag placement.[85] Methods using convection and evaporation were more effective than those involving conduction for the treatment of hyperthermia.[15] In clinical practice, ice water immersion or ice packing are commonly undertaken because it causes heat loss by conduction, as well as by heat consumption by the phase change of melting ice. In healthy volunteers, evaporative cooling techniques (e.g., facial fanning) were associated with decreased thermal sensation and improved thermal comfort.[86]

Despite the continued enthusiasm of some clinicians for ice water immersion, *evaporative cooling is the fastest noninvasive cooling technique in human studies.*[13] [82] [86] To maximize evaporative cooling rates, several factors must be optimized. Air

TABLE 68-3 -- Advantages and Disadvantages of Various Cooling Techniques

Technique	Advantages	Disadvantages
Evaporative	Simple, readily available	Constant moistening of skin required
	Noninvasive	
	Easy monitoring and patient access	
	Relatively fast	
Immersion	Noninvasive	Cumbersome
	Relatively fast	Patient access and monitoring difficult
	Low mortality rates reported	Shivering
		Poorly tolerated by conscious patients
Ice packing	Noninvasive	Shivering
	Readily available	Poorly tolerated by conscious patients
Strategic ice packs	Noninvasive	Relatively slower cooling
	Readily available	Shivering
	Can be combined with other techniques	Poorly tolerated by conscious patients
Cold gastric lavage	Can be combined with other techniques	Relatively slower cooling
		Invasive
		Requires airway protection
		Human experience limited
Cold peritoneal lavage	Rapid cooling	Invasive
		Human experience limited

flow rates must be high (large fans are required). The air must be warm (but *not* humid), as evaporation is decreased at lower temperatures. The entire body surface must be exposed to airflow and continuously moistened with water (ideally the patient is suspended in a mesh sling to expose the back to airflow and moisture). Finally, the temperature of the water used to moisten the skin must be tepid (15°C). If the water is ice cold, evaporation will be slowed. Conversely, if it is hot, conductive heat gain may occur. Studies conducted in heat-stressed laying hens demonstrated superior cooling rates with ventral cooling regimes as compared to dorsal cooling. [87]

Weiner and Khogali[84] have constructed a sophisticated "body cooling unit" (BCU) to maximize evaporative cooling. Patients in the BCU are suspended in a mesh net. High airflow rates (30 m/min) at temperatures of 45°C are maintained both anterior and posterior to the mesh net. Atomized water at

15°C is continuously sprayed on all body surfaces. For emergency departments (EDs) without access to a BCU, temporary units can be set up using shower sprays and fans, providing the ambient temperature in the ED is relatively cool. [88] Dematte and colleagues recommend use of a body-cooling unit as a preferred technique for rapid cooling.[89]

The realities of clinical practice make these conditions impossible to fully replicate. Half the body surface (the back) will usually be unavailable for evaporative cooling. Airflow rates and temperatures are usually limited by the ambient temperature in the treatment facility and by the size and power of the fan available. These realities are reflected in the slower cooling rates achieved with evaporative cooling in a clinical setting.

Procedure

Evaporative cooling is accomplished by undressing the patient completely, positioning a fan or fans (usually at the foot of the bed or stretcher) as close to the patient as possible, and then sponging or misting the skin continuously with tepid 15°C water. A single care provider can continue the technique and monitor the patient once cooling has been initiated. It is important to keep as much body surface as possible moist and exposed to airflow. Covering sheets or clothing will impede skin evaporation and cooling.

Complications

Complications of evaporative cooling are rare and more often attributed to the underlying disorder than to the cooling technique. Wet skin may interfere with electrocardiogram (ECG) monitoring, but this can usually be avoided by using electrodes on the patient's back. Shivering occurs infrequently with this technique when compared with other cooling techniques, because the water is relatively lukewarm. [88] [90] Because the rectal temperature lags behind the core (esophageal) temperature, evaporative cooling should be discontinued when the rectal temperature reaches 39°C. In cases of mild hyperthermia, tympanic temperatures also accurately reflect core temperatures and can be useful in this setting. [91] Continued cooling beyond this temperature may lead to subsequent "overshoot hypothermia" due to continued core temperature drop after active evaporative cooling is discontinued. Shivering indicates that the core temperature has decreased to 37°C or below. [20]

Immersion Cooling

In one of the first studies of heatstroke cooling techniques, Daily and Harrison demonstrated that rats with hyperthermia cooled faster with ice water immersion than with evaporative cooling. [92] Some contemporary sources continue to recommend ice water immersion as the cooling technique of choice for heatstroke. [89] [93] Plattner and colleagues [93] demonstrated cooling rates with ice-water immersion that were six times faster than rates seen with forced air or circulating water.

Costrini and colleagues [95] reported no fatalities in 252 consecutive young marine recruits who were treated for exertional heatstroke over a 15-year period with ice water immersion within 20 minutes of diagnosis. They regard ice water immersion as superior in reducing mortality rates when compared to other conventional methods described in the literature.

Overall, in clinical trials, cold water immersion remains the second fastest noninvasive cooling technique available (see Table 68-2). In situations where an adequate evaporative cooling system is not available, immersion may be the cooling technique of choice. Several factors are important in maximizing the rate of immersion cooling. Conductive heat loss is dependent on cutaneous blood flow to maintain a heat gradient from skin to water. Theoretically, contact with ice water causes skin and subcutaneous (SQ) vasoconstriction, blocking heat exchange and turning these structures into insulators. [96] Intense cutaneous vasoconstriction will impede conductive heat loss. Mekjavic et al. [97] reported that motion sickness actually potentiates core cooling during immersion by attenuating the vasoconstrictor response to skin and core cooling, thereby augmenting heat loss and the magnitude of the decrease in deep body temperature. Careful monitoring is required because this may predispose patients to hypothermia. Magazanik et al. [98] in a canine study, suggested that warmer water (15°C) may actually cool faster than ice water (0°C). The optimal water temperature for cooling human heatstroke patients has not been defined.

Regardless of the water temperature, it is clear that increasing surface area increases conductive heat loss. Maximizing the body surface area in contact with the water will increase cooling rates with immersion cooling. In clinical practice, this means that complete immersion of the trunk and extremities will cool the patient faster than partial immersion of the trunk (back only) with the extremities extended out of the bath.

Procedure

Immersion cooling is accomplished by undressing the patient completely before transfer to a tub of water of a depth sufficient to cover the torso and extremities. Various water containers have been used. A regular bathtub, if available, can be used. Most clinical reports describe tubs that can be moved to the emergency treatment area when needed. A child's plastic wading pool and a decontamination tub or stretcher with waterproof sides and drainage capability are examples of the latter approach. The patient's head must be continuously supported out of the bath. In cases where tubs are unavailable, patients can be placed on water impermeable sheets and placed in a sling apparatus while ice and water are poured into the sling. [89] Temperature and ECG leads must be securely attached to the patient if monitoring is to be continued during immersion. The patient is removed from the bath when the rectal temperature reaches 39°C, because core temperature will continue to drop for a short period, even after the patient is removed. An electronic temperature monitor with a long flexible rectal probe is useful for continuous temperature monitoring during immersion. Studies show that rates of cooling close to 1°C per minute can be achieved. [23]

Complications

The common complications of immersion cooling are patient shivering, cutaneous vasoconstriction, patient discomfort, and the loss of monitoring capability. Shivering generates considerable heat through muscle metabolism. Cutaneous vasoconstriction impedes conductive heat loss. If significant shivering does occur, it can be reduced with benzodiazepine agents such as diazepam. Although the use of phenothiazines such as chlorpromazine has been advocated for shivering in the past, their use is currently discouraged because they also may impair heat loss by their anticholinergic effects on sweat glands, contribute to hypotension via a-adrenergic blockade, lower the seizure threshold, and cause dystonic reactions. In addition, they possess central dopamine-blocking effects

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that may exacerbate symptoms of NMS. [53] Benzodiazepines are also valuable if the patient is hyperthermic secondary to sympathomimetic agents such as cocaine. Magazanik et al. [98] also suggested that warmer water temperatures (15°C) minimize shivering and increase cutaneous blood flow, thereby increasing cooling rates. DeWitte and Sessler [99] reported that shivering occurs only as the body's final defense after maximal arteriovenous shunt vasoconstriction and behavior modifications have proved to be insufficient in maintaining core temperature. Shivering typically occurs after temperatures fall below 37°C. [20]

Patient monitoring is a problem under water. Electrodes can be used on the nonimmersed upper shoulders. ECG artifact often becomes a major problem during vigorous shivering. Immersion cooling is not recommended for patients with unstable cardiac rhythms or patients who are at risk for developing these rhythms. A significant change in cardiac rhythm might go undetected during the labor-intensive process of immersion cooling.

Patient access for resuscitative procedures is also a major problem with this technique. Should the patient develop ventricular fibrillation, he or she must be removed from the bath and dried prior to defibrillation. Invasive and diagnostic procedures (e.g., IV access, radiography) cannot be performed during the cooling period. Care must be taken to avoid displacement of IV lines during placement in and removal from the bath.

As body temperature drops, mental status will improve in many heatstroke victims. When awake, most people find ice water immersion difficult to tolerate. IV sedation may be required.

Finally, this technique is labor-intensive. Several caregivers must be present throughout the process. The patient's head must be maintained out of the bath. If massage is used, one or more individuals will need to immerse their own hands in water to continuously massage the patient. Medications should be given IV, and constant attention to temperature and ECG monitors is also necessary. This cooling technique should be used only if adequate personnel are available.

Whole-Body Ice Packing

Packing the heatstroke victim in ice may enhance conductive heat loss without the attendant logistical problems caused by water immersion (Fig. 68-1). Constant attendance, as required for skin moistening with evaporative cooling and as described for immersion cooling, may not be necessary with ice packing. Kielblock et al. [100] demonstrated in a human study of mild, exercise-induced hyperthermia that whole-body ice packing cooled just as fast as evaporative cooling (see Table 68-2).

Procedure

Whole-body ice packing is accomplished by undressing the patient completely and then covering the extremities and torso with crushed ice. As with any cooling technique, constant temperature monitoring using an electric thermometer and a long, flexible rectal probe is recommended. A large supply of crushed ice will be needed whenever this technique is used. Logistically, ice packing may be problematic. Whole-body ice packing can usually be performed on the ED stretcher without additional equipment. Ideally the patient is placed in a container that facilitates ice contact with the skin and

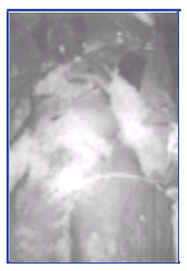

Figure 68-1 It is absolutely essential to rapidly lower the core temperature of a severely hyperthermic patient by instituting cooling techniques as soon as possible. Evaporative cooling (see text) is usually quite effective and technically easy. An alternative approach, albeit poorly studied, is to literally pack the patient in ice. In this case, plastic trash bags were used to hold the ice and to prevent water from dripping on the floor. A child's plastic wading pool is another option for this ice packing technique.

prevents water from dripping onto the floor. This is best accomplished by placing the patient in a child's lightweight plastic pool, which is available in toy stores. Lacking this equipment, plastic cloths or trash bags may be placed under the patient with the edges curled up to form a slinglike apparatus. As with immersion cooling, ECG monitoring can be potentially difficult owing to shivering artifact and displacement of electrodes. Alert patients usually do not tolerate ice packing well, and IV sedation or restraint is usually required. Excessive shivering can be treated with benzodiazepines if the rate of cooling is decreased. The ice is removed, and the patient dried off, when the rectal temperature reaches 39°C.

Strategic Ice Packs

Noakes has suggested that selective placement of ice packs over areas of the body where large blood vessels run close to the skin may be an effective cooling technique.[101] Cooling in these areas occurs despite cutaneous vasoconstriction, owing to direct conductive heat loss from the blood within the vessel, across the vessel wall, subcutaneous tissue, and skin to

the ice. The most common areas used for strategic ice packing are the anterior neck (carotid and jugular vessels), the axilla (axillary artery and vein), and the groin (femoral vessels). There have been numerous reports of successful cooling using ice packs as primary or adjunctive therapy (see Table 68-2).[88] [89] [101] In addition, application of ice packs, although easier to perform than immersion or total-body ice packing, limits the conductive cooling offered by the latter two procedures. [102] However, a study in pigtail monkeys demonstrated that a combination of strategic ice packs with evaporative cooling resulted in faster cooling than either technique alone, although the relative increase achieved by adding ice packs to evaporative cooling was small. [103]

In unconscious patients or in awake patients who can tolerate ice packs without excessive shivering, this technique could be added to evaporative cooling. However, the clinical value of strategic ice packs alone or in combination with other techniques remains to be determined. Anecdotally, during the Chicago heat wave of 1995, the majority of heatstroke patients who presented to EDs survived after being effectively cooled using the evaporation method accompanied by strategic placement of ice packs.

Procedure

This technique is best accomplished by placing large plastic bags filled with crushed ice or an ice water mixture in both axillae and over both femoral triangles. Do not diminish the effectiveness of the ice packs by wrapping them in towels—apply them directly to the patient. If the neck is used, the packs must be placed laterally, with care taken not to compress the trachea or apply excessive weight over the carotid arteries. The neck area should probably not be packed in the presence of carotid bruits or a history of cerebrovascular disease. Some sources advocate rubbing the body surface briskly with plastic bags containing ice after the body has been wet down with water. This is effective, provided it is combined with evaporation therapy. [21]

Complications

Complications of strategic ice packing are limited to shivering and patient discomfort as described previously for whole-body ice packing. The ice packs are removed when the rectal temperature reaches 39°C to avoid excessive core temperature drop. Prolonged direct contact with ice can produce cold injury. However, if the temperature is monitored and the ice packs are removed as soon as the target temperature is reached, such injury is unlikely.

External vs Core Cooling

All of the external cooling techniques described previously are noninvasive and use heat loss by evaporation or conduction across the skin as the primary cooling mechanism. With each of these techniques, dropping of the central temperature will continue even after the technique is discontinued and the skin is dried. This is due to a delay in the establishment of an equilibrium between the cold skin and the core. The amount of "core after-drop" can exceed 2°C. [93] For this reason, cooling is discontinued when the core temperature reaches 39°C.

Because the sites of significant cell damage with heatstroke are centrally located (e.g., liver, kidney, heart), central cooling techniques theoretically are preferable to external techniques. Core cooling techniques studied in both animal and human models include iced gastric lavage, intravascular cooling, bladder lavage, and peritoneal lavage.[93] [104] [105] [106] [107] Central venous cooling is effective in rapidly decreasing core temperatures. [108] Studies conducted in healthy volunteers demonstrated reductions of core temperatures varied according to the temperature of the infused fluid. Subjects receiving 30-minute infusions of fluid at 4°C experienced decreases in core temperatures of 2.5° ± 0.4°C. Subjects receiving 30-minute infusions of fluid at 20°C experienced decreases of 1.4°C (±0.2°C). [108] Clinical trials investigating cooling via the respiratory tract had no significant impact on temperature changes when used exclusively, yet demonstrated effectiveness as an adjunctive measure to other external cooling techniques. [91] Cool air (10°C) was administered via a hood or mask. Cooling via the respiratory tract has been studied in animals but not investigated clinically. [91] Central cooling techniques are necessarily more invasive than external techniques and therefore have the potential for more significant complications.

Cold Gastric Lavage

The stomach lies in close proximity to the liver, great vessels, kidneys, and heart. The gastric mucosa is not subject to the intense vasoconstriction observed on skin exposure to ice water.[109] For these reasons, lavage of the stomach might be expected to be an effective central cooling method. In one human trial, lavage with ice water at a rate of 500 mL/10 minutes was associated with increased abdominal cramping and diarrhea. [93] Human heatstroke victims have been successfully cooled with gastric lavage, but only in combination with external techniques. In practice, this technique is rarely used. Cold gastric lavage seems best suited for use in patients with severe hyperthermia who are cooling at a slow rate with external techniques alone. The presence of an endotracheal tube and the passage of a large-bore gastric tube make rapid lavage without aspiration possible. This technique should be reserved for patients whose airways are protected by endotracheal intubation and who do not have contraindications to gastric tube placement (see Chapter 41 Chapter 42 Chapter 43).

Procedure

Cold gastric lavage is best accomplished by instilling 10 mL/kg of iced tap water into the stomach as rapidly as possible (usually over 30 to 60 seconds). After a 30- to 60-second dwell time, the water is removed by suction or gravity. [107] Cooling will theoretically be faster if a high temperature gradient is maintained in the stomach. To this end, the lavage should proceed quickly. A faster lavage rate is usually maintained if suction is used to withdraw instilled fluid. A large container of ice-temperature water maintained 1 to 1.5 m above the patient's body will facilitate instillation of fluid. This container should be directly connected to the lavage tubing and should ideally allow passage of water but not ice, which may occlude the tube. Since large volumes of water are needed, it is helpful if ice can be added to the container without interrupting the lavage. A large syringe can be used as an alternative to gravity instillation, but this is usually slower.

A simple system that accomplishes this procedure can be devised from readily available equipment in most EDs. A standard lavage setup (for use in drug overdoses) and a large-bore gastric tube are used. The lavage bag is cut open at

the top to allow water and ice to be added. It is then suspended above the patient's body and connected to the orogastric tube by Y tubing with clamps. The other arm of the Y tubing is connected to suction. Using the clamps, ice water can intermittently be instilled by gravity and withdrawn by suction.

Complications

A major potential complication of cold gastric lavage is pulmonary aspiration. The use of a cuffed endotracheal tube minimizes the incidence of this complication. Owing to the large volume of water used and the frequent depression of airway reflexes seen with severe heatstroke, this technique should rarely be used in a patient who is not endotracheally intubated.

If tap water is used, water intoxication, hyponatremia, and other electrolyte disturbances are *potential* complications particularly in pediatric or geriatric patients. Water is absorbed from the stomach and, with large-volume lavage, may pass the pylorus into the small intestine. In canine studies, large-volume gastric lavage with tap water did not cause electrolyte abnormalities.[110] The actual incidence of these potential complications in human heatstroke has not been determined. The use of normal saline instead of tap water would eliminate this potential problem.

Theoretically, the passage of cold water through the esophagus, located directly behind the heart, has the potential to induce cardiac dysrhythmias. Dysrhythmias have not been observed in canine studies or in case reports of human heatstroke victims cooled with this technique.[109] [110]

Cold Peritoneal Lavage

The surface area and blood flow of the peritoneum greatly exceed those of the stomach. Peritoneal lavage is therefore expected to exchange heat much faster than gastric lavage. Peritoneal lavage demonstrates some of the fastest cooling rates ever reported in large animal or human studies (see Table 68-2). A case report of cold peritoneal lavage cooling for hyperthermia following ecstasy ingestion demonstrated rapid cooling.[111] As with gastric lavage, this central cooling technique offers the advantage of directly cooling the core organs that are most susceptible to thermal damage. Unlike with gastric lavage, endotracheal intubation is not required. Peritoneal lavage is used extensively to treat hyperthermia under various conditions and typically decreases core temperatures 5°–10°C/hour.[93] [104] [111]

Peritoneal lavage is a more invasive cooling technique. Surgical placement of the lavage catheter is necessary. Since heat exchange is more efficient across the peritoneum, smaller volumes of fluid can be used. This cooling technique is relatively contraindicated by conditions that preclude placement of a lavage catheter (e.g., multiple abdominal surgical scars) (see Chapter 44).

Peritoneal lavage is the most rapid central cooling technique. It can theoretically be combined with other techniques to speed cooling of the heatstroke patient with refractory hyperthermia. Being the most invasive cooling technique, it requires time, proper equipment, and surgical expertise to institute. Although effective, it is seldom used in clinical practice. Its use is probably best suited to situations in which heatstroke patients are not responding to external cooling and adequate equipment and personnel are readily available.

Procedure

To institute peritoneal lavage cooling, 2 to 8 L of sterile saline is immersed in an ice water bath to cool while the catheter is being placed. A standard peritoneal lavage catheter (as for diagnostic use in trauma patients) is placed using any of the techniques described in Chapter 44 . Standard contraindications apply. Use of a larger peritoneal dialysis catheter may speed fluid instillation and withdrawal. Actual lavage volumes and rates have not been established. One approach is to instill and withdraw 500 to 1000 mL every 10 minutes until adequate cooling is achieved. Rectal temperature may be falsely low during the lavage owing to the presence of cold water about the rectum at the level of the rectal temperature probe.[107] [112] It may be preferable to monitor tympanic membrane or esophageal temperature when using this technique. The lavage is discontinued when core temperature reaches 39°C to avoid excessive core temperature after-drop.

Complications

The potential complications of peritoneal lavage cooling are primarily related to placement of the catheter and include bowel or bladder perforation and placement into the rectus sheath rather than the peritoneum. These potential problems are discussed further in Chapter 44 .

Other Cooling Techniques

Although high-frequency jet ventilation (HFJV) causes core cooling in critically ill patients,[114] efforts to use the respiratory tract to cool heatstroke victims have been unsuccessful. In a canine model of heatstroke, the use of HFJV is shown to be a relatively ineffective cooling technique.[115] Heat loss by convection (air transfer) is relatively inefficient compared with the conductive heat loss mechanism used by other cooling techniques. The use of dry, hot air to maximize evaporative heat loss from the lungs might cause respiratory complications.[114]

In human trials, ice water lavage of the bladder (300 mL iced Ringer's solution/10 min) provided only minimal cooling with rates of 0.8°C (±0.3°C) per hour.[93] Iced water lavage of the rectum would theoretically provide faster cooling rates secondary to the increased surface area and better perfusion; however, it has not been investigated in human trials.[94]

Hemodialysis or partial cardiopulmonary bypass could theoretically be used to cool heatstroke patients. Before the availability of dantrolene in 1979, partial cardiopulmonary bypass was one treatment for malignant hyperthermia.[105] Its specific use in management of heatstroke has not been studied. Drawbacks could potentially include lack of technical expertise as well as preparation time for the procedure.

Cyclic lung lavage using cold perflurochemical lung lavage in animal models is currently under investigation. Benefits include rapid cooling rates of 0.5°C per minute and are minimally invasive in the already mechanically ventilated subject.[116] [117] [118]

In addition to physical cooling techniques, pharmacological agents have demonstrated merit as adjunctive agents in the management of hyperthermia. There are anecdotal reports of enhanced temperature reduction using intravenous ketoralac. In a recent study, Cienki and colleagues[119] demonstrated enhanced temperature decreases using ketorolac, 30 mg IV. All patients received standard treatment for

hyperthermia (e.g., ice packs, iced lavage, circulating air). Patients were randomized to receive ketorolac versus saline. In the group receiving ketorolac, the average rectal temperature after 90 minutes was two times lower than that of those receiving placebo saline (3.7°F vs 1.6°F).

SUMMARY

Rapid cooling is the key step in the emergency management of heatstroke patients. Survival approaches 90% when elevated temperatures are lowered in a timely fashion.[19] [27] The highest documented temperature in the medical literature with survival is 48.8°C (115°F). In this case, the patient was rapidly cooled and recovered without neurologic sequelae.[34] Evaporative cooling appears to be the technique of choice. It combines the advantages of simplicity and noninvasiveness with the most rapid cooling rates achieved with any external technique. It is also logistically easier to institute, maintain, and monitor evaporative cooling than any other cooling technique. If a patient is not cooling rapidly with evaporative cooling, other techniques can be added. Ice packing or strategic ice packing is a common alternative technique that can be rapidly instituted in any ED. If the patient is endotracheally intubated, gastric lavage can be instituted. If facilities and personnel are available, peritoneal lavage cooling can be used as a rapid central cooling technique.

If muscle rigidity is present or malignant hyperthermia is suspected, dantrolene sodium should be administered. In addition, the clinician should have a heightened index of suspicion for NMS and sympathomimetic drug toxicity. Regardless of the cause, a reasonable clinical goal is to reduce the rectal temperature to 40°C or below within 30 minutes of instituting therapy.[89]

Immersion cooling is best limited to centers with the proper equipment and skilled medical personnel experienced in managing hyperthermic patients. This method may also be effective in conditions in which electric power for evaporative cooling is unavailable (e.g., in wilderness settings where bodies of cool water are available nearby and the victim is far from more sophisticated medical care). Central venous cooling with iced saline is a promising cooling technique for rapid cooling for severe hyperthermia. Other cooling techniques require further study before a clear recommendation as to their efficacy can be made.

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Chapter 69 - Ultrasound-Guided Procedures

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Lisa Mackowiak Filippone

Sonographic guidance for invasive procedures is a logical addition to the practice of emergency medicine. As of this writing, however, it is not considered standard of care that the emergency clinician is skilled in, or has access to, bedside ultrasonography. Bedside sonography images deep tissue anatomy, thus increasing the chance of successful performance of a wide range of invasive procedures and minimizing associated complications. Consensus guidelines developed by the American College of Emergency Physicians for use of ultrasound (US) by emergency clinicians include sonographic guidance for procedures as being within the scope of practice for emergency clinicians.[1] This chapter is not meant to be a comprehensive description of the US examination, nor should it be viewed as a tutorial for emergency clinicians unskilled in the use of US. It is assumed that the emergency medicine clinician using US has had some formal training in the performance and interpretation of directed bedside US as outlined in the ACEP 2001 guidelines. The following chapter will describe those procedures within the scope of practice of emergency clinicians for which there is a role for bedside US. The procedures described here are covered in detail elsewhere in the text, and this section will focus primarily on the role of US.

PHYSICS

US images are created by high-frequency sound waves, which are generated and interpreted by a transducer and then converted electronically to form an image on a screen. The transducer is a probe that contains crystals, which change shape and vibrate when an electrical current is applied, creating sound waves. This is referred to as the *piezoelectric* effect. The crystals emit sound for a brief moment, and then wait for the returning echo reflected from the structures in the plane of the sound beam. When the echo is received the crystals vibrate, generating an electrical voltage proportional to the strength of the returning echo. This is then converted electronically into an image on the viewing screen.

Sound waves are reflected back to the transducer from tissue interfaces that have different acoustic impedances (the density of the tissue times the speed of sound in tissue). Tissues of higher density such as bone that interface with lower density substances such as muscle or fluid will reflect nearly all the sound waves, and will appear on the monitor as brightly echogenic (white) structures. Fluid transmits nearly all the sound waves, and will appear black or anechoic. Tissues will vary in their echogenicity or brightness based on their density, compliance, and adjacent structures (Fig. 69-1).

The purpose of using US during a procedure is to allow the clinician to "see" the area of interest below the skin surface. The area on the body surface that will provide the best images is referred to as the *acoustic window*. There are some general principles that help determine the suitability of an area as an acoustic window. Sound waves travel best through structures that are composed of closely packed molecules. Air, because the molecules are widely spaced, is a very poor conductor of sound. Therefore, structures that contain air, such as the lungs and bowel, cannot be imaged with US. In addition, structures that lie beneath an air-filled structure (such as the aorta, which lies beneath loops of small bowel) may not be clearly visualized. In contrast, fluids have tightly packed molecules and conduct sound well. Fluid-filled structures are readily visualized with US because the fluid/tissue interface is highly reflective, creating a clear image. Fluid-filled structures also serve as excellent acoustic windows to structures that lie beneath them. Therefore, procedures involving entry into a fluid-filled space are best suited to sonographic guidance. For each of the procedures listed in this chapter, the optimal acoustic window is described.

INDICATIONS AND CONTRAINDICATIONS

US may be used to guide cannulation of vessels, aspirate fluid collections within cavities (e.g., pericardium, pleurae, bladder, or joints), and locate soft tissue foreign bodies. US may be used to mark the site for skin puncture or provide continuous real-time visualization throughout the procedure. The real and potential applications for bedside sonography continue to expand as technology improves and clinical expertise in the hands of emergency clinicians grows. It is truly an extension of the examining clinician's eyes and hands and may be used to visualize any portion of the anatomy that is amenable to sonographic imaging. US is especially helpful in answering clinical questions regarding depth, size, and nature of subcutaneous masses or collections and determination of the presence of fluid within body cavities.

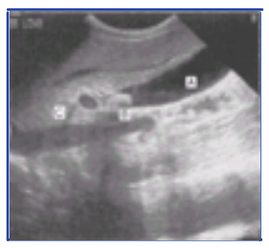

Figure 69-1 Longitudinal image of the gallbladder, demonstrating variability in tissue echogenicity. *A,* The anechoic appearance of fluid. Water, plasma, non-clotted blood, and urine will have the same appearance. *B,* The highly reflective appearance of a calcified stone in the gallbladder. Foreign bodies, needles, and bone will have a similar brightly echogenic appearance. *C,* The relatively hypoechoic appearance of tissue. Clotted blood, particulate material within fluid (lipid or purulent material) will appear the same, with echogenicity intermediate to bone and fluid.

The clinician choosing to use sonography must have training and experience in the use of US technology and image interpretation. Clinical errors directly related to the use of bedside US usually result from incorrect use of the technology or misinterpretation of sonographic images. Technological errors may occur when the wrong probe is used. For example, procedures requiring high resolution (central vein cannulation or foreign body detection) should be guided with a high-frequency probe. Lower frequency probes may not adequately delineate structures and hence lead to error. Imaging of the heart often requires a probe with a small "footprint" that will allow for imaging between the ribs; a larger footprint probe will include rib shadows that may affect the quality of the examination. Inadequate amounts of gel and air bubbles in the probe cover can create artifacts that adversely affect the technical quality of the images. Finally, the sonographer must be familiar with the various settings on the machine that will determine image quality. For example, inappropriate gain settings are common sources of error and image misinterpretation; too much gain may give the appearance of echogenic shadows within spaces. These shadows may then be interpreted as tissue or clot.

Problems with interpretation usually arise when the clinician is attempting to interpret an image that is suboptimal, due to poor patient preparation or positioning, the presence of air-filled structures between the probe and structure of interest, or lack of appreciation of sonographic artifacts. Emergency clinicians are often required to perform procedures under suboptimal conditions, and the same is true for sonography. Bedside sonography is often performed simultaneously with other procedures on a patient who may be unable or unwilling to fully cooperate. The patient may be receiving active chest compressions or be profoundly hypotensive, leaving vessels flaccid with poor flow. There may be subcutaneous air or significant soft tissue swelling between the probe and object of interest, and veins may be filled with clot or scar from previous central lines. For these reasons, the clinician using sonography must adhere to a few important principles. First, do not attempt to interpret a sonographic image that does not clearly depict the structure or organ of interest. Second, interpret the sonographic image in the context of the clinical picture—what you see must make sense to what is happening clinically. Finally, if you are not sure of what you are seeing—obtain an alternative imaging study or expert assistance.

For the remainder of this chapter, it is assumed that the clinician performing a US-guided procedure has appropriate training and experience. To avoid redundancy, this point will not be restated. That is, lack of sonographic training and experience is a contraindication for incorporating bedside sonography with any procedure.

EQUIPMENT

The crystals determine transducer frequencies. Those used most commonly for medical diagnostic imaging range from 2 MHz to 10 MHz. Lower frequency probes are used for viewing deeper structures, such as the heart or the aorta, and larger patients, but produce images that are of lower resolution. Higher frequency probes provide high-resolution images of fairly superficial structures, such as veins and subcutaneous tissues, and are recommended for use in children and very

Figure 69-2 The curvilinear array probe is used for lower frequency (2.0 to 5.0 MHz) scanning of the abdomen and chest. It is best used for cardiac and abdominal imaging. The linear array probe is used for high frequency (6 to 10 MHz) scanning of superficial tissues, vessels, subcutaneous masses, and foreign bodies.

thin adults. The highest possible frequency probe should be used because it will provide superior resolution.

Transducers vary also in the array of their piezoelectric elements, or crystals. The nature of the array will affect the overall field of imaging. The transducer array with the widest range of applications for emergency sonography is the curvilinear array, which has a narrow near field and pie-shaped window, allowing for a small acoustic window and large imaging area. This is ideal for imaging between ribs and curved surfaces. The linear array is used for high-frequency scanning of superficial tissues and is ideal for imaging vessels, subcutaneous masses, and fluid collections (Fig. 69-2).

There are several needle guidance systems available. Attached to the probe of such a system is a metal or plastic device through which the needle passes (Fig. 69-3). While these systems are designed to improve the accuracy of needle insertion, the path of the needle is determined by the probe angle, which can be altered by even subtle hand movements. Needle guidance systems are most useful when the target

Figure 69-3 An ultrasound needle guide is ideally used to guide needle placement into deep or small structures. *(Courtesy of Dymax Corp, a subsidiary of Bard Access Systems.)*

organ is deep or small in size, and are usually not necessary for the majority of emergency department (ED) procedures.

GENERAL APPROACH

Most procedures facilitated by the use of bedside US require the use of two hands. Therefore, the clinician must make a decision whether to have an assistant hold the probe, perform the procedure with one hand while holding the probe in the other (freehand), or use the US probe to simply mark the point of entry prior to the procedure (blind approach). When the procedure requires two hands, it is best to have an assistant hold the probe. This is particularly important when one needs to visualize needle entry into the space or vessel. The freehand approach requires the operator to hold the probe in one hand while performing the procedure with the other. This is best suited for procedures that require only one hand, such as needle aspiration of large joint spaces or abscesses.

The blind approach is best used for large collections of fluid, such as ascites or large pleural effusions. The optimal site is identified prior to the start of the procedure and the area marked with a sterile pen. The depth of the collection is noted by referring to the depth markers to the right of the imaging screen, which indicate the distance from the skin surface to the most anterior margin of the collection or vessel. Depending on the area of interest, it may be necessary to note the patient's position and phase of respiration when identifying the point of entry. This is particularly important when attempting thoracentesis (phase of respiration) or paracentesis (patient positioning).

Catheters, wires, and needles appear as brightly reflective structures within the fluid-filled anechoic space. Keeping the needle perpendicular to the plane of the US beam will maximize the chance of visualizing the needle. Other methods that have been reported to improve visualization include placing the focal zone in the near field and bobbing the needle. The latter requires an in-and-out jiggling type of movement that moves the needle within the sonographic window. [2]

When the probe is left in place during the procedure, it needs to be covered with a sterile rubber sheath (a sterile glove is usually adequate). When available, a transparent sterile drape can also be used. In either case, a layer of acoustic gel should be placed between the probe and sterile cover or drape. Another layer of sterile acoustic gel should be placed between the skin surface and the probe cover or drape.

Central Venous Line Placement

Obtaining vascular access is central to the care of many patients in the ED, particularly those who are critically ill or injured. Peripheral access is often difficult in these patients and central venous catheter placement is necessary. Bedside US has been shown to improve the chance of successful central vein cannulation and to reduce the number of complications. While internal jugular and femoral vein access are amenable to sonographic guidance, the subclavian vein is difficult to visualize sonographically secondary to the presence of the clavicle and will not be described here.

Background

Sonographic guidance (using two-demensional echo) of central vein access was first described in 1986. [3] In 1990, Mallory et al. compared the standard landmark-guided technique to the US-guided technique for cannulation of the internal jugular vein in intensive care unit patients. They demonstrated a 100% success rate in the US-guided group versus a 65% success rate in the landmark group. [4] Another prospective study in 1991 evaluated 160 cardio-thoracic surgical patients requiring right internal jugular vein cannulation. The US group had a greater overall success rate (100% vs. 95%), a higher percentage of first-attempt cannulations (73% vs. 54%), fewer attempts per cannulation (1.4 vs. 2.8), shorter time per cannulation (61 vs. 117 seconds), and fewer carotid punctures (1 vs. 7) compared with a control group using the standard landmark technique. [5] In 1993, Denys et al. conducted the largest prospective trial to date, comparing the results of 302 US-assisted internal jugular vein cannulations with 302 cannulation attempts using the standard external landmark-guided technique. The US group had a higher success rate with fewer attempts, a decrease in the mean time to cannulation, and a reduction in complications including carotid puncture, brachial plexus injury, and hematoma. [6] Another study performed on ED patients by emergency clinicians again demonstrated more successful single-needle-pass punctures of the internal jugular vein using US guidance, particularly in patients with no visual or palpable landmarks. [7]

The benefits of using US for central venous access have also been demonstrated in children. [8] [9] This is no surprise since children's vessels are smaller, cooperation is unpredictable, and tensions are often high when performed under emergent conditions. In addition, compared with older children and adults, studies have demonstrated that younger children have more failed attempts and more complications. [8] [10] [11] [12] In a study of infants under 12 months of age undergoing preoperative central line placement, one study found that cannulation of the internal jugular vein was 100% successful using US guidance (n = 43) vs 77% in infants using the standard landmark-guided technique (n = 52). [8] The time for cannulation was also significantly reduced (3.3 minutes vs 10 minutes) and the incidence of carotid punctures was lower (0% vs 25%) in the US group. [8]

There have been few studies evaluating the use of US for femoral vein access, but the evidence to date is encouraging. In 1997, Kwon et al. demonstrated greater success (100%) in cannulating the femoral vein using US guidance compared with the standard landmark-guided technique (89%) in patients requiring central venous access for hemodialysis. [13] Complication rates were also lower in the US-guided group. [13] Hilty et al. compared success and complication rates of US-guided femoral line placement with that of the standard landmark-guided approach in ED patients during cardiopulmonary resuscitation. The success rate using US was 90% vs 65% in the control group. There were no arterial punctures in the US group; however, the femoral artery was punctured in 20% of blind attempts. [14]

Other vessels besides the internal jugular and femoral veins can be cannulated safely using US guidance. Of particular interest is the brachial vein, which is usually not attempted due to absence of reliable landmarks, deep location, and proximity to important structures (e.g., nerves, brachial artery). A study by Keyes et al. showed that the brachial vein can be safely cannulated using sonographic guidance in patients with limited access (50 intravenous drug users and 21 obese patients). The brachial vein was successfully cannulated in 91% of patients within a mean time of

77 seconds. Reported complications were pain, line failure due to falling out or infiltration (8%), and brachial artery cannulation (2%). [15]

Indications and Contraindications

If sonographic equipment is readily available, the emergency clinician should have a very low threshold for using it to guide central venous cannulation. It is especially helpful when access is required on a patient with distorted anatomy, obesity, history of multiple prior central lines, and hypotension, and when blind attempts have been unsuccessful. The benefits include localization of the vein and artery and demonstration of venous patency through ease of compression and free flow (if Doppler is available). There are no reported contraindications specific to sonographic guidance of venous cannulation.

Equipment

A high-frequency (7.5 MHz to 10 MHz) linear probe is highly recommended for imaging the relatively superficial internal jugular and femoral veins. A needle guide may be helpful for deep or small veins, but may make coordinating imaging and access more difficult for less experienced sonographers (see Fig. 69-3). If a linear probe is not available, any high-frequency probe, such as the endovaginal probe, can be used. A sterile sleeve (or glove) and sterile conductive medium are required. A sterile marking pen can be used to mark the location of the vein if the US probe is removed before puncture (see later in this section). Two persons are usually required—one to perform the US while the other obtains vascular access.

Image Interpretation

The internal jugular vein and carotid artery will appear as relatively superficial structures on the monitor. When viewed in the transverse plane, the internal jugular vein is a thin-walled, circular, anechoic structure anterior and lateral to the carotid artery. However, there is some variability in the relationship between the two

vessels.[16] [17] Denys et al. studied the location of the internal jugular vein relative to the carotid artery in 200 patients. They found that 2% of patients had an internal jugular vein that was positioned medially, overlying the carotid artery. The internal jugular vein was found more laterally than expected in 1% of patients. [16] Turning the patient's head will also alter the relationship between the vein and artery, usually moving the vein medially over the artery. When patent, the internal jugular vein will increase in size with inspiration and Valsalva maneuvers. Light pressure applied with the probe directly over the vessels will easily compress the internal jugular vein and only one anechoic circular structure, the carotid artery, will be seen on the monitor. Once pressure is released, the lumen of the internal jugular vein will reappear. The external jugular vein is not usually seen on the monitor. Because of its superficial location and compressibility, it is usually flattened just by placement of the probe on the neck.

The femoral artery and vein also appear as superficial anechoic circular structures when viewed in the transverse plane. The vein is located medially and the artery is located laterally. As the probe is moved toward the patient's head, this relationship changes, with the vein eventually lying behind the artery. Other sonographic findings helpful in distinguishing the femoral vein are that it is easily compressed and will increase in size by squeezing the thigh. The artery is not readily compressed and can be seen to pulsate. One exception to this may be in patients who are undergoing cardiopulmonary resuscitation, where pulsations will be seen primarily in the femoral vein and not the artery. Coletti et al. described the presence of femoral pulses in canines when cardiopulmonary resuscitation was in progress and the proximal femoral artery was clamped. These pulses disappeared when the proximal femoral vein was clamped and the artery unclamped. [18]

Procedure and Technique

Internal jugular vein

To access the internal jugular vein, the patient is placed in the supine position with the head turned slightly to the opposite side of the vein being cannulated. The lateral aspect of the neck is prepped and draped in the usual fashion (see Chapter 22). A small amount of US medium is placed on the probe. A sterile sleeve or glove is placed over the probe. It is important to remove any air bubbles between the gel on the probe and the sterile cover, as this will create artifacts. Sterile gel is then placed over the cover. A sterile towel or drape can be wrapped around the rest of the probe and cord to help maintain sterility.

The US probe is placed parallel and superior to the clavicle over the groove made by the two heads of the sternocleidomastoid muscle (Fig. 69-4). The beam of the US probe should intersect the carotid artery and the internal jugular vein in a transverse or cross-sectional plane. At this level, the internal jugular vein is usually anterior and lateral to the carotid artery (Fig. 69-5*A* and *B*). Additional maneuvers can help in differentiating the internal jugular vein from the carotid artery (e.g., the vein will markedly increase in diameter with Valsalva maneuvers). Asking the patient to blow on his or her

Figure 69-4 Imaging window for the internal jugular vein and carotid artery. The ultrasound probe should be covered with acoustic gel and then a sterile cover or glove. An additional layer of sterile gel should be placed over the cover. The probe is then placed parallel and superior to the clavicle over the groove made by the two heads of the sternocleidomastoid muscle. The probe should be pointed toward the patient's right. Care should be taken not to apply undue pressure on the probe to avoid compression of the easily collapsible internal jugular vein. Asking the patient to blow on his or her thumb or performing a Valsalva maneuver will increase the luminal diameter of the vein. *(Lydia F. Roberts, Photographer)*

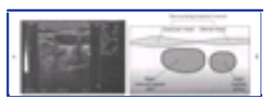

Figure 69-5 The internal jugular vein and carotid artery. *A,* Sonographic image; *B,* Schematic representation. The beam of the ultrasound should intersect the carotid artery and the internal jugular vein in a transverse or cross-sectional plane. The internal jugular vein is easily identified by its compressibility and response to Valsalva maneuvers (i.e., it increases in diameter).

thumb is often helpful in creating a sustained Valsalva maneuver that will readily identify a patent internal jugular vein. If one applies gentle pressure to the vein, it will collapse. On the screen, only the carotid artery lumen will remain. Either color or power Doppler can also readily distinguish a vein from an artery. Pulsatile flow within the carotid artery appears quite different from lower amplitude, phasic venous flow that augments with Valsalva maneuvers. Once visualized, the internal jugular vein should be centered on the monitor screen, allowing the center of the probe to be used as a guide for needle puncture. The needle will first indent the vein. Then the vein will appear to collapse on the monitor as it is entered (Fig. 69-6*A* and *B*). The needle is advanced slightly farther and the vein will re-expand once it has been entered. The needle may seen on the monitor, particularly if the US beam is perpendicular to needle (Fig. 69-7*A* and *B*). Once venous blood is aspirated, the US probe may be removed. A guidewire is then threaded through the needle and the catheter is placed as described in Chapter 22 .

An alternative method, requiring only one operator, is to use the US to locate the underlying vein and then mark its location with a sterile pen. The internal jugular vein is usually a very superficial structure between the two heads of the

Figure 69-6 *A* and *B,* Once the vein is positioned directly under the probe, needle aspiration can be attempted. If the site of skin puncture is correct, the needle will indent the vein and the vein will appear to collapse as it is entered.

sternocleidomastoid muscle, and easily accessed once its location and patency is confirmed with US. Care must be taken not to readjust the position of the patient's head at this time. Rotating the head farther away from the vessel being cannulated will usually cause the vein to move medially over the carotid artery, and increase the risk of inadvertently puncturing it. The probe is removed after the vein has been located and needle puncture is performed.

Femoral vein

The patient is placed supine with the leg extended and slightly rotated externally. The patient is prepped and draped as usual (see Chapter 22), and the US probe is prepared as described earlier. If the patient has a palpable pulse, the US probe is placed over the pulse in a transverse orientation inferior to the inguinal ligament. If the patient does not have a palpable pulse, the probe should be placed approximately midway between an imaginary line drawn from the anterior superior iliac spine to the ipsilateral pubic tubercle just inferior to the inguinal ligament. The vein will be the vessel located medially. Similar maneuvers as described earlier can be done to help distinguish the femoral vein from the artery. These include compression with the probe and squeezing the thigh, which is the "Valsalva equivalent" for the femoral vein and

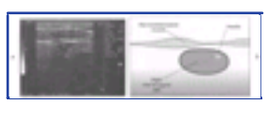

Figure 69-7 *A* and *B,* The needle may be visualized within the vessel lumen, and will appear as a brightly echogenic structure.

will increase the vessel caliber in a patent vein. Once located, the vein should be centered on the screen so that the middle of the probe can be used as a guide for the needle stick. Once the vein is entered the probe can be removed and a guidewire advanced through the needle. Subsequent cannulation of the vein is described in Chapter 22 .

Complications

Complication rates for the standard landmark-guided approach of internal jugular vein cannulation are between 4% and 10%. [19] [20] [21] Reported complications include vascular perforation and laceration, pneumothorax or hemothorax, tracheal perforation, brachial plexus and phrenic nerve injury, hematoma formation, thoracic duct injury, and catheter malposition. Several trials have shown that using US guidance to cannulate the internal jugular vein decreases complication rates, particularly carotid artery puncture.[5] [6] [8] [22] Troianos et al. reported a decrease in the carotid artery puncture rate from 8.43% to 1.39% using standard vs US-guided cannulation of the internal jugular vein.[5] Likewise, Denys et al. demonstrated a similar decrease in carotid artery punctures using US guidance, as well as decreases in brachial plexus irritation and hematoma formation.[6]

Potential complications unique to femoral vein cannulation include retroperitoneal hematoma, bowel perforation, and bladder perforation. Two studies comparing the complication rates of US-guided vs standard femoral vein cannulation have demonstrated reduced complications using US. [13] [14]

Complications unique to using US are rare and usually due to poor technique or attempts to use a low-resolution probe to image the vessels. One commonly encountered problem is in patients with proximate clot or stricture in the vein from prior central lines. In these patients, the vein will be seen and readily accessed, yet there will be little to no flow. A clue to this possibility is a vein that does not easily compress. When in doubt, comparison with the opposite side may also be helpful. In addition, Doppler can identify those vessels with proximate obstruction, but is not essential. Finally, remember that the anatomical relationships between the internal jugular vein and artery will change with head turning, and may place the artery beneath the vein. If the blind approach (i.e., marking the skin and removing the US probe prior to insertion) is used, the vessels should be imaged after final head positioning and the patient kept immobile until the line is secured.

Pericardiocentesis

The role of bedside sonography in the management of patients with suspected pericardial effusion is to establish the diagnosis and to visualize placement of a drainage catheter within the pericardial sac. The diagnosis of a clinically significant pericardial effusion is readily determined by bedside sonography, and is far superior to physical examination alone. Sonography is the study of choice for identification of pericardial effusions, and the availability of bedside US has the potential to shorten time to diagnosis of patients with clinically significant effusions. [23] [24] [25] [26] [27] For the subset of patients who are hemodynamically compromised by their effusion, pericardiocentesis performed under sonographic guidance is a safe and effective alternative to blind aspiration.

Background

Aspiration of the pericardial sac by emergency clinicians is usually a last-ditch effort to resuscitate patients with pulseless electrical activity (PEA) and is often unsuccessful. This is because the diagnosis is usually considered only after the patient has become unstable and pericardiocentesis is performed late in the resuscitation effort. Recent studies have shown that while large pericardial effusions are a rare cause of hemodynamic instability in the ED, a low threshold for performing bedside US may increase detection of effusions before they become hemodynamically significant. [25] [28] [29]

The traditional approach to the patient with a suspected clinically significant effusion has been to blindly access the pericardial sac using the subxiphoid approach. This technique is associated with a variety of complications, including puncture of the liver, lungs, myocardium, and epicardial vessels [30] [31] (see Chapter 16). In hemodynamically stable patients, a surgical window is usually performed in order to avoid the complications of blind aspiration. Over the past decade there has been increased enthusiasm for echocardiographically guided pericardiocentesis. The techniques first described in 1998 [30] are applicable to the ED setting and can be used for management of patients with hemodynamically significant pericardial effusions.

Indications and Contraindications

Patients presenting with acute and subacute cardiac tamponade usually do not display the classic triad of hypotension, neck vein distention, and muffled heart tones. Waiting for their appearance to order a confirmatory test will often delay diagnosis and possibly jeopardize the safety of the patient. The heart should be imaged whenever a clinically significant pericardial effusion is suspected. Scenarios warranting consideration of a pericardial effusion include a patient with hypotension of unclear etiology, particularly those patients with known malignancies, recent myocardial infarction, and end-stage renal disease, and victims of trauma, both blunt and penetrating.[25] [26] [27] [28] [29] Early demonstration of a large pericardial effusion will guide further work-up, support early consultation from either cardiology or cardiothoracic surgery, and facilitate therapeutic drainage for those patients who remain hypotensive in spite of fluid resuscitation. For patients in extremis, the clinician most clinically experienced in both sonography and aspiration of the pericardial sac should perform pericardiocentesis without delay. For patients with large effusions who are relatively stable, management options are greater and may include a pericardial window. Consultation with either cardiology or cardiothoracic surgery is advised prior to performing aspiration on stable patients.

Equipment

The optimal probe frequency is 2 to 4 MHz. It should have a small enough footprint to allow for imaging between the rib spaces, particularly if the parasternal windows are used. For the subxiphoid view, the standard 2 to 3.5 MHz curvilinear probe will provide excellent images.

Acoustic Windows

The cardiac examination is dynamic. The sonographer must identify the imaging window that provides images which best demonstrate the effusion or cardiac chamber of interest. Most frequently used are the subxiphoid and parasternal windows. It is important for the sonographer to develop expertise in using a variety of imaging windows to obtain the necessary clinical information.

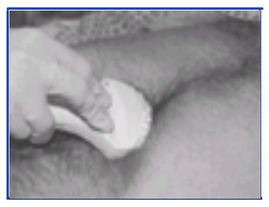

Figure 69-8 To obtain a subxiphoid view of the heart and pericardium, the probe should be placed beneath the xiphoid process with the probe marker pointing to the patient's right. The probe should be angled upward and slightly to the left. *(Lydia F. Roberts, Photographer)*

Subxiphoid view

This view provides the most comprehensive information for a single view, and is therefore the most important acoustic window for the less experienced sonographer to learn. It will readily identify a circumferential pericardial effusion and allow assessment of overall cardiac wall motion. The subxiphoid view can usually be obtained within 1 minute, and may be the only view required. To obtain the subxiphoid view, the probe is placed transversely at the left costal margin at the level of the xiphoid process with the beam aimed at the left shoulder (Fig. 69-8). The angle and rotation of the probe should be adjusted to obtain the appropriate views. The structures closest to the probe will appear at the top of the display and include the liver, diaphragm, pericardial space, and right ventricle (Fig. 69-9*A* and *B*).

Figure 69-9 *A,* Sonographic appearance of a normal heart and pericardium obtained through the subxiphoid window. The right ventricle will be the most anterior cardiac structure, bordered superiorly by the pericardium and diaphragm. *B,* Schematic representation.


Figure 69-10 To obtain a parasternal view of the heart, the probe should be placed adjacent to the left sternal border in the left second or third intercostal space. The patient's head is at the lower edge of the photograph. *(Lydia F. Roberts, Photographer)*

Parasternal view

The next most useful view is the parasternal long axis view. To obtain this view, the transducer is placed in the left parasternal area between the second and fourth intercostal spaces. The plane of the beam should be parallel to a line drawn from the right shoulder to the left hip with the marker pointing to the right shoulder (reverse if the image is not set for cardiac views) (Fig. 69-10). The parasternal view provides excellent images of the left atrium, mitral valve, left ventricle, aortic valve, and the proximal ascending aorta. It is also the best view to identify small dependent collections within the pericardial sac (Fig. 69-11*A* and *B*).

Image Interpretation

The normal pericardium will appear as a single, brightly echogenic stripe adjacent to the myocardium. Fluid within the pericardial space will collect between the visceral and parietal pericardium and will appear as a large, non-beating, anechoic area adjacent to the ventricular myocardium (Fig. 69-12*A* and *B*). A small amount of fluid in the dependent portion of the pericardial space is normal. The sonographic appearance of the clinically significant effusion is distinct. The sonogram will reveal a hyperkinetic heart within a circumferential pericardial effusion, with diastolic collapse of the right-sided chambers. This reflects pressures within the pericardial space that are greater than the right ventricular filling pressure during diastole. The inferior vena cava will be dilated and not show respiratory variation or collapse when the patient is asked to "sniff."

Procedure and Technique

Tsang et al. described the technique for US-guided pericardiocentesis in 1998. [30] The ideal site of skin puncture is where the largest area of fluid accumulation is closest to the skin surface. On US, this is demonstrated by visualizing a large anechoic area at the top of the screen (which is the body area

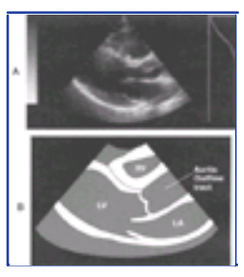
Figure 69-11 *A,* Sonographic appearance of a normal heart and pericardium obtained through the parasternal long axis window. This view is an excellent imaging window for posterior effusions. *B,* Schematic representation.

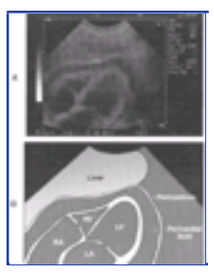
Figure 69-12 *A,* Sonographic appearance of a circumferential pericardial effusion obtained through the subxiphoid window. The effusion is seen as an anechoic stripe surrounding the heart. *B,* Schematic representation.

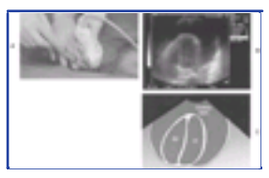
Figure 69-13 Photograph (*A*), sonographic image (*B*), and schematic representation (*C*) demonstrating the sonographic window that places the largest area of accumulated fluid nearest the probe (top of screen). There should be no vital structures between the probe and the pericardial space when the aspirating needle/catheter is placed over the superior border of the rib closest to the anechoic area. *(Lydia F. Roberts, Photographer)*

closest to the probe) and usually corresponds to the left anterior chest wall (rather than the subcostal region) (Fig. 69-13*A*,*B* , and *C*). In addition to being closer to the skin surface, this approach avoids injury to the liver. Inadvertent puncture of the lung is also prevented using this approach, because air in the lung will not conduct sound waves and will prevent visualization of the heart when located immediately beneath the probe. The clinician should avoid choosing a site that might puncture either the internal mammary artery, which lies 3 to 5 cm from the parasternal border, or the neurovascular bundle located at the inferior rib border. The best site should be marked with a sterile pen.

The needle trajectory and depth should be confirmed prior to skin puncture. Repositioning the patient will alter the position of the heart and pericardial sac within the chest and requires reassessment. The skin should be prepared antiseptically and a sterile cover placed over the probe. If time permits, the selected area should be anesthetized with 1% to 2% lidocaine, using the superior border of the adjacent rib as a landmark. The needle should ideally have an "over-the-needle" sheath that allows the needle to be withdrawn after the pericardial space is entered. This helps avoid injury to the heart and other vital structures. A 16- to 18-ga needle that is 5 to 8 cm in length is ideal. A saline-filled syringe should be attached to the needle, and gentle aspiration applied while the needle is advanced. The US probe can remain on the chest wall immediately adjacent to the aspiration site or removed after the fluid is localized.

Once the pericardial space is entered, agitated saline can be injected to confirm needle placement, particularly if the pericardial fluid is grossly bloody or there is any question concerning needle position. A saline echo contrast medium is prepared by using two 5 mL syringes, one with saline and the other air, connected via a three-way stopcock to the needle catheter sheath. Saline in one syringe is rapidly injected between the syringes and then injected into the sheath. Entrance of the agitated saline into the pericardial space can be monitored sonographically, appearing as a brightly echogenic stream.

After confirmation of needle placement, a wire can be passed through the sheath and a dilator (6 to 8 Fr Cordis) placed over the wire. The dilator is removed and an introducer sheath-dilator (6 to 8 Fr Cordis) placed over the wire. Both the wire and the sheath can then be removed and the introducer sheath left in place. The pigtail angiocatheter is inserted through the introducer sheath, and fluid aspirated to confirm placement [30] (see Chapter 16).

Complications

The potential complications associated with US-guided pericardiocentesis are the same as those with blind aspiration and are discussed in detail in Chapter 16 . The use of US will significantly minimize the risk of many of these complications, especially inadvertent puncture of the myocardium, epicardial vessels, and liver. [24] [30] Additional complications uniquely associated with US are due to misinterpretation of the sonographic image. For example, epicardial fat pads are common in obese patients and can be misinterpreted as clot or fluid within the pericardial space. [24] Although they have a similar echogenicity, there are a number of important findings that can aide in differentiating an epicardial fat pad from clot or fluid in the pericardial space.

First, the epicardial fat pad is an anterior structure. Clot in the anterior pericardial space suggests that the effusion is circumferential, and therefore should also be seen in the dependent portion of the pericardial space. This is best

demonstrated using the parasternal long axis view. Alternatively, the probe can be aligned longitudinally so that the inferior vena cava is visualized as it enters the right atrium. The right side of the heart can be seen adjacent to the diaphragm, and blood or fluid within the pericardial sac can be readily identified (as long as it is not loculated).[24]

Second, the inferior vena cava should collapse when the patient sniffs; collapse of less than 50% indicates increased intrathoracic pressure and possibly tamponade. Third, blood clotting is a dynamic process, with clots continuously forming and being broken down. If blood is present within the pericardial sac, careful examination should reveal fronds of clot waving within an anechoic (black) pericardial space. Finally, and most importantly, an anterior fat pad should not cause collapse of the right ventricular free wall. If after careful examination doubt still exists as to the presence of an effusion, hemodynamically stable patients should have a formal echocardiogram or computed tomography scan performed.

Also remember that fluid within the pericardial space is not always pathologic. A small (<0.5 to 0.9 cm) effusion may not be clinically significant, and the sonographer should exercise caution in over reading an effusion, particularly when the patient is hemodynamically stable. The patient who is hemodynamically compromised by a pericardial effusion should have a sizable effusion (the size can vary) with diastolic collapse of the right ventricular free wall, septal bulging, and dilation of the hepatic veins and inferior vena cava. The heart is usually beating rapidly with hyperkinetic wall motion, and may appear to swing to and fro within the pericardial sac.

Transvenous Pacemaker Insertion (TVPM)

Transvenous cardiac pacing can be life-saving in patients with symptomatic bradycardia or heart block. Optimal conditions for placement of a TVPM are the presence of forward flow to float the catheter into the right ventricle and fluoroscopy to guide placement. Unfortunately, ED patients who require emergent pacing are usually hemodynamically compromised by their rhythm and have poor forward flow. In the absence of flow, pacing wires may fail to pass through the tricuspid valve, coil in the right atrium, or fail to make contact with the right ventricular wall. Bedside sonography can readily demonstrate the passage of the pacing wire into the right ventricle, confirm contact with the right ventricular myocardium, and verify successful capture of the ventricle.

Background

Success rates for blind TVPM insertion with capture of the heart are highly variable, ranging from 10% to 90%. [32] [33] [34] Reasons for failure are often difficult to determine without adjunctive imaging, but malposition of the pacemaker wire is likely to play a role. US can readily demonstrate not only the passage of the pacing wire into the right ventricle, but also confirm contact with the myocardium. In two case series, bedside sonography was extremely helpful in guiding pacemaker wire placement and demonstrating capture in patients with hemodynamically significant bradyarrhythmias. [32] [33] Additionally, US can detect complications of blind pacemaker placement, including malpositioning of pacemaker wires and intraventricular septal perforation. [32]

Indications and Contraindications

Patients with symptomatic bradycardia or heart block for whom the placement of a TVPM is clinically indicated are excellent candidates for US-guided pacemaker wire placement. While there are no absolute contraindications, the presence of obesity or hyperinflated lungs (e.g., patients with chronic obstructive pulmonary disease) may interfere with visualization of the heart and pacing wire.

Equipment

A 2 to 3.5 MHz probe should be used to optimally visualize the right ventricle.

Procedure and Technique

The techniques for placing a TVPM are described in Chapter 15 . When using US guidance, the subxiphoid view is the best imaging window because it provides excellent views of the four cardiac chambers, pericardial space, and overall cardiac wall motion. In order to minimize interference with the procedure, it is best performed with the patient supine and with the probe placed distant from the Cordis introducer. Pacing catheter wires are highly reflective of sound waves and appear as bright linear echoes within the heart (Fig. 69-14*A* and *B*). The pacing wire can be tracked as it passes into the right ventricle and placed in the right ventricular apex. Myocardial capture is readily demonstrated sonographically as rhythmic contractions of the heart.

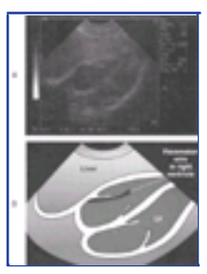

Figure 69-14 Pacing wire within the right ventricle. *A,* Songraphic image. *B,* Schematic representation. The right ventricle is seen through the subxiphoid window, which provides excellent views of the heart without interfering with pacemaker line placement. The pacemaker wire is seen as a brightly echogenic structure within the right ventricle.

Complications

Complications of TVPM insertion are discussed in Chapter 15 . There have been no reported complications specific to the use of US, although experience to date is limited.

Thoracentesis

Clinicians typically have localized fluid within the pleural space by percussion of the patient's chest and listening for dullness. This can be unreliable and inexact, particularly in obese or very muscular patients. US is an excellent tool for identification and aspiration of a pleural effusion, and can detect even small amounts of fluid within the pleural space. It has been shown to be at least comparable, and in some studies superior, to chest radiographs in the detection of pleural fluid. [35] [36] [37] [38] [39] US offers several advantages to the ED management of pleural effusions. The examination is rapid, can be performed at the bedside with suboptimal patient positioning, and percutaneous insertion of the needle is not blind. This reduces the risk of inadvertent puncture of a vessel, lung, or subdiaphragmatic organs.

Background

The use of US to identify the presence of pleural fluid and to guide aspiration is not new, and has become routine for pulmonary and critical care practitioners managing pleural effusions. Prior to the use of US, complications associated with blind thoracentesis included pneumothorax, dry aspiration, inadvertent liver or splenic lacerations, and subcutaneous hematoma. These complications are significantly reduced when performed under sonographic guidance. [40] [41] In addition, US can readily distinguish between tissue masses and fluid in patients with chest radiographs that show a "white lung" or opacifications that do not layer freely.

Indications and Contraindications

Any patient with a suspected pleural effusion is a potential candidate for sonographic imaging. Patients who are at greater risk for complications, such as those with small effusions, underlying malignancies, or for whom there are concerns regarding whether the fluid is free flowing, are ideal candidates. There are no absolute contraindications to the use of US. However, the study may be limited when there is subcutaneous emphysema or massive obesity.

Equipment

A 3.5 to 5.0 MHz probe can readily identify most pleural effusions.

Image Interpretation

In the normal chest, the interface between the air-filled lung and the visceral pleura is highly reflective, and blocks further transmission of sound waves into the chest. The image on the screen will appear as a pattern of bright echoes, which are due to reverberation artifacts. If pleural fluid is present, there will be an anechoic area indicating fluid above the diaphragm, which appears as a brightly echogenic curved line above the liver and spleen (Fig. 69-15*A and B*). Dynamic signs of pleural fluid include flapping movement of the atelectatic lung within the fluid, anechoic appearance of the fluid, and visualization of internal septa or intrapleural debris. [40] [42]

Procedure and Technique

Patient preparation, needle insertion, and fluid removal are discussed in Chapter 9 . All patients should have a chest

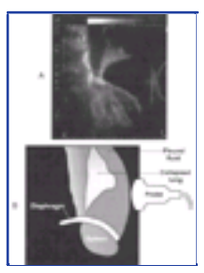

Figure 69-15 Sonographic appearance (*A*) and schematic representation (*B*) of a pleural effusion. Fluid will appear as an anechoic area over the brightly echogenic diaphragm. The collapsed lung can usually be seen as a wedge-shaped echogenic structure moving with respirations within the fluid. Note that the sonographic image has been rotated 90° clockwise to match the schematic drawing.

radiograph prior to starting the procedure. This will demonstrate the presence and general location (e.g., left or right pleural space) of the fluid. The optimal position for thoracentesis is to have the patient sitting with arms resting on a bedside table. Fluid will then collect in a dependent position. The probe should be positioned on the chest wall at the midaxillary line, above either the spleen or the liver (Fig. 69-16). The lung should be seen moving freely within the fluid as the patient breathes. The diaphragm must be visualized to avoid puncture of the liver or spleen; this may be especially difficult on the left side where there is no optimal acoustic window. The kidneys can usually be easily seen and then the probe moved superiorly to identify the diaphragm. The pleural fluid will be seen adjacent to the diaphragm. For large effusions, the thoracentesis needle can be advanced over the rib that is immediately adjacent to the fluid. If the fluid pocket is small, then the aspiration can be performed under directed sonographic guidance by covering the transducer with a sterile glove/cover and puncturing the chest wall directly alongside the transducer.

Complications

Complications of thoracentesis are discussed in Chapter 9 . While rare, complications directly related to use of US may occur in complicated effusions secondary to image misinterpretation. Echogenic material within the fluid can suggest the

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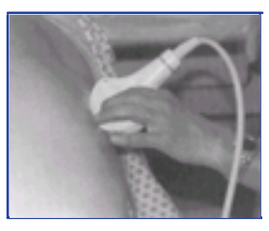

Figure 69-16 Probe positioning for identification of a pleural effusion. The probe should be placed longitudinally over the lateral or posterior chest wall on the side of the suspected effusion. The probe should be moved inferiorly to identify the kidney, liver or spleen, and diaphragm. *(Lydia F. Roberts, Photographer)*

presence of clot, pus, and even bowel depending on the sonographic appearance. Solid masses with few internal echoes may appear as a loculated effusion and care should be taken to identify free fluid. Fortunately, most masses adjacent to the pleura do contain echoes that readily distinguish them from free fluid.

Paracentesis

US is a fairly sensitive tool for identification of fluid within the peritoneal cavity and has virtually replaced diagnostic peritoneal lavage (DPL) in patients with traumatic injuries and suspected hemoperitoneum.[43] In such patients, demonstration of free fluid is presumed to be blood. Most patients will subsequently undergo exploratory laparotomy or computed tomography scanning. Patients requiring paracentesis in the ED usually have atraumatic peritoneal fluid collections. The benefits of paracentesis for these individuals include diagnosis of spontaneous bacterial peritonitis, differentiating transudative from exudative effusions, and therapeutic aspirations for patients with large amounts of ascitic fluid (see Chapter 44).

Background

US is the diagnostic study of choice for demonstration of intraperitoneal fluid. The primary use in the ED is for identification of patients with intraperitoneal blood secondary to both blunt and penetrating trauma. [43] [44] [45] [46] [47] Demonstration of free fluid in these patients usually leads to exploratory laparotomy or computed tomography scanning to further delineate the extent of intra-abdominal injury and rarely results in paracentesis. Patients requiring paracentesis in the ED are generally those with nontraumatic fluid collections requiring either a diagnostic or therapeutic tap. Many of these patients have end-stage liver disease or underlying malignancy. Although relatively rare, intraperitoneal hemorrhage, abdominal wall hematomas, and bowel perforation are reported complications of blind paracentesis (see Chapter 44). Risk factors for complications include coagulopathic states, obesity, small amounts of fluid, and prior surgery. US can minimize these risks by identifying the largest pocket of readily accessible fluid. [48] [49]

Indications and Contraindications

The indications for therapeutic and diagnostic paracentesis are discussed in detail elsewhere (see Chapter 44). Specific indications for sonographic imaging are patients without a known diagnosis of ascites, obesity, patients with prior abdominal surgeries, or failed blind aspirations. US is the study of choice for identification of

ascites, and can readily establish the presence of intraperitoneal fluid. It can also show the size of the collection and identify the best site for aspiration. Ascitic fluid in patients with prior abdominal surgeries may be loculated, and US can identify areas of free-flowing fluid.

Equipment

A 3.5 to 5.0 MHz probe can readily identify free fluid within the peritoneal cavity.

Image Interpretation

Free fluid will appear as an anechoic, black, sharp-edged collection in the most dependent area of the peritoneal cavity. In contrast to the usual "snowstorm" appearance created by the air-filled loops of bowel, large amounts of fluid will appear to fill the peritoneal cavity and floating loops of bowel will be distinct (Fig. 69-17). Blood or pus within the fluid will create echogenic shadows. In addition, pus will usually layer out, creating an echogenic stripe in the dependent portion of the fluid. Blood clots will also layer or appear as fronds floating within the fluid. Structures within the peritoneal cavity, particularly bowel and bladder, can be easily identified and avoided during needle aspiration.

Procedure and Technique

The patient can be imaged in the supine or reverse Trendelenburg position to allow flow of smaller amounts of fluid into the pelvis. A full bladder can be confused with an intraperitoneal fluid collection and should be emptied prior to

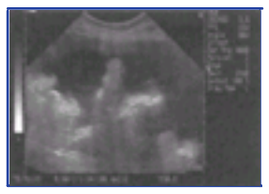

Figure 69-17 Sonographic appearance of peritoneal free fluid. Fluid will appear as a large anechoic area with loops of bowel floating freely. The optimal area for needle insertion is where there is a large pocket of fluid just under the skin surface, closest to the probe.

the procedure. The liver, spleen, bladder, and loops of bowel should be identified to avoid inadvertent puncture. For the best result, the largest pocket of free fluid should be located. This is usually found in the lower quadrant of the abdomen and will appear as a black, anechoic area. Once this area is identified, paracentesis is performed as described in Chapter 44 , with the needle placed directly over the area adjacent to the fluid and carefully avoiding the bladder and inferior epigastric arteries.

Complications

Complications directly related to the use of US are rare, and likely to be due to misinterpretation of the images. A full bladder or large cystic masses within the abdomen (e.g., ovarian) may be confused with intraperitoneal fluid. A clue to differentiation of fluid contained in either the bladder or a cyst is that ascitic fluid will have an irregular appearance due to bowel indentation, whereas the bladder or cyst will have a smooth curved surface outline. In addition, free intraperitoneal fluid will collect dependently, and will have a sharp-edged appearance when bounded by peritoneal reflections. In contrast, fluid contained in the bladder or cyst will have rounded edges.

Joint Aspiration

Joint pain is a common complaint in the ED. Identification of fluid within the joint space has important implications for both diagnosis and treatment. For the patient presenting with a warm, swollen joint, it is often difficult to differentiate cellulitis, soft tissue abscess, or septic/inflammatory arthritis by physical examination alone. US has been widely used by rheumatologists as an adjunct to their clinical examination to define joint and synovial pathology and monitor response to treatment. [50] [51] In the hands of the ED clinician, it can aid in identification of clinically significant fluid within the joint, identify extra-articular sources of pain, and guide aspiration of joint spaces.

Background

US can demonstrate the presence of as little as 1 mL of intra-articular fluid in any joint space that has a sonographic "window," in particular the hip, shoulder, knee, ankle, and wrist.[52] [53] [54] [55] [56] Although some joints, such as the knee, are readily aspirated using the blind approach, other joints are often difficult to aspirate successfully and may require multiple attempts. US will not only confirm the presence of fluid, which in itself is of clinical value, but can also identify the optimal site for joint aspiration.

Additional benefits to sonographic imaging of the joints include characterization of the joint fluid (see later in this section) and identification of extra-articular sources of pain. When no fluid is seen within the joint space, and the patient has pain, swelling, or both, it can be assumed that the source of discomfort is outside the joint space (e.g., involving skin, tendon, bone, ligament, or extra-articular bursa). In experienced hands, US can also diagnose disease of the periarticular tissues. [52]

Indications and Contraindications

Sonographic examination of the joint is indicated whenever identification of fluid within the joint space is clinically relevant. This includes patients presenting with acute joint pain due to trauma, patients with suspected inflammatory or infectious arthritis, and nontraumatic hemarthrosis, particularly those associated with coagulopathic states. There are no reported contraindications to the use of US.

Equipment

The most commonly used probe for assessment of the joint space is a high-frequency linear array probe (7.5 to 10 MHz).

Acoustic Windows

A detailed discussion of the sonoanatomy of all joints is beyond the scope of this section. The knee, shoulder, and ankle are most often involved in traumatic and inflammatory conditions, and the sonographic windows for these joints are described as follows.

The knee

Knee effusions are often difficult to reliably identify on physical examination alone, particularly when the patient is obese or when severe pain limits the examination. The knee should be flexed (approximately 20°) and a roll placed behind the popliteal fossa. The anterior views of the knee begin with a longitudinal scan above the patella, which is the bony landmark for imaging the suprapatellar bursa. [52] [53] The suprapatellar bursa communicates with the knee joint, extending approximately 6 cm above the patella just deep to the quadriceps tendon (Fig. 69-18). The medial and lateral recesses of the knee (extensions of the suprapatellar bursa) should then be viewed in longitudinal and transverse orientation. In a normal joint, the bursa is a thin hypoechoic line no more than 2 mm thick. Fluid within the bursa will appear as an anechoic stripe beneath the quadriceps tendon (Fig. 69-19A).[52] [55] Extra-articular fluid commonly located in the prepatellar bursa can also be readily identified as a fluid collection anterior to the patella.

The shoulder

The shoulder joint can be imaged using anterior or posterior windows. The anterior window, easiest to acquire, consists of longitudinal and transverse scans of the long head of the biceps tendon (Fig. 69-20). The biceps tendon has a synovial sheath, which is an extracapsular extension of the joint synovium. Consequently, fluid within the joint is often demonstrated within the synovial sheath (Fig. 69-21*A* and *B*). For the anterior window, the patient is placed in the sitting position with the elbow adducted and the hand palm-up. Alternatively, the patient can lie prone on a gurney with the arm hanging down. [52] The posterior window is performed with the patient prone or seated leaning forward with the hand on the affected side placed on the opposite shoulder to open up the joint space (Fig. 69-22).

Fluid will preferably accumulate in the posterior recess due to capsular thinning and minimal adjacent structures to prohibit distention (Fig. 69-23*A*, *B*, and *C*). This fluid is usually seen first in the anterior window, but the posterior aspect of the shoulder can be imaged to guide aspiration if that is the preferred approach.

The ankle

The foot should be positioned in plantar flexion, and the transducer oriented longitudinally over the anterior surface of the tibia (Fig. 69-24). Moving the transducer toward the foot and seeing the curved dome of the talus will identify the joint space. An effusion will appear as an increased anechoic space at the anterior aspect of the tibio-talar joint (Fig. 69-25*A*, *B*, and *C*). [52] [54] [56] [57]

Image Interpretation

Three basic types of effusions will be encountered when performing bedside sonography: noninflammatory, post-traumatic,

Figure 69-18 *A,* Probe positioning for sonographic imaging of the suprapatellar bursa. The probe should be placed in the longitudinal position superior to the patella. Following identification of fluid within the bursa, the probe can be rotated transversely to identify the medial and lateral extent of the fluid. *B,* Schematic representation. *(Lydia F. Roberts, Photographer)*

and inflammatory. Simple, noninflammatory joint effusions will appear as an anechoic or hypoechoic area within the joint space. Traumatic effusions are typically complicated by the presence of either free-floating clot or fat lobules. These effusions will have a heterogeneous appearance with echogenic particles or fronds (clot) floating within the hypoechoic joint

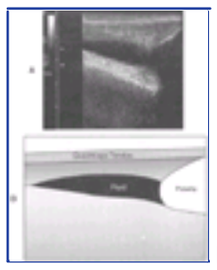

Figure 69-19 *A,* Sonographic appearance of a suprapatellar effusion. Fluid will appear as an anechoic area superior to the patella and beneath the quadriceps tendon. *B,* Schematic representation.

space. In both septic and inflammatory arthritis, the fluid often appears hypoechoic with internal echoes, giving it a particulate appearance. [51] [52] Layering may be seen whenever there is pus, blood, or excess lipid within the effusion.

Procedure and Technique

Arthrocentesis is described in Chapter 54 ; US can be used to locate the effusion and confirm optimal needle placement. Joints should be imaged using a variety of sonographic windows. It is helpful to move the joint under direct sonographic guidance to aid in identification of intra-articular and periarticular structures. Compression of the joint is extremely

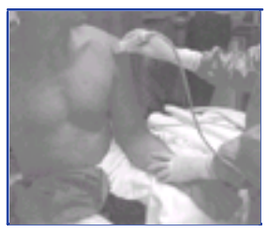

Figure 69-20 Probe positioning for the anterior view of the shoulder joint. The patient's arm should be placed palm up and the elbow flexed and supported. The arm should be rotated externally. The probe should be placed in the transverse position over the anterior aspect of the shoulder and the biceps groove visualized. *(Lydia F. Roberts, Photographer)*

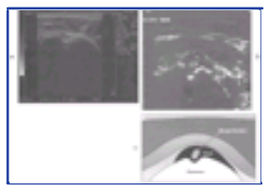

Figure 69-21 Sonographic appearance of the anterior shoulder. *A,* Normal; *B,* Effusion present; *C,* Schematic representation. Fluid within a shoulder will appear as an anechoic area surrounding the biceps tendon within the biceps groove. *(Courtesy of Verena Valley, MD.)*

useful in differentiating fluid (or clot) from tissue or cartilage. The affected joint should always be compared to the contralateral (normal) side by use of the multi-image function on the US machine. Once the effusion is identified, the needle should be placed over the area adjacent to the fluid collection. For small effusions, continuous imaging during the procedure can confirm needle entry into the joint. Alternatively, the location of the effusion can be marked with a sterile marking pen.

Complications

The most common pitfall associated with joint imaging is failure to recognize artifacts common to musculoskeletal US. Knowledge of these artifacts can aid in identification of important pathology (such as reverberation artifact for foreign bodies), but may be problematic if confused with actual structures. Although many important artifacts have already been discussed, one that is most common to musculoskeletal imaging is due to anisotropy of joints and ligaments. This refers to the fact that linear structures (such as tendons and ligaments) must be imaged perpendicular to the plane of the US beam to demonstrate their true echogenicity. [52] [58] [59] Failure to do so will give them a falsely hypoechoic appearance, which may be misleading. The relatively hypoechoic appearance of muscle or tendon surrounding most joints can also be confused with fluid. Fortunately, when an effusion is adjacent to bone, the difference in sonographic appearance of fluid and bone will usually minimize the risk of errors. In addition, movement of associated structures by the patient can be helpful in identifying anatomic structures, and comparison to the unaffected (normal) side can improve the accuracy of image interpretation. [52]

Foreign Body Localization

Detection of foreign bodies retained in soft tissues is often difficult, and missed or delayed identification may lead to infection, inflammation, or both. Of the available imaging modalities, plain radiography is the most widely used to screen for foreign bodies, but will only detect radiopaque objects. Computed tomography and magnetic resonance imaging are alternative imaging modalities that reliably detect most foreign bodies, but are costly, time-consuming, and often unavailable. US has been shown to be a potentially useful adjunct in detection of both radiopaque and radiolucent foreign bodies.

Background

Studies to date vary considerably in their conclusions regarding the utility of US in identification of subcutaneous foreign bodies. Accuracy of the sonographic examination is highly dependent on the composition of the foreign body, how long it has been embedded, composition of adjacent structures, and sonographer skill. For radiopaque substances (e.g., glass, metal), plain radiographs consistently identify a greater proportion of foreign bodies. [60] [61] Manthey et al. had two independent radiologists image 120 chicken thighs embedded

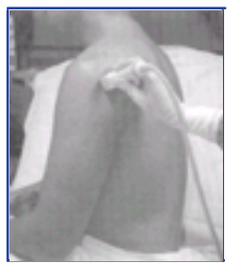

Figure 69-22 Probe positioning for the posterior view of the shoulder joint. The probe should be placed in the transverse position over the posterior aspect of the shoulder. The glenoid rim can be readily identified in this position. *(Lydia F. Roberts, Photographer)*

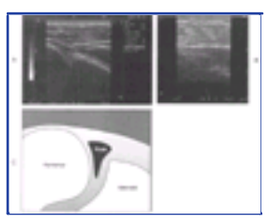

Figure 69-23 Sonographic appearance of the posterior shoulder. *A,* Normal; *B,* Effusion present; *C,* Schematic representation. Free fluid will appear as an anechoic collection medial to the glenoid rim. When fluid is identified in the anterior view, fluid can often be "milked" into the posterior space by moving the shoulder. *(Courtesy of Verena Valley, MD.)*

with either gravel, metal, glass, cactus spine, wood, or plastic. The overall sensitivity and specificity for US in detecting the foreign substances was 43% and 70%, respectively. No individual foreign body reached a US detection rate of 50%. Plain radiography demonstrated radiopaque substances (gravel, glass, metal) 98% of the time, but was unable to reveal any of the radiolucent substances (wood, plastic, cactus spines). [61]

US is superior to plain radiographs in identification of radiolucent objects, specifically wood, plastic, and cactus spines. [62] [63] [64] [65] [66] [67] [68] [69] One large study of high-frequency sonography demonstrated an overall accuracy of 82% (sensitivity 79%, specificity 86%, positive predictive value 85%, and negative predictive value 80%) when US was used to identify wooden foreign bodies embedded in chicken thighs. [70] Another experimental study compared sonography with magnetic resonance imaging in the evaluation of embedded wooden foreign bodies in cadavaric specimens. While the two imaging modalities were comparable in detection of foreign bodies, magnetic resonance imaging was superior to US when the substance was embedded near bone. [68] Other studies using various models (beef cubes, chicken thighs, cadaver hands and legs) and embedded foreign bodies (wood, glass, metal) have shown that while imperfect, sonography can identify radiolucent substances that may be missed on plain radiographs. [64] [66] [67] [68] [69] US appears to have a unique role in identifying foreign bodies that are radiolucent, and is most helpful when a foreign body is positively identified. When exclusion of a retained foreign body is critical and none is seen on US, it may be advisable to obtain an alternative imaging study.

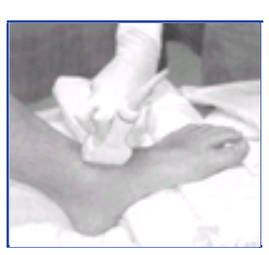

Figure 69-24 Probe positioning for assessment of ankle effusion. The foot should be positioned in plantar flexion, and the transducer oriented longitudinally over the anterior surface of the tibia. Moving the transducer toward the foot and seeing the curved dome of the talus will identify the joint space. *(Lydia F. Roberts, Photographer)*

Figure 69-25 Sonographic appearance of the ankle. *A,* Normal; *B,* Effusion present; *C,* Schematic representation. An effusion will appear as an increased anechoic space at the anterior aspect of the tibio-talar joint. The optimal site for needle placement is where there is an anechoic space immediately under the probe.

Indications and Contraindications

All patients with a suspected foreign body should have a plain radiograph performed initially. If the foreign body is readily visualized, then it is unlikely that sonography will provide any further useful information about the foreign body itself. However, it will demonstrate the presence of fluid around the foreign body and adjacent fluid-filled structures, such as vessels, that may be injured during the extraction process. If there is no clear evidence of a foreign body on plain radiographs, and there is clinical suspicion of a subcutaneous foreign body, then US imaging is indicated.

Equipment

A high-frequency (7 to 10 MHz) linear probe is best for localization of subcutaneous foreign bodies.

Image Interpretation

The appearance of a foreign body will depend on the orientation of the foreign body to the US beam, adjacent tissues, surrounding edema or abscess, and the density of the foreign body itself. In general, all foreign bodies are hyperechoic and will appear as brightly echogenic structures, distinct from surrounding bone or calcifications. Additional clues to the location of a foreign body include acoustic shadowing (for stone, wood, and plastic) and reverberation or comet-tail artifacts (for glass and metal objects) (Fig. 69-26*A* and *B*). Secondary signs associated with some soft tissue foreign bodies may also help in their identification. For example,

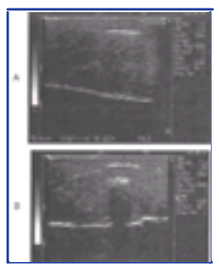

Figure 69-26 Sonographic appearance of a retained metallic foreign body (*A*) and a retained glass foreign body (*B*). Foreign bodies are usually hyperechoic, and will appear as brightly echogenic structures, distinct from surrounding bone or calcifications. Clues to the location of a foreign body will be acoustic shadowing and reverberation artifact.

in the acute setting, air may be trapped within the tract and appears as a brightly reflective trail to the foreign body. Hematomas will have a characteristic hypoechoic appearance. Substances that have been in place for longer than 24 hours may have surrounding edema, which will appear as a sonolucent area surrounding the hyperechoic foreign body. Wooden objects will initially appear as hyperechoic objects with acoustic shadowing, but if left in place will decompose and eventually lose their echogenicity. They are often best recognized by a localized area of inflammation that appears as a heterogeneous fluid collection.

Procedure and Technique

The probe is placed on the skin with a layer of acoustic gel, or for very superficial foreign bodies, a stand-off pad can be used. This is either a gel pad or a 100 to 250 mL bag of saline with the air removed that is placed between the probe and the skin. The use of a stand-off pad will eliminate near-field artifacts and bring the object into the transducer focal zone. The US beam should be angled perpendicular to the area of interest. A particular advantage of US is the fact that the examination can be focused over the entrance of the foreign body or area of maximal tenderness or erythema. The area should be scanned from a variety of angles because a foreign body can be easily missed if the lie is not perpendicular to the beam. Once the foreign body is identified, its relationship to surrounding structures such as bone and vessels should be documented. The exploring hemostat or forceps should be angled parallel to the US beam. [67] It is helpful to have an assistant hold the probe during the extraction procedure. See Chapter 37 , Chapter 38 , and Chapter 52 for specific foreign body removal details.

Complications

Complications directly related to the use of US are usually due to an over-reliance on US as a screening tool for detection of a foreign body. There are many reports of false-positive and false-negative scans, largely due to the presence of artifacts that can mimic a foreign body and failure to detect a foreign body, respectively. The brightly echogenic interfaces between bone and tissue, and vessels imaged "end-on" may be mistaken for a foreign body, and lead to unnecessary wound exploration.[68] Conversely, a foreign body that is isodense with the surrounding tissue, such as a retained wooden splinter, may not be seen. [64] [66] [67] [68] [69] The clinician must rely on clinical judgment and knowledge of potential artifacts when performing musculoskeletal US. In addition, appropriate discharge instructions and close follow-up are imperative.

Bladder Aspiration

Needle aspiration of the bladder may be required when a patient, usually an infant, is unable to provide a sterile urine specimen. The bladder is a superficial structure that is readily identified with US. The role of US is to localize the bladder, demonstrate that the bladder is full, and guide needle aspiration. [71] A secondary role of bladder sonography is to assess bladder volume, which may be particularly helpful when the patient is unable to provide a spontaneous urine sample and the differential diagnosis includes dehydration vs bladder outlet obstruction.

Background

The approach to blind suprapubic bladder aspiration requires localization of the bladder by percussion and palpitation. While relatively straightforward for most children and adults, it may be difficult in infants in whom the bladder is an intra-abdominal organ and in children who are agitated, volume depleted, or have abdominal distention. US has been identified as an extremely useful tool to locate the bladder, estimate bladder volumes, and guide suprapubic aspiration. [72] [73] [74] Recent studies have shown that US guidance increases the likelihood of successful bladder aspiration. In a study of 140 male infants less than 2 years of age, Ozkan et al. demonstrated a 90% success rate in the US-guided group compared with 64% ($p < 0.05$) in the blind aspiration group. [73] There were also fewer passes made in those patients who had the procedure done under ultrasonographic guidance. [73] Another study of similar design looked at the impact of bedside US on aspiration attempts in 59 infants. Aspiration was successful on the first attempt in 26 of 28 patients (93%) with US guidance, but in only 13 of 21 patients (62%) undergoing blind aspiration.[74] In these studies, complications in both the experimental and control groups were rare.

Indications and Contraindications

US-guided aspiration is indicated in the infant, child, or adult for whom blind aspiration is unsuccessful or where localization of the bladder may be difficult due to dehydration or abdominal distention. In the child who is agitated, clear localization of the bladder may facilitate the speed and success of the procedure. Adult patients with prior abdominal surgery or irradiation may have adhesions of the bowel to the bladder, and will also benefit from sonographic guidance. US can also

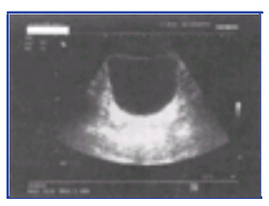

Figure 69-27 Sonographic appearance of the bladder in transverse view. The bladder will appear as an anechoic structure immediately beneath the skin surface, with a characteristic rhomboid appearance on transverse view.

be used to guide placement of cystostomy tubes in adults. There are no contraindications specific to the use of US.

Equipment

A 5 MHz curvilinear probe is usually adequate for pediatric patients. A lower frequency probe (3.5 MHz) should be used for adults.

Procedure and Technique

The bladder should be full for best results. The probe should be placed just above the pubic symphysis and the bladder imaged in both the longitudinal and transverse planes. The bladder will appear as an anechoic structure immediately beneath the skin surface, with a characteristic rhomboid appearance on transverse view (Fig. 69-27). A layer of acoustic gel and a sterile cover should be placed over the probe. A second layer of sterile gel should be placed between the probe cover and the skin surface. The skin should be punctured immediately above the portion of the bladder closest to the skin surface (at the top of the imaging screen). The site can either be marked with a sterile pen prior to skin puncture, or the probe left in place to guide aspiration. For a complete discussion of suprapubic aspiration, see Chapter 56 .

Complications

General complications of bladder aspiration are discussed in Chapter 56 and include viscus perforation, microhematuria, and unsuccessful aspiration. Complications directly related to the use of US are rare, but may occur if aspiration is attempted and the bladder is not full or if there is free fluid in the peritoneal cavity that is mistaken for urine in the bladder. In addition, the outlines of the bladder wall may be difficult to identify when there is peritoneal fluid, particularly if the fluid is loculated. In general, bladder fluid has a curved outline, whereas fluid within the peritoneal cavity will be indented by loops of free-floating bowel.

Fluid Collections and Abscesses

US is a very useful adjunct for identification of subcutaneous masses. It can readily differentiate between solid and fluid-filled masses, estimate the size and depth of the fluid collection and demonstrate the presence of septations or foreign bodies. [51] [65] This is often of clinical value, particularly when the examination suggests a large mass and aspiration is unsuccessful or limited. [75] US can also provide critical information regarding the extent of the collection and proximity to important structures such as blood vessels and vital organs.

Background

US is widely used by both radiologists and rheumatologists to characterize soft tissue collections, which include subcutaneous abscesses and hematomas. It has proven value in differentiating subcutaneous fluid collections from solid masses and identifying the presence of foreign bodies (particularly wood) that may serve as a nidus for infection. [51] [56] [65] It is a logical adjunct to the assessment of subcutaneous masses in the ED, especially when percutanous aspiration or incision and drainage is planned.

Indications and Contraindications

Any subcutaneous mass should be imaged when demonstration of a fluid collection is of clinical significance. It is particularly important when the collection is near vital structures, the extent of the collection is difficult to assess due to location or body habitus, and when prior attempts at aspiration have been unsuccessful. There are no contraindications to the use of US for this purpose.

Equipment

The depth and size of the collection will determine optimal probed selection. Large or deep collections should be imaged with a 3.5 to 5.0 MHz probe. Small or superficial collections will be best seen with higher frequency probes.

Image Interpretation

In the early stages of abscess formation, the affected tissue will contain an increased amount of fluid, which will appear as horizontal bands of hypoechoic or anechoic areas. As the abscess forms, the central area liquefies and the surrounding tissue forms a wall of scar tissue. Sonographically, an abscess appears as a discrete anechoic area often surrounded by a brightly reflective wall (Fig. 69-28). The fluid will usually be black, but will often contain scattered echoes representing purulent material or necrotic debris. The fluid may contain septations, which appear as echogenic walls that traverse the fluid cavity.

Procedure and Technique

Imaging should be performed in both the longitudinal and transverse planes to demonstrate the full depth and extent of the fluid collection or mass. Surrounding structures, particularly blood vessels, should be identified prior to aspiration or incision and drainage. The use of Doppler (either color or power) to demonstrate absence of flow within the collection can add a measure of safety (but is not required). The depth of the collection can be determined by using the depth markers on the right side of the monitor screen. Specific procedural details for drainage of fluid collections are provided in Chapter 38 .

Complications

While image interpretation is usually straightforward, complications may occur due to misinterpretation of like images. Cellulitic areas will have a heterogeneous, layered appearance (alternating bands of anechoic and echogenic layers) that may be mistaken for a discrete fluid collection. Blood vessels, when imaged in the transverse plane, can appear as a cystic fluid collection, undermining the importance of imaging the collection in both the longitudinal and transverse planes. It must also be remembered that fluid collections do not

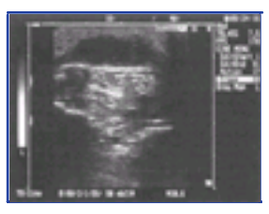

Figure 69-28 Sonographic appearance of a subcutaneous fluid collection. An abscess appears as a discrete anechoic area that may be surrounded by a brightly reflective wall. The fluid will be anechoic, but will often contain scattered echoes representing purulent material or necrotic debris. The fluid may also contain septations, which appear as echogenic walls traversing the fluid cavity.

always indicate abscess, and may be Baker cysts, bursae, or hematomas. Clinical judgment must be exercised during image interpretation.

SUMMARY

US has become a valuable adjunct to the practice of emergency medicine and the spectrum of procedures amenable to sonographic guidance is rapidly expanding. The procedures described in this chapter reflect proven efficacy to the role of emergency US. The principles and approaches described can also be broadly applied to any procedure involving percutaneous cannulation or aspiration of a fluid-filled body cavity. It is likely that future texts will describe an expanded role for sonography in the ED.

Although sonography is now within the scope of practice of many emergency clinicians, care must be taken by the individual to understand the limitations of the sonographic examination and obtain appropriate training before using it to guide clinical practice. Additionally, nonemergency clinician sonographers can and should be used to provide sonographic guidance for procedures when there is no skilled sonographer in the ED, or when there are concerns with the examination performance or interpretation.

Acknowledgment

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Chapter 70 - Bedside Laboratory and Microbiologic Procedures

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ASSESSMENT OF URINE

Obtaining a Urine Specimen

Several methods are available for obtaining a urine specimen. These can be listed in order of increasingly precise collection techniques, which tend to come at the cost of increasing difficulty and/or patient discomfort:

1. *Random voided:* Any specimen provided by the patient.
2. *Midstream voided:* No skin preparation, container placed in urinary stream 2 to 3 seconds after the initiation of micturition.
3. *Clean catch:* Same as item 1, with antiseptic cleansing of the urethral area. In males this is performed by retraction of the prepuce (the labia in females) and cleansing of the meatus in an anterior to posterior direction using three swabs soaked in Betadine (or some other antiseptic solution). For female patients who are physically capable, the ideal position is sitting astride a toilet, facing backward. This helps to separate the labia and position the cup for specimen collection.
4. *Midstream clean-catch* specimen (MSCC): Cleansing as in item 3, with midstream collection as in item 2.
5. *Catheterized:* Obtained from a newly placed catheter.
6. *Suprapubic aspiration* (SPA) (see Chapter 56).

The low levels of bacteriuria induced in 2% to 8% of patients after straight catheterization [262] are generally clinically insignificant. Catheterization does cause minor local injury, as reflected by bacteriuria and low-level hematuria (1 to 3 red blood cells [RBCs]/high-power field [hpf]) in =15% of patients. [103] [262] SPA continues to be advocated by some for neonates in cases in which accurate diagnosis is essential and the risk of infection must be minimized. SPA (see Chapter 56) has a 10% "failure rate" (i.e., no urine obtained after three attempts) in most series and may actually take longer than most methods, especially when multiple attempts are considered. SPA can also cause spuriously lowered leukocyte or bacterial colony counts due to the necessity of maximally filling the bladder before performing the procedure.[215] Surprisingly, older men who underwent both catheterization and SPA strongly preferred the latter procedure because it caused less discomfort. [164]

Recommendations for Urine Collection

Since the mid-1950s there have been proponents and opponents for each technique. In choosing a method for obtaining a urine specimen, the disease under consideration affects both the interpretation of the results and the quality of the specimen required. For the purposes of this discussion, the diagnostic categories can broadly be divided into infectious and non-infectious. With the exception of testing for blood, most of the non-infectious tests (e.g., ketones, glucose, bilirubin, protein) are not affected by collection method. Since the detection of infection is the most common purpose of urine testing in emergency practice, this will be considered first.

Urinary tract infections (UTI) are either symptomatic or asymptomatic. The group to which a patient belongs is the first step in determining which collection method is required (Fig. 70-1). The typical emergency department (ED) patient is in some way "symptomatic." This includes patients with classic signs and symptoms of UTI (e.g., urgency, frequency, dysuria) as well as those with systemic signs of infection (e.g., fever and chills) who are unable to accurately report on symptoms. [263] In addition, ED clinicians cannot afford to miss detecting asymptomatic bacteriuria in high-risk patients (e.g., the immunocompromised, neonates and infants, pregnant patients, diabetics), or in those who require urine cultures because of a history of relapsing, recurrent, complicated, or childhood UTIs. [141]

Whether the nonspecific findings of undifferentiated abdominal pain or fever constitute "symptoms" of UTI has never been studied, although they are frequent indications for urine testing in the ED.[263] However, to interpret tests for UTI it is necessary to estimate the extent to which the patient's symptoms are urinary in origin (making it a test of the "symptomatic" patient), versus non-urinary (making it a screening test). This is because in symptomatic UTI, extremely low levels of bacteriuria (10^2 CFU/mL) and pyuria are of clinical significance.[149] [154] [255] [257] This fact has been obscured by studies on *asymptomatic* patients showing that the threshold for "significant bacteriuria" is =10^5 CFU/mL.[124] [125] [199] Furthermore, specimens are not necessarily contaminated if multiple pathogens are isolated. Up to 50% of symptomatic women may have polymicrobial infections.[255] [257]

It behooves the clinician to obtain specimens as carefully as possible, because contamination is most likely to produce the finding of low-level or polymicrobial bacteriuria, which may be clinically significant in symptomatic patients. Although several studies have failed to demonstrate a statistical difference in contamination rates with various urine collection techniques, most studies show trends in favor of more meticulous and/or invasive collection methods. [110] [163] [164] [166] [226] [275] In cooperative, motivated males and females with symptoms of uncomplicated lower UTI or pyelonephritis who are capable of diligently performing the necessary maneuvers, an MSCC specimen is as accurate as a catheterized specimen, especially when the possibility of urethral and/or prostatic trauma and patient discomfort are considered.[164] In females in whom symptoms are equivocal, and in all patients unable to provide an MSCC specimen, a catheterized specimen is usually warranted.

If no symptoms of UTI are present, the urine examination can be considered as a "screening" test. In the asymptomatic patient, routine screening for bacteriuria is unwarranted in all but two clinical situations: pregnant women and all patients for whom urologic surgery has been scheduled. [209] [256] If only a urine culture is to be performed, some would argue that any spontaneously voided specimen would suffice, since the diagnosis of asymptomatic bacteriuria depends on =10^5 CFU of a single pathogen per milliliter of urine, and with such criteria, contaminants are usually easily identified. [110] However, performing cultures in all such patients is prohibitively costly, so that dipstick and/or urinalysis (UA) is commonly used for screening. [169] [178] With these tests, contamination by bacteria, leukocytes, or erythrocytes can still cause diagnostic

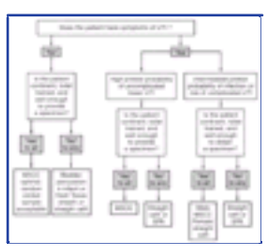

Figure 70-1 Algorithm for deciding method of obtaining urine specimen for evaluation of possible urinary tract infection. MSCC, midstream clean catch; SPA, suprapubic aspiration; UTI, urinary tract infection.

confusion. Thus, the advantages of a less contaminated specimen are worth the minimal extra effort of asking the patient to provide an MSCC sample.

The approach described in Figure 70-1 covers the vast majority of situations. A few circumstances and techniques deserve special mention.

Bladder Percussion and the Midstream Specimen in Infant Males

The emergency clinician is familiar with how frequently a urine stream is generated in infants who are confronted by the alarming ED environment and a cold stethoscope. Rather than wasting a potentially perfect MSCC specimen on a laboratory coat, with an ensuing delay in obtaining urine, the emergency clinician can exploit this response by approaching the patient with an open sterile urine container in hand in case of a spontaneous micturition. The process is facilitated with the application of cold povidone-iodine to the genitalia. Such an approach has been shown to generate a urine sample in a median time of 10 minutes. [5] This is much less than the typical time needed for straight catheterization or suprapubic aspiration, especially since it can be performed concomitant with the history and physical examination. It also circumvents an invasive procedure, with its associated complications, resource expenditures, and stress to both patient and staff. If the urine specimen is not immediately forthcoming, a parent can be instructed to catch the specimen in the sterile container, freeing up ED staff for other tasks.

Two techniques to actively induce voiding in infants have been described. The first, which is useful in newborns, exploits the Perez reflex. [24] After cleansing of the genitalia (with povidone-iodine or some equivalent non-irritative antibacterial agent), the infant is held in one hand while the paraspinal muscles are stroked, cephalad to caudad. This causes extension of the back, and flexion of the hips and induces micturition in less than 5 minutes in the majority of cases. [24]

The second technique is known as "bladder tapping." After urethral cleansing, if there is still no urine, two fingers may be tapped on the suprapubic area at a rate of approximately once per second for a full minute, followed by a minute's rest. The cycle is then repeated until urine is produced. The mean time prior to the production of urine is about 5 minutes. This technique, while not practicable for the staff in a busy ED, can provide an infant's parents with a task that invests them in the clinical process.[266]

Bag Collection in Non-Toilet-Trained Children

The incidence of unsuspected UTI in the febrile neonate or infant is about 3% to 5%. Because a true UTI in an infant or child requires a subsequent evaluation for urinary tract pathology, and the disease may produce significant morbidity (e.g., hypertension, renal disease), one must be certain of the presence or absence of infection in this subgroup. Numerous studies demonstrate the disutility of urine specimens obtained for culture from a collection bag stuck to an infant's perineum. [7] [24] [26] [58] [95] [266] Bag specimens obtained for a routine UA in suspected UTI are equally unreliable, with white blood cell (WBC) concentrations routinely 25-fold higher and bacterial counts 1000-fold higher than in a catheterized specimen. [7] In the occasional situation in which urine is needed exclusively for chemical analysis (e.g., glucose, ketones, urobilinogen, pH, specific gravity), a bag specimen will suffice. The utility of obtaining a bag specimen to "screen" the UA before deciding on antibiotic treatment or whether to send a formal culture is only defensible if the suspicion of UTI is low, as the sensitivity for markers of a UTI (discussed later), is only 50% to 85%.[58] [102] In summary, an

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MSCC, catheter, or SPA specimen is required for an accurate UA or urine culture in neonates and infants.

Urine Specimens from Patients with Chronic Urinary Drainage Systems

Urine obtained from any part of a chronic urinary drainage system is highly inaccurate for bacteriologic purposes. If UTI is suspected, a new catheter should be inserted and fresh bladder urine specimens subsequently obtained. [90] A small study advocating replacement of a chronically applied "Texas sheath" catheter with a fresh one was performed on subjects who did not have symptoms of UTI.[206] Such a method might be sufficiently accurate for screening of asymptomatic patients, but a Foley catheter should be used to obtain urine from patients with sheath catheters who have signs or symptoms of acute UTI.

Urine Dipstick

Urine dipstick tests are available to test for 10 separate parameters. The unassuming appearance and commonplace use of the urine dipstick might lead one to mistakenly underestimate its technical sophistication. Because each colored square on

TABLE 70-1 -- Overview of Urine Dipstick Tests

Test	Sources of Error and Artifact	Comments
Glucose	False-negative with ascorbate, ketones, and increased SG	Hypothermia may cause glycosuria despite hypoglycemia
	False-positive with peroxide and hypochlorite	
Ketones	False-positive with ascorbate, levodopa, valproate, pyridium, *N*-acetyl cysteine, high-protein diet, phenylketones, phthalein compounds	Very susceptible to deterioration with humidity, causing false-negative
Nitrites	False-positive with contamination, pyridium	75% false-negative rate when dipsticks exposed to air for 15 days
	False-negative with high SG, ascorbate, high urine pH, and urine standing in specimen cup >2 hr	
Protein	False-positive with pH>7, and chlorhexidine	Only reliable for albumin (glomerular proteinuria)
	False-negative with low pH, very dilute urine	
Blood	False-positive with povidone-iodine, certain (peroxidase-producing) bacteria, hypochlorite (bleach)	Positive test with speckles or dots implies non-hemolyzed blood
	False-negative with high SG, and high concentrations of urinary nitrites, ascorbate, or captopril.	Positive test with diffuse pattern implies hemolyzed RBC or myoglobin
Bilirubin	False-positive with iodine, stool contamination, chlorpromazine, mefenamic acid	Hard to read with agents causing marked urine discoloration
	False-negative after prolonged standing	
Urobilinogen	False-positive with pyridium, gantrisin, sulfonamides, porphyrin, methyldopa, procaine, aminosalicylic acid, 5-hydroxyindolacetic acid	Use fresh specimen; rapidly broken down by light and in acid urine
	False-negative with gantrisin and pyridium	
Leukocyte esterase	False-positive with vaginal contamination (commonest cause), oxidizing agents, eosinophils in the urine, *Trichomonas*	
	False-negative with high glucose, ketones, protein (especially albumin), pH, or SG and male sex, cephalexin, tetracycline, pyridium, oxalates, ascorbic acid, neutropenia	
pH	Urea splitting bacteria elevate pH	Use fresh specimen: standing raises pH by loss of CO_2
	Run off from protein strip can falsely lower pH	
Specific gravity	Overestimates SG with low pH, ketoacidosis, and protein	Not reliable at SG >1.025

	Underestimates SG due to glucose, urea, or with pH >7	

a urine dipstick involves a complex assay, it is essential to *meticulously follow the manufacturer's instructions for storage and use*.[81] Even with optimal storage and testing conditions, the false-negative and false-positive rates of these tests are problematic. In addition, most of the tests are susceptible to interference from a variety of substances (Table 70-1).

Method

Urine specimens should be tested as soon as possible after they are collected. If urine has been standing, it should be stirred or shaken well, because cells rapidly sink in a container. The test strip should be completely immersed for 1 second or less. The edge of the strip is drawn along the rim of the specimen container and can also be lightly tapped to remove excess urine, thus avoiding mixing of the reagents between different test patches. Hold the strip horizontally or place it on a clean gauze pad until the recommended time has elapsed. Most strips are designed so that all of the tests can be read together after 1 to 2 minutes.

Interpretation

Glucose.

The urine glucose test is normally negative. Urine glucose testing has limited usefulness in quantitative

testing because the serum glucose level at which spillage occurs varies (although in most patients it starts at between 180 and 200 mg/dL). [99] Changes in urine glucose lag behind changes in blood glucose by approximately half the interval between voids. [114] Glycosuria in the absence of hyperglycemia suggests renal tubular dysfunction. Glycosuria may occur in hypothermic patients in the absence of hyperglycemia, and indeed may mask true hypoglycemia in alcoholic patients who are hypothermic.[76]

Ketones.

Ketones are found in the urine during starvation, inadequate carbohydrate intake, diabetic and alcoholic ketoacidosis, isopropyl alcohol poisoning, or glycogen storage disease. Urine testing for ketones is not sensitive to all forms of ketonemia or ketonuria, and the absence of a positive ketone test by dipstick should not categorically dismiss a significant ketotic state. Tests for urine ketones are 5 to 10 times as sensitive to acetoacetate as to acetone. Similar to the serum "acetone test," dipsticks do not detect 5-hydroxybutyrate, which comprises 80 to 95% of the 3 "ketone bodies," and is the predominant form in the setting of ketoacidosis. However, urine ketone testing is significantly *more* sensitive than serum ketone testing.[79] Indeed, there is generally no need to obtain "serum acetones" to diagnose or manage diabetic ketoacidosis (DKA) when urine ketone monitoring is coupled with blood gas and anion gap analyses.

Nitrites.

Normally, urine does not contain nitrites. Nitrites are specific (approximately 95%), but not sensitive (approximately 45%) indicators of UTI. [119] [209] [213] Urinary nitrates are converted to nitrites most strongly by enteric coliform bacteria, explaining the nitrite test's 90% sensitivity in detecting UTI caused by *Escherichia coli*.[148] *Enterococcus*, a moderately frequent urinary pathogen, and *Pseudomonas* species and *Acinetobacter* do not convert nitrates to nitrites and therefore are not detected by the nitrite test. False-negative results also occur because of lack of dietary nitrate, frequent voiding, and/or diuresis. [215] Early morning-voided specimens are ideal (allowing time for conversion of nitrate to nitrite), but they are rarely available in the ED; if possible, a specimen obtained =4 hours after the last voiding is preferred.

Protein.

Proteins with molecular weight below 50,000 to 60,000 daltons can pass through the glomerulus to be reabsorbed in the proximal tubule. Normal protein passage in the urine is less than 150 mg/24 hours, or approximately 10 mg/dL of urine. About 10% to 33% of urinary protein is albumin, 33% is Tamm-Horsfall glycoprotein (secreted by renal tubular cells), and the balance is made up of a variety of immunoglobulins and other proteins. Proteinuria is a finding noted in about 5% of routine urine screens in men.[45] Whether this represents a normal variant is unclear, as 3% to 5% of young healthy adults have postural proteinuria (proteinuria when standing, but not when recumbent).[95] The urine dip-stick detects negatively charged proteins much more strongly than positively charged ones; it is therefore most sensitive to albumin.

In assessing a patient with proteinuria, it is helpful to divide the list of causes into those associated with hematuria and those not so associated. These are listed in Table 70-2 . Proteinuria can be due to either systemic or renal causes. Systemic causes can be physiological after strenuous exercise and in "postural" proteinuria; or pathological due to severe dehydration; hypovolemia; hypotension; hemorrhage; fever; congestive heart failure; severe infection; or hyperproteinemic states, which cause overflow proteinuria. Hyperproteinemic states include multiple myeloma, leukemia, massive intravascular hemolysis, and rhabdomyolysis. Elevated urinary protein due to renal causes, in the absence of any of the systemic conditions listed in Table 70-2 , is much more common. The source is either glomerular, with passage of normally unfiltered proteins; or tubular, with failure to reabsorb physiologically filtered, low molecular weight globulins. The former condition causes albuminuria. Renal tubular proteinuria is characterized by low levels of urinary albumin, and therefore more likely to be missed.

The likelihood that a positive urine protein dipstick reflects renal disease (as opposed to a systemic) etiology heightens with an increasingly positive result, as it does in patients with conditions such as hypertension, diabetes, connective tissue diseases, peripheral edema, gout, or elevated serum creatinine. A study of ED patients with severe acute hypertension identified renal dysfunction (as evidenced by an elevated serum creatinine) with 100% sensitivity using urine dipstick detection of 1+ proteinuria or hematuria.[123] As noted, proteinuria is an incidental finding in 5% of the normal population, so mildly elevated proteinuria in asymptomatic patients without systemic disease or renal insufficiency with otherwise normal urine sediment is likely of no clinical significance. [293]

The urine dipstick test is positive for protein with pyuria of more than 6 WBCs/hpf. [288] This is technically a false-positive finding but is useful when the urine dipstick is being used to screen for a UTI, since the threshold for leukocyte esterase is often much higher. Hematuria only slightly elevates urine protein levels.

"Blood."

The blood section of the urine dipstick shows a positive result in the presence of RBCs, hemoglobin, or myoglobin. Guidelines for distinguishing these are outlined in Table 70-3 . Moderate intravascular hemolysis does not cause hemoglobinuria, as the hemoglobin is tightly bound to haptoglobin and is therefore not filtered. Massive intravascular hemolysis gives rise to free plasma hemoglobin with a molecular weight of 32,000 daltons, which can pass through the glomerulus. Myoglobin has a molecular weight of 17,000 daltons, permitting easy passage through the glomerulus.

Urine in young, healthy volunteers contains <7 RBCs/µL (RBCs/µL).[274] Studies have shown that although the urine dipstick is only 93% sensitive compared to microscopy of unspun urine at the very low level of 1 RBC/hpf, it generally is very sensitive to 10 RBCs/µL. [274] Hence, false-negative results are confined to clinically insignificant hematuria.[133] [188] In asymptomatic men older than 50 years, significant disease can be signaled by intermittent positive dipstick hematuria, mandating careful follow-up of patients with this incidental finding. [184]

Dipsticks are vitiated by humidity and air and give false-negative results after improper storage. [54] Because RBCs lyse rapidly in urine with high pH or low specific gravity, delays in performing a microscopic UA may suggest myoglobinuria or hemoglobinuria. Microscopy or dipstick testing of a *freshly* obtained specimen can clarify this issue. The dipstick pad should be inspected for discrete positive "dots," indicative of nonhemolyzed RBCs. Conversely, urine with high specific gravity or low pH can inhibit the lysis of erythrocytes, which is necessary for the dipstick chemical reaction to occur, thus causing a false-negative result. [160] Povidone-iodine was reported to cause false-positive results in vitro, but a study reproducing typical clinical conditions demonstrated that this does not happen in vivo. [200] Iatrogenically caused trace positive results may occur after straight catheterization in 15%

TABLE 70-2 -- Causes of Proteinuria with and without Hematuria

Proteinuria usually with hematuria
(Generally indicative of glomerular disease; most etiologies in early stages can present without hematuria)
Infectious diseases
Post-streptococcal GN, pneumococcal pneumonia, ABE, meningococcemia, secondary syphilis, hepatitis B, severe viral infections, malaria, toxoplasmosis, Guillaín-Barré, etc.
Multisystem diseases
Vasculitides: Henoch-Schönlein purpura, PAN, Wegener's granulomatosis, Kawasaki disease, etc.
Connective tissue diseases (SLE, RA, scleroderma), neoplasms, etc.
Rhabdomyolysis (usually causes artifactual hematuria)
Goodpasture's syndrome
Cryoglobulinemias
Toxemia of pregnancy
Serum sickness
Proteinuria usually without hematuria
(Generally indicative of tubular/interstitial disease, or of overflow proteinuria. In advanced disease, can develop hematuria.)
Systemic conditions
Fever, post exercise, orthostatic
Diabetes
Amyloidosis
Sarcoidosis
"Overflow states": multiple myeloma, lymphoma, leukemia, rhabdomyolysis, etc.
Renovascular hypertension
Medications, drugs, and toxins
NSAIDs, gold, penicillamine, probenicid, captopril, lithium, cyclosporine, etc.
Heroin
Heavy metal nephropathy: lead, mercury, or cadmium
Renal diseases
Chronic pyelonephritis
Interstitial nephritis
Fanconi's syndrome

of cases, but at such low levels that they should not be a source of confusion with emergency urologic conditions. [103] [262]

Urine bilirubin.

Urine bilirubin represents the filtered, soluble, conjugated form of bilirubin. Unconjugated bilirubin is protein-bound, and does not pass through the glomerulus. Bilirubinuria is therefore due to intrahepatic or extrahepatic cholestasis. Bilirubinuria will be detected significantly earlier than clinical jaundice. Urinary bilirubin excretion is enhanced by alkalosis. A fresh sample of urine should be tested, because bilirubin glucuronide is hydrolyzed when exposed to light. Ascorbic acid and high levels of urinary nitrites decrease the sensitivity of the test to bilirubin.

Urobilinogen.

In a healthy person, conjugated bilirubin is excreted in bile. In the colon it is broken down into a number of compounds, which includes urobilinogen. Most of these compounds are excreted in the stool, giving the characteristic color. A small amount of urobilinogen is absorbed

TABLE 70-3 -- Aids in Distinguishing Hematuria, Intravascular Hemolysis, and Myoglobinuria

	Hematuria	**Myoglobinuria**	**Intravascular Hemolysis**
Serum findings	Color: clear	Color: clear	Color: pink
		Haptoglobin: normal	Haptoglobin: low
Urine appearance	Color: Clear to brown, clears with centrifugation	Color: Clear to red/brown; no clearing with centrifugation	Color: Clear to brown; no clearing with centrifugation
Urine microscopy	RBC, RBC casts, and protein implies glomerular source	Possible occasional RBC and tubular cells secondary to rhabdomyolysis-induced renal damage	Usually unremarkable
	No RBC casts, tubular cells, small protein: nephron source		
	Just RBC: source distal to nephron (e.g., ureterolithiasis)		

from the colon, and if it is not taken up on the first pass through the liver, it enters the circulation. Ultimately, some of this urobilinogen is filtered by the glomerulus to enter the urine, so that it is normal to have zero to moderate levels of urinary urobilinogen on dipstick testing. Most diseases causing hepatocyte dysfunction (viral or drug-induced hepatitis, cirrhosis, congestive heart failure with passive liver congestion, or cholangitis) also cause increased urinary urobilinogen excretion. This is because there is greater impairment of hepatic uptake of urobilinogen than there is of hepatic excretion of conjugated bilirubin. As a qualitative test with a wide range of normal values, it is rarely helpful, but in evaluating a patient with jaundice, it can have diagnostic significance (Table 70-4).

Leukocyte esterase.

This portion of the dipstick test is designed to detect enzymes from the azurophilic granules in neutrophils. Normally the test is negative. Studies report a

TABLE 70-4 -- Relationship between Urinary Bilirubin, Urobilinogen, and Stool Color in the Jaundiced Patient

	Healthy, Normal	Complete Biliary Obstruction	Intravascular Hemolysis	Hepatocellular Disease
Urinary bilirubin	None	Elevated	None	Elevated
Urinary urobilinogen	None or present	None	Present, sometimes large	Normal early, Increased late
Stool color	Normal	Acholic	Normal	Normal

wide and clinically important range of thresholds for dipstick testing sensitivity, from 10 to 100 WBCs/µL urine. [169] [178] [230] Studies suggest that the test is between 50 and 96% sensitive in detecting infection. [83] [85] [149] [151] [211] [217] [230] The specificity for presence of WBCs is between 91 and 99%. [52] [83] [151] [209] [211] The most common cause of a false-positive leukocyte esterase test is vaginal contamination.

pH.

Average daily excretion of 50 to 100 mmol of H^+ in the urine gives rise to a typical urine pH of approximately 6. Dietary protein lowers the urinary pH, whereas fruit and vegetables, especially citrus fruits, tend to increase it. The normal urinary pH ranges from 4.5 to 8. The significance of pH testing is in the assessment of normal renal function. In most states of alkalosis and acidosis, the healthy kidneys maintain homeostasis by conserving or excreting H^+. Failure to do so suggests renal disease, especially renal tubular acidoses. An exception is the "paradoxical aciduria" of hypokalemic alkalosis secondary to volume contraction, hypercorticism, or diuretics, where the highest priority of the renal tubule is to conserve sodium. Note that pH is falsely elevated by the action of urea-splitting bacteria, especially *Proteus* species. This can occur with "stasis" of urine, either in the bladder or in specimen cups awaiting processing in the laboratory. A persistently alkaline urine is seen in patients with struvite (triple phosphate) urolithiasis.

Specific gravity.

The dipstick test for urine-specific gravity assays for the primary urinary cations, sodium and potassium. True specific gravity, which is also dependent on anions, albumin, proteins, urea, and glucose, is therefore not measured. Artifactually low specific gravity readings are obtained in alkaline urine. Acid urine and albumin falsely elevate the specific gravity reading. [1] Some investigators believe that these strips on the dipstick test are of marginal clinical utility at best. [10] Other clinical indicators of a patient's hydration status are probably more reliable. If necessary, a refractive-specific gravitometer or a hygrometer should be used.

Microscopic Urinalysis

Microscopic UA is performed to identify cells, bacteria, and other microbes as well as formed elements such as casts and crystals (Fig. 70-2 (Figure Not Available)). The following discussion will focus on the findings of significance in diagnosing UTI: WBCs, bacteria, and WBC casts. The presence of WBCs with bacteria distinguishes infection from colonization (bacteriuria without pyuria). Some authorities state that significant infection without pyuria occurs in less than 5% of cases, making pyuria a sensitive marker of infection. [172] [195] [253] [254] Other studies do not support reliance on pyuria as an indicator of infection. [102] [125] [146] [149] [154] [164] [224] The presence of WBC casts distinguishes pyelonephritis ("upper UTI") from cystitis ("lower UTI").

Microscopic UA is performed using one of five methods, listed subsequently. Traditionally, the most common technique has been the examination of unstained centrifuged urine (technique 3). It has the advantage of concentrating formed elements that might otherwise be missed. The disadvantage is that the presence and quantity of elements in a specimen will vary depending on many uncontrolled factors: the volume of the specimen, the time and speed of centrifugation, the fragility of the formed elements, the volume of the "drop" in which the pellet is resuspended after removal of the supernatant, and the size of the microscope's "high-power field" (hpf). [36] [125] [256] [277]

1. *Examination of unspun urine in a hemocytometer counting chamber.* The hemocytometer is a precisely milled slide etched with measured squares, allowing for the exact enumeration of cells in each square. Because the distance between the etched surface and the coverslip is exactly known, it is possible to determine the number of cells per unit volume of specimen. Enough fresh unspun urine is placed on the slide to fully cover the counting area, and the cells are counted. There should usually be less than 1 WBC/µL, [80] [195] although more than 10 WBCs/µL and more than 5 RBCs/µL are unequivocally abnormal. The threshold for diagnosing UTI is usually set at = 8 WBCs/µL. [36] [80] [95] [252] [257] Bacteria do not sink to the surface of the hemocytometer, so that counting them through the many focal planes of the chamber is not possible, although methods to estimate the bacterial count per unit volume have been described. [224] [277] Casts should be approximately 125-fold less numerous than WBC, so they should not be found in the hemocytometer unless by chance. [134]
   The hemocytometer is accurate, fast (time need not be spent in staining or centrifugation), and easy to master. Its two major drawbacks are cost (around $150 for the slide and cover slip) and fragility (these are both easily destroyed if dropped). These problems may be insurmountable given the equally cramped budgets and laboratory facilities of many EDs. In the identification of formed elements other than WBC and RBC, the hemocytometer is less accurate than spun urine microscopy since these elements appear in very low concentrations.
2. *Examination of unspun, unstained urine placed on a regular microscope slide* is a qualitative method sometimes used in the diagnosis of UTI. Using 1 organism/hpf as a positive result, the sensitivity and specificity in detecting 10^5 CFU/mL are between 60% and 90%.[118] This method only identifies 1 WBC/hpf with the highly pyuric state of 250 WBCs/µL, [252] leading some to advocate the use of more than 1 WBC/*low-power* field as a criterion for infection. [195]

3. *Examination of unstained, centrifuged urine* is performed by centrifuging 10 mL of urine at approximately 450 g (1000 to 4000 rpm) for 3 to 5 minutes. Roughly 9 mL of supernatant is poured off, and the pellet is resuspended in the remaining fluid. This suspension is placed on a slide with a coverslip and examined. The larger formed elements, especially casts, tend to migrate to the edge of the coverslip, and they can be seen with low magnification. One or two casts, depending on the clinical context, may be normal; more are not. The morphology of the more commonly encountered formed elements of urine sediment is shown in Figure 70-2 (Figure Not Available) . The significance of each is beyond the scope of this text, but this information can be found in a standard textbook of clinical laboratory procedures and diagnostic testing. [99] [114] When examining centrifuged urine, more than 5 WBCs/hpf seen in the middle of the coverslip has traditionally been taken as indicative of abnormal pyuria. Most authors have estimated that 10 WBCs/µL are equivalent to approximately 1 WBC/hpf; thus, this oft-cited threshold for diagnosing UTI is actually equivalent to 50 WBCs/µL. [36] [80] [195] [252] [257] Since the gold standard for diagnosis of UTI is =8 WBCs/µL, many infections will escape detection using this method.
   Various numbers of bacteria per high-powered field have been used as criteria for the diagnosis of UTI. A threshold of 10 to 20 organisms/hpf has been recommended to rule out bacteriuria at the 10^5 CFU/mL level.[118] This threshold does not exclude infection in symptomatic patients.

4. *Examination of Gram-stained, uncentrifuged urine* is also a semiquantitative measurement. It is estimated that 1 bacterium/hpf is equivalent to 10^5 CFU/mL in bacterial culture. [99] [118] [125] [147] [236]
5. *Gram stain of centrifuged urine* is probably the optimal technique, short of culture, in the assessment of bacteriuria. It is more than 95% sensitive and more than 60% specific to 10^4 CFU/mL—an order of magnitude lower concentration of bacteriuria than the previously described methods. [118] [167] Detection of 1 organism/oil-immersion field constitutes a positive result. [118] [228] Specificity is increased to 95% if 5 organisms/hpf are seen. [118]

Figure 70-2 (Figure Not Available) *1,* Red and white blood cells (RBCs and WBCs); *2,* WBCs with bacteria in an infected urine specimen; *3,* fine granular cast; *4,* WBC cast (seen in intrinsic renal diseases such as pyelonephritis and glomerulonephritis: note the discernible nuclei and cell boundaries); *5,* RBC cast (the distinct and uniformly spherical shape of the erythrocyte is visible); *6,* epithelial cast (present in tubular disease); *7, Trichomonas vaginalis; 8,* budding yeast forms; *9,* calcium oxalate crystals; *10,* phosphate crystals; *11,* urate crystals; *12,* cystine crystal (indicative of cystinuria). *(1, 3–9, and 12 from Urine under the Microscope. Montclair, NJ, ROCOM Press, 1975; 2 from Birch DF, et al: A Color Atlas of Urine Microscopy. Chapman & Hall, 1994; 10 and 11 from Netter FH, Shapter RK, Yonkman FF: The Ciba Collection of Medical Illustration. 6:80, 1973.)*

Summary of Tests Used in the Diagnosis of UTI

The three tests commonly used to evaluate a patient for the presence or absence of UTI are urine dipstick, microscopic UA, and urine culture. Each represents an increasing degree of expense, delay, and resources. A brief discussion of their relative strengths and weaknesses ensues.

Urine Dipstick

Dipstick testing of urine is faster than microscopic UA; is less labor-intensive, thus cheaper; and circumvents multiple sources of potential and proven error. [135] Is it sufficiently accurate to replace it? Using *either* leukocyte esterase (LE) *or* nitrites to indicate infection, the dipstick is still only 50 to 90% sensitive to culture-proven infection.[6] [167] [169] [212] [213] [217] This is clearly not adequate to rule out infection in symptomatic patients, but may be acceptable in screening populations of asymptomatic patients, because such a group is likely to have a low prevalence of disease so that the test will have a serviceably high negative predictive value of 95 to 99%.[135] [169] [213] [214] In symptomatic patients, sensitivity should be enhanced by taking a positive result in *any one* (or more) of *either* LE *or* nitrites *or* protein *or* blood as indication of a UTI.[91] [174] [192] [236] [240] Sensitivity can also be augmented by allowing more time before reading the strip.[169] [240] An approach that maximizes sensitivity is indicated in symptomatic females for whom empiric treatment is recommended and the prevalence of disease high. In such patients a completely negative dipstick should prompt a careful search for another source of infection, but not deter from empiric treatment, since it is still inadequately sensitive to rule out infection.[174] [236] Clearly, maneuvers to enhance sensitivity will diminish specificity, which may be as low as 26%, [263] leading some to advocate microscopic UA for all urines abnormal by dipstick.[25] However, as discussed later, this probably adds little to a carefully performed dipstick test.

Microscopic Urinalysis

If the urine dipstick has such poor specificity when it is used as a test with adequate sensitivity, should it be discarded altogether, and microscopic UA relied upon in its stead? Apart from the hemocytometer, the most reliable method for identifying significant bacteriuria is oil-immersion microscopy of Gram-stained, centrifuged urine.[118] Despite this, the practice in most hospital laboratories is to examine the resuspended pellet of unstained, centrifuged urine. The problems with this method have been discussed. In various studies a range between 1 and 10 organisms/hpf or 5 WBCs/hpf has been considered a "positive" test (as the threshold number rises, so does specificity, at the price of sensitivity).[118] [219] In aggregate, the accuracy of microscopy in the diagnosis of UTI is similar to that of the dipstick alone, with 22% false-positive, and 23% false-negative rates when compared to culture.[53] [89] [277] Thus microscopic UA cannot rule out infection in symptomatic patients. The specificity of pyuria will be improved when viewed as a marker of all genitourinary infections including urethritis, prostatitis, epididymitis, vaginitis, and cervicitis. These diagnoses must always be entertained in patients with urinary symptoms, especially if the urine is sterile with pyuria.

Urine Culture

Cultures are indicated for any potentially complicated UTI. This includes those in children; men; women with recurrences or relapses; the immunocompromised; patients with urinary tract pathology, including stones and possible pyelonephritis; and pregnant patients. They are usually recommended around the 16th week of gestation. As previously discussed, every effort should be made to obtain a high-quality specimen owing to the difficulty of distinguishing contamination from significant low-count bacteriuria in symptomatic patients, and in view of the expense of cultures and treatment, if the culture is positive.

Conclusion

It is well established that female patients with symptoms of uncomplicated lower UTI can be treated without culture.[39] [46] [119] [129] [140] [208] [256] Treatment is generally benign, and 95% of urine cultures that are positive will grow out a limited number of organisms with predictable antibiotic susceptibilities. [46] [231] [256] Many authors recommend dipstick and/or microscopic UA in this group.[119] [208] [256] While these tests cannot rule out disease in symptomatic patients, if they are negative, alternative diagnoses should be considered more carefully. Symptomatic patients with negative dipstick and/or UA can be treated empirically, with cultures sent and instructions for follow-up.[119] Urine testing is more likely to alter management as well as increase clinical accuracy in patients whose symptoms are equivocal (intermediate pretest probability).[263] The UA will also help in equivocal situations where it can help in distinguishing pyelonephritis (WBC casts), vaginitis (rarely pyuria or hematuria on careful MSCC or catheterized specimen), and urethritis (usually pyuria, rarely hematuria), from cystitis (pyuria and often hematuria). [140] [256] In the ED, the ease of obtaining a dipstick argues for its use without UA since it is equally sensitive, unless formed elements such as cells, casts, crystals, trichomonas, or other parasites are suspected.[151] [169] In patients with pyelonephritis, urine cultures are probably warranted because they will alter therapy in about 5% of cases. [270] In most asymptomatic patients, a negative dipstick or UA has sufficient negative predictive value to rule out disease unless the patient is pregnant or getting urologic surgery. Special clinical considerations in some patients (e.g., the immunocompromised, diabetic females at high risk for UTI) may mandate adjustments of this approach.[169] In febrile infants and children, pyuria less consistently accompanies infection, rendering the dipstick and UA less sensitive than in adults. [7] [95] [102] Cultures are recommended for febrile infants younger than 2 months.[58]

TESTING FOR PREGNANCY

Pregnancy tests are based on the detection of ß-human chorionic gonadotropin (ß-hCG) in serum or urine. ß-hCG is secreted by trophoblastic cells of the placenta starting from the time of implantation of the blastocyst. Qualitative serum tests and the urine test detect ß-hCG levels of between 15 and 25 mIU/mL. [187] The concentration of hCG is usually lower in urine than in serum, which accounts for the slight advantage of serum tests in detection of early pregnancy. Optimal urine pregnancy tests are obtained on first-voided concentrated morning specimens. Home urine pregnancy test kits detect levels of ß-hCG of about 50 mIU/mL in the urine.

ß-hCG levels of 5 to 8 mIU/mL correspond to the 9th to 11th day after ovulation (23 to 25 days after the first day of the last normal menstrual period). In a viable intrauterine pregnancy, the ß-hCG level doubles approximately every 2 days during the first 4 weeks of gestation, reaching a serum level of 25 to 300 mIU/mL, detectable by virtually all ß-hCG-based pregnancy tests in urine or blood, on or around the first day of the missed menstrual period. The doubling rate declines to every third day thereafter, [179] [246] with the quantitative ß-hCG reaching a peak of around 100,000 to 200,000 mIU/mL between the 10th to 14th gestational weeks and declining to around 10,000 to 20,000 mIU/mL for the rest of the pregnancy (Table 70-5). There is a wide range of ß-hCG levels among different women at the same stage of gestation, making definite clinical determinations on the basis of a single quantitative test impossible. [59] Furthermore, since antigens in each patient vary, there is up to a full two-fold range of discordance in simultaneously measured ß-hCG levels. [55] [56] This is likely to be of clinical significance only when hCG levels are extremely low. False-positive tests have been described in association with molar pregnancy, choriocarcinoma, teratoma, occasional malignancies outside the genitourinary tract, and very high levels of proteinuria. There is one report of a positive urine hCG (but normal serum hCG) associated with tubo-ovarian abscess. [161]

The rate of decline of quantitative ß-hCG after gestation varies depending on the reason for the conclusion of the pregnancy. After a term delivery, ß-hCG falls to zero in

TABLE 70-5 -- Quantitative Immunometric Assay for ß-hCG

	mIU/mL (IU/L)
Males and nonpregnant females	<5
Pregnant females (wk gestation)	
=1:	5–50
2:	50–500
3:	100–10,000
4:	1000–30,000
5:	3500–115,000
6–8:	12,000–270,000
12:	15,000–220,000

From 3rd edition Roberts and Hedges, p 1225, Table 74-4. Values differ slightly from Table 70-6 due to use of gestational age and different patient population.

2 weeks. After surgery for an ectopic pregnancy the range is 1 to 31 days, with a median of 8.5 days; after a first-trimester spontaneous abortion, the range is 9 to 35 days (median, 19 days); and after first-trimester elective abortion the range is 16 to 60 days (median, 30 days). [99]

Although a previous quantitative ß-hCG level is rarely available to the emergency clinician, doubling rates are an important part of the assessment of a healthy first-trimester pregnancy. Fetal nonviability, ectopic pregnancy, and intrauterine demise are signaled by abnormalities in the predicted rise in quantitative ß-hCG. [28] [62] [120] A serum quantitative ß-hCG level that does not increase by 66% every 48 hours has a 75% chance of being due to a nonviable pregnancy. [120] [121] The ß-hCG levels in a healthy intrauterine pregnancy (IUP) and associated sonographic findings are listed in Table 70-6 .

The association between ß-hCG levels and gestational dates has led to the concept of the "discriminatory zone." In a normal pregnancy with a quantitative ß-hCG level of >1000 to 1500 mIU/mL, a double decidual sac should, at the very least, be sonographically identifiable by transvaginal ultrasound. [27] The discriminatory threshold for identifying early IUP by transabdominal ultrasound is considered to be =6500 mIU/mL. If these ultrasound findings are absent at these thresholds, the pregnancy is almost certainly abnormal, with a significant possibility of ectopic. However, the converse—that ectopic pregnancy will always be accompanied by a

TABLE 70-6 -- Relationship between Gestational Age, Quantitative ß-hCG Levels, and Ultrasound Findings

Time Elapsed from First Day of Last Normal Menstrual Period	**Quantitative ß-hCG Level (mIU/mL) Using the IRP**	**Ultrasound Findings**
<28 days	5–50	
4–5 wk	50–500	From about 4.5 wk and ß-hCG =1000–1500, TVU can show DDS to confirm IUP
5–6 wk	100–10,000	Definitely abnormal if TVU does *not* show DDS after 5 wks or with ß-hCG >1500. Yolk sac with quant ß-hCG >7000, EGA 5–6 wk
		TAU should show viable IUP if quant ß-hCG >6500
6–7 wk	1000–30,000	Fetal pole, cardiac activity 5.5–7 wk, quant ß-hCG >10,000
7–8 wk	3500–115,000	Yolk sac 5–6 wk or ß-hCG >7200; fetal pole/heart 5.5–7 wk or ß-hCG >11,000–17,000
8–10 wk	12,000–270,000	
>10 wk	270,000–15,000	

TAU: transabdominal ultrasound

TVU: transvaginal ultrasound

DDS: double decidual sac

EGA: estimated gestational age

IUP: intrauterine pregnancy

ß-hCG level =1000 mIU/mL—is *not* true. Only 15% of ectopic gestational sacs examined pathologically have evidence of an embryo. This observation, combined with

a variety of pathologic characteristics and sites of implantation, leads to highly variable quantitative ß-hCG levels in ectopic pregnancies. [65] In general, an ectopic pregnancy elaborates quantitatively small amounts of ß-hCG, with 1% of ectopic pregnancies having a quantitative ß-hCG of <10 mIU/mL and up to 8% having ß-hCG levels of <25 mIU/mL.[225] About 10% of ruptured ectopic pregnancies are associated with a ß-hCG level less than or equal to 100 mIU/mL. [65] Overall, about 25% of ectopics will be diagnosed with a ß-hCG of <1000 mIU/mL,[57] although in one series, at the time of operation, 50% of women with unruptured ectopic and 40% with ruptured ectopic pregnancies had serum ß-hCG levels of <1000 mIU/mL.[65] Patients with abdominal and/or pelvic pain or vaginal bleeding and a ß-hCG level =1000 mIU/mL have a fourfold increased risk of ectopic pregnancy compared with those with the same symptoms and a ß-hCG level =1000 mIU/mL.[122]

Thus, it can be seen that the "discriminatory zone" can only be used to identify patients in whom ultrasound should definitely demonstrate signs of healthy IUP: a transvaginal ultrasound that fails to show an IUP with a ß-hCG level =1000 is >86% specific for ectopic pregnancy. [28] While there will be an increased rate of non-diagnostic ultrasounds below this level, a sonographic diagnosis will still be possible in a third or more of patients. [12] [61] Thus, the ß-hCG titers provide a basis for interpreting ultrasounds, *but not a basis for deciding whether or not to get them*.[57] [122] [177]

BLOOD CULTURES IN THE EMERGENCY DEPARTMENT

Indications

Blood cultures are indicated when there are clinical findings suggestive of an otherwise unidentifiable bacteremic state (Table 70-7). Traditional use of blood cultures in patients with pneumonia and pyelonephritis is of increasingly questionable utility. [100] [270] [283] [290] It should be noted that 25% of patients with documented bacteremia have periods without fever.[196] In the elderly the proportion is even higher, with 50% of bacteremic patients over 65 having a temperature between 97.1°F (36.2°C) and 100.9°F (38.3°C); and at least

TABLE 70-7 -- Summary of Indications for Obtaining Blood Cultures

Patients with fever and any of the following:
Unexplained alterations in mental status, functional status, or autonomic status in previously healthy patient between the ages of 5 and 65 years, or
No source, if <2 years, >65 years, or immunocompromised, or
Age <2 months
Patients with or without fever and any of the following:
Rigors, or
toxic or "septic" appearance: (i.e., unexplained hypotension, altered mental status, shock), or
suspicion of infectious endocarditis, or
serious focal infections (e.g., meningitis, septic arthritis, osteomyelitis)

13% with no documented temperature greater than 99.1°F (37.3°C) at any time. [47] [77] [86] [243] In the elderly, increasing age, vomiting, altered mental status, presence of a Foley catheter or urinary incontinence, and greater than 6% band forms are predictive of positive blood culture results. [77] [222] The subjective impression of "having fever" in adults is not a reliable indicator of the presence of fever, although the subjective impression of "no fever" is much more likely to be accurate. [38] Prediction models to improve the use of this costly laboratory procedure have been developed, [14] but lack widespread validation or acceptance. [193] [296]

In children, the indications for blood cultures are under active investigation. Traditional teaching that blood cultures are indicated for all patients younger than 2 years with fever >38.6°C (>101.5°F) and without obvious source is being modified by the effectiveness of *Haemophilus influenzae* type b (HIB) vaccination. In the post-HIB era, the commonest etiology of "occult bacteremia" is the pneumococcus, accounting for >75% of cases, the majority of which resolve spontaneously. [3] [157] Bacteremia is more likely the higher the temperature and WBC count, the lower the patient's age, and the more rapid the onset of illness. [35] [93] [113] [150] In infants younger than 2 months with temperatures higher than 38°C (>100.5°F), some authorities would recommend blood cultures regardless of the presence or absence of a source, although this approach is subject to modification by experienced clinicians based on the patient's age and clinical setting. A child with a normal temperature in the ED and a history from the parents of tactile fever needs to be approached in the same way as a patient with fever documented on physical exam for several reasons. First, bacteremic children, like adults, have intermittent fever, with up to 50% afebrile rates in children with demonstrated bacteremia. [138] Second, parents' tactile impression of fever has been shown to be highly reliable. [11]

The Controversy Regarding "Outpatient Blood Cultures"

There is a long-standing debate regarding the utility of outpatient blood cultures (i.e., blood cultures on patients who are discharged from the ED pending results). Arguments for and against outpatient blood cultures are summarized in Table 70-8 .

TABLE 70-8 -- Summary of Arguments for and Against the Performance of Outpatient Blood Cultures

Arguments against outpatient blood cultures:
1. Low true-positive rates.
2. True positives are rarely clinically significant.
3. High false-positive rates are expensive and time-consuming for both patient and health care system.
4. Difficulty of ED follow-up makes positive blood culture results a medicolegal liability.
Arguments for outpatient blood cultures:
1. Permits outpatient management in certain groups with low prevalence of disease with financial, psychological, and nosocomial cost savings for patients and society.
2. Allows initiation of antibiotics without fear of a potentially irrevocably lost opportunity for blood cultures (especially with infectious endocarditis).
3. May give useful etiologic information and antibiotic susceptibility profiles about diseases under outpatient treatment.

The data on the subject is still inconclusive. Opponents cite medicolegal issues, problems with follow-up, high contamination rates, low rates of positive cultures, and even lower rates of frequency of patients in whom therapy is changed because of culture results. [72] [250] [261] Proponents also cite medicolegal concerns, positive rates similar to those seen with inpatient blood cultures, cost savings, and the benefit of diagnosing significant, yet subtle, bacteremic states (such as endocarditis). [73] [244] They also point out that the high false-positive rates seen in many ED series should be an indictment of poor technique, not of the test itself.

On the basis of current data, it would seem to be fiscally extravagant to admit all patients in whom a bacteremic state is possible, and injudicious to deny blood cultures solely on the basis of a patient not appearing "toxic enough" to warrant admission. Outpatient blood cultures, with due attention to collection technique, patient selection, and diligent follow-up, would therefore seem to be an appropriate component of emergency medicine practice.

Technique of Obtaining Blood Cultures

Studies have demonstrated sources of contamination at every stage of the process of obtaining and processing blood cultures. In addition to obvious sources of contamination from the patient's and the phlebotomist's skin, antiseptic agents [21] [201] and gloves[297] have been implicated. Some authorities have argued that the primary source of contamination is in the laboratory processing of specimens. [237] However, the consensus is that the most common source of contamination is the process of phlebotomy and inoculation of blood culture bottles. [112] [281] Obviously this is the single step over which emergency clinicians have control, either directly, or via protocols of technique for blood culture phlebotomy. Contamination rates are typically between 1.5 and 3%, [3] [74] [186] although many ED series show much higher rates than this, reflecting the difficulty of obtaining effective blood cultures in the ED setting. [251] [261] [271]

A high degree of sensitivity is required of blood cultures. Many significant bacteremic illnesses have been documented with as little as one colony-forming unit (CFU) per 10 mL of blood.[98] [132] Human skin has a bacterial concentration between 10^3 and 10^6 CFU/mL, on the forearm and groin, respectively. [235] Designed to detect vanishingly low concentrations of bacteria, the test is clearly susceptible to false-positive results (impaired specificity) when blood must necessarily be obtained by

passing a needle through the skin. Eighty percent of the skin flora are transient, superficial, and removable; 20% inhabit the sebaceous ducts and hair follicles, and are not removable without destroying the skin.[170] [235] The former group are predominantly gram-positive and gram-negative aerobes and are the target of skin disinfectants.

The primary agents for skin disinfection are iodine compounds, alcohols, chlorhexidine, and hexachlorophene. Iodine solution remains a gold standard, killing bacteria, fungi, protozoa, and viruses, but has been replaced in many institutions due to concerns about skin burns and allergic reactions. Reports of the former were probably due to the use of 7% solution: The risk of a burn or an allergic reaction is thought to be negligible with the currently available 2% preparation. [96] [281] The most effective cleansing agent is tincture of iodine, which is a mixture of 2% iodine solution and 70% alcohol. [137] [165] [259] [260] Povidone-iodine 10% solution (Betadine) has a much lower free iodine concentration than iodine solution and is therefore less potent. Iodine is superior to hexachlorophene and chlorhexidine in killing gram-negative bacteria. Iodine, like other antiseptic agents, is inhibited by the presence of organic matter, emphasizing the need for thorough skin cleansing before the application of any skin disinfectant.

Ethyl or isopropyl alcohol should be used in 60 to 80% solution. Alcohol prep pads, which generally contain 70% isopropanol, have solved traditional concerns regarding evaporation of alcohol from cotton balls stored in jars. Alcohol is a less powerful germicide than iodine in vitro, and kills only 90% of surface bacteria after a full 2 minutes with reapplication to prevent drying.[96] Alcohol foam applicators can avoid premature drying. Alcohol is inactive against fungi, spores, and viruses; however, in vivo studies of blood culture contamination rates have shown it to compare favorably with iodine. [157] [259] Because iodine solution is often not available, and iodophor solutions are less potent, alcohol still has an important place in skin antisepsis. In addition, alcohol is an excellent solvent, so that alcohol pads are a good tool for skin preparation prior to the application of iodine compounds.

Chlorhexidine (Hibiclens) and hexachlorophene (pHisoHex) are antiseptics that are more effective against gram-positive than gram-negative bacteria. Both agents have intradermal absorption, which causes prolonged antimicrobial activity and is the basis of their popularity as surgical scrub and operative site preparations. This also makes them preferable agents where indwelling lines, especially central lines, are being placed. [173] For routine blood culture phlebotomy, they are not as effective as alcohol and iodine combinations, although they are superior to povidone-iodine solution. [186] Most studies show chlorhexidine to be more potent than hexachlorophene, and it has not been associated with induction of seizures in infants.

The results of studies of contamination rates with various skin preparation agents are not unanimous. Table 70-9 presents a consensus skin preparation protocol. Optimal results seem to be obtained by alcohol-iodine mixtures.[165] [232] [235] [260] The most important concept in skin disinfection is that bacteria do not die at the instant of contact with disinfectant agents. Iodine (2%), which is twice as potent as 10% povidone-iodine, requires at least 90 seconds in contact with the skin to

TABLE 70-9 -- Skin Preparation and Technique for Drawing Blood Cultures

1. Cleanse the skin with alcohol swabs three times, or until the swabs appear entirely free of surface dirt after application.
2. Allow to dry.
3. Apply 10% povidone iodine or (preferably) 2% iodine solution or (ideally) 2% tincture of iodine in 70% alcohol, three times, in centrifugal circles from the anticipated site of venipuncture.
4. After third swab, allow to dry at least 60 seconds.
5. During this period, prepare lids of blood culture bottles with iodine and/or alcohol; lay out sterile gloves, and use paper wrapper as a sterile field for the needle and syringe (not necessary if using a vacutainer system).
6. Wipe off dry iodine at venipuncture site with alcohol. Substitute chlorhexidine if placing an indwelling catheter.
7. Obtain at least 20 mL of blood to place in 2 bottles. (This is one blood culture set.)
8. Inoculate bottles without changing needles.

kill 90% of surface bacteria.[96] In many ED patients it will be necessary to use alcohol prep pads to remove gross dirt and debris from phlebotomy sites before initiating the steps of the formal skin preparation.

Special Considerations in Obtaining Blood Cultures

"Changing the Needle" After Phlebotomy

In considering this issue, it is important to emphasize the distinction between *needle changing* and *needle recapping*.[284] The latter is a well-established risk to health care workers; it contravenes standard recommendations for universal precautions, *and should not be performed*. Needle replacement using the standard needle removal device on "sharps" containers is an unquantified risk, but clearly much less dangerous than recapping.

Based on very little scientific data,[233] it was long considered essential to change the phlebotomy needle before inoculating blood culture bottles. With increasing awareness of the risk of bloodborne infections associated with needle-stick injuries, this practice has come under scrutiny. One large, well-controlled study showed no difference in contamination rates with or without needle changing.[144] Other studies show a clear trend *toward* a lower contamination rate with needle change, without reaching statistical significance.[51] [112] [158] [245] In conclusion, not changing needles before blood culture bottle inoculation is acceptable practice for obtaining routine blood cultures. However, in those situations in which the results of the blood culture are of paramount importance (e.g., suspected infectious endocarditis, for which empiric antibiotic coverage will be started immediately), it is suggested that needles be changed prior to inoculation of culture bottles, but not recapped.

Special Access Sites

Some studies show that newly placed intravenous catheters are an acceptable source of blood culture specimens, providing that the usual measures are taken in skin preparation,[111] [245] although other series have less favorable results.[74] Previously placed IV catheters (central and peripheral) and arterial lines have been compared with fresh percutaneous phlebotomy for blood cultures in several studies. Chronically placed lines either trend toward, or show, statistically significant increased contamination rates,[37] [75] [265] [272] [273] [294] with the exception of carefully tended central venous access ports in cancer patients, which may have increased sensitivity in identifying bacteremia (possibly due to the fact that the catheters themselves are often a source of bacteremia in those patients).[63] Thus, with the possible exception of chronic venous access ports in cancer patients, chronically indwelling vascular lines should, in most situations, be avoided as a source of blood cultures.

Heel Stick in Neonates

This technique resulted in recovery rates of bacteria equivalent to phlebotomy in two studies.[139] [176] Since approximately 25% of bacteremic infants have less than 25 CFU/mL of blood, this proportion (25%) will be missed if = 0.2 mL is obtained for culture. For this reason, heel stick should be considered as a source of last resort for blood culture.

Intraosseous Specimens

This technique may also be used when phlebotomy is impossible.[205]

Timing of Blood Cultures

In most circumstances, the timing of blood cultures is moot in the ED. Most patients are sick enough to warrant the initiation of empiric antibiotics, or are well enough for discharge, so that two or more sets need to be drawn immediately. The situation in which the timing of blood cultures might become a consideration is in a patient

requiring admission but in whom the diagnosis of bacteremia is sufficiently in doubt that antibiotic therapy is not to be initiated empirically.

Contrary to the time-honored tradition of obtaining blood cultures during a fever spike, the data show a trend toward a higher proportion of true positive cultures in patients whose blood is drawn in the 12 hours *before* a fever spike.[20] [281] Furthermore, excepting infectious endocarditis, most clinically significant bacteremia is thought to be intermittent, so that multiple sets of cultures obtained at one time would heighten the risk of missing the period of bacteremia. [197] [280] Therefore, for patients admitted to the hospital with the tentative diagnosis of sepsis, it is theoretically advantageous not to draw all three sets of blood cultures in the ED, but to space them over the following 12 to 24 hours.[49] However, if administration of antibiotics is clinically indicated, two or three sets of cultures separated by 20 to 30 minutes are obtained in the ED before initiation of antibiotic therapy.

Blood Culture Volumes

Volumes in Adults

A large number of studies almost uniformly demonstrate that the sensitivity of blood cultures is directly related to the volume of blood cultured. [9] [34] [94] [109] [183] [227] [238] [269] In a representative study, Ilstrup and Washington showed that 20 mL and 30 mL of blood yielded, respectively, 38% and 62% more true-positive results than 10 mL.[109] Mermel and Maki showed that each additional mL of blood yields an average of 3% more true-positive results. [183] This finding is also consistent with the fact that 40% of adults with bacteremia have less than 1 CFU/mL of blood, and that 20% have less than 1 CFU/10 mL. [98] [132] Alternatively expressed, if 10 mL of blood is obtained for culture, 20% of patients with continuous bacteremia will be missed. Since most bacteremia is intermittent, and since endogenous factors in blood will cause some inhibition of bacterial growth even with modern lysis- and filtration-centrifugation techniques, the false-negative rate in clinical practice will always be significantly higher. On purely mathematical grounds, 10 mL per set of blood cultures is an absolute bare minimum for culture. In adults, most authorities recommend at least 30 mL of blood per culture site/set. [131] [282] [287]

To ensure dilution of the blood's antibacterial properties (e.g., immunoglobulins, complement, WBCs), blood should be placed in a concentration less than 10% (i.e., not > 1 part of blood to 10 parts of medium).[282] Thus, if 30 mL of blood is obtained from one site, it should be equally divided into three of the usual 100-mL broth bottles.

Volumes in Children

A blood volume of 30 mL from a 70 kg adult is equivalent to 0.5 mL of blood in a 3.5 kg neonate. Fortunately (for the validity of the blood culture), it has been shown that levels of bacteremia are typically 10-fold higher in infants than adults [64] [176] [258] ; and that the sicker the child, the greater the likelihood of a high level of bacteremia.[64] Although one study[198]

failed to show any effect on the rate of detection of bacteremia with increasing volumes of blood specimens, many studies have suggested that small culture volumes are at increased risk of false-negative results, just as in adults. [70] [111] [264] [292] Furthermore, the studies showing high CFU/mL rates were performed on neonates. As the immune system matures during infancy, levels of bacteremia might be expected to fall toward those seen in adults. In two reviews, it was recommended to obtain a similar volume of blood with respect to body mass as would be drawn in adults: approximately 1 mL/2.5 kg, or 4 mL per 10 kg body mass. [44] [207] These recommendations are summarized in Table 70-10 .

How Many Sets of Blood Cultures Should Be Tested?

A *set* of blood cultures is the sample obtained from a single site. A one mL specimen from a neonate placed in an aerobic bottle, and a 30 mL specimen from an adult divided between fungal, aerobic, and anaerobic bottles, are both a *single set* of blood cultures. Two or more sets of blood cultures make up a *series*. [8] [49] The information derived from the blood culture sets is pooled in such a way as to make both the sensitivity and specificity of the series greater than that of the component sets. Sensitivity is enhanced because even with continuous bacteremia, an individual set is usually only 80% sensitive. [8] Specificity is improved by determining whether pathogens that are also frequently contaminants are found in more than one set of the series.

While this conceptual process is applied to all blood culture series, the focus of inquiry varies depending on the infectious process being ruled in or out. For example, in an elderly patient with fever and purulent urine, it is extremely unlikely that the causative organism is a typical skin contaminant. The usual causes of "false-positive" blood cultures will therefore be easily recognized, thus lowering the false-positive rate for the series, and making for a test with intrinsically higher specificity. At the same time, with typical pathogens in this clinical context being nonfastidious organisms, sensitivity is typically around 99% with two sets of 20 mL blood per set.[287] Conversely, in a patient with a prosthetic heart valve, fever, and signs of septic emboli, many likely pathogens are also skin contaminants (this phenomenon lowers the specificity of each individual blood culture set), so at least two sets of cultures must be *positive* with such organisms before the overall test (i.e., of the *series*) is considered positive. At the same time, this clinical picture makes the pretest probability of disease very high (diminishing the negative predictive value of a negative set), so that an extremely sensitive overall test (i.e., series) will be needed to adequately rule out disease. Thus, in

TABLE 70-10 -- Optimal Specimen Volumes to Be Drawn per Blood Culture Set in Children *

Age Group/Weight (kg)	Ideal Volume of Specimen per Set (mL)
Neonates	1–2
Infants (5–10)	2–4
Children (7–20)	3–8
Children > 20	10
Children > 40	20
Adults and children > 60	30

*Rule of thumb: 4 mL of blood per 10 kg weight.

TABLE 70-11 -- Numbers of Blood Culture *Sets* to Be Obtained for an Adequate *Series* in Various Clinical Situations in Adults

No. of Sets (Minimum)	Clinical Context
2 sets	Etiology is likely to be easily distinguished from contaminants and pre-test probability of bacteremia is low to moderate
3 sets	Skin contaminants are possible causes of infectious process, *or* pre-test probability of bacteremia is high, *or* infectious endocarditis is a consideration, but with low to moderate pre-test probability
4 sets	Infectious endocarditis AND either moderate to high pre-test probability or the patient has recently been on antibiotics

this clinical context, most authorities would recommend four sets of blood cultures, with good volumes in each. [8] [280] Except in infants, single sets of blood cultures are of insufficient sensitivity or specificity to be of any utility, and should not be drawn. [8] [130] [202] [223] [280] [281] Recommended numbers of sets of blood cultures as they relate to the pre-test probability of disease, as well as causative organism are summarized in Table 70-11 .

Aerobic versus Anaerobic (vs Other) Bottles

Anaerobic infections, by nature, tend to occur in poorly perfused tissues or locations, frequently evolving into abscesses, which further isolate them from the bloodstream, decreasing the likelihood of bacteremia, and making them intrinsically elusive to blood cultures. In addition to these pathophysiological considerations, a significant decrease in the proportion of positive blood cultures owing to anaerobic organisms has been widely reported over the past 15 years. [67] [82] [168] [194] [239] The vast majority of anaerobic bacteremias occur in clinically identifiable situations listed in Table 70-12 .

In recent series, anaerobic pathogens account for only 1 to 5% of positive blood cultures. [67] [194] [239] [271] [280] Clinically significant isolates—those that could not have been predicted on the basis of the clinical picture, or that alter management—are much rarer. [168] Thus, with a typical true-positive blood culture rate of 3 to 7% and with only 3% of positive blood cultures being anaerobic, approximately 500 to 1000 series of blood cultures need to be drawn for every positive anaerobic blood culture. This is borne out by empiric observations showing positive anaerobic blood cultures in 1.4 per 1000 patients receiving blood cultures. [50] Of these, more than 90% had clinical indications of anaerobic infection, so that 10,000 blood culture series would be needed to generate a single anaerobic result to alter clinical management.[50] In addition to

TABLE 70-12 -- Infectious Processes Which can Cause Anaerobic Bacteremia

Odontogenic head and neck infections
Aspiration pneumonia
Abdominal/pelvic infections
Deep soft tissue infections (e.g., myofascitis)
Sepsis with decubitus ulcers or necrotic tissue

TABLE 70-13 -- Blood Culture Bottle Types to Be Used in Various Clinical Settings

Clinical Situation	Bottles to Be Obtained
Children <12 yr	Aerobic bottles only unless patient has peritonitis or fasciitis (in which case draw standard aerobic and anaerobic cultures)
Adults and children >12 yr	If anaerobic infection is unlikely, use aerobic bottles only unless the patient is immunocompromised
	In the immunocompromised, consider a bottle for fungal culture (usually effective in aerobic bottles, but consult the laboratory)
	If anaerobic infection is possible, each set consists of 1 aerobic and 1 anaerobic bottle per set

the very limited clinical utility of anaerobic cultures, they are likely to actually diminish the sensitivity of the test to aerobic bacteremia (>95% of cultured pathogens), which increases by about 3% per additional milliliter of blood. [183] [239] The use of blood culture specimens in anaerobic bottles will also diminish the likelihood of identifying fungal infections, which are increasingly common, especially in immunocompromised patients. [197]

The arguments for a selective use of anaerobic blood cultures are compelling. Based on these considerations, and analysis of a number of review articles, [49] [190] [197] [207] [239] [271] [280] Table 70-13 suggests guidelines for inoculation of blood obtained for culture.

Identifying Contaminants

The emergency clinician must be prepared for telephone calls from the laboratory with positive results of cultures obtained on previous shifts. "False-positive" blood cultures can be due to true contamination, but also may be caused by the intermittent bacteremia that occurs in normal, healthy people. This situation has been complicated by increasingly common identification of *Staphylococcus epidermidis* and *Streptococcus viridans* and fungi as pathogens in blood culture series. [63] [117] [242] [298] The expense of false-positive blood cultures has been estimated at $900 per episode for discharged patients, and more than $5,000 per episode for in-patients. These costs emphasize the importance of good technique in obtaining blood cultures. [15] [234]

Distinguishing contaminants from clinically significant bacteremia is based both on microbiologic information and the patient's clinical condition. Features of false-positive blood culture results are listed in Table 70-14 .[8] [40] [287] Notwithstanding these guidelines, it would probably be prudent to contact discharged patients with positive blood cultures even when contamination is suspected on a microbiological basis, to ensure that their condition is improving.

Fungal Cultures

Generally, fungi are difficult to isolate in blood cultures, and it may take 4 to 6 weeks to obtain a positive yield. If a fungemia is suspected, it is best to discuss culture media and technique with the laboratory before cultures are taken. Cultures

TABLE 70-14 -- Features Suggestive of Contaminant ("False Positive") Blood Culture Results

1. Coagulase-negative staphylococci (*S. epidermidis*) or *S. viridans* in a single bottle in patients not suspected of infectious endocarditis, and without chronic indwelling intravenous access catheters, are usually contaminants.
2. Corynebacteria (previously known as "diphtheroids"), propionibacterium acne, and bacillus species are usually contaminants, but can be pathogenic in the immunocompromised.
3. Multiple organisms in a series suggests contamination.
4. Species that grow out after prolonged culture have a higher likelihood of being contaminants. Conversely, early-growing bacteria have a much higher likelihood of being pathogens.[3] [145]
5. The patient's symptoms have resolved or are inconsistent with sepsis (beware with infectious endocarditis, which can have an indolent course).
6. A primary source (e.g., sputum, urine) has a different pathogen isolated.

of bone marrow are occasionally positive in deep mycoses when blood cultures are negative.

BEDSIDE TESTS FOR GASTROINTESTINAL HEMORRHAGE

Detection of Blood in the Stool

Bedside fecal blood tests make use of the peroxidase-like activity of hemoglobin. The test card is impregnated with a compound that exhibits a blue color reaction when oxidized. The original test used guaiac, but current tests use more sensitive and more reliable dyes. The addition of hydrogen peroxide developer solution will oxidize the dye in the presence of a peroxidase (e.g., hemoglobin).

Testing for occult blood in the stool is associated with false-positive and false-negative results, but in its primary role in emergency medical practice the test is usually reliable in detecting significant acute gastrointestinal (GI) hemorrhage. [114] Low pH, heat, dry stools, reducing substances (e.g., ascorbate), and antacids can cause false-negative findings. [116] [175] [220] Slow bleeding in the upper GI tract in which heme can be converted (denatured) to porphyrin during transit through the gut may not be identified by stool testing. False-positive results have been attributed to the ingestion of partly cooked or large quantities of meat (dietary sources of myoglobin and hemoglobin), and peroxidase-rich food. [87] [220] Most vegetables contain peroxidase, including (in decreasing order) broccoli, turnips, cantaloupe, red radishes, horseradish, cauliflower, parsnips, Jerusalem artichokes, bean sprouts, beans, lemon rind, mushrooms, parsley, and zucchini. [87] However, a simple in vivo study convincingly calls into question the possibility of peroxidase's passing through the stomach without being denatured. [185] False-positive tests can also be caused by the presence of povidone-iodine solution in concentrations less than 0.1% (a 1% dilution of the 10% solutions commonly available at the bedside). False-positive fecal occult blood tests are uncommon, and a positive test should be considered evidence of the presence of blood until proven otherwise. Routine iron supplementation should *not* be considered as a cause for a false-positive Hemoccult test, [6] [180]

although iron does (like bismuth preparations) cause the stools to appear black on gross examination. Despite this, early in vitro studies demonstrating an artifactual false-positive effect of iron are still frequently cited.

Normal GI blood loss is limited to less than 2.5 mL/day, which translates to less than 2 mg of hemoglobin per gram of stool (0.2% by weight). [2] The sensitivity of the Hemoccult test varies both with the concentration of hemoglobin present in the stool, and the extent to which hemoglobin is exposed to the proteolytic effects of the digestive tract. The Hemoccult test is 37% sensitive to stool containing 2.5 mg hemoglobin per gram of stool, but 95% sensitive when the concentration is 20 mg Hb/g of stool, indicating that low to moderate levels of blood may be missed. [99] The test is much more likely to detect lower GI hemorrhage than an identical rate of upper GI bleeding due to the 100-fold diminution of peroxidase activity of blood during transition through the GI tract. [71] Impaired detection of hemoglobin may also occur as a result of dilution due to diarrheal illness. [99] [114] [191]

Method

The stool specimen is smeared onto the reagent area on the card and a drop of developer is added. Because the reaction must occur in an aqueous medium, a drop of water should be added to very dry specimens and allowed to moisten them before addition of developer. Adding water will increase the false-positive rate, however. [175] [220] Formation of a blue color on the paper anywhere around or under the specimen within 60 seconds should be considered a positive result.

Testing for Gastric Blood

Heme tests designed for use on stool specimens can be unreliable when applied to gastric juices, with an increasingly high false-negative rate (low sensitivity) as pH decreases. [155] Thus, while a positive test of gastric contents using a fecal Hemoccult card is likely to be accurate, a negative test with the fecal Hemoccult card does not rule out the presence of blood. The Gastroccult card uses a modified guaiac developer containing buffers to neutralize gastric acid, thereby facilitating accurate hemoglobin detection. The test works on the same basis as the fecal guaiac test using the properties of hemoglobin as a peroxidase. In product testing, the Gastroccult card was 100% sensitive in detecting specimens of =500 parts per million of blood by volume, equivalent to 0.05%, or 0.25 mL of blood in 500 mL of gastric contents. Polyethylene glycol and high concentrations of iron (in an in vitro study simulating iron overdose) prevent both fecal occult blood tests from detecting blood. [106]

Method

Apply a drop of gastric aspirate to the test area. Apply two drops of developer to the sample. Look for formation of a blue dye within 1 minute. Do *not* use fecal blood test developer. In a specimen that is already a bilious green, the test is only considered positive if new *blue* color is formed. The Gastroccult card also contains a pH testing strip located close to the occult blood testing area, which might be useful in testing emesis after an acid or alkali ingestion. False-positive results might be expected to occur (although studies to investigate this have not been performed) with meats and peroxidase-rich foods. False-negative reactions are likely in the presence of reducing substances, such as ascorbic acid. The accuracy of Gastroccult should not be affected by the presence of cimetidine or sucralfate. [106]

DIAGNOSTIC AND THERAPEUTIC TOXICOLOGIC BEDSIDE PROCEDURES

The management of patients who present with an altered mental status can be challenging, especially if the clinician suspects drug overdose or poisoning. These patients often present with no available history or an inaccurate history. [295] Therefore, clinicians must rely heavily on physical examination findings and other sources of information to diagnose or confirm their clinical suspicions of poisoning or overdose. [48]

The hospital toxicology laboratory can be valuable in select cases. Limited screening tests for commonly ingested drugs are available, and ascertaining levels of specific drugs (e.g., acetaminophen, lithium, digoxin, phenytoin) can be helpful. However, most hospital laboratories are not equipped to perform timely analytic procedures for the thousands of possible drugs or toxins. In fact, the results obtained from the drug screening panels that most hospitals use have been shown to rarely influence medical management of adult ED patients. [127] [128] In select pediatric patients, on the other hand, the use of drug screens may have more of an impact on medical management.[18]

Diagnostic bedside testing for specific poisons or toxins has the advantage of being cost-effective and timely. When applied appropriately, certain bedside tests provide immediate information to the clinician and can have a significant and timely influence on medical management. This section discusses bedside diagnostic and therapeutic toxicologic procedures.

Noninvasive Diagnostic Procedures

Amatoxin: Meixner Test

The ingestion of several types of mushrooms (e.g., *Amanita phalloides*) can be fatal. The most poisonous of these are the mushrooms containing amatoxins. Patients who have ingested these mushrooms often complain of gastrointestinal symptoms consisting of nausea, vomiting, diarrhea, and abdominal cramping beginning 6 to 8 hours after ingestion. They often bring in specimens of the mushrooms chopped, crushed, cooked, or mixed with stool or gastric contents. Standard hospital laboratories cannot confirm or exclude the diagnosis of amatoxin poisoning; therefore, treatment decisions must be made on clinical grounds. [204] [276]

Meixner reported a simple colorimetric test for detecting amatoxins that can be used on gastric contents, stool, or actual mushroom samples. The basis of this test is the acid-catalyzed color reaction of amatoxins with lignin, a complex organic compound found in wood pulp. Cheaper grades of paper (e.g., newsprint or the white pages of a telephone book) contain high amounts of lignin. Although there have been no extensive reports of in vivo studies, in vitro tests have shown this method to be highly sensitive and relatively specific for amatoxins. [23] [156] Psilocybin-containing mushrooms can cause false-positive results for amatoxin. [22]

The procedure for a qualitative detection of amatoxin consists of squeezing a drop of liquid from a fresh mushroom sample or squashing a piece of fresh mushroom onto a piece of newspaper. If a stool or a gastric sample is the only

available specimen, the sample is mixed with reagent grade methanol (99.8%). The methanol will extract the amatoxin. If the samples are mixed with methanol, they should then be centrifuged and filtered. Place a drop of the liquid extract on the newspaper. Gently air dry all specimens at room temperature and avoid direct sunlight. Add two to three drops of concentrated hydrochloric acid (37%) to the dried specimen. Use an adjacent area for a control. High amounts of amatoxin in the dried samples exhibit a blue color in 1 to 2 minutes. Small amounts of amatoxin show a blue color in the sampled area in 10 to 20 minutes. Note that this procedure has *not* been proven effective using other bodily secretions, such as blood or urine. [23]

Mothball Identification

Present day commercial mothballs are composed of either paradichlorobenzene or naphthalene. Paradichlorobenzene is nontoxic whereas naphthalene can cause a significant hemolytic reaction in patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency and in neonates. [229] In the past, mothballs were also produced from camphor, which can cause central nervous system (CNS) depression and seizures in overdoses. Fortunately, these mothballs are no longer commercially available, although they may still exist in some households. A rapid differentiation between these groups of mothballs can expedite patient management and disposition. Several bedside tests that take advantage of the physical and chemical properties of these agents have been used to differentiate between them.

TABLE 70-15 -- Diagnostic Odors

Characteristic Odor	Responsible Drug or Toxin
Acetone (sweet, fruity; pear-like)	Lacquer, ethanol, isopropyl alcohol, chloroform, diabetic ketoacidosis, alcoholic ketoacidosis, trichloroethane, paraldehyde, chloral hydrate, methylbromide, *Pseudomonas* infections
Alcohols	Ethanol, (congeners) isopropyl alcohol
Ammonia-like	Uremia
Automobile exhaust	Carbon monoxide (odorless, but associated with exhaust)
Beer (stale)	Scrofula
Bitter almond	Cyanide
Carrots	Cicutoxin (or water hemlock)
Coal gas (stove gas)	Carbon monoxide (odorless, but associated with coal gas)
Disinfectants	Phenol, creosote
Eggs (rotten)	Hydrogen sulfide, carbon disulfide, mercaptans, disulfiram, *N*-acetylcysteine
Feculent	Intestinal obstruction
Fish or raw liver (musty)	Hepatic failure, zinc phosphide, hypermethioninemia, trimethylaminuria
Fruit-like	Nitrites (e.g., amyl, butyl), ethanol (congeners), isopropyl alcohol
Garlic	Phosphorus, tellurium, arsenic, parathion, malathion, selenium, dimethyl sulfoxide (DMSO), thallium
Halitosis	Acute illness, poor oral hygiene
Hay	Phosgene
Mothballs	Naphthalene, *p*-dichlorobenzene, camphor
Peanuts	*N*-3-pyridyl-methyl-*N*-*p*-nitrophenyl urea (Vacor)
Pepper-like	*O*-chlorobenzylidene malonitrile
Putrid	Anaerobic infections, esophageal diverticulum, lung abscess, scurvy

Rope (burned)	Marijuana, opium
Shoe polish	Nitrobenzene
Sweating feet	Isovaleric acid acidemia
Tobacco	Nicotine
Vinegar	Acetic acid
Vinyl-like	Ethchlorvynol (Placidyl)
Violets	Turpentine (metabolites excreted in urine)
Wintergreen	Methyl salicylate

From Chiang WK: Otolaryngologic principles. In Goldfrank LR, Flomenbaum NE, Lewin NA, et al (eds): Goldfrank's Toxicologic Emergencies. 5th ed. East Norwalk, Conn, Appleton & Lange 1994, p 374.

From 3rd edition Roberts and Hedges, p 1232, Figure 74-13.

1. Paradichlorobenzene is heavier than naphthalene, which is heavier than camphor. In lukewarm tap water, camphor will float while naphthalene and paradichlorobenzene will sink. In a solution of 3 tbsp of table salt thoroughly dissolved in 4 oz of lukewarm water, camphor and naphthalene will float and paradichlorobenzene will sink. [143]
2. Paradichlorobenzene has a lower melting point than naphthalene. Paradichlorobenzene mothballs will melt in a water bath at 53°C whereas naphthalene requires a water bath >80°C. [221]
3. Paradichlorobenzene is described as "wet and oily," whereas naphthalene is described as having a "dry" appearance. Paradichlorobenzene is familiar to many people as a cake of disinfectant used in urinals and diaper pails.

Body Secretion Analysis

Careful analysis of patients' bodily secretions, odor, and urine color can help identify certain toxins. Some characteristic smells and urine colors are noted in Table 70-15 and Table 70-16 .

Bedside Toxicologic Tests on Urine

Ethylene glycol.

Evaluation of the urine of patients who may have been exposed to ethylene glycol can be helpful. Microscopic inspection of urine for *calcium oxalate crystals* (a metabolic by-product of ethylene glycol metabolism) may be helpful in the diagnosis of ethylene glycol exposure. The presence of either envelope-shaped calcium dihydrate crystals or needle-shaped calcium monohydrate indicates high oxalate

TABLE 70-16 -- Drugs That Color Urine Yellow

Yellow
Quinacrine (atabrine) in acid urine
Riboflavin (large doses)
Yellow-green
Methylene blue, see blue
Yellow-orange
Fluorescein sodium
Yellow-pink
Cascara* in alkaline urine, see yellow-brown, brown, black
Senna* in alkaline urine, see yellow-brown, brown
Yellow-brown
Cascara* in acid urine, see yellow-pink, brown, black
Nitrofurantoin* (Furadantin and others), see brown
Orange
Phenazopyridine* (Pyridium), see red
Orange-red
Rifampin (Rifadin, Rifamycin, Rimactane)
Pink
Phenothiazines,* see red, red-brown
Phenytoin* (Dilantin), see red, red-brown
Red
Anthraquinone in alkaline urine
Deferoxamine (Desferal)
Methyldopa (Aldomet), see brown, black
Phenazopyridine* (Pyridium), see orange
Phenothiazines,* see pink, red-brown
Phenytoin* (Dilantin), see pink, red-brown
Red-purple
Phenacetin,* see brown
Red-brown
Phenothiazines,* see pink, red
Phenytoin* (Dilantin), see pink, red

Brown
Cascara* in alkaline urine, see yellow-brown, yellow-pink, black
Levodopa (Dopar)
Methocarbamol* (Robaxin), see green, black
Metronidazole (Flagyl)
Methyldopa* (Aldomet), see red, black
Nitrofurantoin* (Furadantin and others), see yellow-brown
Phenacetin,* see red-purple
Quinine,* see black
Senna* in alkaline urine on standing, see yellow-brown, yellow-pink
Blue
Methylene blue,* see green
Triamterene (Dyrenium), fluorescent
Blue-green
Amitryptyline (Elavil, Endep)
Green
Indomethacin (Indocin) from liver damage
Methocarbamol* (Robaxin), see brown-black
Black
Cascara* in alkaline urine on standing, see yellow-brown, yellow-pink, brown
Iron sorbitex* (Jectofer), see brown
Methocarbamol* (Robaxin), see brown, green
Methyldopa (Aldomet), see red, black
Quinine,* see brown
From Thoman M: Vet Hum Toxicol 1982;24:55. Used with permission.

From 3rd edition Roberts and Hedges, p 1233, Figure 74-14.

*Drug imparts more than one color to urine and is listed under each color it adds.

levels in the serum (Fig. 70-3). Calcium monohydrate crystals can be easily confused with sodium urate crystals; therefore, the presence of the dihydrate crystal tends to be more specific for ethylene glycol ingestion. The absence of these crystals does not rule out significant ethylene glycol ingestion, because the excretion of these may occur late in the ingestion (more than 6 hours) and occasionally does not occur at all. [107] [108] [115]

Visual inspection of urine under a Wood's lamp or ultraviolet light to ascertain *fluorescence* may also be helpful in the diagnosis of ethylene glycol exposure. Antifreeze is the most common source of ingested ethylene glycol. Fluorescein, a fluorescing material, is often placed in commercially available antifreeze to enable mechanics to detect radiator leaks with a Wood's lamp or other ultraviolet light source. Fluorescein is a nontoxic inert vegetable dye that is eliminated unchanged in the urine. Therefore, high levels of fluorescein in urine suggest significant ethylene glycol ingestion. However, a lack of fluorescein does not rule out a significant exposure, because not all antifreezes contain fluorescein or high concentrations of fluorescein in relation to ethylene glycol. False-positive findings can occur if certain plastic urine containers are used. [291]

To perform the test, fill glass test tubes with (1) the test urine sample, (2) a positive control urine sample (containing fluorescein) and (3) a negative control urine sample (not containing fluorescein). Inspect all three samples for fluorescence under a Wood's lamp in a dark room. The use of positive and negative controls may increase sensitivity and specificity from 49 and 75% to a sensitivity and specificity of 100%. [278] [291] Fluorescein is readily available in most EDs since fluorescein-containing strips are commonly used in ophthalmologic procedures (see Chapter 64).

Salicylates.

Several qualitative bedside tests have been developed to detect salicylates in urine. These include 10%

Figure 70-3 "Prism-shaped" calcium monohydrate crystals *(right)* resembling hippurate or urate crystals, and octagonal calcium dihydrate crystals *(left)*. *(Illustration by NJ Miller.)*

ferric chloride solution, Trinder solution, and Phenistix reagent strips. All tests are rapid, inexpensive, and sensitive.

Ferric chloride and Trinder solution both have sensitivities of 100% with serum salicylate levels of 5 mg/dL. False positives can occur with both tests in the presence of acetoacetic acid, acetone, and phenylpyruvic acid. Thus they may be falsely positive in patients with diabetic, alcoholic, or starvation ketoacidosis. Phenol-containing drugs such as diflunisal, sulfasalazine, and salicylamide may also produce false positives. A positive result, therefore, requires a confirmatory quantitative serum salicylate assay. [286]

The *ferric chloride test* is a commonly used rapid, qualitative, urinary screening procedure. To perform this test, several drops of 10% ferric chloride are added to 1 or 2 mL of urine that has been collected in a test tube. The immediate appearance of a bluish purple color signifies that salicylates are present in urine. This test is very sensitive, and as few as two aspirin taken within 24 hours will give a positive result. It requires 90 to 120 minutes from time of ingestion for this reaction to become positive in the urine of patients with normal renal function, so when tested before this time, the results may be misleading. [33]

The *Trinder test* uses a mixture of mercuric chloride and ferric nitrate in deionized water. To perform this test, 1 mL of urine is mixed with 1 mL of Trinder solution. A violet or purple color signifies the presence of salicylates. Acetoacetic acid and high levels of phenothiazines may give false-positive results. [136]

Phenistix reagent strips were originally developed to detect phenylketonuria. However, Phenistix strips also turn brown in the presence of salicylates. False-positive findings for salicylates can occur if phenothiazines are present. [29]

Bedside Toxicologic Tests on Oral Secretions and Breath

Ethyl alcohol.

There are several bedside devices to measure alcohol concentrations in bodily fluids. Measurements of alcohol concentration in expired air or saliva have been shown to correlate well with blood alcohol concentrations in the appropriate settings.

Breath alcohol analyzers were developed in the 1950s and are presently used in law enforcement. These devices typically use an infrared spectral analysis to determine the concentration of alcohol in expired air. Almost all the alcohol found in expired air at the level of the mouth is secondary to alcohol diffused from the bronchial system rather than the alveolar system. [101] Minor alterations in breathing patterns can cause large variations in readings. Thus, uncooperative patients who do not exhale properly may give an inaccurate reading. Other causes of inaccurate readings include the use of alcohol-containing products including ingesting them, belching or vomiting, use of inhalers, poor technique, or restrictive pulmonary pathology. [69] [152] [159]

A more recent technology for bedside measurement of alcohol concentration has been the use of a dipstick-like device to measure alcohol concentrations in saliva. These devices use an enzymatic reaction involving alcohol dehydrogenase to measure alcohol concentrations. [16] Patients who are dehydrated (a common occurrence in alcohol-intoxicated patients) are frequently unable to provide adequate saliva samples and inaccurate readings have occurred in patients with high blood alcohol concentrations. [19] [126]

Bedside Toxicologic Tests on Blood

Methemoglobinemia.

Patients with methemoglobinemia will often have a normal partial pressure of oxygen (pO_2) on routine arterial blood gas analysis, a normal *calculated* hemoglobin saturation, a nondiagnostic pulse oximeter reading, and cyanosis that does not clear with O_2 administration. Bedside visual inspection of venous or arterial blood may be helpful in the diagnosis of methemoglobinemia. Methemoglobinemia occurs when normal hemoglobin is exposed to an oxidant stress (Fe^{2+} converted to Fe^{3+}). If the erythrocytes cannot handle this stress, such as in the presence of G6PD deficiency, hemoglobin remains in an oxidized state (methemoglobin), causing a color change in the molecule. Methemoglobin levels higher than 15% are reported to cause a cyanotic appearance in a patient. [60]

The evaluation procedure for methemoglobinemia is to place a drop of sample blood on a white background (a white coffee filter is appropriate) in a well-lit environment. Next to this, place a drop of normal blood as a comparison control sample. Blood with methemoglobinemia appears "darker" or "chocolate-brown." [97]

This method relies on the ability of the examiner to distinguish color changes and therefore may have a degree of interobserver variance. Methemoglobin levels of less than 10% may only slightly alter the color of blood and thereby cause a false-negative finding. Methemoglobin levels of between 12% and 14% may cause a false-negative reading 50% of the time. However, at methemoglobin levels of 35% or higher, the identification of methemoglobinemia by visual inspection is quite accurate. [97] At this level, most patients are obviously cyanotic and significantly symptomatic.

Invasive Diagnostic Procedures

Several invasive diagnostic bedside procedures can be useful in the assessment of possible drug overdoses. The basic premise of these procedures is that patients who have been exposed to a certain drug or poison will respond in a particular fashion if given a diagnostic challenge dose of another particular drug or true antidote.

Naloxone

Naloxone hydrochloride (Narcan) is an opioid receptor antagonist that has the ability to reverse the effects of chemical agents affecting all opioid receptor sites, particularly respiratory and CNS depression. Because of this, a trial of naloxone has been recommended for all patients with CNS depression. [68] Certain clinical findings such as miosis, decreased respiratory rate, and evidence of illicit drug use can predict many patients who will respond to a diagnostic challenge dose of naloxone. [104]

If a patient's mental status improves significantly after a dose of naloxone, the patient should be considered to have been exposed to an opioid substance. This is true even if a laboratory drug screen is negative for opioids. One English study of laboratory drugs of abuse screens had false-positive rates of 4% and false-negative rates of 8%. [42] Furthermore, many of the synthetic opioid agents, such as fentanyl, propoxyphene, meperidine, methadone, and pentazocine may not be detected by the routinely used immunoassay drug screen. [78] Although cases have been reported of patients with other nonopioid overdoses (such as alcohol or phencyclidine) responding to

naloxone, those single observations have not been confirmed in controlled animal or human studies.

The traditional challenge dose of naloxone in an adult or child is 2 mg every 2 minutes IV until a response is achieved or 10 mg is given. [105] Some clinicians prefer to use much smaller doses (0.1 to 0.2 mg) and titrate to effect. This may partially reverse opioid overdose-related symptoms and confirm the diagnosis without precipitating the opioid withdrawal syndrome seen in patients with opioid dependency. Most patients with an opioid overdose will exhibit some response to 1 to 4 mg of naloxone, but some massive overdoses may require larger amounts. A patient who does not respond at all to 10 mg of naloxone probably does not have a pure opioid overdose.

The high doses of naloxone presently recommended are needed to reverse many synthetic narcotic agents, such as propoxyphene and methadone. Lower doses can be given (0.4 to 0.8 mg in adults or 0.01 mg/kg in children) to reverse known opioid-induced respiratory depression without reversing analgesia. Because naloxone has a half-life between 30 and 60 minutes, a continuous drip of naloxone can be used to avoid resedation. A reasonable choice is to set the hourly IV dose at two-thirds of the initial bolus dose that achieved the desired reversal effect. For example, a patient who satisfactorily responded to 1.5 mg of naloxone might receive a naloxone solution of 10 mg of naloxone in 500 mL of normal saline at a rate of 1 mg (50 mL)/hour IV. [88] Nalmefene, a long-acting opioid receptor antagonist that has a terminal half-life of roughly 11 hours, can also be given to patients with suspected overdoses. Theoretically, a single dose of nalmefene will be effective longer than the effects of heroin or most abused opiate substances. The initial recommended dose is 1.0 to 1.5 mg IV.

Naloxone and nalmefene have minimal significant side effects, other than precipitating withdrawal from patients addicted to opioids. Unlike alcohol withdrawal, naloxone-induced opioid withdrawal in the adult is short-lived and is usually not life-threatening. Withdrawal can be avoided if lower initial doses of naloxone or nalmefene are given and then are slowly titrated upward to the desired effect.

Flumazenil

Flumazenil is a competitive benzodiazepine receptor antagonist that has the ability to reverse the CNS and respiratory depression caused by all currently commercially available benzodiazepines. The use of flumazenil as a *routine diagnostic bedside challenge in all obtunded patients is discouraged,* and its use in the setting of possible benzodiazepine overdose is controversial. Unlike naloxone, flumazenil can have significant side effects in certain subsets of patients. [104] These include precipitating seizures or a withdrawal syndrome in benzodiazepine-dependent patients. To minimize the chance of seizures, flumazenil should be avoided in patients who may have ingested epileptogenic drugs (e.g., cyclic antidepressants, cocaine, theophylline, lithium, carbamazepine, isoniazid). [248]

In suspected benzodiazepine overdoses where patients present with obtundation and have no history of seizures or suspicion of involvement of epileptogenic agents, flumazenil can be administered IV at a dose of 0.2 to 0.5 mg/min. Most benzodiazepine-overdosed patients show mental status improvement with 1 mg of flumazenil and almost all respond to 3 to 5 mg. It is prudent to use small, escalating doses given very slowly (maximally, 0.5 mg/min). Larger doses can be given at one time as a bolus, although this increases side effects such as anxiety, agitation, and emotional lability; it also increases the chances of precipitating withdrawal in benzodiazepine-dependent patients.[247] Fortunately, seizures that occur after flumazenil use are usually transient and can frequently be controlled with additional benzodiazepines. In rare cases, higher doses of benzodiazepines, barbiturates, and phenytoin may be required. [248]

If a patient responds to flumazenil with an improvement in depressed mental status, this only suggests that the patient is under the influence of a benzodiazepine. Flumazenil can partially reverse the effects of many other agents or conditions that affect the ?-aminobutyric acid (GABA) pathway, such as zolpidem and hepatic encephalopathy[13] [92] [153] [275] ; however, it does not have any significant effect on alcohol, barbiturates, and other non-benzodiazepine sedative-hypnotics.

Physostigmine

Physostigmine is an acetylcholinesterase inhibitor that can penetrate into the CNS and thus reverse both the central and peripheral effects of anticholinergic agents. In the majority of patients with anticholinergic toxicity, no laboratory tests are available to rapidly confirm the diagnosis, and testing for specific drugs is limited. A clinical picture that may consist of mydriasis, dry and flushed skin, dry mucous membranes, urinary incontinence, absent bowel sounds, tachycardia, hyperthermia, hallucinations, agitation, and seizures suggests an anticholinergic toxicologic syndrome. A rapid and dramatic response to physostigmine often confirms a diagnosis of anticholinergic toxicity. In these patients, physostigmine reduces much of the CNS toxicity of the agents and decreases the degree of agitation and confusion. [17] [43] [201] The use of physostigmine as a diagnostic challenge can be helpful in select situations, but similar to flumazenil, the *routine use of physostigmine as a diagnostic bedside challenge in all obtunded patients should be discouraged*.

As a diagnostic challenge or therapeutic intervention, physostigmine can be administered IV under constant cardiac monitoring at a dose of 1 to 2 mg in adults and 0.02 mg/kg in children, over 5 minutes. Some clinicians empirically pretreat with a benzodiazepine to prevent seizures, but this practice has not been proven effective or necessary. Because the half-life of physostigmine is 30 to 60 minutes, a repeat dose of 2 mg can be given as clinically indicated.

Similar to flumazenil, physostigmine has been reported to interact detrimentally with cyclic antidepressants, often causing life-threatening dysrhythmias. Physostigmine also can cause an excess of acetylcholine and a resultant cholinergic crisis. This syndrome includes salivation, lacrimation, urination, defecation, bradycardia, bronchorrhea, and seizures. Dysrhythmias, including asystole, have also been reported. [210] For this reason, 1 mg of atropine IV should be readily available to reverse potential cholinergic excess when using physostigmine.

Deferoxamine

Deferoxamine is an organic compound derived from the bacterium *Streptomyces pilosus*. Deferoxamine can chelate iron and can be used as therapy or as a diagnostic challenge in patients with iron overdoses. Patients who have unstable vital signs or significant GI or CNS symptoms usually require therapeutic doses of deferoxamine. *Asymptomatic* patients with a

history of iron overdose usually require supportive care only. Patients with persistent but mild symptoms, such as vomiting and diarrhea, may be given a diagnostic challenge dose of deferoxamine. A diagnostic challenge is preferential over ancillary laboratory testing because tests such as iron levels and total iron binding capacity in the setting of iron overdose can be inaccurate, misleading, and time-consuming. [162] [241] [268]

A diagnostic challenge dose of deferoxamine is administered IM or IV over 45 minutes at doses of 40 to 90 mg/kg up to a maximum of 1 g in children and 2 g in adults. Deferoxamine can also be administered IV as a constant drip of 15 mg/kg/hour. A positive result occurs when chelated iron in the form of ferrioxamine appears in the urine. This usually causes the urine to turn a reddish orange or "vin rosé" color in 2 to 3 hours after initiation of treatment. The color change is qualitative only and has no prognostic significance. Color change caused by ferrioxamine is pH and concentration dependent, and false-negative test results occur. [181] [218]

Chronically administered deferoxamine has been reported to have multiple adverse effects, such as adult respiratory distress syndrome (ARDS), visual defects, and enhancement of *Yersinia enterocolitica* infections. In the setting of the single challenge dose, flushing, erythema, tachycardia, urticaria, and hypotension caused by rapid administration of deferoxamine are the most serious side effects.[289]

Invasive Therapeutic Procedures

The indications and rationale for use of certain therapeutic procedures in toxicology are often misunderstood.

Alkalinization of Urine and Blood

Alkalinization of urine consists of manipulating the pH of urine to enhance excretion of certain drugs (Table 70-17). Weak acids remain in ionic form in a basic milieu. The ionic form often prevents reabsorption of that drug in the proximal tubule, and urinary alkalinization can therefore promote elimination in the urine. For certain drugs, this can play a significant role in their elimination. For example, salicylate elimination increases proportionately to the urinary flow rate, but it increases *exponentially* with increases in the urinary pH.[189] [216]

Recommendations differ on the actual method or formula to achieve urinary alkalinization. No body of literature exists that supports one method of urinary alkalinization over another.[285] In general, this procedure should be titrated to the patient's fluid and acid-base status to achieve a urinary pH of 7.5 to 8.0. Many authors recommend the use of a constant infusion of a relatively isotonic solution consisting of 3 ampules of sodium bicarbonate (44 mmol/ ampule) added to 1 L of 5% dextrose in water (D_5 W). Another reasonable formula is to begin with a bolus of two ampules of IV sodium bicarbonate, or 1 to 2 mmol/kg of body weight. The bolus is

TABLE 70-17 -- Drugs That Have Increased Elimination with Urinary Alkalinization

Chlorpropamide
2,4-Dichlorophenoxyacetic acid
Formate
Methotrexate
Phenobarbital
Salicylates

followed with a constant infusion of three ampules of sodium bicarbonate in 1 L of D_5 W solution with 20 to 40 mmol of potassium (if the patient has normal renal function) infused at 100 to 300 mL/hour. Although repetitive boluses of sodium bicarbonate ampules also can be used, this may increase the chances of hypernatremia, hypokalemia, relative hypocalcemia, fluid overload, and alkalemia. All of these are potential adverse effects of aggressive urinary alkalinization. The actual amount of fluids and bicarbonate administered requires titration to the patient's clinical condition. Therefore careful monitoring of electrolyte, pH, and fluid status is encouraged.[267]

Urinary alkalinization can sometimes be difficult to achieve or maintain. Hypovolemia is probably the leading cause of an inability to achieve alkaline urine. Other theoretical causes are hypokalemia and hypochloremia. Several authors have suggested that in patients with severe salicylate poisoning, urinary alkalinization may

be difficult if not impossible to achieve with reasonable doses of bicarbonate. [285]

Ethanol Infusion

Recently, fomepizole (4-methylpyrazole) has been approved by the FDA for the treatment of ethylene glycol poisonings. It has also been used successfully in treating methanol poisonings.[31] [182] Compared to the traditional treatment of toxic alcohol poisoning, namely ethanol, fomepizole has the advantages of ease of use, fewer side effects (specifically hypoglycemia), and ability to maintain therapeutic levels. [30] [32] However, owing to the cost and the logistics of stocking this antidote, many hospitals may not have this drug readily available.

Ethanol can be used as a therapeutic intervention in patients with methanol or ethylene glycol poisoning due to ethanol's much greater affinity for alcohol dehydrogenases. These enzymes metabolize methanol and ethylene glycol to even more toxic by-products. However, with serum ethanol levels of 100 mg/dL, minimal amounts of ethylene glycol or methanol are metabolized by alcohol dehydrogenases. [31] [32] [41] Ethanol infusions are not useful in the treatment of isopropyl alcohol poisoning.

Ethanol can be administered orally or IV (Table 70-18). Intravenous ethanol has the advantages of obtaining therapeutic levels rapidly, ensuring complete absorption, limiting chances of aspiration, and avoiding gastritis. A 5% concentration of ethanol, which can be given in a peripheral vein, requires the use of large fluid volumes. In a 70-kg patient, a loading dose requires 1.4 L of 5% solution, with a maintenance dose of 700 mL/hr. In contrast, oral loading can be achieved using much lower volumes. However, oral loading can be difficult in the uncooperative or unconscious patient or if vomiting or GI hemorrhage is present. A therapeutic level is reached slower with oral loading.

Ethanol metabolism can vary widely, and ethanol is dialyzable. Therefore, it may be difficult to maintain appropriate ethanol levels during dialysis therapy of ethylene glycol or methanol. Frequent measurements of ethanol should be obtained and the infusion adjusted accordingly. [84] When patients are given ethanol infusions, CNS depression and hypoglycemia are common adverse effects (the latter is particularly true in diabetics and children). Serial levels of ethanol and glucose should be obtained. If IV ethanol is given, careful attention to cardiopulmonary status should be maintained. [142] [249]

TABLE 70-18 -- Ethanol in Methanol or Ethylene Glycol Poisoning [*]

Intravenous Ethanol: Loading Dose (using a 10% ethanol solution)[a] (A 10% volume/volume concentration yields approximately 100 mg/mL)						
	Volume of loading dose (given over 1–2 hr as tolerated)[a]					
	10 kg	*15 kg*	*30 kg*	*50 kg*	*70 kg*	*100 kg*
Loading dose of 1000 mg/kg of 10% ethanol (infused over 1–2 hours as tolerated); *assumes a zero ethanol level to start*	100 mL	150 mL	300 mL	500 mL	700 mL	1000 mL
Aim is to produce a serum ethanol level of 100–150 mg/dL						
Oral Ethanol: Loading Dose (A 20% volume/volume concentration yields approximately 200 mg/mL)						
	Volume of loading dose					
	10 kg	*15 kg*	*30 kg*	*50 kg*	*70 kg*	*100 kg*
Loading dose of 1000 mg/kg of 20% ethanol,[b] diluted in juice; may be administered orally or via nasogastric tube; *assumes a zero ethanol level to start*	50 mL	75 mL	150 mL	250 mL	350 mL	500 mL
Aim is to produce a serum ethanol level of 100–150 mg/dL						
Intravenous Ethanol: Maintenance Dose (using a 10% ethanol solution)[c] (A 10% volume/volume concentration yields approximately 100 mg/mL. Infusion to be started immediately following the loading dose. Aim is to maintain serum ethanol level of 100–150 mg/dL [†])						
	Infusion rate (mL/hr for various weights)[c]					
Normal maintenance range	*10 kg*	*15 kg*	*30 kg*	*50 kg*	*70 kg*	*100 kg*
80 mg/kg/hr	8	12	24	40	56	80
110 mg/kg/hr	11	16	33	55	77	110
130 mg/kg/hr	13	19	39	65	91	130
Approximate maintenance dose for chronic alcoholic						
150 mg/kg/hr[‡]	15	22	45	75	105	150
Range required during hemodialysis						
250 mg/kg/hr[‡]	25	38	75	125	175	250
300 mg/kg/hr[‡]	30	45	90	150	210	300
350 mg/kg/hr[‡]	35	53	105	175	245	350
Oral Ethanol: Maintenance Dose (A 20% volume/volume concentration yields approximately 200 mg/mL; infusion to be given each hour immediately following a loading dose; aim is to maintain serum ethanol level of 100–150 mg/dL; [†] each dose may be diluted in juice and given orally or via nasogastric tube)						
	Infusion rate (mL/hr [§] ***for various weights***[¶])					
Normal maintenance range	*10 kg*	*15 kg*	*30 kg*	*50 kg*	*70 kg*	*100 kg*
80 mg/kg/hr	4	6	12	20	28	40
110 mg/kg/hr	6	8	17	27	39	55
130 mg/kg/hr	7	10	20	33	46	66
Approximate range for chronic alcoholic or for patient receiving continuous oral activated charcoal						
150 mg/kg/hr	8	11	22	38	53	75
Range required during hemodialysis						
250 mg/kg/hr	13	19	38	63	88	125
300 mg/kg/hr	15	23	46	75	105	150
350 mg/kg/hr	18	26	52	88	123	175

*Note: *Concentrations higher than 10% are not recommended for IV administration. Concentrations higher than 30% are not recommended for oral administration.* The dose schedule is based on the premise that the patient initially has a *zero* ethanol level. The aim of therapy is to maintain a serum ethanol level of 100 to 150 mg/dL, but constant monitoring of the ethanol level is required because of wide variations in endogenous metabolic capacity. Ethanol is removed by dialysis, and the infusion rate of ethanol must be increased during dialysis. Prolonged ethanol administration may lead to *hypoglycemia*. Note: 10% ethanol for infusion may be difficult to find in the hospital pharmacy. To formulate 10% ethanol for infusion (1) remove 50 mL from a 1-L bottle of 5% ethanol/D_5W and replace it with 50 ml of 100% ethanol, or (2) remove 100 mL from a 1-L bottle of D_5W and replace it with 100 ml of 100% ethanol.

[a] If a 5% ethanol solution is used, *double* the volume of the loading dose.

[b] Equivalent to a 40 proof solution.

[c] If a 5% ethanol solution is used, *double* the volume rate; monitor closely for potential volume overload.
†Serum ethanol levels should be monitored closely.
‡At higher infusion rates, it may be necessary to administer by volume rather than by mL/hr.
§For a 30% concentration, divide the amount by 1.5.
¶Rounded off to nearest milliliter.

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Chapter 71 - Standard Precautions and Infectious Exposure Management

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Body fluid contamination of health care workers is a frequent occurrence in the emergency department (ED). In a prospective study of ED health care workers, skin and clothing contamination with body fluids occurred in 1 of 35 patient visits. [30] These fluids may contain various transmissible infectious diseases, as the prevalence of HIV infection, hepatitis, and other communicable diseases can be high in certain ED patient populations. [5] [24] [27] [43] For example, investigators from an inner city ED reported a patient seroprevalence of 6% for HIV infection, 18% for hepatitis C, and 5% for hepatitis B surface antigen. [26] One in four patients tested positive for at least one of these diseases. Patient characteristics were found to be poor predictors for hepatitis positivity, making it more difficult to identify which patients pose a risk to health care workers.

Unfortunately, compliance with standard precautions, formerly known as universal precautions, is far from universal. [3] [20] [25] Baraff and Talan[3] reported poor compliance, even in the setting of treating critical trauma patients. Compliance rates were 75% for gloves, 27% for gowns, 19% for eyewear, and only 2% for masks. Despite having recently received education about standard precautions, Hammond et al [20] also reported low compliance rates during invasive procedures and with high-risk patients. Compliance improved when equipment was organized and placed in trauma resuscitation rooms. In 1985, this combination of high-risk illness with low compliance barrier use prompted the Centers for Disease Control and Prevention (CDC) to recommend guidelines for the protection of health care workers. [7] In 1991, these recommendations were enacted into law by mandate of the Occupational Safety and Health Administration (OSHA).

Figure 71-1 *A,* Recapping a needle by holding the cap in the hand is the most common way to sustain a needle puncture. *B,* It is best to discard the needle/syringe without recapping, but an alternative is to partially recap *without* holding the guard (needle cap), so that at least 80% of the needle is covered before completing the recapping with the second hand.

The primary focus of the CDC guidelines is to reduce mucocutaneous body fluid exposures by encouraging hand washing and barrier protection. However, these measures do little to protect from percutaneous exposures, which are the most efficient exposures in the transmission of hepatitis and HIV. [31] [32] The current strategy for risk reduction in the ED includes immunization against hepatitis B virus, use of standard precautions (including re-engineered safety products), and prompt initiation of post-exposure prophylaxis (PEP) when appropriate.

STANDARD PRECAUTIONS GUIDELINES

Appropriate precautions for all patient contact must be viewed as a consistent practice or "way of life" in the ED. The following guidelines, based upon the CDC recommendations, should be used when there is any possibility of body fluid contact:

Barrier Precautions

1. *Gloves* should be used for any patient contact with the risk of body fluid exposure. Both cutaneous and percutaneous exposures can be reduced by the use of gloves. In an animal model, Mast et al.[33] reported a 46 to 86% reduction in the volume of blood transferred via needlestick injury when the needle first punctured a glove. Fisher et al[17] compared the biomechanical performance of powder-free, latex, and nitrile examination gloves. The nitrile examination gloves exhibited greater puncture resistance, despite being thinner than the latex examination gloves.
2. *Mask and protective eyewear* should be used when exposure to body fluid aerosols is possible (e.g., wound irrigation, traumatic chest wound).
3. *Gown and shoe covers* should be worn when there is the risk of large splash volumes of body fluids (e.g., chest tube, thoracotomy).

Sharps Precautions

Most importantly, this means no recapping, bending, or breaking of needles. If needle recapping is deemed necessary, a single-handed technique should be used (Fig. 71-1). A safer alternative is immediate disposal of the needle into an approved sharps container without recapping. In an observational study

of ED employees, the rate of needle recapping was 34%, and most practitioners used a two-handed technique. [22] There are various re-engineered products for use in the ED, including retracting scalpels, auto-capping needles, and needle-less intravenous systems. A survey of infection control professionals at Iowa and Virginia hospitals found that implementation of such devices was the most common action taken to decrease percutaneous injuries. [4]

Respiratory Precautions

During contact with patients with suspected or confirmed pulmonary tuberculosis, providers should wear a National Institute of Safety and Health (NIOSH)-approved N-95 particulate respirator. These masks are designed to efficiently filter 1- to 5-µm particles. A more costly and less comfortable alternative is the use of a HEPA-filtered mask. In addition, such patients should be placed into respiratory isolation in a room with negative pressure, high circulation (optimally at least 12 air changes per hour), and external exhaust. Procedures resulting in increased release of infectious droplets, such as sputum induction, should be avoided in the ED. Potentially infectious patients should wear a surgical-type mask themselves, especially during transportation outside of the respiratory isolation room (e.g., to radiology). [9]

Hand washing.

Any skin surface coming into contact with body fluids should be washed immediately with soap and water.

Annual education.

All workers should receive a mandatory annual review of infection control and safe practices.

OCCUPATIONAL DISEASE EXPOSURE

Occupationally acquired infections cause considerable morbidity and mortality among health care workers despite OSHA requirements for precautions. Given the often occult presentation of disease in the ED patient population, emergency health care workers are at high risk for significant exposure from many pathogens. Owing to the high prevalence of particular diseases among ED patients and specific concerns for pathogens associated with high morbidity and mortality, this chapter focuses on HIV, hepatitis B and C viruses (HBV, HCV), and tuberculosis.

TABLE 71-1 -- Recommendations for Hepatitis B Prophylaxis Following Percutaneous or Permucosal Exposure

	Source		
Exposed person	***HBsAg-positive***	***HBsAg-negative***	***Source unknown or not available***
Unvaccinated	HBIG × 1[*] and HB vaccine series	Initiate HB vaccine series	Initiate HB vaccine series
Vaccinated			
Known responder	No treatment	No treatment	No treatment
Known nonresponder	HBIG × 2 or	No treatment	If known high-risk source, treat as if source were HBsAg-positive
	HBIG × 1 and initiate revaccination[§]		
Response unknown	Test exposed person for anti-HBs	No treatment	Test exposed person for Anti-HBs
	1. If adequate,[†] no treatment		1. If adequate, no treatment
	2. If inadequate, HBIG × 1 plus vaccine booster		2. If inadequate, HB vaccine booster and recheck titer in 1 to 2 months

Adapted from Updated U.S. public health service guidelines for the management of occupational exposures to HBV, HCV, and HIV and recommendations for postexposure prophylaxis. MMWR Recomm Rep 50:1, 2001.

*HBIG dose 0.06 mL/kg IM.
§§The option of giving one dose of HBIG and reinitiating the vaccine series is preferred for nonresponders who have not completed a second three-dose vaccine series. For persons who previously completed a second vaccine series but failed to respond, two doses of HBIG are preferred.
†Adequate anti-HBs is = 10 mIU/mL.

Hepatitis B Virus

HBV Transmission

HBV is a well-recognized occupational risk for health care providers, and multiple studies have documented the high prevalence of hepatitis among ED patients. [16] [26] [28] Despite the attention focused on transmission of HIV, the infectivity of HBV is significantly higher because of the virulence of the organism and relatively small inoculum required for disease transmission.[18] Percutaneous injuries are among the most efficient modes of HBV transmission, but many infected health care workers do not recall a specific injury. [19] [39] Many body fluids other than blood contain hepatitis B surface antigen, but the levels of infectious HBV particles in blood-free body fluids are 100 to 1000 times lower than blood itself. Because implementation of the CDC's standard precautions, along with the OSHA regulations for barrier protection and pre-exposure vaccination, the incidence of HBV transmission has sharply declined. [14]

To understand the risk of HBV transmission resulting from occupational exposure, one must be familiar with a few key serologic markers for HBV. Hepatitis B surface antigen (HBsAg) is a marker of active infection in the source patient. From a practical standpoint, HBV can be transmitted when HBsAg is present, and is generally not transmissible when this marker is absent. Hepatitis B surface antibody (HBsAb) is a protective antibody against HBV. In vaccinating health care workers, the goal is to stimulate the immune system to produce a sufficient quantity of this antibody. Hepatitis Be antigen (HBeAg) can be found in the bloodstream of HBV-infected individuals during times of peak virus replication. When a source is positive for HBeAg, their bloodstream contains a much larger number of infectious HBV particles. If a non-immune individual sustains a needle stick from an HbsAg-positive patient, the risk of HBV transmission is dependent upon the HBeAg status of the source. [50] The risk of clinical hepatitis is approximately 2% (range, 1% to 6%) if HBeAg is absent, compared with a risk of 22% to 31% if HBeAg is present. [14]

HBV Post-exposure Management

Post-exposure prophylaxis following exposure to an HBsAg positive source may require hepatitis B vaccine, HBIG, both, or neither (Table 71-1). This is dependent upon the vaccination and antibody response status of the exposed health care worker. HBIG is derived from pooled human plasma and

provides passive immunization for non-immune exposed individuals. This preparation is very safe and is not known to transmit disease. [14] When HBIG is used for PEP, ideally it should be given within 24 hours following exposure and it is of questionable value beyond 7 days. [8] In some cases, hepatitis B vaccine is also used for post-exposure prophylaxis. Health care workers who have any chance of exposure to infectious body fluids should be routinely vaccinated against hepatitis B. Adverse reactions to the hepatitis B vaccine are generally quite mild, and it is even safe to give during pregnancy. Primary immunization consists of an initial intramuscular (IM) injection, with subsequent IM vaccinations at 1 and 6 months. Antibody levels (HBsAb) should be checked at 4 to 6 weeks after the series is completed, and the desired titer is at least 10 mIU/mL. Vaccinated individuals who achieve this antibody level are referred to as "responders" and may be immune for life. While about 25% to 50% of vaccine responders demonstrate a decline in HBsAb antibody levels to below 10 mIU/mL within 5 to 7 years, these individuals are still protected against clinical disease. This results from a robust immune system memory or anamnestic response. [23] Post-exposure prophylaxis with these agents is not contra-indicated during pregnancy or lactation. Health care workers who have previously been infected with HBV are immune to re-infection, so PEP is not indicated in such individuals.

Hepatitis C Virus

HCV Transmission

Approximately 1.8% of Americans (3.9 million) are infected with hepatitis C virus, and many of these individuals are unaware they are infected. HCV is often acquired from injection drug use, and was once commonly transmitted by blood transfusion (now rare with modern screening). While HCV can be transmitted sexually, this a minor route. Percutaneous transmission is most efficient. The incidence of seroconversion following an HCV-positive needle stick is about 1.8% (estimates range from 0 to 7%).[14] Mucous membrane transmission of HCV is possible but much less common. It is useful to remember that the risk of HCV transmission following a needle stick is similar to that of HBV transmission when the source is HBeAg negative. When seroconversion does occur, 80% of patients will demonstrate antibodies at 15 weeks and 97% at 6 months following exposure. While the clinical course of hepatitis C virus is often asymptomatic or mild, approximately 85% of patients will

develop chronic hepatitis, 10% to 20% cirrhosis, and 1% to 5% hepatocellular carcinoma. [10] [11] [12]

HCV Post-exposure Management

Unfortunately, post-exposure prophylaxis for HCV exposure is currently not available. HCV exhibits a high degree of genetic heterogeneity and a very rapid mutation rate, making the development of vaccine extremely difficult. The use of post-exposure immune globulin is probably not helpful, and there are currently no clinical trials of agents such as interferon or ribavirin for HCV post-exposure prophylaxis. [14]

Human Immunodeficiency Virus

HIV Transmission

According to the CDC, through June 2000, there were 56 cases of occupational HIV transmission to health care workers in the United States. Additionally, another 138 health care workers demonstrated HIV seroconversion, which may have been occupationally related. [13] The risk of contracting HIV from working in the ED depends upon the prevalence of HIV in the local patient population. One study reported an annual HIV seroconversion risk of 1/3800 for high-prevalence EDs and 1/55,000 for low-prevalence EDs.[32] Wears et al.[49] estimated the cumulative career risk of contracting HIV from occupational exposure in a high-prevalence ED to be as high as 1.4%. The author suggests, however, that this risk can be reduced with adequate precautionary measures. When seroconversion occurs, HIV antibodies can be detected as early as 3 weeks after exposure, and are almost always present by 6 months. Seroconversion at 6 to 12 months is rare, but has been reported with HIV and HCV virus co-infection. Acute retroviral syndrome is a clinical manifestation of HIV seroconversion that occurs in approximately 80% of newly infected individuals at a median of 25 days after exposure. The presentation of acute retroviral syndrome is similar to mononucleosis, with fever, lymphadenopathy, and rash.

The overall risk of HIV seroconversion is about 1/300 (0.3%) following needlestick and less than 1/1000 for mucous membrane exposures. Cardo et al. [6] demonstrated that the risk for HIV seroconversion following needlestick injuries is not uniform. Seroconversion was found to be more likely for deep injuries (odds ratio [OR] = 15), if blood was visible on the device (OR = 6.2), if the needle had been used in a source patient's artery or vein (OR = 4.3), or if the source patient suffered from terminal AIDS (OR = 5.6). It is essential to gather information regarding the nature of the injury to "risk stratify" the exposure.

HIV Post-exposure Management (CDC, 2001)

Evidence supporting post-exposure prophylaxis.

In 1998, the U.S. Public Health Service recommended the use of post-exposure prophylaxis (PEP) for selected HIV exposures. [11] [12]

These recommendations were based upon a number of animal and human studies suggesting that post-exposure prophylaxis may be effective. While animal studies are mixed in both methodology and outcomes, PEP with various agents has successfully prevented HIV infection. In human studies, the use of anti-retroviral agents during pregnancy decreased perinatal HIV transmission by 67%. [15] In addition, when children born to HIV-positive mothers were given HIV PEP within 48 hours of birth, HIV transmission was also decreased.[47] While perinatal exposures are different than occupational needle sticks, this evidence supports the concept of a "window of opportunity" during which PEP may prevent HIV transmission to an exposed individual. The most important human study of the efficacy of PEP is a CDC-sponsored case-control study undertaken in the United States, France, the United Kingdom, and Italy. [6] This investigation compared 33 health care workers who seroconverted following HIV exposure with 665 control health care workers who did not seroconvert following HIV exposure. About 90% of patients in this study were exposed via hollow bore needles. When post-exposure zidovudine (AZT) was used, the risk for HIV infection was reduced by 81% (95% confidence interval, 48% to 94%). While the study methodology is limited by its retrospective design and the potential for recall bias, these results strongly support the efficacy of AZT for PEP. Currently there are no published randomized controlled human trials of agents for

HIV PEP. Given the results of the CDC case-control study, it is highly unlikely that such trials will ever be published, as the use of a control group is now considered unethical.

Selecting patients for PEP.

In June 2001, the U.S. Public Health Service published updated recommendations regarding the use of HIV PEP. [14] In general, the decision to use PEP depends on the type of exposure and the source HIV status. The first step in determining if PEP is indicated is to assess the exposure severity. Percutaneous exposures can be categorized as less severe or more severe. A less severe exposure involves a solid needle, a superficial injury, and without blood visible on the device. All other percutaneous injuries are categorized as more severe. Exposure to mucus membrane and non-intact skin are categorized as either small volume (few drops of blood) or large volume (a major blood splash). There are no reported cases of HIV seroconversion following blood exposure to intact skin.

Following assessment of the exposure severity, one must next determine the potential infectivity of the source. PEP should only be considered for blood and body fluid exposures from a source who is known to be or likely to be HIV-positive. Exposures from an HIV-negative source do not require PEP. Testing of sharp instruments for HIV is not recommended or reliable. HIV-positive source patients are categorized as either lower risk (class 1) or higher risk (class 2). Class 1 patients have asymptomatic HIV infection and a low viral load (less than 1500 RNA copies/mL). Higher risk patients include those with symptomatic HIV, AIDS, acute seroconversion, or a high viral load. Once exposure severity and source HIV status are determined, Table 71-2 and Table 71-3 can be used to guide the proper PEP regimen. For skin and mucous membrane exposures, the PEP regimen chosen falls into three general categories. For small volume exposures from an HIV class 1 source, the basic regimen (two drugs) should be *considered*. If either the exposure is of large volume or the source is HIV class 2, then the basic regimen should be *recommended*. In cases where there is both a large volume exposure and an HIV class 2 source, the expanded regimen (three drugs) should be recommended. For most percutaneous exposures, the expanded regimen should be recommended. However, for less severe percutaneous exposures from an HIV class 1 source, the basic regimen should be recommended.

A number of special circumstances may arise when determining the need for HIV PEP. When a source is known, but his or her HIV status is pending, the use of PEP should be decided on a case-by-case basis. When the source is high-risk, PEP can be initiated and then stopped or modified once the HIV status is determined. When a source can be identified, but his or her HIV status is unknown and will not become available, PEP is generally not recommended. However, use of the basic two-drug regimen should be considered if the source has HIV risk factors. Sometimes an exposure will occur where the source is completely unknown. While PEP is generally not recommended for such exposures, the basic regimen should be considered if the exposure occurred in an HIV-likely setting (e.g., exposure to a discarded needle on an AIDS ward).

Choice of PEP medications.

When HIV PEP is administered, a minimum of two drugs is recommended. While there is no direct evidence that combination PEP regimens are beneficial, concerns about antiretroviral resistance and the synergistic effects of antiviral medications when treating patients with AIDS supports such an approach. As discussed earlier, the U.S. Public Health Service recommends using a basic (two-drug) PEP regimen for lower risk HIV exposures and an expanded (three-drug) PEP regimen for higher risk exposures. The basic PEP regimen consists of two nucleoside reverse transcriptase inhibitors, usually zidovudine (AZT) plus lamivudine (3TC). Alternative basic regimens include 3TC plus d4T (stavudine) or d4T plus ddI (didanosine). When using the expanded PEP regimen, either a protease inhibitor or a non-nucleoside reverse transcriptase inhibitor is added to the basic regimen. Most commonly either nelfinavir or indinavir is preferred as the third drug. A number of second-line and alternative agents may be chosen for HIV PEP. Expert consultation is recommended if antiretroviral resistance is suspected. An important resource for emergency clinicians is the national clinicians' postexposure prophylaxis hotline at UCSF/San Francisco General Hospital. Expert consultation can be obtained by calling 888-448-4911.

PEP timing, duration, and side effects.

HIV exposure should be considered a true emergency, and PEP should be initiated as soon as possible after exposure, ideally within 1 hour. Animal studies indicate

that the efficacy of PEP diminishes with delayed initiation.[42] HIV PEP regimens consist of a 4-week course of therapy. In the ED, patients can be prescribed the first 3 days of medications, as long as outpatient follow-up is arranged. Side effects are experienced by about 50% of health care workers taking PEP, causing approximately 33% of health care workers to discontinue therapy prematurely.[43] Table 71-4 lists some of the major side effects experienced when taking PEP agents. Depending upon the choice of PEP medications, patients should also be prescribed antiemetics and antidiarrheal agents when PEP is initiated. A very useful and practical resource that can be used in the ED is the UCLA needle-stick Web site, www.needlestick.mednet.ucla.edu. This Web site allows one to enter information about a specific patient exposure. It then advises the clinician of the appropriate tests to obtain, recommends a PEP regimen, and provides printable discharge instructions and prescriptions.

Tuberculosis

Tuberculosis Transmission

During the mid-1980s the United States experienced a resurgence in tuberculosis (TB), especially among HIV-positive patients. This disease poses a serious risk to both public health and health care workers. Tuberculosis is transmitted by infectious droplets 1 to 5 µm in size. Primary infection occurs when one to three organisms are inhaled into the alveoli, where they begin to replicate. Host defenses usually stop infection within 2 to 10 weeks, and the patient enters the latent period. During this time, patients are not contagious and are asymptomatic. Reactivation occurs when cell-mediated immunity wanes, and patients are again contagious. This can be due to advancing age, HIV infection, steroid use, malignancy, malnutrition, or other causes of immune suppression. The lifetime risk of reactivation is 10%, with about half of this risk occurring in the first 2 years after primary infection. Patients with increased infectivity include those with pulmonary or laryngeal TB, an active cough, positive sputum smears for acid-fast bacilli, cavitation on chest radiographs, and those on inadequate therapy. Children are overall less contagious than adults, but can still transmit

TABLE 71-2 -- Recommended HIV Postexposure Prophylaxis for Percutaneous Injuries

Infection Status of Source					
Exposure type	***HIV-positive class 1****	***HIV-positive class 2****	***Source of unknown HIV status**†*	***Unknown source**§*	***HIV-negative***
Less severe¶	Recommend basic 2-drug PEP	Recommend expanded 3-drug PEP	Generally, no PEP warranted; however, consider basic 2-drug PEP‡ for source with HIV risk factors£	Generally, no PEP warranted; however, consider basic 2-drug PEP‡ in settings where exposure to HIV-infected persons is likely	No PEP warranted
More severe¢	Recommend expanded 3-drug PEP	Recommend expanded 3-drug PEP	Generally, no PEP warranted; however, consider basic 2-drug PEP‡ for source with HIV risk factors£	Generally, no PEP warranted; however, consider basic 2-drug PEP‡ in settings where exposure to HIV-infected persons is likely	No PEP warranted

Adapted from: Updated U.S. Public Health Service Guidelines for the Management of Occupational Exposures to HBV, HCV, and HIV and Recommendations for Postexposure Prophylaxis. MMWR Recomm Rep 50:1, 2001.

*HIV-Positive, Class 1—asymptomatic HIV infection or known low viral load (e.g., <1500 RNA copies/mL). HIV-Positive, Class 2—symptomatic HIV infection, AIDS, acute seroconversion, or known high viral load. If drug resistance is a concern, obtain expert consultation. Initiation of postexposure prophylaxis (PEP) should not be delayed pending expert consultation, and, because expert consultation alone cannot substitute for face-to-face counseling, resources should be available to provide immediate evaluation and follow-up care for all exposures.
†Source of unknown HIV status (e.g., deceased source person with no samples available for HIV testing).
§Unknown source (e.g., a needle from a sharps disposal container).
¶Less severe (e.g., solid needle and superficial injury).
‡ The designation "consider PEP" indicates that PEP is optional and should be based on an individualized decision between the exposed person and the treating clinician.
£If PEP is offered and taken and the source is later determined to be HIV-negative, PEP should be discontinued.
¢More severe (e.g., large-bore hollow needle, deep puncture, visible blood on device, or needle used in patient's artery or vein).

TABLE 71-3 -- Recommended HIV Postexposure Prophylaxis for Mucous Membrane Exposures and Non-intact Skin* Exposures

Infection Status of Source					
Exposure type	***HIV-positive class 1**†*	***HIV-positive class 2**†*	***Source of unknown HIV status**§*	***Unknown source**‡*	***HIV-negative***
Small volume£	Consider basic 2 drug-PEP¶	Recommend basic 3-drug PEP	Generally, no PEP warranted; however, consider basic 2-drug PEP¶ for source with HIV risk factors¢	Generally, no PEP warranted; however, consider basic 2-drug PEP¶ in settings where exposure to HIV-infected persons is likely	No PEP warranted
Large volume	Recommend basic 3-drug PEP	Recommend expanded 3-drug PEP	Generally, no PEP warranted; however, consider basic 2-drug PEP¶ for source with HIV risk factors¢	Generally, no PEP warranted; however, consider basic 2-drug PEP¶ in settings where exposure to HIV-infected persons is likely	No PEP warranted

Adapted from Updated U.S. Public Health Service Guidelines for the Management of Occupational Exposures to HBV, HCV, and HIV and Recommendations for Postexposure Prophylaxis. MMWR Recomm Rep 50:1, 2001.

*For skin exposures, follow-up is indicated only if there is evidence of compromised skin integrity (e.g., dermatitis, abrasion, or open wound).
†HIV-Positive, Class 1—asymptomatic HIV infection or known low viral load (e.g., <1500 RNA copies/mL). HIV-Positive, Class 2—symptomatic HIV infection, AIDS, acute seroconversion, or known high viral load. If drug resistance is a concern, obtain expert consultation. Initiation of postexposure prophylaxis (PEP) should not be delayed pending expert consultation, and, because expert consultation alone cannot substitute for face-to-face counseling, resources should be available to provide immediate evaluation and follow-up care for all exposures.
§Source of unknown HIV status (e.g., deceased source person with no samples available for HIV testing).
Unknown source (e.g., splash from inappropriately disposed blood).
‡Large volume (e.g., major blood splash).
£Small volume (e.g., a few drops).
¶The designation "consider PEP" indicates that PEP is optional and should be based on an individualized decision between the exposed person and the treating clinician.
¢If PEP is offered and taken and the source is later determined to be HIV-negative, PEP should be discontinued.

TABLE 71-4 -- Primary Side Effects Associated with Antiretroviral Agents

Antiretroviral Class/Agent	**Primary Side Effects and Toxicities**
Nucleoside reverse Transcriptase Inhibitors (NRTIs)	
Zidovudine (Retrovir; ZDV; AZT)	Anemia, neutropenia, nausea, headache, insomnia, muscle pain, and weakness
Lamivudine (Epivir; 3TC)	Abdominal pain, nausea, diarrhea, rash, and pancreatitis
Stavudine (Zerit; d4T)	Peripheral neuropathy, headache, diarrhea, nausea, insomnia, anorexia, pancreatitis, increased liver function tests (LFTs), anemia, neutropenia, and ascending neuromuscular weakness

Didanosine (Videx; ddI)	Pancreatitis, lactic acidosis, neuropathy, diarrhea, abdominal pain, and nausea
Abacavir (Ziagen; ABC)	Nausea, diarrhea, anorexia, abdominal pain, fatigue, headache, insomnia, and hypersensitivity reactions
Nonnucleoside Reverse Transcriptase Inhibitors (NNRTIs)	
Nevirapine (Viramune; NVP)	Rash (including cases of Stevens-Johnson syndrome), fever, nausea, headache, hepatitis, and increased LFTs
Delavirdine (Rescriptor; DLV)	Rash (including cases of Stevens-Johnson syndrome), nausea, diarrhea, headache, fatigue, and increased LFTs
Efavirenz (Sustiva; EFV)	Rash (including cases of Stevens-Johnson syndrome), insomnia, somnolence, dizziness, trouble concentrating, and abnormal dreaming
Protease Inhibitors (PIs)	
Indinavir (Crixivan; IDV)	Nausea, abdominal pain, nephrolithiasis, and indirect hyperbilirubinemia
Nelfinavir (Viracept™; NFV)	Diarrhea, nausea, abdominal pain, weakness, and rash
Ritonavir (Norvir™; RTV)	Weakness, diarrhea, nausea, circumoral paresthesia, taste alteration, and increased cholesterol and triglycerides
Saquinavir (Fortovase; SQV)	Diarrhea, abdominal pain, nausea, hyperglycemia, and increased LFTs
Amprenavir (Agenerase; AMP)	Nausea, diarrhea, rash, circumoral paresthesia, taste alteration, and depression
Lopinavir/Ritonavir (Kaletra)	Diarrhea, fatigue, headache, nausea, and increased cholesterol and triglycerides

Adapted from Updated U.S. Public Health Service Guidelines for the Management of Occupational Exposures to HBV, HCV, and HIV and Recommendations for Postexposure Prophylaxis. MMWR Recomm Rep 50:1, 2001.

the disease. Extrapulmonary TB is only contagious in the cases of an open skin lesion or oral cavity involvement. [9]

Depending upon the patient population and geographic location, ED personnel can be at high risk for occupational TB infection. In a 1993 study at a county hospital in Los Angeles, it was reported that 31% of ED workers became PPD-positive during employment, including 20% of attendings, 32% of nurses, and 33% of residents.[44] PPD conversion risk was found to be 6% after 1 year of ED employment, 14% after 2 years, and 27% after 4 years. EDs typically care for higher risk patients—those who are homeless, foreign-born, recently incarcerated, or chronically debilitated. Overcrowding can lead to extended waiting periods and delays in admissions. The clinical presentation of TB in ED patients is often atypical, and this can lead to delayed diagnosis. [45] [46] This is especially true for HIV-infected patients, where symptoms may mimic *Pneumocystis carinii* pneumonia (PCP), skin tests are often negative, chest radiographs are commonly atypical, and sputum tests may be less sensitive.[2] [21] [25] [36] [37] [38] [41] In one ED study of patients with TB, the mean time from ED registration to respiratory isolation was 6.5 hours, and 46% of patients were first isolated on the hospital ward.[34]

Preventing TB exposure requires a multifaceted approach.[9] Proper ED ventilation plays a key role, and inadequate ventilation has been a contributing factor in many nosocomial outbreaks. Ideally, there is single-pass airflow from waiting rooms to the outside. Within the ED, air should flow from clean areas to less clean areas, not vice versa. If patients with TB are seen frequently, there should be at least one true respiratory isolation room available. Such rooms are recommended to have at least 12 air changes per hour and should be "negative pressure" (air flows *into* the room from other ED areas). Other engineering approaches to TB infection control include the use of HEPA filters and upper room ultraviolet light irradiation.

ED personnel should be familiar with the appropriate use of respiratory protection against TB. Surgical masks (e.g., string-tie masks) are indicated for source control. These should be placed on potentially contagious patients to decrease the production of infectious droplets into the air. Because air can leak around such masks, they are not optimal for health care worker protection. In late 1995, NIOSH certified a new class of masks known as N-95 particulate respirators. [40] These filter 1-µm-size particles with at least 95% efficiency, and are the preferred mask for health care workers. They are available in a variety of shapes, sizes, and manufacturers. Before these masks were available, HEPA-filtered masks were used for respiratory protection. While these are very effective, they are more costly and can make breathing uncomfortable.

Attempts should be made to initiate respiratory isolation of patients with suspected pulmonary tuberculosis as soon as possible in the ED, ideally at triage. Screening protocols at triage can detect patients with more classic presentations of TB, but unfortunately, reported protocols are only moderately sensitive and somewhat cumbersome.[45] [46] Immediate respiratory isolation should be considered for patients with high-risk chief complaints, such as HIV-positive patients with cough, those with hemoptysis, or patients with a history of TB presenting with cough or fever. The best guideline is to initiate respiratory isolation as soon as TB is considered as a possible diagnosis. Masks should be placed on such patients *prior* to obtaining chest radiographs.

Tuberculosis Post-exposure Management

Health care workers who are exposed to patients with active pulmonary TB should be referred to either employee health care or their primary care clinician for follow-up testing and treatment. To establish the health care worker's baseline PPD status, skin testing is usually performed within days following exposure. If the baseline test is negative, a follow-up skin test is then performed 3 months later to determine if PPD-conversion has occurred. A positive baseline test indicates previous exposure or infection. Health care workers who convert their PPD to positive (5-mm induration) after an exposure should undergo chest radiography to screen for active pulmonary TB. If active disease is present, treatment should be initiated with at least four anti-tuberculous medications. PPD-converters who do not have active disease should be considered for chemoprophylaxis. When deciding whether or not to initiate chemoprophylaxis, one must balance the potential benefit of TB prevention with the risk of medication-associated hepatitis. In general, chemoprophylaxis should be given in the case of a recent PPD-conversion (within 2 years), a known TB contact, a patient who is medically predisposed to TB, HIV-infected or using intravenous drugs, or younger than 35 years. This means that occupationally exposed health care workers who are PPD-converters should usually receive chemoprophylaxis, regardless of age. The preferred regimen for HIV-negative persons is daily isoniazid (INH) for 9 months, although acceptable alternative regimens can be considered. [1] For exposures to multi-drug resistant TB (MDR-TB), expert consultation is advised for selecting an individualized regimen. Baseline and serial liver function testing is not necessary in most cases, but close monitoring for clinical symptoms suggestive of hepatotoxicity is very important.

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Chapter 72 - Educational Aspects of Emergency Department Procedures

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The psychomotor skills required to perform many emergency department (ED) procedures are complex and the acquisition of these motor skills is a highly individualized educational process. Although many of these skills are difficult to learn, emergency clinicians can become extremely competent in their performance when provided with adequate training and sufficient practice.

EDUCATIONAL THEORY AND PROCEDURAL SKILL TRAINING

According to the behaviorist theory, the learning of a motor skill can be divided into three phases: a cognitive phase, when the student learns the individual procedural steps; an associative phase, when these specific steps are mastered and integrated together; and an autonomous phase, when the entire skill can be executed smoothly and without any cognitive input.[19]

Elsewhere in the literature, four types of learning have been described as essential for acquiring competence in performing emergency surgical procedures.[11] All four types of learning are essential to mastering a procedural skill. They include:

Memorization of facts.

The clinician must memorize the critical steps of the procedure and their sequence.

Application of rules.

The clinician must be able to apply rules in novel situations, such as the indications and contraindications of performing the procedure.

Visual cue recognition.

The clinician must be able to recognize visual cues, primarily the critical structures of human anatomy.

Psychomotor coordination.

The clinician must be able to coordinate the mind and the body when performing the procedure.

Procedures can be taught to trainees in a consistent and organized manner by observing a set of eight steps for procedural learning as described by Chapman[11] and shown in Box 72-1 . These steps incorporate the four types of learning that are important to understand when teaching a procedural skill or when testing for procedural competency.

Long-Term Procedural Skill Retention

Long-term retention of procedural competency is also an important aim of training because some decay of procedural skills is naturally observed in complicated psychomotor tasks. Educators would like to know how long the effect of procedural training will last and how often clinicians need to practice in order to retain speed and accuracy for resuscitative procedures. Information may be lost from short-term memory (memory that is held for less than 30 seconds) through decay and interference, but once committed to long-term memory, it is assumed to be virtually permanent.[2] [58] This is analogous to certain learned skills that are retained indefinitely, such as riding a bicycle. These skills are never forgotten most likely owing to a process called *overlearning*. Retention of skills depends, in part, on the adequacy of the initial educational experience.[64] Research studies of motor learning and control have shown that execution of active movements is best recalled when the movements are practiced actively rather than following a passive demonstration.[39] [57] For most ED procedures, which are rarely overlearned, practice may be required in order to retain those skills.[11] One study demonstrated that airway management skills decline early after initial training but performance may be maintained effectively with independent practice and periodic feedback.[37]

Box 72-1. Eight Steps of Procedural Skill Learning

1. Identify an acceptable skill level.
2. Identify when to perform the procedure.
3. Select the instruments and equipment needed to perform the procedure.
4. Identify the critical steps of the procedure.
5. Memorize the sequential order of the steps.
6. Develop a mental image of performing the procedure.
7. Practice procedural movements with feedback.
8. Assess procedural competence.

From Chapman[11]

PROCEDURAL TRAINING IN THE EMERGENCY DEPARTMENT

Bedside Teaching

Procedural training at the bedside is an important part of learning a new skill in the ED (Fig. 72-1). Ideally the trainee has undergone formal training in the procedure prior to attempting the new skill on a human patient. This may include listening to lectures, watching videotapes, using computerized instructional materials, and reading and reviewing procedural texts and atlases. Having an appropriate clinical setting and adequate time to explain the procedure in detail optimizes bedside teaching. The instructor should review the procedure beforehand with the trainee. This may include such components as the indications, contraindications, relevant anatomy, procedural steps, and complications. The review may take place outside the patient's room or at the bedside. The trainee then performs the procedure while the instructor observes and provides guidance and assistance if needed. Following the procedure, the instructor should examine the patient and assess the adequacy of the procedure. This may then be followed by a discussion of problems that occurred during the procedure, suggestions for improvement, and a review of postprocedural care.

Figure 72-1 Procedural training at the bedside is an important part of learning a new skill. *A,* This ankle dislocation requires prompt relocation in the ED. *B,* The instructor first reviews the procedure with the trainee, including indications, contraindications, procedural steps, and complications. The trainee then performs the reduction while the instructor observes and provides guidance during the procedure. *C,* Following the procedure, the instructor examines the patient and assesses the adequacy of reduction.

PROCEDURAL TRAINING DURING EMERGENCIES

Procedural training in the ED is often complicated by the exigent circumstances under which many procedures are performed. When a patient arrives with a life-threatening condition that mandates emergent intervention, procedures must be accomplished as quickly and competently as possible, and they are frequently done by the most experienced member of the resuscitation team.[29] [61] Consequently, emergency medicine (EM) faculty are seldom afforded the time and circumstances necessary to explain the rationale and details of life-saving surgical procedures to residents in training. [48] [49] The adage "see one, do one, teach one" is particularly unconscionable in the ED, [67] where procedures must be performed rapidly and competently in order to be lifesaving. Hedges [26] wrote that in the case of an unfavorable teaching scenario, it may be better for the educator to teach while he or she does the procedure. He observes that while this may be less satisfying for the trainee, there are still many things to be learned from observing the patient-clinician interaction, procedural setup, critical actions, and postprocedural care.

PROCEDURAL TRAINING FOR UNCOMMON PROCEDURES

Furthermore, the ability for clinicians to practice certain critical resuscitative procedures on human patients in the ED may be limited because they are performed so infrequently. Residents, therefore, have limited opportunity to train and acquire procedural competency and speed in their performance.

There has been a decline in the rate of performance of certain resuscitative procedures in recent years due to a number of factors. Recent critical analyses of patient outcomes following ED thoracotomy have identified key predictors of successful outcome, most notably penetrating trauma to the chest, which have also virtually eliminated performing this procedure in victims of blunt trauma. [5] [16] With clinicians now using a more selective approach for thoracotomy, procedural competency is more difficult to obtain in the ED setting. In addition, Chang et al. [9] reported a significant decline in the rate of cricothyroidotomies performed on trauma patients following the beginning of a new EM residency training program. They attribute this to several possible causes, including the widespread application of rapid sequence intubation techniques, the presence of supervisory EM faculty 24 hours a day, and the diminished concern regarding orotracheal intubation of patients whose cervical spines have not been radiographically cleared. There is also competition for procedures between EM residents and residents from other specialties. [59] In fact, a 1995 survey completed by program directors of EM residencies found that, overall, emergency clinicians perform only 50% of 10 index procedures in the ED while residents from other specialties perform the remainder. [22] Finally, how often residents have the opportunity to perform surgical procedures is often determined by chance.[38]

A recent survey of the experience of EM residents found that a significant number of residents perform fewer than five each of certain critical resuscitative procedures during their training. [24] This confirms the findings of earlier published reports that certain emergency procedures, particularly venous cutdown, thoracotomy, and cricothyroidotomy, are performed infrequently. [20] [54]

ASSESSING PROCEDURAL COMPETENCY

Chapman and others have argued that, with so few procedures being done and without training alternatives, it is unlikely that emergency medicine residents will have achieved procedural competency during their training. [9] [14] The actual number of procedures necessary to ensure competency remains unknown. A study by Chapman failed to show a significant correlation between the clinician's recollection of previous procedural experience and actual thoracotomy performance as assessed by written, computerized, or animal models. Similarly, knowledge of thoracotomy content and procedural steps did not predict procedural competency. [10] Typically, the number of procedures previously performed is taken as predictive of procedural competency; however, these findings suggest that procedural competency cannot be predicted by previous experience. Despite these findings, many hospitals use the number of procedures previously performed as a criterion for hospital credentialing and thereby granting the privilege to perform procedures independently. [12] Some surgical residencies have adopted competency-based education and report success using fresh cadavers as models to achieve rapid improvement in surgical skills. [44]

The acquisition of complicated motor skills is highly individualized and may depend upon the quality of each procedural experience rather than the number of procedures performed.[24] For example, if procedures have been taught or practiced incorrectly, experience is not a useful predictor of procedural competency. It has been demonstrated that when one performs a manual skill incorrectly, even if only once, it is much more difficult to relearn the correct technique. [63] Therefore it is critically important to observe students closely and correct them as they practice a procedure. If procedures are learned incorrectly without the benefit of testing and remediation, these errors may be passed on to other residents and students through the teaching process. [12] In the words of Red Auerbach, the legendary coach of the Boston Celtics, "Practice does not make perfect... . Perfect practice makes perfect." [63]

The learning curve is steep for certain procedures. In a report by Konrad, initial intubation success rates for anesthesia residents in the operating room may be as low as 50% with their first 10 patients, and the curve does not rise to 90% until a mean of 57 attempts. [36]

Regardless of the reasons, when procedures are performed infrequently, competency becomes an issue and complications may be prevalent. Cricothyroidotomy is an example of an uncommon procedure and published reports of complication rates vary widely. [33] [45] [50] [56] However, despite a high incidence of complications, successful cricothyroidotomy is an essential skill to be able to perform, because the alternative is failing to establish an airway, which in many situations results in certain death.[60]

Chapman is an advocate of establishing procedural competency expectations for emergency procedures and resuscitations to ensure the competency of EM graduates. [14] There are currently no published competency criteria for emergency procedures and resuscitations and a minimum number of procedures required by the American Board of Emergency Medicine (ABEM) has not been established. ABEM has likewise not established a mechanism for testing procedural competency, and must rely upon residencies to provide the necessary training, evaluation and assurance of competency. [4] Braen and Munger[4] described early efforts of the ABEM to evaluate procedural skills, but these were not implemented, primarily due to a lack of reliable equipment and resources to examine large pools of candidates in a high-stakes national examination. To standardize testing there must be an assurance that examiners will be able to reliably evaluate candidates. Bullock et al. [7] demonstrated good inter-rater reliability by both expert and non-expert observers when they were given structured checklists to assess procedural skills. Custalow et al.[17] likewise observed excellent inter-rater reliability among expert reviewers for the evaluation of critical step performance of saphenous vein cutdown, thoracotomy, and cricothyroidotomy. Ideally, a national procedural competency examination might incorporate animal, cadaver, and virtual reality simulation laboratories to evaluate candidates. [14]

EDUCATIONAL ALTERNATIVES FOR PRACTICING PROCEDURES

To ensure residents' procedural competency upon completion of residency training and with limited available clinical experience, educators must explore alternative methods for training. Many educational methods have been tried. Unfortunately, many alternatives to clinical experience have substantial limitations and few have been tested for their efficacy and applicability to the performance of procedures

on human patients. The following is a description of the currently available educational techniques. As mentioned earlier, it is ideal to introduce the procedure via formal review before the trainee practices the skill using one of the following procedural models. This may include listening to lectures, watching videotapes, using computerized instructional materials, and reading and reviewing procedural texts and atlases.

Volunteers

Human volunteers (paid or unpaid) are commonly used for teaching procedures. They are useful for practicing noninvasive procedures such as casting and splinting (Fig. 72-2) and certain minimally invasive procedures such as intravenous catheter or nasogastric tube insertion but they are clearly not appropriate for most invasive procedures.

Living Patients in Operating Rooms

Many EM residency programs provide opportunities for residents to practice endotracheal intubations during anesthesia rotations in the controlled setting of the operating room. However, when there is also an anesthesia residency training program at the hospital, residents may compete for these cases.

Mannequins

Mannequins, such as the popular "Resusci-Annie," are available to teach many procedures including cardiopulmonary resuscitation, peripheral venipuncture, arterial puncture, intraosseous infusion, umbilical vessel catheterization, central line catheterization, lumbar puncture, endotracheal intubation (Fig. 72-3), needle thoracostomy, urinary catheter placement, suturing, and emergency childbirth. Stratton found that paramedics trained in endotracheal intubation using a mannequin-only teaching program achieved equal success rates in field intubations compared to those receiving additional training on human cadavers. [62] Mannequins can serve as useful adjuncts

Figure 72-2 Volunteers are ideal models for teaching certain noninvasive procedures such as splinting and casting.

Figure 72-3 Mannequins are available to teach many procedures, including endotracheal intubation.

for teaching procedures but are limited by the lack of certain physical properties of living tissue, including hemorrhage, a beating heart, and breathing lungs. Most mannequins are designed for durability and not for realistic simulation. They are very different from the human patient in which pliable structures (such as the tongue and epiglottis) bend easily and move with the instruments. [41]

Video Imaging

Levitan developed the Airway Cam Direct Laryngoscopy Video System in which a miniature video camera is worn by the trainee, thus allowing a supervisor to assist. The advantage is that instructors can provide real-time feedback to trainees. The trainee's performance can be recorded and reviewed. [40] The complicated anatomy of the larynx can also be demonstrated better with a camera directed on an actual patient than on a mannequin. In a study of initial success rates of paramedic trainees in an operating room setting, Levitan et al. [42] observed a mean improvement in intubation success rates of 41% after paramedics watched a 26-minute instructional videotape made using this video camera system.

In another study medical students were found to perform emergent thoracotomy on the dog and pig models with surprising accuracy after only watching a 30-minute videotape demonstration of this procedure. The presentation of complicated anatomy to novices appears to be an important prerequisite to performing procedures accurately.[11] [12]

Virtual Reality and Medical Simulators

Virtual reality is a promising technology that has enormous educational potential for teaching procedures, but it is still in the early stages of development. [27] In a review of virtual reality and simulation technology, Reznek et al. note that to be an effective teaching tool, a simulator must provide both educationally sound and realistic feedback to a user's questions,

decisions, and actions.[55] Force and tactile feedback have proven to be the most difficult parts of the virtual environment to simulate. While not in widespread use in emergency medical education at this time, modern human patient simulators have been developed or are currently under development for endotracheal intubation, chest compression, electrical cardioversion, cricothyroidotomy, chest tube insertion, pericardiocentesis, diagnostic peritoneal lavage, emergency thoracotomy, lumbar puncture, skin suturing, insertion of peripheral venous/arterial catheters, and insertion of central venous lines (Fig. 72-4).[55]

Cadaver Laboratories

Fresh cadavers (bodies of individuals who have given consent for their bodies to be used for teaching) can be useful to teach certain procedures. The disadvantages are cost, limited availability, and the potential risk for transmission of disease. [48] Preserved cadavers are more readily available and these laboratories offer the

advantage of a standardized reproducible laboratory experience under the guidance of an instructor with no risk to patients. [66] Disadvantages of preserved cadavers are that they do not bleed and they suffer from tissue adherence due to the preservation process, so while excellent for learning the anatomy of procedures, they may not completely simulate the in vivo experience. [13]

Newly Deceased Patients

Newly deceased patients (those who have not expressly given consent for the use of their bodies) have also been used to practice performing procedures. A survey of program directors of EM and critical care training programs found that 63% of EM programs allow procedures to be performed on newly dead patients. [8]

The performance of invasive procedures on the newly dead may be limited by certain debated ethical concerns. [1] [8] [23] [25] [30] [31] [32] [47] [53] Whether consent should be obtained prior to performing procedures on the newly dead is a question that has been discussed extensively in the literature. Advocates of practicing procedures without consent believe that the dead

Figure 72-4 Human patient medical simulator. *A,* In the control room the educator is able to create and monitor clinical scenarios. *B,* The trainee performs electrical cardioversion on a human patient simulator and receives feedback from his actions by watching the monitor (successful conversion of ventricular tachycardia to sinus rhythm).

have no claim to autonomy and that this is a function of personhood. Iserson states that consent for procedures on the newly dead is therefore an inappropriate extension of patient autonomy. Secondly, given the absence of suitable alternative training models, it is in society's best interest to have adequate numbers of medical care providers who are experienced in life-saving procedures. Finally, families are often less altruistic on behalf of their relatives than the individual would be, and requesting permission from relatives may supersede the individual's own wishes and create excessive emotional barriers between the provider and a distraught family.[30] [31] [32]

Those who advocate obtaining consent claim that practicing procedures on the newly dead without consent is unlawful and unethical. Goldblatt [23] wrote that proxy consent by a family member is required when patients cannot give consent themselves and that the next of kin have "quasi-property rights" for the body of a dead family member. He suggested that by framing a request to the family such as, "What would the deceased have wanted?" the protective instincts of the next of kin may be decreased and permission granted.

In a survey involving theoretical clinical scenarios, people responded that they would agree to after-death procedures on themselves in 75% of cases and on their relatives in 70% of cases. However, without prior consent they would allow such procedures only 40% of the time on themselves and 50% on their family members. [43] In actual clinical practice, consent given by family members has traditionally been lower. Families gave consent for intubation of newly deceased infants in 73% of cases,[3] for retrograde wire intubation of newly deceased adults in 59% of cases, [46] and for cricothyroidotomy of newly deceased adults in 39% of cases. [51]

Isolated Animal Tissues

Many schools teach basic wound closure techniques using fresh animal tissue such as pig's feet (Fig. 72-5).[34] Isolated sheep or pig tracheas are useful to practice the technique of cricothyroidotomy but lack the external anatomy of the cricothyroid membrane in situ. Others have experimented with the use of chicken or turkey bones to teach the technique of intraosseous line placement.

Figure 72-5 Isolated pig's feet are useful models to teach basic wound closure techniques.

Live Animal Laboratories

Live animal laboratories provide for a realistic, stimulating teaching environment that imparts a level of understanding vastly different from other educational methods.[18] Although no alternative to performing procedures on a human patient is completely suitable, animal laboratories can provide a reasonable training alternative in which procedures may be performed with close supervision and under controlled conditions (Fig. 72-6).[61] Animal laboratories offer the advantages associated with performing procedures on living, bleeding tissues. Many feel that there is no substitute for this experience and that these learning experiences cannot be duplicated by

Figure 72-6 In the live animal laboratory an instructor assists while the trainee performs a venous cutdown.

simulations.[18] Students experience a feeling of responsibility and receive immediate feedback for their actions. [35] Moreover, animal laboratories may instill confidence in performing procedures on real patients. A survey of clinicians participating in a swine procedure laboratory reported a significant change in their comfort level in performing six resuscitative procedures.[52]

A survey of American medical colleges found that 92 of 126 medical schools (73%) use live animal laboratories at some point in their regular curriculum. [34] In this study, more than half of the schools without animal laboratories cited increasing expense of animals and equipment as the primary reason for not continuing them. Only two schools noted pressure from animal rights activists or student complaints as significant issues in discontinuing animal laboratories. [34]

The time, space, and money required to provide animal laboratory training for residents are considerable, yet the effectiveness of animal laboratory training as compared with other training techniques has not been well investigated. In one study, an animal procedure laboratory emphasizing skill repetition improved tube thoracostomy procedural skills and speed, and these skills were retained on retesting of subjects 18 days later. However, all participants in this study received animal laboratory training and there was no comparison to subjects receiving instruction using conventional training techniques. [29] A 2002 study by Custalow et al.[17] compared residents who had received animal laboratory training to those without animal laboratory training and found significant long-term improvements in procedural competency and speed in the performance of resuscitative procedures in those who received animal laboratory training.

One disadvantage of using animals for medical training, however, is the considerable cost involved. Furthermore, many question whether an animal model provides the best available simulation of the performance of procedures in humans. Finally, debate continues on the appropriate balance between animal rights and the benefits of using them for medical training and research. [15] [21] [29] [65]

CONCLUSION

There are numerous educational alternatives for teaching ED procedures, each with advantages and disadvantages. Optimally, residencies will have the space and resources to use a combination of training techniques to ensure competent performance of procedures by EM graduates. Educators may then find new ways to teach time-proven techniques, refine these techniques, or even develop new ones such as the Brofeldt four-step cricothyrotomy. [6] [28] Many questions remain unanswered regarding the best educational methods, but EM has the foundation for building a comprehensive instructional system to ensure the competency of trainees and maintenance of procedural skills throughout life. With repetition and reinforcement, these skills may then be committed to long-term memory for retrieval when they are most needed in the ED.

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Emergency clinicians are frequently required to perform conversions and calculations in the emergency department (ED). Several formulas and tables are presented to guide those computations.

TEMPERATURE-CONVERSIONS FROM CELSIUS TO FAHRENHEIT

Fahrenheit (F) and Celsius (C) are the units most commonly used. Table A-1 is a list of approximate F/C equivalents. The formulas for conversion between units are as follows:

°F = (°C × 1.8) + 32

°C = (°F - 32) / 1.8

Example: A mother reports her child's temperature is 104°F. To convert to °C:

°C = (104°F - 32) / 1.8 = 40°C

Example: A child's temperature in the ED is 39.6°C. His mother wants to know what the temperature is in °F. To convert to °F:

°F = (39.6°C × 1.8) + 32 = 103.3°F

TABLE 1 -- Approximate Fahrenheit and Celsius Equivalents

°C	***°F***
30	86
32	89.6
34	93.2
35	95
35.5	95.9
36	96.8
36.5	97.7
37	98.6
37.5	99.5
38	100.4
38.5	101.3
39	102.2
39.5	103.1
40	104
40.5	104.9
41	105.8

WEIGHT-CONVERSION FROM POUNDS TO KILOGRAMS

Patients frequently express body weight in pounds, but many medical calculations require conversion to kilograms. The formulas for conversion between these two units are

1 kg = 2.2 lb

kg = lb/2.2 lb

Example: A dehydrated child requires intravenous fluid. The clinician plans to infuse a fluid bolus of 20 mL per kilogram. The patient's mother states that the child weighs 35 lb.

kg = 35 lb/2.2 = 16 kg

The fluid bolus is 16 kg × 20 mL = 320 mL

ESTIMATION OF A CHILD'S WEIGHT

Weight in kg = (2 × age) + 8

Example: A 3-year-old child needs rehydration. The estimated weight in kg is (2 × 3) + 8 = 14 kg.

CALCULATION OF THE MEAN ARTERIAL PRESSURE

Calculation of the mean arterial pressure (MAP) provides a weighted average of the systolic blood pressure (SBP) and the diastolic blood pressure (DBP). It is a determination of tissue perfusion pressure and is normally 70 to 100 mm Hg in adults. To determine the MAP:

MAP = [SBP + (2 × DBP)]/3

Example: An elderly, hypertensive patient is diagnosed with an acute hemorrhagic stroke. The neurologist recommends lowering the MAP to <130 mm Hg. The patient's current blood pressure is 210/120. To calculate the current MAP:

MAP = [210 + (2 × 120)]/3 = 150 mm Hg

QT AND QTC INTERVALS

The QT interval on the electrocardiogram (ECG) represents the period of ventricular electrical activity from activation to repolarization. The most important determinant of the QT interval is the heart rate. As the heart rate increases, the QT interval shortens. To calculate the rate-corrected QT interval (QTc), divide the QT interval by the square root of the R-R interval (the interval between the R on two consecutive QRS complexes). The interval is represented in sec or msec:

QTc = QT/vR-R

QTc = the normal corrected QT interval, which is normally =0.40 sec for men and =0.44 sec for women.

R-R = the R-R interval is calculated as 60 divided by the heart rate (in beats per minute).

Table A-2 shows the normal range of the QT interval for adults.

Example: A 21-year-old male ingested a large quantity of amitriptyline (tricyclic antidepressant) tablets. His ECG reveals a QT interval of 0.37 seconds and a heart rate of 120 beats/min. The QTc is calculated as follows:

QTc = QT interval/vR-R

R-R interval = 60/120 = 0.50

QTc = 0.37/v0.50 = 0.37/0.71 = 0.53 sec = 520 msec

The patient's QTc is significantly prolonged for his heart rate and indicates significant cardiac effects from a tricyclic antidepressant overdose.
Causes of prolonged QT interval are shown in Table A-3 .

TABLE 2 -- Normal Range of QT Interval for Adults

Heart Rate (bpm)	***Normal QT Range (sec)***
40	0.42–0.53
50	0.37–0.48
60	0.34–0.44
70	0.31–0.41
80	0.29–0.38
90	0.28–0.36
100	0.27–0.34
110	0.25–0.32
120	0.24–0.31
130	0.23–0.30
140	0.22–0.29
150	0.21–0.28

TABLE 3 -- Conditions/Medications That May Cause QT Prolongation and/or Induce Torsades de Pointes

Conditions That May Cause QT Prolongation	
Metabolic abnormalities	• Hypokalemia
	• Hypocalcemia
	• Hypomagnesemia
Bradyarrhythmias	• Complete atrioventricular block
	• Any bradyarrhythmia, even transient
Starvation	• Anorexia nervosa
	• "Liquid protein" diets
	• Gastroplasty and ileojejunal bypass
	• Celiac disease
Nervous system injury	• Subarachnoid hemorrhage
	• Thalamic hematoma
	• Right neck dissection or hematoma
	• Pheochromocytoma
Medications That Can Prolong the QT Interval and/or Induce Torsades de Pointes	

Antipsychotics	• Chlorpromazine*
	• Haloperidol—QT, TdP
	• Mesoridazine—QT, TdP
	• Pimozide—QT, cases
	• Quetiapine—QT
	• Risperidone—QT
	• Thioridazine—QT, TdP
	• Ziprasidone—QT
Antiarrhythmics	• Amiodarone—QT, TdP
	• Disopyramide—QT, TdP
	• Dofetilide—QT, TdP
	• Flecainide† —QT, TdP
	• Ibutilide—QT, TdP
	• Procainamide—QT, TdP
	• Quinidine—QT, TdP
	• Sotalol—QT, TdP
Antidepressants	• Amitripyline‡ -cases
	• Desipramine‡ -cases
	• Doxepin‡ -cases
	• Fluoxetine—QT, TdP
	• Imipramine‡ —cases
	• Paroxetine—TdP
	• Sertraline† —QT, TdP
	• Venlafaxine—QT
Anticancer	• Arsenic Trioxide—QT, TdP, cases
	• Tamoxifen—QT
Misc. cardiac	• Bepridil—QT, TdP
	• Isradipine—QT
	• Moexipril/hetz—QT
	• Nicardipine—QT
GI stimulant	• Cisapride—QT, TdP
Antibiotics	• Clarithromycin-cases
	• Erythromycin—QT, TdP
	• Gatifloxacin—QT
	• Levofloxacin—TdP
	• Moxifloxacin—QT
	• Sparfloxacin—QT, TdP
Antimigraine	• Naratriptan—QT
	• Sumatriptan—QT
	• Zolmitriptan—QT
Miscellaneous	• Dolasetron—QT
	Droperidol—QT, TdP, cases
	• Fascarnet—QT
	• Felbamate—TdP
	• Fosphenytoin—QT
	• Halofantrine—QT, TdP
	• Indapamide—QT, cases
	• Levomethadyl—QT
	• Octreotide—QT
	• Pentamidine—QT, TdP
	• Salmeterol—QT
	• Tacrolimus-cases
	• Tizanidine—QT

QT: Prolongation is mentioned in the FDA-approved labeling as a known action of the drug.

TdP: The FDA-approved labeling includes mention of cases or a risk of Torsades de Pointes.

Cases: There are case reports of TdP in the medical literature.

Reference: Drugs that prolong the Q-T interval and/or induce Torsades de Pointes. (Resource on World Wide Web) http://www.torsades.org accessed September 12, 2000.

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*Nonspecific QT changes
†Association not clear
‡Tricyclic antidepressants used alone or in combination with drugs that prolong the QT interval may predispose patients to cardiac arrythmias.

PREDICTED PEAK EXPIRATORY FLOW RATE

The peak expiratory flow rate (PEFR), measured in liters/minute (L/min) is a useful means of assessing airway obstruction. It is measured by having a patient exhale maximally through a peak flow meter. Normal values range from 350 to 600 L/min. A patient's prognosis in asthma exacerbations can be followed by comparing the initial and post-treatment PEFR. Patients with an initial PEFR of <20% predicted or with a subsequent value of <60% predicted after initial therapy may require further evaluation or treatment, or both. Many patients monitor PEFR on themselves and are able to state a personal best, which is the preferred standard for that individual. Other patients may be monitored using an estimated PEFR. PEFR is primarily based on a patient's gender, age, and height. While graphs and tables are available to provide values across a range of ages and heights, PEFR can also be approximated using the following formulas for adults:

PEFR in males (L/min) = {[(Height in inches × 0.139) + 1.58] - (Age × 0.041)} × 60

PEFR in females (L/min) = {[(Height in inches × 0.094) + 2.24] - (Age × 0.03)} × 60

Example (male): A 50-year-old male is 72 inches tall.

PEFR in males (L/min) = {[(72 in × 0.139) + 1.58] - (50 × 0.041)} × 60 = (11.59 - 2.05) × 60 = 572 L/min

Example (female): A 45-year-old female is 62 inches tall.

PEFR in females (L/min) = {[(62 in × 0.094) + 2.24] - (45 × 0.03)} × 60 = (8.07 - 1.35) × 60 = 403 L/min

ENDOTRACHEAL INTUBATION AND MECHANICAL VENTILATION

Patients with respiratory failure may require endotracheal intubation and mechanical ventilation in the ED. The following are guidelines for choosing the endotracheal (ET) tube size and for calculating the initial ventilator settings.

Selecting the Endotracheal Tube Size (Also See Chapter 4)

Adults: Select the largest diameter ET tube that can be tolerated for adults.
Size 7.5 mm cuffed ET tube is well tolerated by most adult female patients.
Size 8.0 mm cuffed ET tube is well tolerated by most adult male patients.
An uncuffed ET tube should be used for children under the age of 8. There are a number of techniques for estimating the appropriate ET tube size in children. The formula most commonly used is:

ET size (mm) = (Age in years/4) + 4

Example: A 5-year-old male needs emergent intubation. To select the appropriate size tube:

ET size (mm) = (5/4) + 4 ˜ 5.0 endotracheal tube (uncuffed)

To estimate the depth of insertion for a child older than 2 years:

Depth of insertion = 3 × internal diameter of the endotracheal tube

Determining Initial Ventilator Settings (See Chapter 8)

The recommended initial ventilator settings follow. Adjustments in these ventilator settings may be made according to the patient's clinical situation:
Tidal volume (TV) = 6–12 mL/kg

Rate = 10–12 breaths per minute for adults, 16–20 breaths per minute for children and 20–30 breaths per minute for infants.

FiO_2 = 50%–100% initially. Reduce the FiO_2 as quickly as possible to avoid oxygen toxicity to the lungs.

I:E ratio = 1:2. To allow complete exhalation, the inspiratory to expiratory ratio should be at least 1:2.

Example: A 6-year-old female with asthma has respiratory distress and altered mental status, requiring ET intubation. Her weight is 20 kg. To prepare for intubation and mechanical ventilation:

ET tube size = 6/4 + 4 = 5.5 mm tube, uncuffed

Tidal volume = 12 mL/kg × 20 kg = 240 mL
Respiratory rate = 16 breaths per minute
FiO_2 = 100%
I:E ratio = 1:2

RENAL FUNCTION

Creatinine Clearance

Creatinine clearance (Cl_{Cr}) is best calculated using a collection of urine over a 24-hour period. However, if the patient's creatinine clearance is in steady state (i.e., without recent change), it is possible to estimate Cl_{Cr} by using a formula that incorporates serum creatinine, weight, age, and gender:

Cl_{Cr} (men) = [(140 - age in years) × (lean body weight, kg)]/[72 × (serum creatinine in mg/dL)]

Cl_{Cr} (women) = [(140 - age in years) × (lean body weight, kg)]/[72 × (serum creatinine in mg/dL)] × 0.85

Normal values: 74–160 mL/min

Mild renal impairment: 40–60 mL/min

Moderate renal impairment: 10–40 mL/min

Severe renal impairment: <15 mL/min (= indication for renal dialysis)

Example: A 64-year-old woman has upper abdominal pain tenderness. A CT scan is planned, but the radiologist is concerned about the risk of intravenous contrast. The patient's serum creatinine is 1.8 mg/dL and she weighs 75 kg. To calculate her Cl_{Cr}:

Cl_{Cr} (women) = [(140 - 64) × (75 kg)/72 × (1.8 mg/dL)] × 0.85 = 37.4 mL/min

The patient has moderate renal impairment.

Fractional Excretion of Sodium

The fractional excretion of sodium (Fe_{Na}) calculation predicts the likelihood that a patient with acute renal failure has acute tubular necrosis (ATN). In ATN, the kidneys have difficulty reabsorbing sodium and concentrating urine. The following formula takes advantage of this by using plasma sodium and creatinine concentrations (P[Na] and P[Cr]) and spot urine sodium and creatinine levels (U[Na] and U[Cr]):

$Fe_{Na} = (U[Na] \times P[Cr])/(P[Na] \times U[Cr]) \times 100$

Fe_{Na} > 2% suggests post-glomerular failure

Fe_{Na} < 1% suggests prerenal azotemia (volume depletion).

Example: A middle-aged male was found lying on the ground and has ulcers on all pressure points on his back, buttocks, and legs. A urinary catheter is placed and yields a small amount of dark urine, which tests positive for blood on dipstick. He is found to have acute renal failure. Plasma and urine electrolytes and creatinine analyses are performed. The results are P[Na] = 134 mmol/L, P[Cr] = 3.5 mg/dL, U[Na] = 108 mmol/L, U[Cr] = 68 mg/dL. What is the most likely cause of his renal failure, ATN/myoglobinuria, or dehydration?

$Fe_{Na} = [(108 \times 3.5)/(134 \times 68)] \times 100 = 4.1$

The patient's Fe_{Na} is 4.1 (which is >2%) and suggests that ATN is the most likely the cause of his renal failure and myoglobinuria.

Calculation of the Anion Gap

The anion gap (AG) is an estimate of the amount of negatively charged (unmeasured) ions in the serum that are not bicarbonate (HCO_3^-) and chloride (Cl^-). The AG is calculated by subtracting the sum of HCO_3^- and Cl^- values from the sodium (Na^+), which is the major positive charge in the serum. Potassium (K^+) is not used in the calculation because most of the body's potassium is intracellular and there is a relatively small amount of K^+ in the serum. An elevated AG usually means that there is some unmeasured anion, toxin, or organic acid in the blood. The AG is normally 8 to 12 mmol/L:

$AG = Na^+ - (Cl^- + HCO_3^-) = 8$ to 12 mmol/L

An increase in the AG is usually associated with acidosis, referred to as an anion gap acidosis.
Table A-4 lists many substances that can cause an anion gap acidosis.

Example: A suicidal young male drank an unknown amount of antifreeze. His electrolyte levels are Na^+ 144, K^+ 3.1, Cl^- 108, and HCO_3^- 14. The anion gap is calculated as follows:

$AG = [144 - (108 + 14)] = 22$ mmol/L

The anion gap is abnormally elevated, presumably due to ethylene glycol ingestion.

TABLE 4 -- Substances Associated with High Anion Gap*

Aspirin
Methanol, metformin
Uremia
Diabetic ketoacidosis
Paraldehyde, phenformin
Isoniazid, iron
Lactate (multiple causes)
Ethylene glycol
Carbon monoxide, cyanide
Alcoholic ketoacidosis
Toluene

*Follows the mnemonic A MUDPILE CAT.

Calculating the Osmolal Gap

Serum osmolality can be measured in the laboratory by freezing point depression. The measured serum osmolality is normally higher than the calculated osmolality and the difference is termed the osmolal gap (OG). The osmolal gap is normally 5 to 10 mOsm/kg. If there is a higher gap, the osmols unaccounted for may represent methanol, ethylene glycol, isopropyl alcohol, or other solutes (Table A-5). To calculate serum osmolaity (Osm_{calc}) and the osmolal gap:

$Osm_{calc} = 2 \times (Na^+) + [BUN\ (mg/dL)/2.8] + [glucose\ (mg/dL)/18] =$ normally 280—295

$OG = Osm_{meas} - Osm_{calc}$

TABLE 5 -- The Effect of Some Solutes on Serum Osmolality

Each mg/dL of:	*Increases Serum mOsm/kg by:*	*For Each Serum mOsm/kg Increase Due to:*	*The Corresponding mg/dL Change is (= mol wt/10):* t
Methanol	0.31	Methanol	3.2
Ethanol	0.22	Ethanol	4.6
Acetone	0.17	Acetone	5.8
Isopropyl alcohol	0.17	Isopropyl alcohol	6.0
Ethylene glycol	0.16	Ethylene glycol	6.2
Glycerol	0.11	Glycerol	9.2
Mannitol	0.05	Mannitol	18.2

Adapted from Kullig K, Duffy JP, Linden CH, et al: Toxic effects of methanol, ethylene glycol and isopropyl alcohol. Top Emerg Med 6(2):16, 1984.

Table A-5 shows the effect of some solutes on serum osmolality. In general, the increase in osmolality caused by a solute can be calculated by dividing its serum concentration by the tabulated value.

Example: An intoxicated patient has serum chemistry results as follows: Na^+ 142, K^+ 4.5, Cl^- 100, HCO_3^- 22, Glucose 90, BUN 14. His ethanol level is 240 and his measured serum osmolality is 348. To calculate his serum osmolaity:

$$\text{Osm}_{\text{calc}} = 2\,(142) + (14/2.8) + (90/18)$$
$$= 284 + 5 + 5$$
$$= 294\ \text{mOsm/kg}$$

To evaluate for the effect of ethanol, refer to Table A-5 , adding the alcohol level divided by 4.6:

$$= 240/4.6 + 294\ \text{mOsm/kg}$$
$$= 52 + 294\ \text{mOsm/kg}$$
$$= 346\ \text{mOsm/kg}$$

Finally, calculate the osmolal gap:

$OG = Osm_{meas} - Osm_{calc} = 348 - 346 = 2$

Factitious Hyponatremia

Factitious hyponatremia may be due to hyperglycemia. In this hyperosmolal state, glucose tends to stay in the extracellular fluid, drawing water out of the cells and into the extracellular fluid. Serum sodium is decreased by about 1.6 mmol/L for each 100 mg/dL of excess glucose. To calculate the corrected sodium:

Corrected Na^+ (mmol/L) = Measured Na^+ (mmol/L) + [1.6 × [(measured glucose in mg/dL - 100)/100]

Many labs automatically make this adjustment so it is important to check with your lab to determine the necessity of this correction.

Example: An obtunded elderly man appears dehydrated. His sodium level is 126 mmol/L and his glucose is 1000 mg/dL.

$$\text{Corrected } Na^+ = 126\ \text{mmol/L} + [1.6 \times (1000 - 100)/100]$$
$$= 126 + 14.4 = 140.4\ \text{mmol/L}$$

His corrected Na^+ suggests that he has factitious hyponatremia due to hyperglycemia.

Free Water Deficit in Hypernatremia

Elevation of serum sodium concentration is proportionate to free water deficit when volume is depleted. Since 60% of the adult body is water, total body free water deficit is calculated using measured Na^+ , desired Na^+ , and body weight in kg. To calculate the free water deficit:

Ideal total body water (TBW) = 60% × weight in kg

Current TBW = (desired serum Na^+ × ideal TBW)/measured Na^+

Free water deficit = ideal TBW - current TBW

Example: An elderly man is brought to the ED in a coma. He has signs of severe dehydration. His ideal body weight is 70 kg. His serum Na^+ is 165 mmol/L. To determine his free water deficit:

Ideal TBW = 0.6 (70 kg) = 42 L

Current TBW = 140 mmol/L × 42 L/165 mmol/L = 35.6 L

Free water deficit = 42 L - 35.6 L = 6.4 L

Fluid correction for hypernatremia should take place over 48 to 72 hours to avoid the potential for cerebral edema.

Calculation of the Sodium Deficit In Hyponatremia

The following formula may be used to calculate the Na^+ deficit in hyponatremia:

Na^+ deficit = 60% × (weight in kg) × (desired Na^+ - measured Na^+)

Symptoms related to hyponatremia are variable and the severity of symptoms should guide therapy. Sodium replacement is most commonly given as isotonic saline, which contains 154 mmol of Na^+ per liter. Patients who are severely symptomatic may require 3% saline solution that contains 513 mmol of Na^+ per liter. The volume of solution needed to replace the Na^+ deficit (in mmol) can be calculated using the concentrations in the saline solutions listed earlier.

Example: A young man is seizing on arrival to the ED. He is known to have schizophrenia and compulsive water drinking. His Na^+ is 116 mmol/L. He weights 65 kg. To determine his sodium deficit:

$$Na^+\ \text{deficit} = 0.6 \times (65\ \text{kg}) \times (140 - 116)$$
$$= 936\ \text{mmol of } Na^+$$

This amount of Na^+ deficit can be administered as approximately 6 liters of isotonic saline or 1.8 liters of hypertonic saline. Na^+ should be replaced very slowly to

avoid the possibility of inducing central pontine myelinolysis (CPM), which results from overaggressive correction of sodium.

pH and Changes in Serum Potassium Levels

Serum potassium (K^+) levels change with the acid-base status. In acidemic states, K^+ moves out of cells as H^+ moves in, thus raising serum K^+ levels. In alkalemic states, K^+ moves into cells as H^+ moves out, thus lowering serum K^+ levels. The change in K^+ varies inversely with pH at the following rate:

Serum K^+ concentration increases 0.6 mmol/L for each 0.1 unit decrease in pH.

Serum K^+ concentration decreases 0.6 mmol/L for each 0.1 unit increase in pH.

Calculating the Corrected Calcium Level

Approximately 50% of serum calcium is bound to serum proteins (primarily albumin), 40% is in the free ionized state (the physiologically active form), and 10% is mixed with serum anions (phosphate, bicarbonate, citrate, and lactate). For this reason, serum calcium is lowered about 0.8 mg/dL for every decrease in albumin of 1 g/dL. To correct for decreased albumin (at levels <4 g/dL), the following formula can be used:

Corrected Ca^{2+} (mg/dL) = serum Ca^{2+} (mg/dL) + (0.8 × [4.0 - serum albumin {g/dL}])

Example: A malnourished man has a serum calcium level of 7.5 mg/dL and a serum albumin level of 2 g/dL. To calculate his corrected calcium level:

Corrected Ca^{2+} (mg/dL) = 7.5 mg/dL + [0.8 × (4.0 - 2.0 g/dL)] = 7.5 + 1.6 = 9.1 mg/dL

Calculating Pediatric Intravenous Maintenance Fluids

To calculate maintenance intravenous fluids for a pediatric patient, use the following formula:

4 mL/kg/hr for the first 10 kg, plus
2 mL/kg/hr for the second 10 kg, plus
1 mL/kg/hr for each further kg

Example: A 5-year-old boy weighs 19 kg and requires maintenance intravenous fluids. To calculate his intravenous fluid rate:

4 mL/kg/hr for the first 10 kg = 4 mL × 10 kg/hr = 40 kg/hr, plus
2 mL/kg/hr for the second 10 kg = 2 mL × 9 kg/hr = 18 kg/hr
40 kg/hr + 18 kg/hr = 58 kg/hr

Fluid Replacement for Burn Victims

Various formulas for intravenous fluid resuscitation in burns have been recommended. The Parkland formula is commonly used, and is calculated as follows:

Replacement fluid = (4 mL) × (weight in kg) × (% body surface area burned)

The total volume should be given in the first 24 hours with half the fluid given in the first 8 hours and the remaining half given in the next 16 hours. Clinical parameters including urine output, vital signs, and central venous pressure or pulmonary capillary wedge pressure should be monitored carefully to assess the adequacy of resuscitation.

Example: A 65-kg woman has second- and third-degree burns covering 35% of her body. To determine her anticipated 24-hour fluid resuscitation needs:

Replacement fluid (in liters) = (4 mL) × (65) × (35) = 9100 mL

ARTERIAL BLOOD GAS ANALYSIS

The Alveolar-Arterial Oxygen Gradient

The alveolar-arterial oxygen gradient (A-a gradient) is the difference between the partial pressure of oxygen in the alveolar air ($P_A O_2$) and the arterial blood ($P_a O_2$). It is used primarily to differentiate between hypoxia due to hypoventilation (in which the A-a gradient is normal) and hypoxia due to ventilation-perfusion mismatch (in which the A-a gradient is abnormal). In conditions where there is abnormal oxygen exchange between the alveoli and the arterial blood, the A-a gradient will be increased.

Calculating the Partial Pressure of Oxygen in the Alveolar Air ($P_a O_2$)

The partial pressure of oxygen in the alveolar air ($P_A O_2$) cannot be directly sampled and is therefore calculated from the alveolar air equation. Because it is difficult to determine the actual amount of oxygen delivered to the alveoli when the patient is breathing supplemental oxygen, the equation is most accurate when the patient is breathing room air.

$P_A O_2 = [FiO_2 \times (\text{barometric pressure} - PH_2O)] - PCO_2/RQ$

*Administer 50% of volume over first 8 hours and remainder over subsequent 14 hours post burn.

$P_A O_2$ = partial pressure of oxygen in the alveolar air

FiO_2 = 0.21 = fraction of the inspired air that is oxygen (21% for room air)

Barometric pressure (at sea-level) = 760 mm Hg

PH_2O = 47 mm Hg (partial pressure of water vapor at 37° and sea level)

PCO_2 = as measured by arterial blood gas (ABG) analysis

RQ = 0.8 (respiratory quotient, the ratio of CO_2 produced per unit of oxygen consumed)

To simplify the equation assuming sea-level barometric pressure and room air:

$P_A O_2 = [0.21 \times (760 - 47)] - (PCO_2/0.8)$
$P_A O_2 = 150 - (PCO_2/0.8)$

Calculating the A-a Gradient

To determine the A-a gradient, the partial pressure of oxygen in the arterial blood ($P_a O_2$) (as determined by the ABG) is subtracted from the calculated partial pressure of oxygen in the alveolar air ($P_A O_2$):

A-a gradient = $P_A O_2 - P_a O_2 = [150 - (PCO_2/0.8)] - P_a O_2$

Calculating the Age Correction for the A-a Gradient

The normal A-a gradient is less than 10 mm Hg in a young person; however, since alveolar oxygen diffusion decreases with age, an increase in the A-a gradient is expected with age. To estimate this change, use the following formula:

Age-corrected A-a gradient = [(age/4) + 4]

Example: A 60-year-old man is found confused at home. He is tachypneic and diaphoretic. Oxygen saturation is 92% by pulse oximetry. A room air ABG showed: pH = 7.48, PCO_2 = 27, PO_2 = 72. To calculate the A-a gradient:

A-a gradient = (150 - 27/0.8) - 72 = 116 - 72 = 44 mm Hg

Age adjusted A-a gradient: (age/4) + 4 = (60/4) + 4 = 19

The A-a gradient is abnormally elevated beyond the adjustment for age, suggesting a ventilation-perfusion (V/Q) mismatch.

ACID-BASE BALANCE

Using the combination of an ABG sample and serum electrolyte levels, a patient's acid-base status can be evaluated. Faced with pH, PCO_2, and HCO^-_3 data, the clinician may determine the primary acid-base disturbance and whether the compensation is appropriate. The following basic steps are required.

Knowledge of normal values:

Normal serum pH = 7.40

Normal PCO_2 = 40 mm Hg

First, determine the pH status. Acid-base changes are either metabolic or respiratory. Simple disturbances are categorized by examining the pH and the PCO_2 and HCO^-_3 (Table A-6). If the pH is less than normal (below 7.35), the patient is acidemic. If the pH is above normal (above 7.45), the patient is alkalemic. If the patient is acidemic and the PCO_2 is elevated (>45 mm Hg) as a primary disorder, then respiratory acidosis is present. If the patient is alkalemic and the PCO_2 is decreased (<35 mm Hg) as a primary disorder, then respiratory alkalosis is present. If the patient is acidemic and the arterial HCO^-_3 level is less than normal (<22 mm Hg) as a primary disorder, then a metabolic acidosis is present. If the patient is alkalemic and the HCO^-_3 level is greater than normal (>26 mm Hg) as a primary disorder, then a metabolic alkalosis is present.

Acid-base homeostasis is normally maintained because the disruption of one element provokes a proportional change, or compensation, in the other. Alteration of pH is limited and not entirely corrected by normal mechanisms. The degree and timing of compensation is determined by the primary disturbance itself and by individual physiology. Respiratory compensation for metabolic disorders is rapid and occurs by adjusting the PCO_2. Full metabolic compensation for a respiratory disturbance requires renal adjustment of HCO^-_3 and takes 3 to 5 days. The predicted compensatory responses for the primary disturbances are shown in Table A-6 . It is important to remember that

physiologic compensatory mechanisms may themselves be compromised or overwhelmed by the acid-base disorder (Fig. A-1).

TABLE 6 -- Acidosis and Compensatory Response

Primary Disturbance	***Predicted Compensatory Response***
Metabolic acidosis	? in PCO_2 = 1.3 × ? in HCO^-_3
Metabolic acidosis	? in PCO_2 = 0.6 × ? in HCO^-_3
Respiratory acidosis	*Acute:* For every PCO_2 ? of 10 mm Hg, ? by 1 mmol/L
	Chronic: For every PCO_2 ? of 10 mm Hg, HCO^-_3 ? by 4 mmol/L
	Acute: For every PCO_2 ? of 10 mm Hg, HCO^-_3 ? by 2 mmol/L
	Chronic: For every PCO_2 ? of 10 mm Hg, HCO^-_3 ? by 5 mmol/L

Adapted from Rutecki GW, Whittier FC: Acid-Base interpretation. Consultant, Nov 1991, pp 44–59.

Example 1: A 58-year-old woman has had profuse diarrhea for 1 week. Initial laboratory data include the following:

Sodium, 133 mmol/L

Potassium, 2.8 mmol/L

pH, 7.26

Chloride, 118 mmol/L

PCO_2 , 13 mm Hg

HCO^-_3 , 5 mmol/L

1. Acidemia is present (pH < 7.40)
2. The primary process is metabolic (HCO^-_3 < 22 mmoL, and PCO_2 is not increased)
3. Compensation: In primary metabolic acidemia, the formula that checks for compensation is

? PCO_2 = 1.3 × ? HCO^-_3
? PCO_2 = 1.3 × (25 - 5) = 26 mm Hg
The predicted PCO_2 is 40 - 26 = 14
The actual PCO_2 is 13 mm Hg
Compensation is normal

Example 2: A 74-year-old nursing home resident is admitted to the hospital with hypotension (96/70) and fever (39°C). He has had a positive urine culture for *Escherichia coli* and two positive blood cultures with the same organism. The laboratory values are as follows:

Sodium, 138 mmol/L

Potassium, 3.2 mmol/L

Chloride, 105 mmol/L

pH, 7.49

PCO_2 , 25 mm Hg

HCO^-_3 , 22 mmol/L

1. Alkalemia is present (pH > 7.44)
2. The primary process is respiratory (PCO_2 <40 mm Hg and the HCO^-_3 was not increased).
3. Compensation: The decrease in the PCO_2 is 40 - 25 or 15 mm Hg. The formula for an expected decrease in HCO^-_3 is 2 mmol for every 10 torr decrease in the PCO_2 . In this instance the expected decrease in HCO^-_3 , for the 15 mm Hg decrease in PCO_2 is 3 mmol/L, which is nearly identical to the actual decrease (i.e., 25 – 22). Therefore, only acute respiratory alkalemia is present with normal compensation.

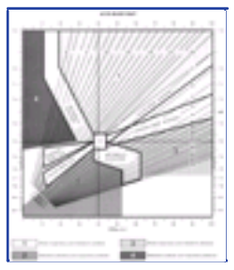


Figure 1 Acid-base map.

THE TRAUMATIC LUMBAR PUNCTURE

A traumatic lumbar puncture is suggested by progressive clearing of the cerebrospinal fluid (CSF) and a decreasing red blood count (RBC) from tubes one to three. The ratio of RBC to white blood count (WBC) should reflect the ratio in the peripheral blood. Subarachnoid hemorrhage is considered if cerebrospinal fluid (CSF) is markedly bloody and unchanged after several milliliters of fluid are removed from the puncture site. The centrifuged sample in subarachnoid hemorrhage is frequently xanthochromic. The following formula may be used to estimate the true CSF WBC:

True CSF WBC = Measured CSF WBC × [(CSF RBC × peripheral blood WBC)/peripheral blood RBC]

The approximate normal ranges for whole blood in adults (male and female):

RBC = 3.5-6 × 10^6 /µL approximate normal range for peripheral blood RBC

WBC = 5-11 × 10^3 /µL approximate normal range for peripheral blood WBC

When peripheral blood cell counts are normal, the CSF from a traumatic lumbar puncture should contain around 1 WBC per 700 RBCs.

DIAGNOSTIC PROBABILITY

The probability of obtaining a certain test result, in the presence or absence of a particular disease entity, for a given population with a given disease prevalence is represented by Table A-7 .

No medical test is totally accurate. The parameters listed in Table A-7 , when available, can help guide a clinician's test selection. When this information is not available, it may be difficult to identify random laboratory errors or detection failures. Knowledge of disease prevalence, combined with the sensitivity and specificity of the test, yields the positive (or negative) predictive value of that test. For a given sensitivity and specificity, predictive value is directly proportional to prevalence. Hence, even a test with high sensitivity and specificity may not detect a rare disease. This underscores the importance of pre-test clinical evaluation.

TABLE 7 -- Definitions of Commonly Used Epidemiologic Terms

Prevalence	= (a + c)/(a + b + c + d)	= incidence of disease in population tested
Sensitivity	= a/(a + c)	= probability of positive test result, disease present
Specificity	= d/(b + d)	= probability of negative test result, disease absent
False-negative rate	= c/(a + c)	= probability of negative test result, disease present
False-positive rate	= b/(b + d)	= probability of positive test result, disease absent
Positive predictive value	= a/(a + b)	= probability of disease presence, test positive
Negative predictive value	= d/(c + d)	= probability of disease absence, test negative
Overall accuracy	= (a + d)/(a + b + c + d)	= probability of a "true" test result

	Disease State		
Test Result	**Present**	**Absent**	
Positive	a (true positive)	b (false positive)	a + b = all positive tests
Negative	c (false negative)	d (true negative)	c + d = all negative tests
	a + c = all patients with disease	b + d = all patients without disease	a + b + c + d = all patients tested

Modified from Goldman L: Quantitative aspects of clinical reasoning. In Isselbacher KJ, Braunwald E, Wilson JD (eds): Harrison's Principles of Internal Medicine, 13th ed. New York, McGraw-Hill, 1994, p 44.

Acknowledgment

The editors and authors wish to acknowledge the contributions of M. John Mendelsohn to this chapter in previous editions.

Figure 1-1 Examples of abnormalities of the arterial pulse compared with the normal pulse. The normal pulse pressure is approximately 30 to 40 mm Hg. The pulse contour is smooth and rounded. (The notch on the descending slope of the pulse wave is not palpable.) *(From Bates B: A Guide to Physical Examination and History Taking, 4th ed. Philadelphia, JB Lippincott, 1987.)*

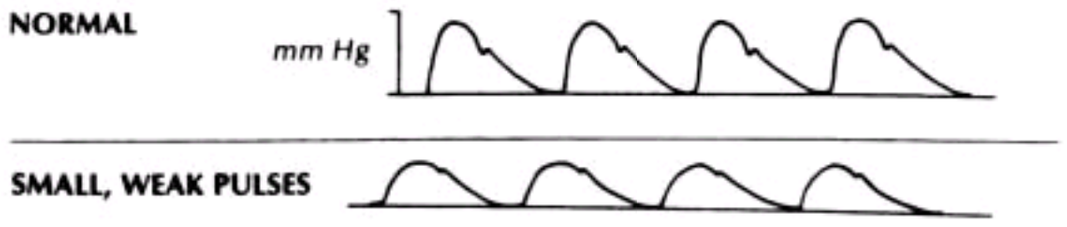


Figure 1-2 Korotkoff sounds. Systole—first audible sound. Diastole—sound disappears. *(From Burnside JW, McGlynn TJ: Physical Diagnosis, 17th ed. Baltimore, Williams & Wilkins, 1986.)*

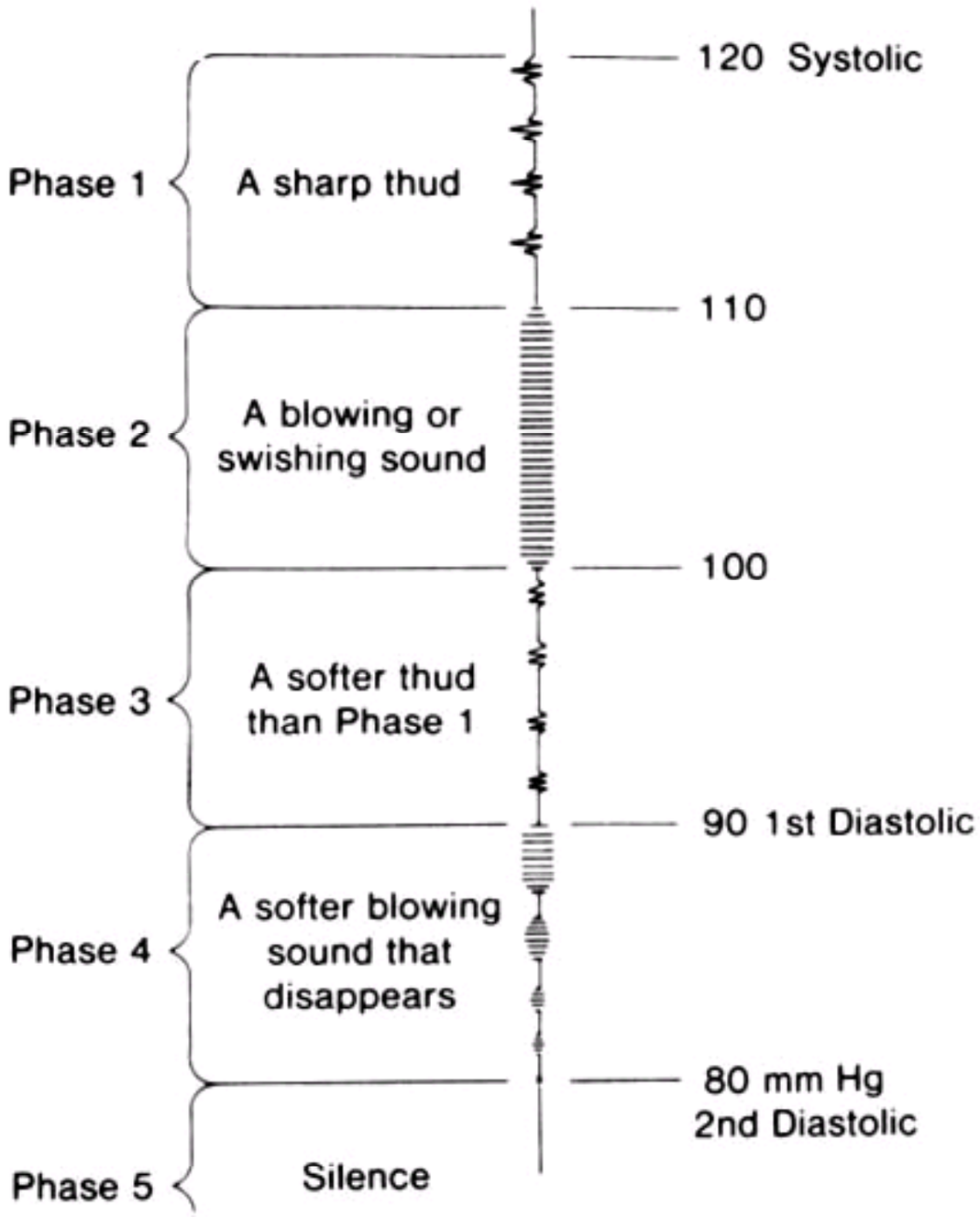
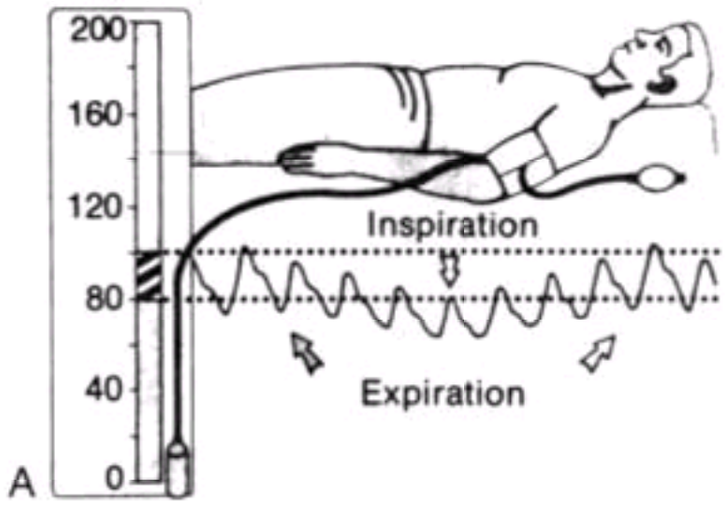


Figure 1-3 *A*, Measurement of pulsus paradoxus. Note that the systolic pressure varies during the respiratory cycle. *(From Stein L, Shubin H, Weil M: Recognition and management of pericardial tamponade. JAMA 225:504, 1973. Copyright 1973, American Medical Association. Reproduced by permission.)* *B*, Technique for the measurement of pulsus paradoxus.



A

PROCEDURE FOR THE MEASUREMENT OF PULSUS PARADOXUS

Patient should be reclining at a 30 to 45° angle and instructed to breathe normally.

1. Inflate standard blood pressure cuff until Korotkoff sounds over brachial artery disappear.
2. Lower pressure in cuff a few millimeters of mercury per second until first Korotkoff sounds appear during expiration.
3. Maintain pressure at this level and observe disappearance of sounds during inspiration. Record this cuff pressure.
4. Very slowly lower cuff pressure unitl Korotkoff sounds are heard throughout the respiratory cycle. Record this cuff pressure.
5. The difference between pressures recorded in the 2 previous steps is then recorded as the measurement (in mm Hg) of pulsus paradoxus. A pulsus paradoxus >12 mm Hg is abnormal. (see text).

B

Figure 1-4 Pocket Doppler stethoscope (model BF4A). *(Courtesy of Medsonics Inc, Los Altos, CA.)*

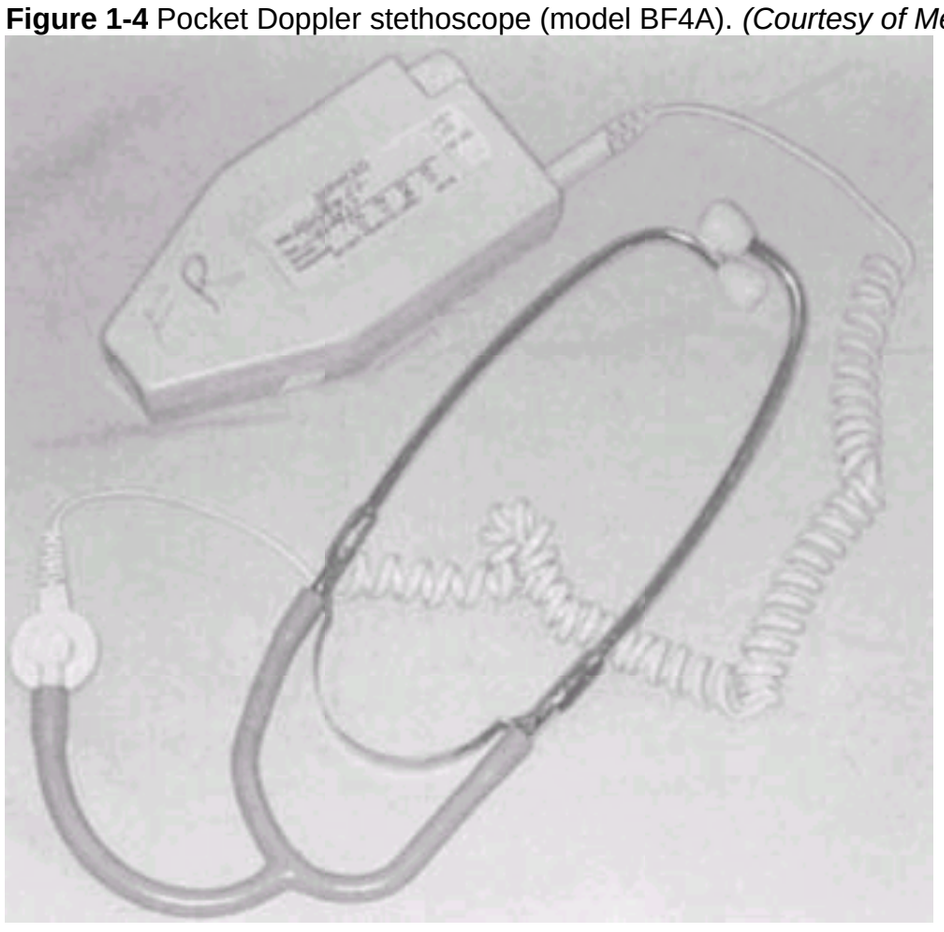


Figure 1-5 Ultrasonic Doppler flow detector with speaker and probes (model 811). *(Courtesy of Parks Medical Electronics, Aloha, OR.)*

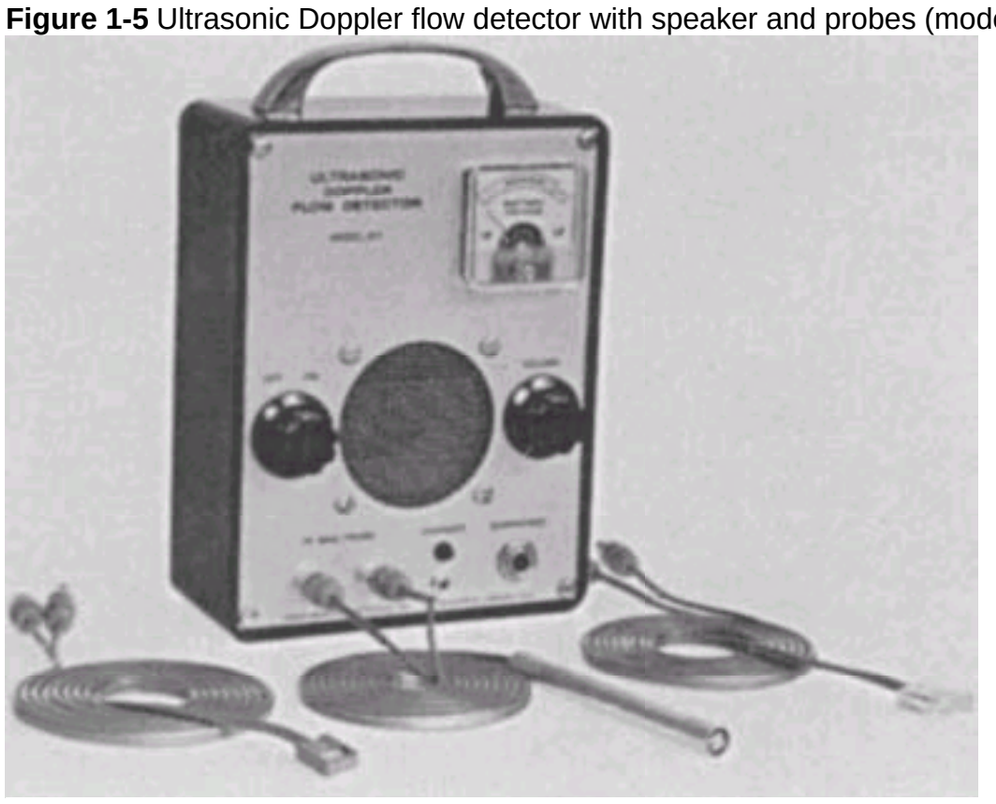


Figure 1-6 Typical pressures in a normal subject. Findings, based on resting pressures, show no evidence of occlusive disease of the large- or medium-sized arteries. Significant findings (normal) are as follows: (1) Ankle-to-brachial pressure index =1.0. (2) All pressure gradients <30 mm Hg. (3) Upper thigh pressure at least 40 mm Hg above brachial pressure. *(From Doppler Evaluation of Peripheral Arterial Disease: A Clinical Handbook. Fredericksburg, VA, Sonicaid, Inc. Reproduced by permission.)*

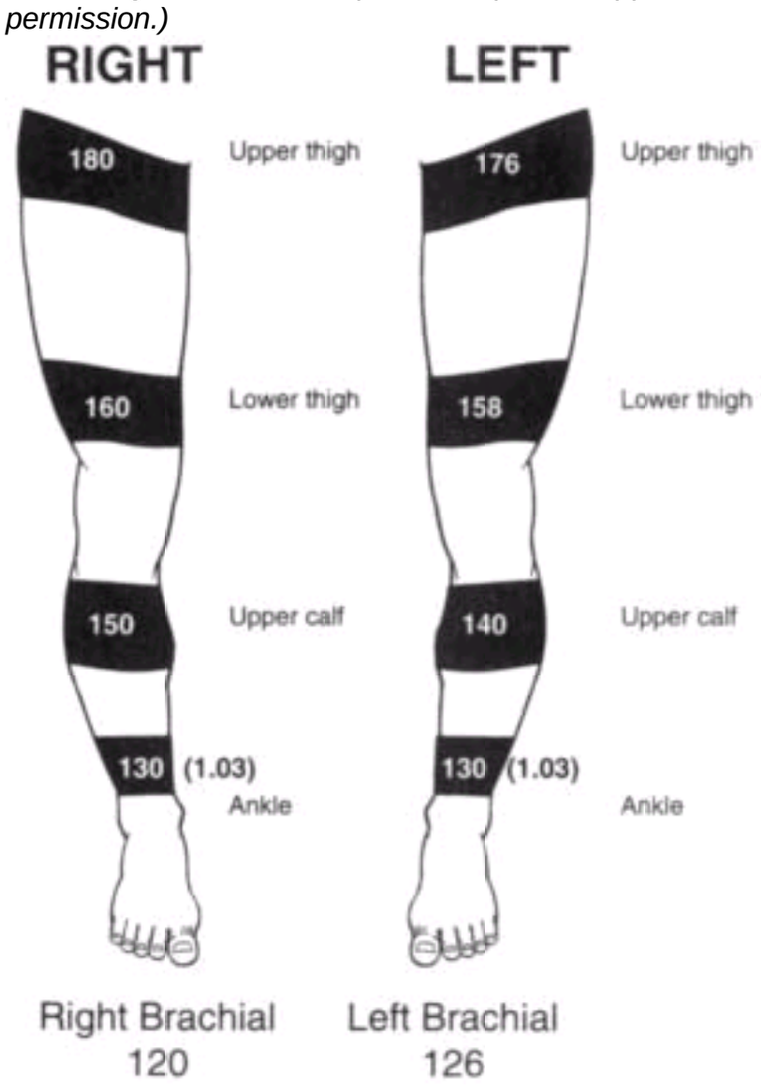


Figure 1-7 Typical pressures in a patient with obstruction of the popliteal or tibial arteries. Significant findings are as follows: (1) Ankle-to-brachial pressure index <0.9 in right leg. (2) Abnormally high gradient from ankle to below knee and again from below to above knee in right leg. (3) Upper thigh pressures are 50 mm Hg higher than brachial pressures, consistent with normal flow at the aorta-iliac level. Findings are suggestive of a right popliteal occlusion or an anterior and posterior tibial occlusion, or both. *(From Doppler Evaluation of Peripheral Arterial Disease: A Clinical Handbook. Fredericksburg, VA, Sonicaid, Inc. Reproduced by permission.)*

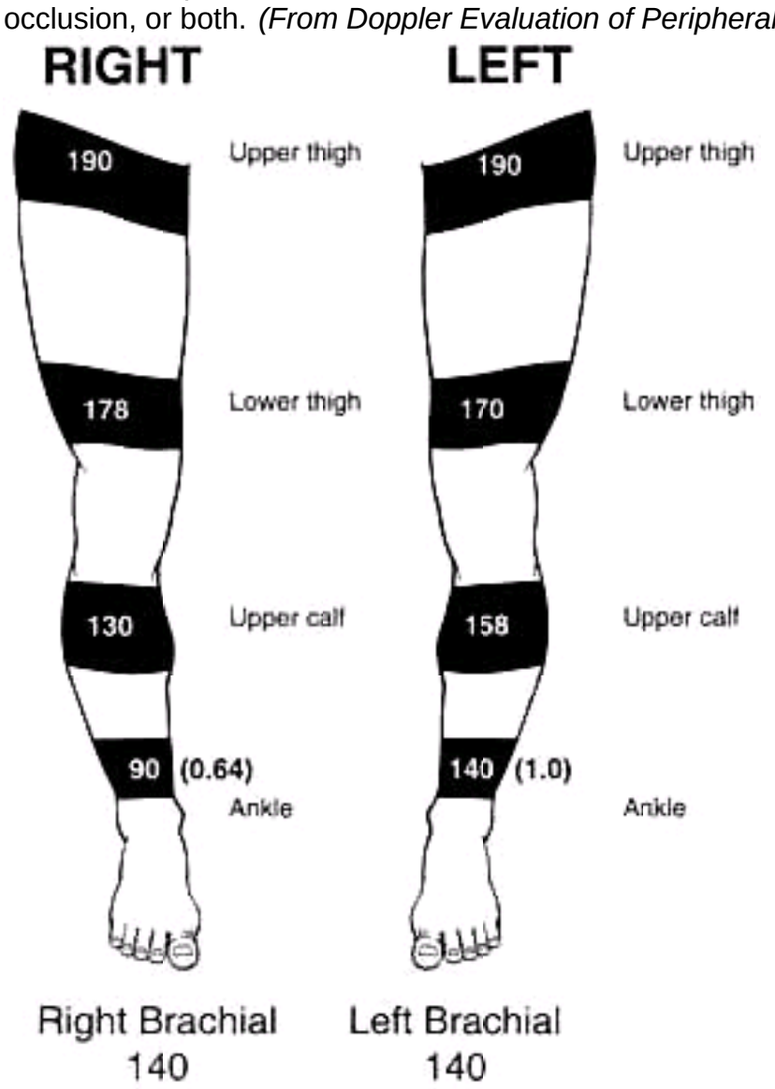


Figure 1-8 Typical pressures in a patient with obstruction of the abdominal aorta or bilateral iliac obstruction. Significant findings are as follows: (1) Ankle-to-brachial pressure index <0.9. (2) All segmental gradients <30 mm Hg. (3) Both upper thigh pressures relatively low with respect to brachial pressure. Findings are suggestive of severe aorto-iliac occlusive disease. *(From Doppler Evaluation of Peripheral Arterial Disease: A Clinical Handbook. Fredericksburg, VA, Sonicaid, Inc. Reproduced by permission.)*

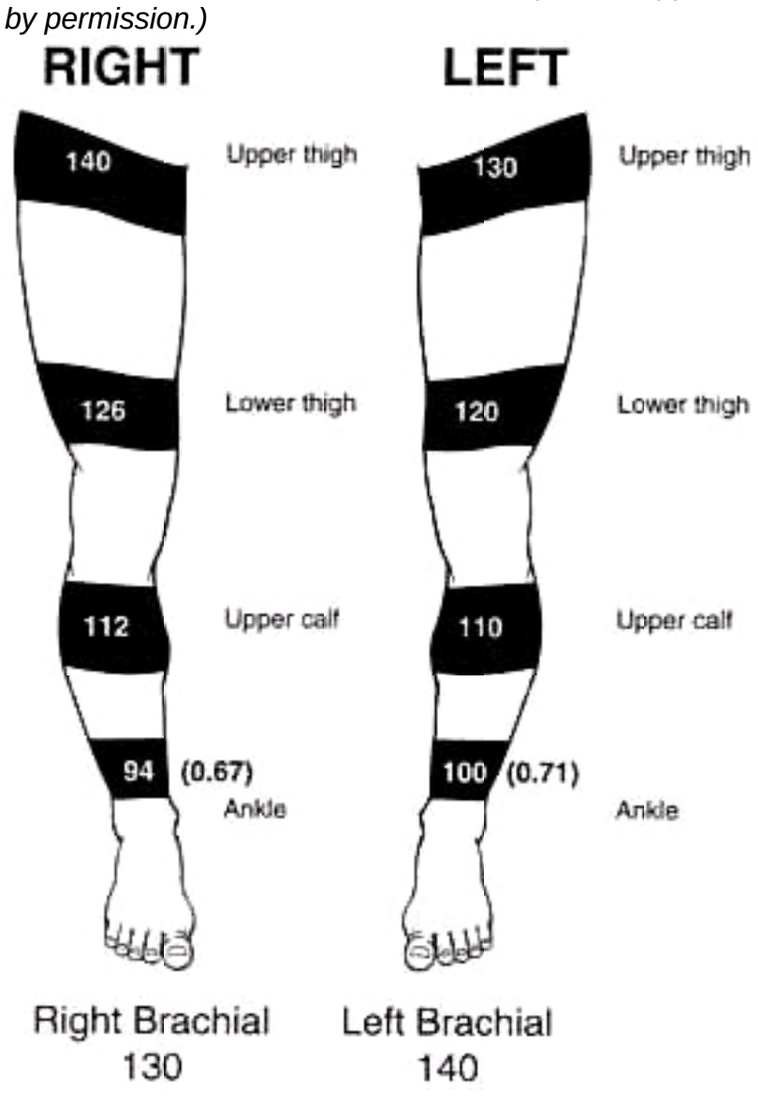


Figure 1-9 The effect of hemorrhage on cardiac output and arterial pressure. *(From Guyton AC: Textbook of Medical Physiology, 6th ed. Philadelphia, WB Saunders, 1981. Reproduced by permission.)*

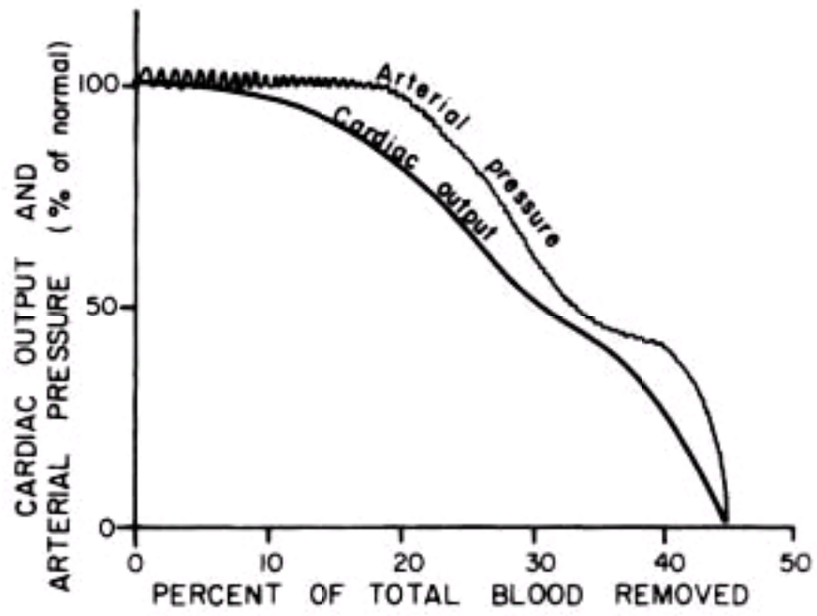


Figure 1-10 Relationship between oral and urinary temperatures constructed as a nomogram. Urinary temperatures are consistently within 1 to 1.5°C of simultaneously obtained oral temperatures and within 2°C of rectal temperatures. To use the nomogram, locate the oral temperature on the horizontal axis (*A*) and draw a perpendicular line to intersect the diagonal border of the graph (*B*). Follow the dotted horizontal line to the right to determine the expected urinary temperature on the longitudinal axis (*C*). The far right-hand scale (*D*) provides a 99% confidence level for the lower range of expected urinary temperatures and finding a value below the 99% confidence level is essentially diagnostic of a factitious fever. *(From Murray HW, Tuazon CU, Guerrero IC, et al: Urinary temperature: A clue to early diagnosis of factitious fever. N Engl J Med 296:23, 1977. Reproduced by permission of the New England Journal of Medicine.)*

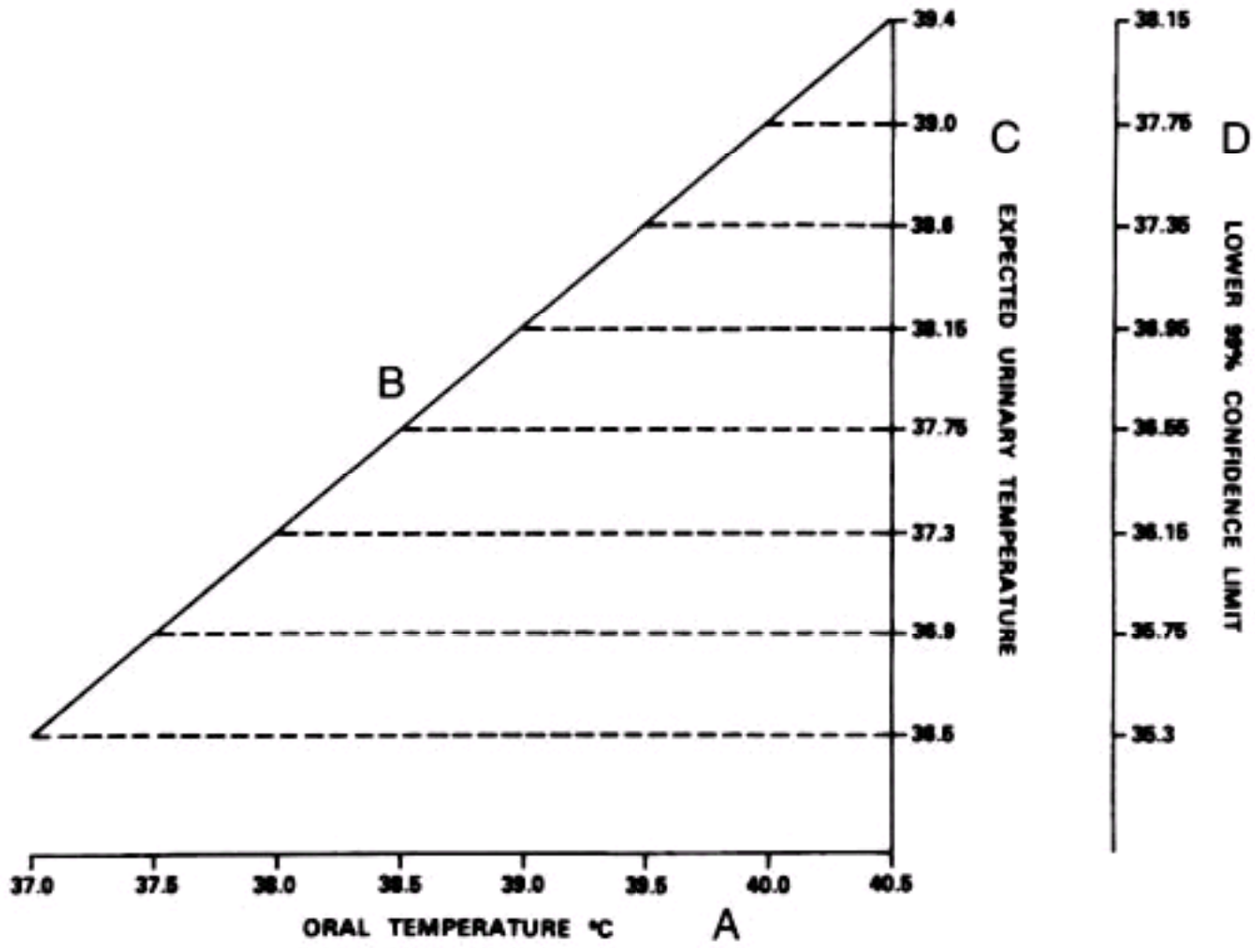


Figure 1-11 Temperature conversion scale. To change Celsius (centigrade) to Fahrenheit, multiply the Celsius temperature by 9/5 and add 32. To change Fahrenheit to Celsius, subtract 32 from the Fahrenheit number and multiply by 5/9.

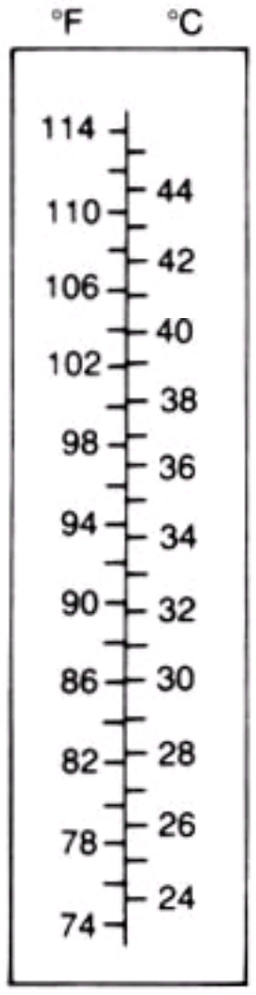


Figure 2-1 Time-forced vital capacity (FVC) is the volume of gas forcibly expelled following a maximal inspiration. Forced expiratory volume in 1 second (FEV_1) is the volume of gas expelled during the first second of the forced expiration. The other lung volumes obtainable are the tidal volume (TV), which is the volume of gas moved during quiet respiration; the inspiratory reserve volume (IRV), which is the volume of gas that can be inspired in addition to the tidal gas volume; and the expiratory reserve volume (ERV), which is the volume of gas that can be forcibly expired at the end of a tidal expiration. Some gas cannot be expired and remains in the chest. This is known as the residual volume (RV).

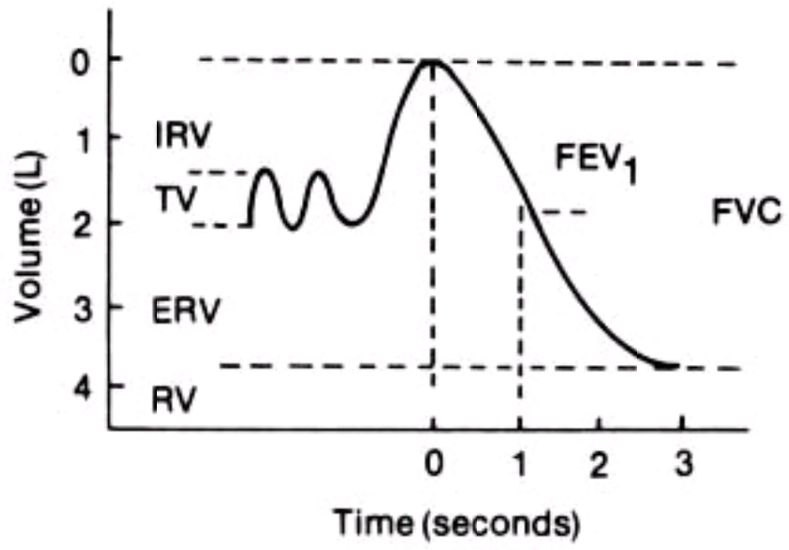


Figure 2-2 Wright peak flow meter. After resetting the dial to zero, the patient inhales fully and exhales forcefully through the disposable paper mouthpiece. The best of three attempts is recorded. A tight seal of the lips around the mouthpiece is required.

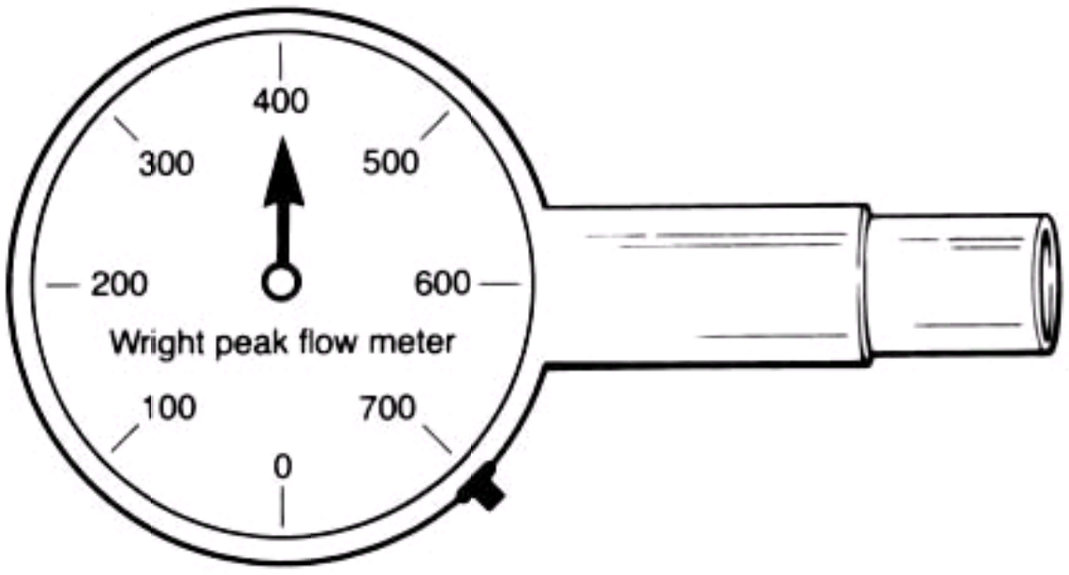


Figure 2-3 Mini-Wright peak flow meter. The indicator arrow is moved back to zero. The patient inhales fully and then exhales forcefully through the disposable paper mouthpiece. The best of three attempts is recorded. Patient cooperation and a tight seal of the lips around the mouthpiece are required.

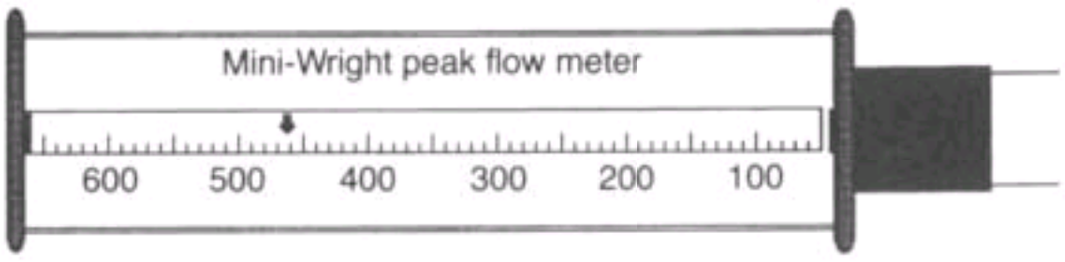


Figure 2-4 The Respiradyne portable spirometer. The device is turned on or "cleared" from the last effort. The patient exhales forcefully into the handheld mouthpiece following maximal inhalation. The "sequence" button permits selection of the desired spirometry measurements. The best of several tries should be recorded.

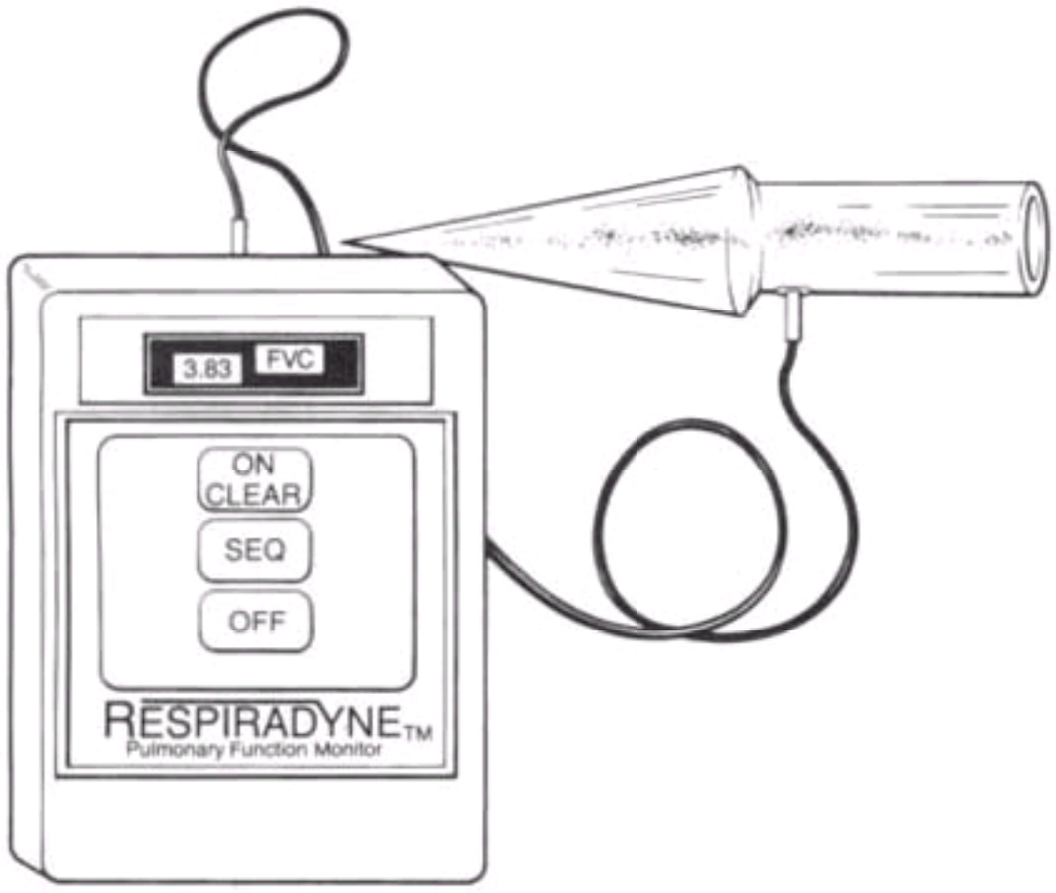
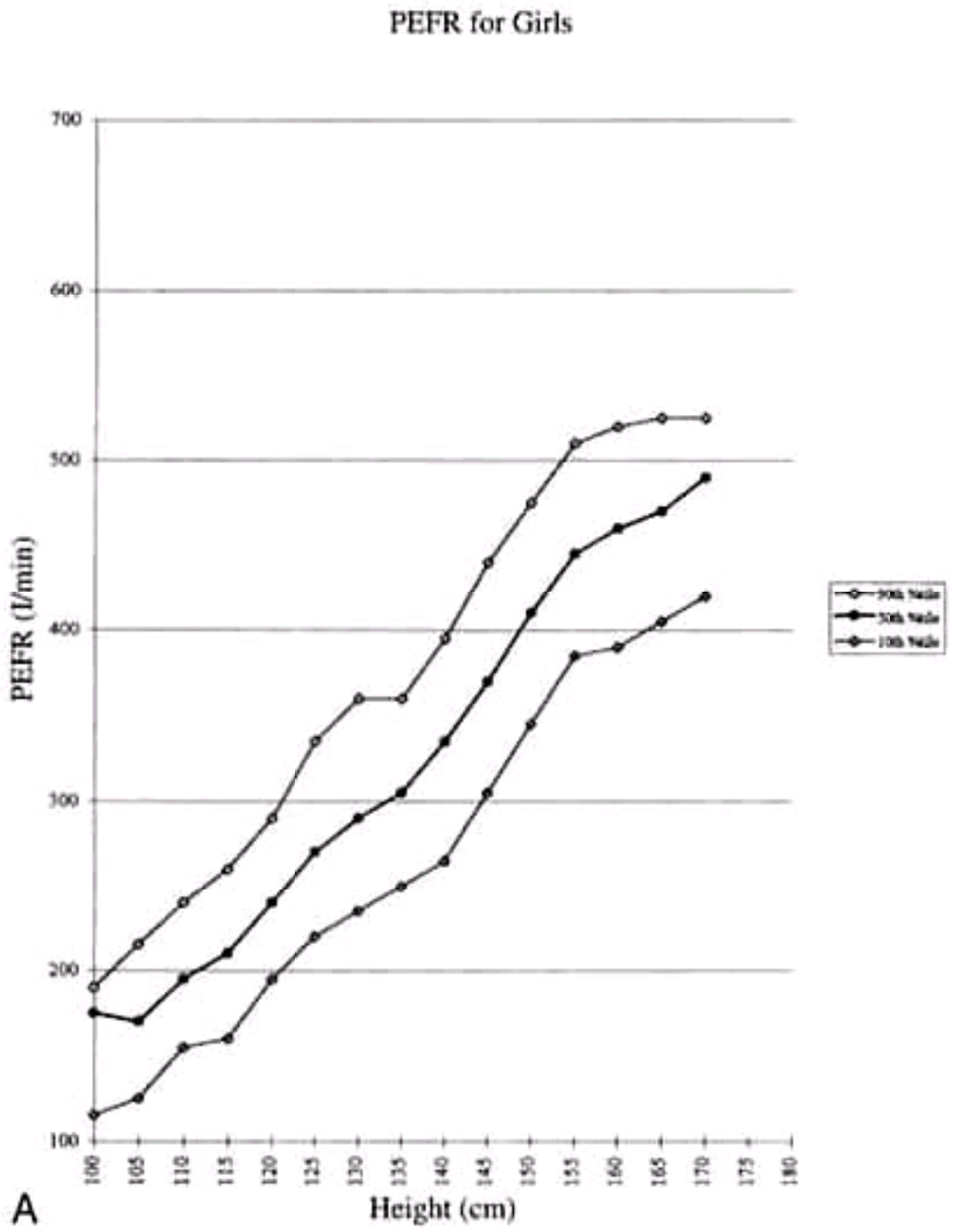


Figure 2-5 Peak expiratory flow rate vs height for girls (*A*) and for boys (*B*). These figures were constructed from Carson and coworkers' unsmoothed data obtained from 2752 healthy children in Dublin. *(From Carson JWK, Hoey H, Taylor MRH: Growth and other factors affecting peak expiratory flow rate. Arch Dis Child 64:96, 1989.)*



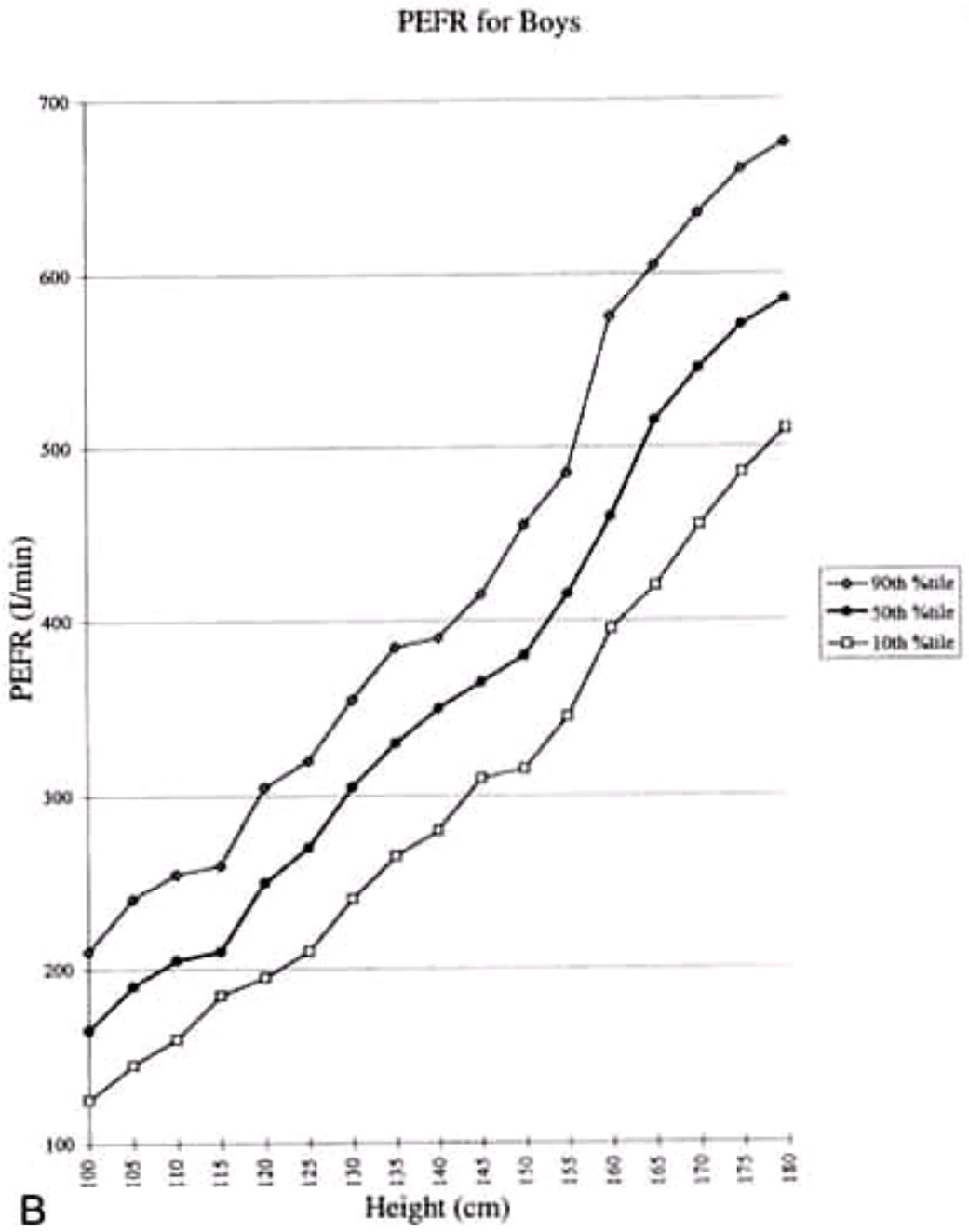


Figure 2-6 *A*, Pulse oximeter sensors attached to digits. *B*, Pulse oximeter sensors attached to the dorsum of the nose and the earlobe. Only one site is measured at a time.

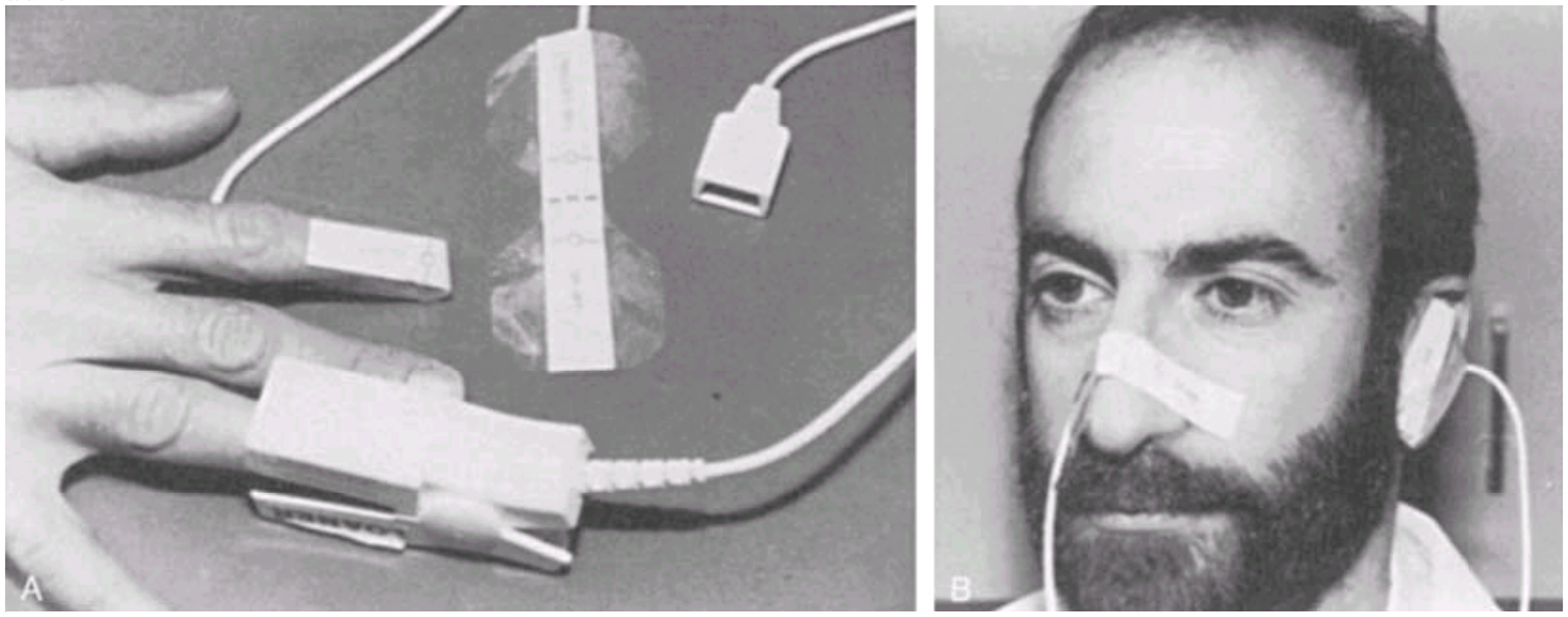


Figure 2-7 Factors influencing light absorption through pulsatile vascular bed. *(From McGough EK, Boysen PG: Benefits and limitations of pulse oximetry in the ICU. J Crit Illness 4:23, 1989.)*

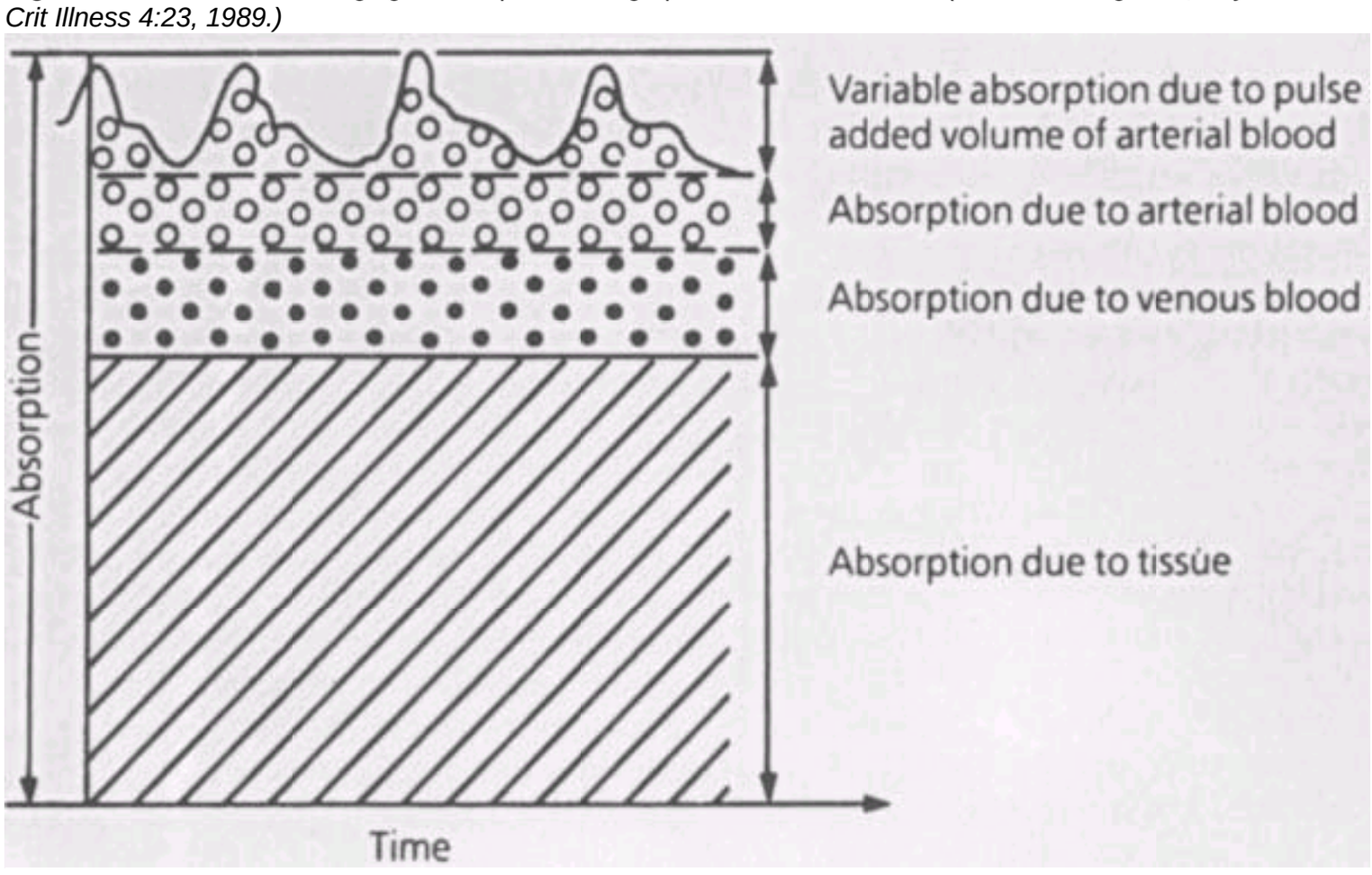


Figure 2-8 Oxyhemoglobin dissociation curve.

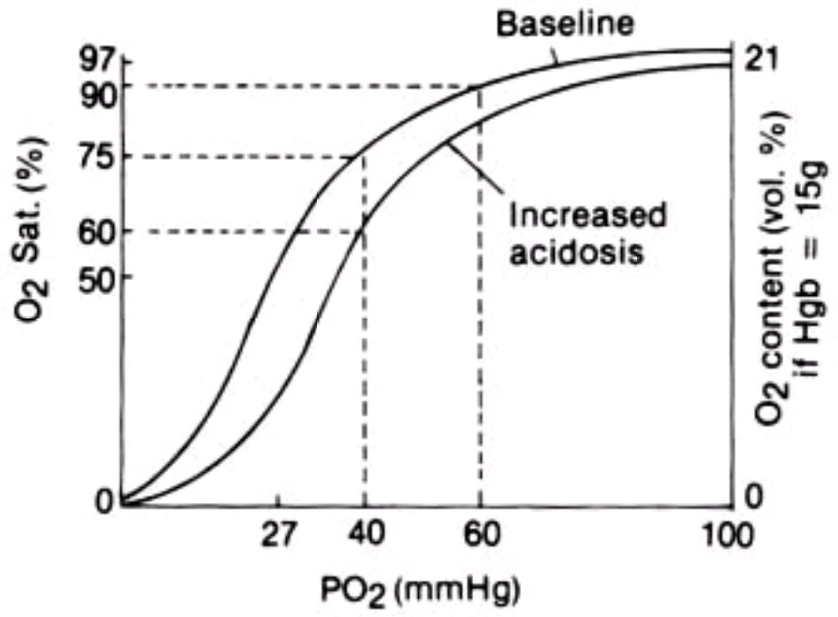


Figure 2-9 Schema of mainstream analyzer used for capnography. Transducer to measure CO_2 is placed in-line of airway connector. Infrared light source (not indicated) resides opposite photodetector in transducer. *(From Szalfarski NL, Cohen NH: Use of capnography in critically ill adults. Heart Lung 20:363, 1991.)*

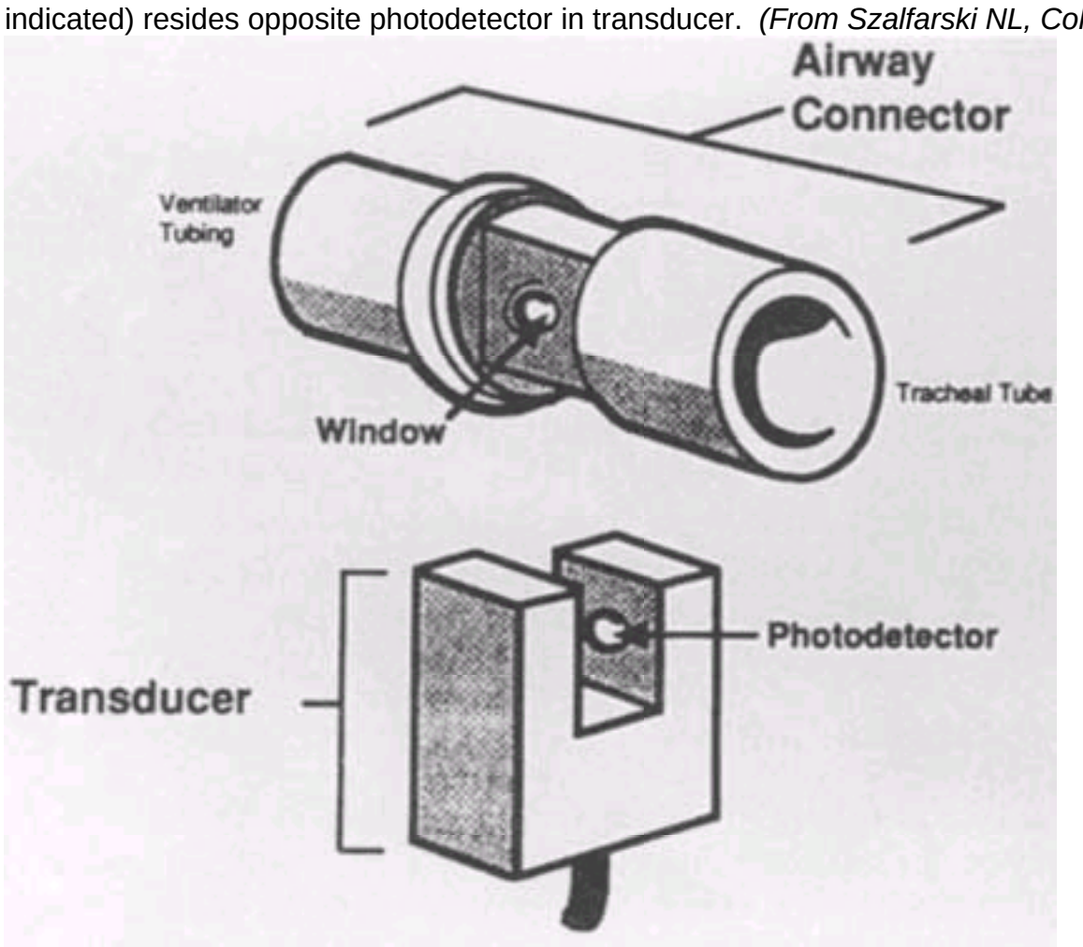


Figure 2-10 Schema of side-stream CO_2 sampling technique in capnography. *(From Szalfarski NL, Cohen NH: Use of capnography in critically ill adults. Heart Lung 20:363, 1991.)*

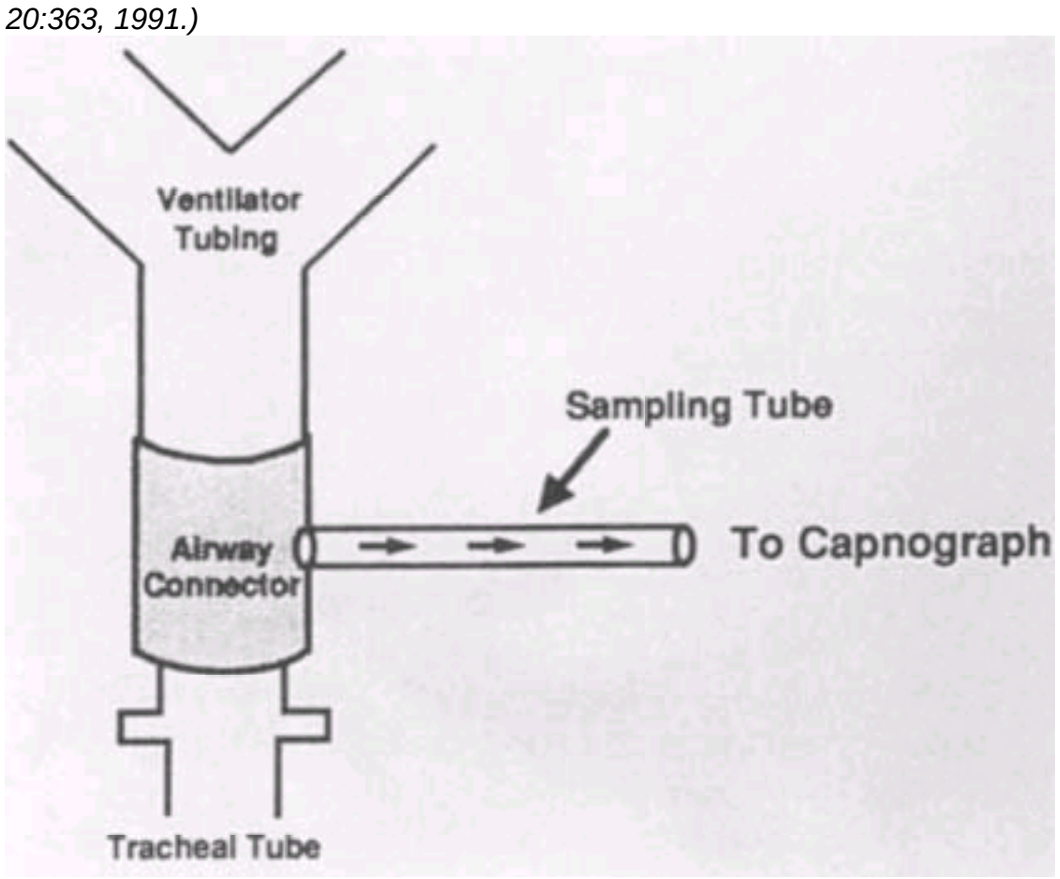
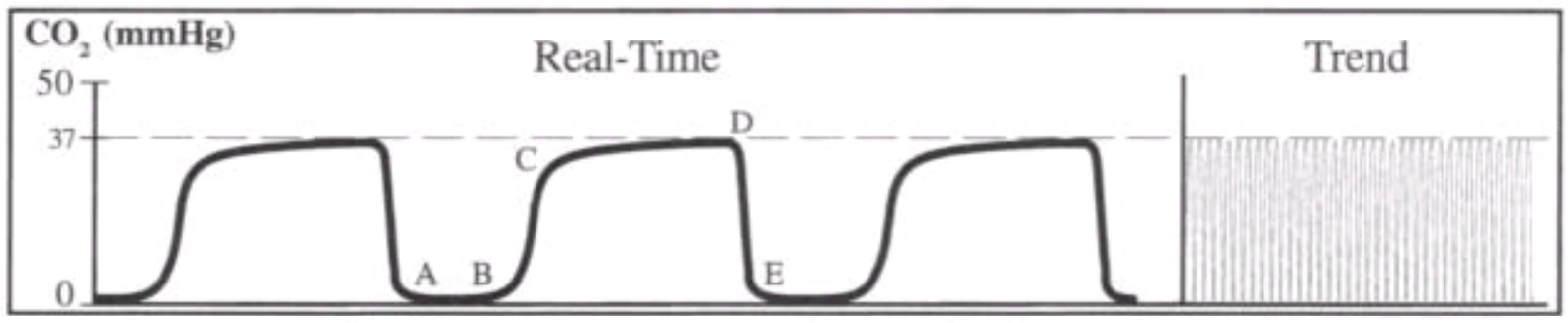


Figure 2-11 The four phases of a normal capnogram. Exhalation begins at point *B*. The steep ascending limb (segment *B–C*) represents rapidly exchanging alveoli. Segment *C–D* is the alveolar plateau representing uniformly ventilated alveoli, with a nearly constant CO_2 concentration. $PetCO_2$ is the highest point on the alveolar plateau (point *D*) and immediately precedes the next inspiration. Segment *D–E* rapidly descends to the original baseline (zero in normal subject) during inspiration (segment *A–B*). *(Courtesy of Novametrix Medical Systems, Inc., Wallingford, CT.)*



The "normal" capnogram is a waveform which represents the varying CO_2 level throughout the breath cycle.

Waveform Characteristics:

A–B	Baseline
B–C	Expiratory Upstroke
C–D	Expiratory Plateau
D	End Tidal Concentration
D–E	Inspiration Begins

Figure 2-12 Capnogram of patient with adult respiratory distress syndrome and pneumonia. Lack of a normal alveolar plateau is evident. *(From Szalfarski NL, Cohen NH: Use of capnography in critically ill adults. Heart Lung 20:369, 1991.)*

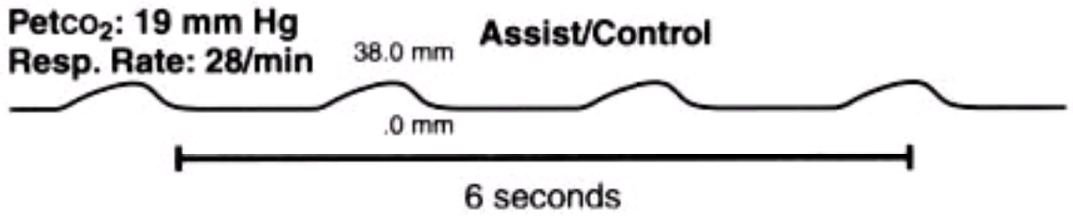


Figure 2-13 Esophageal capnogram after ingestion of carbonated beverage. *A*, CO_2 is clearly present as the esophagus is ventilated. The CO_2 concentration rapidly dissipates to the nonphysiologic range. *B*, The normal tracheal capnogram obtained simultaneously from the same experimental animal is shown. *(Modified from Garnett AR, Gervin CA, Gervin AS: Capnographic waveforms in esophageal intubations: Effect of carbonated beverages. Ann Emerg Med 18:387, 1989.)*

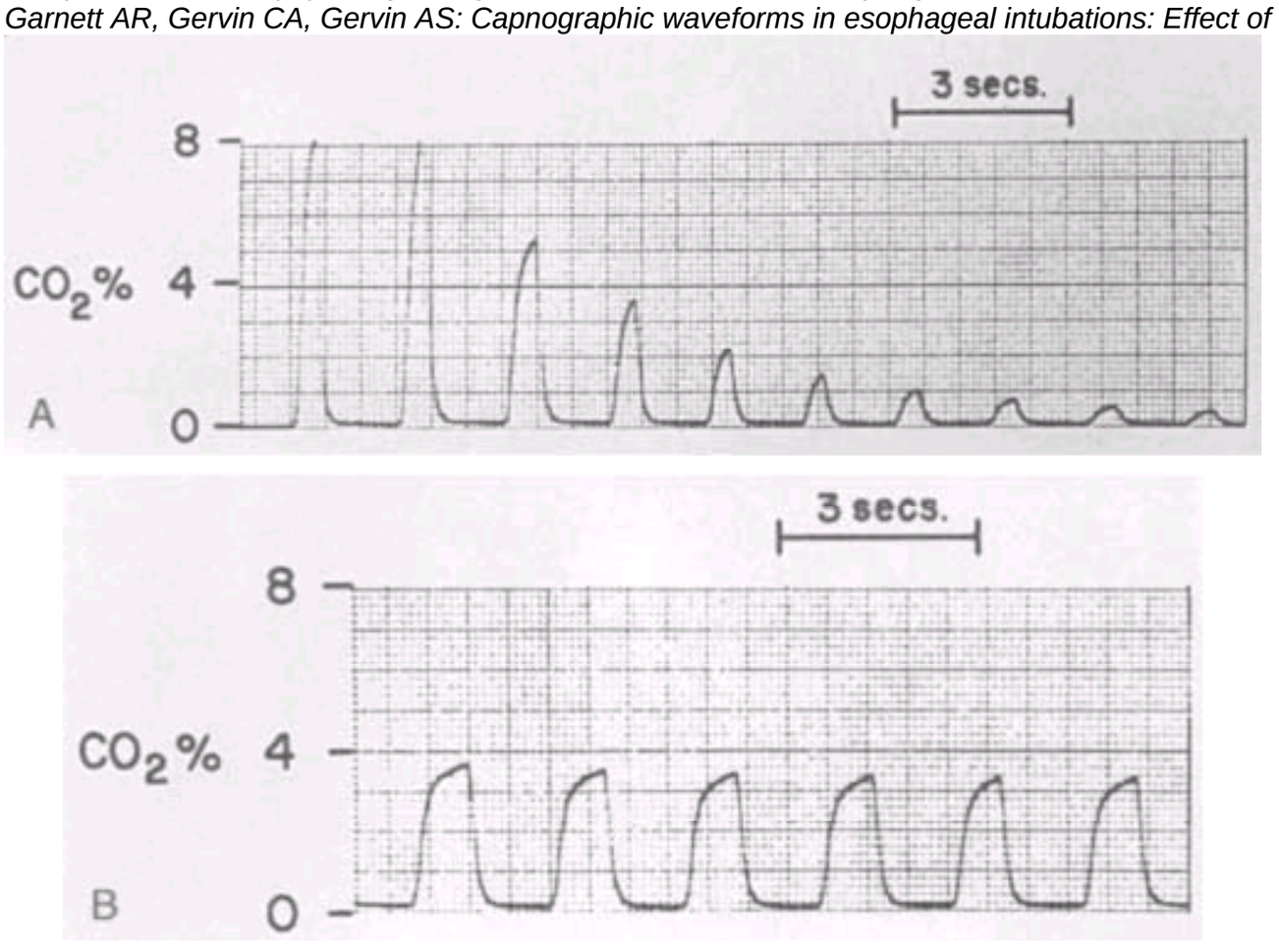
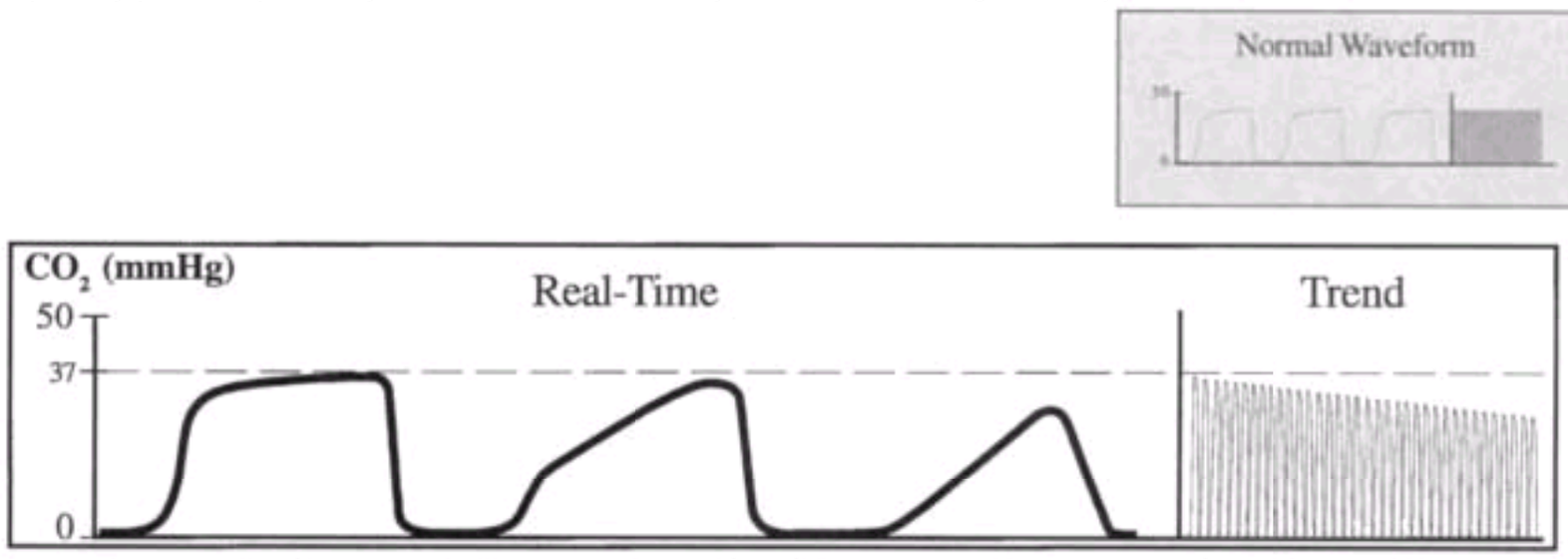


Figure 2-14 Partial endotracheal tube occlusion and other obstructions in the breathing circuit or airway. Note slurred upstroke of CO_2 waveform and absence of expiratory plateau. *(Courtesy of Novametrix Medical Systems, Inc., Wallingford, CT.)*

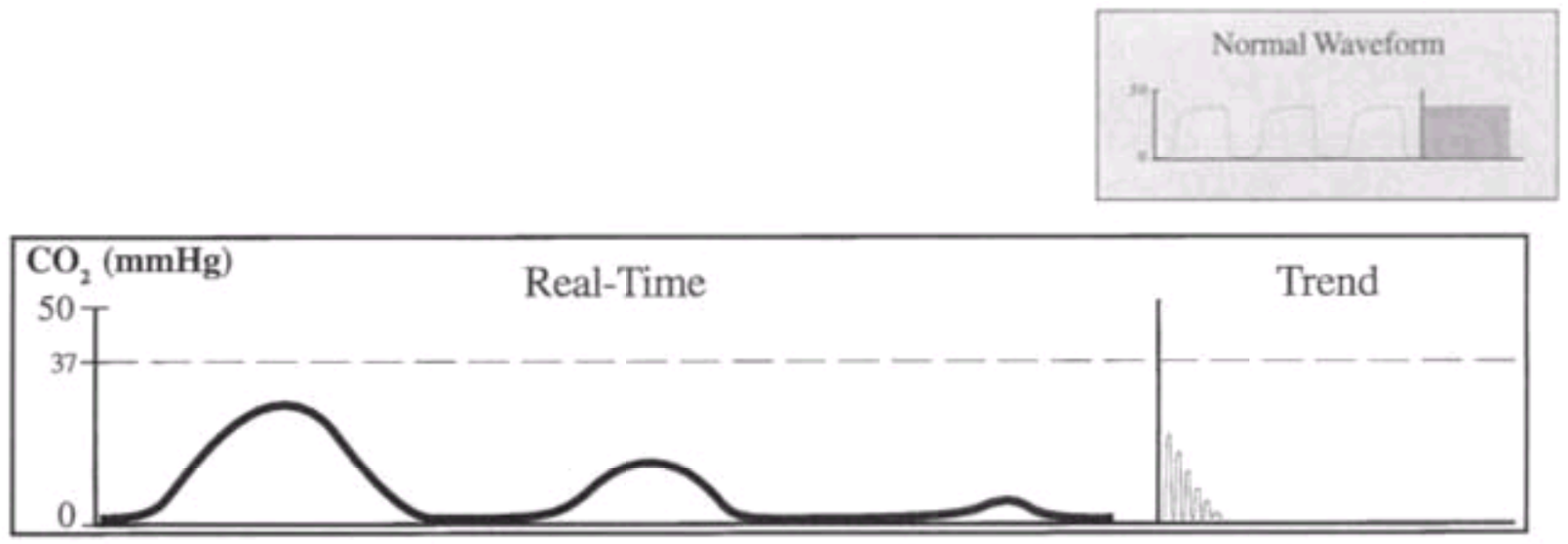


Obstructed expiratory gas flow is noted as a change in the slope of the ascending limb of the capnogram (the expiratory plateau may be absent).

Possible Causes:

- Obstruction in the expiratory limb of the breathing circuit
- Presence of a foreign body in the upper airway
- Partially kinked or occluded artificial airway
- Bronchospasm

Figure 2-15 Malposition of endotracheal tube in the esophagus. Note loss of capnographic waveform. *(Courtesy of Novametrix Medical Systems, Inc., Wallingford, CT.)*



Waveform Evaluation:

A normal capnogram is the best available evidence that the ET tube is correctly positioned and that proper ventilation is occurring. When the ET tube is placed in the esophagus, either no CO_2 is sensed or only small transient waveforms are present.

Figure 2-16 Occlusion of the endotracheal tube. Note that occlusion of a properly placed tube yields the same flat capnogram as esophageal intubation or accidental extubation. *(From Murray IP, Modell JH: Early detection of endotracheal tube accidents by monitoring carbon dioxide concentration in respiratory gas. Anesthesiology 59:345, 1983.)*

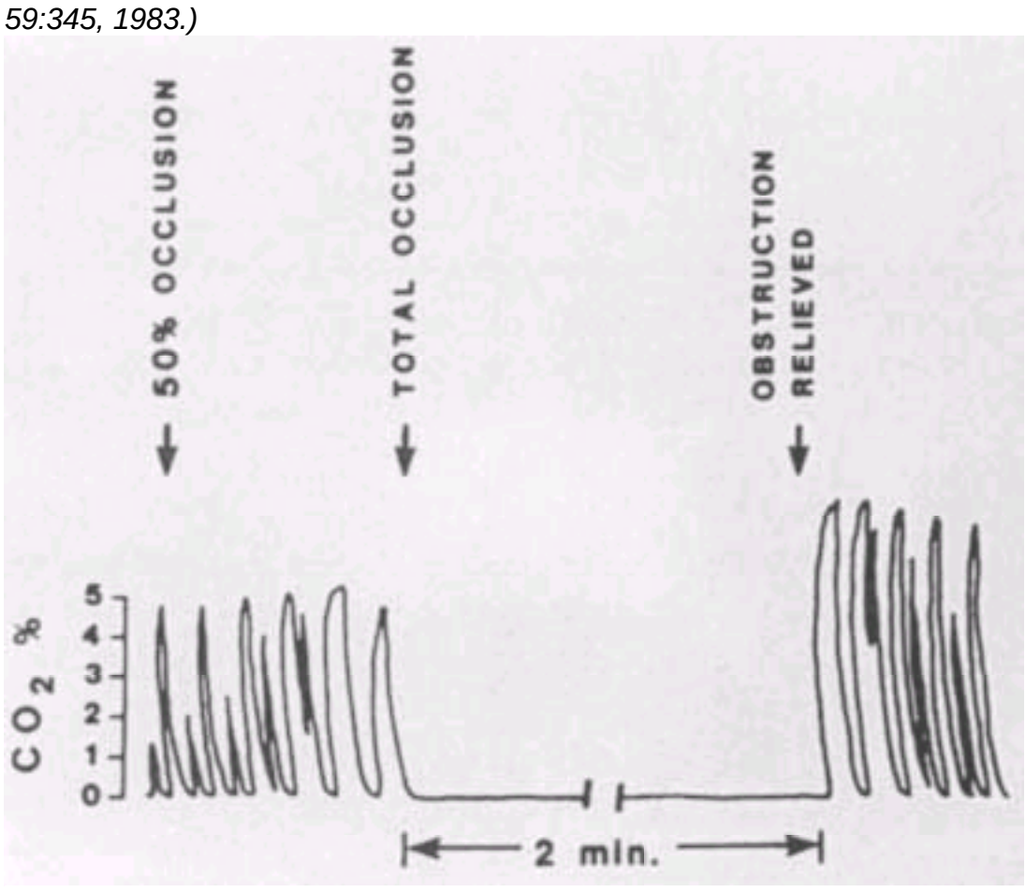
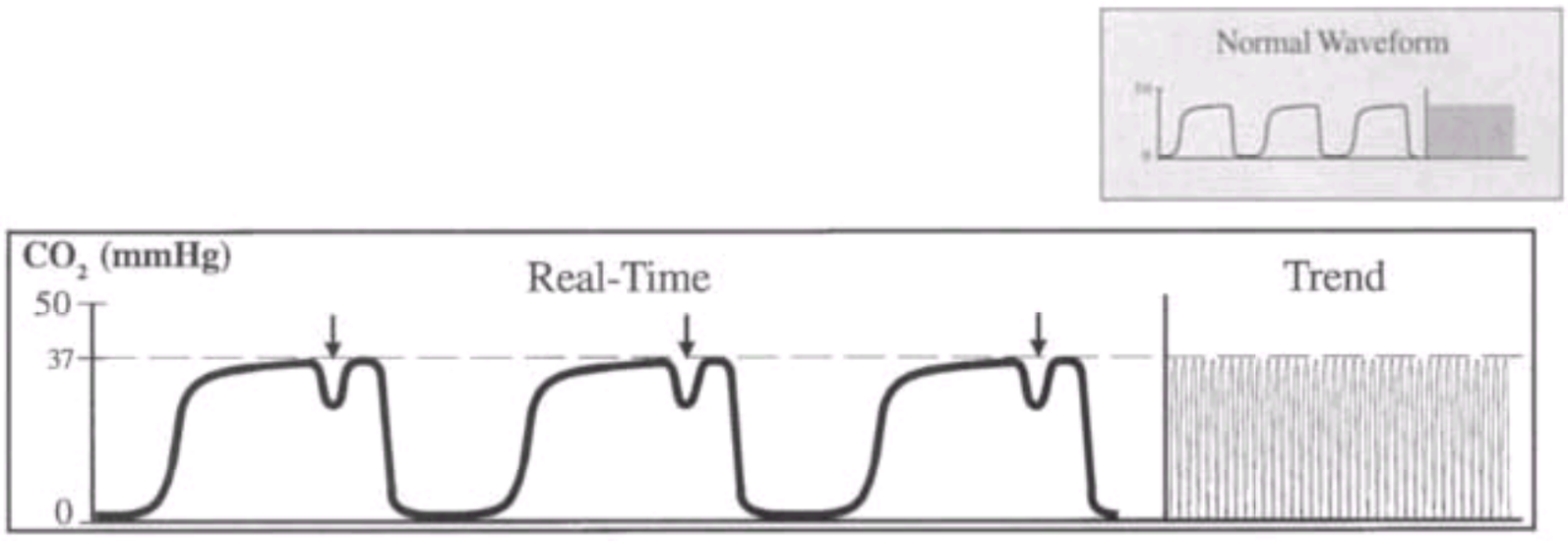


Figure 2-17 Capnogram of patient receiving assist-control ventilation and vecuronium paralyzation. *(Courtesy of Novametrix Medical Systems, Inc., Wallingford, CT.)*

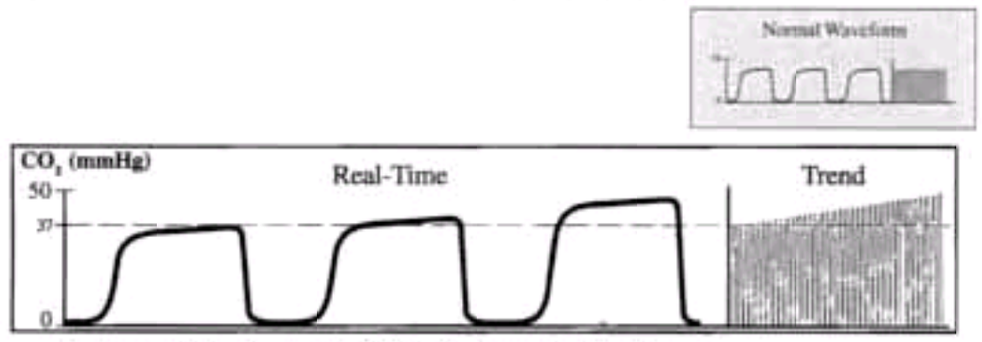


Clefts are seen in the plateau portion of the capnogram. They appear when the action of the muscle relaxant begins to subside and spontaneous ventilation returns.

Characteristics:

- Depth of the cleft is inversely proportional to the degree of drug activity
- Position is fairly constant on the same patient but not necessarily present with every breath

Figure 2-18 *A–F,* Various abnormal capnography patterns and their causes. *(Courtesy of Novametrix Medical Systems, Inc., Wallingford, CT.)*

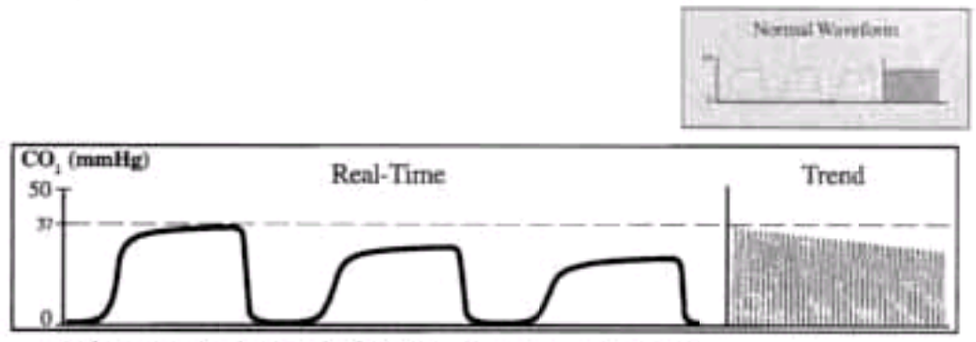


An increase in the level of $ETCO_2$ from previous levels.

Possible Causes:

- Decrease in respiratory rate (hypoventilation)
- Decrease in tidal volume (hypoventilation)
- Increase in metabolic rate
- Rapid rise in body temperature (malignant hyperthermia)

A

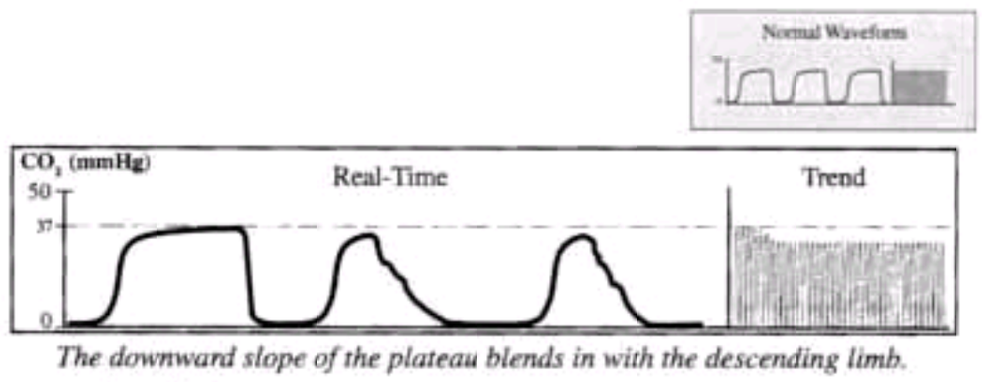


A decrease in the level of $ETCO_2$ from previous levels.

Possible Causes:

- Increase in respiratory rate (hyperventilation)
- Increase in tidal volume (hyperventilation)
- Decrease in metabolic rate
- Fall in body temperature

B

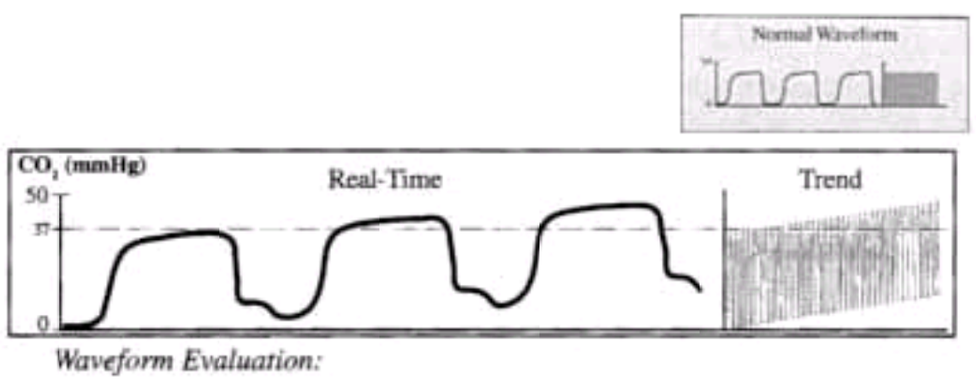


The downward slope of the plateau blends in with the descending limb.

Possible Causes:

- A leaky or deflated endotracheal or tracheostomy cuff
- An artificial airway that is too small for the patient

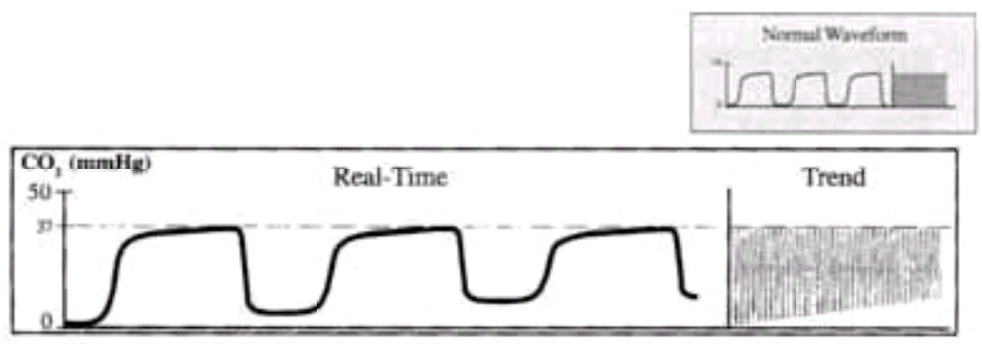
C



Waveform Evaluation:

- Baseline elevated
- Abnormal descending limb of capnogram
- Allows patient to rebreathe exhaled gas

D

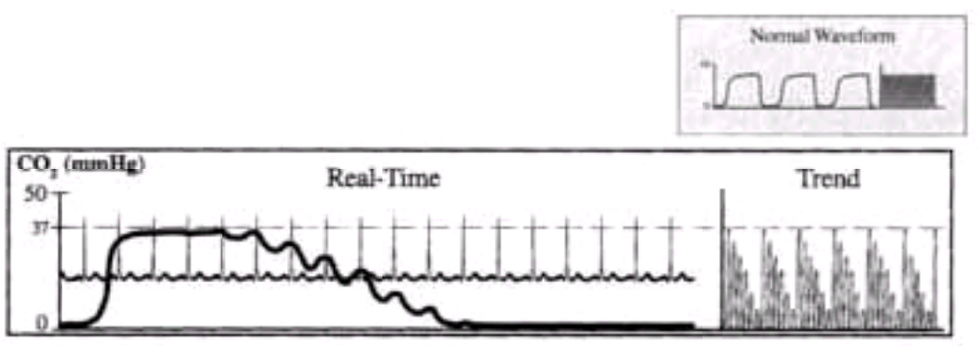


Elevation of the baseline indicates rebreathing (may also show a corresponding increase in $ETCO_2$).

Possible Causes:

- Faulty expiratory valve
- Inadequate inspiratory flow
- Malfunction of a CO_2 absorber system
- Partial rebreathing circuits
- Insufficient expiratory time

E



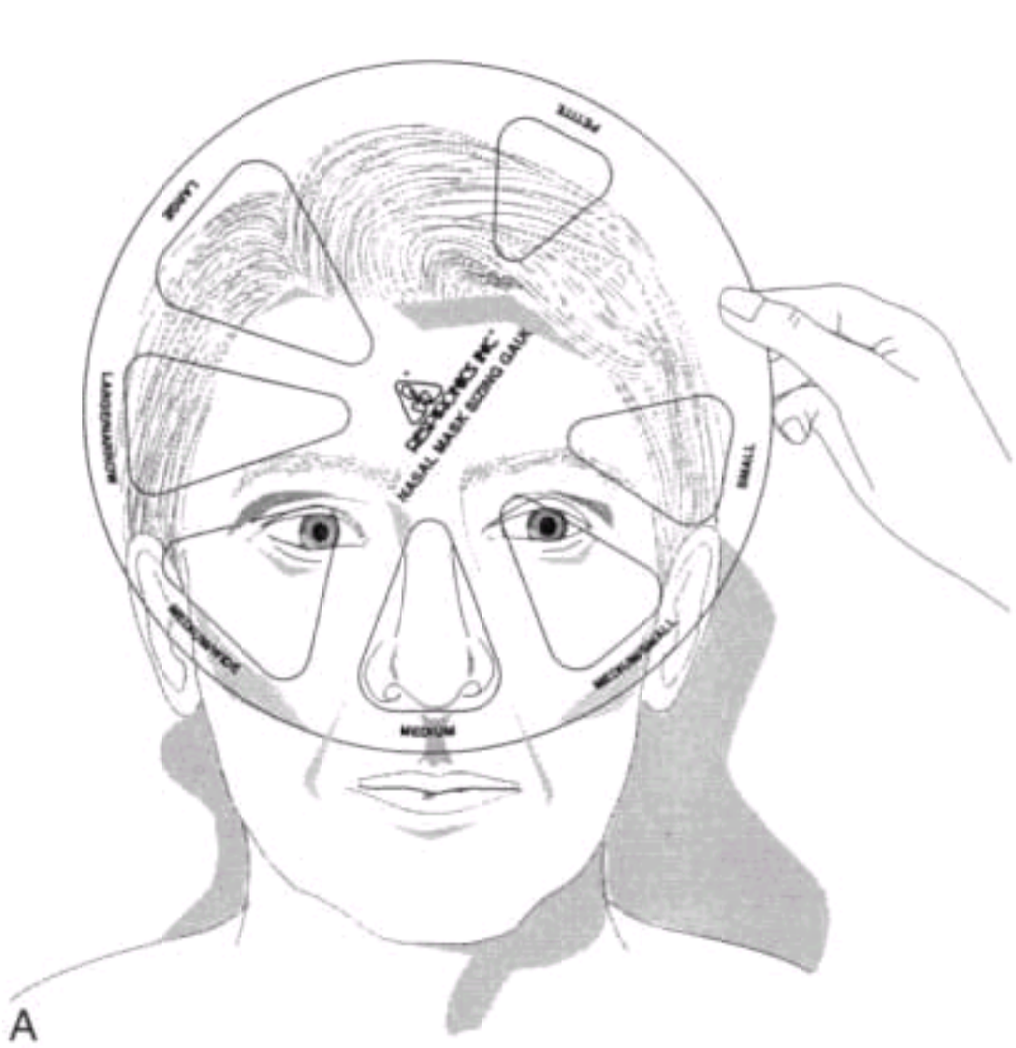
Cardiogenic oscillations appear during the final phase of the alveolar plateau and during the descending limb. They are caused by the heart beating against the lungs.

Characteristics:

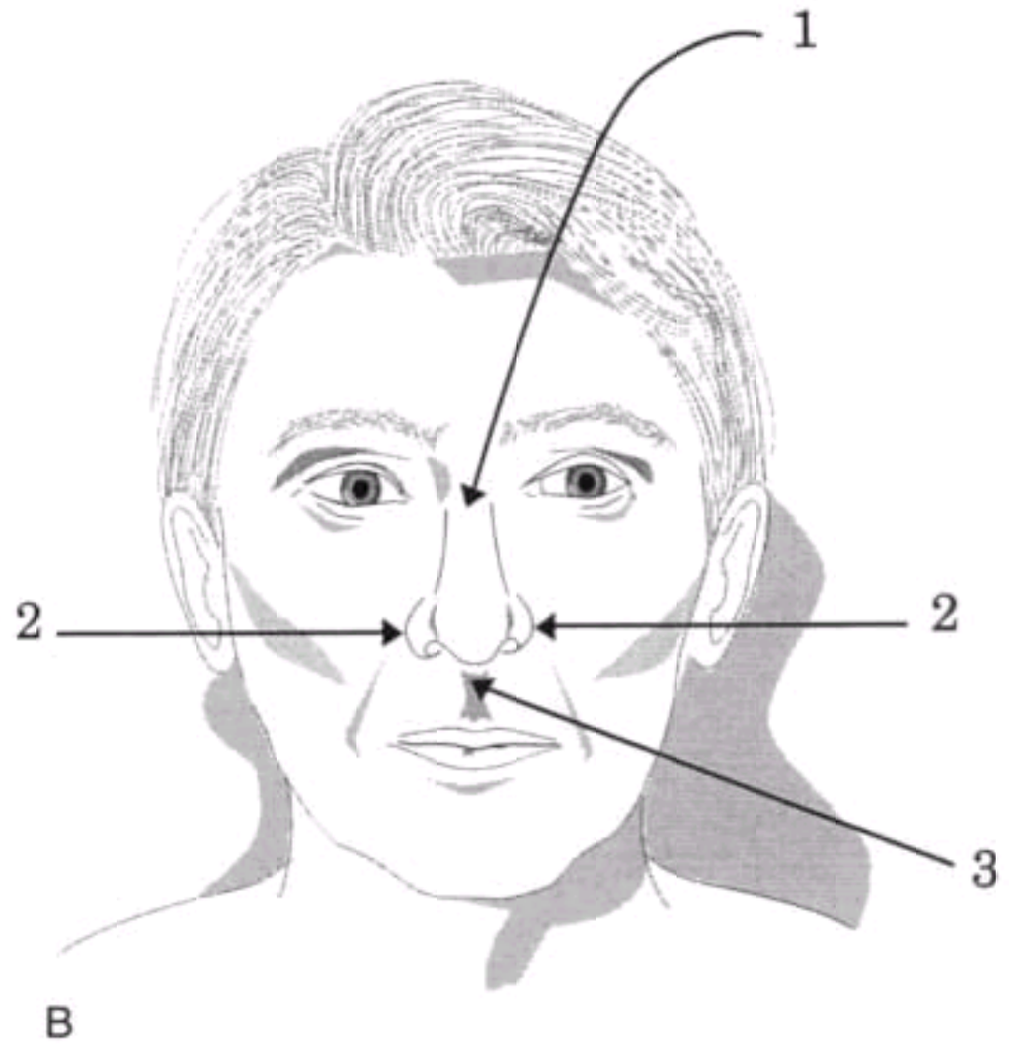
- Rhythmic and synchronized to heart rate
- May be observed in pediatric patients who are mechanically ventilated at low respiratory rates with prolonged expiratory times

F

Figure 2-19 Use of template to size nasal mask for BiPAP System. *A*, The nasal mask sizing gauge is placed over the patient's nose. The size that comes close to, but does not touch, the nose in three locations is selected. *B*, The sites to avoid direct contact: (1) just above the junction of the nasal bone and cartilage; (2) on the sides of both nares; and (3) just below the lowest point of the nose, above the lip. *Remember to use the smallest size mask that will sufficiently cover the nasal area.* Too small a mask will produce skin discomfort or injury. Too large a mask will increase air leak. *(Courtesy of Respironics Inc., Murrysville, PA.)*

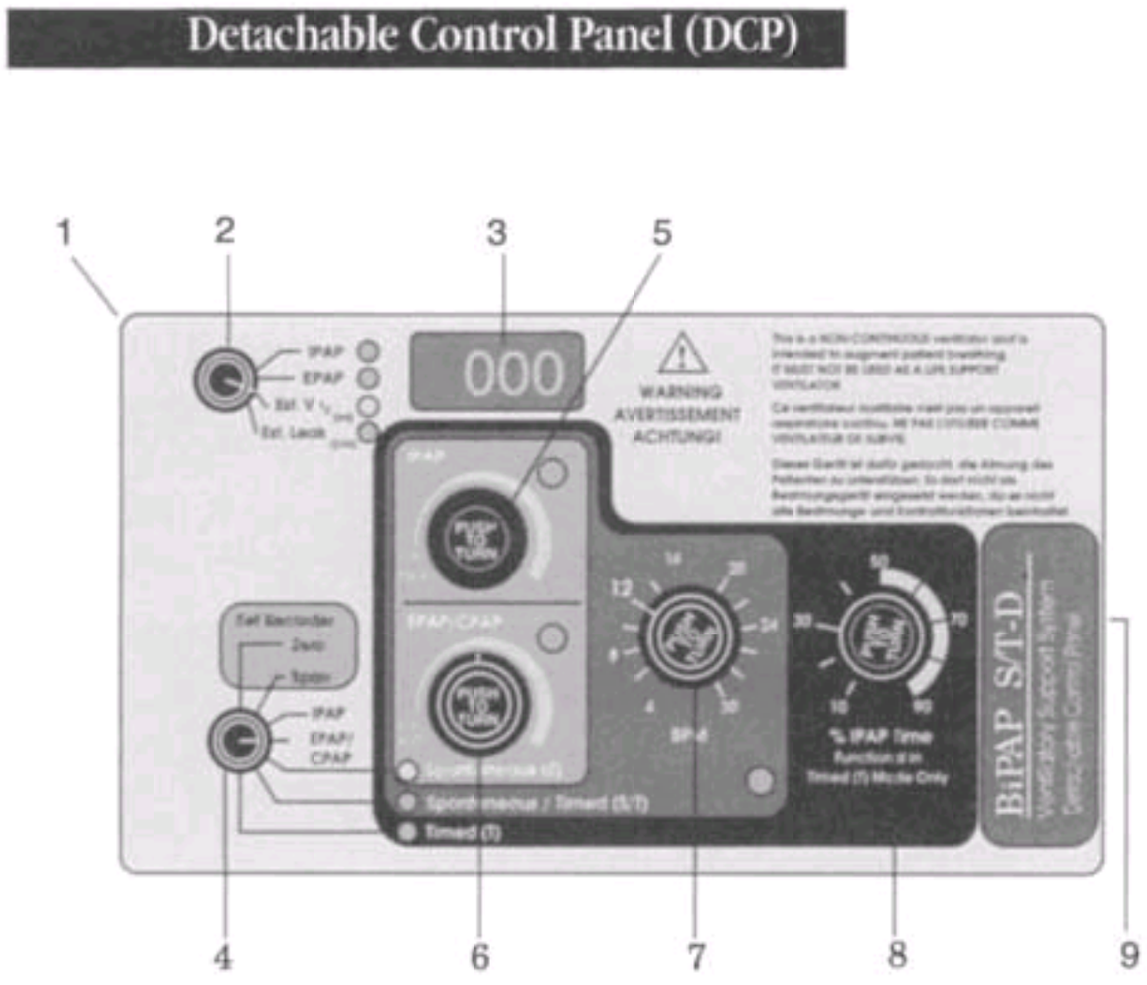


A



B

Figure 2-20 Control panel of BiPAP System. Note control setting for ventilation mode in lower left corner (see text for use of settings). For spontaneously breathing patients in respiratory distress, the "spontaneous" mode is generally selected and IPAP and EPAP settings selected. *(Courtesy of Respironics Inc., Murrysville, PA.)*



1 Puts controls at fingertips of hospital staff.

2 **Display Selector** determines the type of information shown on the Digital Display:
- **IPAP** setting displays delivered inspiratory pressure level
- **EPAP** setting displays delivered expiratory pressure level
- **Estimated Tidal Volume** setting displays estimated exhaled tidal volume
- **Estimated Leak** setting displays estimated circuit leak

3 **Digital Display** provides data for patient monitoring:
- Provides continuous monitoring of estimated exhaled tidal volume
- Allows verification of circuit pressures
- Offers capability to monitor circuit leaks
- Indicator lights specify which display parameter is active

4 **Function Selector** sets one of four different modes of operation:
- **Spontaneous (S) mode.** The unit cycles between IPAP and EPAP in response to the patient rate. The difference between IPAP and EPAP is the delivered pressure support level.
- **Spontaneous/Timed (S/T) mode.** The unit cycles as in the S mode. In addition, if the patient fails to initiate an inspiration within the timed interval, the unit will cycle to IPAP based on the Breaths Per Minute (BPM) control setting.
- **Timed (T) mode.** The unit cycles between IPAP and EPAP levels based solely on set BPM and % IPAP Time Controls.
- **Continuous Positive Airway Pressure (CPAP) mode.** Allows system to be used for CPAP delivery.

5 **IPAP control** sets prescribed inspiratory pressure level. Range: 4 cm H_2O to 20 cm H_2O.

6 **EPAP control** sets prescribed PEEP level. Range: 4 cm H_2O to 20 cm H_2O.

7 **Breaths Per Minute (BPM) control** sets the number of breaths per minute. Range: 4 to 30 breaths per minute. Active in S/T and T modes.

8 **% IPAP Time control** works in conjunction with the BPM control and determines the fraction of the respiratory cycle spent in IPAP. Range: 10% to 90%. Active only in T mode.

9 **Isolated DC analog outputs** (on side) for recorder include:
- Estimated Tidal Volume
- Pressure
- Total Flow
- Estimated Patient Flow

Figure 2-21 BiPAP System nasal mask in place on patient. *(Courtesy of Respironics Inc., Murrysville, PA.)*

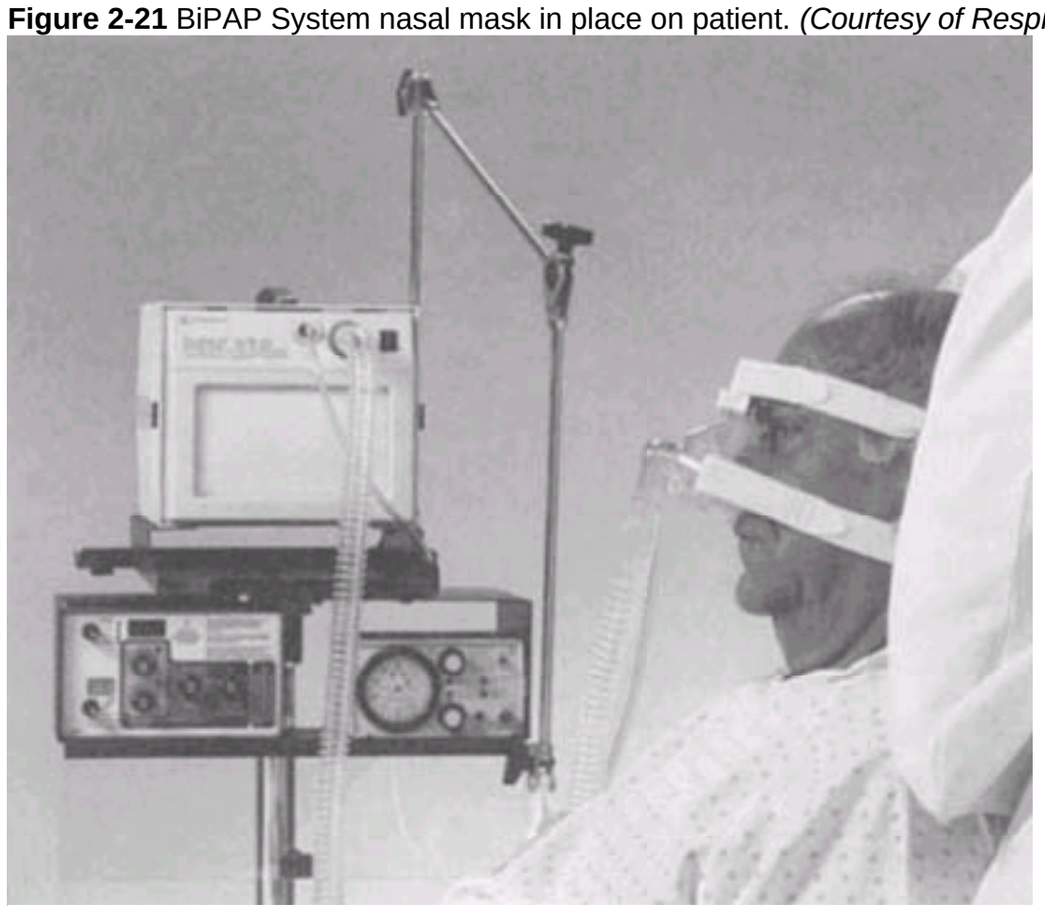


Figure 3-1 Nonsurgical airway management algorithm.

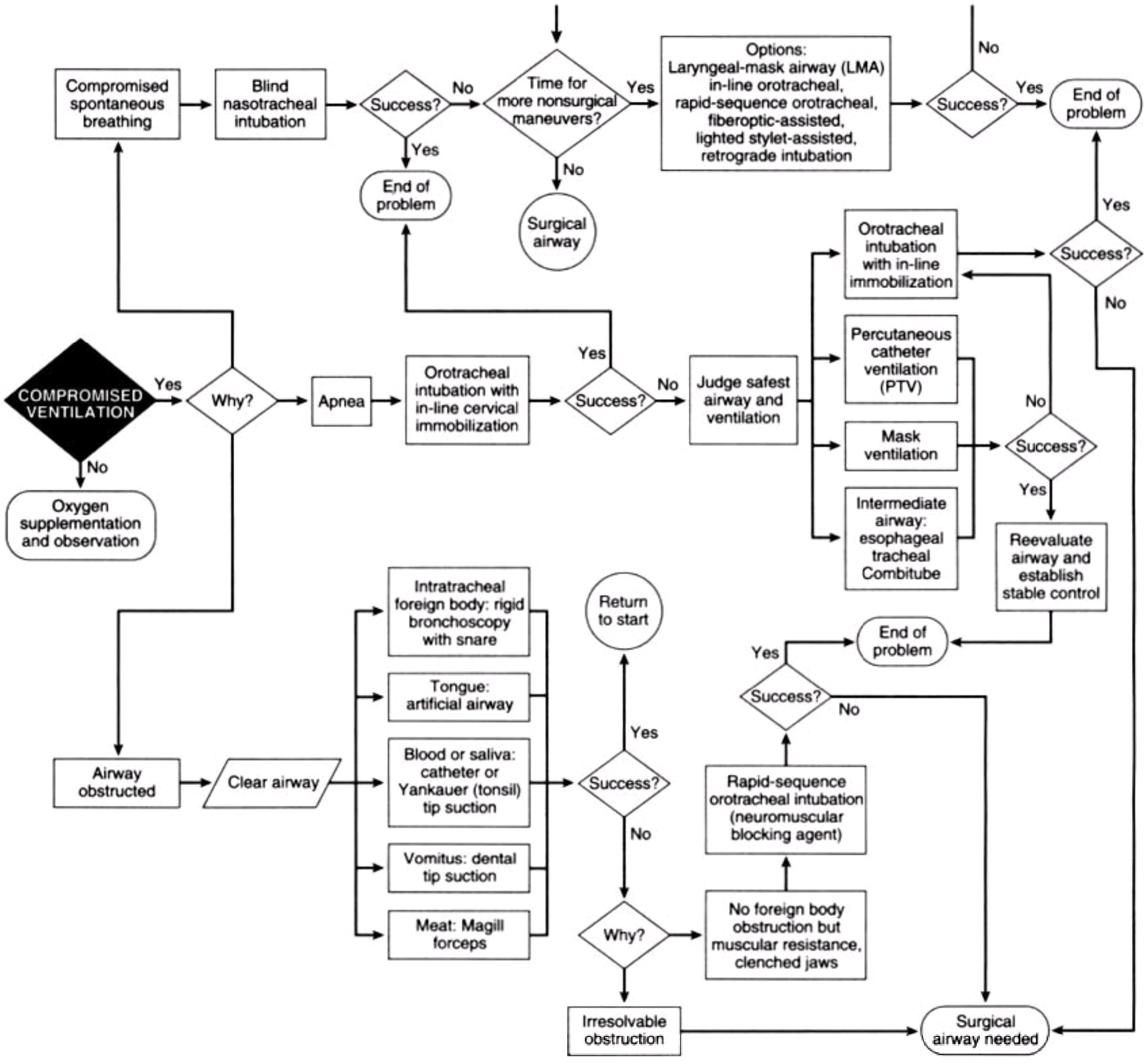


Figure 3-2 Surgical airway management algorithm.

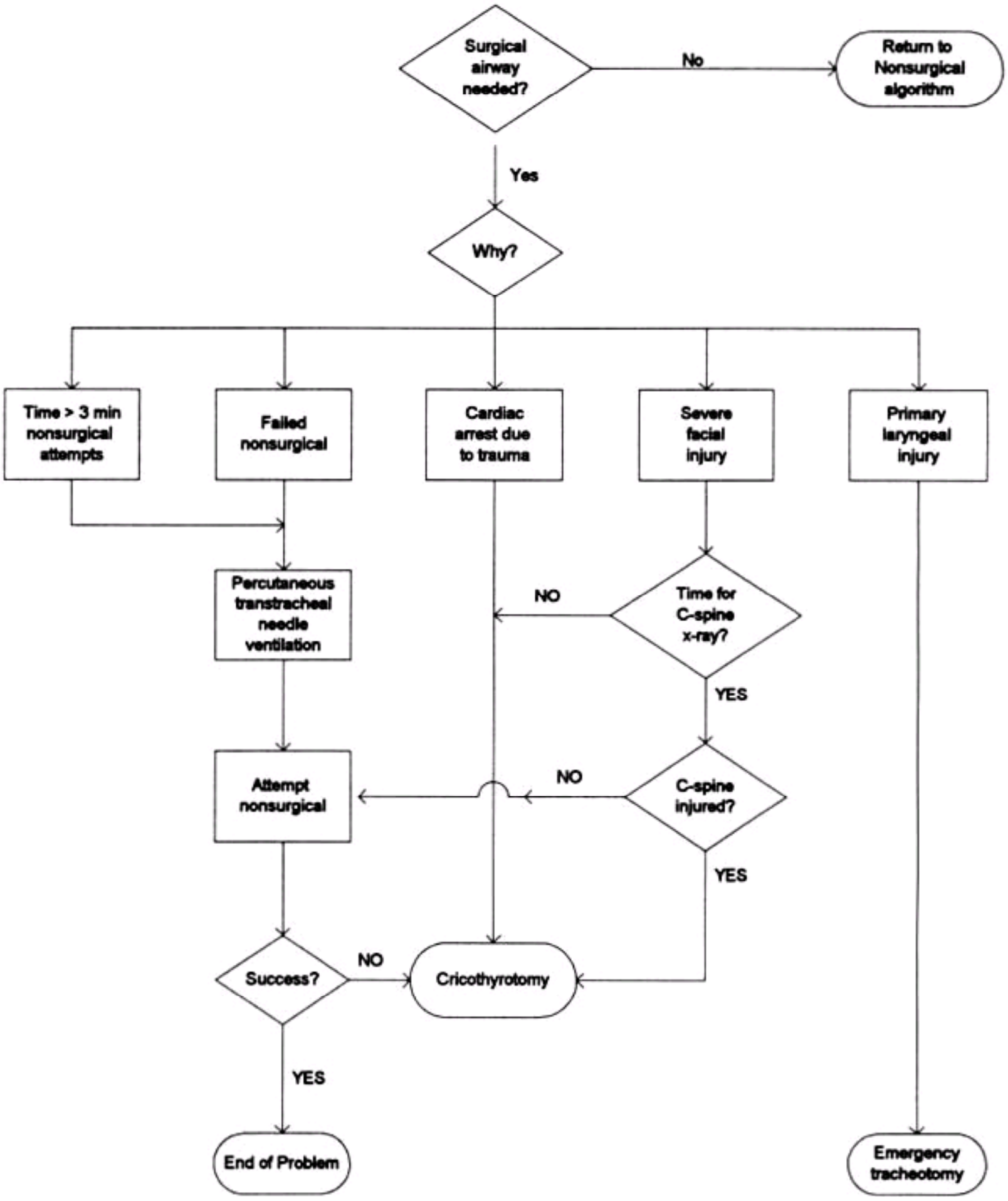


Figure 3-3 Illustration of maneuvers for opening the airway. *A,* Neck lift. *B,* Chin lift. *C,* Jaw thrust. *(From Guildner CW: Resuscitation—opening the airway: A comparative study of techniques for opening an airway obstructed by the tongue. JACEP 5:588, 1976. Reproduced by permission.)*

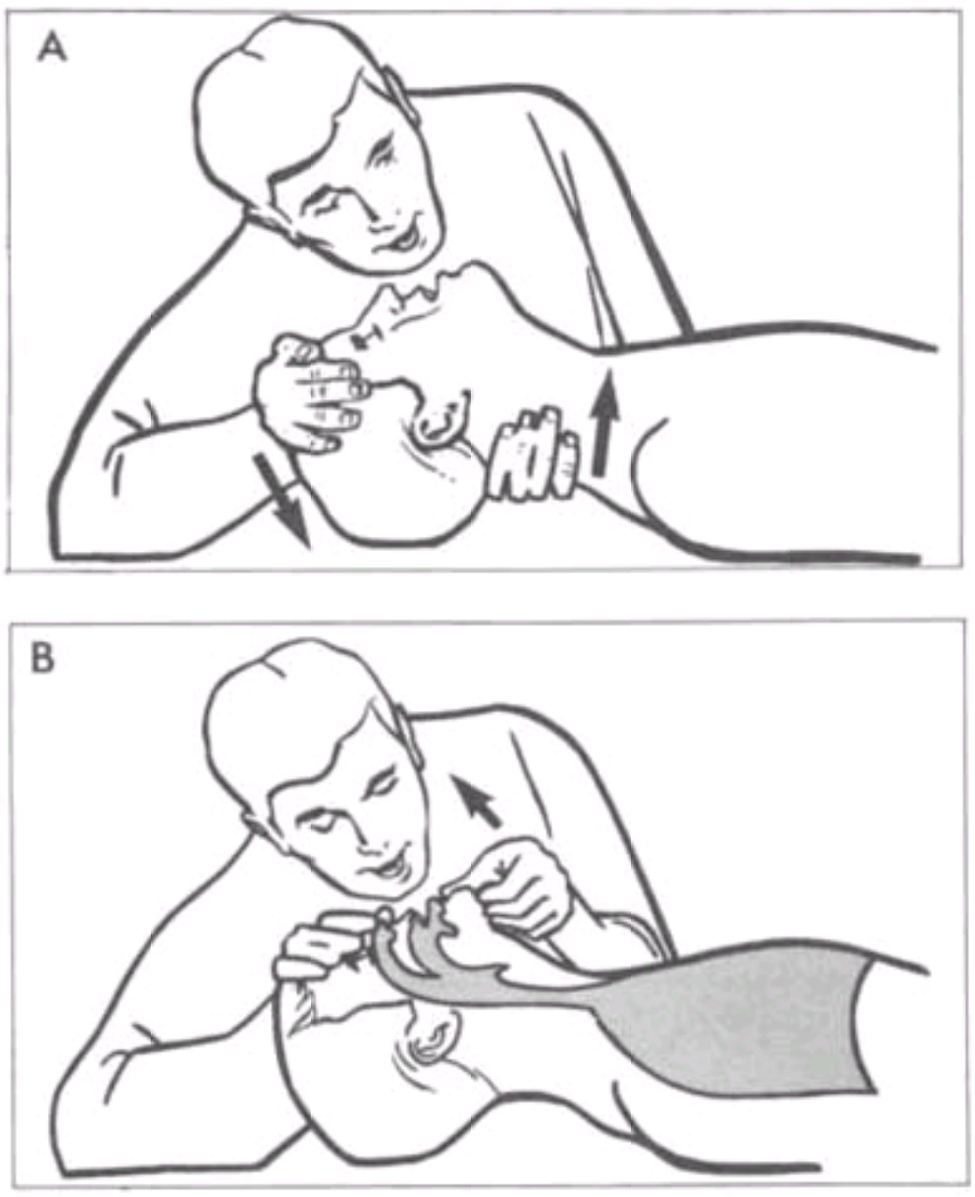


Figure 3-4 Three types of suction tips: dental, tonsil, and catheter. *(From Clinton JE, Ruiz E: Trauma Life Support Manual. Minneapolis, MN, Hennepin County Medical Center, 1982.)*

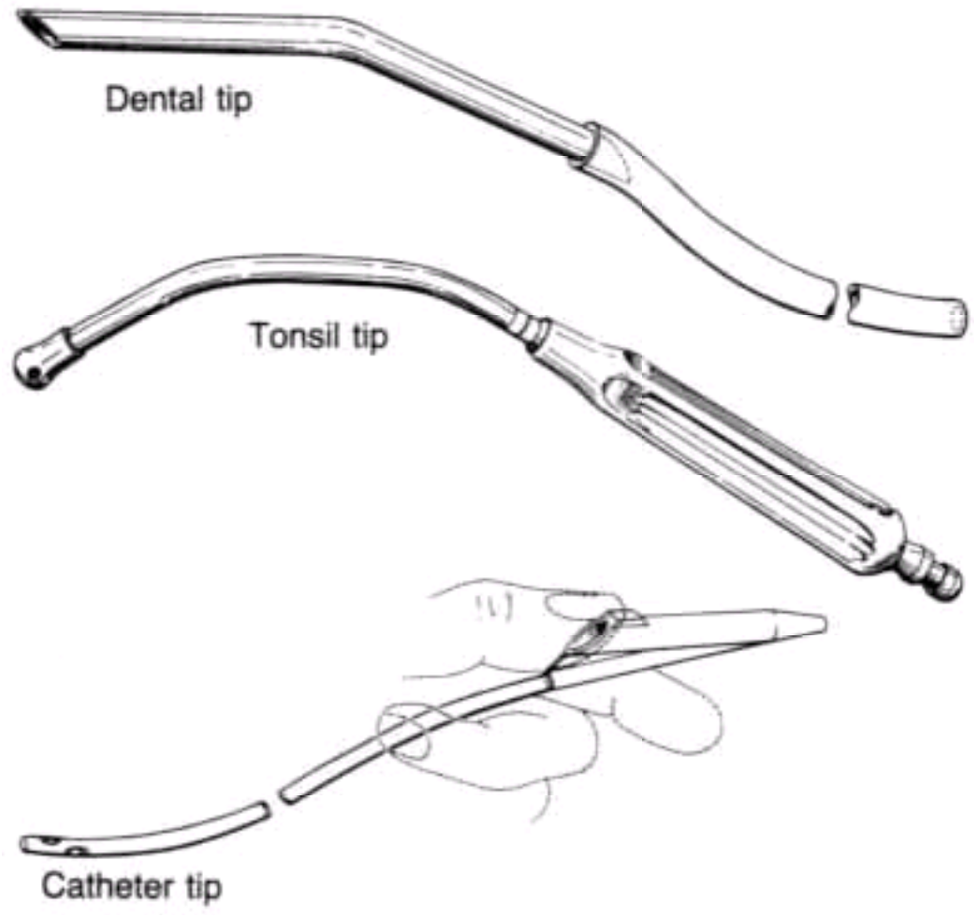


Figure 3-5 The Ruben suction booster, which is designed to allow high-capacity suctioning through the endotracheal tube during intubation. Schematic diagram: *A,* Tracheal tube connection. *B,* Connection to suction. *C,* Introducer opening in the closed position. *D,* Opening that is kept closed when suction is needed through the tracheal tube. Note: All suction should be done under direct vision. *(From Ruben H, Hansen E, MacNaughton FI: High-capacity suction technique. Anaesthesia 34:349, 1979. Reproduced by permission.)*

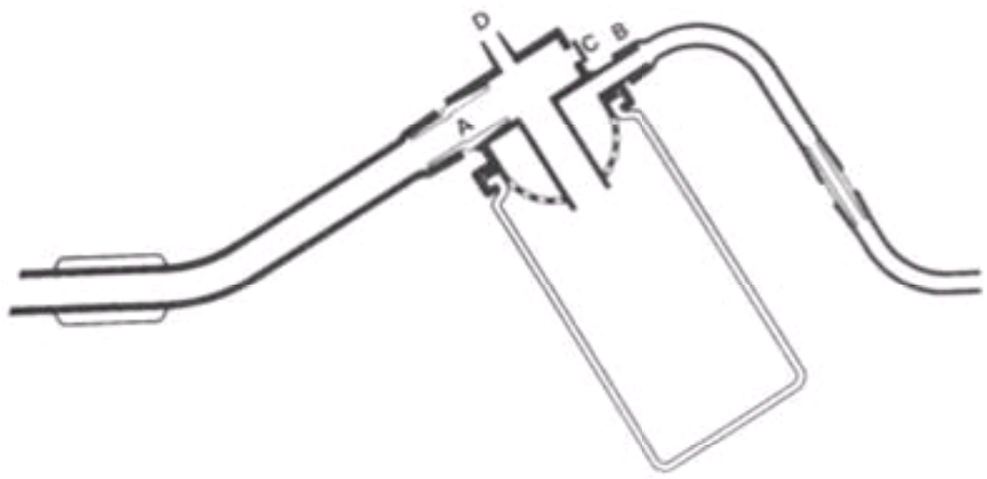


Figure 3-6 Intracranial intubation. Lateral skull x-ray showing nasogastric tube placed into brain through skull fracture. *(From Clinton JE, Ruiz E: Trauma Life Support Manual. Minneapolis, MN, Hennepin County Medical Center, 1982.)*

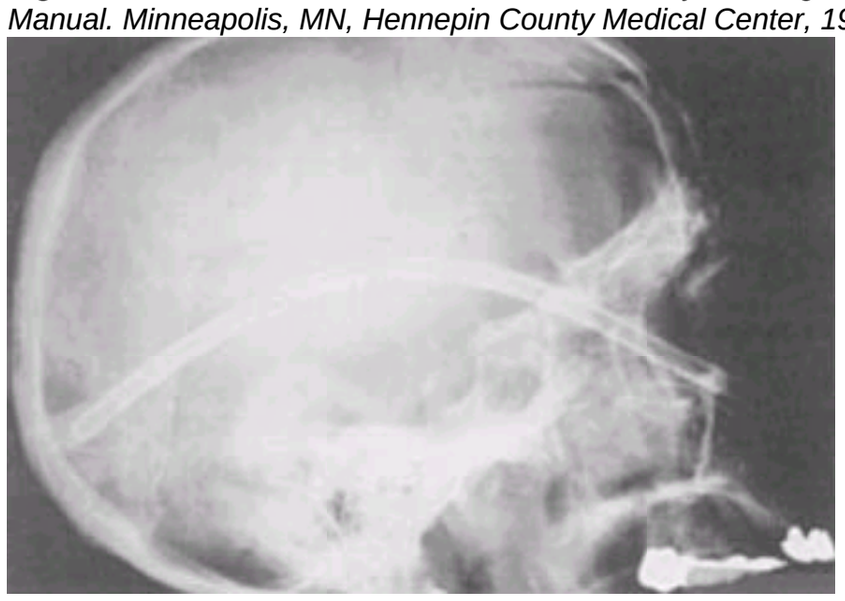


Figure 3-7 Simple artificial airways: oropharyngeal and nasopharyngeal. *(From Clinton JE, Ruiz E: Trauma Life Support Manual. Minneapolis, MN, Hennepin County Medical Center, 1982.)*

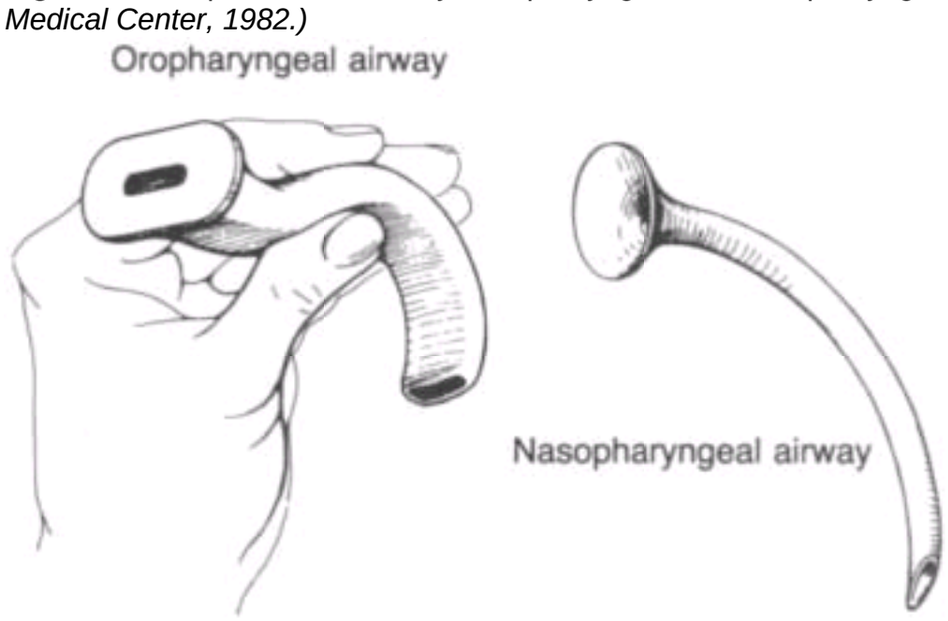


Figure 3-8 Some clinicians place a nasopharyngeal tube to dilate the nasal passage prior to blind nasotrachael intubation.

Figure 3-9 Bag-valve-mask ventilation is very difficult for one person to do *(A),* and it frequently fails to deliver adequate tidal volumes, especially during cardiopulmonary resuscitation. With the two-person method *(B),* one person uses both hands to hold the mask firmly against the face and extend the head. The other person uses both hands to squeeze the bag. Dentures are generally left in place to help provide a better-fitting mask. *(From Jesudian MC, Harrison RR, Keenan RL, Maull KI: Bag-valve-mask ventilation: Two rescuers are better than one. Crit Care Med 13:122, 1985.)*

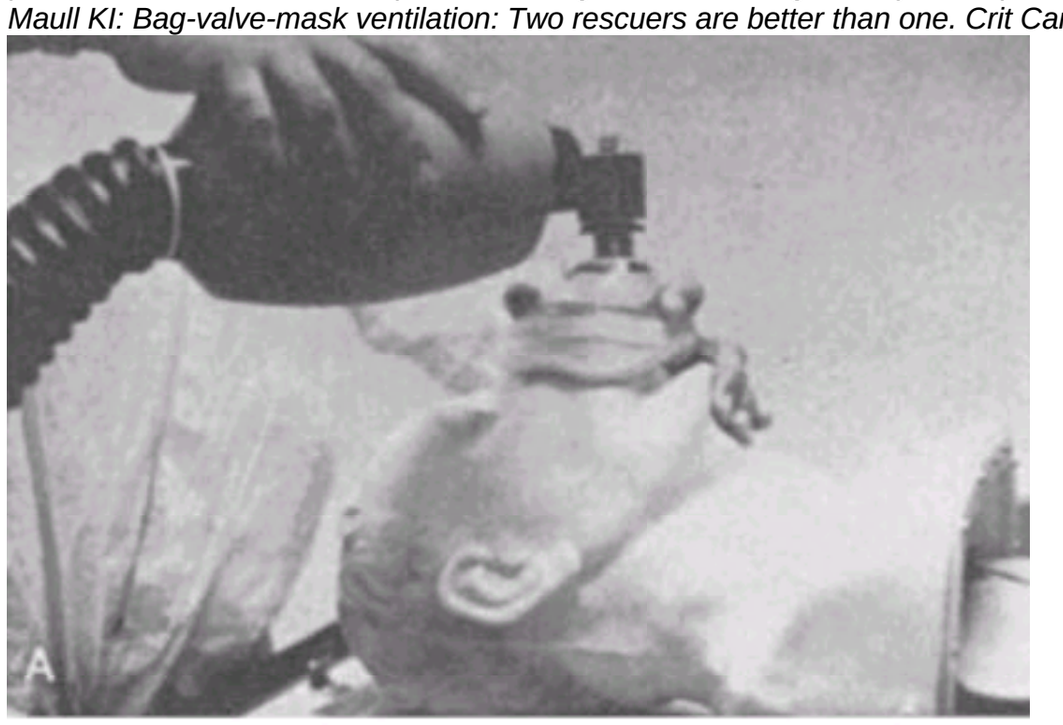
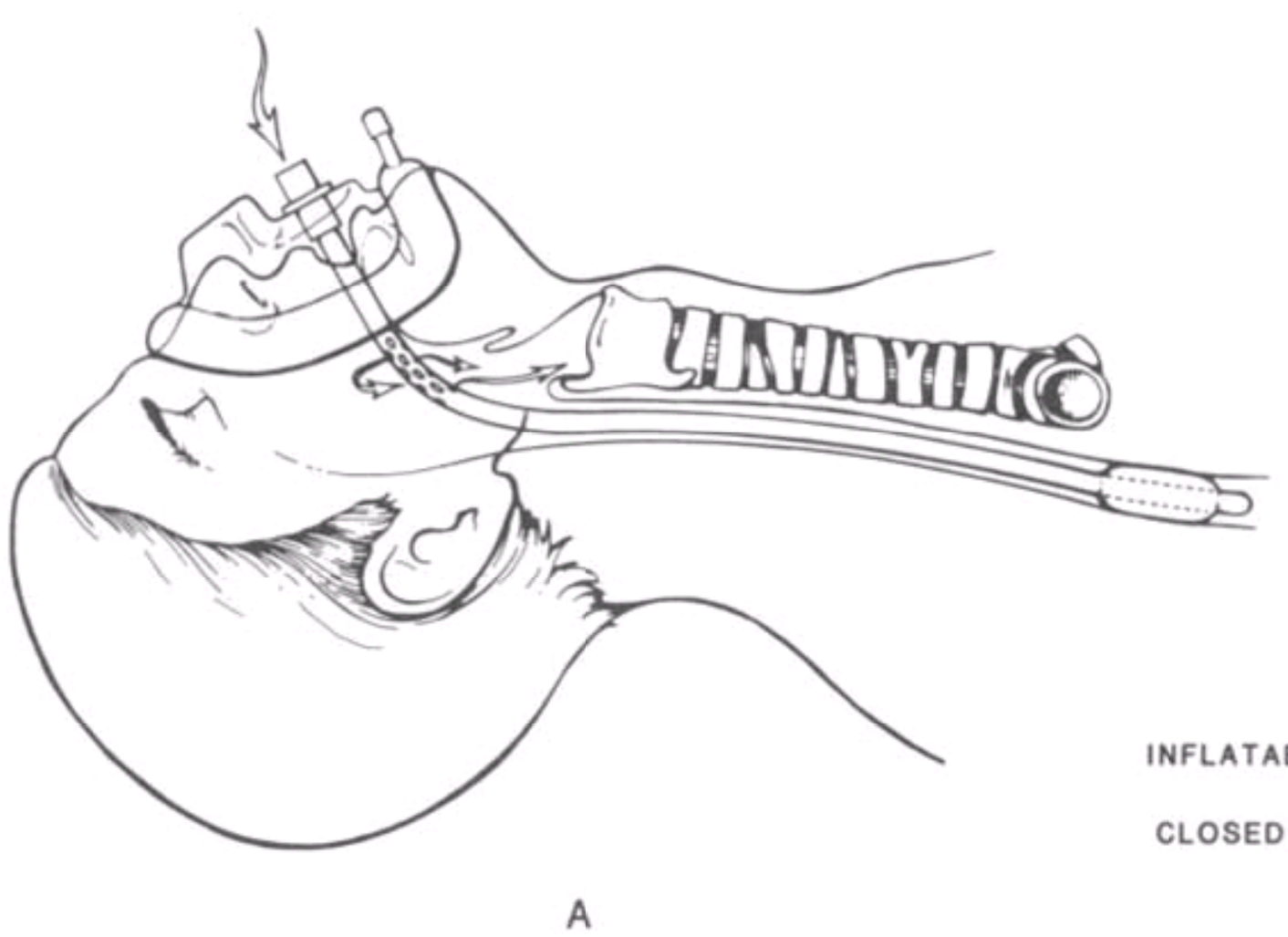
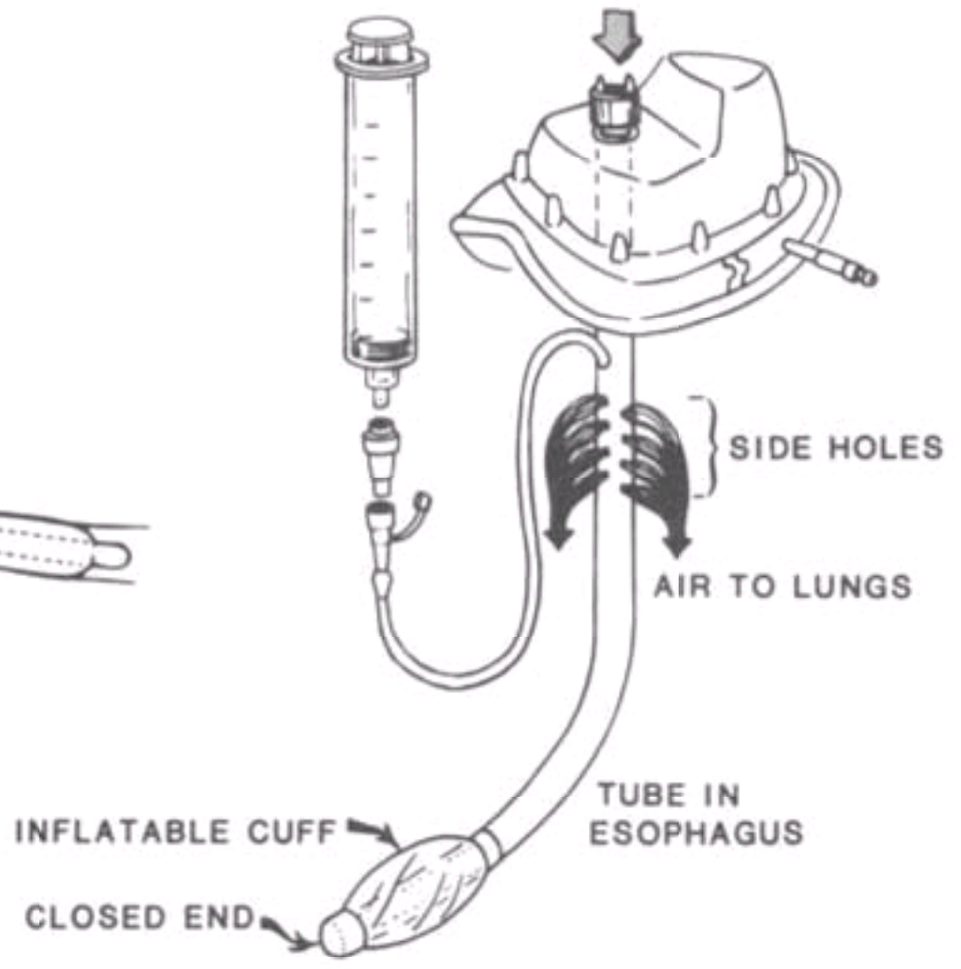


Figure 3-10 *A,* Esophageal obturator airway. Correct placement of the esophageal airway with the cuff inflated in the esophagus caudad to the bifurcation of the trachea. *(From Clinton JE, Ruiz E: Trauma Life Support Manual. Minneapolis, MN, Hennepin County Medical Center, 1982.) B,* Esophageal obturator airway. *(From Jacobs LM: The importance of airway management in trauma. J Natl Med Assoc 80:873, 1988.)*

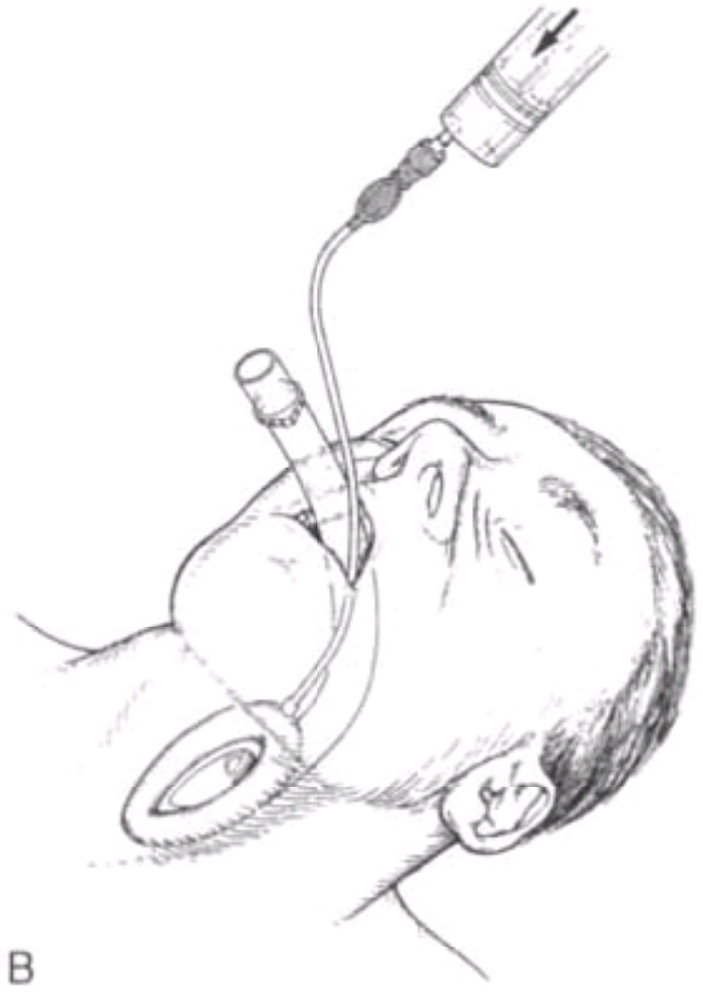


A



B

Figure 3-11 Laryngeal mask airway (LMA). *A*, inflated LMA outside of the body. *B*, LMA in place with cuff overlying larynx. *C*, LMA placement into the pharynx. *D*, LMA placement using the index finger as a guide. *(From Basket PJF, Brain AIJ: The use of the LMA. In Basket PJF, Brain AIJ (eds): Cardiopulmonary Resuscitation Handbook. London, Intavent Research, 1994.)*



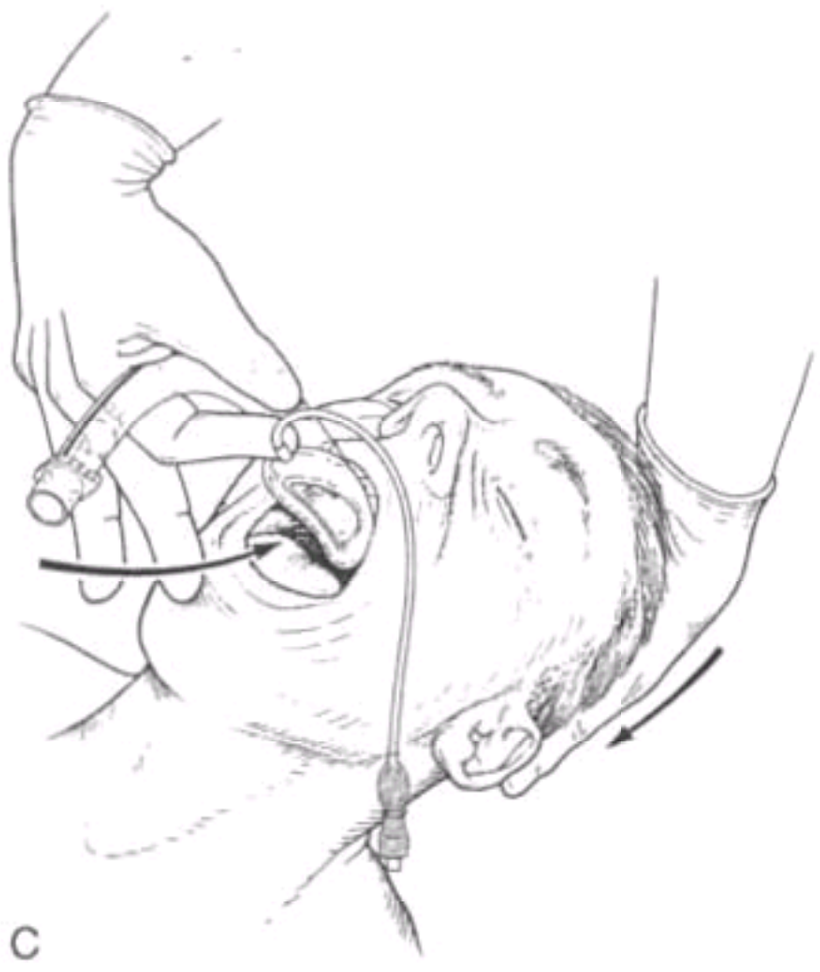


Figure 3-12 Blind passage of an endotracheal tube through the laryngeal mask airway. *(Courtesy of the Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

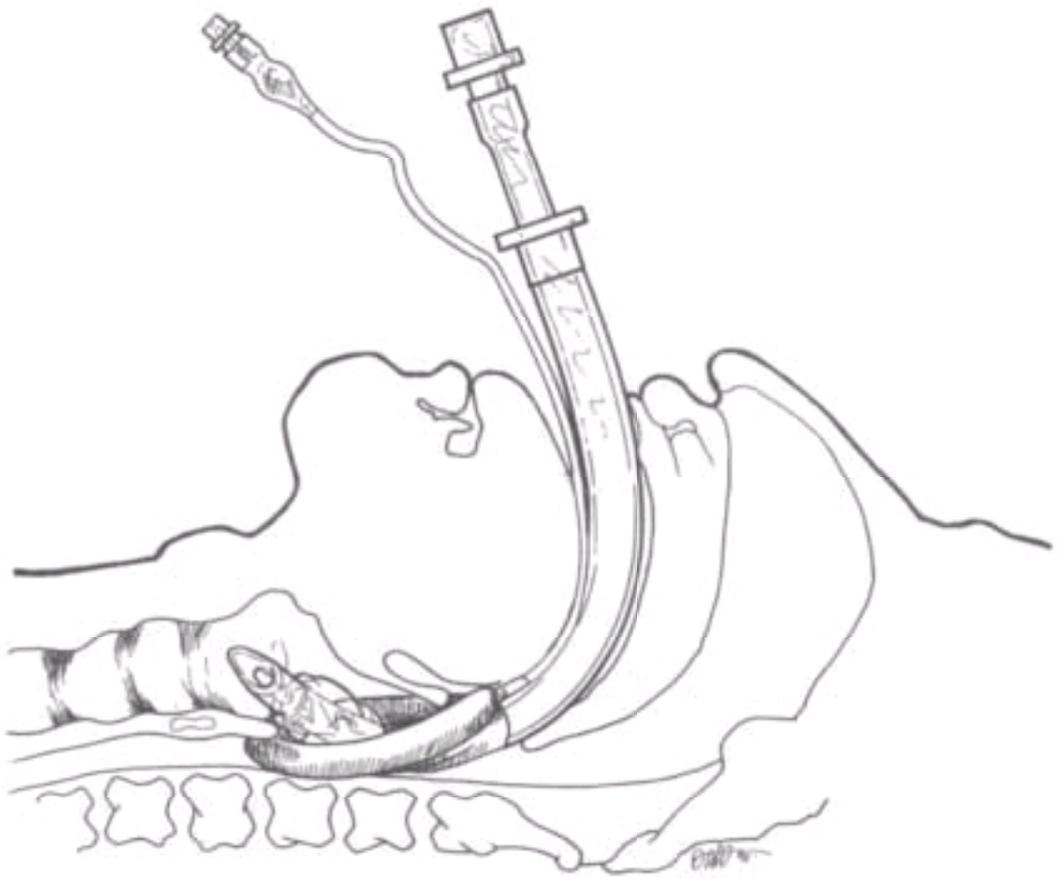


Figure 3-13 Esophageal-tracheal Combitube (ETC) airway. *(From Frass M, Frenzer R, Zdrahal F, et al: The esophageal tracheal Combitube: Preliminary results with a new airway for cardiopulmonary resuscitation. Ann Emerg Med 16:770, 1987.)*

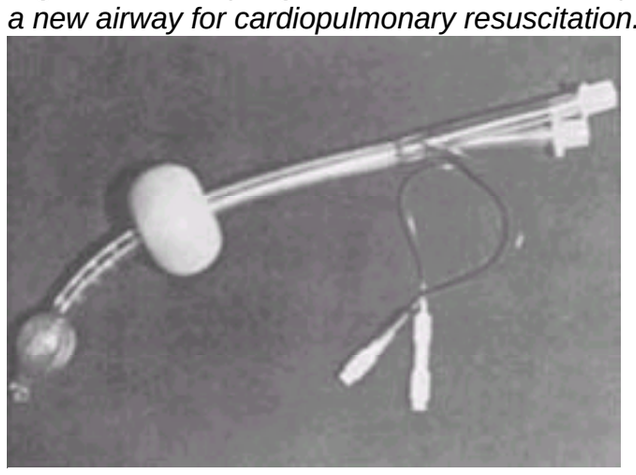


Figure 4-1 Adult airway cart. Equipment and materials are visible, labeled, and accessible. *(Concept and design by Dr. Ernest Ruiz, Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

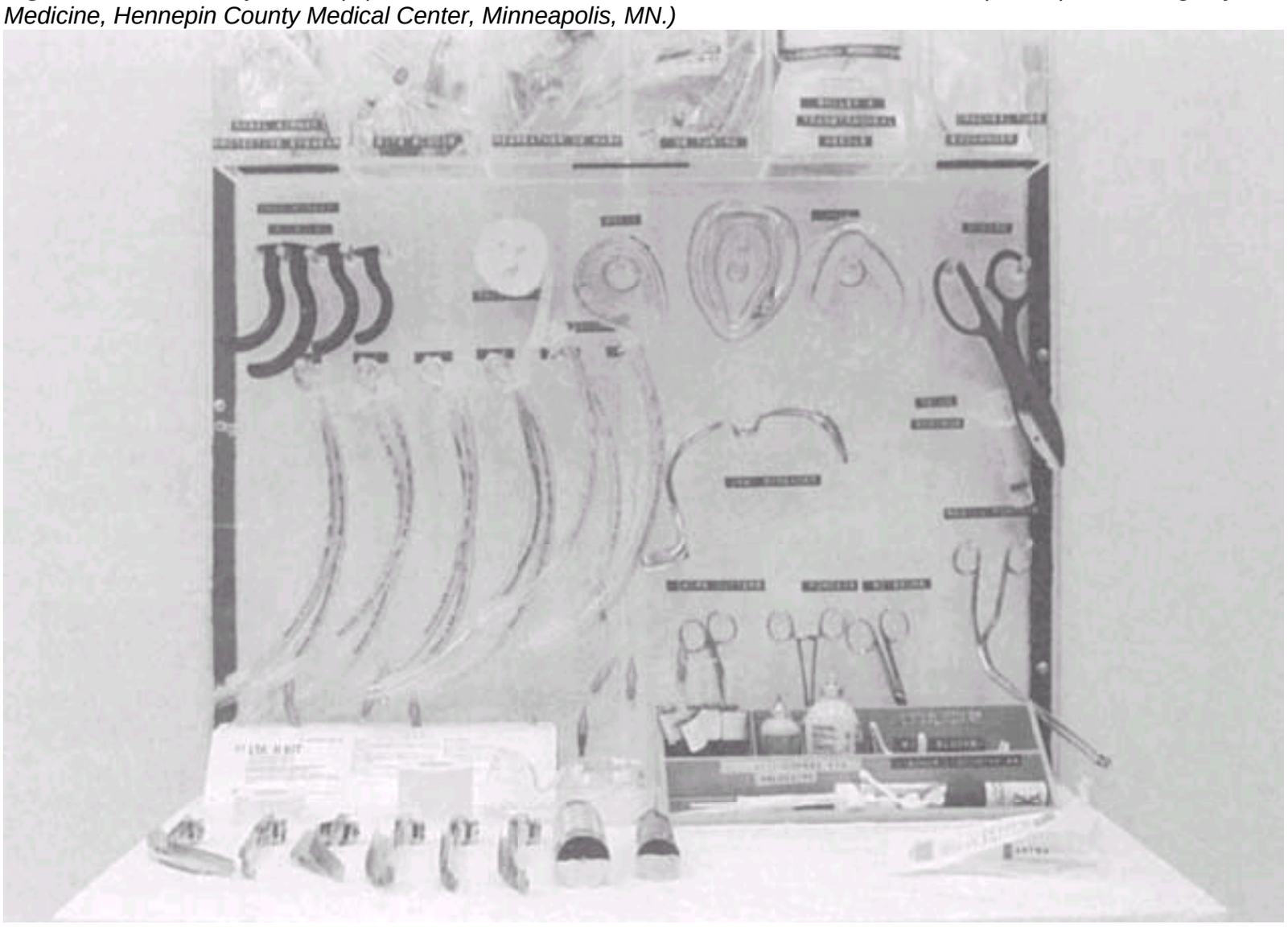


Figure 4-2 *A*, Lateral (sagittal) view of the upper airway. *(A from Snell RS, Smith MS (eds): Clinical Anatomy for Emergency Medicine. St Louis, Mosby-Year Book, 1993, p 16.) B*, View of larynx, epiglottis, and vocal cords as seen with laryngoscope (not shown) in place.

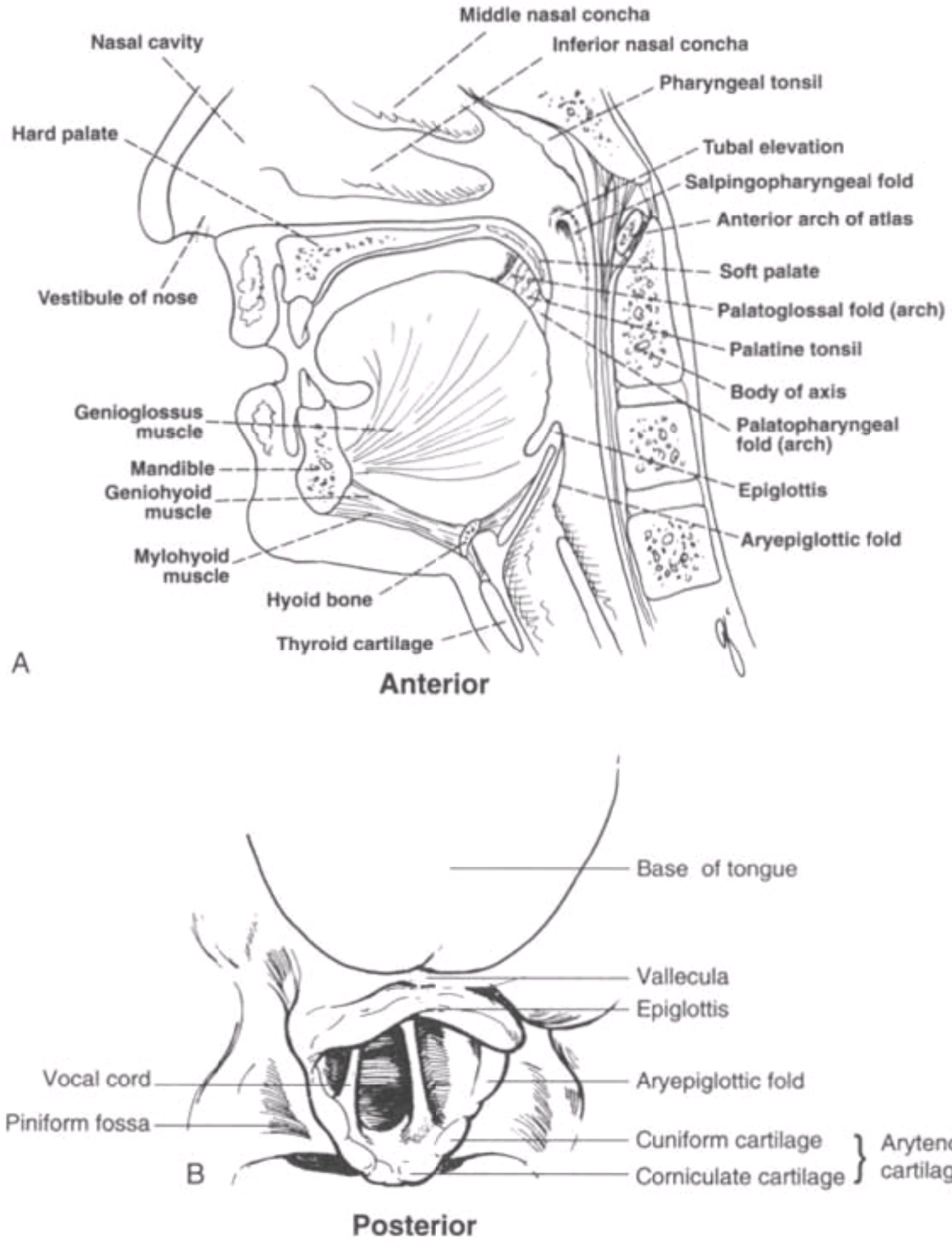


Figure 4-3 Pediatric endotracheal tube size estimation using fingernail width of the 5th finger.

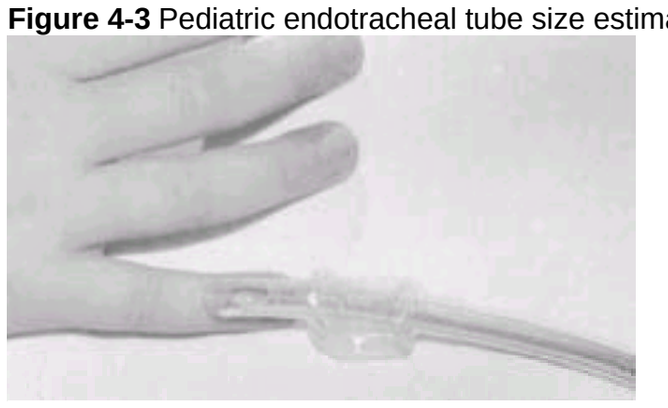


Figure 4-4 Head positioning for tracheal intubation. *A,* Neutral position. *B,* Head elevated. *C,* "Sniffing" position, with flexed neck and extended head. Note how flexing the neck and extending the head to line up the various axes allows for intubation. This position creates the shortest distance and straightest line between the teeth and vocal cords.

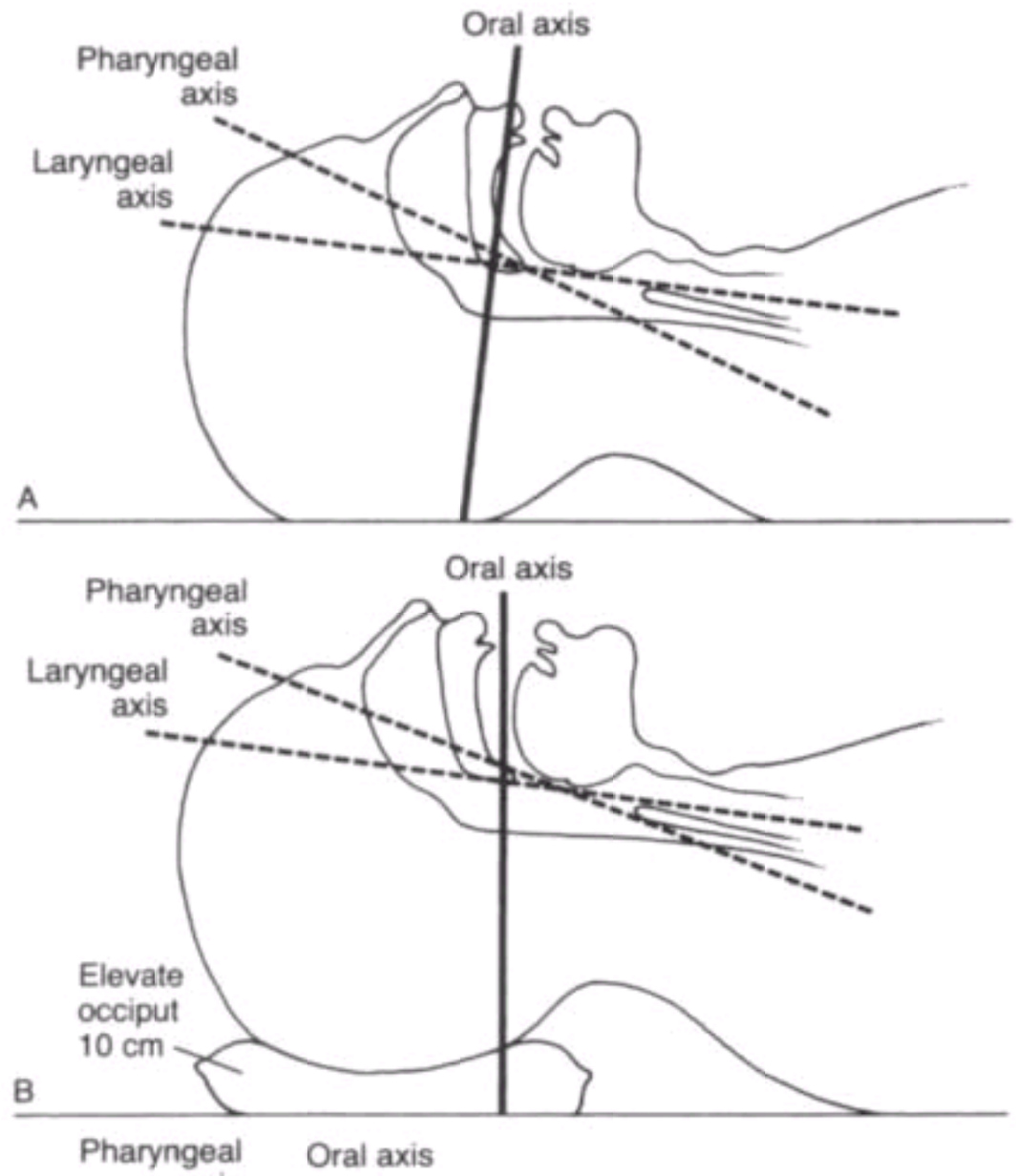
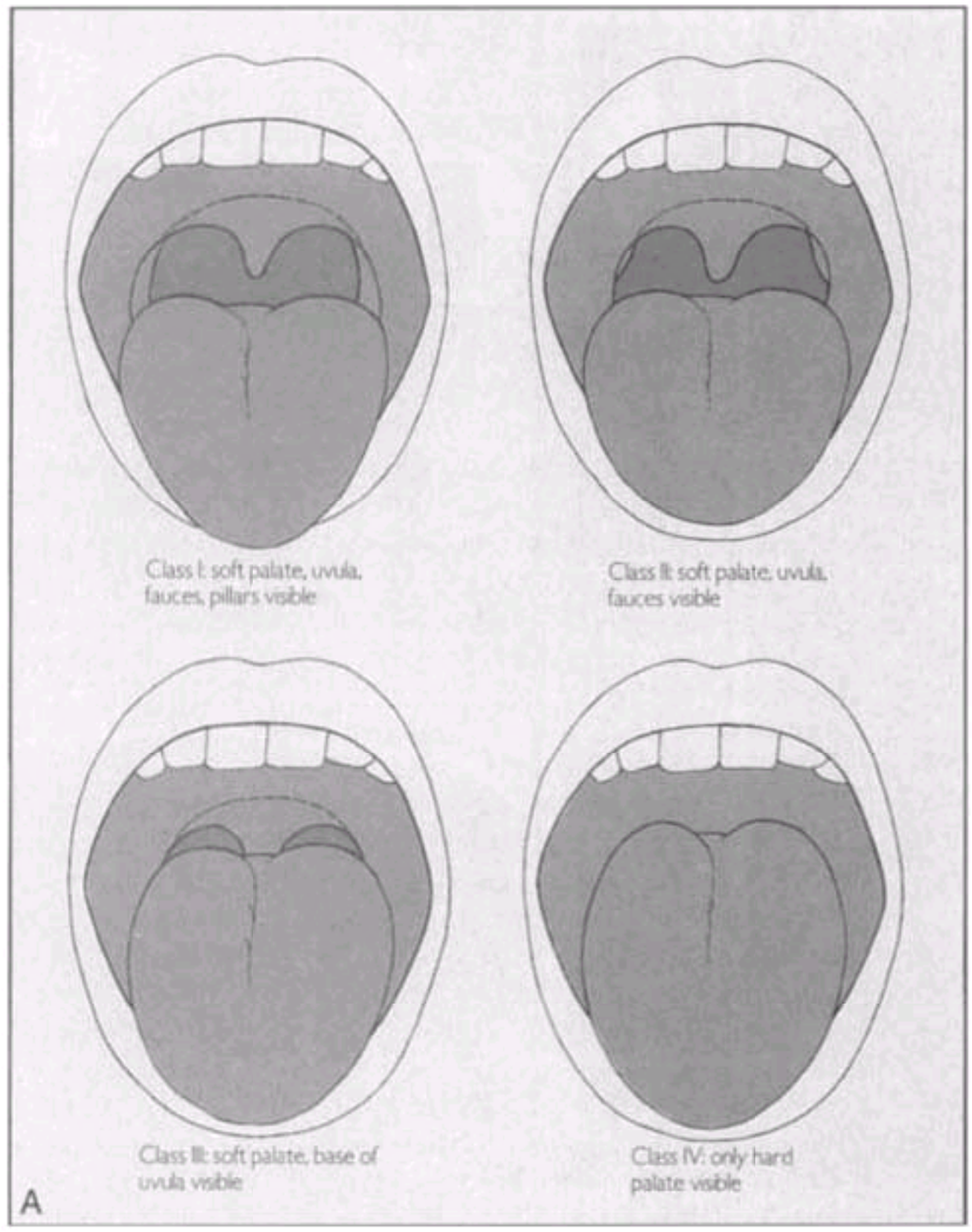


Figure 4-5 *A*, Mallampati classification predicting difficulty of intubation based on visibility of intraoral structures: Intubation should pose no difficulty in patient in class I or II, moderate difficulty in class III, and severe difficulty in class IV. *(A from Whitten CE: Preintubation evaluation: Predicting the difficult airway. Emerg Med 21:111, 1989.) B*, A difficult intubation also may be expected if the distance from the mentum of the chin to the hyoid bone in an adult is less than three fingerbreadths when the head is extended. Patients with a shortened distance often have a hypoplastic or poorly developed mandible or a short, thick neck, resulting in an anterior larynx that may be best approached with the straight blade and cricoid pressure.



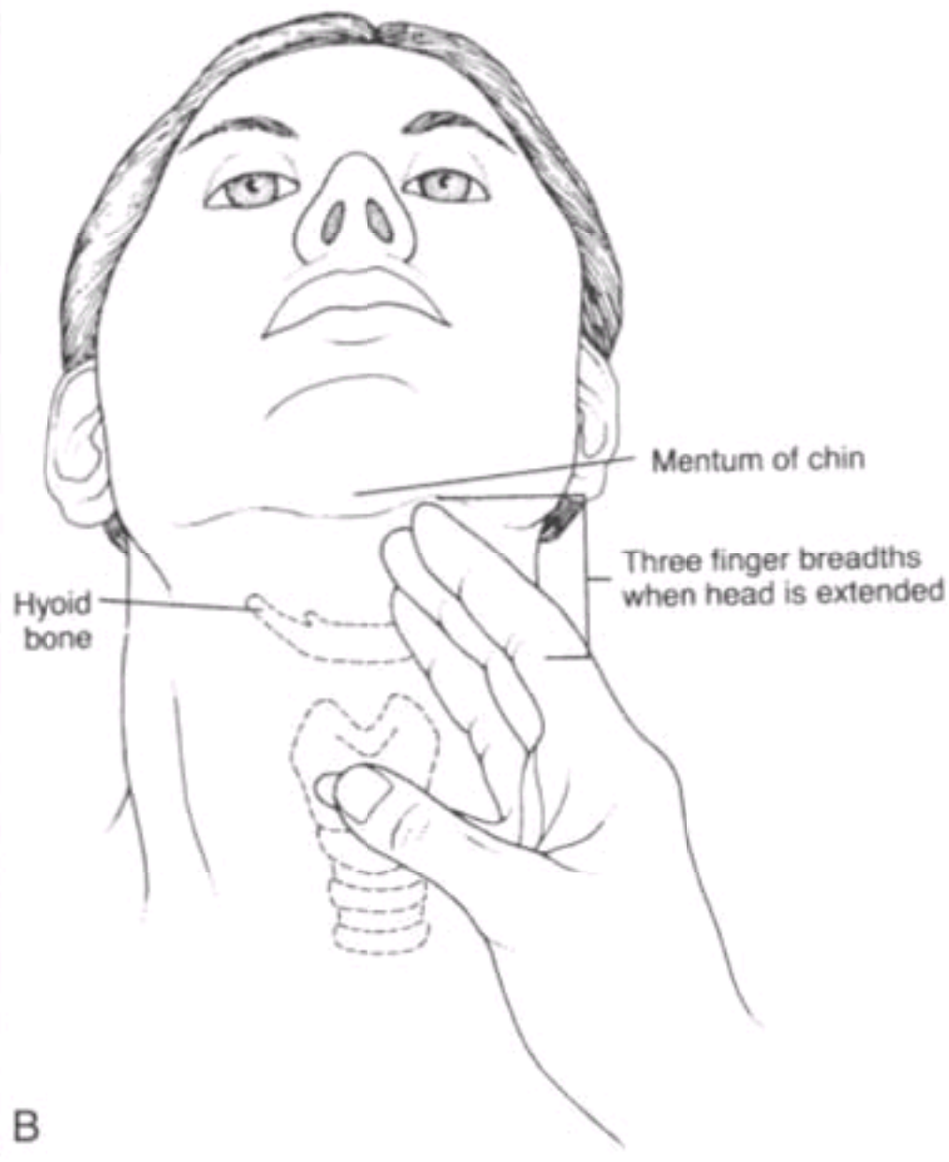


Figure 4-6 Proper positioning of clinician, patient, and assistant for tracheal intubation.

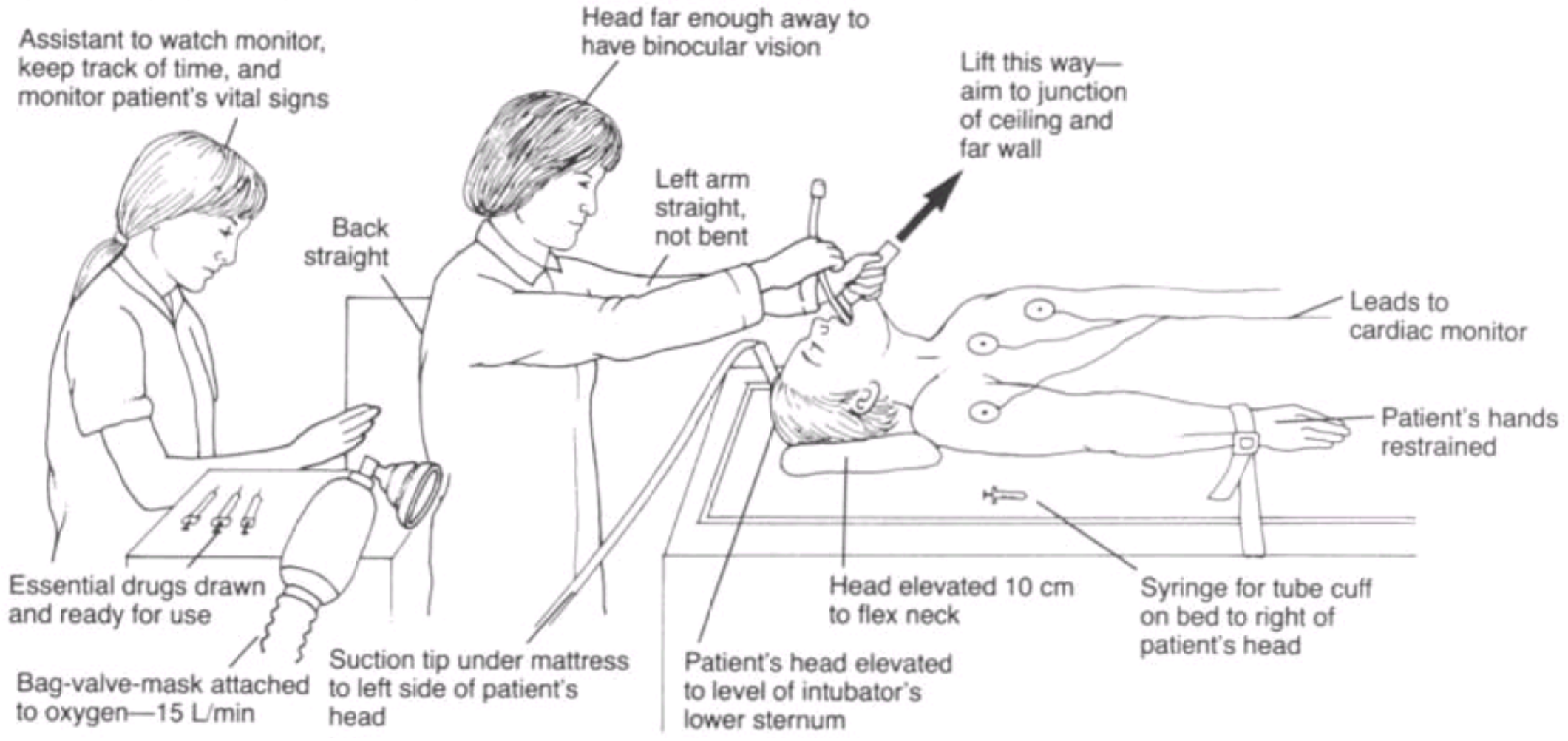
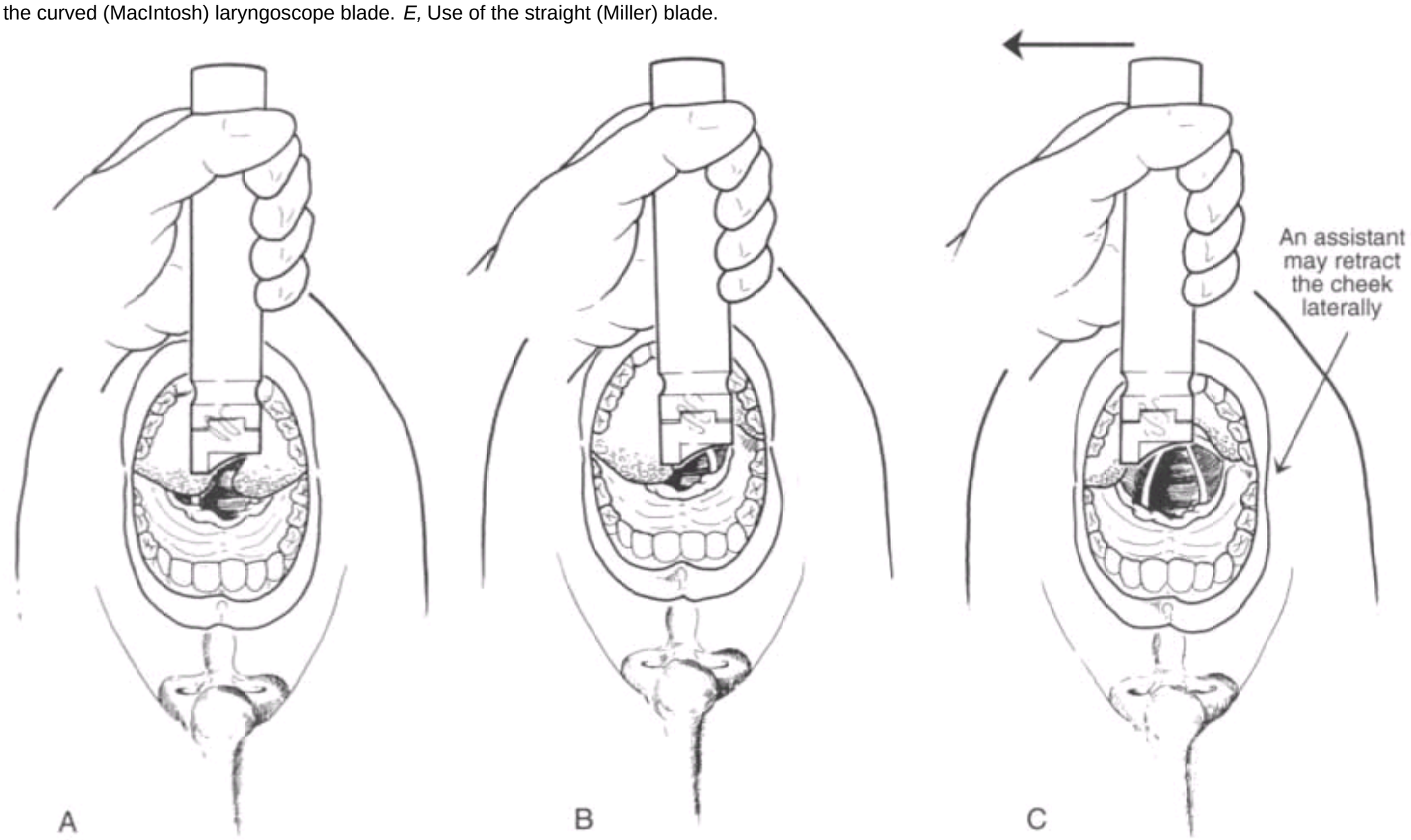


Figure 4-7 Common problems encountered with the laryngoscope: *A,* Laryngoscope blade under the middle of the tongue, with the sides of the tongue hanging down and obscuring the glottis. *B,* Tongue not pushed far enough to the left, obscuring the glottis. *C*, Correct blade position with tongue elevated and to the left. *D,* Use of the curved (MacIntosh) laryngoscope blade. *E,* Use of the straight (Miller) blade.



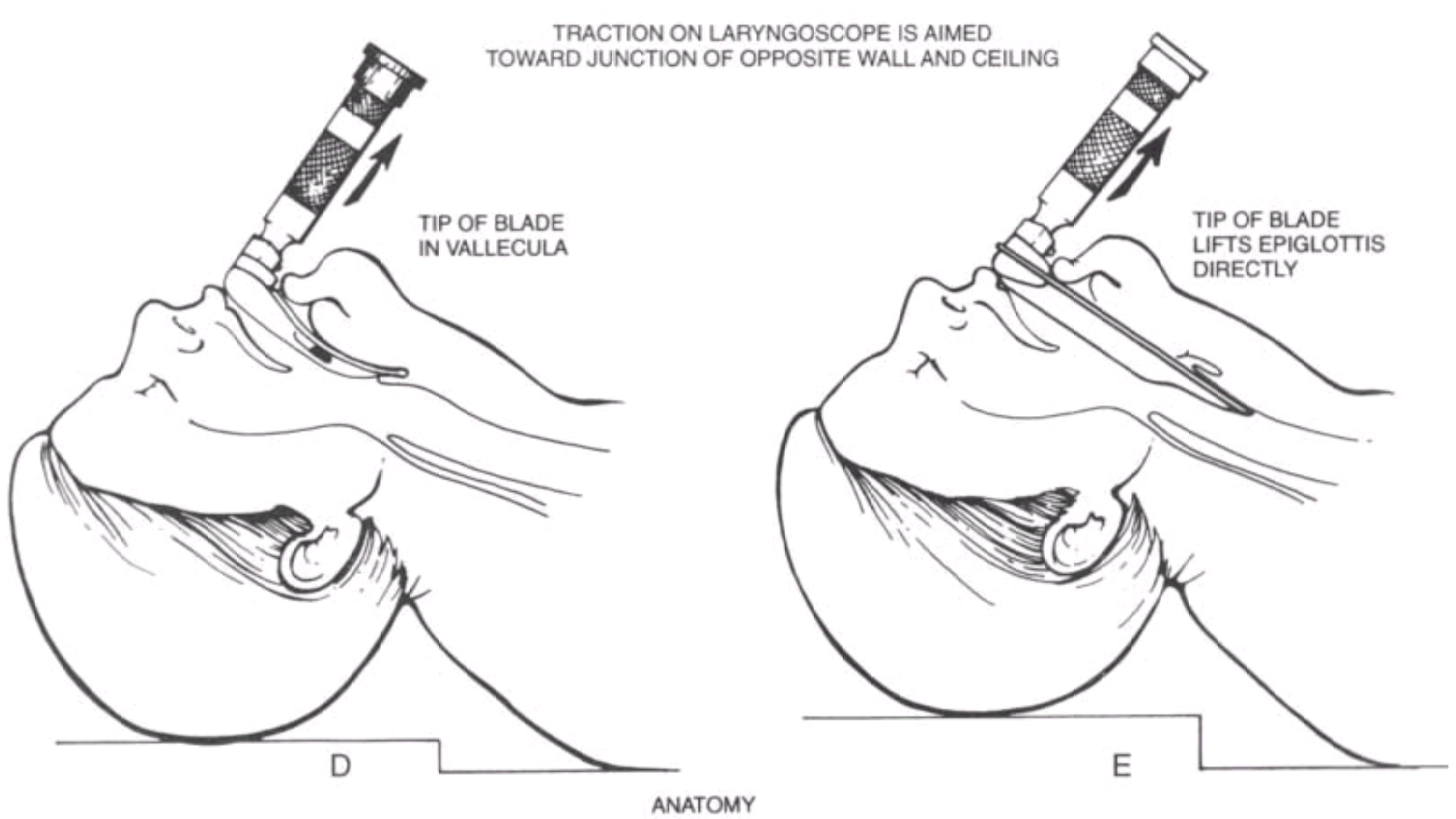


Figure 4-8 The epiglottis is elevated and visualization of the vocal cords is improved by pressure on the hypoepiglottic ligament and external laryngeal manipulation. *(Courtesy of Richard M. Levitan, MD. Airway Cam Technologies Inc., Wayne, PA. Used by permission.)*

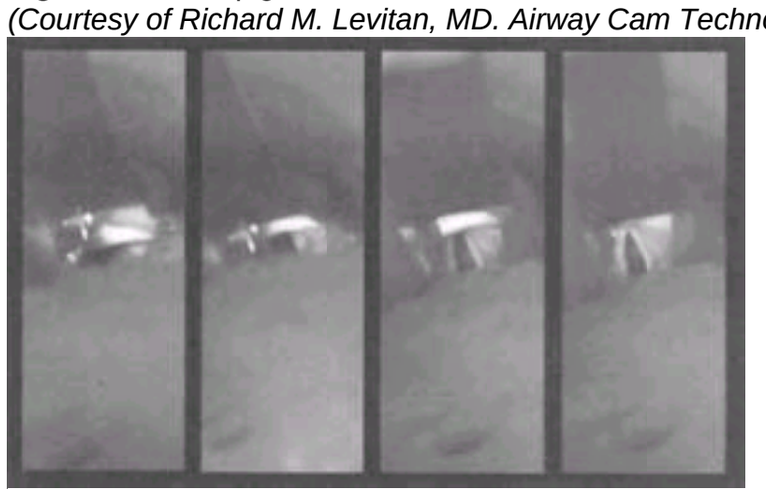


Figure 4-9 Two views (*A* and *B*) of technique for taping the tracheal tube. It is important to secure the tracheal tube properly. The method illustrated can be replaced by using a commercial holder or tracheostomy cloth tie. Avoid taping the lips.

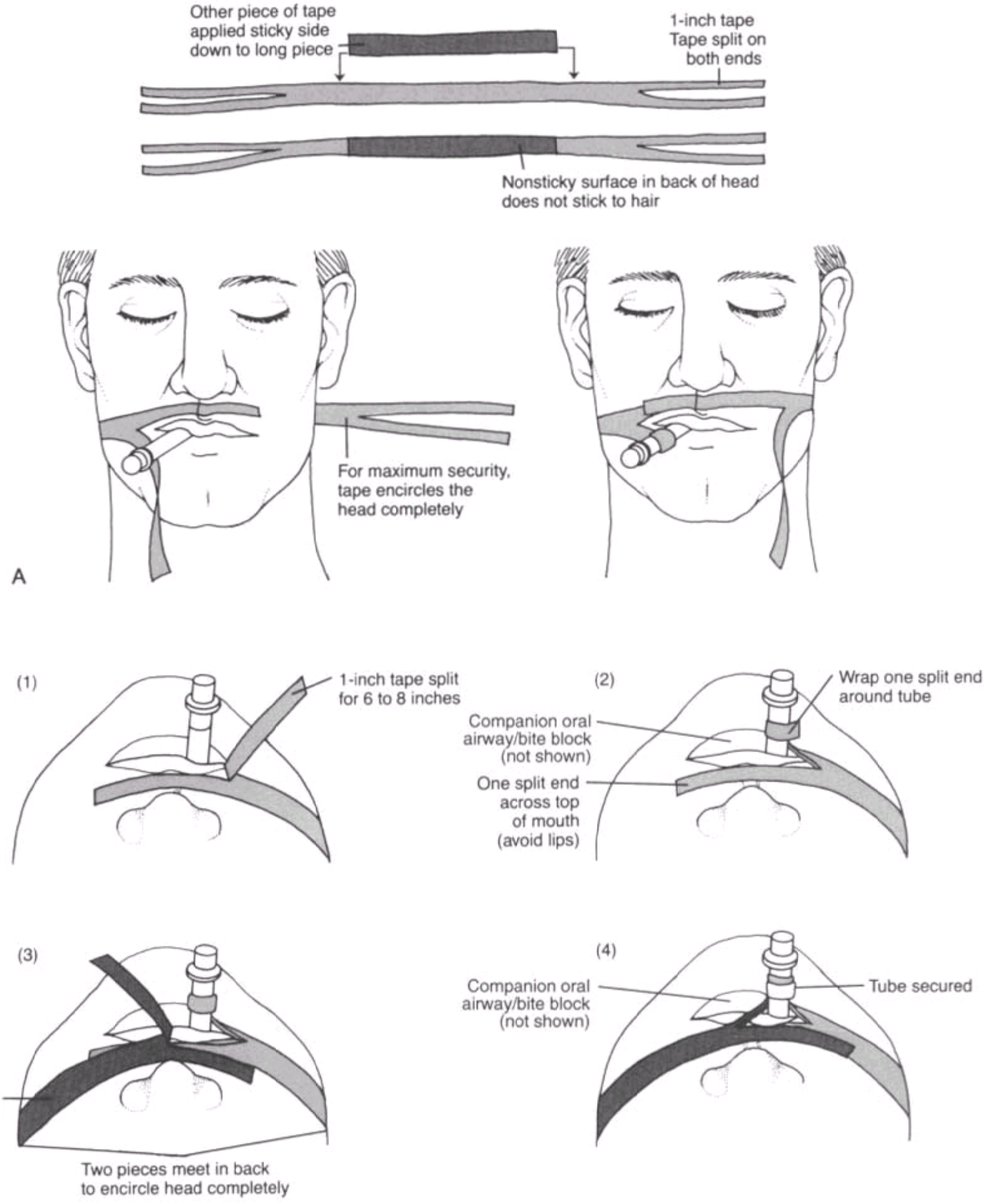


Figure 4-10 This disposable endotracheal tube holder provides a firm anchor on which to tape the tube, while also serving as a bite block. *(Courtesy of Precision Medical, Northampton, PA.)*

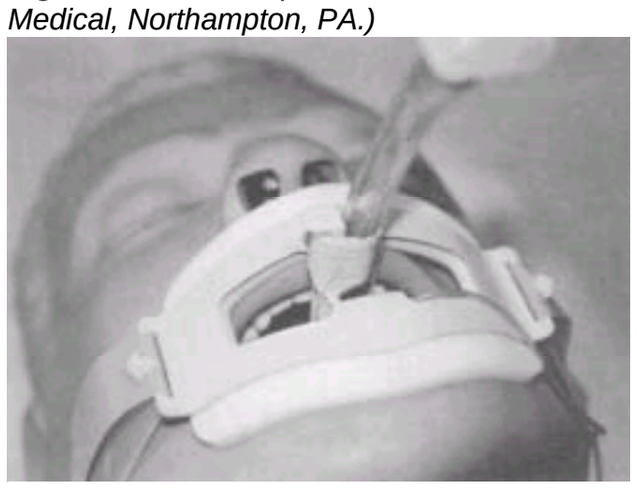


Figure 4-11 Sagittal sections of the neck of an adult (*A*) and an infant shortly after birth (*B*). Different vertebral levels in these age groups are shown. Note that in children, the neck is shorter and the larynx is located more cephalad. *(From Snell RS, Smith MS (eds): Clinical Anatomy for Emergency Medicine. St Louis, Mosby-Year Book, 1993, p 16.)*

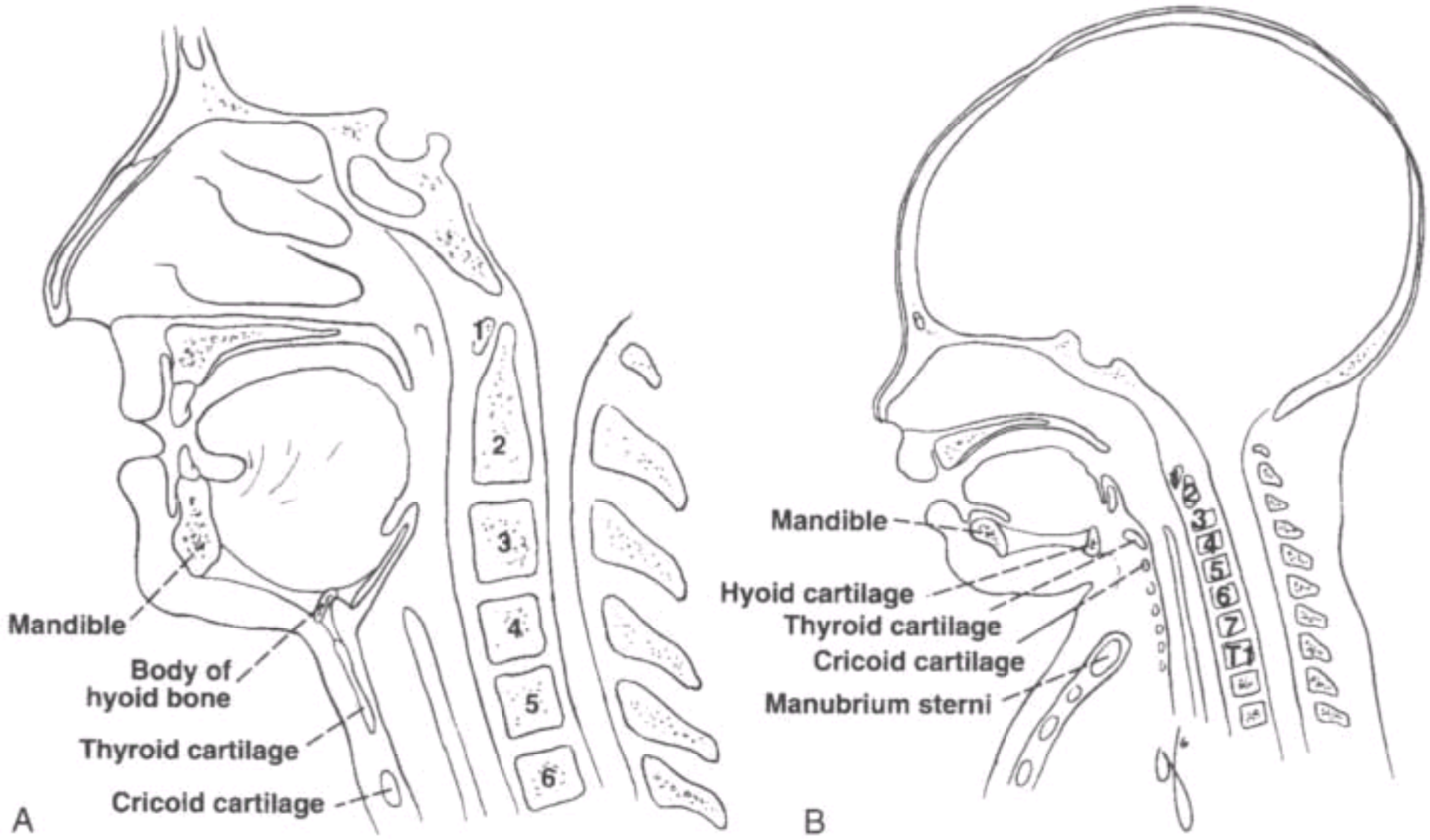


Figure 4-12 Oral intubation in a child, using a straight blade. The proportionately large, floppy head of the child may present some difficulty, and an assistant may be required to hold the child's head straight.

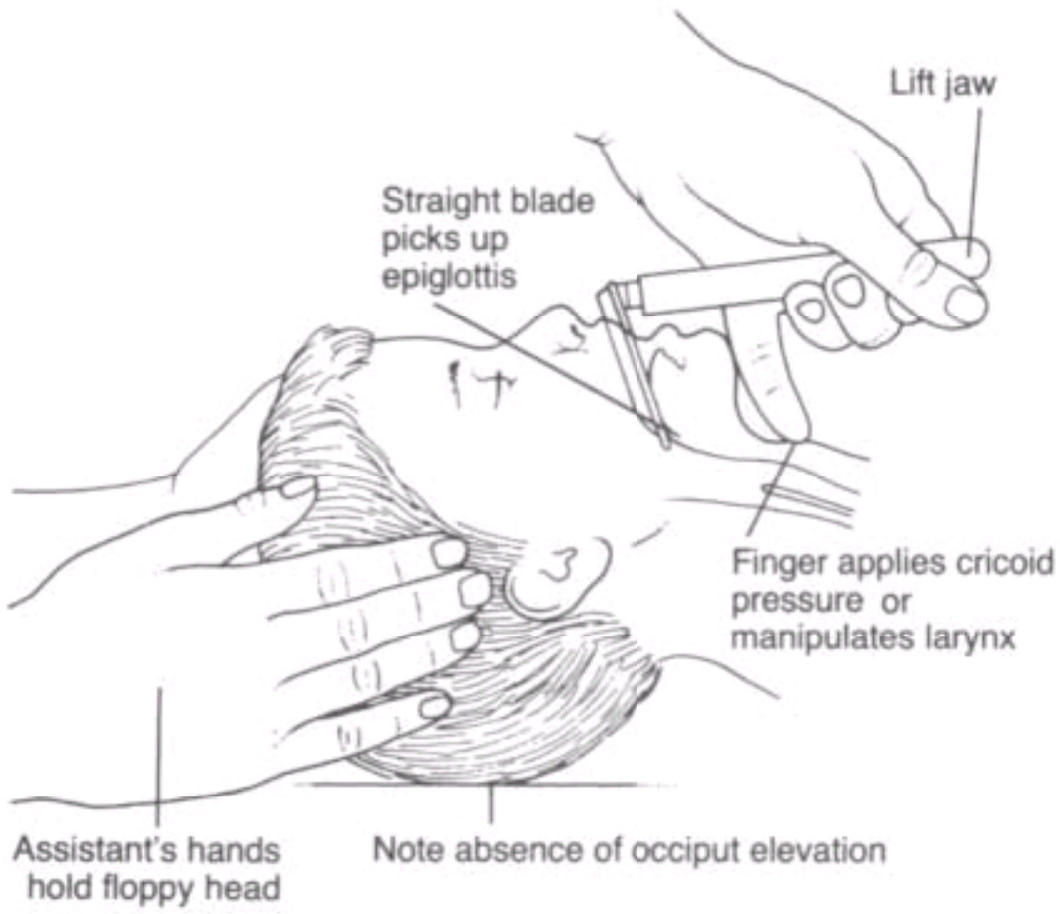


Figure 4-13 Bullard laryngoscope. Anatomically shaped laryngoscope visualizing the glottis; tracheal tube is mounted on the attached stylet for easy tracheal passage. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

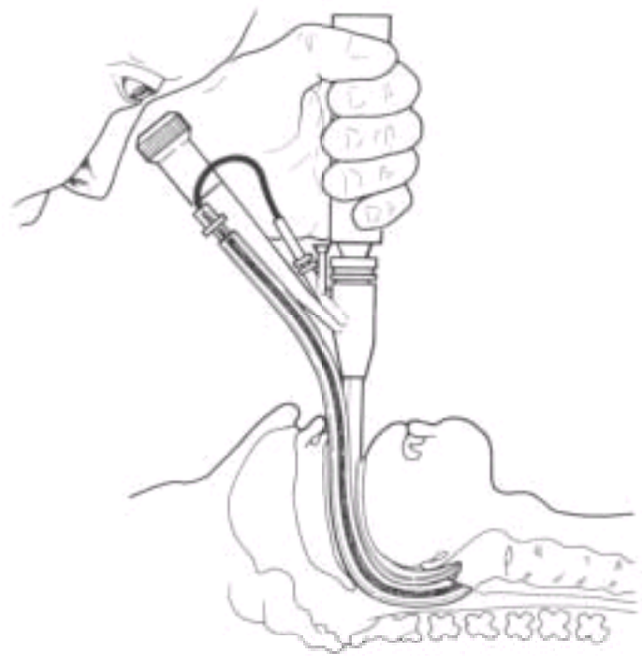


Figure 4-14 Common problem with blind tracheal tube passage through the larynx. *A,* Tip of tube caught on the arytenoid cartilage. *B,* Rotation of tube 90° counterclockwise orients the bevel of the tip posteriorly and allows passage into the larynx. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, MN.)*

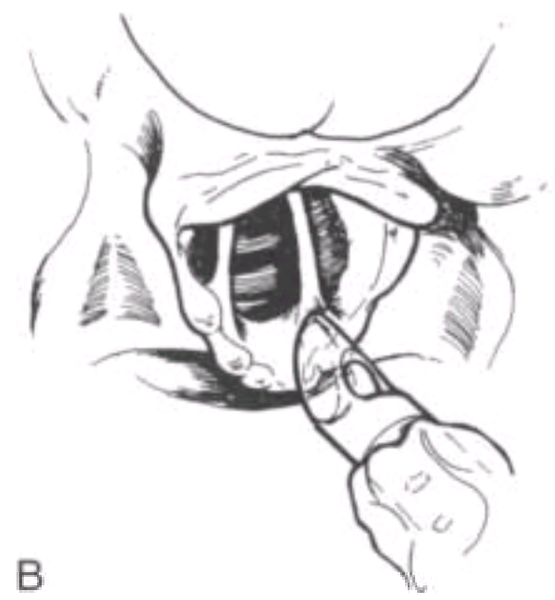


Figure 4-15 Use of suction catheter to aid in passage of nasotracheal tube caught at laryngeal inlet. The suction catheter is passed down the tracheal tube and into the trachea. The tracheal tube is then passed over the suction catheter, and the catheter is removed. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

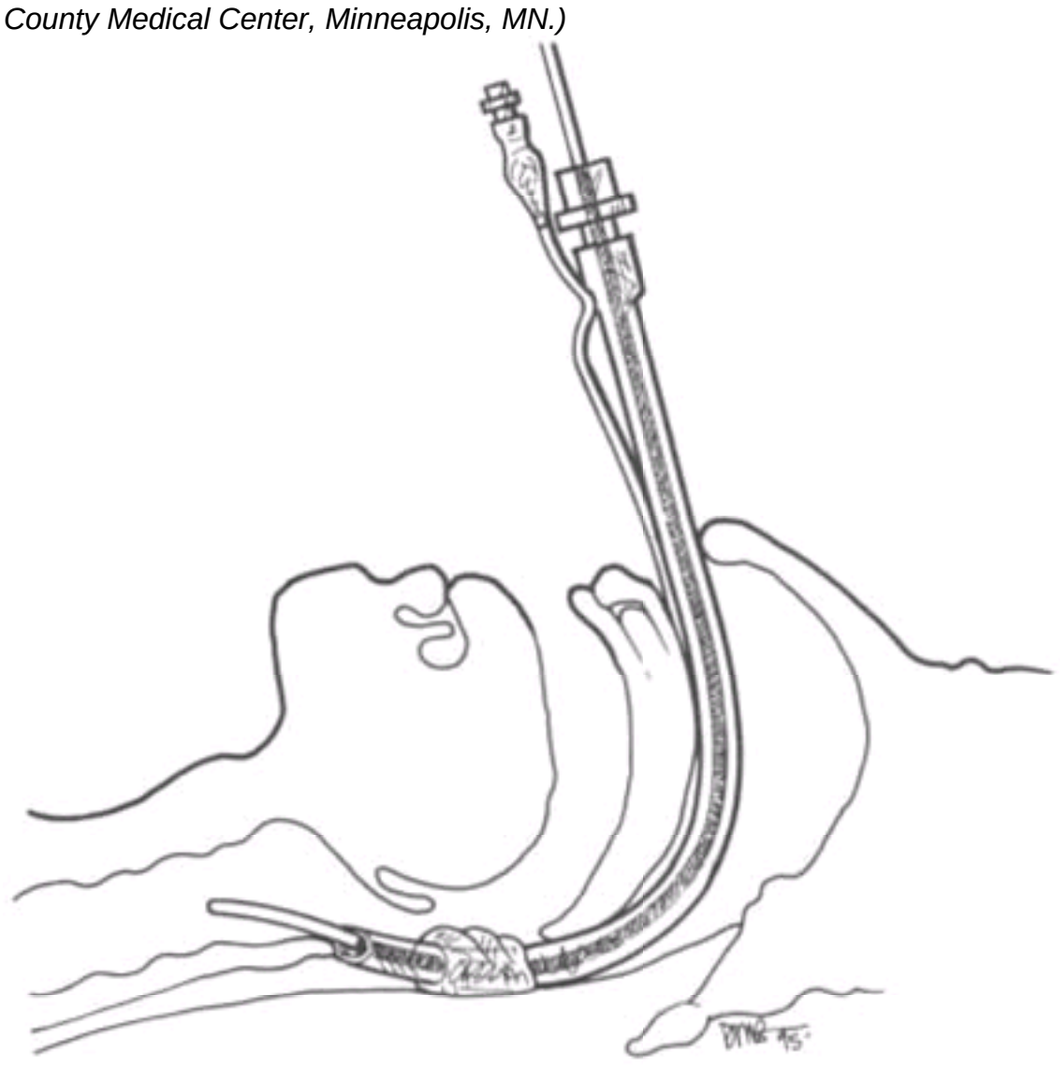


Figure 4-16 Use of tracheal tube cuff inflation to aid in nasotracheal intubation. *A,* Tracheal tube is pulled back after esophageal passage. *B,* Once breath sounds are heard, cuff is inflated with 15 mL of air and is readvanced into the laryngeal inlet. Once seated in the inlet, the cuff is deflated and the tube is advanced into the trachea. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

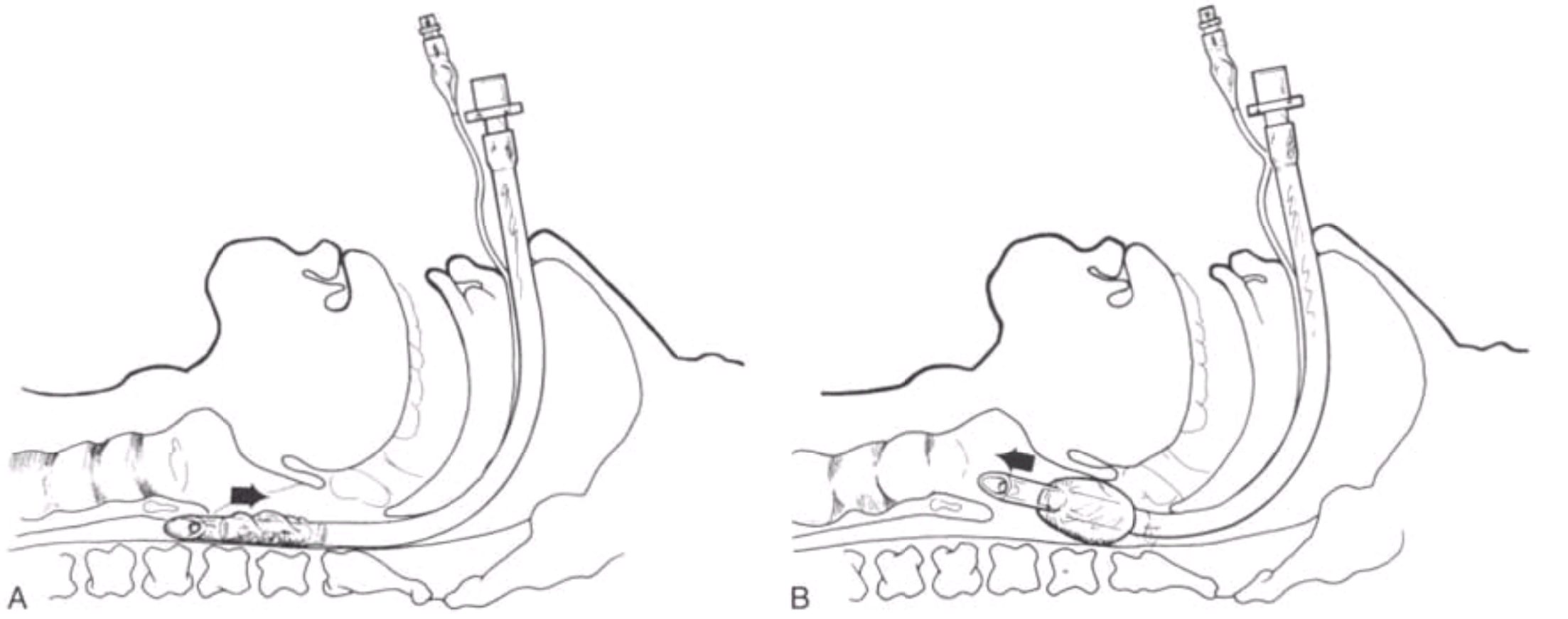


Figure 4-17 Nasotracheal intubation with the aid of a laryngoscope and Magill forceps. Note that the forceps do not pull the tube—they only serve to guide the tip of the tube through the vocal cords while an assistant advances the tube. The cuff is frequently damaged if it is grasped.

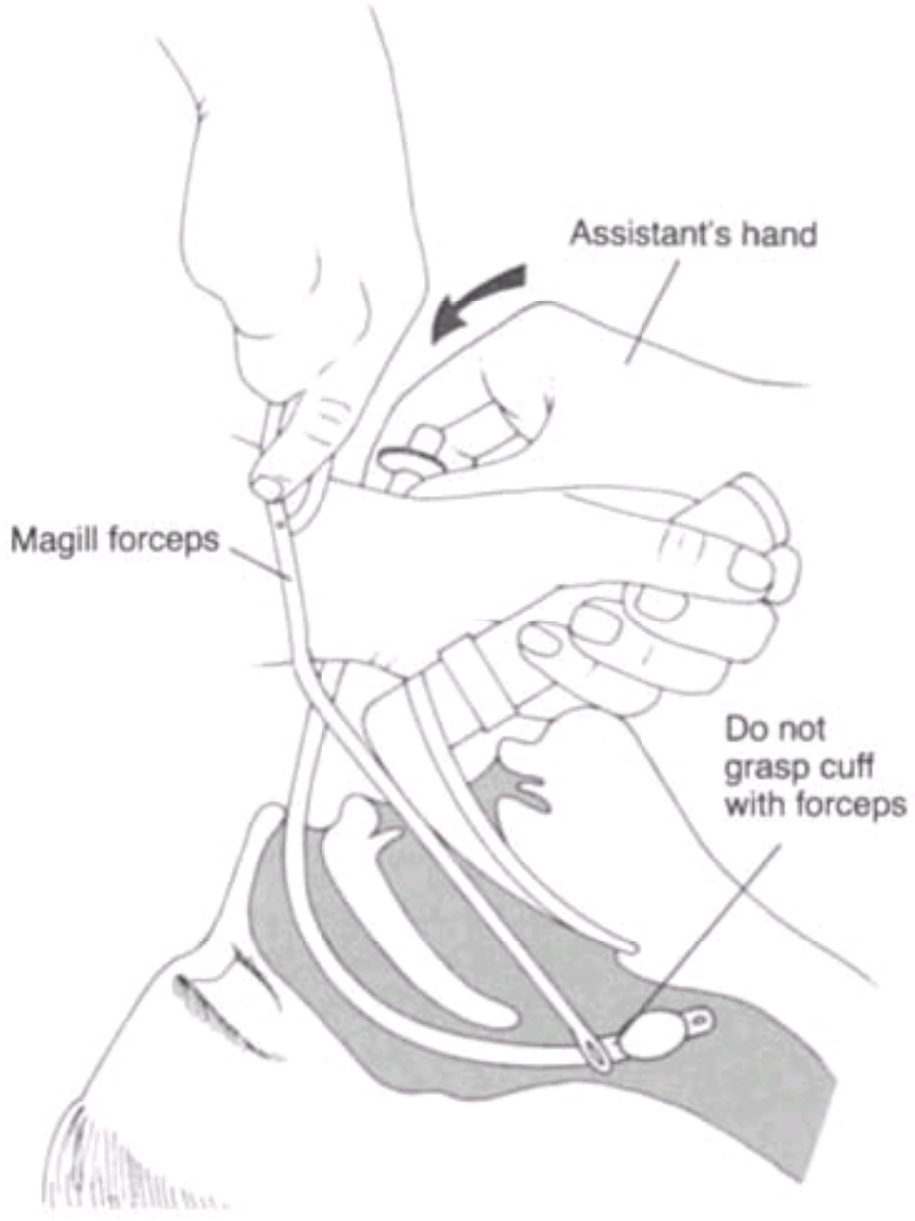


Figure 4-18 Digital intubation. The tracheal tube is cradled between the index and middle fingers and guided into the glottic opening. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

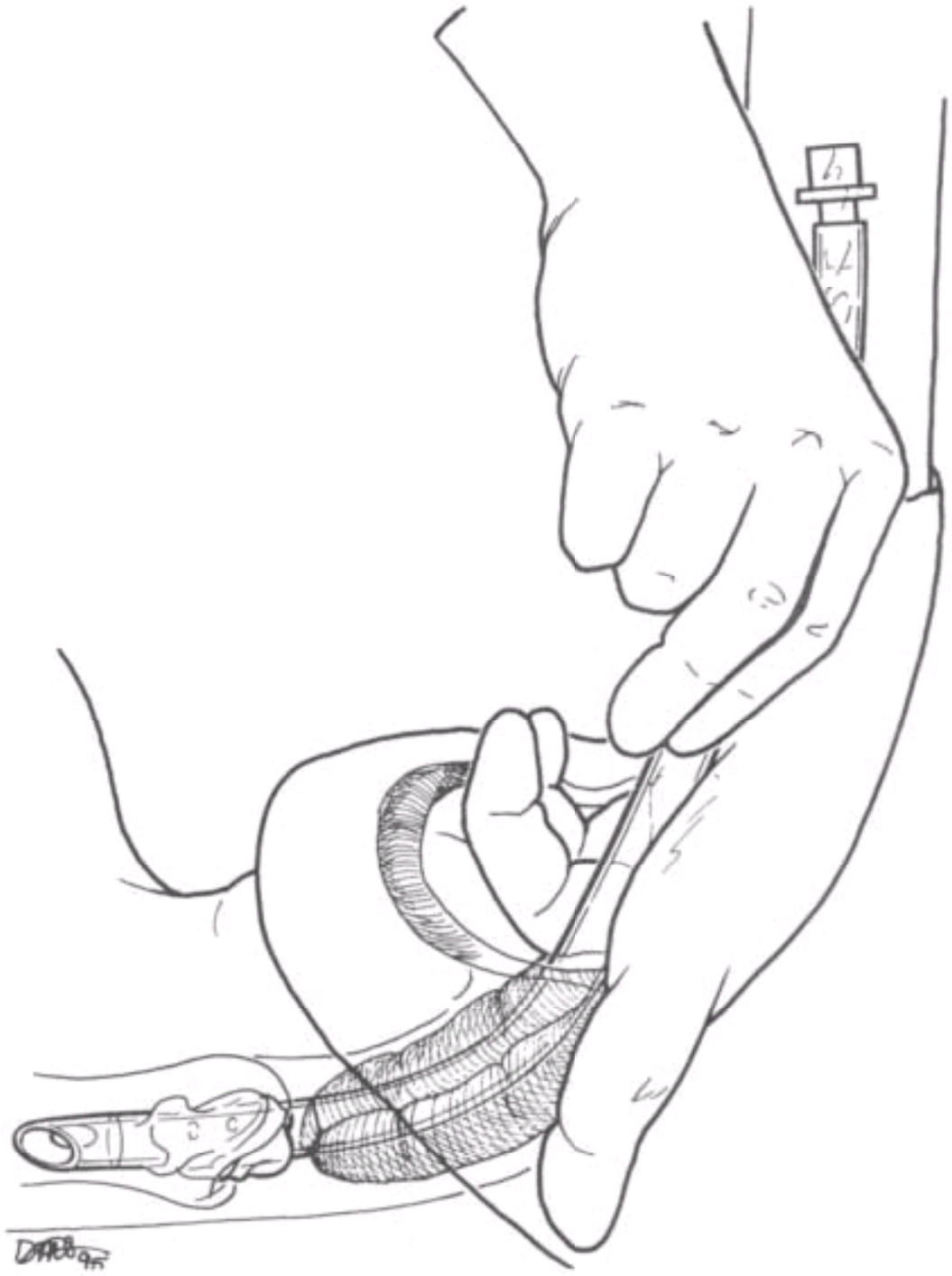


Figure 4-19 Digital intubation in the neonate. The tube is guided using only the index finger to palpate the epiglottis and laryngeal inlet. A stylet is optional. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

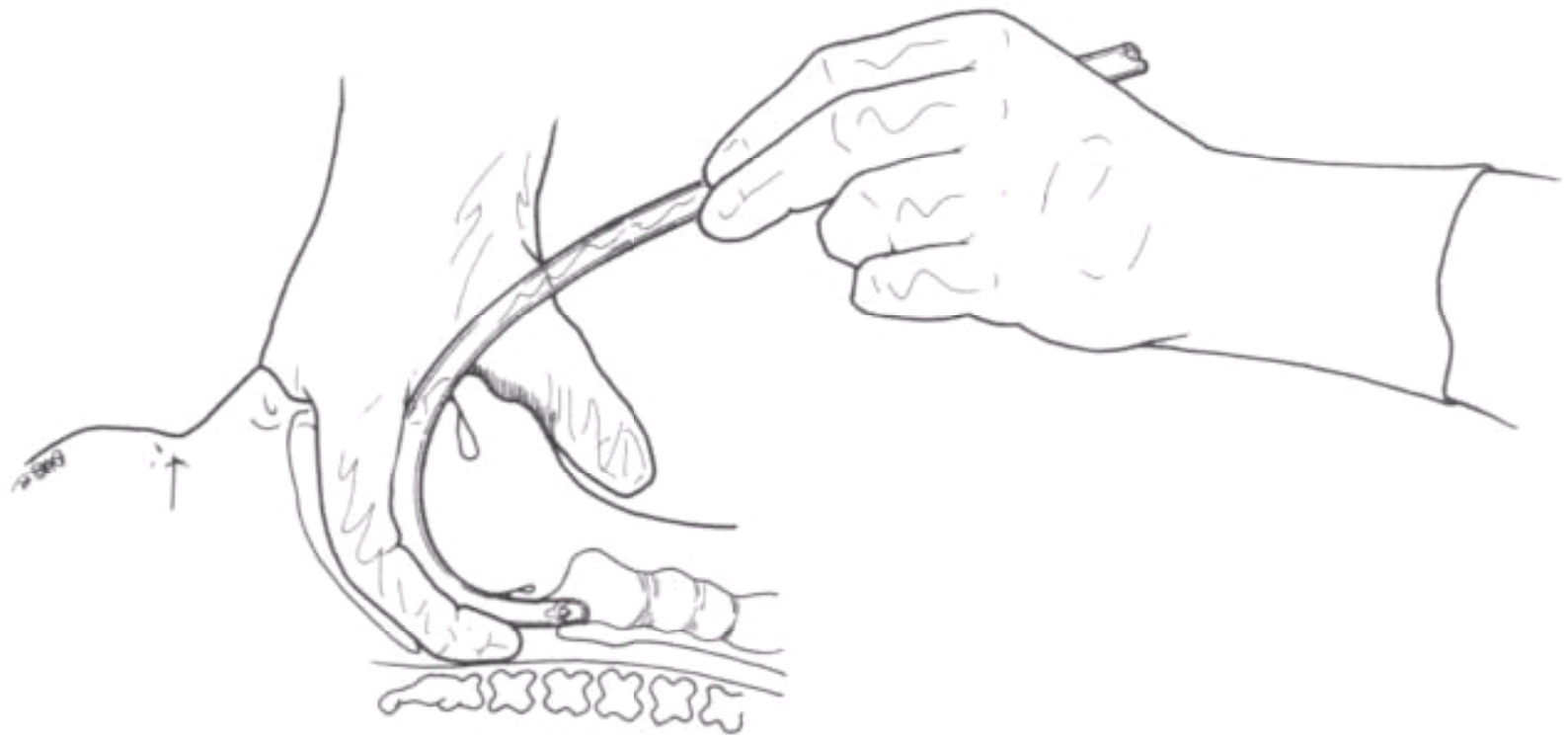


Figure 4-20 Lighted stylet intubation. Use of the Trachlight for endotracheal intubation using transillumination of soft tissues as a guide to placement. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

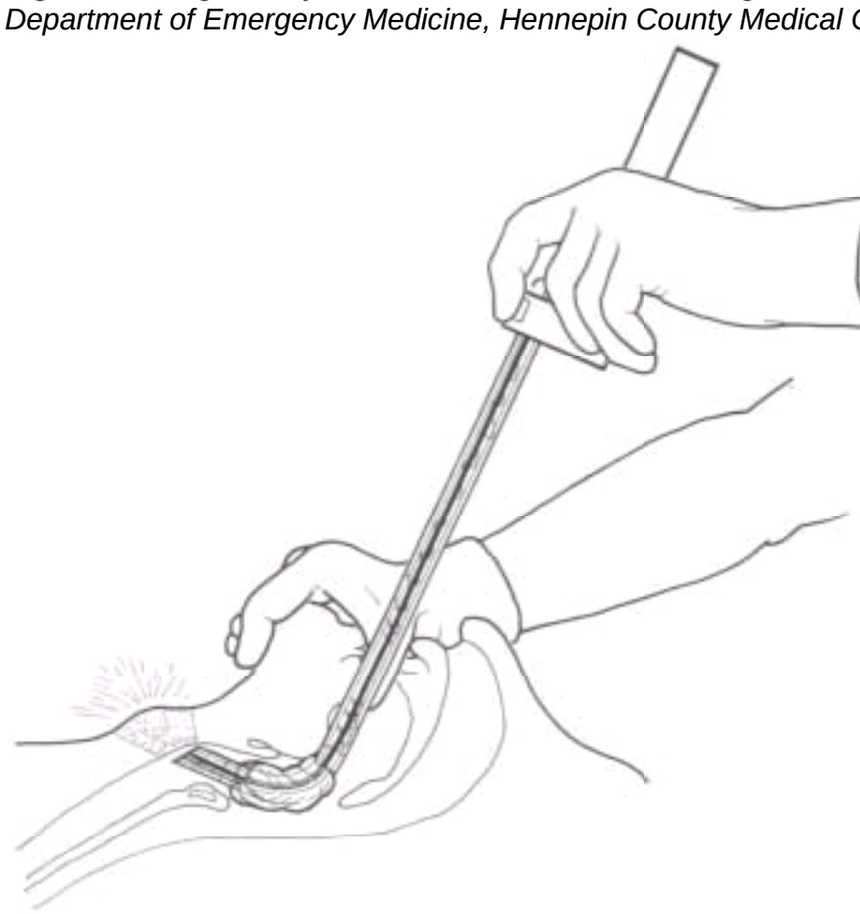


Figure 4-21 Fiberoptic nasotracheal intubation. Note use of the fiberoptic scope as a guide over which the tracheal tube is passed. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

Figure 4-22 Fiberoptic orotracheal intubation. A fiberoptic scope is passed through a hollow oral airway device to help position the scope and tube and to protect them from being bitten during fiberoptic oral intubation. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

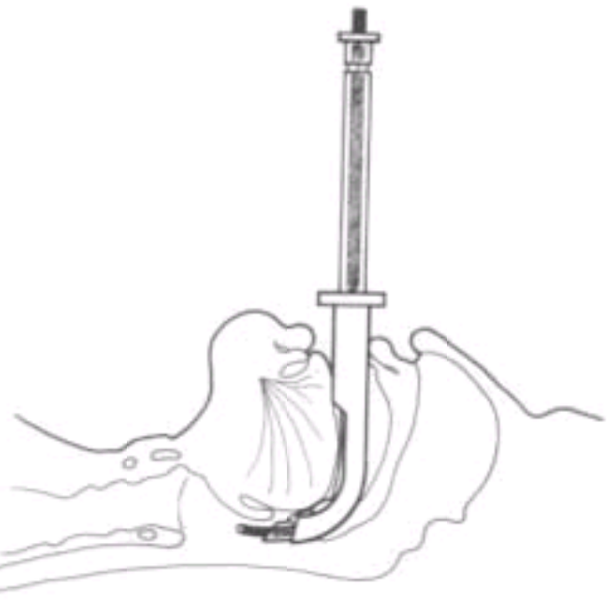


Figure 4-23 Retrograde intubation using guidewire and antegrade sheath. *A*, Placement of a needle through the cricothyroid membrane (note: needle bevel is oriented cephalad). *B*, Placement of the J-wire directed cephalad through the translaryngeal needle. *C*, Passing antegrade sheath over the guidewire. *D*, Removal of guidewire with advancement of sheath into trachea. *E*, Advancement of tracheal tube over sheath into trachea. *F*, Removal of the sheath. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

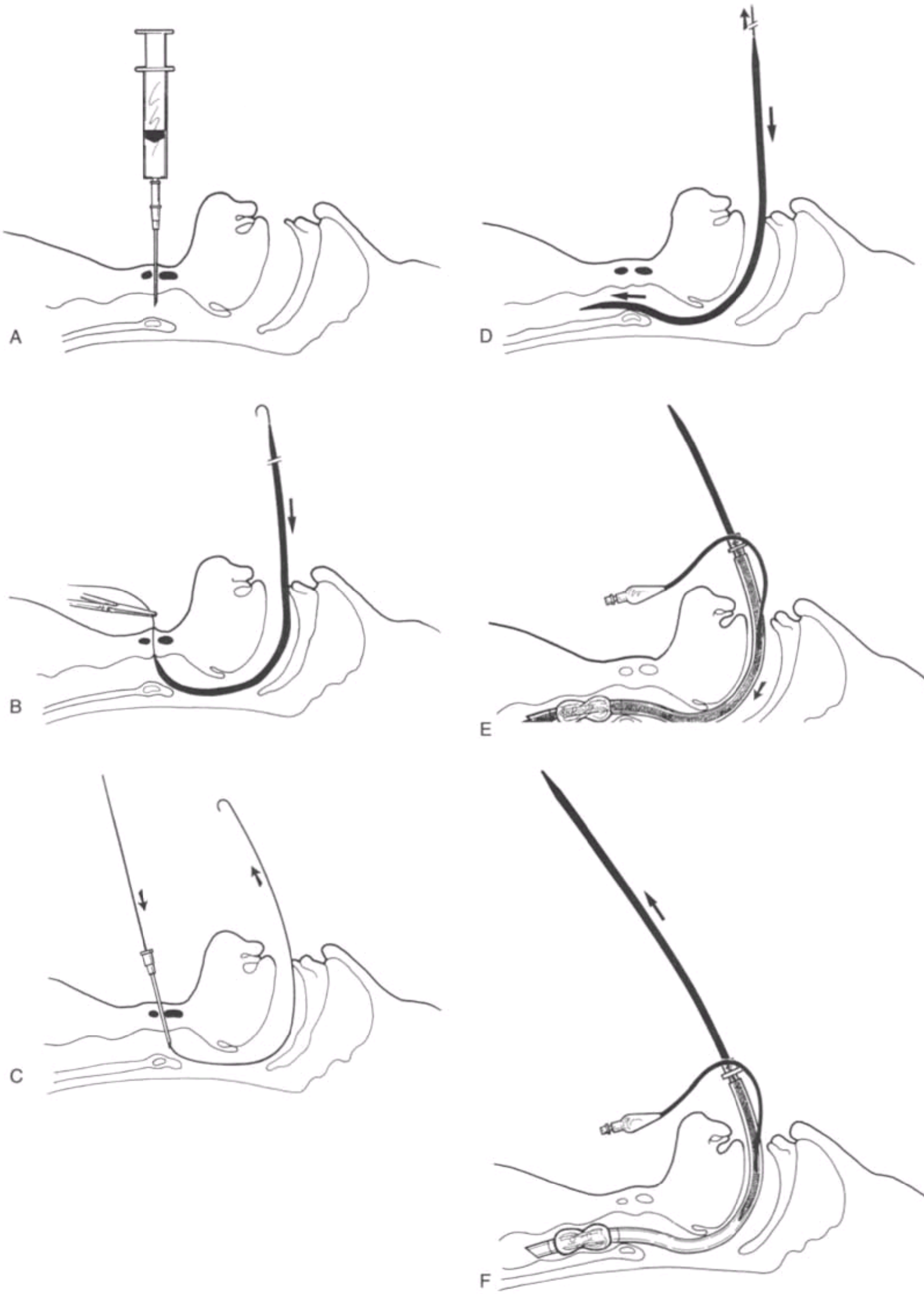


Figure 4-24 Fiberoptic scope as an aid in retrograde intubation. Once the retrograde wire (long) is placed and retrieved superiorly, the wire is threaded through the tip of the fiberoptic scope and out through the suction port. The scope is then advanced until it passes through the vocal cords, stopping at the anterior laryngeal wall. The wire is withdrawn through the suction port, the fiberoptic scope is advanced into the trachea, and the tracheal tube advanced over the scope into the trachea. The fiberoptic scope is then withdrawn. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

Figure 4-25 Replacing damaged tracheal tube. *A,* Placement of tracheal tube exchanger down damaged tube and deep into trachea (*open arrow*) followed by removal of tracheal tube (*solid arrow*). *B,* Replacement tube is passed over tube exchanger into trachea, followed by removal of tube exchanger. *(Courtesy of Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN.)*

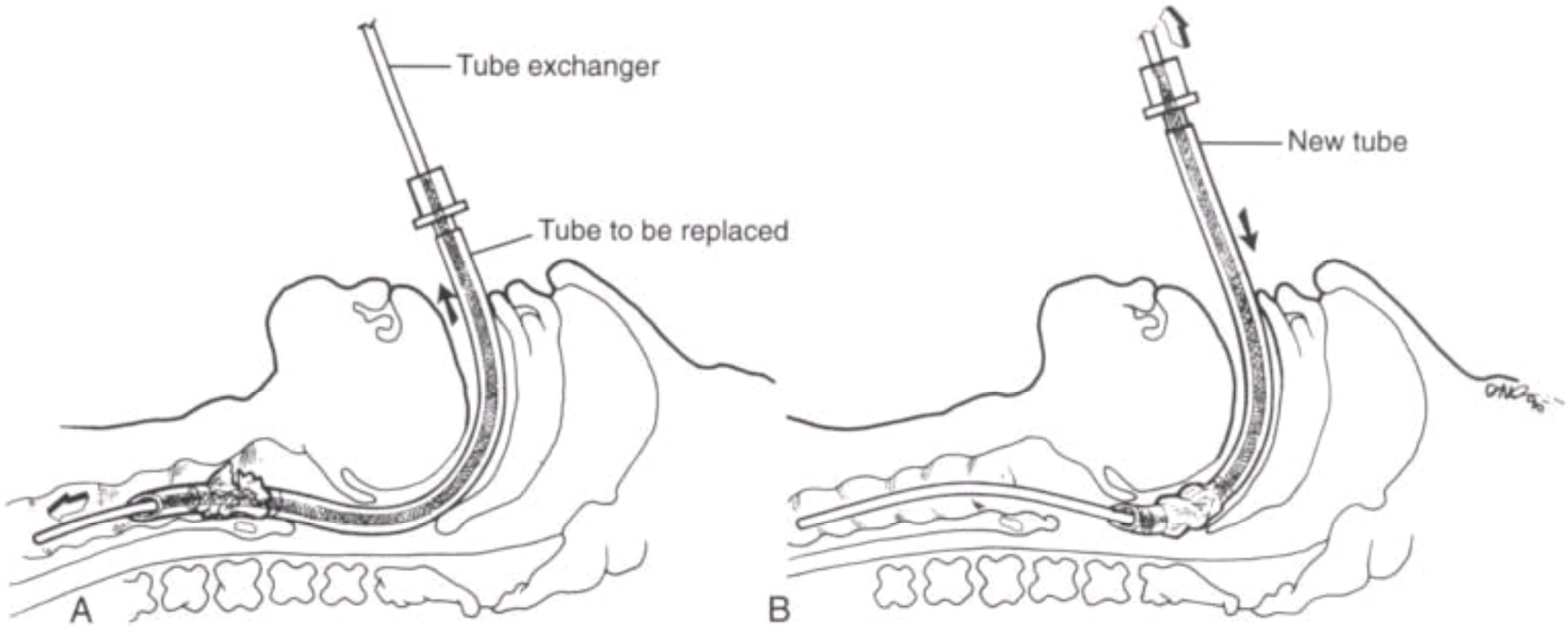
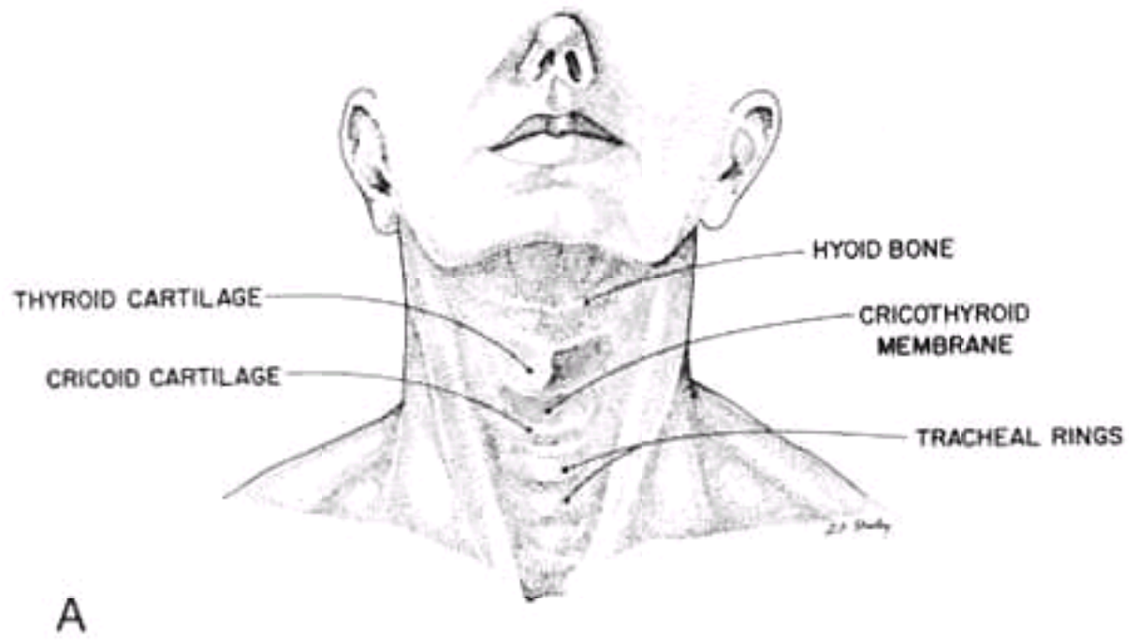
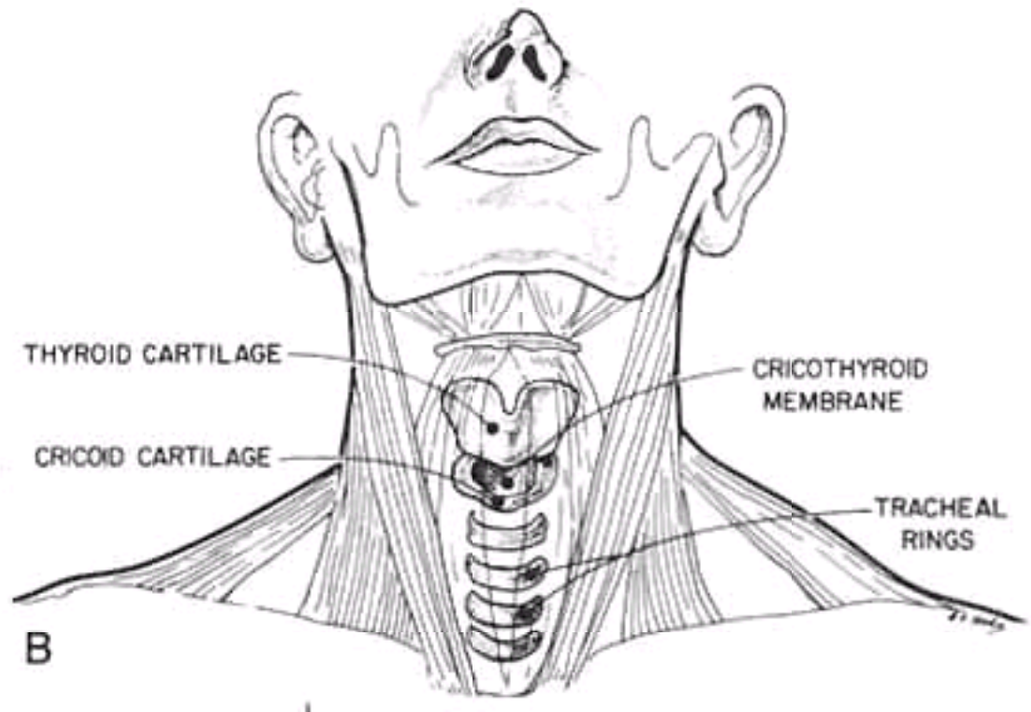


Figure 6-1 Anatomy of the neck. *A*, Surface anatomy of the neck, showing important external landmarks. *B*, Anterior view of the neck, showing various internal structures (overlying superficial skin and structures removed to show cricothyroid membrane). *C*, Lateral view of the neck, showing various structures.





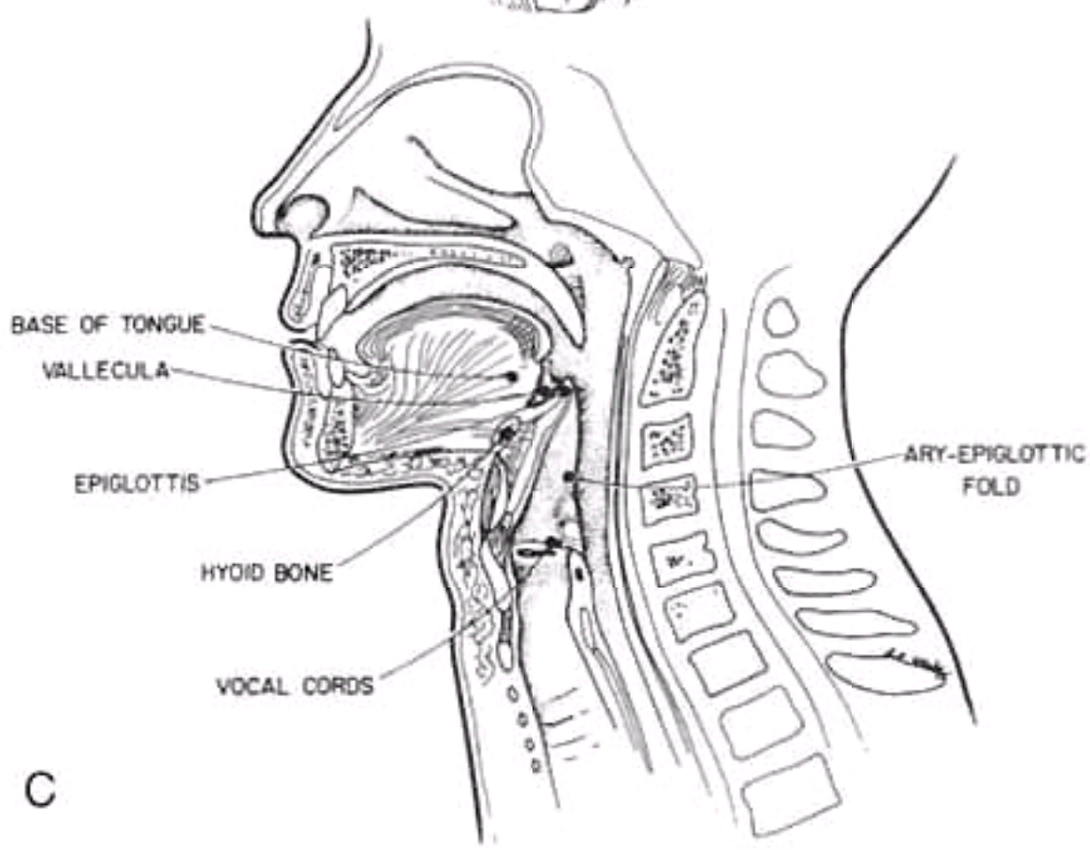


Figure 6-2 Modification of an endotracheal tube for use as a tracheostomy tube. *A*, An appropriate-sized endotracheal tube is cut to an appropriate length. *B*, An adapter is attached to the cut end before the modified endotracheal tube is inserted into the larynx via the opening in the cricothyroid membrane.

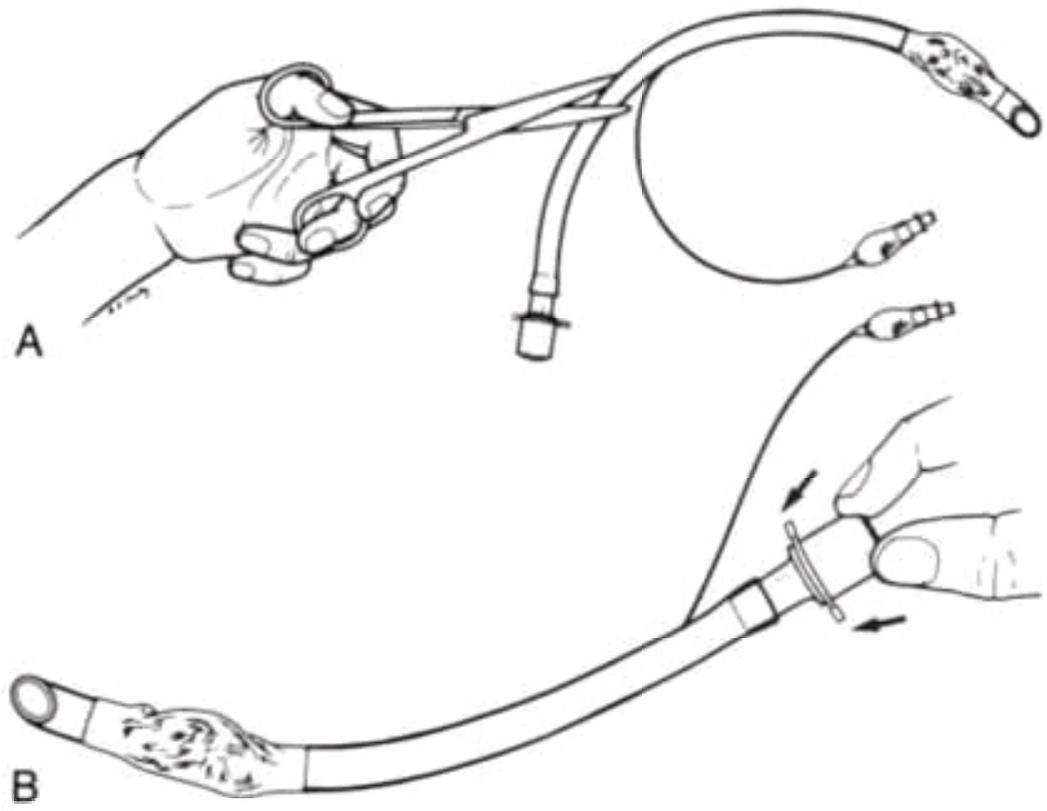


Figure 6-3 The procedure for surgical cricothyrotomy. *A*, Locate the cricothyroid membrane. The insert shows further anatomic details of the region. *B*, A longitudinal skin incision is made over the cricothyroid membrane. *C*, The larynx is stabilized with the thumb and middle finger or a tracheal hook (held in the nondominant hand) while an incision is made in the cricothyroid membrane. *D*, The surrounding anatomy of the neck is shown, with the incision being made in the cricothyroid membrane. *E*, After the incision in the cricothyroid membrane is widened using hemostats, curved Mayo scissors, or the blunt end of the scalpel, the tracheostomy tube is inserted between the curved hemostats or tracheal dilator. *F*, Lateral view, showing insertion of the tracheostomy tube.

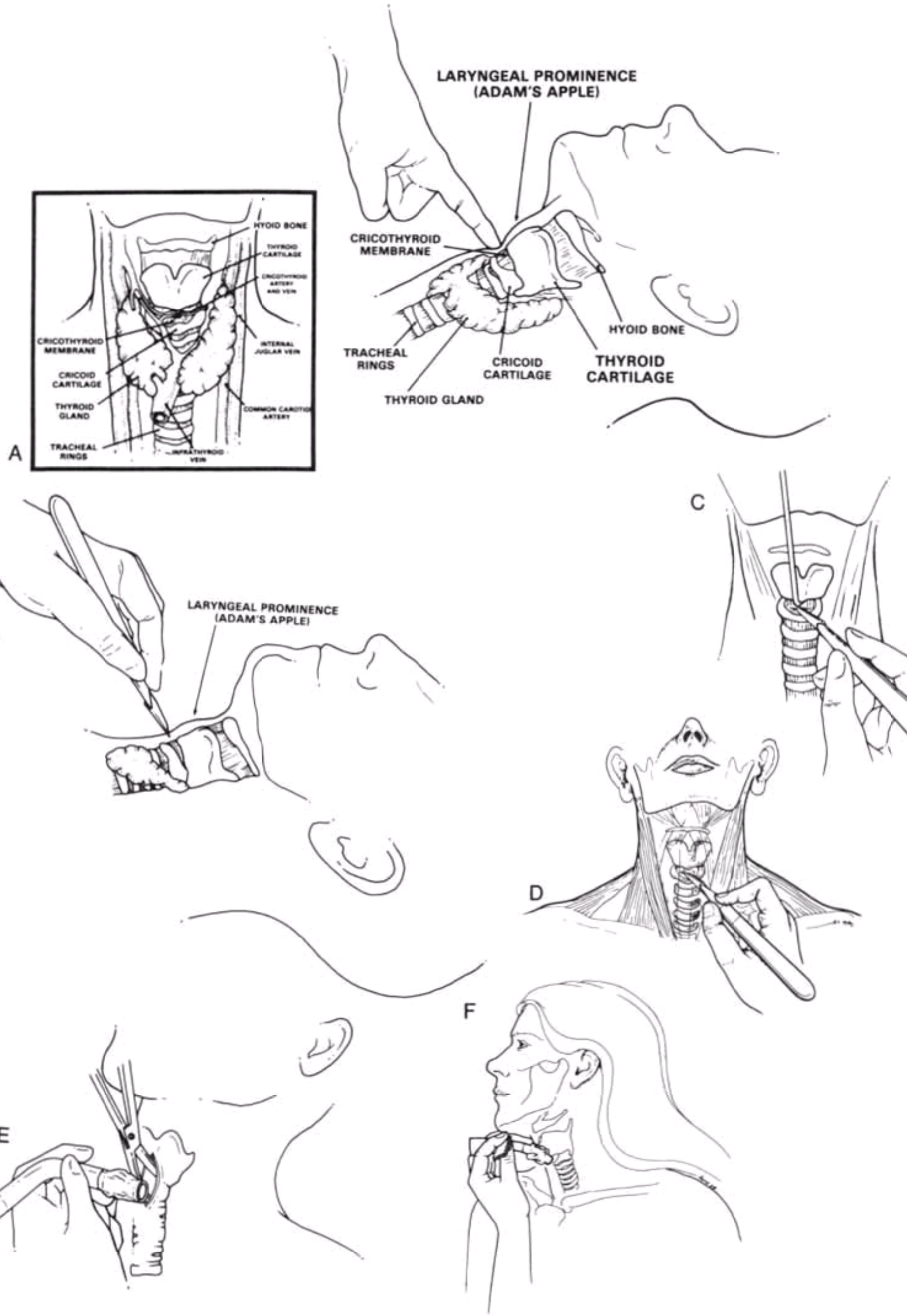


Figure 6-4 Rapid, four-step cricothyrotomy technique. *A,* Step 1: Palpation (location of cricoid membrane externally). *B,* Step 2: Incision (horizontal incision of skin and soft tissues through cricoid membrane). *C,* Step 3: Traction (caudal traction on cricoid ring). *D,* Step 4: Intubation (passage of tracheal tube). *(Reproduced from Brofeldt BT, Panacek EA, Richards JR. An easy cricothyrotomy approach: the rapid four-step technique. Acad Emerg Med 3:1060, 1996.)*

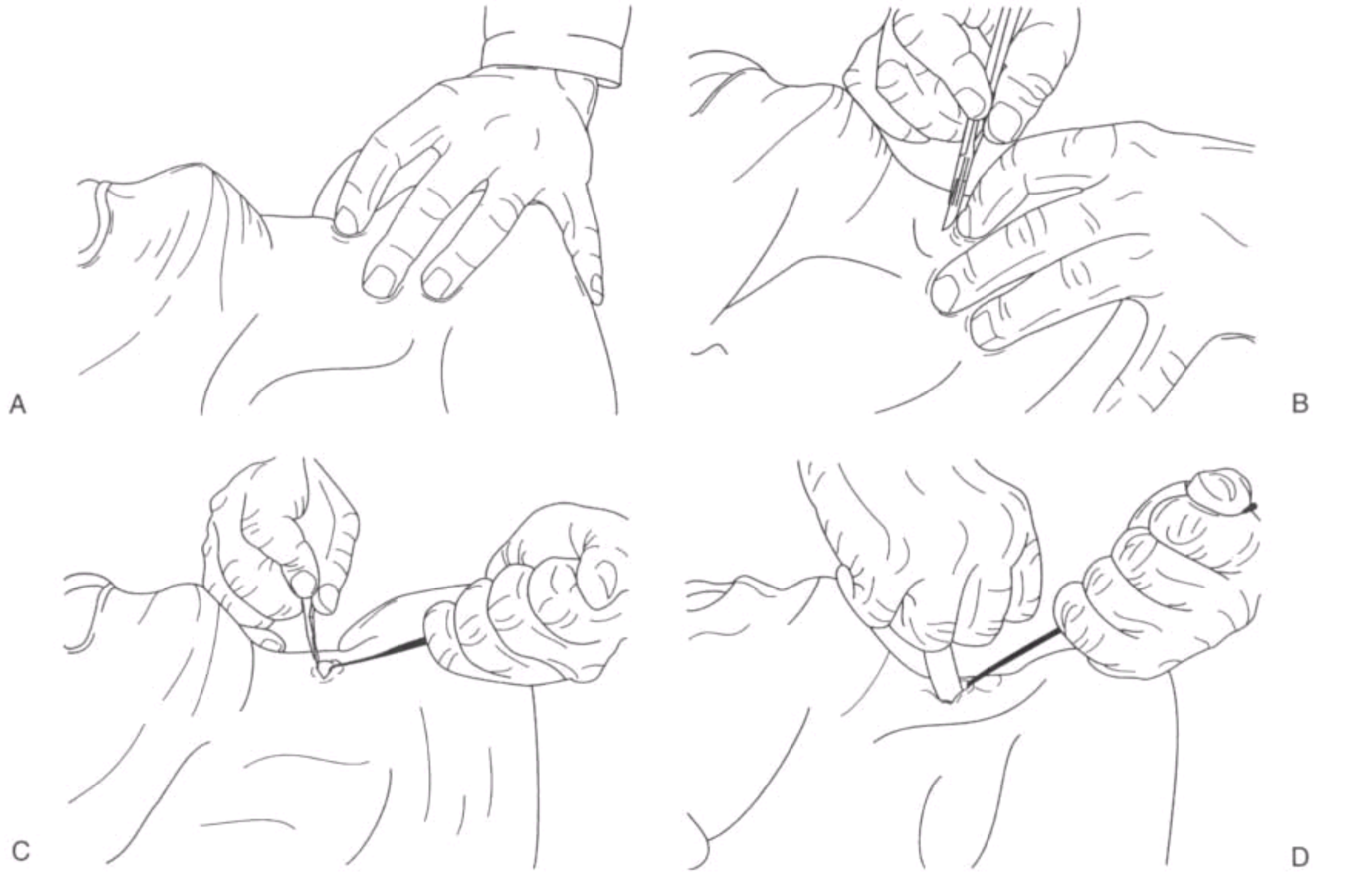
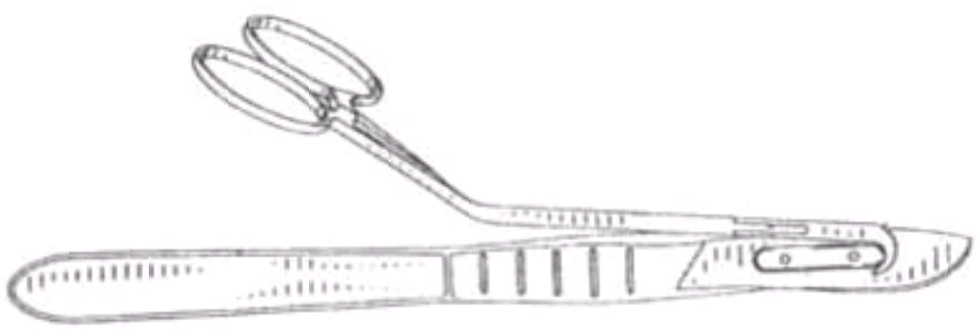


Figure 6-5 "Bair Claw" device. *A,* Device shown without scalpel. During the tracheal tube intubation procedure, the device is used as a caudally directed tracheal hook with slight spreading of the arms of the hook. In this manner the device both stabilizes the distal trachea and provides exposure of the lumen. *B,* Device shown attached to scalpel. During the initial incision, the device is attached to the No. 20 scalpel and passively placed through the cricothyroid membrane during the initial horizontal incision. The "claws" of the device are then released and the scalpel removed. The device is then rotated into position and used as outlined above. *(Davis DP, Bramwell KJ, Hamilton RS, et al. Safety and efficacy of the rapid four-step technique for cricothyrotomy using a Bair Claw. J Emerg Med 19:125, 2000.)*

A



B

Figure 6-6 Modification for a patient with a massive neck swelling (see text). *A*, Landmarks in the neck. *B*, The procedure for a patient with a massive neck swelling uses a spinal needle to find and identify the hyoid bone; the skin incision is then made in the midline, moving inferiorly from the midpoint of the hyoid bone past the thyroid cartilage until the cricothyroid membrane is exposed. Care is needed to *avoid* a high incision through the thyrohyoid membrane above the thyroid cartilage.

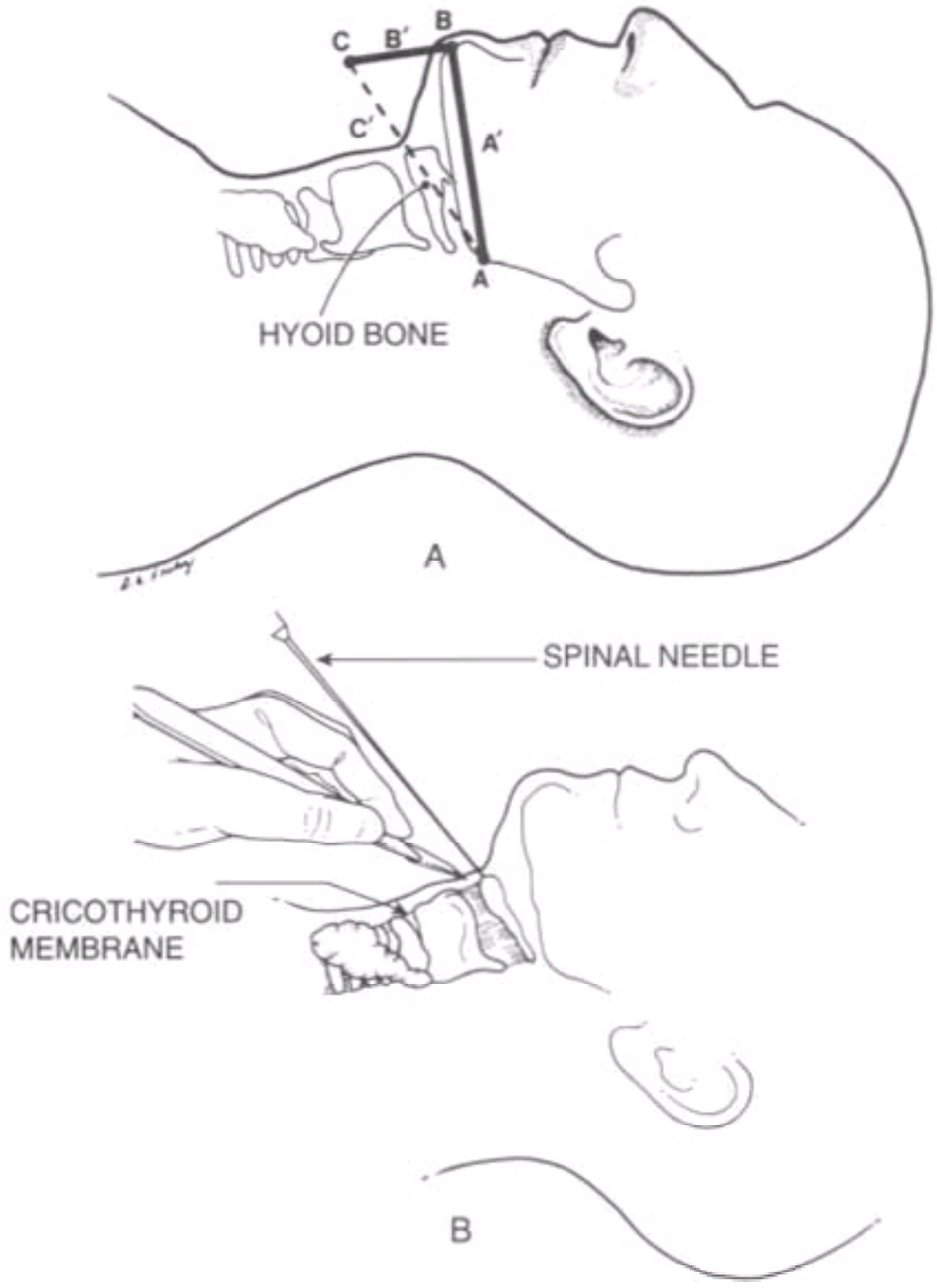


Figure 6-7 The equipment for translaryngeal ventilation. One-way valve used to control ventilation. Note that tubing is attached at the neck to the hub of the cricothyrotomy device, or if a tracheostomy is in place, the insufflation catheter can be passed into the tracheostomy tube.

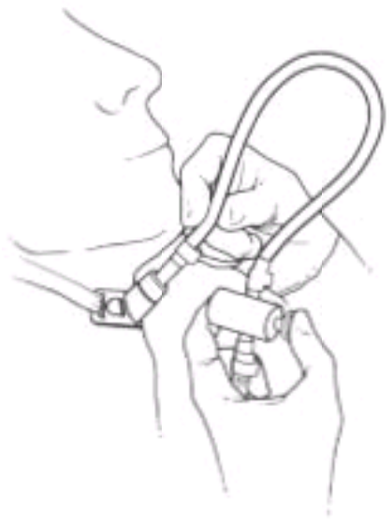


Figure 6-8 A simple setup for translaryngeal ventilation using standard equipment found in any emergency department. This setup is inadequate for adults. High-pressure (50 psi) ventilation systems (see Fig. 6-7) are optimal. Even with the pressure relief valve on the bag-valve device turned off, only a suboptimal pressure can be developed. However, this technique may be satisfactory in infants and small children.

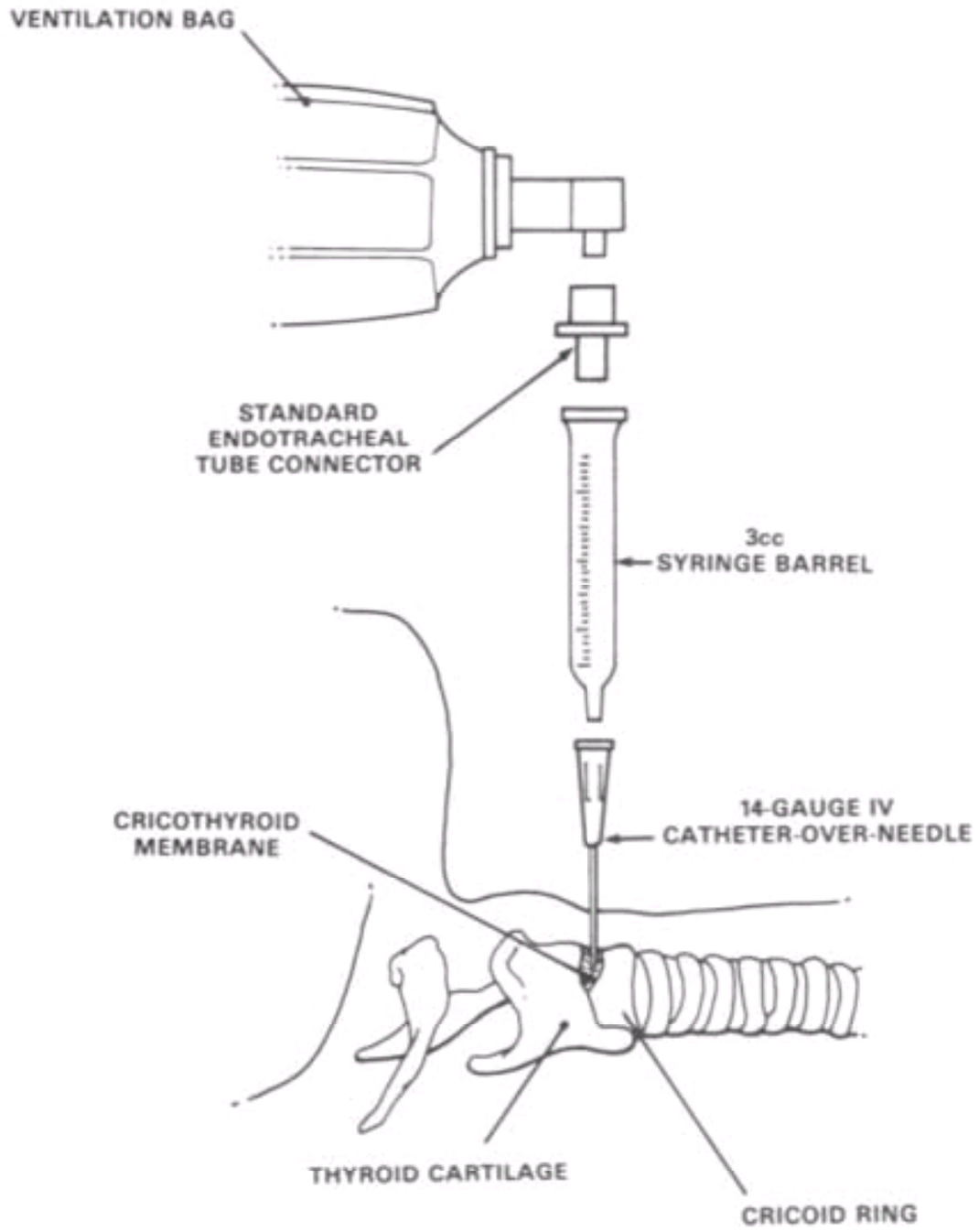
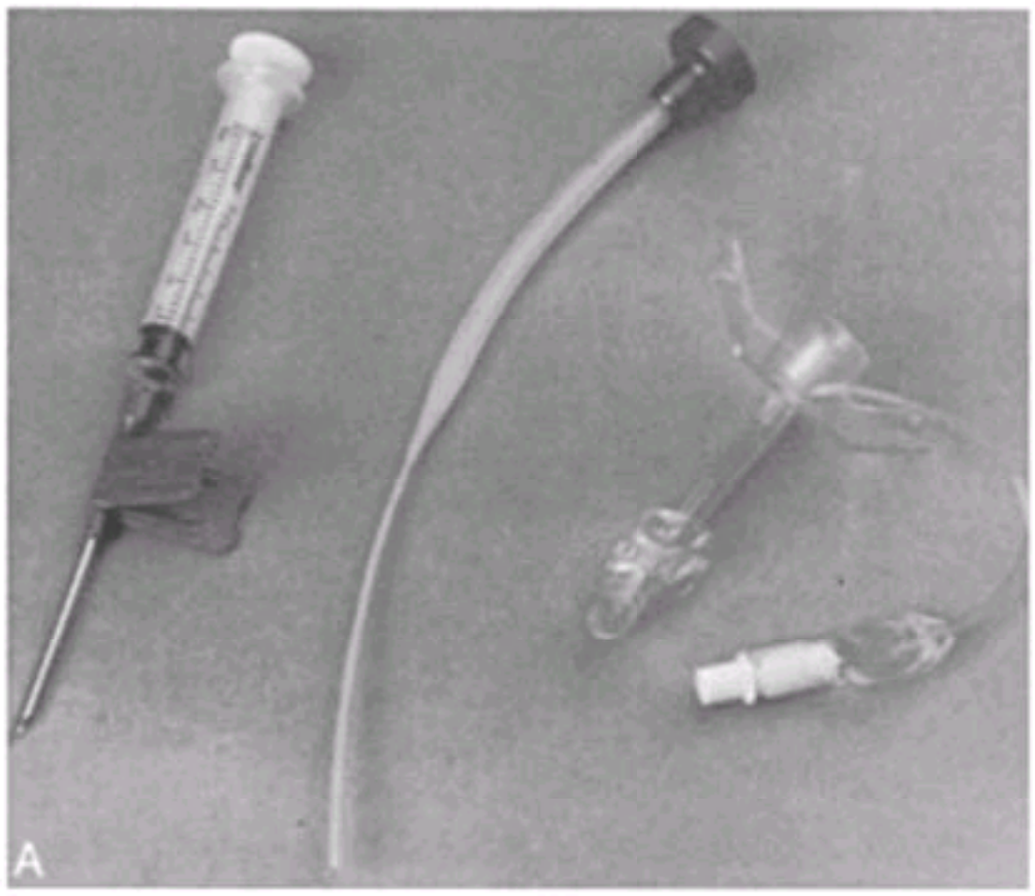
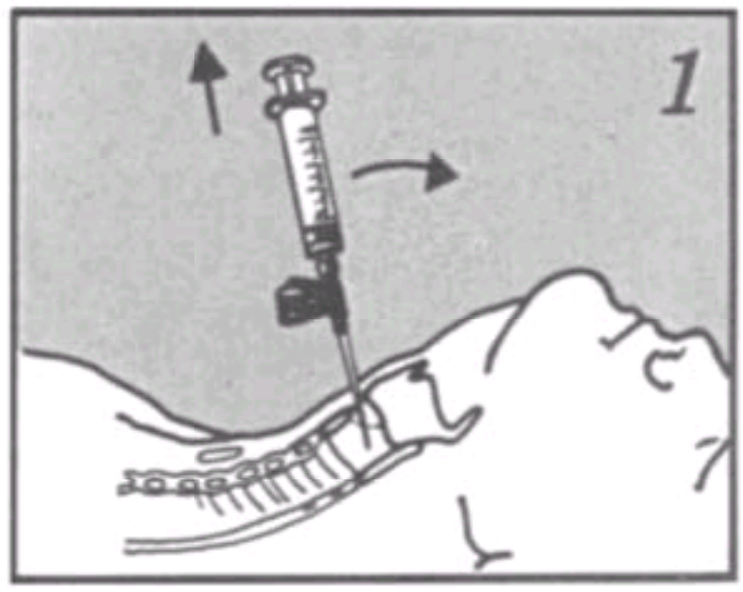
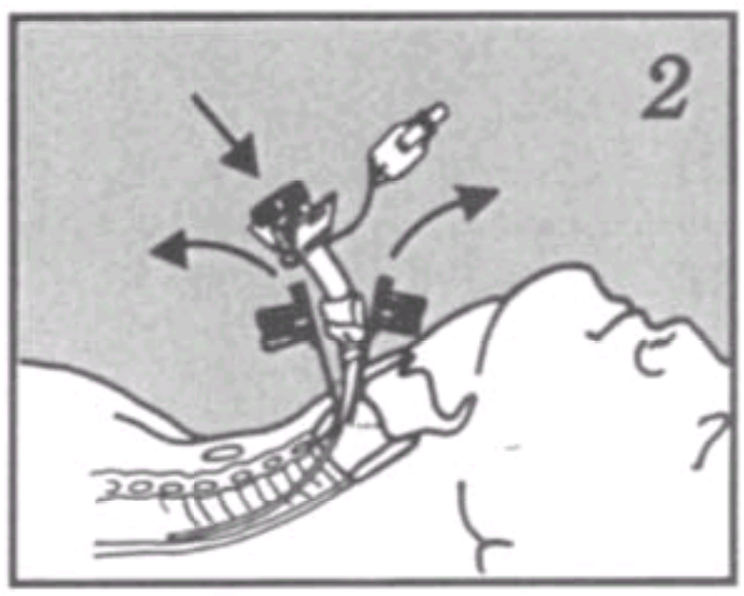


Figure 6-9 *A*, Commercial device used in performing a cricothyrotomy with a modified Seldinger technique. The adult Pertrach percutaneous emergency airway provides a means of rapidly inserting a tube into a patient's trachea (through either the cricothyroid membrane or trachea) in certain clinical situations. It provides an adequate airway that can, by its standard 15-mm adapter, be used to allow the patient to breathe. The unit allows full control of the airway with inflation of the cuff. The pediatric device is provided uncuffed in three sizes for infants and children of various sizes. The 15-mm adapter is part of each unit. (Tracheostomy *only* is recommended in children and infants.) *B*, Method of establishing an airway with the Pertrach cricothyrotomy kit: (1) The 14-ga needle is inserted into the trachea through a cricothyroid membrane or upper trachea. Placement in the airway is verified by drawing air. The syringe is removed. (2) The dilator is placed to its hilt into the tracheostomy tube, and both are inserted as a single unit. The leader of the dilator is inserted into the trachea through the needle, which is then split and removed. The dilator and trachea tube are inserted into the trachea. Note that a small skin incision at the puncture site is needed to facilitate passage of the tracheal tube. (3) The dilator is removed. The cuff can then be inflated and a respirator applied. *(Courtesy of Pertrach, Inc., Clarksburg, WV 26301.)*







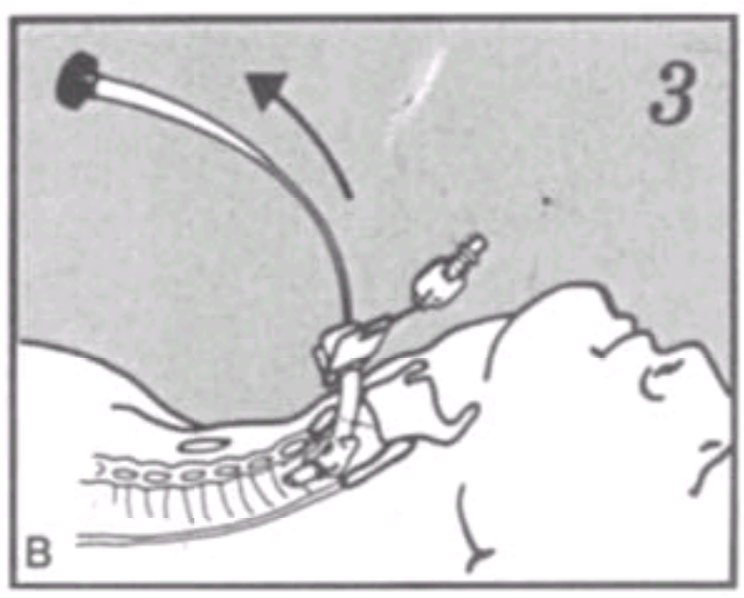


Figure 6-10 The procedure for transtracheal ventilation. *A*, The cricothyroid membrane is punctured with the needle or catheter aimed caudally at approximately a 45° angle. *B*, The inner needle has been removed, and the catheter is hooked up to a manual in-line device and then to a source of oxygen. A valve device or tubing modification as shown in Figure 6-7 is also required.

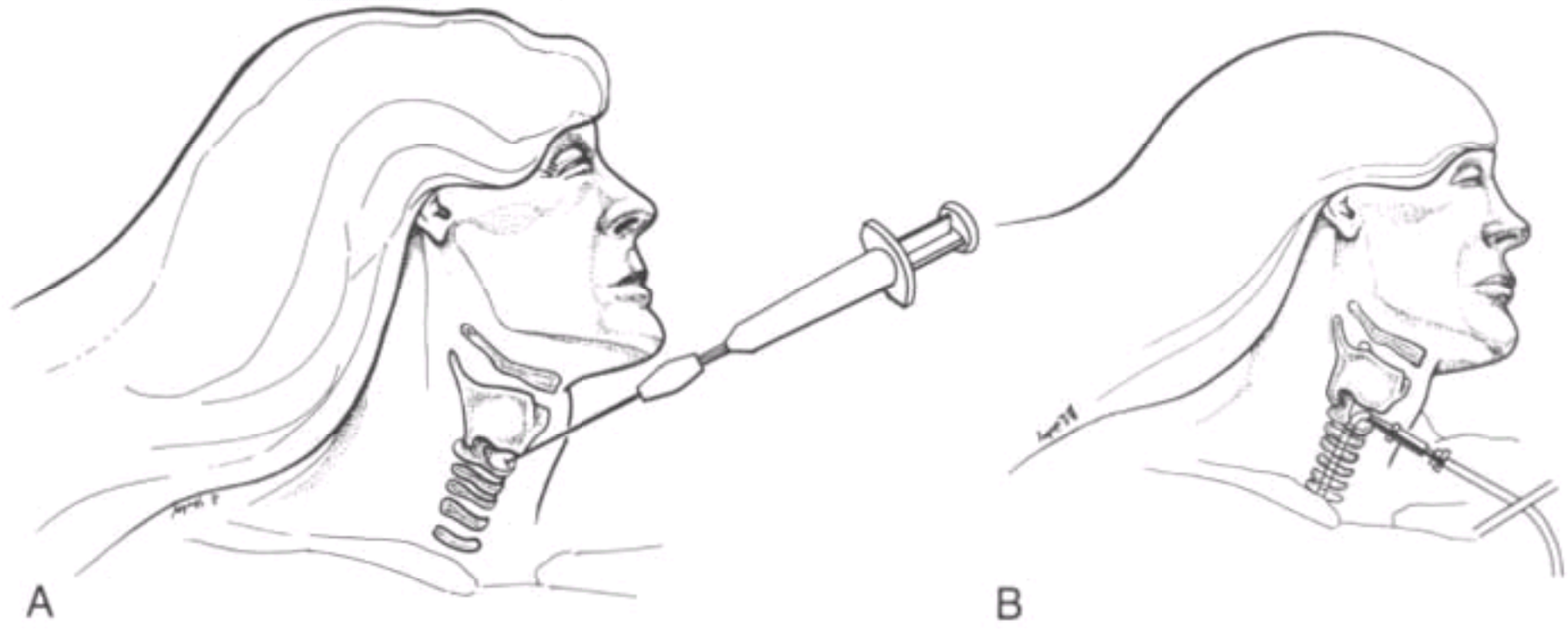


Figure 7-1 Control of innominate artery bleed by digital pressure. When major bleeding occurs and a cuffed tracheostomy tube is present, overinflation of the tube cuff may temporize (see text). When unsuccessful or a cuffed tube is not available, use the illustrated maneuver; digital pressure should be applied to the anterior tracheal wall through the tracheostomy. The index finger is placed within the trachea and then pulled against the anterior tracheal wall, allowing the airway to remain partially open. The artery is compressed between the index finger and the thumb—placed over the anterior neck. Digital compression of the innominate artery is a temporizing procedure, until definitive (operative) management of the bleed is obtained.

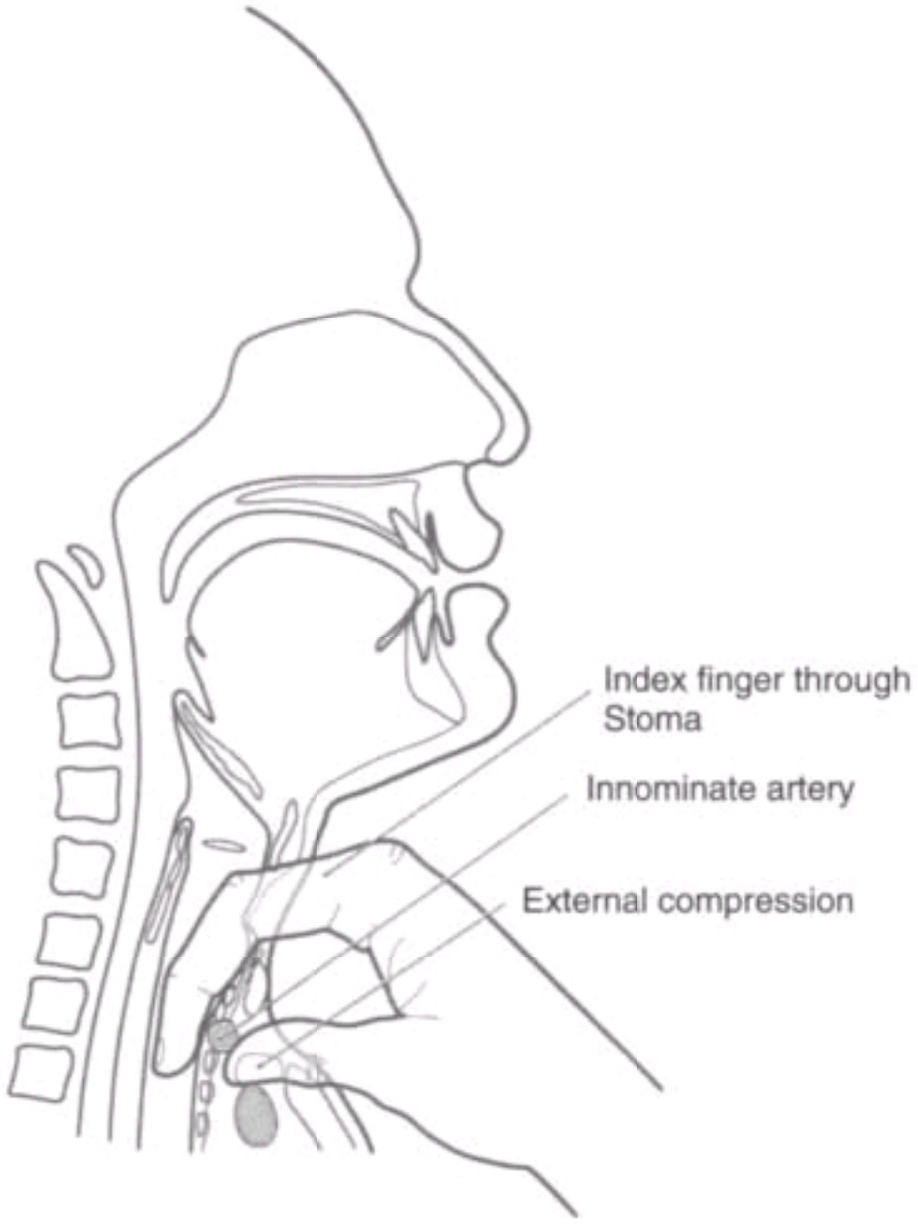


Figure 7-2 Tracheostomy tube *(top)* with inner cannula and attached ambu bag adaptor *(middle)* and obturator *(bottom)*.

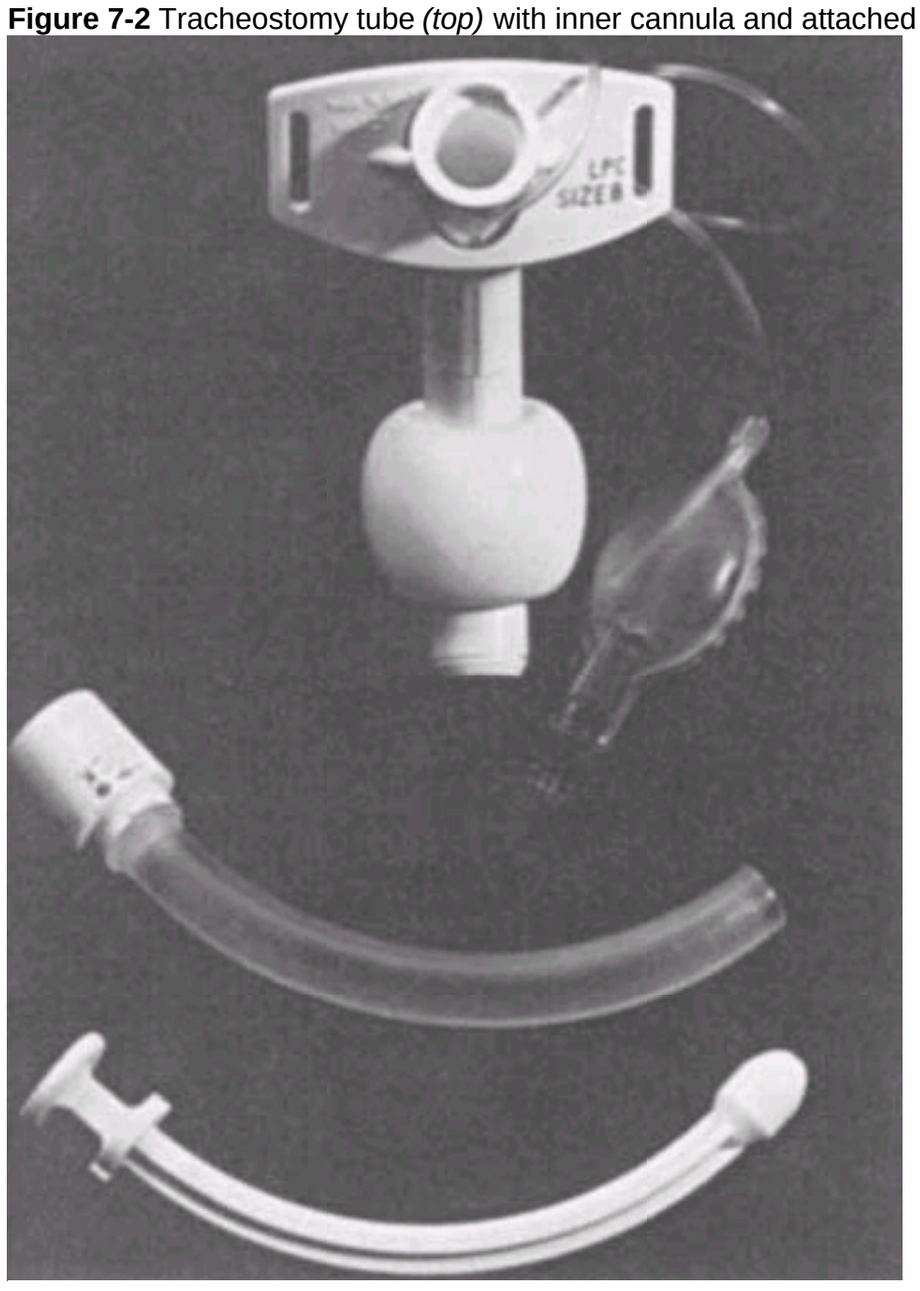


Figure 7-3 Changing a tracheostomy tube. *A,* Before the old tube is removed, a small red rubber catheter (or other guide catheter) is passed into the proximal trachea. *B,* The tracheostomy tube has been removed over the catheter, and only the catheter remains in the trachea. The catheter serves as a guide for easy and atraumatic insertion of a new tube. Note that the neck should be slightly hyperextended. *C,* A new tracheostomy tube, without the obturator is threaded over the guide catheter; once the tube is in place, the catheter is removed. Similarly, if the tracheostomy tube has already been removed, the catheter may be passed through the stoma before a new tube is advanced. Note that an obturator or inner cannula is *not* used when changing a tube with this technique.

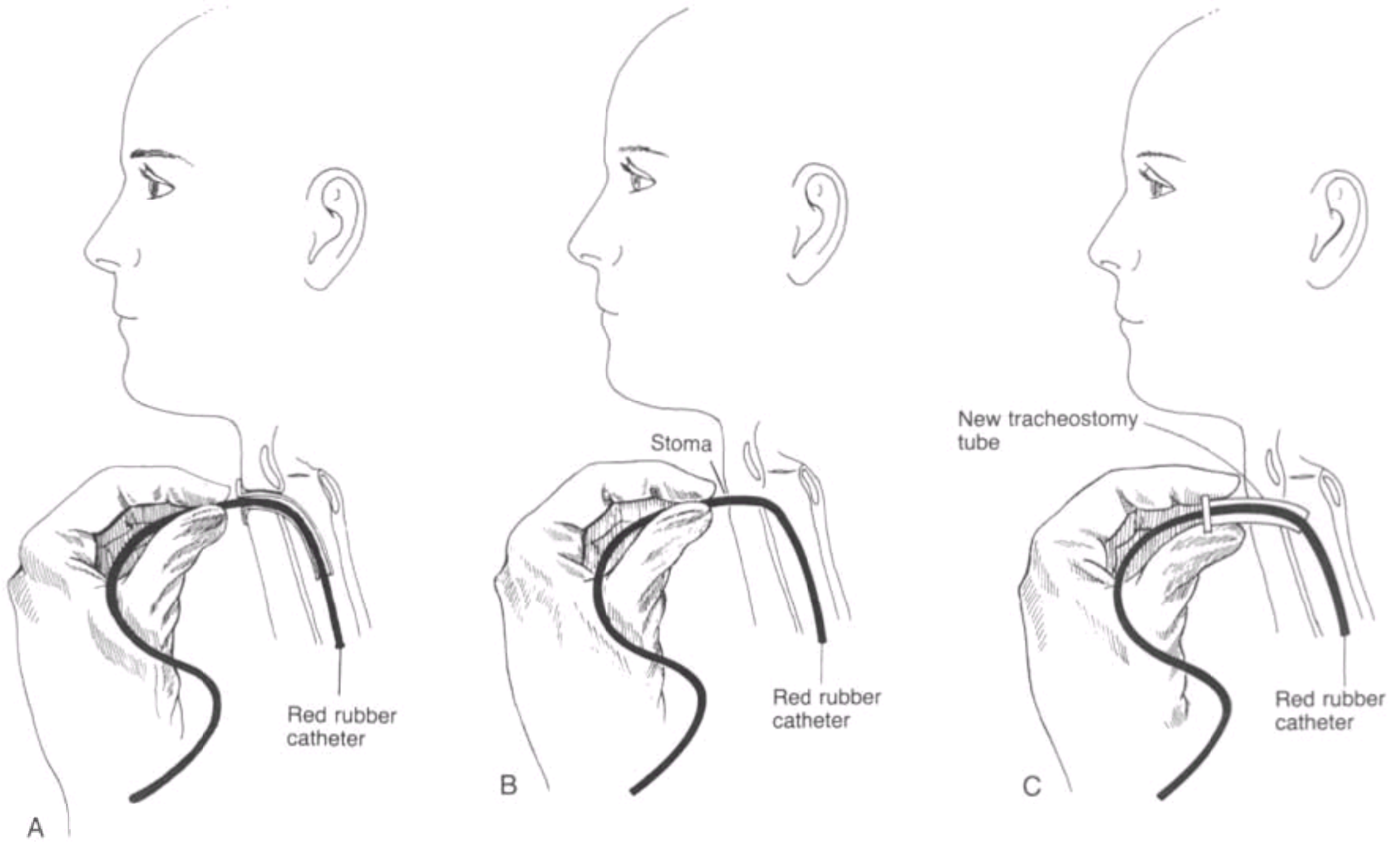


Figure 7-4 The tracheostomy tube should be inserted with a gentle rocking motion of the wrist. Insertion should follow the curve of the tube, as diagrammed above. If any resistance is met during insertion, the clinician should discontinue insertion and identify the obstruction. A pediatric laryngoscope or other light source may be helpful in visualizing the tracheal lumen prior to insertion. *(Adapted from Hackeling et al: Emergency care of patients with tracheostomies: A 7-year review. Am J Emerg Med 16:681, 1998.)*

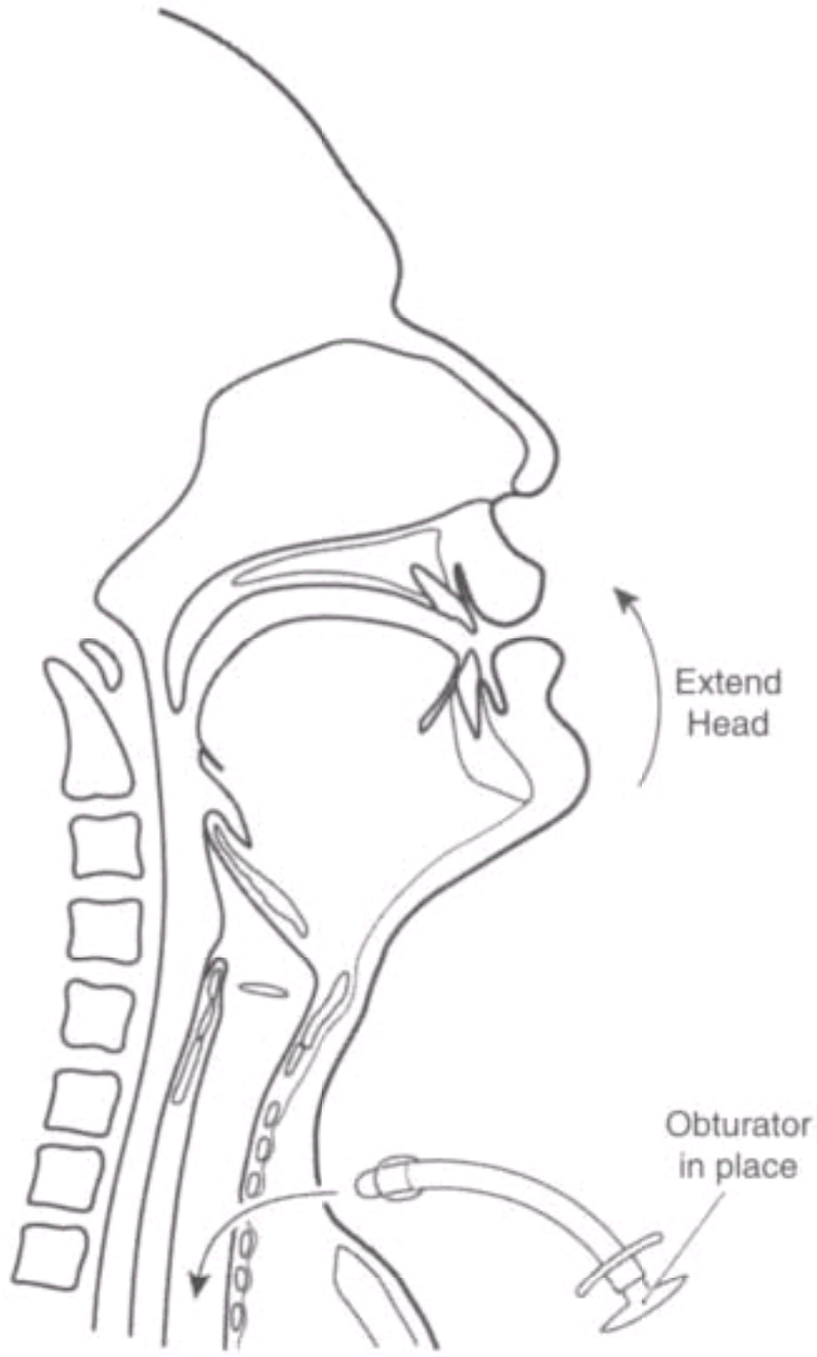


Figure 7-5 Transtracheal aspiration. *A*, Position of patient. *B*, Anatomic landmarks. *C*, Technique of puncture. The Intracath needle or J-wire introducer needle is inserted just above the cricoid cartilage through the cricothyroid membrane with its bevel up at a 45° angle to the skin. *(From Eknoyen G: Medical Procedures Manual. Chicago, Year Book Medical Publishers, 1981. Reproduced by permission.)*

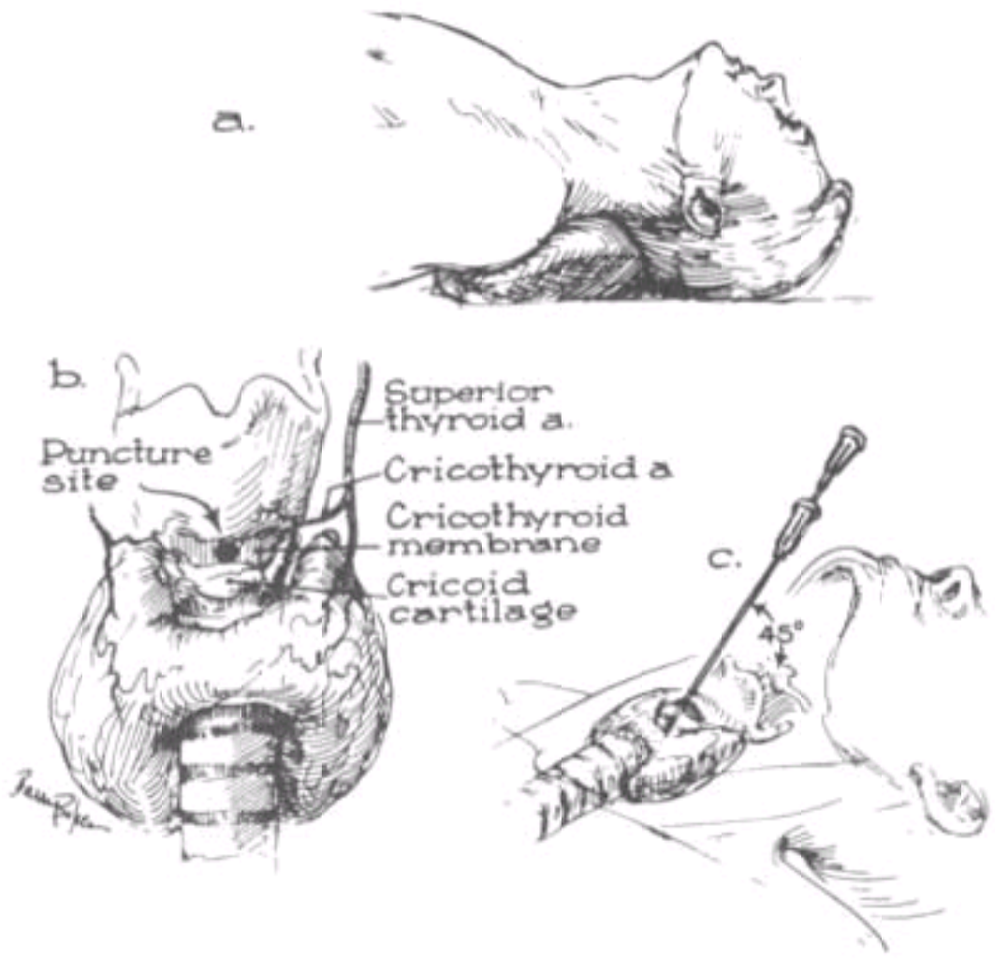


Figure 8-1 Alveolar interdependence. Note that alveoli are not round in shape; instead, they are polygons. Polygons have corners and may have two opposing surfaces that may adhere to one another via surface tension. Surfactant works to reduce this surface tension and allow alveoli to open with reduced shear stress at the junction of closed and open alveoli. Alveoli are connected via the pores of Kohn. This allows opening alveoli to pull a relatively closed alveolus open while equalizing pressure between adjacent alveoli. The central alveolus on the right is fairly closed in the upper diagram, but is pulled open by its neighbors as they expand and accept gas.

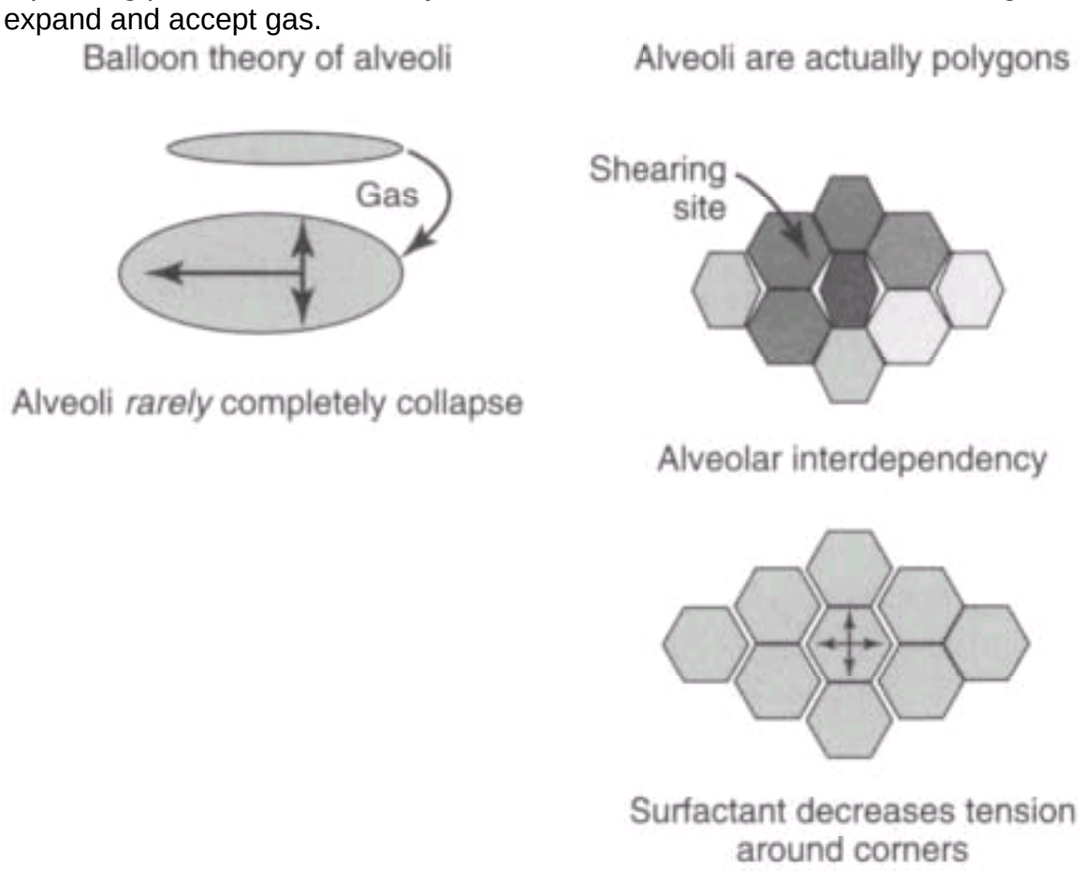


Figure 8-2 Dynamic pressure-volume loop. Note that as soon as there is delivered pressure to the airway, there is an increase in measured tidal volume. The lower arrow denotes inspiration while the upper arrow indicates exhalation. This indicates that the airways are open and do not need to be forced open by increasing the pressure in the airway. If this latter case were true, then the P-V loop would initially be flat along the X-axis.

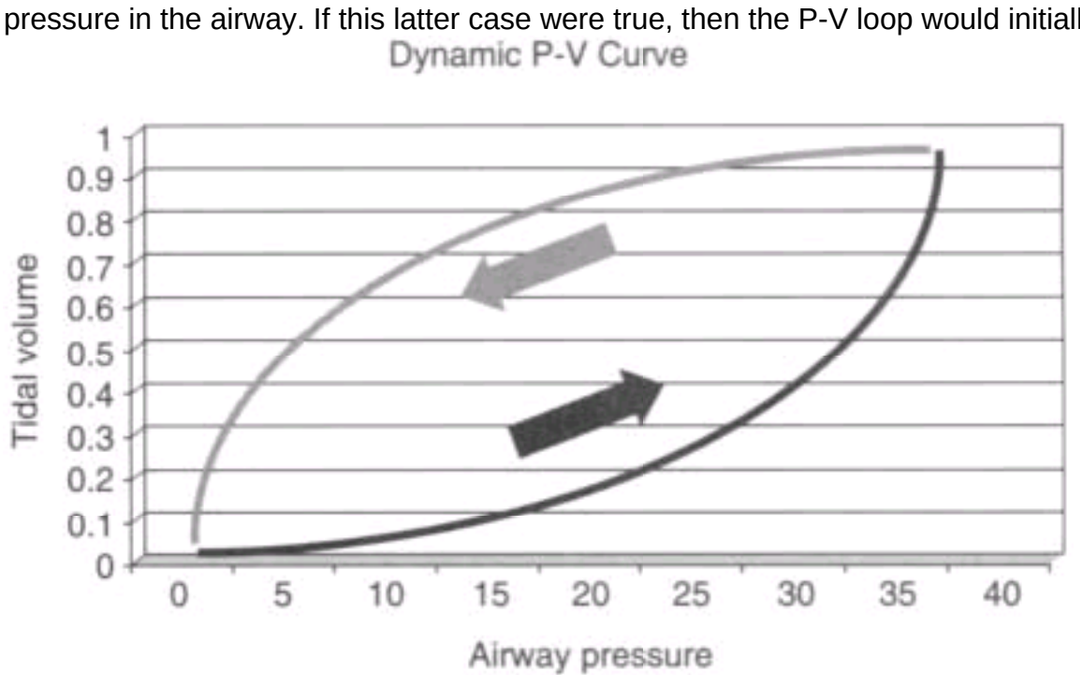


Figure 8-3 Inadequate PEEP and the P-V loop. Compare this curve to that in Figure 8-2 . Note that the loop is initially flat (lower segment) along the X-axis. Once the airway pressure is high enough to open the alveolar units, each increase in airway pressure is matched by a corresponding increase in tidal volume.

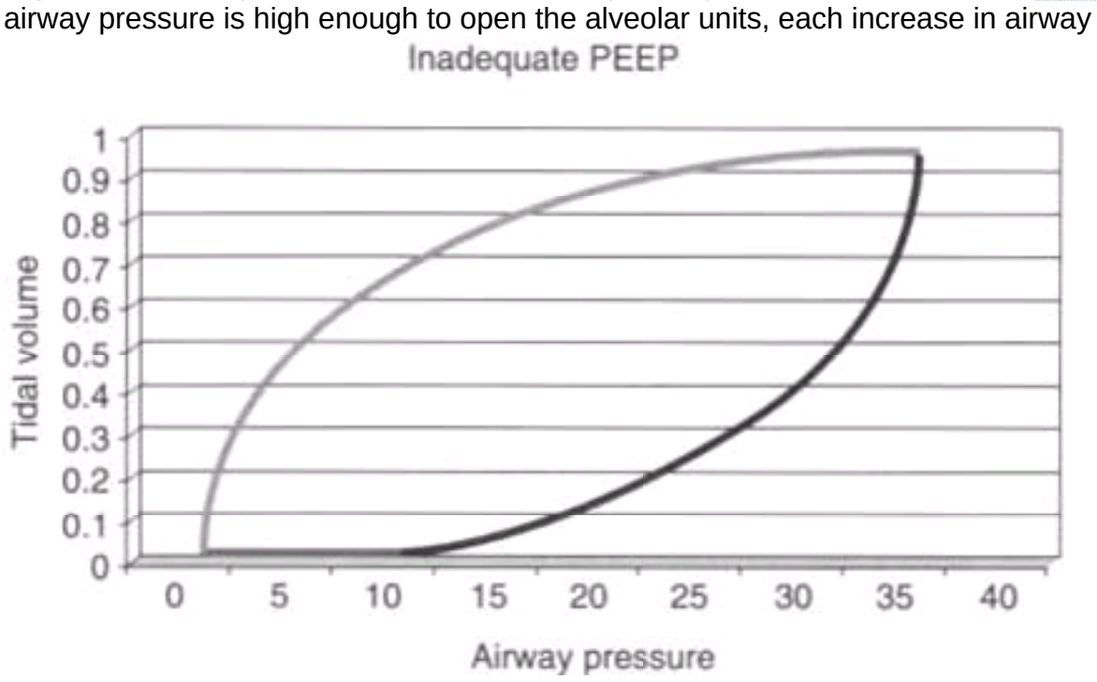


Figure 8-4 Identifying auto-PEEP with the flow-time trace. The X-axis is time while the Y-axis is flow rate in L/min. Deflections above the X-axis indicate inspiration, while deflections below represent exhalation. In this example, the flow pattern is ramp (decelerating). Note that as the respiratory rate increases (decreased time for exhalation), the flow has not yet returned to baseline (a), indicating incomplete exhalation when compared to the following breath (b).

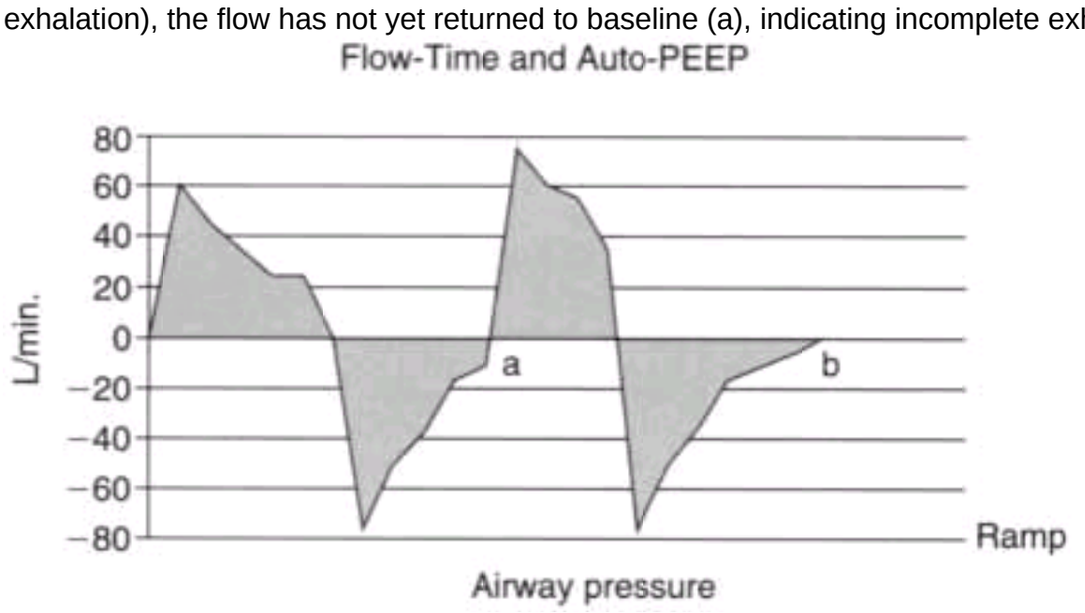


Figure 8-5 Effect of flow rate on inspiratory and expiratory times. Note that as the flow rate changes, there are corresponding alterations in the effective time for inspiration and exhalation. Deflections above the X-axis (time) indicate inspiration, while those below indicate exhalation. The delivered tidal volume for each cycle is the same, but the inspiratory and expiratory times are different.

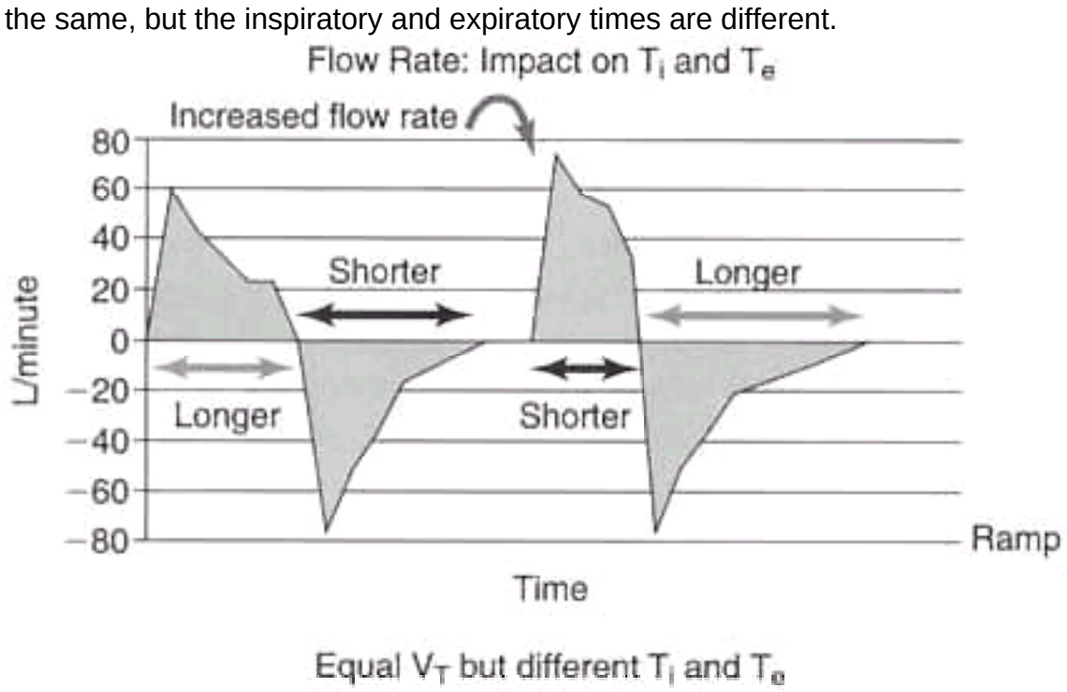


Figure 8-6 Airway Pressure Release Ventilation (APRV)—airway pressure-time and flow-time traces. Note that the peak airway pressure (P_{aw}-high) is maintained for a long period. This phase establishes oxygenation (T_{high}). There is a short period of release when most CO_2 is cleared (T_{low}). Note that the bottom trace is flow over time. The combined time for the T_{high} and T_{low} is known as a phase cycle. Note that the number of phase cycles is not the respiratory rate as patients breathe within the entirety of the T_{high}. As the release phase is initiated, the flow rate is identified as negative and is of a high rate (here approximately 7.5 L/min), consistent with significant alveolar recruitment. During the high CPAP phase, the patient is allowed to exhale (negative deflections on the flow-time trace). Thus, APRV is quite dissimilar from traditional cyclic ventilation. This unique mode is made possible by a floating valve system.

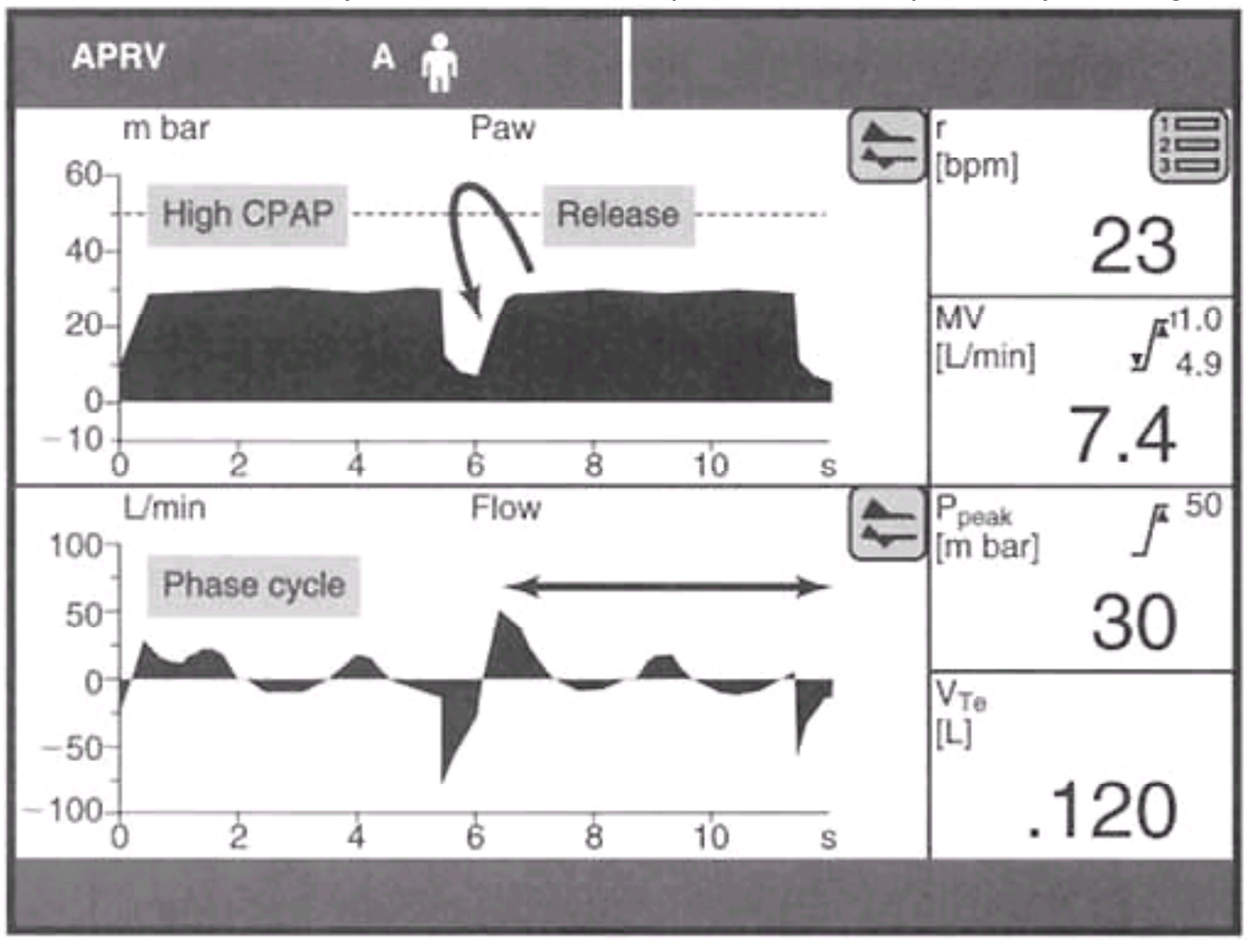


Figure 8-7 Chest geometry and prone position. Note that ovoid chest geometry results in equal volumes above and below the transverse axis leading to no change in recruited lung volume with pronation. Triangular chest geometry leads to a significant increase in lung volume with prone positioning. This is by virtue of the large volume of lung that lies posterior to the transverse plane with the patient in the supine position. Increases in cardiac volume reduce the available retrocardiac lung volume and impair the effectiveness of prone positioning with regard to pulmonary recruitment.

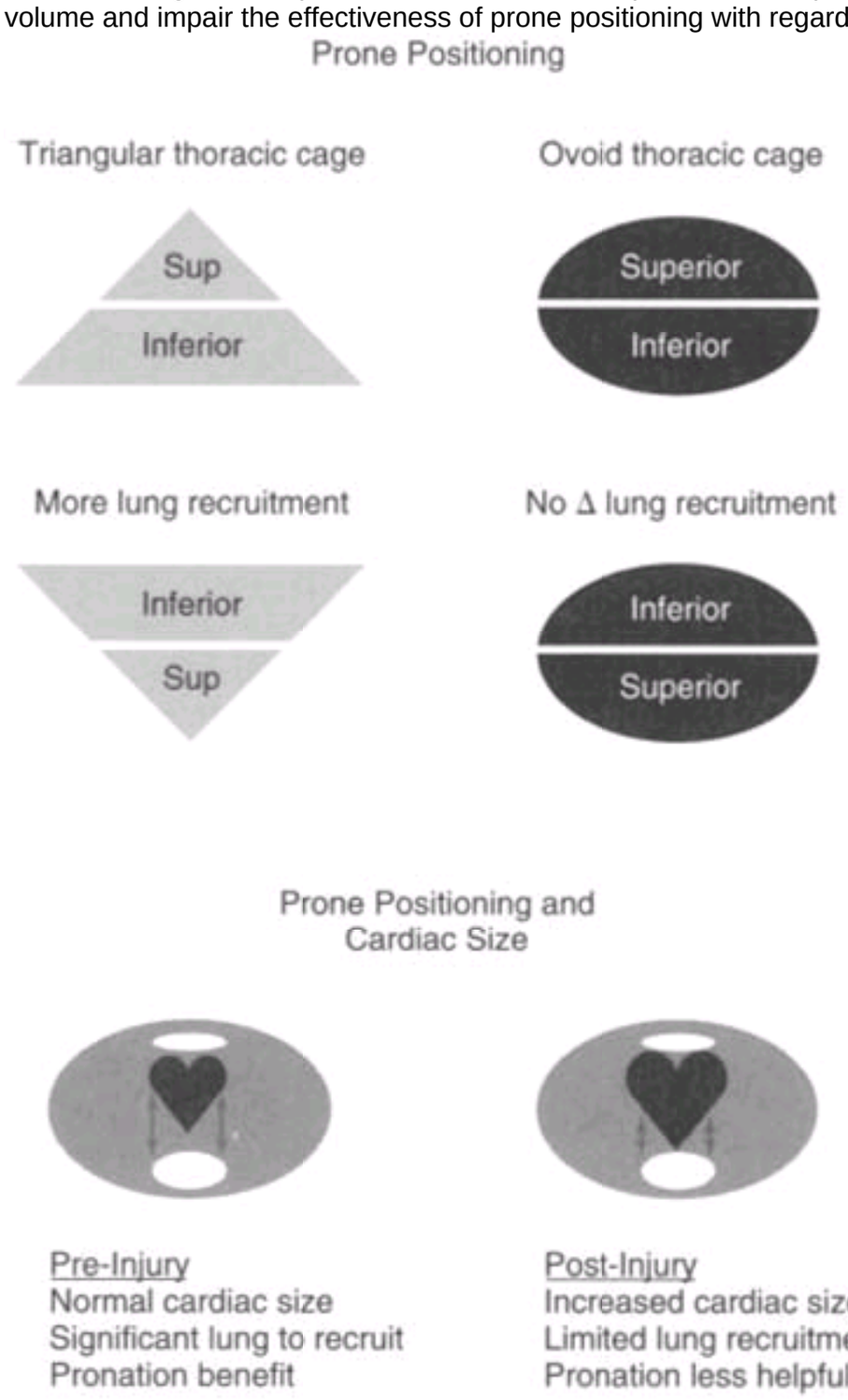


Figure 8-8 Alveolar overdistension is reflected in the increase in airway pressure without any concomitant increase in tidal volume. This pressure-volume curve pattern approximates a "bird's beak" profile.

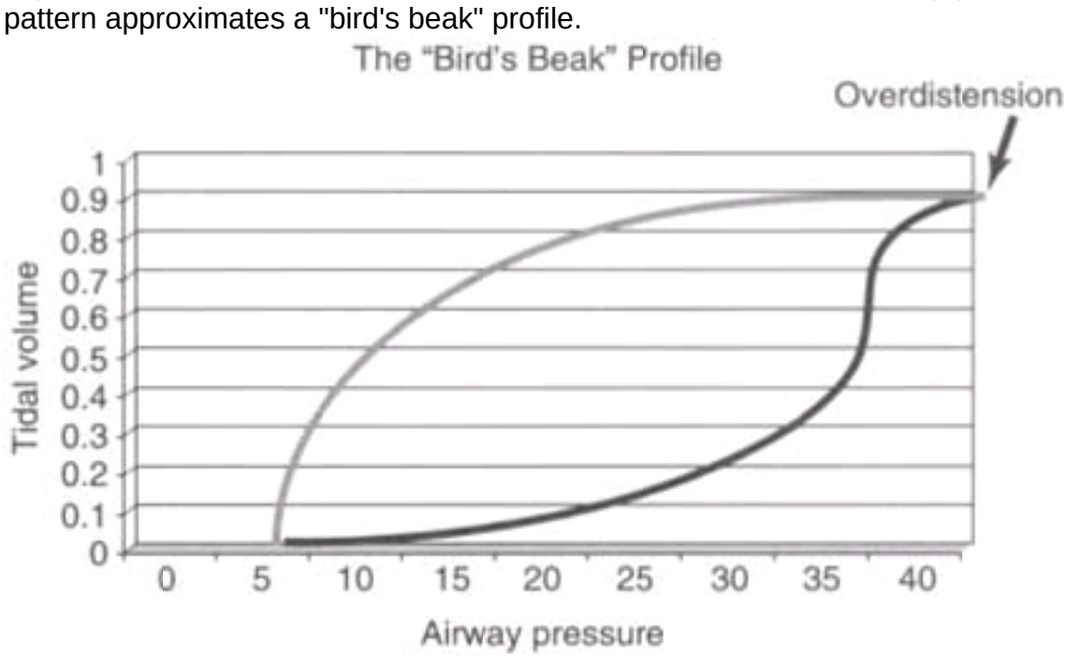


Figure 8-9 Mean airway pressure and the pressure-time trace. Note that the greater the maximum airway pressure and the longer the T_i, the greater the area under the curve (AUC) described by the positive-pressure (inspiratory) limb of the respiratory cycle. The increase in mean airway pressure (area under the curve) is the principal correlate of oxygenation in volume or pressure-cycled ventilation.

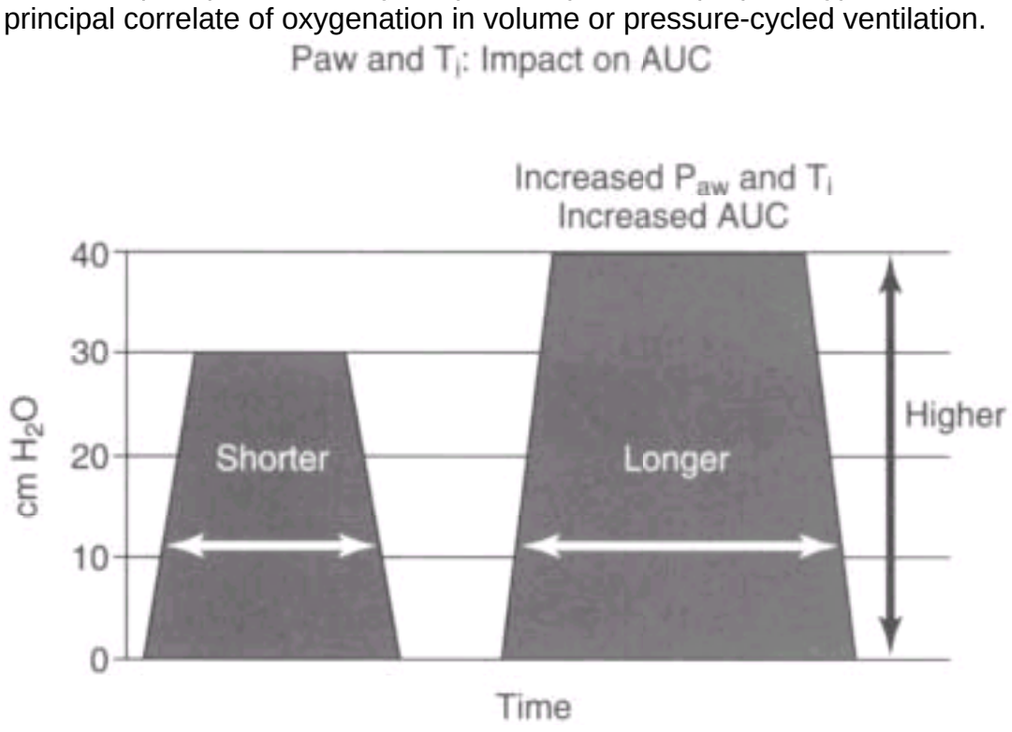


Figure 8-10 Confounders in the diagnosis of pneumonia. Fever, leukocytosis, radiographic infiltrate, and sputum production do not necessarily indicate the diagnosis of pneumonia. Multiple other causes should be considered as well so that one does not apply antibiotics when there is no infectious agent to address. ALI = acute lung injury, ARDS = acute respiratory distress syndrome.

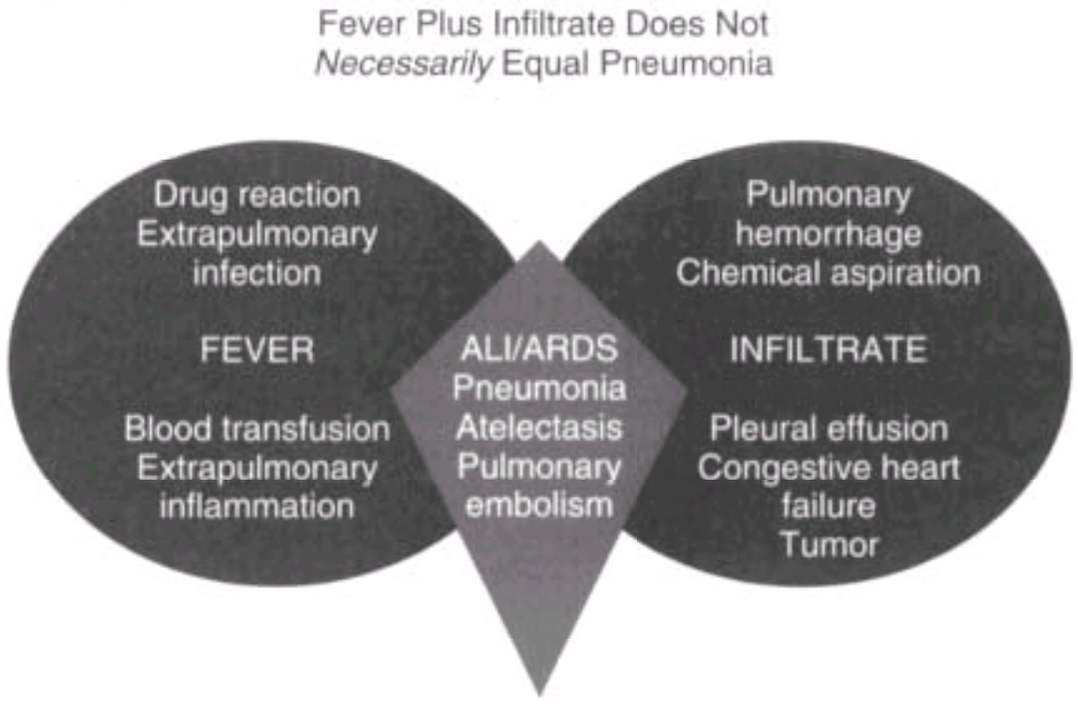


Figure 8-11 The Bispectral Index monitor (Aspect Medical, Nantucket, MA). This is a pole-mounted device that attaches to the patient's forehead and provides a modified single-lead electroencephalogram whose power spectrum undergoes a Fourier transformation to yield a numeric representation of the level of sedation.

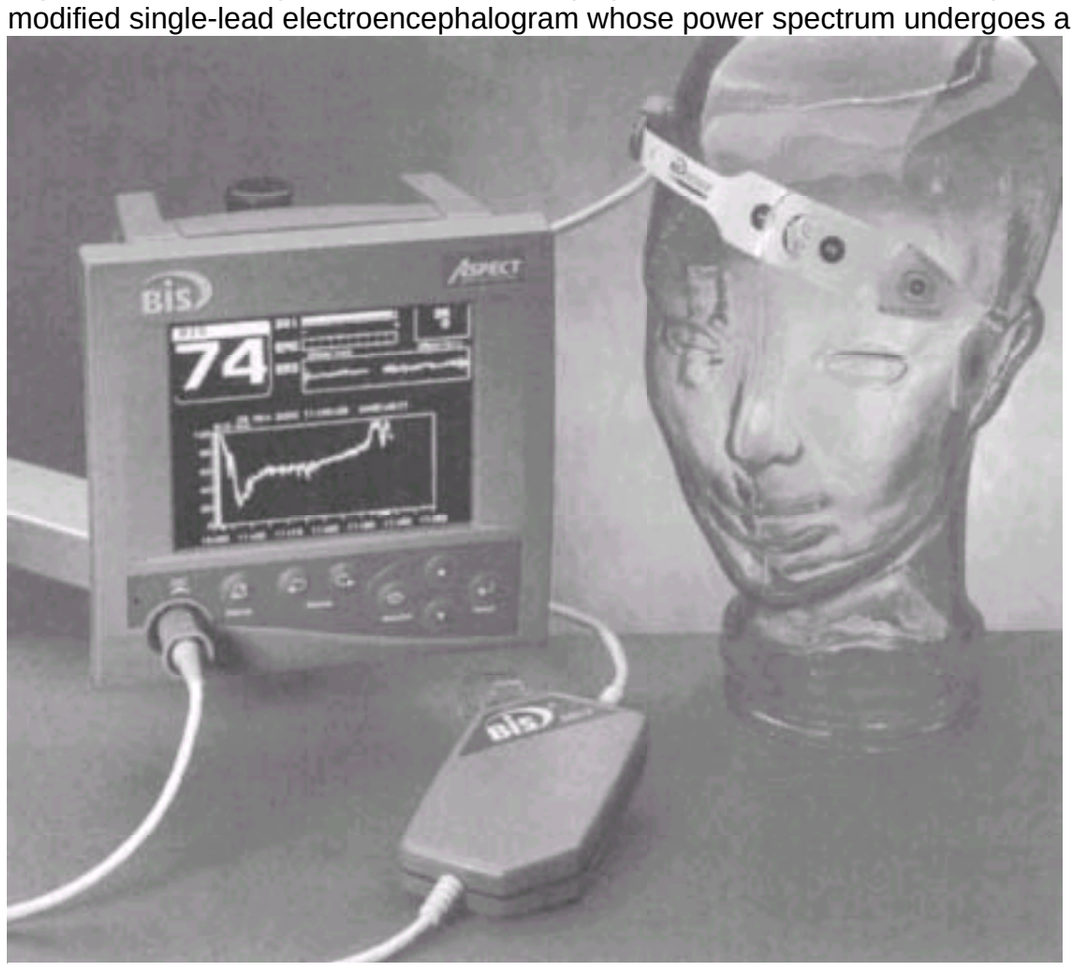


Figure 8-12 Volume guarantee ventilation. Note that the ventilator uses the prior breath to determine how much support is required to achieve the desired tidal volume and remain within the set pressure limit.

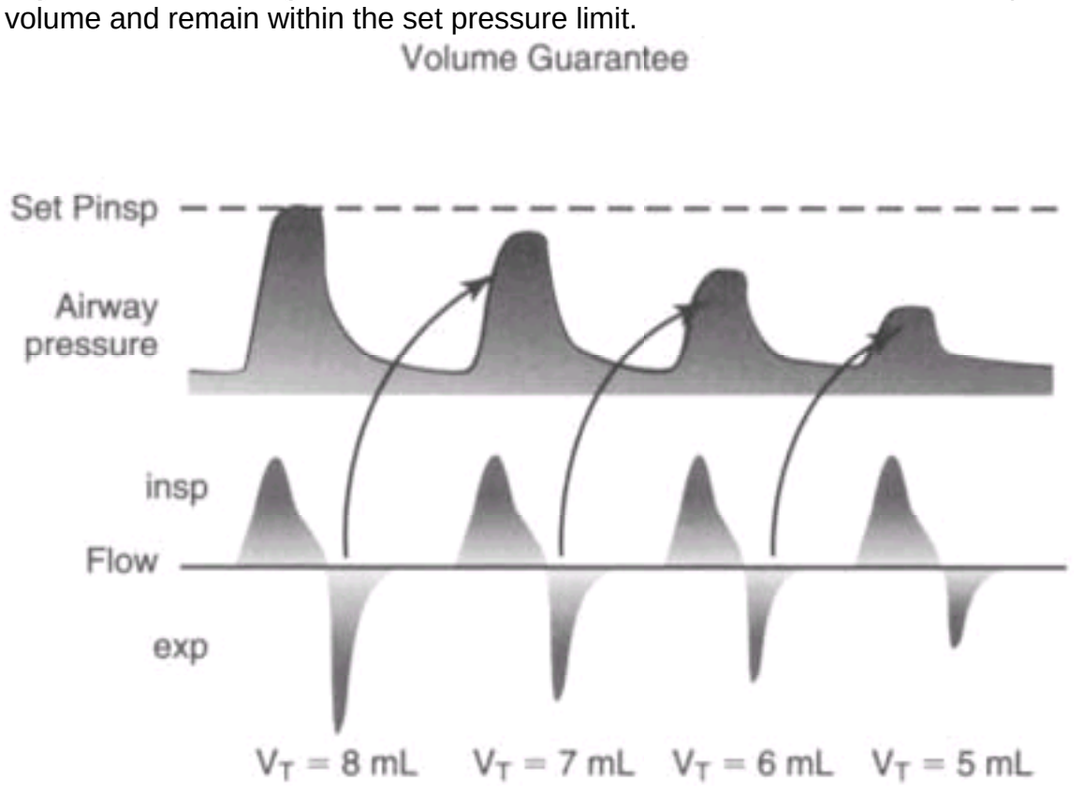


Figure 8-13 Pressure support volume guarantee ventilation. As the compliance of the lung (C lung) improves, the positive inspiratory pressure (PIP) decreases while maintaining a constant tidal volume (V_T). When the pressure support volume is reduced to an acceptable minimum level, weaning has been achieved and the patient should be evaluated for liberation from mechanical ventilation.

PSV + VG: The Concept of "Autoweaning"

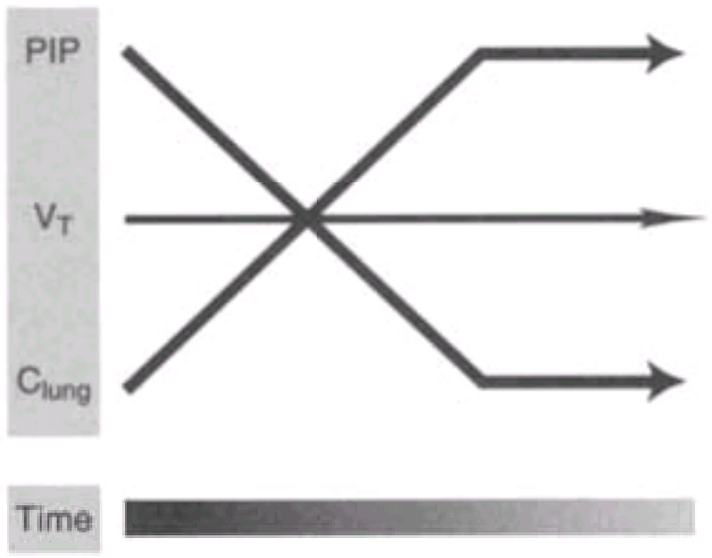


Figure 8-14 *Continuous positive airway pressure* (CPAP) mask. This is a nasal CPAP mask and has a low-pressure bladder around the rigid mask. There is a hook-and-loop closure strap system to secure the mask to the patient. Care must be taken to ensure a proper fit. The goal is to achieve a tight seal without creating undue pressure on the skin, nose, or other bony prominences of the head, thus avoiding pressure necrosis.

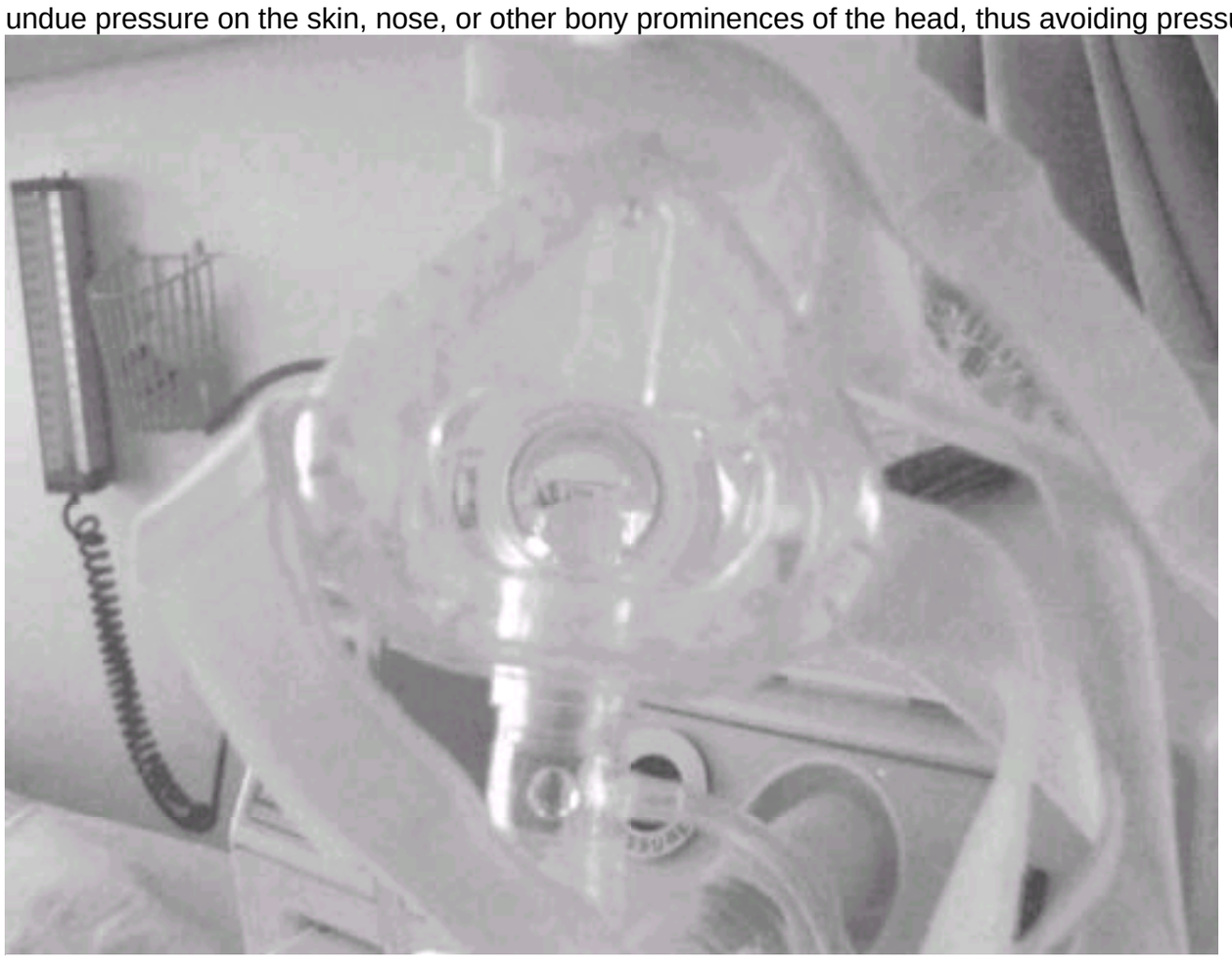


Figure 8-15 The BiPAP machine. Each hospital may have a different BiPAP device. The key features are an O_2 connection to bleed-in O_2, a rate control, and separate controls for the level of inspiratory positive pressure (IPAP) and expiratory positive pressure (EPAP), as well as separate timing controls for IPAP and EPAP. Clinicians should familiarize themselves with the unique features of the device available at their institutions.

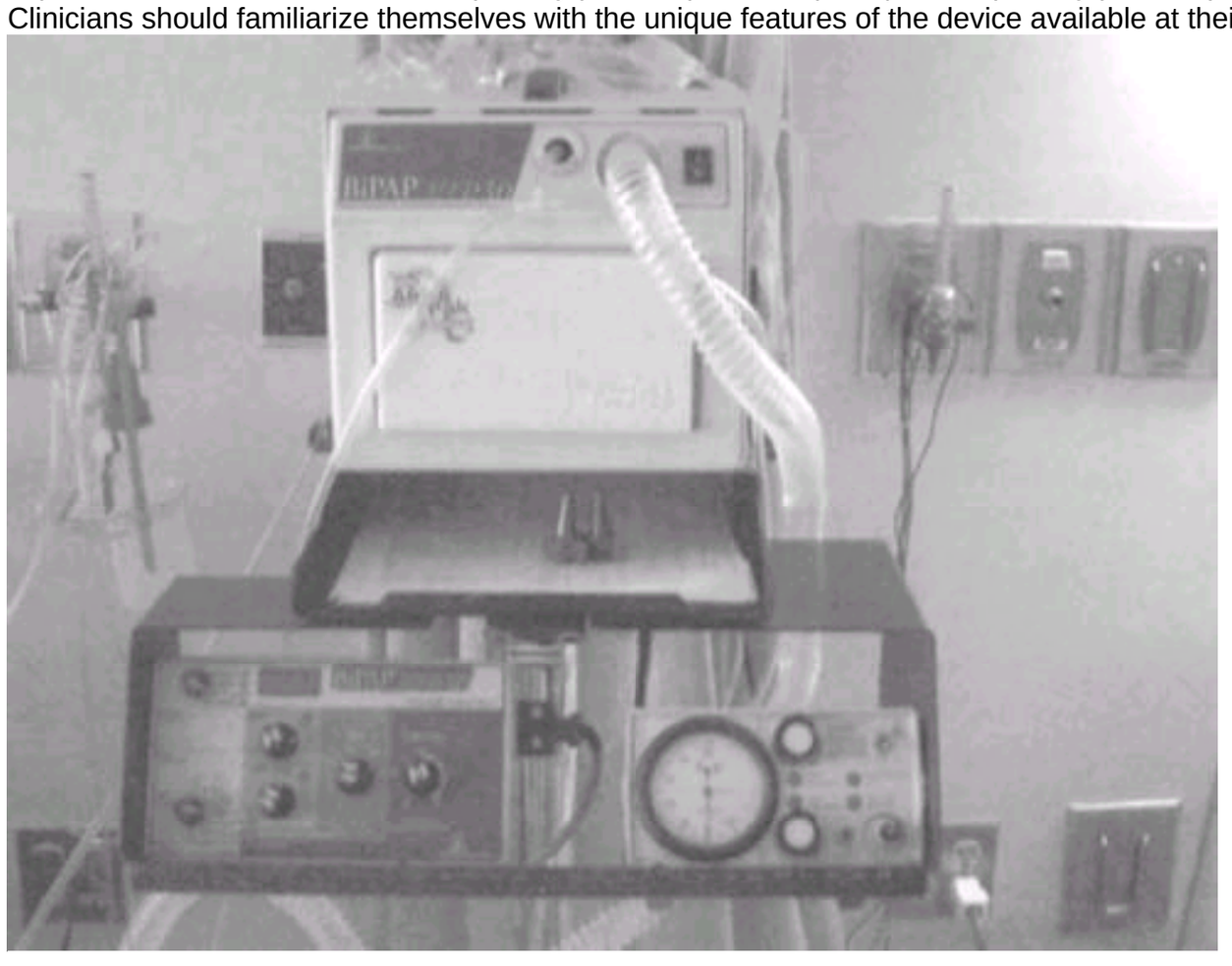


Figure 8-16 The end-tidal CO_2 ($ETCO_2$) trace. This diagram of an $ETCO_2$ capnograph represents normal exhalation flow dynamics. As exhalation progresses, the measured CO_2 rises and eventually plateaus. At the end of the plateau, immediately before the downstroke of the trace is the point at which the $ETCO_2$ value is measured and reported (i.e., point "D"). No other values are reported on the $ETCO_2$ capnometer. Thus in the absence of the capnograph, all other information is unavailable to the clinician. Abnormalities in endotracheal tube position, blockage of the airway (complete or partial), as well as correct endotracheal tube placement, are identifiable by evaluating the $ETCO_2$ tracing. *(Reproduced with permission from Drager Medical.)*

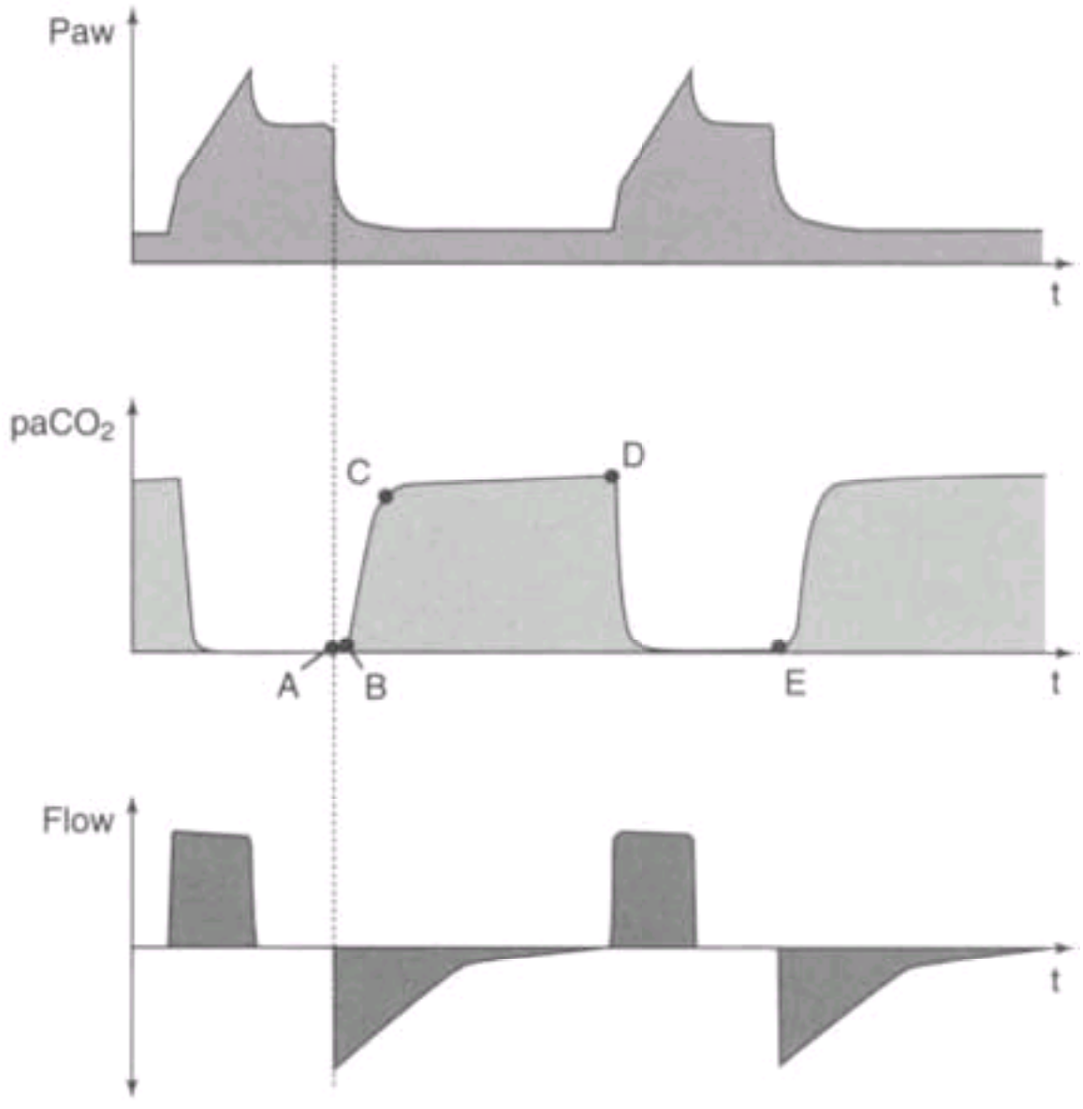


Figure 8-17 The $ETCO_2$ tracing in COPD—identification of auto-PEEP. In this diagram of an $ETCO_2$ trace in a patient with COPD who is being ventilated at a high respiratory rate, one may detect the presence of auto-PEEP based on the abnormal expiratory flow pattern. A similar suggestion could be obtained by interrogating the flow-time trace as well.

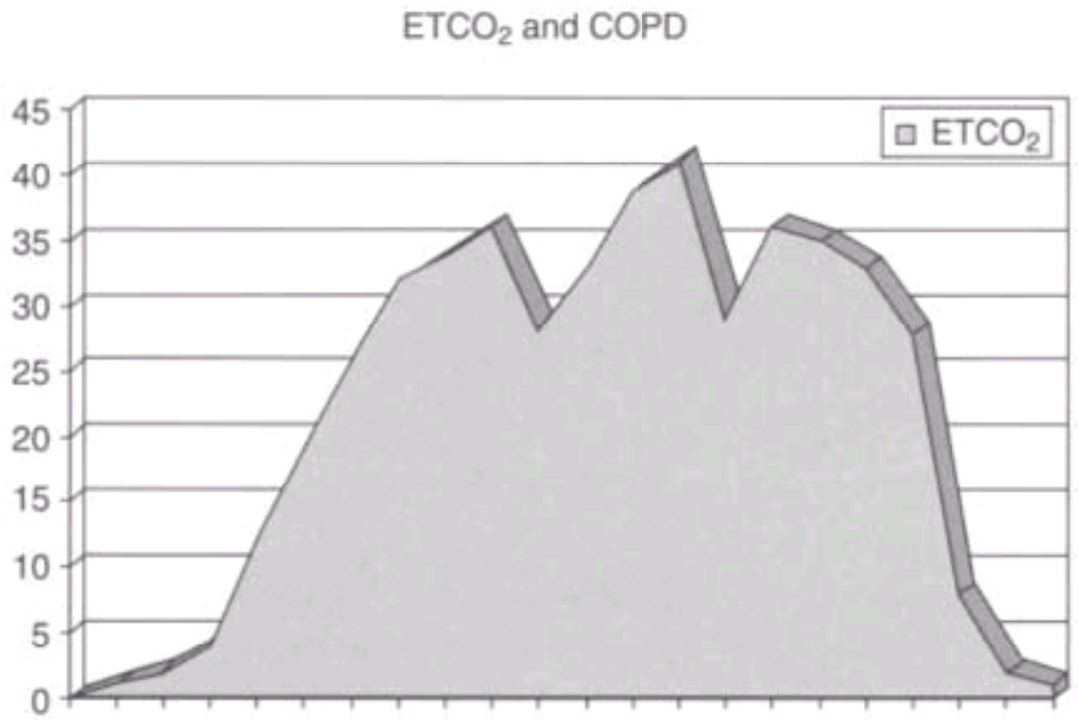


Figure 8-18 The Puritan-Bennett 7200 Model ventilator. Note that on this particular ventilator, the controls are grouped into three distinct areas of the ventilator faceplate. To the left of the picture are display areas in green that indicate pressures (top), rate and I:E ratio (middle), and volumes (bottom). At the very top of the green section is an analog pressure meter to confirm pressures in the case of a liquid-crystal display (LCD) malfunction. The center portion (blue) houses the data entry keypad, the alarm limit-setting buttons, waveform selection, sighs, and 100% O_2 for suctioning button. Note that the "++" button is the selector to change between options that are displayed in the horizontal LCD bar above the keypad. Function 10 is the function to set the pressure support and is accessed by using the "++" key until the number 10 appears. One must then press Enter to access the function. The top of the blue section has the PEEP/CPAP knob, as well as the displays for peak flow, FIO_2 , set rate, and tidal volume. The gray section to the right indicates the alarm status as well as the alarm silence button.

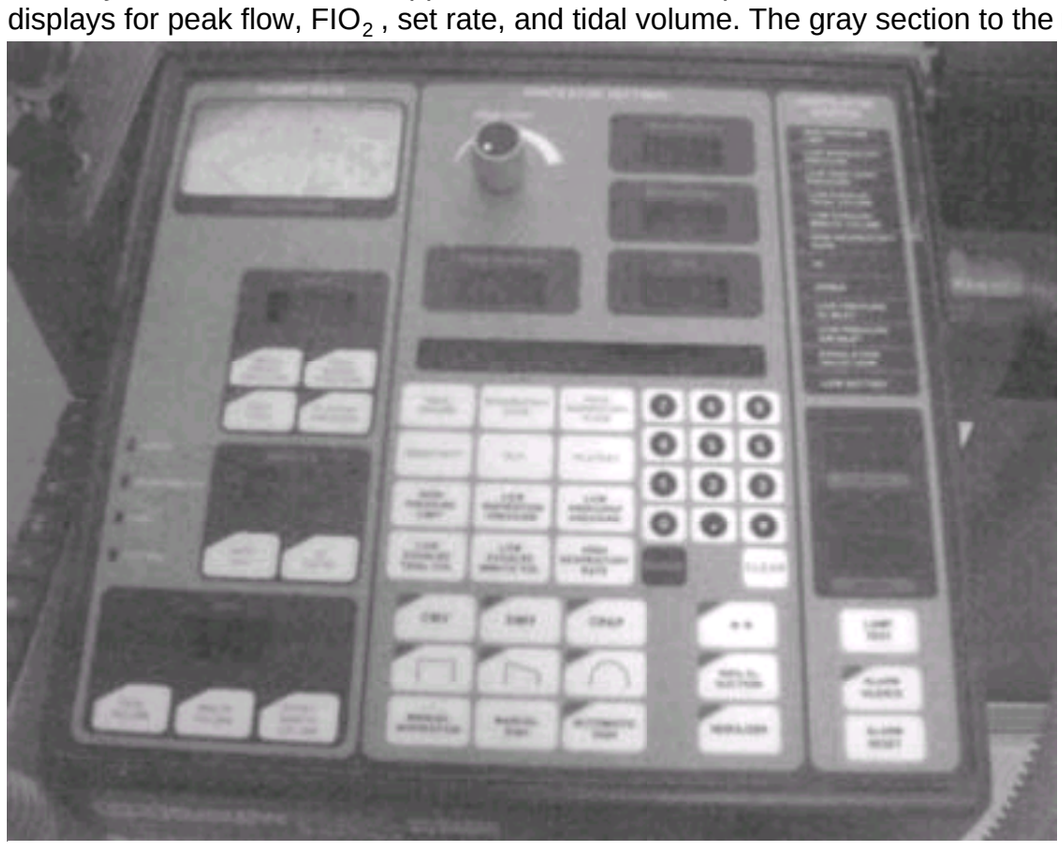


Figure 8-19 Faceplate for the Drager Evita 4 Pulmonary Workstation. Note that the liquid-crystal display (LCD) screen is colored and has controls embedded in the screen to adjust the individual mode settings. The righthand controls on the front of the device control the kind of screen display, the silence button, and the standby button.

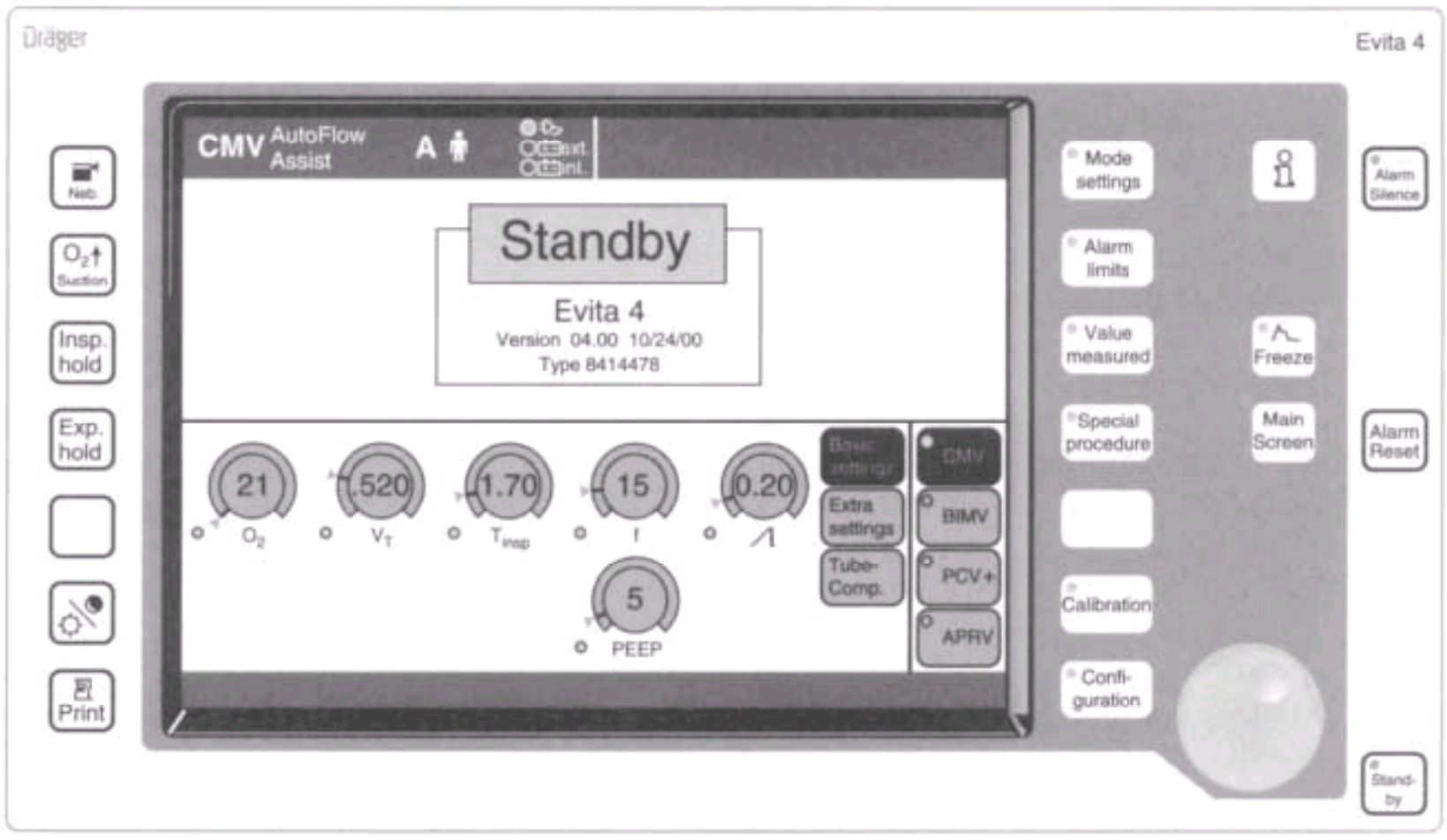


Figure 8-20 The pressure-volume curve on the Adult Star ventilator. Note that the curve is identical to the type of curve one would obtain on the E $_4$ with the exception of color and the ability to freeze a reference curve for comparison. Here it is accompanied by the flow-time loop, as well.

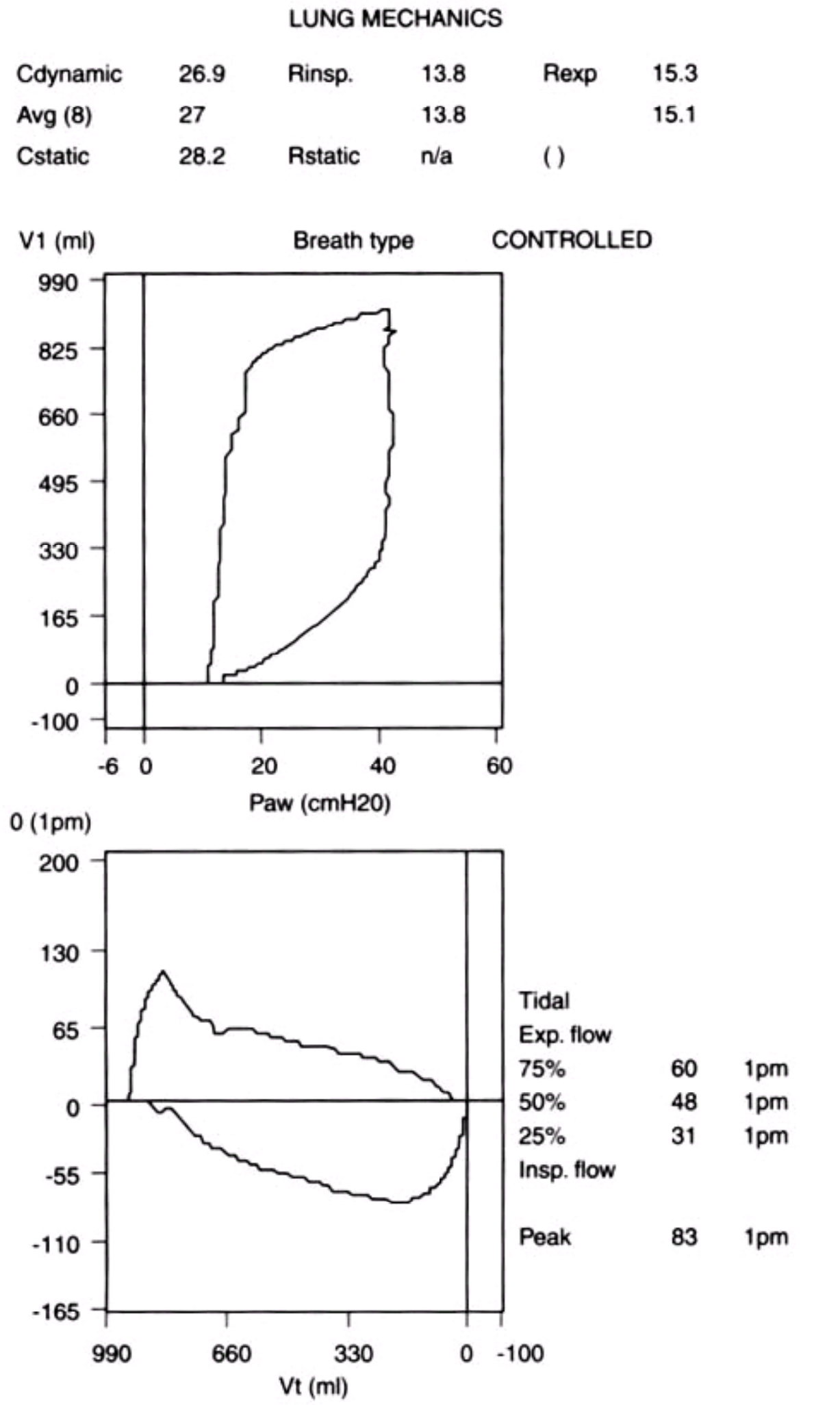
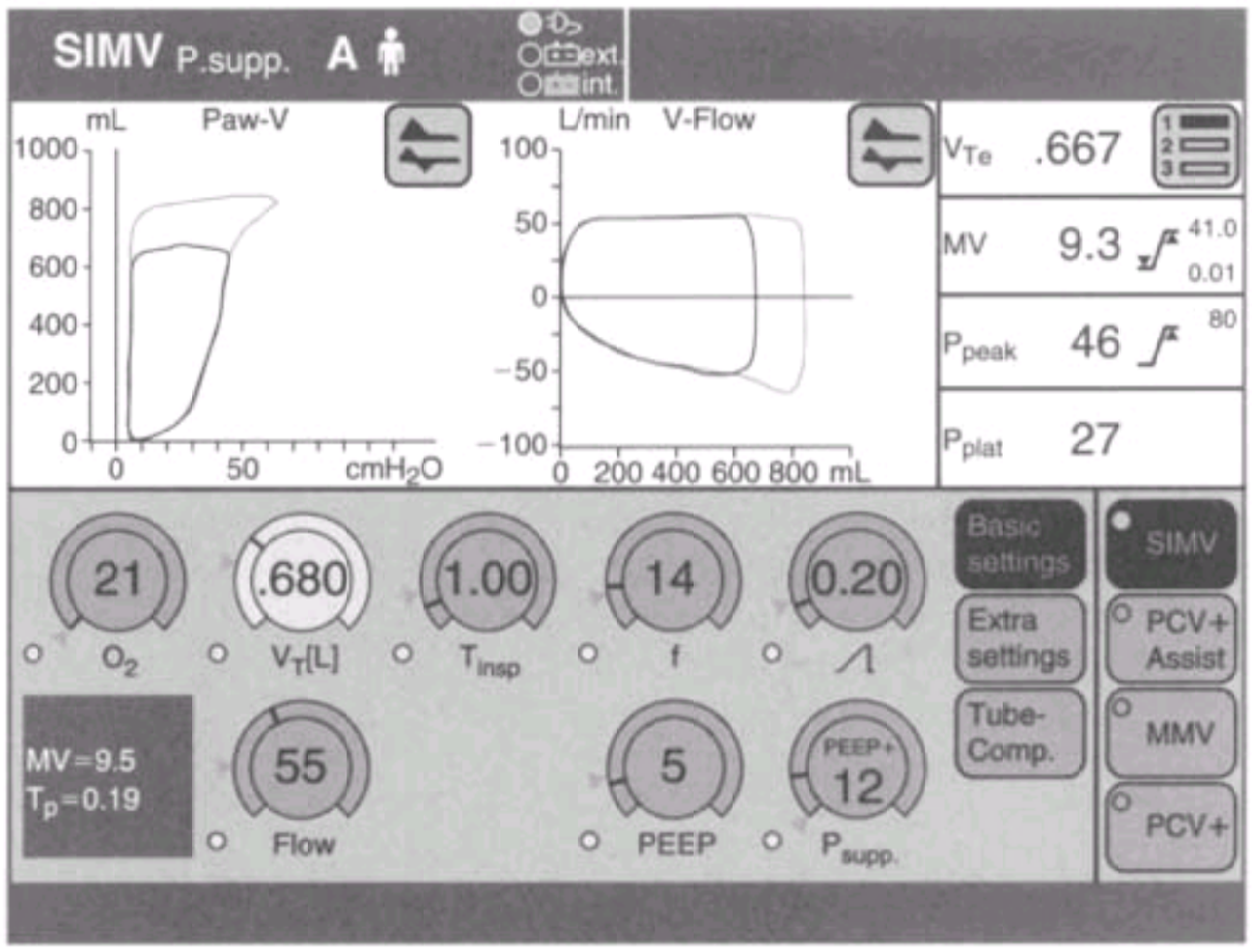


Figure 8-21 Pressure-volume curve on the E_4 . This display may be brought up as a solo display, or integrated into other multi-display screens as indicated above. One accesses the menu on the lower right of the screen by activating the "values measured" button on the right of the faceplate (not the liquid-crystal display [LCD] screen). Then press Loops. The device will either display a continuous set of loops, or a reference and a single breath (yellow button illuminated on the appropriate green icons).



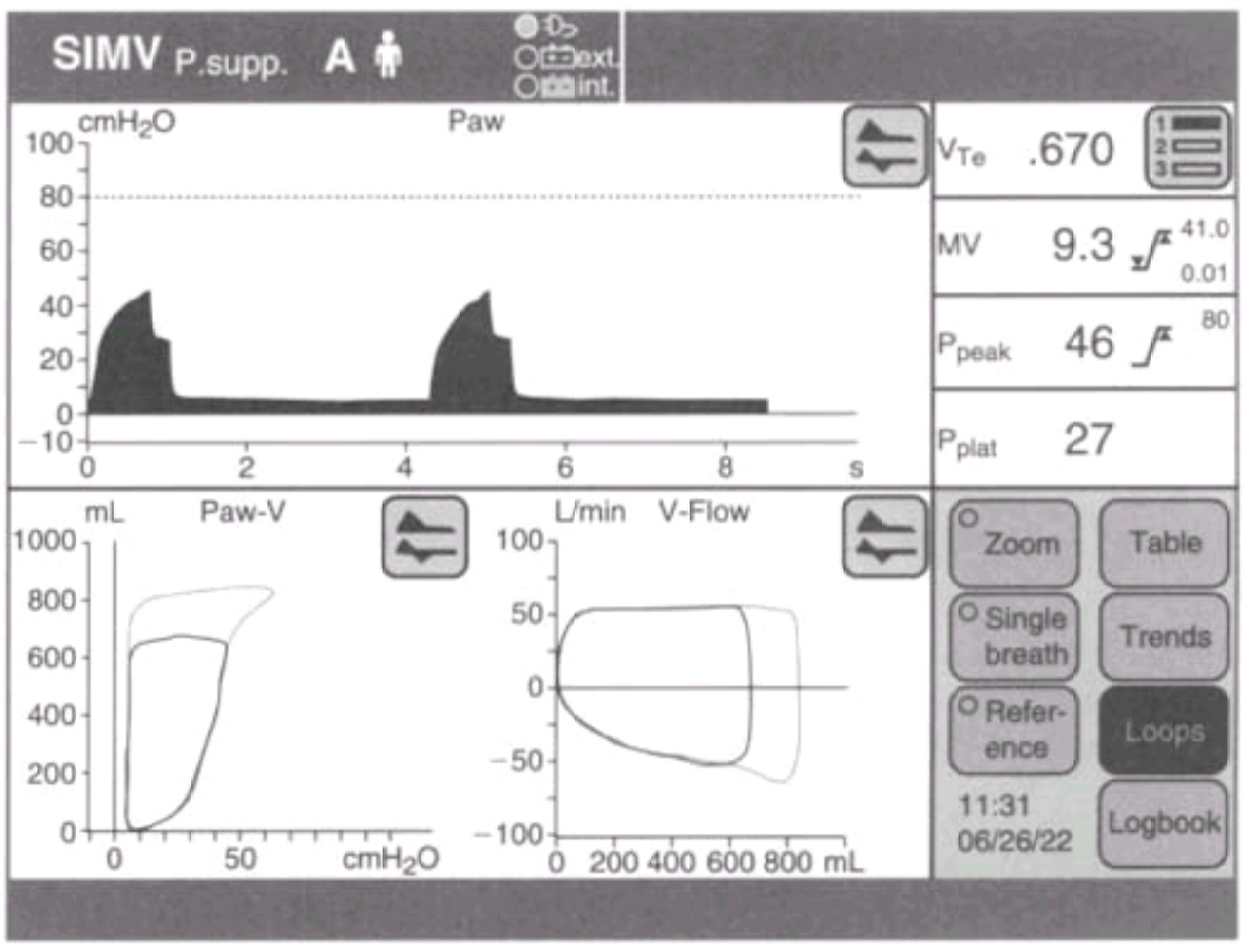


Figure 8-22 Intrinsic PEEP (PEEPi). The E_4 will measure the intrinsic PEEP as well as the trapped volume responsible for that PEEPi. The function is accessed by using the "special procedure" button on the righthand side of the faceplate, activating the "start" button on the liquid-crystal display [LCD] screen, and then depressing the knob to initiate the procedure. In this example, the PEEPi is 0.4 cm H_2O pressure and represents a volume of 155 mL (0.155L) of gas.

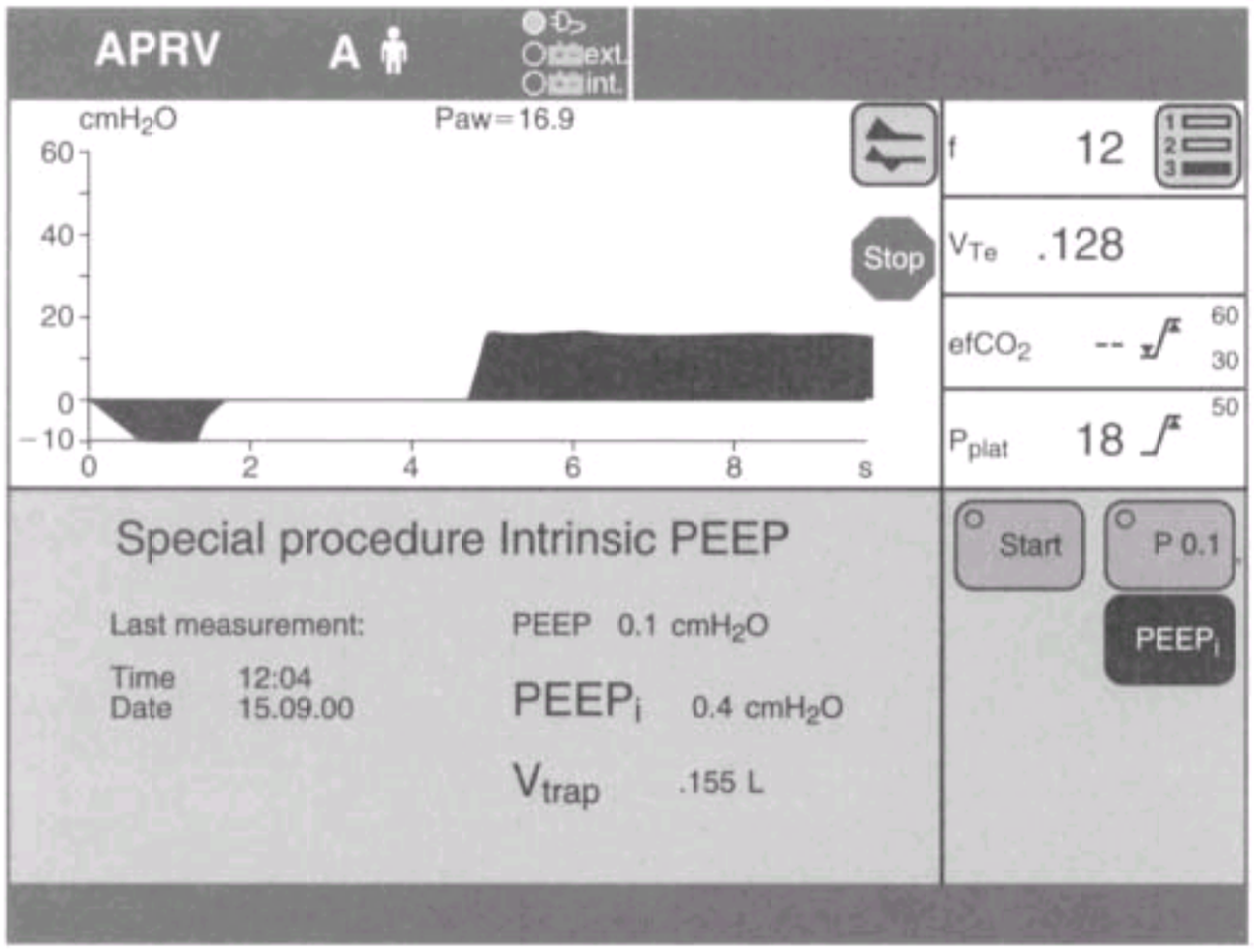


Figure 9-1 Schematic of relationships between the parietal and visceral pleura.

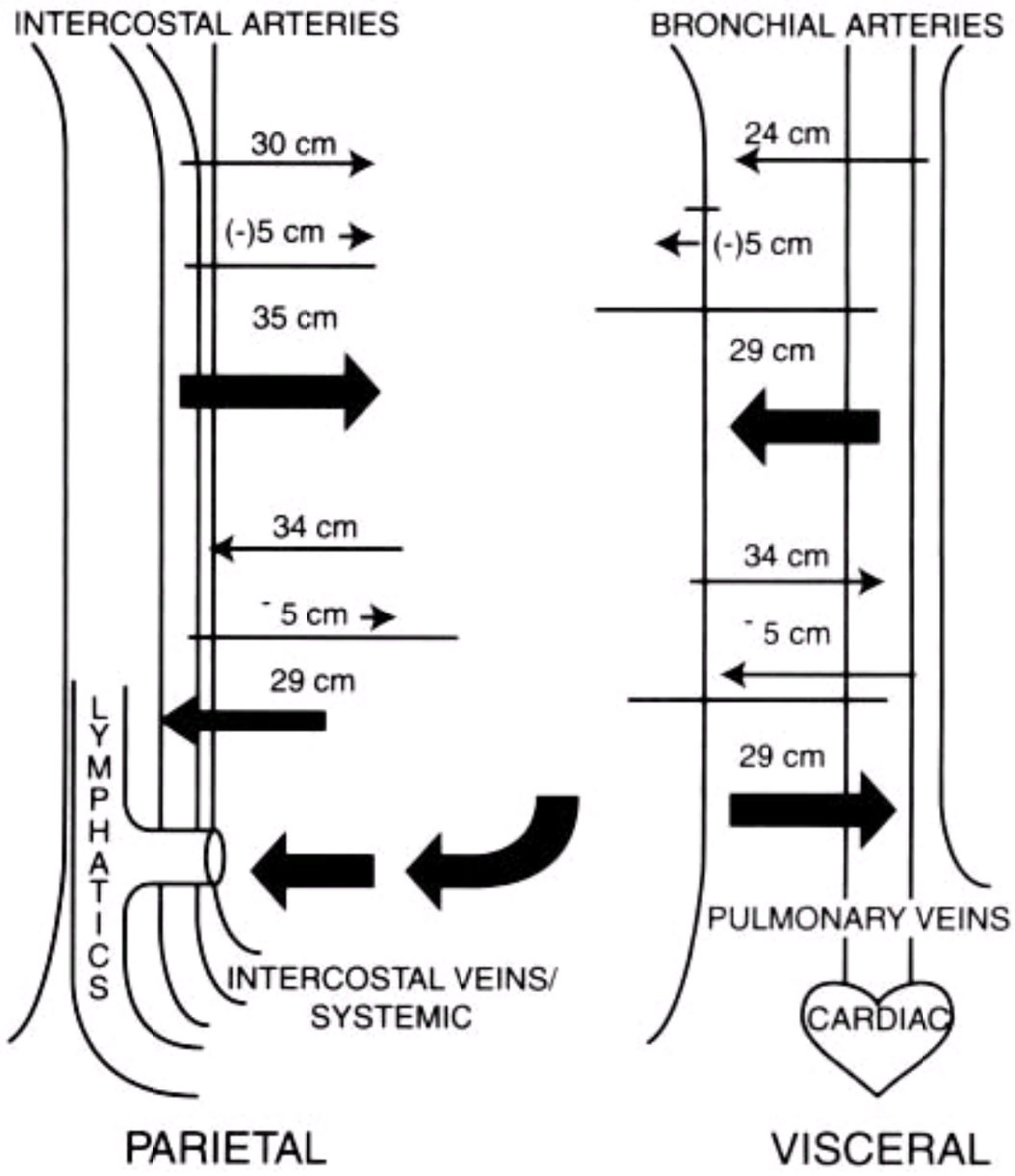


Figure 9-2 Right-sided pleural effusion. Posterior-anterior (PA) radiograph demonstrates blunting of right costophrenic angle. Both PA and lateral radiographs demonstrate the appearance of a meniscus.

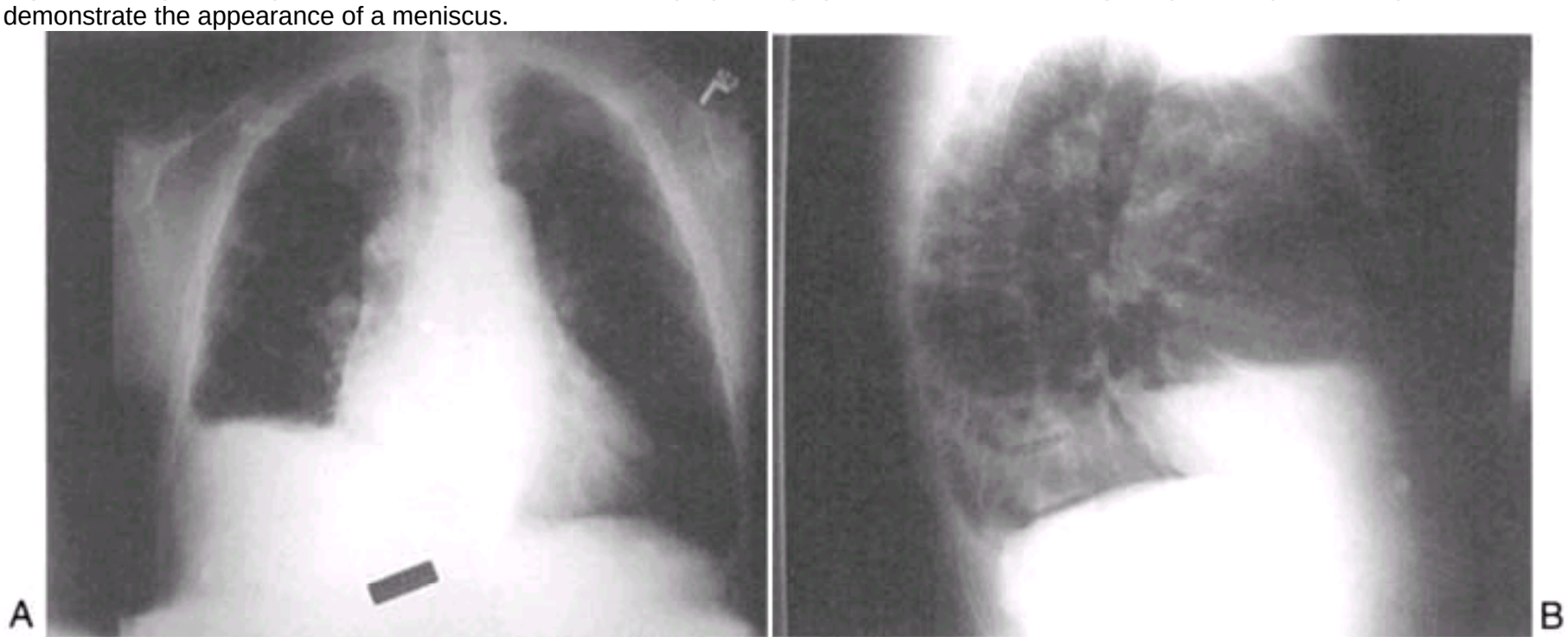


Figure 9-3 Loculated pleural effusion. This posterior-anterior radiograph demonstrates the D-shaped appearance of a right-sided loculated pleural effusion.

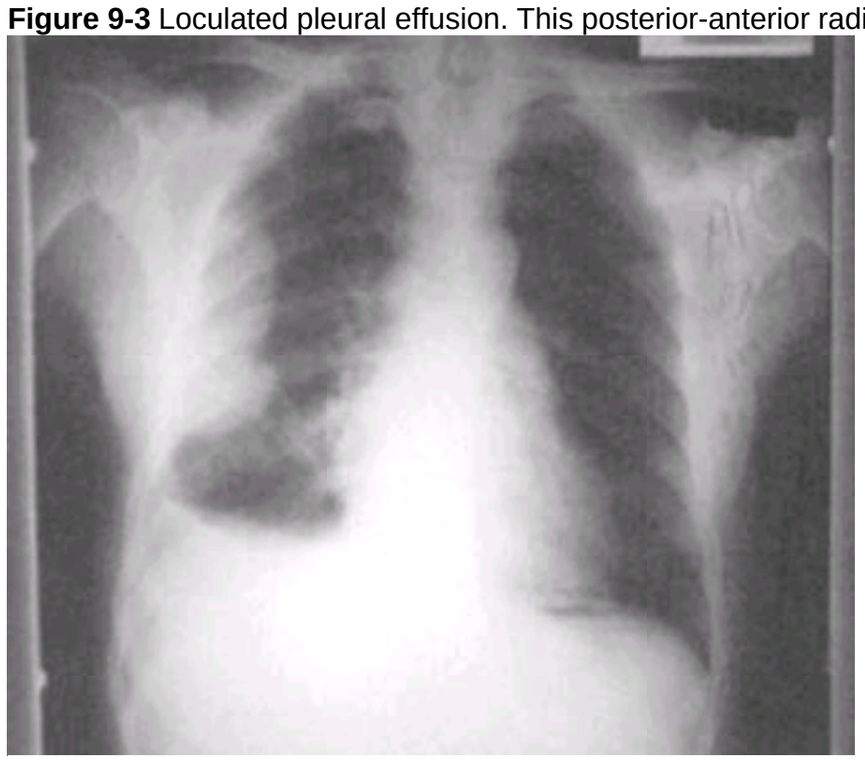


Figure 9-4 Subpulmonic effusion. This posterior-anterior radiograph demonstrates an elevated left hemidiaphragm that peaks more laterally than expected. A decubitus radiograph in the same patient demonstrates the free-flowing fluid.

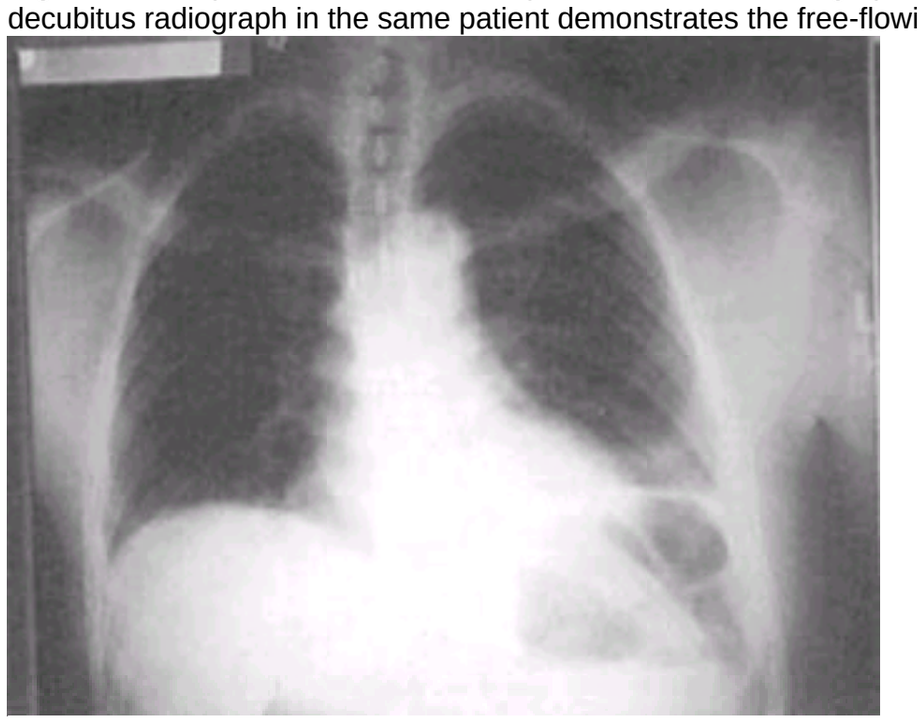


Figure 9-5 Bilateral decubitus radiographs. This patient's right-sided pleural effusion appeared on the posterior-anterior radiograph. Bilateral decubitus views demonstrate the free-flowing nature of the effusion and allow visualization of the lung parenchyma.

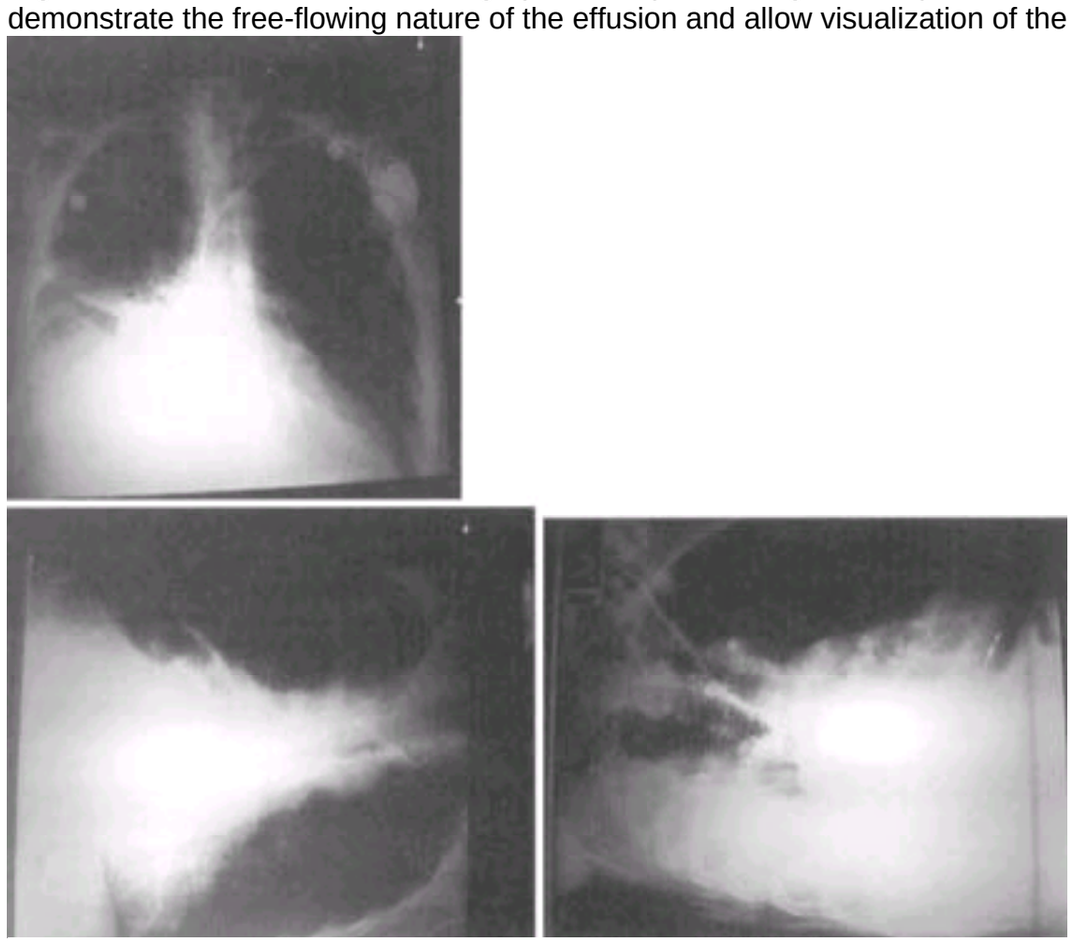


Figure 9-6 Massive pleural effusion. A repeat radiograph after thoracentesis demonstrates underlying mass.

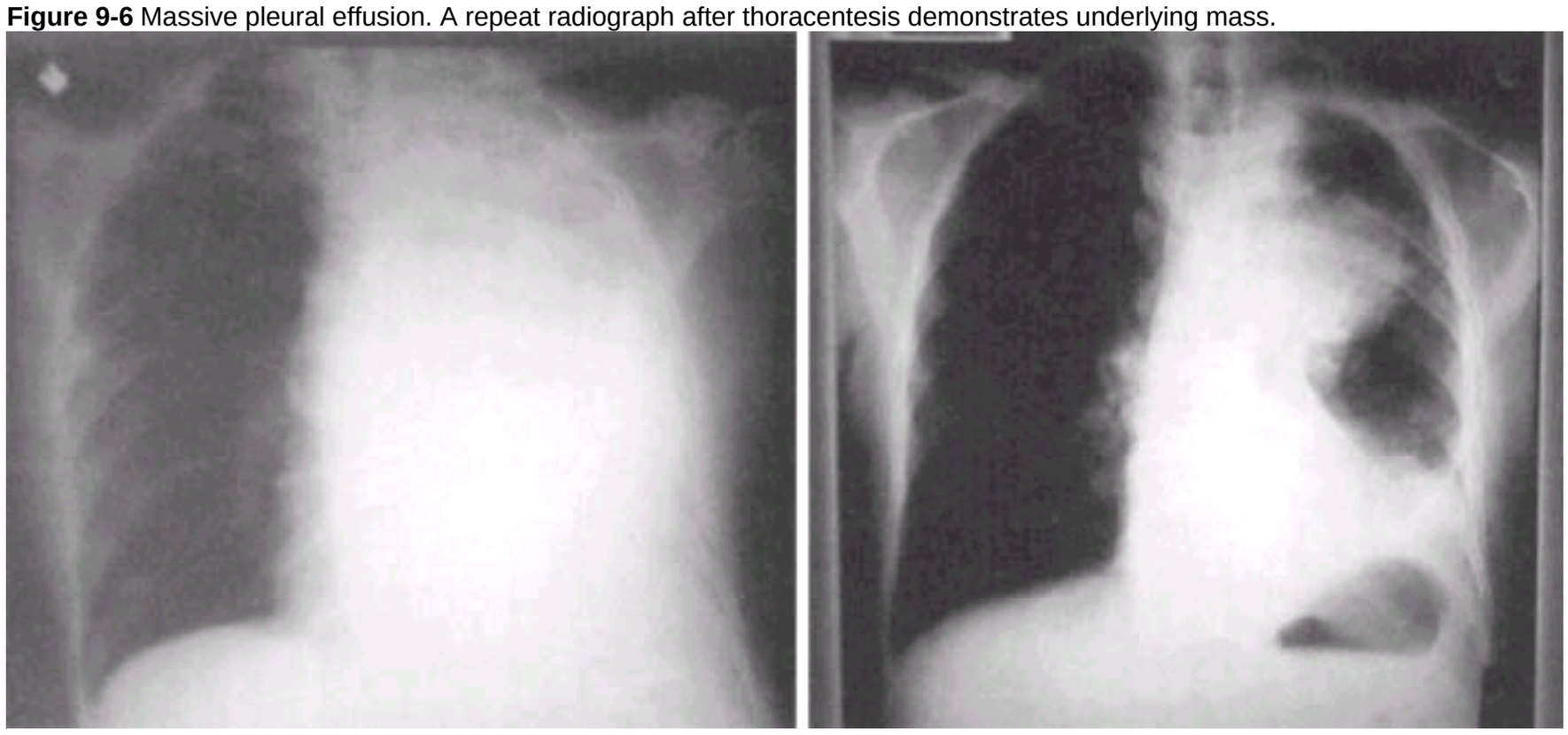


Figure 9-7 Pleural effusion. Sickle appearance of a free-flowing pleural effusion on computed tomography scan.

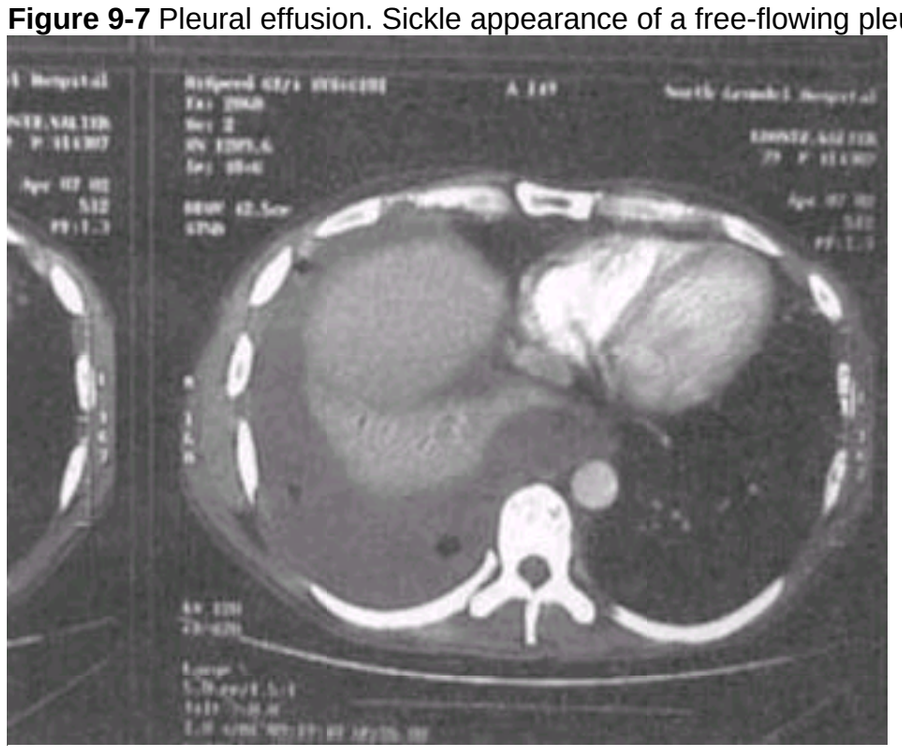


Figure 9-8 The bare area sign. A computed tomography scan demonstrates the obliteration of the potential space behind the liver with pleural fluid.

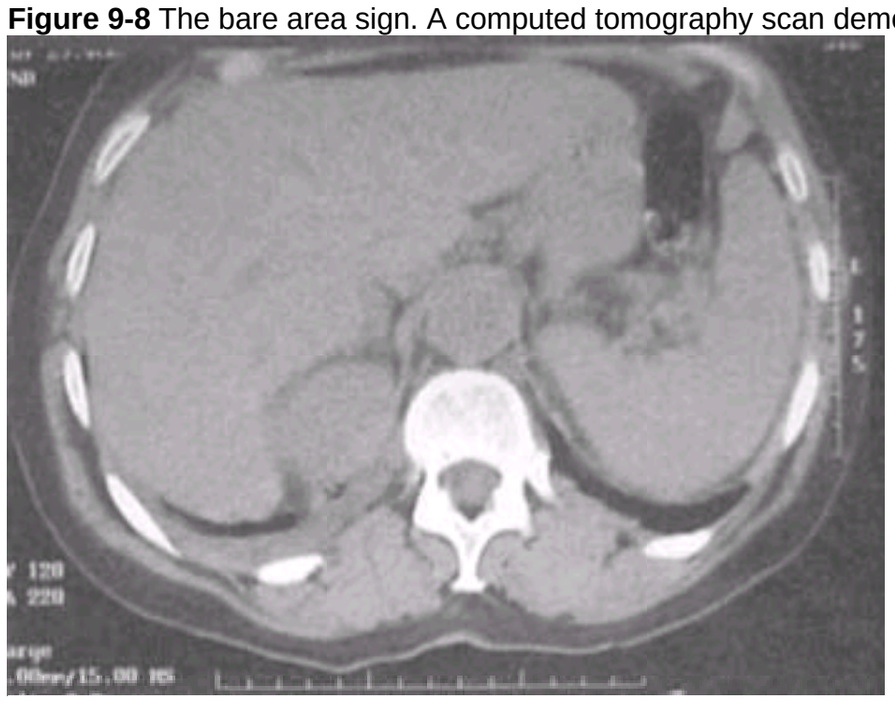


Figure 9-9 Pleural effusion. Ultrasound demonstrates the appearance of pleural fluid.

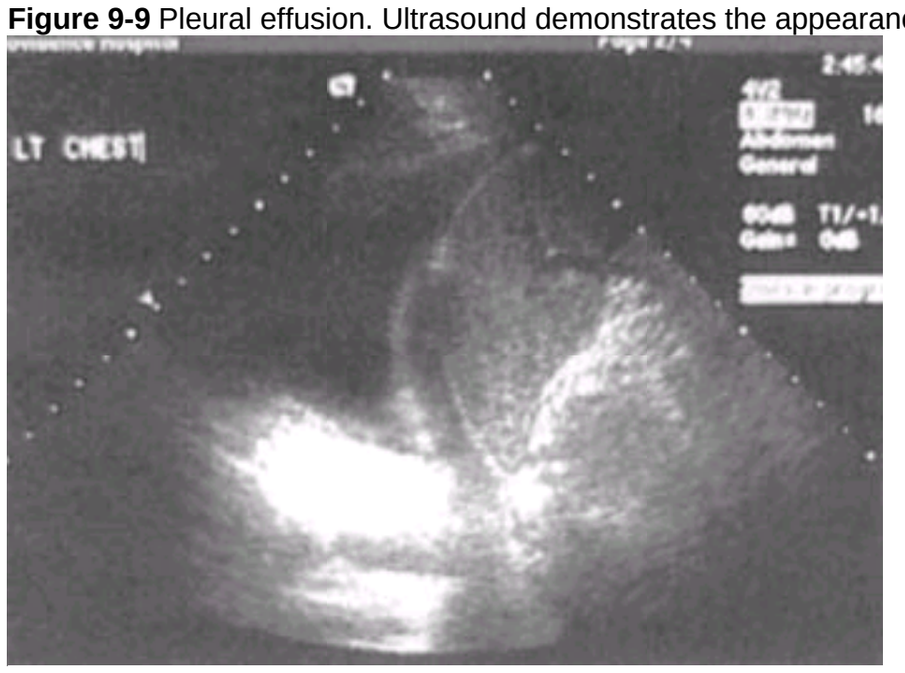


Figure 9-10 Techniques for through-the-needle catheter thoracentesis. Separate intercostal spaces depict the steps. *A,* Needle placement into pleural space. *B,* Catheter insertion through needle. *C*, Withdrawal of needle after catheter advancement.

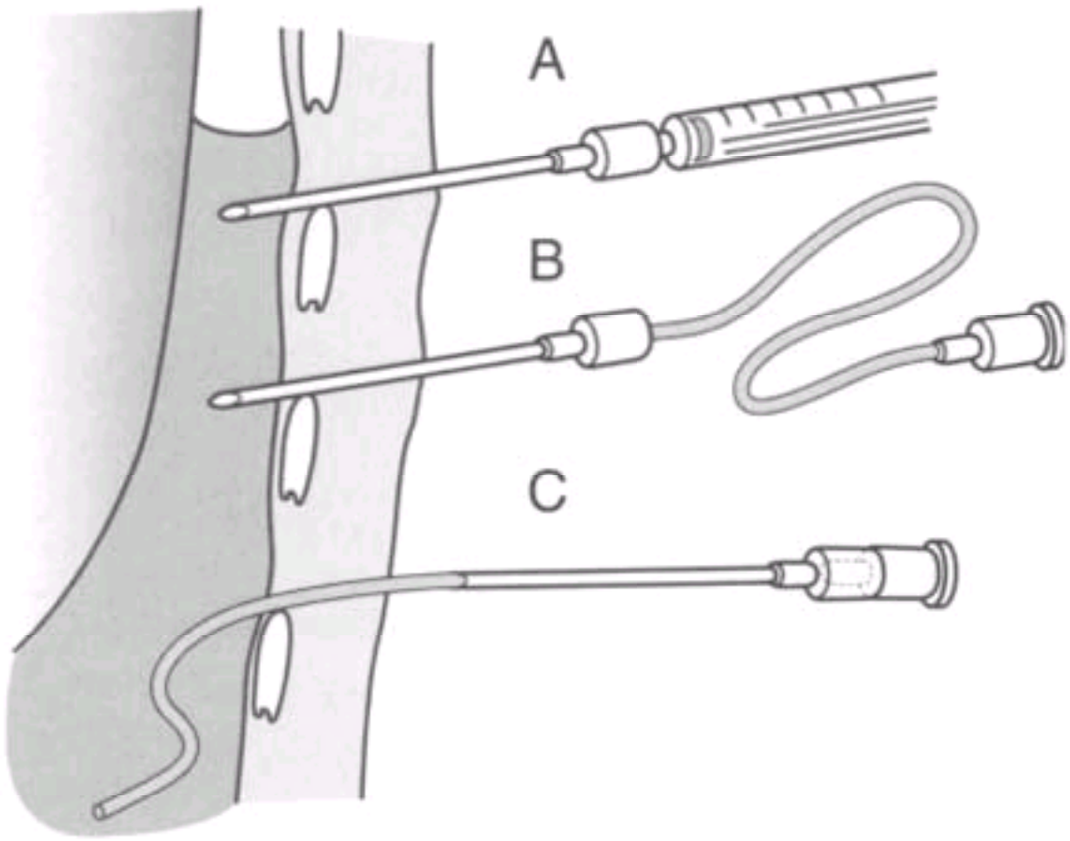


Figure 9-11 Positioning of patient for drainage of pleural effusion. *A,* Upright positioning. The broken line indicates midscapular line. *B,* Lateral decubitus positioning. The broken line indicates posterior axillary line. *C,* Supine patient positioning. The broken line indicates mid-axillary line.

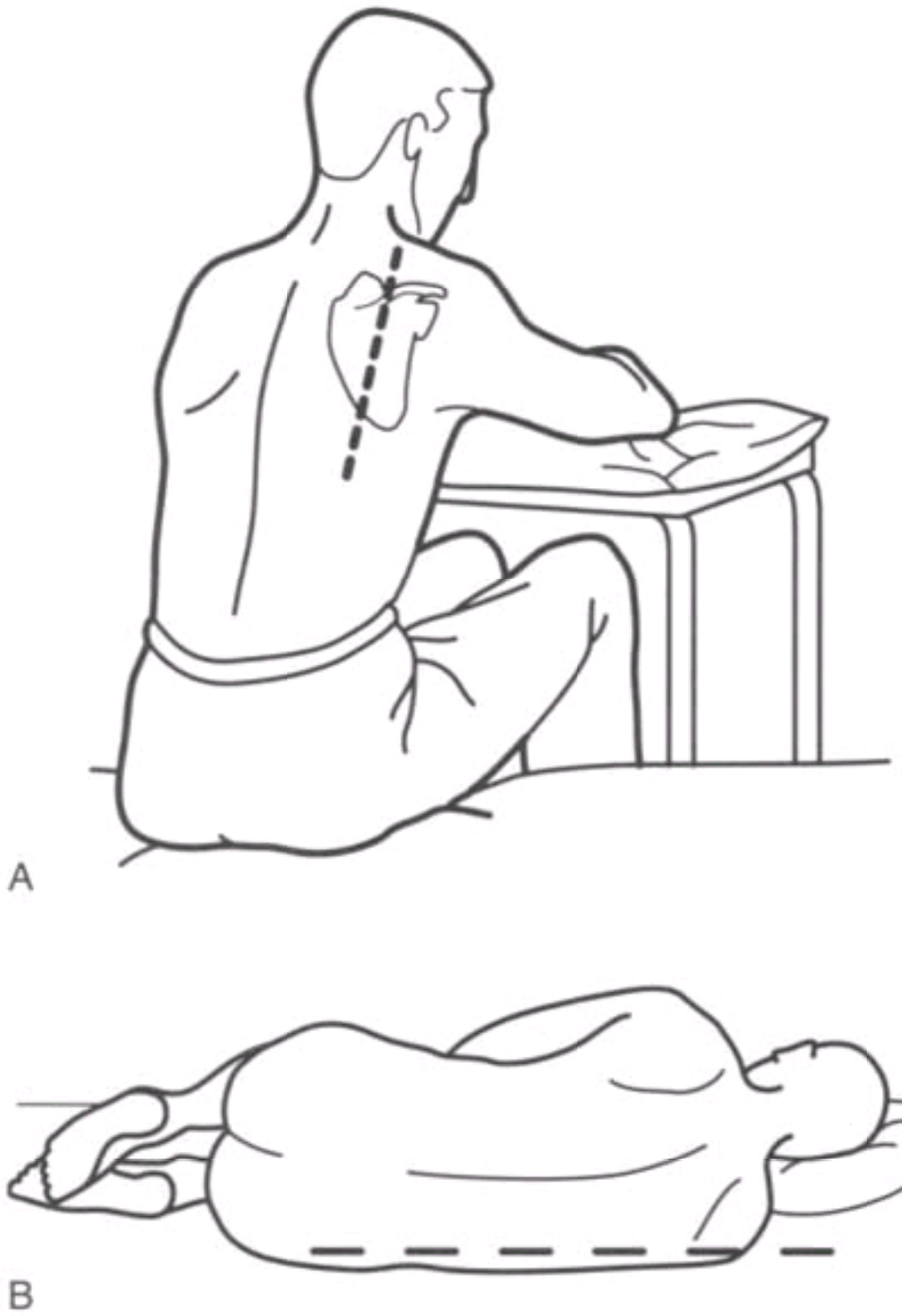


Figure 9-12 "Walking" the anesthetic needle over the superior aspect of the rib.

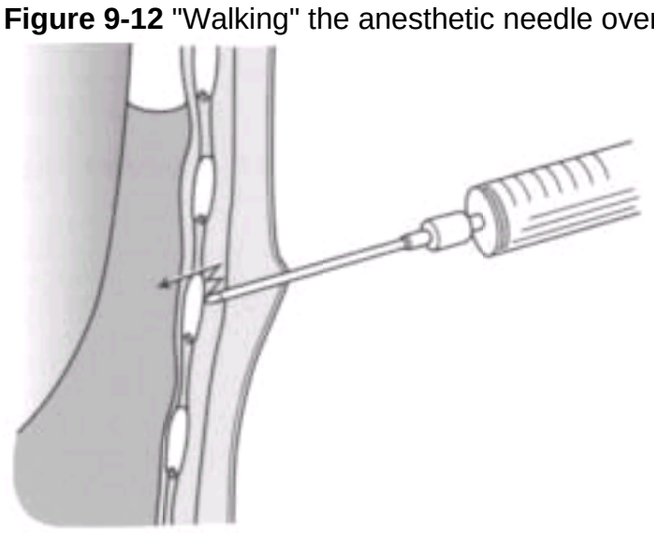


Figure 10-1 Simple pneumothorax, anteroposterior chest radiograph view. Note absence of lung marking on the affected side and deviation of the trachea contralaterally.

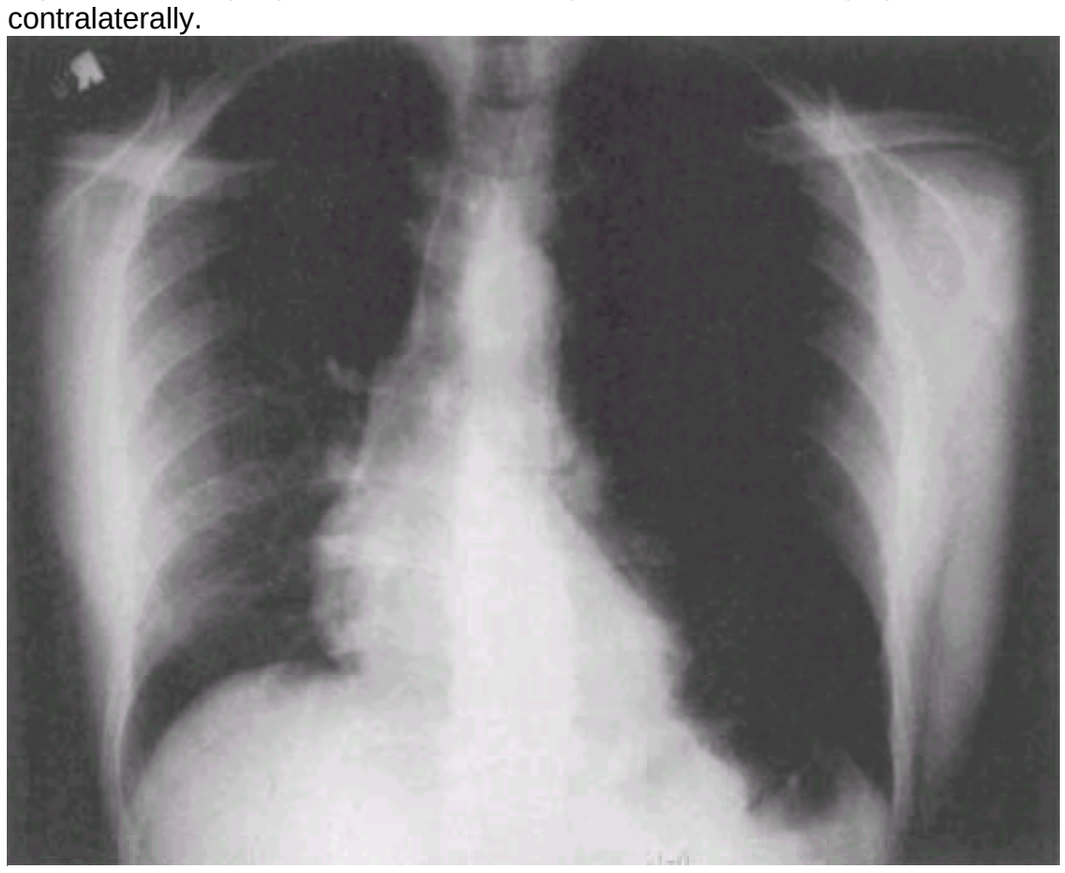
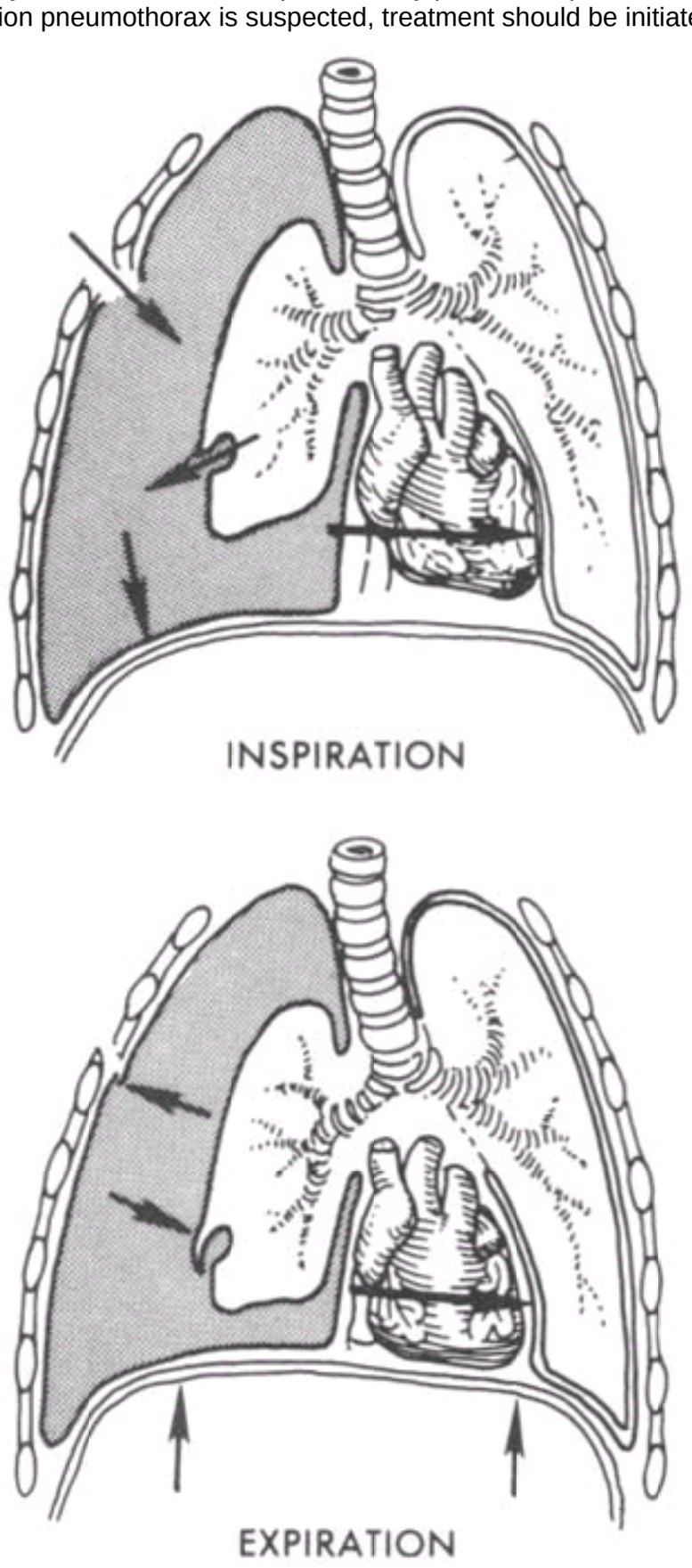
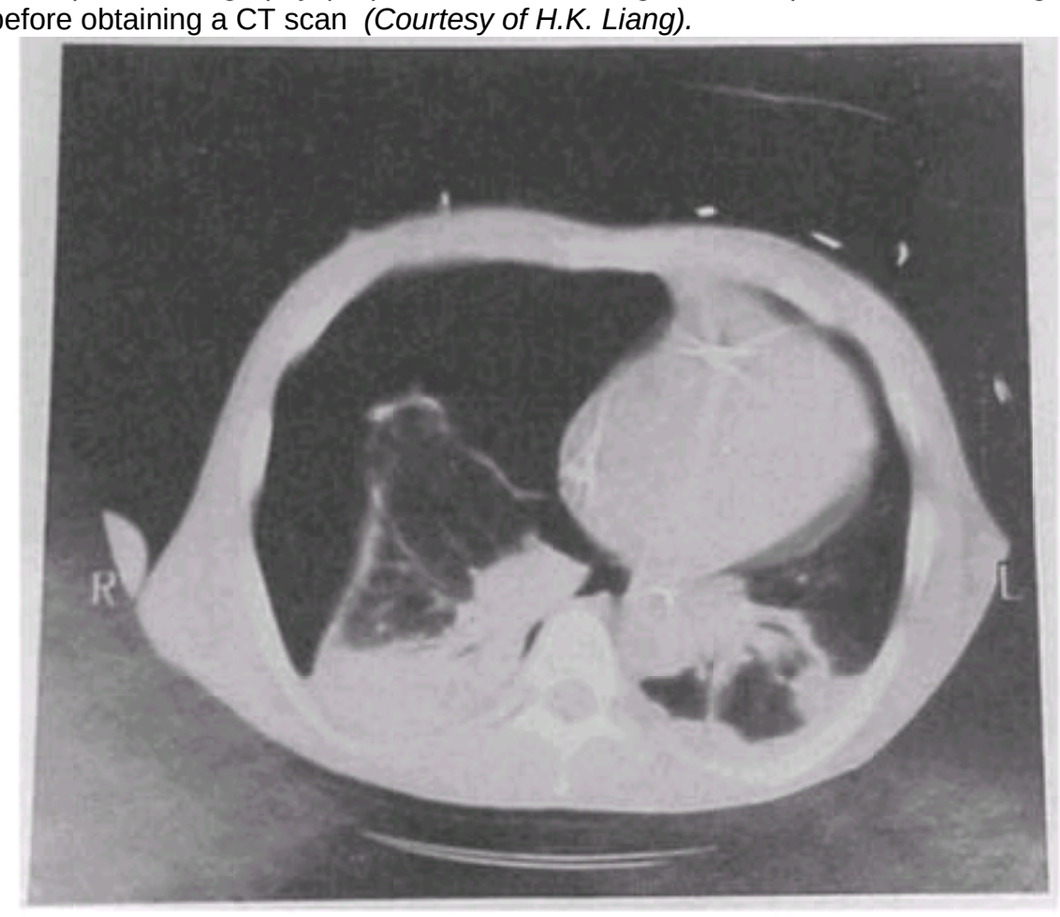


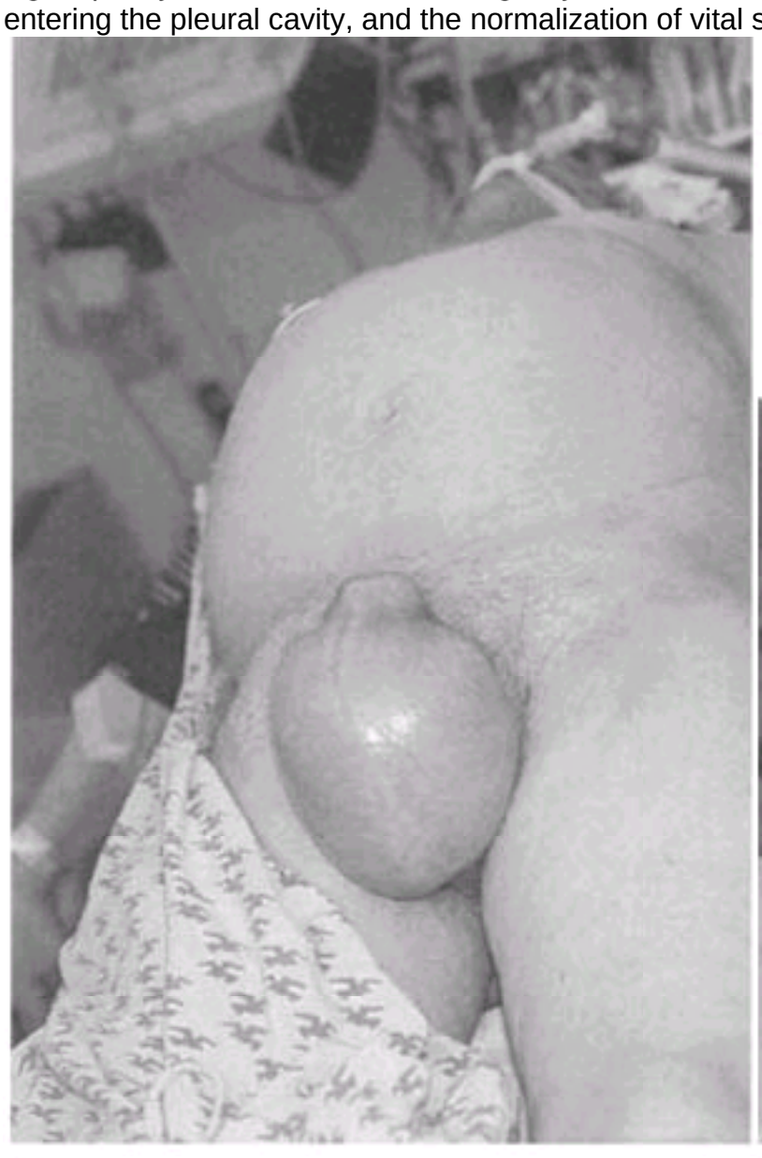
Figure 10-2 *A*, Tension pneumothorax. With inspiration air enters the pleural space from the outside or from a puncture in the lung, or both. Upon expiration, the air leaks are closed, trapping increasing amounts of air in the pleural space. Cardiac filling and eventually cardiac output are compromised. *(From Vukich DJ, Markovchick VJ: Pulmonary and chest wall injuries. In Rosen P, Barkin RM, Braen CR, et al (eds): Emergency Medicine: Concepts and Clinical Practice. St Louis, Mosby-Year Book, 1988. Reproduced by permission.) B*, A thoracic computed tomography (CT) scan demonstrating a tension pneumothorax. In general, whenever a tension pneumothorax is suspected, treatment should be initiated before obtaining a CT scan *(Courtesy of H.K. Liang).*



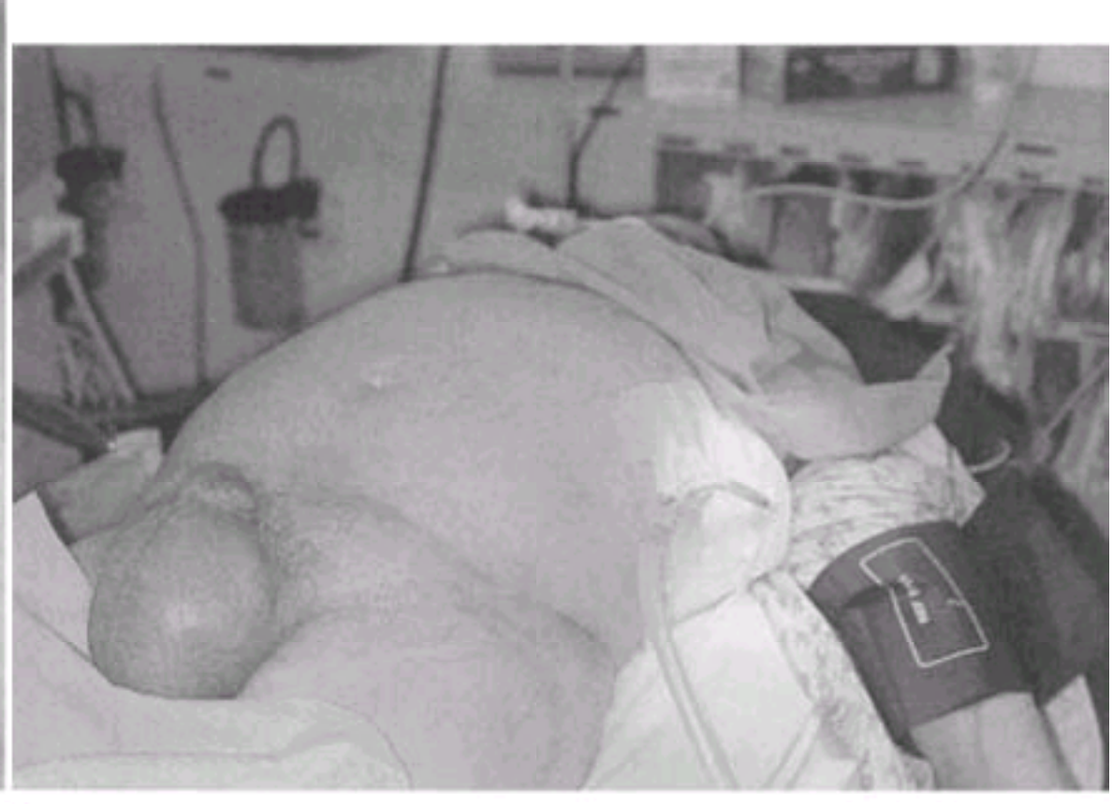


B

Figure 10-3 *A*, Following a successful cardiopulmonary resuscitation and intubation from respiratory arrest, this patient began to deteriorate. Marked subcutaneous emphysema was noted in the scrotum and abdominal wall. Curiously, little air could be palpated in the chest or neck, the usual place for air to accumulate. Assuming that the air must have come from the lungs, the scenario led to the clinical diagnosis of a tension pneumothorax. *B*, Following the placement of a chest tube, the vital signs quickly stabilized. Due to the urgency of the situation, the thoracostomy tube was performed before obtaining an x-ray, and the rush of air obtained upon entering the pleural cavity, and the normalization of vital signs, retrospectively confirmed the tension pneumothorax.

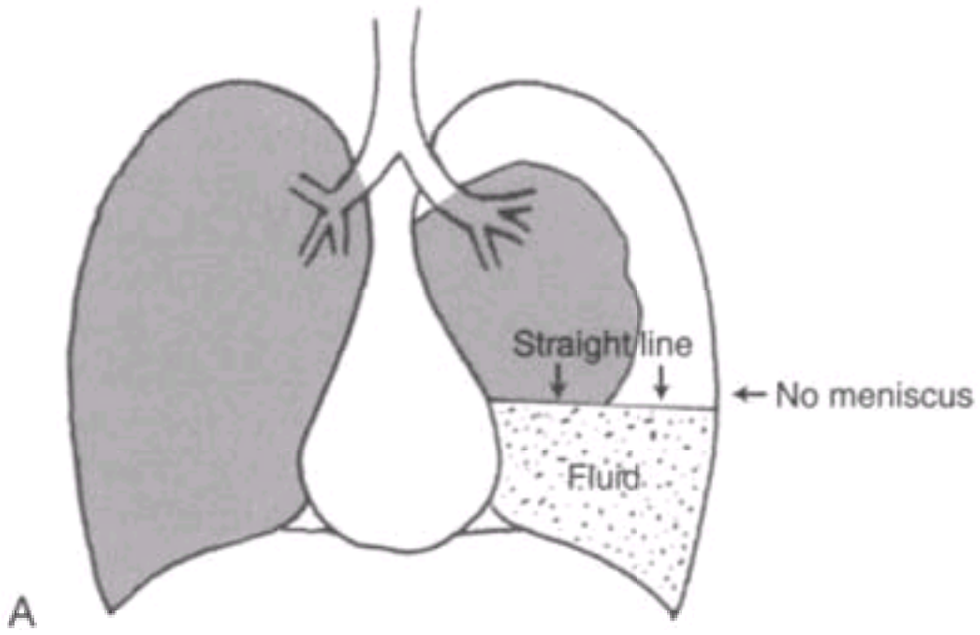


A

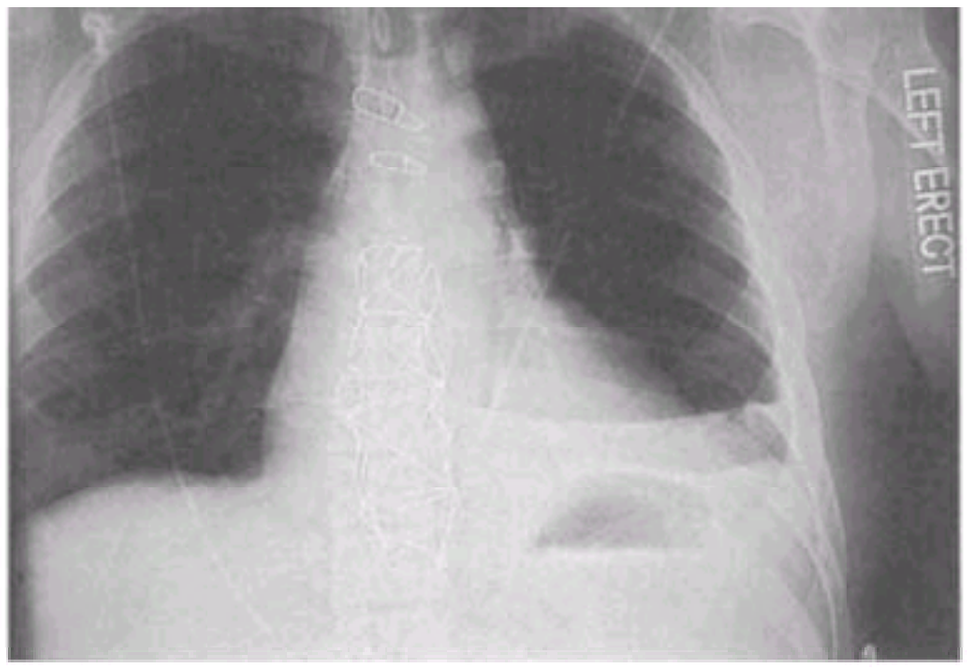


B

Figure 10-4 *A*, Hemopneumothorax. Note that whenever the top of the accumulated fluid is seen as a straight line as opposed to a meniscus, a pneumothorax must also be present. *B*, Following a stab wound to the chest, this radiograph demonstrated an air-fluid level (straight line) in the left hemithorax. A 10% pneumothorax is difficult to see on the poor quality film, but the air-fluid level means that a pneumothorax must be present. Otherwise, the fluid would form a meniscus tracking up the lateral chest wall. On a supine film, this subtle finding may not be appreciated.



A



B

Figure 10-5 Nomogram for the prediction of pneumothorax size from an average interpleural distance (see text). The interpleural distance is measured in centimeters from the outer edge of the collapsed lung to the inner aspect of the chest wall (i.e., parietal pleura to visceral pleura). *(From Rhea JT, DeLuca SA, Greene RE: Determining the size of pneumothorax in the upright patient. Radiology 144:733, 1982. Reproduced with permission.)*

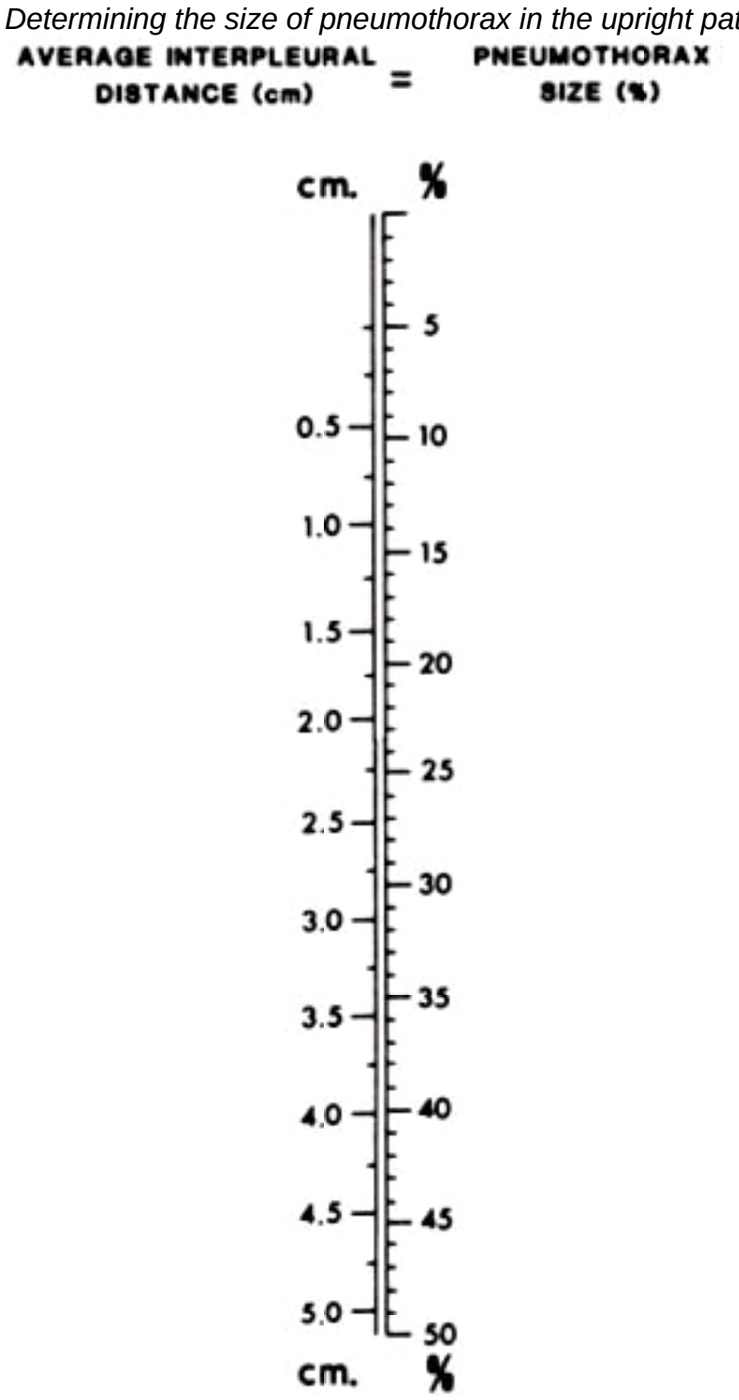


Figure 10-6 During a resuscitation involving a tension pneumothorax, there may be no time for a chest tube and needle decompression may not be effective rapidly enough. Under these circumstances the pleural cavity can be vented in seconds. This assumes that the patient is intubated. *A*, A No. 10 scalpel is used to make a deep incision in the skin and subcutaneous tissue over the fourth or fifth rib in the anterior axillary line. *B*, A long *closed* Kelly clamp (or scissors) is inserted over the top of the rib and stabbed into the pleural space. A pop is usually felt. *C*, Once the instrument is in the pleural space, it is opened wide to create a rent in the parietal pleura. Air should immediately vent. If the patient is intubated, normal cardiorespiratory function can be maintained. A chest tube is then inserted. (See also Figure 10-11 Figure 10-12 Figure 10-13 Figure 10-14 .)

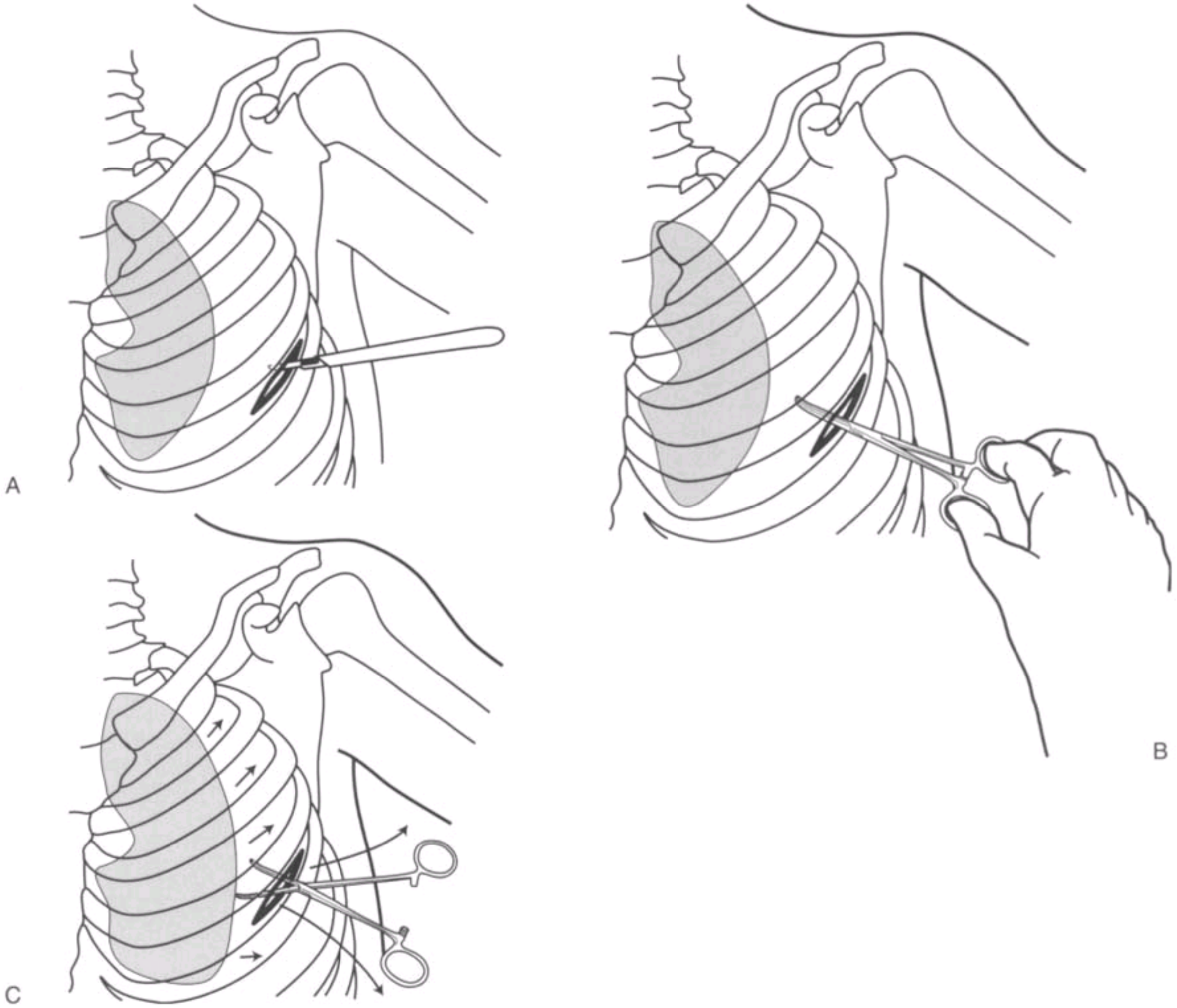


Figure 10-7 Standard sites for tube thoracostomy. *A*, The second intercostal space, midclavicular line. *B*, The fourth or fifth intercostal space, midaxillary line. The midaxillary line placement is usually preferable for all chest tubes, regardless of pathology. Always avoid the breast tissue, even in prepubescent girls. Note that placing the tube too far posteriorly will not allow the patient to lie down comfortably.

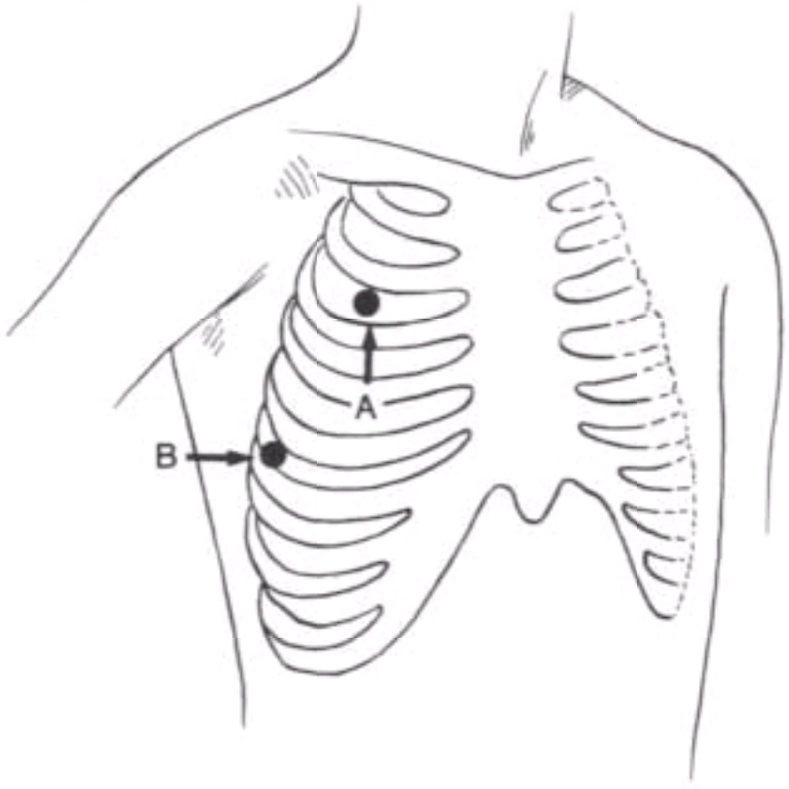


Figure 10-8 To insert a chest tube, the patient is placed semi-erect with the ipsilateral shoulder abducted and preferably restrained. Supplemental oxygen is recommended.

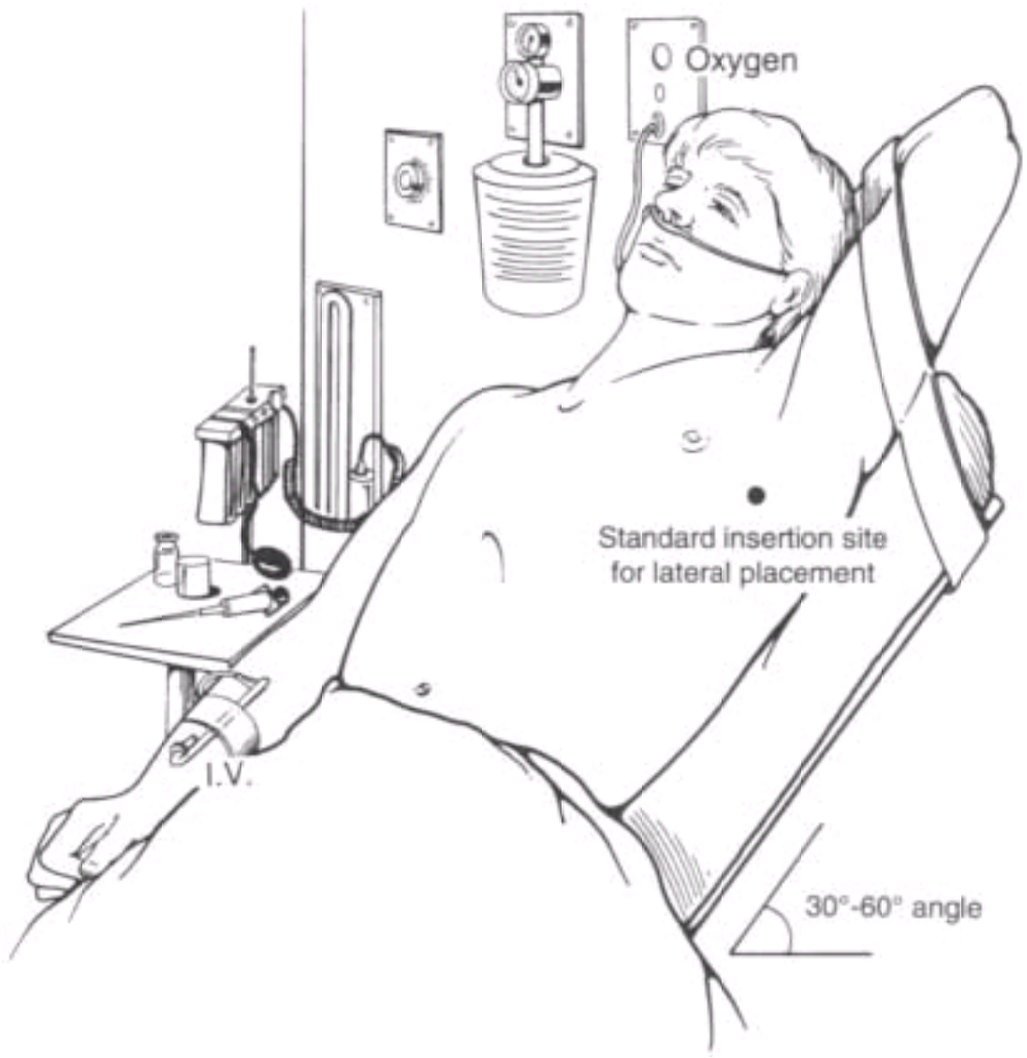


Figure 10-9 Insertion of a chest tube can be relatively painless with proper infiltration of the skin *and pleura* with local anesthetic. The liberal use of 1% lidocaine with epinephrine (maximum lidocaine dose, 5 mg/kg) is recommended. *(Redrawn from Hughes WT, Buescher ES: Pediatric Procedures, 2nd ed. Philadelphia, WB Saunders, 1980, p 234.)*

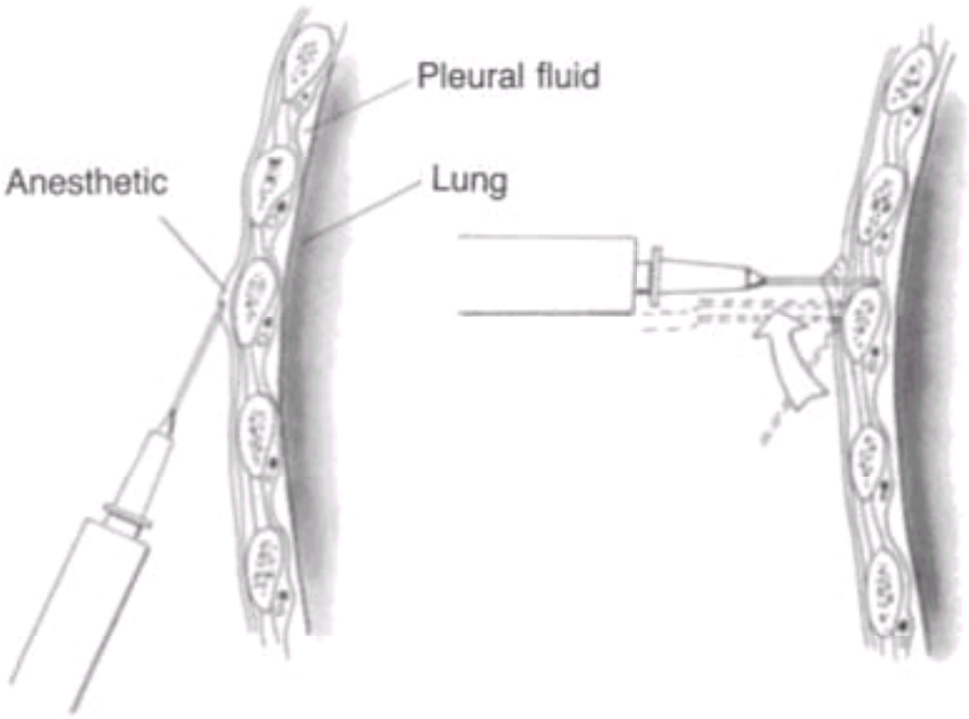


Figure 10-10 Use of the anesthetic needle to puncture the parietal pleura and establish the presence of blood or air in the pleural space. This procedure is not only diagnostic, but also may be a temporary therapeutic maneuver in a patient with tension pneumothorax. *(Redrawn from Richards V: Tube thoracostomy. J Fam Pract 6:631, 1978.)*

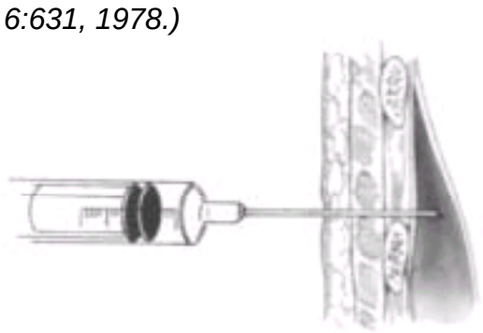


Figure 10-11 The skin wound is made one intercostal space below the space through which the tube will pass *(A)*. By spreading tissue with scissors *(B)*, dissection is carried subcutaneously to the pleural lining. The pleura is entered with a blunt Kelly or Tonsillar clamp *(C)*. A common error in technique is to attempt to insert a large chest tube through a skin incision that is too small. *(Redrawn from Hughes WT, Buescher ES: Pediatric Procedures, 2nd ed. Philadelphia, WB Saunders, 1980, p 237.)*

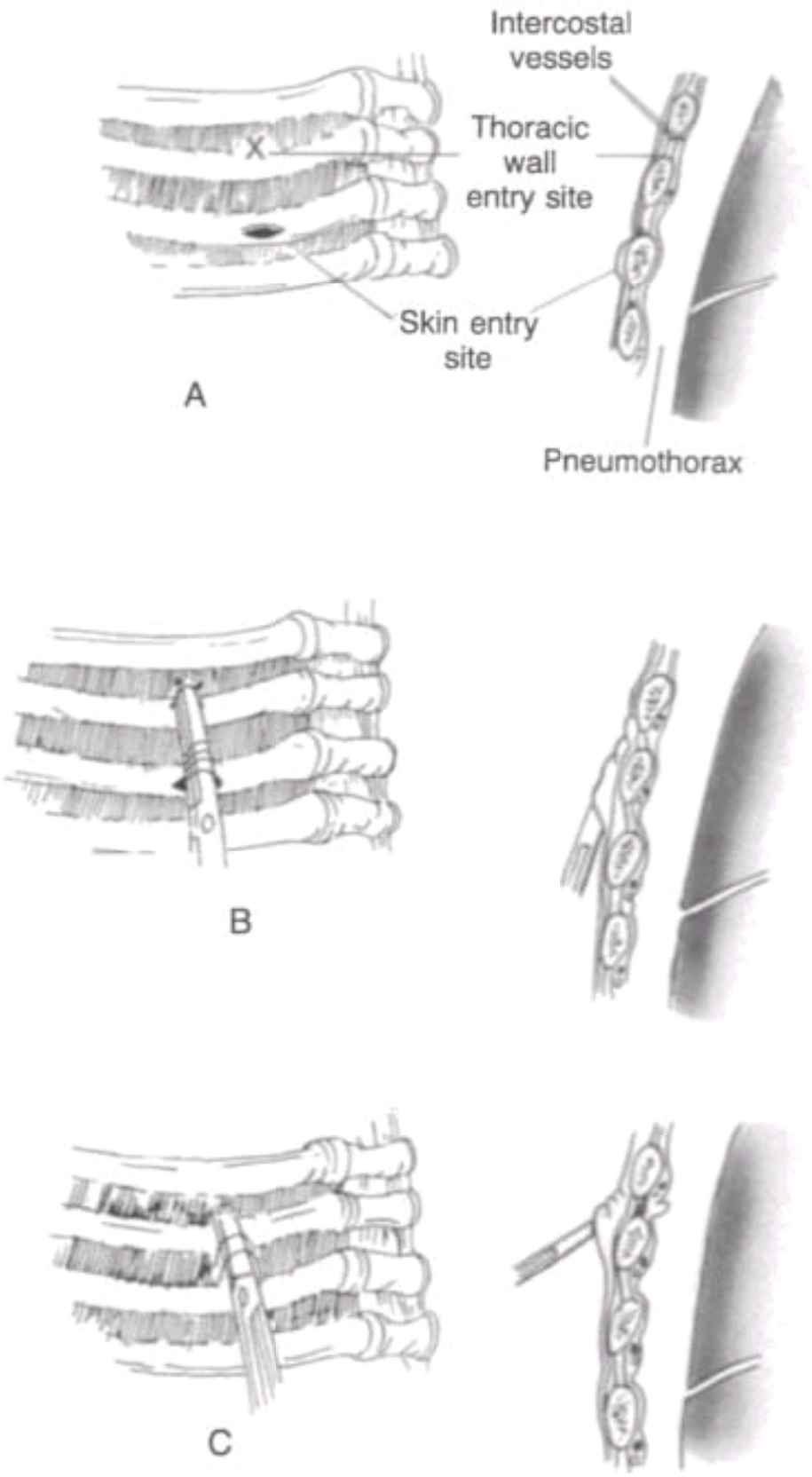


Figure 10-12 Location of the intercostal neurovascular bundle, running inferiorly and slightly medial to the rib. *(From Millikan JS, Moore EE, Steiner E: Complications of tube thoracostomy for acute trauma. Am J Surg 140:739, 1980.)*

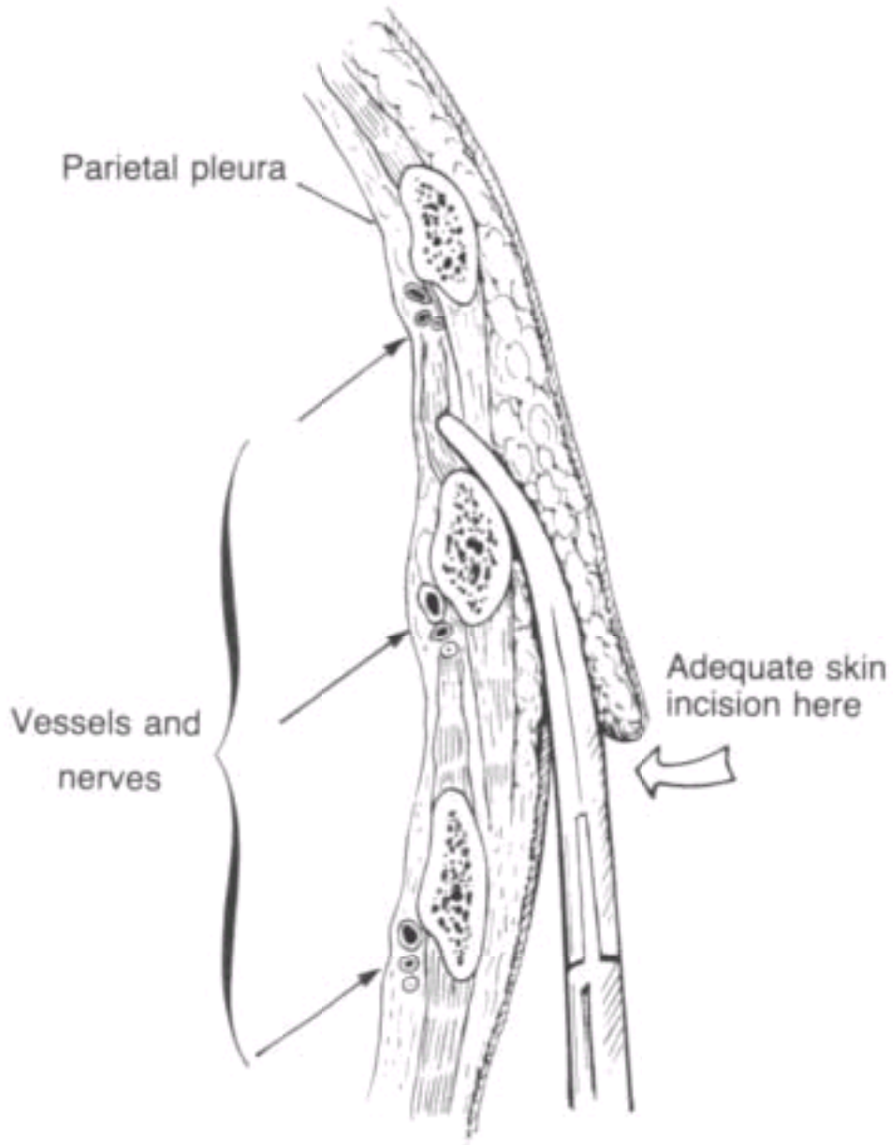


Figure 10-13 One accomplishes blunt dissection by forcing the closed points of the clamp forward, then spreading the tips and pulling back with the points spread. Note that the clamp should be spread in a direction parallel with the ribs (See Fig. 10-6); not perpendicular to the ribs as suggested in the figure. The drawing emphasizes the placement of the instrument *over* the rib. A rush of air or fluid signifies penetration into the pleural space. *(From Bricker DL: Safe, effective tube thoracostomy. ER Reports 2:49, 1981.)*

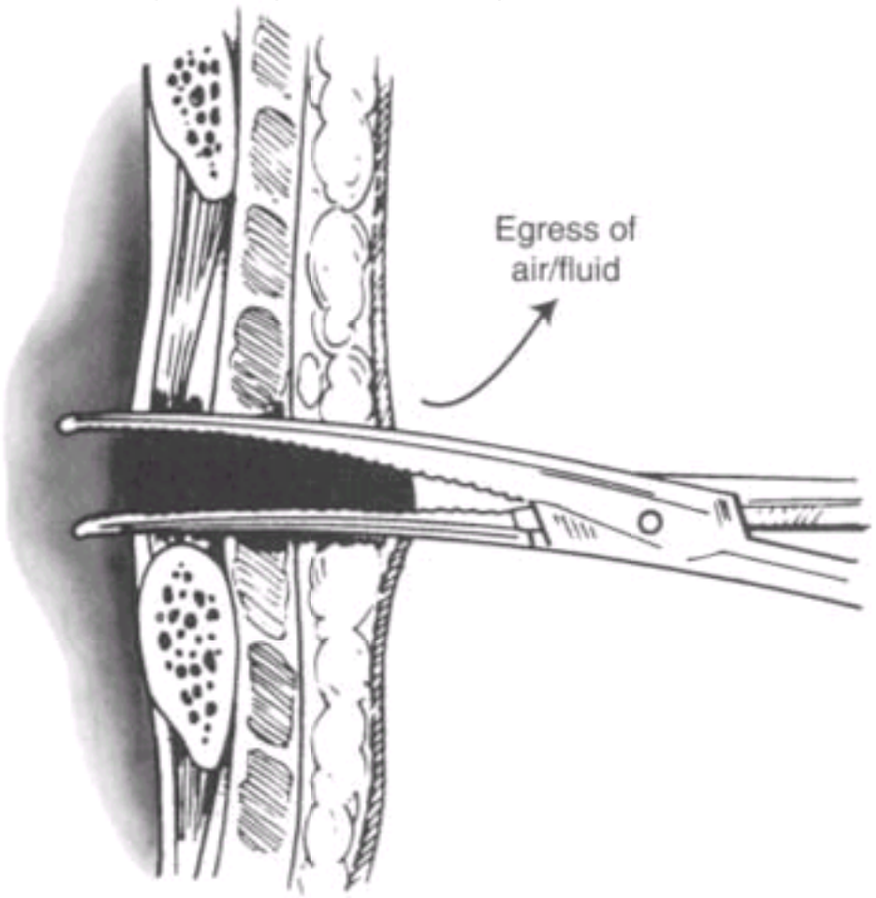


Figure 10-14 Using the finger as a guide to ensure entry into the pleural cavity, one places the tip of the tube into the pleural cavity. *(From Millikan JS, Moore EE, Steiner E: Complications of tube thoracostomy for acute trauma. Am J Surg 140:739, 1980.)*

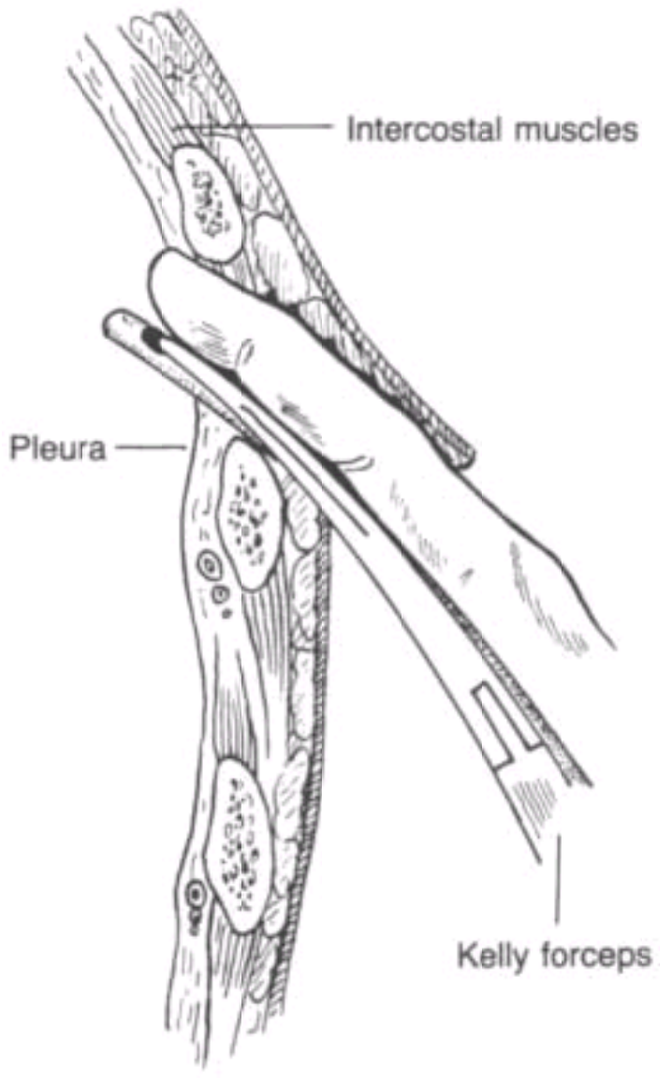


Figure 10-15 The tube is grasped with the curved clamp, with the tube tip protruding from the jaws.

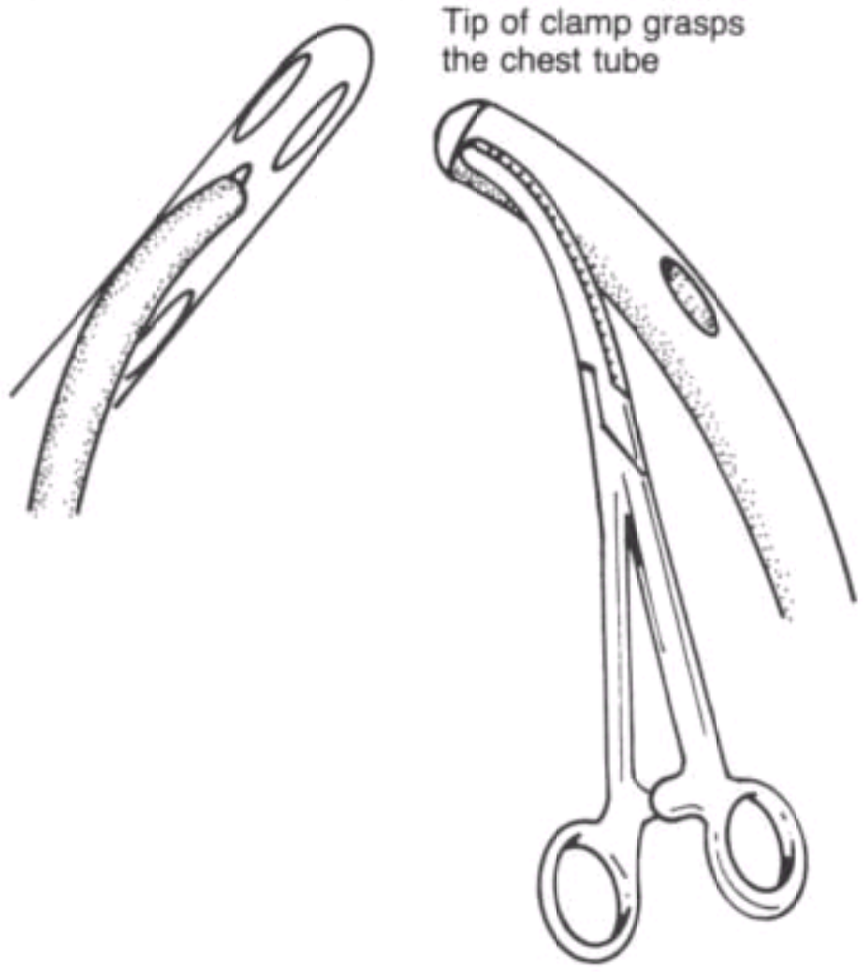


Figure 10-16 Subcutaneous (SQ) placement of a chest tube (*arrows*) can occur, as the tube can dissect through tissue planes with relative ease. If this tube had been directed posteriorly, the radiograph would erroneously "confirm" intrapleural placement despite the tube being SQ throughout its entire course.

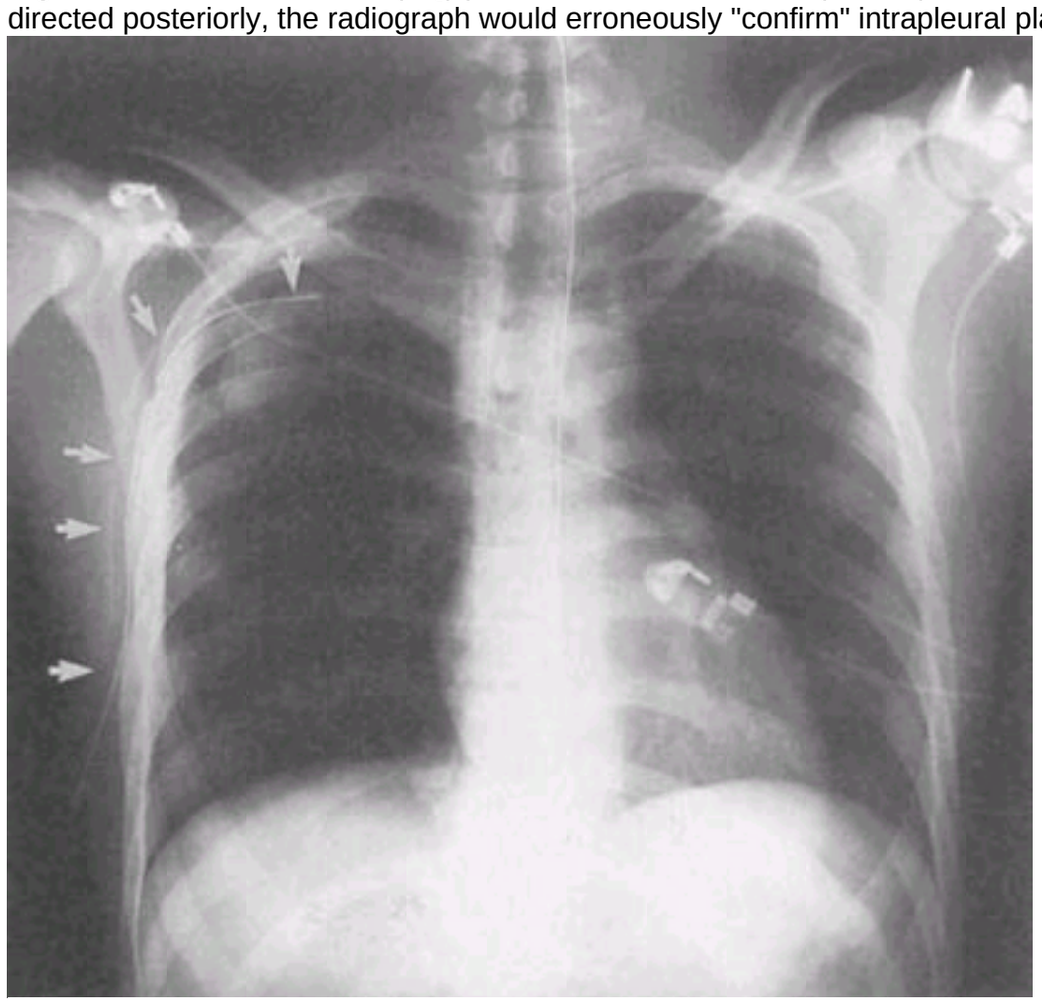


Figure 10-17 A "stay" suture is first placed next to the tube to close the skin incision. *A,* The knot is tied securely and the ends, which will subsequently be wrapped around the chest tube, are left long. *B,* The ends of the suture are wound twice about the tube tightly enough to indent the tube slightly and are tied securely.

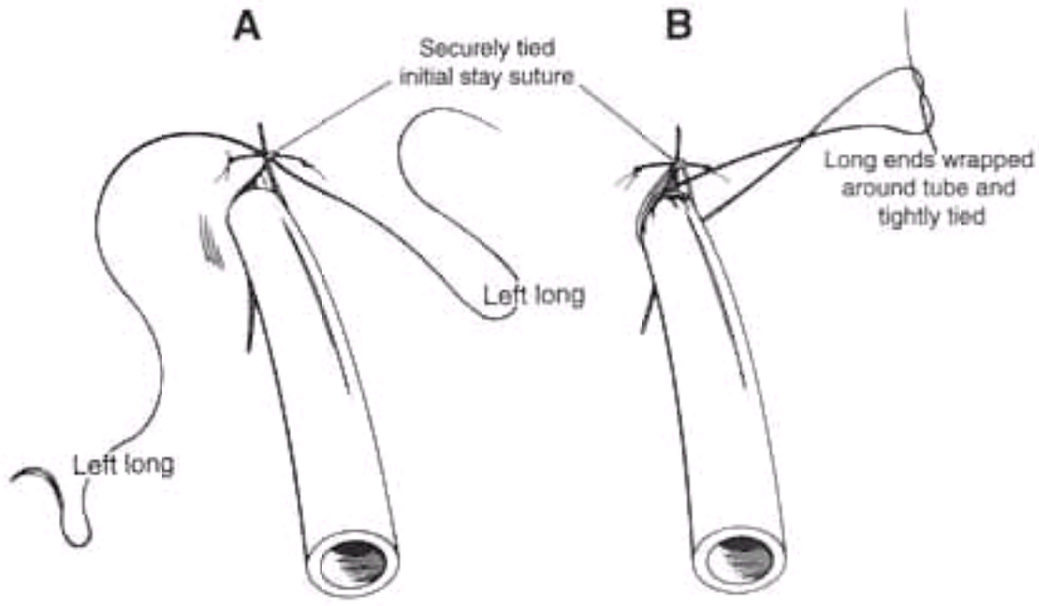


Figure 10-18 *A,* A horizontal mattress suture is placed around (above and below) the tube and is held only with a surgeon's knot. *B,* The loose ends are also wrapped around the tube and are tied loosely in a bow to identify the suture. This suture will be untied and used to close the skin incision after tube removal.

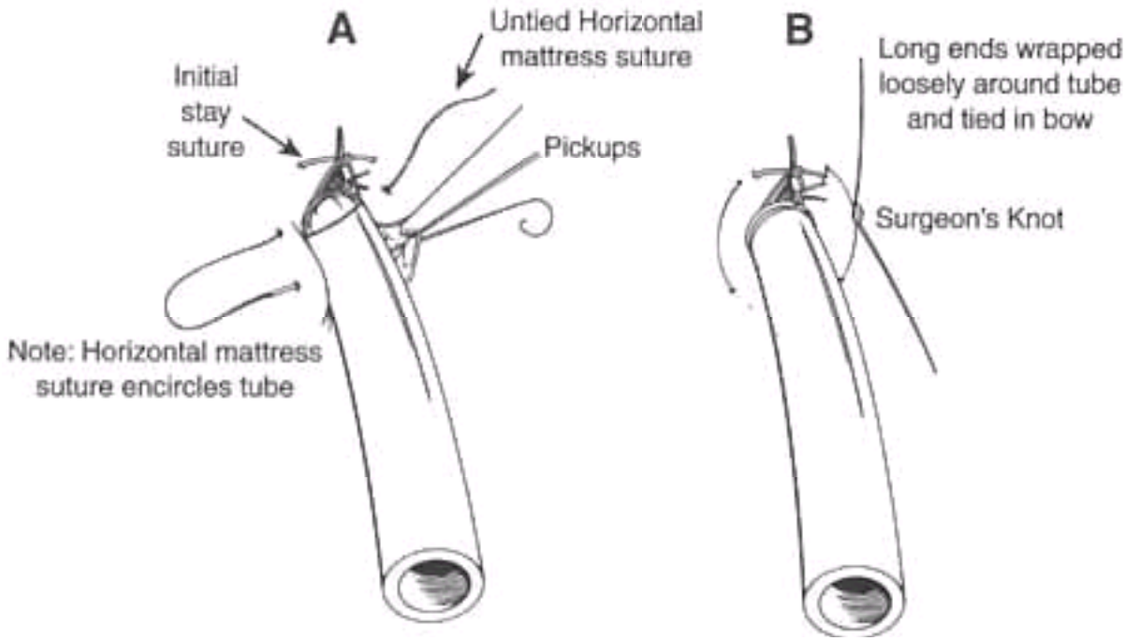


Figure 10-19 A dressing consisting of gauze sponges with a Y-cut is applied to the entry site to provide an air-tight seal. Two pieces are placed at angles.

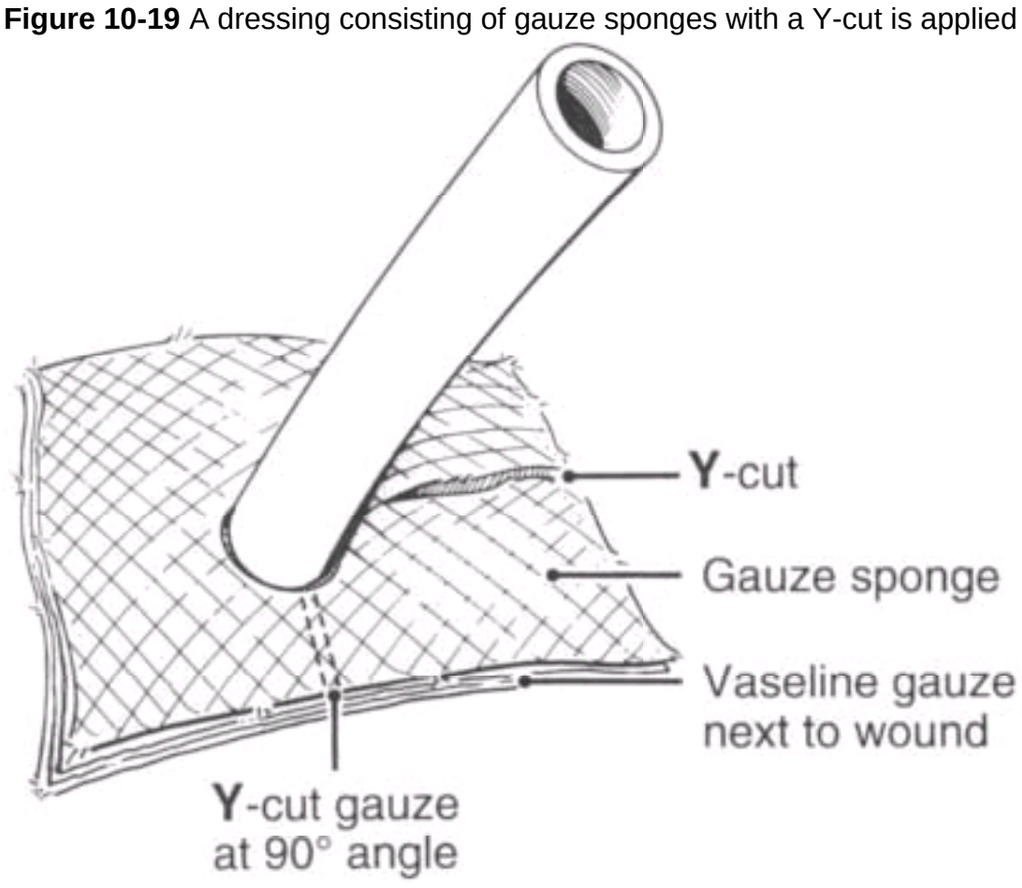
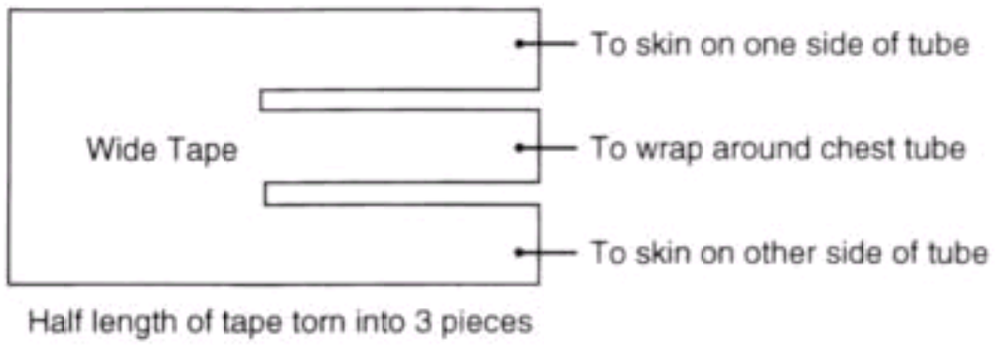


Figure 10-20 *A*, The distal half of a wide piece of tape is longitudinally split into three pieces. The two outside pieces are placed on the skin on either side of the tube, and the center strip is wrapped around the chest tube itself. *B*, This process may be repeated with a similar piece of tape placed at a 90° angle. The tape is securely anchored to the skin (benzoin is optional, but the skin must be clean and dry), and the torn tape is wrapped around the tube. Each anchoring piece is covered by another piece of tape.

A.



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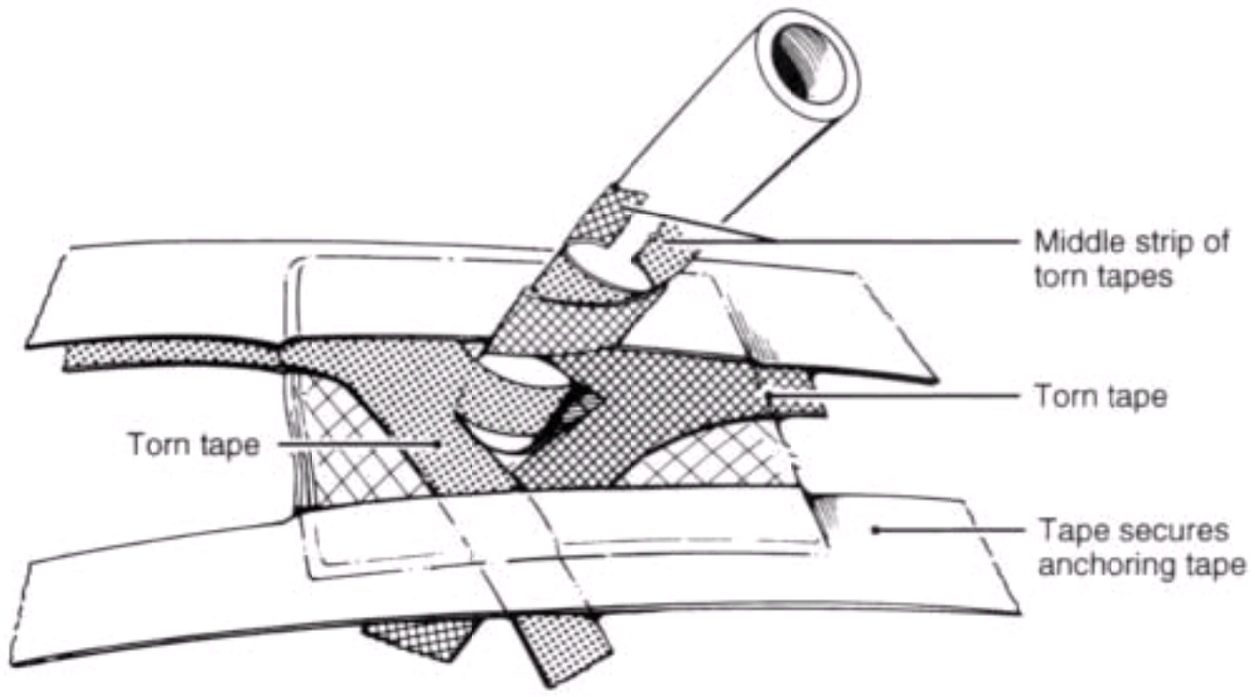


Figure 10-21 A one-way Heimlich valve alone is usually sufficient to treat a pneumothorax, but should *not* be used to treat a hemothorax.

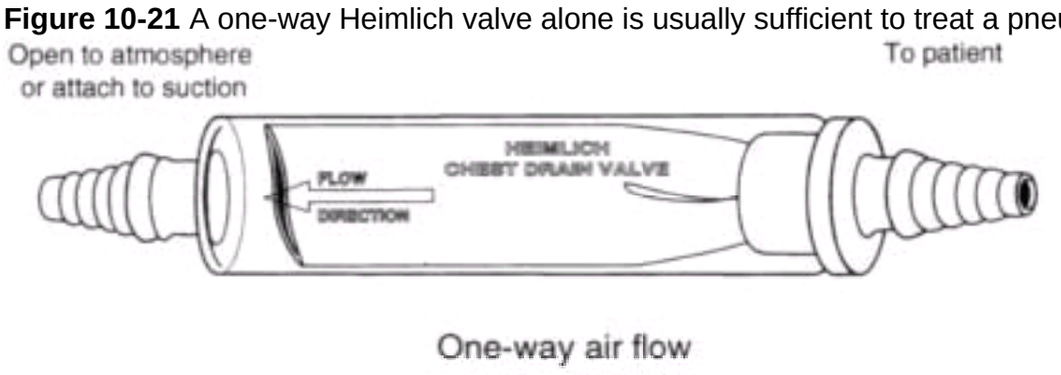


Figure 10-22 Single-bottle (water seal) collection device.

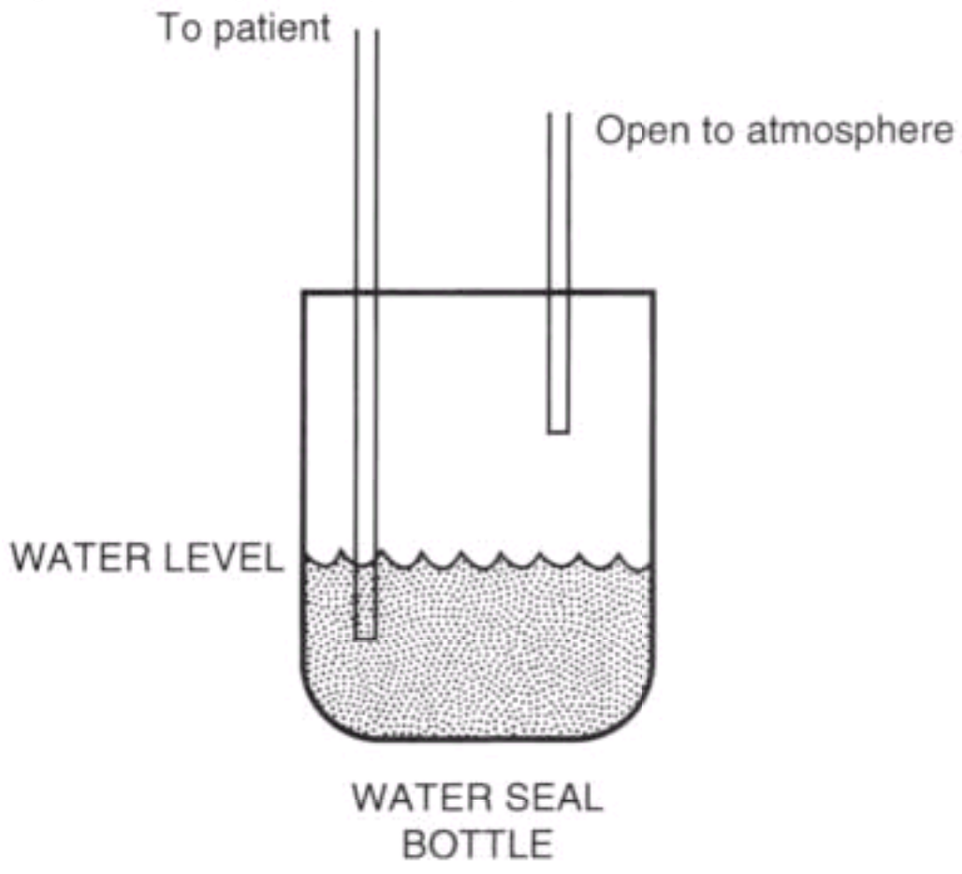


Figure 10-23 A two-bottle system. The "trap" reservoir proximal to the water seal keeps the accumulating drainage from affecting the water seal pressure.

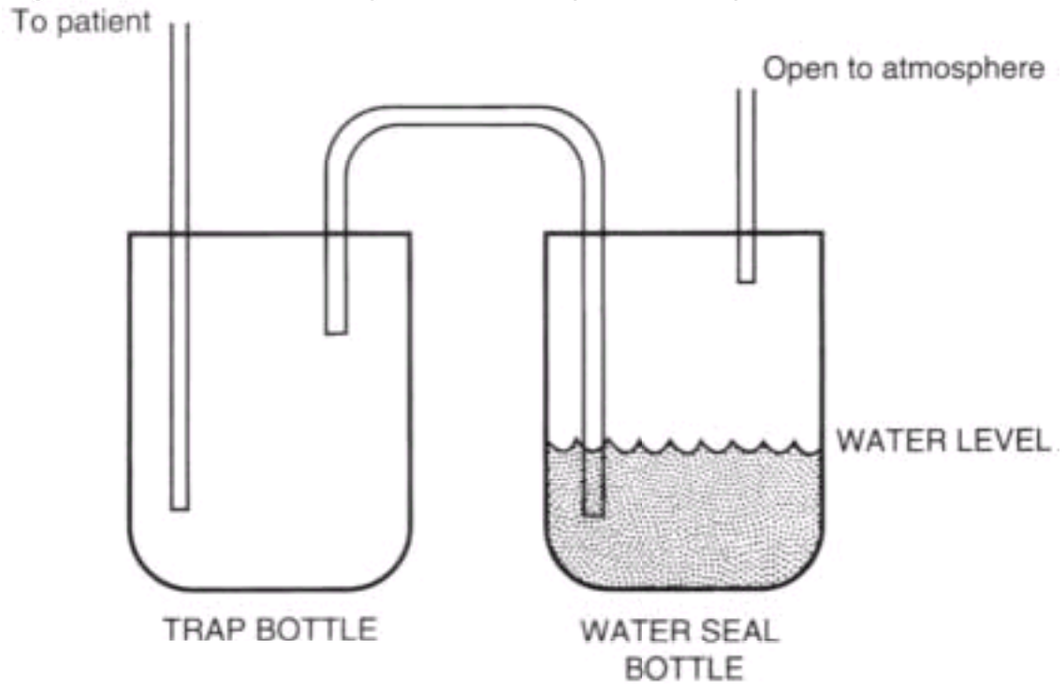


Figure 10-24 A 3-bottle system with fluid trap bottle, water seal, and adjustable vent tube. The height of the column of water in bottle 2 regulates the amount of suction applied, independent of the pressure on the suction valve. *(From Miller KS, Sahn SA: Chest tubes: Indications, technique, management and complications. Chest 91:258, 1987.)*

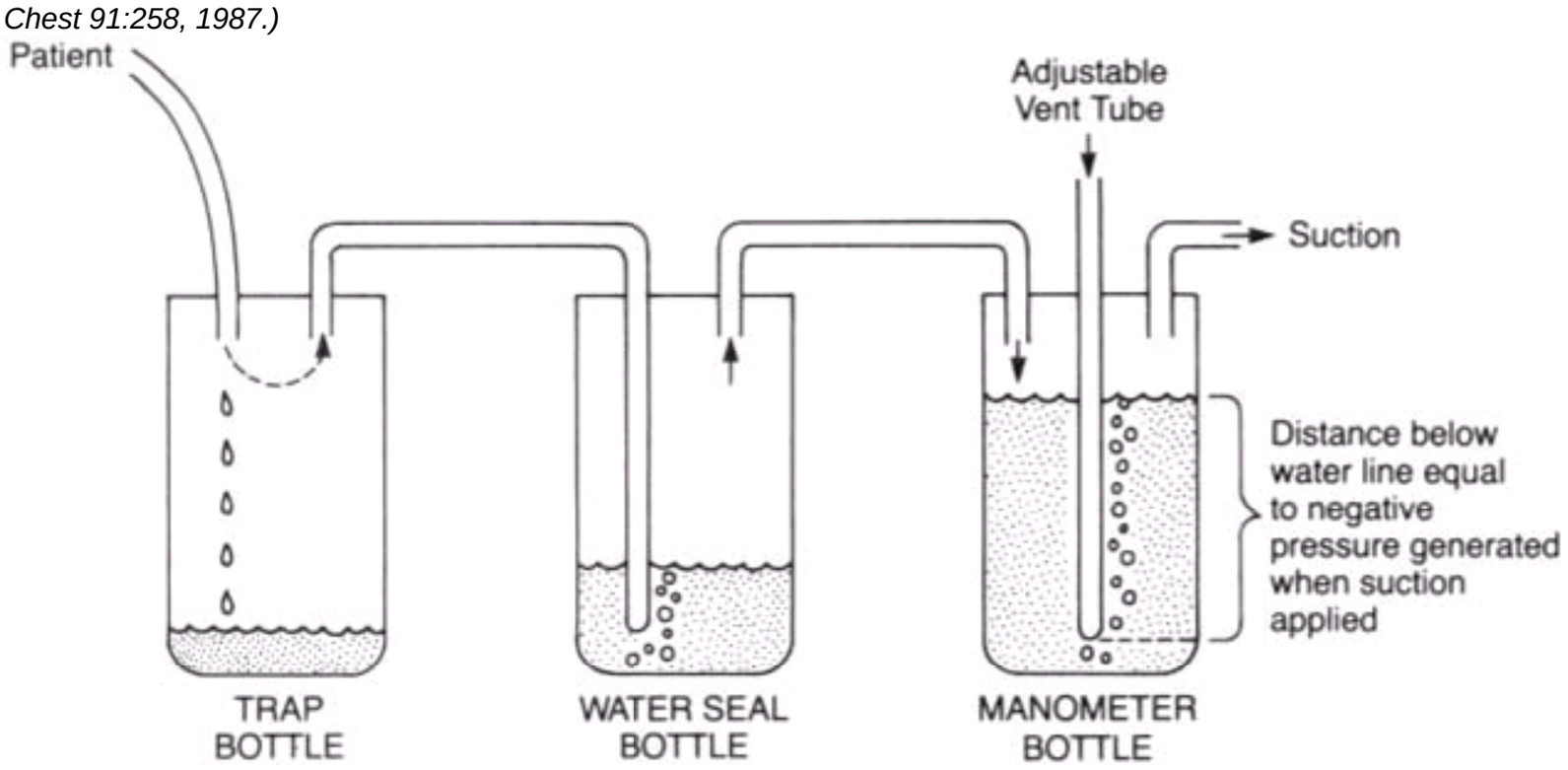


Figure 10-25 Diagram of a commercially available chest drainage system. Included is a 2500-mL fluid collection chamber, a suction control device, an air leak indicator, and a fluid sampling port. A separate attachment is available for autotransfusion. *(Courtesy of Davol, Inc, a division of CR Bard, Cranston, RI.)*

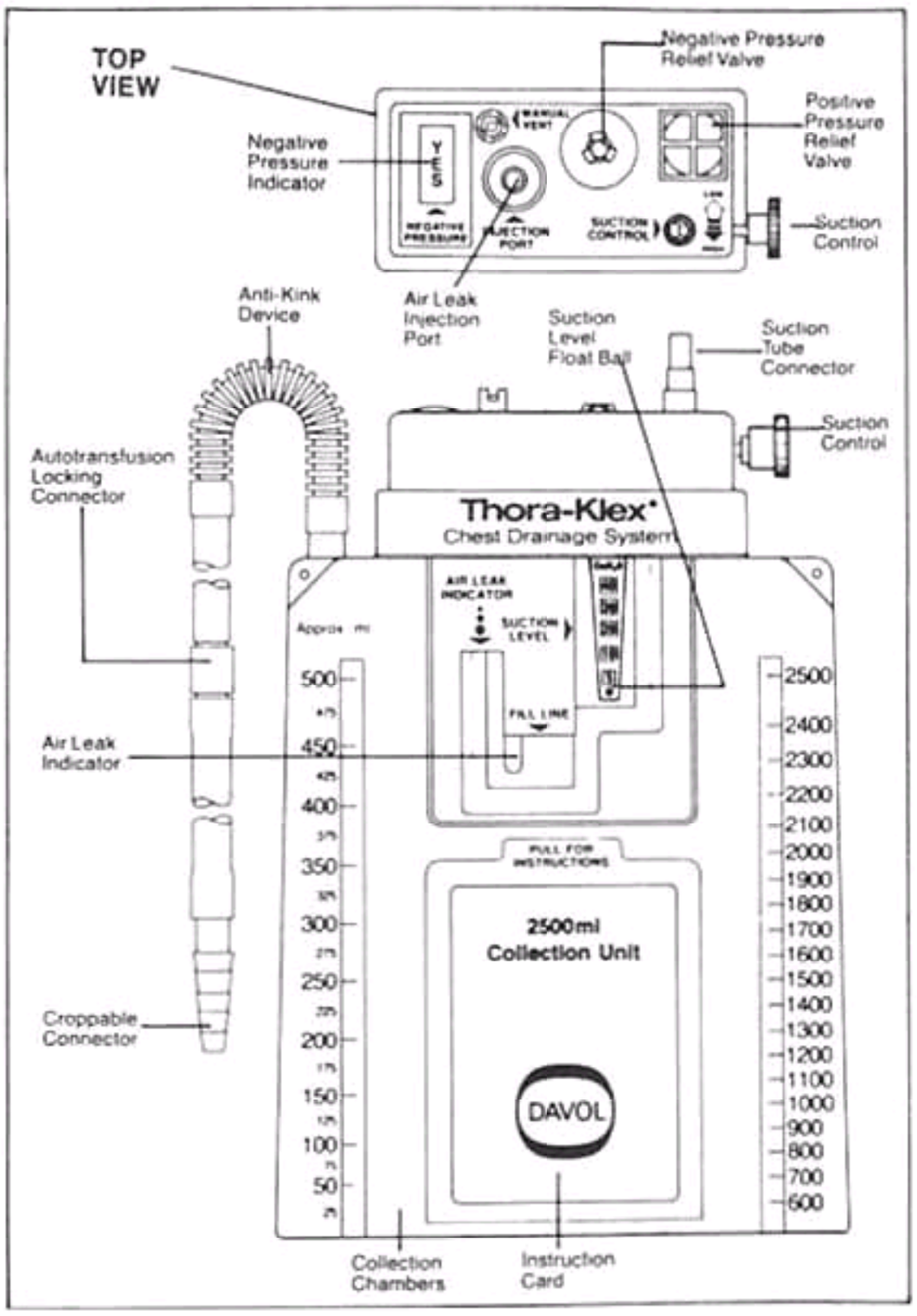


Figure 10-26 Dependent loops of fluid-filled tubing require positive intrapleural pressure greater than the vertical height of the fluid-filled loop for drainage to occur. *(From Batchelder TL, Morris KA: Critical factors in determining adequate pleural drainage in both the operated and non-operated chest. Am Surg 28:298, 1962.)*

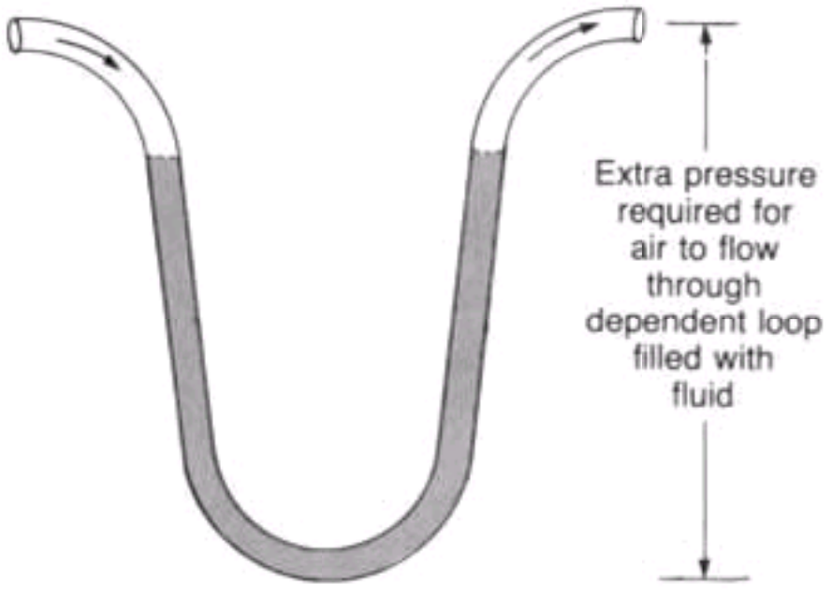


Figure 10-27 Aspiration of a pneumothorax (caused by subclavian vein catheterization) with an Arrow 14 Fr Percutaneous Cavity Drainage Catheterization Kit. This 23-cm pigtail multi-hole catheter is ideal for such purposes. Air can be aspirated from the catheter part with a syringe or the catheter can be attached to suction or a Heimlich valve. This catheter is not used for patients on a ventilator, those with continuing air leaks, or with a hemothorax. It is ideal for stable patients who have a primary pneumothorax or a collapse that can be expected to be stable if the lung is reexpanded (such as intravenous drug use induced, minor blunt trauma, secondary to central venous catheter insertion). *A*, Seldinger-type catheter kit demonstrating pigtail catheter and all necessary equipment, including local anesthesia, introducing needle and syringe, scalpel, guidewire, and dilator. *B*, After generous local anesthesia, the introducing syringe is advanced in *a straight line over the top of the fifth rib* until air is aspirated. Unless a straight track is created it will be difficult to advance the floppy catheter, and a tunneling approach cannot be used. *C*, The guidewire is advanced into the pleural space and the introducing needle is removed. *D*, Following puncture of the skin at the site of wire insertion with a scalpel, a dilator is advanced over the wire to create a track for the catheter. *E*, The pigtail catheter is advanced over the wire through the dilated tract, assuming its pigtail configuration when it is in the pleural space. A twisting motion may be needed to advance the catheter through the subcutaneous tissues. *F*, The catheter is advanced to the hilt and secured to suction. This catheter may be removed after a period of observation or the suction may be maintained for a few days. If used for a few days, the catheter will become clogged with mucus or blood, which may be cleared by injecting sterile saline through the device.

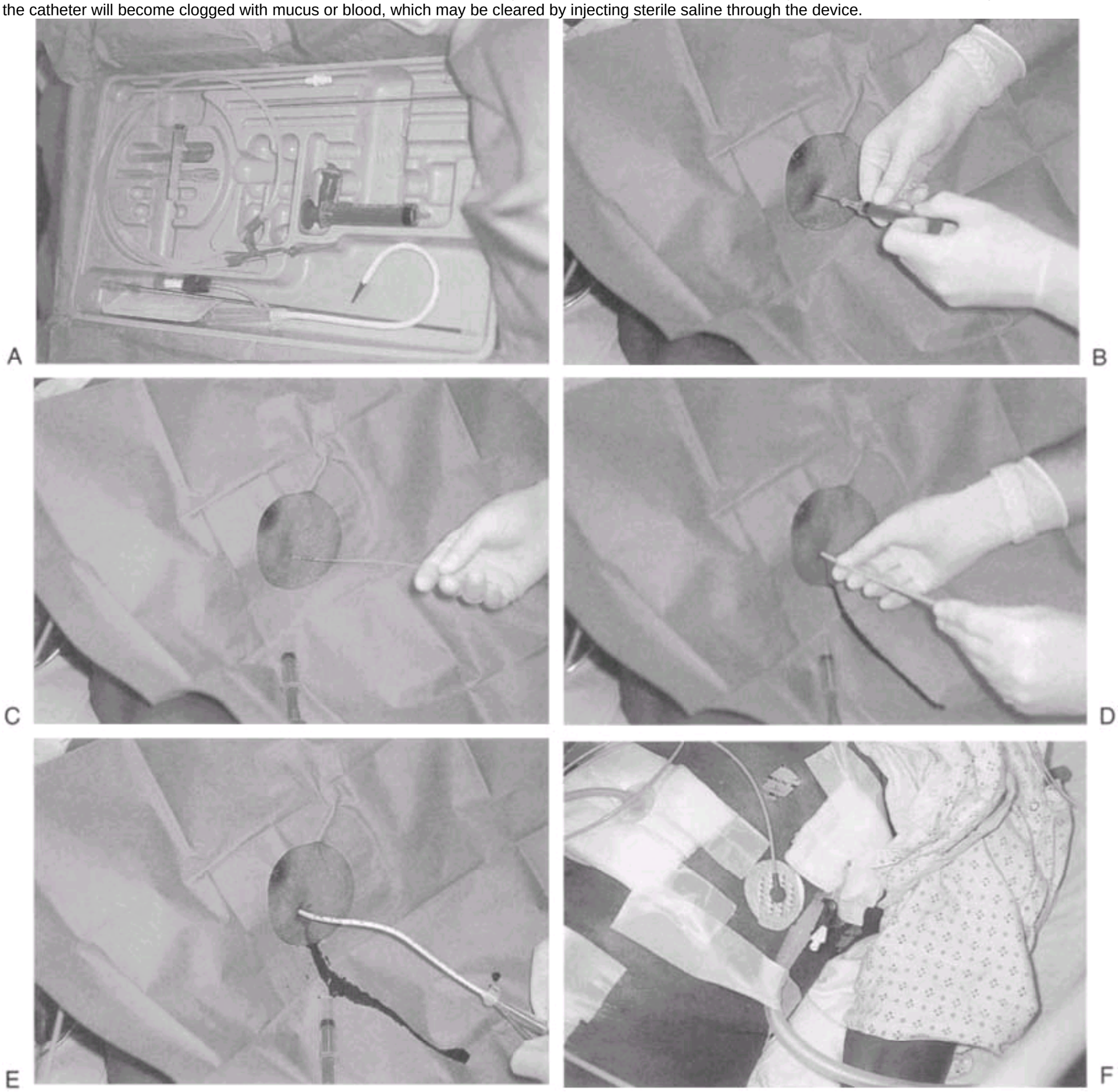
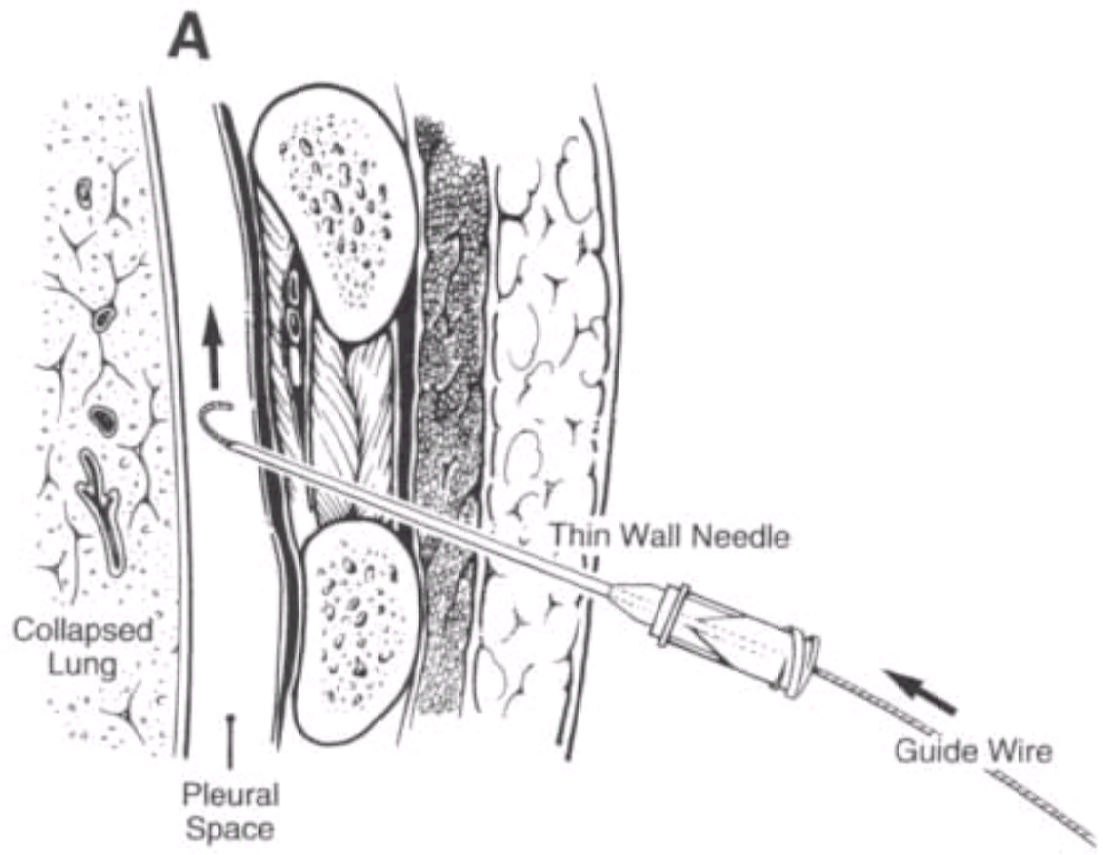
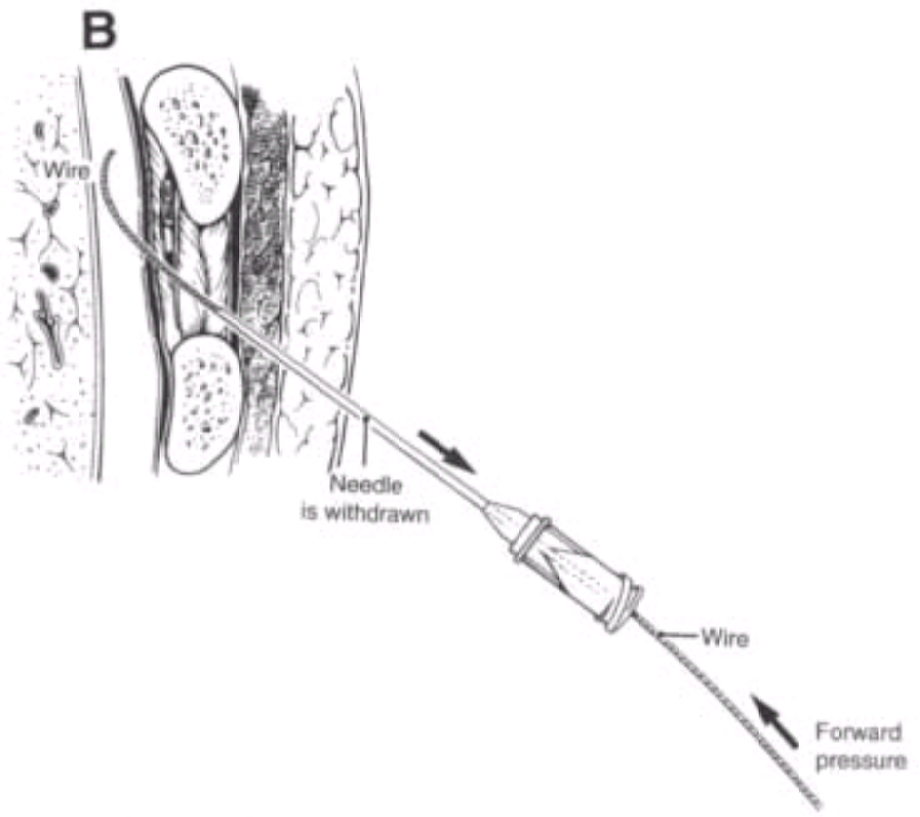
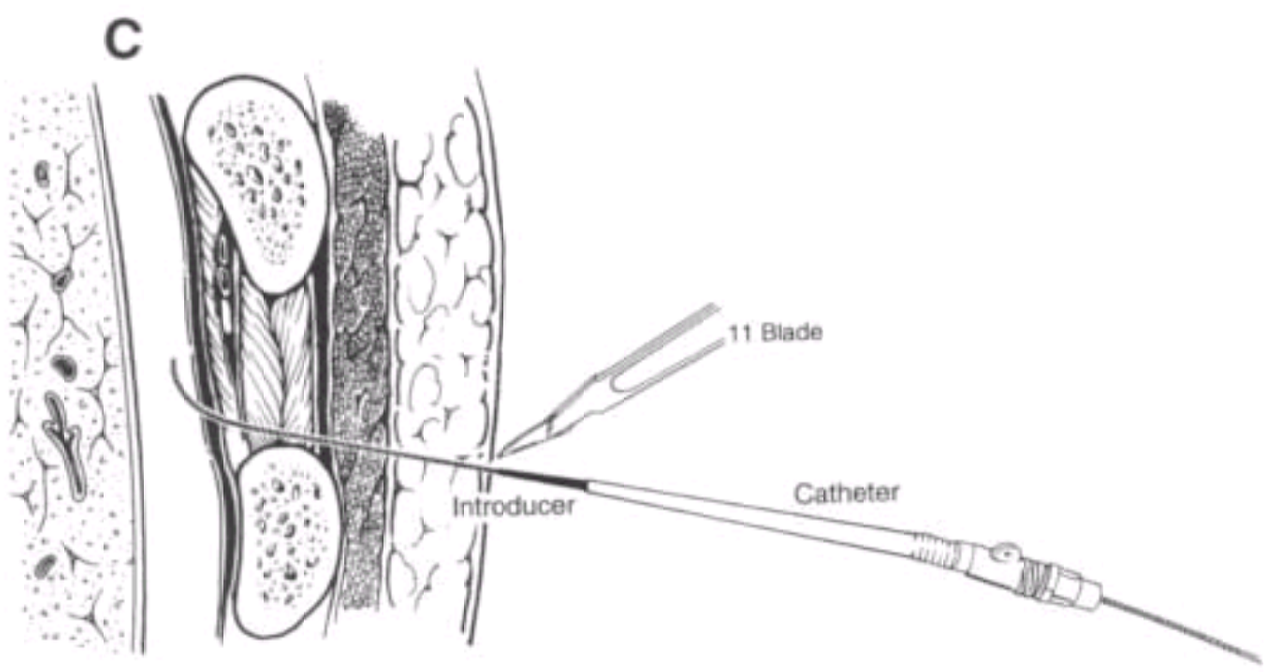
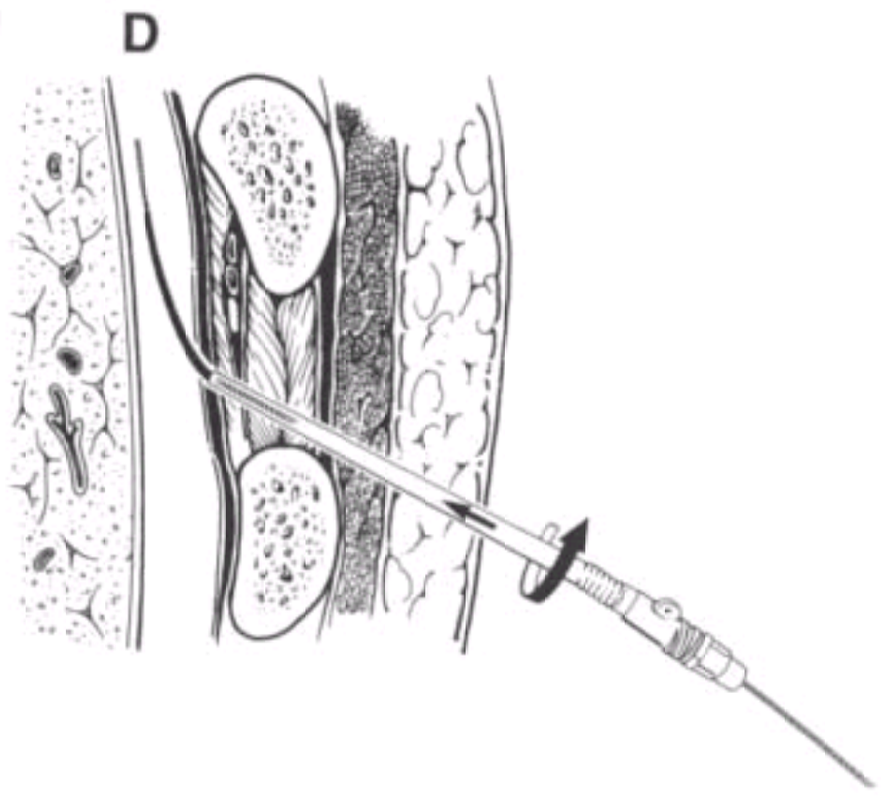


Figure 10-28 Seldinger technique for aspiration catheter insertion. *A*, Guidewire is passed through a needle over the rib into the pleural space. *B*, The needle is removed with the wire in the pleural space. *C* and *D*, A nick is made in the skin with a No. 11 blade, and the introducer and catheter are threaded into the pleural space. A twisting motion may be helpful. *E*, The guidewire and introducer are removed, leaving the catheter in the pleural space.









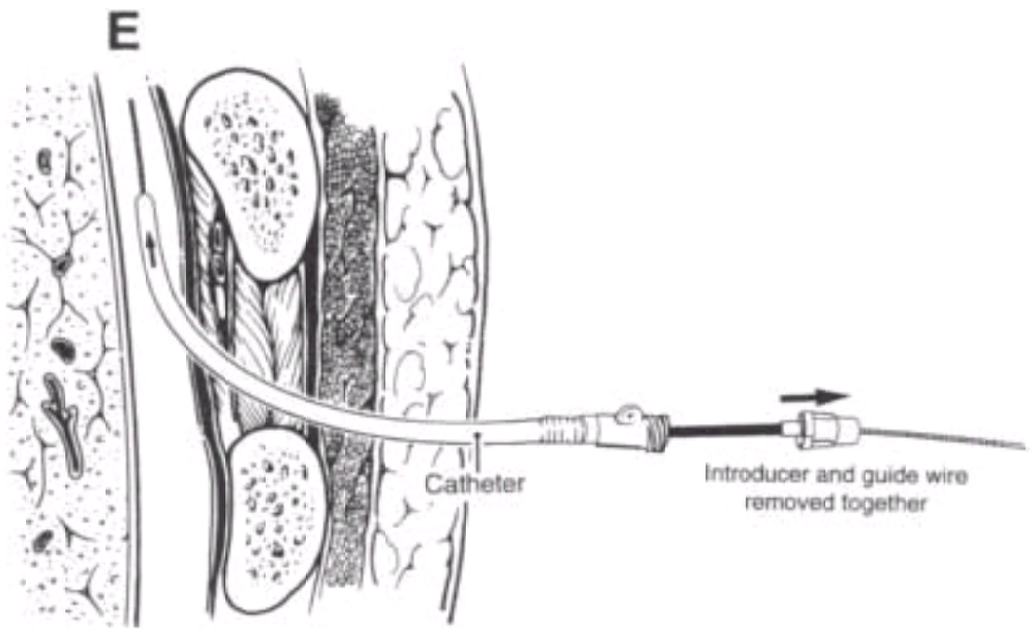


Figure 10-29 Over-the-needle catheter aspiration. *A,* A small nick is made in the skin with a No. 11 blade. The over-the-needle catheter is inserted through the nick into the pleural space. Proper placement is confirmed by the free flow of air into the attached syringe. *B,* The catheter is then threaded over the needle into the pleural space, and the needle is withdrawn.

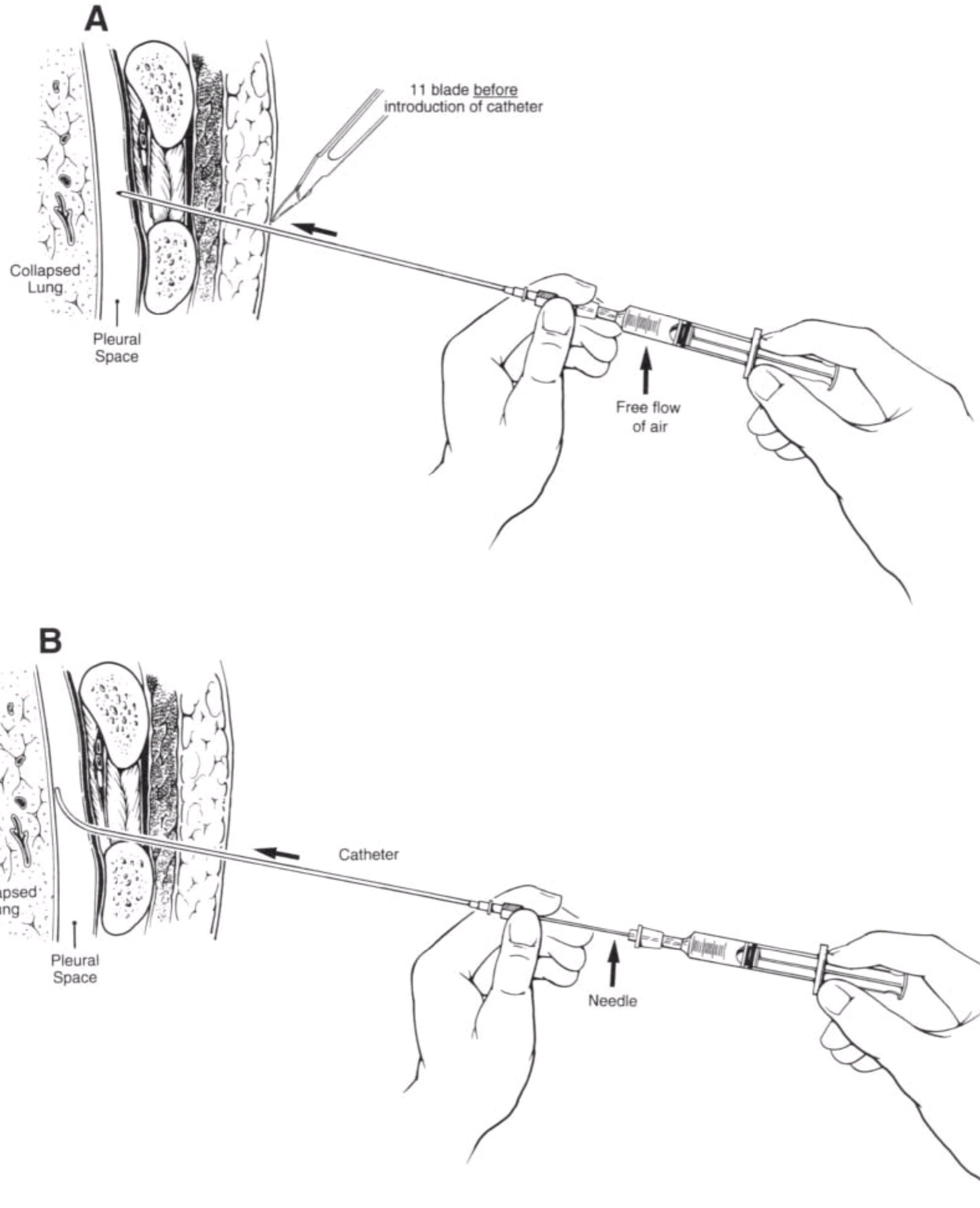


Figure 11-1 Anatomy of the carotid sinus reflex. Carotid receptors send impulses to the medulla by way of the sinus nerve of Hering and cranial nerve IX. Efferent nerves are shown on the right. *(Adapted from Scher AM: Control of arterial blood pressure. In Ruch TC, Patton HD: Physiology and Biophysics, vol 2, 20th ed. Philadelphia, WB Saunders, 1974.)*

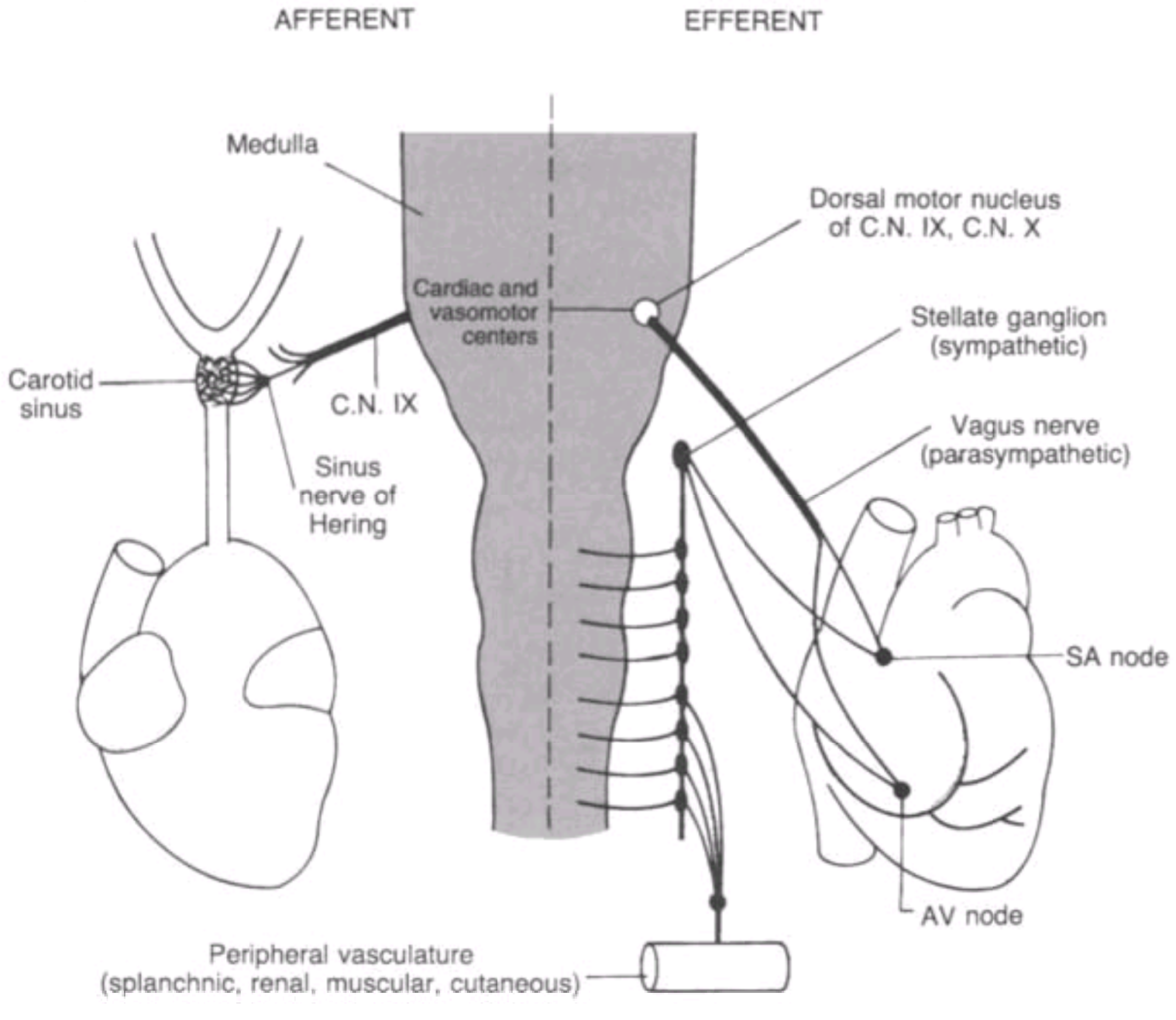


Figure 11-2 Ventricular Tachycardia. Carotid sinus massage (CSM) slows atria but not ventricles, thus establishing the presence of atrioventricular dissociation, supporting the diagnosis of ventricular tachycardia. The QRS measures 0.16 sec. Note the atrial rate slowing from 102 to 88 beats/min while the ventricular rate is unaffected. *(From Lown B, Levine SA: Carotid sinus—Clinical value of its stimulation. Circulation 23:766, 1961. Reproduced by permission.)*

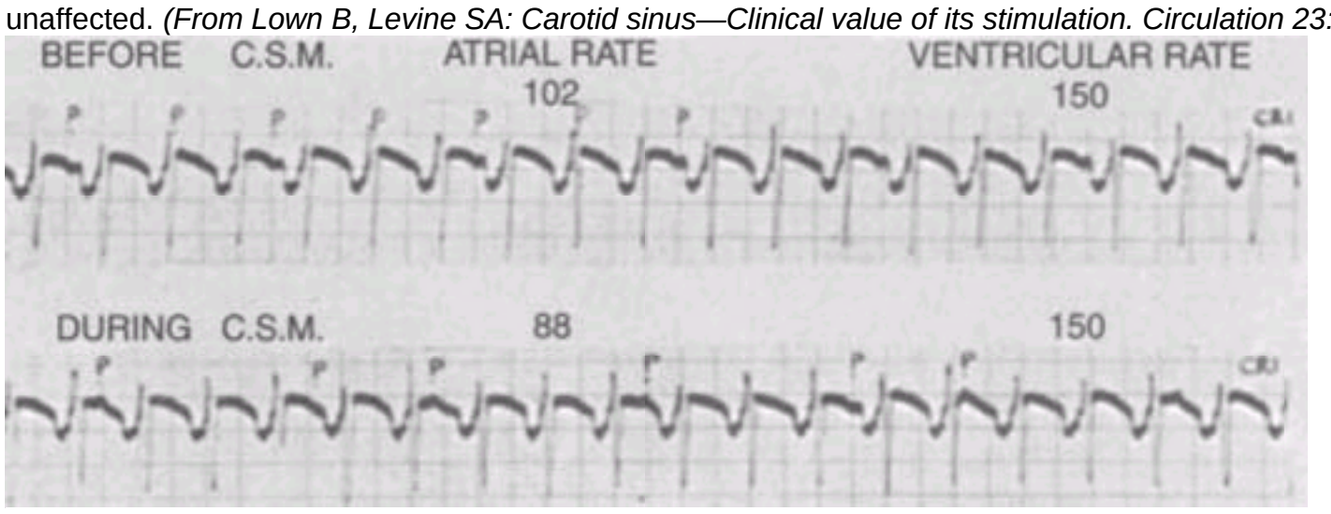


Figure 11-3 Paroxysmal atrial tachycardia with variable block. Carotid sinus pressure uncovers P waves hidden in the ventricular complex. The upper strip resembles atrial flutter or atrial fibrillation with ventricular ectopic beats. The lower strip shows paroxysmal atrial tachycardia with variable block at an atrial rate of 166 beats/min. *(From Lown B, Levine SA: Carotid sinus—Clinical value of its stimulation. Circulation 23:766, 1961. Reproduced by permission.)*

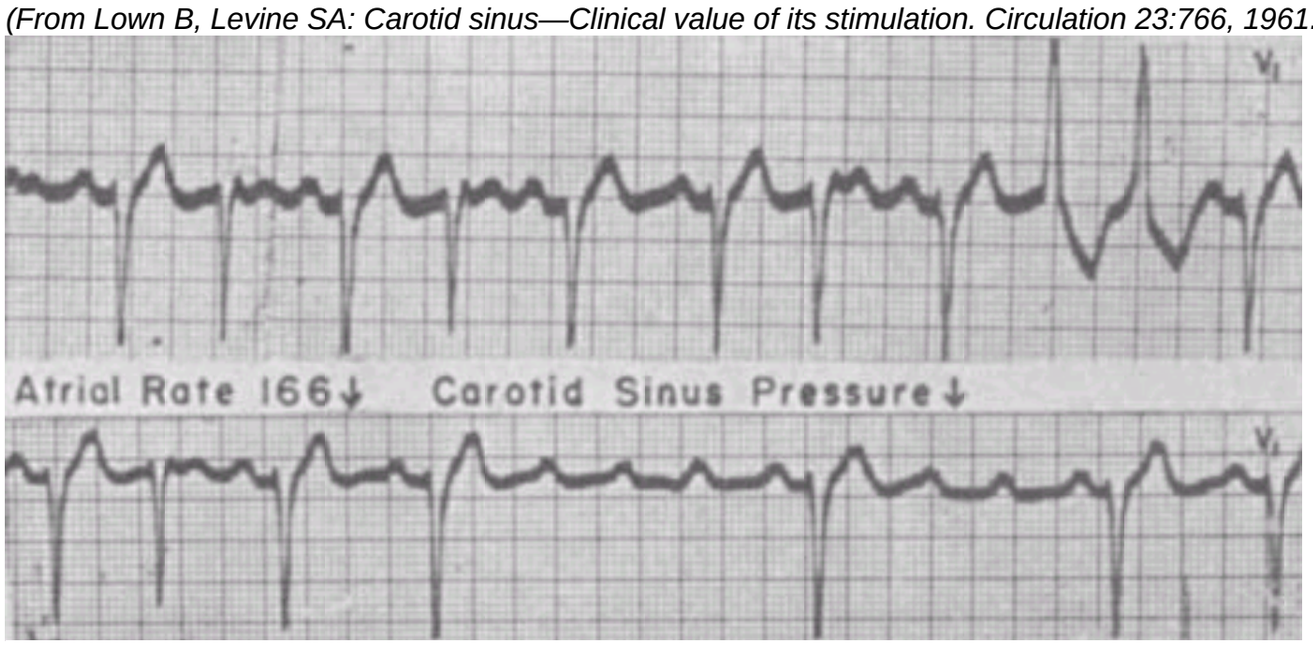


Figure 11-4 Sinus tachycardia. The sinus P wave is obscured within the descending limb of the T wave. Carotid sinus massage (CSM) transiently slows the sinus rate and exposes the P wave. The rate then increases. The strips are continuous. *(From Silverman ME: Recognition and treatment of arrhythmias. In Schwartz GR, Safar P, Stone JH, et al (eds): Principles and Practice of Emergency Medicine, vol. 2. Philadelphia, WB Saunders, 1978. Reproduced by permission.)*

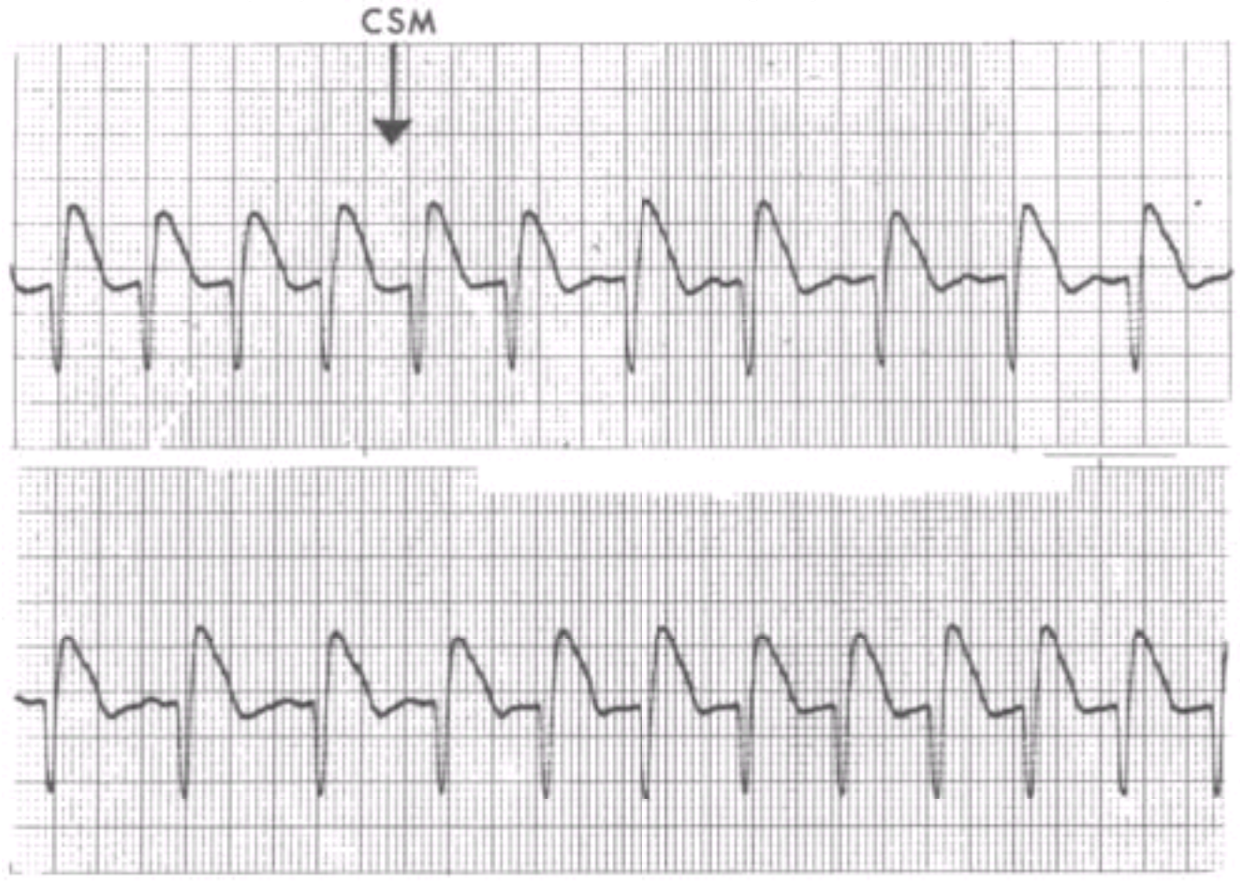


Figure 11-5 Sinus tachycardia with high-degree atrioventricular block. Arrows indicate sinus P waves. Strips II-a to II-d are continuous. The basic rhythm is sinus, but marked first-degree atrioventricular block is present. High-degree (advanced) atrioventricular block associated with transient slowing of sinus rate is produced by carotid sinus stimulation (CSS). *(From Chung EK: Electrocardiography, 2nd ed. New York, Harper & Row, 1980. Reproduced by permission.)*

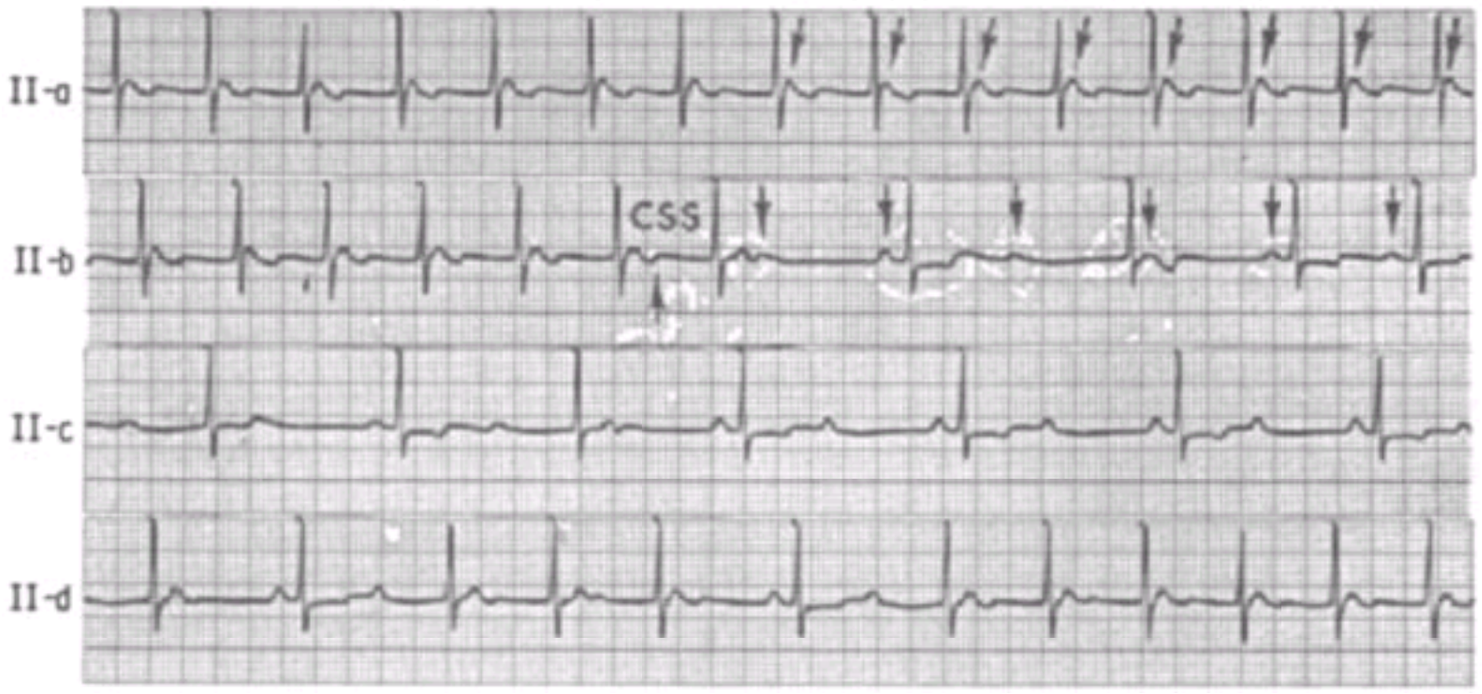


Figure 11-6 Paroxysmal atrial tachycardia. Carotid sinus massage (CSM) abolishes the dysrhythmia and results in a period of sinus suppression with a junctional (J) escape beat, followed thereafter by a sinus rhythm. Prolonged periods of asystole may produce anxiety in the clinician who is waiting for the resumption of a sinus pacemaker. *(From Silverman ME: Recognition and treatment of arrhythmias. In Schwartz GR, Safar P, Stone JH, et al (eds): Principles and Practice of Emergency Medicine, vol 2. Philadelphia, WB Saunders, 1978. Reproduced by permission.)*

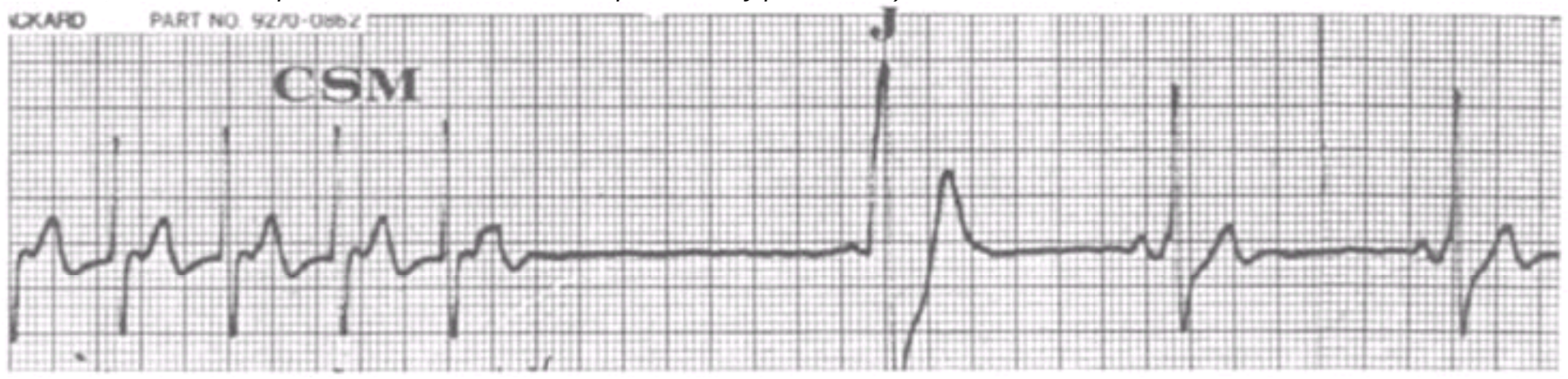


Figure 11-7 Atrial flutter. Carotid sinus stimulation (CSS, *downward arrow*) produces marked slowing of the ventricular rate in atrial flutter. Note the obvious flutter waves with an atrial rate of 300 and a long period of ventricular standstill. Strips are continuous. *(From Chung EK: Electrocardiography, 2nd ed. New York, Harper & Row, 1980. Reproduced by permission.)*

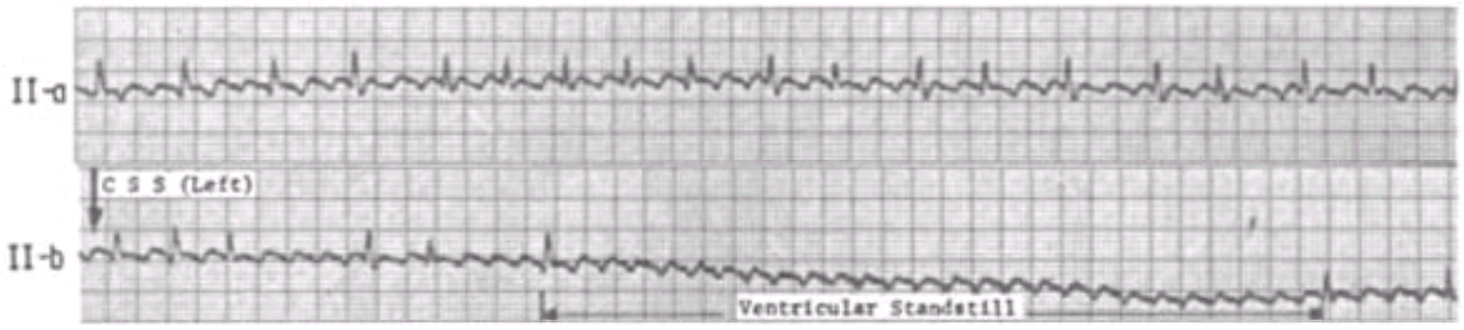


Figure 11-8 Atrial fibrillation. Carotid sinus massage (CSM) slows the ventricular response transiently, revealing the fibrillating baseline. The ventricular rate subsequently accelerates. *(From Silverman ME: Recognition and treatment of arrhythmias. In Schwartz GR, Safar P, Stone JH, et al (eds): Principles and Practice of Emergency Medicine, vol. 2. Philadelphia, WB Saunders, 1978. Reproduced by permission.)*

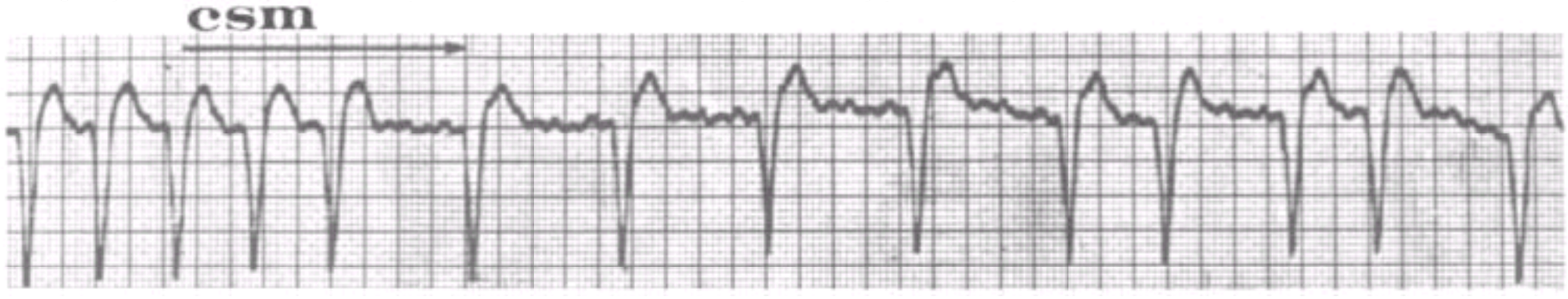


Figure 11-9 Occult premature ventricular contractions. Carotid sinus massage (CSM) reveals ventricular extrasystoles, thereby explaining the cause of palpitation in this case. *(From Lown B, Levine SA: Carotid sinus—Clinical value of its stimulation. Circulation 23:766, 1961. Reproduced by permission.)*

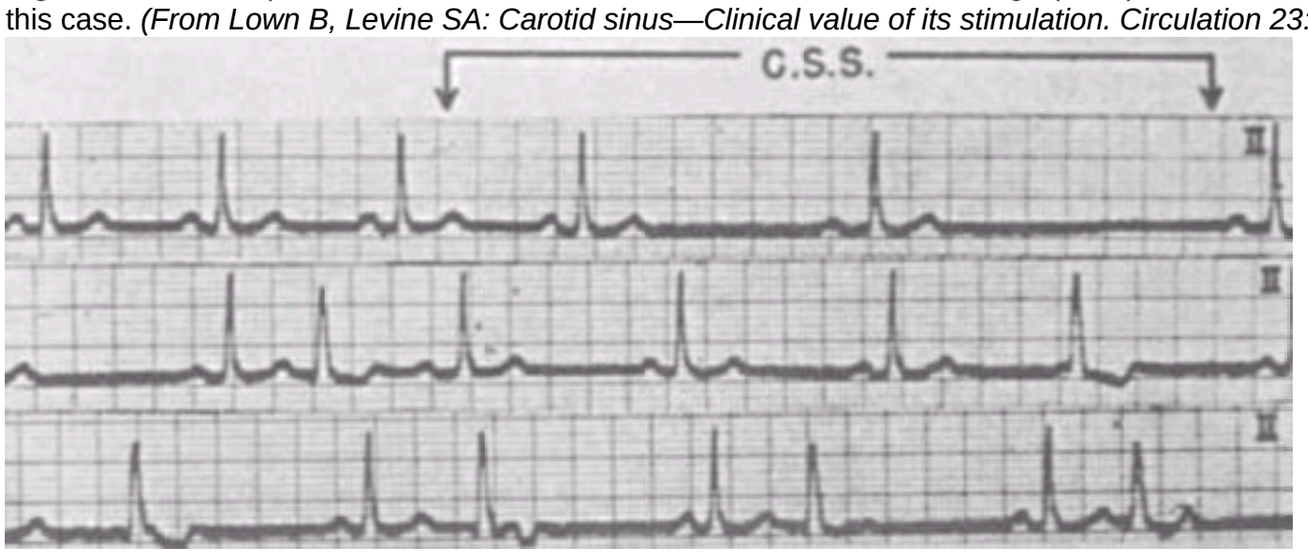


Figure 11-10 A run of ventricular tachycardia is seen immediately after a supraventricular dysrhythmia is terminated by cardiac sinus massage (CSM). The patient remained asymptomatic, and a normal sinus rhythm was established spontaneously within a few seconds.

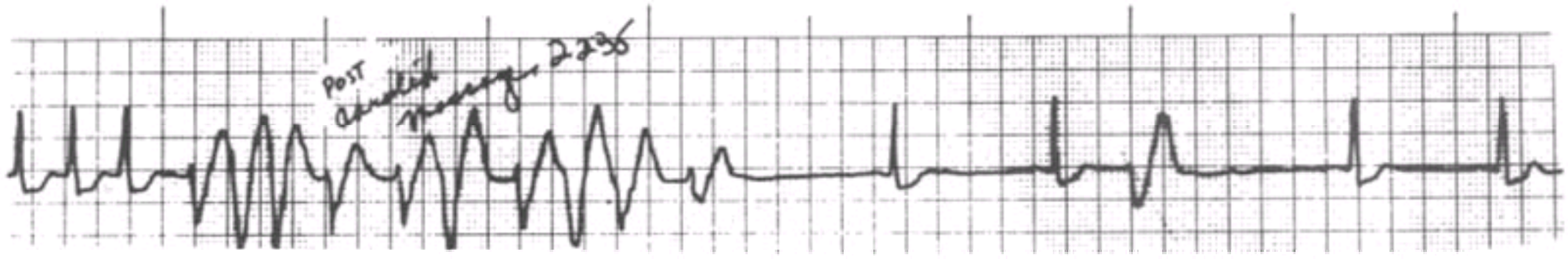


Figure 11-11 Hyperreactive carotid sinus reflex. Gentle pressure was applied to the carotid sinus for 3 seconds, resulting in a pause in sinus rhythm of approximately 7 seconds. This syndrome may be the cause of syncope. *(From Bigger JT Jr: Mechanisms and diagnosis of arrhythmias. In Braunwald E (ed): Heart Disease, vol 1. Philadelphia, WB Saunders, 1980. Reproduced by permission.)*

Figure 11-12 Acceleration of ventricular rate by carotid sinus stimulation (CSS). Continuous tracing. Upper strip shows 2:1 atrioventricular block: Atrial rate at approximately 102/min; ventricular rate at approximately 51/min. The second and third strips were recorded during and after CSS, when the atrial rate was reduced to 68/min; a 1:1 response occurs. *(From Lown B, Levine SA: Carotid sinus—Clinical value of its stimulation. Circulation 23:766, 1961. Reproduced by permission.)*

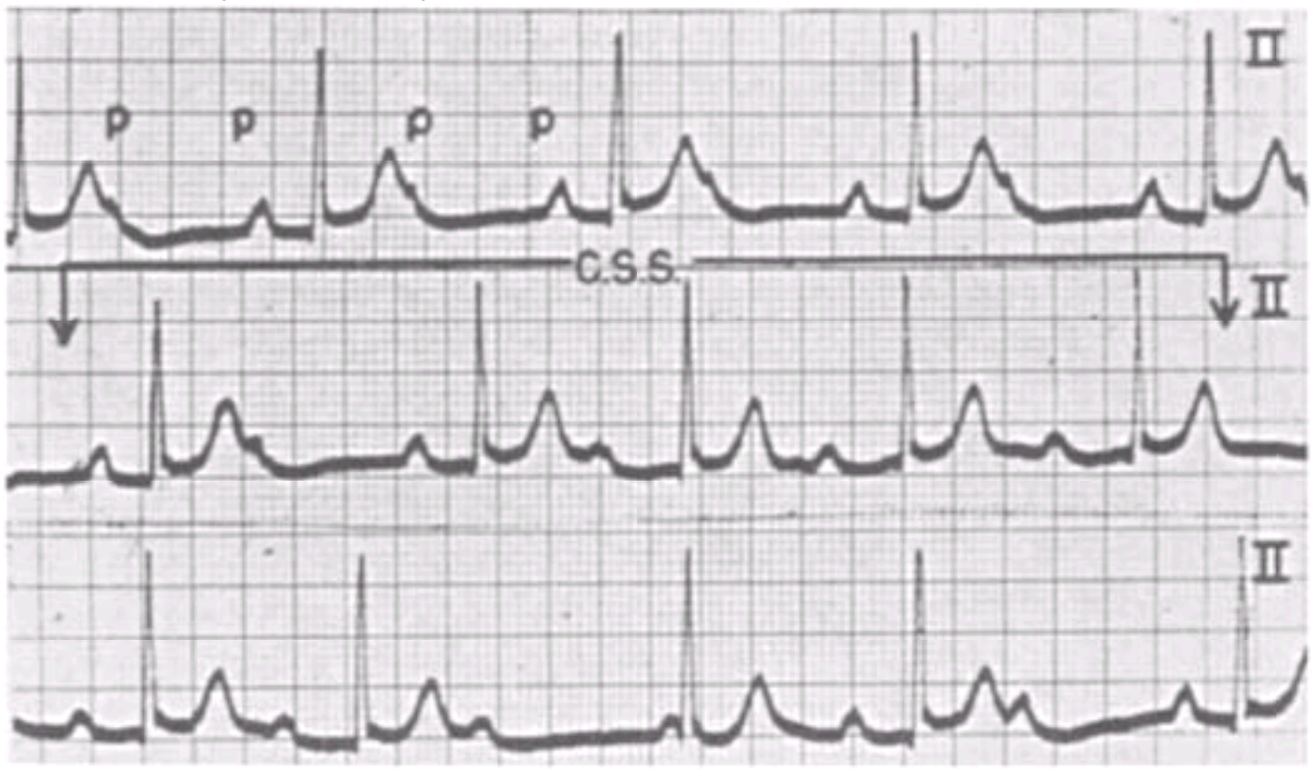


Figure 12-1 Success versus time; graph depicting relationship between survival to hospital discharge after ventricular fibrillation (VF) cardiac arrest and time interval between collapse and defibrillation. There is a 7% to 10% decrease in survival for each minute of VF duration. *(From Larsen MP, Eisenberg MS, Cummins RO, Hallstrom AP: Predicting survival from out-of-hospital cardiac arrest: A geographic model. Ann Emerg Med 22:1652, 1993. Reproduced with permission.)*

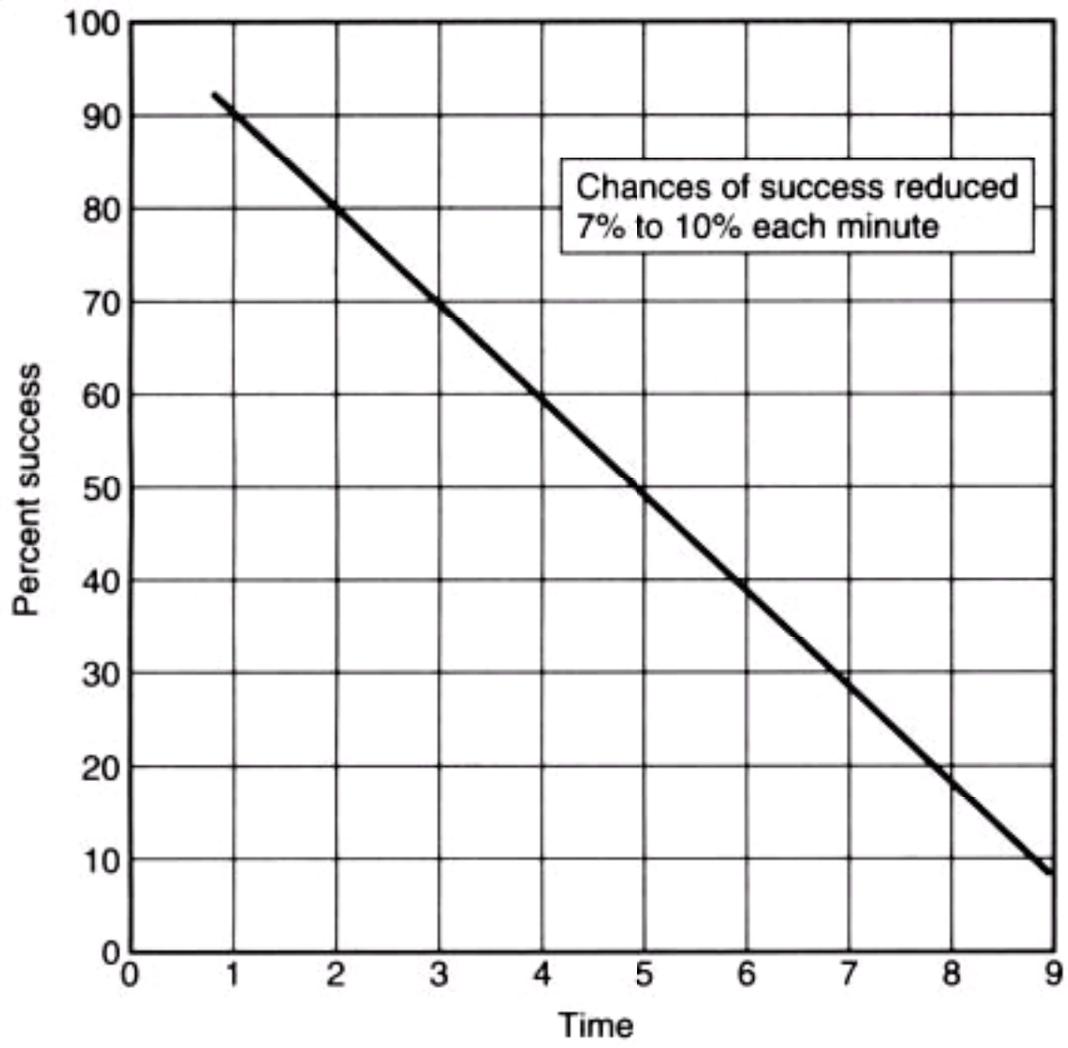


Figure 12-2 Leads aVR, aVL, and aVF from an animal with electrically induced ventricular fibrillation (VF). From onset to 4.5 minutes, VF waves were obvious in all 6 frontal plane leads. At 5.0 and 5.5 minutes, lead aVR was a straight line. Note that aVR is the electrical sum of leads aVL and aVF. By 6 minutes, the null vector had changed, and VF was again evident in all 6 frontal plane leads. *(From Ewy GA, Dahl CF, Zimmerman M, et al: Ventricular fibrillation masquerading as ventricular standstill. Crit Care Med 9:841, 1981. Reproduced by permission.)*

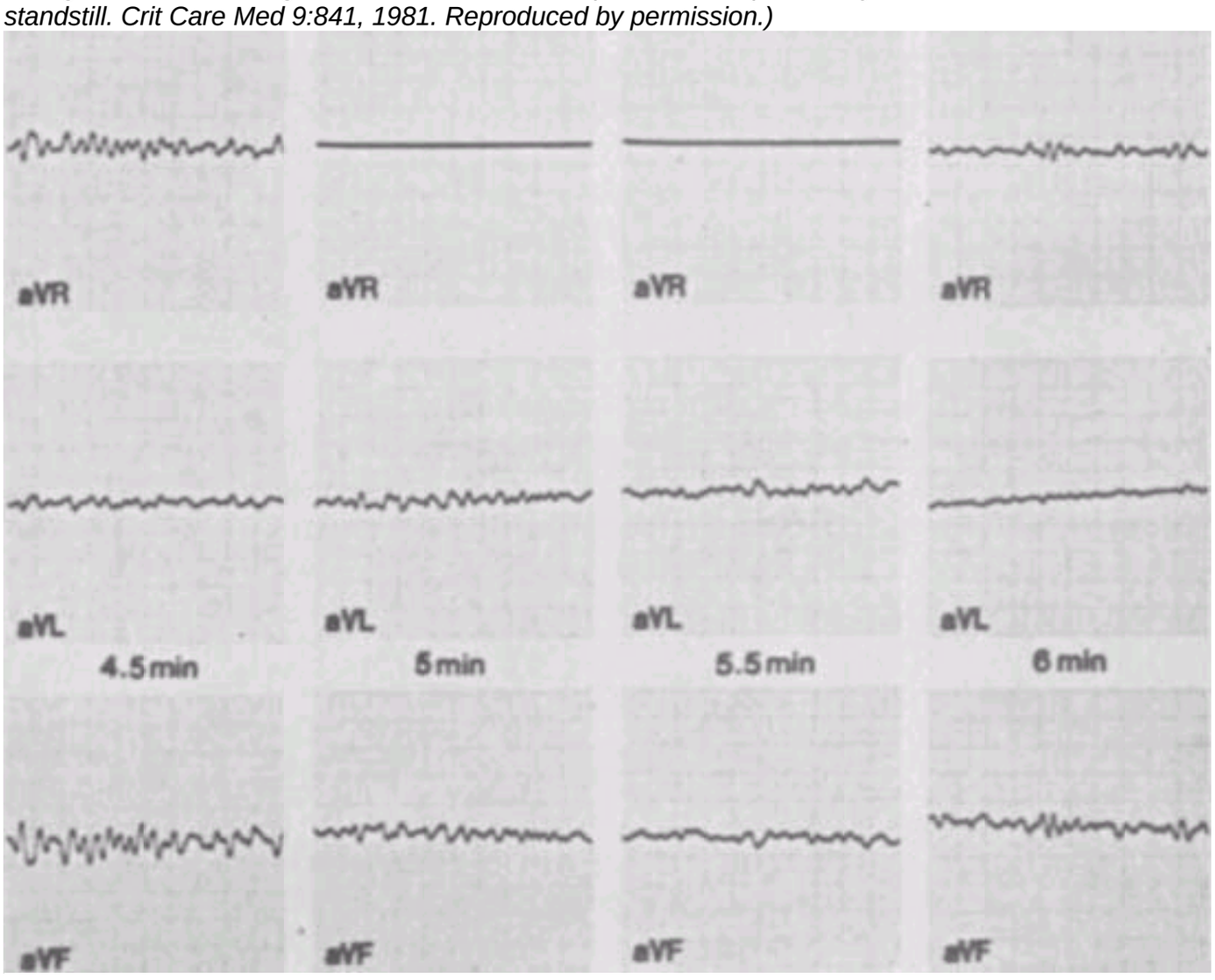


Figure 12-3 Phases of vulnerability for atrium and ventricle. Note that an alternating current shock of 0.20 seconds may end at the T wave even when synchronized with the R wave of the electrocardiogram. *(From Resnekov L: Theory and practice of electroversion in cardiac dysrhythmias. Med Clin North Am 60:325, 1976. Reproduced by permission.)*

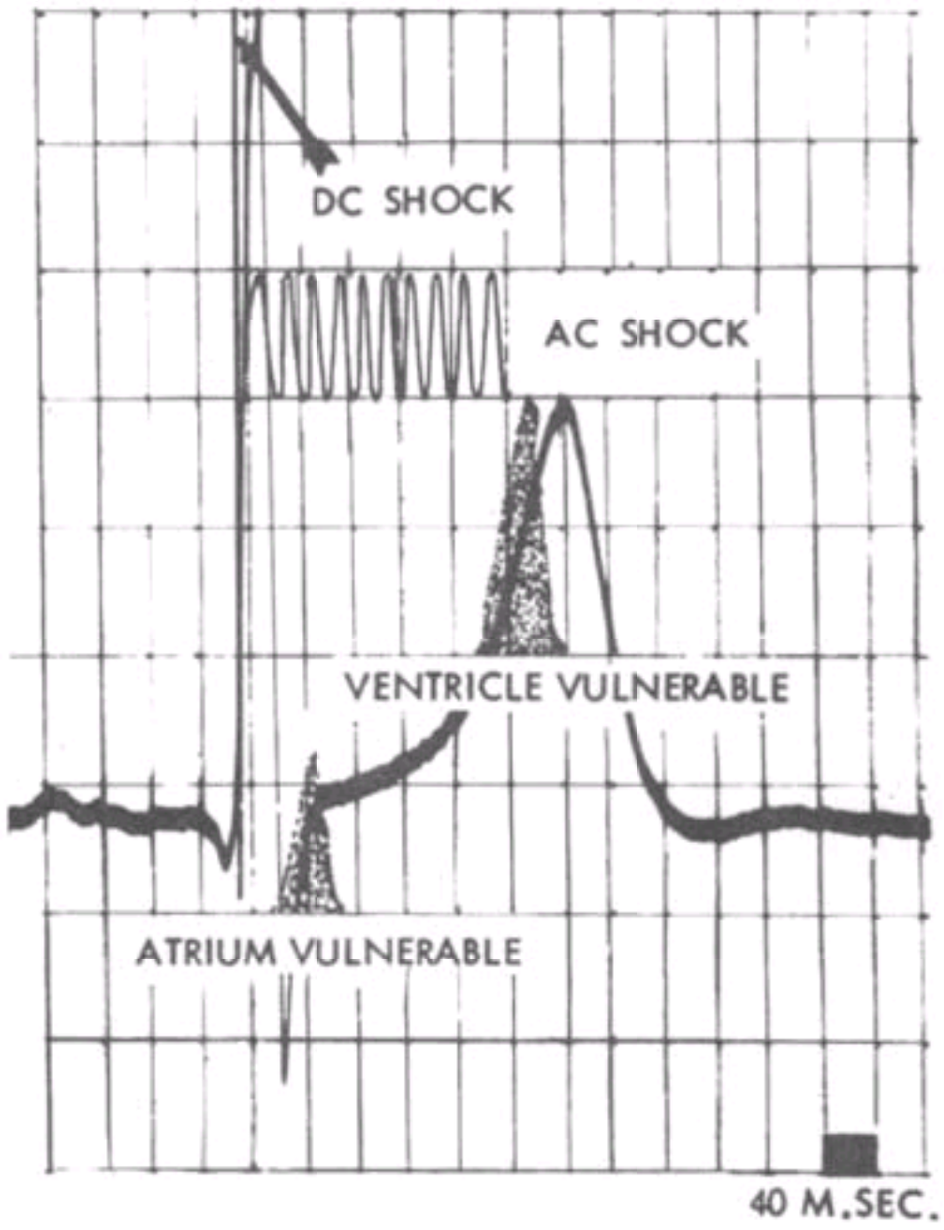


Figure 12-4 Monophasic damped sine waveform (MDS) (Energy delivered versus time/msec.) *(Adapted courtesy of Cardiac Science, Inc., Irvine, CA.)*

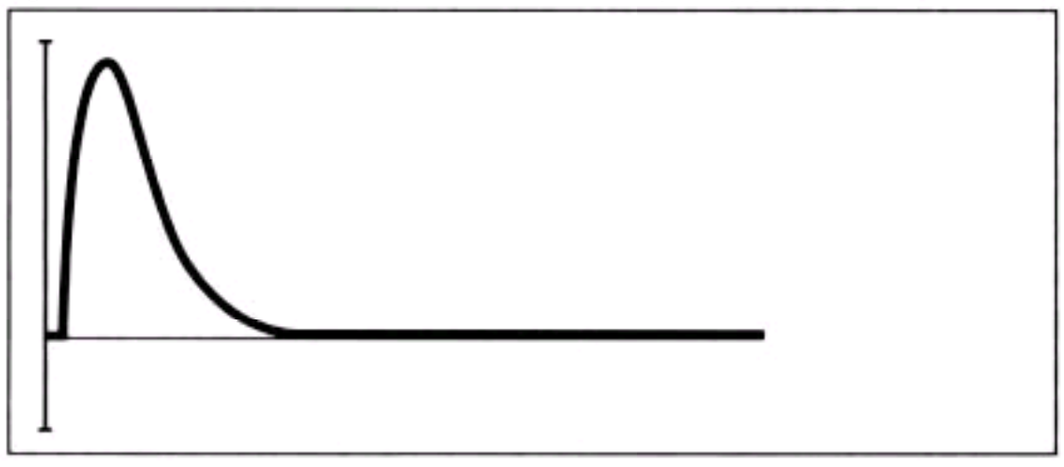


Figure 12-5 Monophasic truncated exponential waveform (MTE) (Energy delivered versus time/msec.) *(Adapted courtesy of Cardiac Science, Inc., Irvine, CA.)*

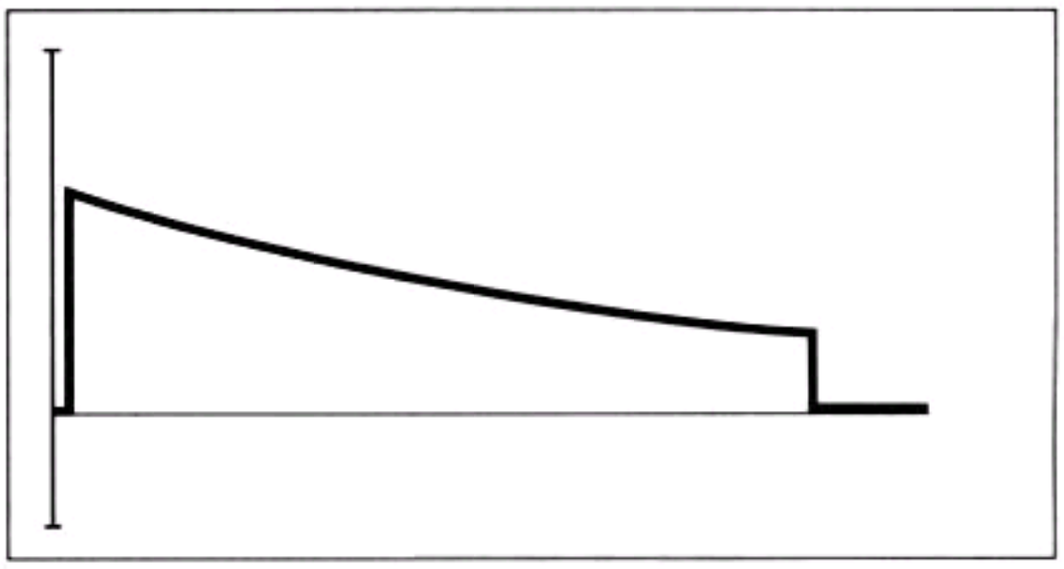


Figure 12-6 Biphasic truncated exponential waveform (BTE) (Energy delivered versus time/msec.) *(Adapted courtesy of Cardiac Science, Inc., Irvine, CA.)*

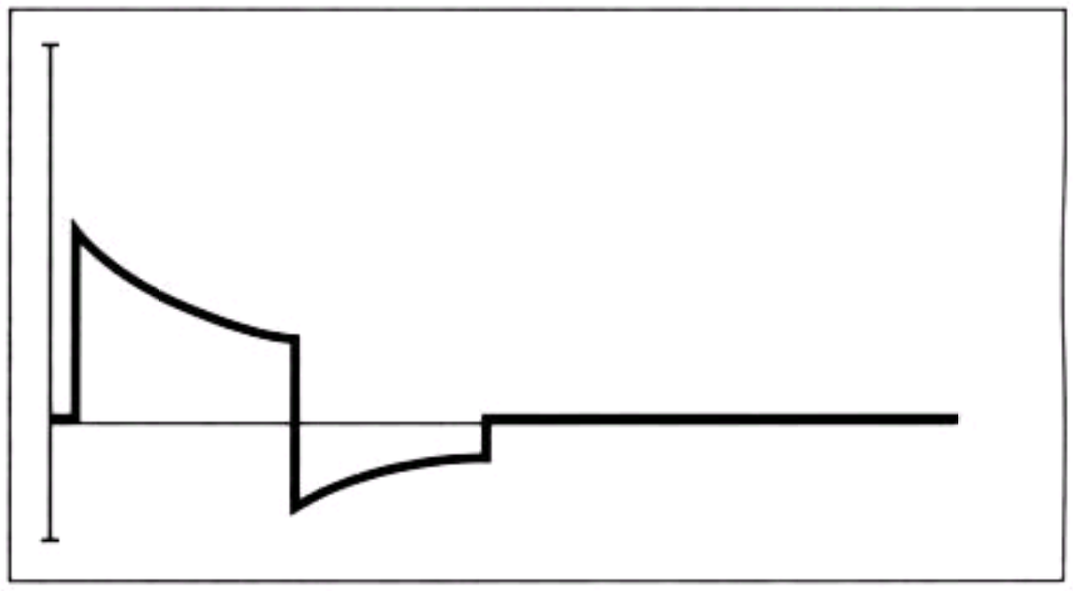


Figure 12-9 Ventricular fibrillation/pulses VT algorithm. *(From Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 102(Suppl 1):I-147, 2000.)*

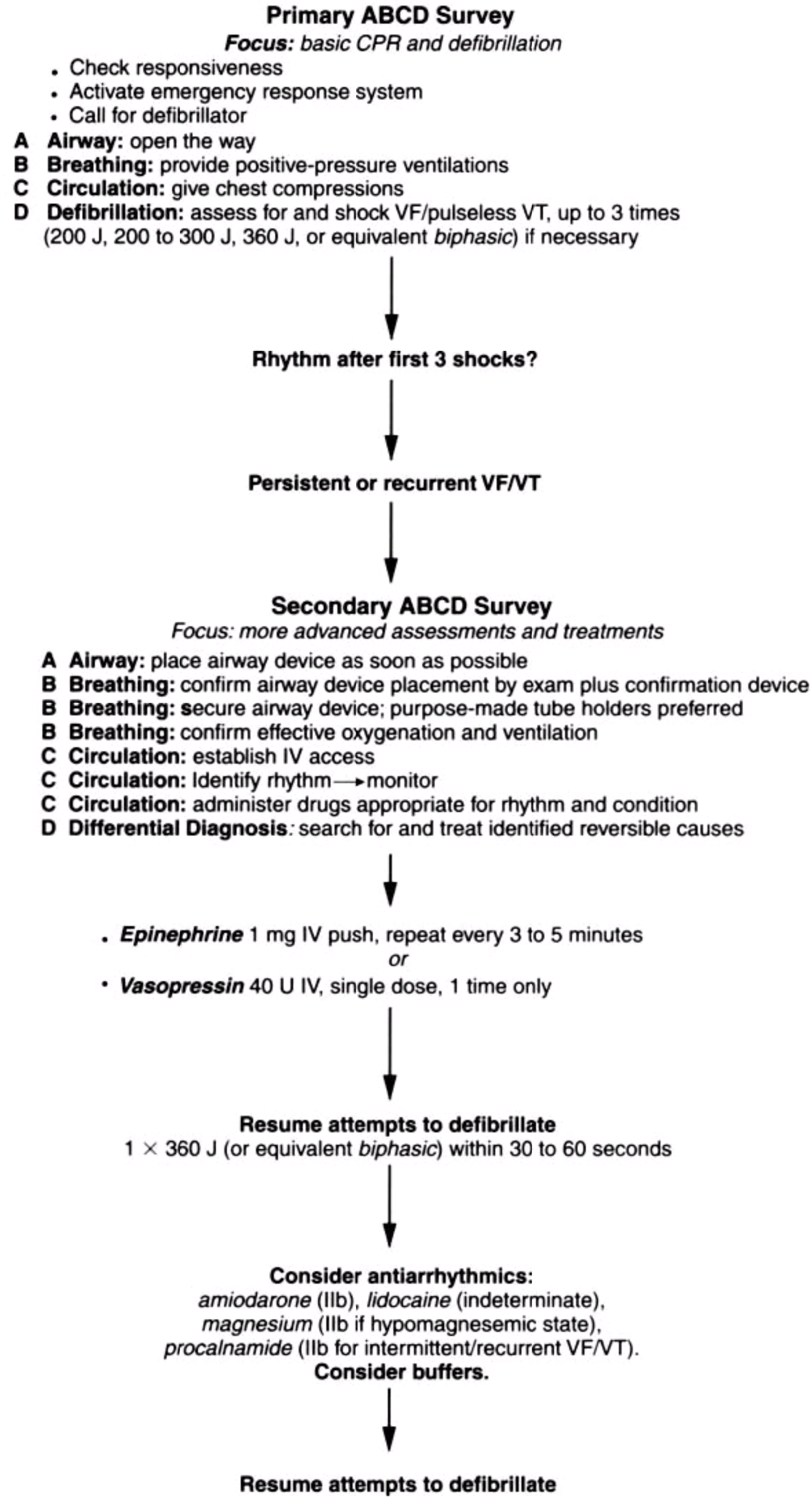


Figure 12-10 Anterolateral paddle electrode position. *(From Suratt PM, Gibson RS: Manual of Medical Procedures. St Louis, CV Mosby, 1982. Reproduced by permission.)*

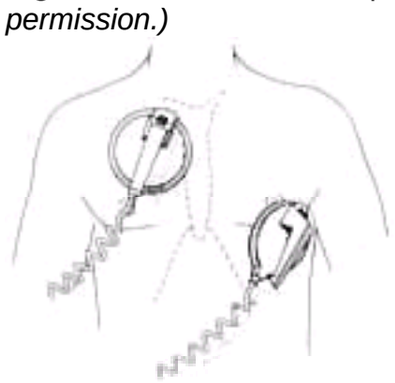


Figure 12-11 Anteroposterior paddle electrode position. Use this placement in tall, thin individuals with retrosternal cardiac location. *(From Suratt PM, Gibson RS: Manual of Medical Procedures. St Louis, CV Mosby, 1982. Reproduced by permission.)*

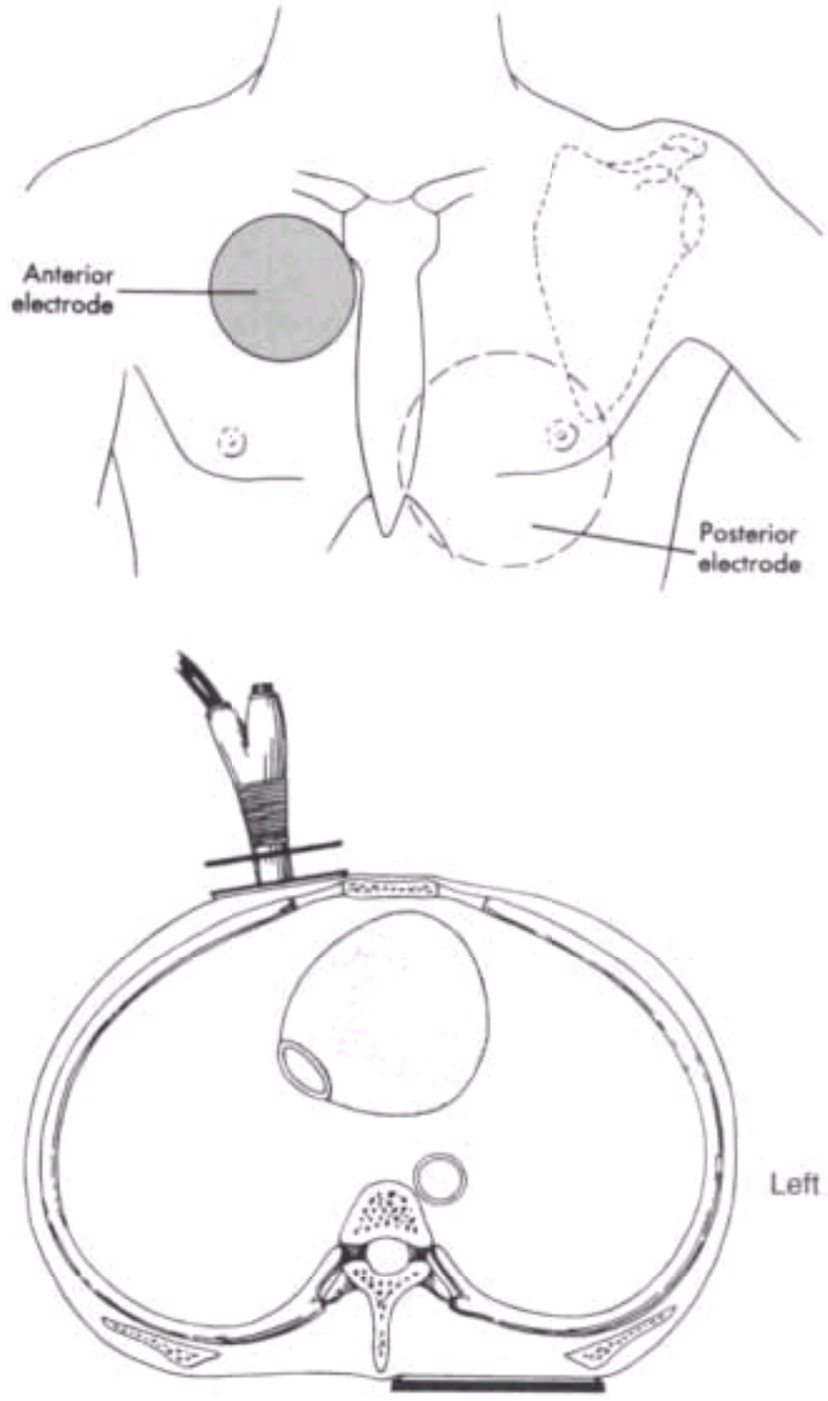


Figure 12-13 Automatic external defibrillator treatment algorithm for emergency cardiac care. *(From AHA Guidelines 2000, Circulation 102(suppl):1, 2000. Reproduced with permission.)*

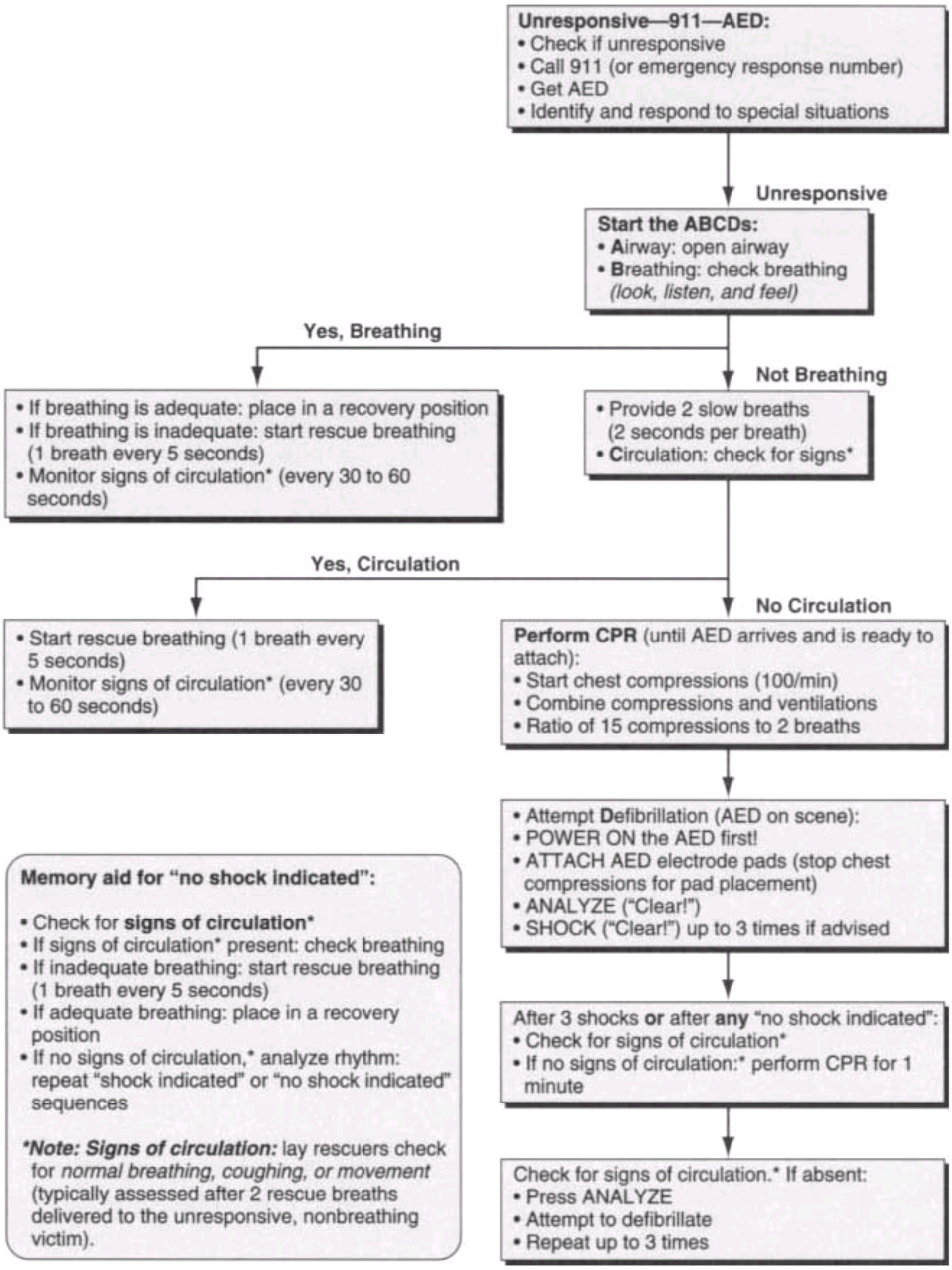


Figure 12-14 Correct automatic external defibrillator electrode placement on victim. *(From AHA Guidelines 2000, Circulation 102(suppl):1, 2000. Reproduced with permission.)*

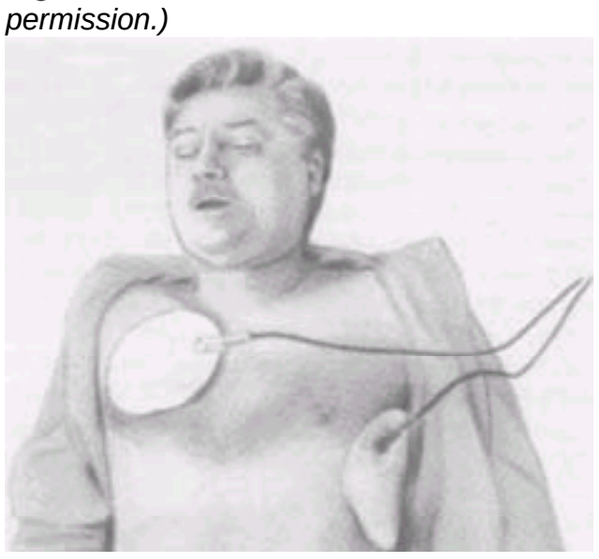


Figure 12-15 Pediatric pulseless arrest Pediatric Advanced Life Support treatment algorithm. *(From AHA Guidelines 2000, Circulation 102(suppl):1, 2000. Reproduced with permission.)*

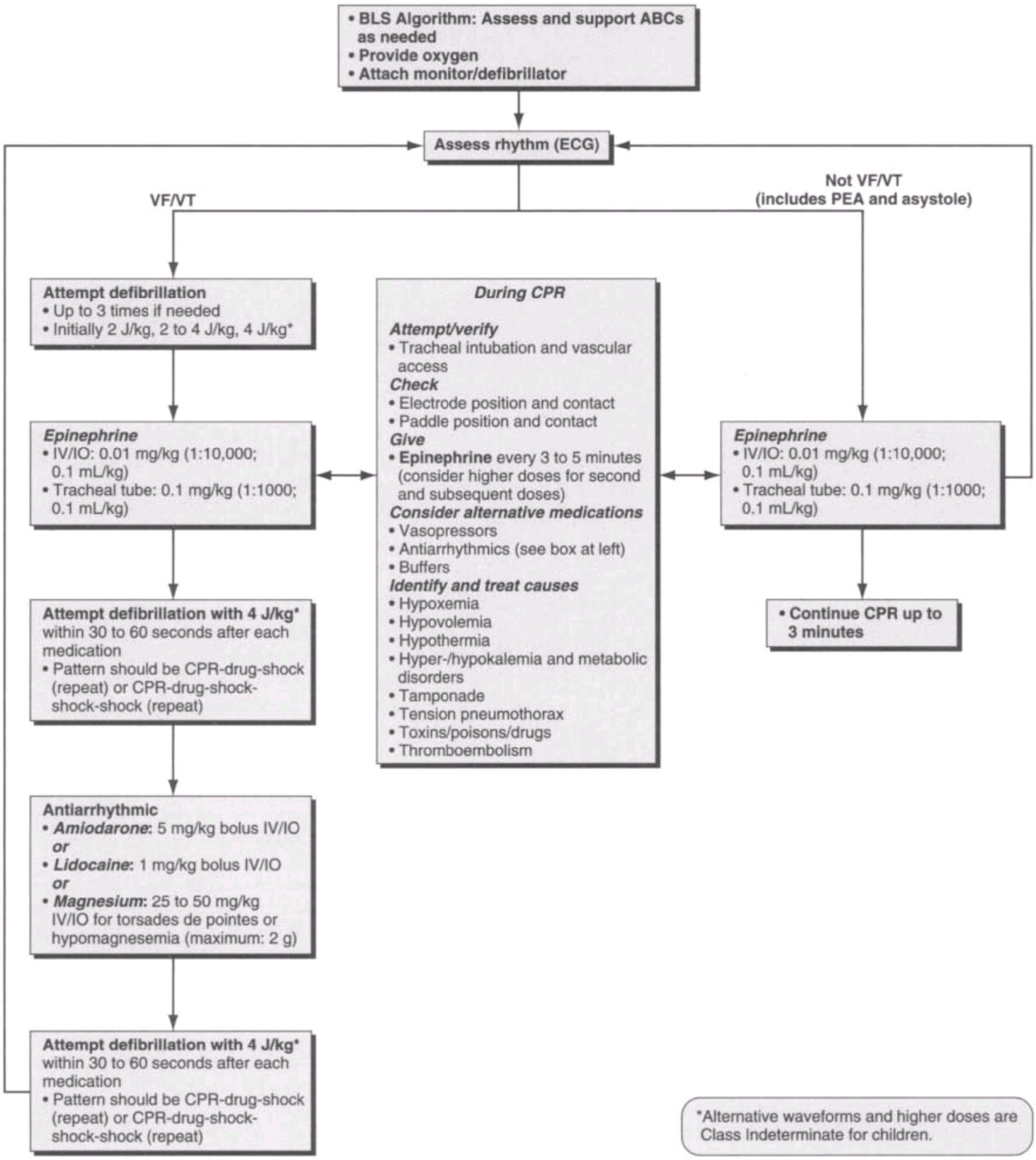


Figure 13-1 Radiographs of an implanted pacemaker showing battery and lead wires. *A*, standard view. *B*, Close-up view.

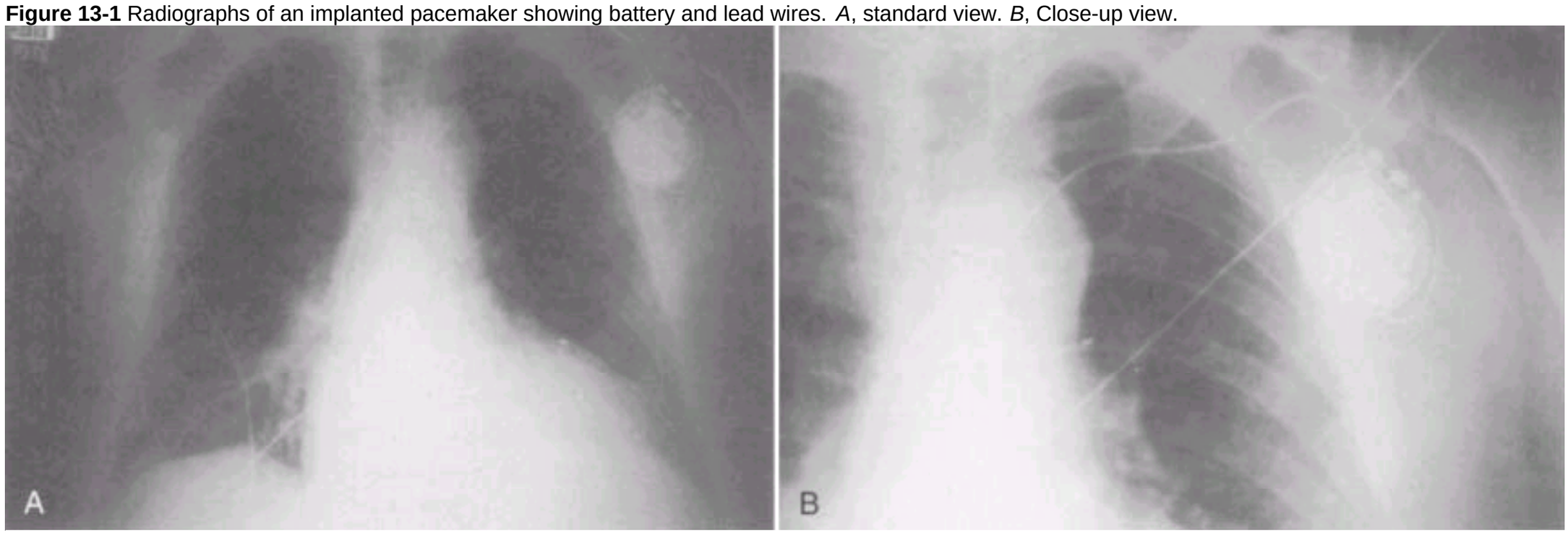


Figure 13-2 Automatic implantable cardioverter-defibrillator (AICD). *(Courtesy of Medtronics Inc., Minneapolis, MN.)*

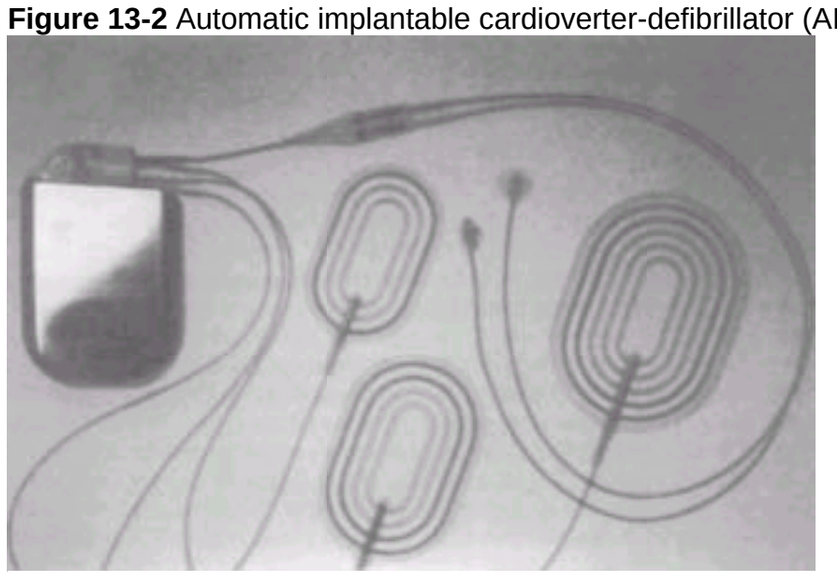


Figure 13-3 Posteroanterior *(A)* and lateral *(B)* chest radiographs demonstrating typical appearance of AICD with ventricular patches.

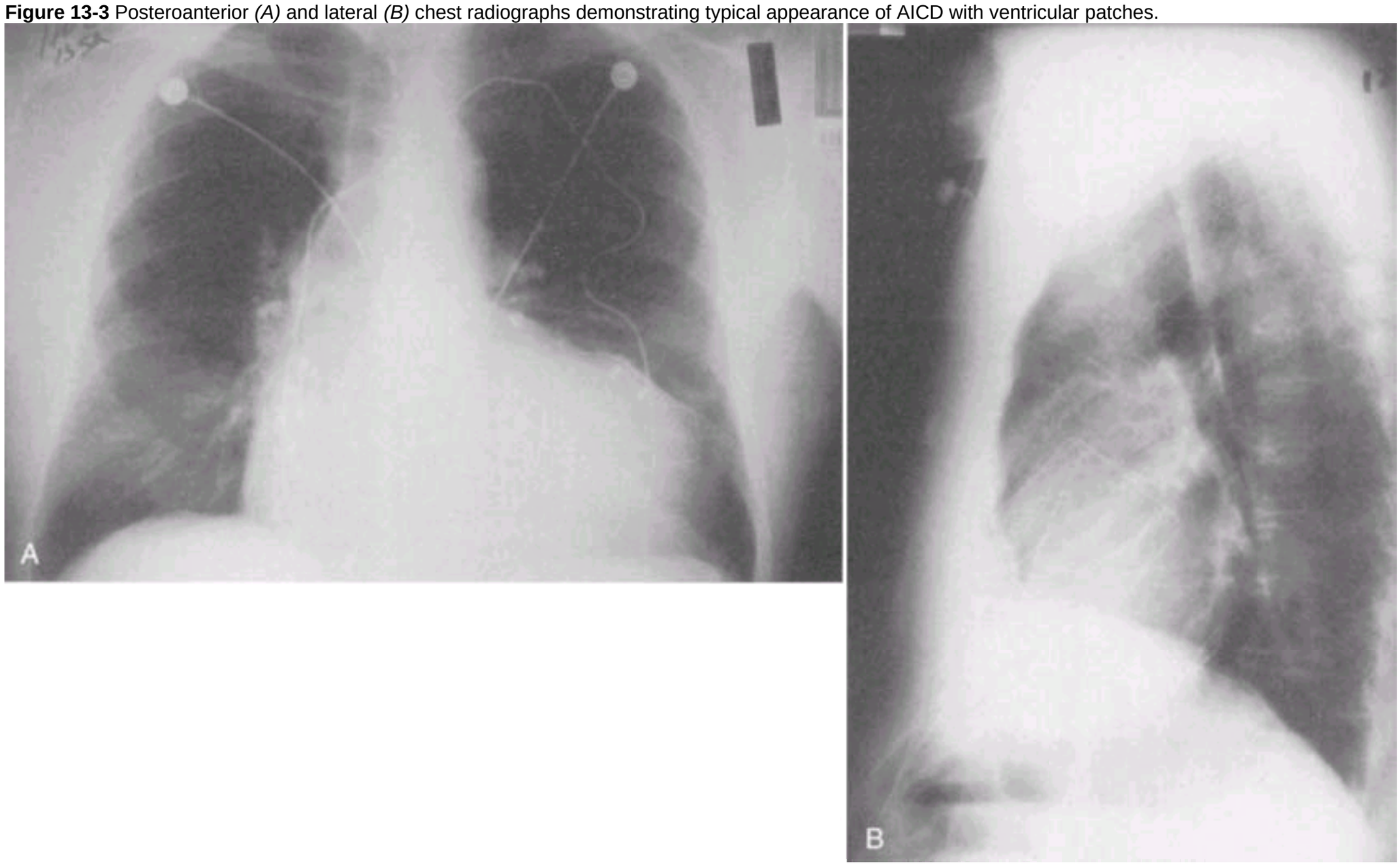


Figure 13-4 Posteroanterior *(A)* and lateral *(B)* chest radiographs demonstrating typical appearance of single-lead transvenous AICD.

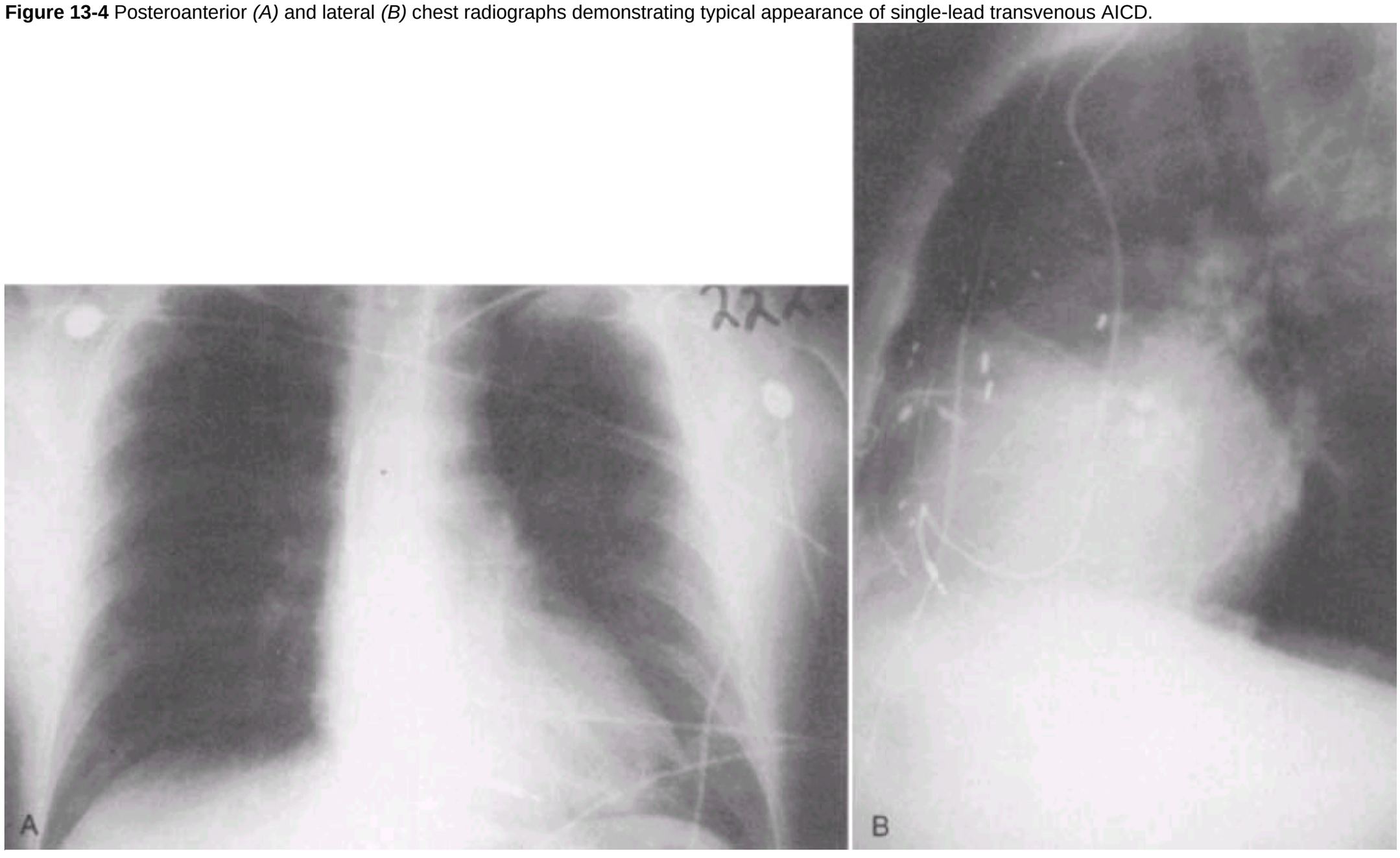


Figure 13-5 Typical external appearance of AICD implanted in the abdominal wall. *(Courtesy of Lawrence B. Stack, MD. From Munter DW, DeLacey WA: Automatic implantable cardioverter-defibrillators. Emerg Med Clin North Am 12:579, 1994. Used with permission.)*

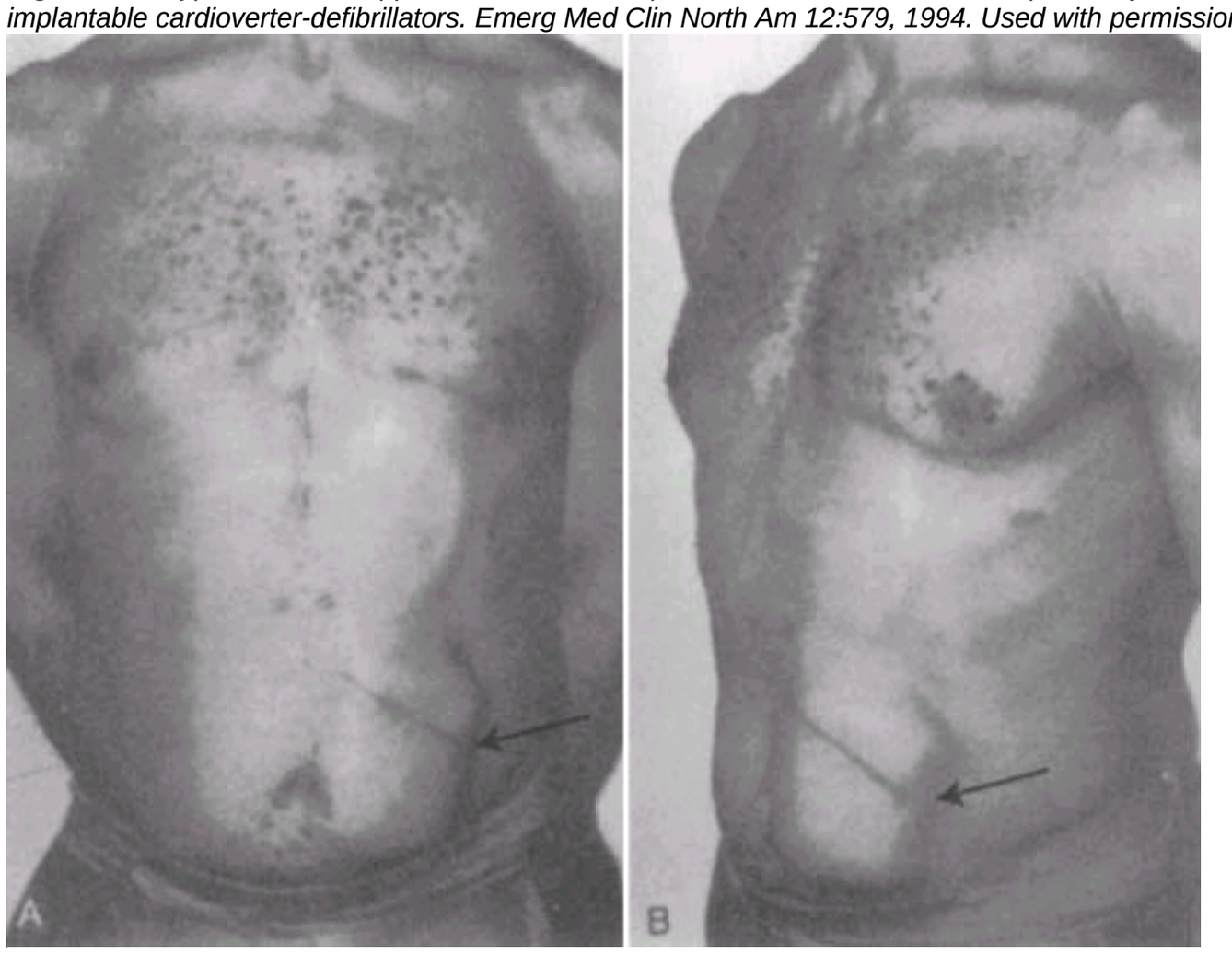


Figure 13-6 Radiographic appearance of the implanted AICD in the abdominal wall.

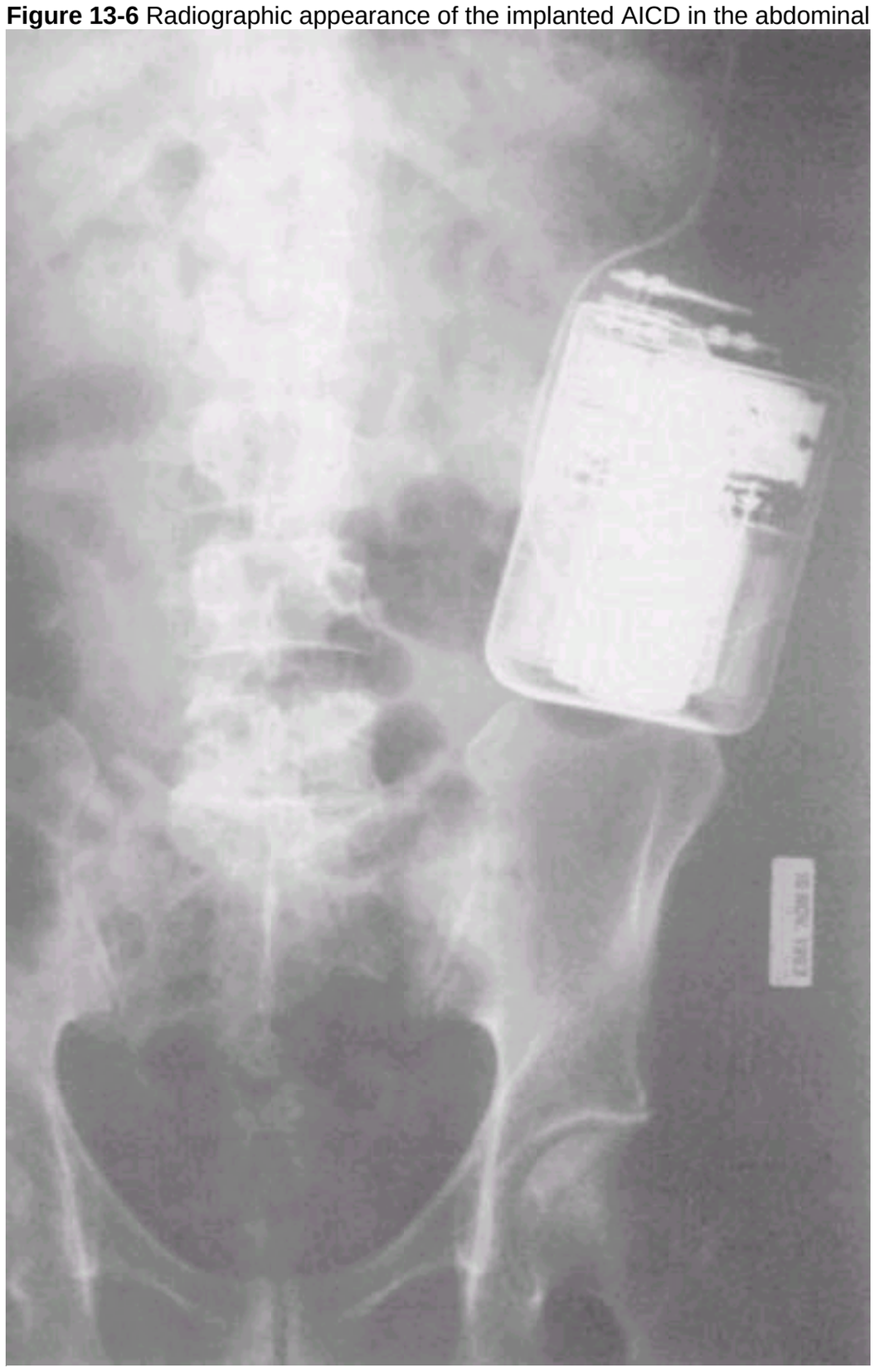


Figure 13-7 *A* and *B*, Appearance of fracture of atrial "J" retention wire. *A*, Radiographic appearance of fracture of atrial "J" retention wire. *B*, Diagram of fracture site. *(Courtesy of Telectronics Pacing Systems, Englewood, CO.)*

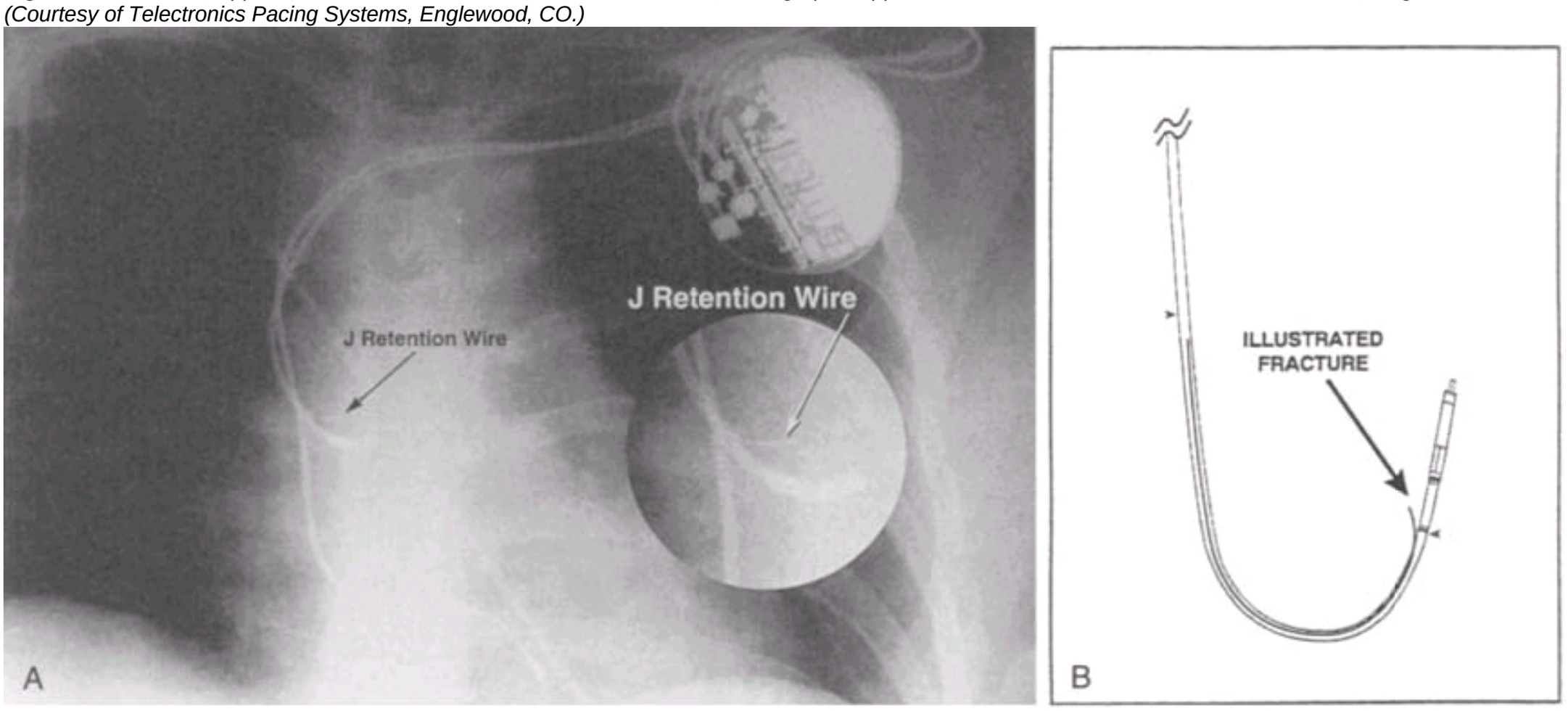


Figure 13-8 Ring magnet.

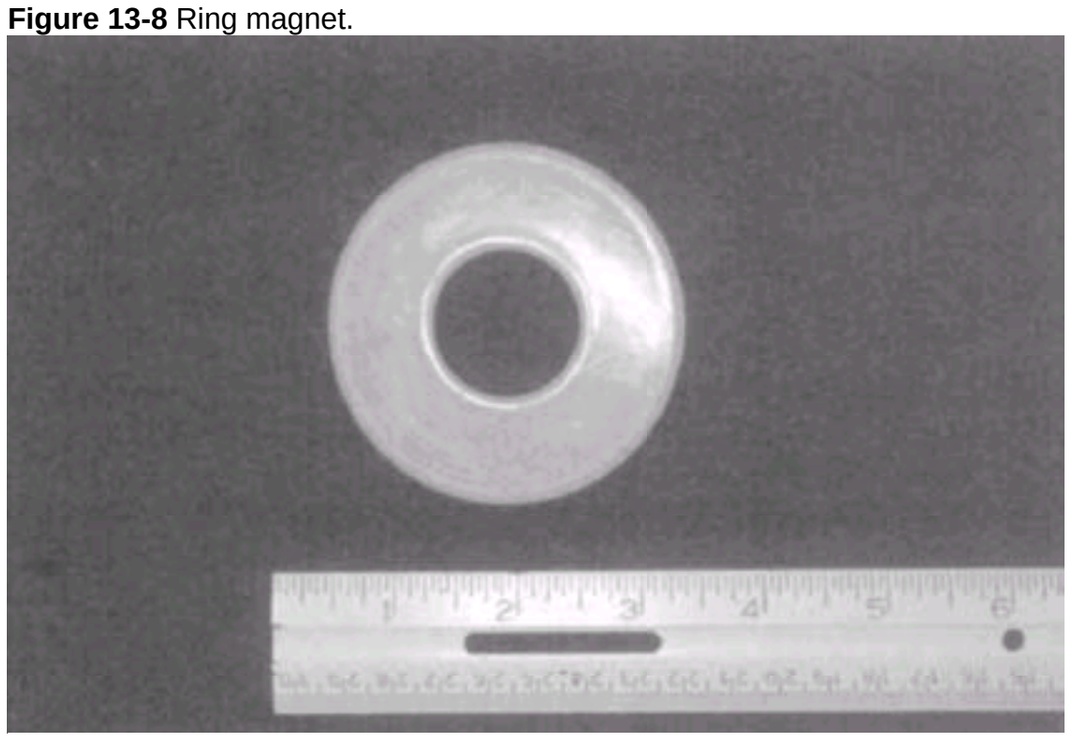


Figure 13-9 Typical location of pacemaker pocket in upper chest wall.

Figure 13-10 Application of ring magnet over pacemaker generator.

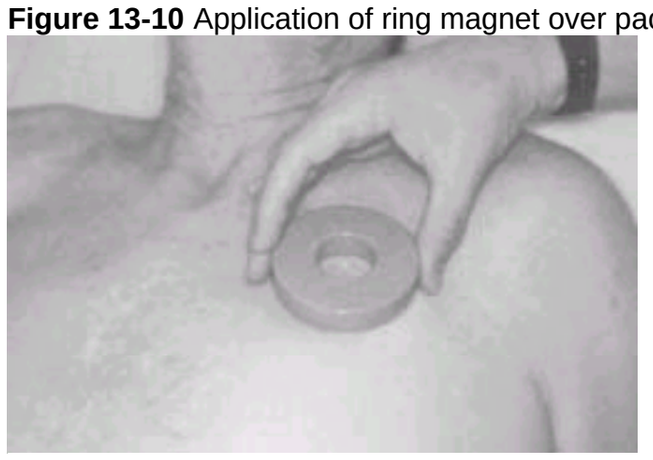


Figure 13-11 Electrocardiogram of patient with a nonfiring pacemaker. Intrinsic cardiac rate is 80 beats/min, and no pacemaker activity is seen.

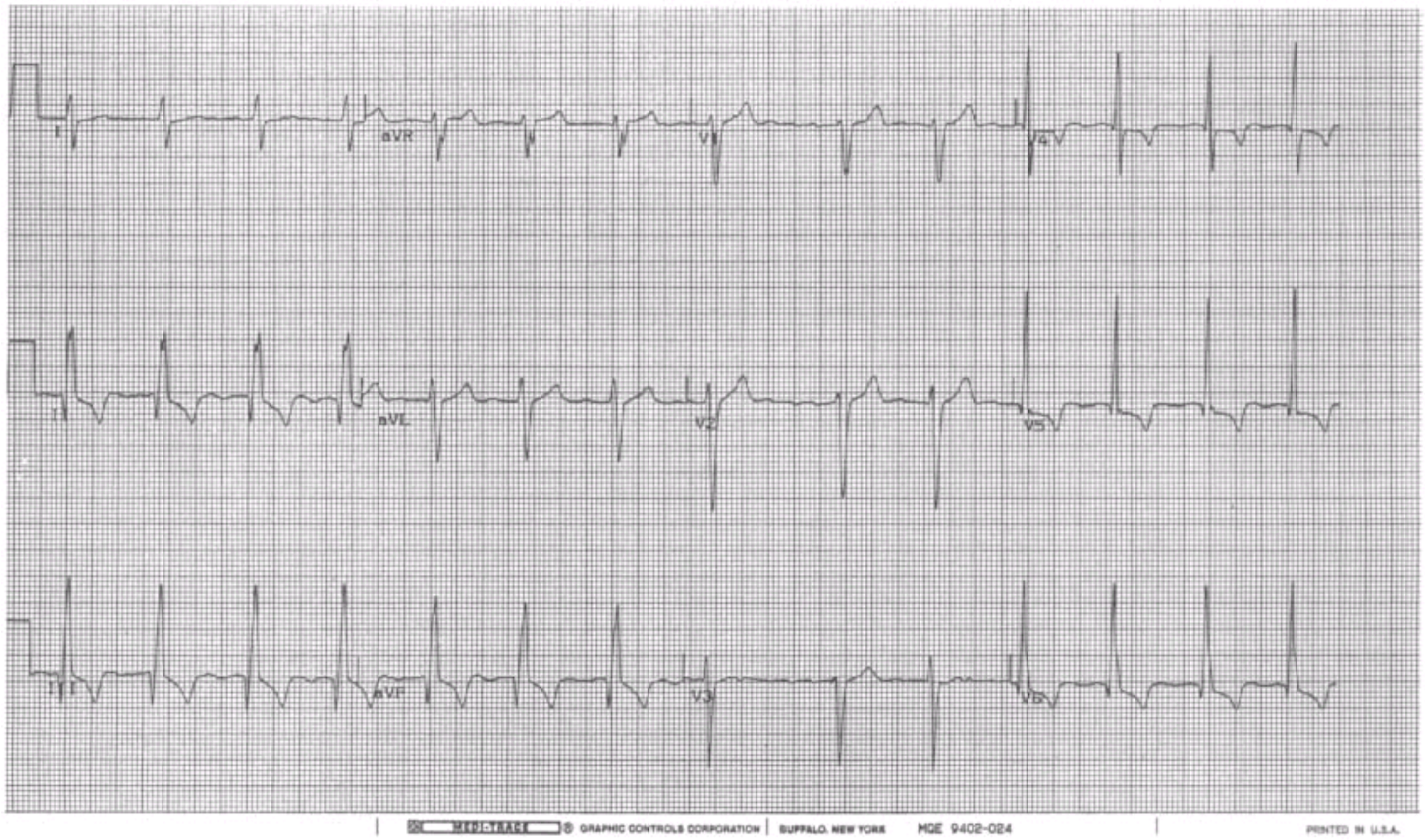


Figure 13-12 Electrocardiogram of same patient with magnet applied over pacemaker, producing a paced rhythm. Pacer spikes are evident (*arrows*) and the magnet rate is 85 beats/min. Note the left bundle-branch bundle typical of a pacer lead in the right ventricle.

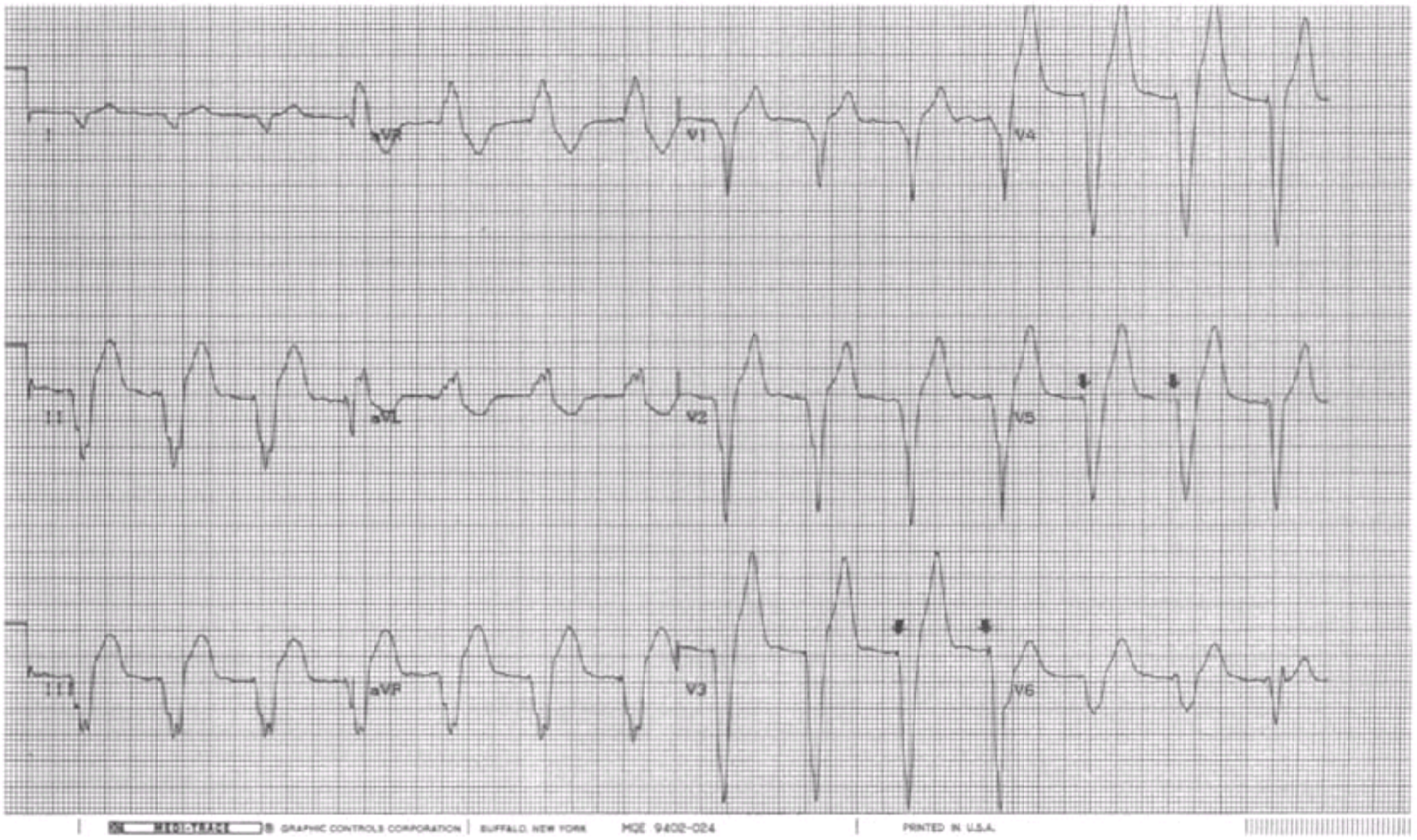


Figure 13-13 Placement of magnet on AICD.

Figure 14-1 Bipolar limb leads. Leads I, II, and III are shown as a triangle, known as Einthoven's triangle. Left arm (LA), right arm (RA), and left leg (LL) placement is shown. These bipolar leads are oriented such that the positive poles lie inferiorly and to the left (given that the bottom apex of the triangle is directed toward the left leg)—as does the major electrical vector of the heart.

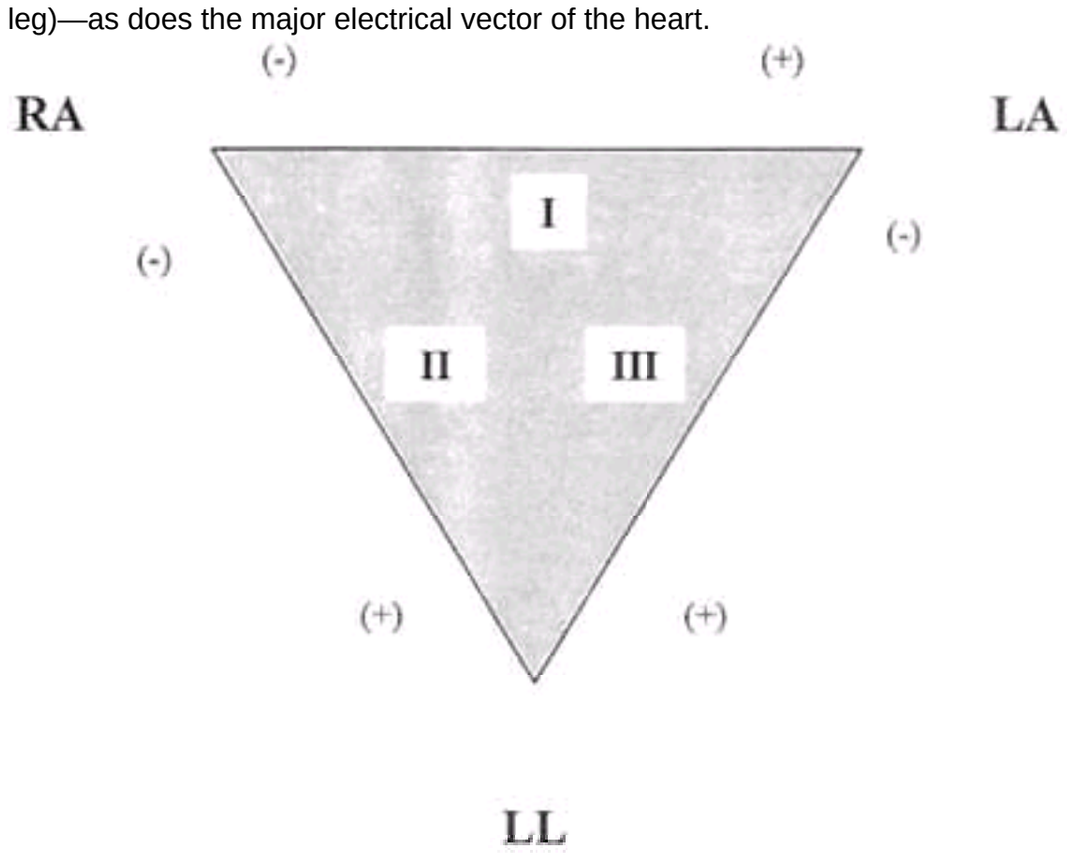


Figure 14-2 Hexaxial system of limb and augmented leads in the frontal plane. Each lead is separated by 30° in this frontal plane representation of the limb and augmented leads. Augmented leads are shown in boldface. Arrows denote positive polarity. Note that the inferior leads (II, III, aVF) logically lie at the bottom of this figure, and the lateral leads (I, aVL) lie on the left side of the figure, where the lateral aspect of the heart is located were this to be superimposed on a patient.

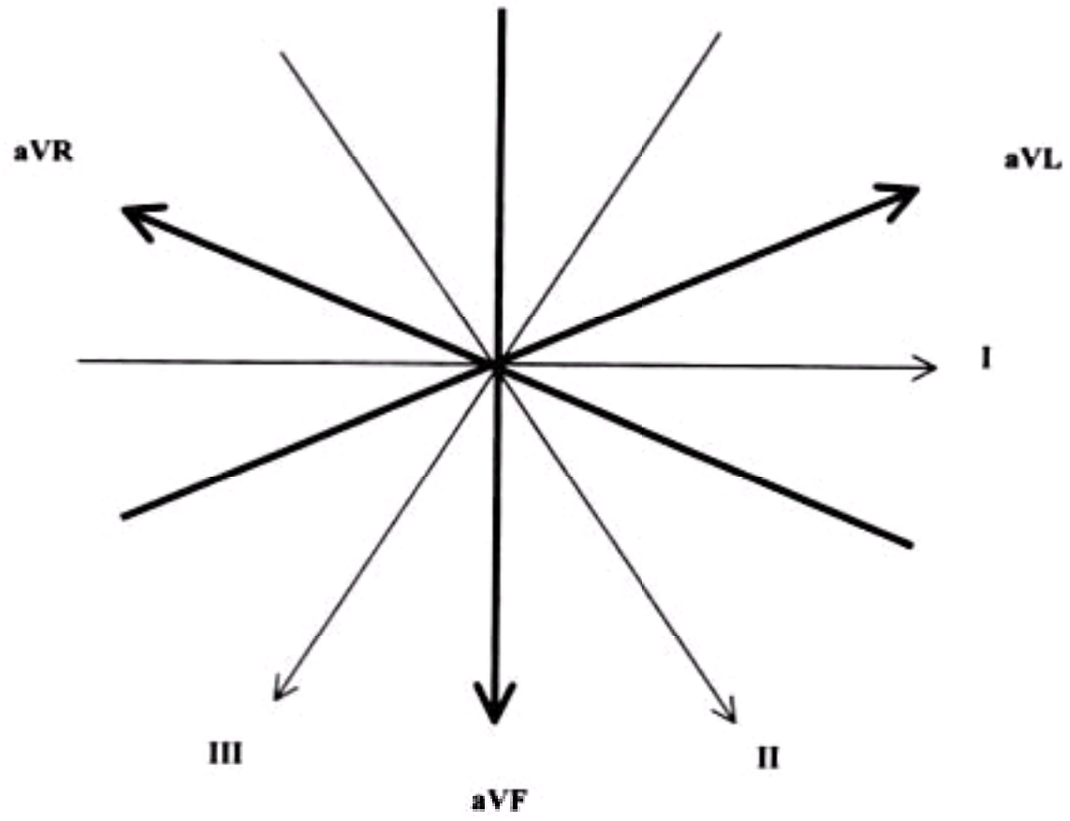


Figure 14-3 Precordial lead placement for the standard 12-lead ECG. If multiple or repeat ECG tracings are anticipated, the original lead placements should be marked on the patient's chest wall or stick-on leads should be left in place after the ECG wires are removed.

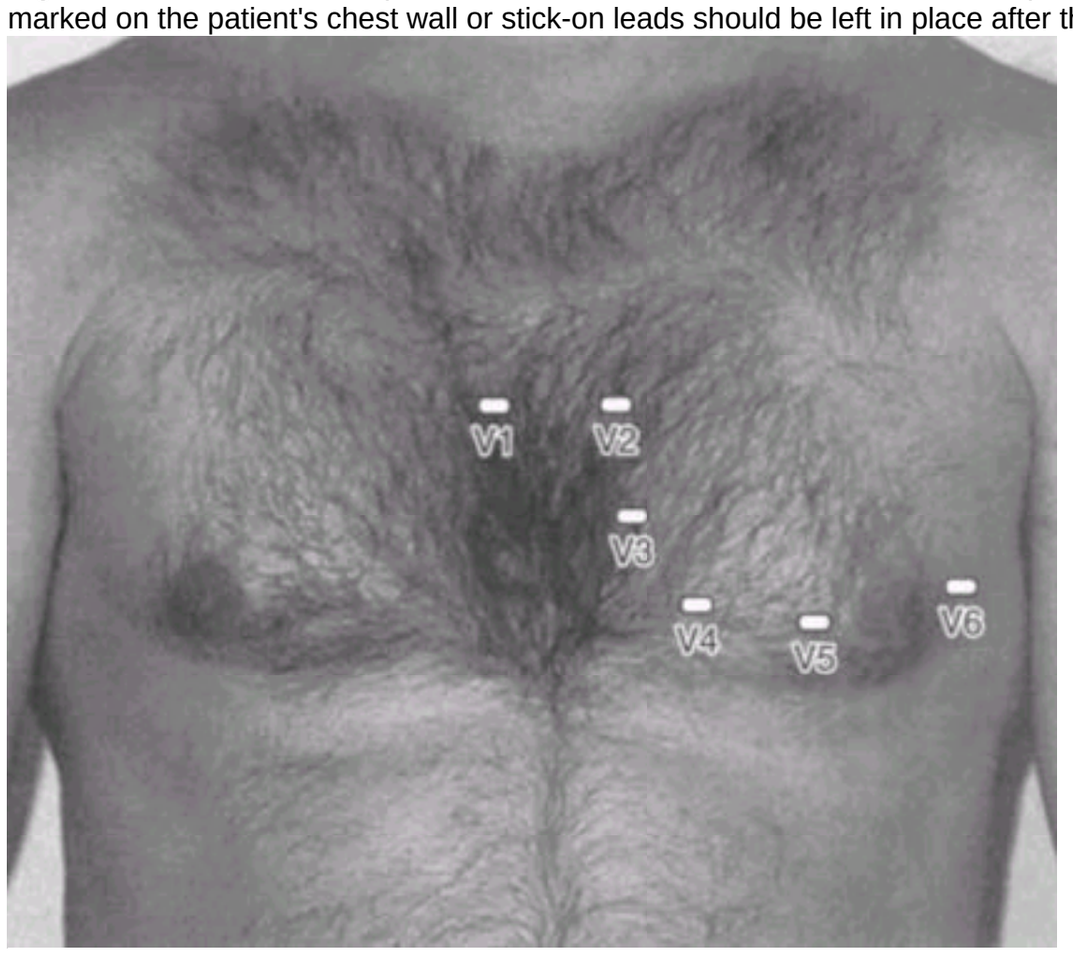


Figure 14-4 *A,* Normal 10 mm/mV calibration. Note the box-shaped mark to the left of the complexes; this is a graphic representation of the calibration for the tracing. This parameter should be routinely noted before ECG interpretation. Note the change in *B*. *B,* Abnormal 20 mm/mV calibration. The calibration in this tracing was (inexplicably and unexpectedly) changed to 20 mm/mV by the ECG, not by the operator. When compared with a baseline ECG, it appeared that the patient had developed voltage criteria for left ventricular hypertrophy as well as ST segment elevation. *A,* which was recorded minutes later with correction of calibration to the standard 10 mm/mV, was unchanged from baseline tracings.

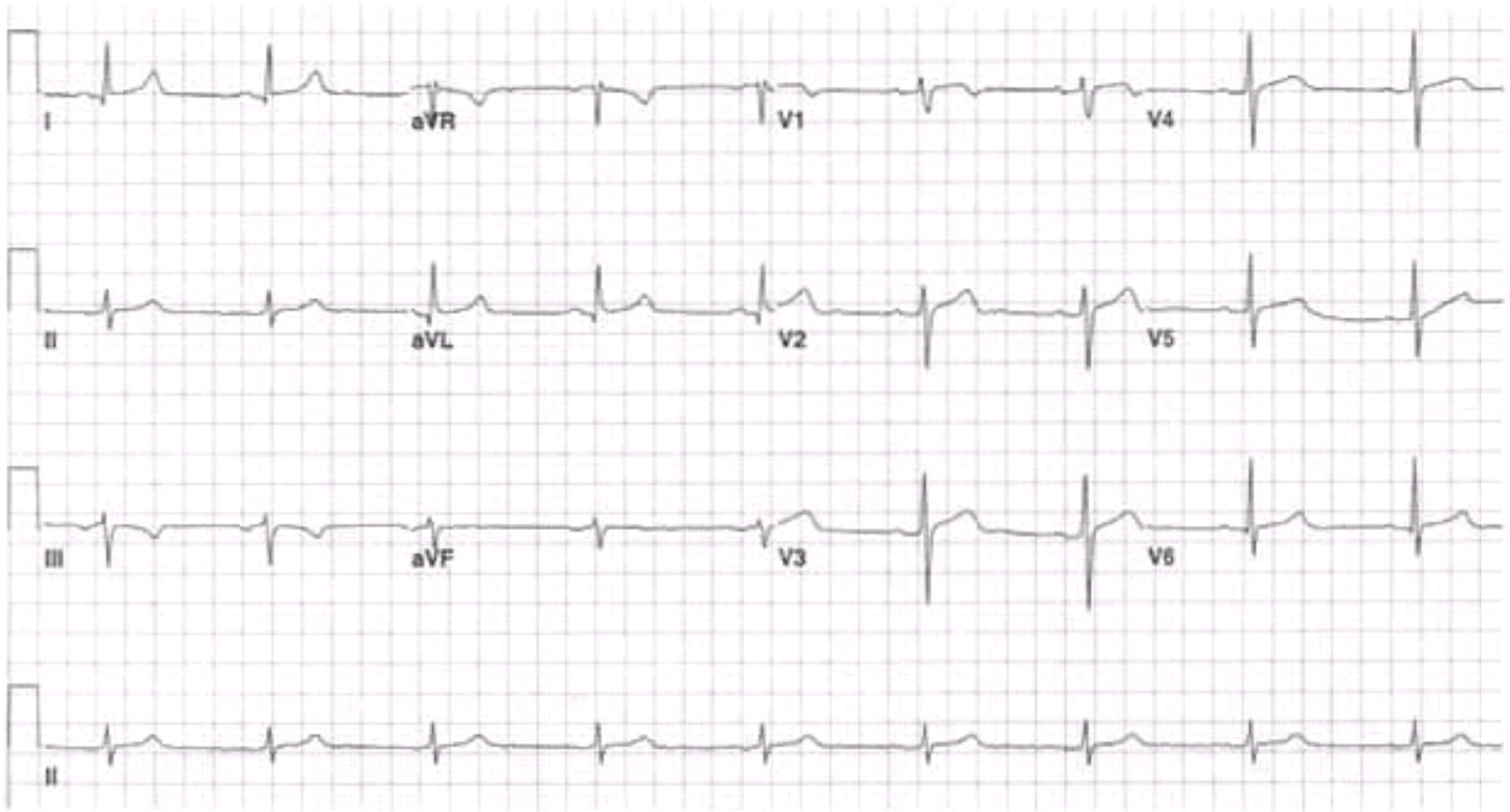


Figure 14-5 Posterior lead placement. Leads V7, V8, and V9 are placed on the same horizontal plane as V6, with V7 at the posterior axillary line, V8 at the tip of the left scapula, and V9 near the border of the left paraspinal muscles.

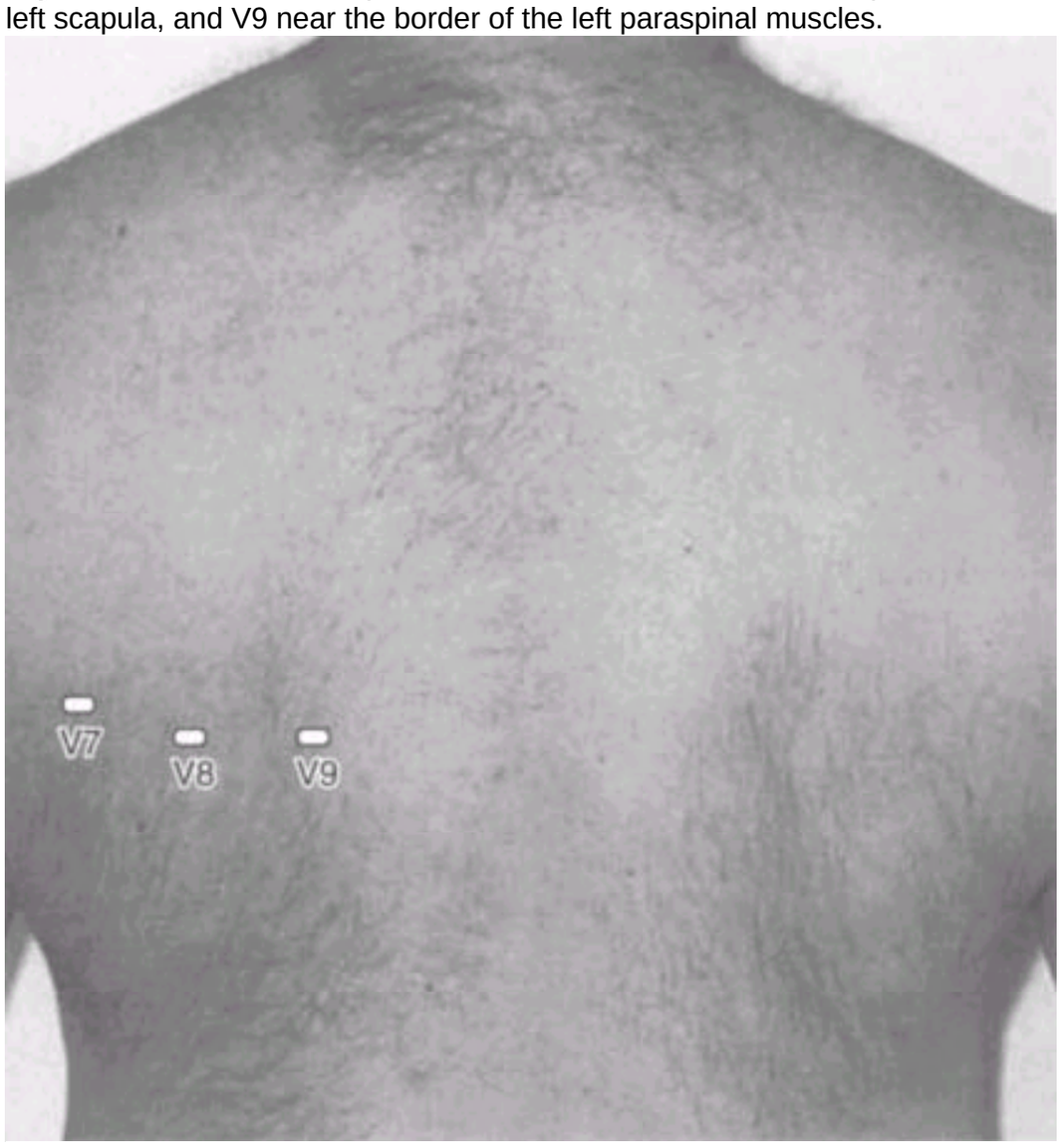


Figure 14-6 Right-sided lead placement. Right-sided leads RV1–RV6 are placed on the chest as a mirror image of the standard precordial leads.

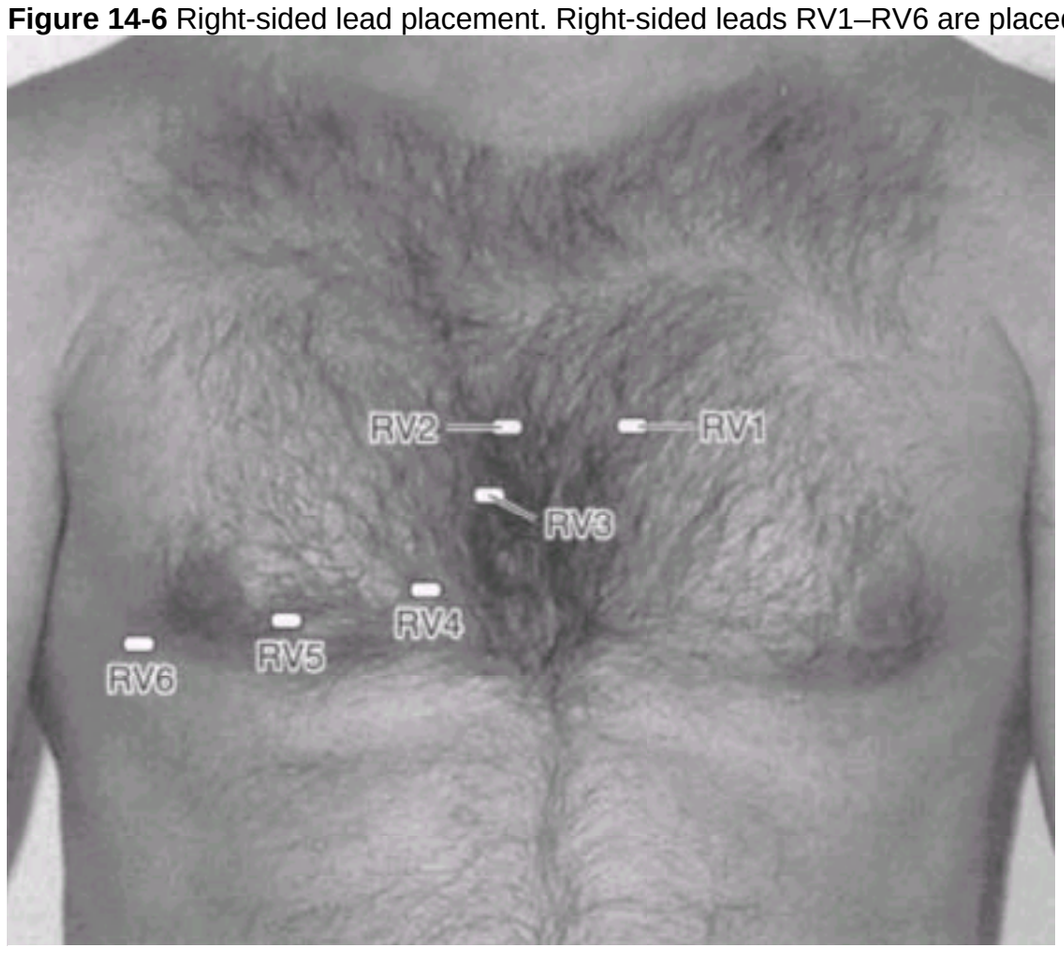


Figure 14-7a *A,* Baseline ECG of patient before development of abnormal rhythm (10 mm/mV). Note the P wave morphologies, especially in leads I, II, and V1. *B,* ECG during ectopic atrial tachycardia (10 mm/mV). Note the change in P wave morphology, especially in lead V1.

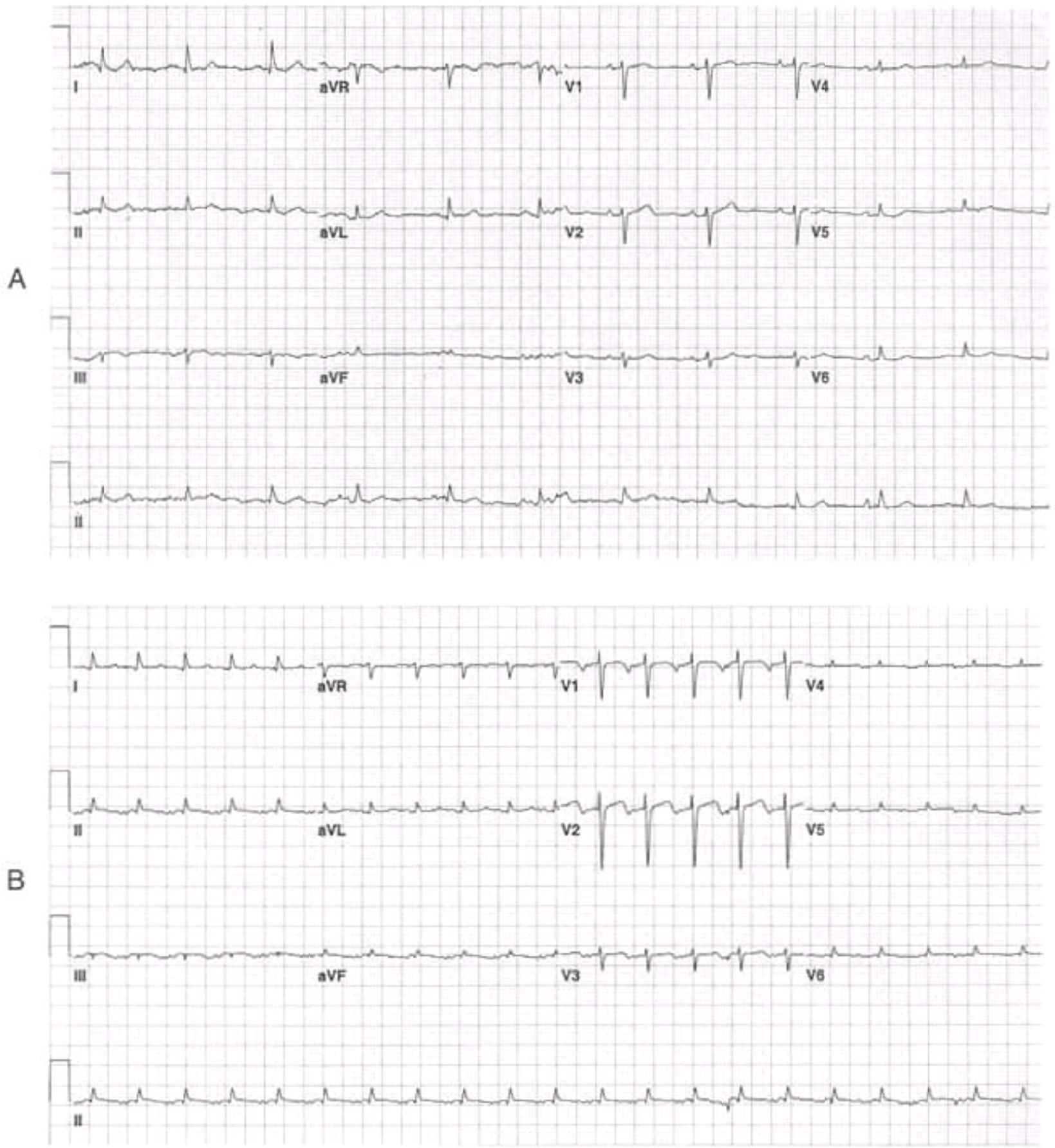
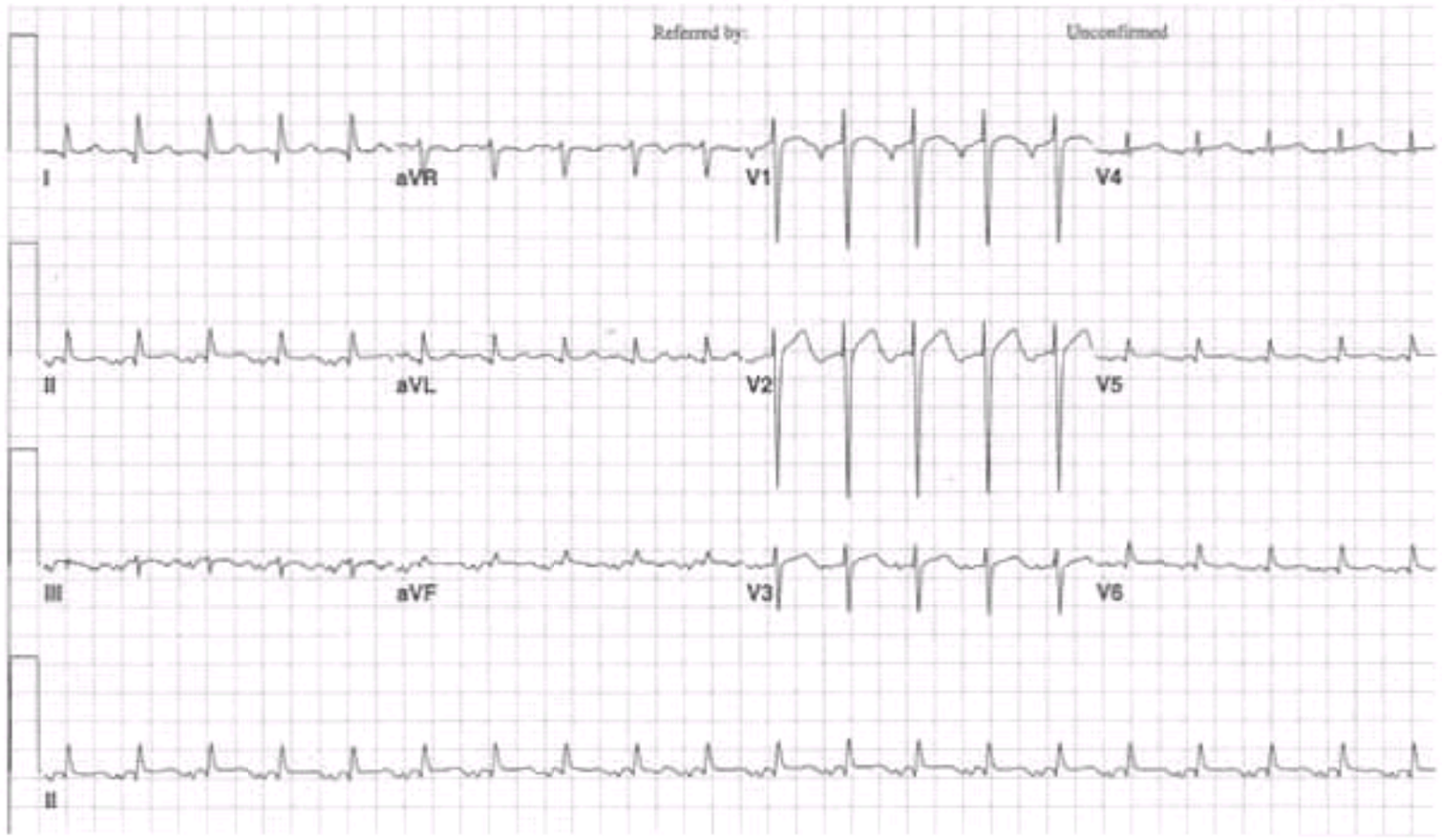


Figure 14-7b *C,* ECG during ectopic atrial tachycardia (20 mm/mV). The P waves are now easier to see in all leads. *D,* ECG after reversion to normal atrial focus (20 mm/mV). Contrast these accentuated P waves to those in *C*.

C



D

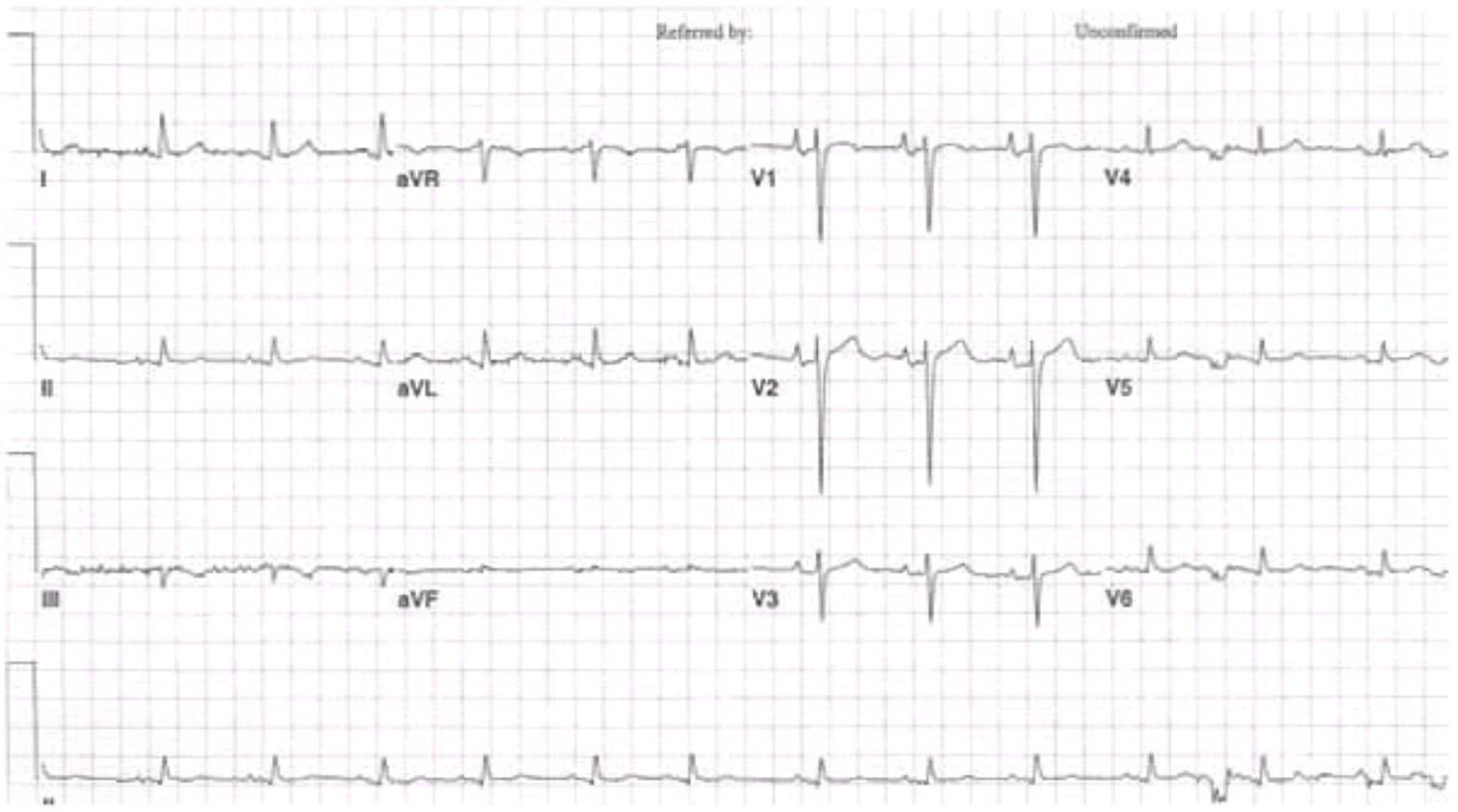
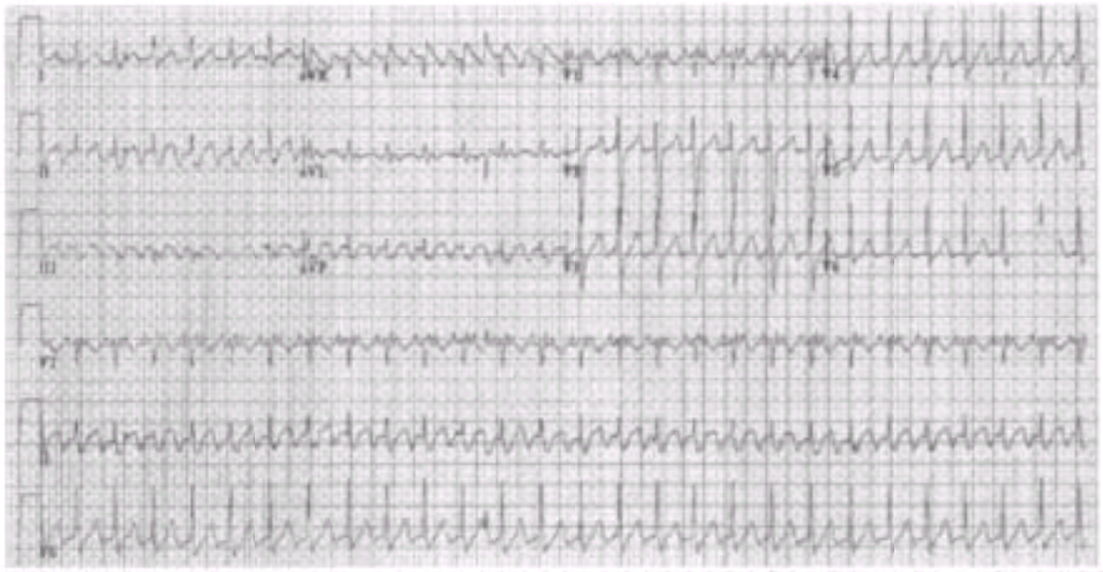


Figure 14-8 *A,* ECG with tachycardia at normal paper speed (25 mm/sec). Because of the rapid rate, the actual P waves are difficult to discern, thus making rhythm determination difficult. The computerized interpretation is sinus tachycardia with first-degree AV block. *B,* ECG with tachycardia at double paper speed (50 mm/sec). With increased paper speed, atrial P wave activity is accentuated, demonstrated atrial flutter with a 2:1 AV block.

A



B

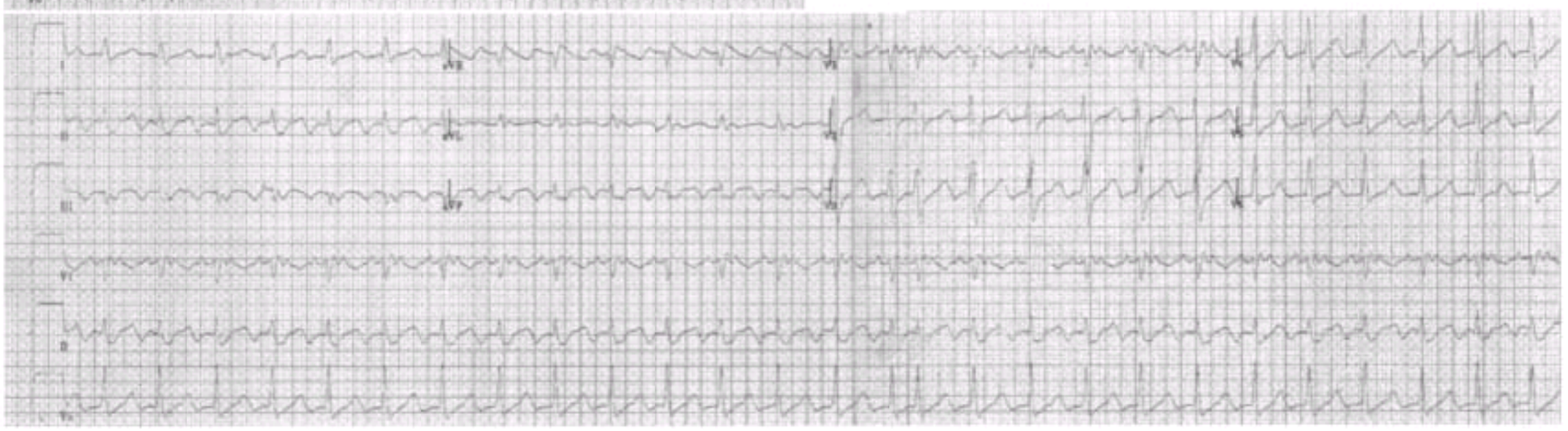


Figure 14-9 Alternative leads. The figure displays three of the more commonly used alternative lead strategies for atrial rhythm clarification (Lewis leads, vertical sternal or Barker leads, and MCL1), and ST/T wave monitoring (MCL6).

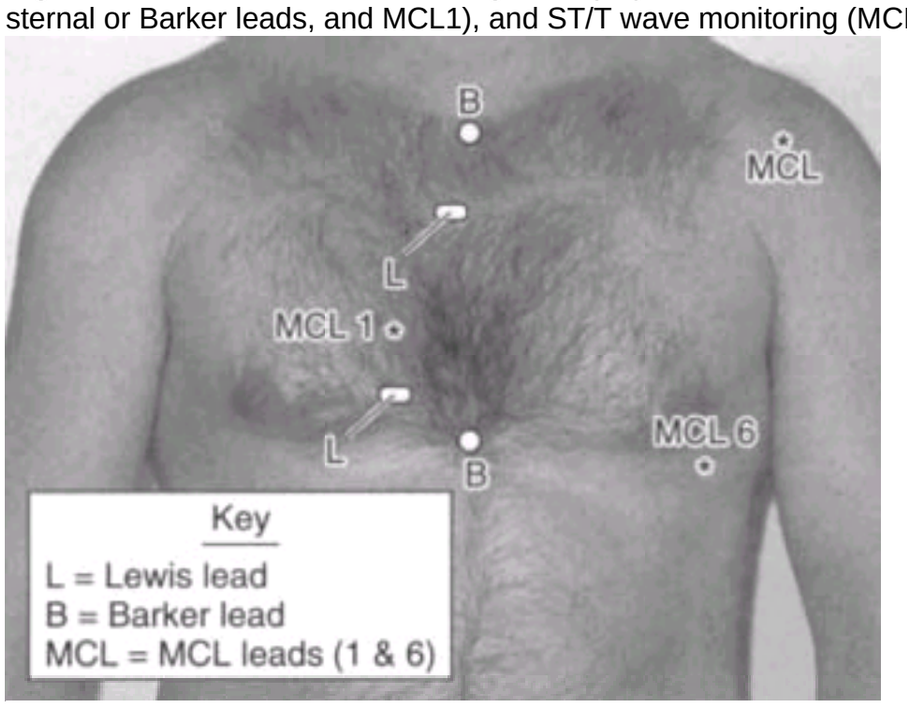


Figure 14-10 Arm lead reversal (LA ⟷ RA). The most common of limb lead reversals, the clues lie in leads I and aVR. Lead I features a negative P wave, as well as a principally negative QRS complex and T wave. This could suggest dextrocardia, but the precordial leads demonstrate normal transition, which is not consistent with dextrocardia. Note also the unusual appearance of aVR in this tracing.

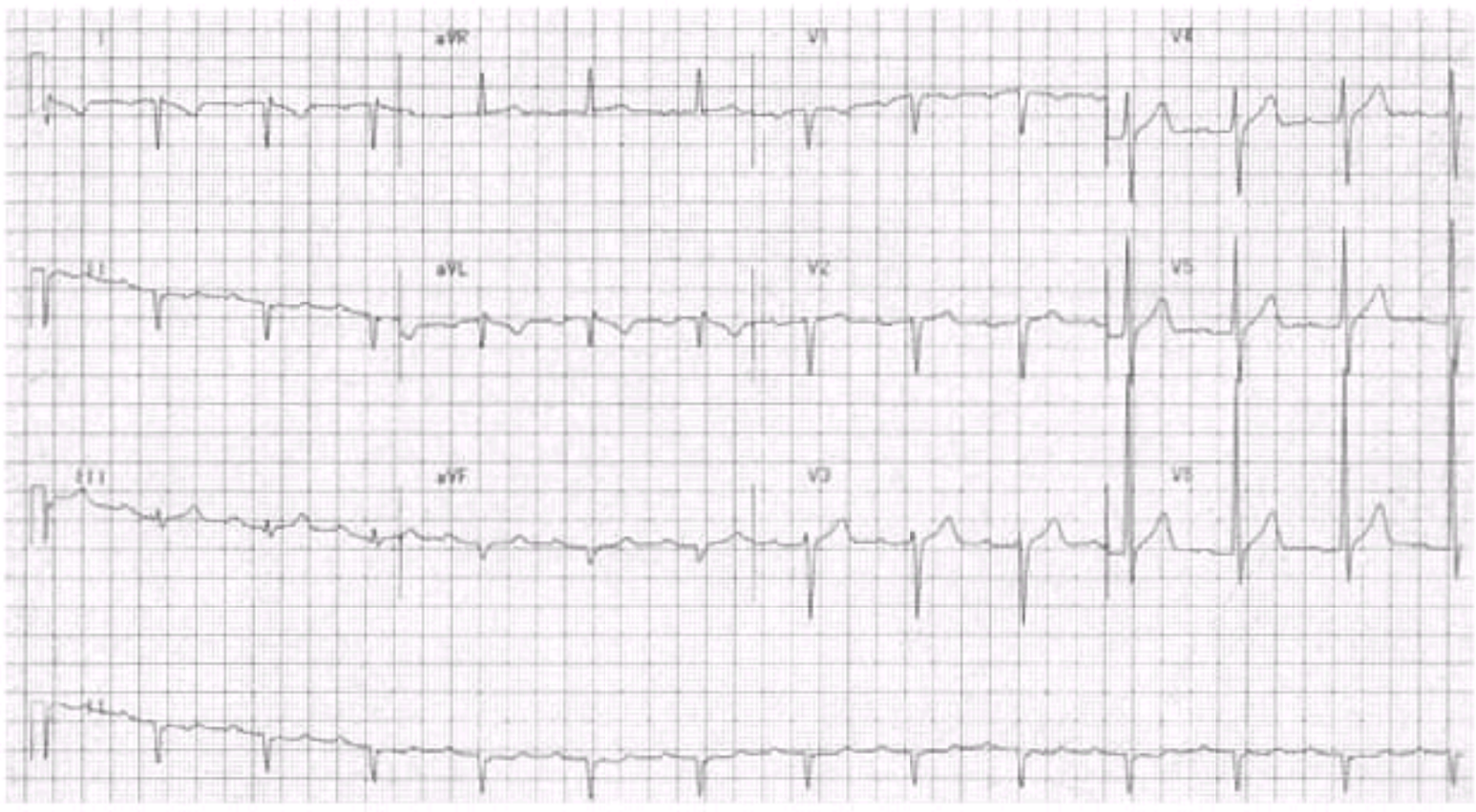
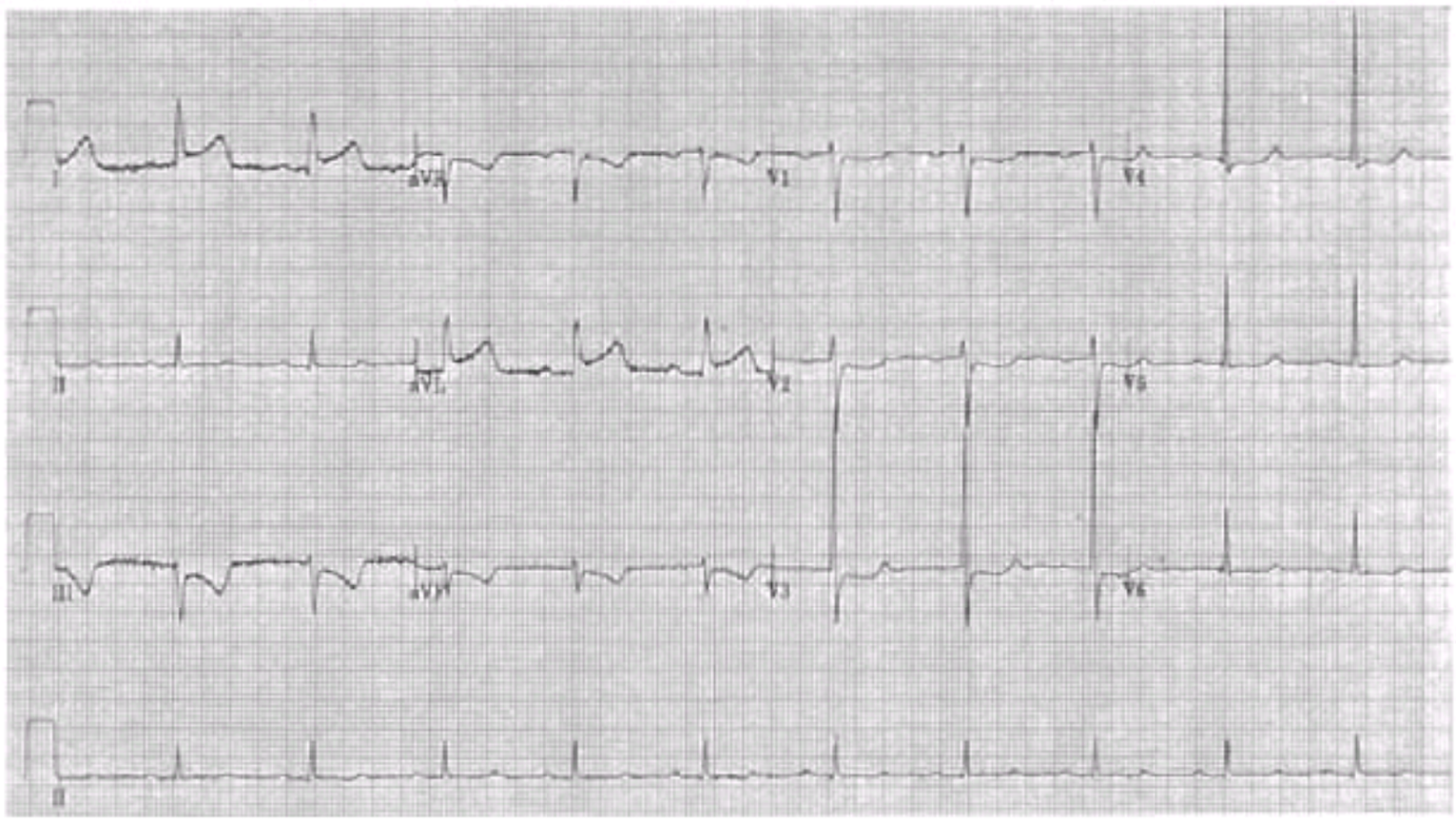


Figure 14-11 *A,* Limb lead reversal (LA ⟷ LL). A patient with a history consistent with ACS was brought to the ED after this ECG was recorded in a clinic. Leads I and aVL suggest an acute high lateral infarct but, surprisingly, there are no corresponding changes in leads V5 and V6. The deep T wave inversions in III and aVF were at first thought to be inferior ischemia or reciprocal changes (see also *B*). *B,* Correction of lead reversal (LA ⟷ LL). After the leads were reconnected, this tracing reveals an acute inferior wall MI, as well as deep T wave inversion in aVL—a harbinger of acute inferior MI. Comparing this tracing with that in *A,* note the following: lead I ⟷ lead II; lead aVL ⟷ aVF, and lead III is inverted. Thus, inferior changes become lateral, and lateral become inferior.

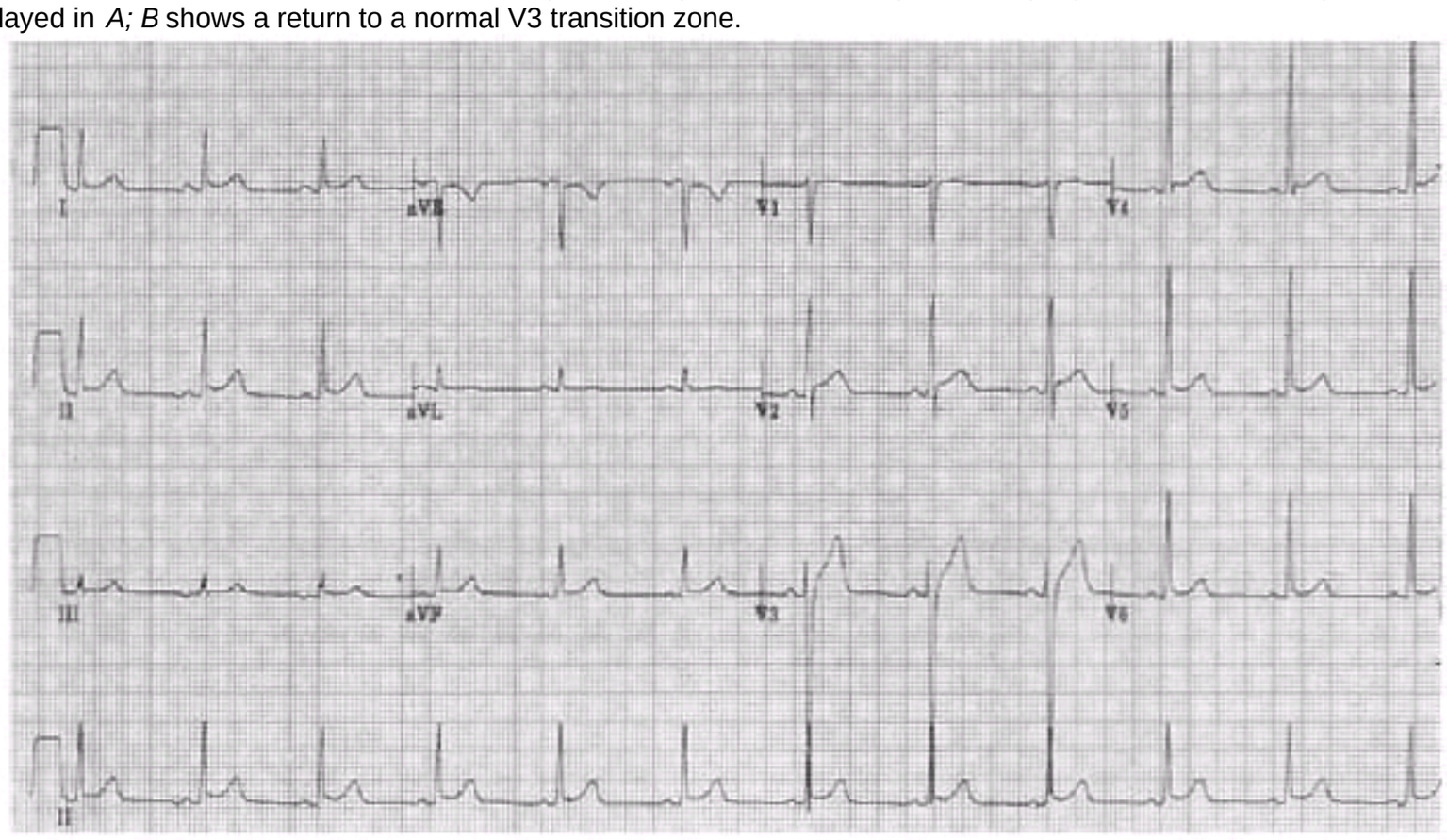
A



B

Figure 14-12 *A* and *B*, Precordial lead reversal (V2 ⟷ V3). Note the usual precordial progression of R wave growth in leads V2 and V3 is disrupted in the tracing displayed in *A; B* shows a return to a normal V3 transition zone.

A



B

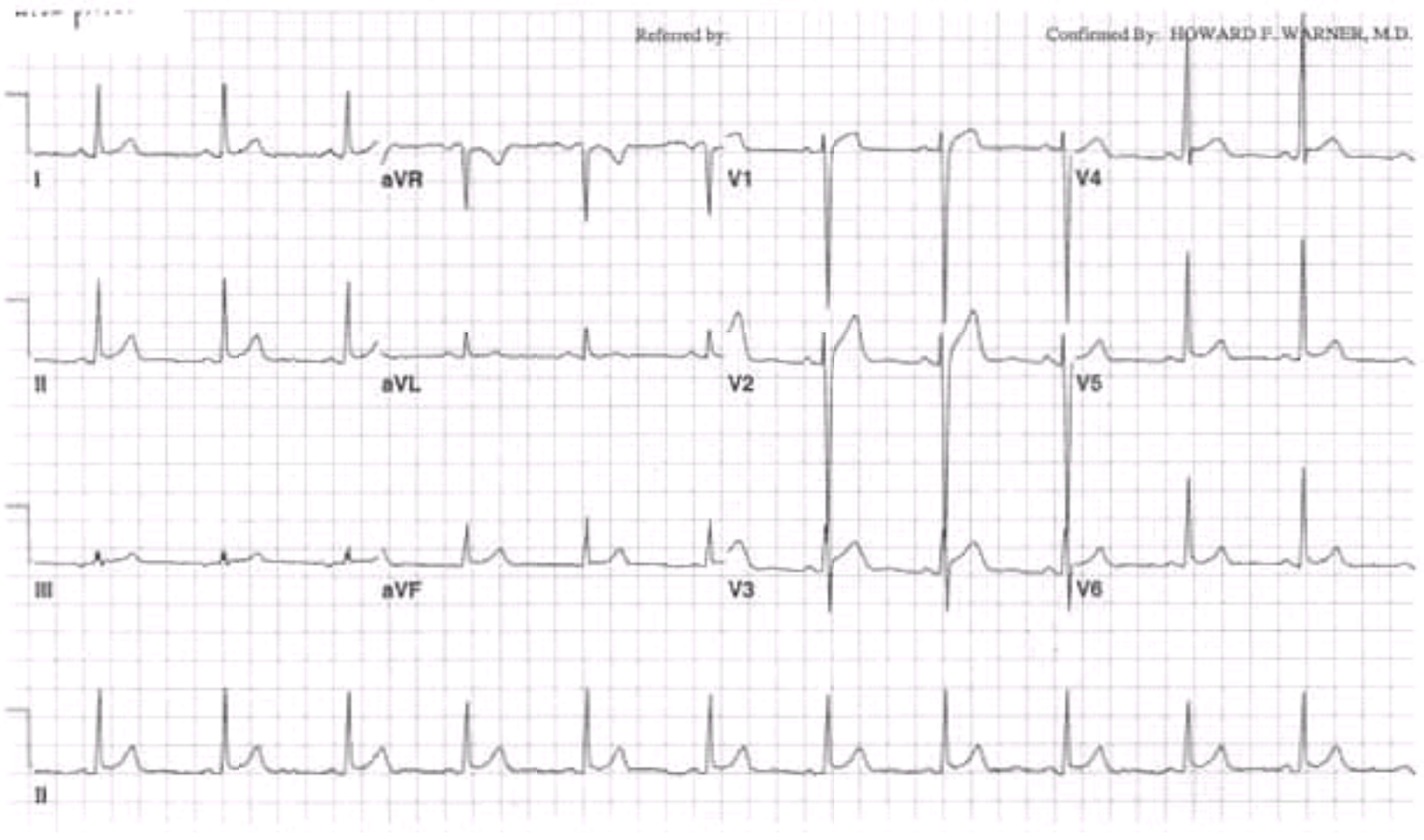


Figure 14-13 Artifact due to physiologic cause. The patient's monitor was alarming due to a perceived heart rate of >200 beats/min, and the computerized alert system called this ventricular tachycardia. The patient, who has Parkinson's disease, was without complaint. The ECG demonstrates a marked artifact, giving the appearance of atrial flutter in lead V1.

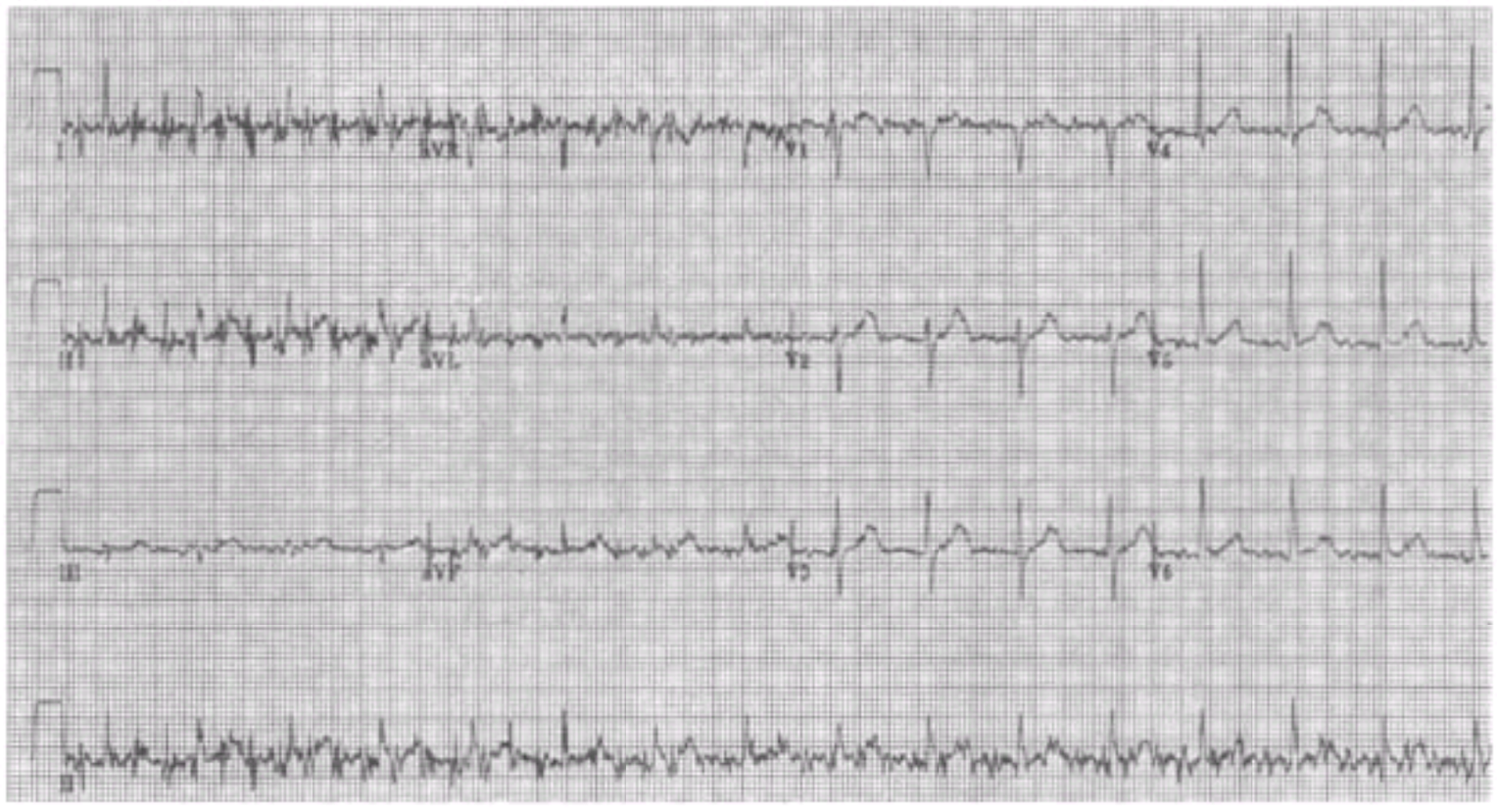


Figure 15-1 Pacemaker energy source controls and connections.

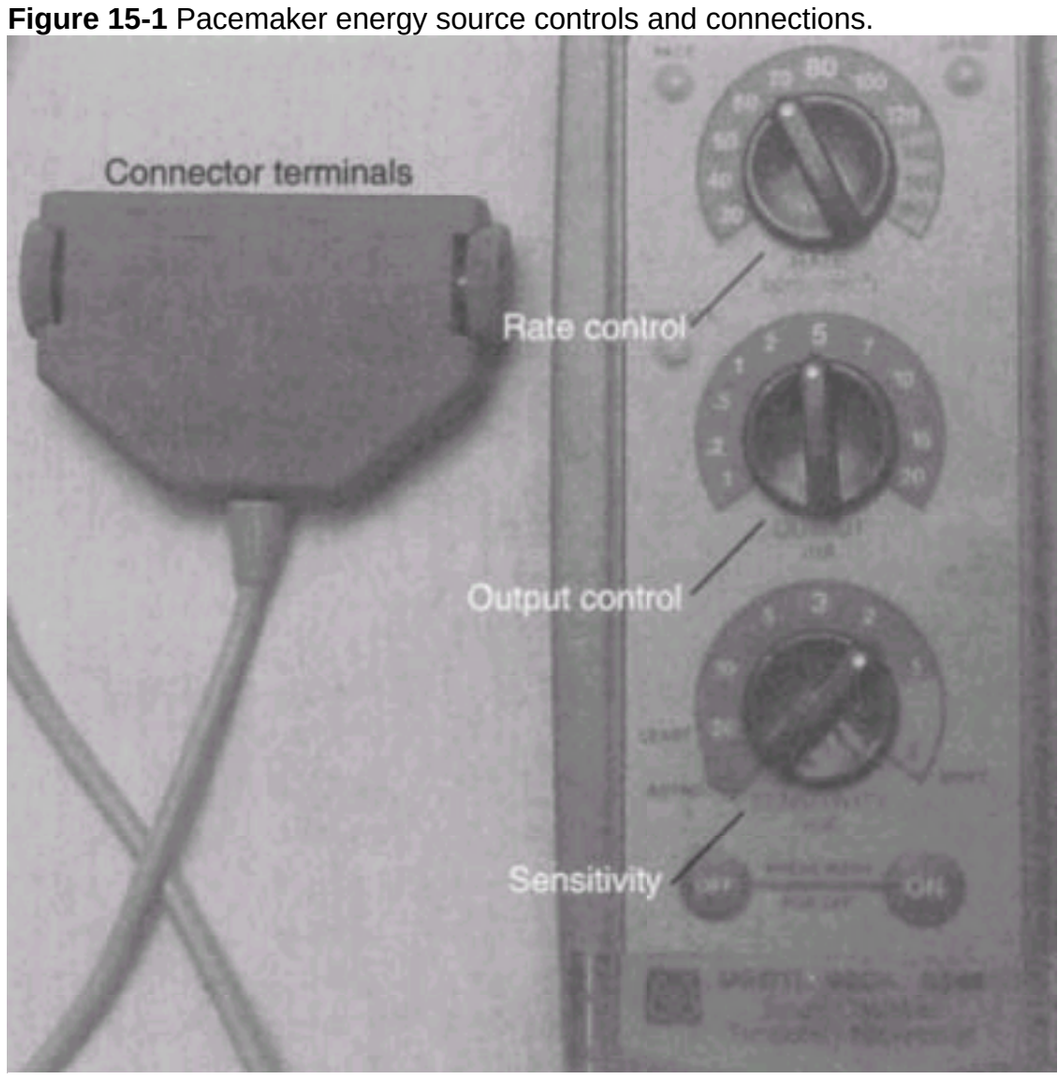


Figure 15-2 Balloon-tipped pacing catheter.

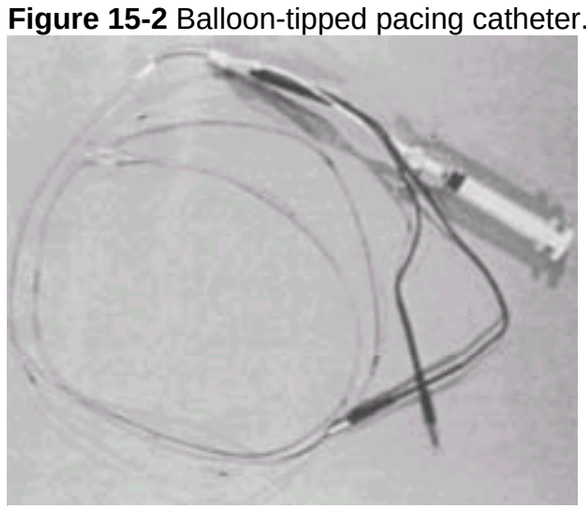


Figure 15-3 Position of an electrocardiogram device during femoral vein insertion of a pacemaker catheter. Operator is on right side of patient facing cephalad.

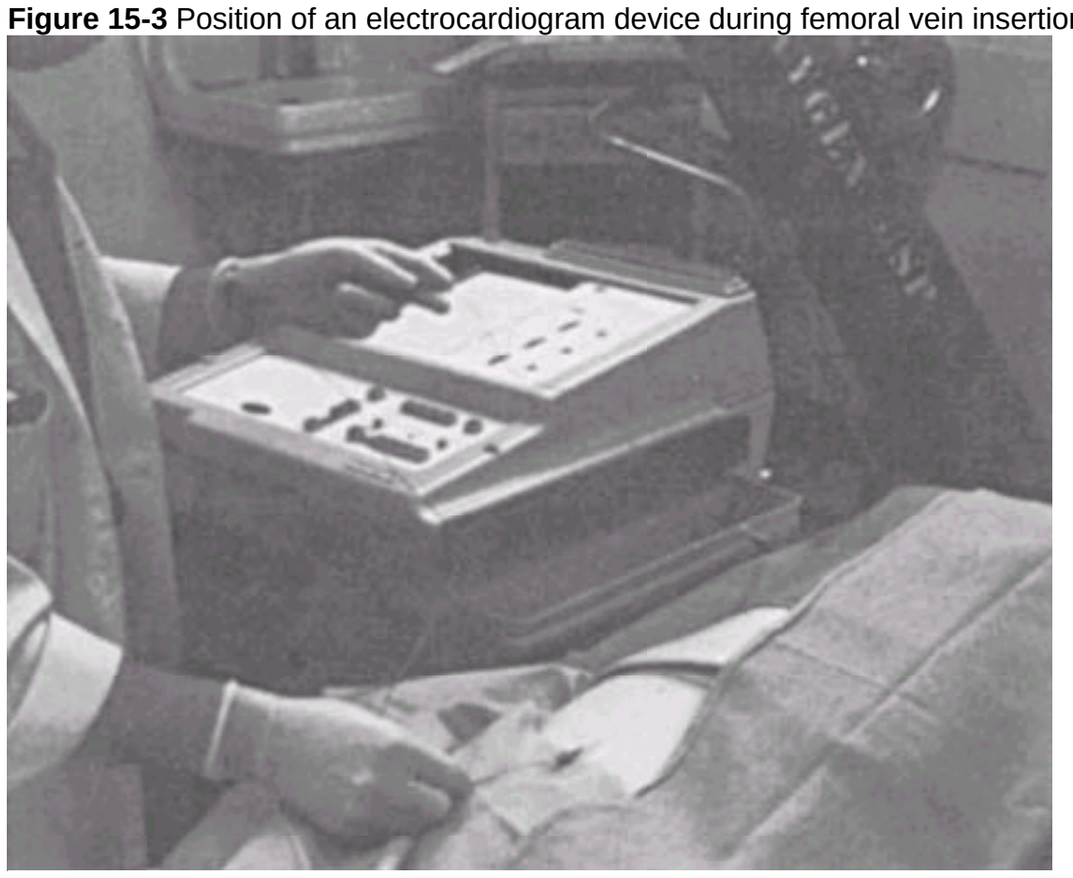


Figure 15-4 Insertion of the pacing catheter through the introducer sheath.

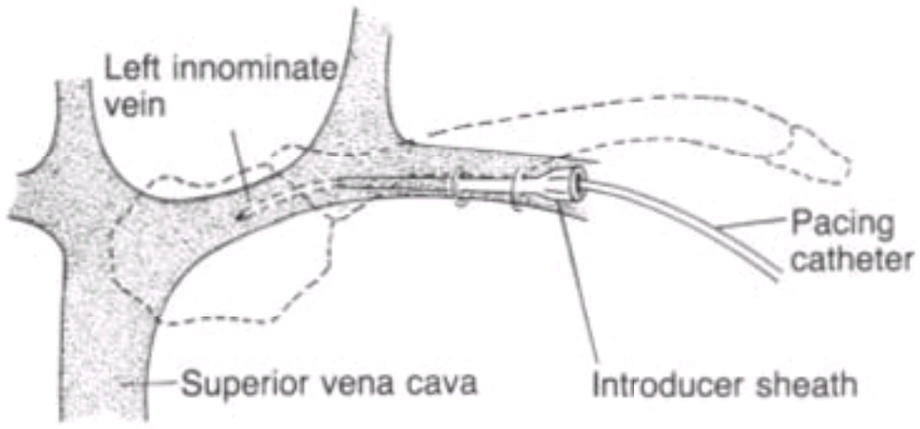


Figure 15-5 Using alligator clips to connect the *negative* lead of the pacemaker catheter to the V lead of an electrocardiographic machine.

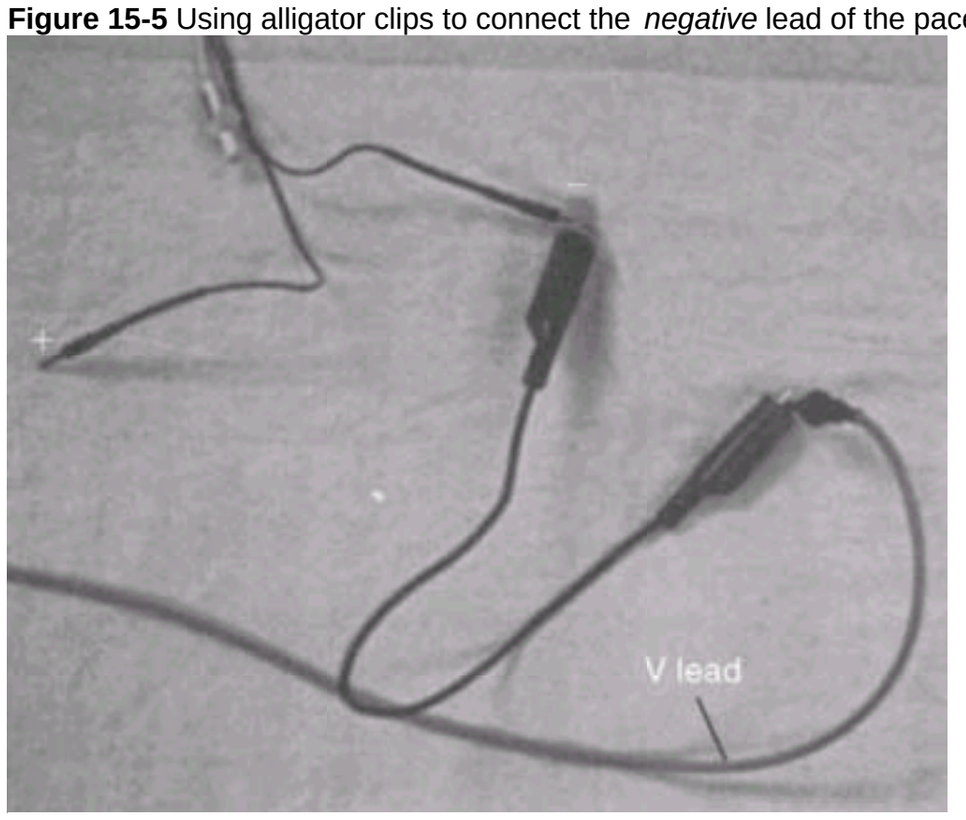


Figure 15-6 *A–K,* Intracardiac electrocardiography: Electrical signals of atrial and ventricular depolarization and repolarization from different vascular and intracardiac locations (see text). *(A–F and H–K from Bing OH, McDowell JW, Hantman J, et al: Pacemaker placement by electrocardiographic monitoring. N Engl J Med 287:651, 1972. G from Goldberger E: Treatment of Cardiac Emergencies, 3rd ed. St. Louis, CV Mosby, 1982, p 252.)*

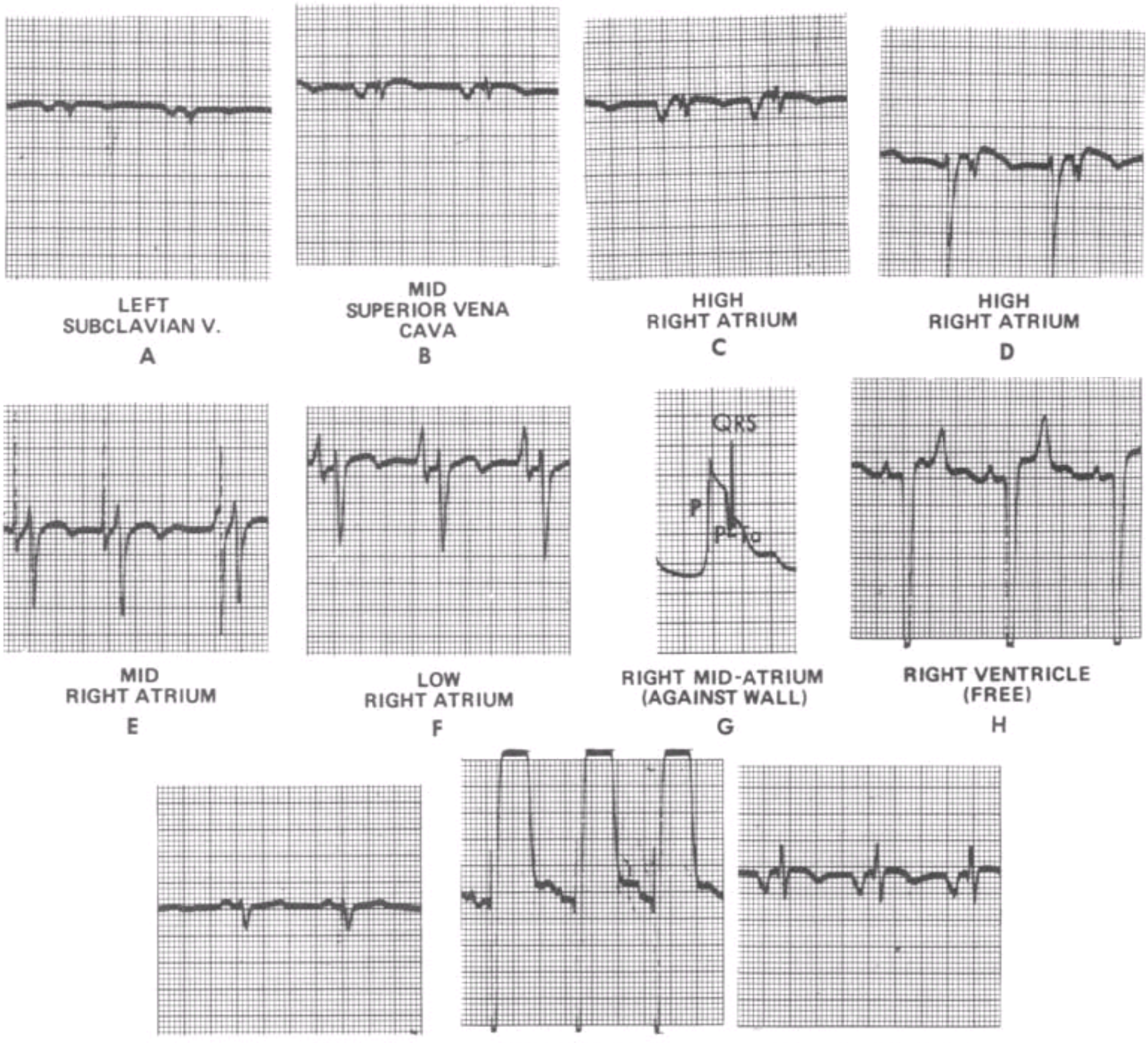


Figure 15-7 Testing unipolar sensing with a bipolar system. *(From Goldberger E: Treatment of Cardiac Emergencies, 3rd ed. St. Louis, CV Mosby, 1982.)*

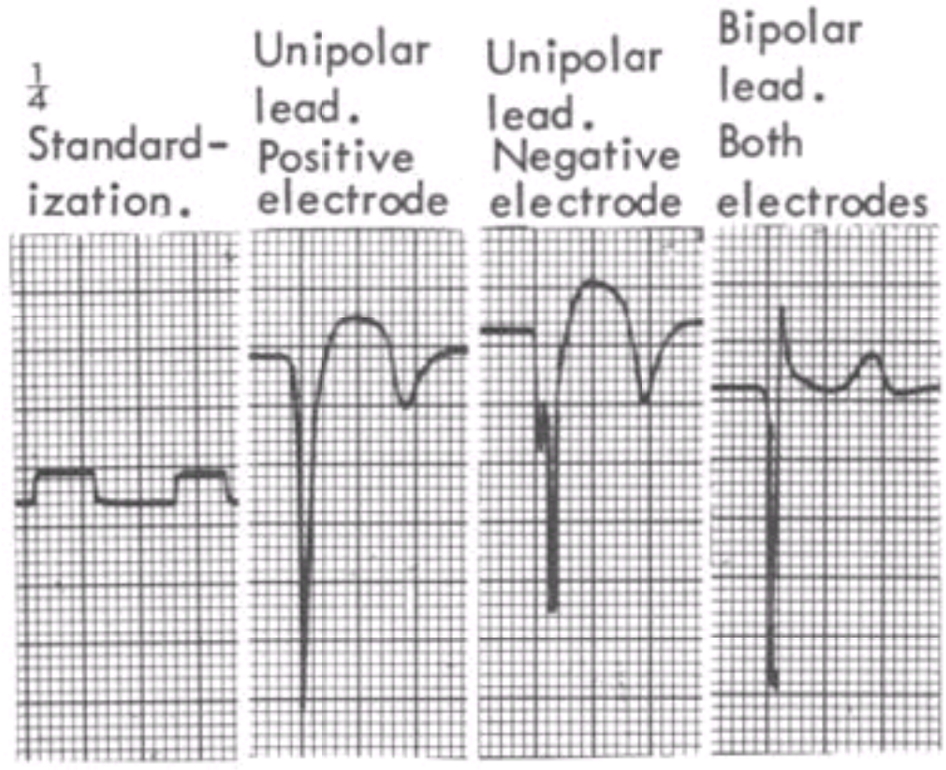


Figure 15-8 Pulling back the introducer sheath (cannula). RA, right atrium; RV, right ventricle; LV, left ventricle.

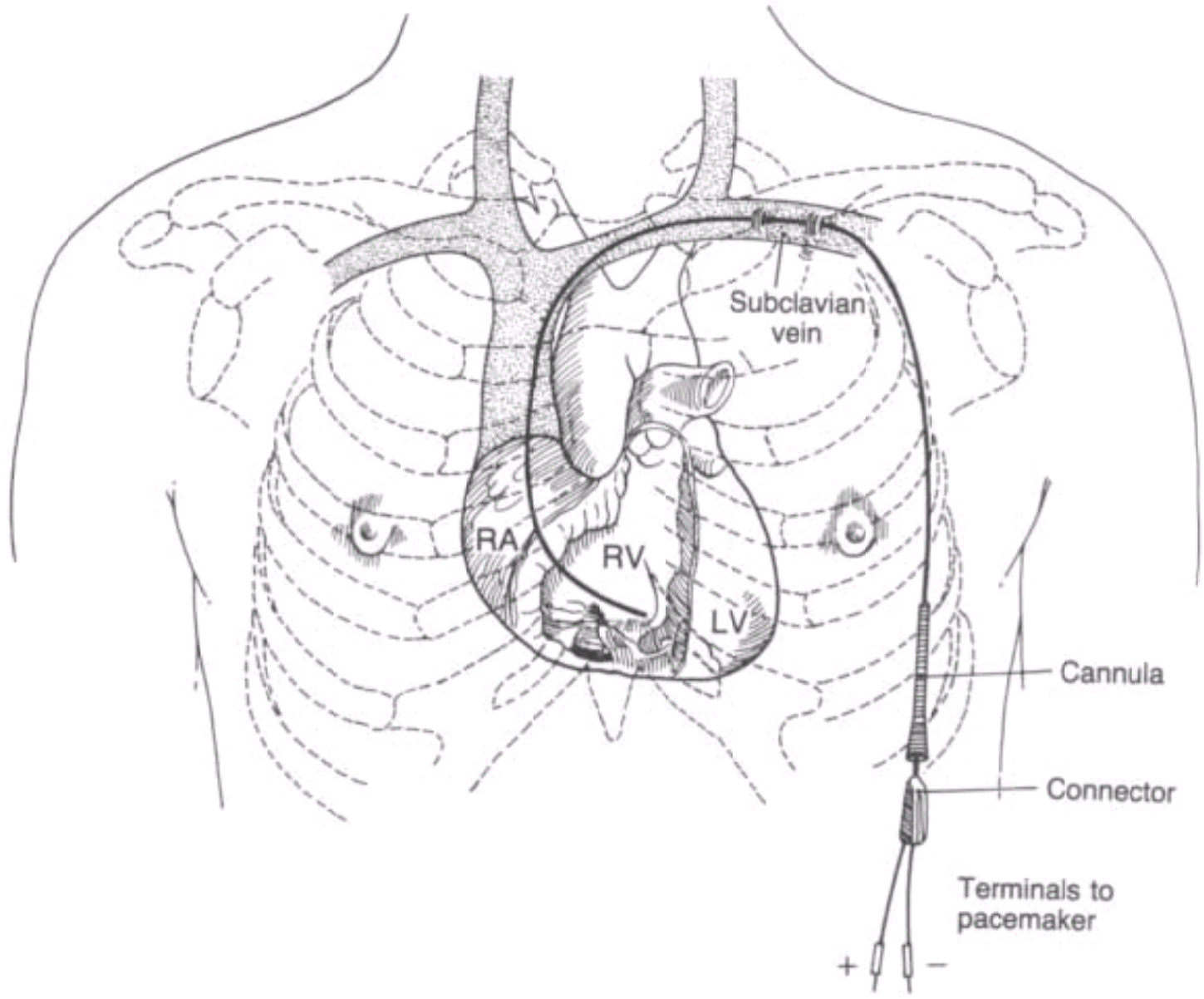


Figure 15-9 Normal pacemaker position on posteroanterior *(A)* and lateral *(B)* chest films. *(From Goldberger E: Treatment of Cardiac Emergencies, 3rd ed. St. Louis, CV Mosby, 1982.)*

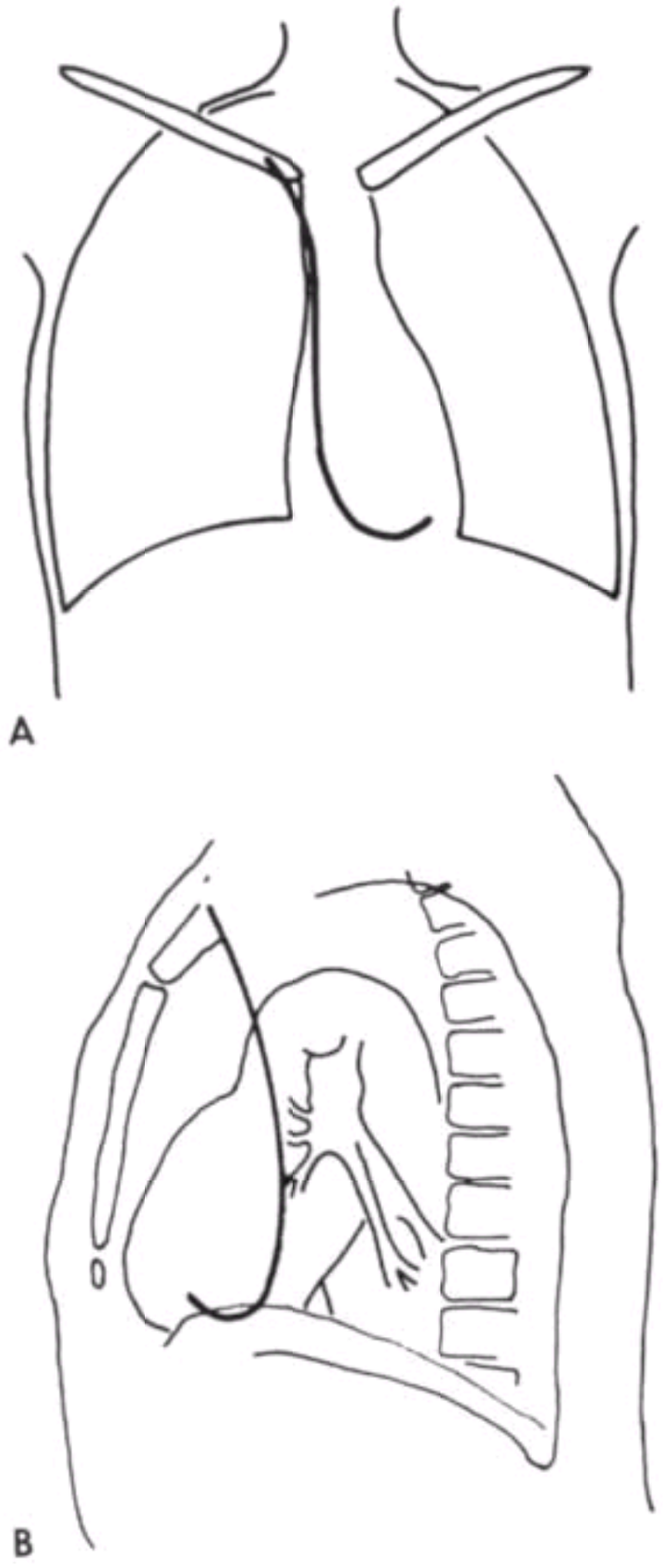


Figure 15-10 Electrocardiogram pattern of right venticular pacemaker.

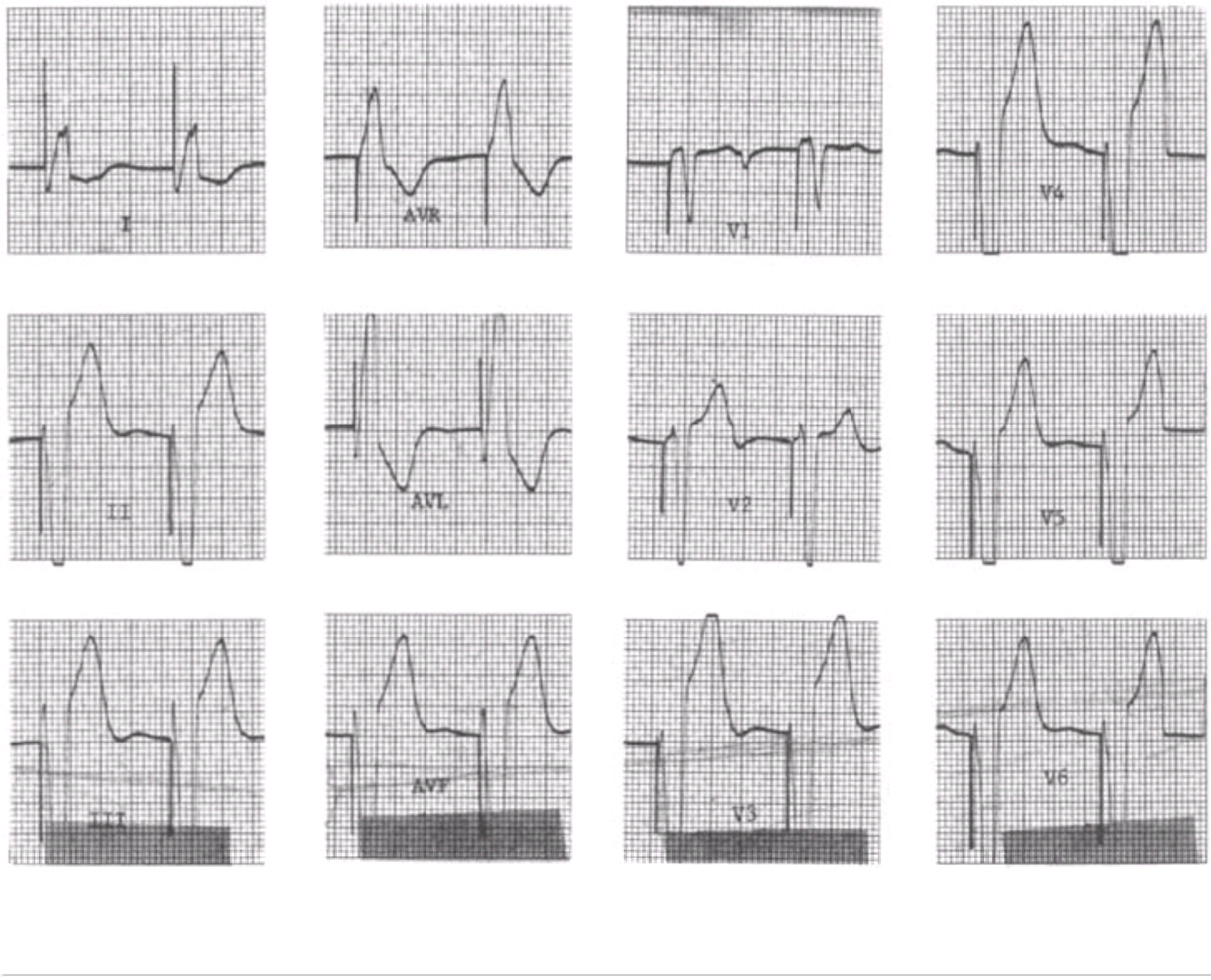


Figure 15-11 Coronary sinus pacing. Note the paced right bundle-branch block pattern.

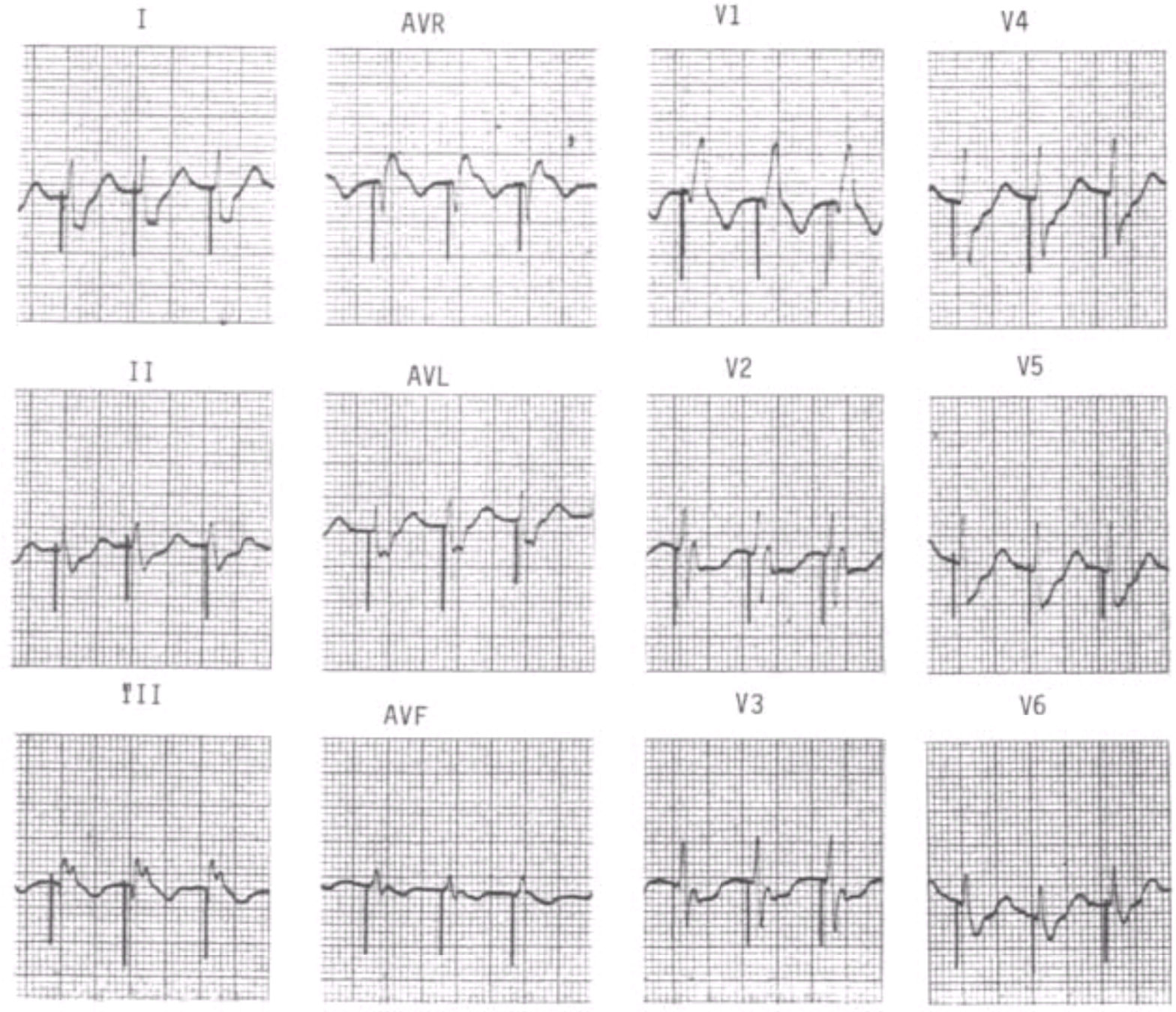


Figure 15-12 Coronary sinus position. *A,* Posteroanterior view. *B,* Lateral view. *(From Goldberger E: Treatment of Cardiac Emergencies, 3rd ed. St. Louis, CV Mosby, 1982.)*

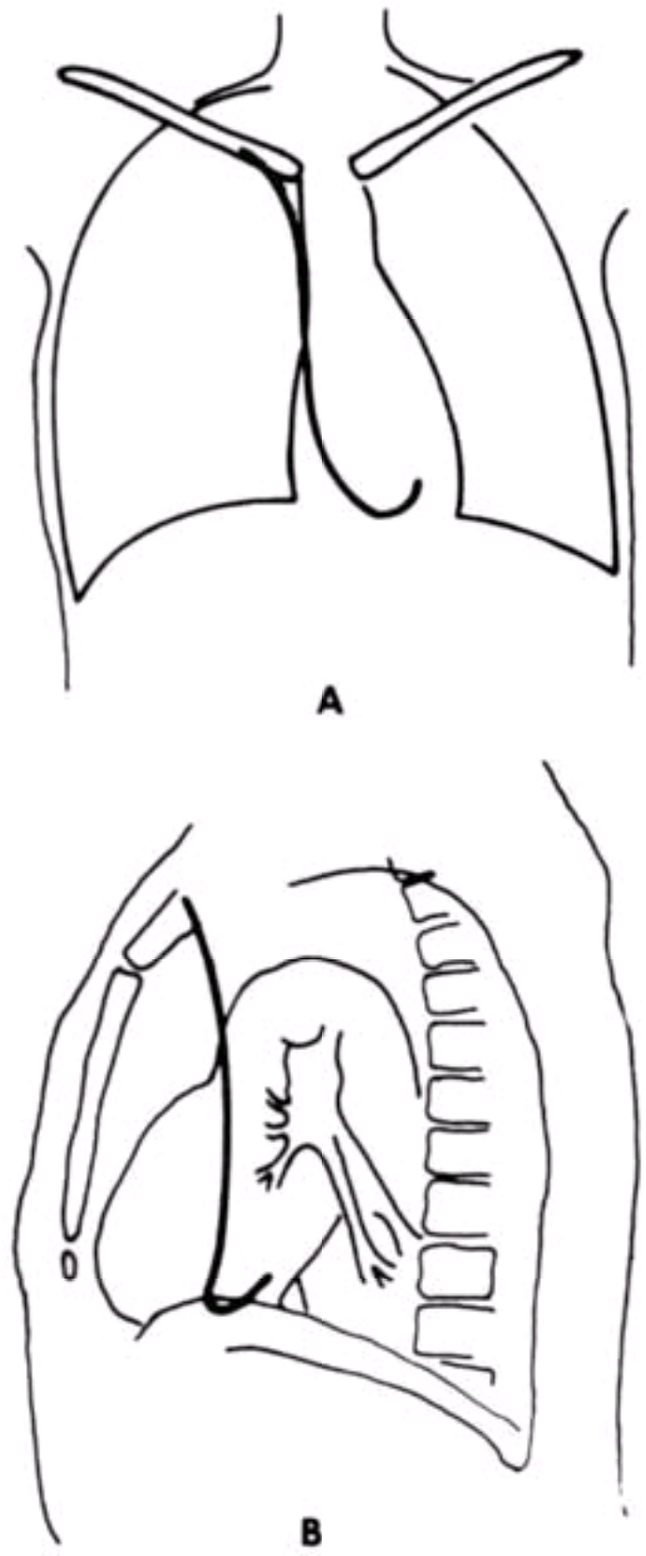


Figure 15-13 A pacing catheter that is outside or abuts the cardiac silhouette and is not properly positioned within the right ventricular cavity suggests myocardial perforation. *(From Tarver RD, Gillespie KR: The misplaced tube. Emerg Med, Feb 29, 1988, p 97.)*

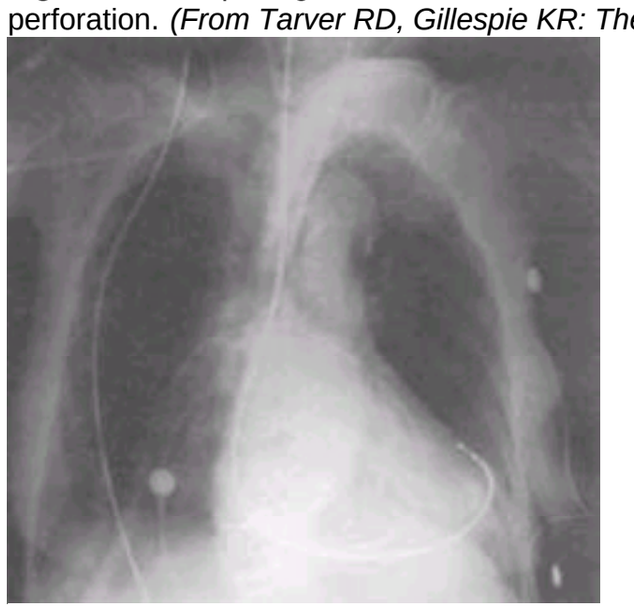


Figure 15-14 Combined defibrillator-transcutaneous pacemaker unit (Zoll-PD). The unit defibrillates through standard hand held paddles and has additional cable connections for electrocardiograph monitoring electrodes and for pacing electrodes. *(Courtesy of ZMI Corporation, Cambridge, Mass.)*

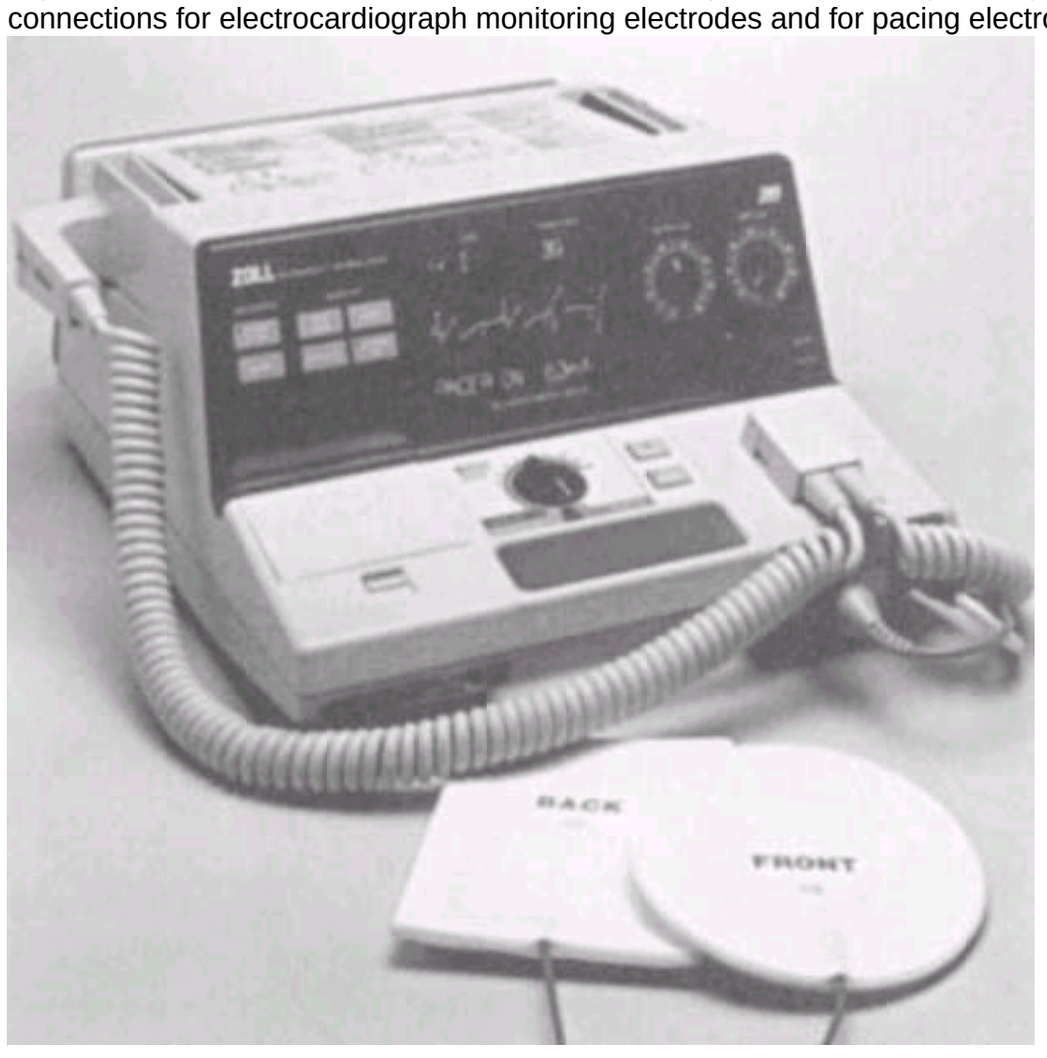


Figure 15-15 "Stand alone" transcutaneous pacemaker (Zoll NTP). This unit has a built-in monitor and strip chart recorder. *(Courtesy of ZMI Corporation, Cambridge, Mass.)*

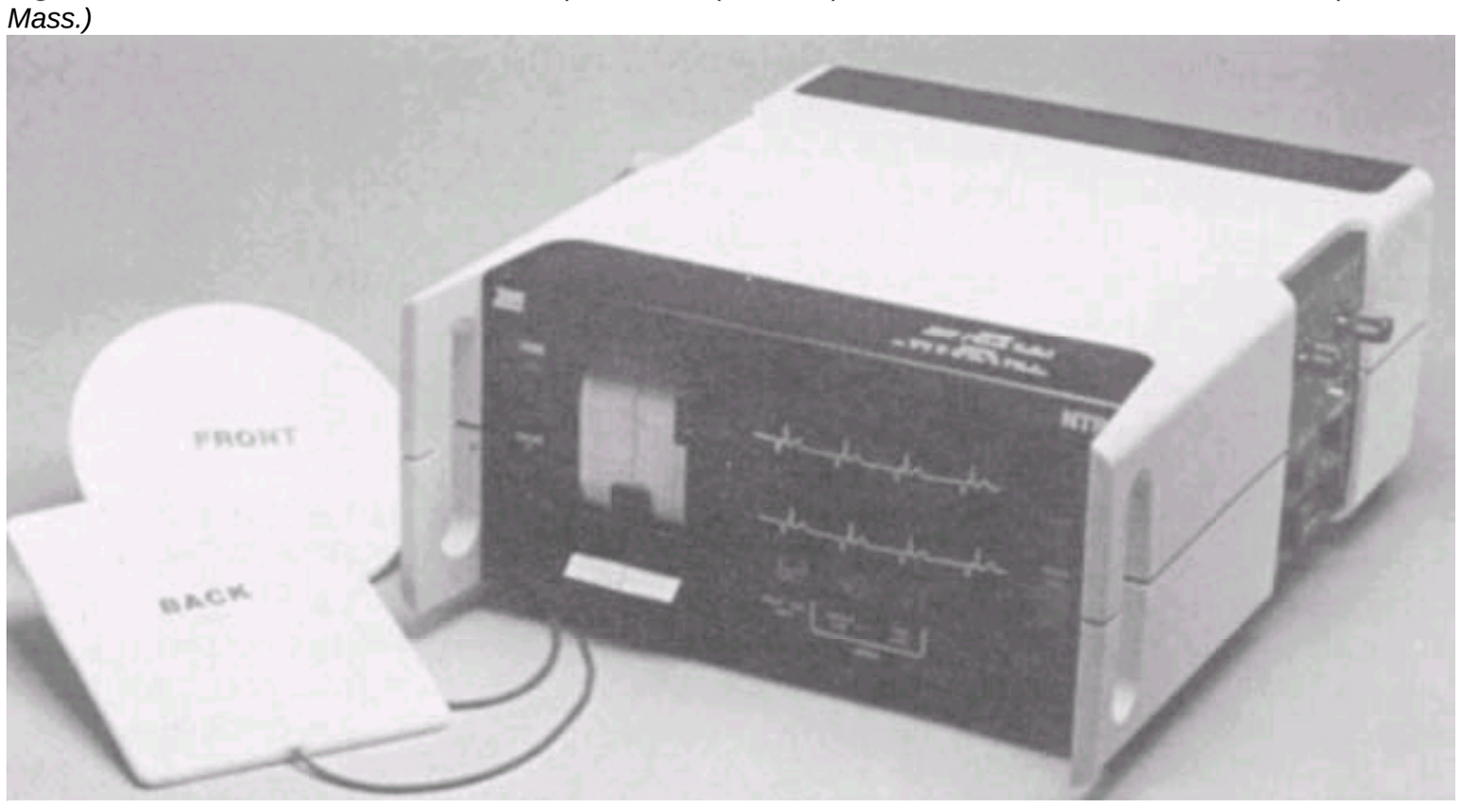


Figure 15-16 The top three rhythm strips *(A–C)* are taken from a standard wall-mounted electrocardiograph monitor. They all demonstrate large pacer spikes without capture. The underlying rhythm cannot be determined and could be treatable ventricular fibrillation. The bottom rhythm strip *(D)* demonstrates a tracing on the same patient with the external pacer monitor (special dampening). Note that the pacing spikes are much smaller, and it is easily seen that the underlying rhythm is asystole, without pacer capture. The presence of a T-wave after the QRS complex is a good indicator of ventricular capture.

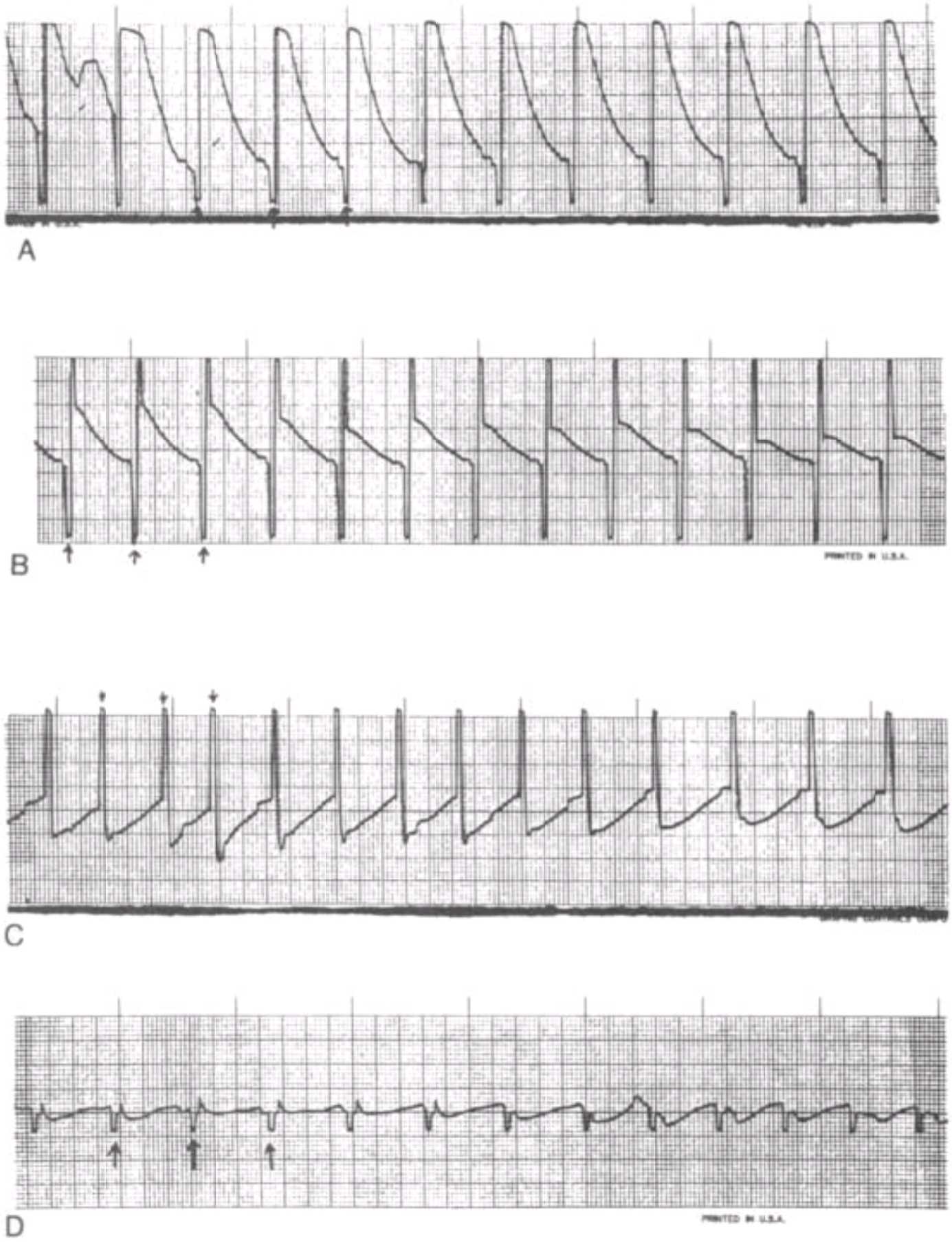


Figure 15-17 Correct placement of transcutaneous pacemaker electrodes (see text). *(Courtesy of ZMI Corporation, Cambridge, Mass.)*

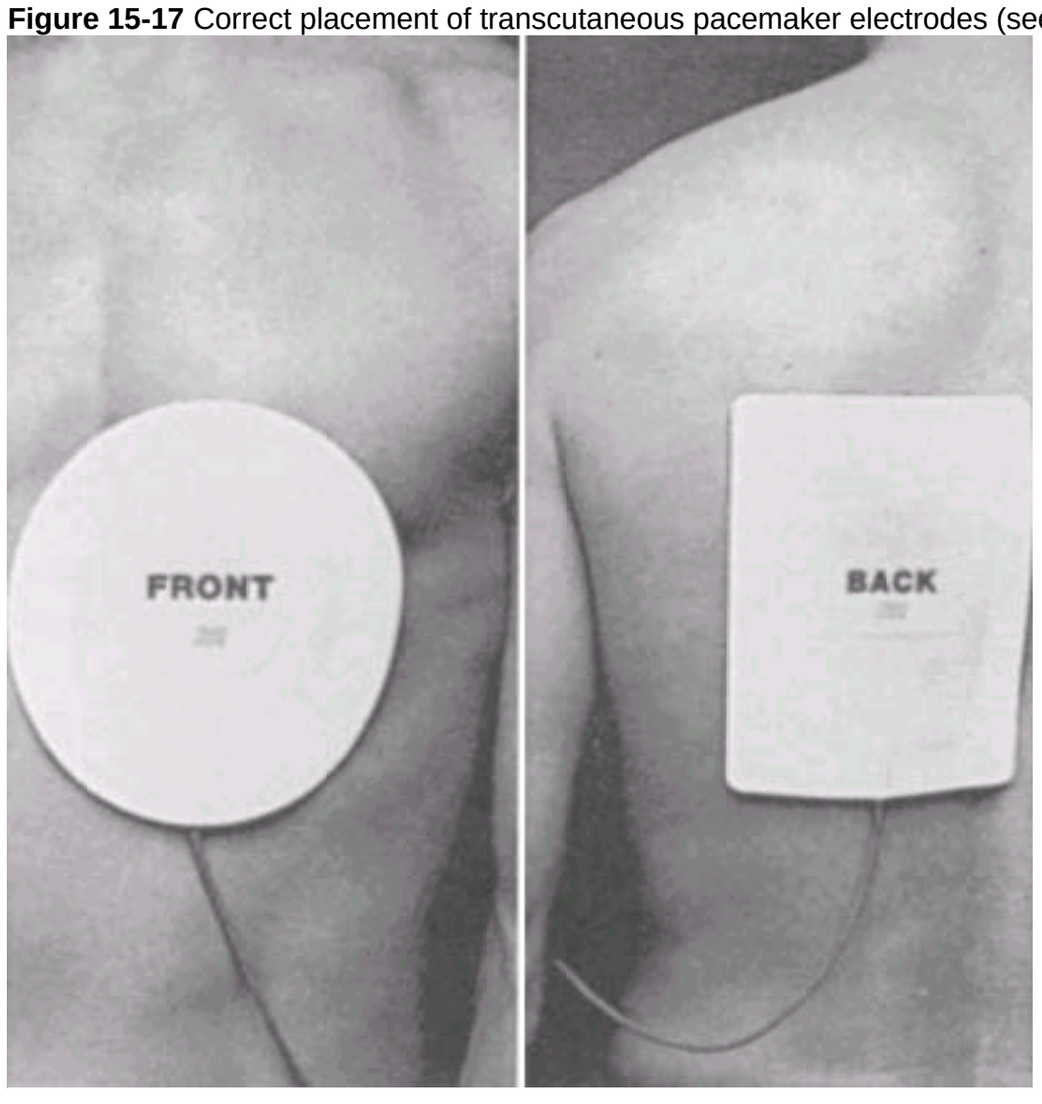


Figure 15-18 Rate and output selections.

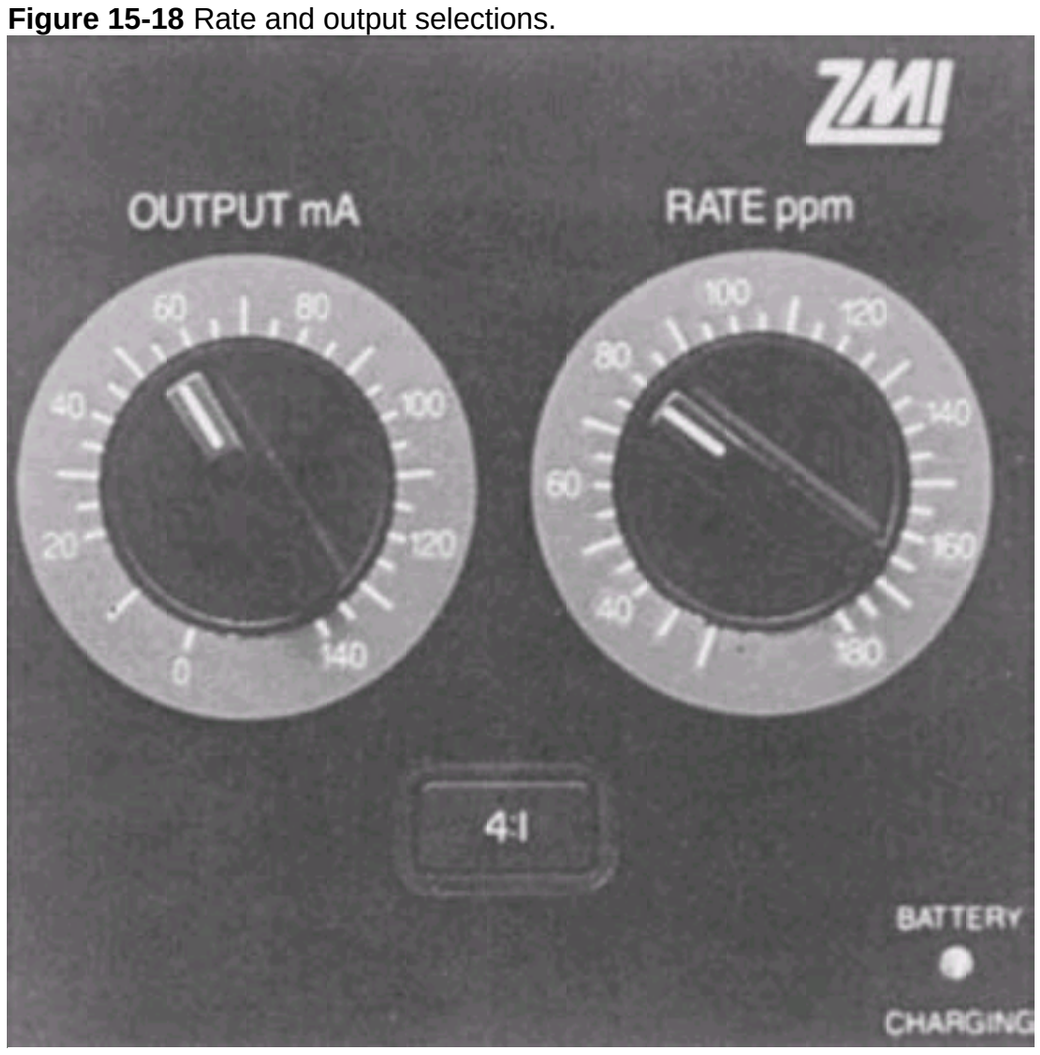


Figure 15-19 Assessing electrocardiogram capture with TCP. Note that the monitor has been adapted to accommodate the large pacing artifact so as not to obscure the underlying ventricular activity.

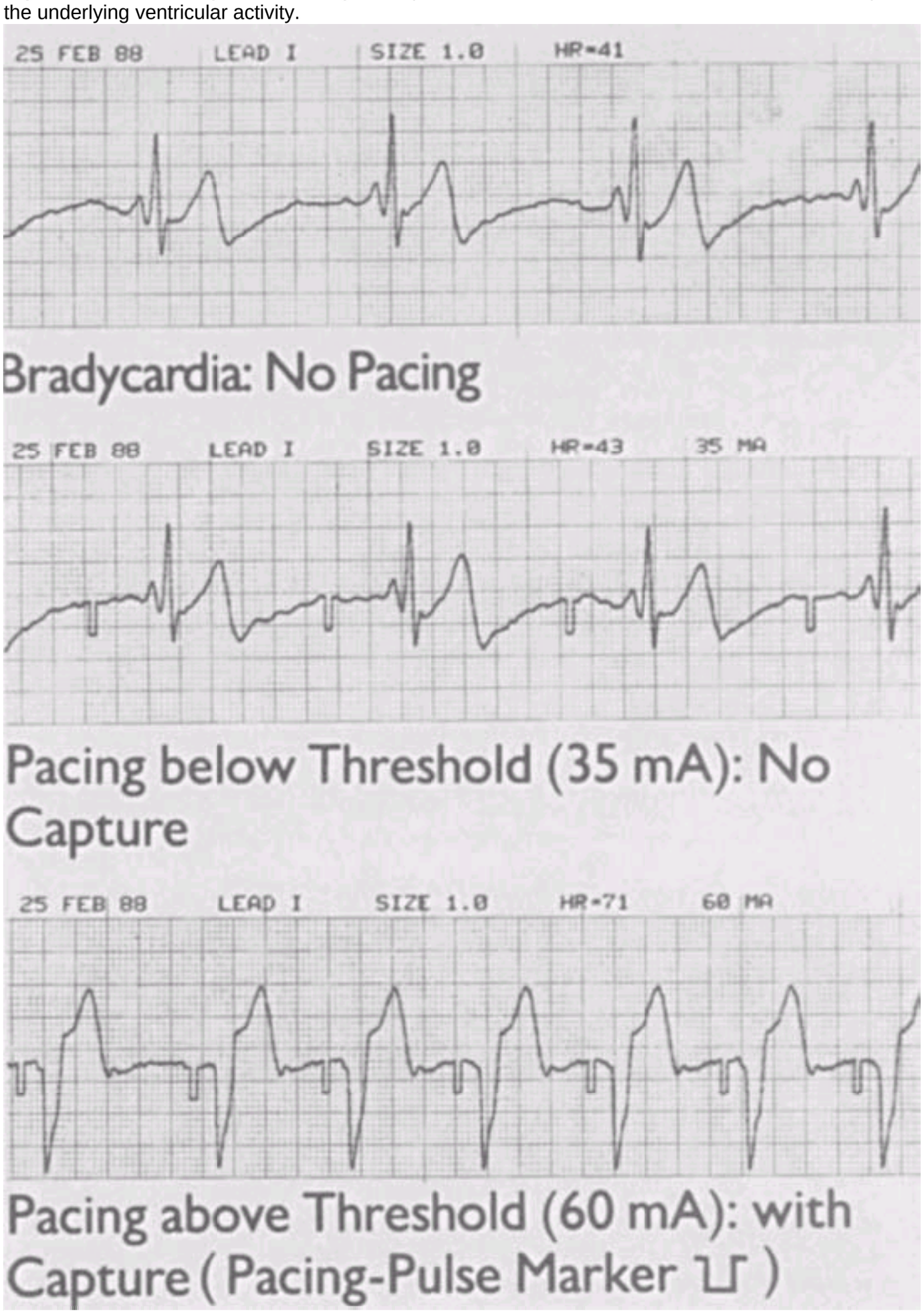


Figure 16-1 Production of cardiac tamponade by injections of saline into the pericardial sac. Although pericardial space can acutely accommodate 80 to 120 mL of fluid without a significant increase in pericardial pressure, note steep increases in pressure and drop in blood pressure at about 200 mL of saline. Once critical volumes are reached, very small increases cause significant hemodynamic compromise. *(From Fowler NO: Physiology of cardiac tamponade and pulsus paradoxus. II: Physiological, circulatory, and pharmacological responses in cardiac tamponade. Mod Concepts Cardiovasc Dis 47:116, 1978. Reproduced by permission of the American Heart Association, Inc.)*

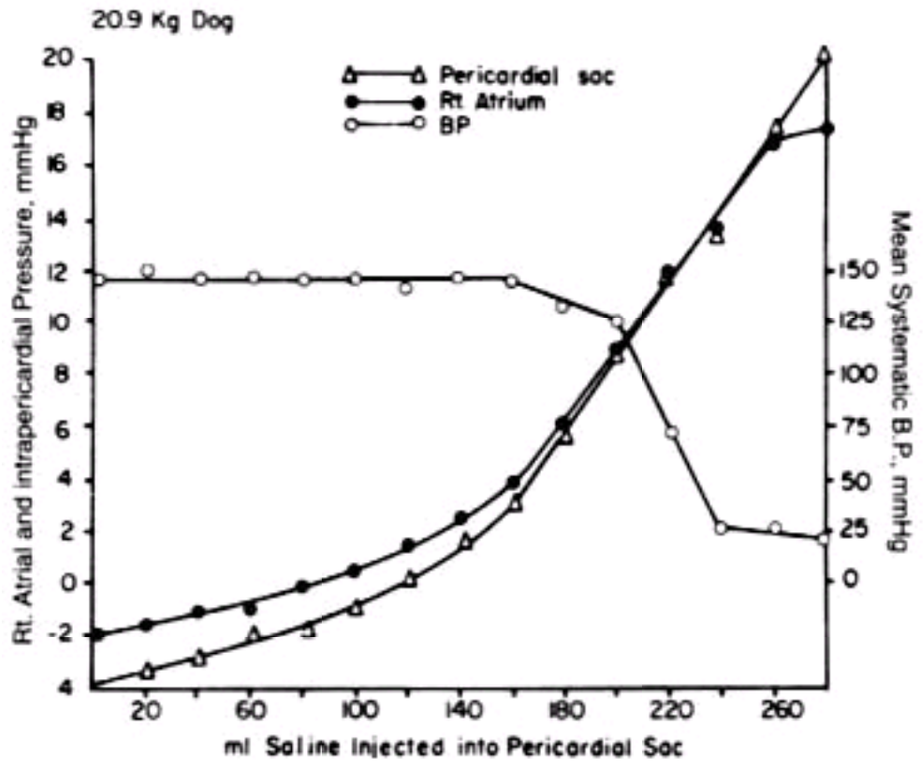


Figure 16-2 Relationship of intrapericardial pressure to volume of pericardial fluid. Note that pressure drops rapidly when a small amount of fluid is removed. *(From Pories W, Gaudiani V: Cardiac tamponade. Surg Clin North Am 55:573, 1975. Reproduced by permission.)*

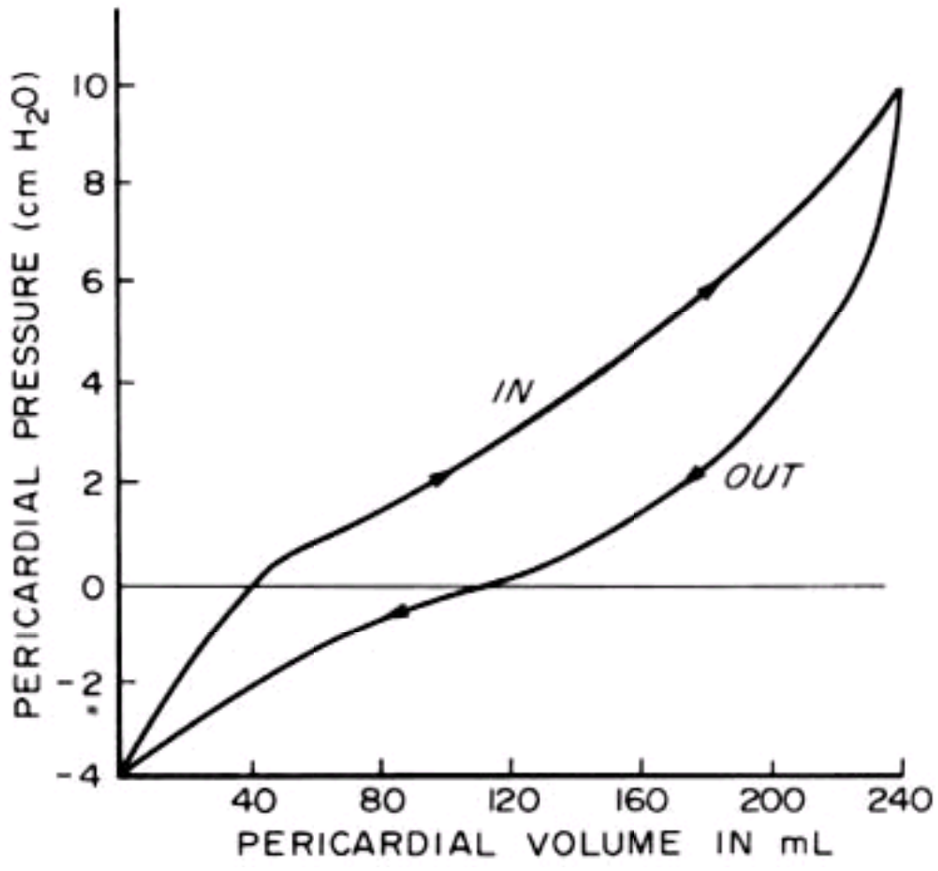


Figure 16-3 Summary of physiologic changes in tamponade. RV, right ventricle. *(From Shoemaker WC, Carey JS, Yao ST, et al: Hemodynamic monitoring for physiological evaluation, diagnosis, and therapy of acute hemopericardial tamponade from penetrating wounds. J Trauma 13:36, 1973; and Spodick D: Acute cardiac tamponade: Pathologic physiology, diagnosis, and management. Prog Cardiovasc Dis 10:65, 1967. Reproduced by permission.)*

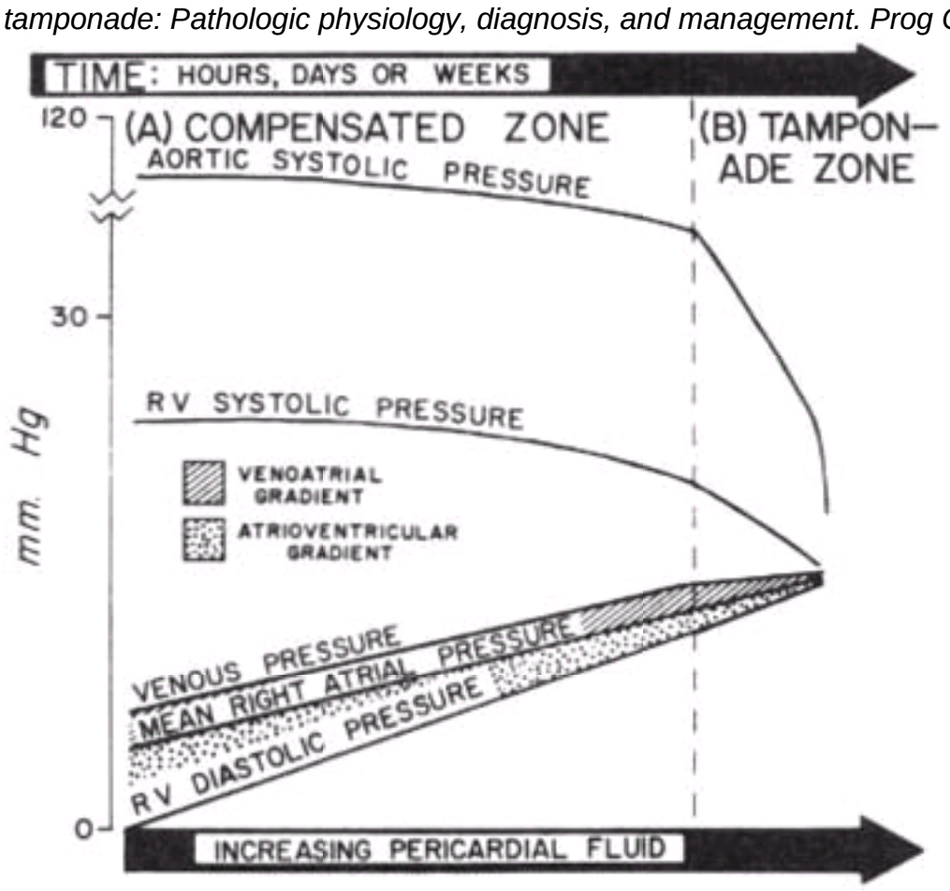


Figure 16-4 Normally systolic blood pressure drops slightly during inspiration. To measure *pulsus paradoxus*, the patient breathes normally while lying at a 45-degree angle. The blood pressure cuff is inflated well above systolic pressure and slowly deflated. When the pulse is first heard only during expiration, this is the upper value. The cuff is deflated until the pulse is heard during both inspiration and expiration, and this is the lower value. The difference in the two values is the amount of *pulsus paradoxus*. A difference of more than 12 mm Hg is abnormal.

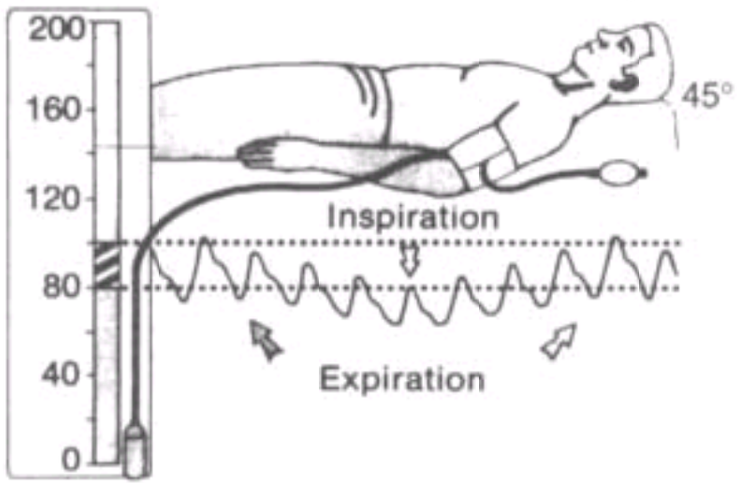


Figure 16-5 Epicardial fat pad sign. The water density space between the radiolucent epicardial fat and the mediastinal fat represents the pericardium and its contents and should be 2 mm or less. An increase suggests pericardial fluid or thickening. *A,* Left anterior-oblique chest film. *B,* Lateral chest film. In acute tamponade, the chest radiograph has very minimal diagnostic value.

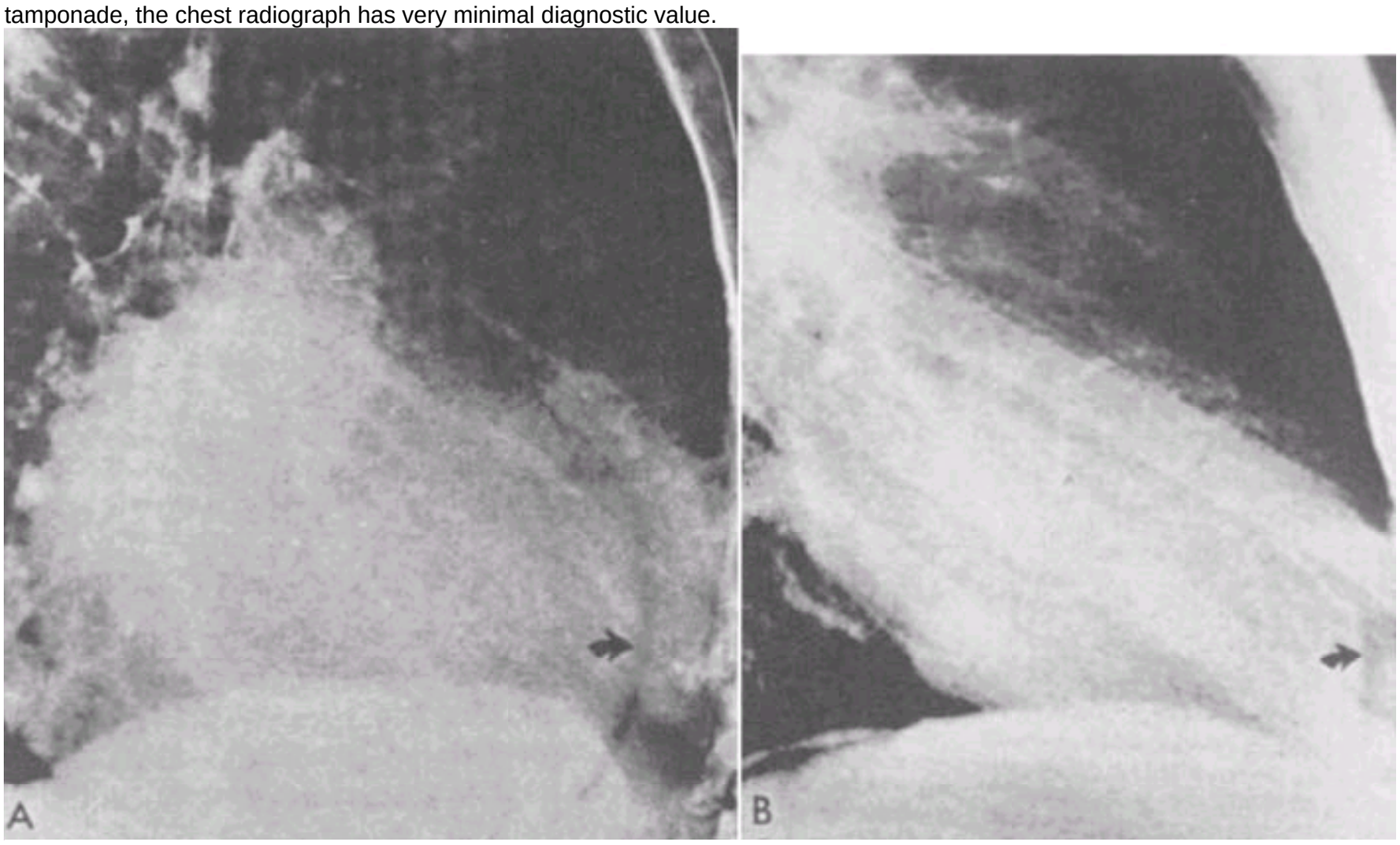


Figure 16-6 Overall, the electrocardiogram (ECG) has a low sensitivity for pericardial effusion or tamponade, but PR depression, low voltage, or electrical alternans may be seen. Lewis lead ECG showing total electrical alternans of both amplitude and configuration of P and QRS complexes. This is rarely seen but is highly suggestive of tamponade. Note that electrical alternans may not be evident in standard ECG leads. *(From Sotolongo RP, Horton JD: Total electrical alternans in pericardial tamponade. Am Heart J 101:854, 1981. Reproduced by permission.)*

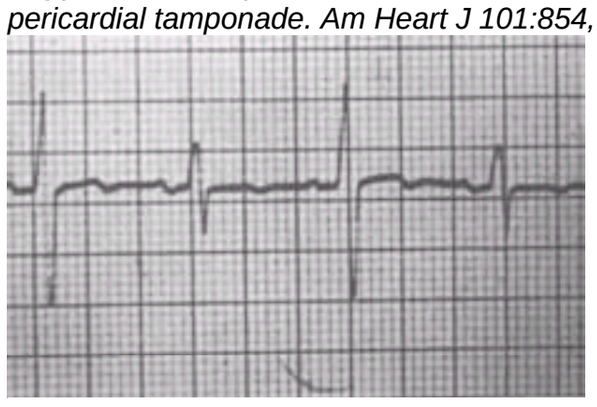


Figure 16-7 Management of *nontraumatic* cardiac tamponade. IV, intravenous line; CVP, central venous pressure; ECG, electrocardiogram.

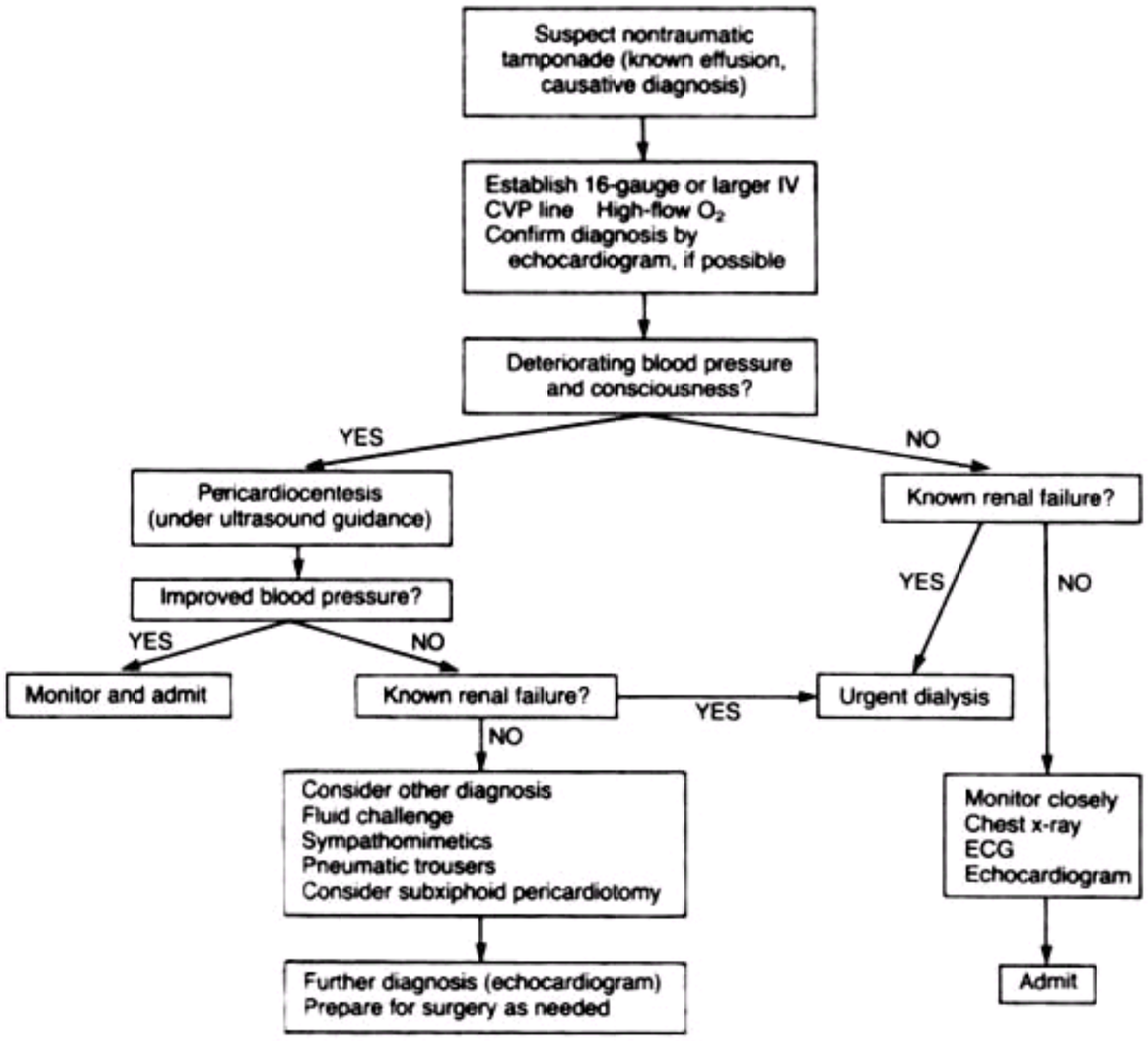


Figure 16-8 Management of *traumatic* cardiac tamponade. IV, intravenous lines; CVP, central venous pressure; ECG, electrocardiogram; R/O, rule out.

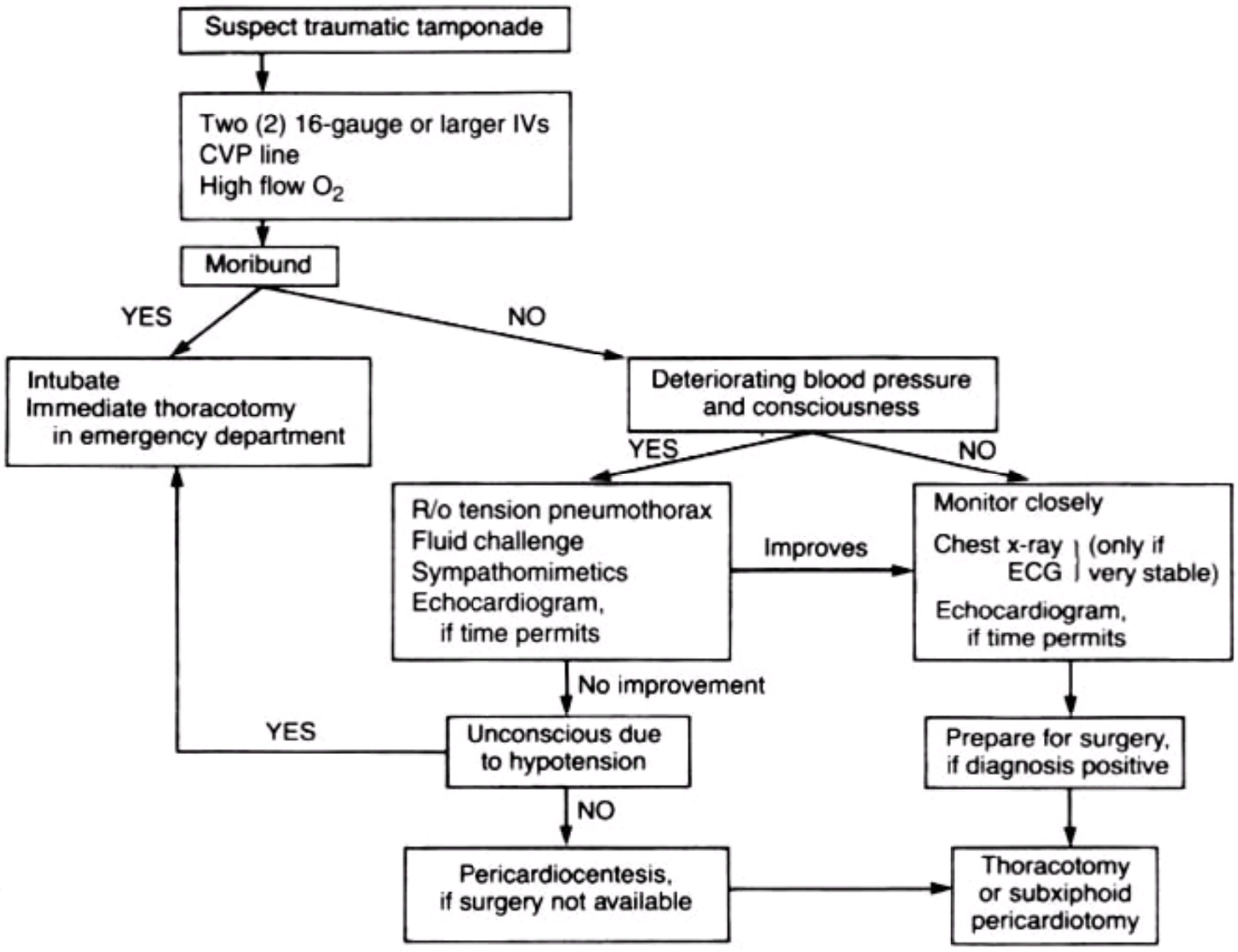


Figure 16-9 Equipment for emergent pericardiocentesis: long, 18-ga spinal needle; wire with alligator clips for connection to the electrocardiograph machine; and syringe (three-way stopcock optional). Sterile skin preparation and local anesthetic are also required.

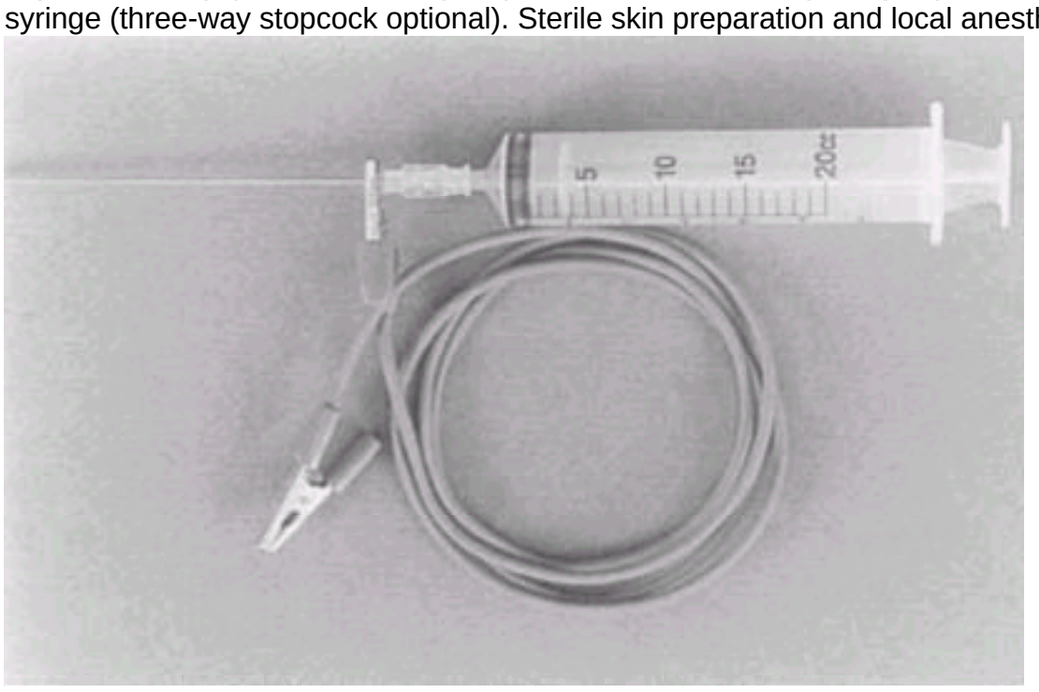


Figure 16-10 An example of the contents of a prepackaged pericardiocentesis set: finder needle, Seldinger wire, dilator, catheter guide, and pigtail catheter. Sterile skin preparation and local anesthetic are also required.

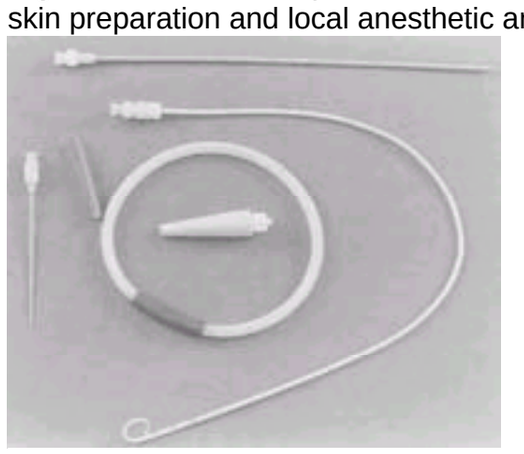


Figure 16-11 Parasternal approach for pericardiocentesis. The patient is depicted in a supine position, although a preferable position would be sitting at a 45° angle, if the patient's clinical condition permits. Note ECG monitoring via alligator clamp attached to lead.

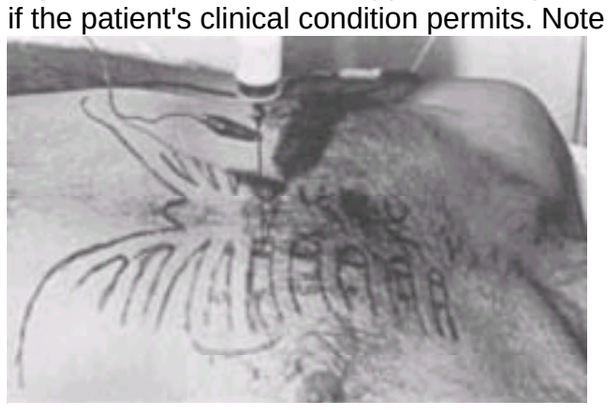


Figure 16-12 *A* and *B*, Xiphosternal approach for pericardiocentesis. The needle is aimed for the sternal notch or the left shoulder. Note the electrocardiography monitor. Although the patient is shown in a supine position, a preferable position would be sitting at a 45° angle, if the patient's condition permits. This general approach is also used for intracardiac injection of advanced cardiac life support drugs.

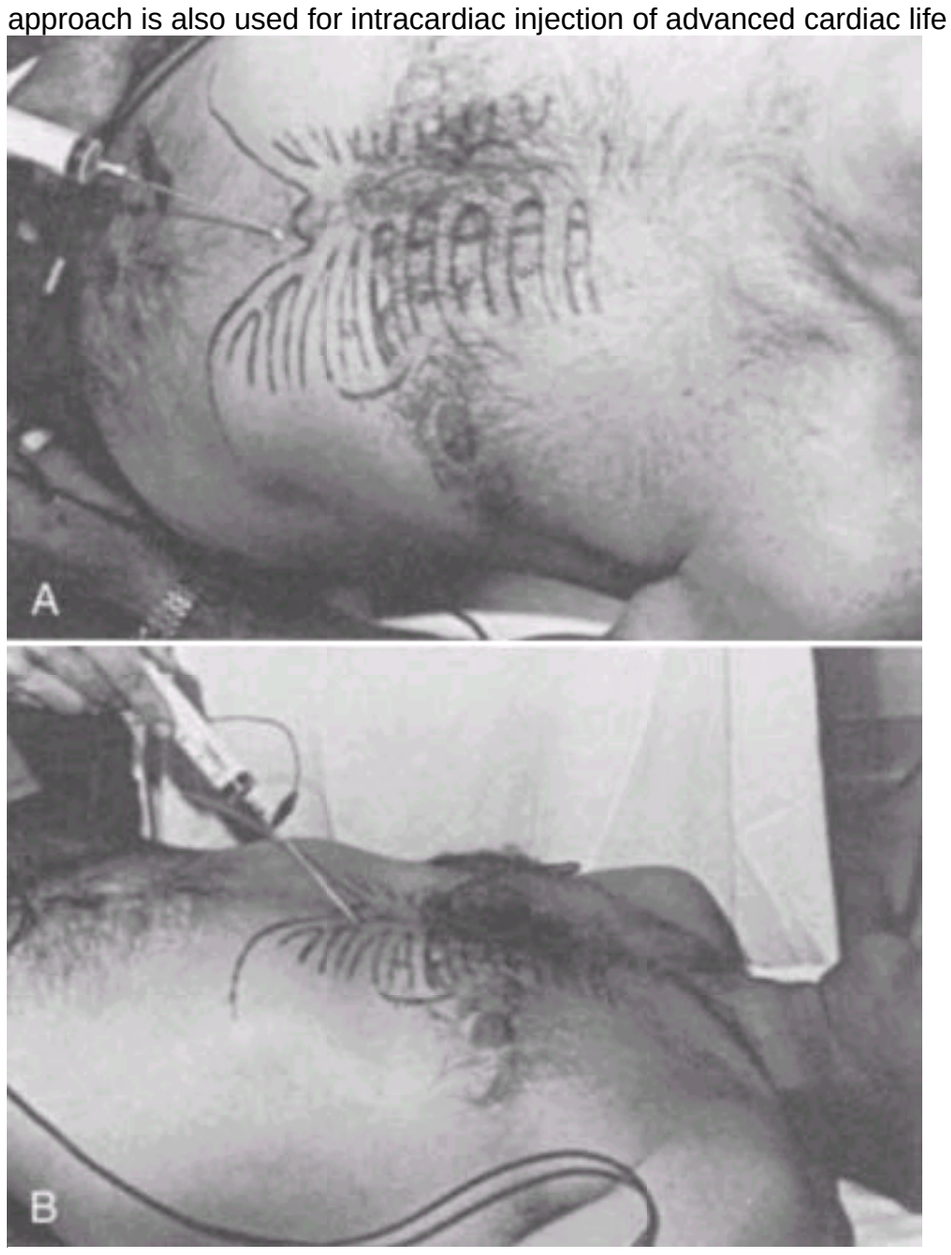
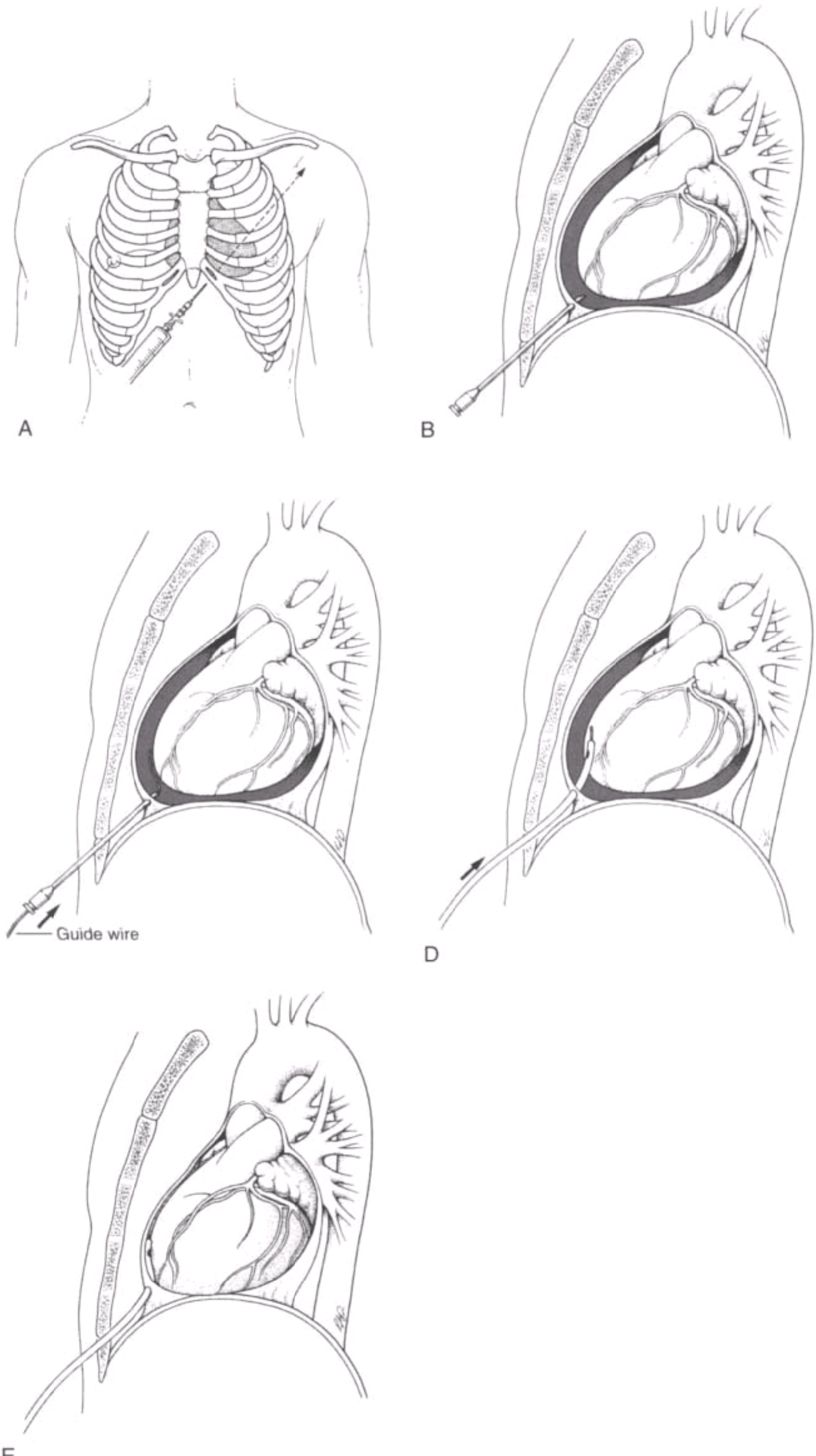


Figure 16-13 Subxyphoid approach to catheter placement into pericordial space. A short needle (16- or 18-ga) is inserted into the left xiphocostal angle perpendicular to the skin and 3 to 4 mm below the left costal margin *(A)*. After advancing the needle to the inner aspect of the rib cage, the needle's hub is depressed so that the needle points toward the patient's left shoulder. The needle is then cautiously advanced about 5 to 10 mm until fluid is reached *(B)*. The fingers may sense a distinct "give" when the needle penetrates the parietal pericardium. Successful removal of fluid confirms the needle's position. The syringe is then disconnected from the needle, and the flexible tip of the guide wire is advanced into the pericardial space *(C)*. The needle is withdrawn and replaced with a soft, multihole pigtail catheter (No. 6 to 8 Fr) using the Seldinger technique. After dilating the needle tract, the catheter is advanced over the guide wire into the pericardial space *(D)*. Once the catheter is properly positioned, aspiration of fluid should result in rapid improvement in blood pressure and cardiac output, a decrease in atrial and pericardial pressures, and a decrease in the degree of any paradoxical pulse *(E)*. Electrical alternans, if present, also decreases or disappears. *(From Spodick DH: The technique of pericardiocentesis. J Crit Illness 2:91, 1987.)*



E

Figure 16-14 Current of injury. There is an obvious change in the electrocardiogram when the pericardiocentesis needle touches the epicardium. Following slight withdrawal *(arrow)*, the ST-segment elevation diminishes.

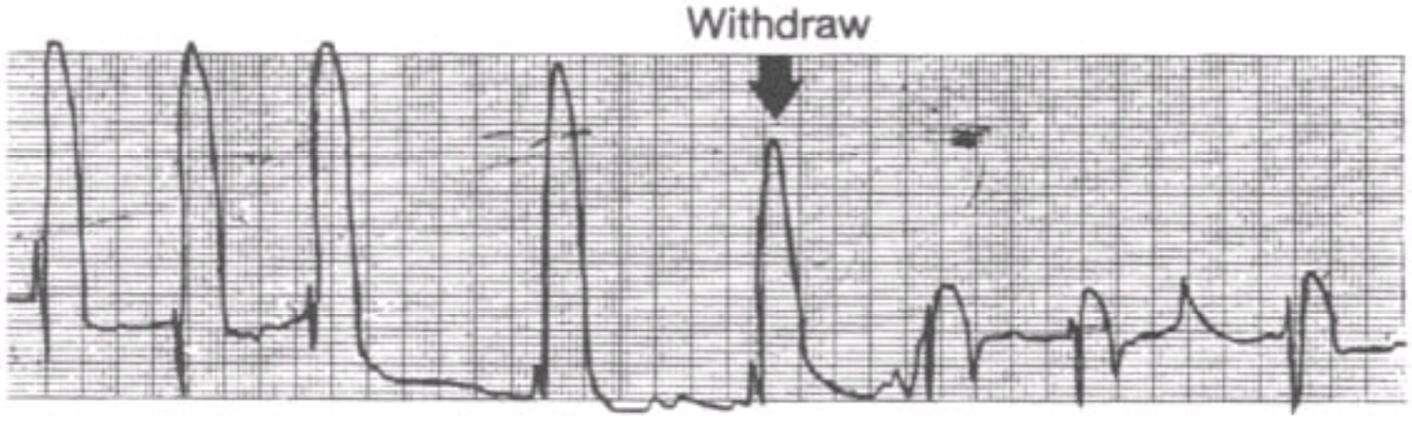


Figure 17-3 The Thumper Cardiopulmonary Resuscitator.

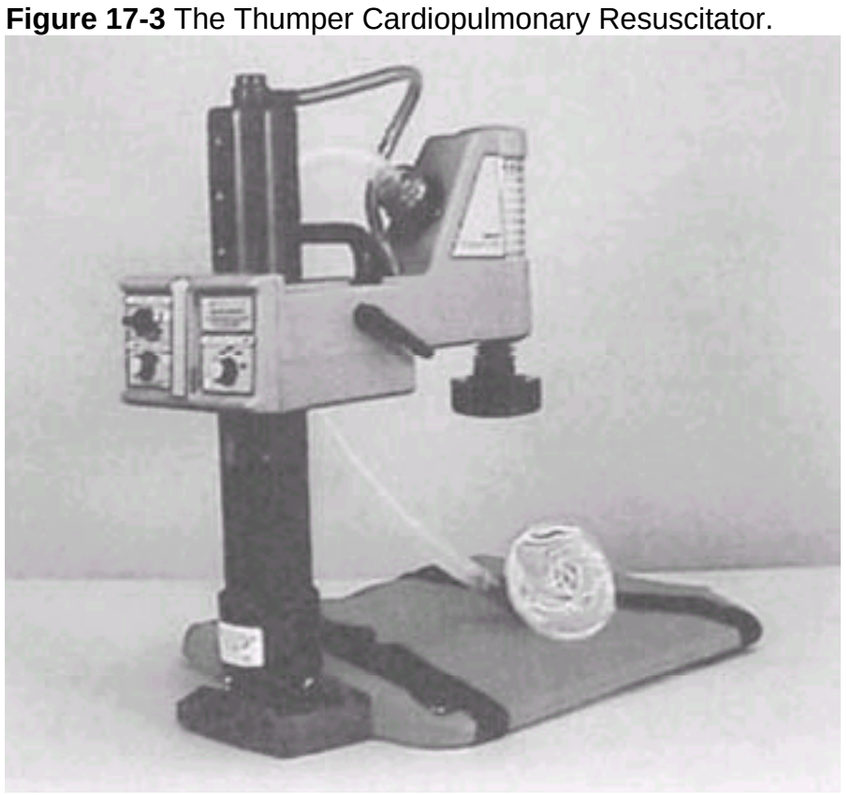


Figure 17-4 An artist's conception of basic rescuers performing interposed abdominal compression cardiopulmonary resuscitation. For clarity, both rescuers are shown on the same side of the victim. With two rescuers, the first compresses the chest and performs ventilation while the second compresses the abdomen. With three rescuers, ventilation, chest compression, and abdominal compression are performed by each individual. Ideally, the rescuer performing chest compressions is on the victim's right side and the rescuer performing abdominal compressions is on the victim's left side. *(From Voorhees WD, Niebauer MJ, Babbs CF: Improved oxygen delivery during cardiopulmonary resuscitation with interposed abdominal compressions. Ann Emerg Med 12:128, 1983. Reproduced by permission.)*

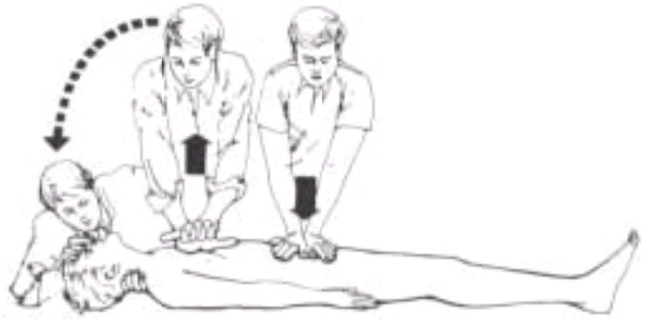


Figure 17-5 A comparison of vest cardiopulmonary resuscitation (CPR) and standard manual CPR. With vest CPR, a pneumatic system inflates and deflates a bladder surrounding the chest. The compression phase results in circumferential compression as opposed to point compression during manual CPR. The vest CPR system is equipped with defibrillator electrodes that monitor the patient's electrocardiogram as well as allow defibrillation without removal of the vest.

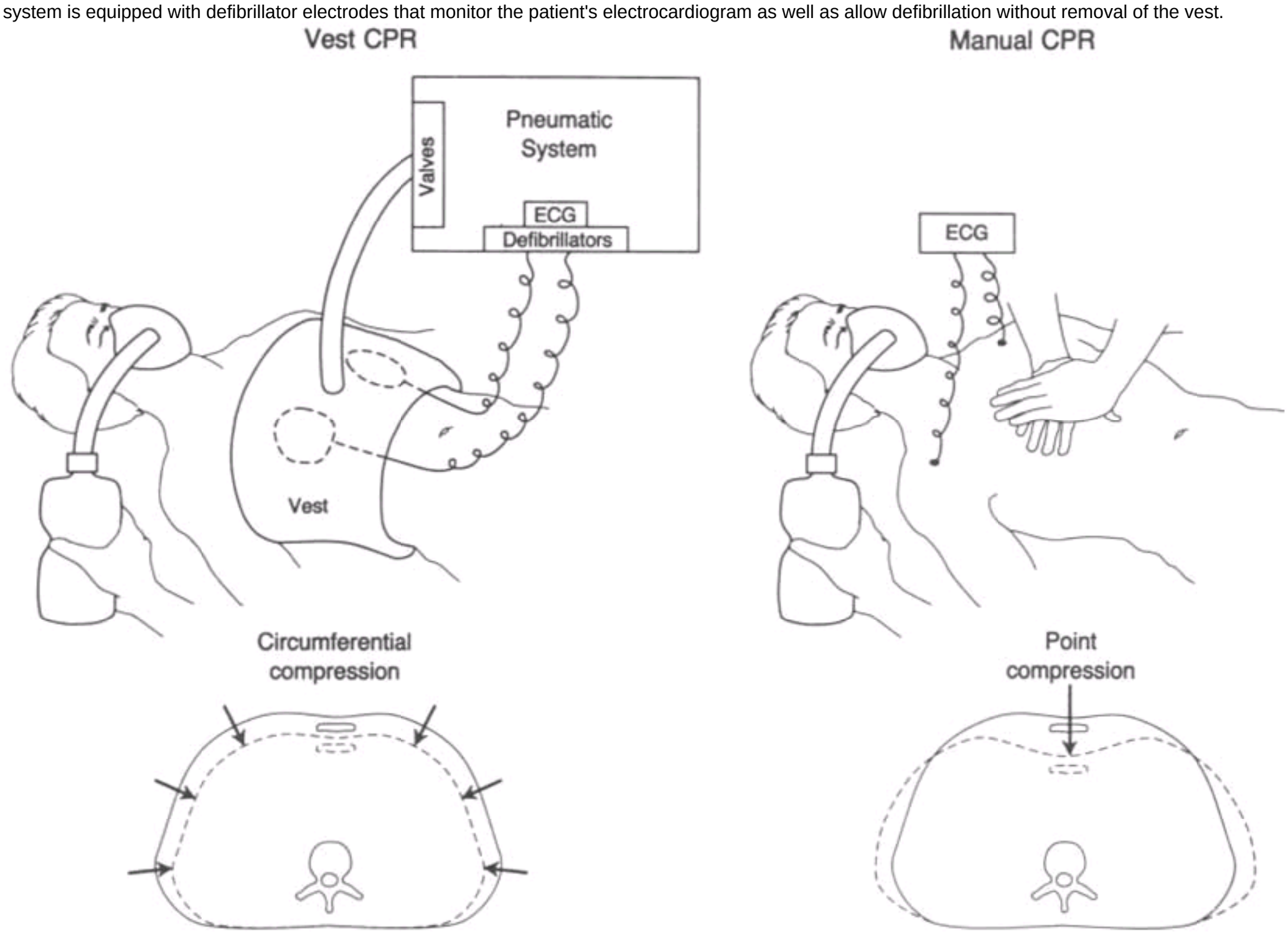


Figure 17-6 The active compression-decompression (ACD) device uses a suction cup positioned at mid-chest at the level of the nipples. The device is pushed downward into the chest during the compression phase of CPR. The force of compression can be approximated by the gauge within the handle. During the decompression phase, the handle is actively pushed up and away from the chest while the suction keeps the device attached. This active withdrawal characterizes the decompression phase of CPR. The value of this adjunct is uncertain.

Figure 17-7 Lifestick Resuscitator used for active compression-decompression cardiopulmonary resuscitation (ACD-CPR).

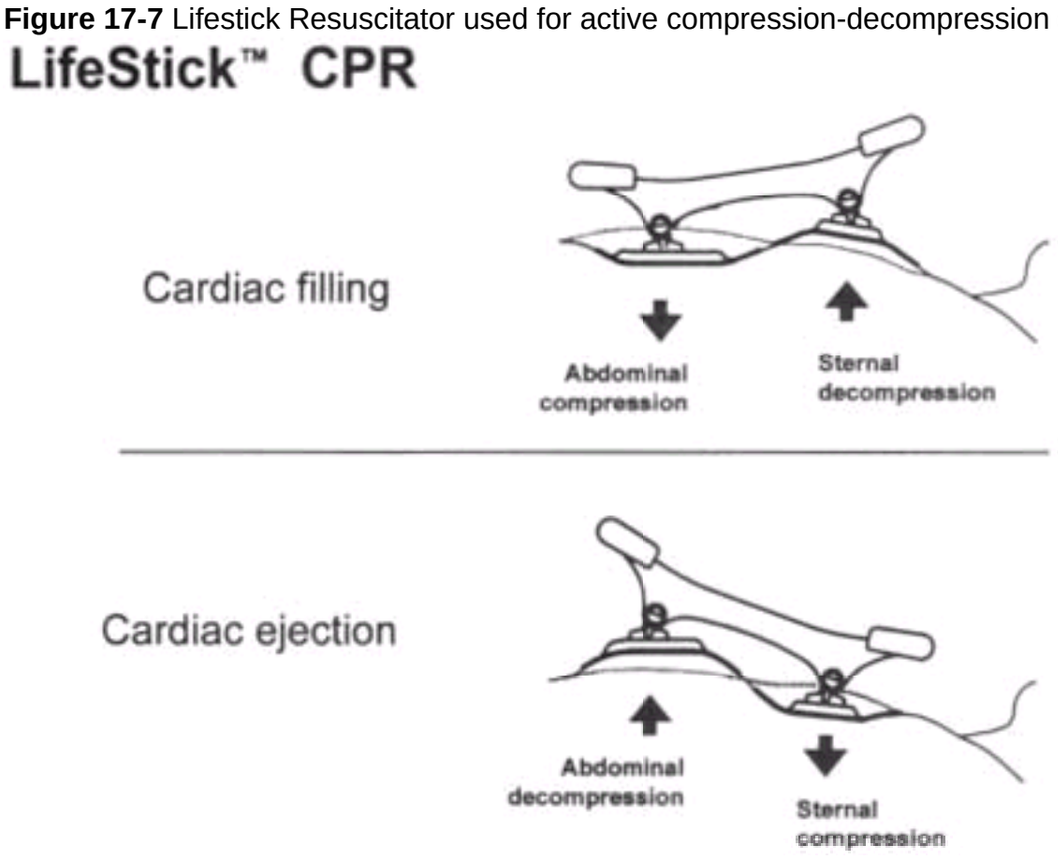


Figure 18-1 An emergency department thoracotomy is a dramatic and heroic intervention that has a very low survival rate. In the proper setting, its use can be advocated (see text). In this case of multiple gunshots, the abdomen was also opened in an attempt to cross-clamp the abdominal aorta, but no readily reversible pathology of the chest or abdomen was found.

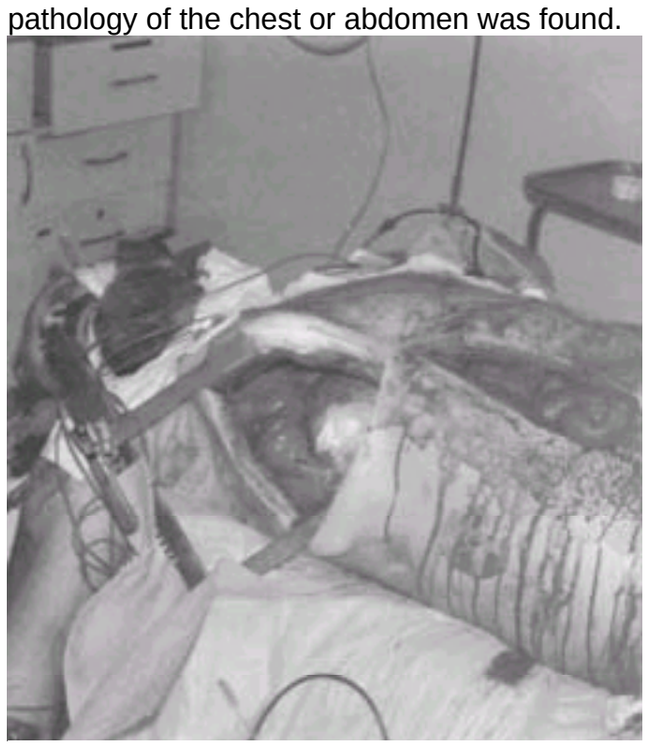


Figure 18-2 Bedside ultrasound demonstrating hemopericardium.

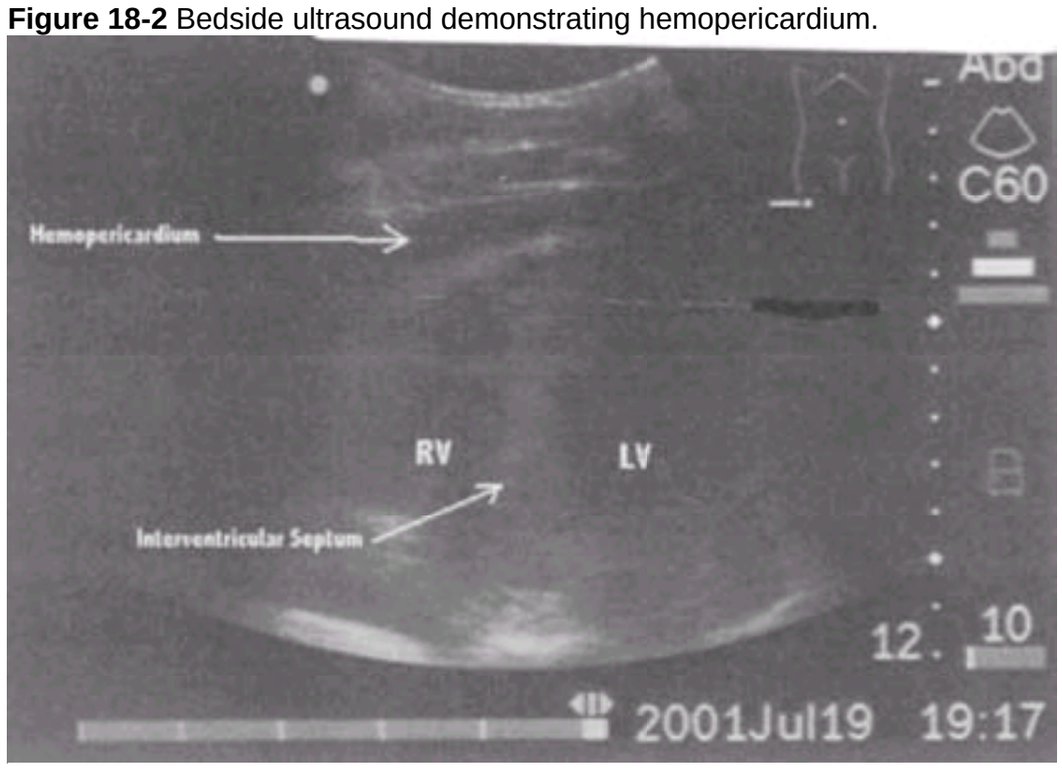


Figure 18-3 An algorithmic approach to chest trauma. QRS, organized electrical activity; VFib, ventricular fibrillation; (+)TAP, pericardial tap yielding blood; US, ultrasonography.

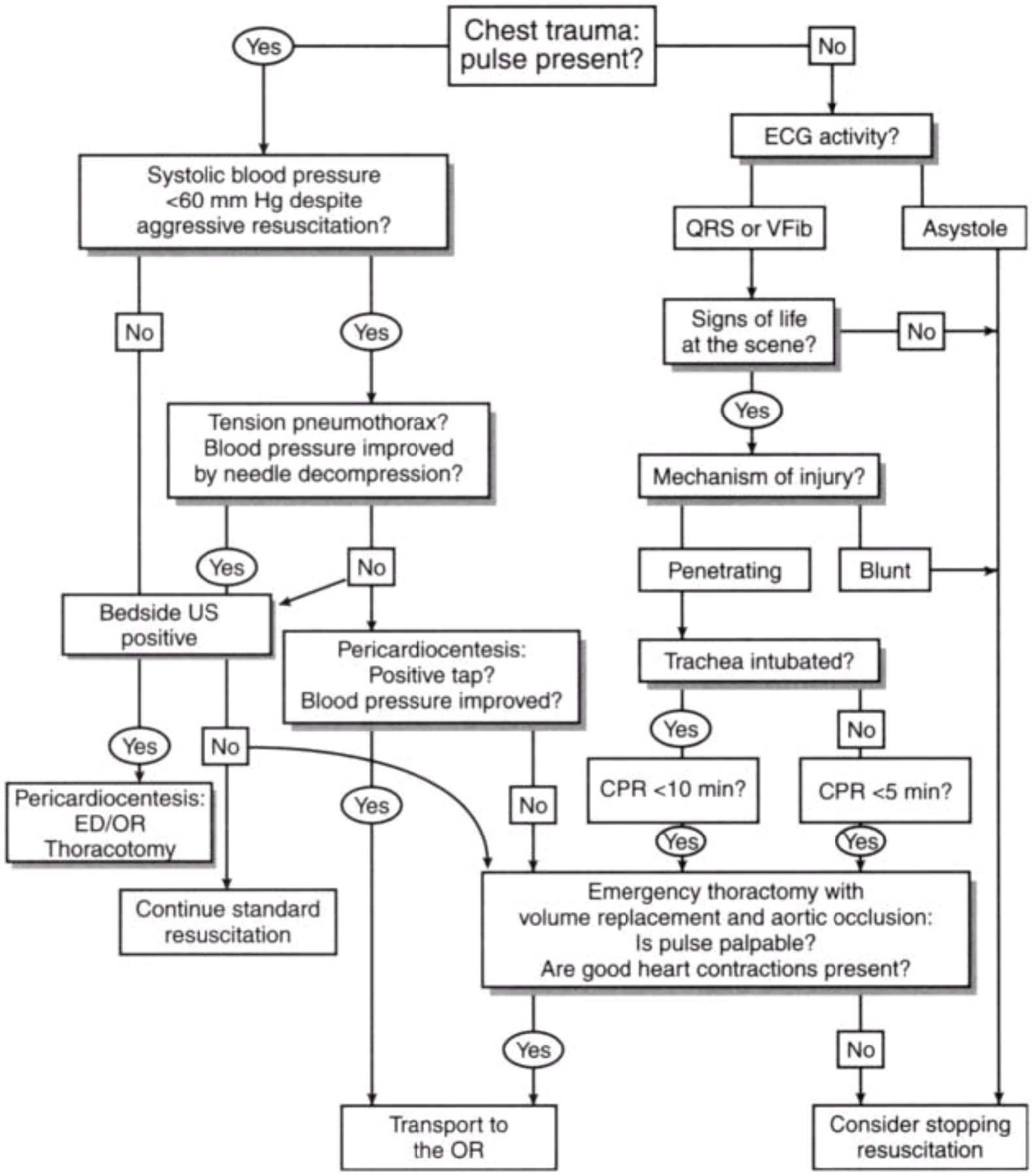


Figure 18-4 Left anterolateral thoracotomy. *A,* Several towels or sandbags are placed under the left scapula and the arm is raised above the head. The patient should be intubated. A nasogastric tube can be inserted to facilitate differentiation of the esophagus from the aorta. *B,* The left anterolateral submammary incision is the suggested initial approach. Ideally the incision is made between the fourth and fifth ribs. Generally the incision is just inferior to the nipple (male) or along the inframammary fold (female). The incision begins on the sternum and extends to the posterior axillary line, where it should be deep enough to partially transect the *latissimus dorsi* muscle. *C, Dashes* indicate the incision site of the inframammary fold in women.

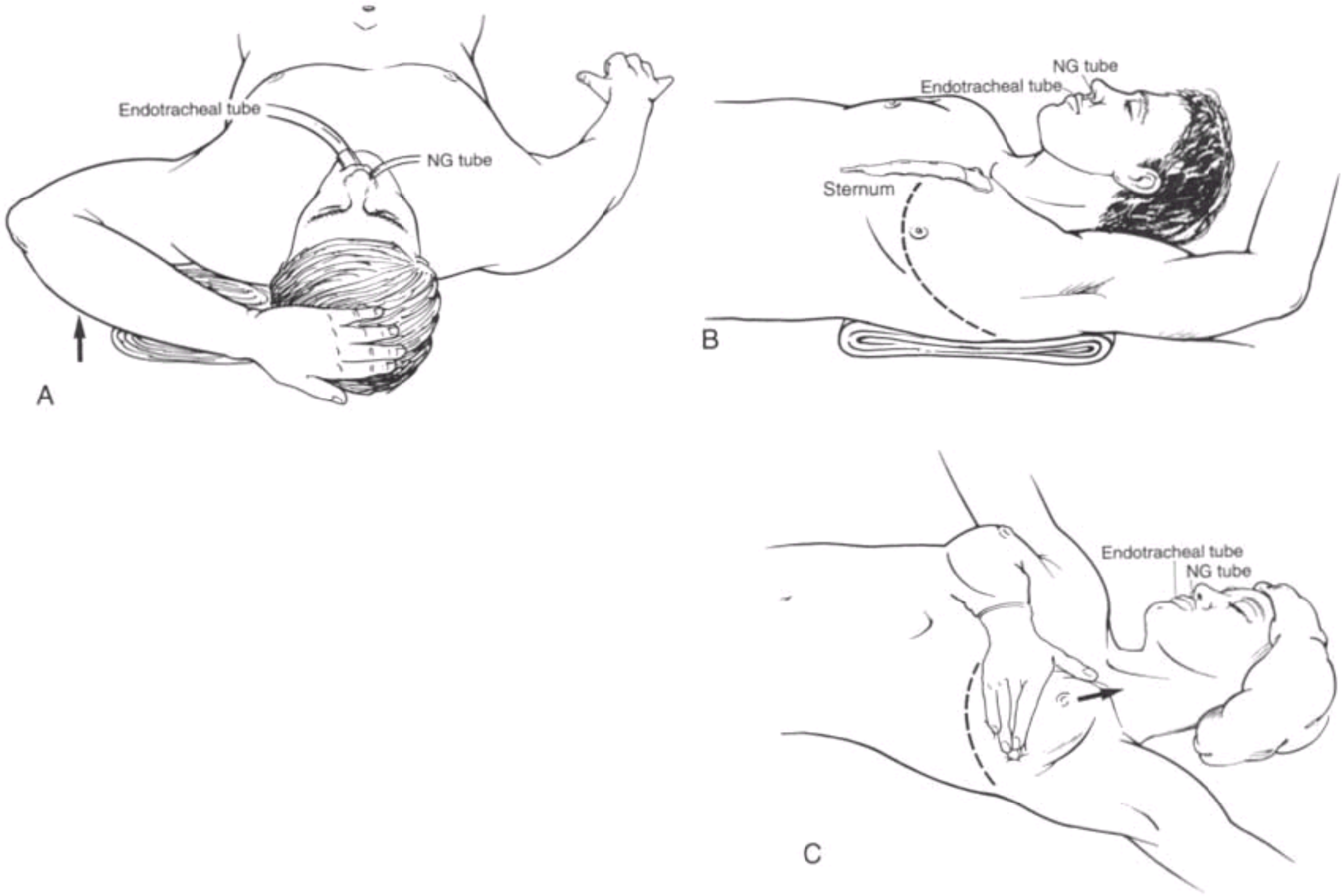


Figure 18-5 *A*, When entering the pleural cavity, it is important to make the incision on top of the rib to avoid the intercostal vessels. Once a hole has been made into the pleural space, the incision is widened with blunt scissors by cutting the intercostal muscles. The fourth and fifth fingers of the operator's free hand are inserted into the pleural space to fend off the lung as the scissors divide the intercostal muscle. Momentary cessation of ventilation will collapse the lung. Alternatively, the right mainstem bronchus can be intubated, which permits continuous ventilation and oxygenation without inflating the left lung into the operating field. *B,* The incision must always be carried to the posterior axillary line to maximize exposure. The rib spreader should be placed with the handle laterally. Because it can be difficult to determine if tamponade has occurred using visual inspection alone, the pericardium must be opened to definitively determine if tamponade is present. Using tissue pick ups with teeth, the operator must press hard against the pericardium to engage it within the tissue pick ups. The incision is started near the diaphragm and anterior to the phrenic nerve, which is easily identified as a thick tendon-like structure. Using blunt scissors, the incision is carried to the root of the aorta.

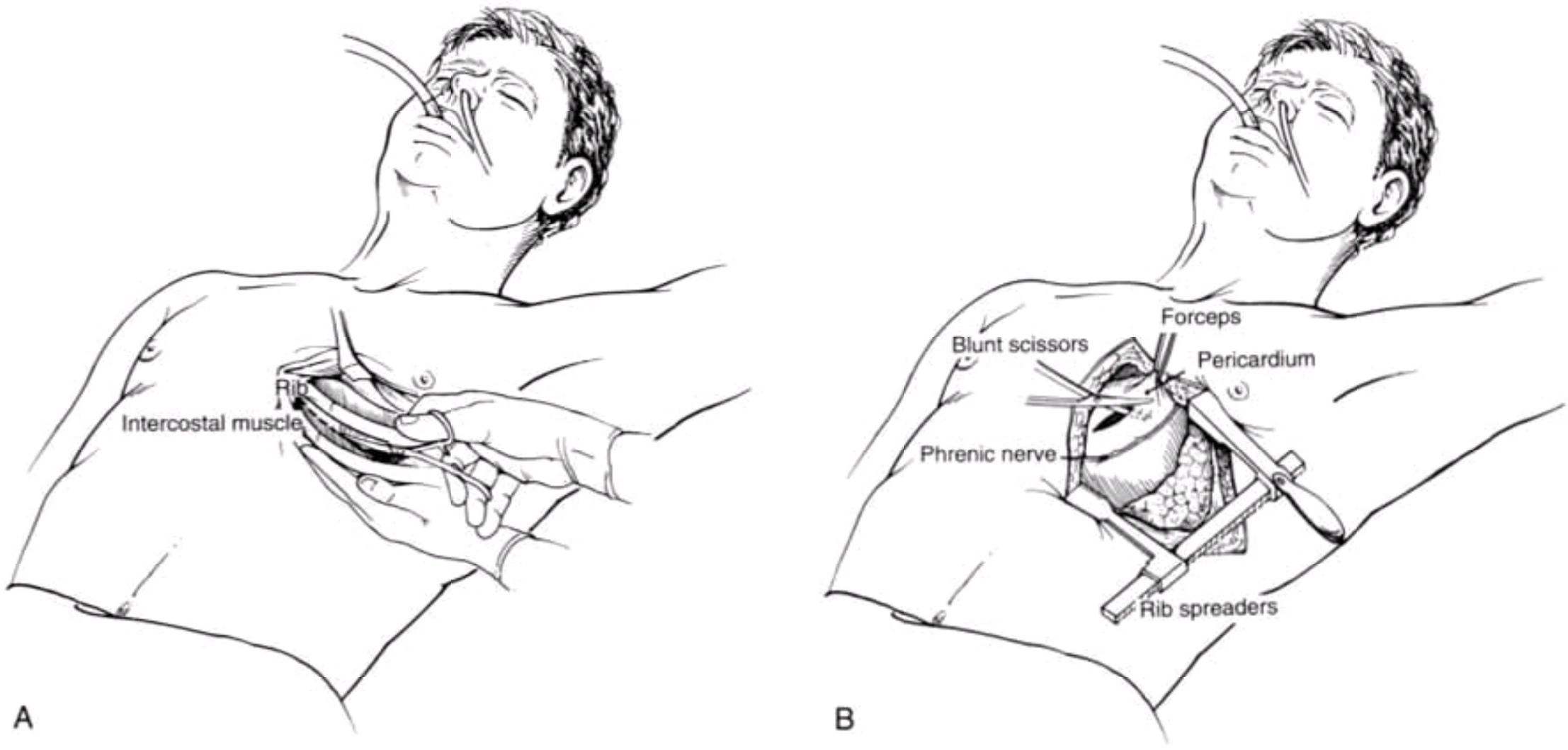


Figure 18-6 Two-handed method of cardiac massage. The ventricles are compressed toward the interventricular septum. Note how the hands flank the left anterior descending artery, which overlies the septum. Avoid using excessive finger *tip* pressure or lifting the heart, which slows ventricular filling by distorting the soft atrial caval junction.

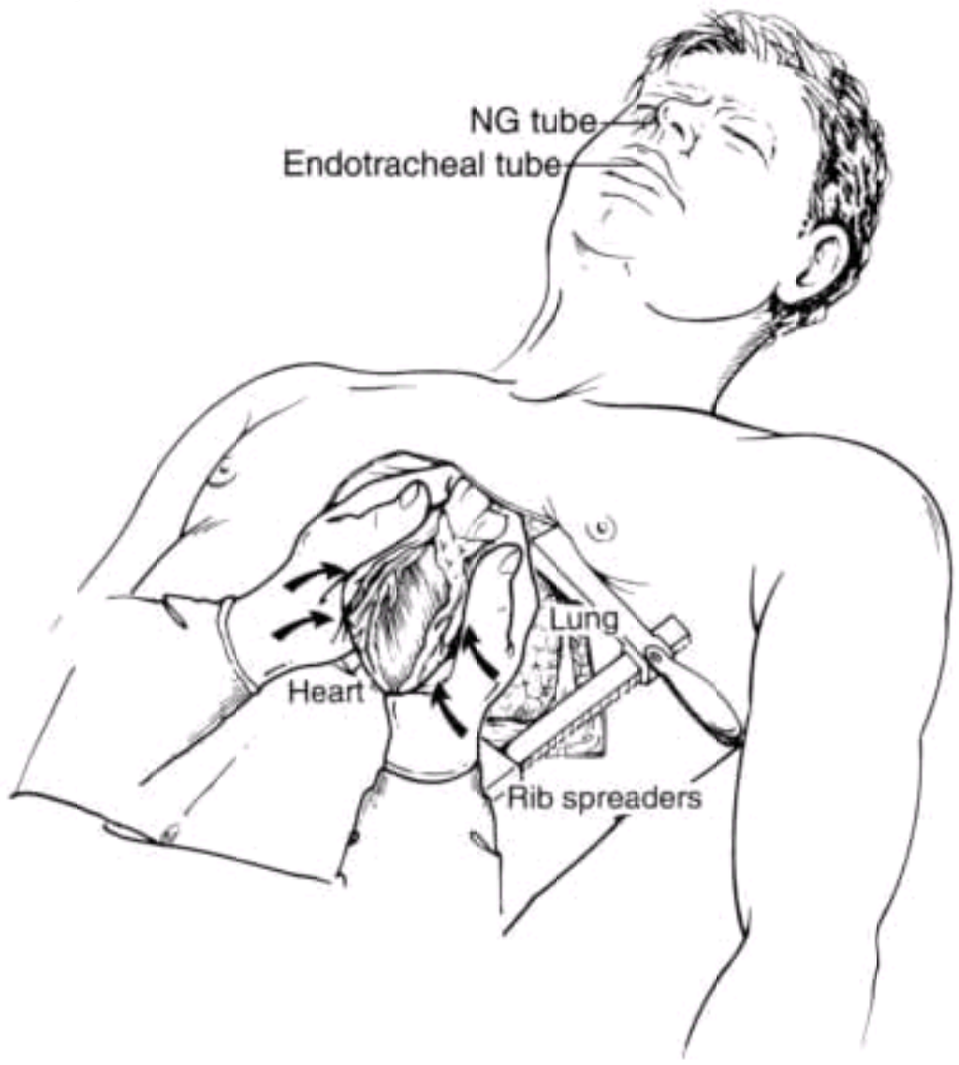


Figure 18-7 Technique of cardiac stapling to temporarily control hemorrhage. An assistant can approximate tissues with fingertip pressure or, as illustrated, two half-horizontal sutures can be used to approximate the wound edges and reduce bleeding. A skin stapler with wide (6 mm) staples is used to place staples 5 mm apart. This technique may be used for atrial and ventricular lacerations. Following stabilization of the patient's condition, the wound is revised in the operating room.

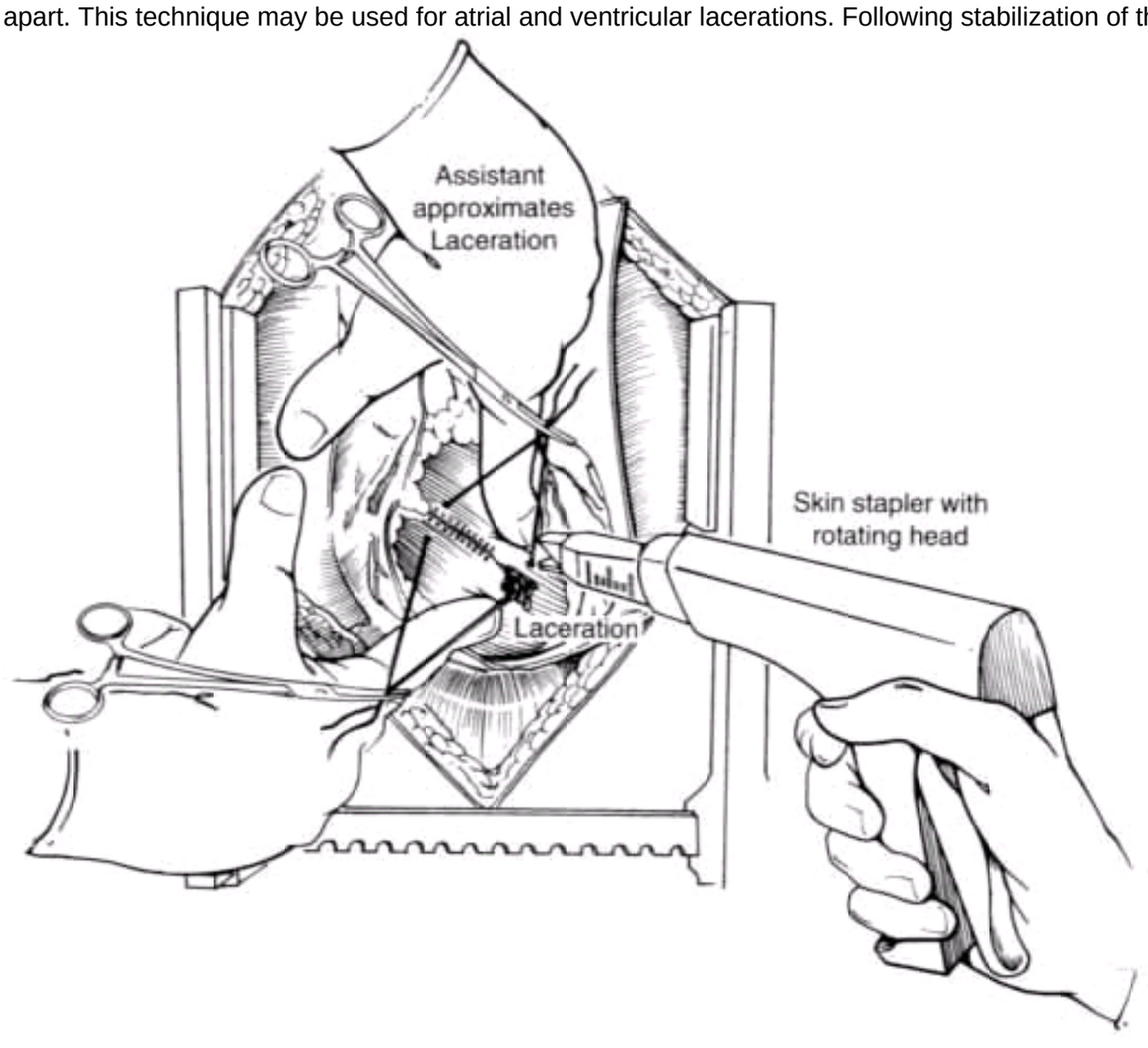


Figure 18-8 *A*, Technique of repair. Multiple horizontal mattress sutures are placed 6 mm from the wound edge before tying. The wound is closed just enough to stop the bleeding. Teflon pledgets are used for reinforcement. Closure without pledgets incurs the risk of sutures ripping through the contracting myocardium. Similarly, the use of simple vertical sutures should be discouraged because of the risk of suture dissection through the myocardium. *B,* For repairs near a coronary artery, care is taken to pass the suture under the artery. Note that rectangles of pericardial tissue may be substituted for ready made Teflon pledgets.

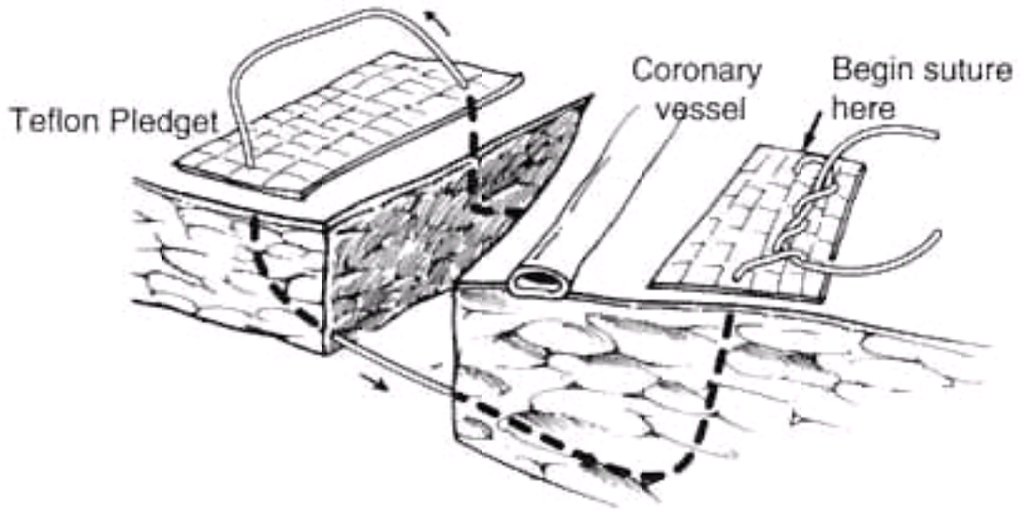


Figure 18-9 Hemorrhage control using two widely placed incomplete mattress sutures. An assistant then crosses the two "half-horizontal" sutures to bring the wound edges into apposition. By controlling the hemorrhage in this manner, the assistant's hands are outside of the operative field, fully exposing the wound edges. This facilitates a more orderly closure of the wound. Following repair of the wound, the sutures may either be removed or tied to each other.

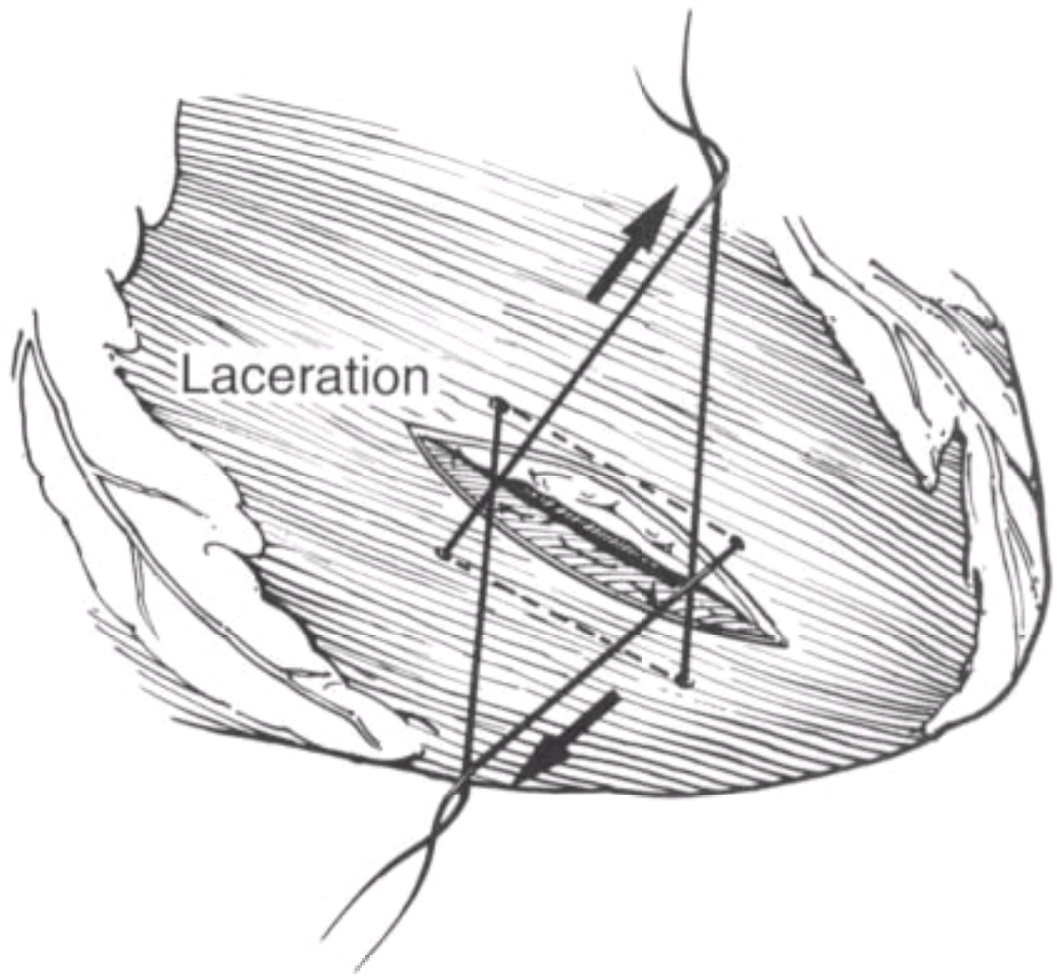


Figure 18-10 Sauerbruch maneuver: the *method of choice* for reducing heavy bleeding from cardiac wounds. Venous inflow occlusion is achieved by using the first and second or second and third fingers as a clamp.

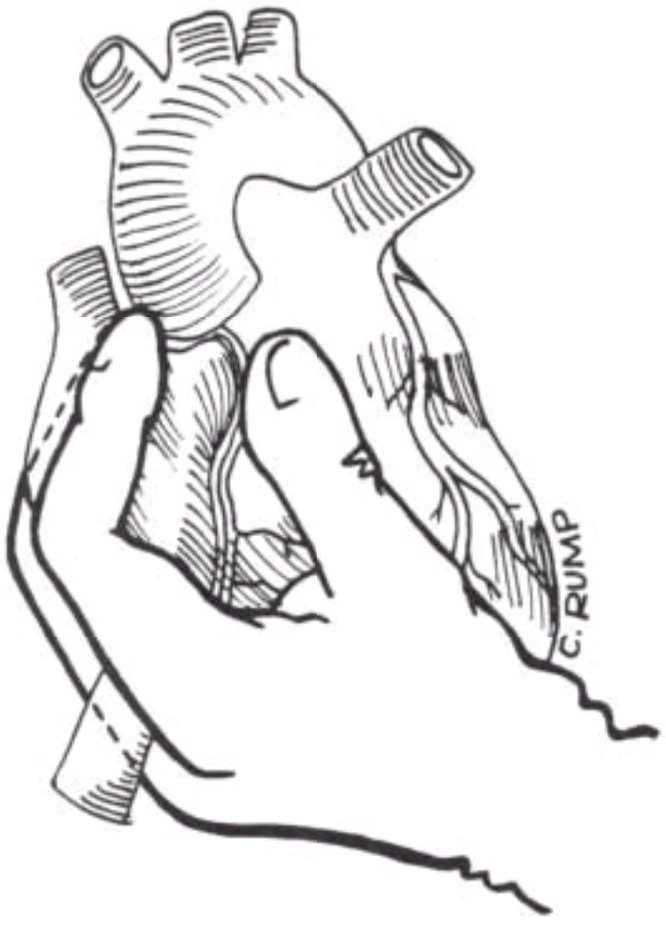


Figure 18-11 Serial illustration. Gentle traction on an inflated Foley catheter will control hemorrhage and allow easy repair. The balloon is inflated with saline, and care is taken to avoid rupturing the balloon with the suture needle. This technique is particularly useful with injuries of the inferior cavoatrial junction, with posterior wounds, and during cardiac massage. Volume loading can be obtained by infusion of blood or crystalloid solutions through the lumen of the catheter. Care should be taken to avoid an air embolus through the lumen of the catheter during placement.

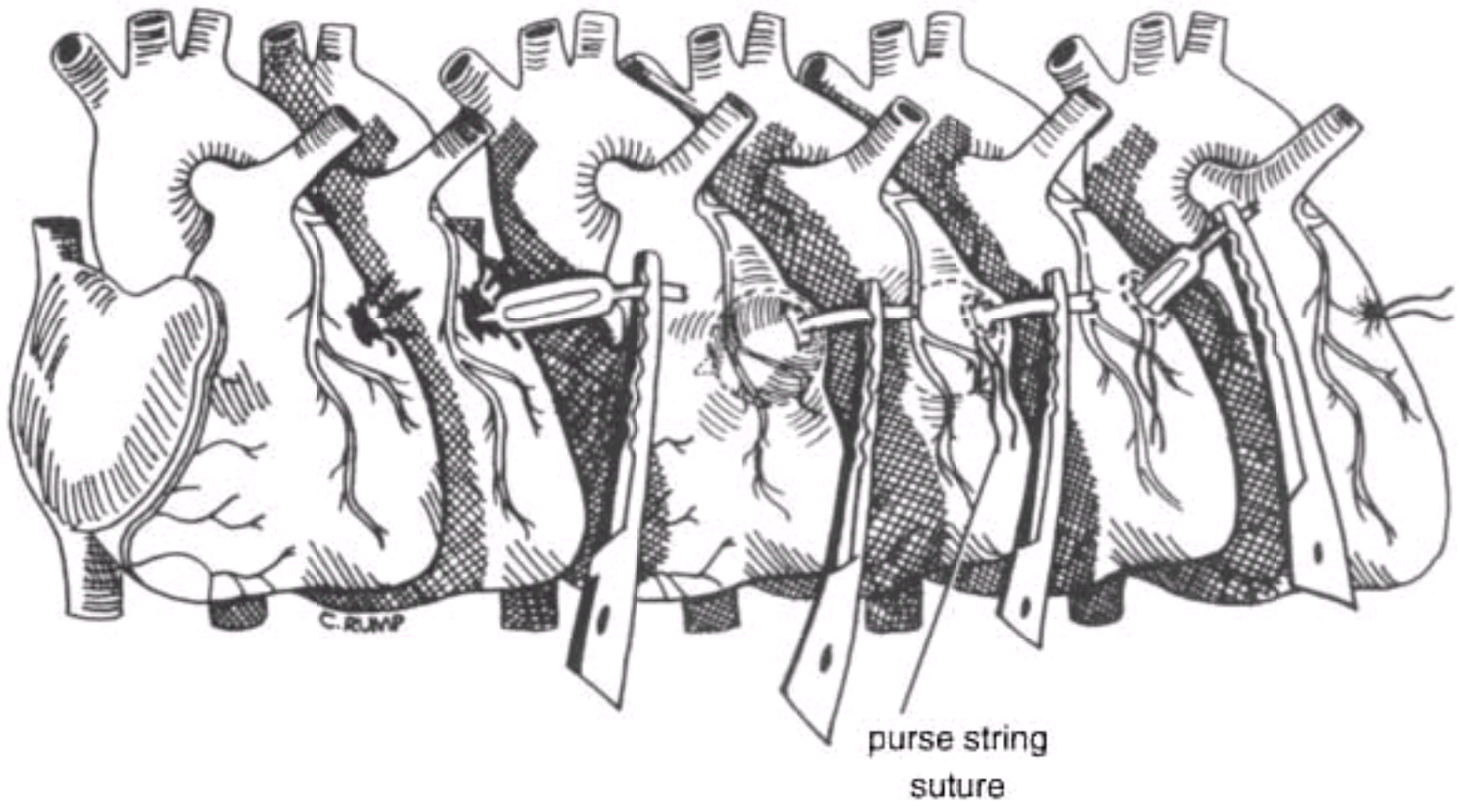


Figure 18-12 Technique for elective fibrillation: 20 J (watt-sec) is delivered through internal defibrillating paddles placed perpendicular to the epicardium. Coronary vessels should be avoided during paddle placement.

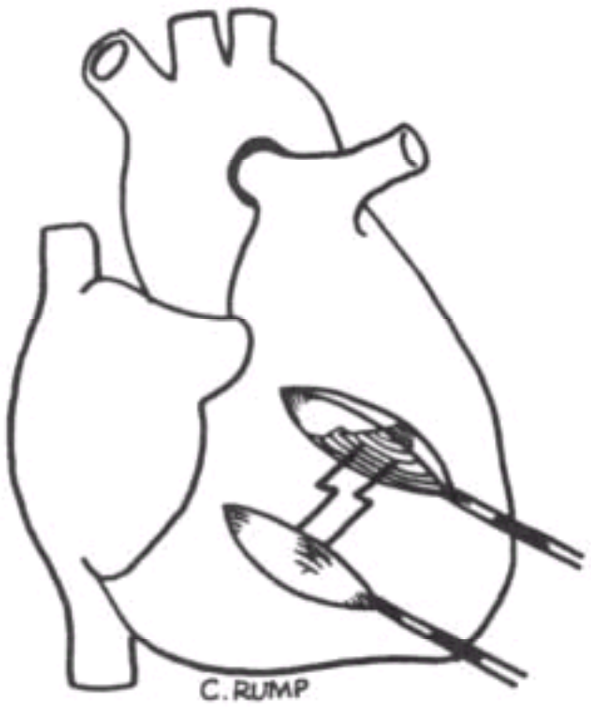


Figure 18-13 Use of a partial occluding clamp in different locations for control of bleeding and subsequent repair.

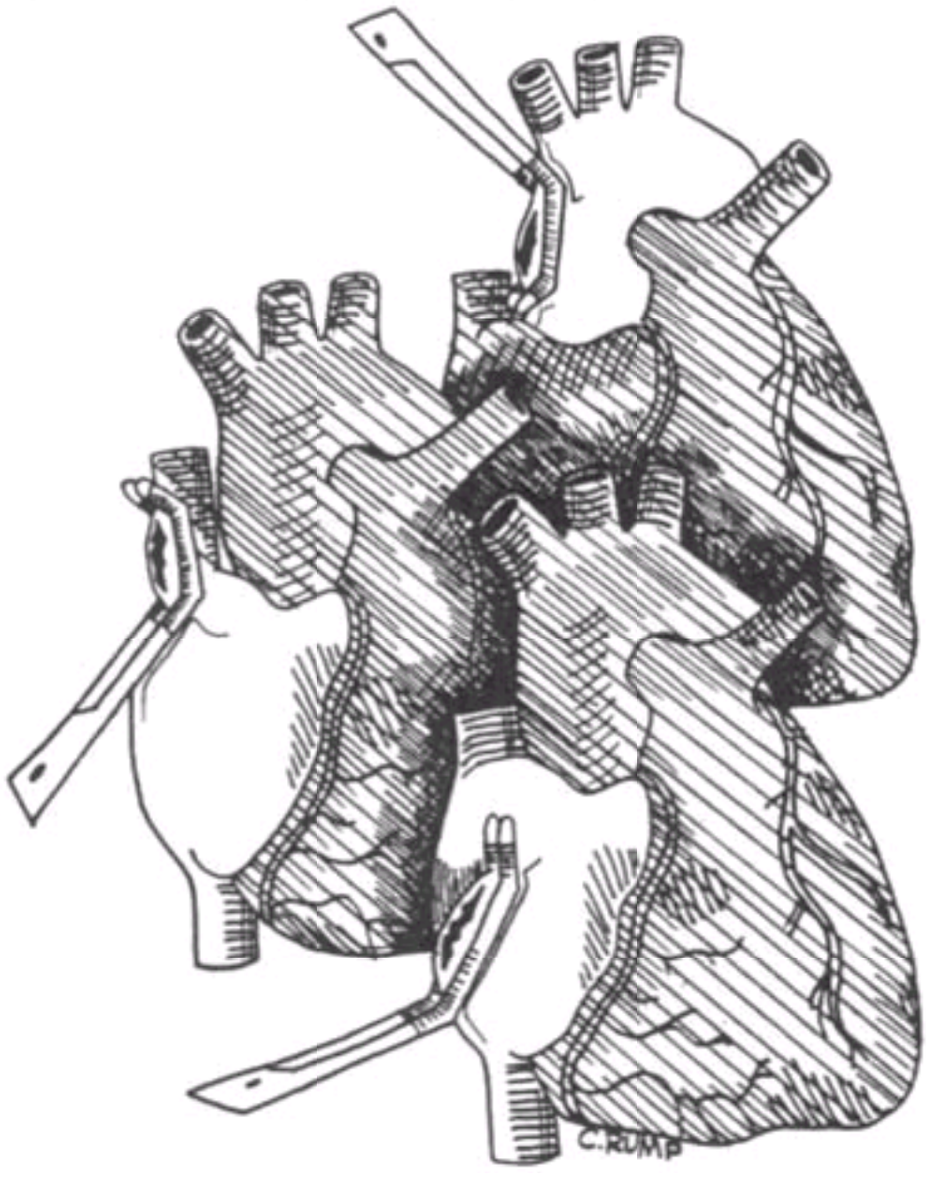


Figure 18-14 Wounds of the inferior cavoatrial junction are difficult to manage with simple vascular clamping. *A–D,* Use of a Foley catheter provides satisfactory control.

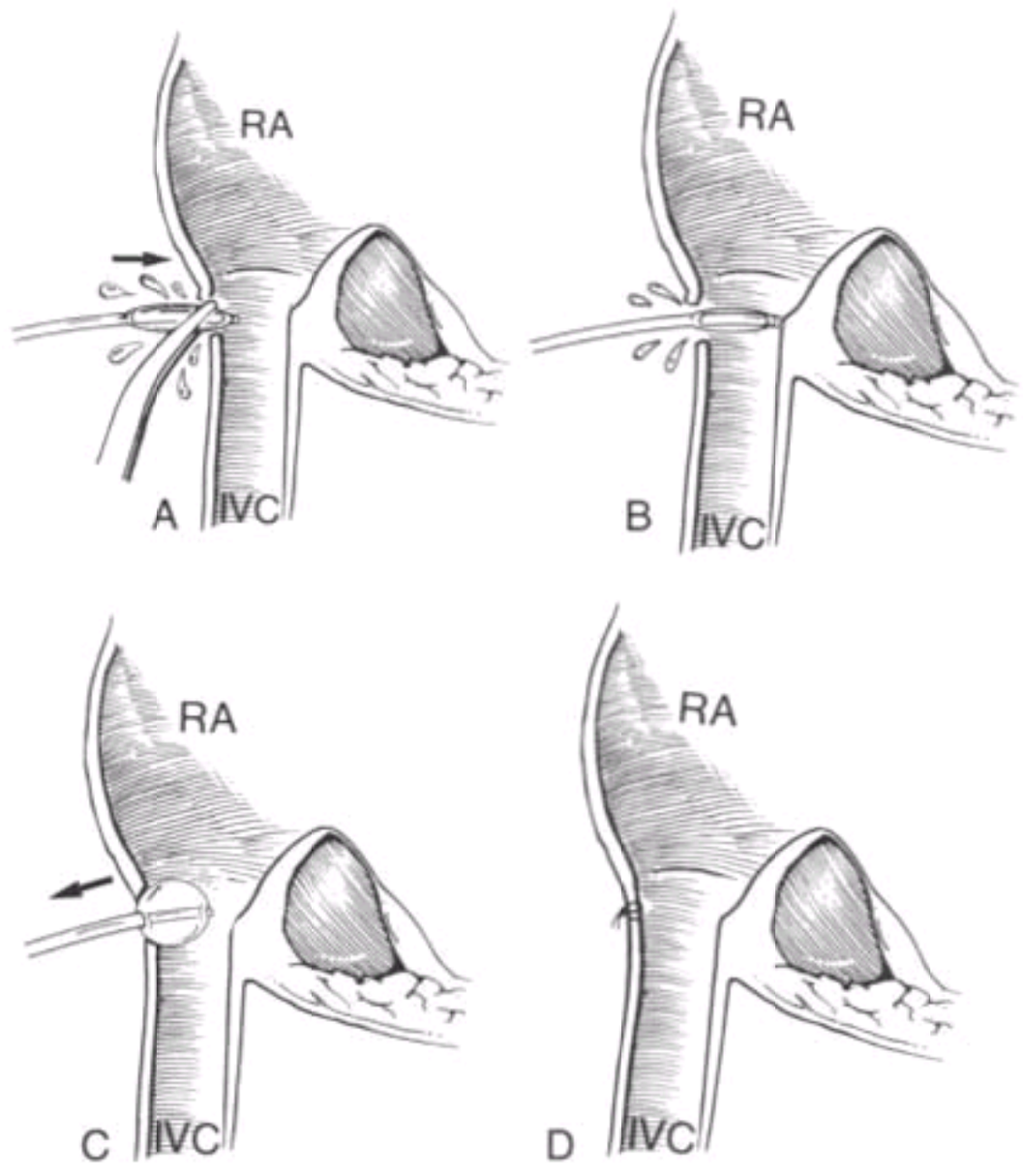


Figure 18-15 Cross-clamping for control of subclavian bleeding is difficult and time-consuming. Compression with laparotomy pads in the apical pleura from below and the supraclavicular fossa from above will control hemorrhage while the patient's condition is stabilized and the patient is transported to the operating room.

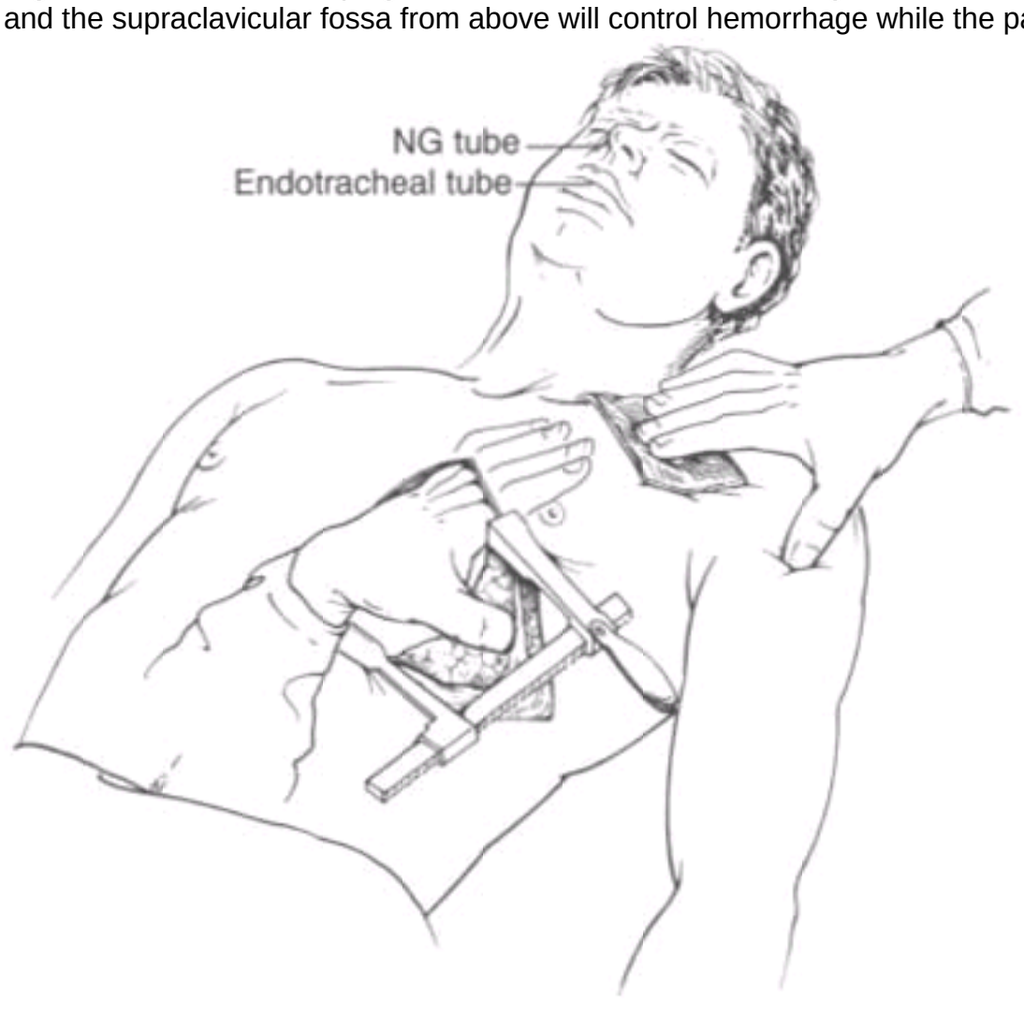


Figure 18-16 *A*, Modified umbilical cord clamp. *B,* Modified umbilical cord clamp securing a catheter in the right atrial appendage.

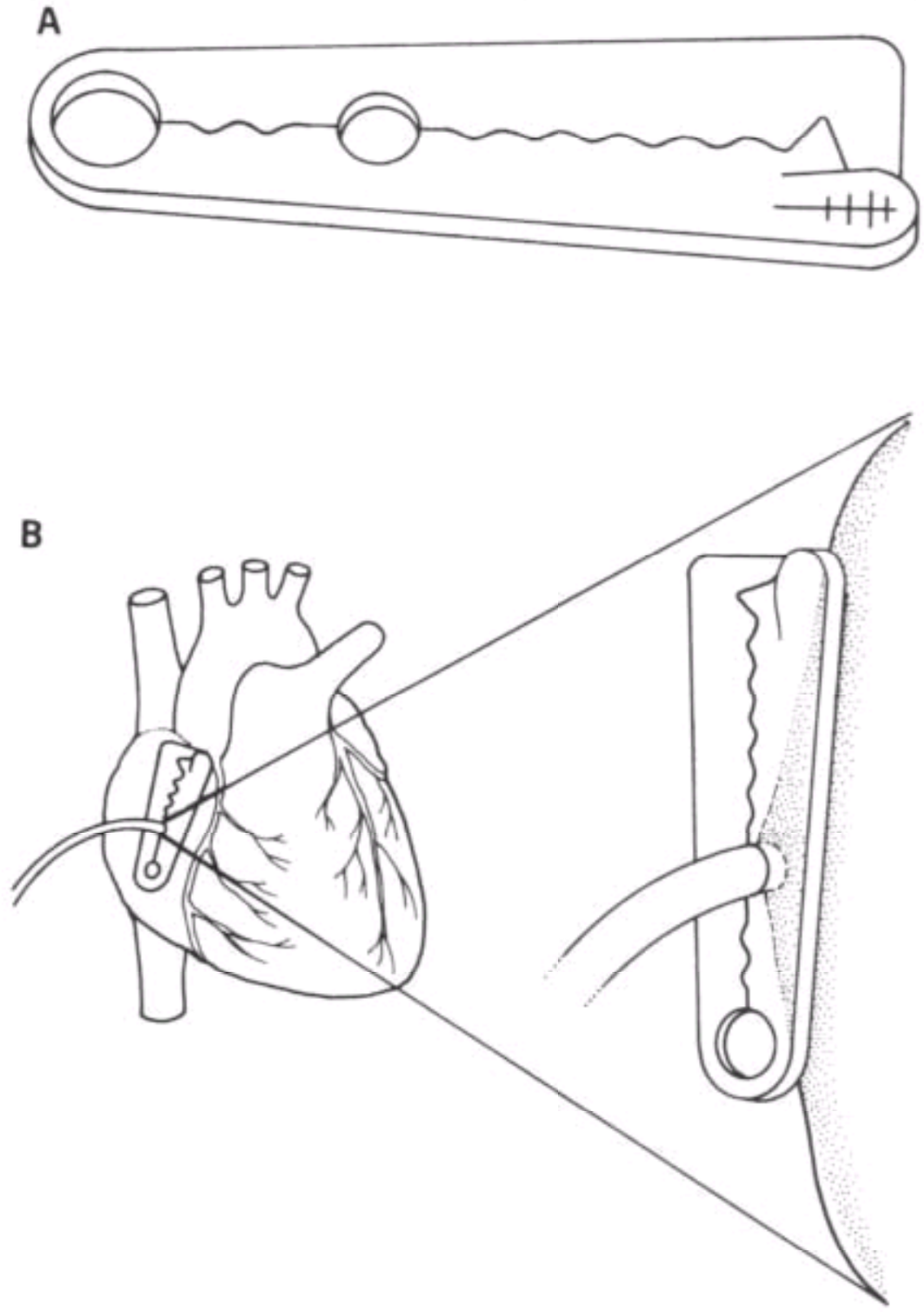


Figure 18-17 Manual cardiac massage and cross-clamping of the aorta to increase coronary and cerebral perfusion selectively.

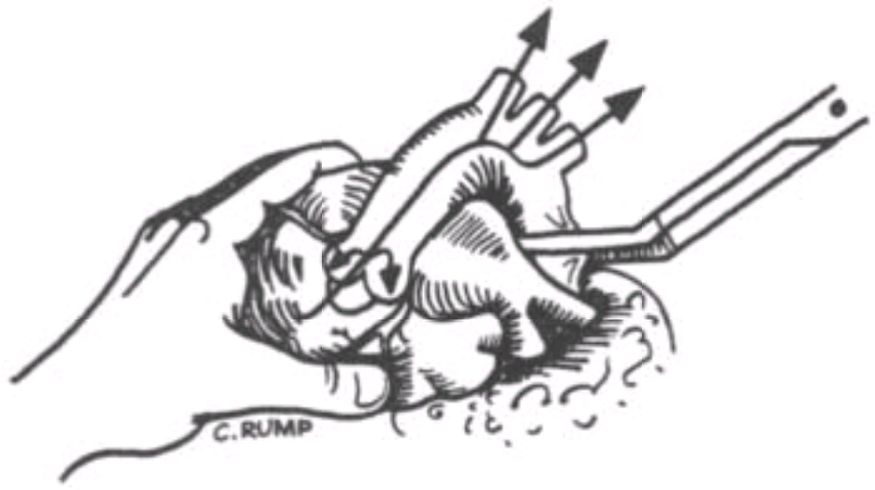


Figure 18-18 Traumatic rupture of the aorta. Three clamps are required for control. Back-bleeding will occur if fewer than three clamps are used.

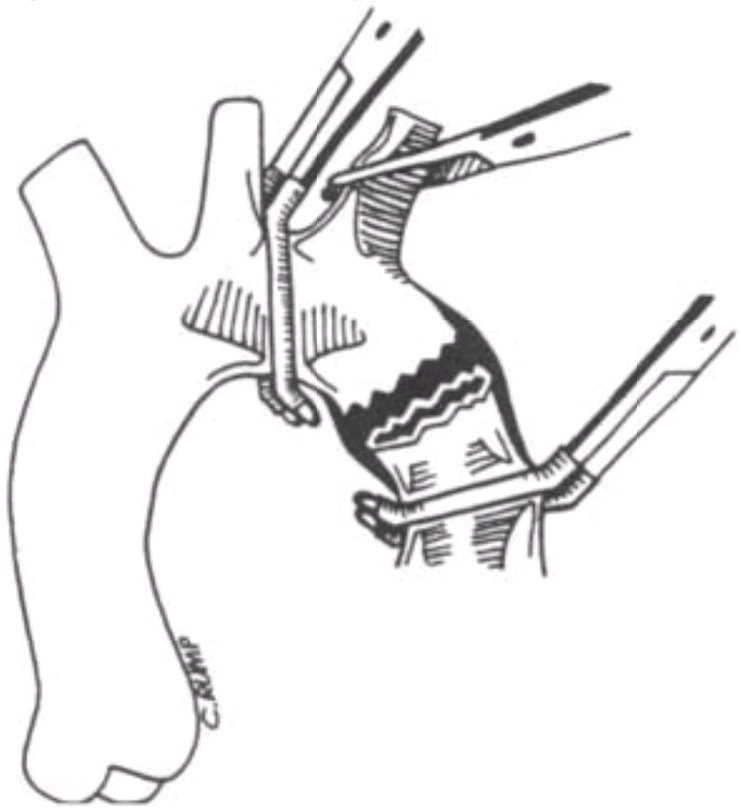


Figure 18-19 Adequate exposure of the descending aorta may require division of the inferior pulmonary ligament.

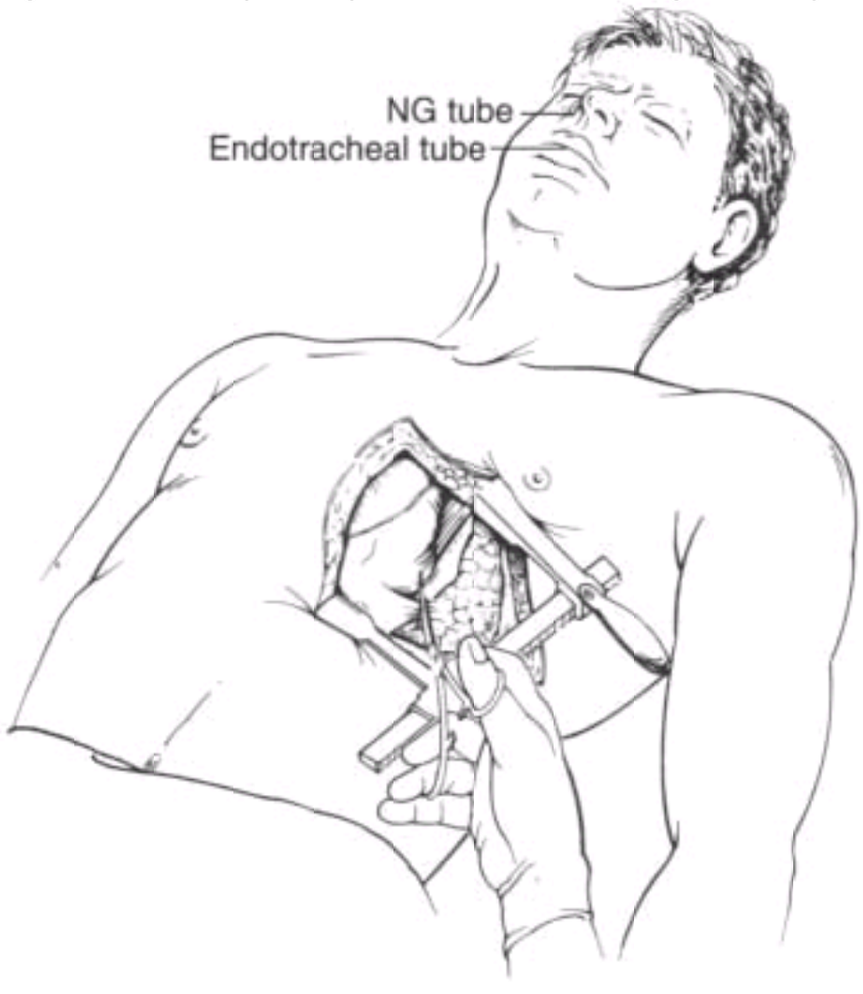
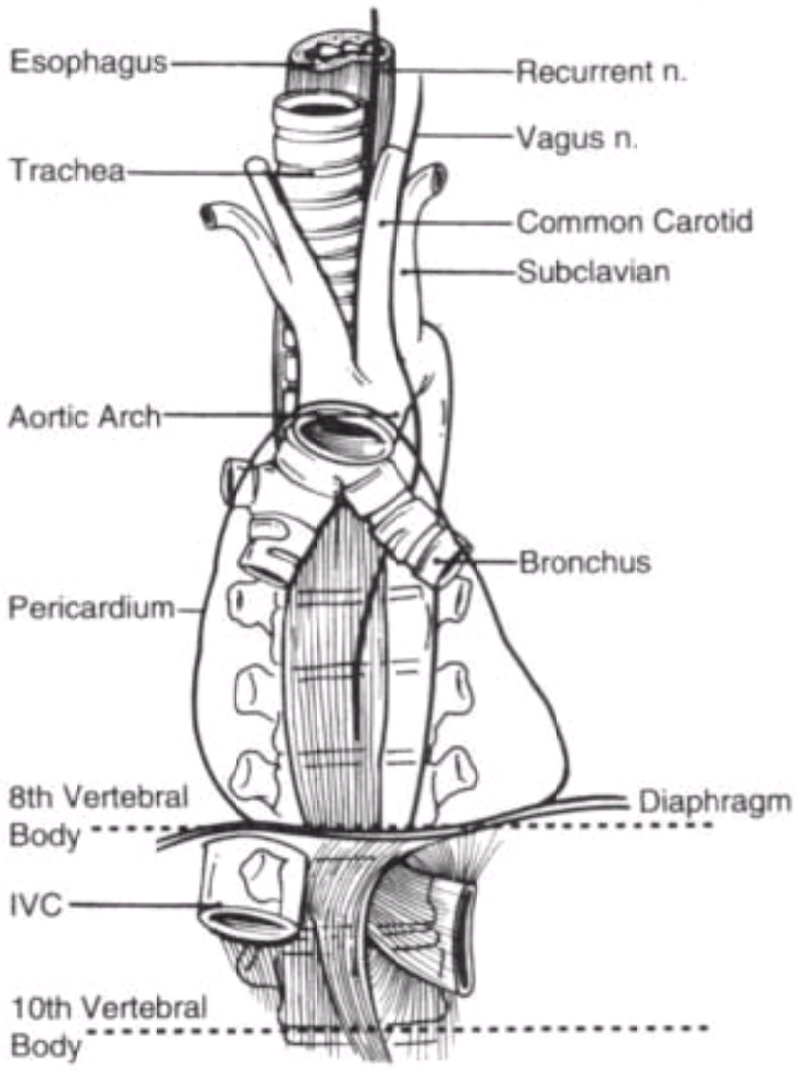


Figure 18-20 *A*, Identification of the aorta: in the posterior mediastinum the aorta lies directly anterior to the vertebral bodies. The esophagus is anterior and slightly medial to the aorta. In the lower thorax, both are covered on the anterolateral surface by mediastinal pleura, which must be dissected prior to isolating the aorta for cross-clamping. *B,* Aortic cross-clamping: Using blunt dissection, one spreads the pleura above and below the aorta. The vessel should be fully mobilized and clearly separated from the esophagus before clamping. It may be difficult to differentiate the aorta from the esophagus. The aorta is the more posterior structure and is in contact with the vertebral bodies. Passage of a nasogastric tube from above will often aid the rapid identification of the esophagus.



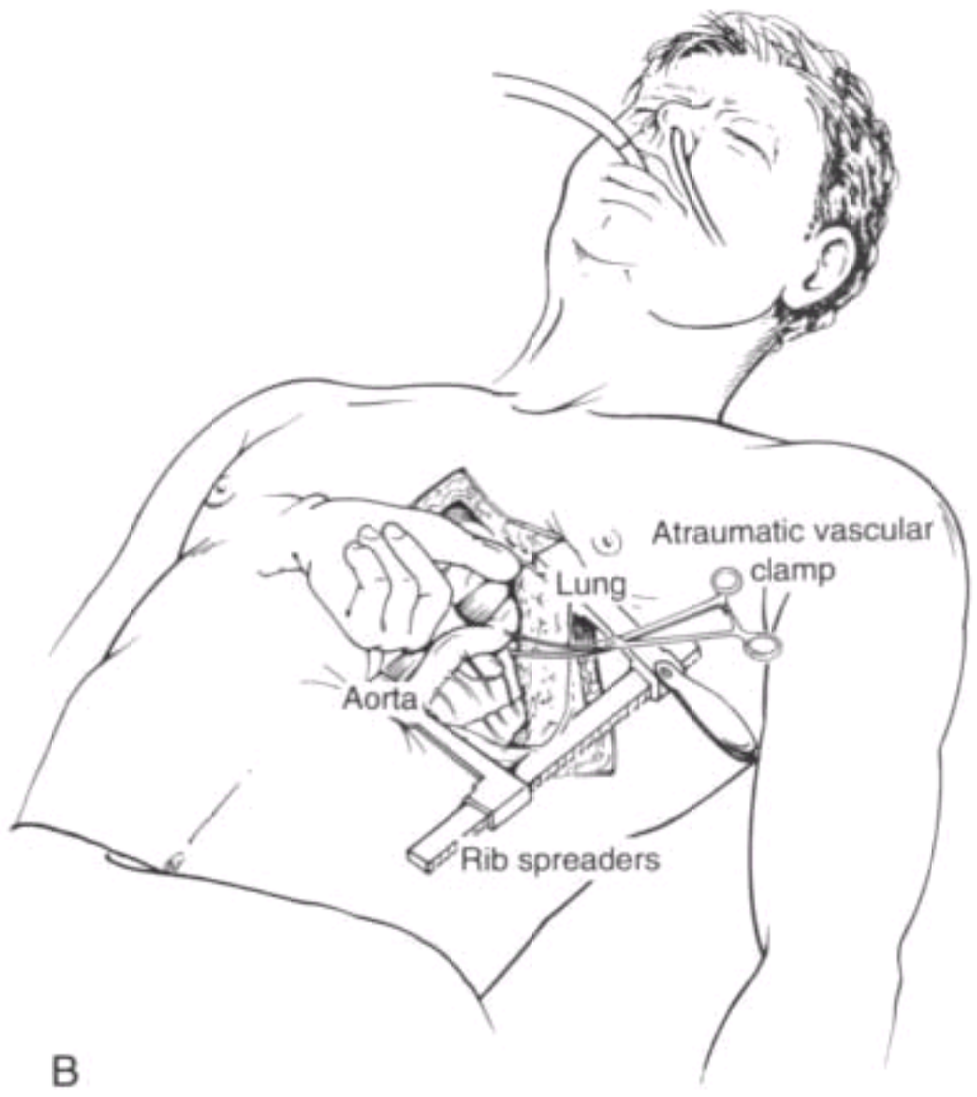


Figure 18-21 The use of a Conn aortic compressor is the method of choice for aortic occlusion because it is fast, does not interfere with the operative field, and is associated with minimal risk of injury. Alternatively, the more awkward technique of direct digital occlusion can be used. *(Courtesy of Pilling Company, Ft. Washington, PA.)*

Figure 19-1 Acceptable sites for heel stick puncture illustrated by shaded areas. *(Adapted from the Textbook of Pediatric Emergency Procedures; Henretig FM, King C [eds]. Philadelphia, Williams & Wilkins, 1997.)*

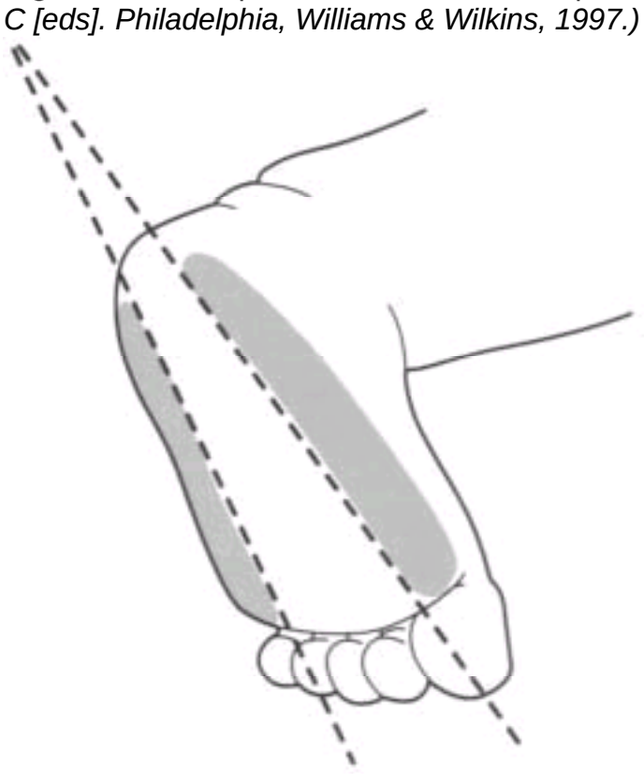
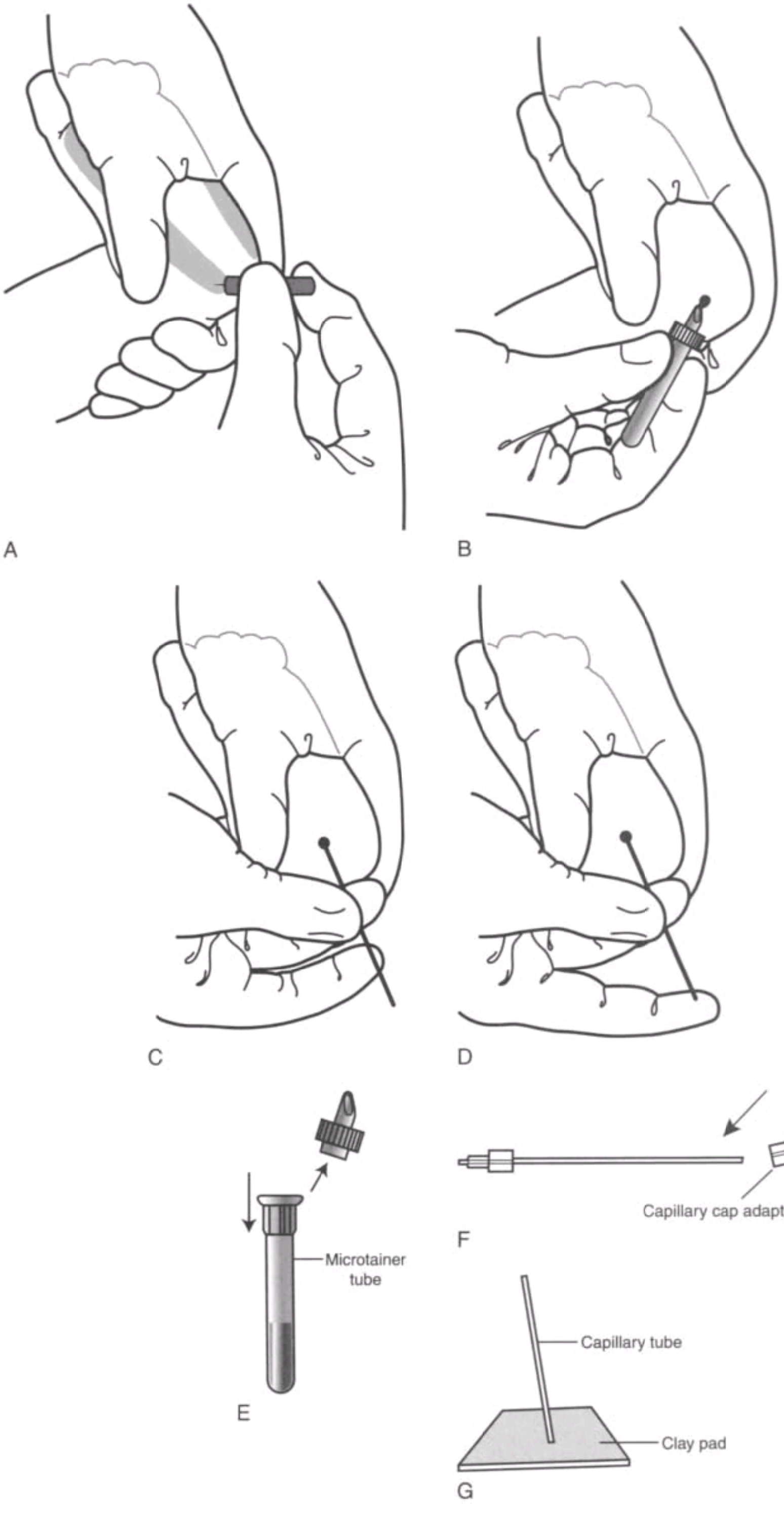


Figure 19-2 *A*, The heel stick is performed on the lateral or medial aspect of the heel. *B*, The collector end of the Microtainer is touched to the drop of blood, and blood is allowed to flow down the wall of the tube to the bottom; or *C*, a heparinized capillary tube is placed in the drop of blood, and the proximal end of the inverted tube is allowed to fill by capillary action. *D*, The blood is maintained in the capillary tube using the index finger to maintain capillary tension on the end of the tube. *E*, When using a microtainer tube, the Microtainer is capped. *F*, when using a capillary tube, the ends are capped or (*G*) sealed with wax or clay. Avoid squeezing the foot, and keep the proximal end of the Microtainer below the puncture site. *(Adapted from the Textbook of Pediatric Emergency Procedures; Henretig FM, King C [eds]. Philadelphia, Williams & Wilkins, 1997.)*



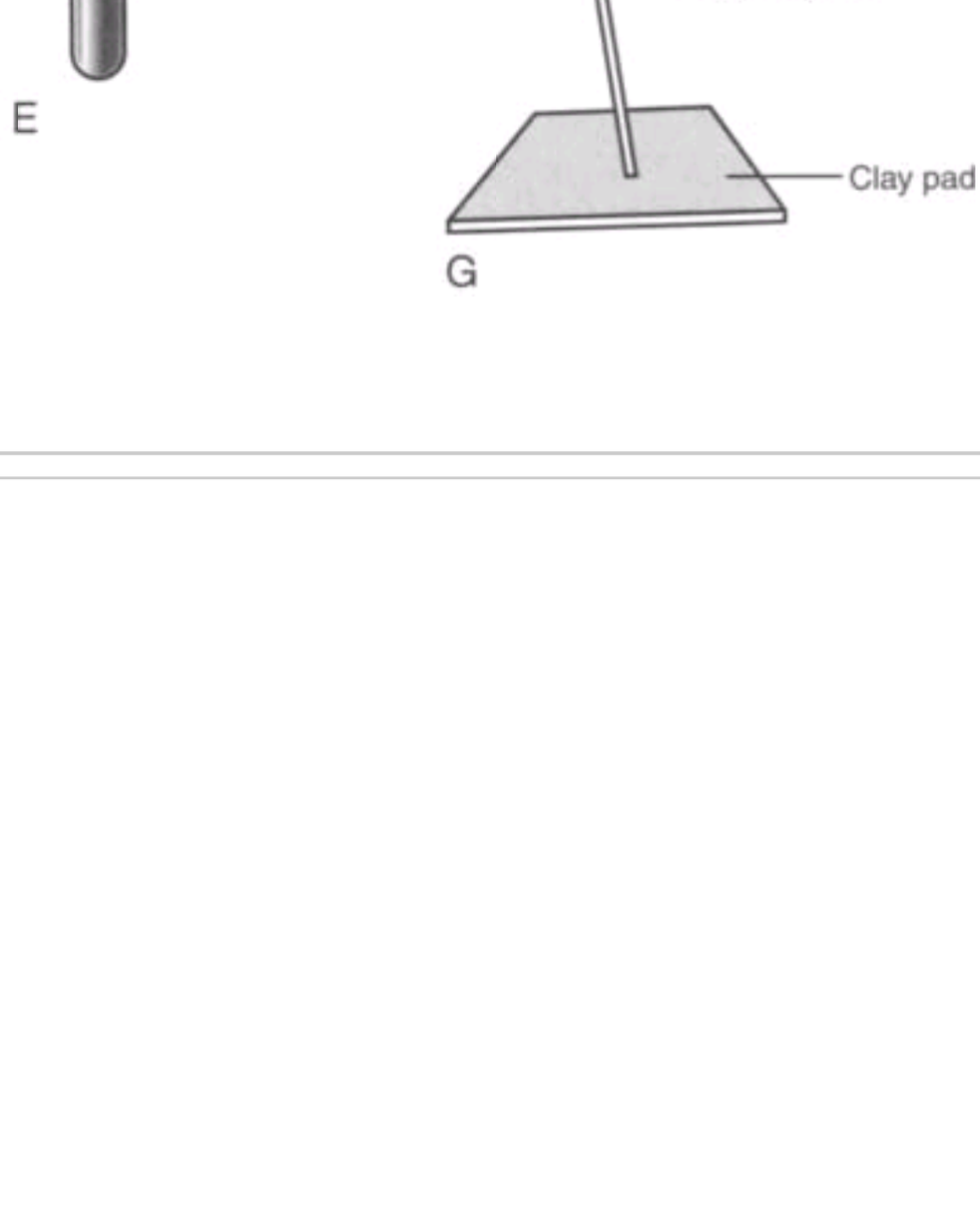

E
Clay pad
G

Figure 19-3 Venous access sites in the neonate and young infant. If venous access is unavailable, arterial blood may be used for most laboratory tests, including blood cultures.

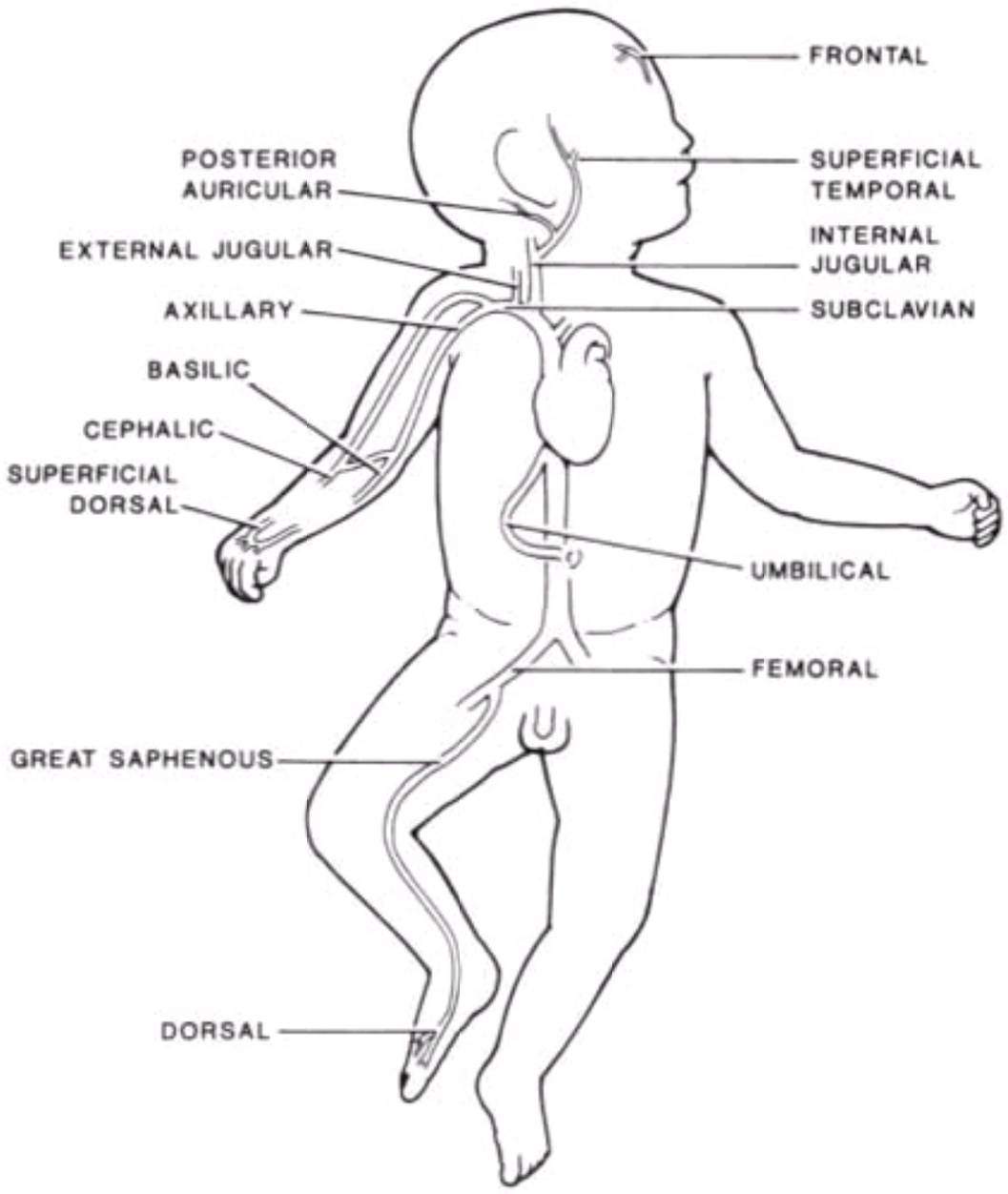


Figure 19-4 *A*, Technique for obtaining blood by antecubital venipuncture with a butterfly needle and a syringe. Once blood is obtained, the butterfly needle may serve as an infusion site. Note that this procedure often requires two persons to carry it out—one to hold the arm and insert the needle and the other to aspirate the blood. *B*, As an alternative to a syringe, a Vacutainer system may be used to apply suction. The Vacutainer needle punctures the sealed end of the butterfly set. Use of this method helps to prevent the premature clotting of blood that may occur if there is a delay in filling the collection tubes.

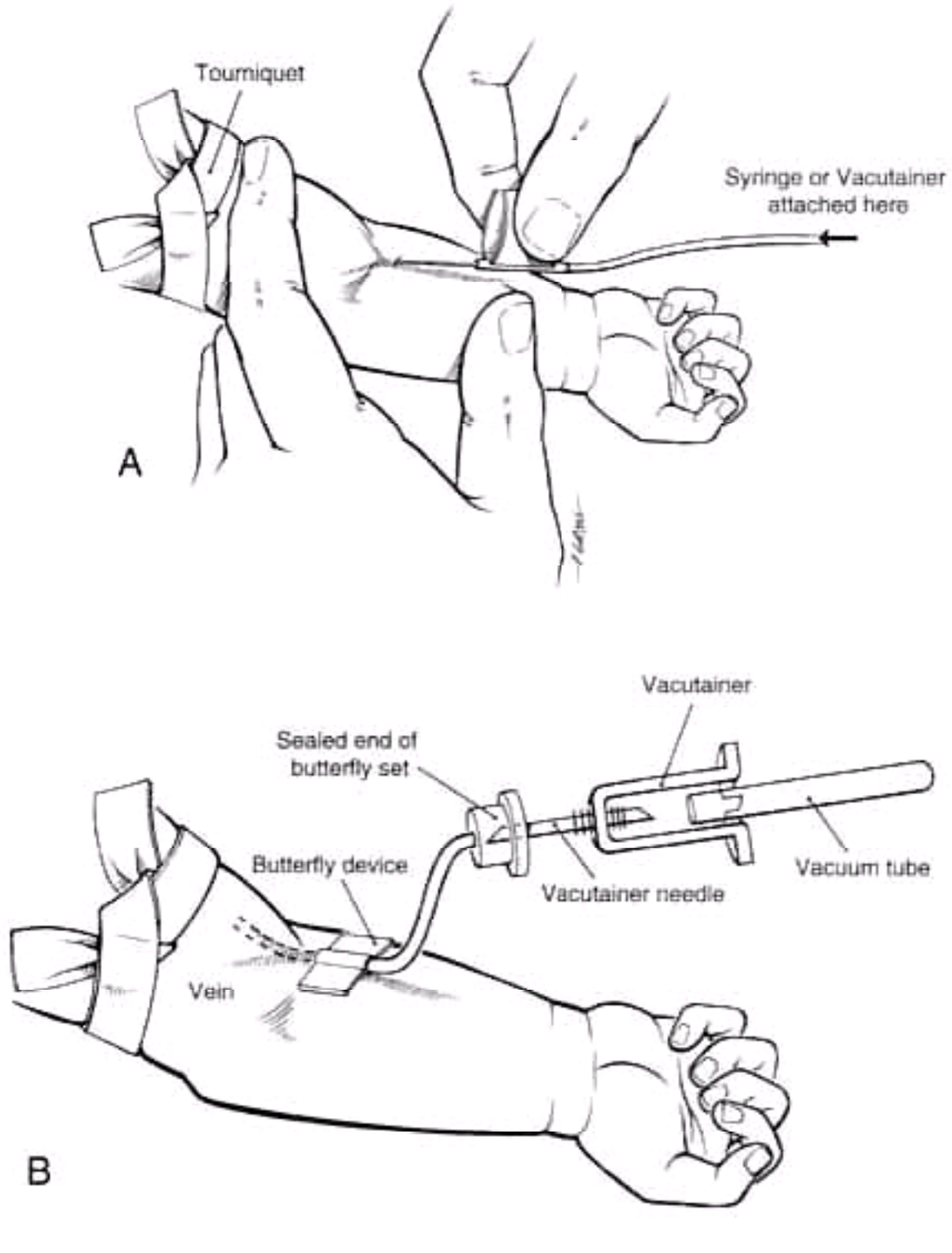


Figure 19-5 External jugular venipuncture. A syringe or a butterfly needle may be used. Venous distention is aided when an assistant's finger occludes the vein, or when the infant cries. The neck is extended, either over the side of the bed or by placing a rolled towel under the shoulders. This procedure requires two persons. Gloves should be worn.

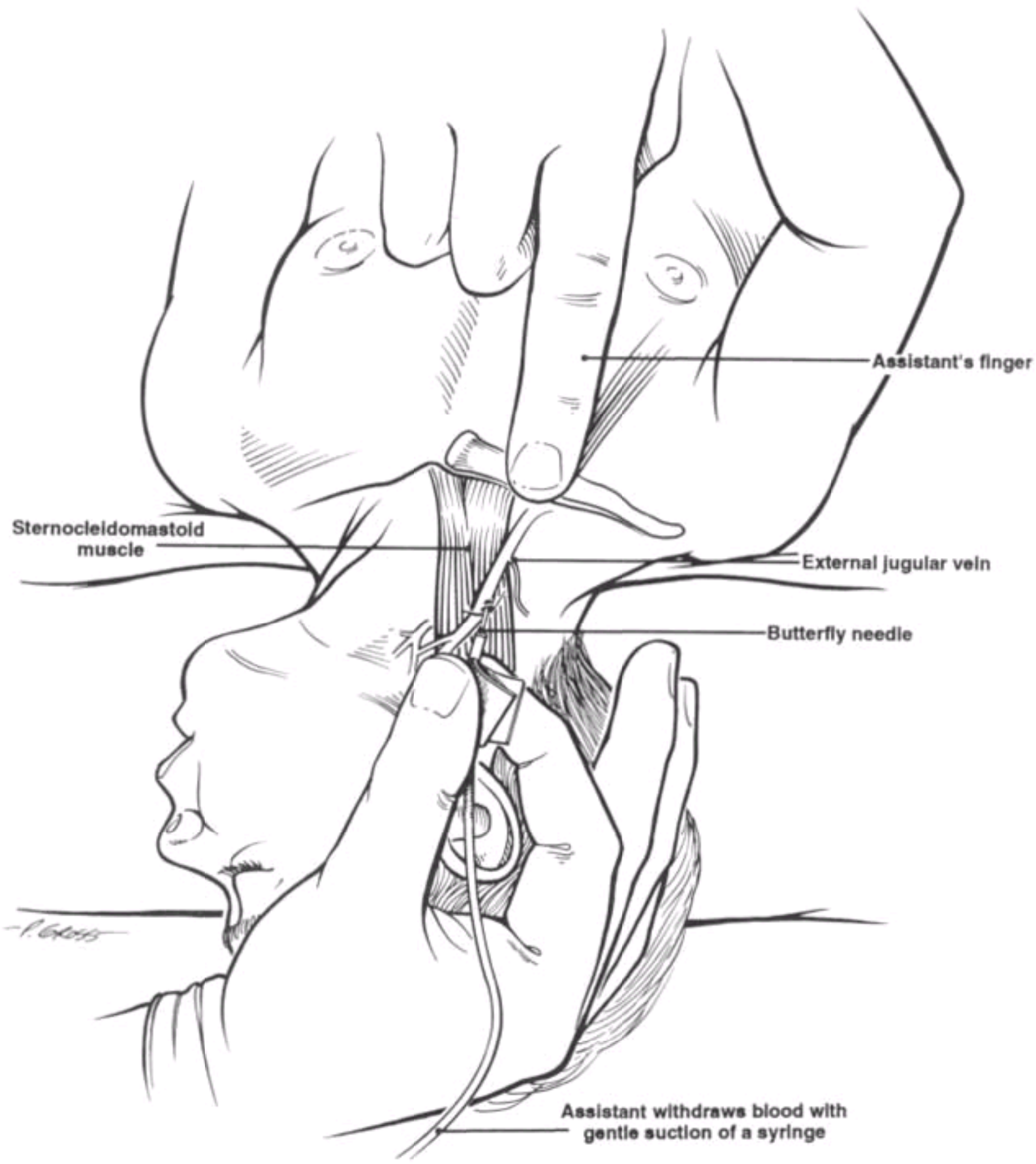


Figure 19-7 Anatomy of the volar surface of the wrist and the palm. The radial artery is preferred for sampling procedures.

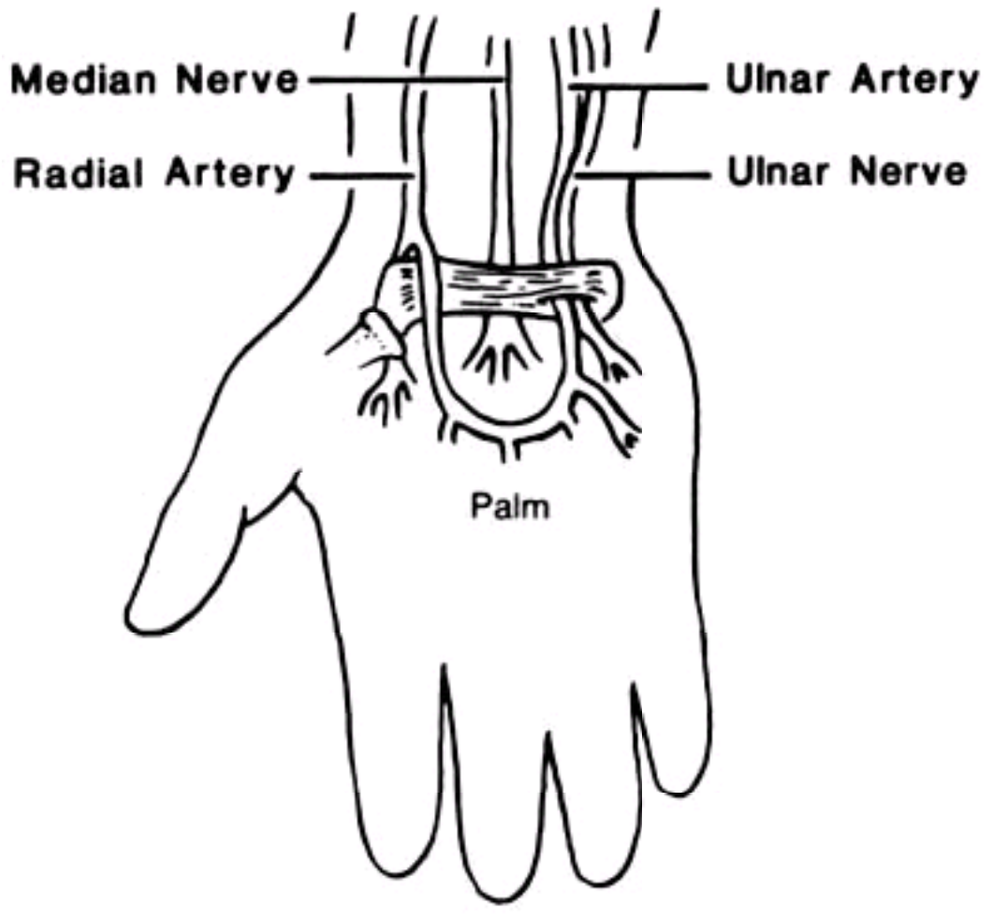


Figure 19-8 For arterial blood sampling, the needle should be inserted under the skin at a 30° to 45° angle. A butterfly needle and syringe are used if larger volumes of blood are required. The wrist is held dorsiflexed by the nondominant hand.

Figure 19-9 Resistance met during passage of the blood gas needle usually indicates contact with bone. The needle should be withdrawn slowly. If the needle has traversed both walls of the artery, blood will be obtained as the needle is slowly withdrawn into the arterial lumen.

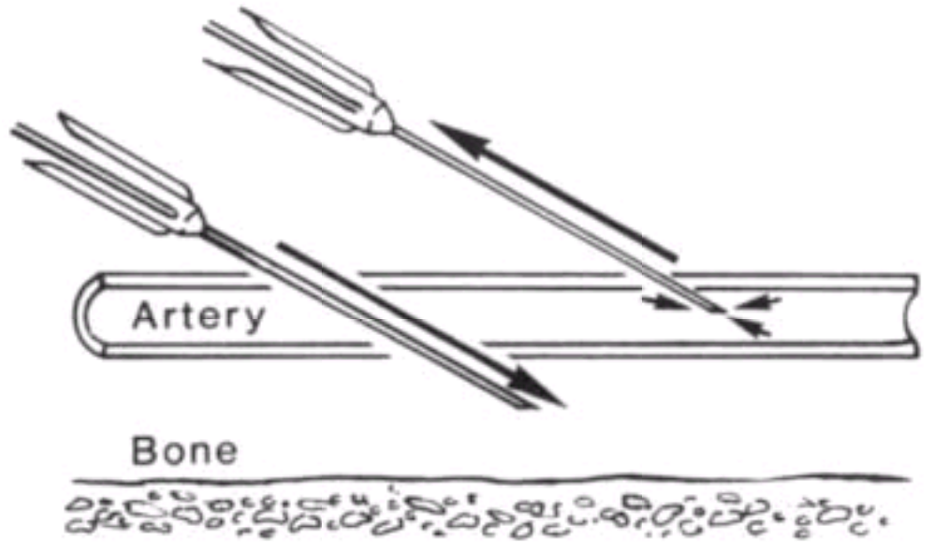


Figure 19-10 Technique for peripheral venous catheterization. *A*, The catheter is directed at a 10° to 20° angle toward the insertion site and advanced until blood return is seen in the catheter and hub. The stylet is removed, and the T-extension tubing is attached. *B*, Taping technique for butterfly and intravascular catheters using a crisscross pattern. *C*, Hand and forearm secured to an arm board. *D*, Covering with the plastic wrapper from the T-extension tubing for protection.

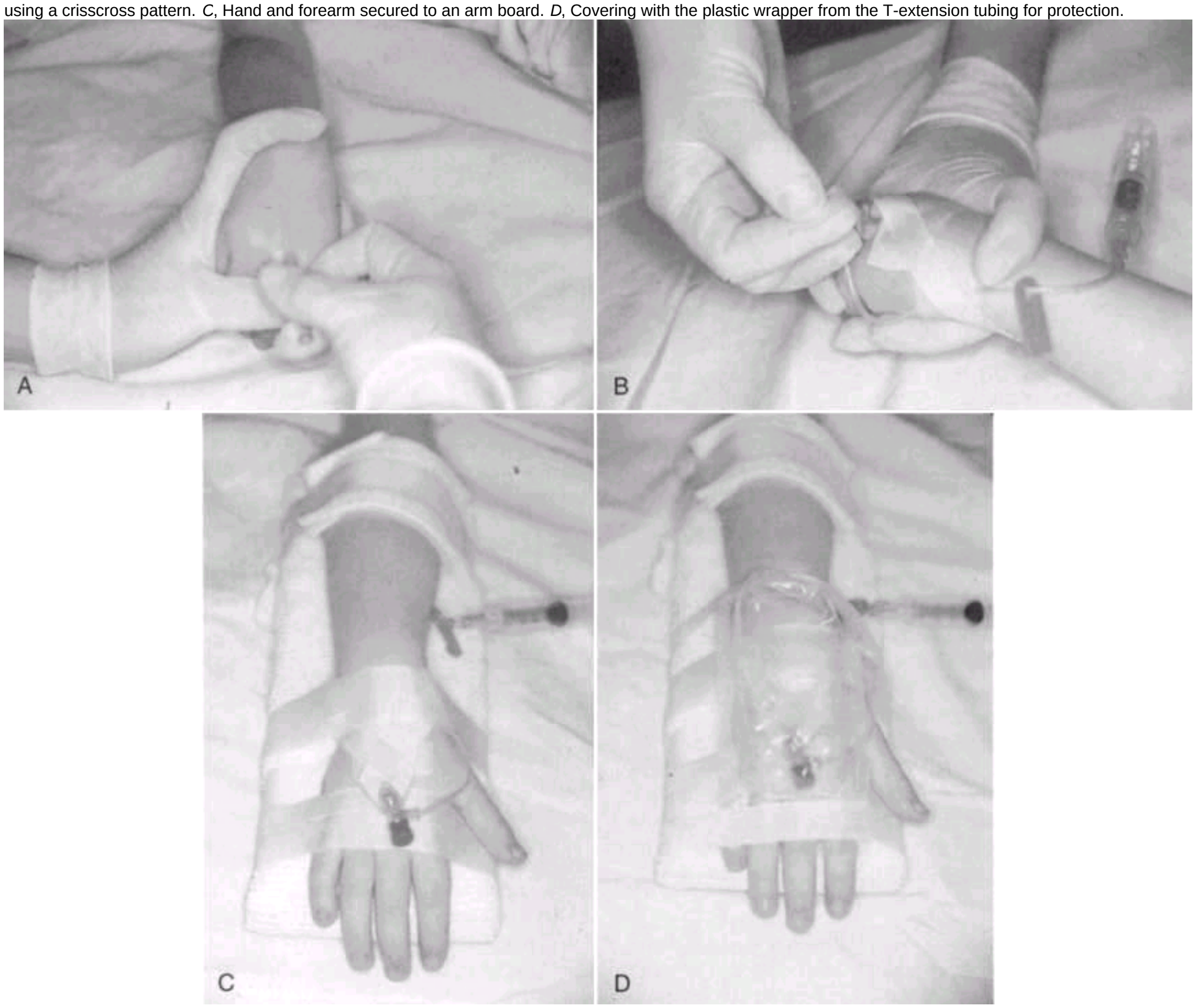


Figure 19-11 Using a rubber band as a tourniquet to distend the scalp veins, the needle is introduced approximately 0.5 cm distal to the anticipated site of the vessel puncture. Gloves should be worn.

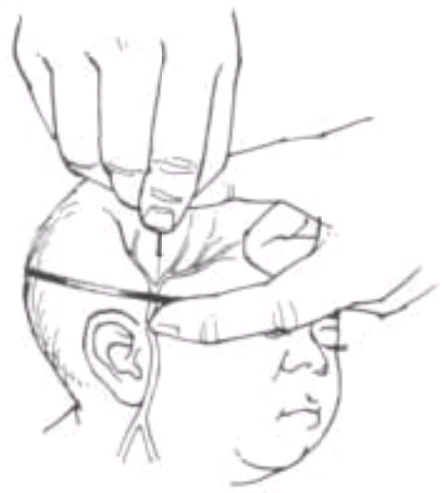


Figure 19-12 Protecting the intravenous line with a plastic medicine cup.

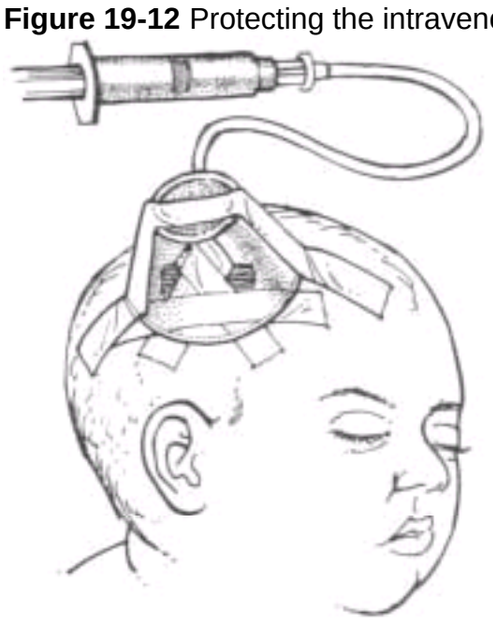


Figure 19-13a Venous cutdown (saphenous vein). *A*, Immobilization of the ankle and the site of skin incision. *B*, A curved hemostat scoops up the vein. The point of the hemostat should be kept against the bone. *C*, The vein is dissected free. *D*, With a proximal and distal tie to stabilize the vein and control bleeding, an incision is made in the upper one third of the vein. *E* and *F*, The infusion catheter is threaded into the vein lumen and advanced.

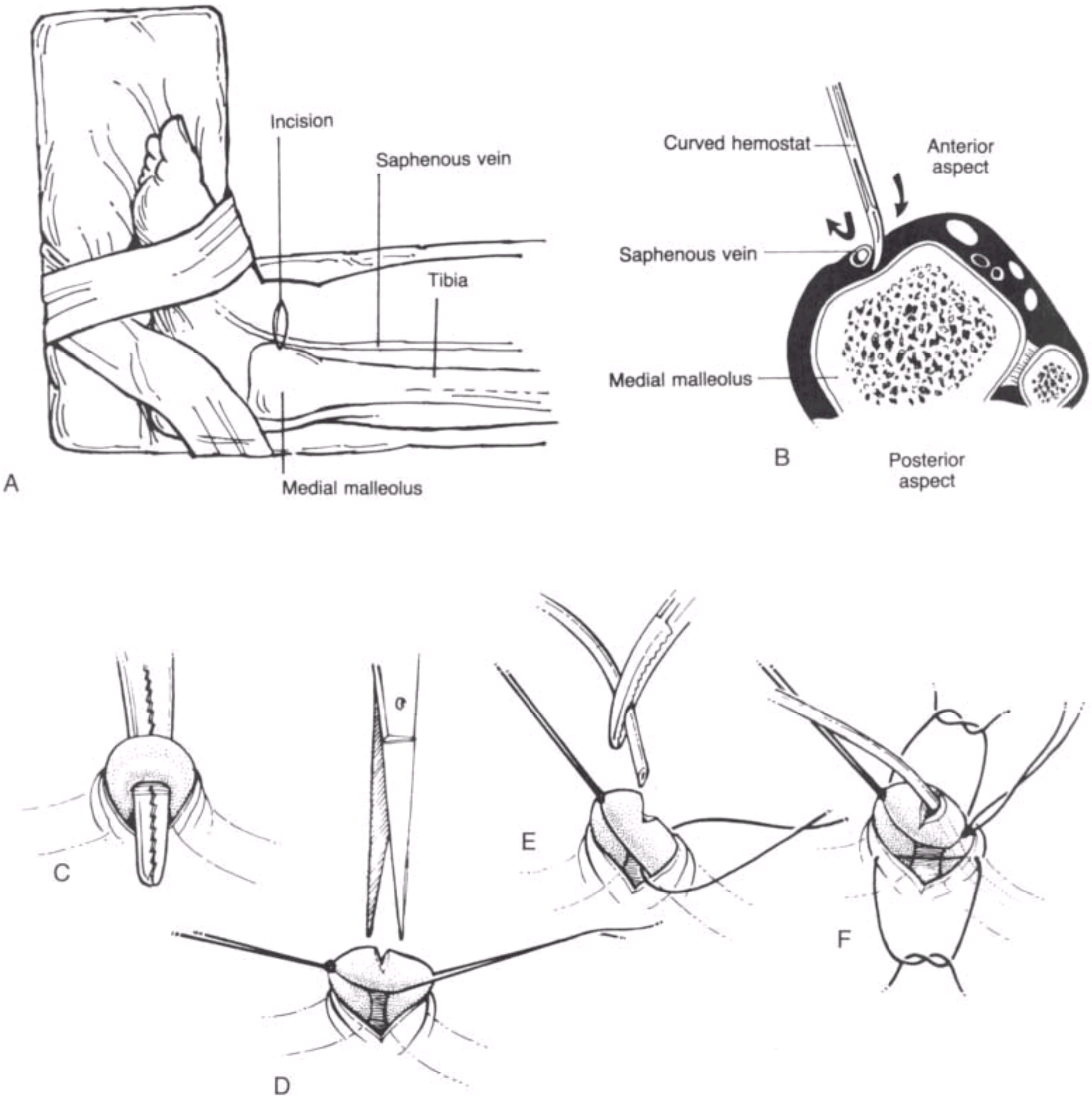
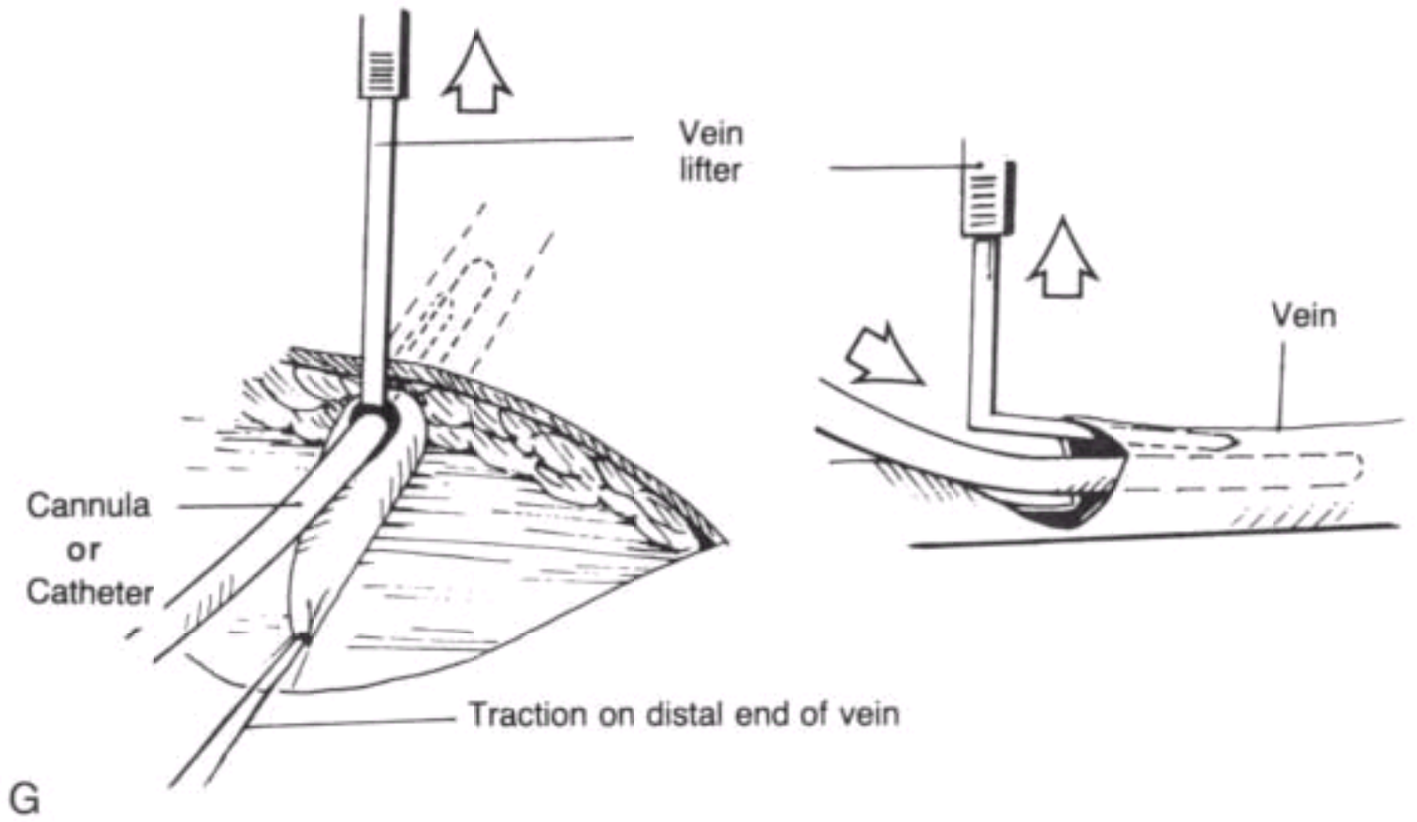


Figure 19-13b *G*, A vein lifter/dilator facilitates placement of the catheter into the vein lumen. *H*, The incision is sutured, and the catheter is secured. *(C from Suratt PM, Gibson RS: Manual of Medical Procedures. St. Louis, CV Mosby, 1982. Reproduced by permission.)*



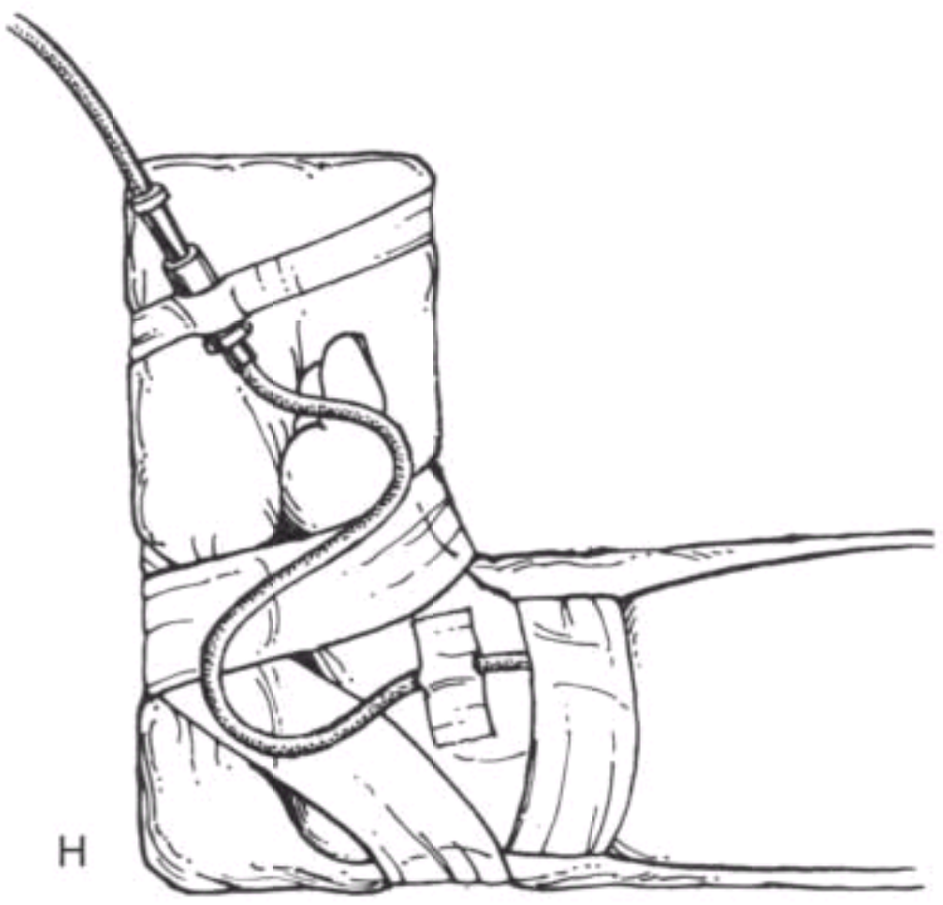


Figure 19-14 The mini-cutdown procedure using a standard intravenous catheter over-the-needle system is technically easier than the full cutdown and may be preferred in an emergency.

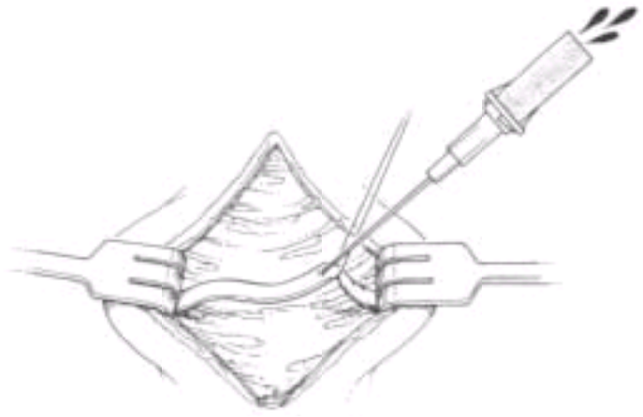


Figure 19-15 Technique for inserting a femoral venous catheter. *A*, A 21-ga butterfly catheter is used to enter the femoral vein, and the guidewire is passed through the butterfly needle into the proximal vein. Note that the tubing has been removed from a standard butterfly set. *B*, A small incision is made alongside the wire, and the dilator is advanced over the wire and into the vein. *C*, The catheter is advanced over the wire and into the vein. *D*, The wire is removed and the catheter secured. Note that many commercial kits have a self-contained 21-ga needle, making modification of a butterfly needle catheter unnecessary.

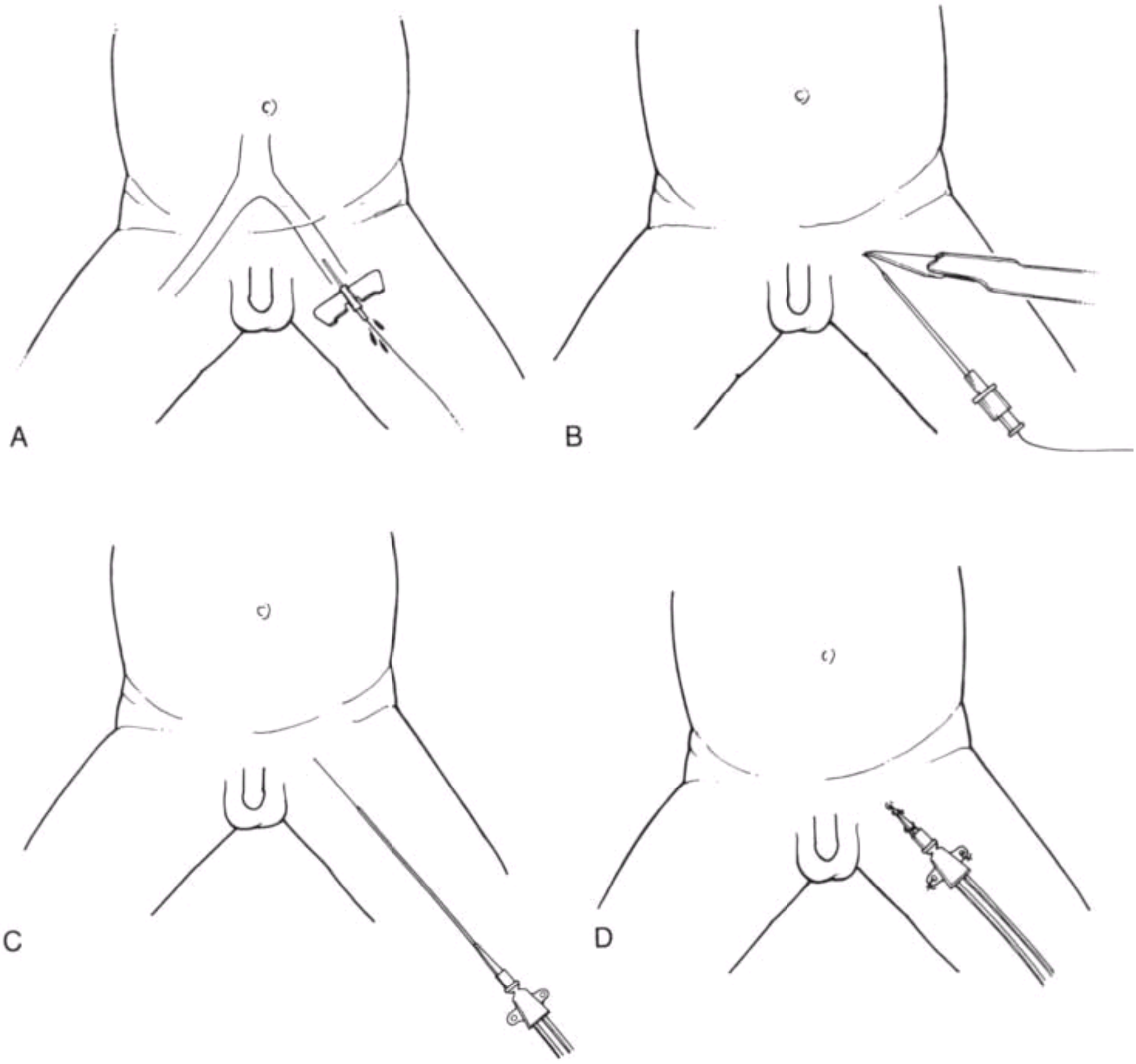


Figure 19-18 Techniques for insertion of central catheters from peripheral veins. *A*, A tape measure is used to determine the catheter length. *B*, Placement of the catheter through the specialized breakaway butterfly-type introducer needle.

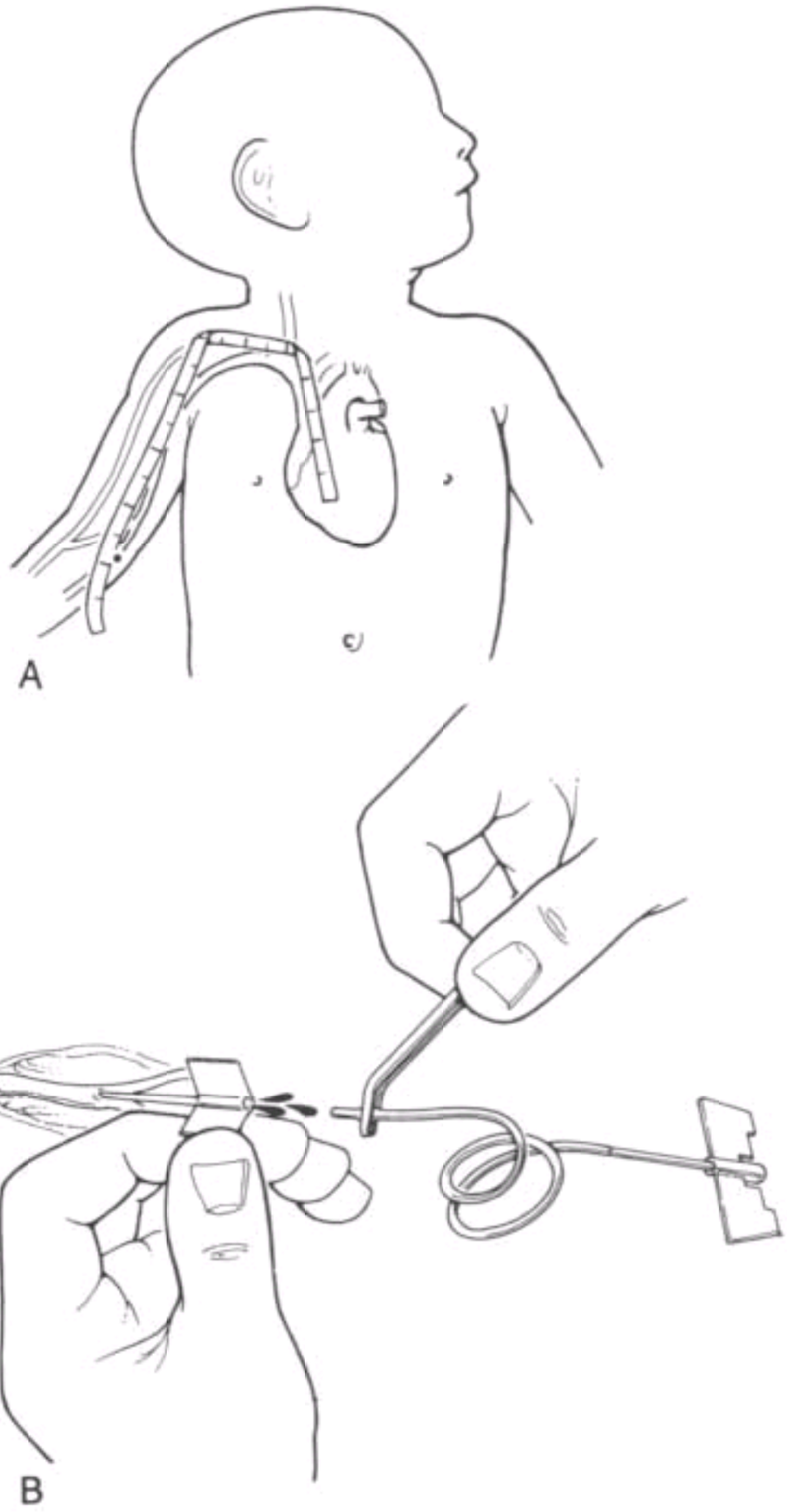


Figure 19-19 When placing an umbilical vein catheter, a pursestring suture or umbilical tape is passed around the base of the cord to provide hemostasis and to anchor the line.

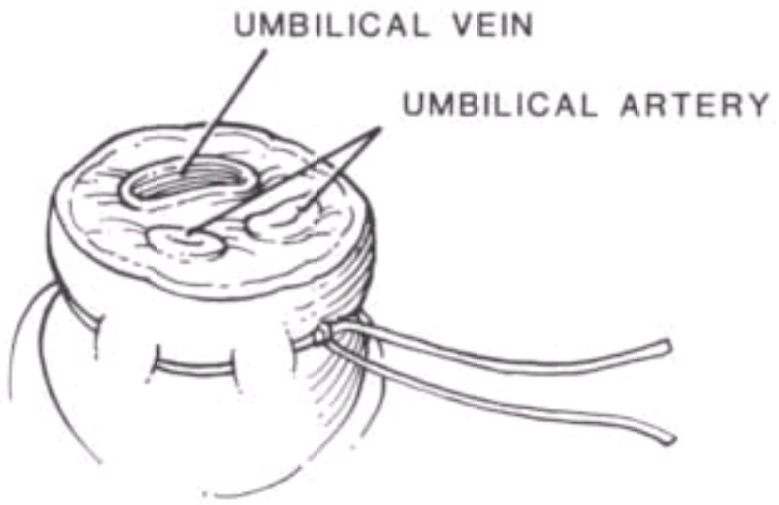


Figure 19-20 An umbilical vein catheter is directed toward the head and remains anterior until it passes through the *ductus venosus* into the inferior vena cava. *(From Ludwig S, Fleisher GR: Textbook of Pediatric Emergency Medicine. Baltimore, Williams & Wilkins, 1994.)*

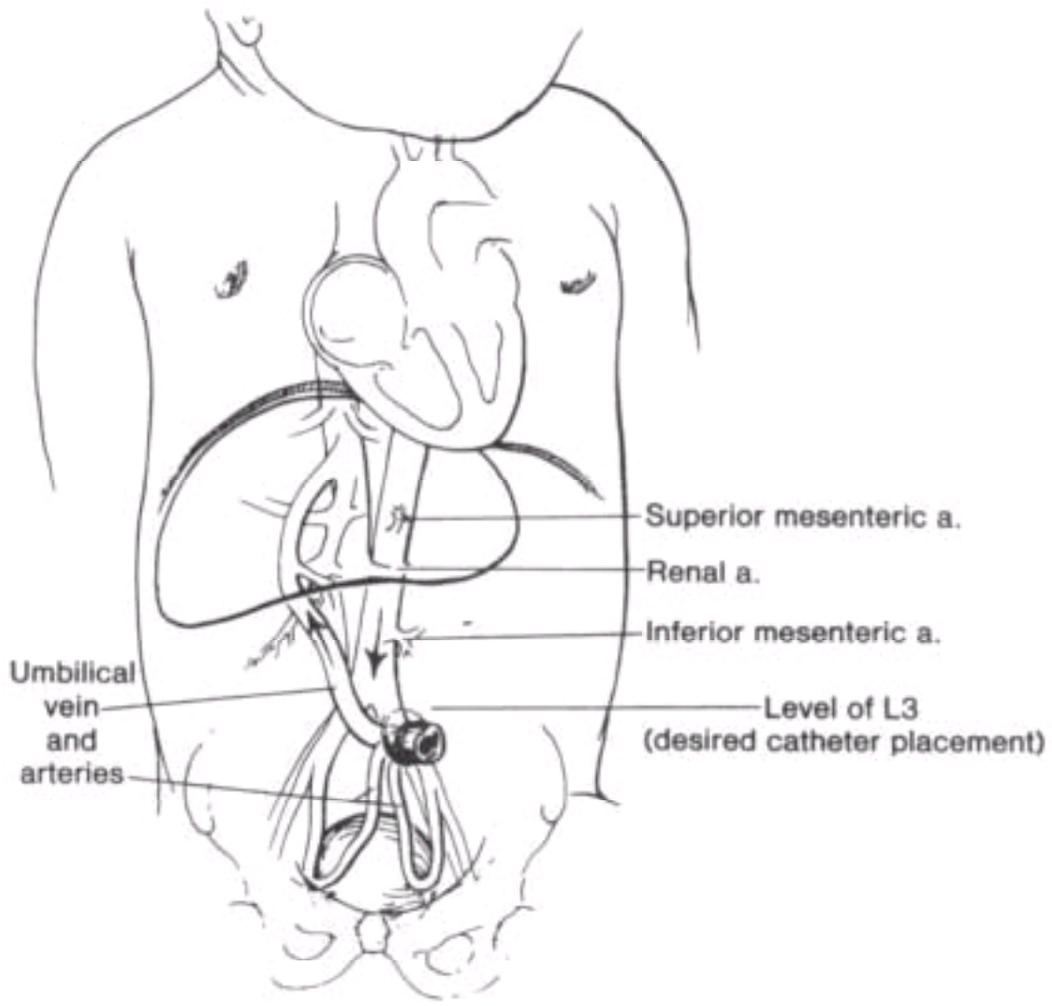


Figure 19-21 After measuring the shoulder-to-umbilicus length, a standardized graph can be used to determine the appropriate length of the umbilical venous catheter (*A*), or umbilical arterial catheter (*B*). The venous catheter should be inserted into the inferior vena cava below the level of the right atrium. The appropriate length of the arterial catheter depends on whether a "high" or "low" line is desired (see text for explanation). *(From The Johns Hopkins Hospital, Nechyba C, Gunn VL: The Harriet Lane Handbook: A Manual for Pediatric Home Officers, 16th ed. St. Louis, CV Mosby, 2002.)*

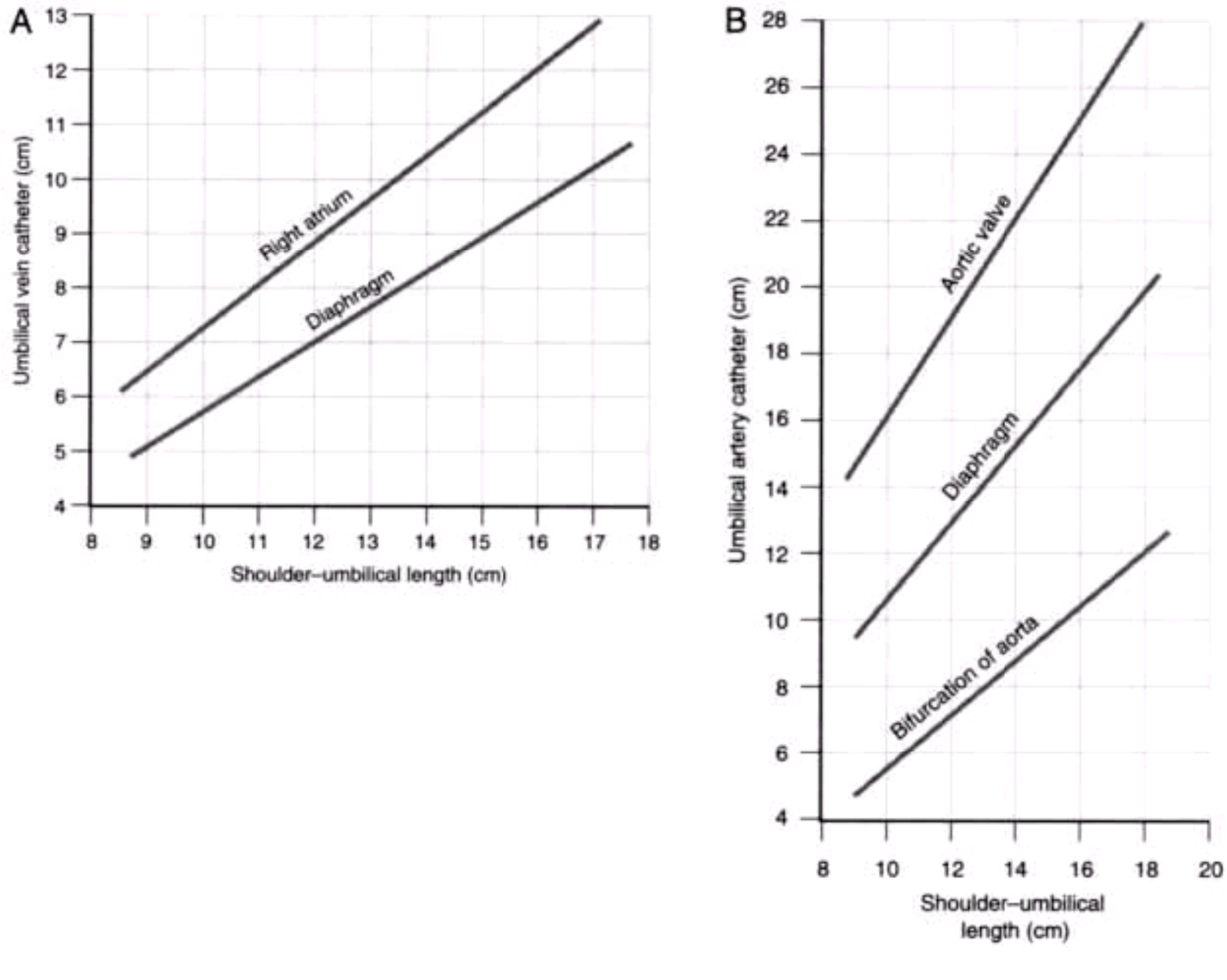


Figure 19-22 The umbilical cord is grasped with a curved hemostat near the selected artery. The umbilical artery is then dilated with a curved iris forceps.

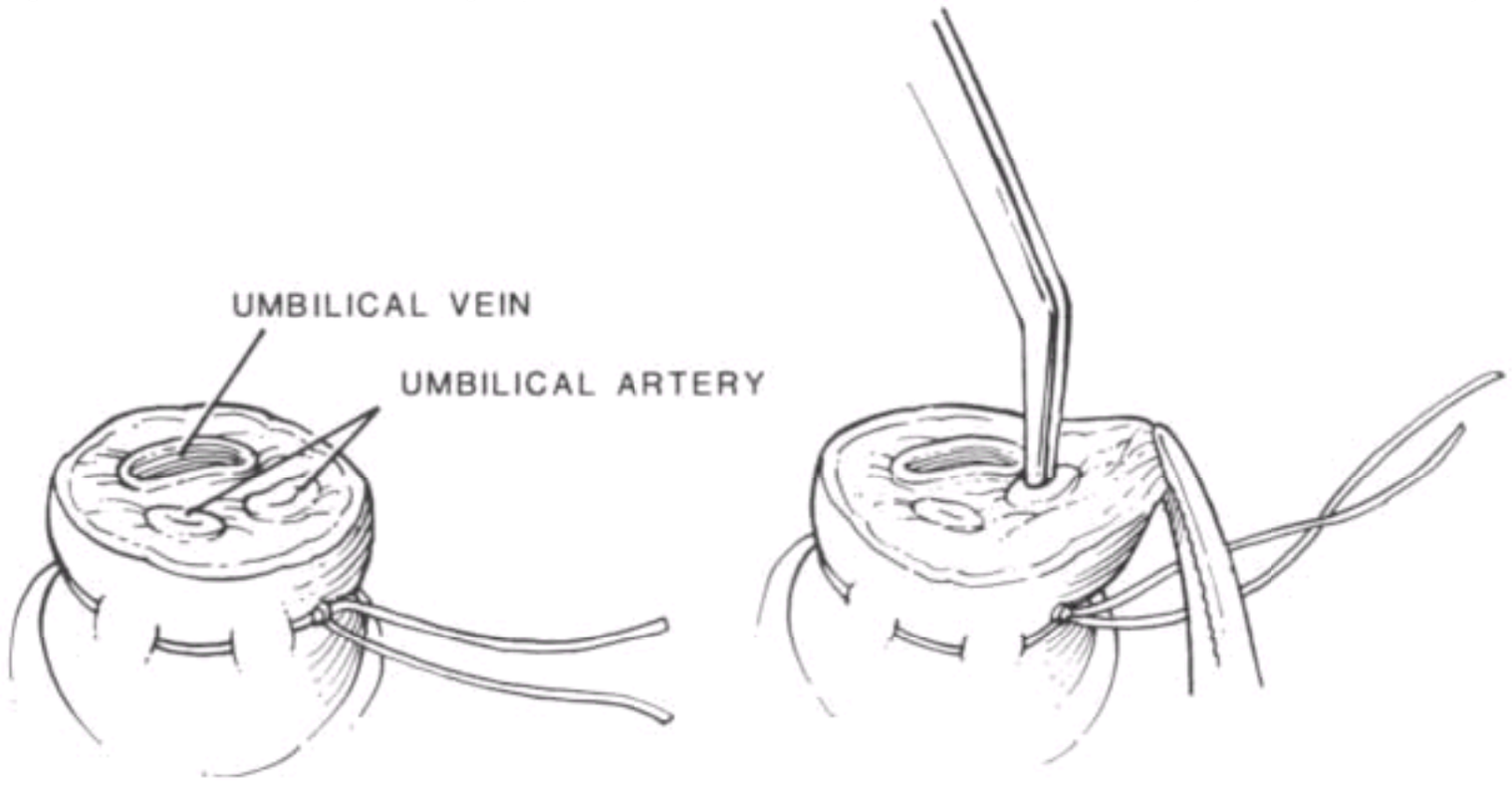


Figure 19-23 The catheter is introduced into the dilated artery and advanced toward the feet. The suture placed around the base of the cord is tied to the catheter.

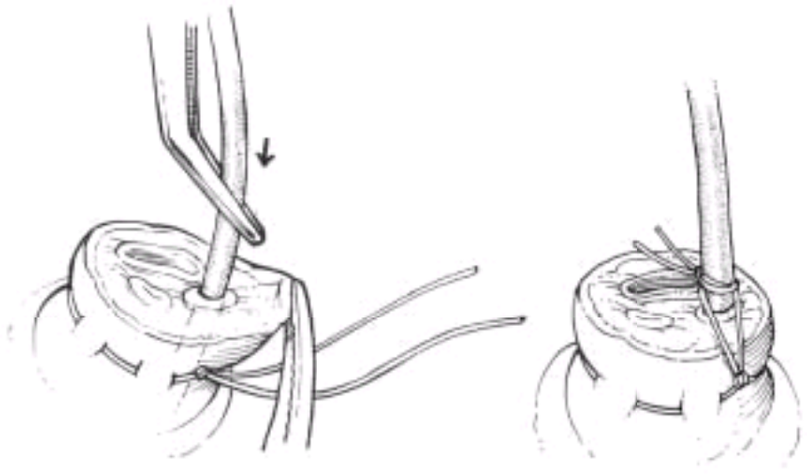


Figure 19-24 The tape is pleated above and below the catheter.

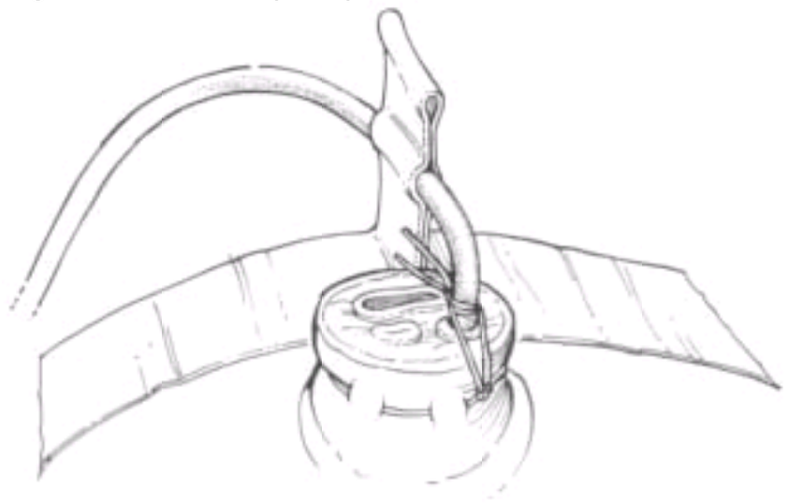


Figure 19-25 The umbilical artery catheter makes a loop downward before heading cephalad (schematic drawing of a radiograph interpretation).

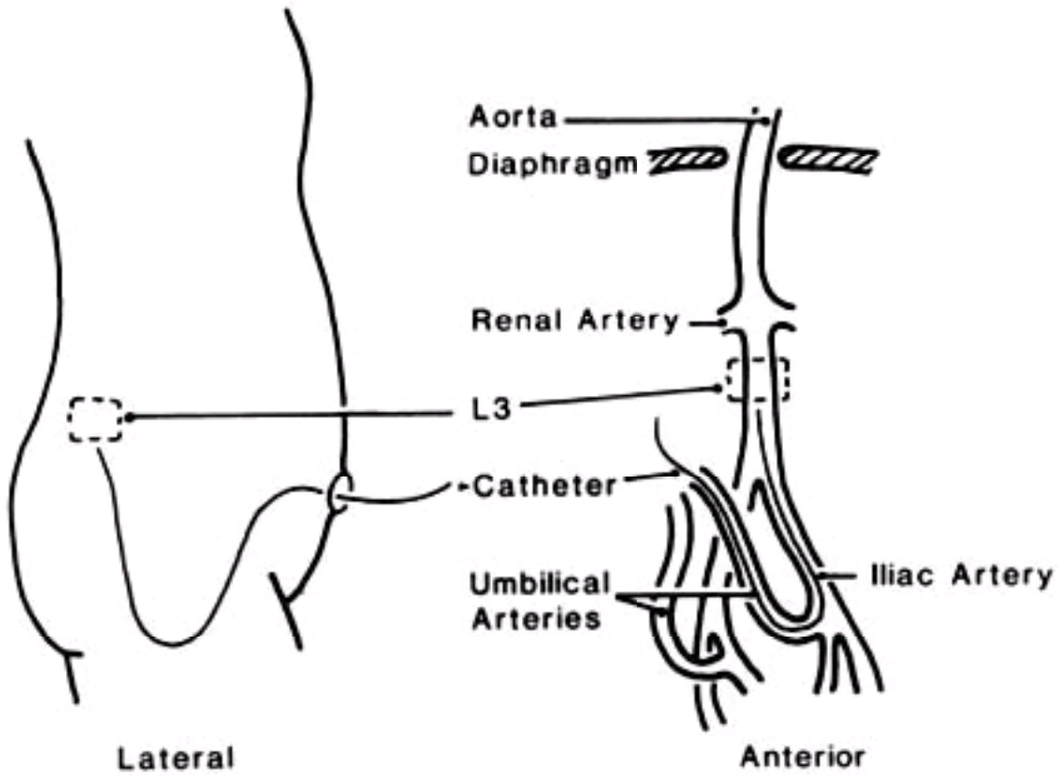


Figure 19-26 The catheter assembly is introduced into the radial artery through skin at a 10- to 20-degree angle. This is a smaller angle than is used for simple arterial puncture. Gloves should be worn.

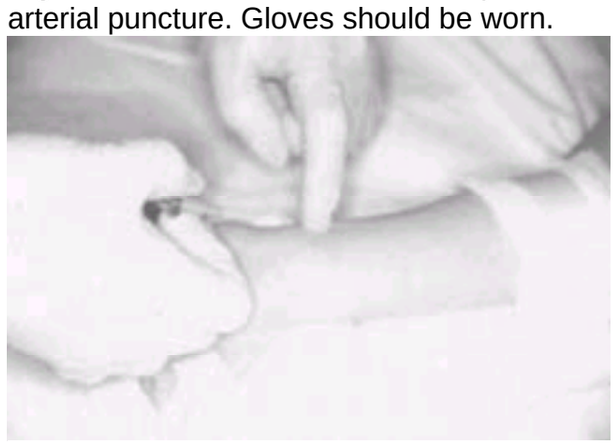


Figure 19-27 One technique of taping the arterial catheter. The arm board should be well padded and secured.

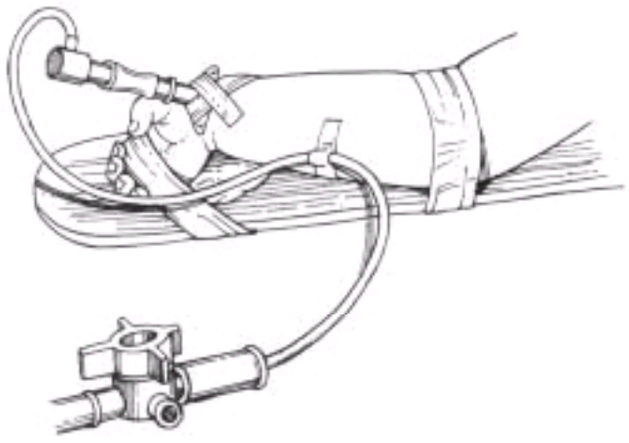


Figure 19-28 Anatomy of the posterior tibial artery and surrounding structures.

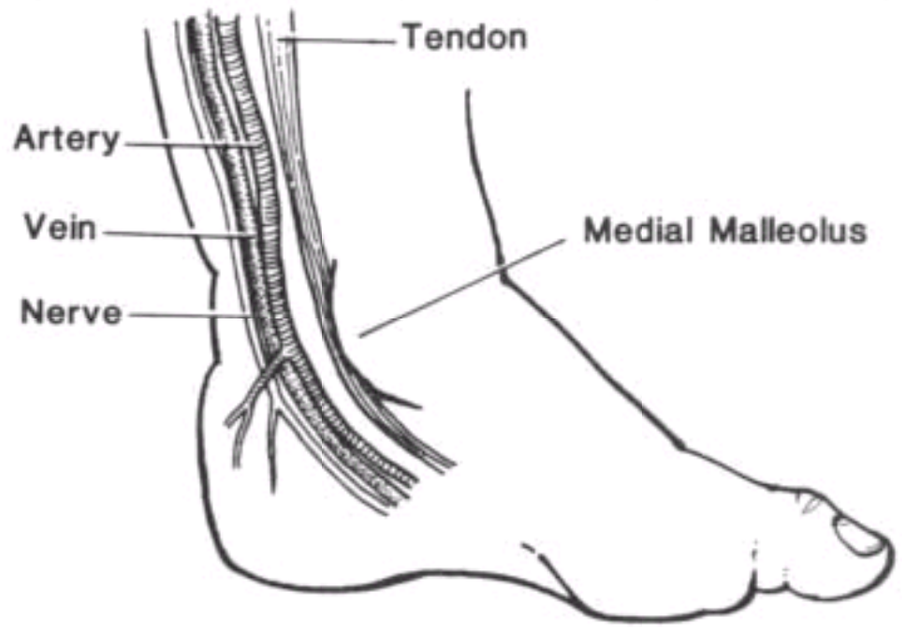


Figure 19-29 *A*, Posterior tibial artery cutdown technique. With the foot prepared and immobilized, a 5- to 7-mm incision is made in the skin posterior to and at the midline of the medial malleolus. *B*, A curved forceps and a silk suture are inserted beneath the posterior tibial artery, which courses just posterior to the medial malleolus.

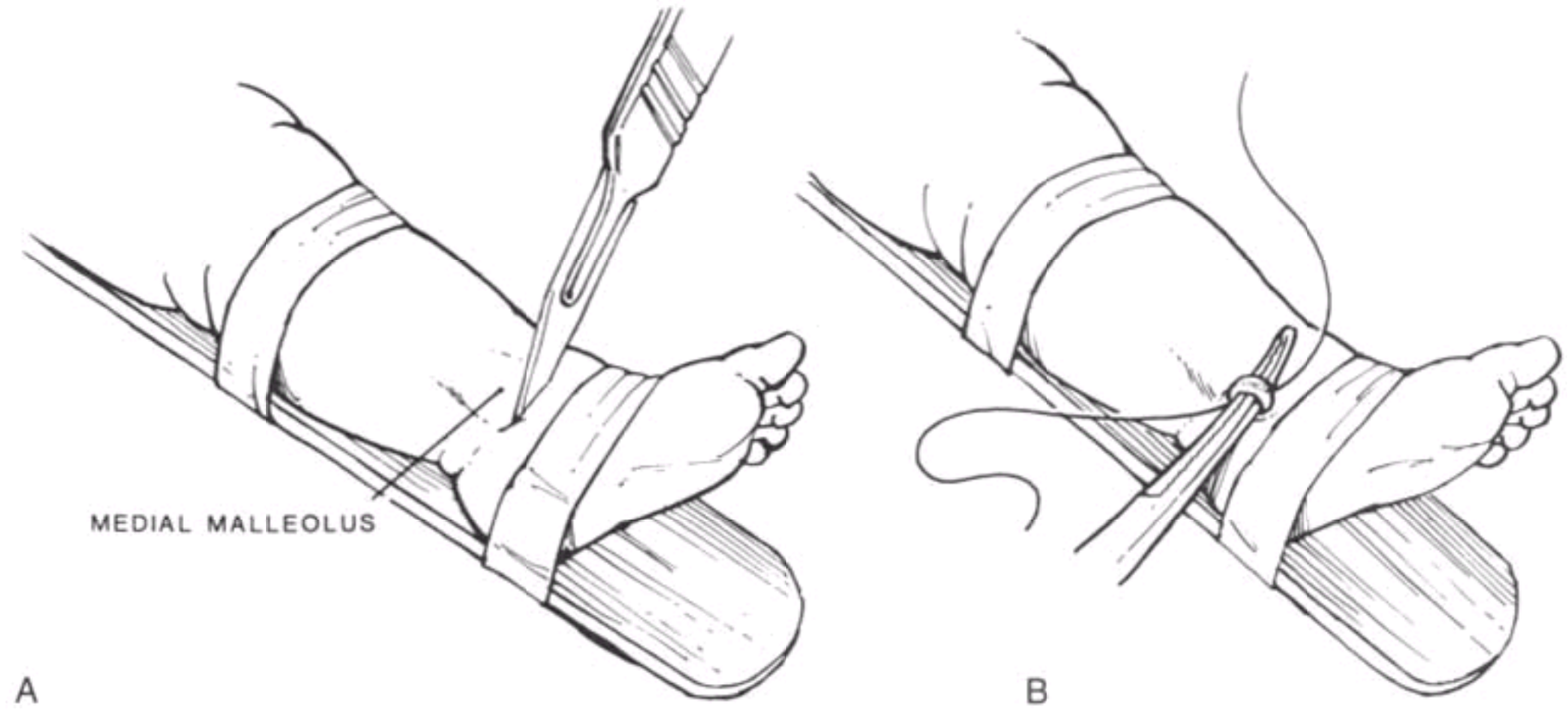


Figure 19-30 Technique of inserting the arterial catheter. A silk tie is used *only to stabilize the artery during cannulation*. It is never tied. The catheter is inserted under direct vision without making an incision in the vessel.

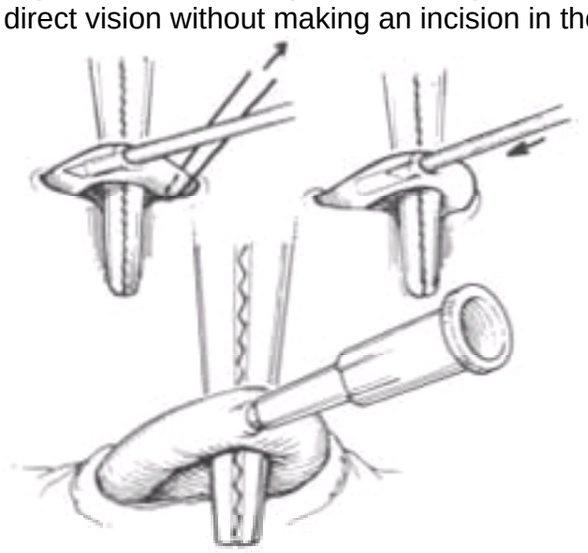


Figure 19-31 The skin incision is closed, and the catheter and connector are secured to the heel with tape.

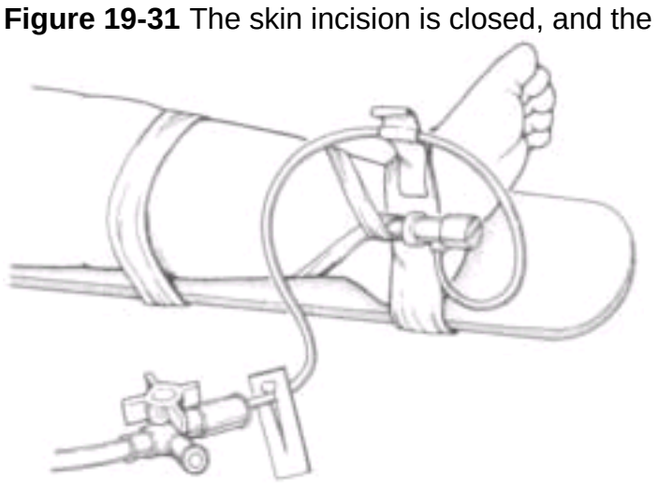


Figure 20-1 Catheters for arterial cannulation. A standard intra-catheter for arterial cannulation may be used for direct vessel cannulation. Newer devices with a modified guidewire and quick-flash identifier (Arrow International, Inc. Reading, PA) or a needle with an attachable guidewire to aid cannulation are also available.

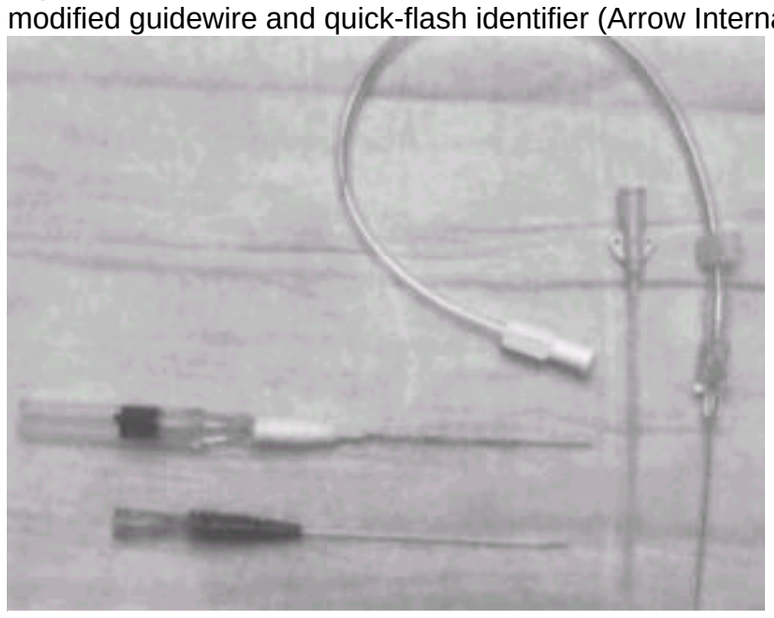


Figure 20-2 Assembly technique for anaeroid manometer system. The middle and proximal extension tubings contain heparinized saline. The middle extension tubing is arranged to form a fluid meniscus at the same level as the heart when the proximal stopcock is closed to the middle tubing (i.e., no pressure input). The distal extension tubing is filled with air and held vertically so that there is no saline contamination of the manometer at maximal pressures. Approximately 10 to 12 cm of air in the distal and middle tubings is optimal. The same system can be used with a mercury manometer in place of the anaeroid manometer. Sterility of the extension tubing and stopcocks is essential. *(From Zorab JSM: Continuous display of the arterial pressure: A simple manometric technique. Anaesthesia 24:433, 1969. Copyright © 1969 by the Association of Anaesthetists of Great Britain and Ireland. Reproduced by permission.)*

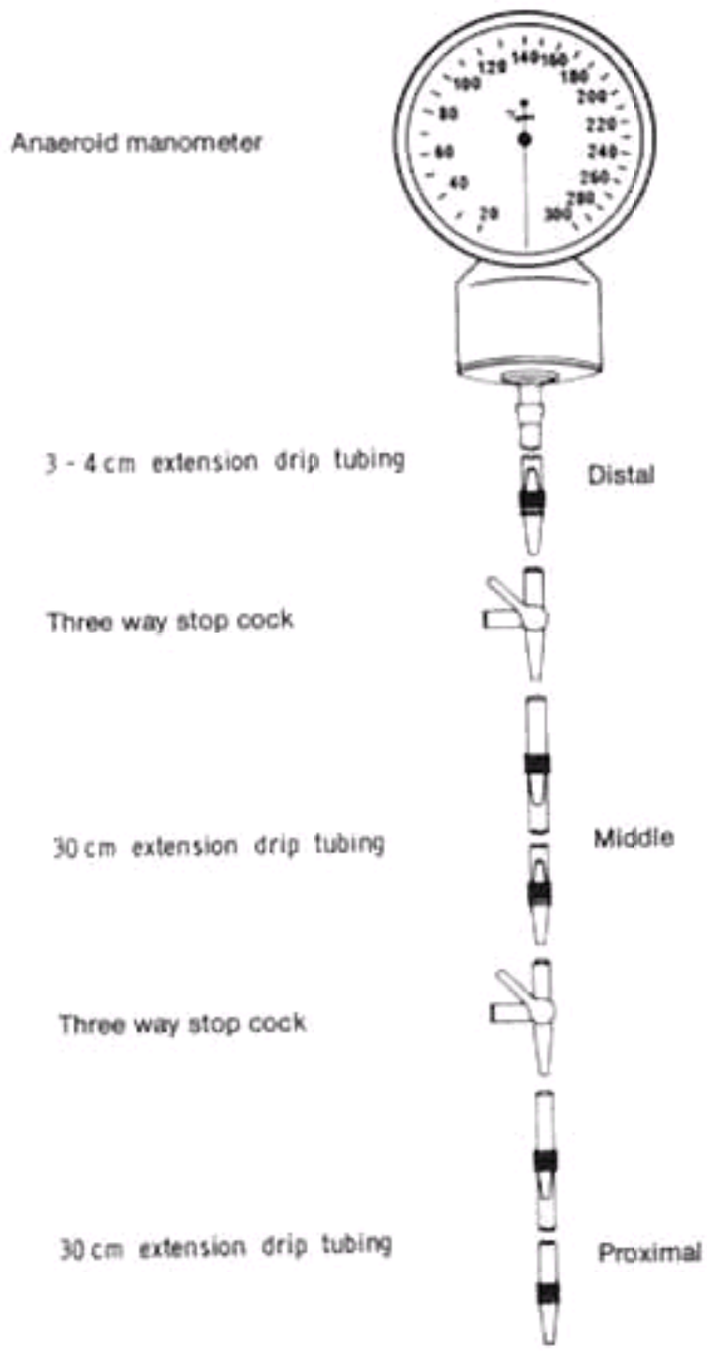


Figure 20-3 Arterial pressure monitoring systems. *A,* System for continuous flush. Heparin (2 mL of a 1:1000 unit solution) is added to a 1-L bag of normal saline, and the bag is pressurized to 300 mm Hg using a metered blood pump (not shown). The continuous flush device is set to deliver 3 mL/hour of the heparinized saline. A mechanical pressure transducer is depicted. The transducer device is a sterile, inexpensive, fully assembled monitor that can be used during patient transfer. Alternatively, the electronic transducer depicted in *B* may be used. *B,* System for manual flush. A heparinized saline flush solution can be injected manually through a syringe at the proximal or distal port. The transducer dome should be maintained at the level of the patient's heart. *(From Beal JM [ed]: Critical Care for Surgical Patients. New York, Macmillan, 1982. Reproduced by permission.)*

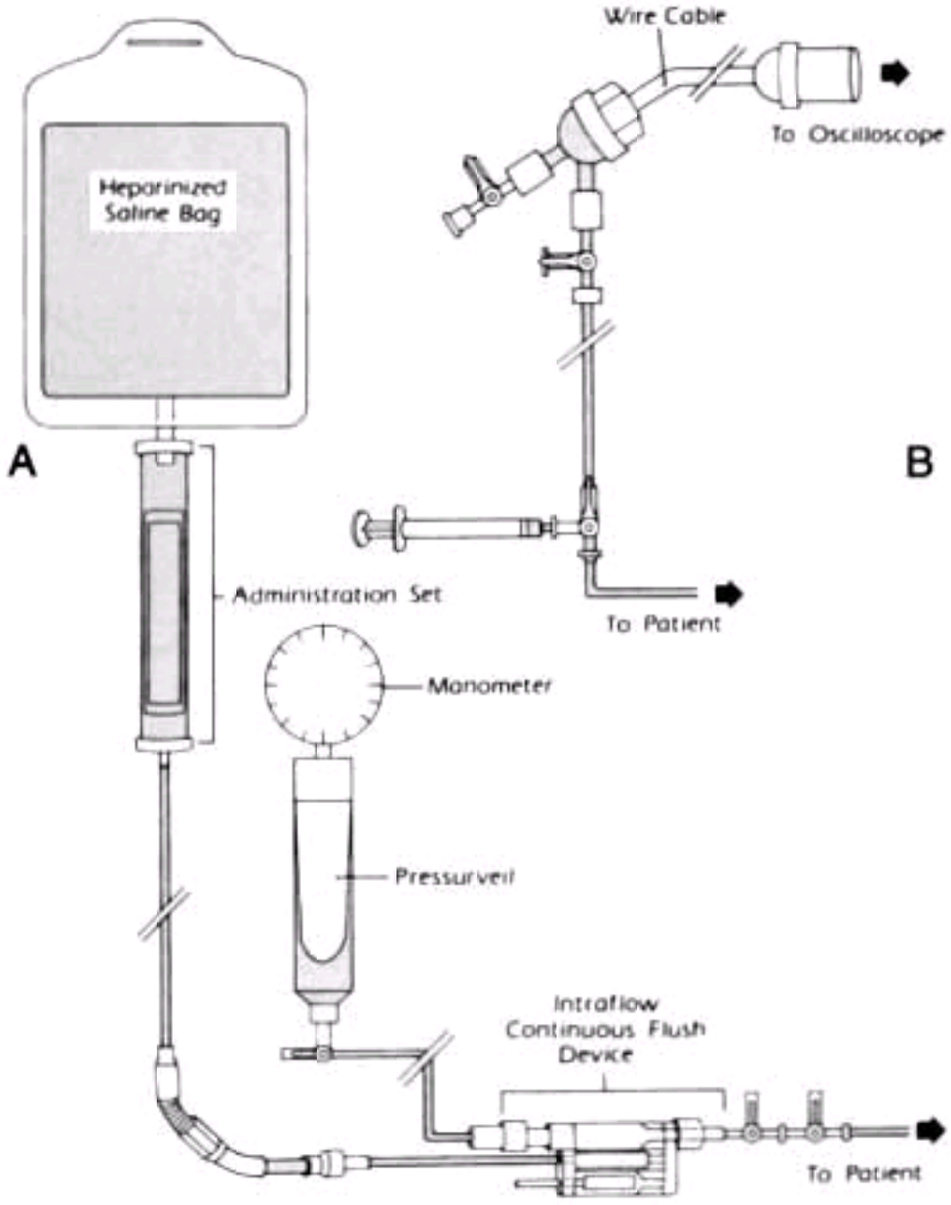


Figure 20-4 Arterial puncture: The syringe-needle is held at a 30 to 45° angle and should enter with the bevel up and advanced slowly. The landmark is best located by palpating the pulse using the index and middle fingers placed immediately proximal to the needle entry site. The needle should *not* be passed between the fingers due to risk of operator self-injury. *(Picture courtesy of JMM, with permission.)*

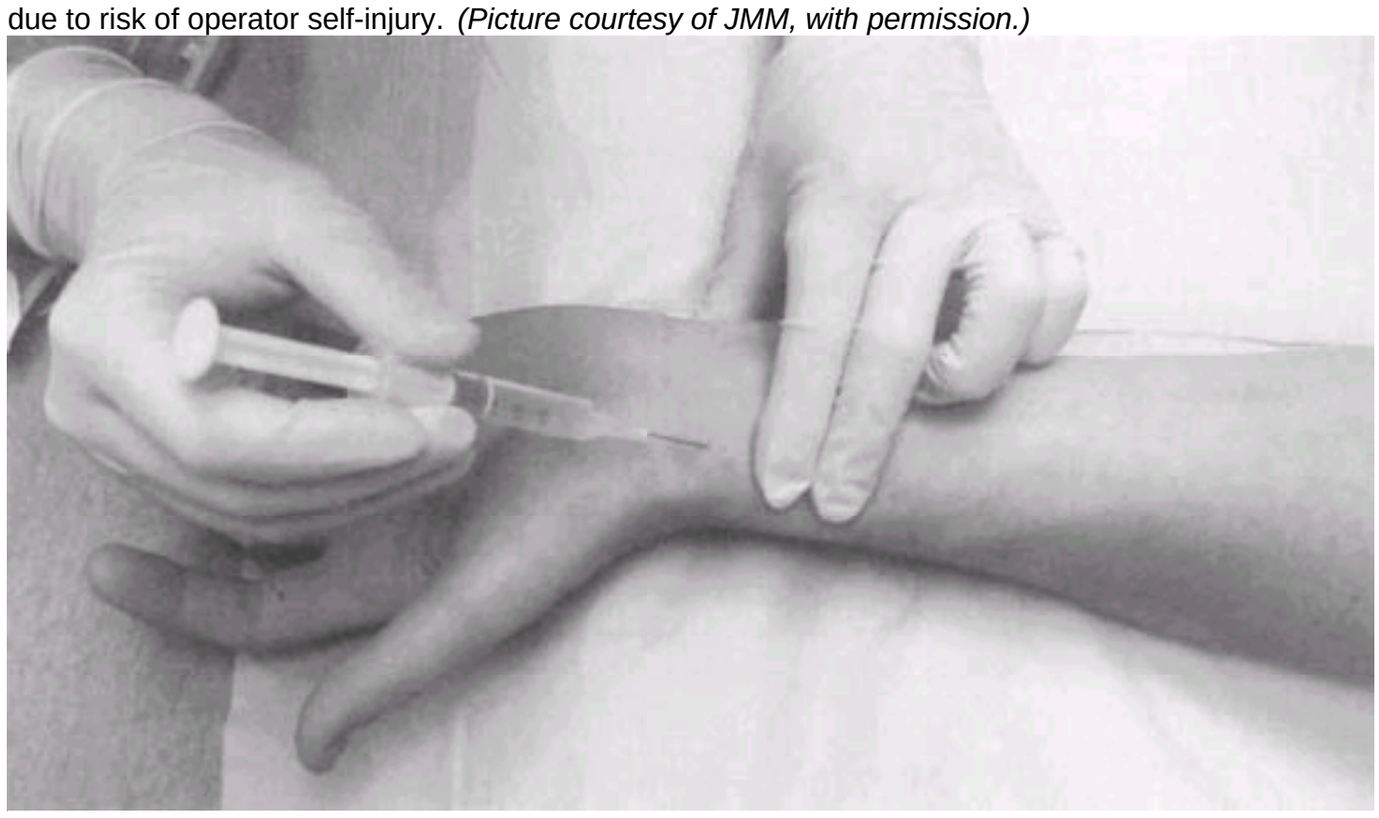
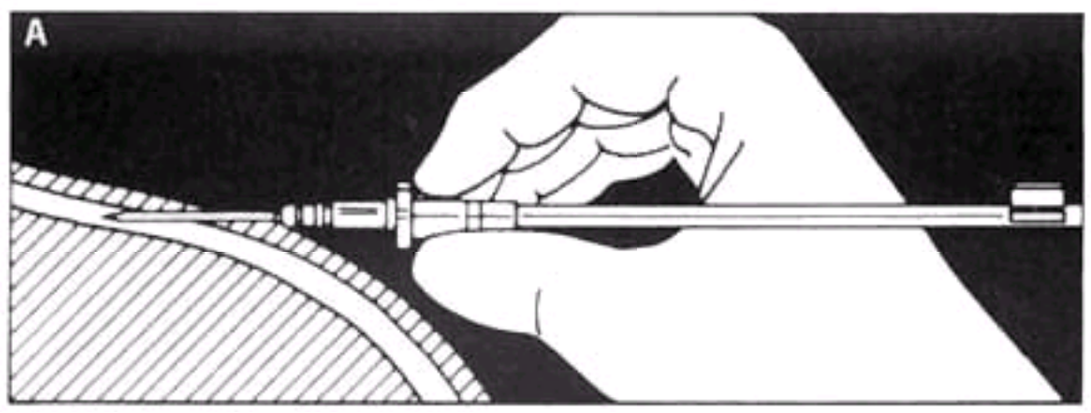


Figure 20-5 Removal of air bubbles from the syringe. Air bubbles are finger-tapped to the top of the syringe. An alcohol swab is placed over the top of the needle. The plunger is advanced to expel air while drops of blood are collected on the alcohol swab. After removal of the bubbles, the syringe is capped and sent to the laboratory.

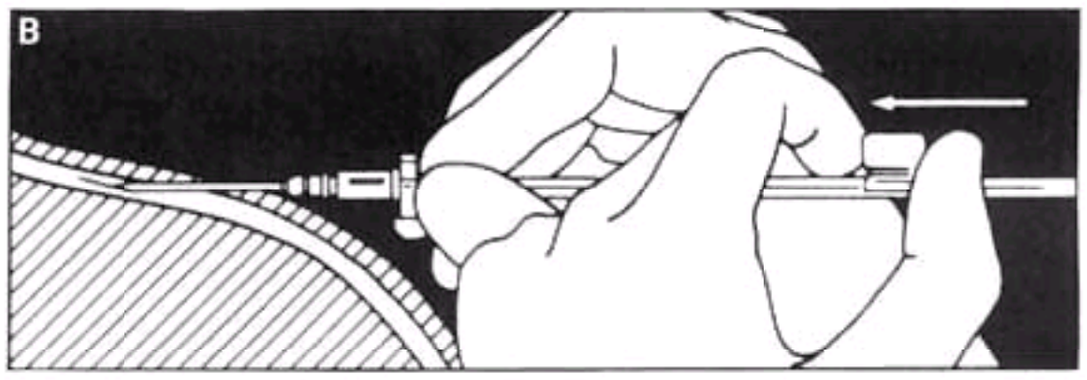
Figure 20-6 Step-by-step arterial cannulation, using the guidewire technique (Arrow arterial catheterization kit). *(Courtesy of Arrow International, Inc., Reading, PA.)*

General instructions for use of Radial Artery set.

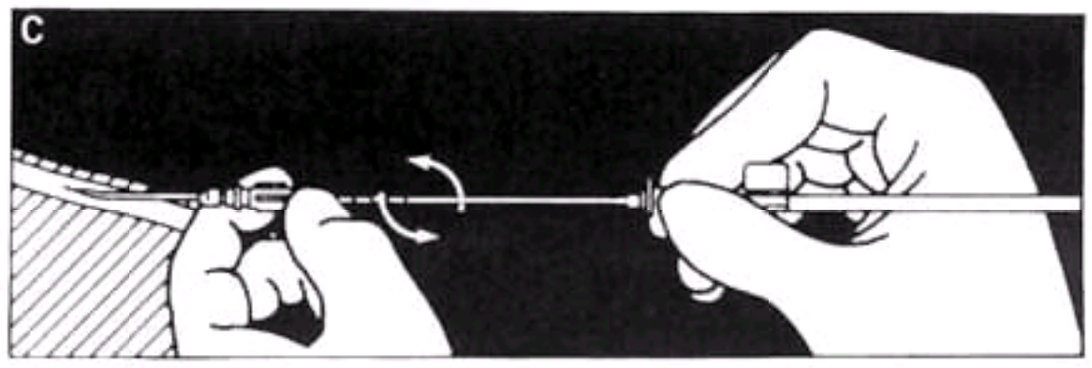
1. Prepare puncture site in preferred manner.
2. Peel open package and remove entire unit.
3. Remove protective shield. Trial advance and retract spring-wire guide through needle via actuating lever to ensure proper feeding. *Note:* Catheter hub wing clip can be "snapped" out of groove and removed if desired. This allows suture ring on hub to be optionally used for attachment to skin after placement.
   CAUTION: Before insertion, actuating lever must be retracted proximally as far as possible so as not to inhibit blood flashback.
4. Puncture vessel using a continuous, controlled, slow forward motion, being careful to avoid transfixing both vessel walls. Blood flashback in clear hub of introducer needle indicates successful entry into vessel *(A)*.
   CAUTION: If both vessel walls are punctured, advancement of spring-wire guide could result in inadvertent subarterial placement.



5. Stabilize position of introducer needle and carefully advance spring-wire guide (via actuating lever) distally as far as possible into vessel *(B)*. Reference mark on clear feed tube indicates approximate actuating lever advancement position at which soft tip of spring-wire guide coincides with tip of needle.
   CAUTION: If resistance is encountered while advancing spring-wire guide, *do not force feed and do not retract spring-wire guide while in vessel* (to avoid damaging wire). Withdraw entire unit and attempt new puncture.



6. Advance the entire placement unit a maximum of 1 to 2 mm farther into the vessel.
7. Firmly hold clear introducer needle hub in position and advance catheter forward to track spring-wire guide into vessel. If difficulty is encountered during catheter advancement, a slight rotating motion of catheter hub may be helpful *(C)*.



8. Hold catheter in place and remove introducer needle, spring-wire guide, and feed tube assembly. Free blood flow indicates successful placement in vessel.
   CAUTION: Do not reinsert needle into catheter.
9. Attach desired stopcock, injection cap, or connecting tubing to catheter hub.
10. Secure catheter to patient in preferred manner, using wing clip or suture ring as described in Step 3.
11. Cover puncture site with suitable dressing.

Figure 20-7 Femoral vessel cannulation kit. (Arrow International, Inc., Reading, PA.) A 16-ga single lumen vessel with a guidewire for Seldinger placement includes entire procedural needs with local anesthetic, subcutaneous puncture syringe, needle, scalpel, and suture material.

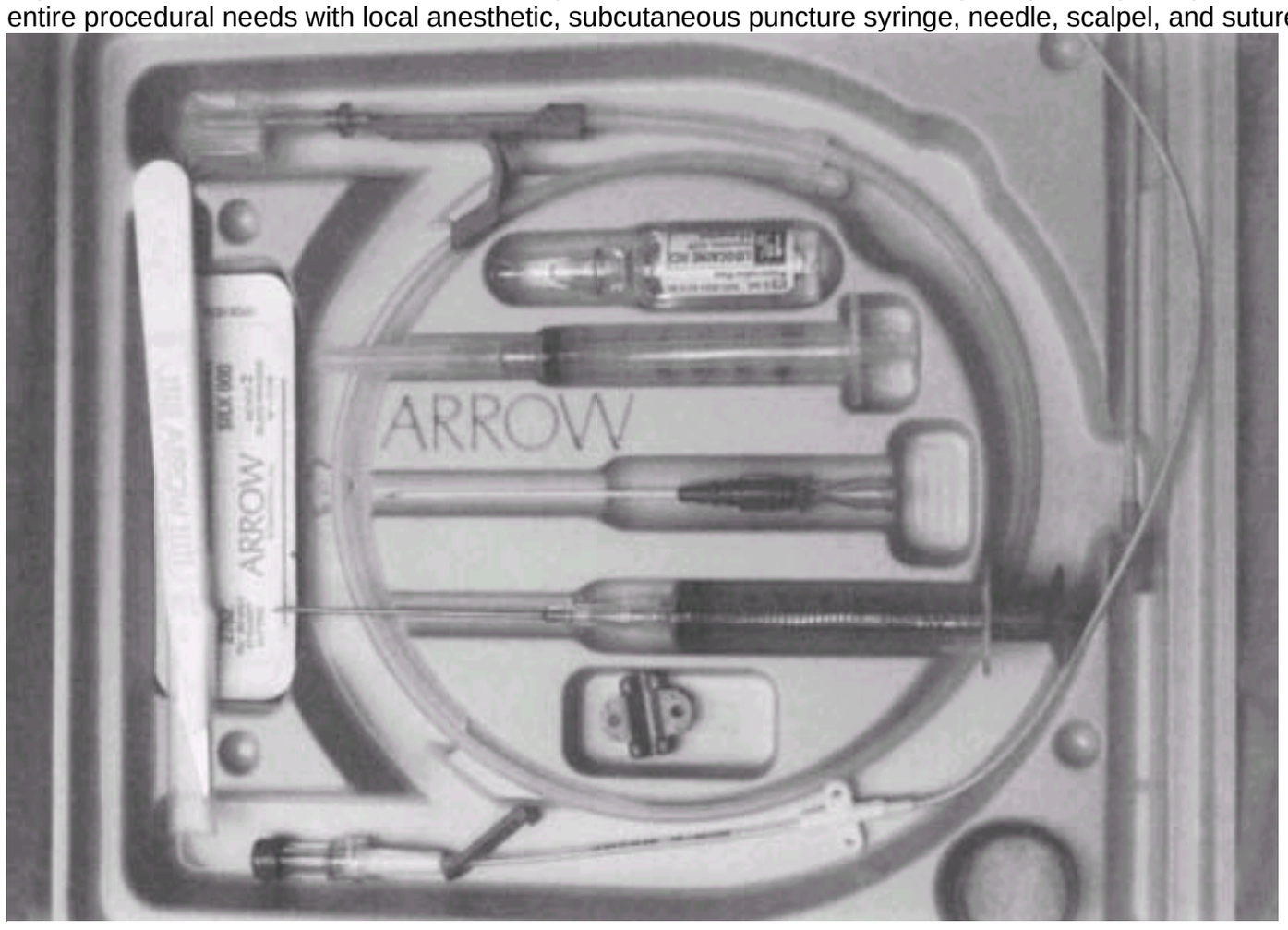


Figure 20-8 Placement of an arterial line using the cutdown technique. Note that the catheter enters the surgical wound percutaneously to minimize bacterial entry into the healing wound and permit better stabilization of the catheter. Catheter entry of the vessel is more parallel to the vessel than is illustrated. Ligatures are used only to *temporarily* isolate the artery and to control bleeding. *The artery should not be tied off.* The catheter is secured by suturing the hub to the skin (see Fig. 20-9).

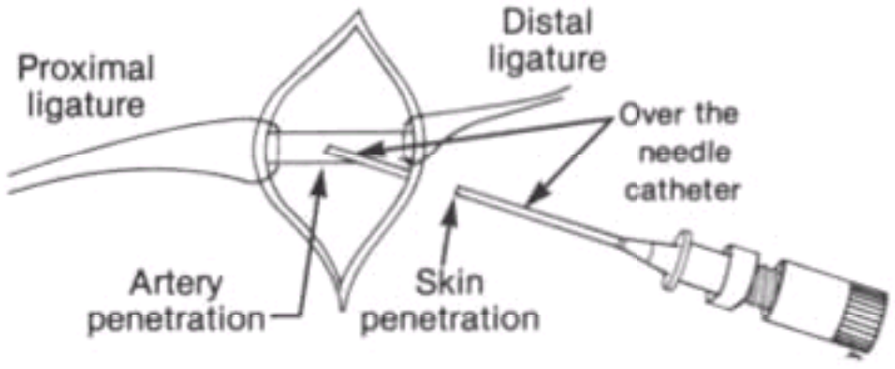


Figure 20-9 Illustration of a technique for securing a vascular catheter to adjacent skin.

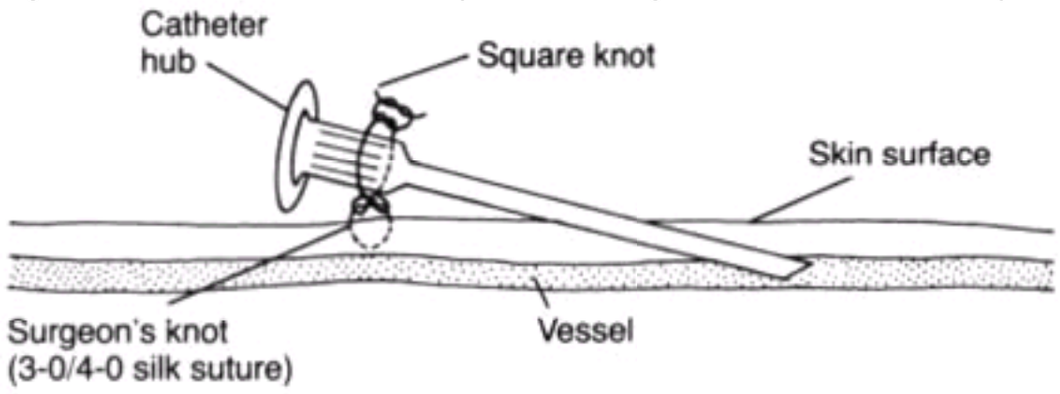


Figure 20-10 Pressure transducer fluid monitoring set; a pressurized saline (heparinized solution may be substituted) connected to an intravascular line and arterial catheter, which is then fixed to a three-way stopcock. The fluid wave is directly monitored by the electormechanical transducer, which records pressure cycles and displays multiple hemodynamic values.

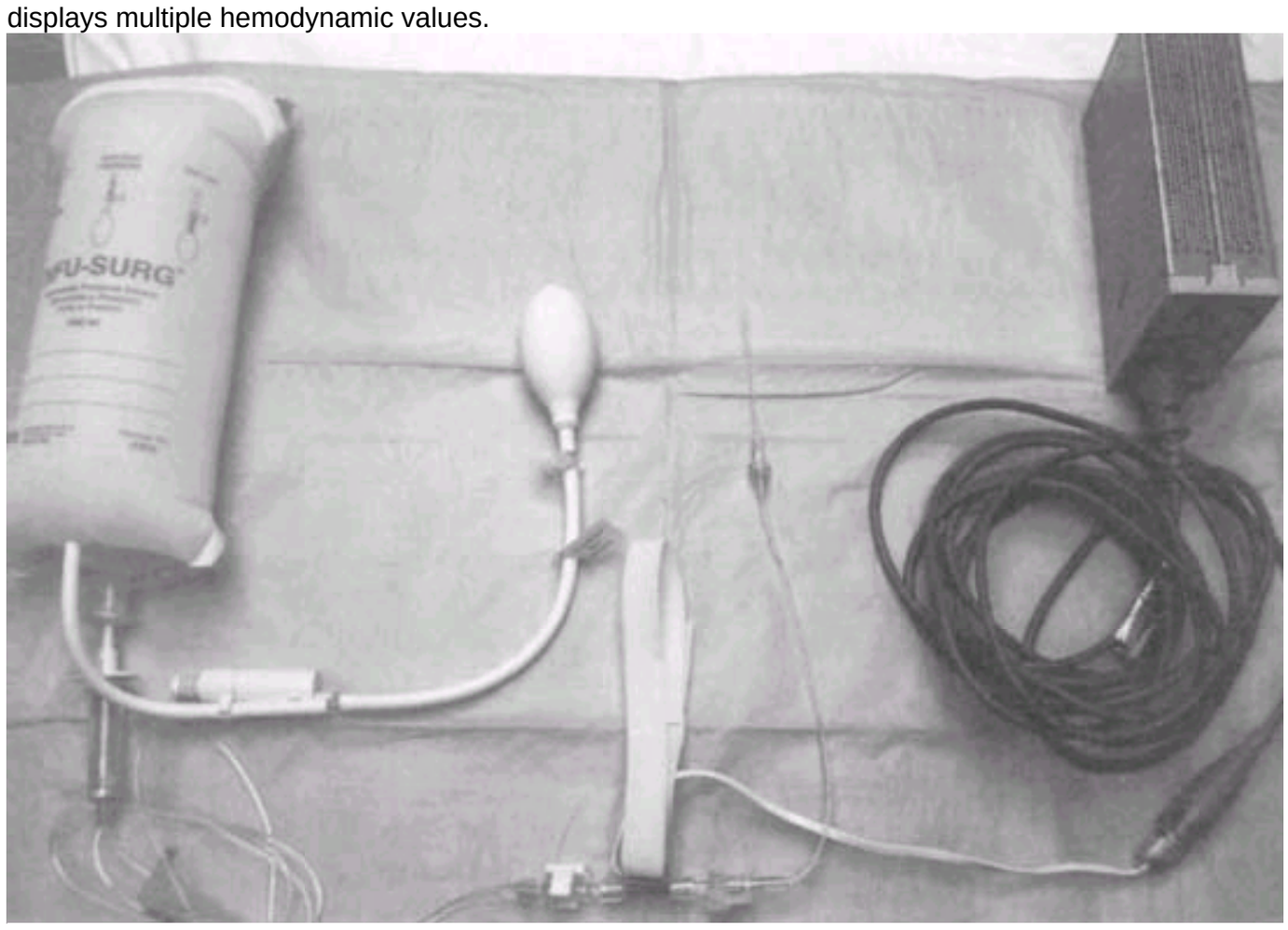


Figure 20-11 Arterial anatomy of the hand and wrist. *(From Ramanathan S, Chalon J, Turndorf H: Determining patency of palmar arches by retrograde radial pulsation. Anesthesiology 42:758, 1975. Reproduced by permission.)*

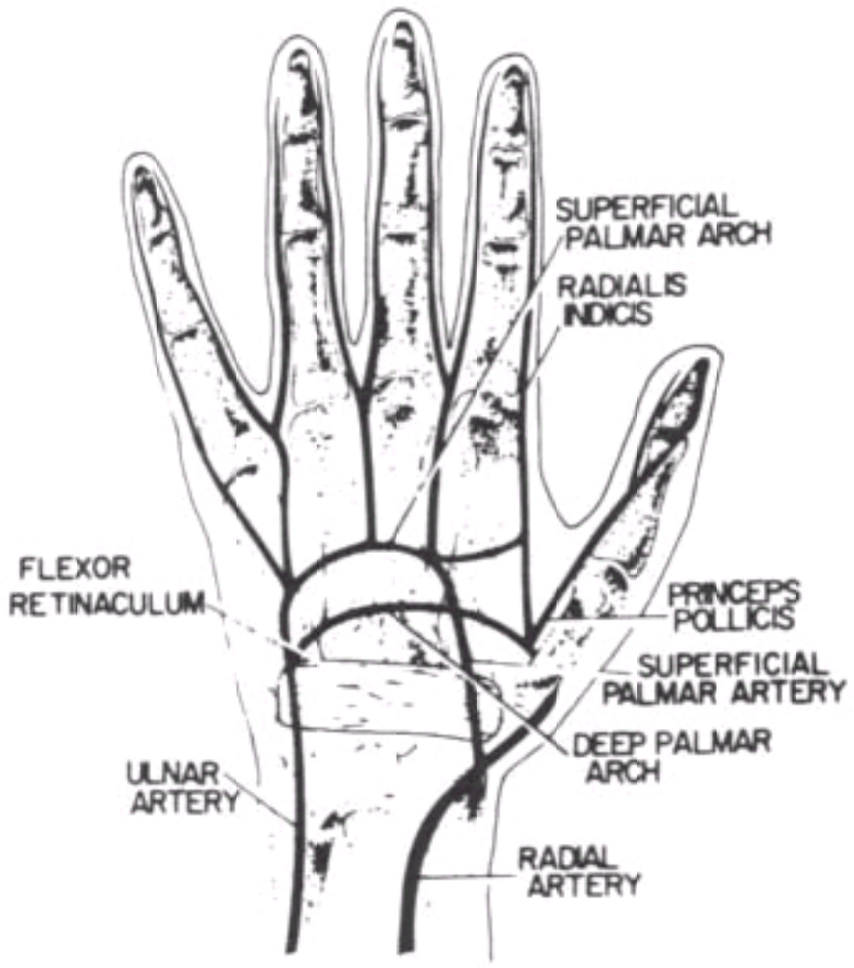


Figure 20-12 Allen test. Before puncturing the radial artery it is important to identify a competent ulnar artery. This can be done as follows: (*1*) The examiner compresses both arteries, and the patient repeatedly makes a tight fist to squeeze all the blood out of the hand. (*2*) The patient then extends the fingers, and the examiner observes the blanched hand. (*3*) Compression of the ulnar artery is released, and the examiner observes the hand filled with blood. If filling does not occur within 5 to 10 seconds, radial artery puncture should not be done. If brisk filling occurs, the test is then repeated with release of the radial artery to assess radial artery patency. If both radial and ulnar arteries demonstrate patency, the wrist may be used for arterial puncture. *(From Schwartz GR [ed]: Principles and Practice of Emergency Medicine. Philadelphia, WB Saunders, 1978, p 354. Reproduced by permission.)*

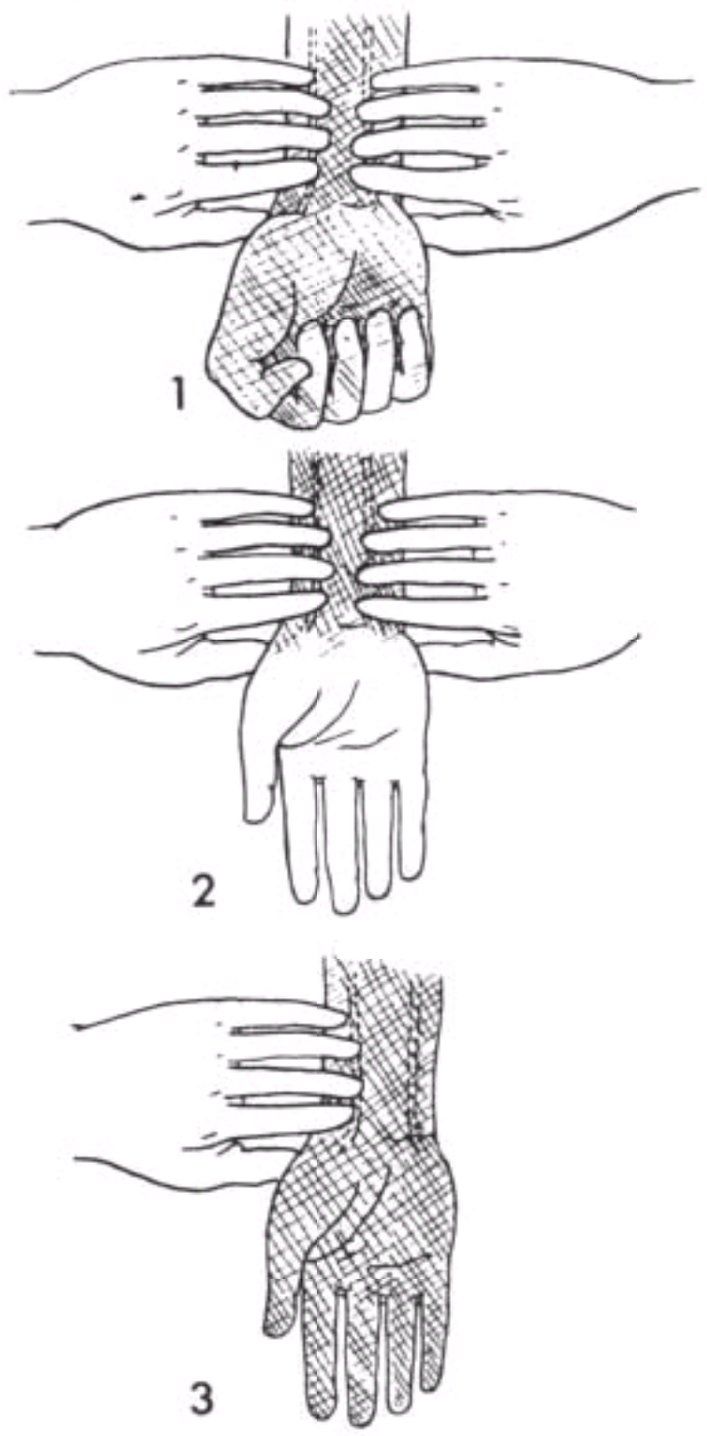


Figure 20-13 Percutaneous arterial cannulation at the wrist. The catheter unit is advanced 1 to 2 mm into the vessel lumen after blood first appears in the flash chamber. While the needle is fixed, the catheter is threaded over the needle. *(From Beal JM [ed]: Critical Care for Surgical Patients. New York, Macmillan, 1982. Reproduced by permission.)*

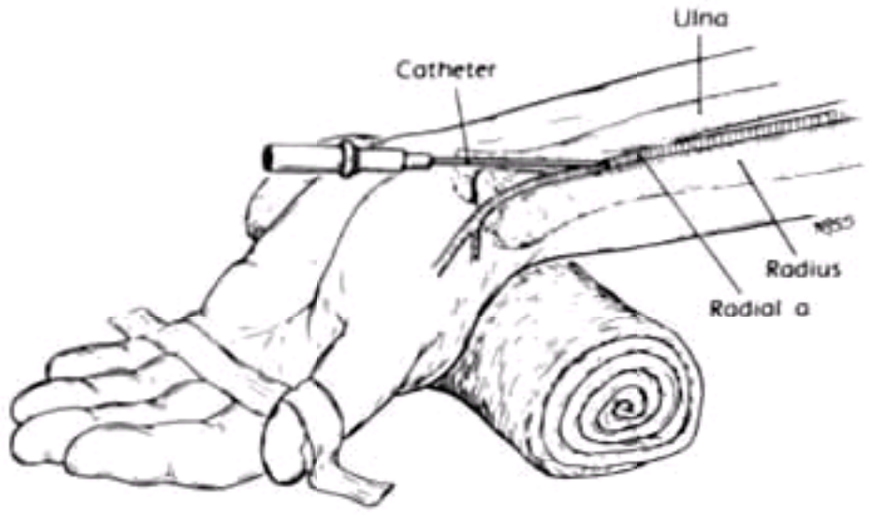


Figure 20-14 The right brachial artery and its branches. *(From Christensen JB, Telford IR: Synopsis of Gross Anatomy. New York, Harper & Row, 1966. Reproduced by permission.)*

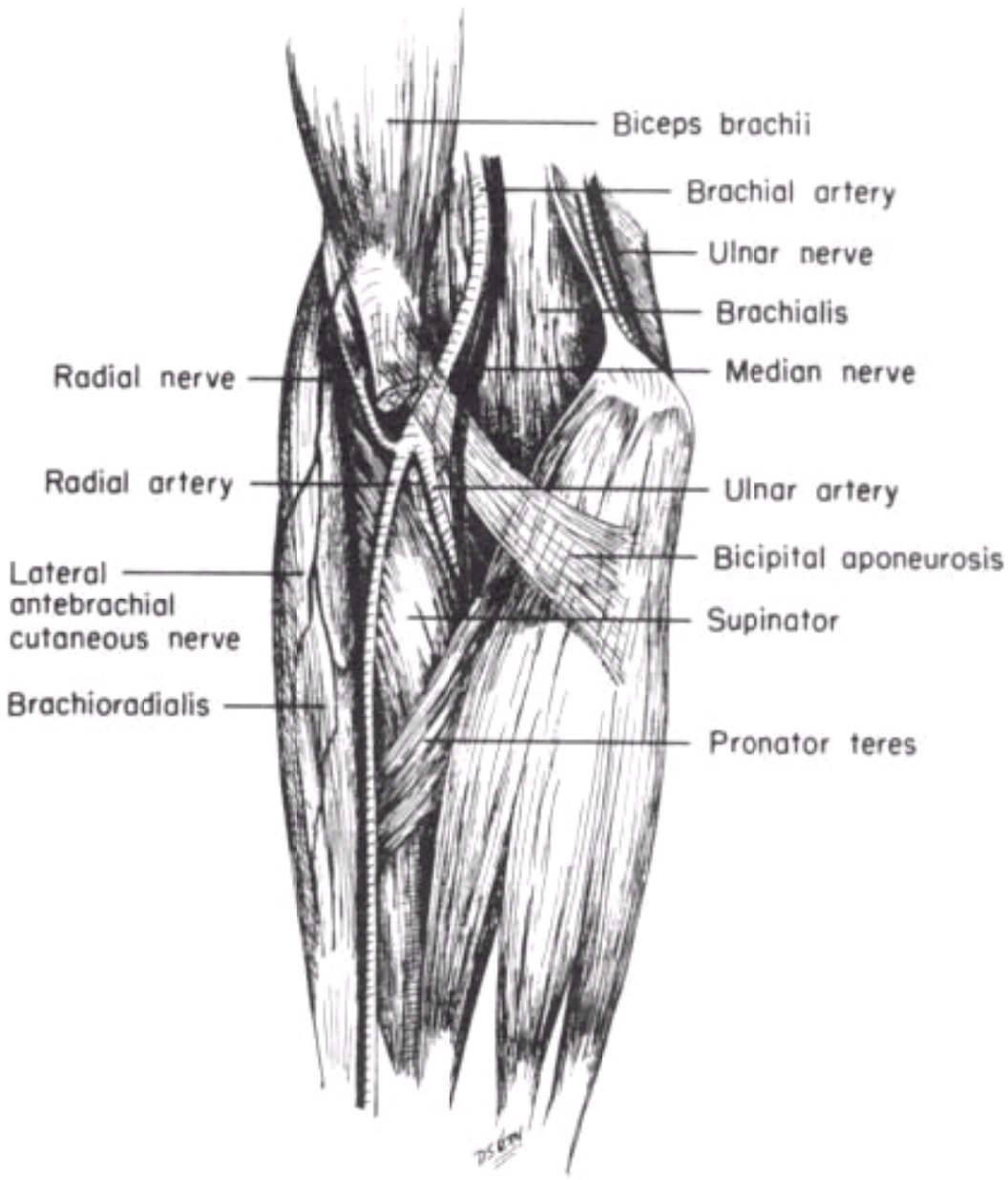


Figure 20-15 A 20-ga catheter in the dorsalis pedis artery, illustrating the relationship to surrounding tendons. The catheter is secured with Steri-Drape. Splinting is not needed. *(From Johnstone RE, Greenhow DE: Catheterization of the dorsalis pedis artery. Anesthesiology 39:655, 1973. Reproduced by permission.)*

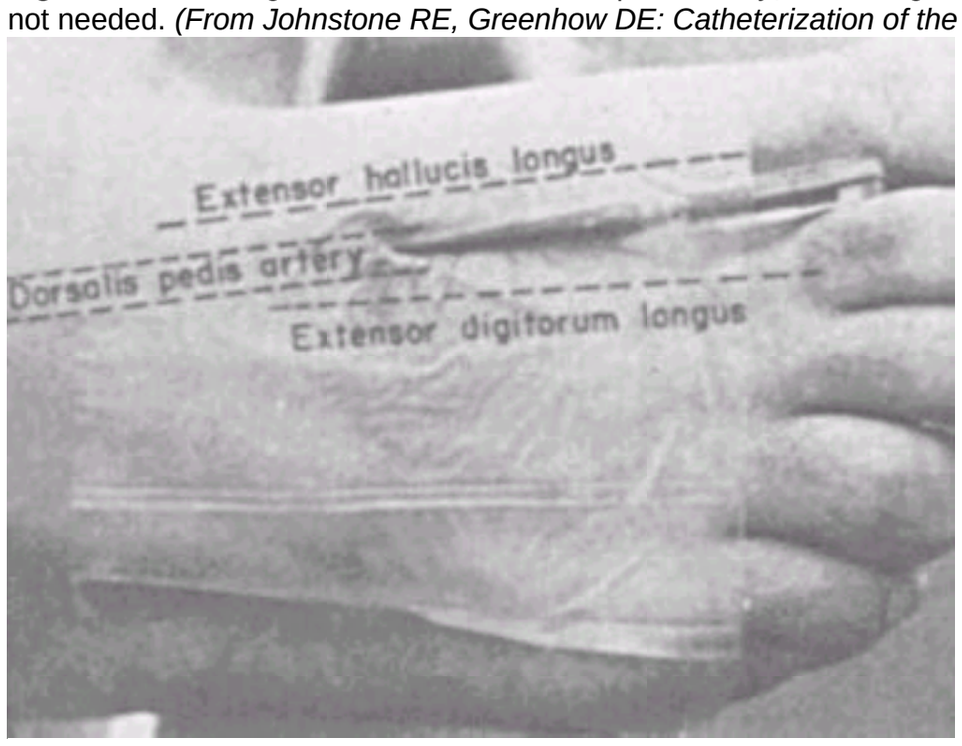


Figure 20-16 The right femoral vessels and some of their branches. The femoral nerve (not shown) lies lateral to the artery and may be deep to the artery. *(From Warwick R, Williams PL [eds]: Gray's Anatomy, 35th ed. Edinburgh, Churchill Livingstone, 1973, p 676. Reproduced by permission.)*

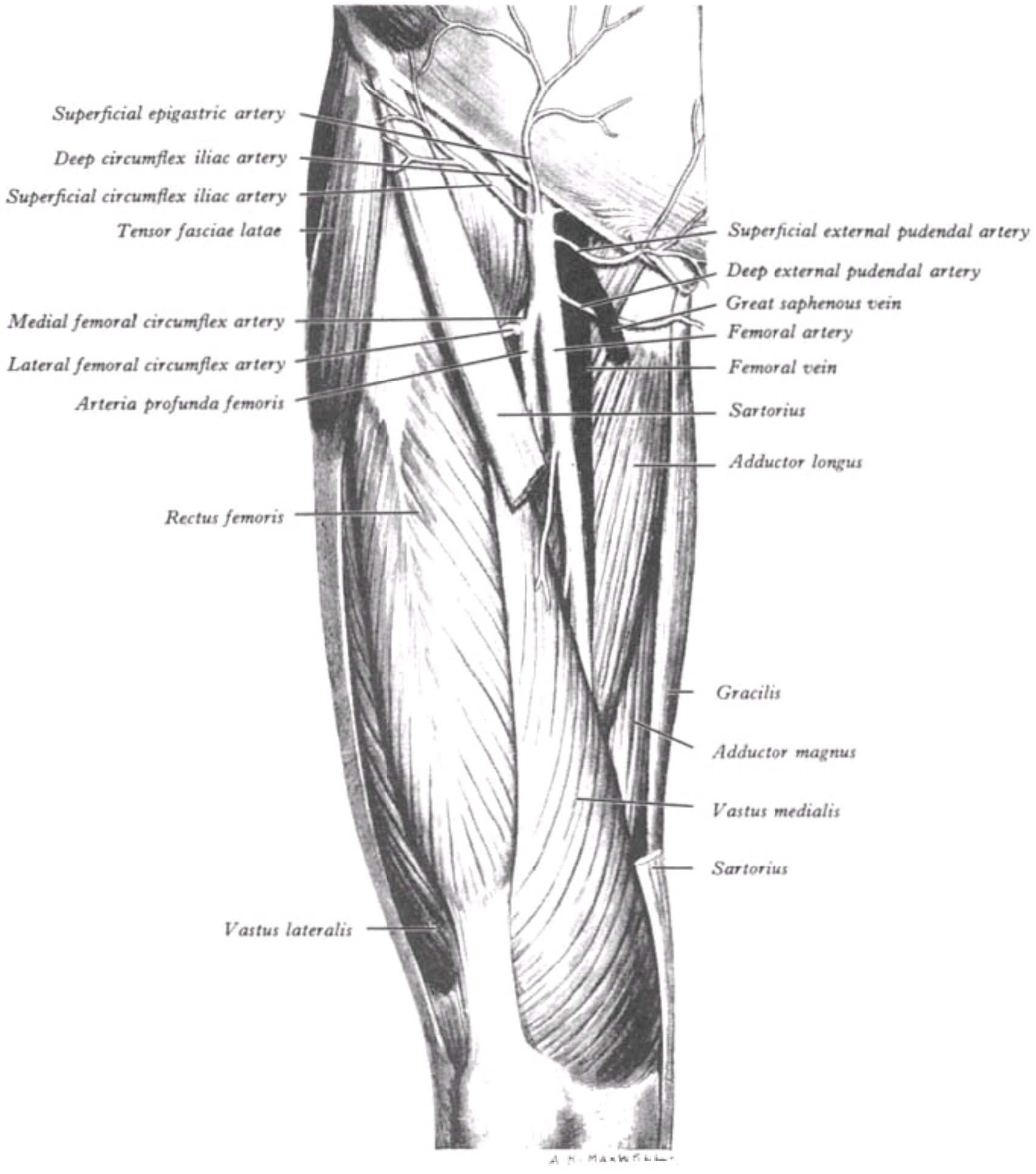


Figure 21-1 Anatomy of veins in hands and arms for intravenous cannulation. *(Adapted from Millam DA: How to insert an IV. Am J Nurs 79:1268, 1979.)*

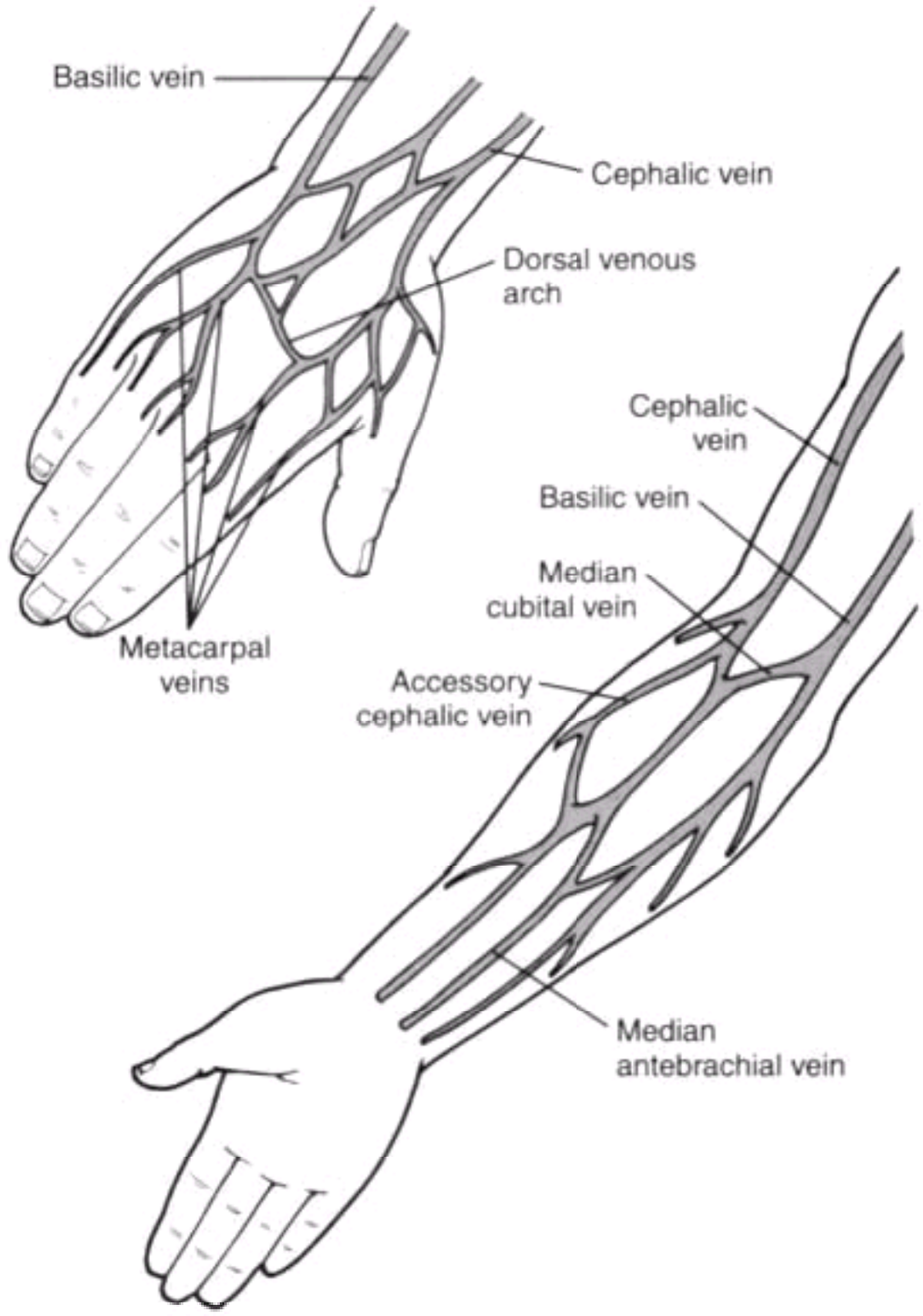


Figure 21-2 The external jugular vein may be cannulated in the same manner as any other peripheral vein, which often negates the need for central venous catheterization. This site is especially useful to obtain blood and to infuse fluids and medication in obese adults, infants, and IV drug users. The major disadvantages are that flow is very dependent on the position of the neck (a significant problem in children and obtunded or restless adults), and valves may abut against the tip of the catheter. Air embolism from a disconnected catheter is another potential hazard with this access route.

Figure 21-3 Materials for intravenous insertion. *A* and *B*, Tape for securement. *C*, Gloves as part of universal health precautions. *D*, Saline lock. *E*, Saline. *F*, Tape roll. *G*, Gauze. *H*, Tegaderm. *I*, Alcohol swabs. *J*, Butterfly needle, if needed. *K*, Intravenous tubing. *L*, Iodine swabs. *M*, Syringe for phlebotomy. *N*, Angiocatheter. *O*, Prepped saline flush. *P*, Tourniquet.

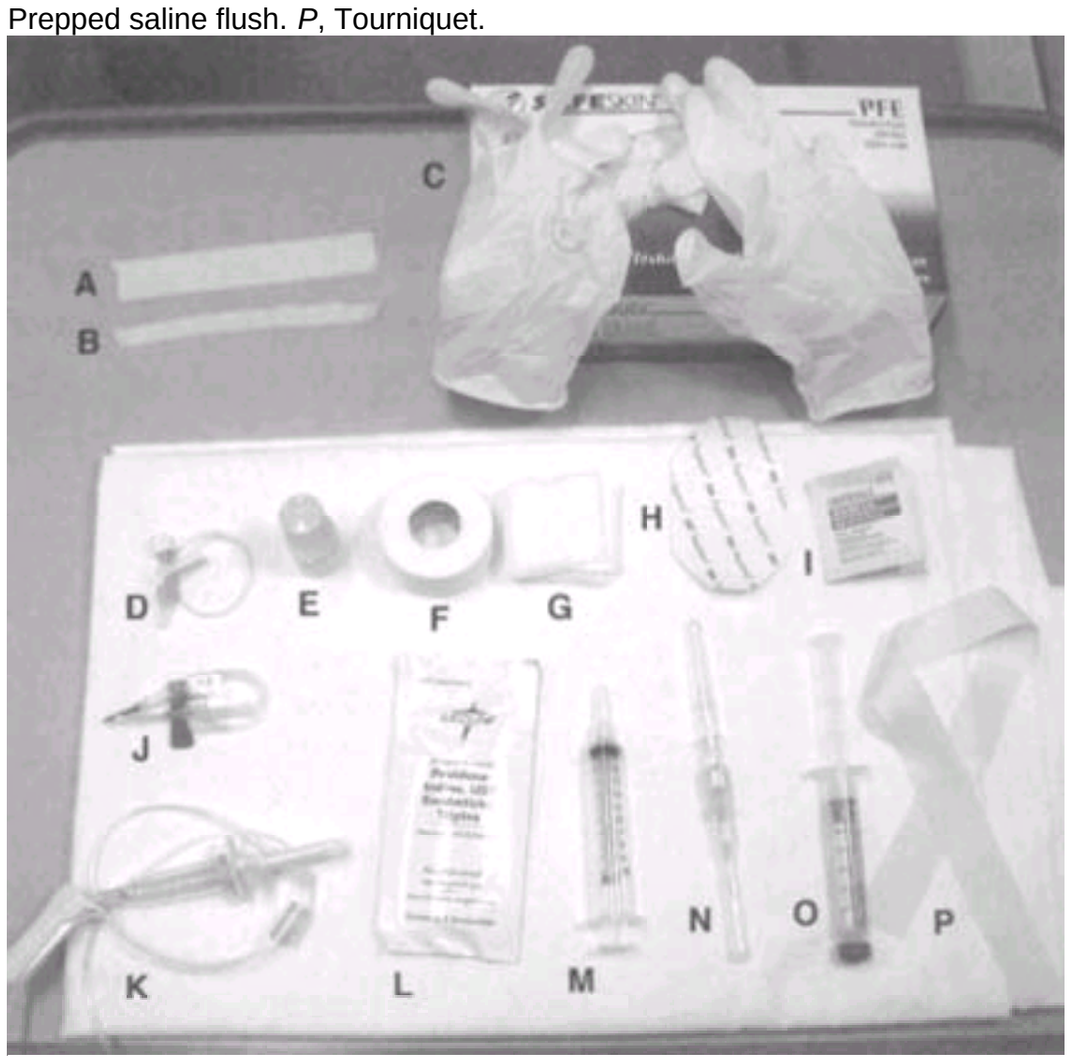


Figure 21-4 Insertion of spiked end of IV tubing into IV bag.

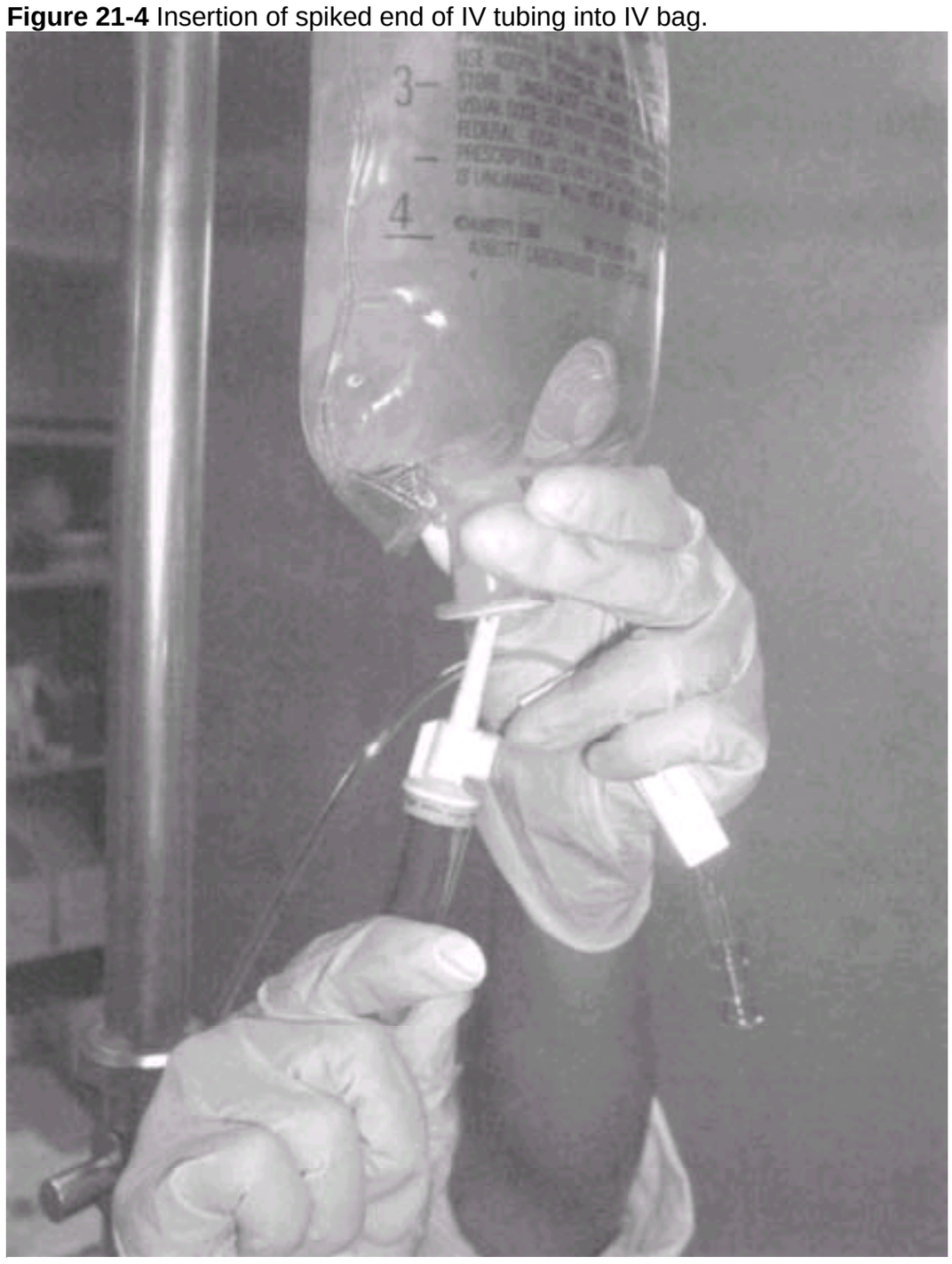


Figure 21-5 Pinching drip chamber to fill bulb halfway before infusing fluid.

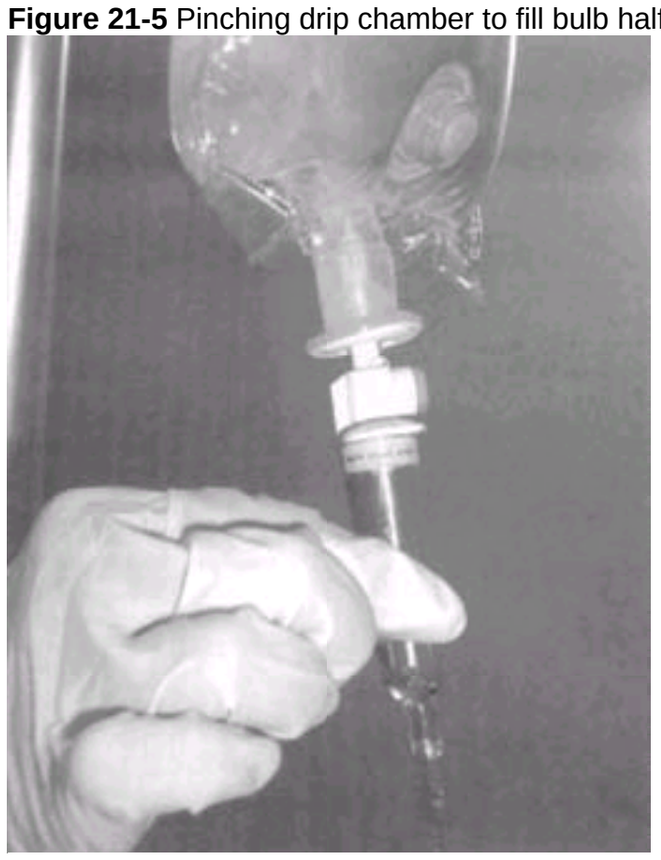


Figure 21-6 Flushing IV tubing.

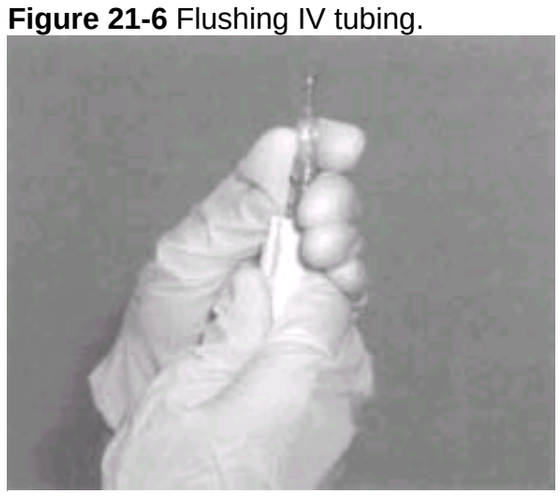


Figure 21-7 Flushing saline lock.

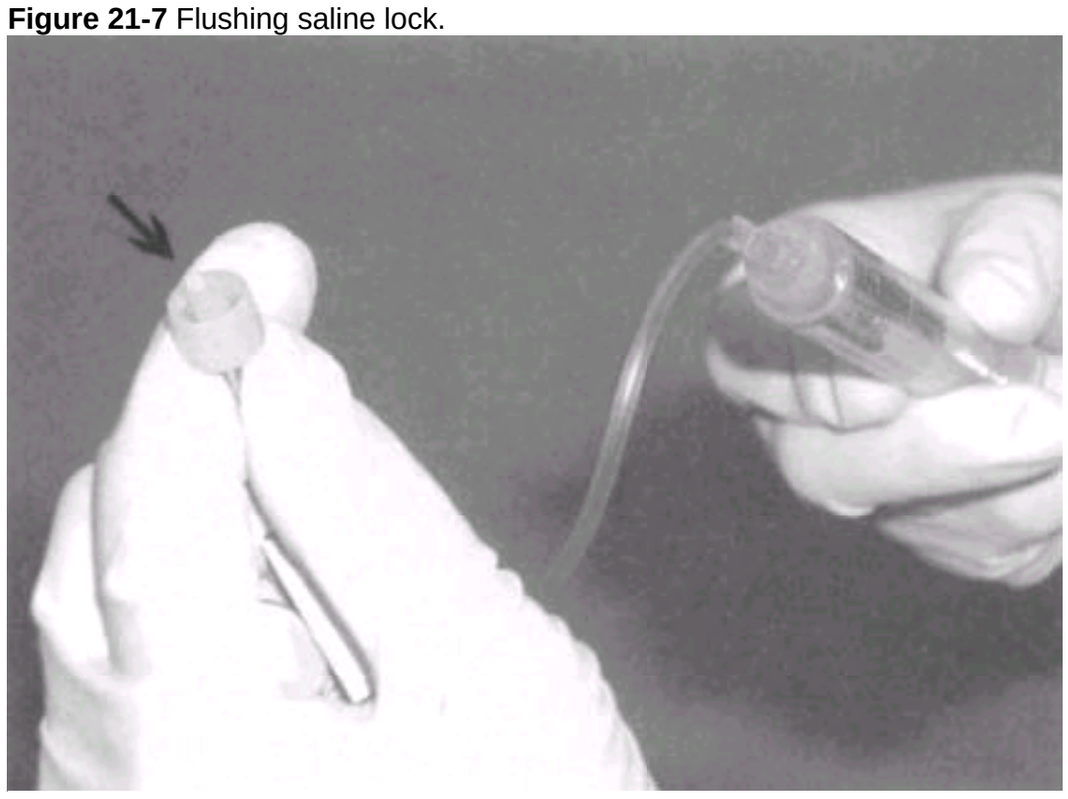


Figure 21-8 Application of tourniquet: Place tourniquet 3 to 4 cm proximal to insertion site.

Figure 21-9 Crossing tourniquet ends and applying tension.

Figure 21-10 Tucking middle portion of one end snugly under opposite end to make loop.

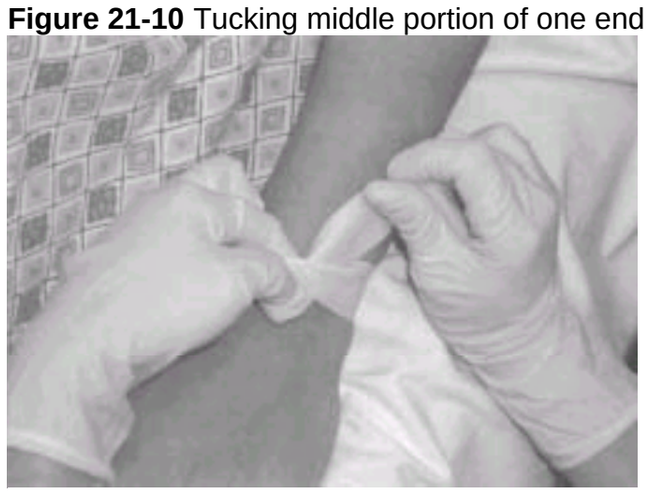


Figure 21-11 Distal portion of tucked end free for one-hand release of tourniquet.

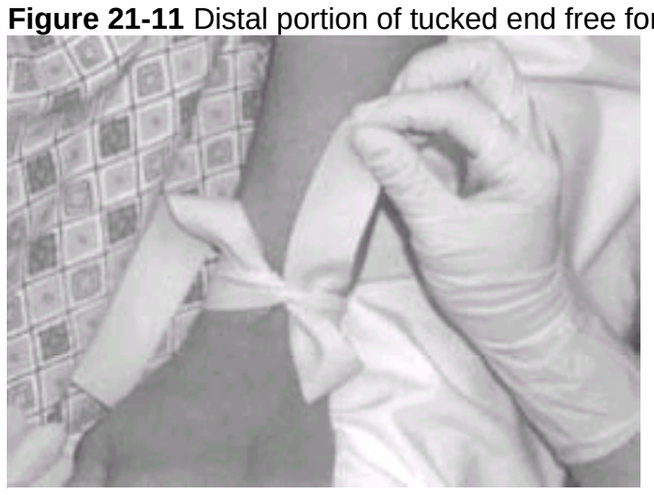


Figure 21-12 Prepping insertion site with alcohol.

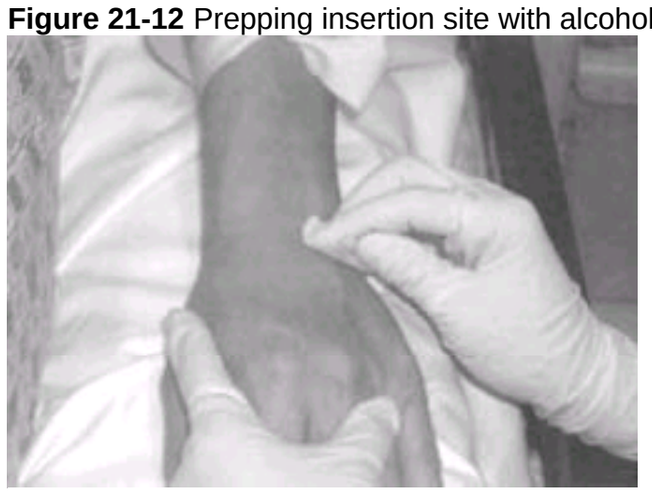


Figure 21-13 Grasping skin and pulling taut to apply traction.

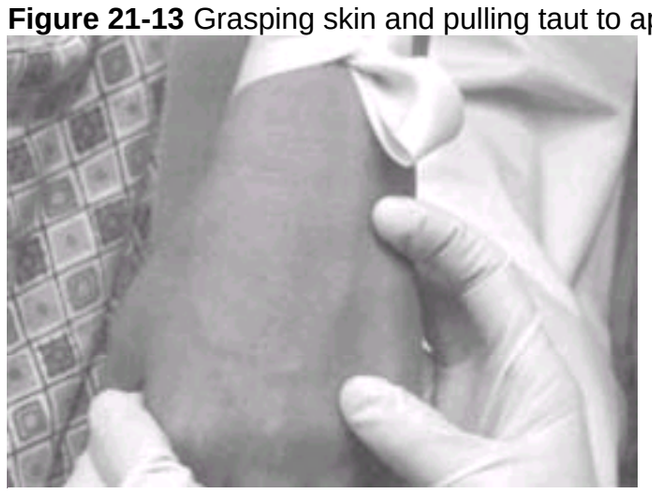


Figure 21-14 Insertion of catheter.

Figure 21-15 Advancing catheter and removing needle.

Figure 21-16 Phlebotomy.

Figure 21-17 Attaching IV saline lock.

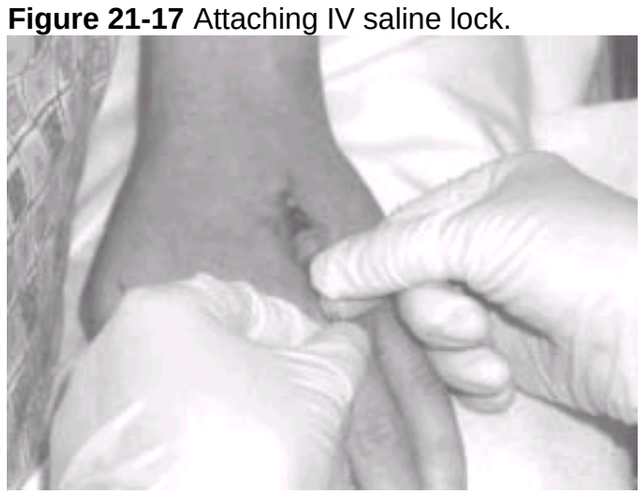


Figure 21-18 Securing IV down: placing tape under hub of catheter, sticky side up.

Figure 21-19 Crossing ends of tape over top of hub.

Figure 21-20 Transparent polyurethane dressing.

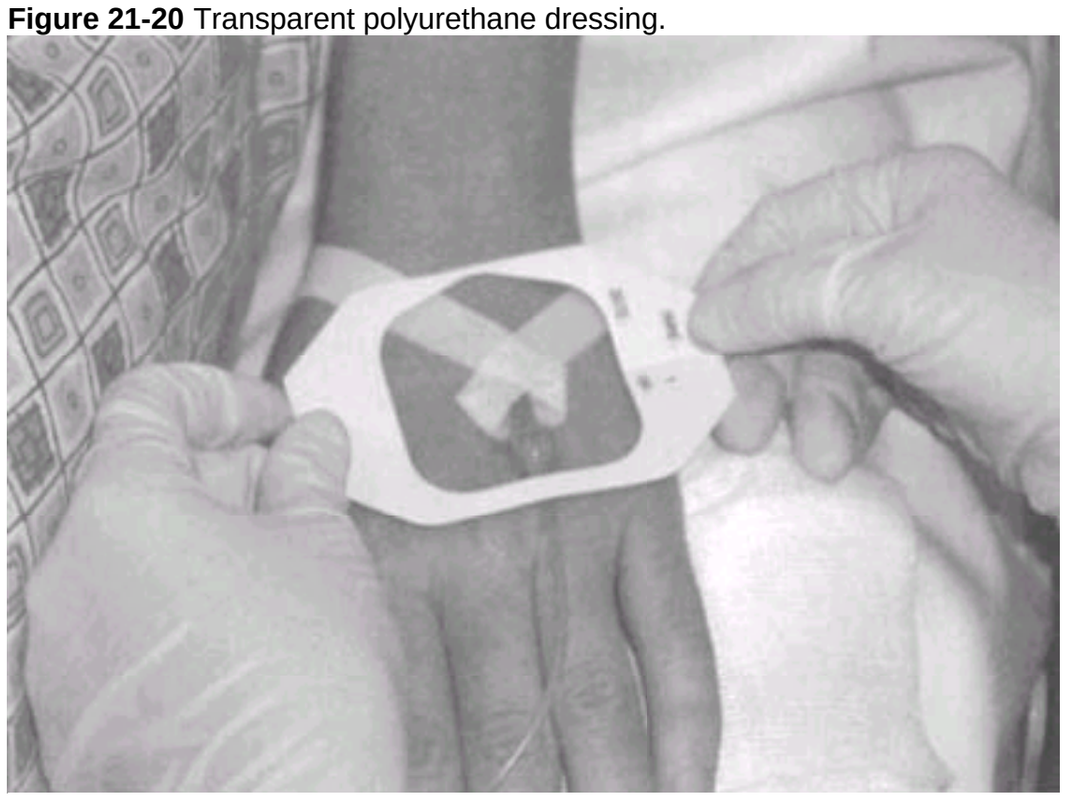


Figure 21-21 Attachment of IV saline lock to angiocatheter. This can be used for IV fluids or medications.

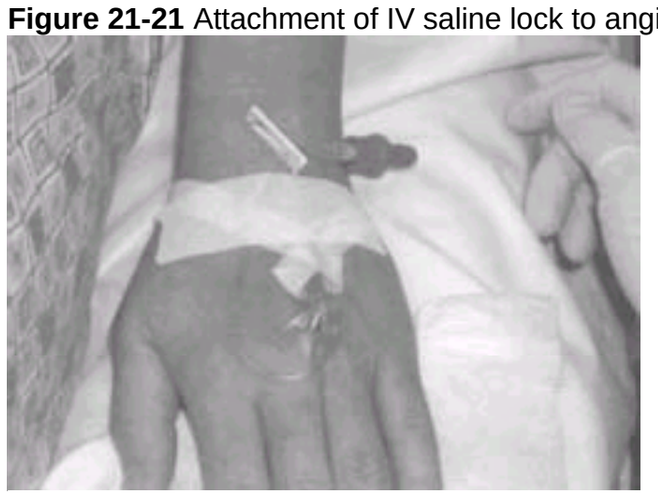


Figure 21-22 Securing the saline lock.

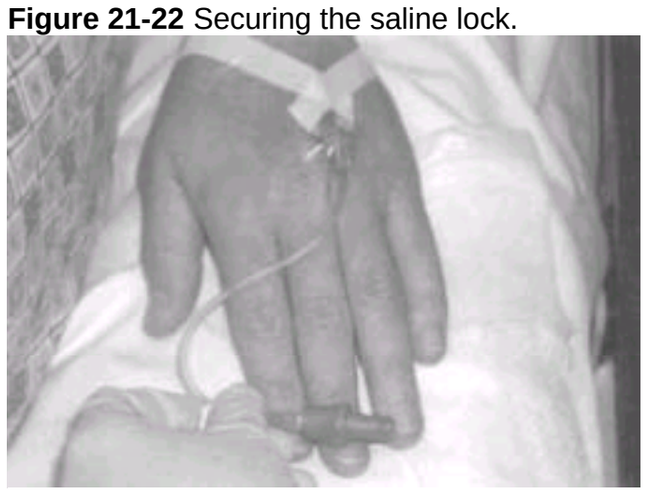


Figure 21-23 Attaching the IV tubing for infusion.

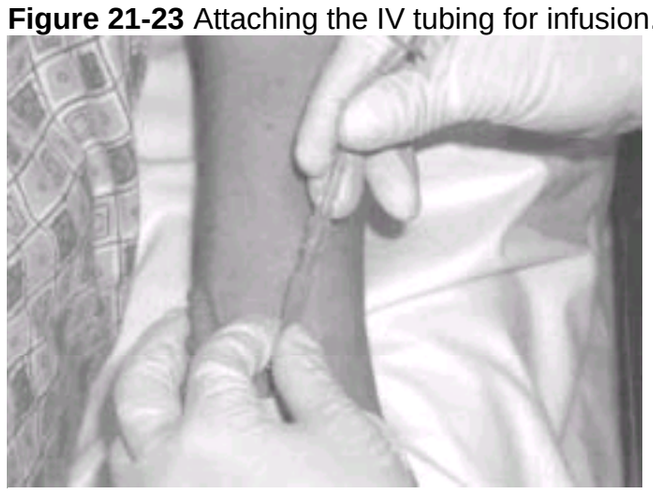


Figure 21-24 *A*, Anatomy of the vessels of the arm. *B*, Corresponding ultrasound image obtained with an Aloka 7.5-MHz ultrasound probe on the right arm. BA, brachial artery; BAV, basilic vein; DBV, deep brachial vein. *(From Keyes LE, et al: Ultrasound-guided brachial and basilic vein cannulation in emergency department patients with difficult intravenous access. Ann Emerg Med 34:711, 1999.)*

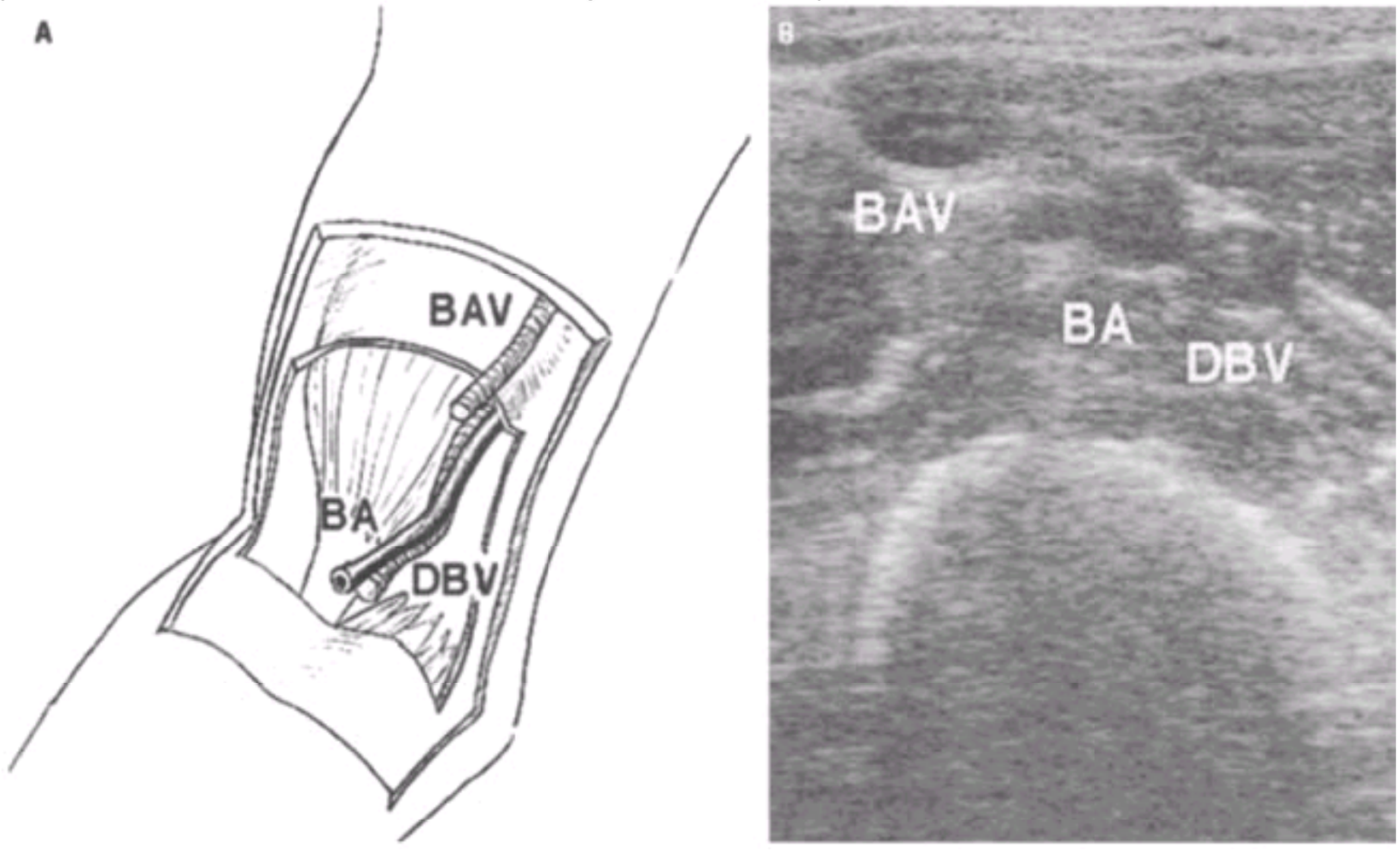


Figure 21-25 Extravasation of phenytoin. Pain at the infusion site or the alarm of an infusion pump requires inspection of the IV site for extravasation when irritating solutions are infused. In this case, an unconscious patient received a concentrated phenytoin infusion for status epilepticus, but his comatose state did not allow him to complain of pain. Over a few days, a skin slough occurred, requiring many weeks to resolve. This could have been avoided with the use of a more dilute solution (i.e., <1 mg/mL phenytoin to saline solution) or a central vein. The top panel shows the site one day after the phenytoin extravasation. The bottom panel was taken on follow up approximately a month later. *(Pictures courtesy of Dr. Mahesh Shrestha.)*

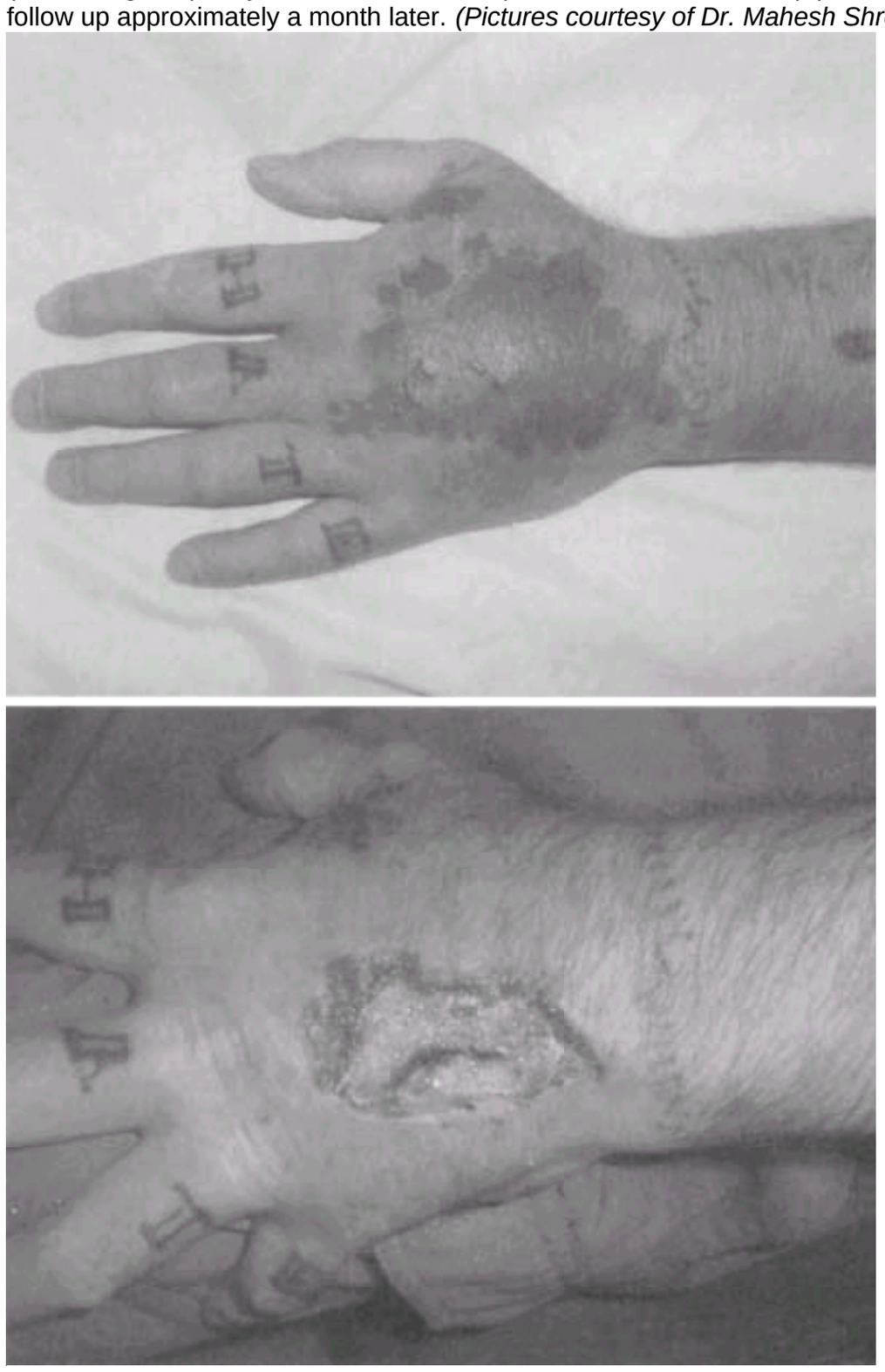
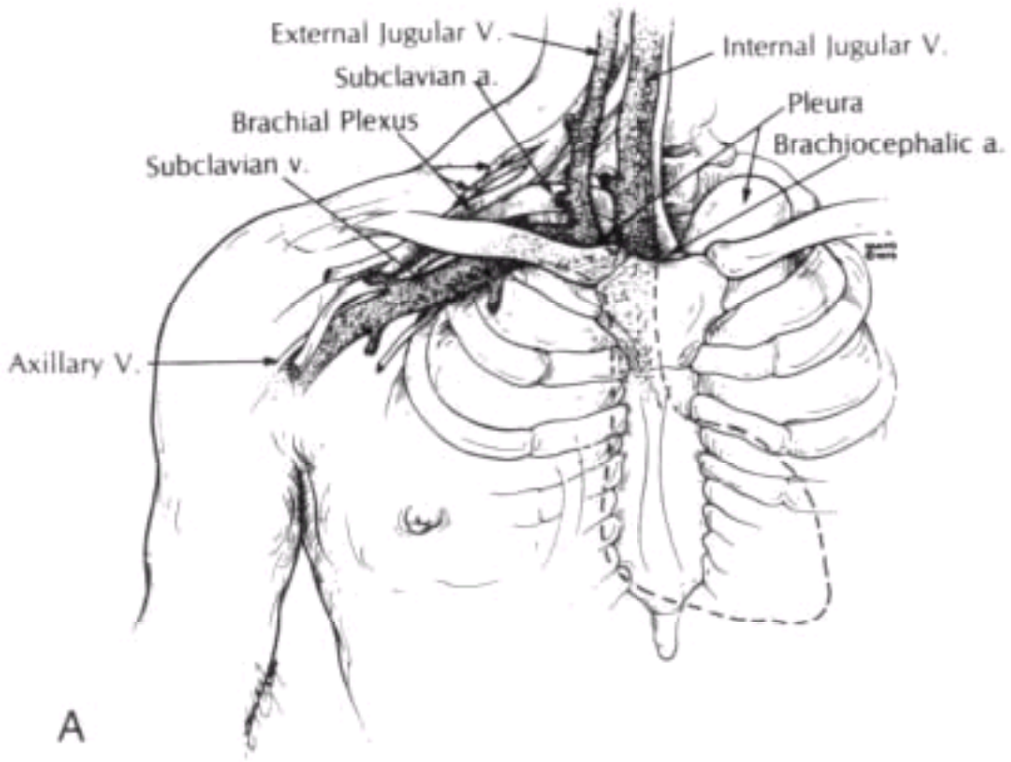


Figure 22-1 *A* and *B,* Subclavian vein and local anatomy. *C,* Subclavian area, sagittal section. *(A from Linos D, Mucha P, von Heerden J: The subclavian vein: A golden route. Mayo Clin Proc 55:316, 1980. B from Davidson JJ, Ben-Hur N, Nathan H: Subclavian venipuncture. Lancet 2:1140, 1963. C from Brahos G: Central venous catheterization: SC approach. J Trauma 17:873, 1977. Reproduced by permission.)*



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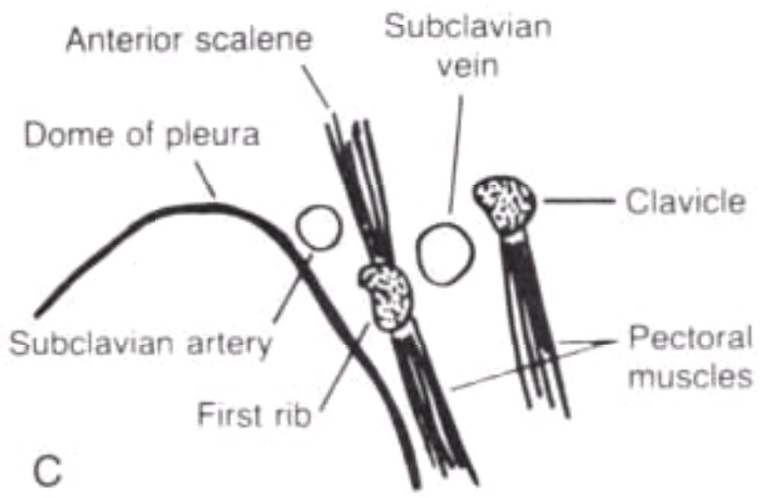

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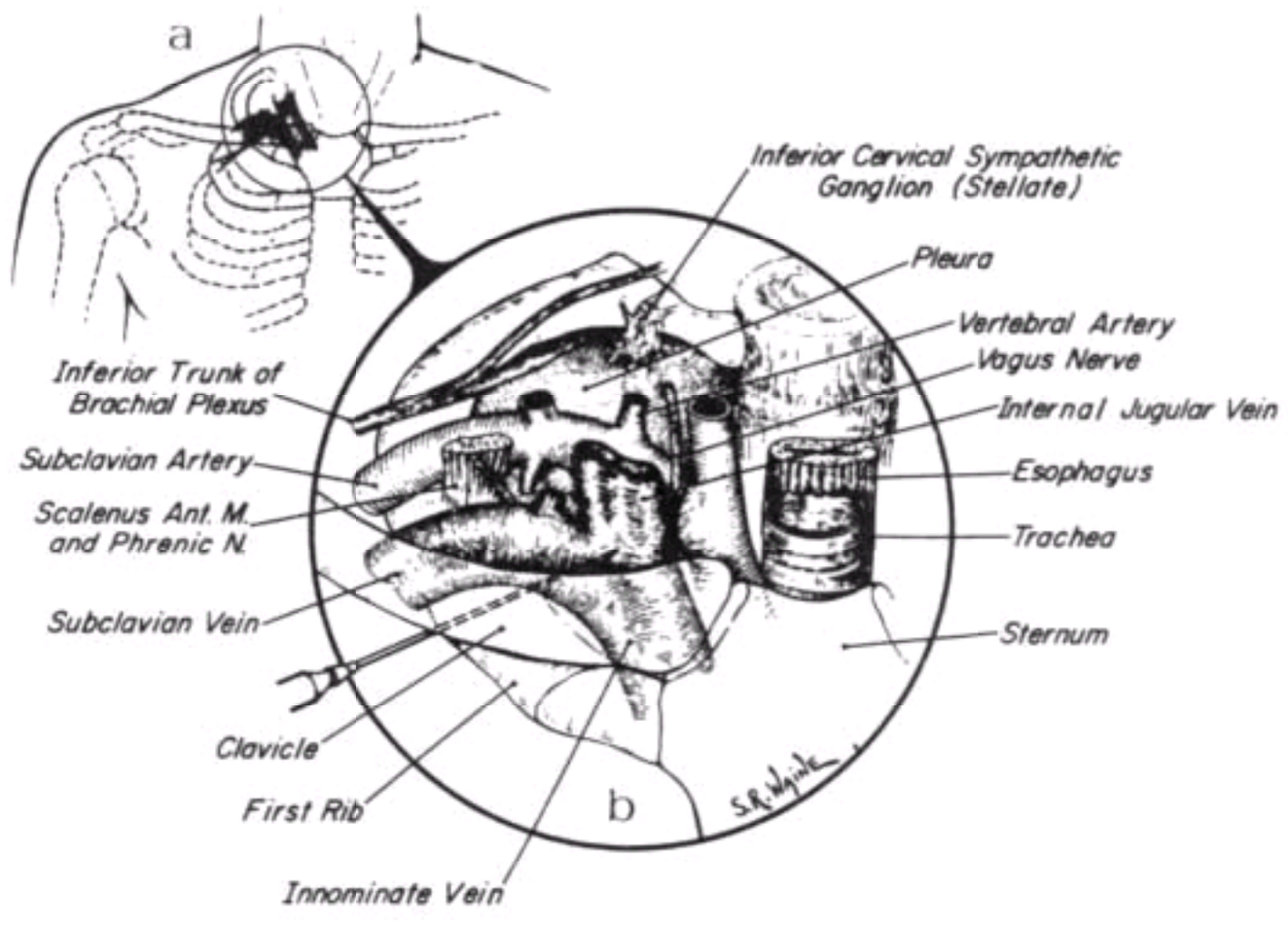


B

a, Point of insertion and direction of needle.
b, Oblique anterosuperior veiw of root of neck.

Figure 22-2 Structures in a dissection of the neck. The superficial veins and the sternocleidomastoid muscle have been removed, as have the submandibular gland and a segment of the facial vein. The cutaneous nerves have been cut down to short stumps arising from the second, third, and fourth cervical nerves. The internal jugular vein is drawn somewhat more medial in this illustration than is commonly found. *(From Hollinshead WH: Textbook of Anatomy, 3rd ed. New York, Harper & Row, 1974, p 765. Reproduced by permission.)*

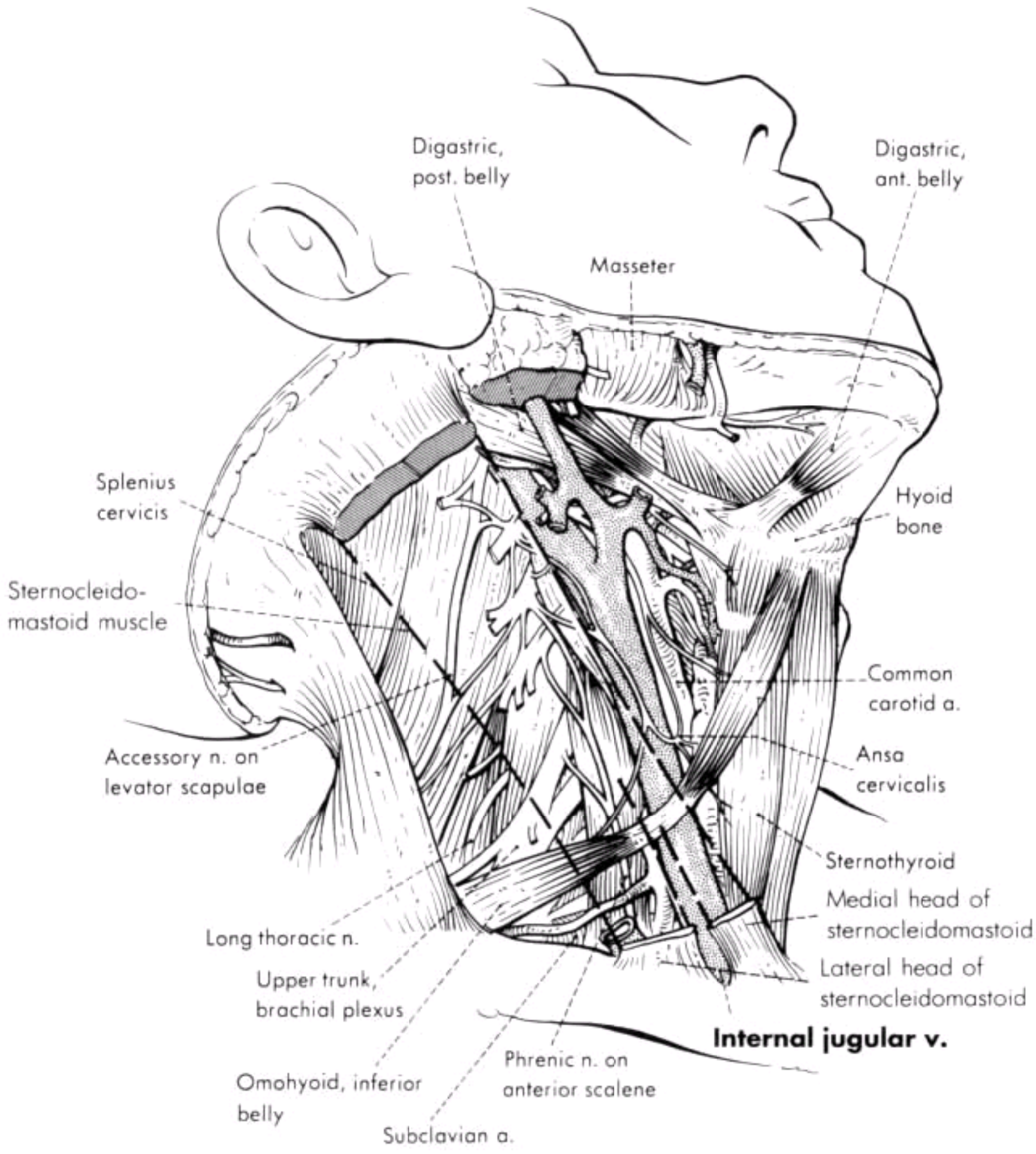


Figure 22-3 The right femoral vessels and some of their branches. The femoral nerve (not shown) lies lateral to the artery and may be deep to the artery. *(From Warwick R, Williams PL [eds]: Gray's Anatomy, 35th ed. Edinburgh, Churchill Livingstone, 1973, p 676. Reproduced by permission.)*

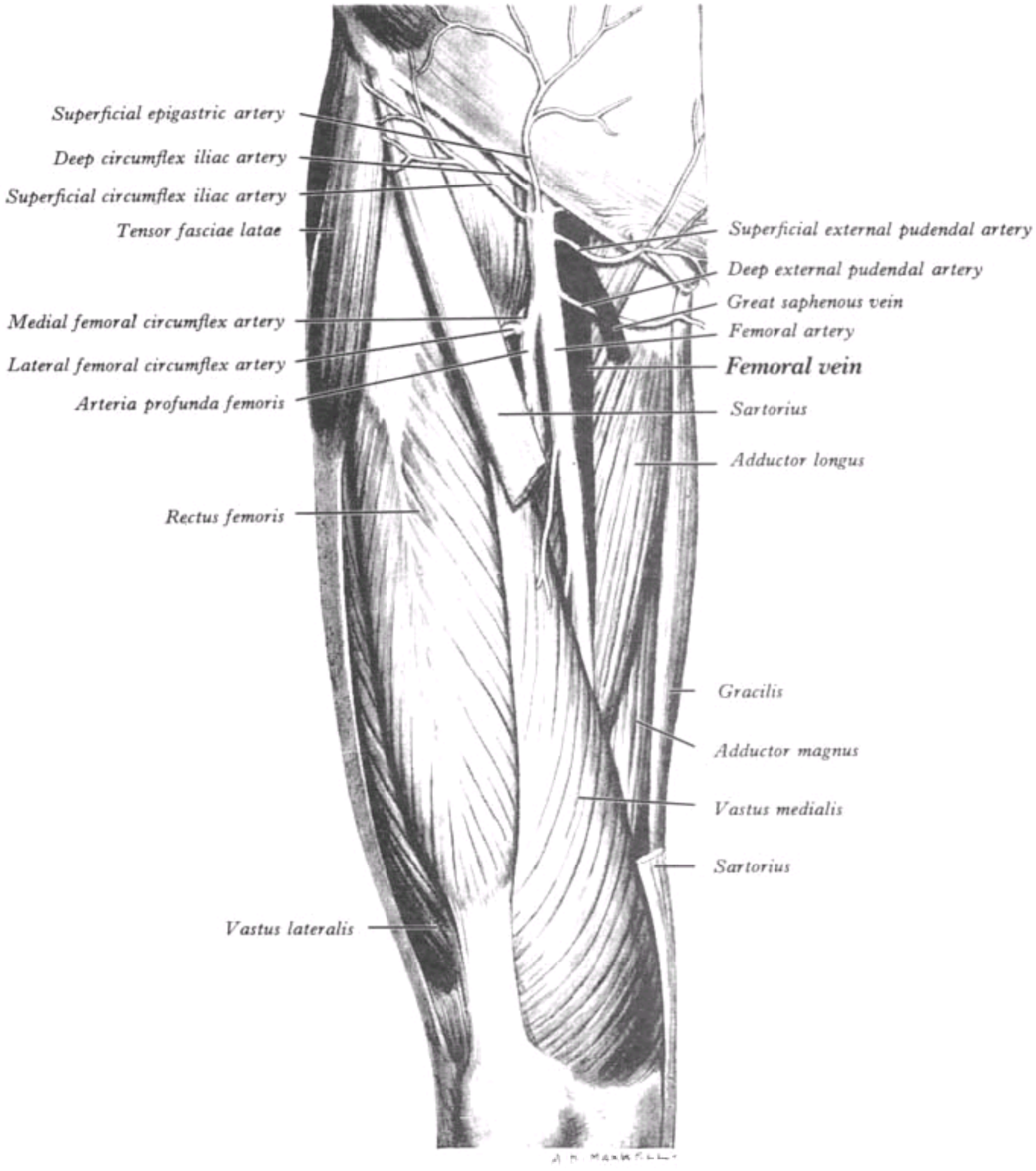


Figure 22-4 Major veins of the upper half of the body. *(From Hedges JR: Vascular access. Curr Top Emerg Med 2:1, 1981. Reproduced by permission.)*

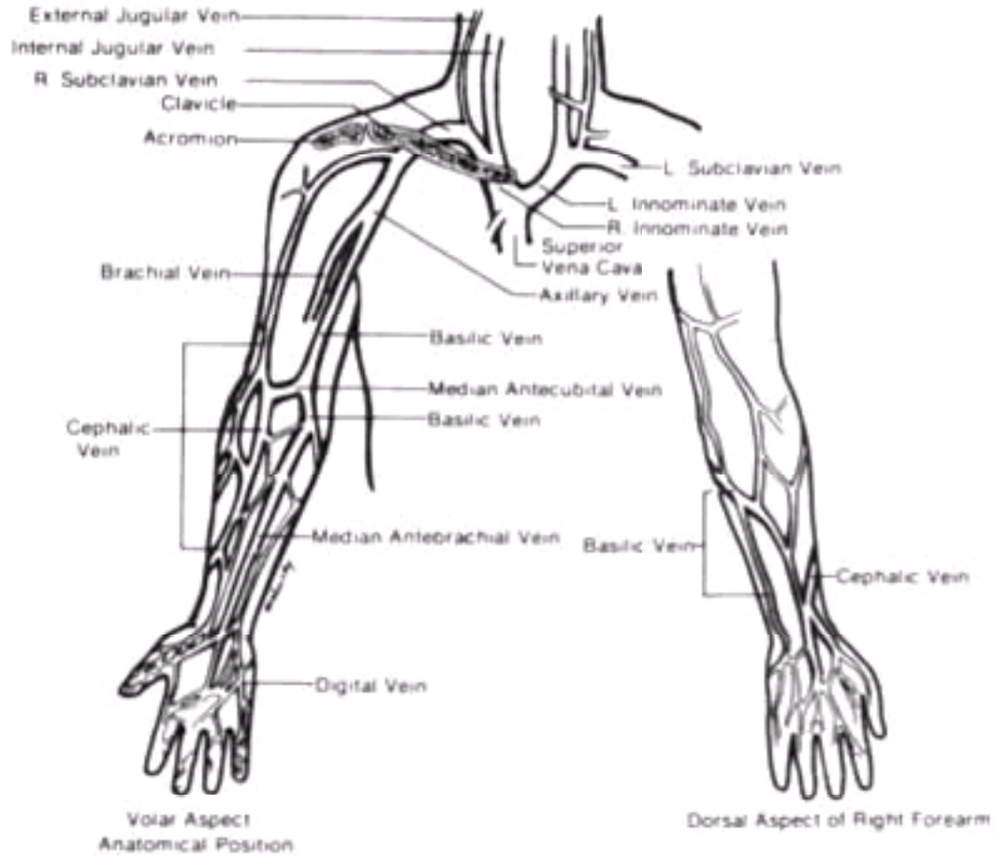
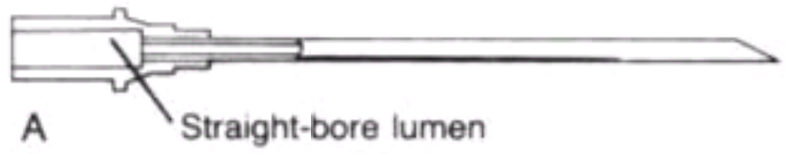


Figure 22-5 Introducing needles. *A,* Ordinary needle with a straight-bore lumen. *B,* Seldinger needle with a tapered lumen, allowing easy entry of the guidewire.



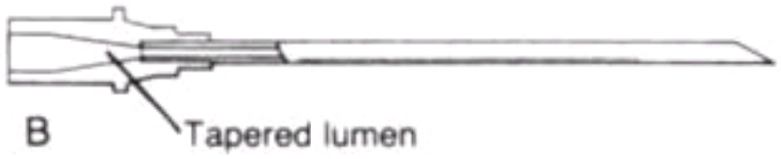


Figure 22-6 *A*, Guidewire internal structure: safety wire (a), core wire (mandrel) (b), coiled wire (c), flexible tip (d). *B*, Flexible end of a straight-spring guidewire knotted on a vessel dilator (a), bent junction of the rigid and flexible portions of a straight-spring guidewire with protrusion of the central core (arrow) (b), partially fractured tip (arrow) of a J-spring guidewire (c). *(From Schwartz AJ, Horrow JL, Jobes DR, Ellison N: Guide wires—A caution. Crit Care Med 9:348, 1981. ©1981 Williams & Wilkins, Baltimore. Reproduced by permission.)*

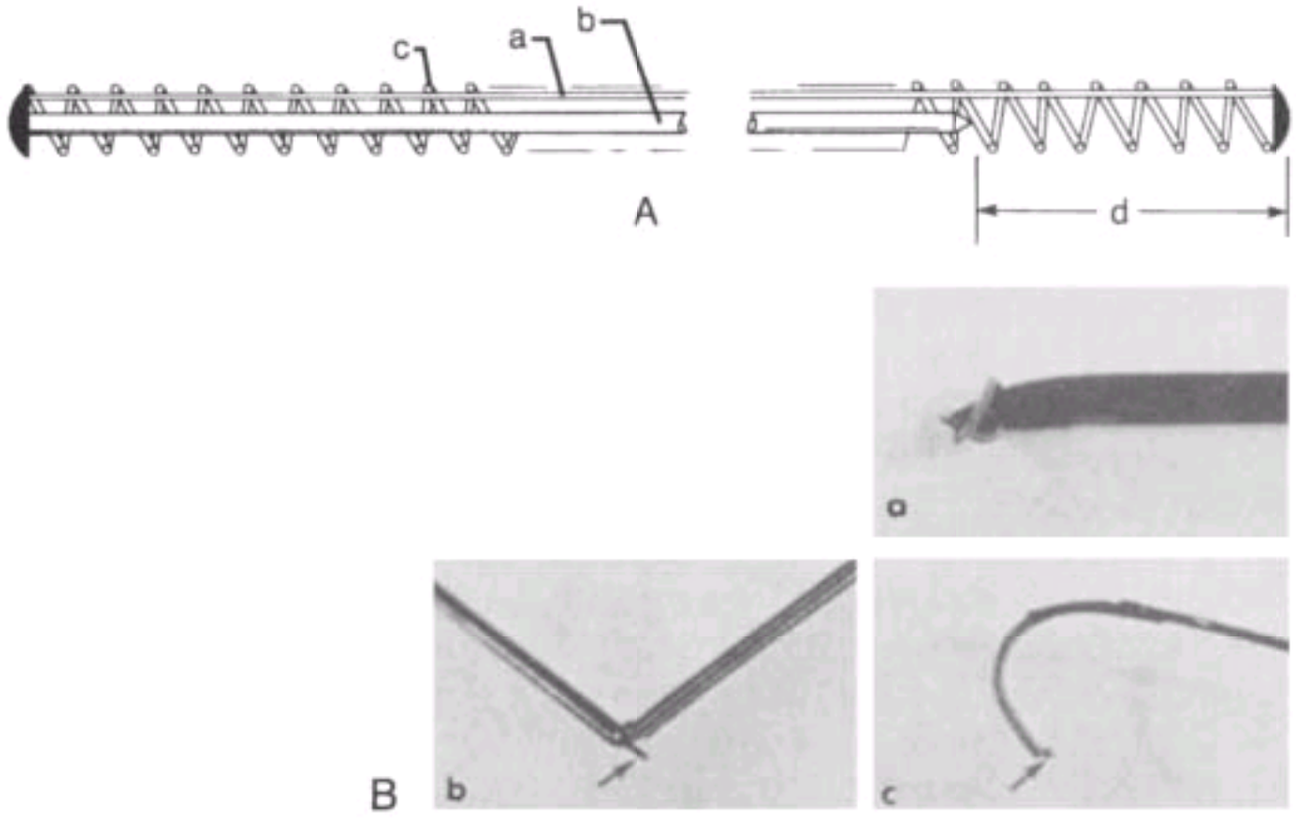


Figure 22-7 Desilets-Hoffman sheath introducer. *A*, Guidewire. *B*, Sheath-introducer. *C*, Dilator. *D*, Assembled device.

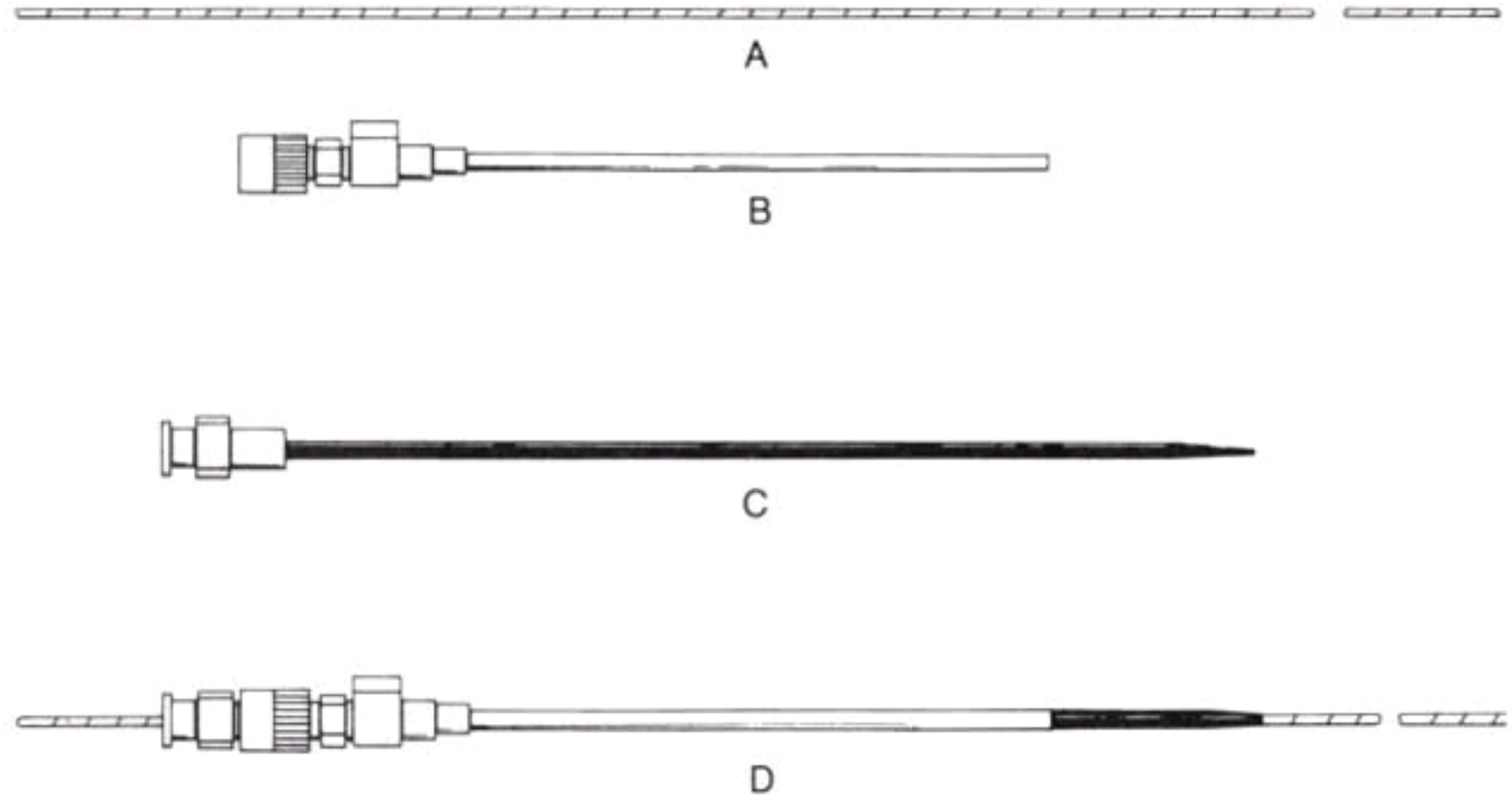


Figure 22-8 Procedure for placement of Seldinger-type guidewire catheter. *A*, The selected vessel is cannulated with a thin-walled needle, or an existing IV catheter is chosen to be changed with the wire technique. *B*, The guidewire is threaded through the vessel, with the flexible end first, into the lumen of the vessel. If a J wire is used, a sleeve will facilitate entry into the needle (see Fig. 22-9). *C*, The needle is removed so that only the wire now exits from the vessel. *D*, The skin entry site is enlarged with a No. 11 scalpel. *E*, The catheter sheath and the dilator are threaded over the wire and advanced to the skin. The wire must be visible through the back of the device. *F*, If the proximal wire is not visible, it is pulled from the skin through the catheter until it appears at the back of the catheter. *G*, The sheath and the dilator are advanced as a unit into the skin with a twisting motion. It is best to grasp the unit at the junction of sheath and dilator to prevent bunching up of the sheath. The wire (at the back of the catheter) must be held while the sheath and dilator are advanced as a unit. *H*, Once the sheath and the dilator are well within the vessel, the guidewire and the dilator are removed. Contrary to the illustration, latex gloves should be worn.

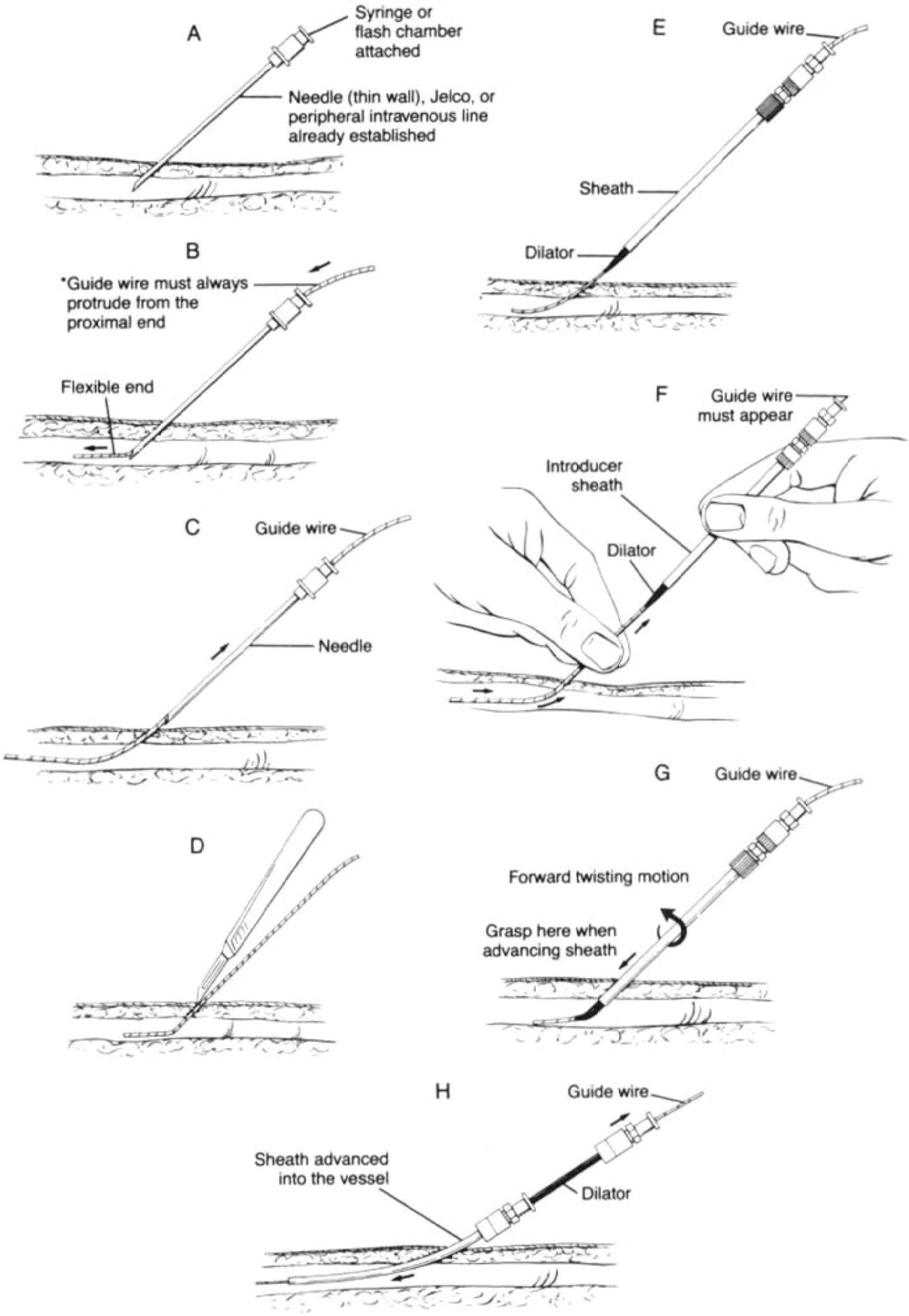


Figure 22-9 J wire. *A*, Plastic sleeve in retracted position, demonstrating the J tip. *B*, Plastic sleeve is advanced to straighten the curve to allow easy introduction into the needle hub. In an emergency, care must be taken to not misplace or throw away the sleeve. Without it, placing the J wire into the hub of the needle is very difficult.

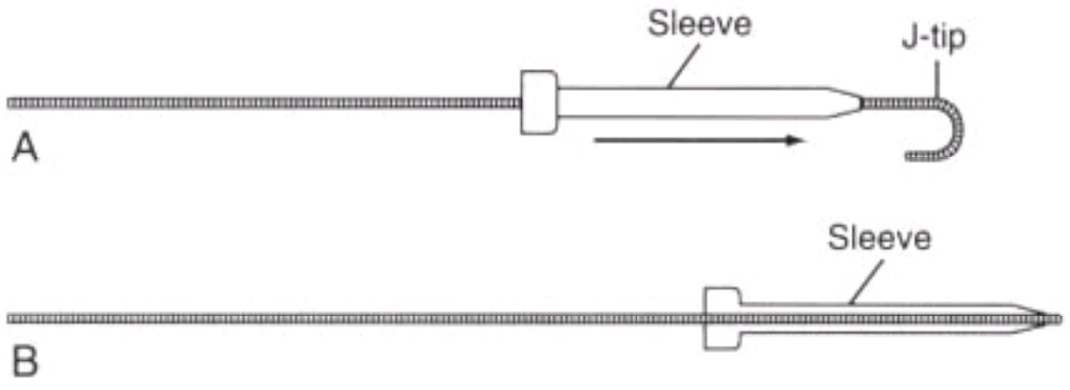
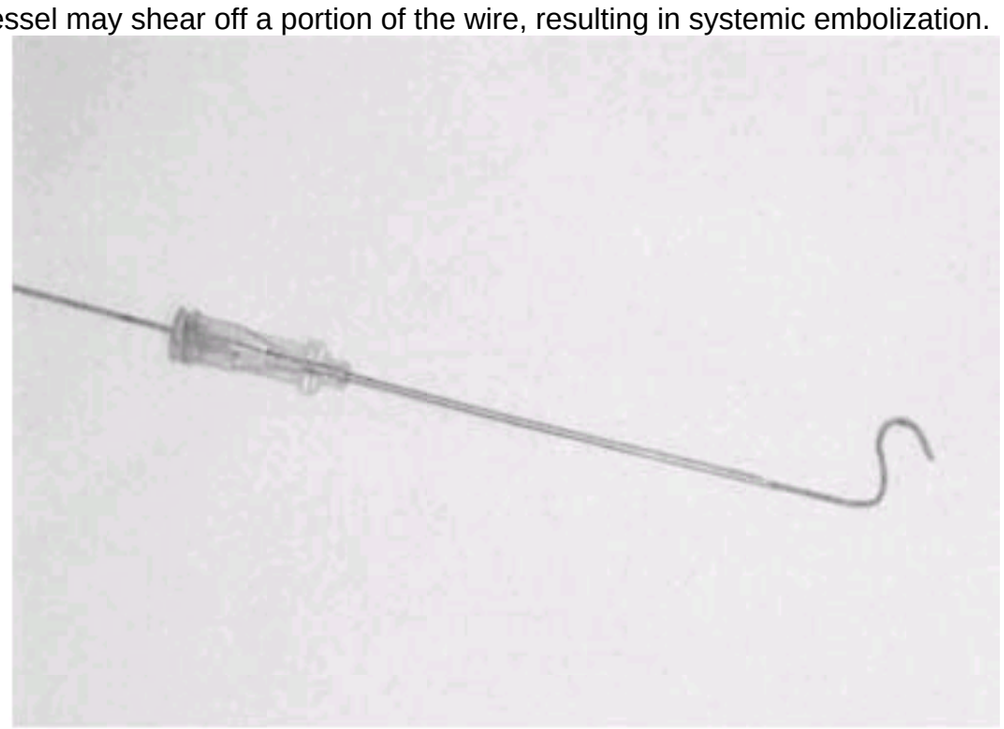


Figure 22-10 Although newer guidewires are more resistant to shearing, if a guidewire will not advance, it is best to withdraw both the needle and wire in one motion. These pictures demonstrate a permanently deformed guidewire that could not be advanced. Withdrawing the wire with the indwelling introducer needle in place within a vessel may shear off a portion of the wire, resulting in systemic embolization.

A



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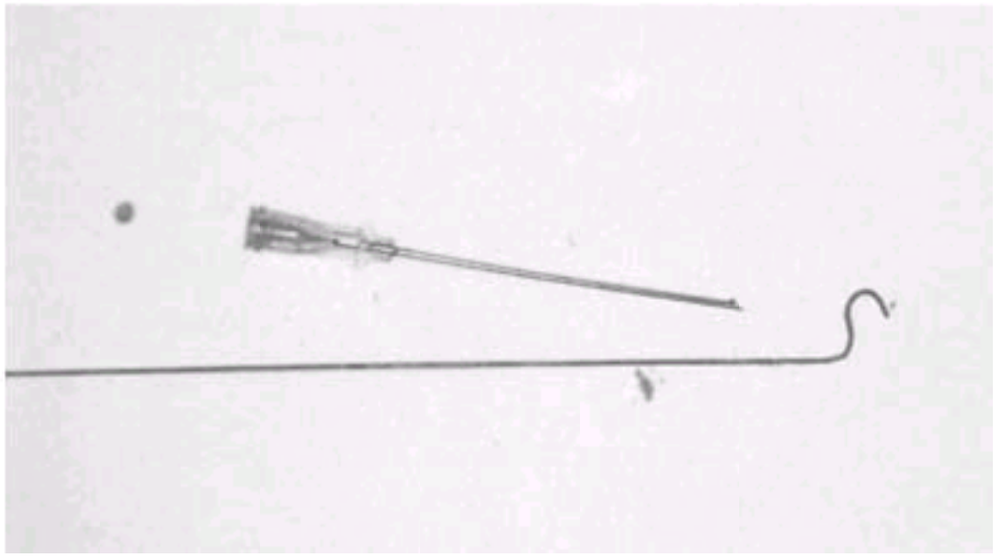


Figure 22-11 Needle bevel orientation using supraclavicular and infraclavicular venipuncture. The orientation of the needle bevel may help in positioning the catheter properly by guiding the direction of the catheter during advancement. If the bevel is aligned with the markings on the syringe, the orientation of the bevel is always certain.

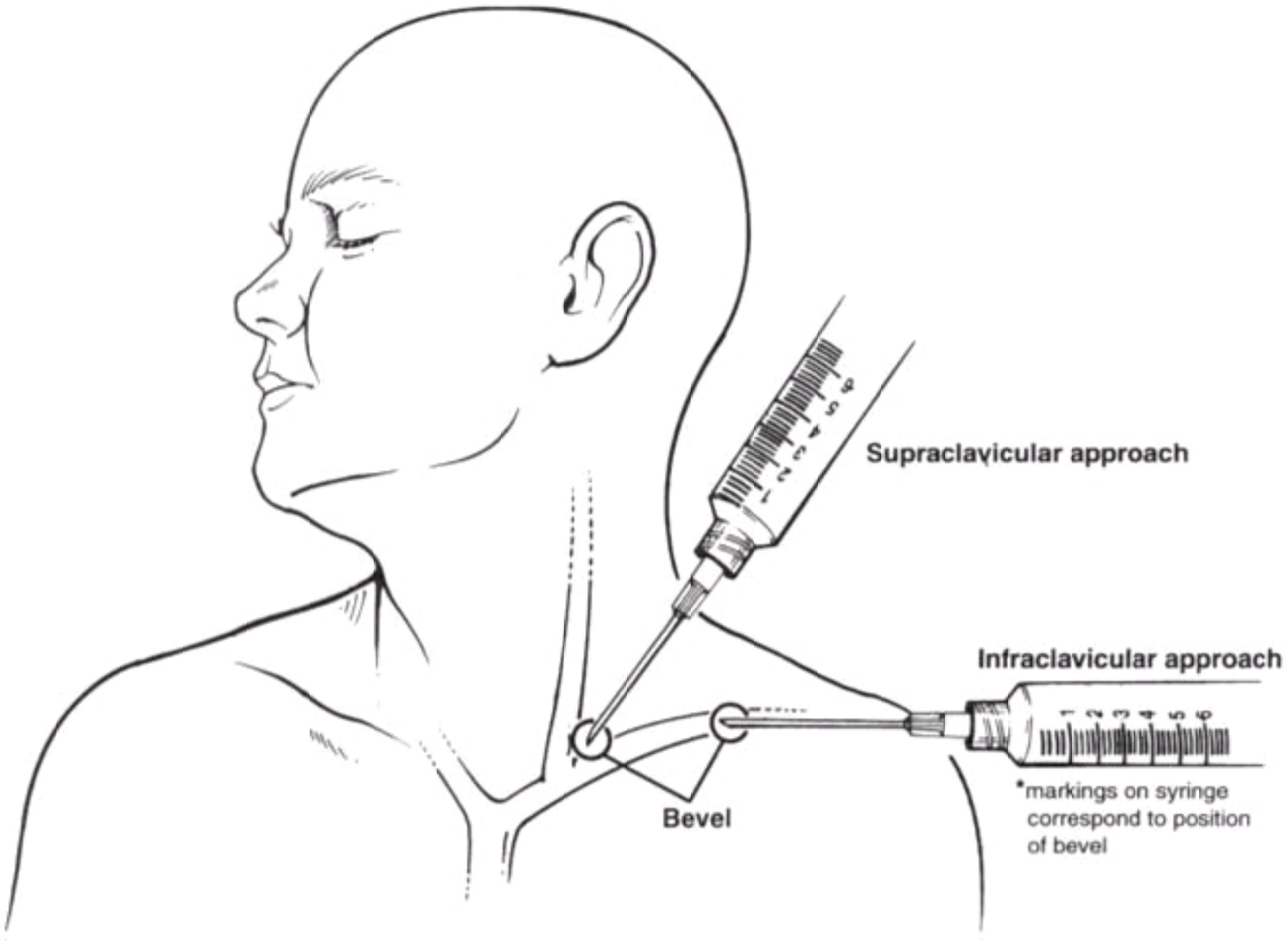


Figure 22-12 Hand position during subclavian venipuncture. Note that surgical gloves should be worn during this procedure, in contrast to the illustration. *(From Linos D, Mucha P, von Heerden J: Subclavian vein: A golden route. Mayo Clin Proc 55:318, 1980. Reproduced by permission.)*

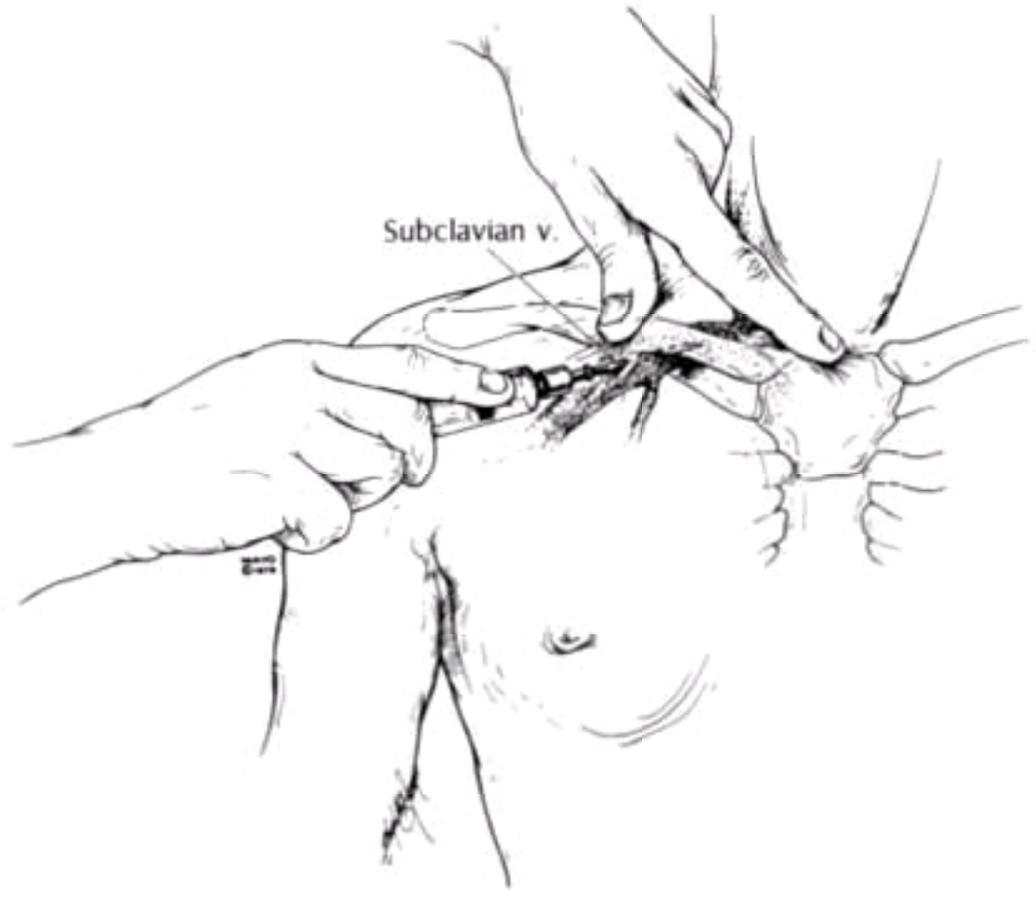
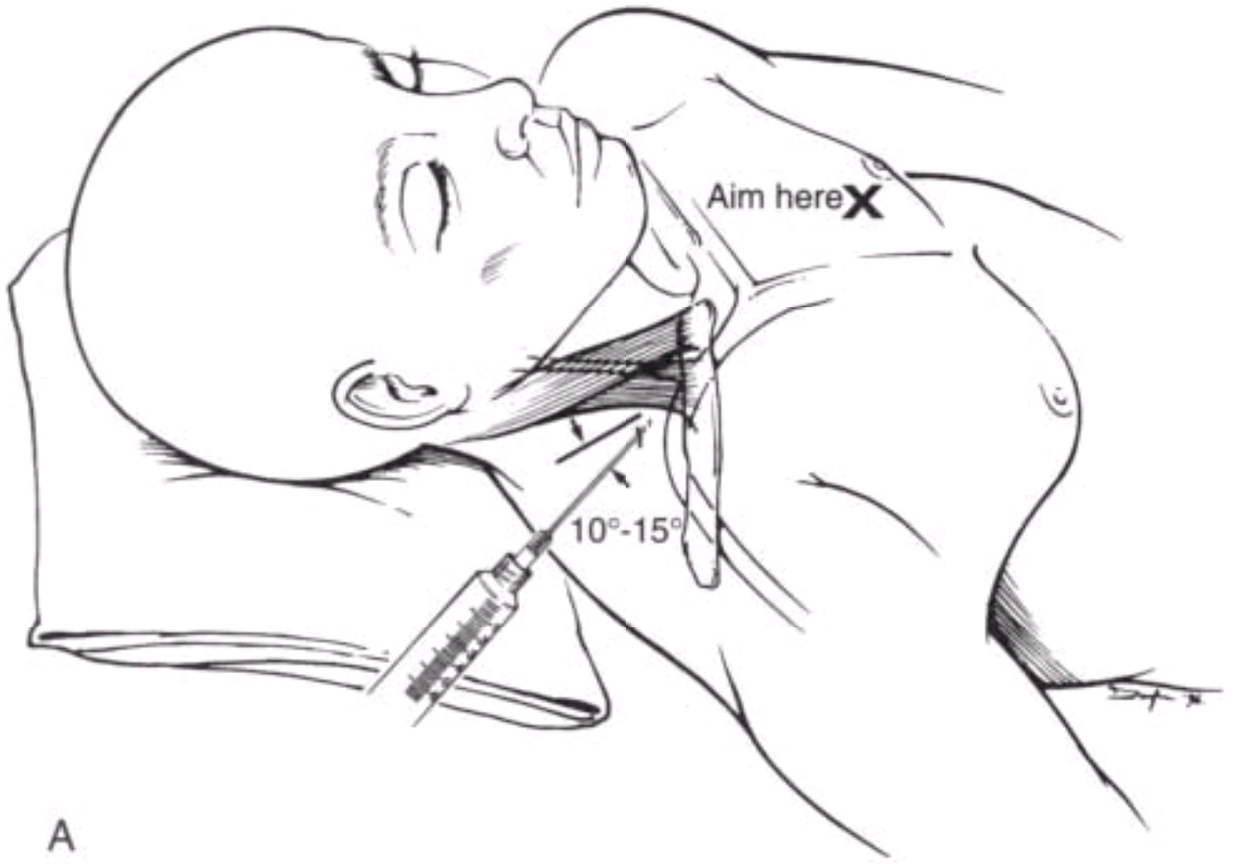
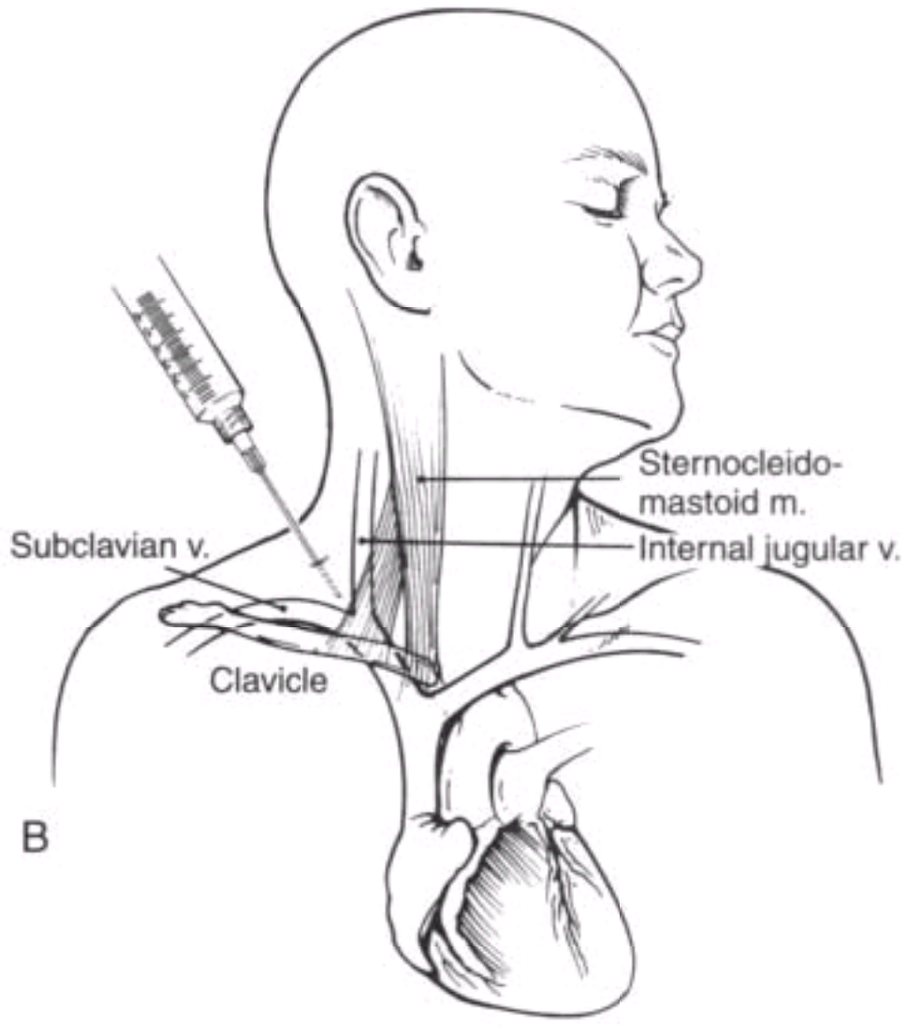


Figure 22-13 *A* and *B*, For the supraclavicular approach, the needle is inserted above and behind the clavicle, bisecting the angle made by the clavicle and the lateral border of the sternocleidomastoid muscle (clavisternomastoid angle). The point of entry is 1 cm lateral to the clavicular head of the muscle and 1 cm posterior to the clavicle. The needle traverses an avascular plane, puncturing the junction of the subclavian and internal jugular vein behind the sternoclavicular joint. The right side is preferred because of a direct route to the superior vena cava and the absence of the thoracic duct. The needle is directed 45° from the sagittal plane and 10° to 15° upward from the horizontal plane, aiming toward the contralateral nipple. Note that the vein is just posterior to the clavicle at this juncture. *C*, Sagittal view of the supraclavicular area. As the subclavian vein passes over (and somewhat anterior to) the first rib, it is separated from the subclavian artery by the anterior scalene muscle. The dome of the pleura is posterolateral to the confluence of the great veins.





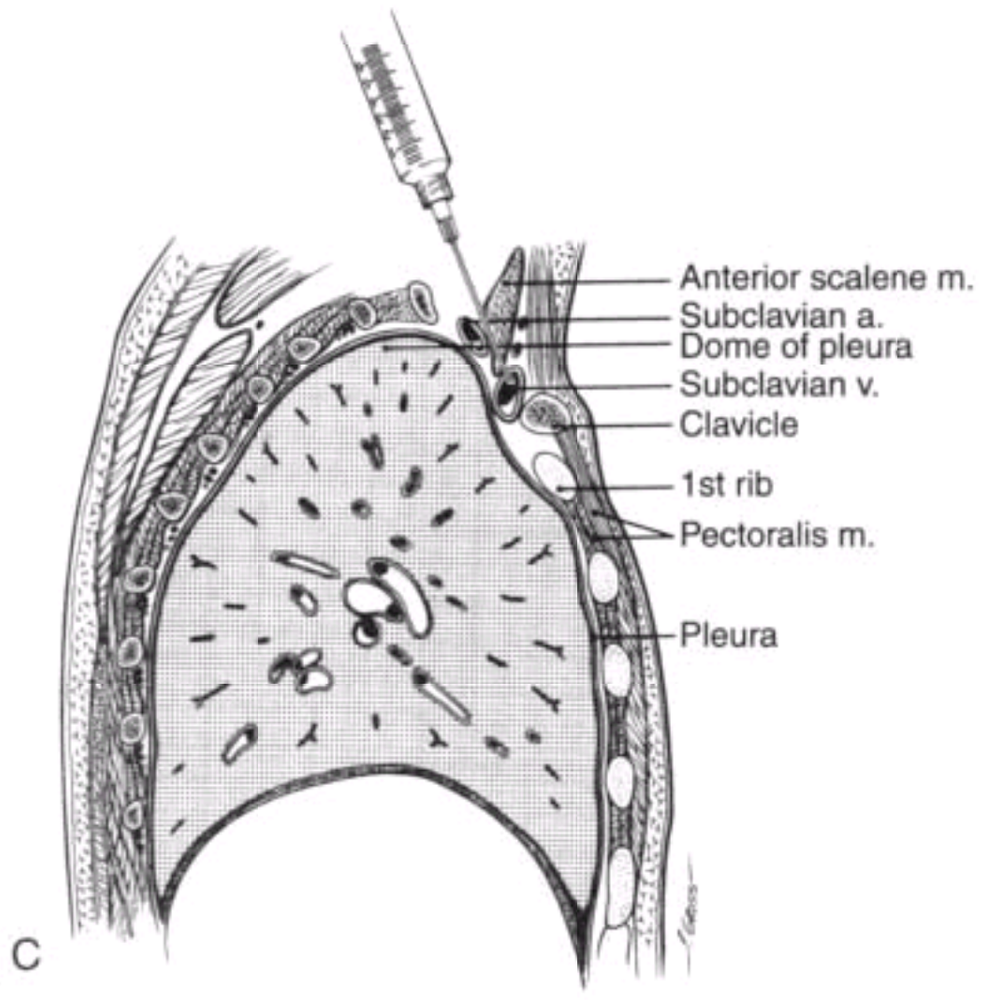
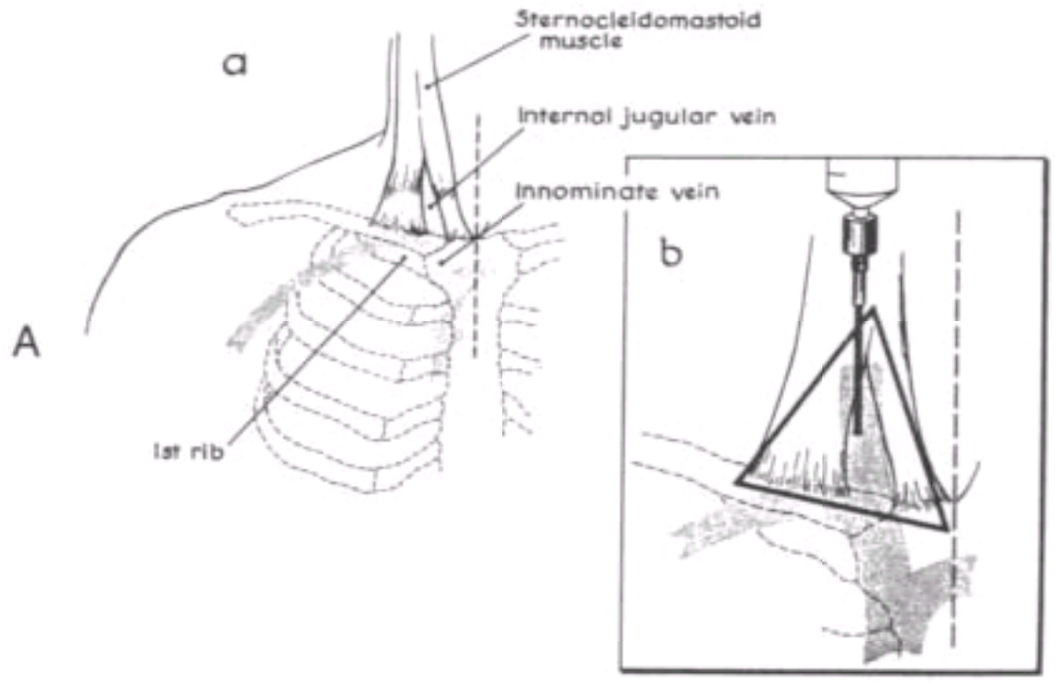
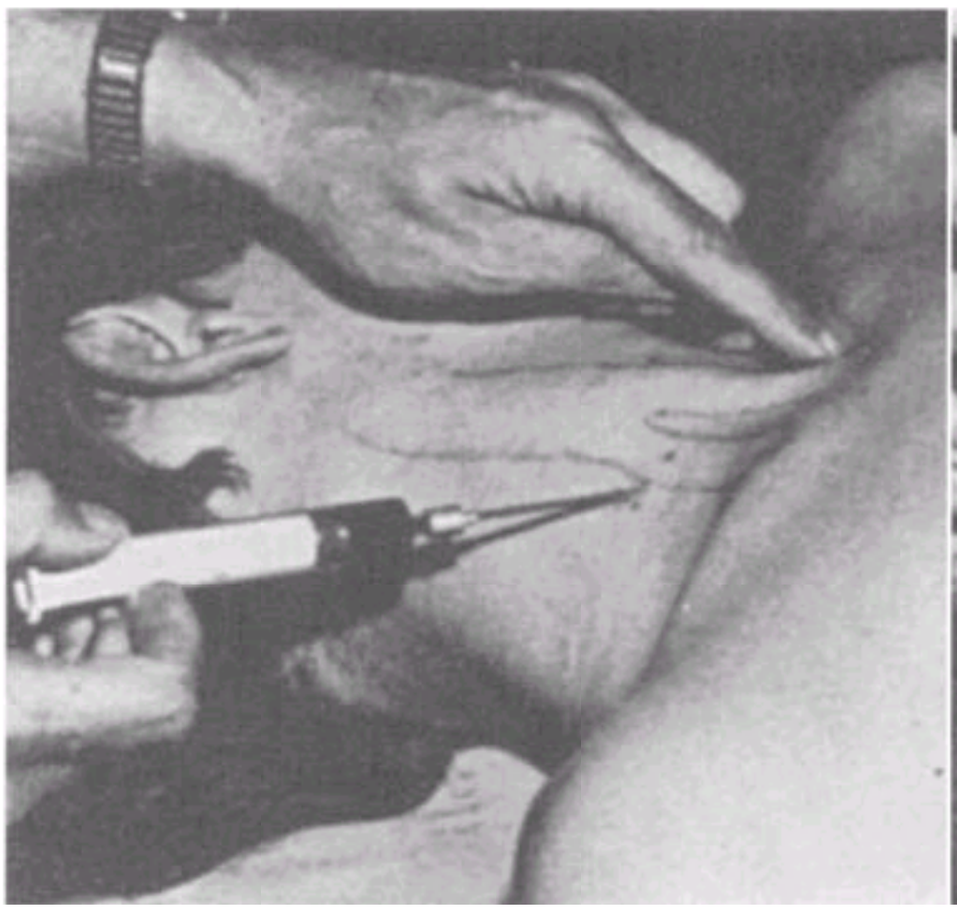


Figure 22-14 Internal jugular vein approaches. *A*, Central approach. *B*, Posterior approach. *C*, Anterior approach. Note that contrary to the photographs, gloves should be worn during the procedure.





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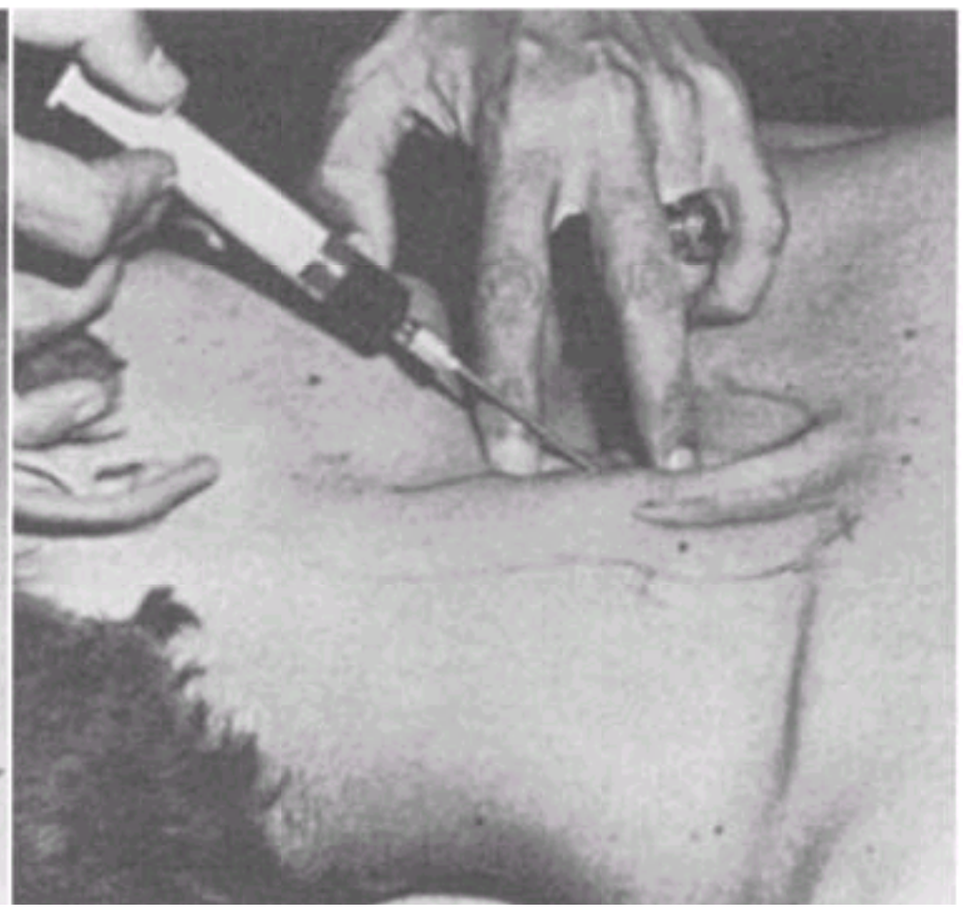


C

Figure 22-15 Insertion of a catheter over a wire via the external jugular vein. Successful passage may require many attempts and manipulations of the J wire to navigate turns and valves. *(From Blitt CD, Wright WA, Petty WC: Central venous catheterization via the external jugular vein, a technique employing the J-wire. JAMA 229:817, 1974. Reproduced by permission.)*

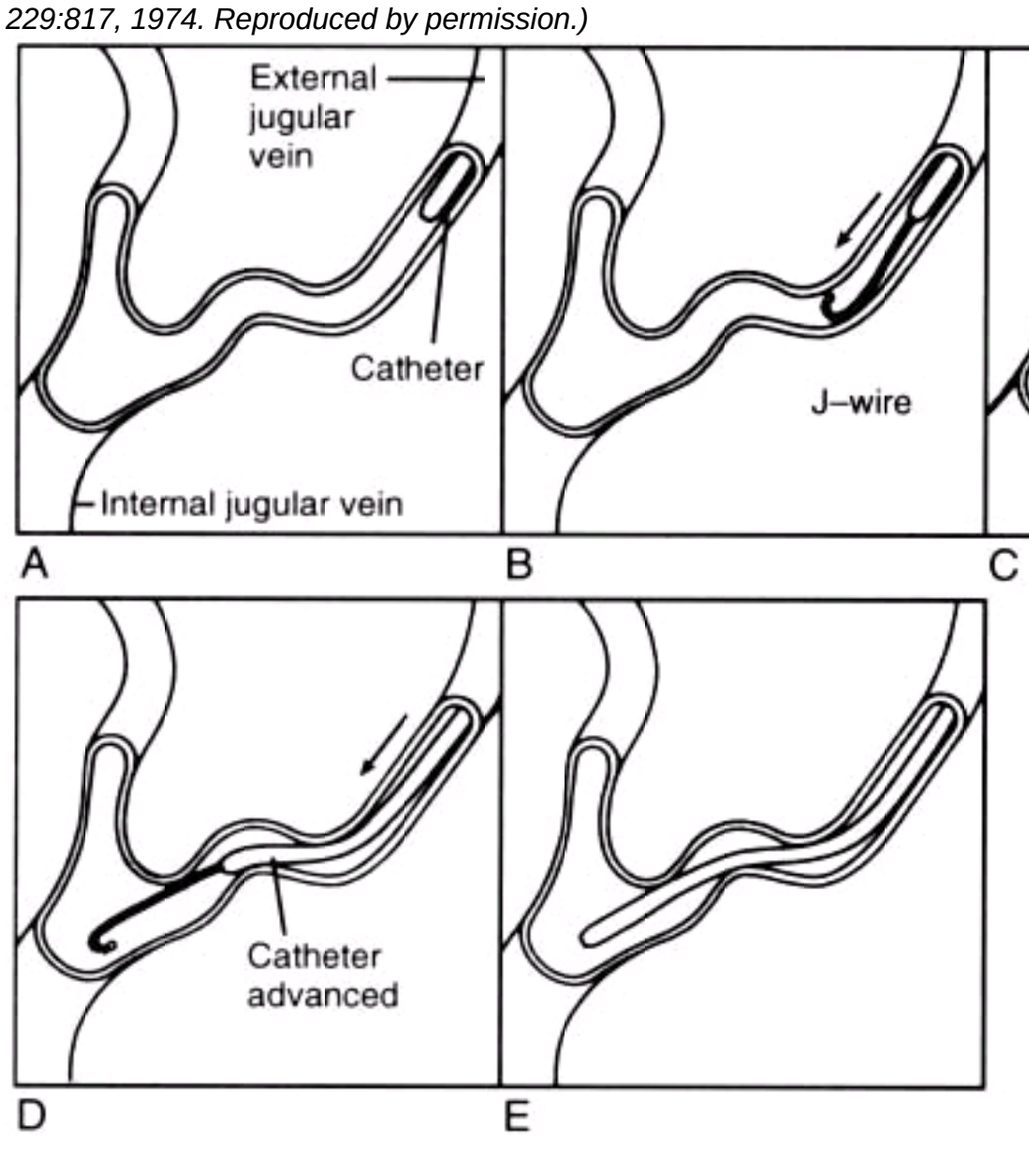


Figure 22-16 Internal jugular line secured by looping around the ear. *(From Boulanger M, et al: Une nouvelle voie d'abord de la veine jugulaire interne. Can Anaesth Soc J 23:609, 1976. Reproduced by permission.)*

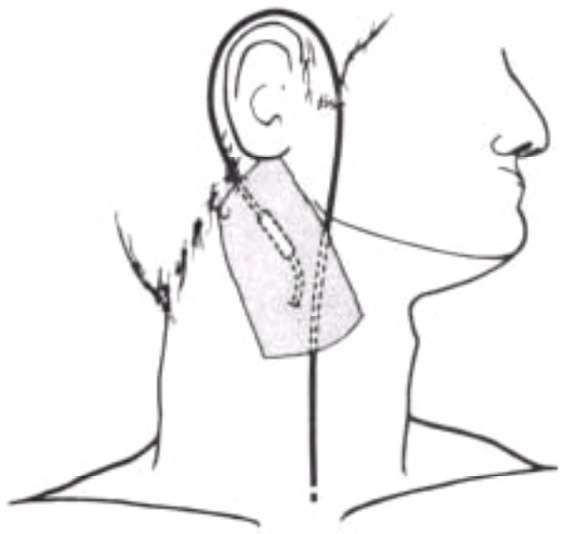


Figure 22-17 A chest film showing the proper catheter tip placement in the superior vena cava *(arrow)*. The tip should not lie within the right atrium or the right ventricle.

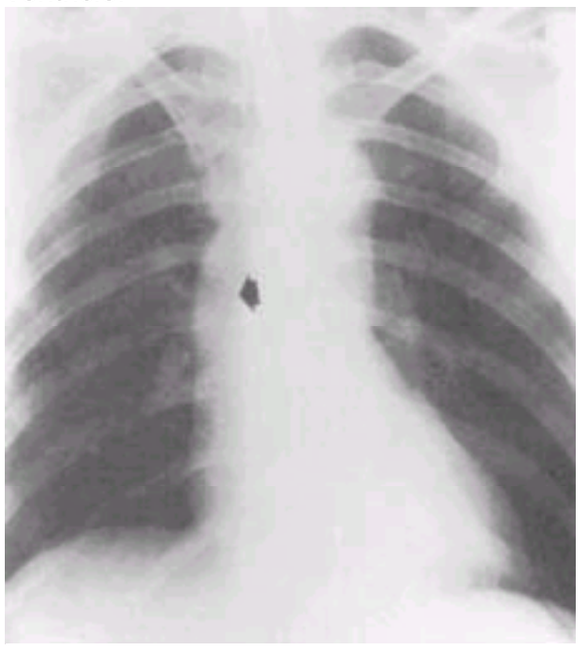


Figure 22-18 A chest radiograph should be routinely taken to assess position of a central catheter introduced via the chest or neck. *A*, In this case a poorly positioned patient produced a rotated and oblique film, and the catheter (black arrow) appeared, at first glance, to be in the correct position in the right subclavian vein. The supine position of the patient did not allow identification of an early hydrothorax (white arrow). *B*, A repeat radiograph shows the obvious intrapleural position of the catheter, and a large hydrothorax after infusion of 2 liters of saline.

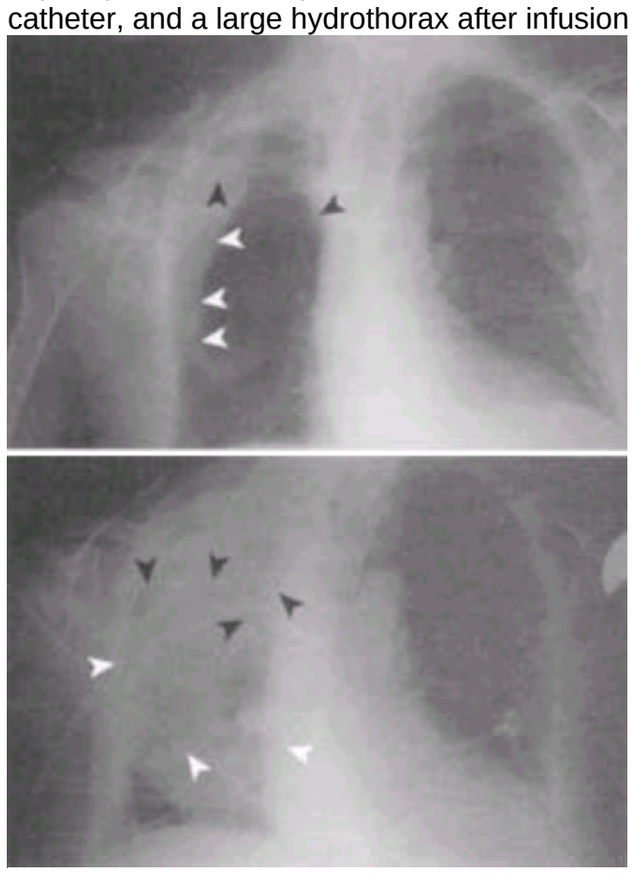


Figure 22-19 *A*, Surface ultrasound-directed central vein identification. A handheld transducer allows noninvasive localization of veins, in this case the subclavian. The device shown includes a needle guide, which allows simultaneous visualization and penetration of a targeted vessel. *B,* Surface ultrasound image of the subclavian artery (left) and vein (right). *(Courtesy of Irene R. Skolnick, Dymax Corp, Pittsburgh.)*

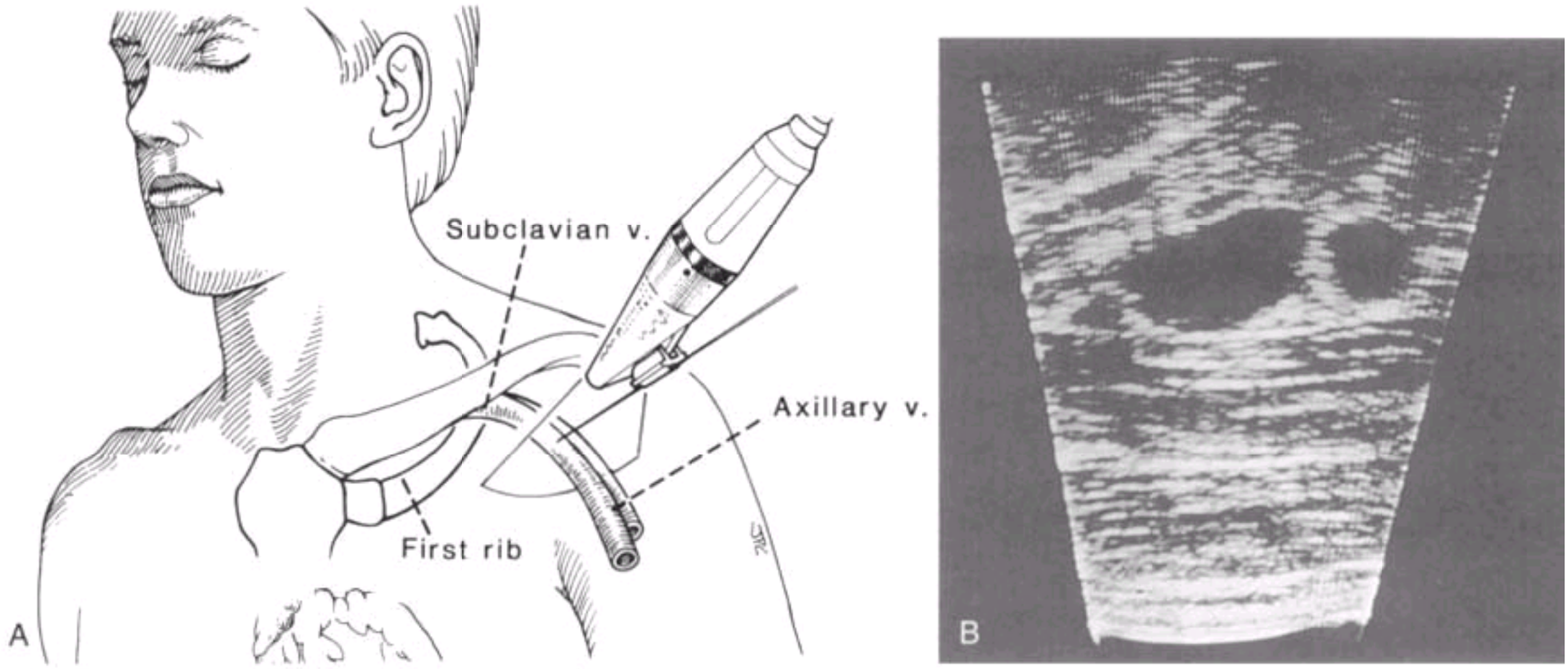


Figure 22-20 *A,* A femoral vein catheter is more prone to deep vein thrombosis and infection than a subclavian/internal jugular line, but it is a standard access route in the emergency department. Strict attention to sterile procedure and limiting use for a few days will negate most of the negatives of this approach. *A*, Significant hemorrhage can occur after puncture of the femoral artery, but this area is readily compressed. The femoral route may be the route of choice in the patient with a coagulopathy who requires a central line. The femoral vein will accept: (*B*) a triple-lumen catheter or (*C*) a large sheath introducer.

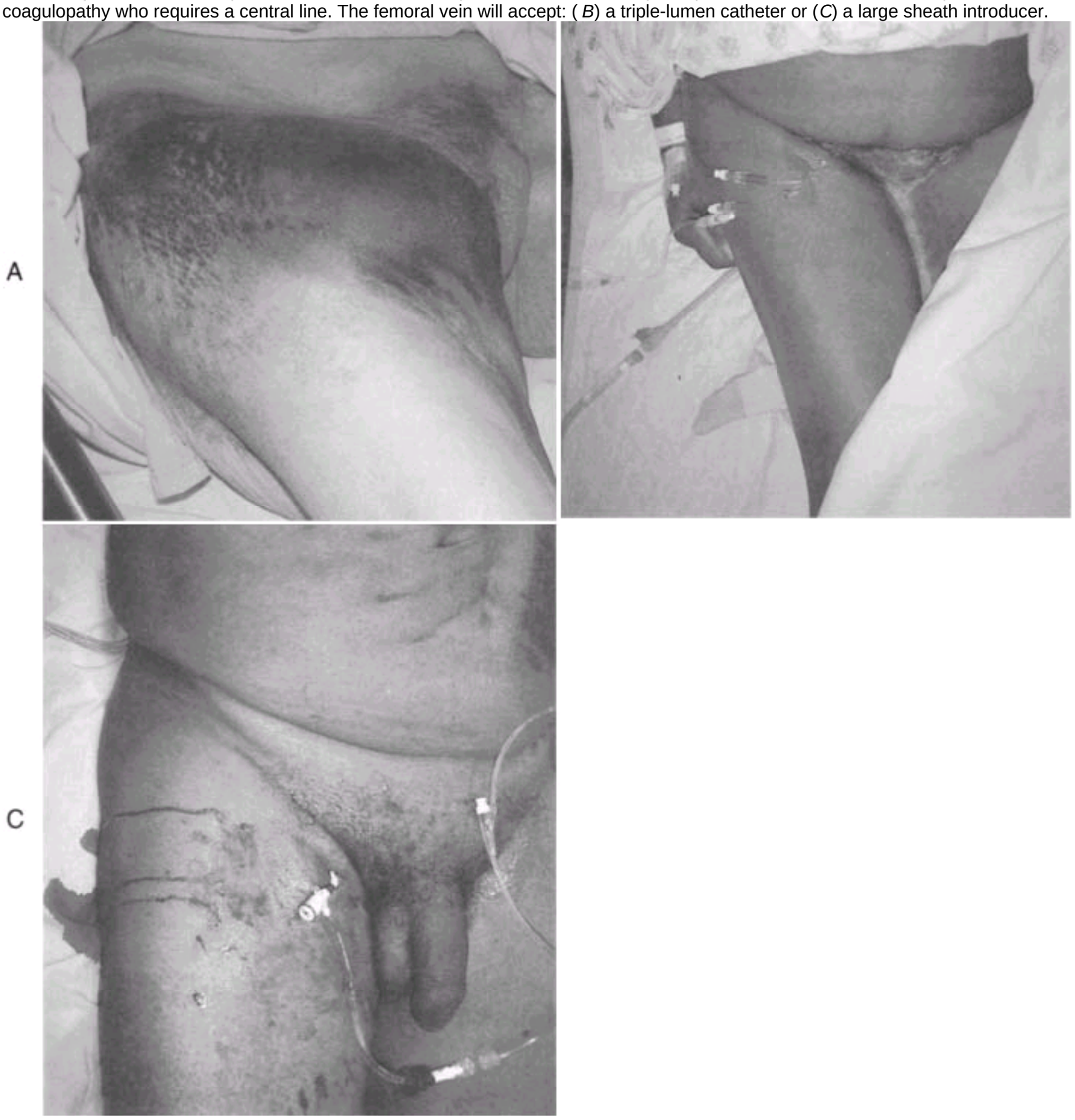


Figure 22-21 A subclavian line can easily be advanced into the neck (internal or external jugular vein). Malpositioned catheters should be discovered and replaced as soon as possible. Infusing saline into this catheter is not harmful, but hyperosmotic and vasoactive medications should not be given through this catheter for prolonged periods of time.

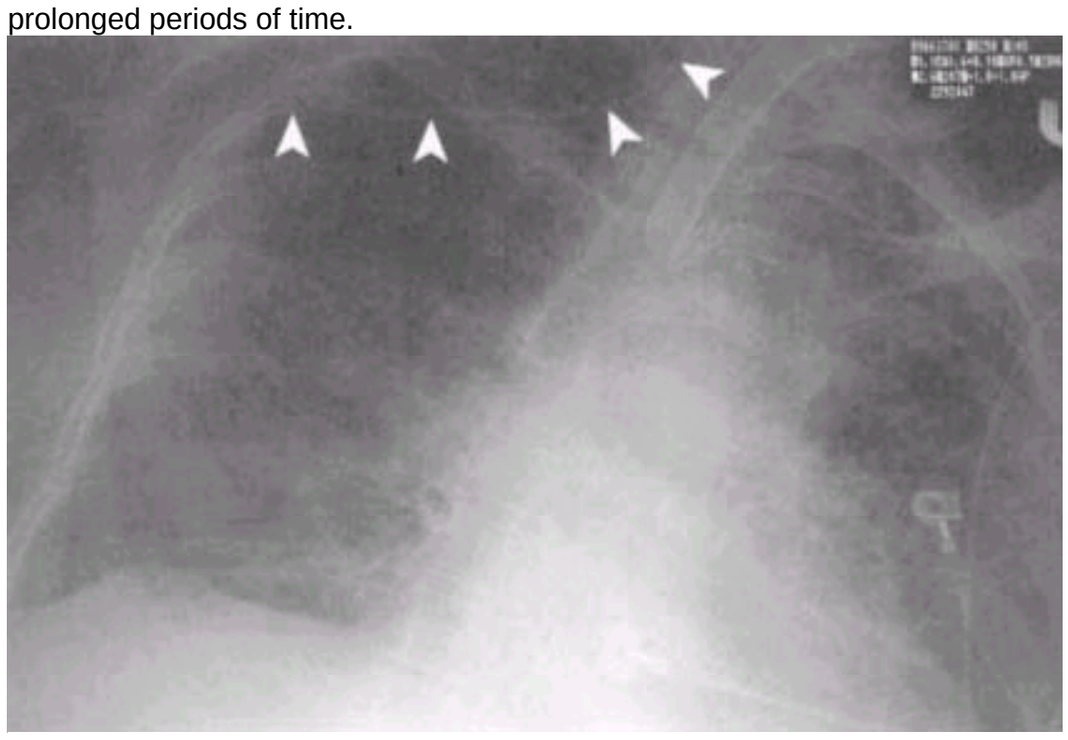
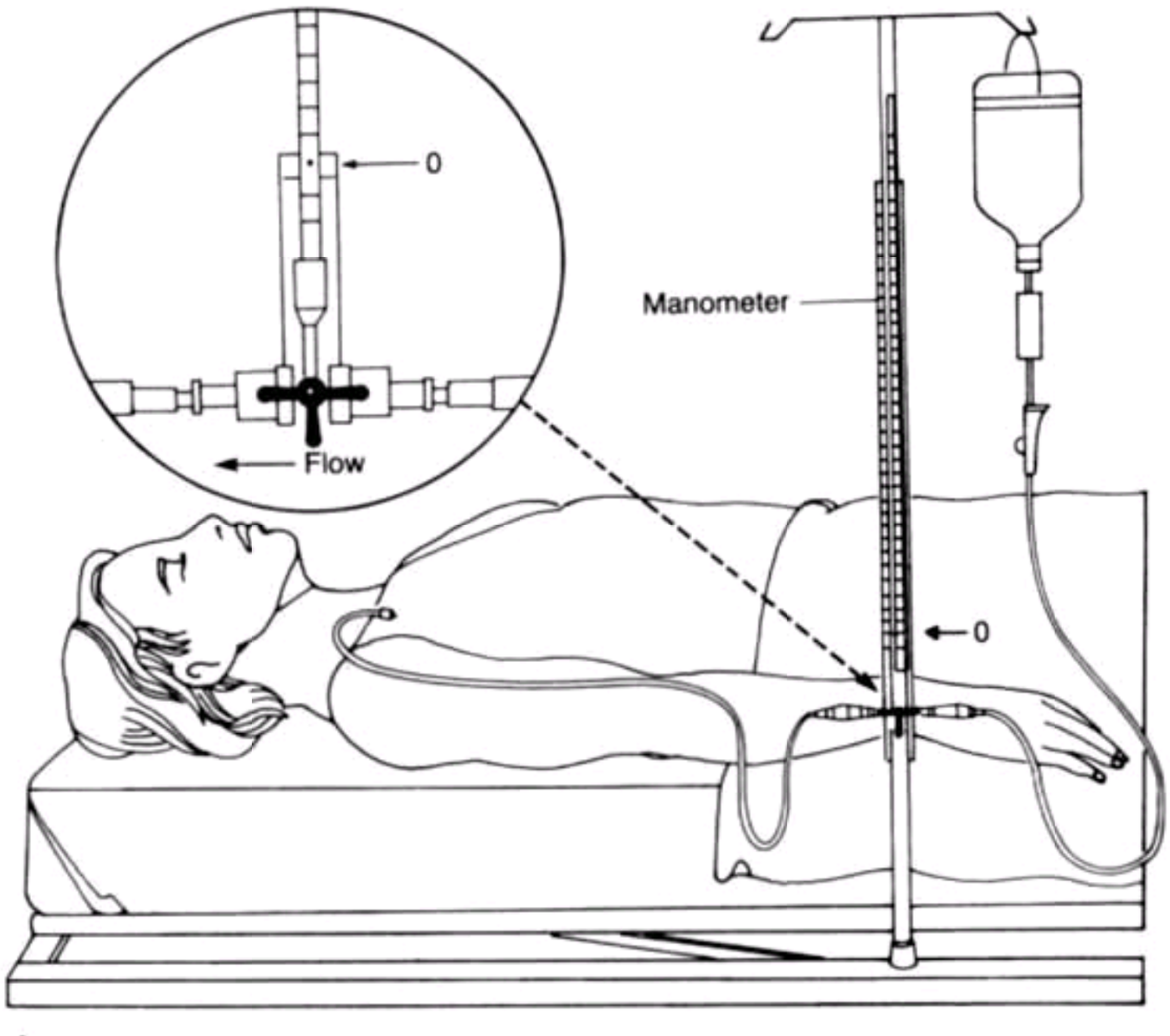
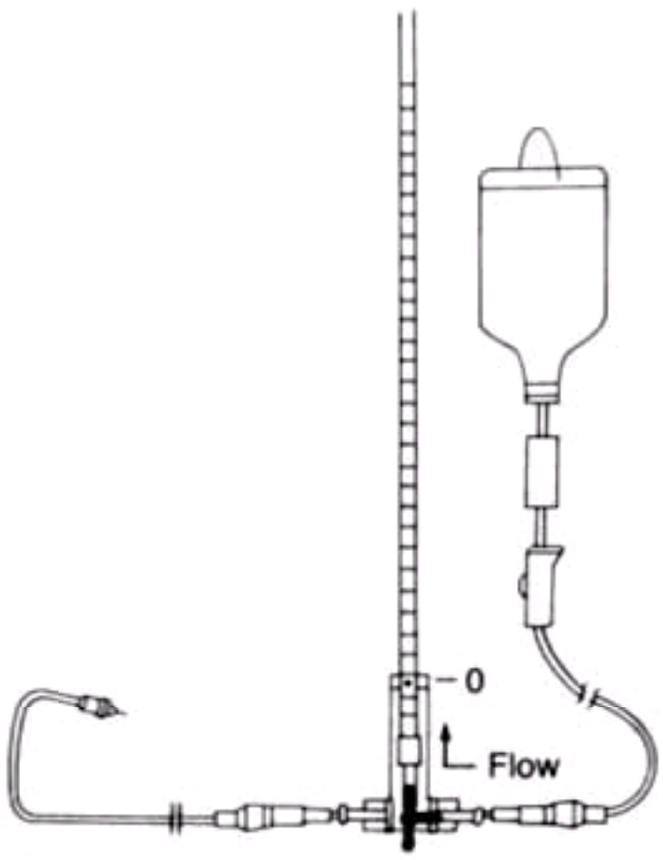


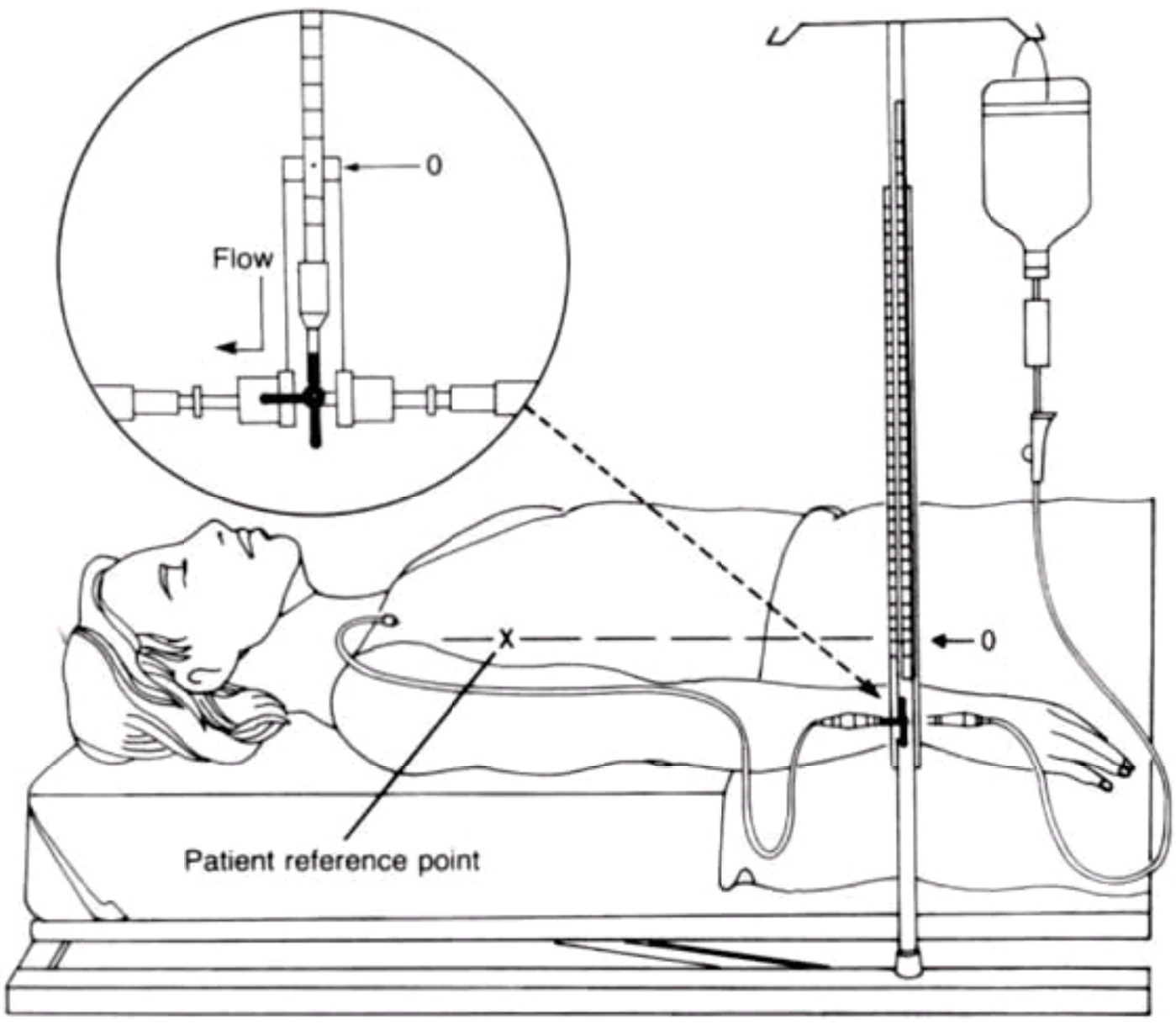
Figure 22-22 *A*, Simple manometry column used to measure CVP at the bedside. The stopcock is turned to direct the flow to the patient, bypassing the manometer. This is the position that is maintained to keep the catheter patent. The tubing is always flushed before connecting it to the patient's central venous pressure catheter. *B*, The stopcock is turned to fill the manometer to 25 cm H_2O. *C*, The stopcock is opened to the patient, and the column of water in the manometer is allowed to fall and stabilize before a reading is taken. Note that the zero mark is horizontally aligned with the tricuspid valve (midaxillary line in a supine patient).



A



B



C

Figure 22-23 General configuration of an intravascular pressure transducer. A working understanding of these devices, particularly regarding proper setup, zeroing, and line debubbling, will maximize their effectiveness and accuracy.

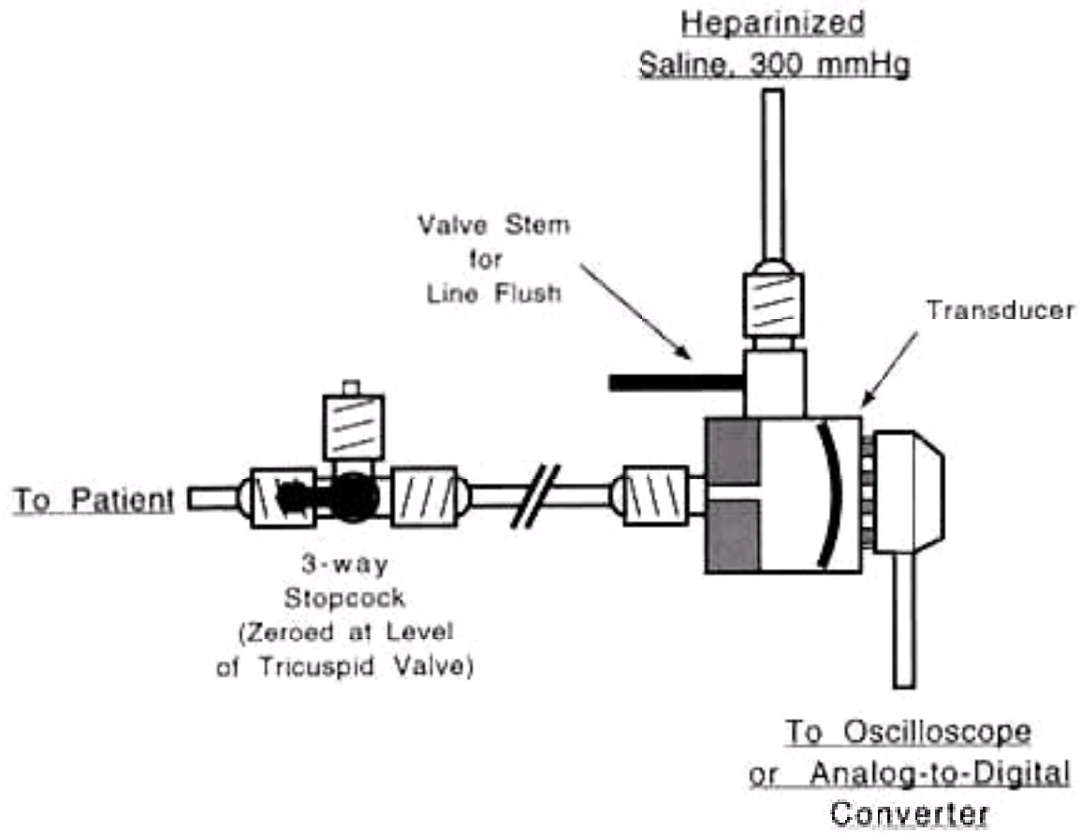


Figure 23-1 Superficial veins of the lower limb.

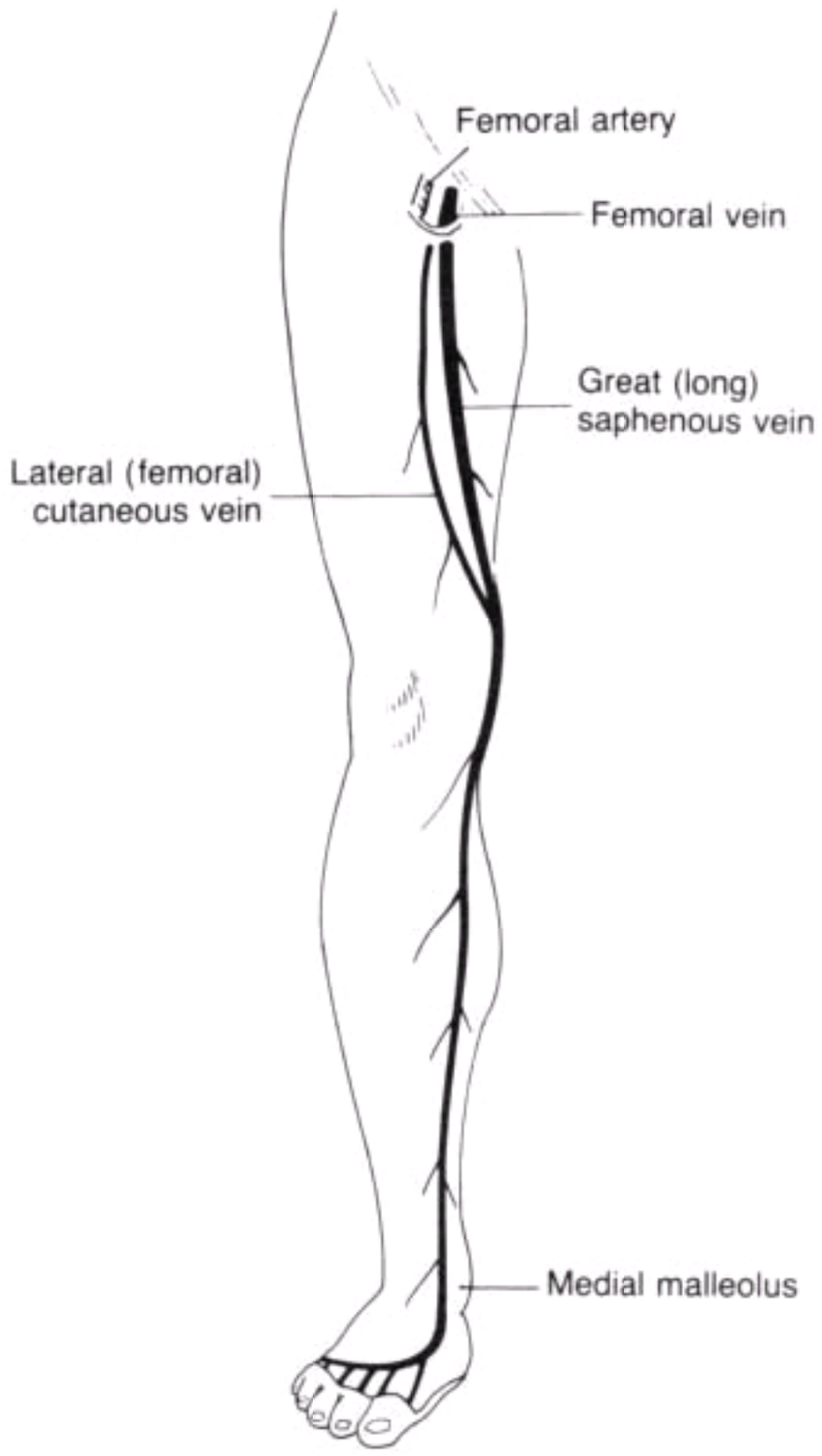


Figure 23-2 Veins of the upper limb.

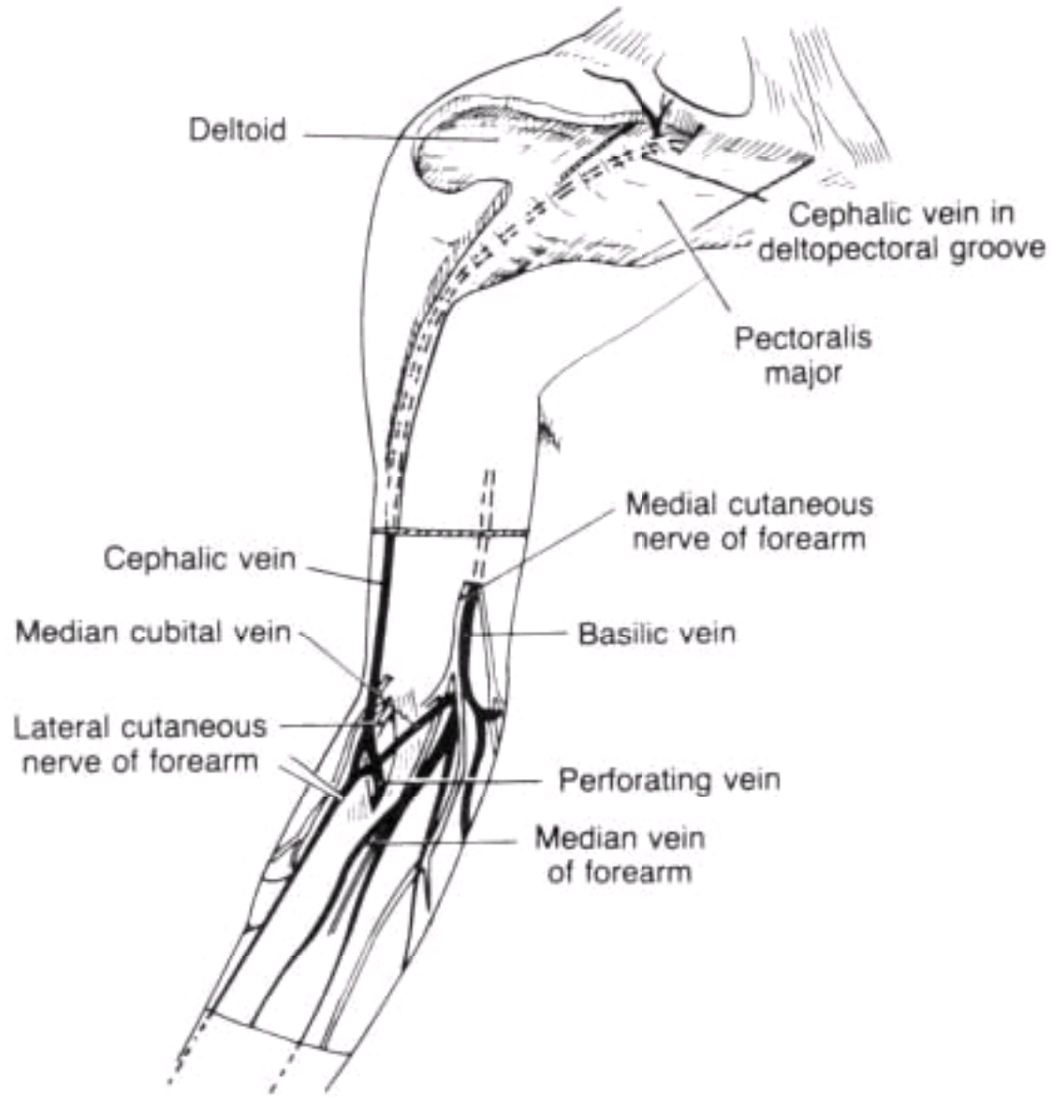


Figure 23-3 Venous cutdown tray. Note the small plastic vein dilator-lifter *(arrows)*, which is especially useful in children.

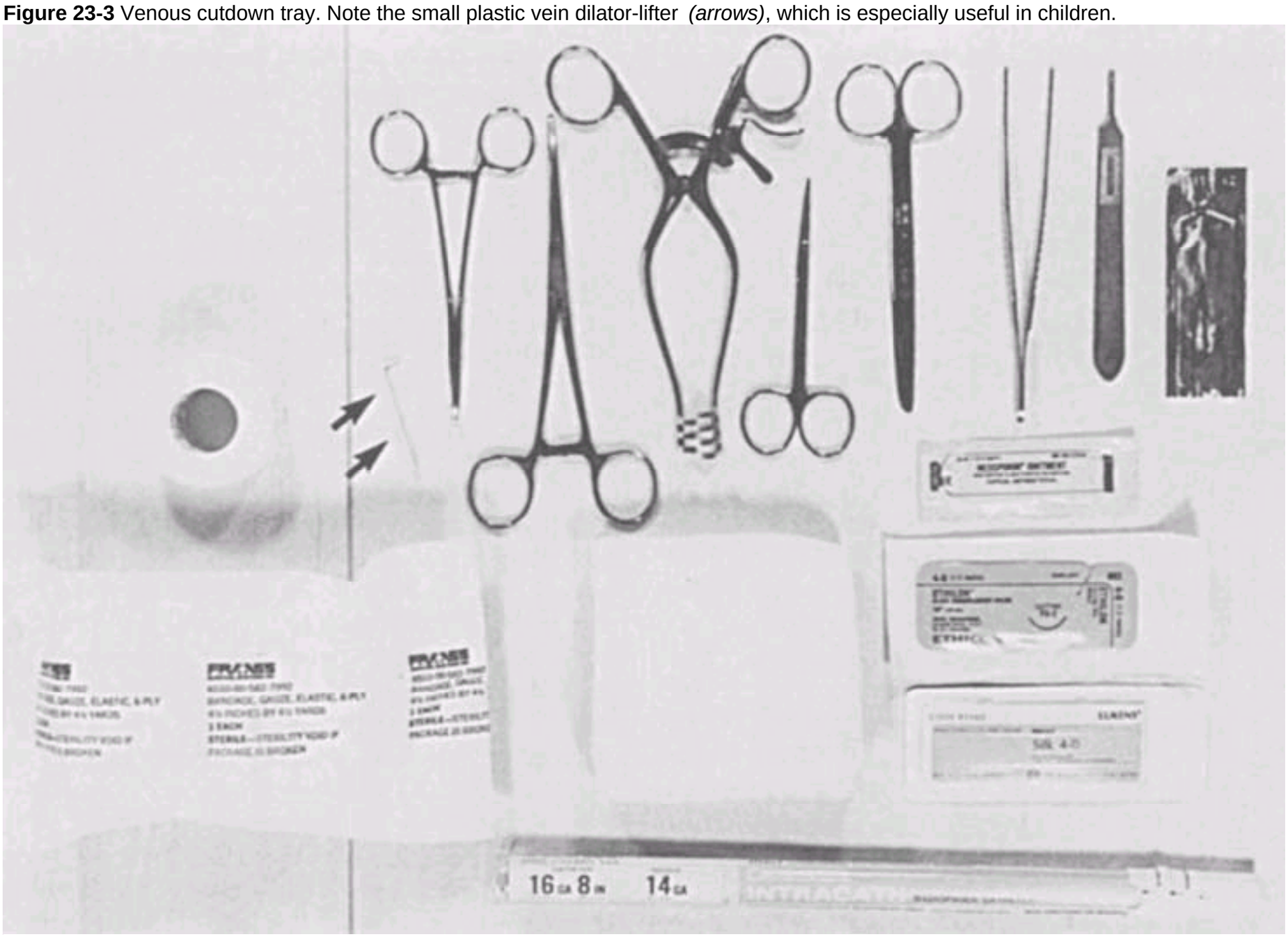


Figure 23-4 A skin incision is made perpendicular to the course of the vein.

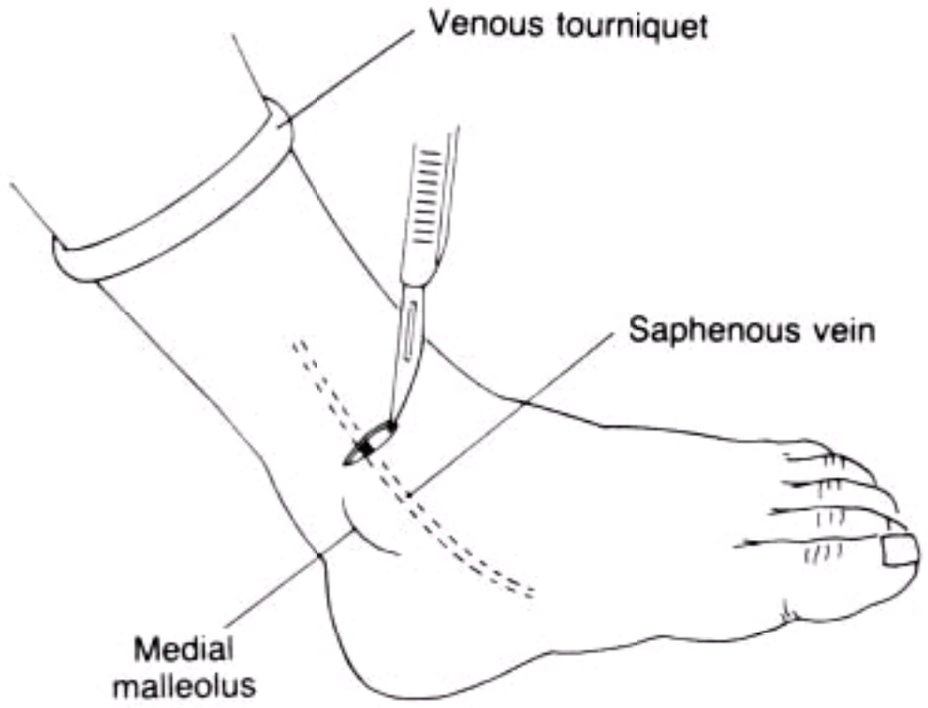


Figure 23-5 The skin is retracted and the vein exposed by blunt dissection. *(From Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

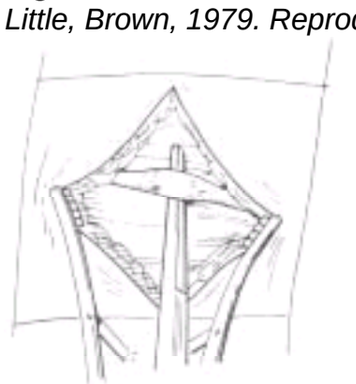


Figure 23-6 Proximal and distal ties are passed under the vein. If the vein is to be sacrificed, the distal suture is tied to prevent bleeding, and the ends are left long to help stabilize the vein during cannulation. The proximal tie is not tied at this point, but traction on it will control back-bleeding. *(From Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

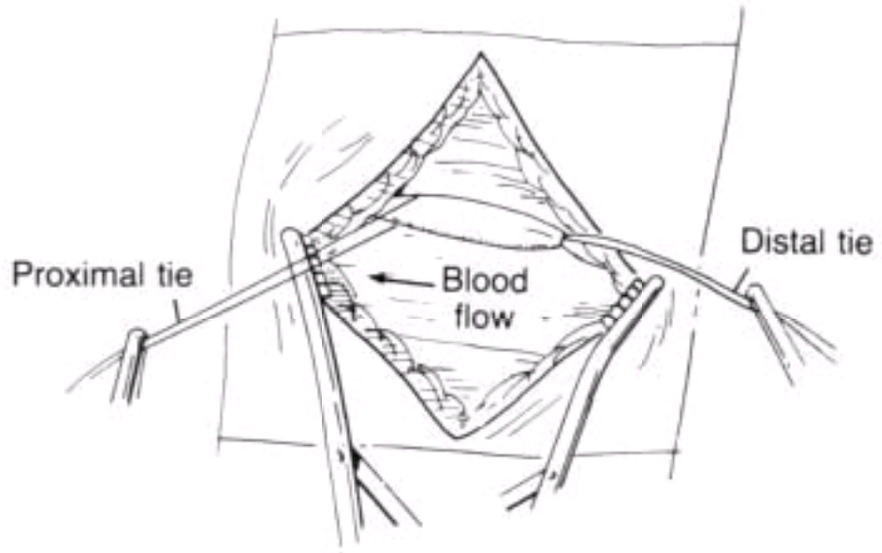


Figure 23-7 The vein is stretched flat and incised at a 45° angle. Approximately one third of the lumen must be exposed. Traction on the proximal tie will control back-bleeding. *(From Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

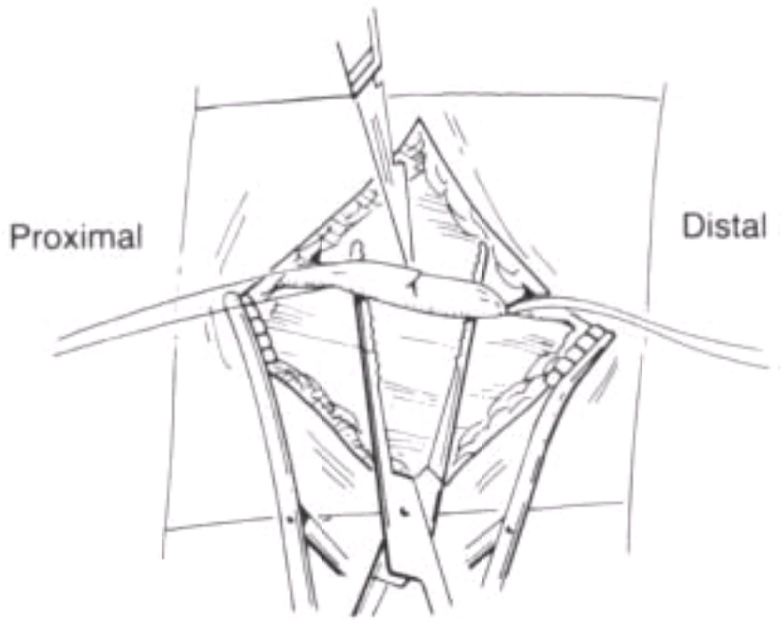


Figure 23-8 Use of the Intracath needle to produce a separate stab incision. The cannula is introduced into the wound by retrograde passage through the introducing needle. *(From Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

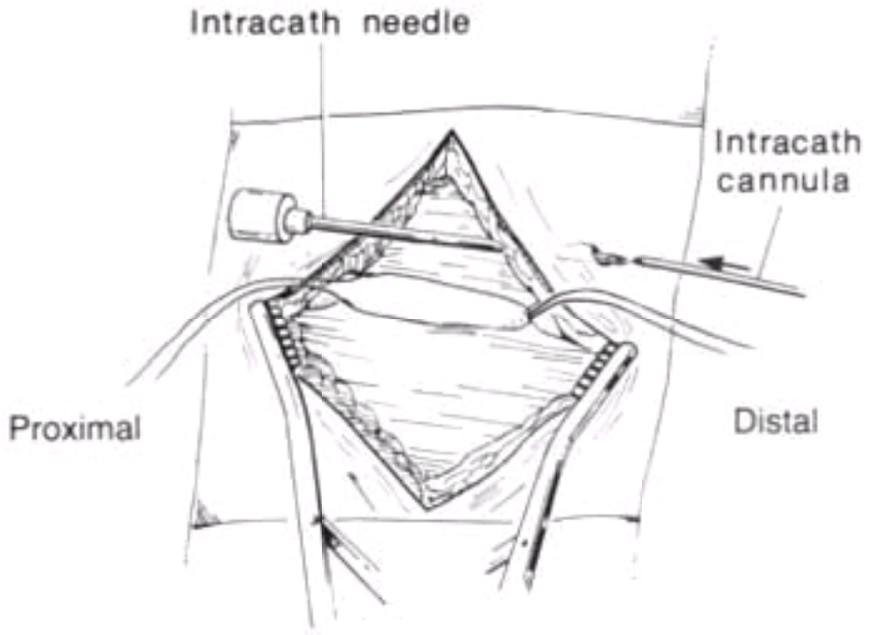


Figure 23-9 A cannula threaded through the stab incision. The Intracath needle has been withdrawn following introduction of the cannula into the wound. *(From Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

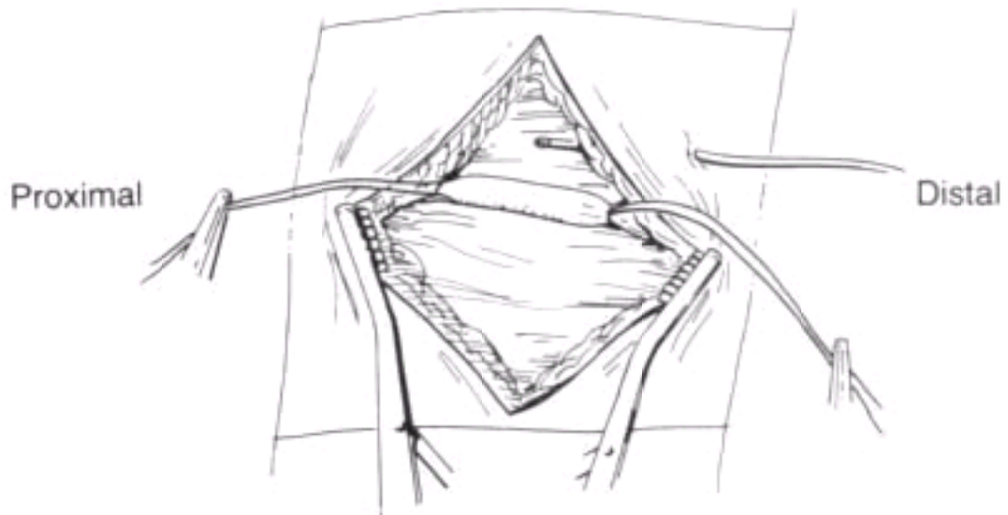


Figure 23-10 Threading the catheter with the aid of a venous dilator (lifter). This is technically the most difficult part of the procedure. The lifter is especially helpful in small veins.

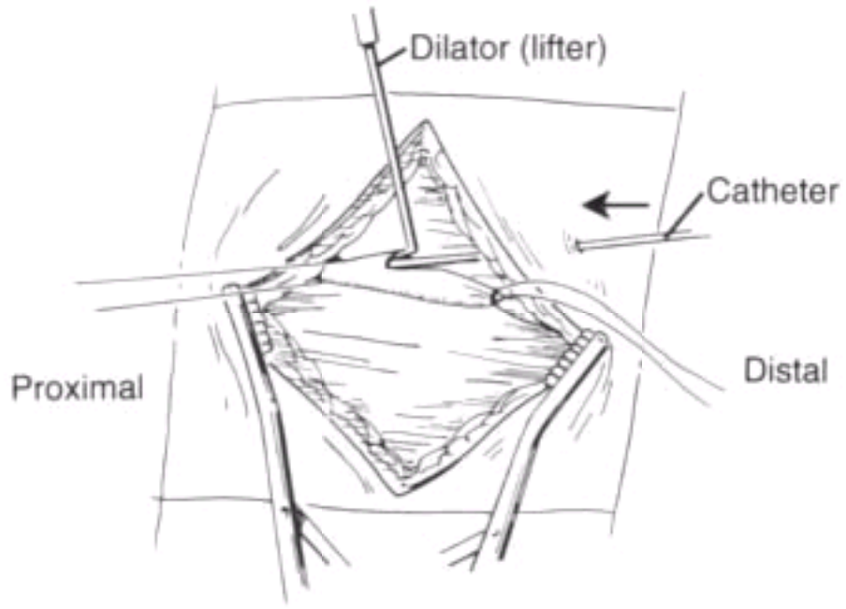


Figure 23-11 In larger veins, a mosquito hemostat can facilitate the placement of the cannula by opening the lumen and providing countertraction.

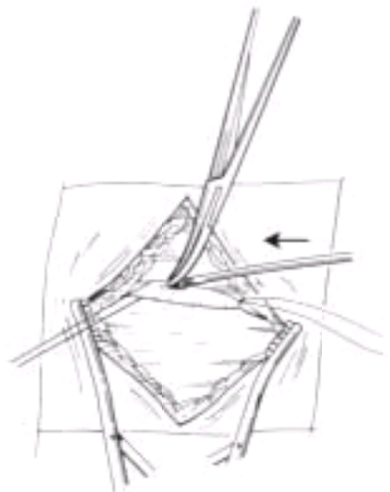


Figure 23-12 The incision is closed, and the catheter is sutured in place.

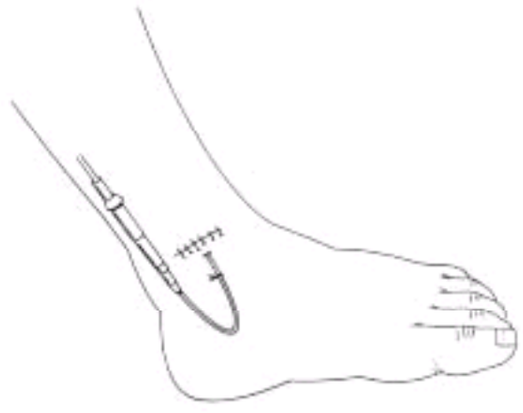


Figure 23-13 The cutdown site is securely dressed and splinted.

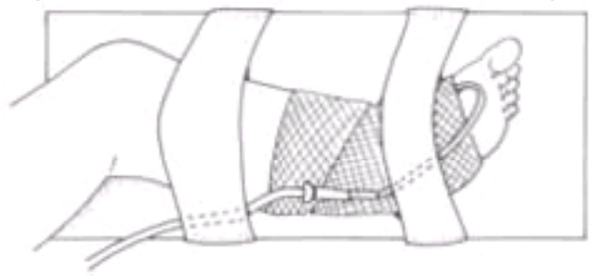


Figure 23-14 The mini-cutdown technique is an alternative to the venous cutdown method. The vein is cannulated under direct vision using standard percutaneous catheters. A separate entry site (shown) may be used, or the vein can be cannulated through the skin incision. Note that the vein is not tied off with this technique. A standard Angiocath IV set also may be used instead of the through-the-needle catheter shown here.

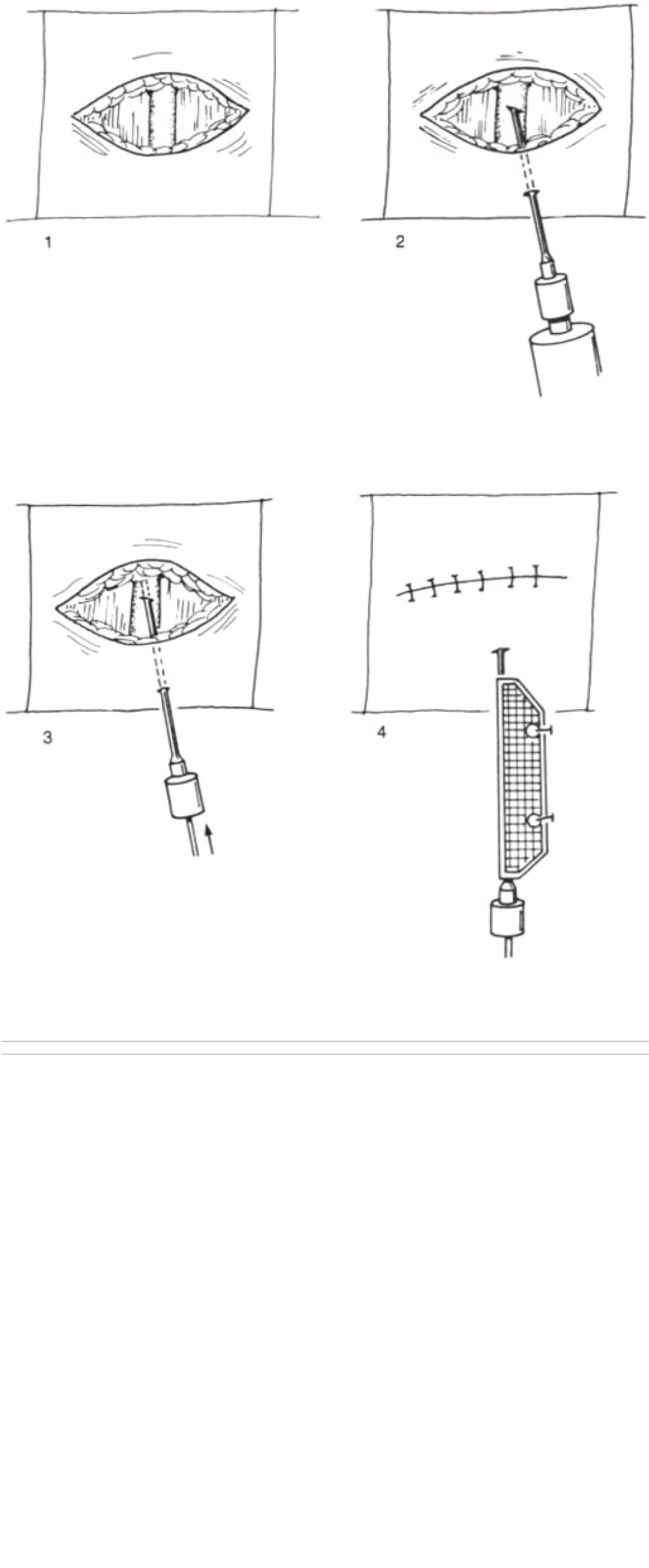
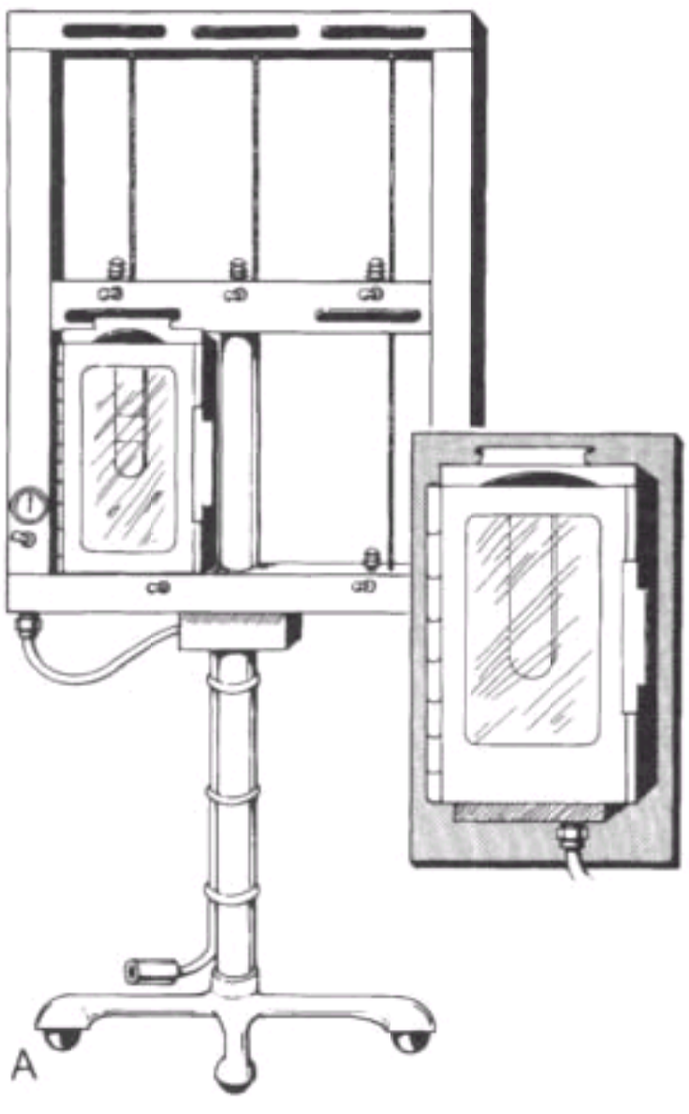


Figure 24-1 Three types of external pressure devices. *A*, Infuser-1. *(As modified in Iserson KV, Reeter A, Woods W, Criss E: Pressurization of IV bags: A new configuration and evaluation for use. J Emerg Med 3:89, 1985.) B*, Infusable *(Biomedical Dynamics Corp., Minneapolis, MN)*. *C*, Alton Dean infuser *(North Salt Lake City, UT)*.



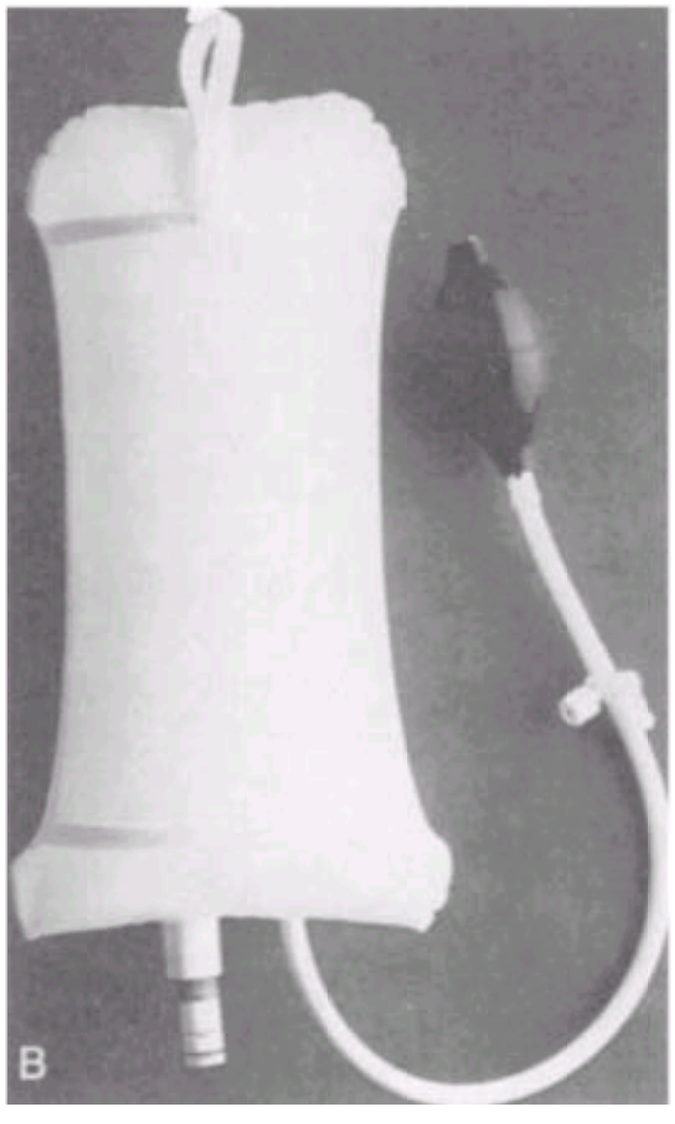


Figure 24-2 (1) During all handling of blood, standard universal precaution procedures should be followed. (2) Remove a 250-mL bag of normal (N) saline from the 70°C incubator. Check the bag for clarity, discoloration, particulate matter, obvious decreased volume, and cracking of the outer wrapper. Do not use if problems are found. (3) Check the incubator thermometer to ensure that its temperature is between 65° and 75°C. Do not use if above 75°C. (4) "Spike" the packed red blood cell (PRBC) unit from the blood bank with one end of a Fenwal plasma transfer set (No. 4C2243). Spike the other end of the transfer set into the 70°C, 250-mL N saline bag. Open the clamp, raise the saline bag above the level of the PRBC bag, and manually squeeze the saline (or use external pressure bag or other external pressure device) into the PRBC bag. (5) A towel or potholder-type kitchen glove may be used to squeeze the 70°C saline bag, if desired. (6) After all of the 70°C saline is transferred into the PRBC bag, tightly close the clamp on the transfer set tubing. (7) Do not remove the transfer set tubing spike from the PRBC bag (to prevent contamination). The now-empty saline bag may be removed from the other end of the transfer set and discarded, or simply left in place. (8) On the patient's side, spike the unused part of the PRBC bag (now admixed with 70°C saline) with one limb of the Y-type blood set already attached to the patient. The admixture temperature will be approximately 37°C. (9) Close the clamp on the other limb of the Y set, which has been infusing standard warm or room-temperature normal saline into the patient. (10) If the blood is to be rapidly infused, pressurize the external pressure bag or device. (11) Open the clamp on the PRBC admixture bag to begin infusion of 37°C PRBC unit into the patient. (12) Additional units of warm-admixed PRBC may be prepared using the same technique. Close the Y set clamp, remove the empty admixed PRBC bag, and replace it with the second unit. Open the clamp and begin infusing the second unit. (13) Infuse the blood into the patient as rapidly as necessary. (14) For each unit of warm-admixed PRBC infused, document in the patient's record that the patient received "1 unit of PRBC admixed with 250 mL of N saline at an infusion temperature of approximately 37°C." Include start/stop times of infusion and the name of the person preparing and administering the infusion. *(From Iserson KV, Knaut MA, Anhalt D: Rapid admixture blood warming: Technical advances. Crit Care Med 18:1138, 1990. © 1990, Williams & Wilkins, Baltimore.)* Following admixture, the infused blood will be between 35° and 40°C. *Cautions:* (1) Keep the blood bag hanging when adding saline. (2) If the blood is admixed, it must be infused or discarded to avoid contamination. (3) Do not mix the blood manually or shake the bag. (4) *Do not* infuse 70°C saline *directly* into patients!

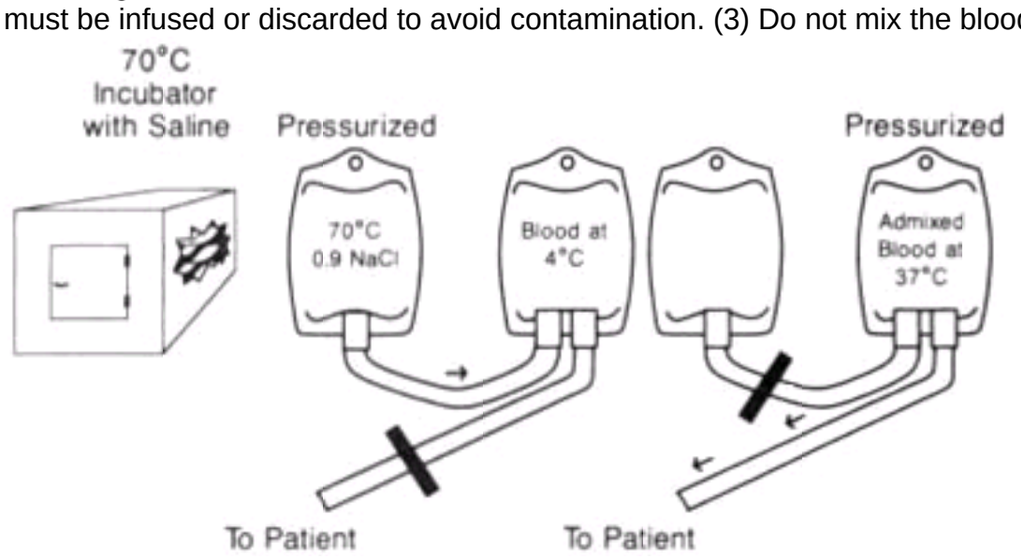


Figure 25-1 Broviac pediatric single-lumen catheter (4.2 Fr) with Dacron cuff.

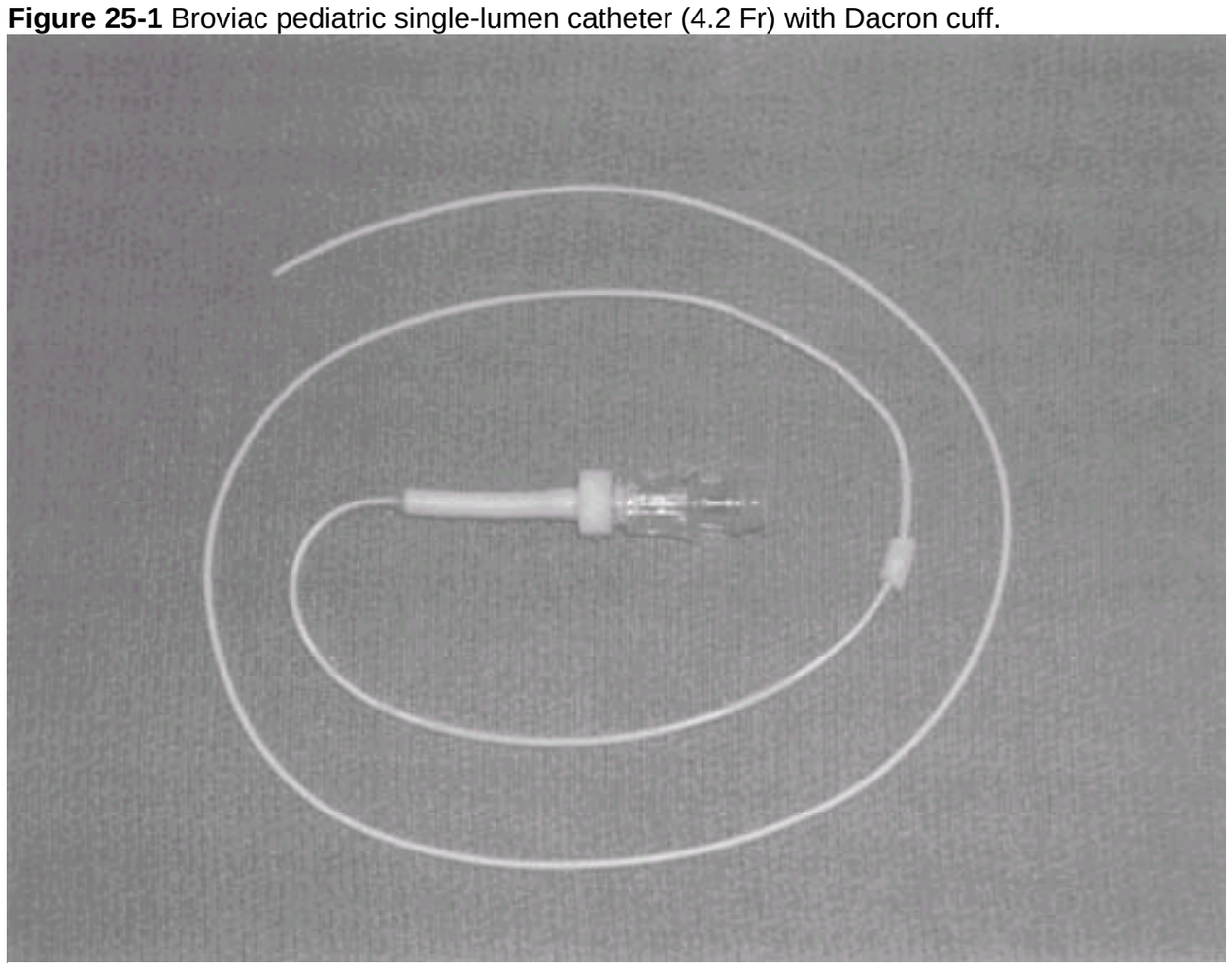


Figure 25-2 Permacath (Quinton Instrument Co.) double-lumen catheter (13.5 Fr).

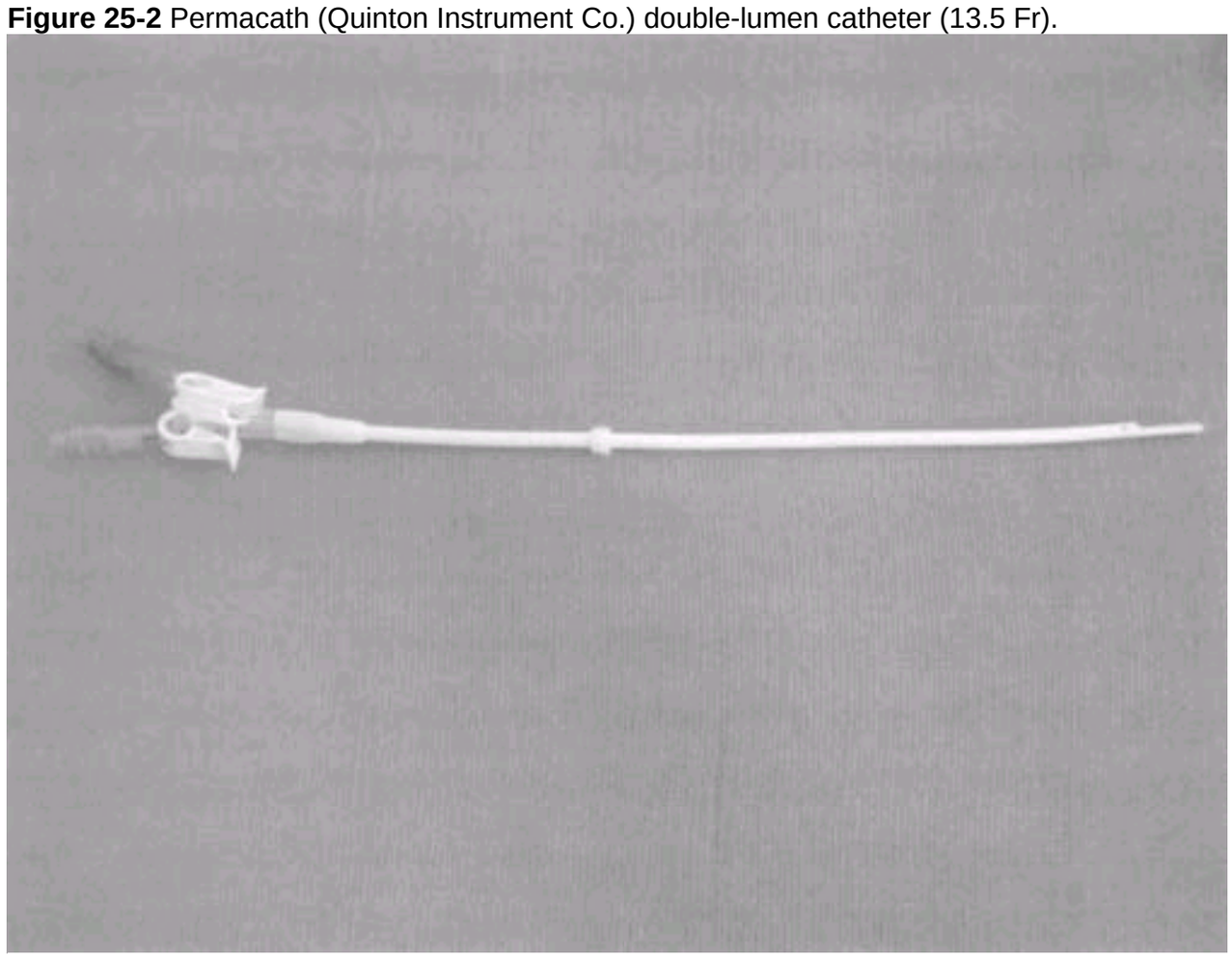


Figure 25-3 Norport System single-lumen catheter and specially tapered Huber needle.

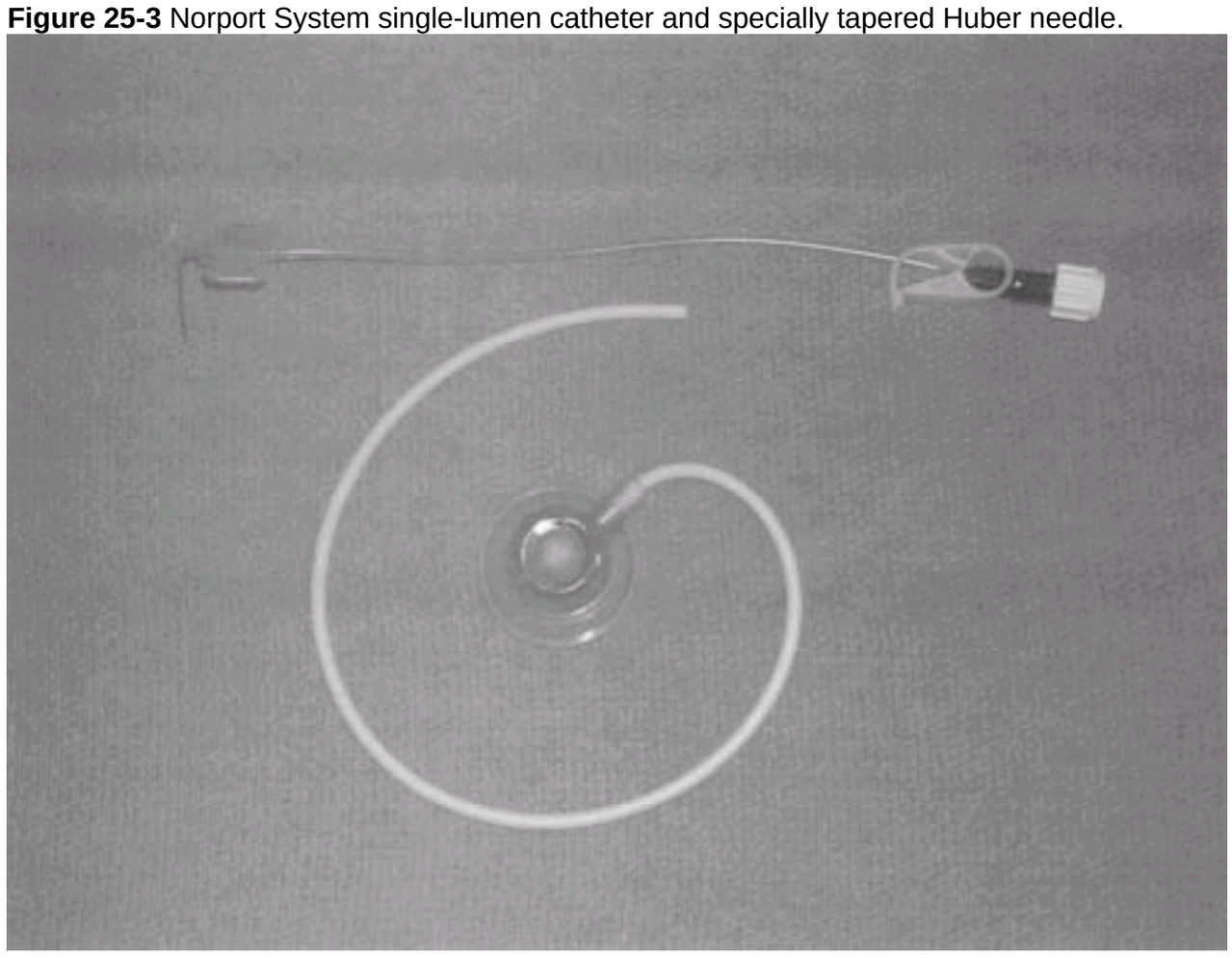


Figure 25-4 Port-A-Cath double-lumen catheter segment.

Figure 25-5 Port-A-Cath system (Deltec, Inc., St. Paul, MN). The Port-A-Cath system is accessed by inserting a Huber needle through the skin and portal septum.

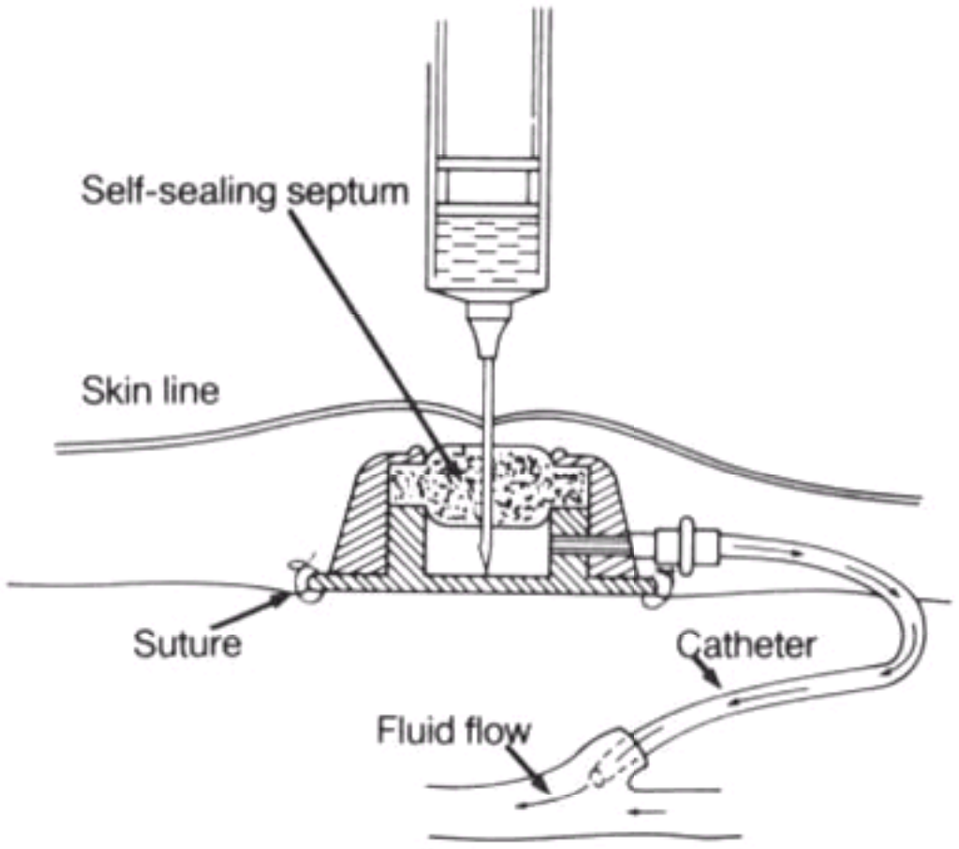


Figure 25-6 Double-lumen peripherally inserted central catheter (5.0 Fr, 18 ga).

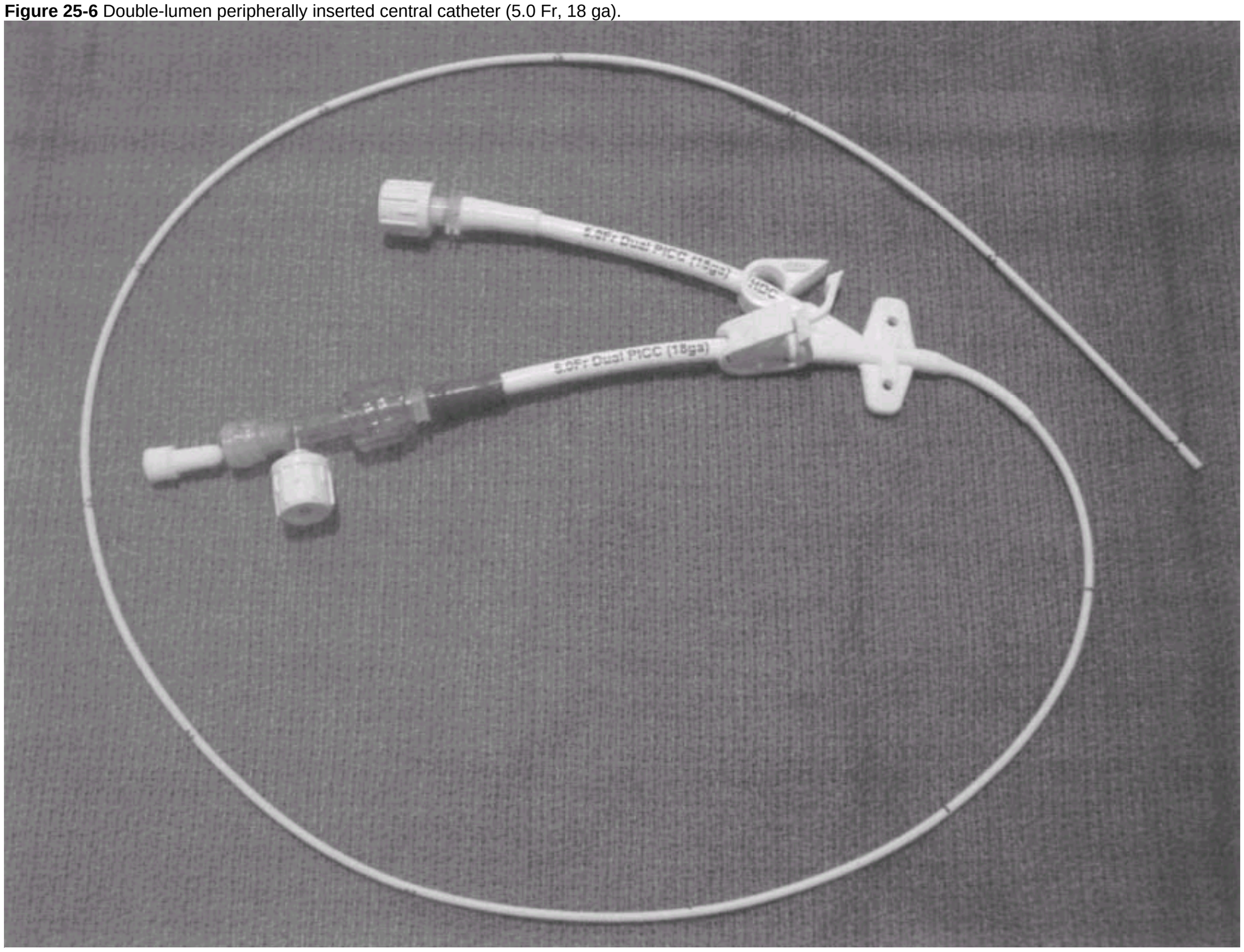


Figure 25-7 Catheter repair. A hollow lumen connector and outer sleeve are placed to splice a replacement catheter to the remaining catheter segment.

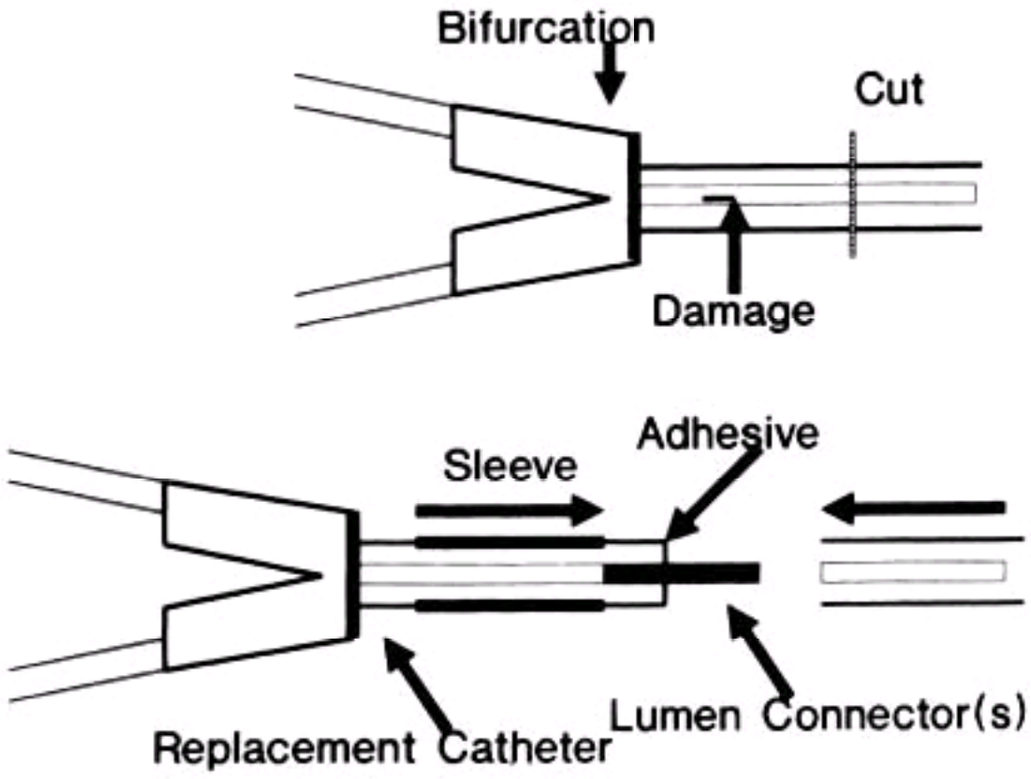


Figure 26-1 Schematic diagram illustrating the venous drainage from the marrow of a long bone with an intraosseous (IO) needle in place.

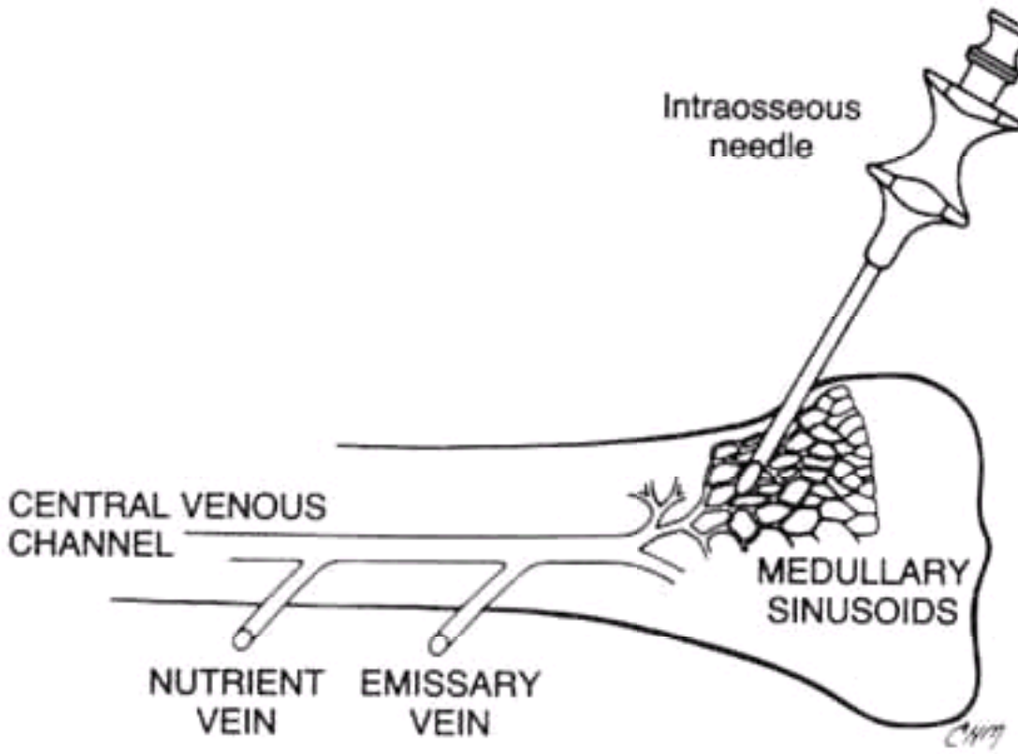


Figure 26-2 Serum diazepam levels (ng/mL, mean ± SE) graphed for the intraosseous (IO) *(shaded area)* and intravenous (IV) *(blackened area)* groups as a function of time when injected during normal perfusion. Initially the IV drug level is slightly higher, but overall the difference between the two routes of administration is not significant.

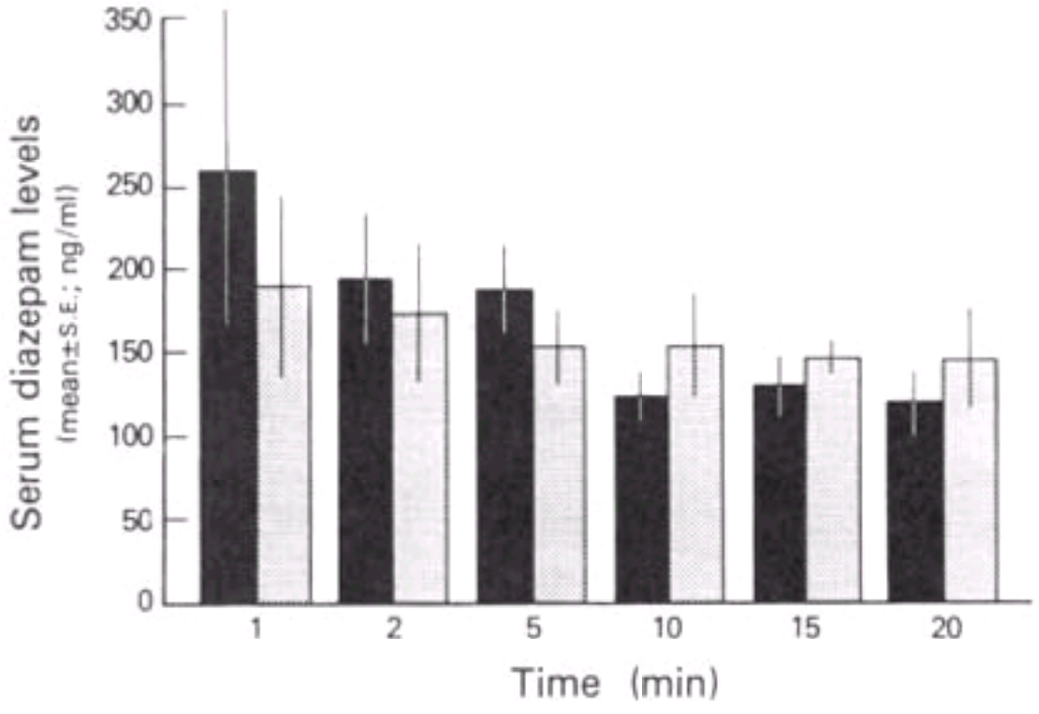


Figure 26-3 Needles used for intraosseous (IO) infusion. *Left to right,* Illinois bone marrow aspiration needle, Illinois sternal/iliac aspiration needle, Jamshidi disposable sternal/iliac aspiration needle, Cook IO needle with 45° trocar, and Sur-Fast IO needle.

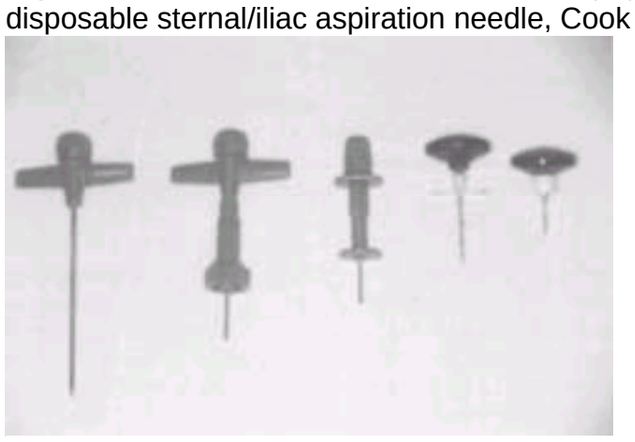


Figure 26-4 The FAST sternal intraosseous (IO) infusion device. *(Courtesy of Pyng Medical Corporation, Richmond, British Columbia.)*

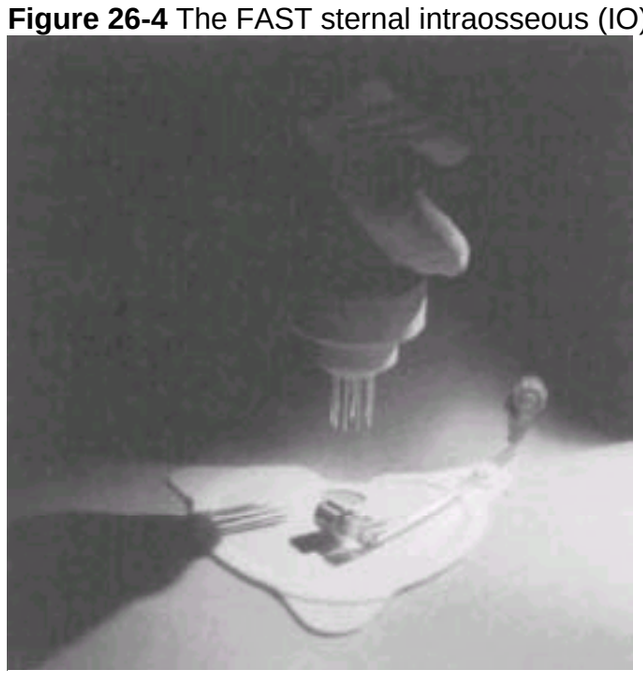


Figure 26-5 The Bone Injection Gun (BIG).

Figure 26-6 Schematic diagram demonstrating intraosseous (IO) insertion sites. *A*, The proximal tibia. The IO needle is inserted 1 to 3 cm distal to the tibial tuberosity and over the medial aspect of the tibia. *The bevel of the needle is directed away from the joint space. B,* The distal tibia. The IO needle is inserted on the medial surface of the distal tibia at the junction of the medial malleolus and the shaft of the tibia, posterior to the greater saphenous vein. The needle is directed cephalad, away from the growth plate. *C,* The distal femur. The IO needle is inserted 2 to 3 cm above the external condyles in the midline and directed cephalad away from the growth plate.

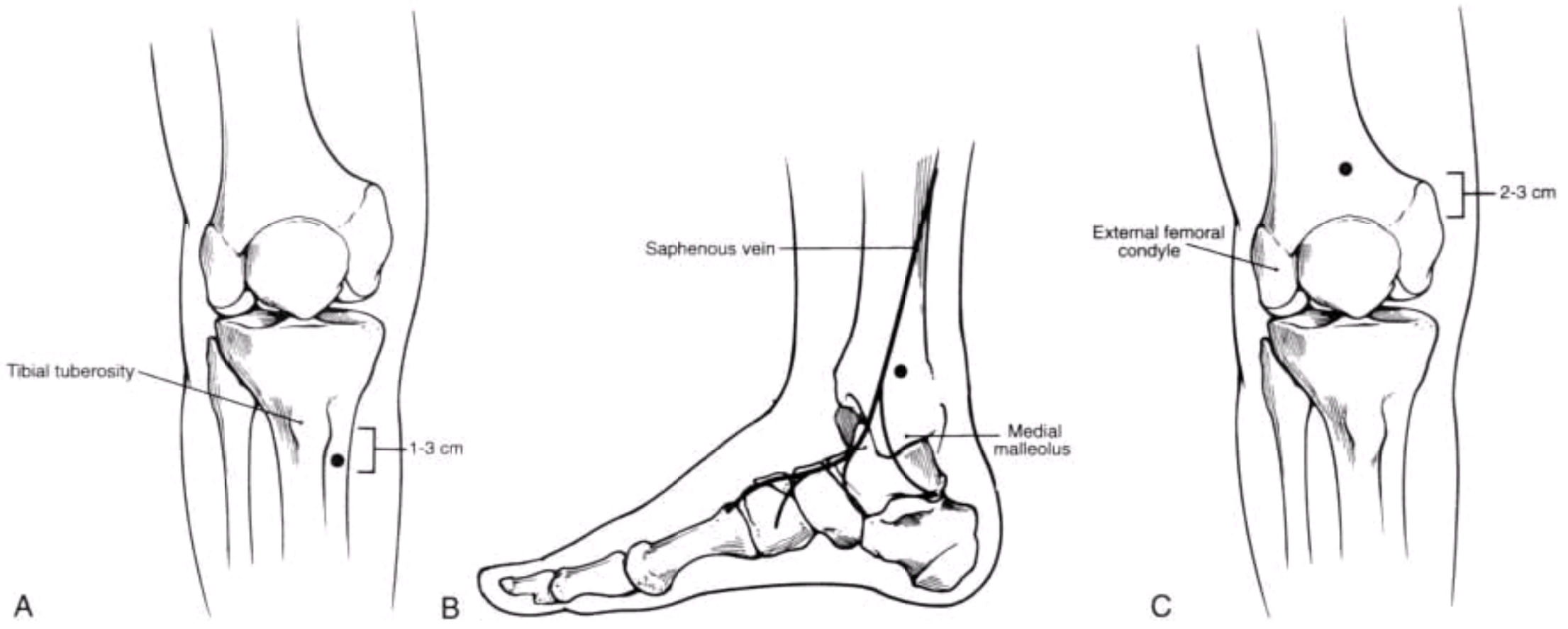


Figure 26-7 Insertion of an intraosseus (IO) needle. The fingers and thumb are wrapped around the proximal tibia to stabilize it. A towel (not shown) may be placed behind the knee for support. The needle is grasped firmly in the palm, and a rotary motion is applied with moderate pressure. The plastic sleeve can be adjusted to prevent it from being forced too deeply into the bone or through the bone. Note that the needle is directed away from the joint space.

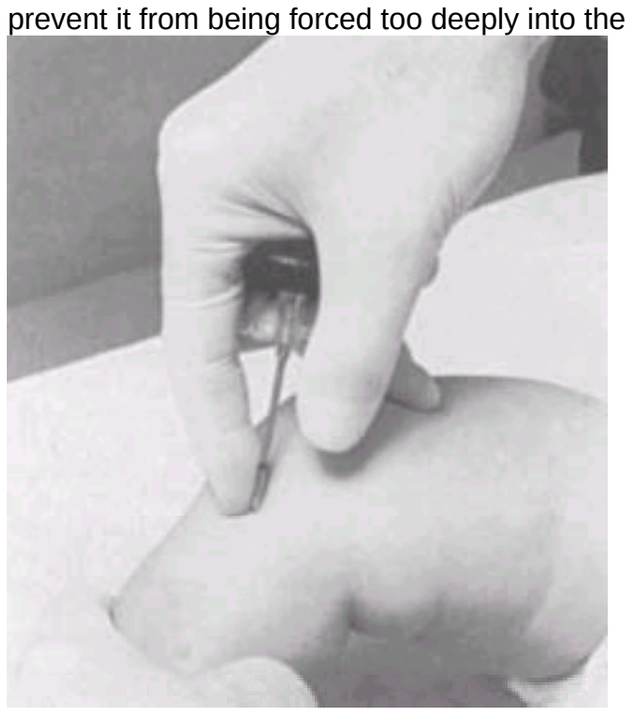


Figure 26-8 Intraosseus (IO) needle in situ. *A*, The needle and tubing are secured with tape and the extremity is immobilized on a leg board. A long leg board may be needed with an active child. *B*, The needle is protected from accidental dislodgement by wrapping a stretch bandage around the extremity and placing a small plastic drinking cup around the needle.

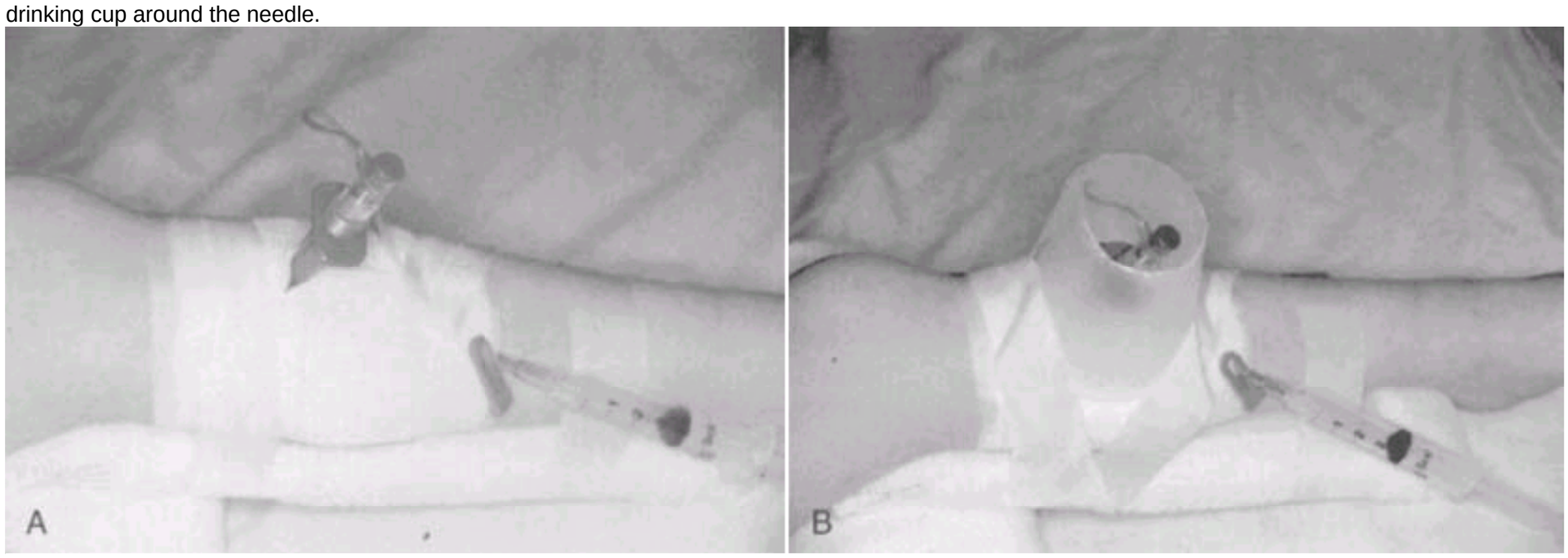


Figure 26-9 The FAST sternal intraosseous (IO) infusion device comes packaged with all necessary equipment.

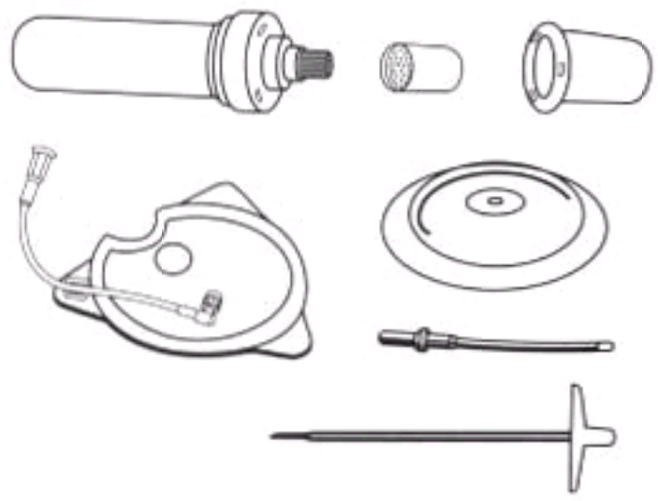
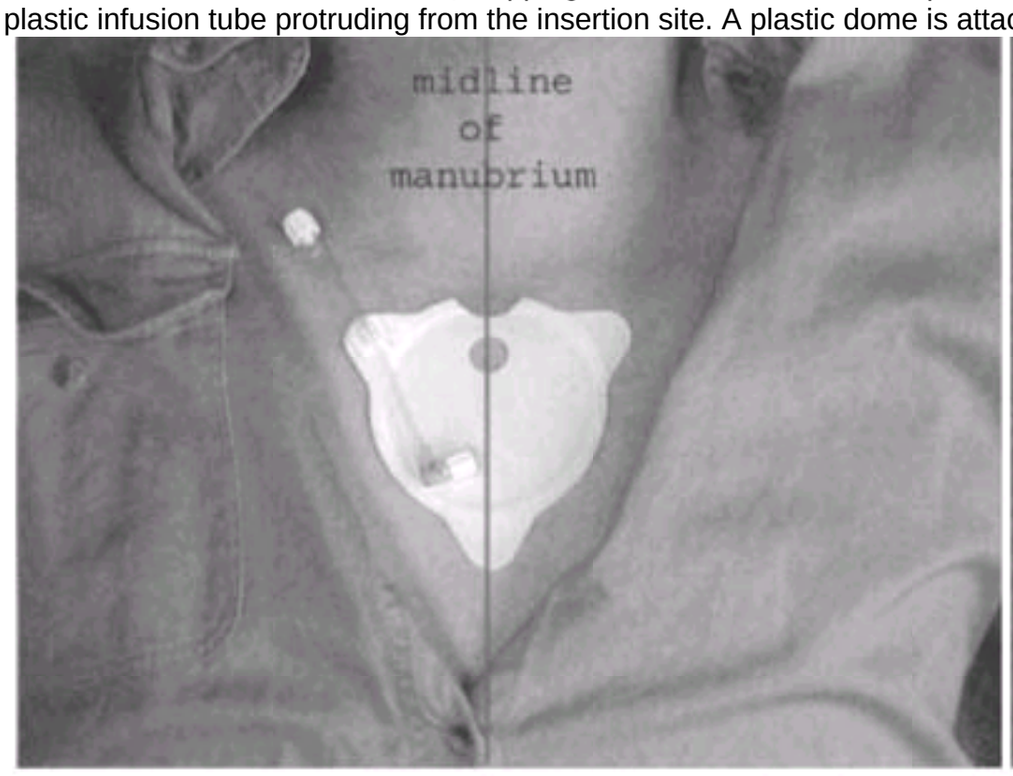
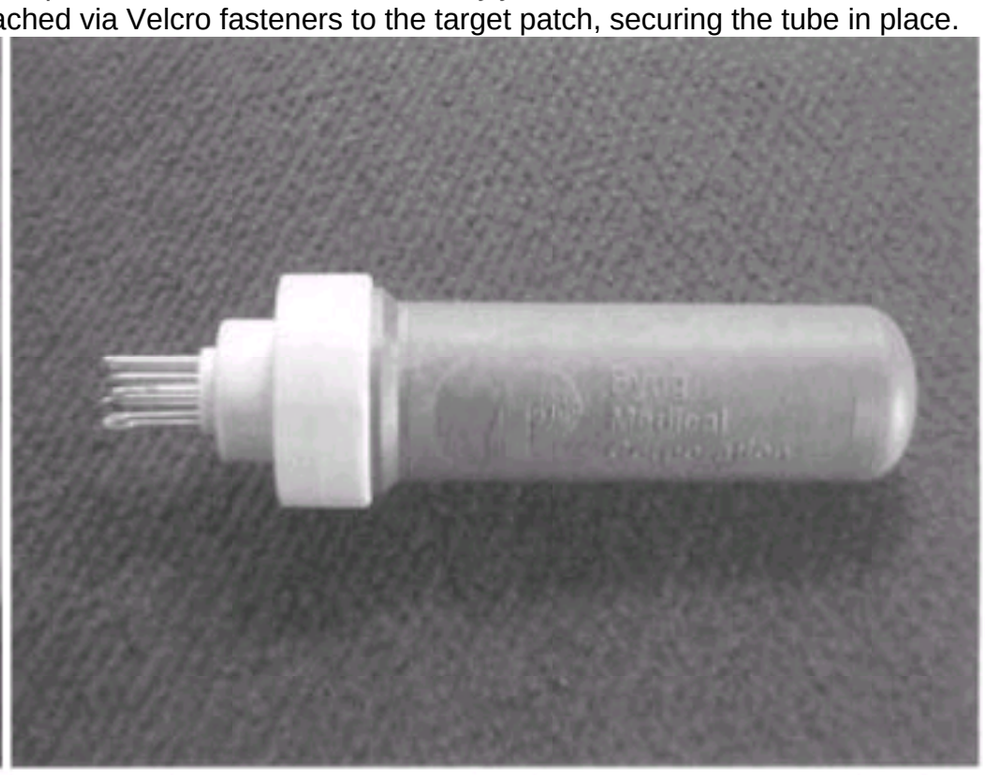


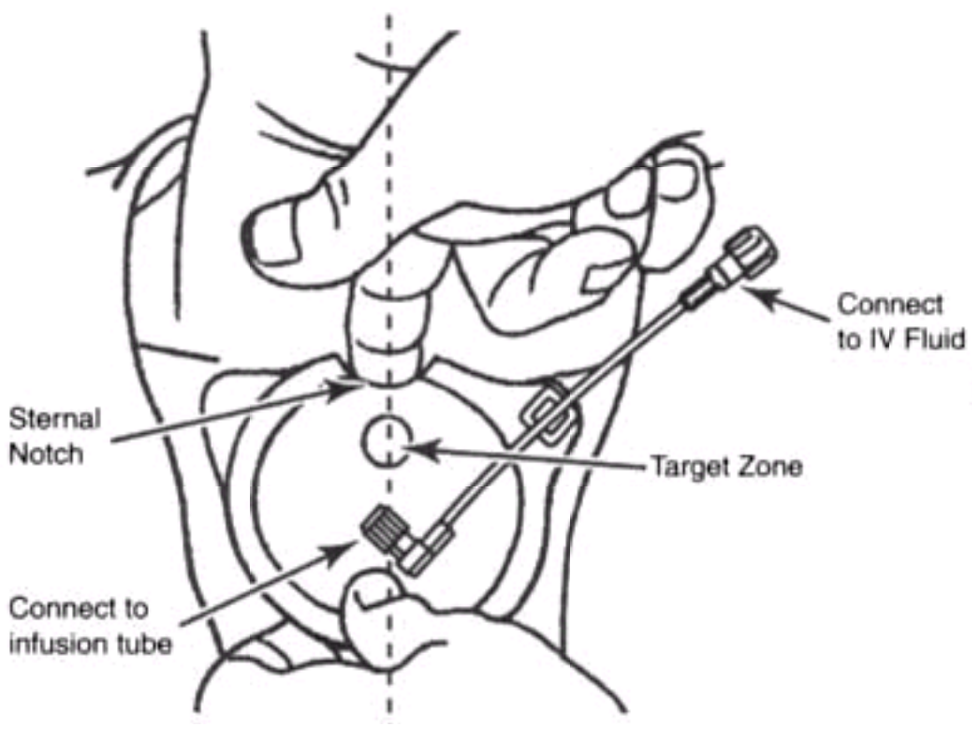
Figure 26-10 *A*, After prepping the overlying skin with iodine and alcohol, an adhesive target patch is placed over the midline of the manubrium with the target zone hole approximately 1.5 cm below the sternal notch. *B*, The introducer, which contains a "bone cluster" of needles, is placed in the center of the target zone. *C*, Pressure on the handle releases an inner needle upon which a plastic infusion tube with a small metal tip is loaded. The central needle advances exactly 5 mm beyond the circular cluster of needles, stopping at the cortex. The metal tip is now positioned in the cortex-medullary junction. Withdrawal of the handle leaves only the plastic infusion tube protruding from the insertion site. A plastic dome is attached via Velcro fasteners to the target patch, securing the tube in place.

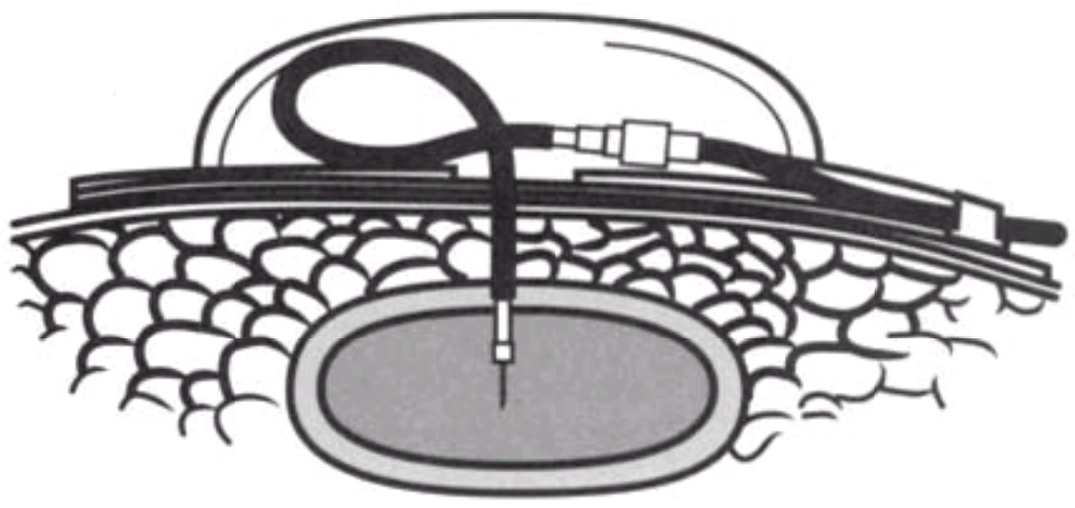


A



B





C

Figure 26-12 Schematic diagram of possible problems encountered with intraosseous (IO) infusion. *A*, Incomplete penetration of the bony cortex. *B*, Penetration of the posterior cortex. *C*, Fluid escaping around the needle through the puncture site. *D*, Fluid leaking through a nearby previous cortical puncture site.

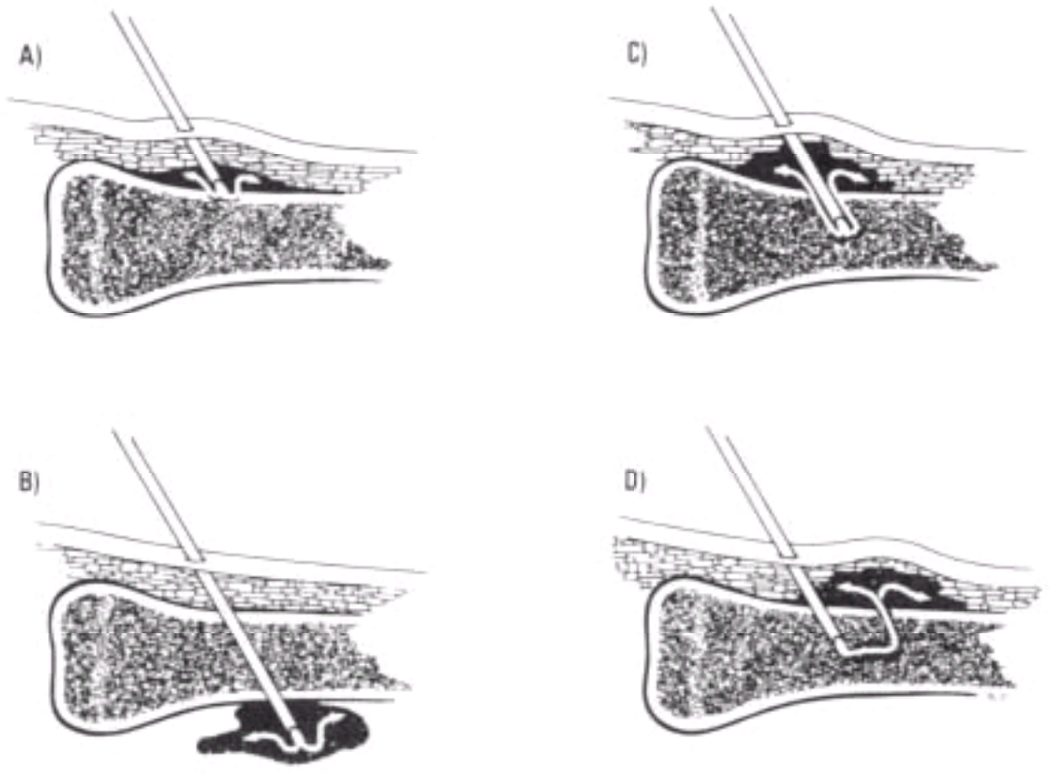


Figure 26-13 Radiograph of bilaterally misplaced IO needles with penetration through the posterior tibial cortices.

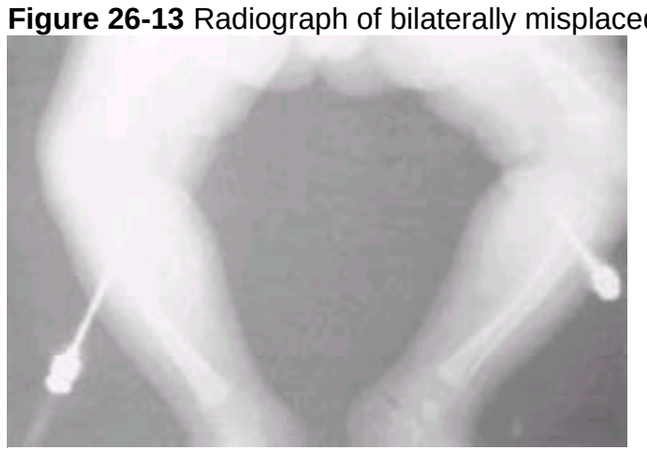


Figure 26-14 Radiograph *(A)* and bone scan *(B)* of the tibia demonstrating an inflammatory reaction 4 days after the patient received intraosseous (IO) phenytoin and phenobarbital. The periosteum is elevated along the length of the bone, mimicking osteomyelitis on the plain films and the bone scan. A diagnosis of osteomyelitis requires either clinical evidence of infectious toxicity or positive cultures (blood or periosteal aspirate).

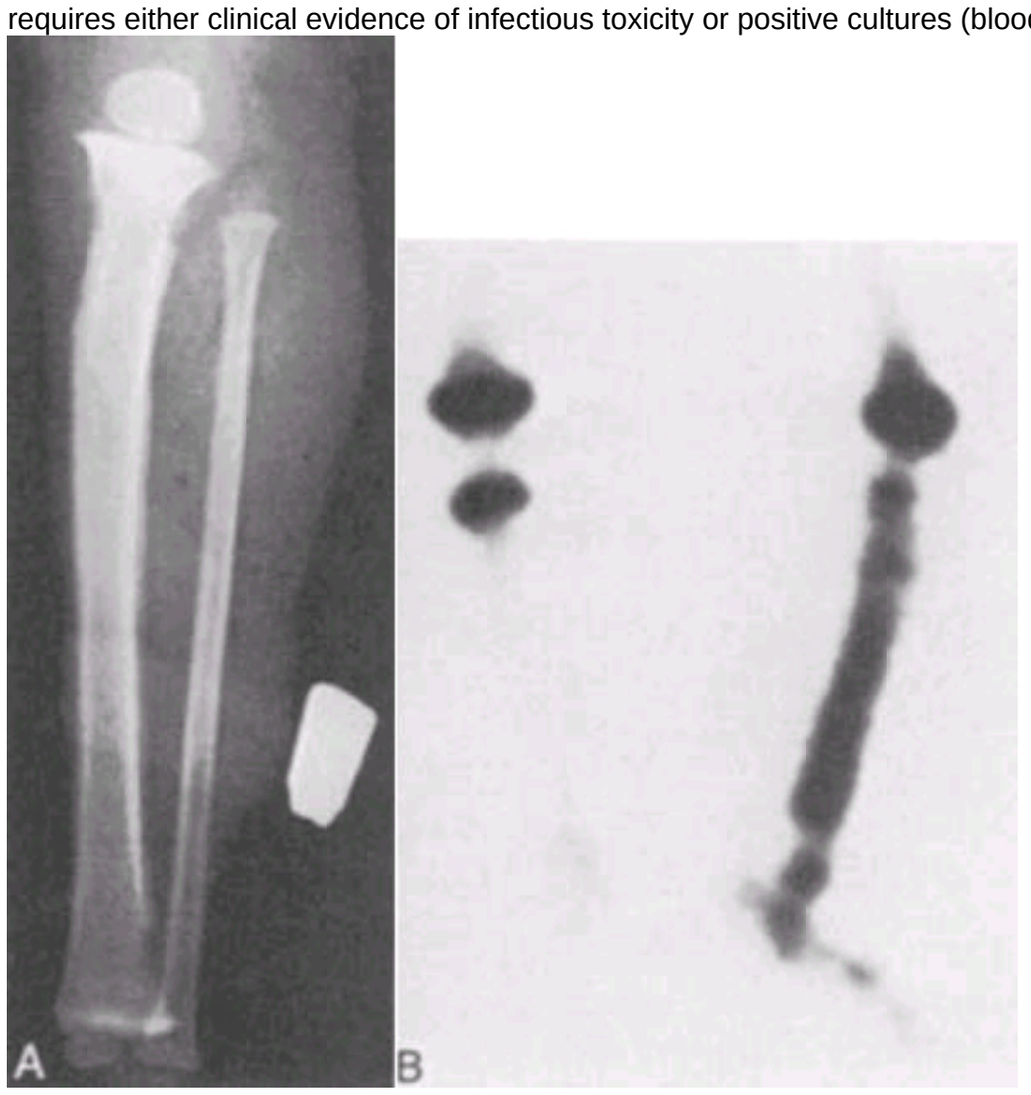


Figure 27-1 Example of a prefilled syringe, which is frequently used in endotracheal therapy.

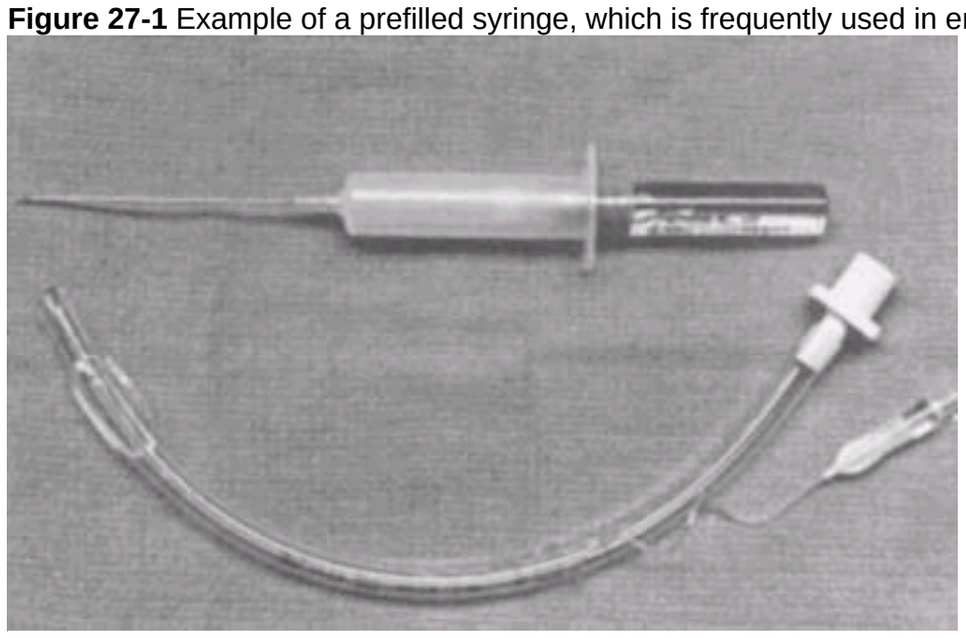


Figure 27-2 Use of a central venous catheter for endotracheal drug delivery.

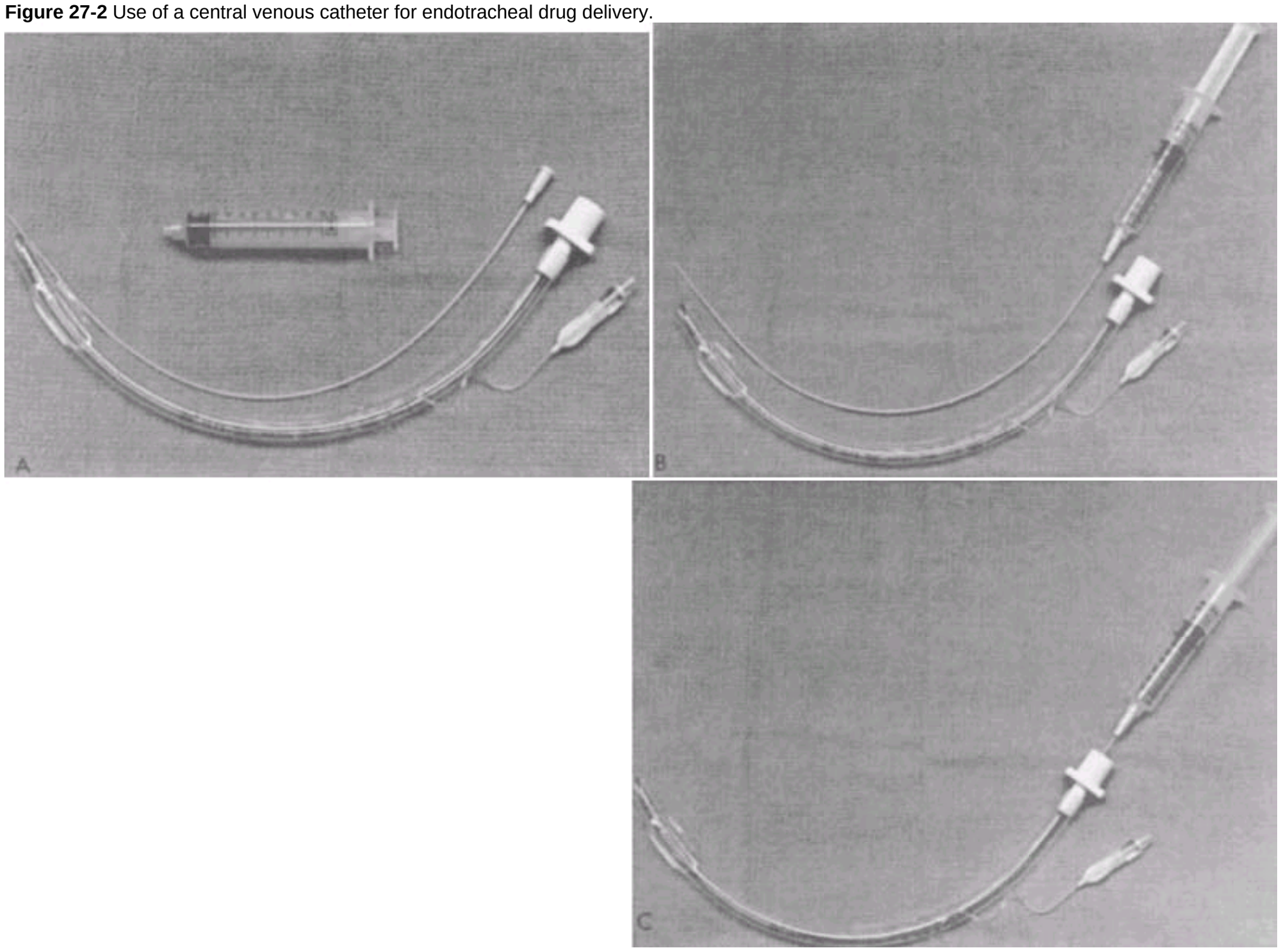


Figure 27-3 Method of drug injection through the endotracheal tube wall.

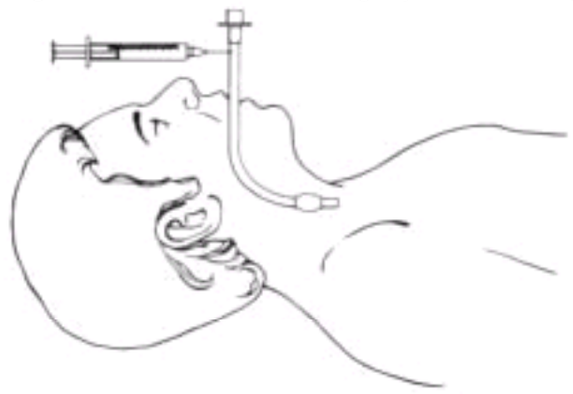


Figure 28-1 Technique of autotransfusion from traumatic hemothorax. *A*, Blood is collected into sterile blood collection bottle with 400 mL normal saline. *B*, After blood is collected, the chest tube is disconnected from the collection bottle. *C*, Blood is infused directly from the blood collection bottle while a new chest tube drainage bottle is connected to the chest tube. Or, *D*, blood collected from the chest tube is transferred into a sterile blood bag. *E*, Blood transferred into blood bag is infused into patient. *(From Symbas PN: Extraoperative autotransfusion from hemothorax. Surgery 84:722, 1978.)*

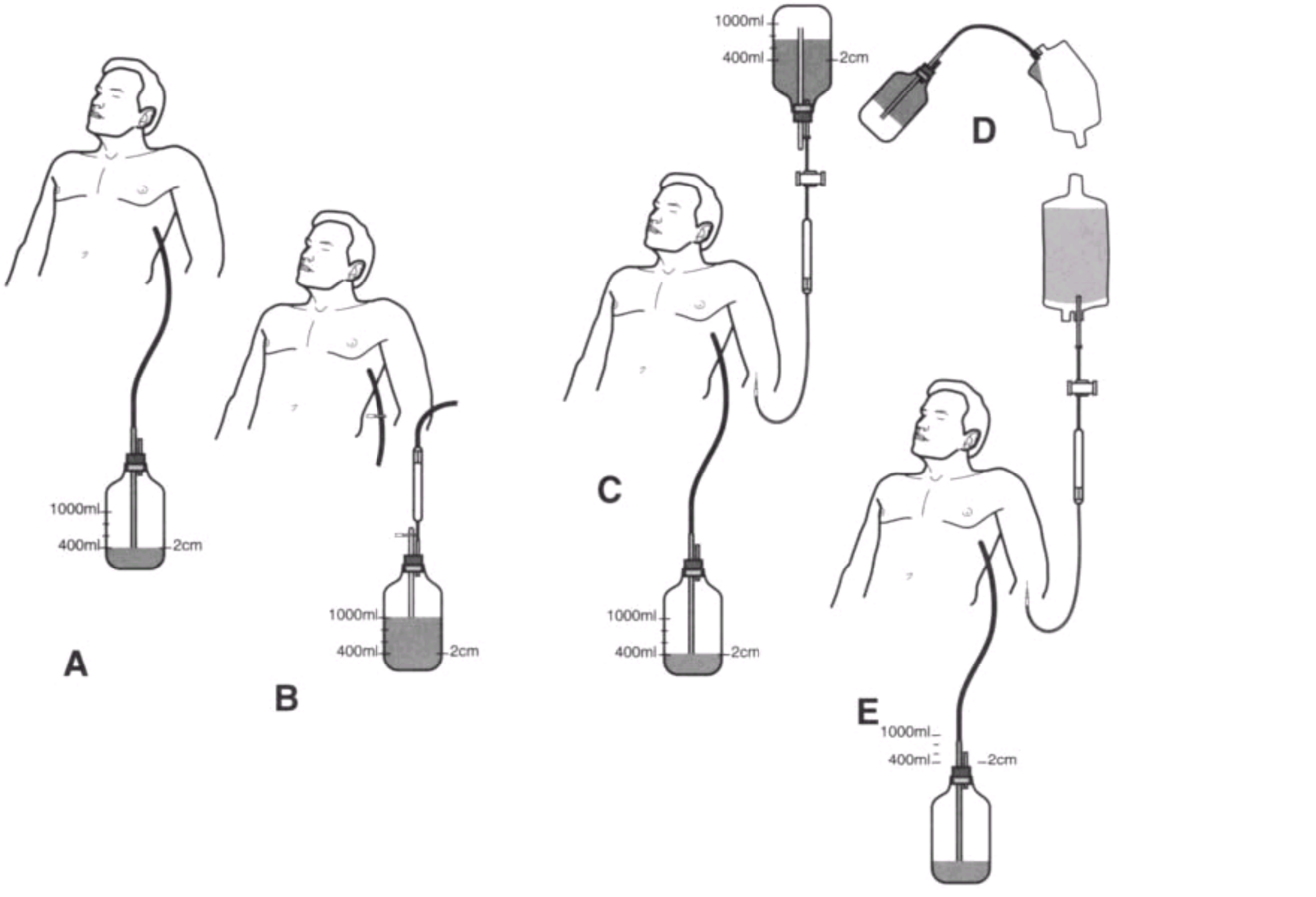


Figure 28-2 Heimlich valve connected directly to Abbott Receptal Bag. *(From Von Koch L, Defore WW, Mattox KL: A practical method of autotransfusion in the emergency center. Am J Surg 133:770, 1977.)*

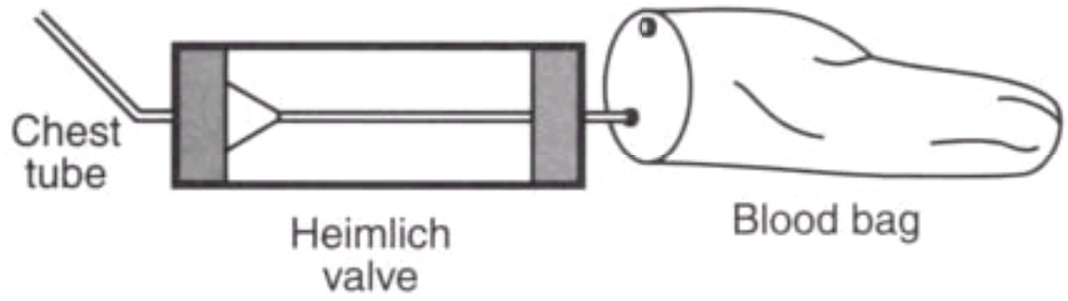


Figure 28-4 Attaching the metal hanger to the Pleur-Evac system, which will hold the blood collection bag. *(Courtesy of Deknatel, Inc., Fall River, MA 02720. Reproduced by permission.)*

Figure 28-5 The blood collection bag that is attached to the standard Pleur-Evac system. *(Courtesy of Deknatel, Inc., Fall River, MA 02720. Reproduced by permission.)*

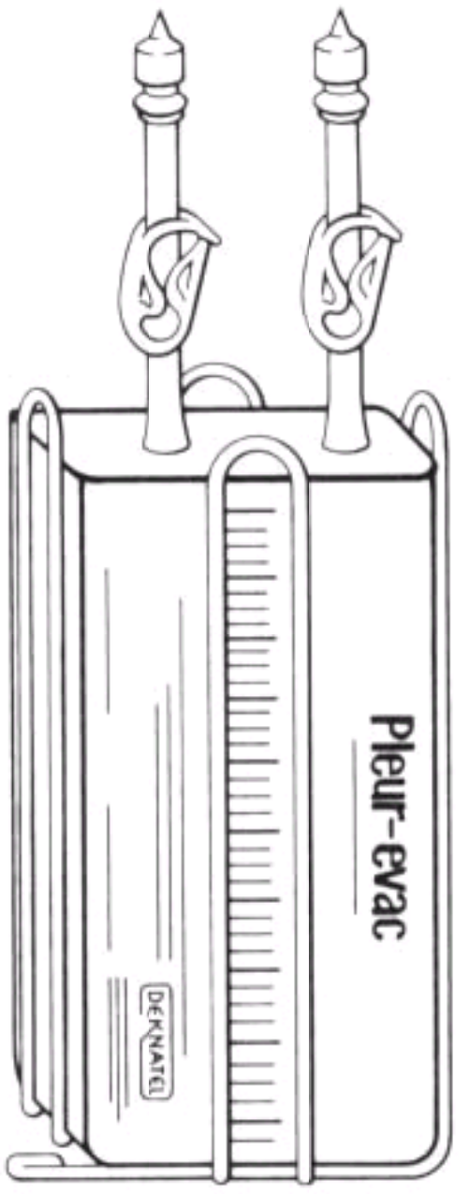


Figure 28-6 Preparation for reinfusion. After air is vented from the collection bag to minimize the chance of air embolism, insert the free recipient arm *(L)* of the prepared Y-type infusion line into the stem of the collection bag. *(Courtesy of Abbott Laboratories, Abbott Park, IL 60064. Reproduced by permission.)*

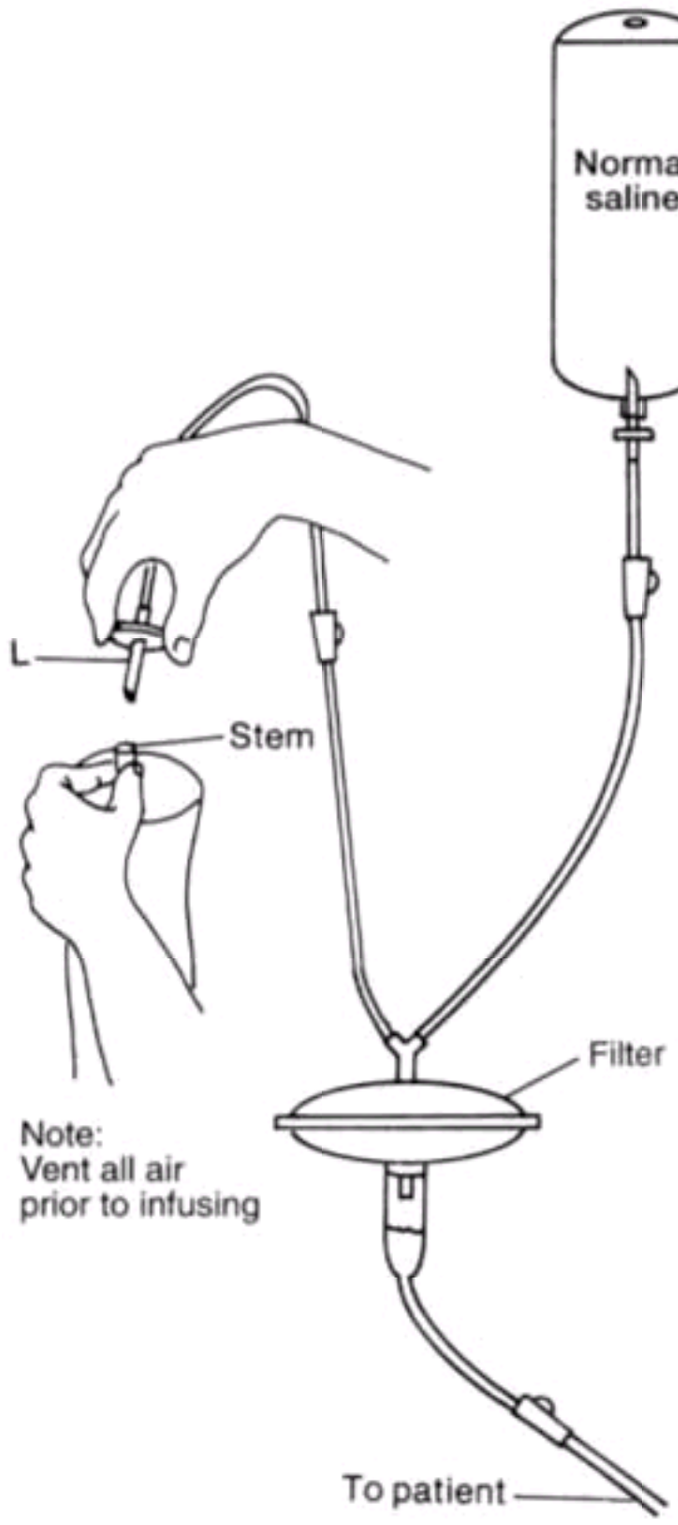


Figure 28-7 Reinfusion of collected blood with the Pleur-Evac system. A second collection may be obtained while this blood is infusing. *(Courtesy of Deknatel, Inc., Fall River, MA 02720. Reproduced by permission.)*

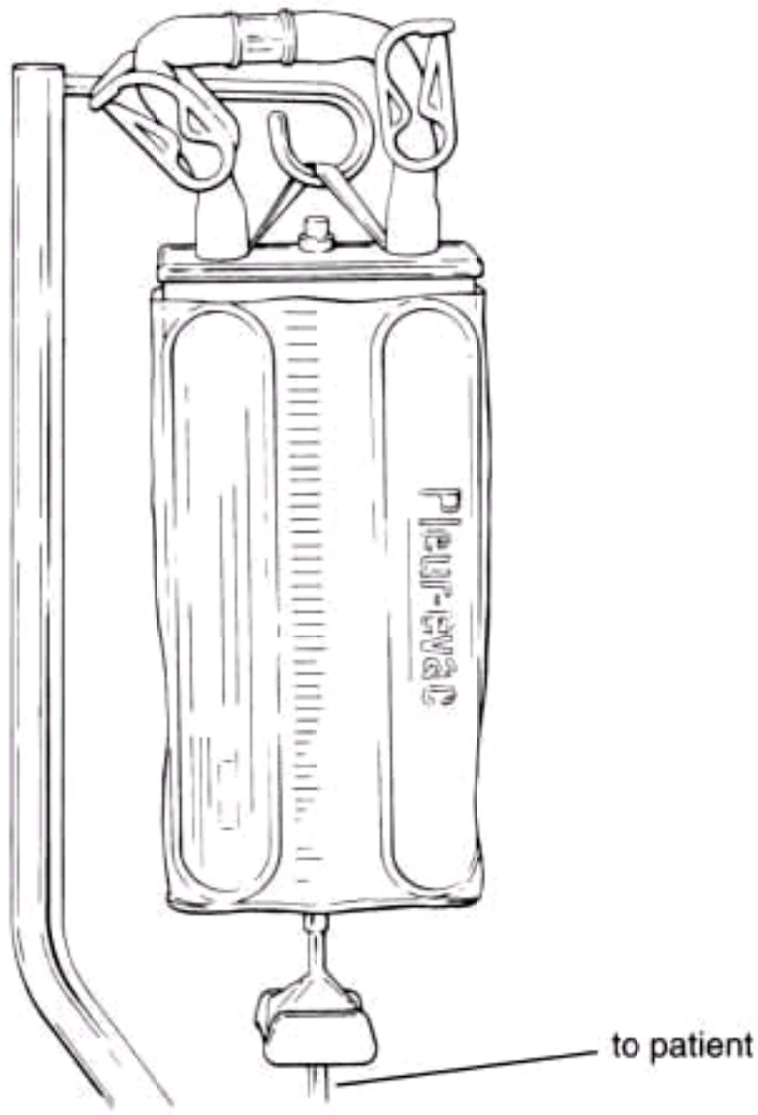


Figure 28-8 Atrium chest tube and autotransfusion system (ATS) prior to connection. *A*, Female ATS connector. *B*, Patient chest tube connector. *C*, Male ATS connector. *D*, Chest tube drain connector. *(Courtesy of Atrium Medical Corporation, Hudson, NH 03051.)*

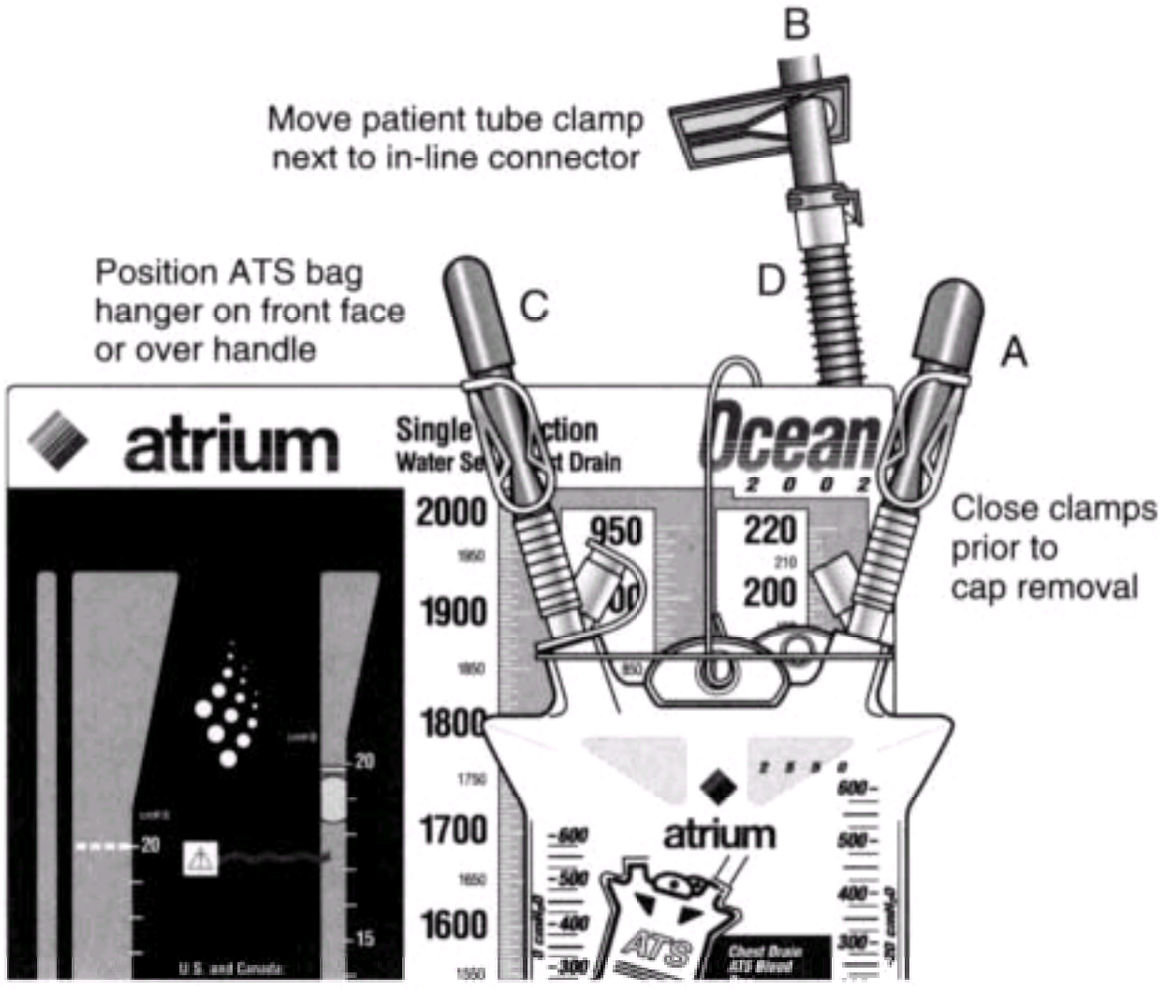


Figure 28-9 Separation of the patient chest tube and chest tube drainage system by depression of connector lock and pulling to separate. *(Courtesy of Atrium Medical Corporation, Hudson, NH 03051.)*

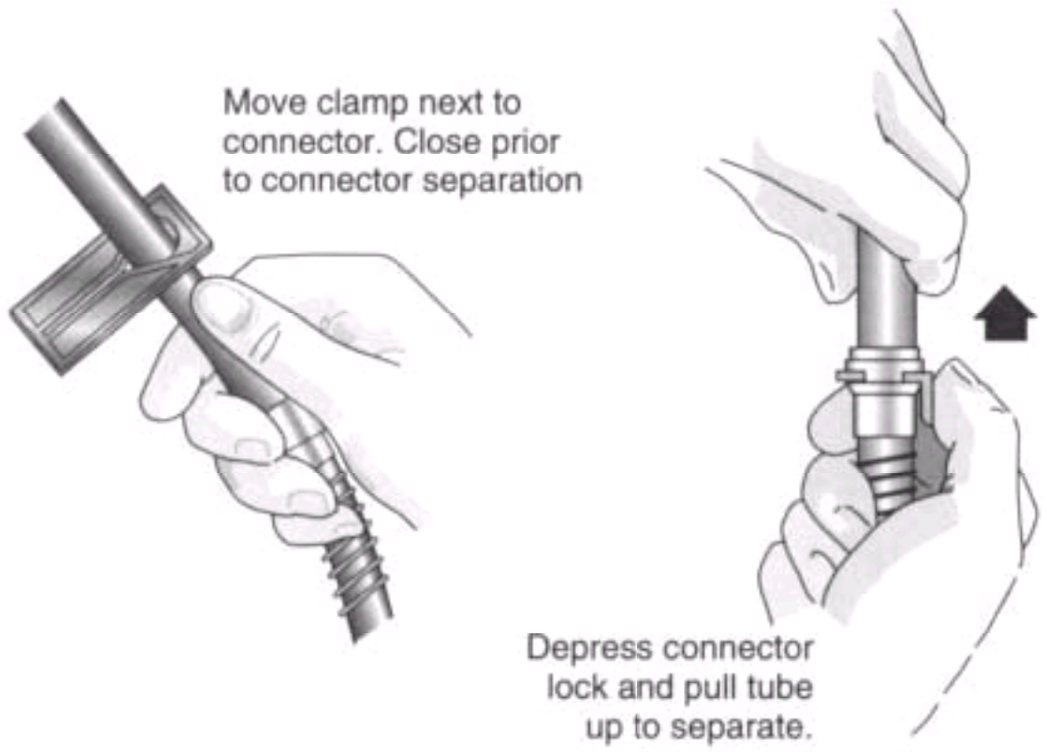


Figure 28-10 Atrium chest tube and autotransfusion system (ATS) during blood collection. *A*, Female ATS connector. *B*, Patient chest tube connector. *C*, Male ATS connector. *D*, Chest tube drain connector. *(Courtesy of Atrium Medical Corporation, Hudson, NH 03051.)*

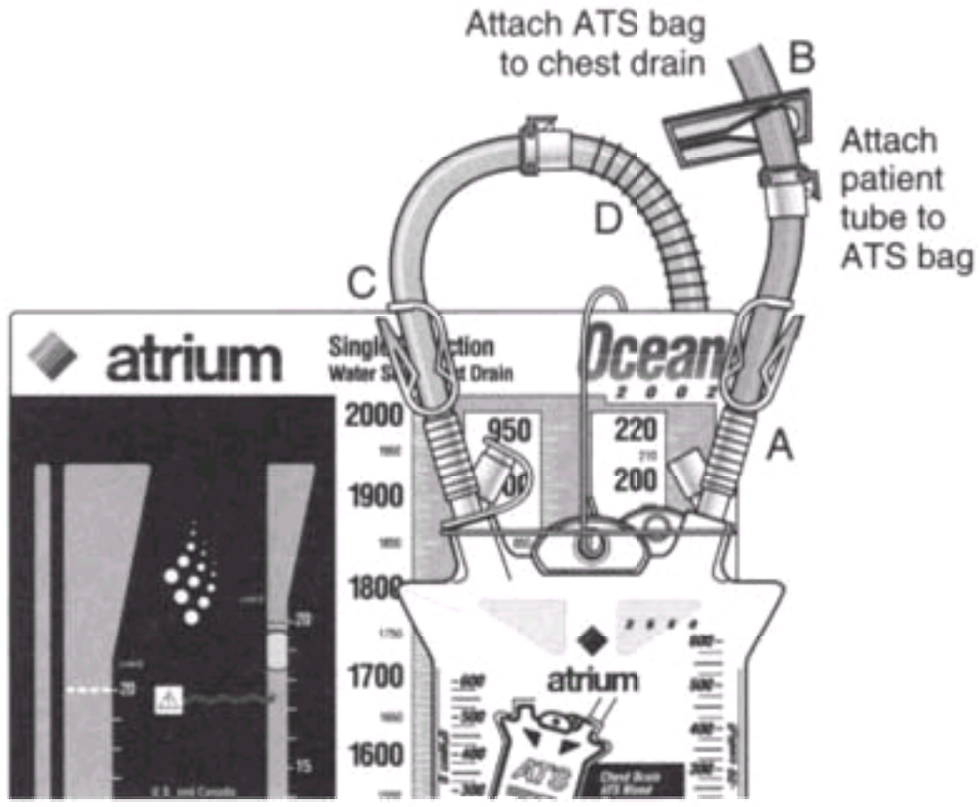


Figure 28-11 Atrium chest tube and autotransfusion system after blood collection. *A*, Female ATS connector. *B*, Patient chest tube connector. *C*, Male ATS connector. *D*, Chest tube drain connector. *(Courtesy of Atrium Medical Corporation, Hudson, NH 03051.)*

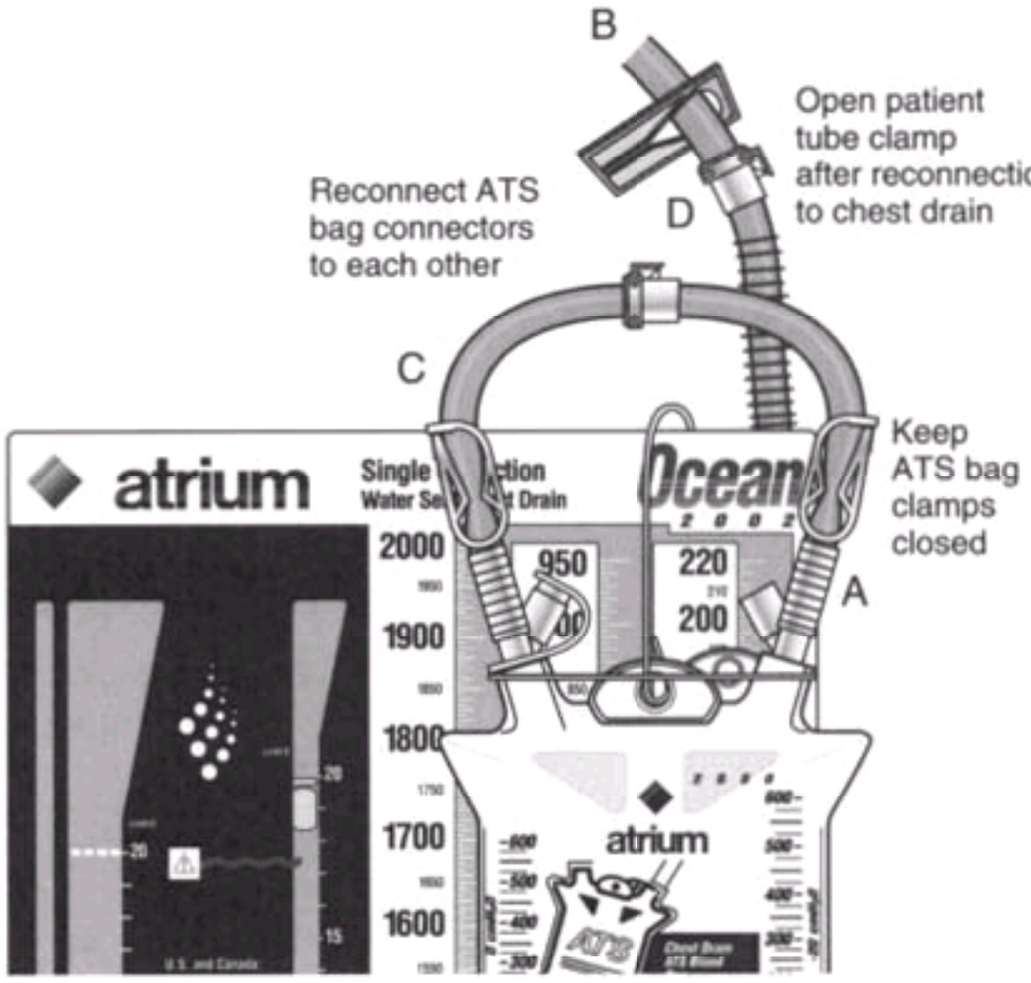


Figure 28-12 Atrium continuous autotransfusion system. *A*, Autotransfusion system (ATS) access line. *F*, ATS access line clamp. *(Courtesy of Atrium Medical Corporation, Hudson, NH 03051.)*

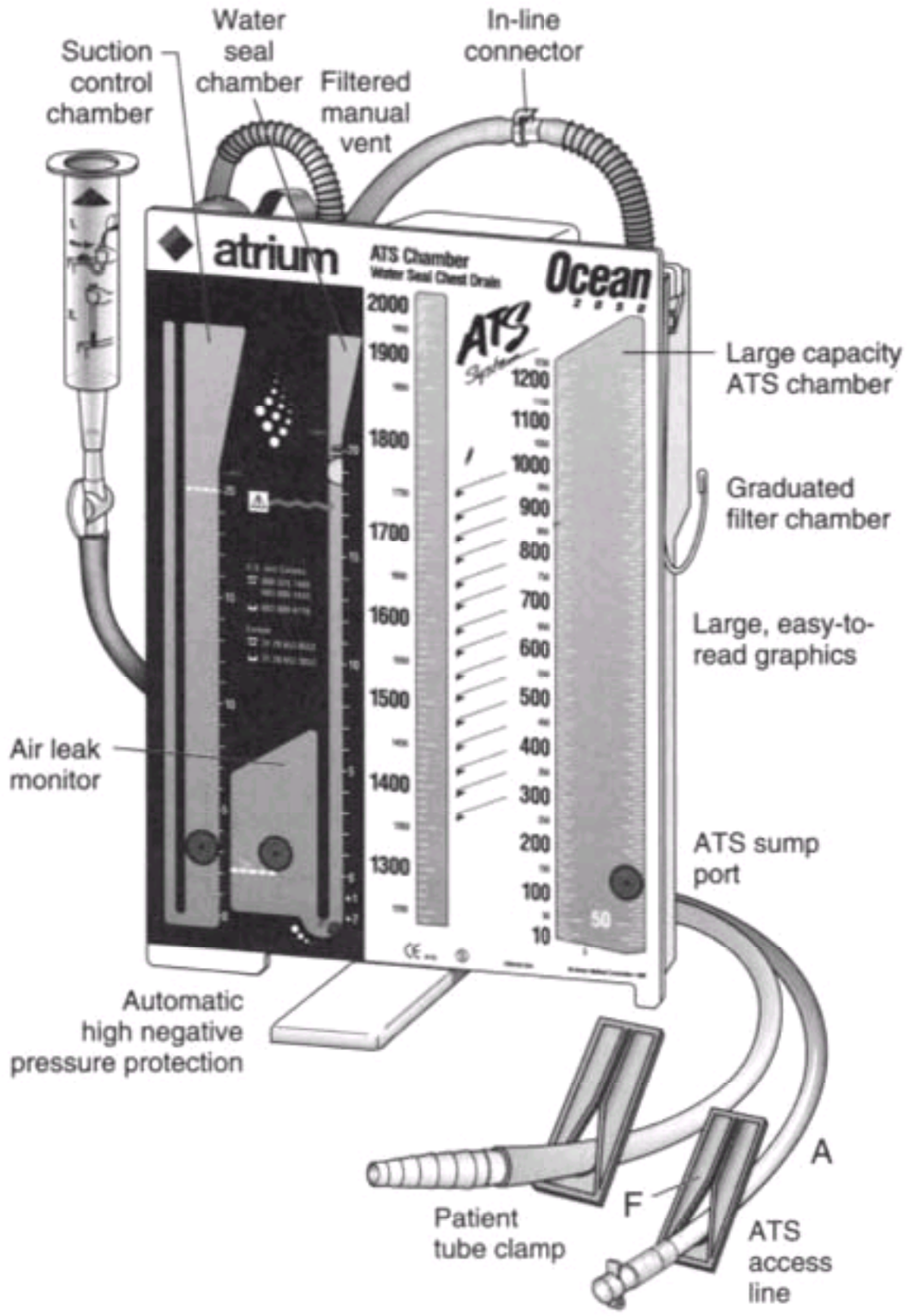


Figure 28-13 Infusion pump set up for continuous autotransfusion. *A*, Autotransfusion system (ATS) access line. *B*, End of ATS access line. *C*, Blood filter. *D*, IV tubing with "spike up" position. *E*, Self-priming IV pump. *G*, line to patient IV access. *(Courtesy of Atrium Medical Corporation, Hudson, NH 03051.)*

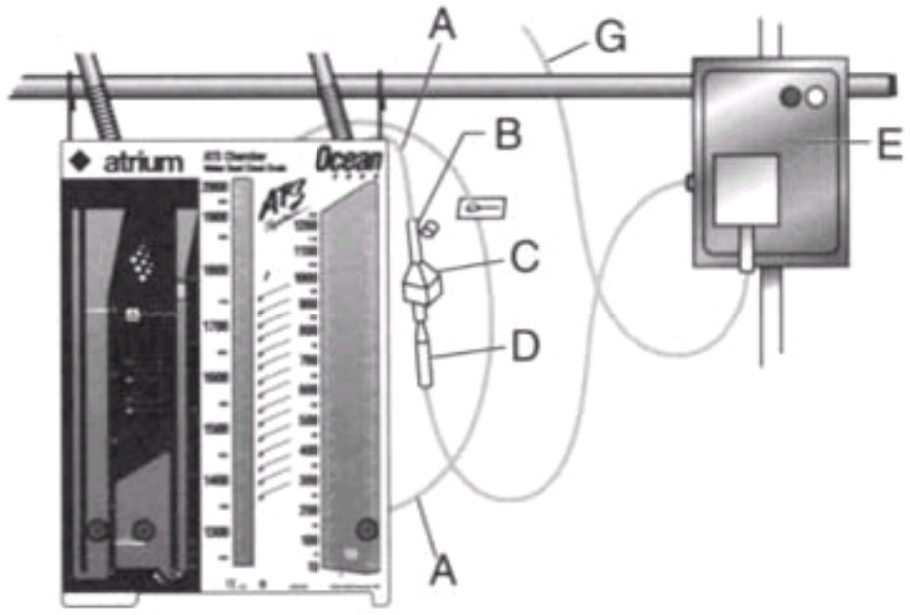


Figure 28-14 Autotransfusion connection to patient. *A*, Autotransfusion system (ATS) access line. *B*, End of ATS access line. *C*, Blood filter. *D*, IV tubing with "spike up" position. *E*, Self-priming IV pump. *G*, Patient IV access. *(Courtesy of Atrium Medical Corporation, Hudson, NH 03051.)*

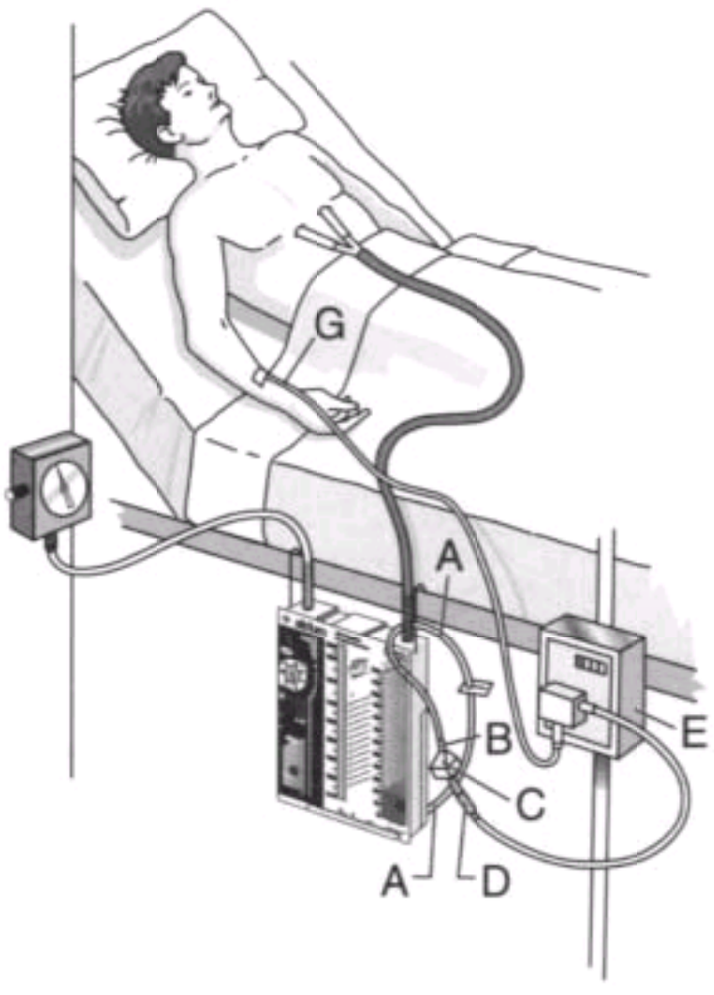


Figure 28-15 Abbott Receptal disposable collection apparatus. *A,* Anticoagulant volume control burette (fill with 50 mL of citrate phosphate dextrose [CPD] anticoagulant). *B,* Chest tube. *C,* Latex drainage tubing. *D,* Male-to-male connector. *E,* End of drainage tubing with side port. *F,* Inlet port of red liner cap attached to collection canister. *G,* Collection liner bag. *H,* Downstream suction hose (do not exceed 60 mm Hg of suction). *J,* Liner lid tubing connector. *K,* Canister tee. *N,* Liner stem with protective cap. *(Modified from Young GP, Purcell TB: Emergency autotransfusion. Ann Emerg Med 12:180, 1983. Reproduced by permission.)*

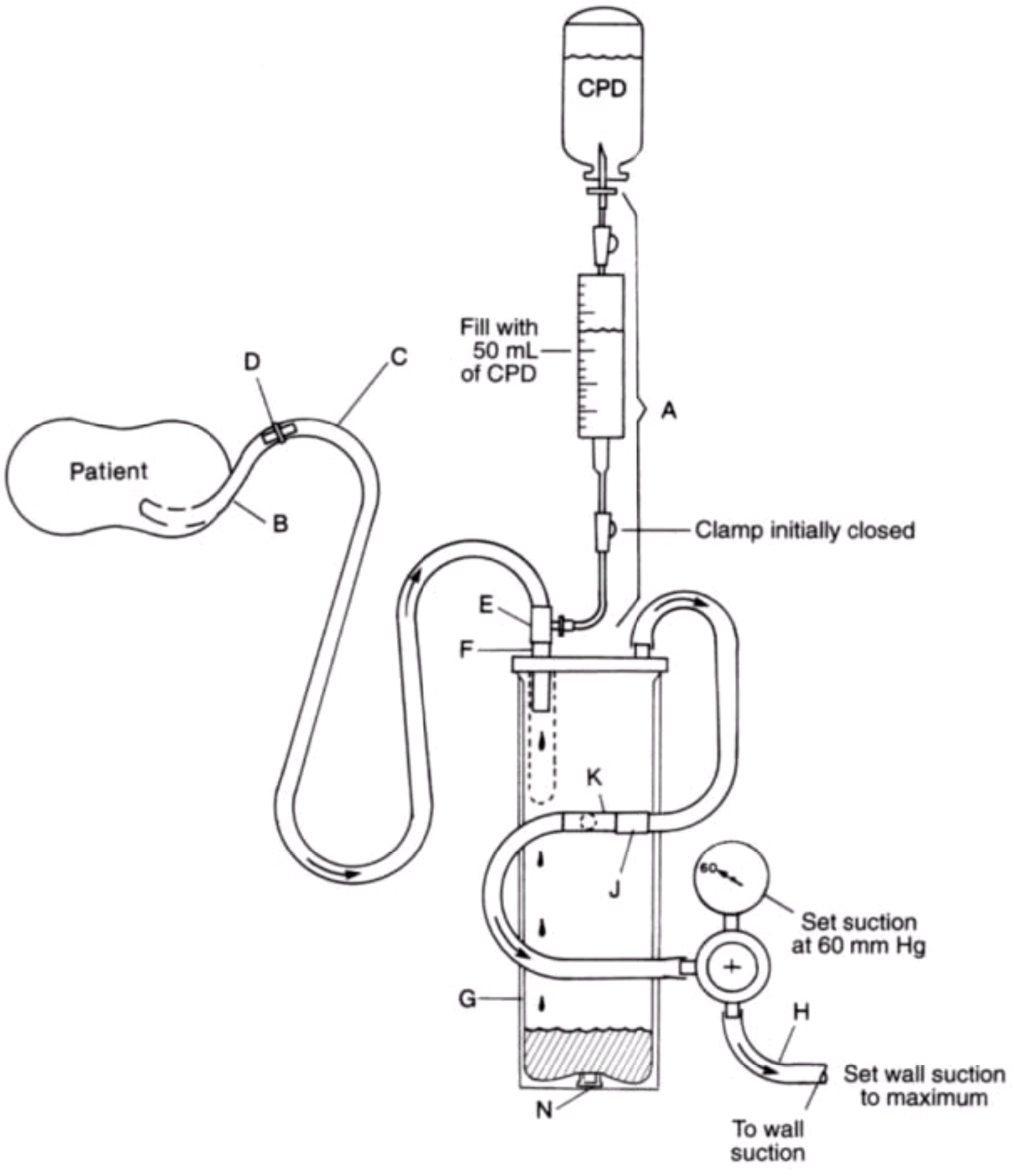


Figure 28-16 Abbott Receptal collection apparatus. Detail of canister connections before attachment of anticoagulant line. *(Courtesy of Abbott Laboratories, Abbott Park, IL 60064. Reproduced by permission.)*

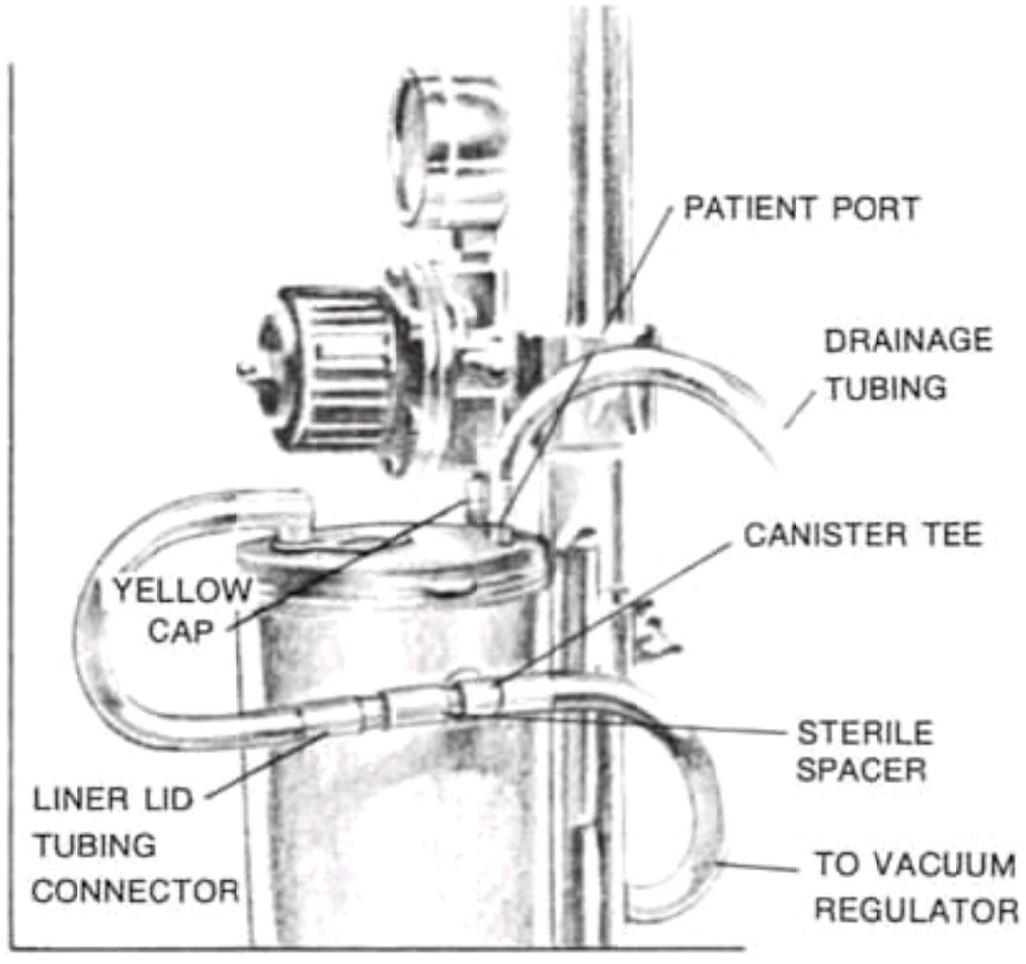


Figure 28-17 Proper attachment of anticoagulant line for Abbott Receptal device. The preferred anticoagulant is citrate phosphate dextrose (CPD) in a 1:7 volume ratio of anticoagulant to blood. *(Courtesy of Abbott Laboratories, Abbott Park, IL 60064. Reproduced by permission.)*

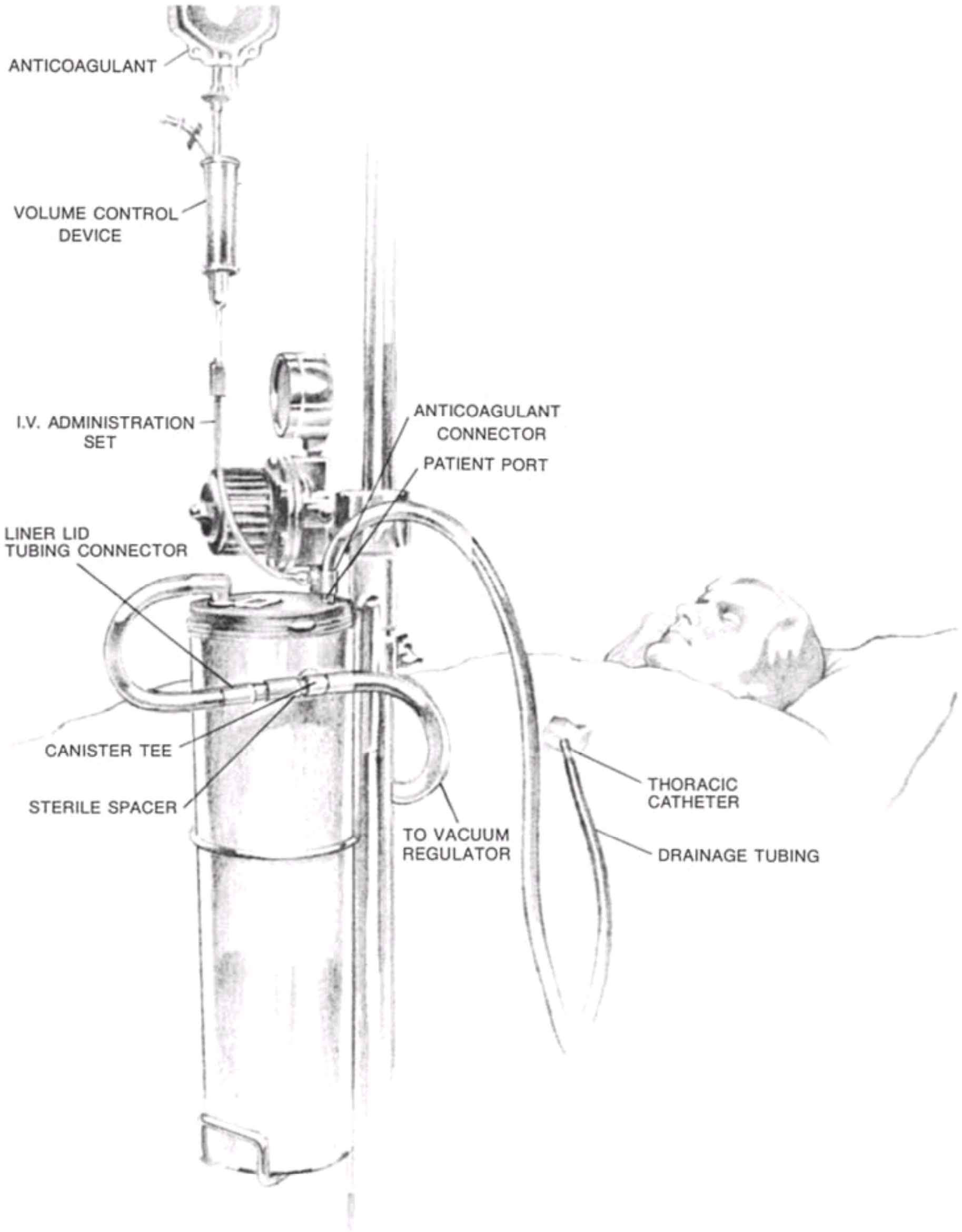


Figure 28-18 Reinfusion apparatus. Inlet port of liner cap (*F*), Y-type blood infusion line with in-line 40-µm filter (*I*), liner lid tubing connector (*J*), liner bag connection with infusion line (*L*), attached hanger tab at top of liner bag (*M*), and normal saline IV fluid for priming and maintaining patency of the system during liner bag changes (*N/S*). *(Modified from Young GP, Purcell TB: Emergency autotransfusion. Ann Emerg Med 12:180, 1983. Reproduced by permission.)*

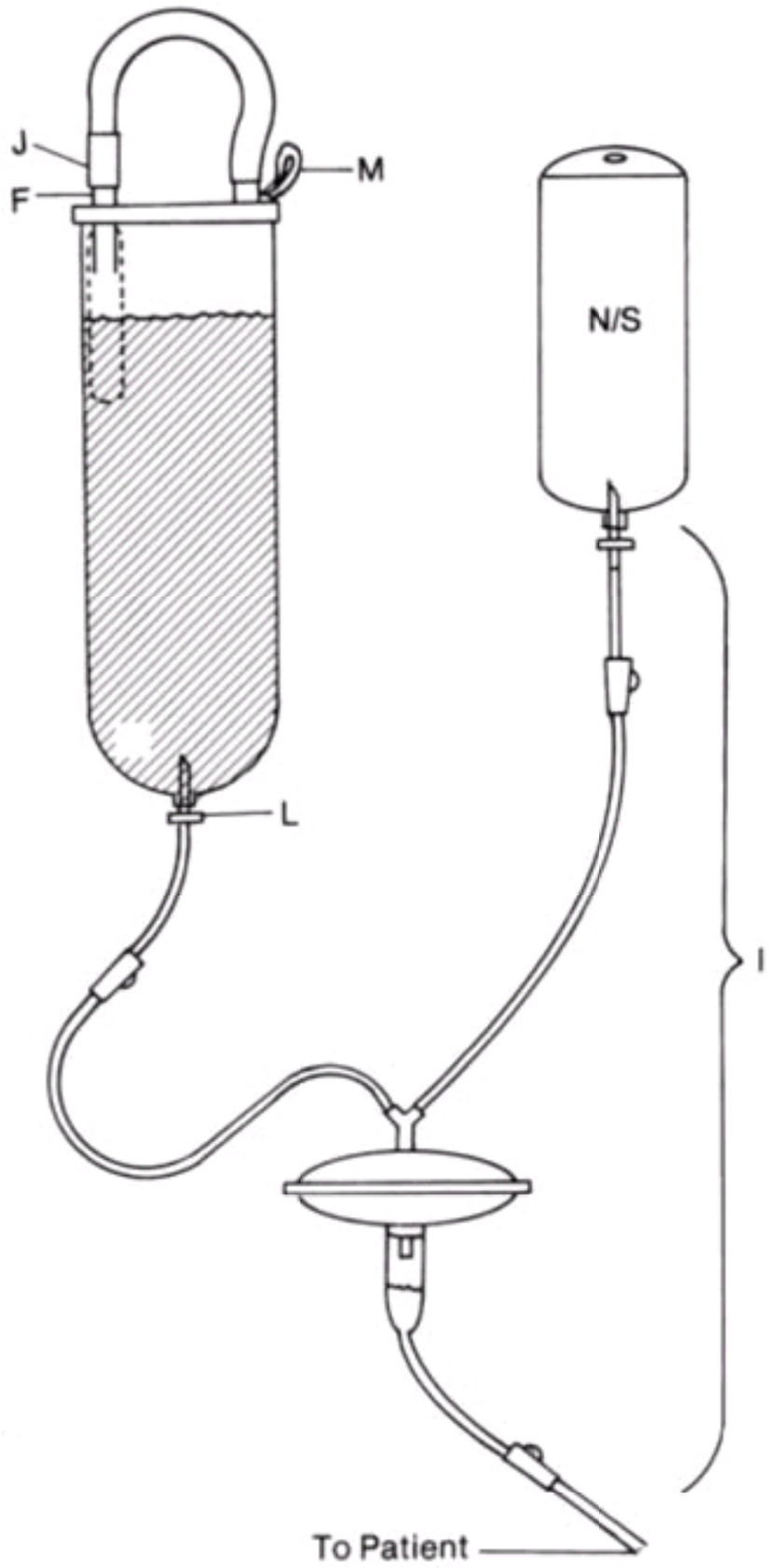


Figure 28-19 Preparation for reinfusion. Removal of liner lid tubing connector (*J*) from canister tee (*K*). Inlet port (*F*) is shown. *(Courtesy of Abbott Laboratories, Abbott Park, IL 60064. Reproduced by permission.)*

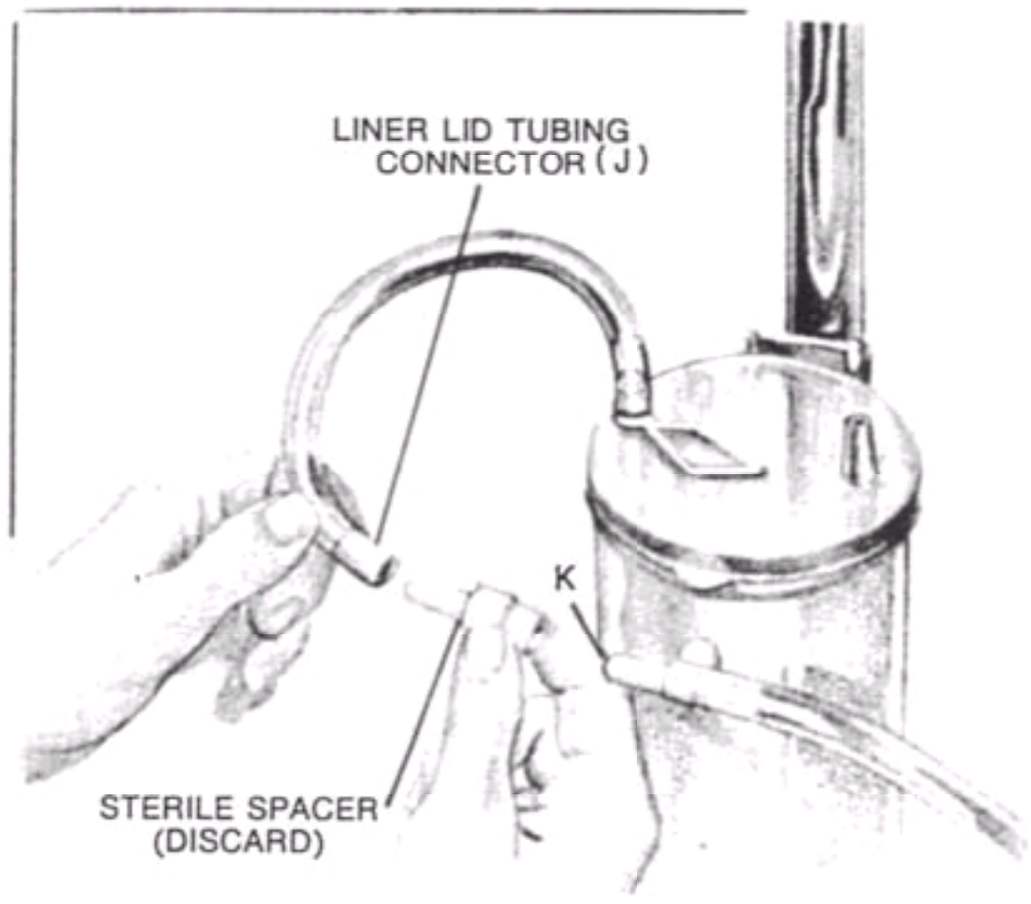


Figure 28-20 Preparation for reinfusion. Removal of liner assembly from the canister after connecting the liner lid tubing connector (*J*) to the inlet port of the liner cap. *(Courtesy of Abbott Laboratories, Abbott Park, IL 60064. Reproduced by permission.)*

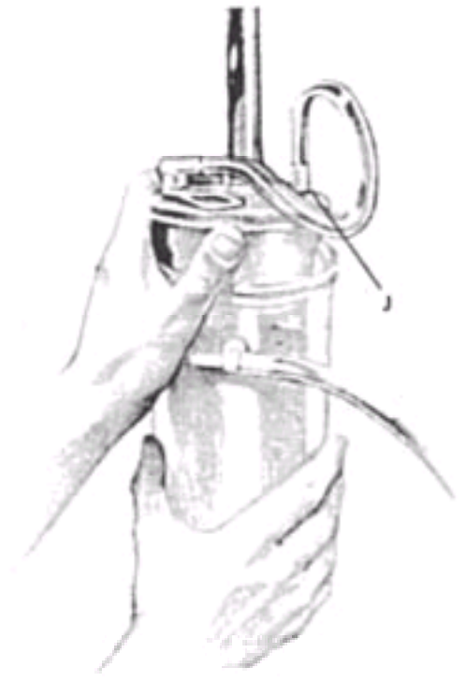


Figure 28-21 Straightening the liner bag before insertion into the canister. *(Courtesy of Abbott Laboratories, Abbott Park, IL 60064. Reproduced by permission.)*

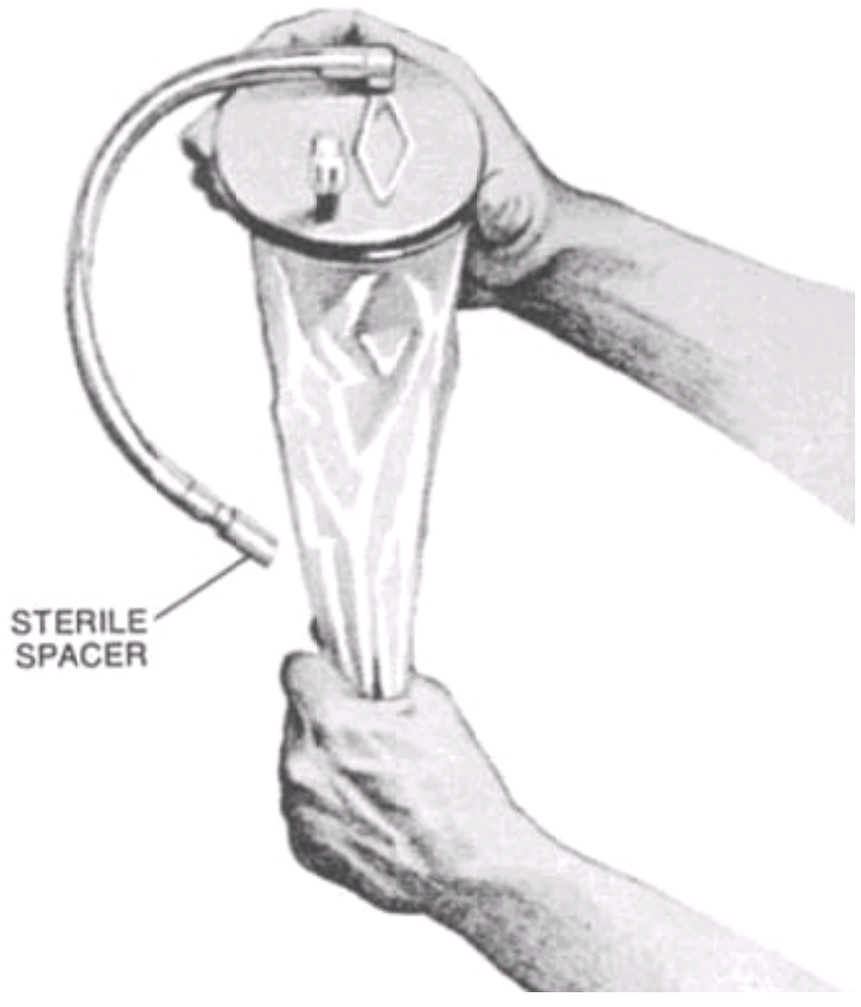


Figure 28-22 Proper placement of the liner bag lid on the canister. *(Courtesy of Abbott Laboratories, Abbott Park, IL 60064. Reproduced by permission.)*

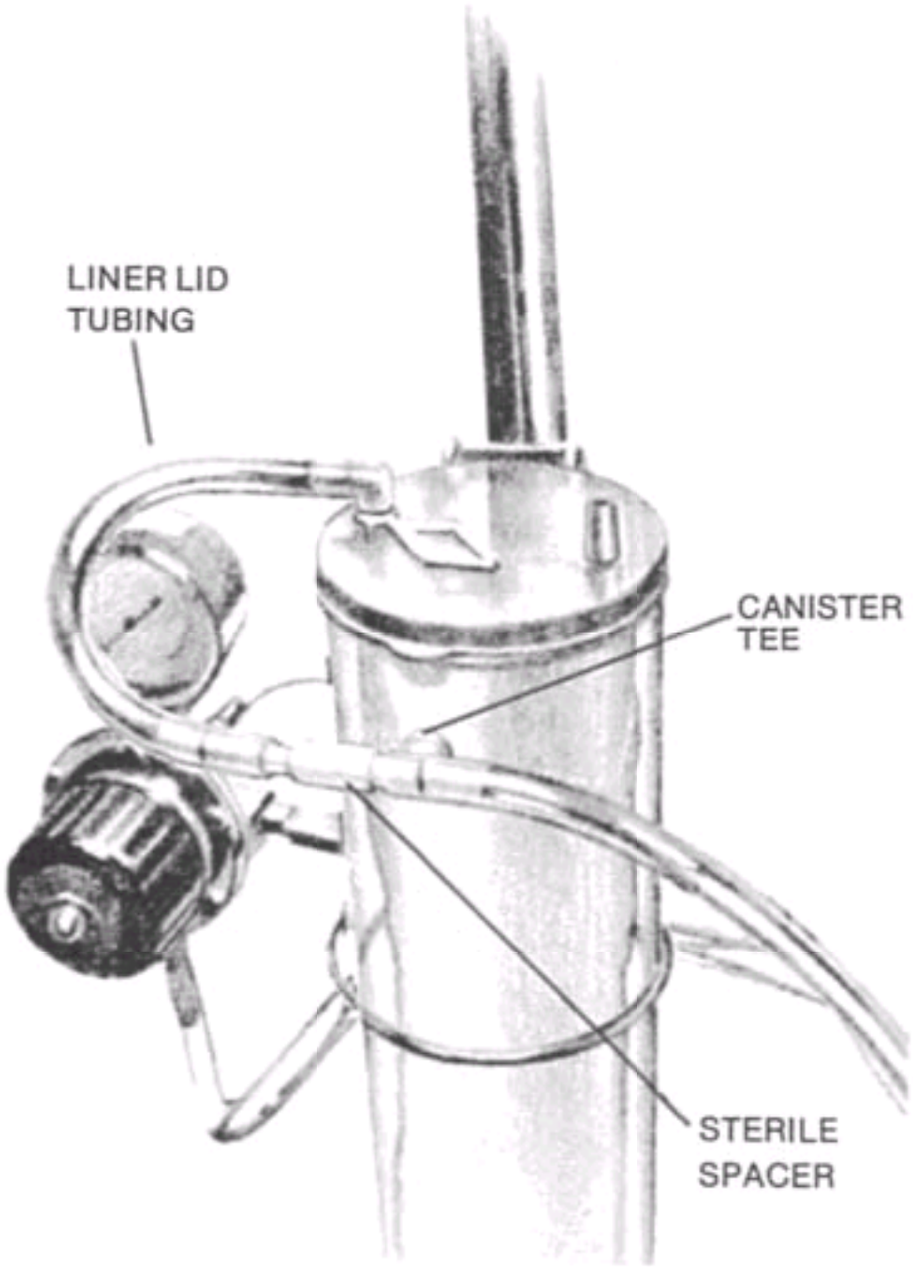


Figure 28-23 Boehringer Autovac System, collection configuration. *(Courtesy of Boehringer Laboratories, Inc., Norristown, PA. Reproduced by permission.)*

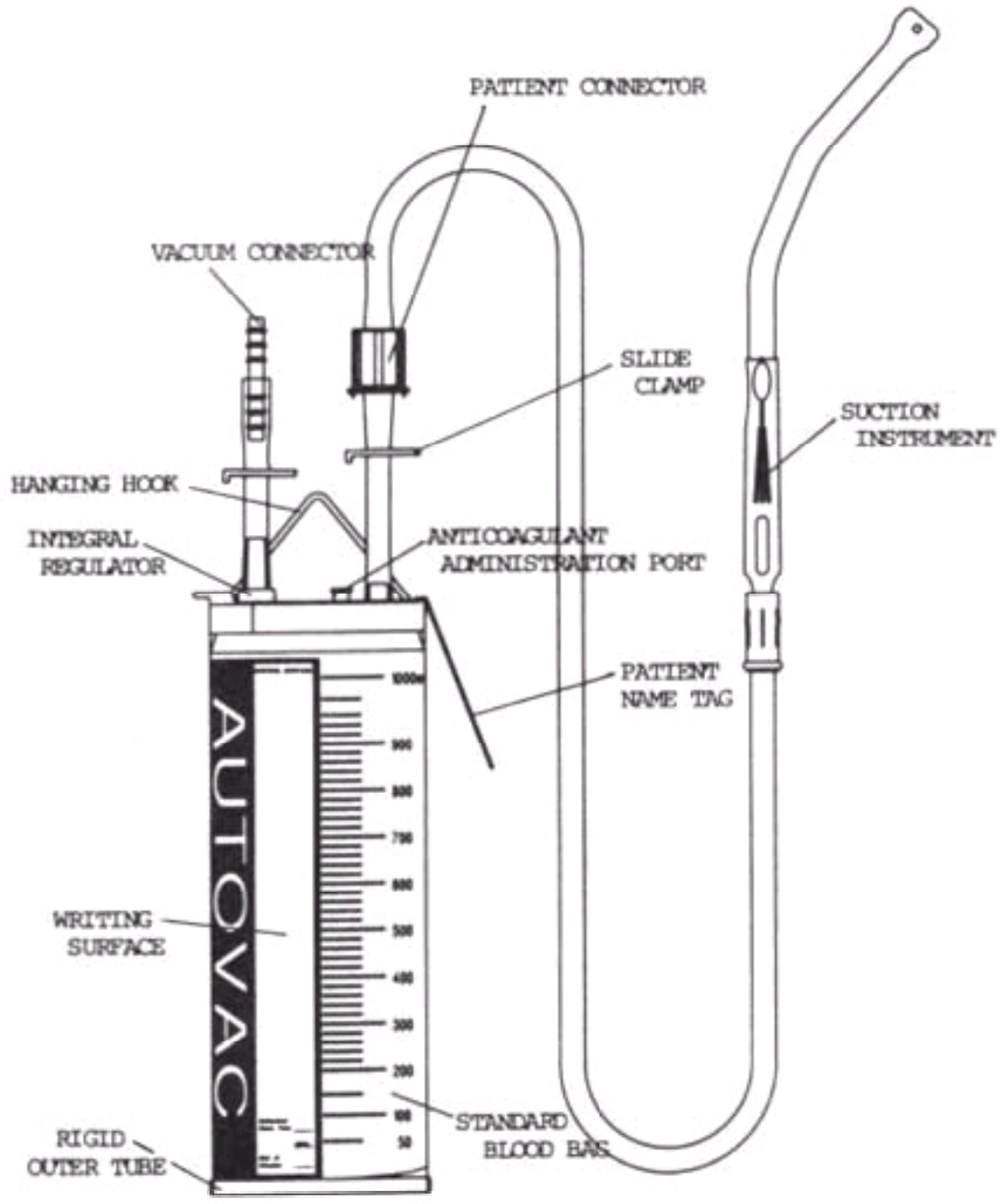


Figure 28-24 Boehringer reinfusion apparatus. *(Courtesy of Boehringer Laboratories, Inc., Norristown, PA. Reproduced by permission.)*

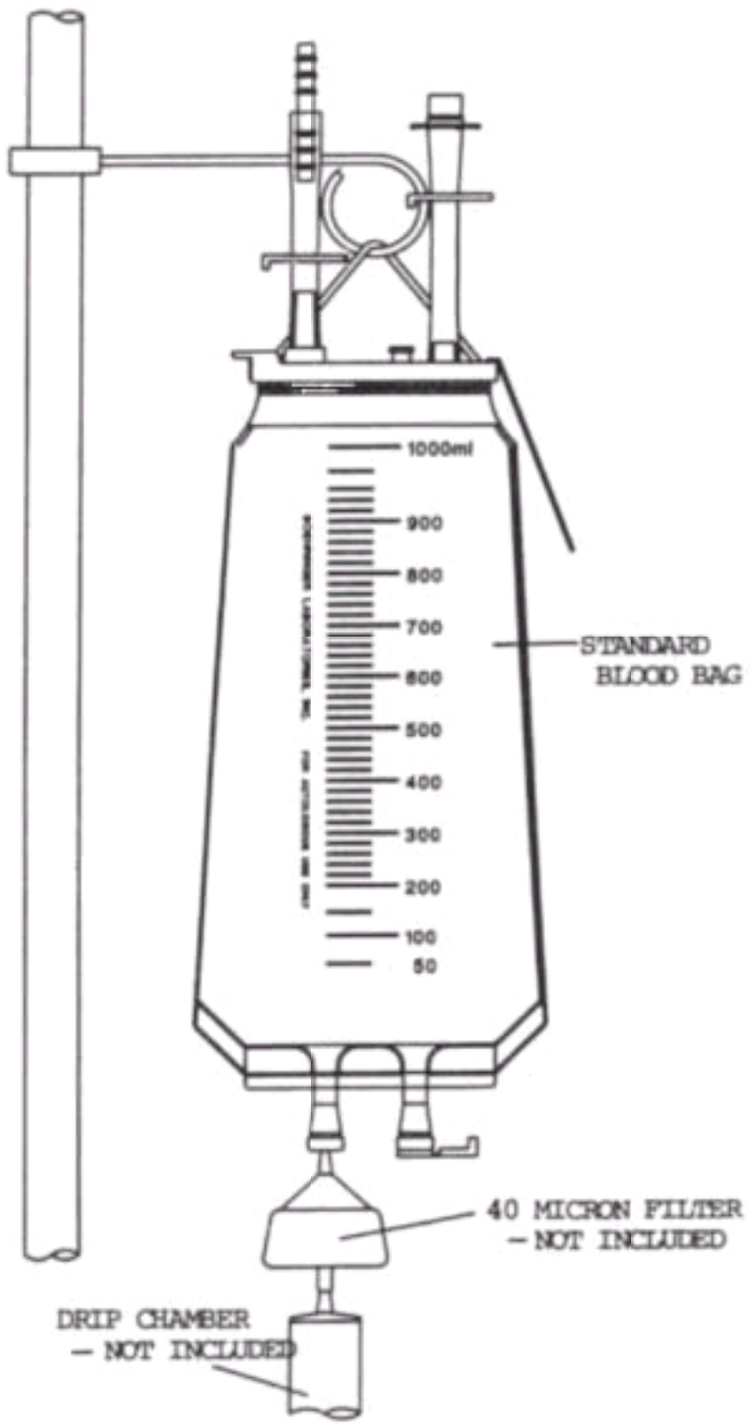


Figure 28-25 The bloodless surgery station. *(Courtesy of Harvest Blood Conservation, Inc., Plymouth MA 02360.)*

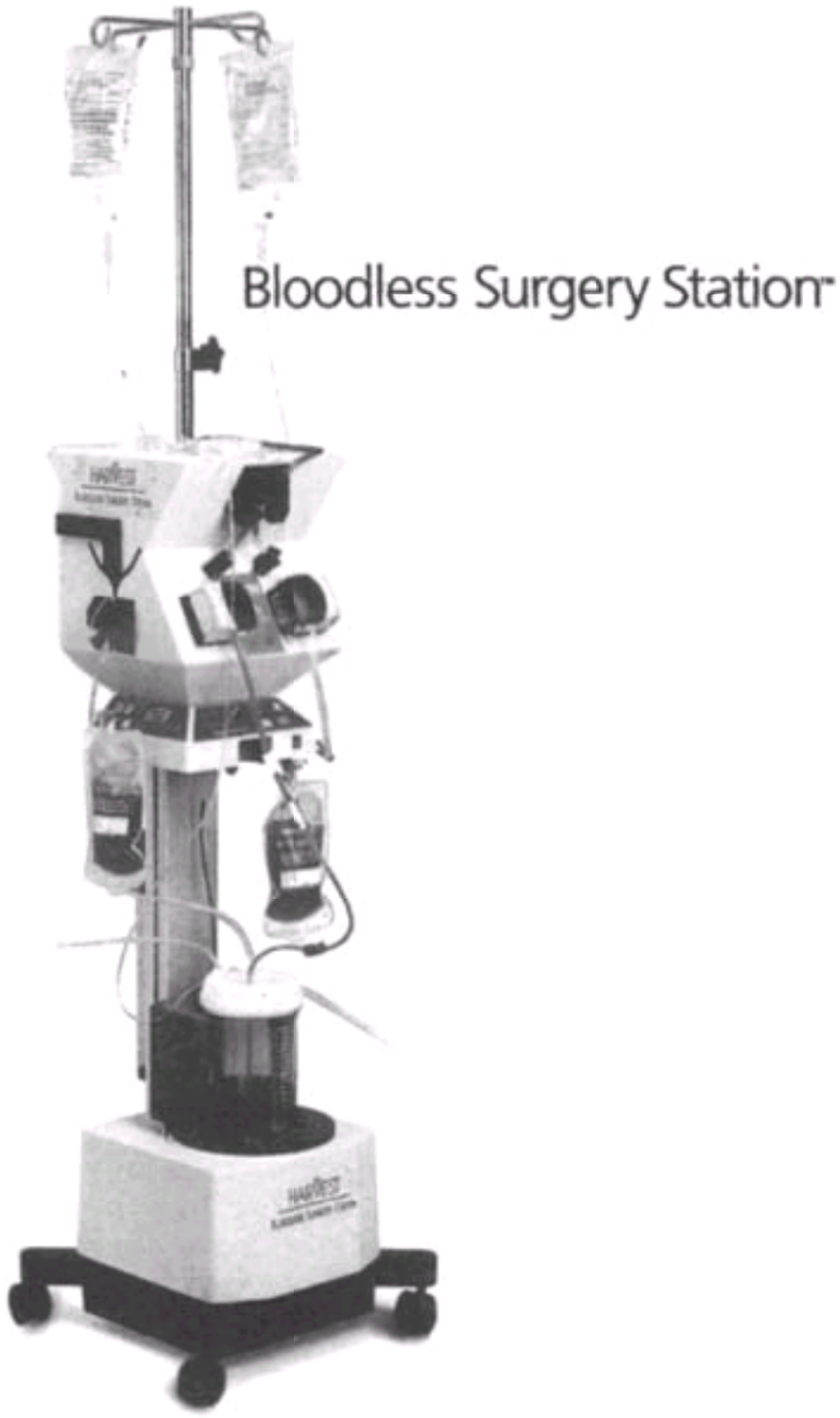


Figure 29-1 The Typenex blood recipient identification system. The identity of the patient and the blood sample are ensured by numbered labels on the tube and on the bracelet. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

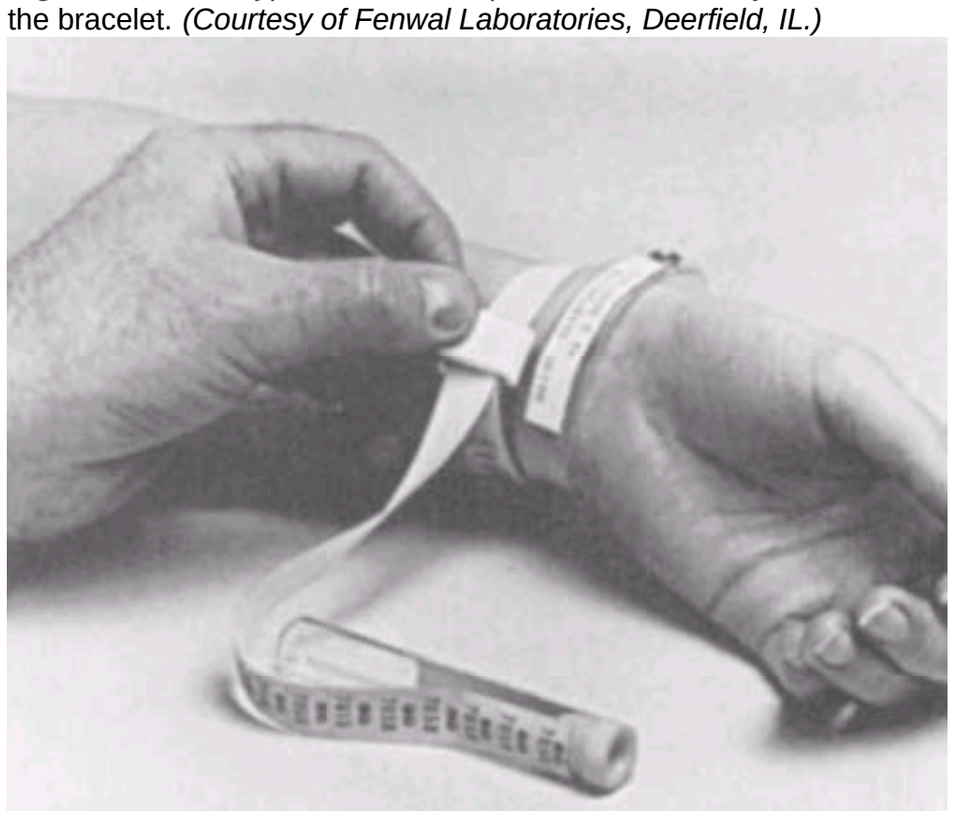


Figure 29-2 In the blood bank, crossmatched units of blood are identified with numbered labels from the patient's blood sample. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

Figure 29-3 Before administration of the blood unit, the numbered labels on the patient's bracelet and on the unit of blood are checked for identity. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

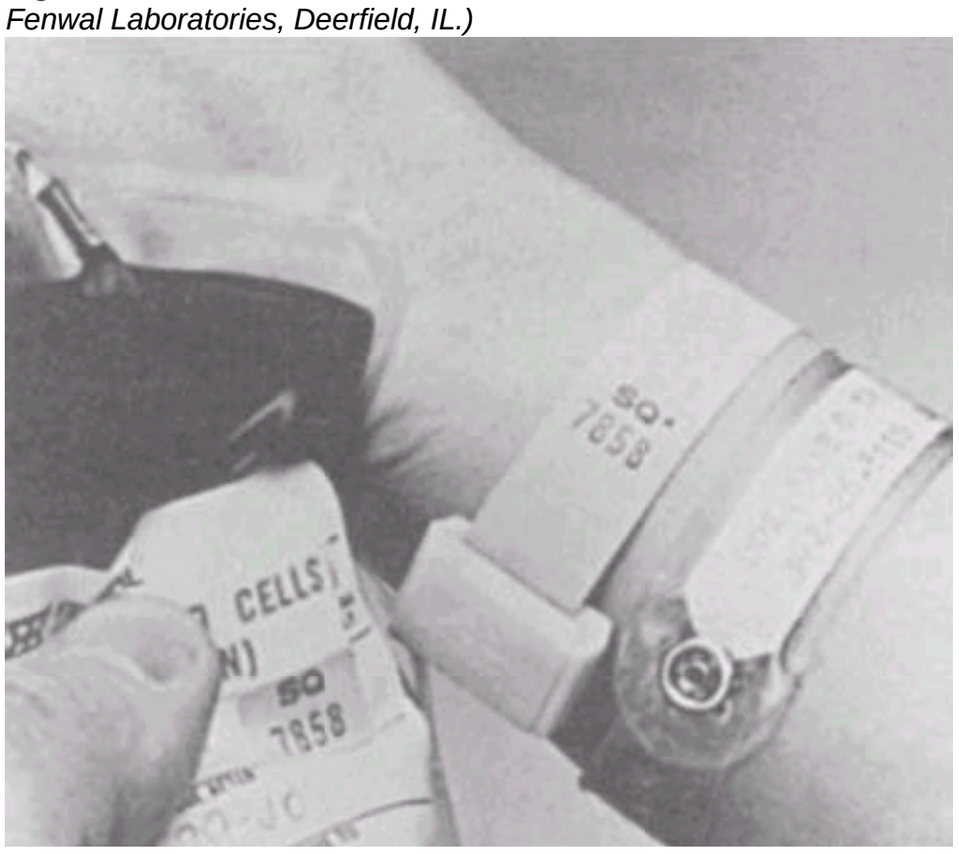


Figure 29-4 An example of a blood administration Y set with two adapters for insertion into a unit of blood or saline; note the in-line filter. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

Figure 29-5 One upper adapter has been inserted into a bag containing normal (0.9%) saline. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

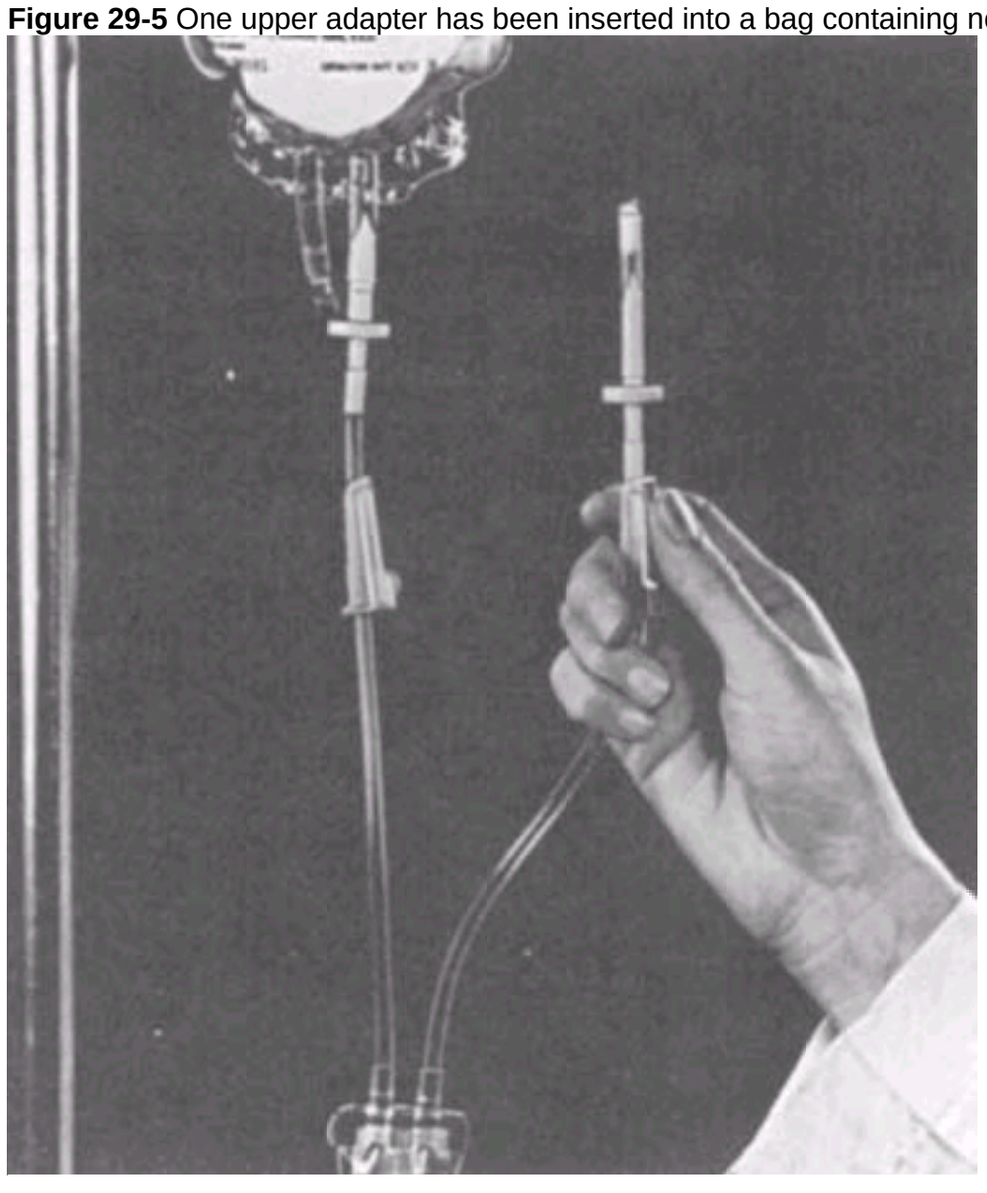


Figure 29-6 The entry site of the unit of blood, into which the other upper adapter of the Y set should be inserted. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

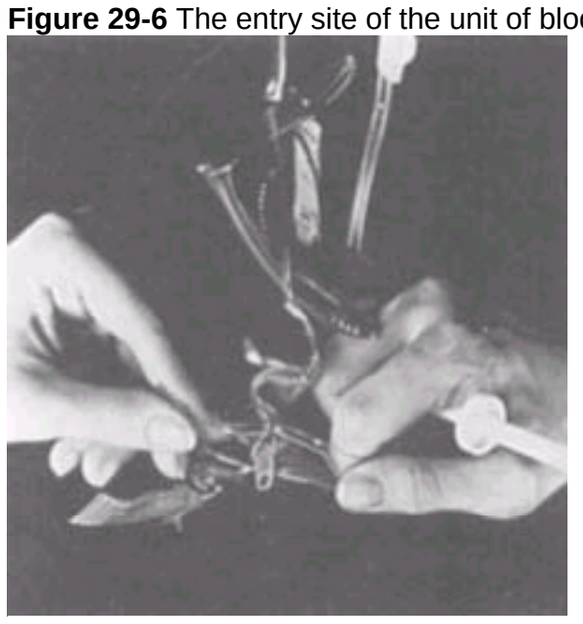


Figure 29-7 Inserting the hard plastic spike of the upper adapter. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

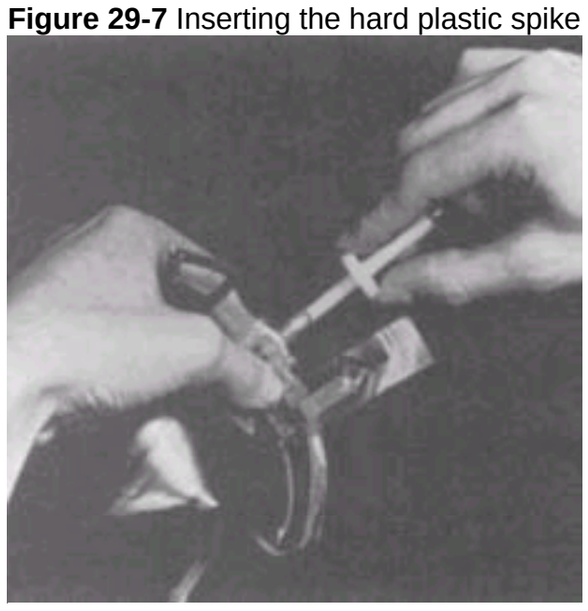


Figure 29-8 After the plastic tubing has been primed with saline, the blood flows through into the patient. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

Figure 29-9 Administration of blood through a microaggregate filter attached between the unit of blood and the hard plastic spike of the administration set. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

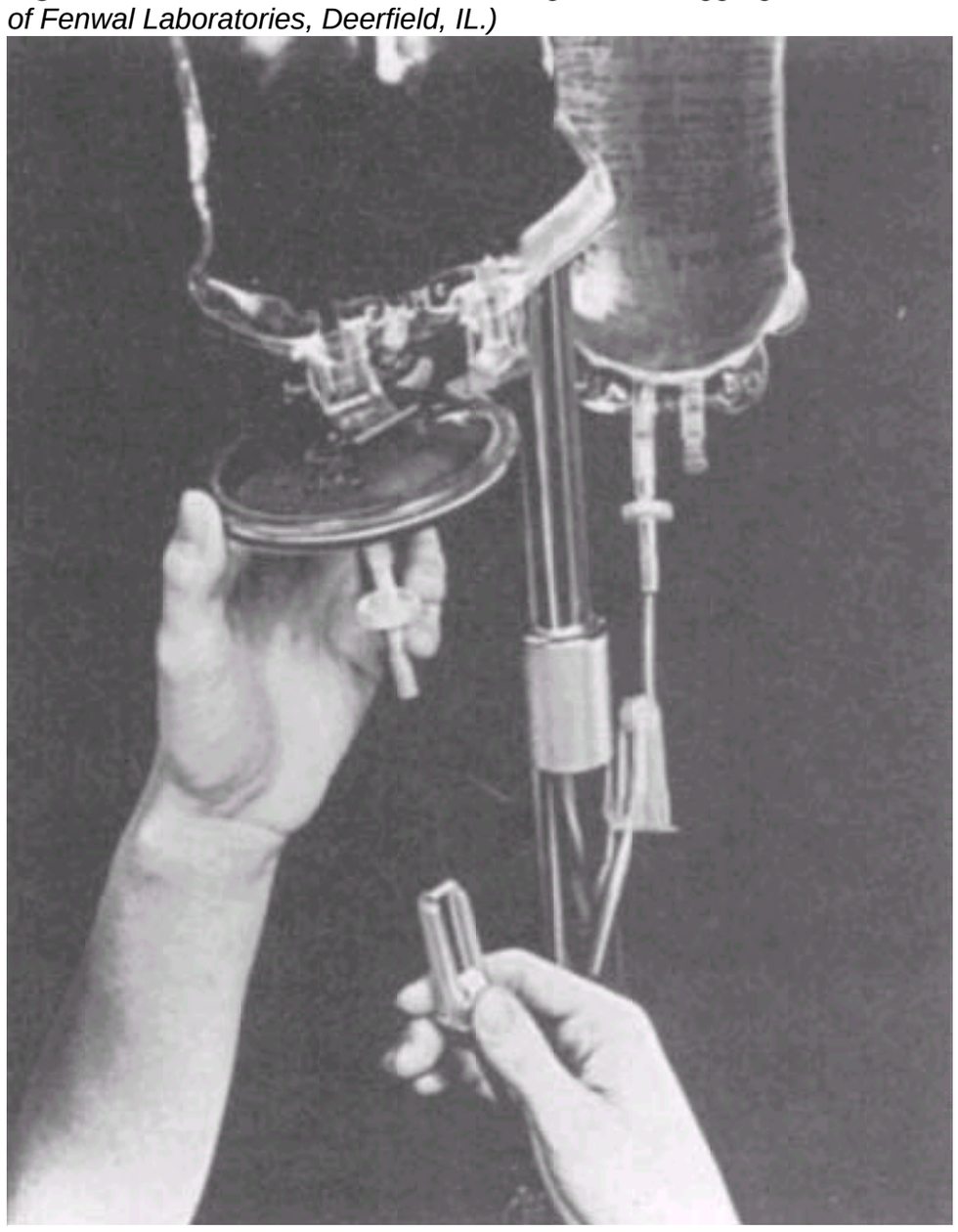


Figure 29-10 A controlled-pressure administration device for rapid infusion of blood products. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

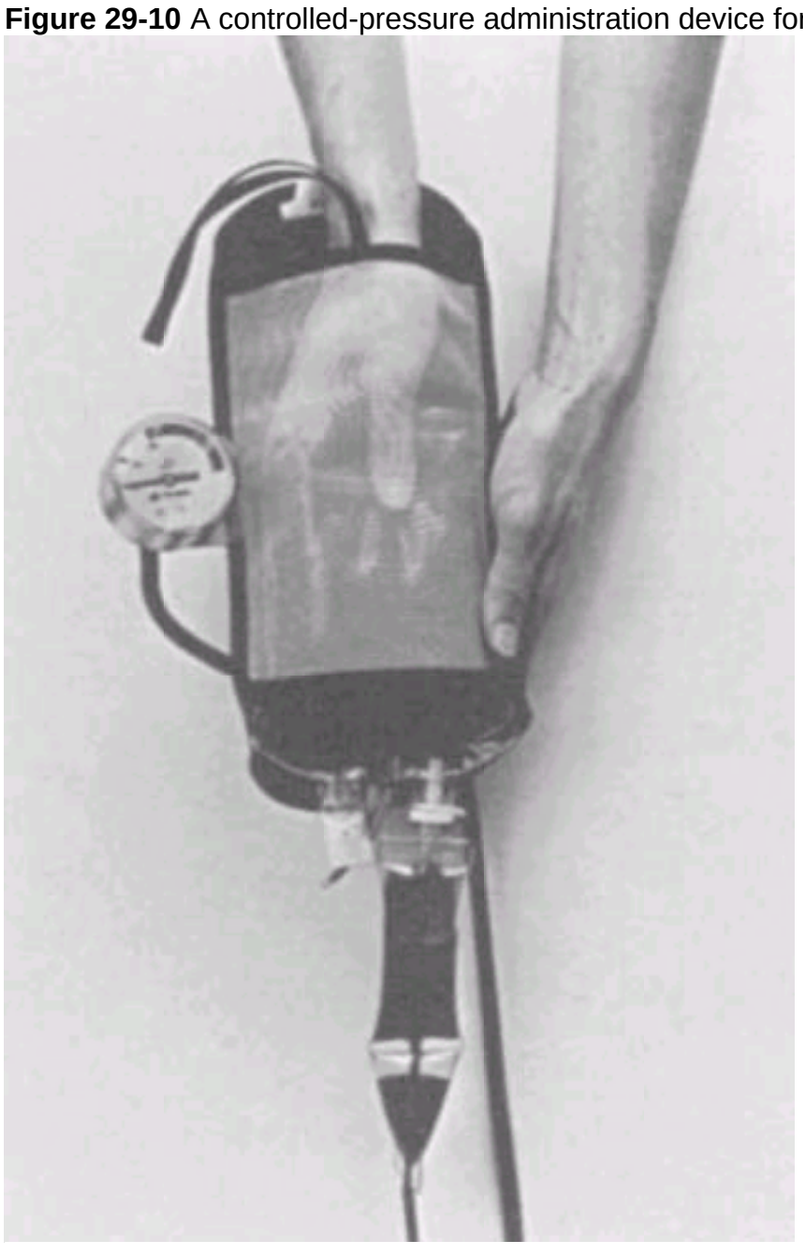


Figure 29-11 A rubber bladder is pumped up, and the blood unit is squeezed uniformly against a reinforced mesh. *(Courtesy of Fenwal Laboratories, Deerfield, IL.)*

Figure 30-1 Schwann cell sheath of unmyelinated *(left)* and myelinated *(right)* nerve fibers. *(From Wildsmith JAW: Peripheral nerve and local anesthetic drugs. Br J Anaesth 58:692, 1986. Reproduced by permission.)*

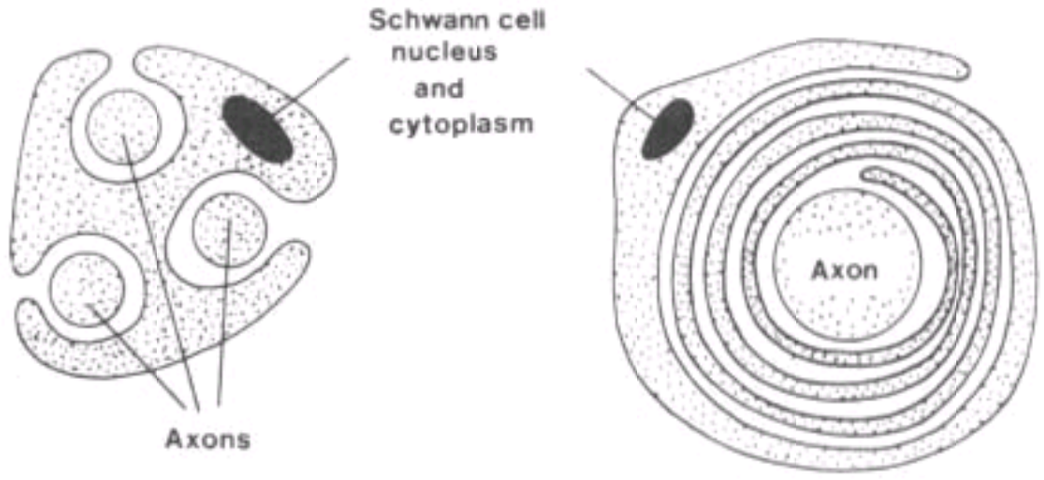


Figure 30-2 Axon membrane. *(From Wildsmith JAW: Peripheral nerve and local anesthetic drugs. Br J Anaesth 58:692, 1986. Reproduced by permission.)*

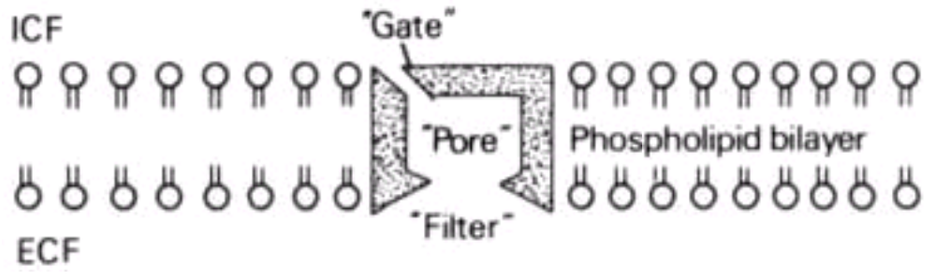


Figure 30-3 Cross section of peripheral nerve. *(From Wildsmith JAW: Peripheral nerve and local anesthetic drugs. Br J Anaesth 58:692, 1986. Reproduced by permission.)*

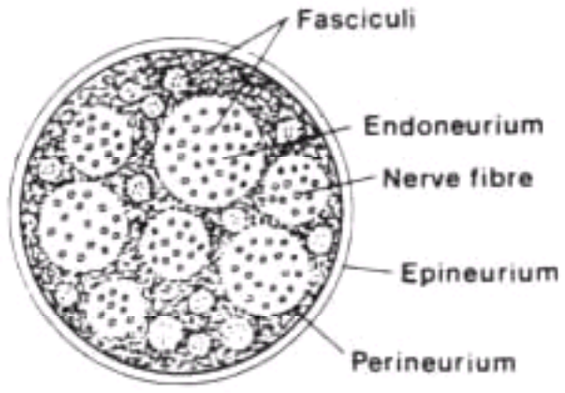
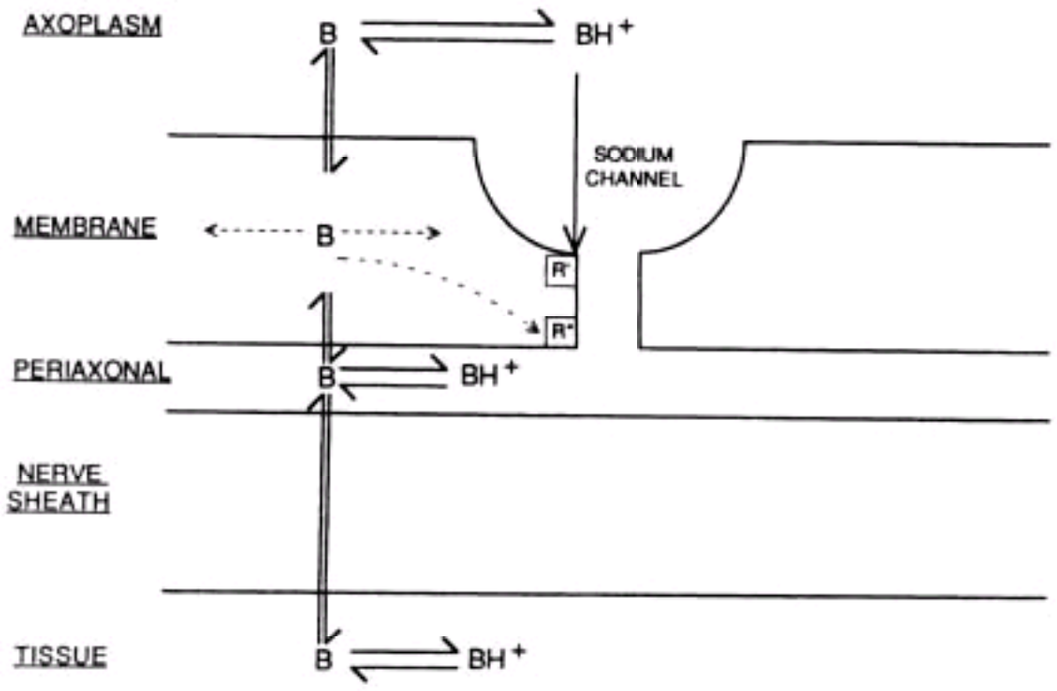


Figure 30-4 Mechanism of action of local anesthetic agents (see text for details). *(Modified from Ritchie JM: Mechanism of action of local anesthetic agents and biotoxins. Br J Anaesth 47:196, 1975. Reproduced by permission.)*



B = Uncharged lipid soluble base BH = Charged cationic form

R' = Known receptor R" = Speculative receptor

⇌ = Equilibrium based on pKa and pH

→ = Proven pathway ⇢ = Speculative pathway

Figure 30-6 *A,* Except in the setting of gross contamination, wounds should be anesthetized by inserting the needle through the cut edges, not through the intact skin. Patients often will not feel a 25-gauge or smaller needle passed into the subcutaneous tissue when it is advanced slowly through the cut edge. However, pain generally occurs with tissue distention by the anesthetic, and hence, injection should be slow and deliberate. *B,* If a wound is grossly contaminated, the anesthetic may be introduced through the intact skin. The operator should limit the number of needle sticks. The needle is first introduced at a point in line with the wound and beyond the wound edge (*1*), and while the anesthetic is slowly injected, the needle is advanced to include 1 entire side of the wound (if possible) to a point well past the opposite end of the wound. The other side may be anesthetized by passing the needle through the area already infiltrated by the first injection (*3*), making the skin puncture painless. A 3.8-cm (1.5-in.) 27-gauge needle is a good choice. If the needle is not long enough to encompass the entire wound, the skin is painlessly punctured at a midway point that has already been anesthetized (*2*).

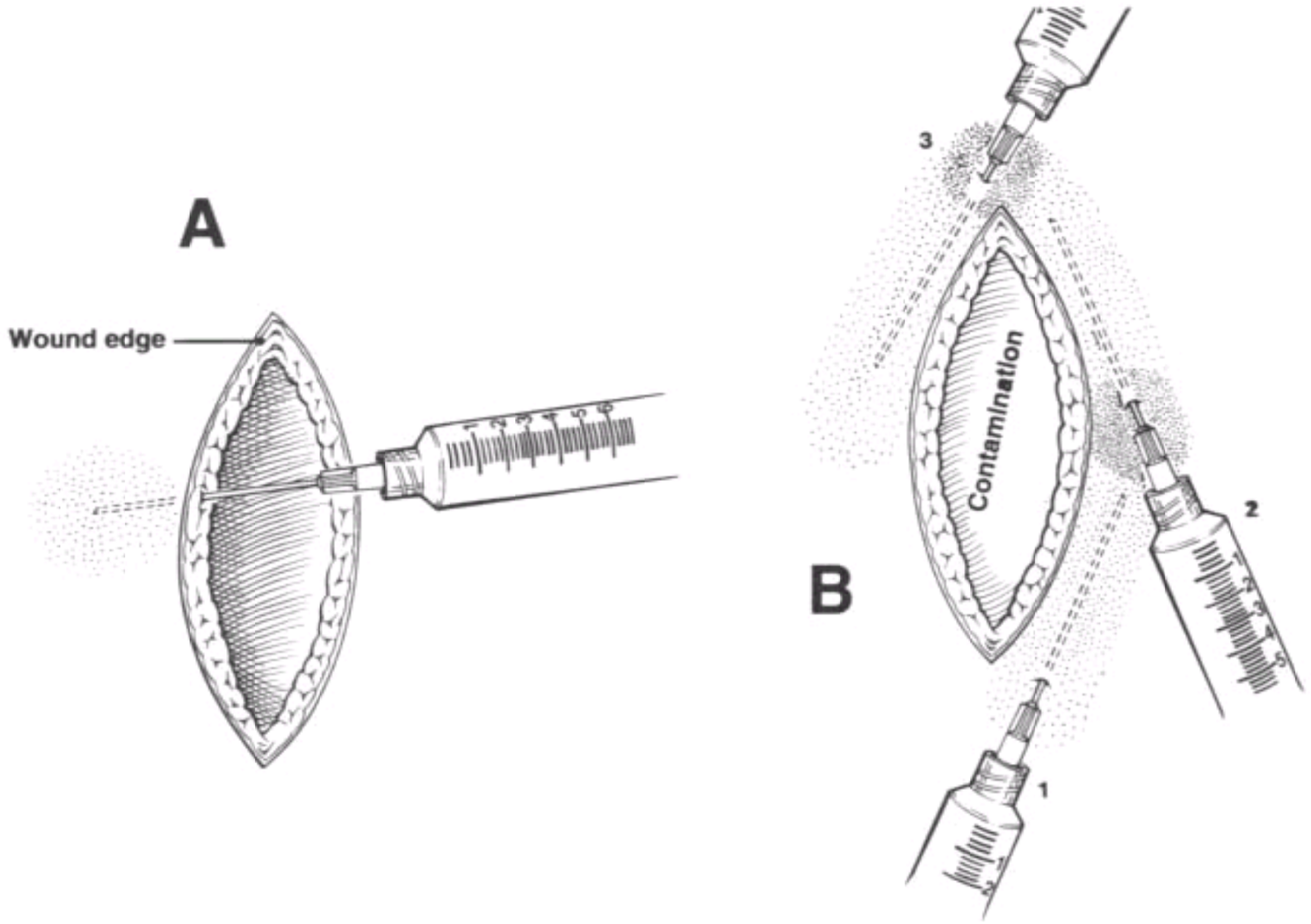


Figure 31-3 *A,* Local anesthesia—basic setup for intraoral application using an aspirating dental syringe. An alternative for a dental syringe is a 3-mL Luer-Lok syringe with a 1 ½ inch 25- or 27-ga needle. *B* and *C,* Intraoral dental blocks may be minimally painless if a topical anesthetic numbs the mucosa. Topical anesthesia is applied prior to injection, illustrated here for an inferior alveolar nerve block. After drying the mucosa with gauze, the pterygomandibular triangle is painted with topical anesthetic. Benzocaine is liberally sprayed into two cotton swabs and held in place by closing the teeth and lips for a few minutes after painting the area of needle penetration. The mucosa is dried with gauze before applying the topical anesthetic.

A

B

C

Figure 31-4 *A,* Proper technique for removal of the disposable hypodermic needle from the dental aspirating syringe. *B,* Incorrect technique involves removal of the carpule adapter, rendering the syringe unable to accept new Carpules.

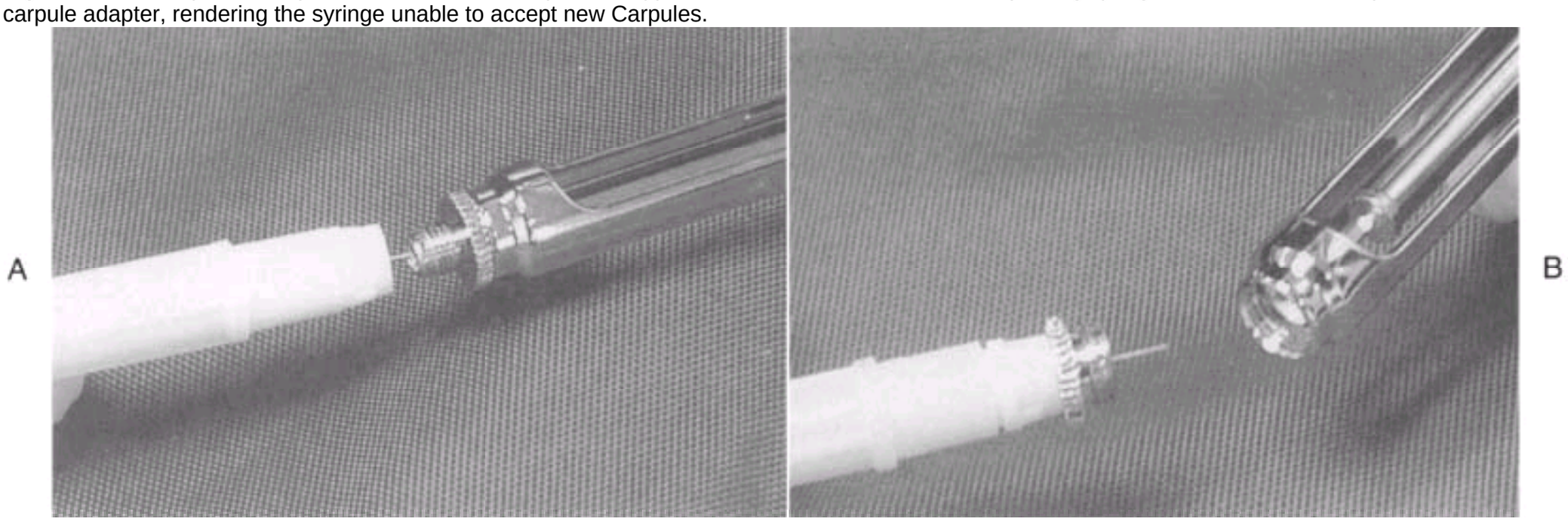
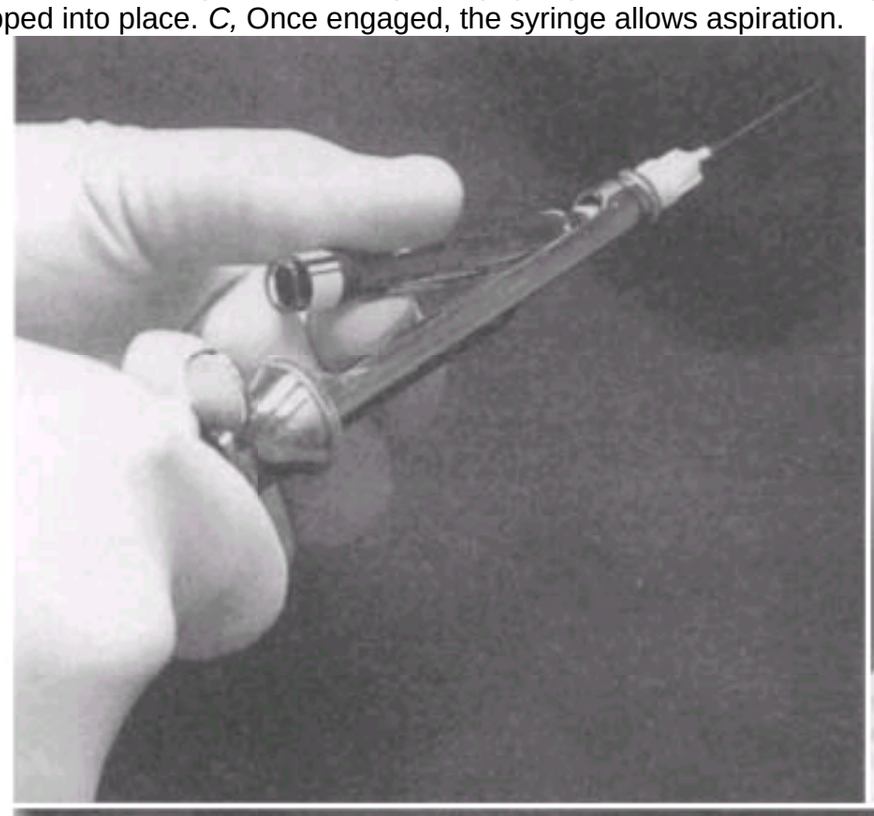
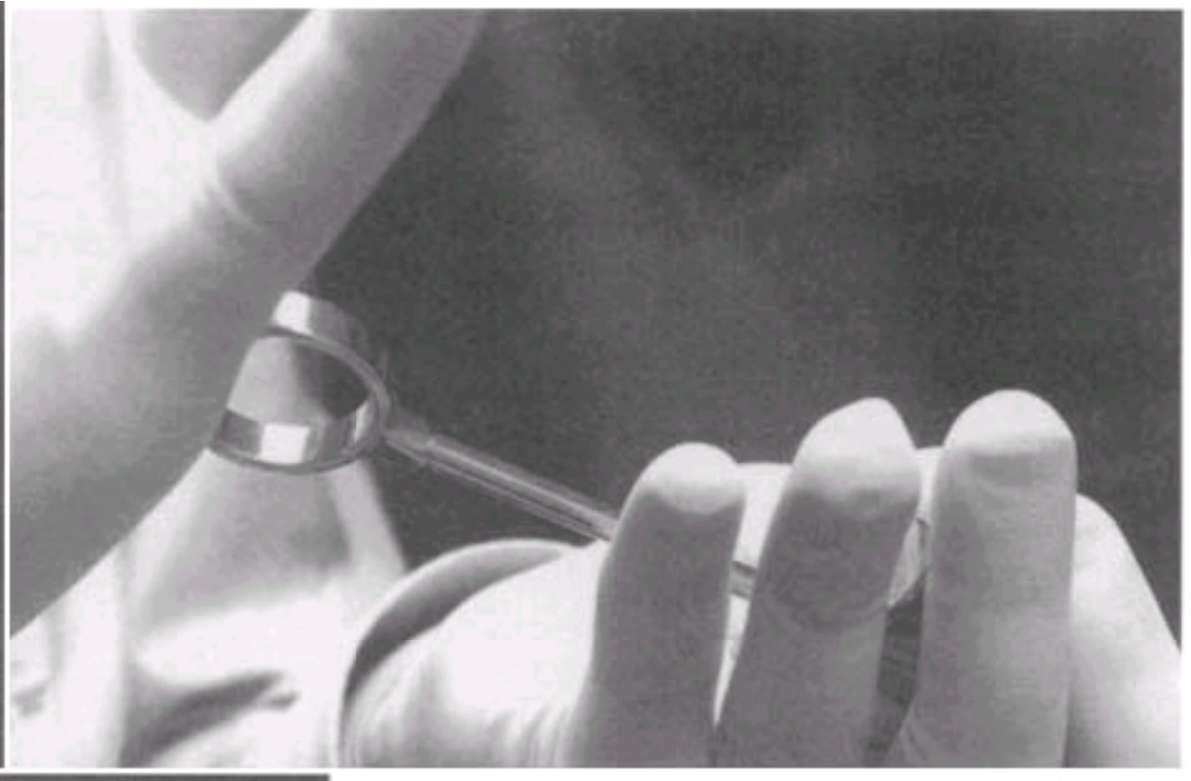


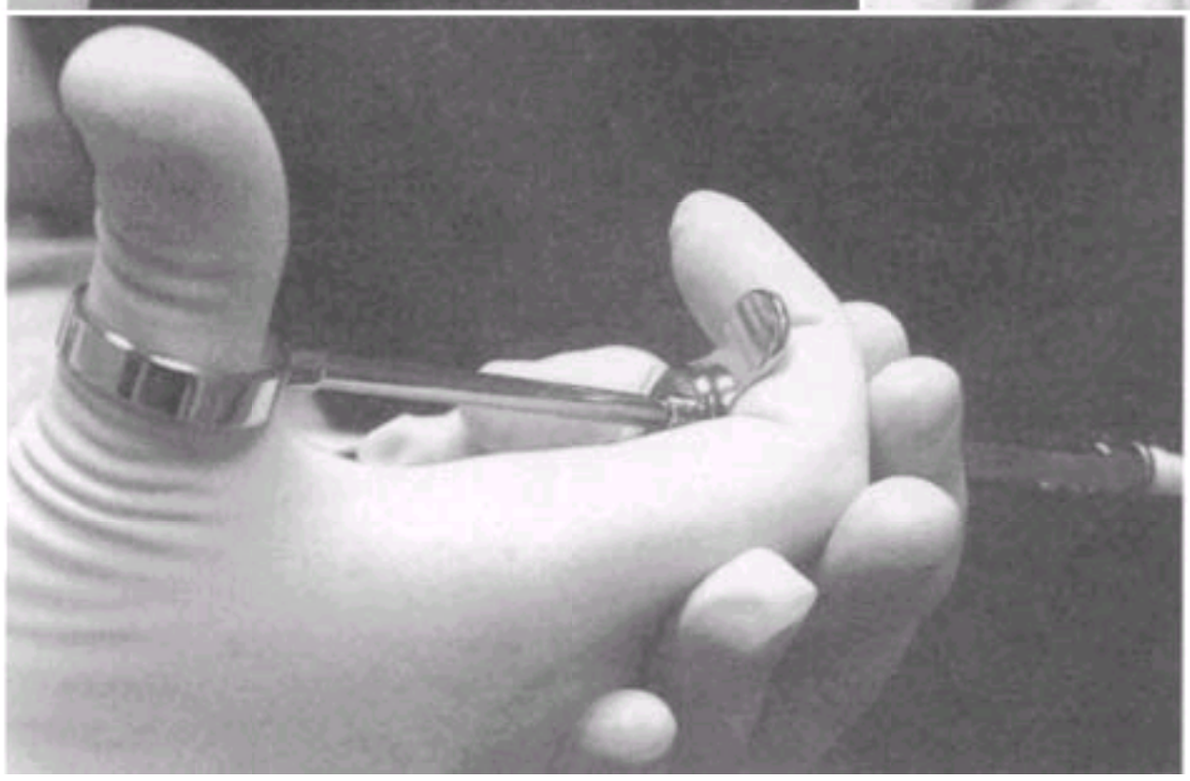
Figure 31-5 Loading the dental aspirating syringe. *A,* The end of the syringe is pulled back. When released, the Carpule cartridge engages the needle. *B,* The barb is snapped into place. *C,* Once engaged, the syringe allows aspiration.



A

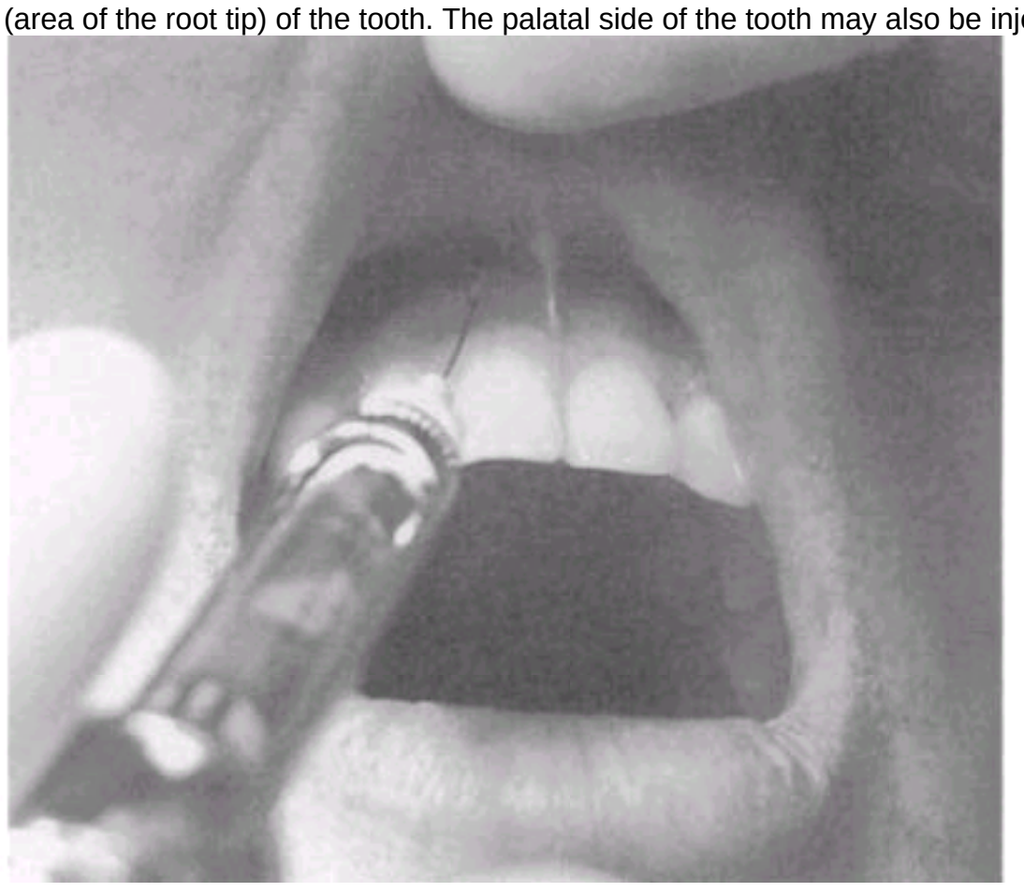


B

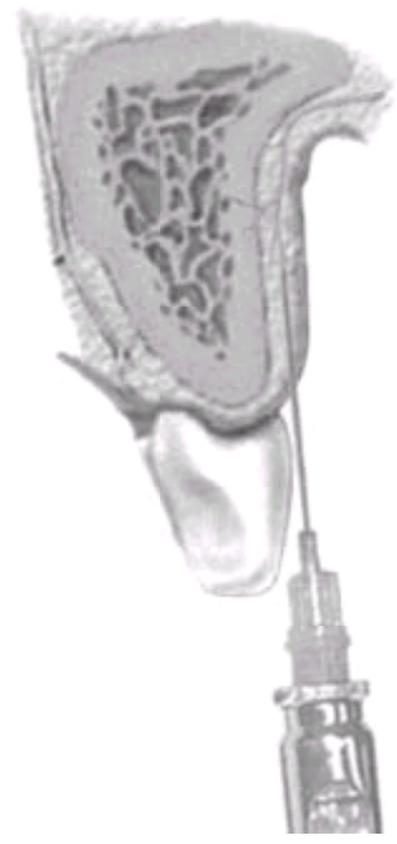


C

Figure 31-6 Topical anesthetic is applied to the dried mucosa before injection. *A,* Supraperiosteal injection technique above the incisors for anesthesia of the upper lip or individual teeth. *B,* Diagrammatic representation of supraperiosteal injection. The aim is to deposit the anesthetic right next to the periosteum at the level of the apex (area of the root tip) of the tooth. The palatal side of the tooth may also be injected.



A



B

Figure 31-7 Posterior superior alveolar nerve block.

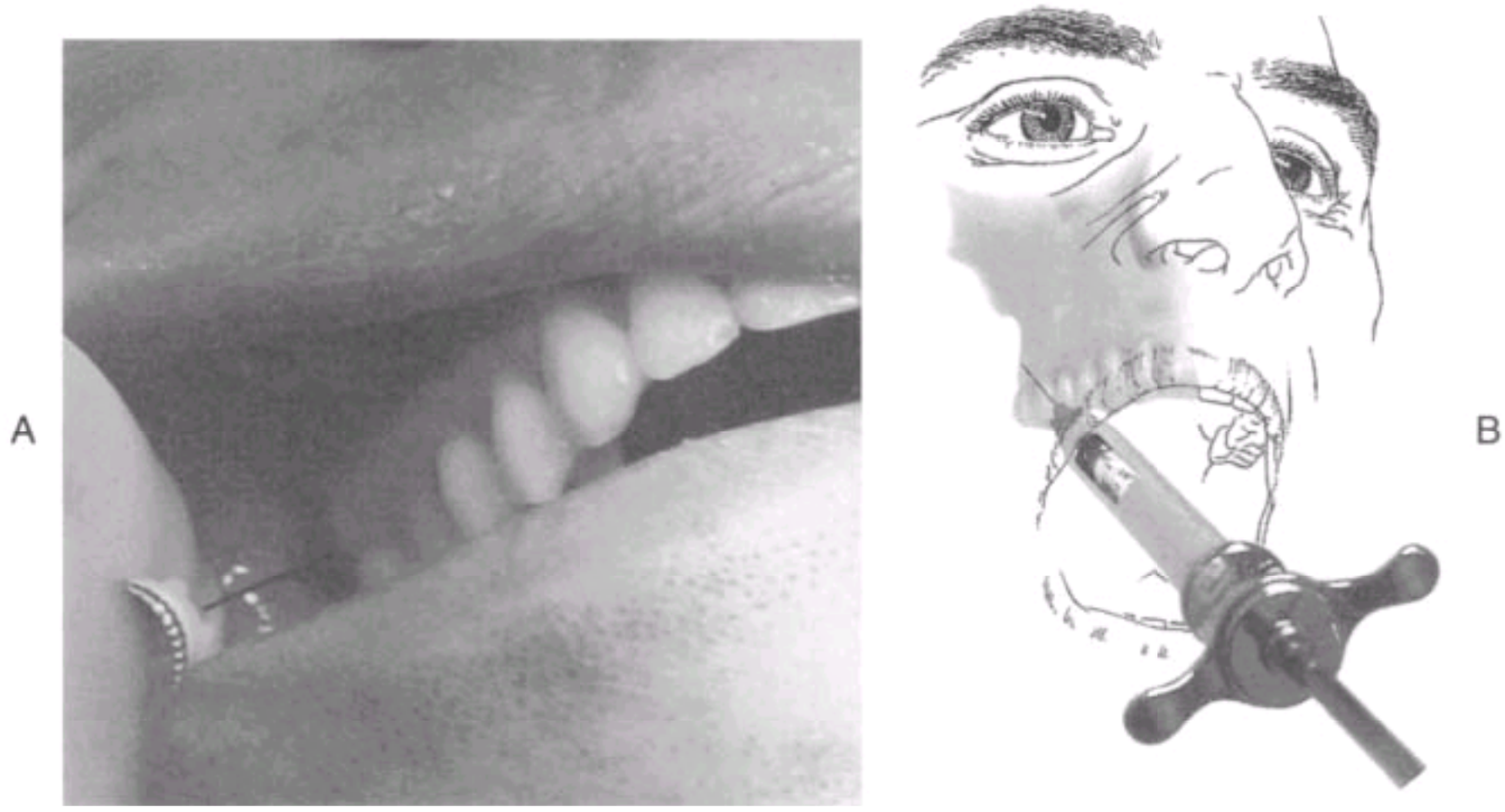


Figure 31-8 Middle superior alveolar nerve block.

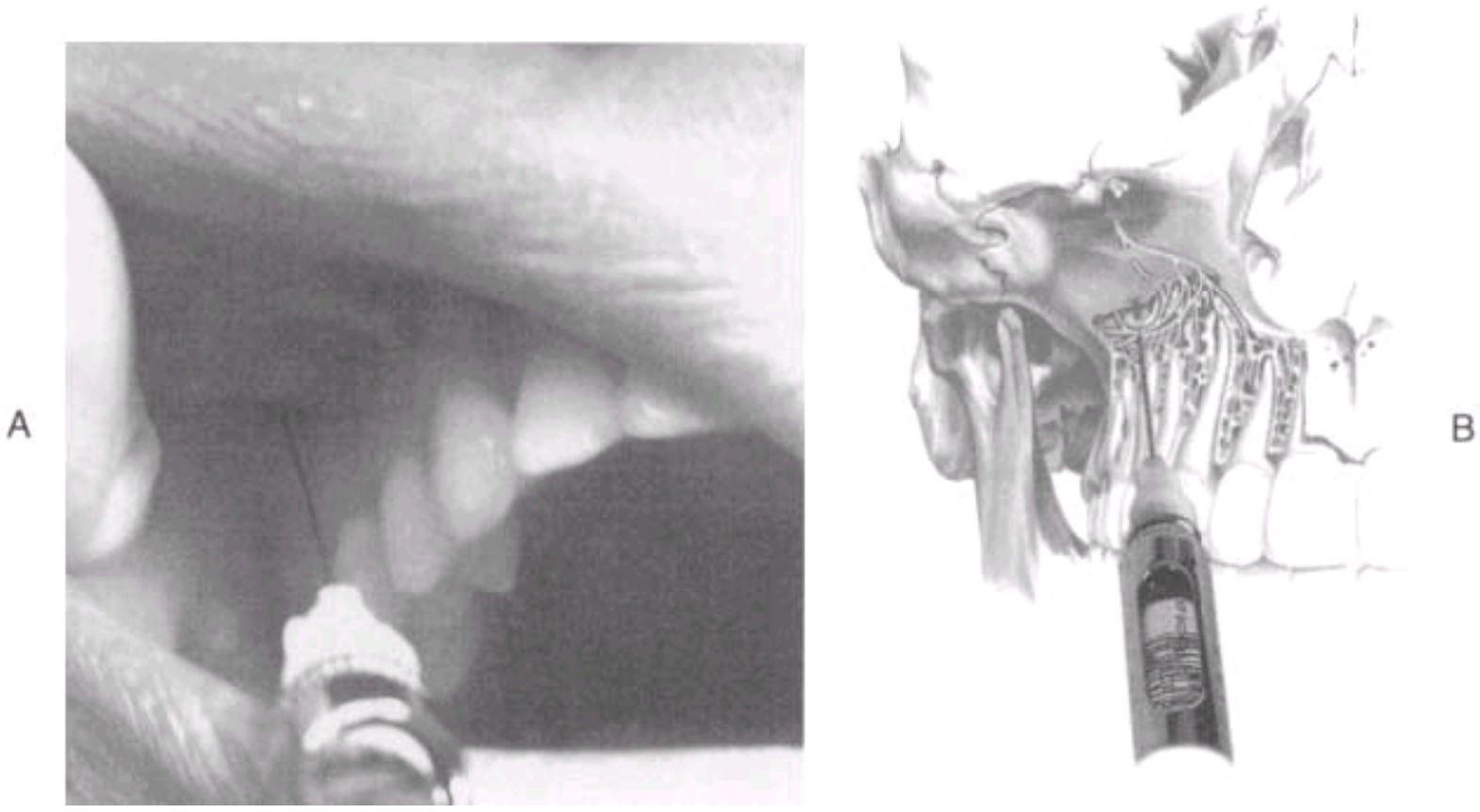


Figure 31-9 Anterior superior alveolar nerve block.

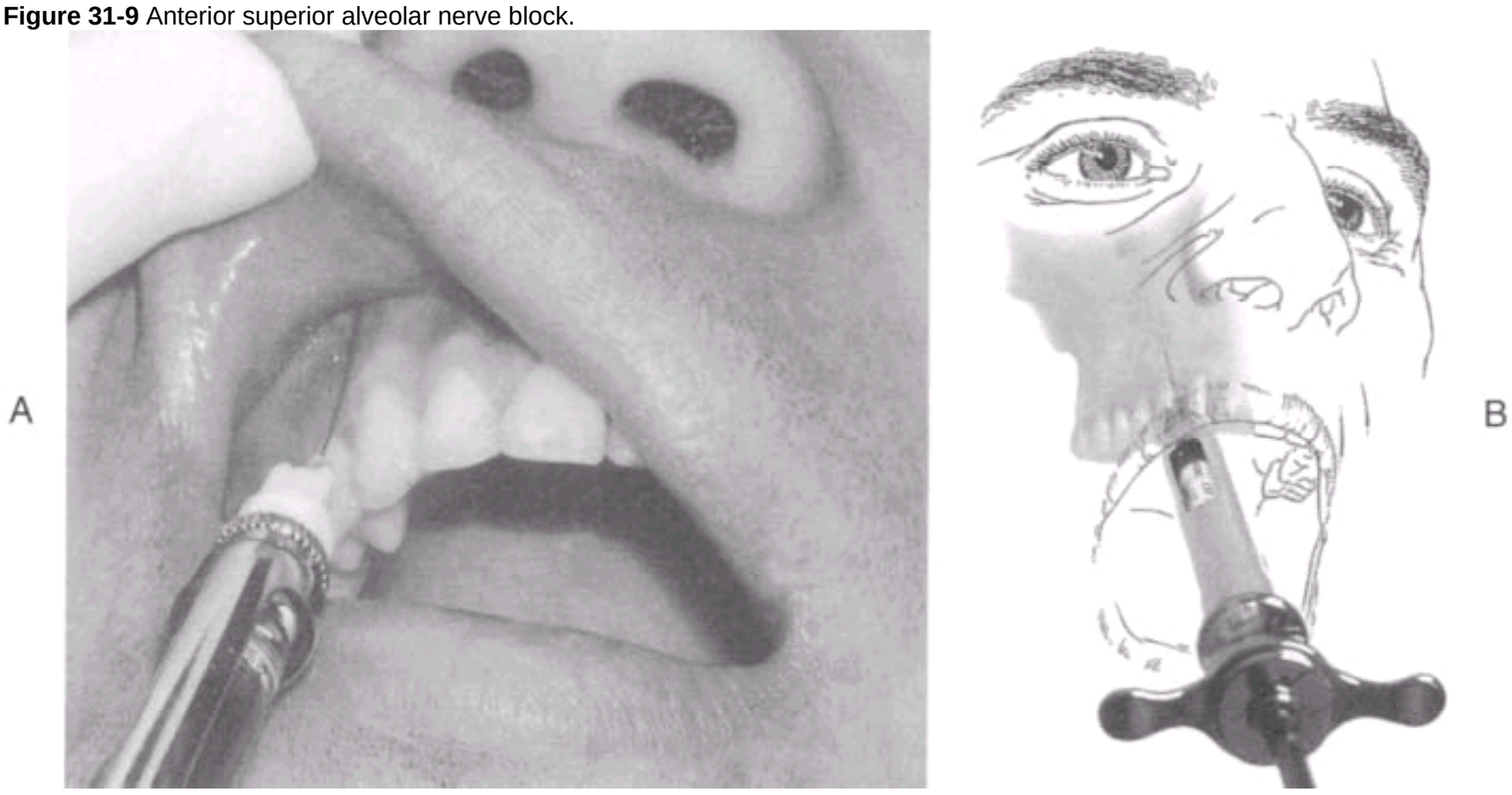


Figure 31-10 Area of anesthesia of a unilateral infraorbital nerve block. Anesthesia includes the lower eyelid and the upper lip.

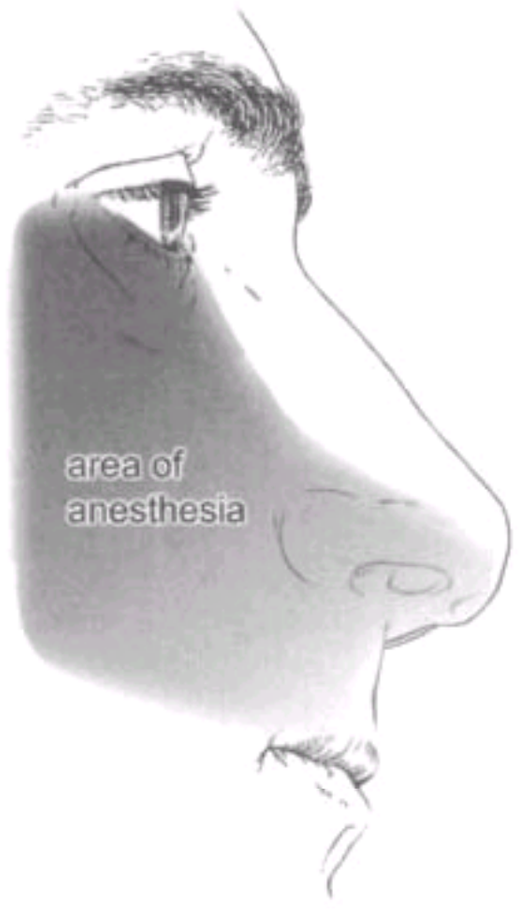


Figure 31-11 *A,* Intraoral approach for infraorbital nerve block. *B,* The position of the infraorbital foramen *(arrow)* on the inferior portion of the infraorbital ridge. During infiltration, the needle should be near but not within the foramen. *C,* Incorrect infraorbital injection technique may result in needle entry into the orbit. *(Adapted from Eriksson E: Illustrated Handbook in Local Anesthesia. Philadelphia, WB Saunders, 1980.)*

A

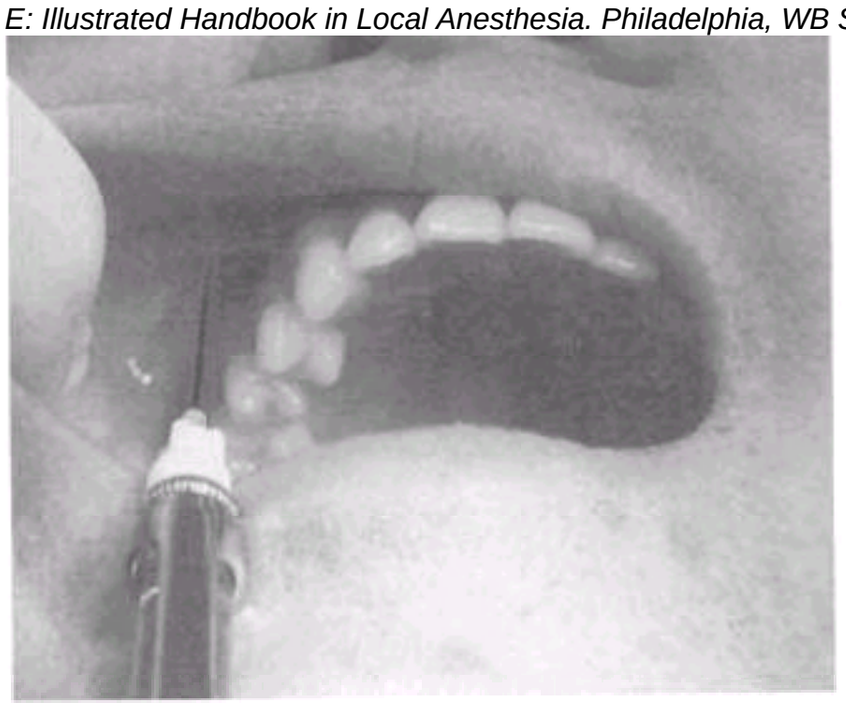
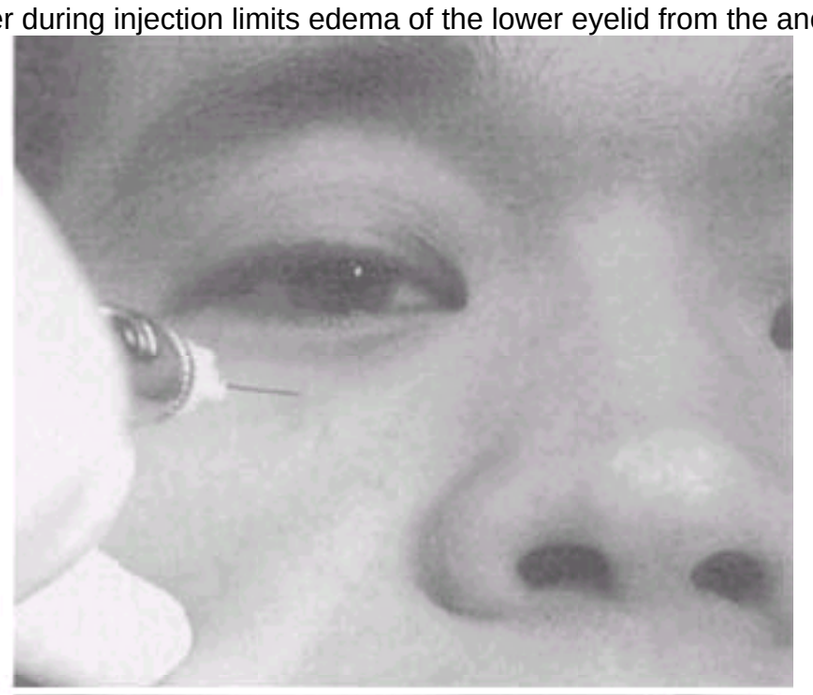


Figure 31-12 *A,* The extraoral approach to the infraorbital nerve. This procedure is more difficult than the intraoral approach, especially when attempting to obtain anesthesia of the upper lip. *B,* During injection, a finger placed above the needle and below the eyelid will limit swelling of the lower lid. Continual pressure with a finger during injection limits edema of the lower eyelid from the anesthetic solution.

A



B

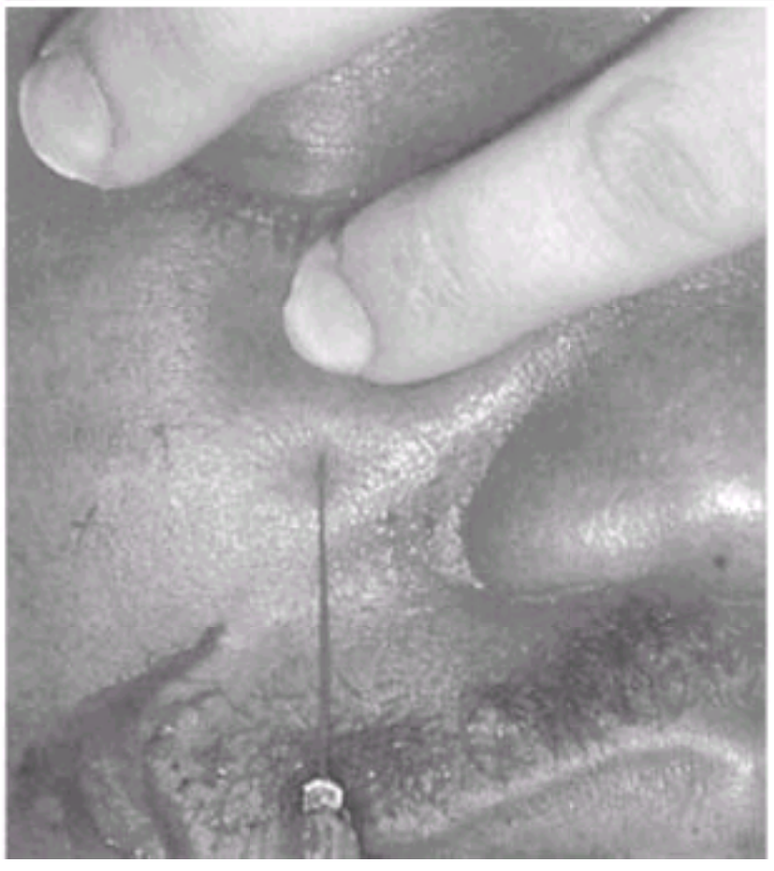
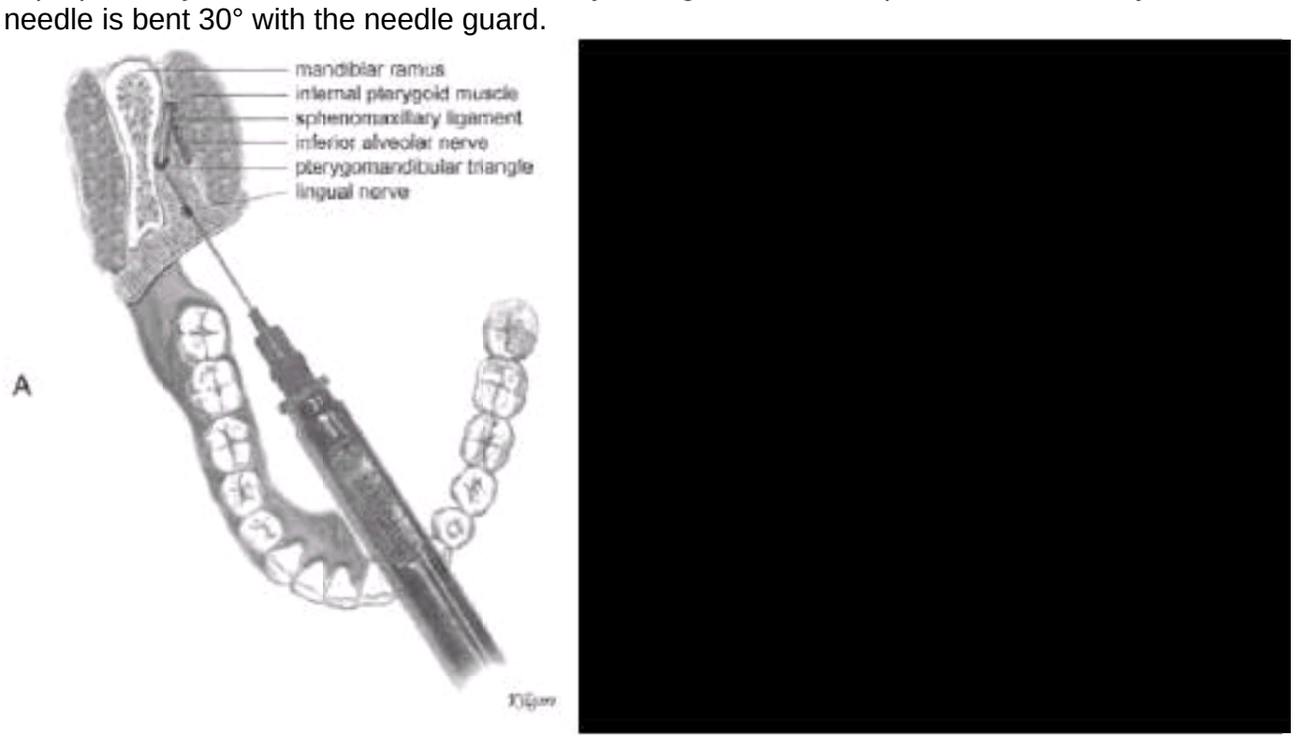
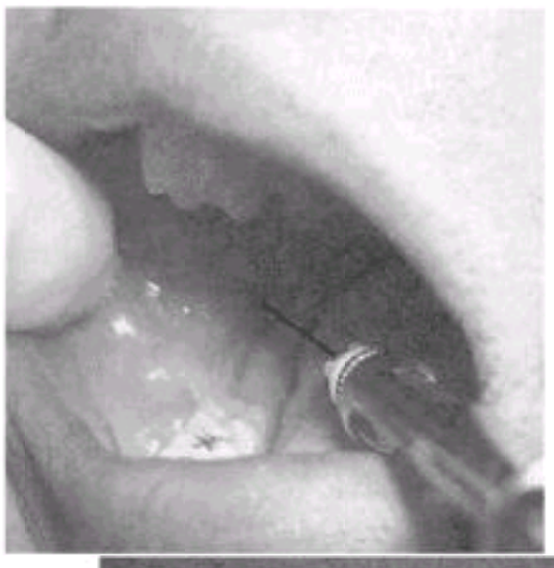
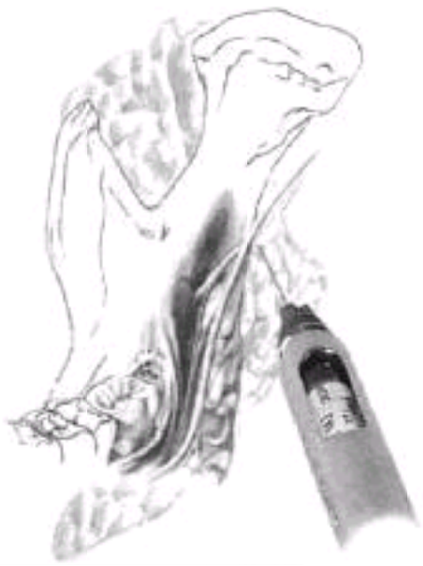


Figure 31-13 Inferior alveolar nerve block. *A,* Transverse illustration of the anatomy of the pterygomandibular triangle and direction of needle *before injection*. *B,* Anatomy of the inferior alveolar nerve block (intraoral approach) *(Adapted from Eriksson E: Illustrated Handbook in Local Anesthesia. Philadelphia, WB Saunders, 1980. Reproduced with permission)*. The greatest depth of the anterior border of the ramus of the mandible, the coronoid notch, is identified with the left index finger or left thumb. *C,* The ramus is grasped between an intraorally placed thumb (positioned on the coronoid notch) and extraorally positioned index finger. The pterygomandibular triangle may then be well visualized. Note the angle of the syringe during injection, with the syringe overlying the first and second premolars on the opposite side of the mandible. Failing to appreciate this orientation is one common cause of failure. The operator should be able to feel the needle contacting the bony surface of the inner mandible. Also note that the injection is 1 cm *above* the level of the teeth. *D,* Directing the needle too far posteriorly during the inferior alveolar nerve block technique will result in entry into the area of the parotid gland. Anesthesia of the seventh nerve may result. This occurs because from an improper entry orientation and is corrected by noting *C*. *E,* To compensate for difficulty in obtaining the correct approach with a straight needle, a 25-ga 1 ½-inch needle is bent 30° with the needle guard.




C


D

E

Figure 31-14 Mental infiltration technique. *A,* The correct supraperiosteal approach is an *infiltration* technique. *B,* Actual introduction of the needle into the mental foramen may produce neurovascular damage, and therefore infiltration only is recommended. *(Adapted from Eriksson E: Illustrated Handbook in Local Anesthesia. Philadelphia, WB Saunders, 1980.)*

A

Figure 31-15 The sources of sensory nerve supply to the scalp.

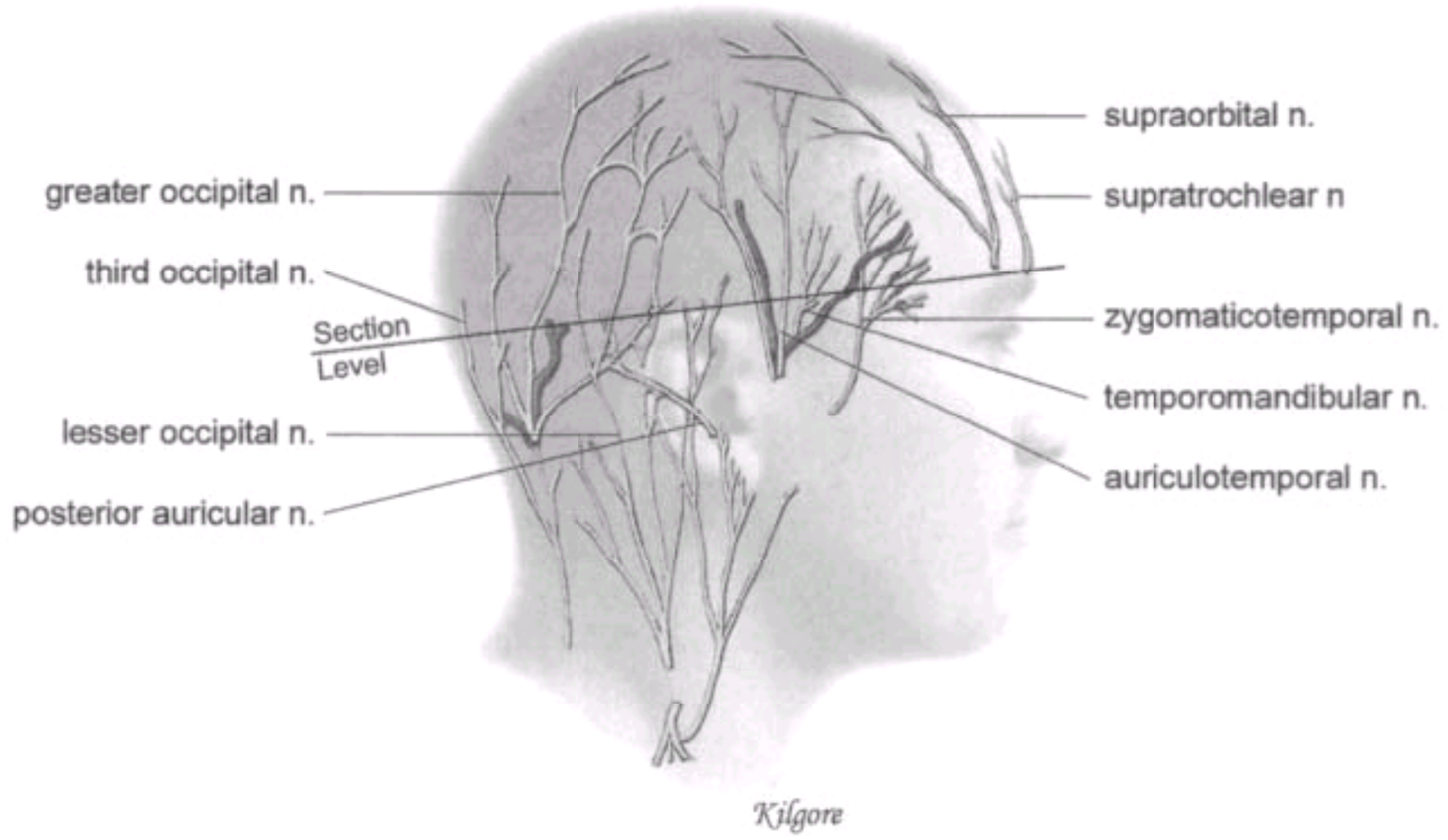


Figure 31-17 Anatomic relationship of the greater and lesser occipital nerves and the adjacent structures at the posterior aspect of the head. Note the proximity of the occipital artery, which is immediately lateral to the greater occipital nerve.

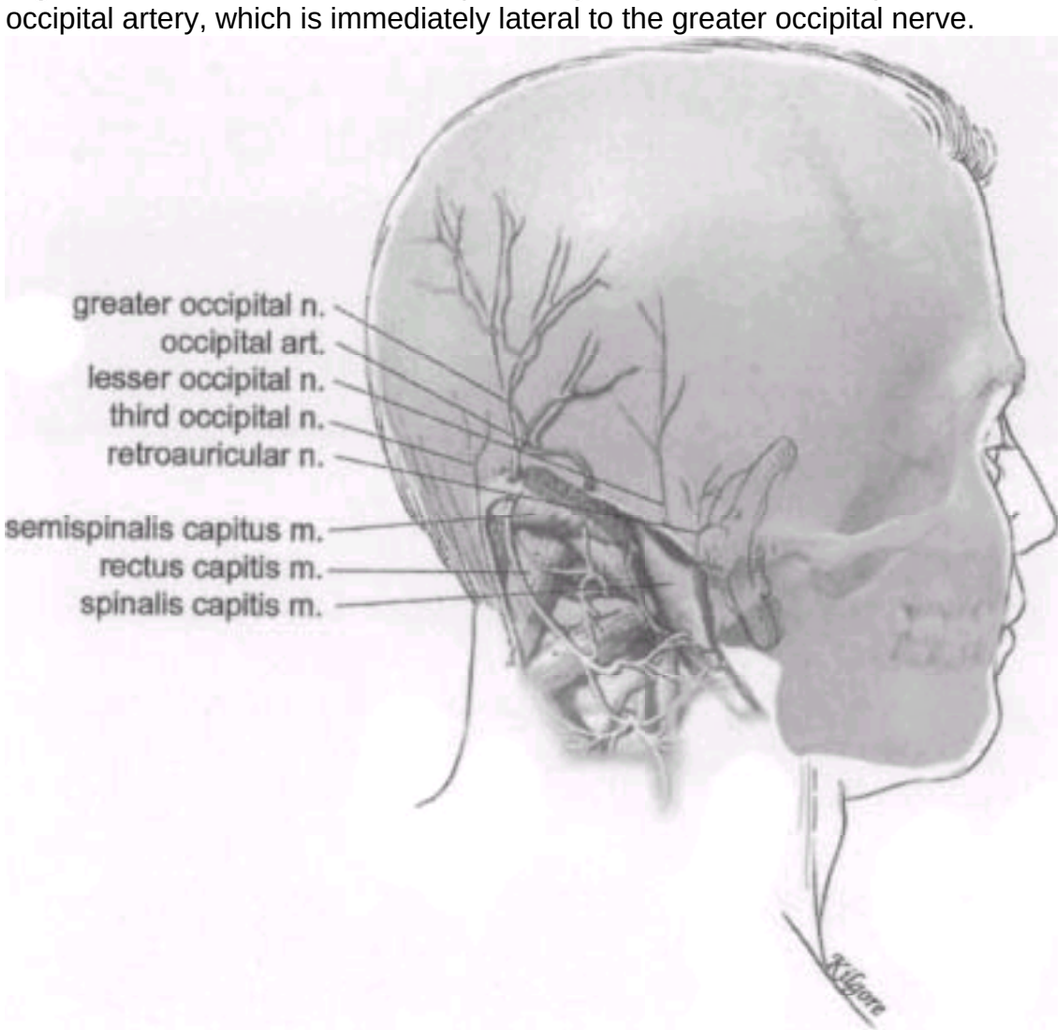


Figure 31-18 *A,* The greater occipital nerve is blocked on a line 3 cm lateral to the external occipital protuberance and the base of the occipital bone. *B,* The lesser occipital nerve is blocked by injection of 2 to 3 mL of anesthetic solution along the posterior border of the mastoid process of the temporal bone.

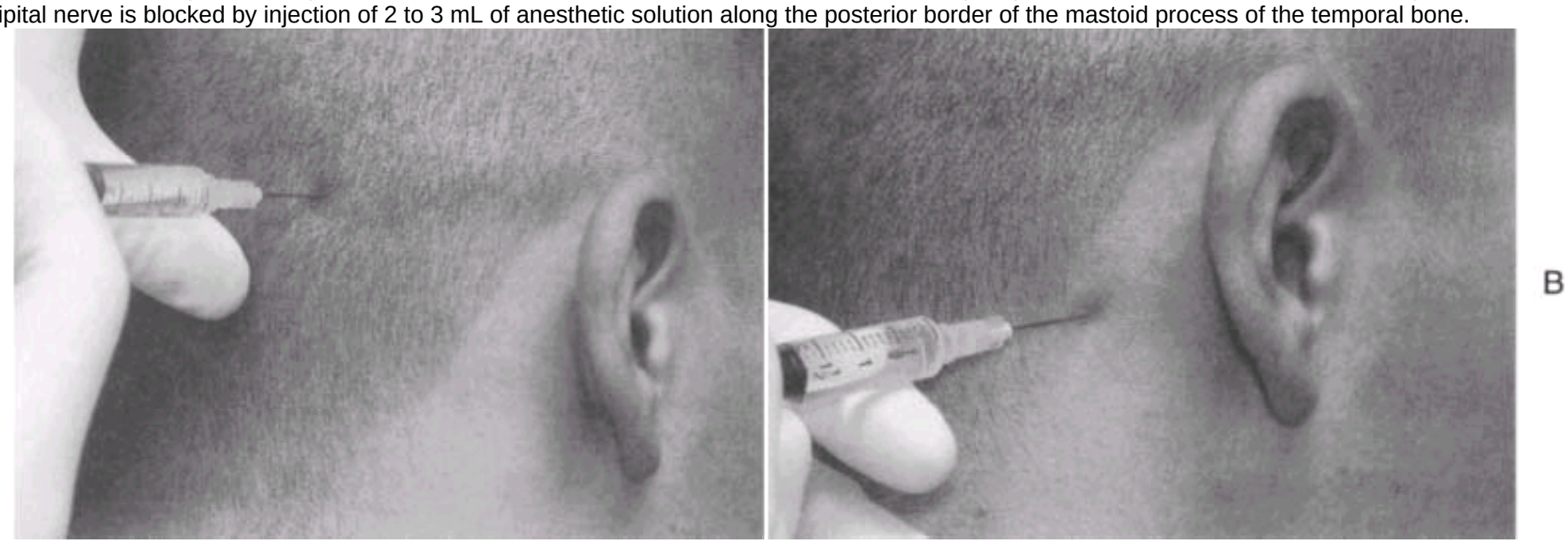


Figure 31-19 This patient had multiple small pieces of glass embedded in the forehead from a windshield injury. Removal was accomplished painlessly with bilateral supraorbital and supratrochlear nerve blocks.

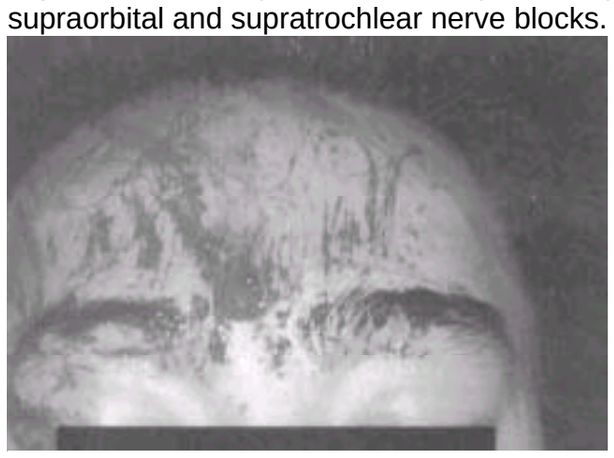


Figure 31-21 A field block of the forehead will anesthetize both the lateral and medial branches of the supraorbital nerve as well as the supratrochlear nerves. The resultant area of anesthesia is represented by the shaded area of the left forehead.

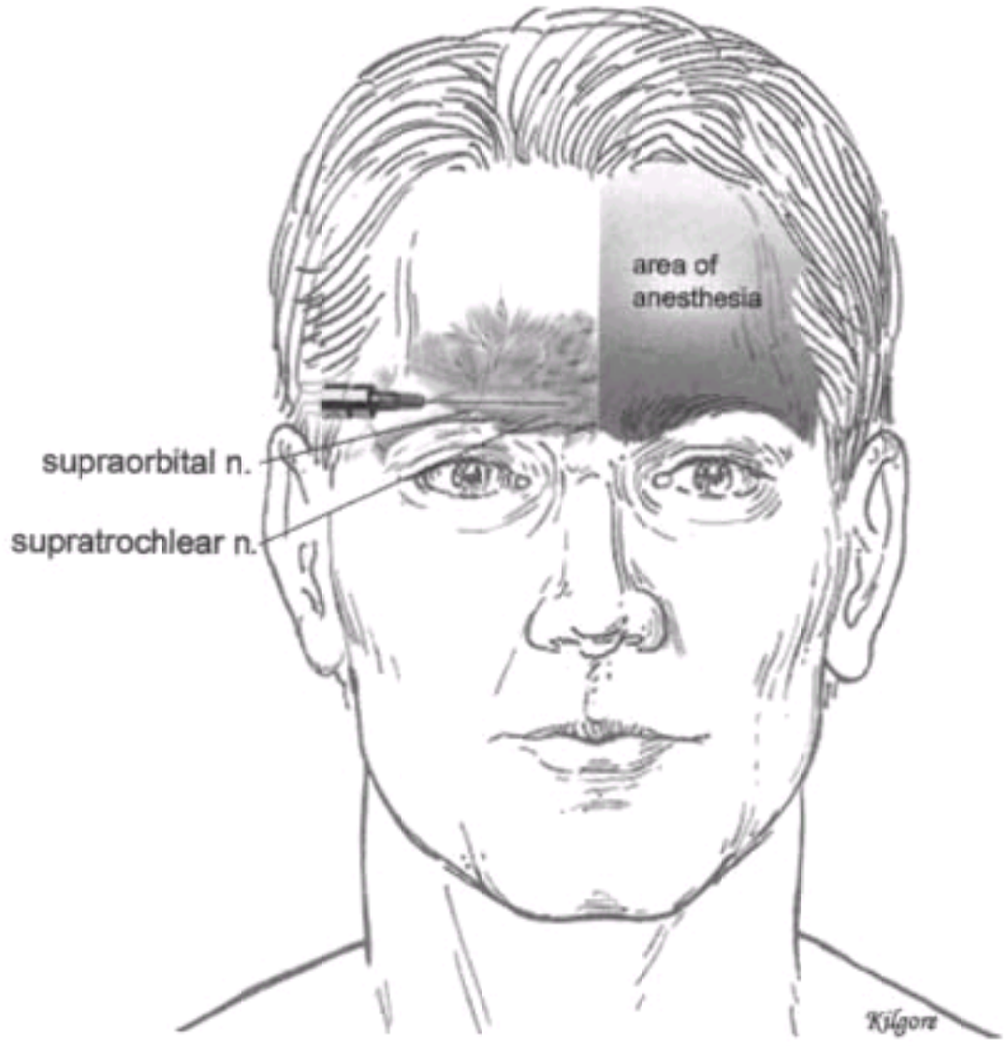


Figure 32-1 Area of anesthesia and cutaneous distribution of the intercostal nerve. *(From Moore DC: Regional Block: A Handbook for Use in the Clinical Practice of Medicine and Surgery, 4th ed. Springfield, IL, Charles C Thomas, 1971, p 159. Reproduced with permission.)*

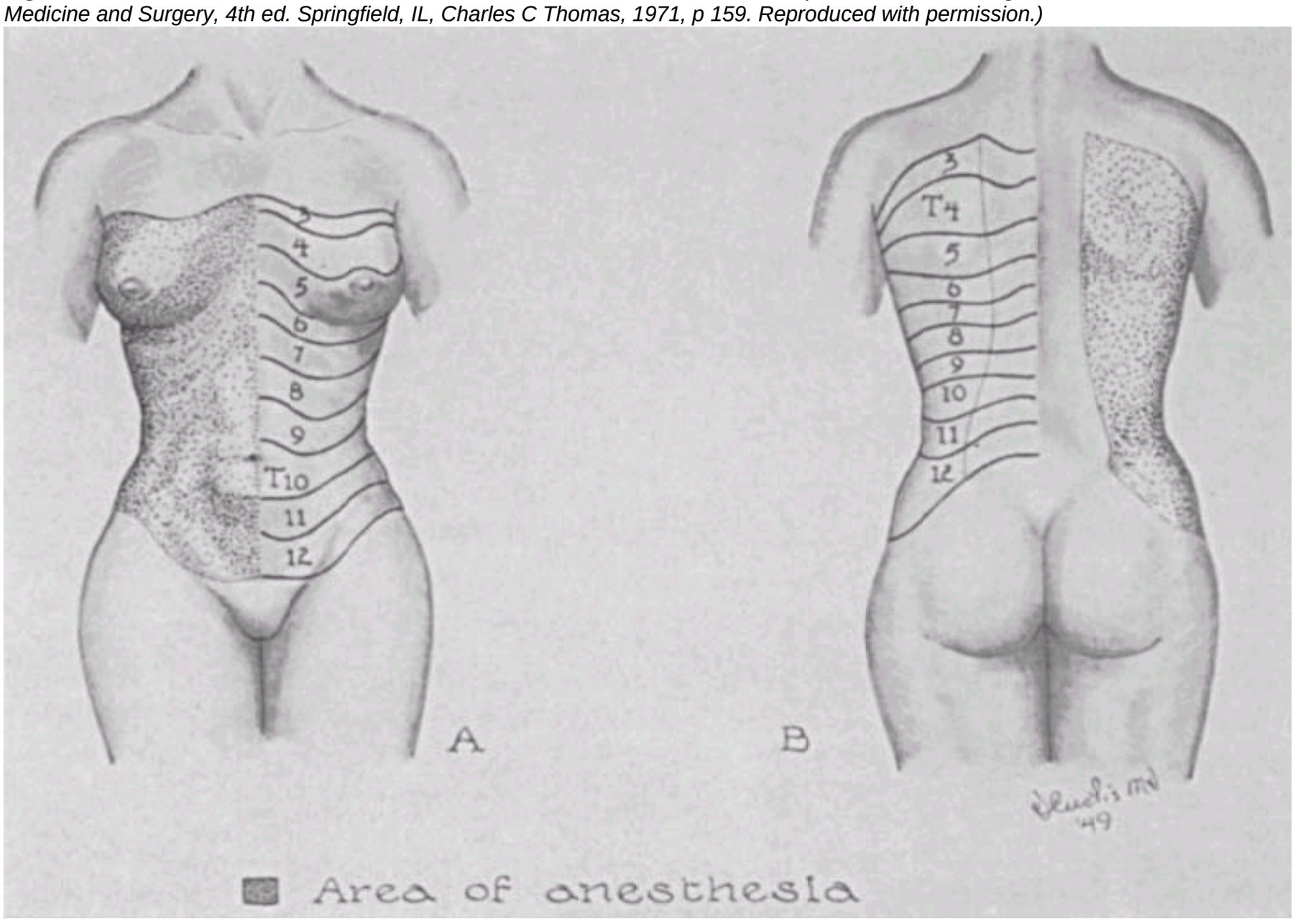


Figure 32-2 Cross section of the chest wall, demonstrating branches of the intercostal nerve, as well as sections of the chest wall at points between which the intercostal block is usually performed: at the posterior angle of the ribs (*A*) and at the midaxillary line (*B*). A block situated at the posterior axillary line is common (see text). *(From Moore DC: Regional Block: A Handbook for Use in the Clinical Practice of Medicine and Surgery, 4th ed. Springfield, IL, Charles C Thomas, 1971, p 146. Reproduced with permission.)*

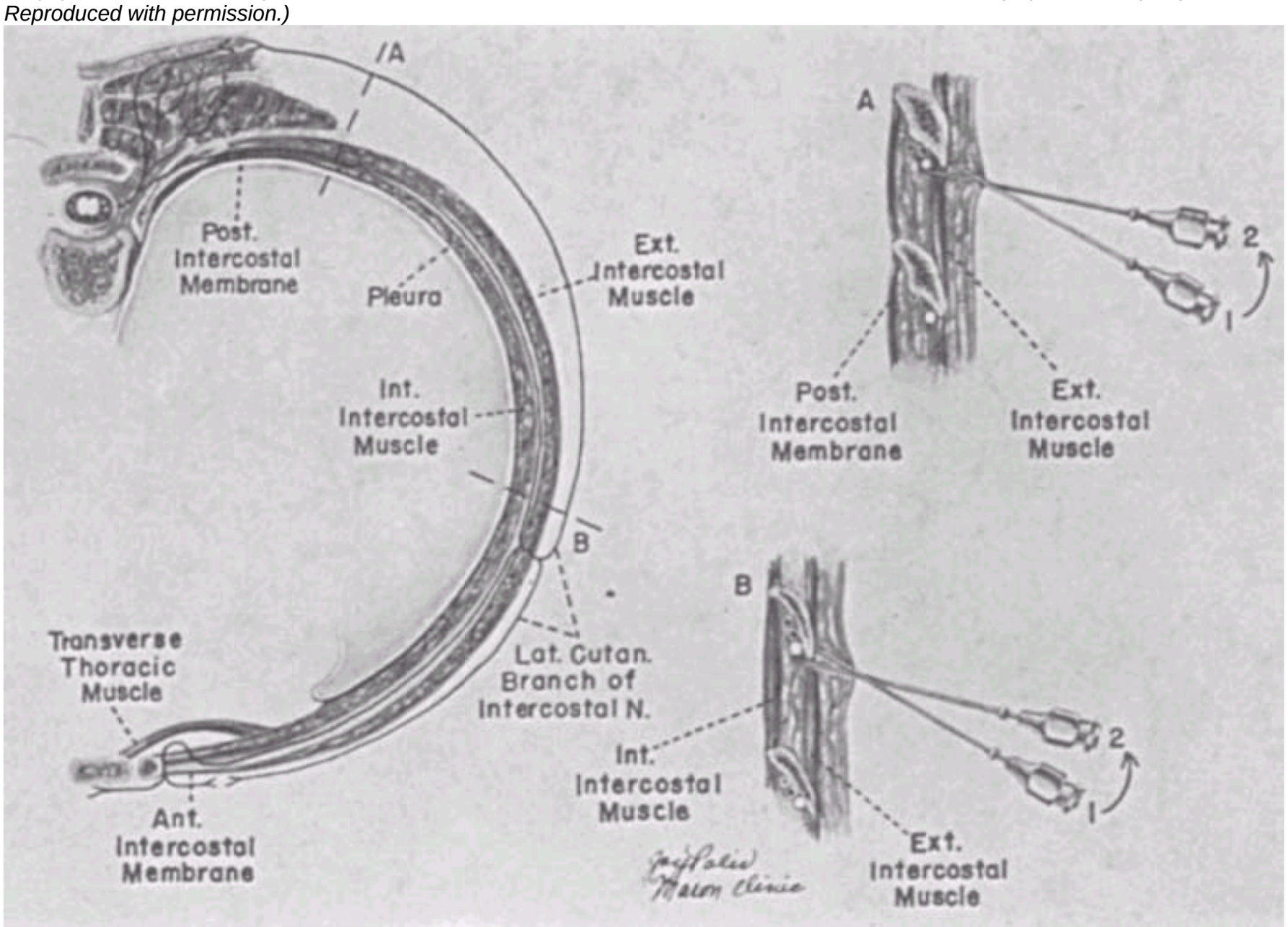


Figure 32-3 Method of retracting skin and the proper needle insertion site for intercostal block. See text for details. *(From Moore DC: Regional Block: A Handbook for Use in the Clinical Practice of Medicine and Surgery, 4th ed. Springfield, IL, Charles C Thomas, 1971, p 153. Reproduced with permission.)*

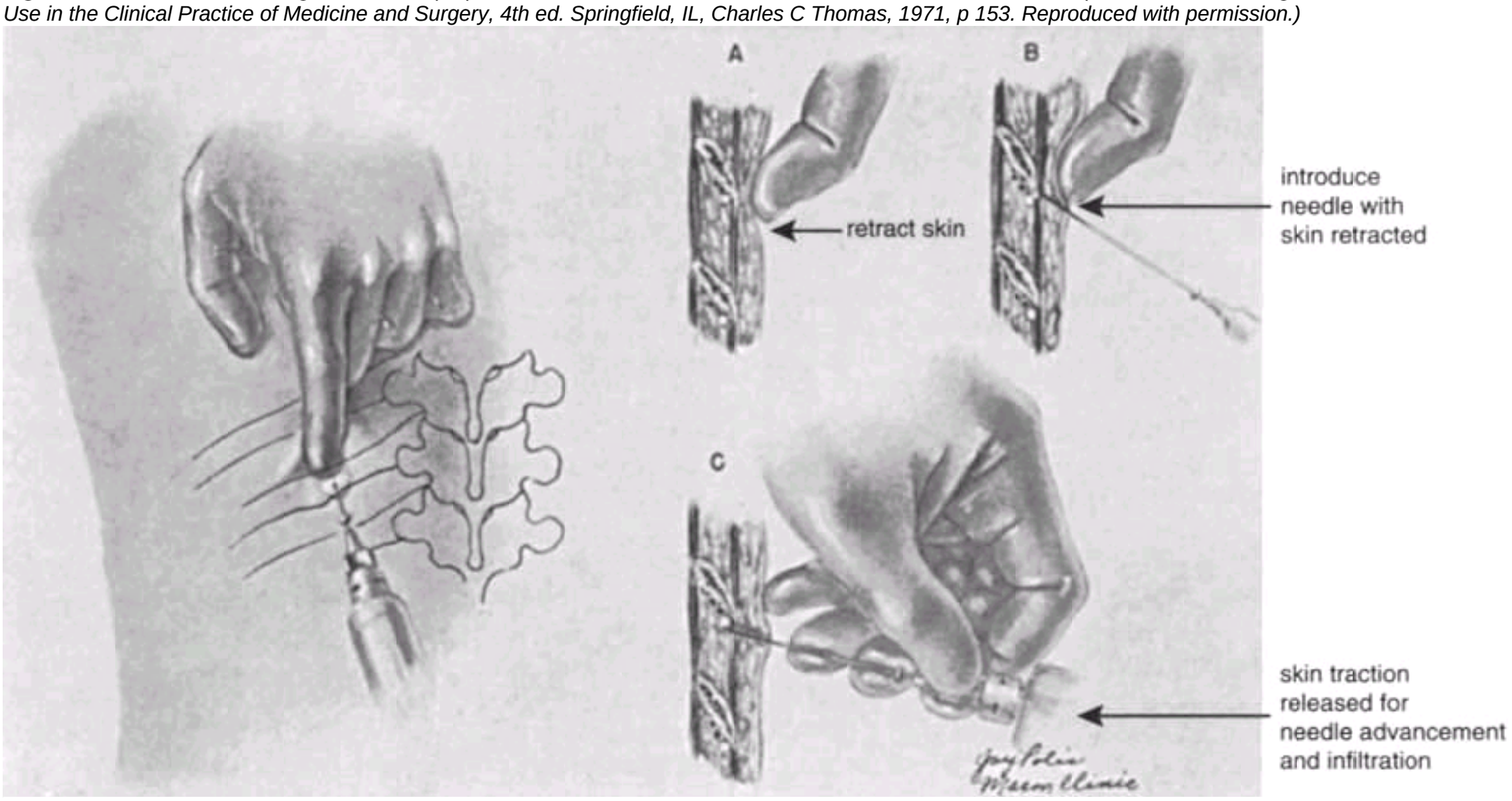
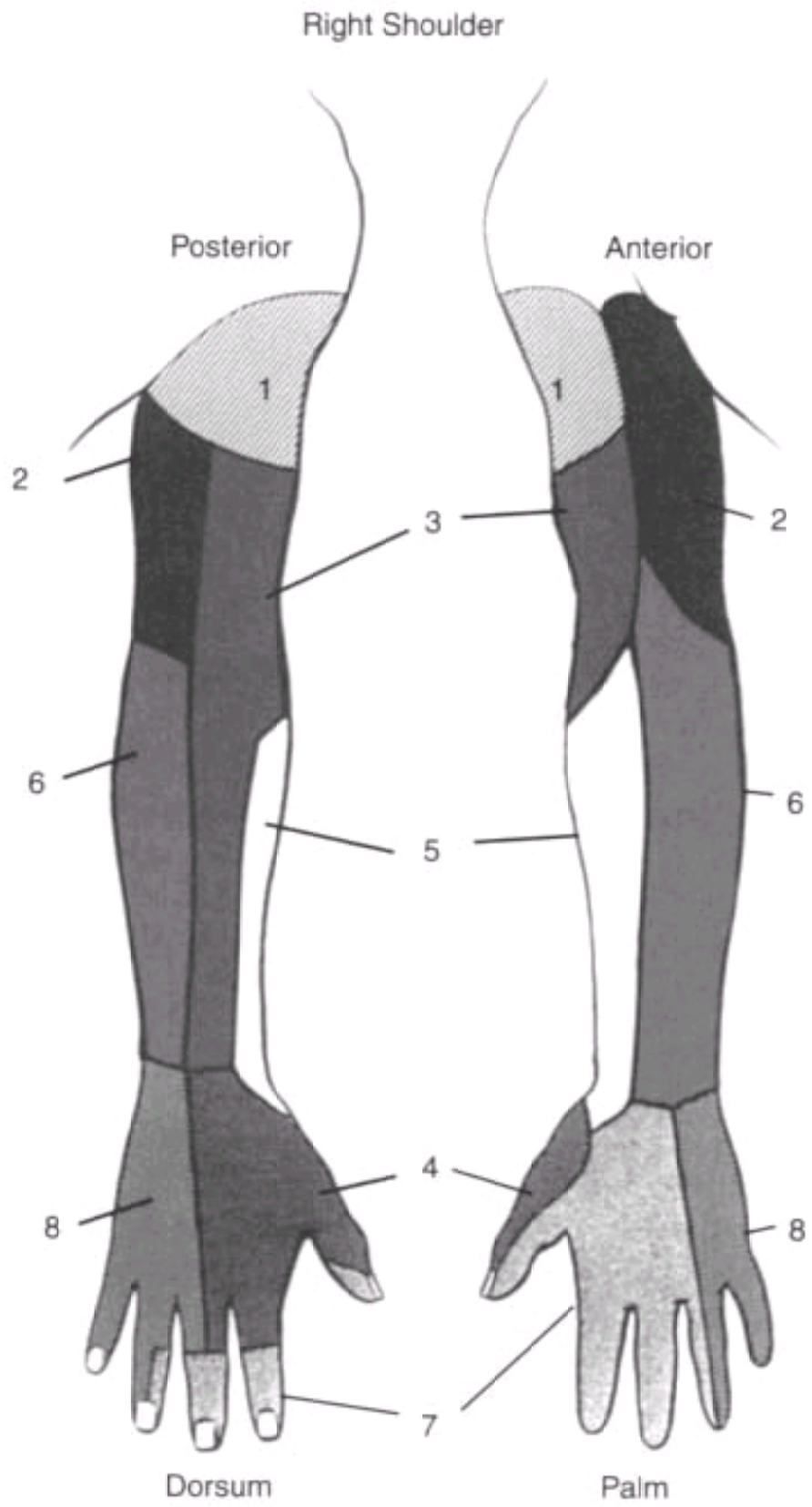


Figure 32-4 Cutaneous nerve supply of the right upper limb. *(From Bridenbaugh LD: The upper extremity: Somatic blockade. In Cousins M, Bridenbaugh PO [eds]: Neural Blockade in Clinical Anesthesia and Management of Pain, 2nd ed. Philadelphia, JB Lippincott, 1988, p 412. Reproduced with permission.)*



Upper Lateral Cutaneous Nerve of Arm (1)

Medial Cutaneous Nerve of Arm and Intercosto-Brachial (2)

Cutaneous Branches of Radial Nerve (lower lateral cutaneous nerve of forearm & posterior cutaneous nerves of arm and forearm) (3)

Superficial Radial Nerve (4)

Lateral Cutaneous Nerve of Forearm (5)

Medial Cutaneous Nerve of Forearm (6)

Median Nerve (7)

Ulnar Nerve (8)

Figure 32-5 Cross section of the wrist looking cephalad. The arrow points to the (covered) median nerve. Shaded triangle depicts the area infiltrated with anesthetic. Note the relatively superficial position of the median nerve, just radial to the palmaris longus. *(Redrawn from Adriani J: Labat's Regional Anesthesia: Techniques and Clinical Applications. 3rd ed. Philadelphia, WB Saunders, 1967, p 234. Reproduced with permission.)*

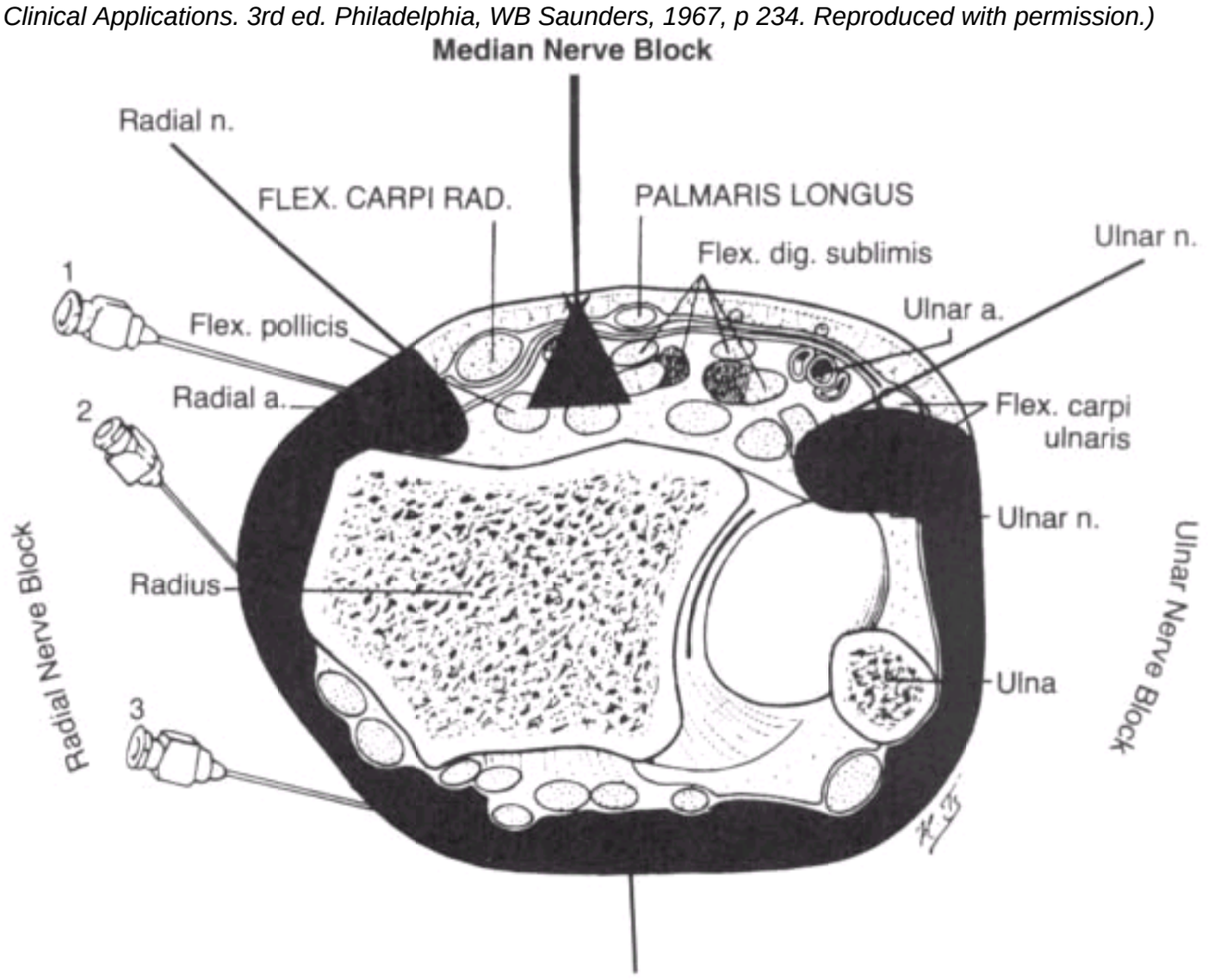


Figure 32-6 Landmarks and anatomy of the median and ulnar nerve block. The median nerve is more superficial than often expected. It lies to the radial side of the palmaris tendon.

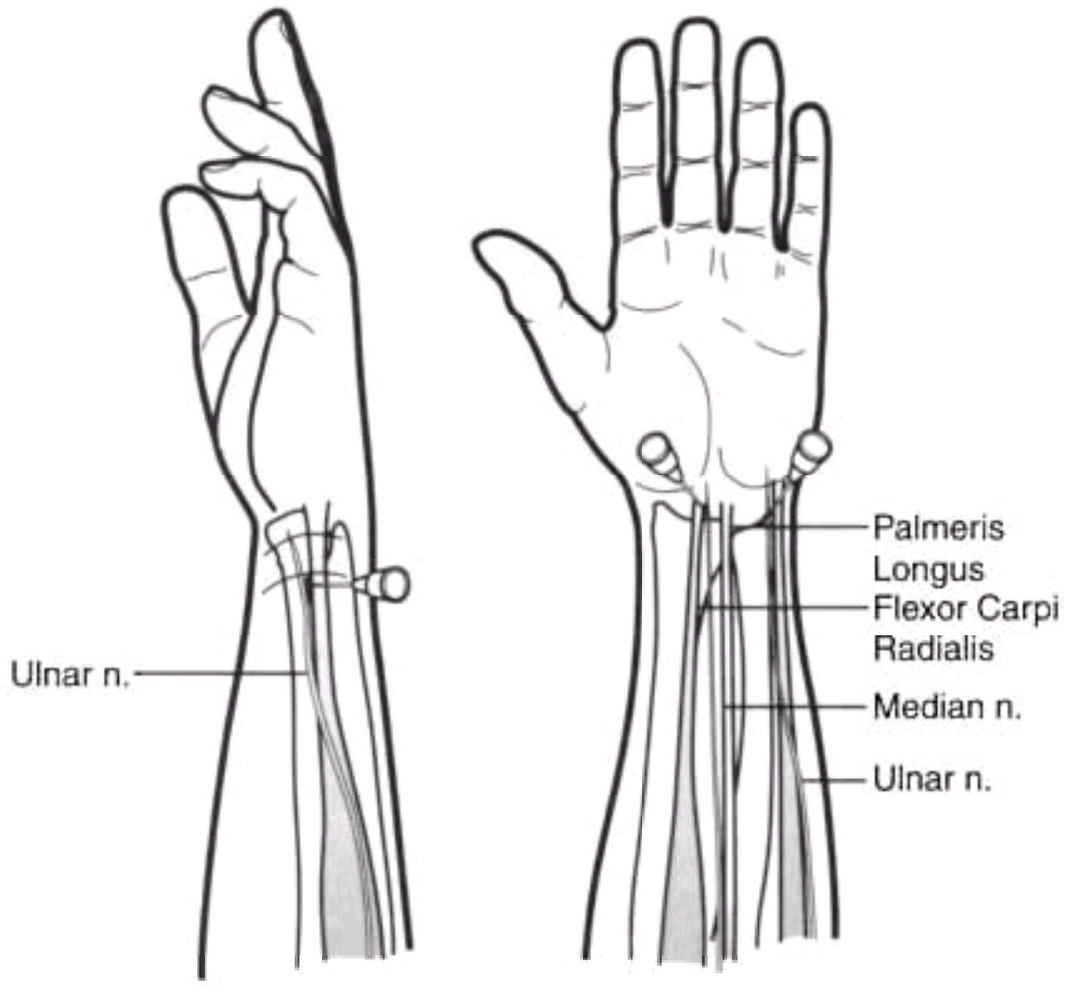


Figure 32-7 Ulnar nerve block at the elbow. *A,* Positioning patient for ulnar nerve block at the elbow. The patient is supine, the elbow flexed, and the medial epicondyle and ulnar groove are exposed. *B,* The ulnar nerve passes between the olecranon (*) and medial epicondyle (•) of the humerus. Avoid injecting large amounts of anesthesia directly into the ulnar groove. Instead, place anesthetic proximal to the groove. Alternatively, the ulnar nerve may be blocked at the wrist.

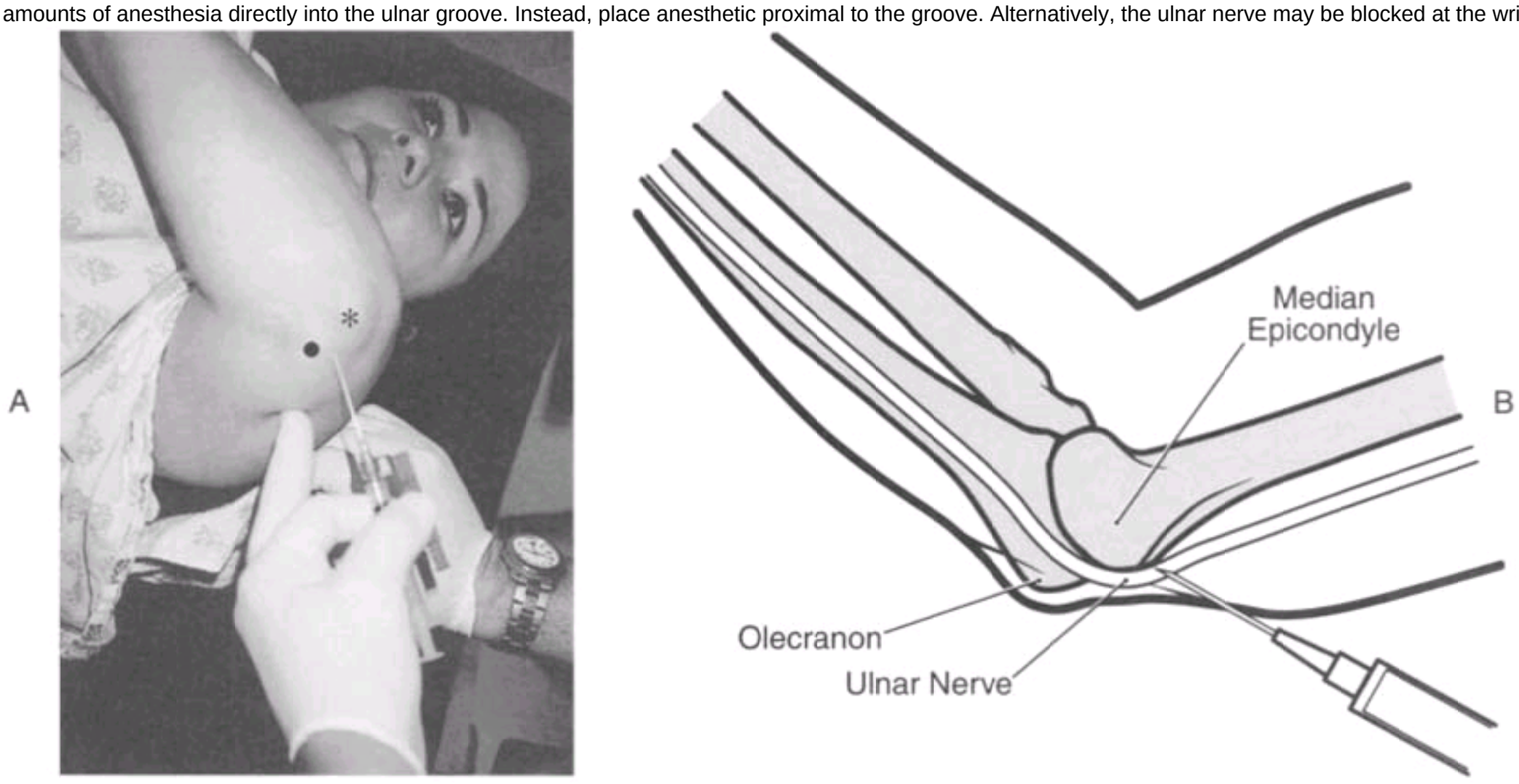


Figure 32-8 Cross section of the elbow, demonstrating (1) the median nerve, (2) the radial nerve, and (3) the ulnar nerve. *(From Adriani J: Labat's Regional Anesthesia: Techniques and Clinical Applications. 3rd ed. Philadelphia, WB Saunders, 1967, p 234. Reproduced with permission.)*

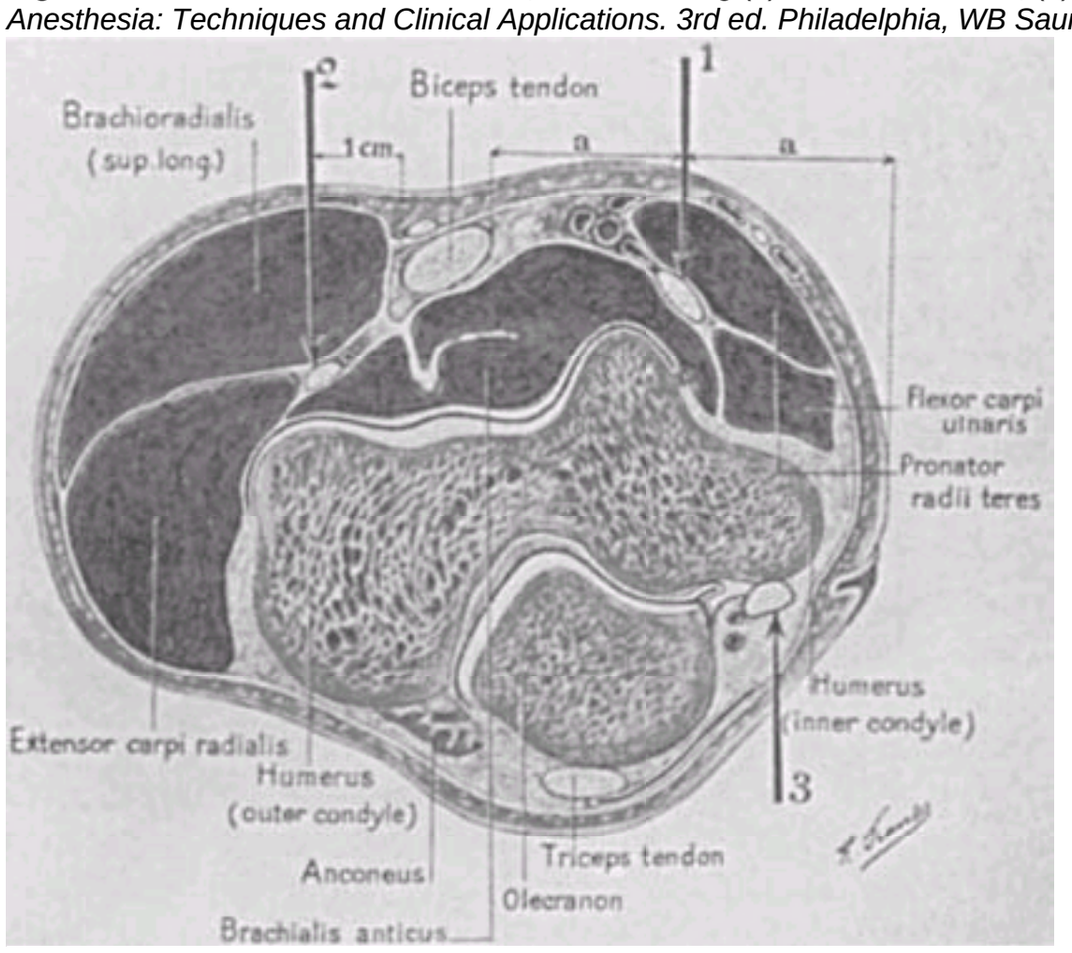


Figure 32-9 Anatomy for nerve blocks at the elbow and wrist. Black circles on skin indicate site of puncture. *(From Adriani J: Labat's Regional Anesthesia: Techniques and Clinical Applications, 3rd ed. Philadelphia, WB Saunders, 1967, p 226. Reproduced with permission.)*

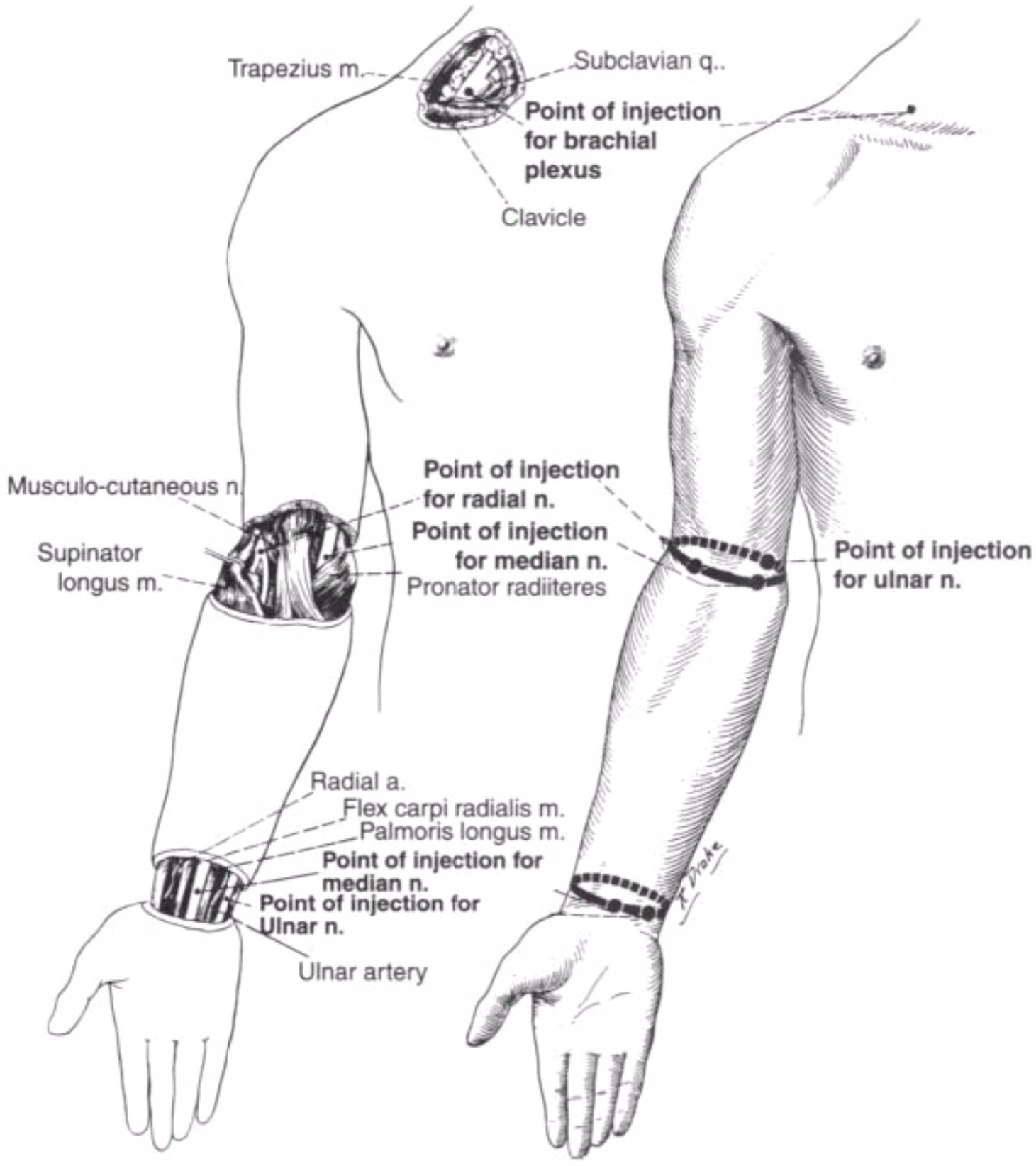


Figure 32-10 Distribution, anatomy, and blockade of the radial nerve at the wrist.

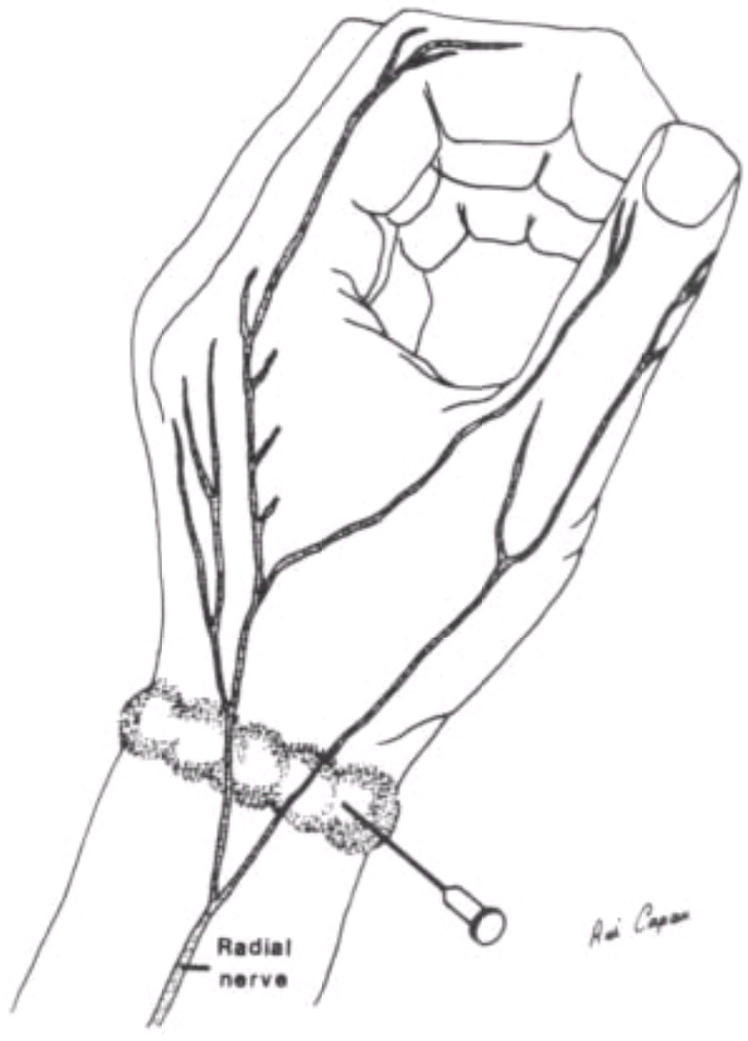
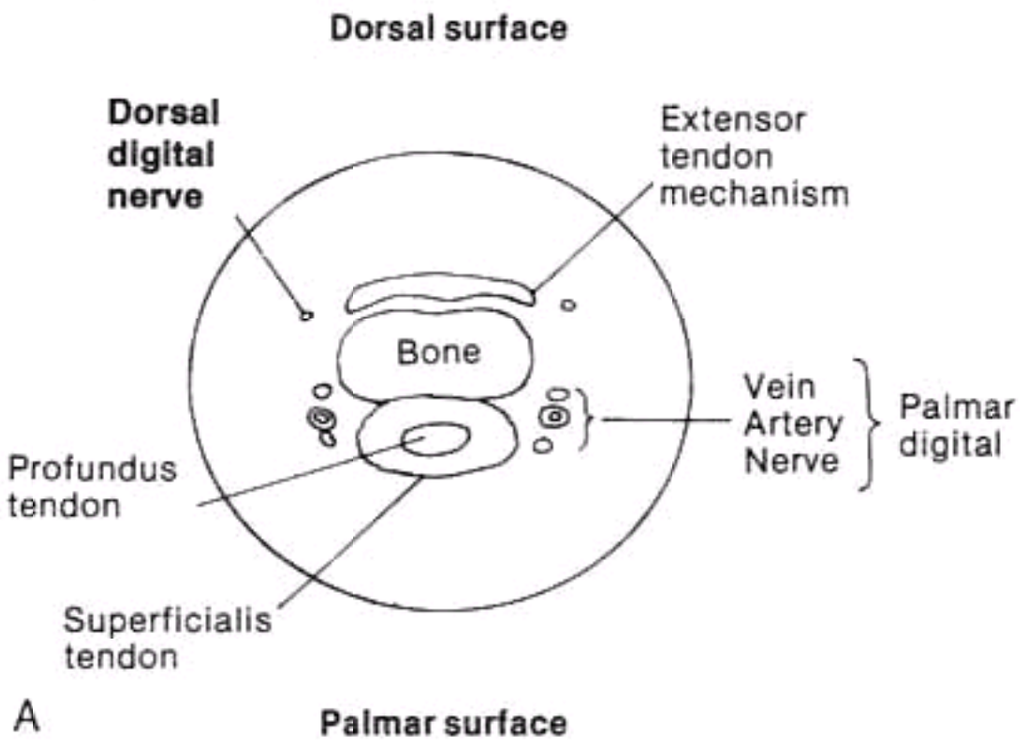


Figure 32-11a *A,* Schematic cross section of the phalanx, demonstrating the relationship of the nerves to the bone. If the anatomy is examined (*B* and *C*), it is obvious that in order to perform a digital block of the thumb and fifth finger, all four digital nerves (two volar, two palmar) must be blocked. Since almost the entire second, third, and fourth fingers are supplied by only the palmar branch (note that each palmar nerve curves around to the dorsum from the palmar surface), only the palmar nerves must be blocked to obtain anesthesia for all but the skin of the proximal dorsal digit. The exception is the ulnar side of the fourth finger, which receives some of its distal dorsal innervation from the dorsal nerve. *D,* On the palmar aspect, the digital nerves are almost adjacent to the flexor tendon, a position that is more palmar than is usually appreciated. *(From Grant's Atlas of Anatomy, 9th ed. Baltimore, Williams & Wilkins, 1978. Reproduced with permission.)*



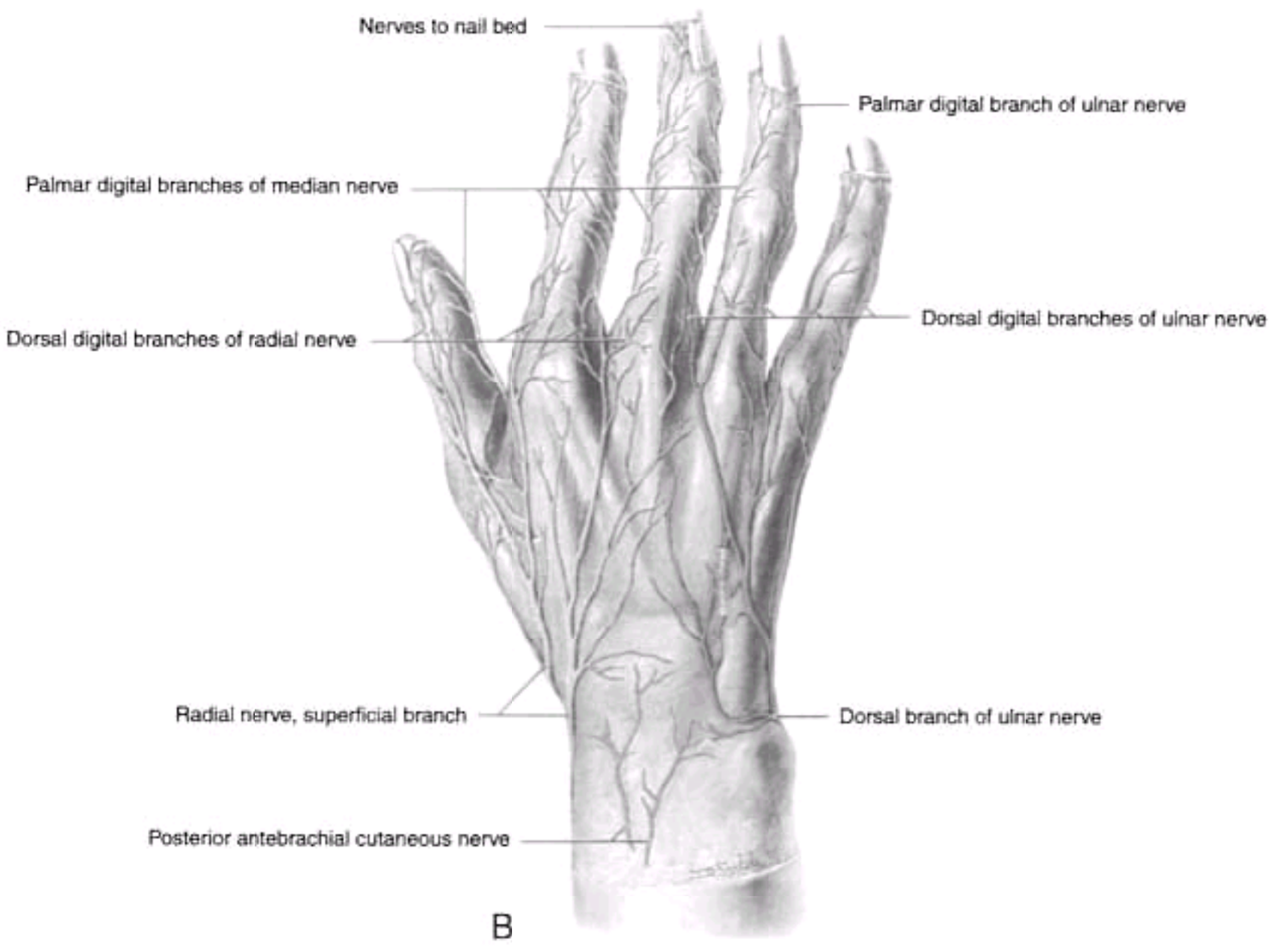


Figure 32-11b

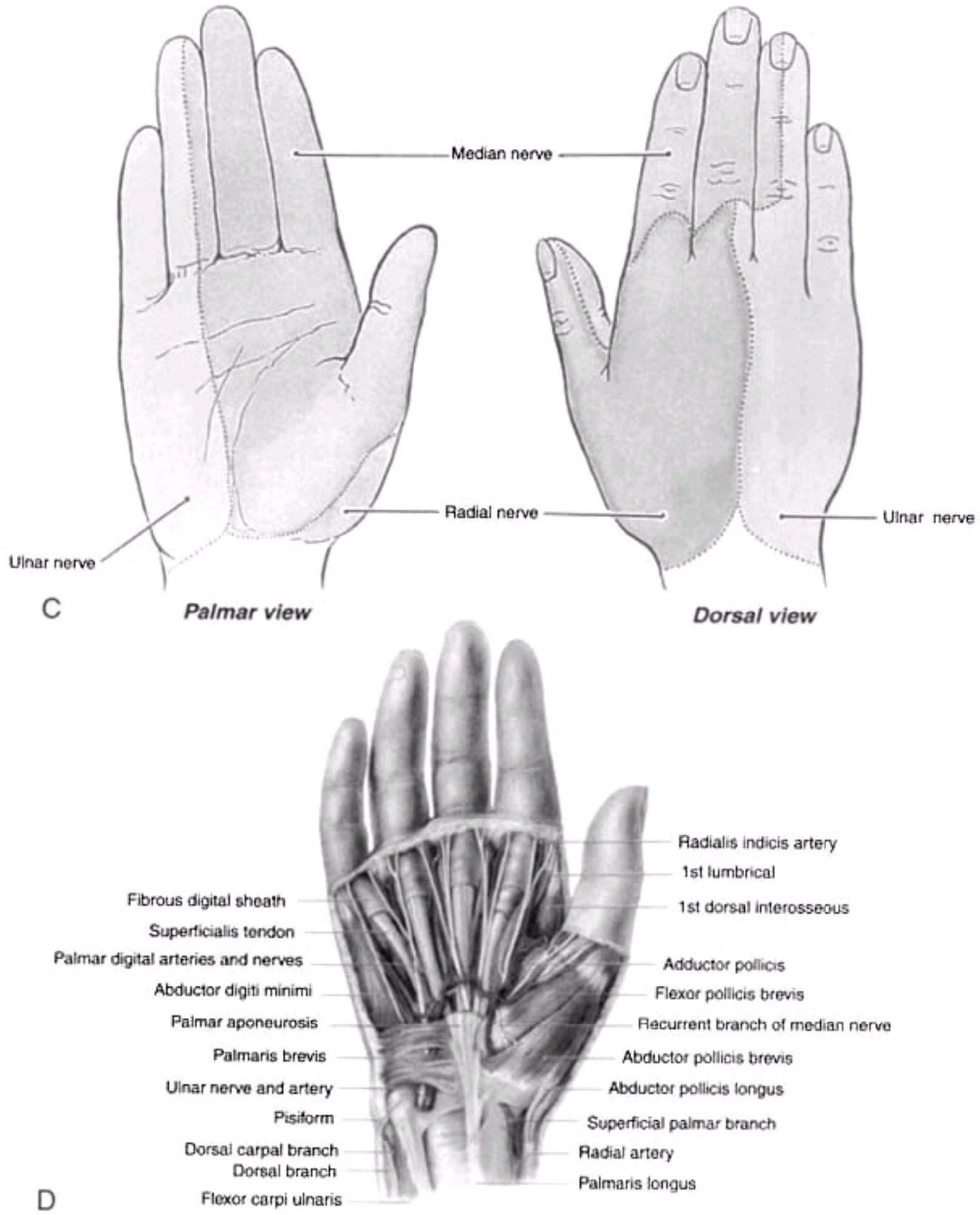
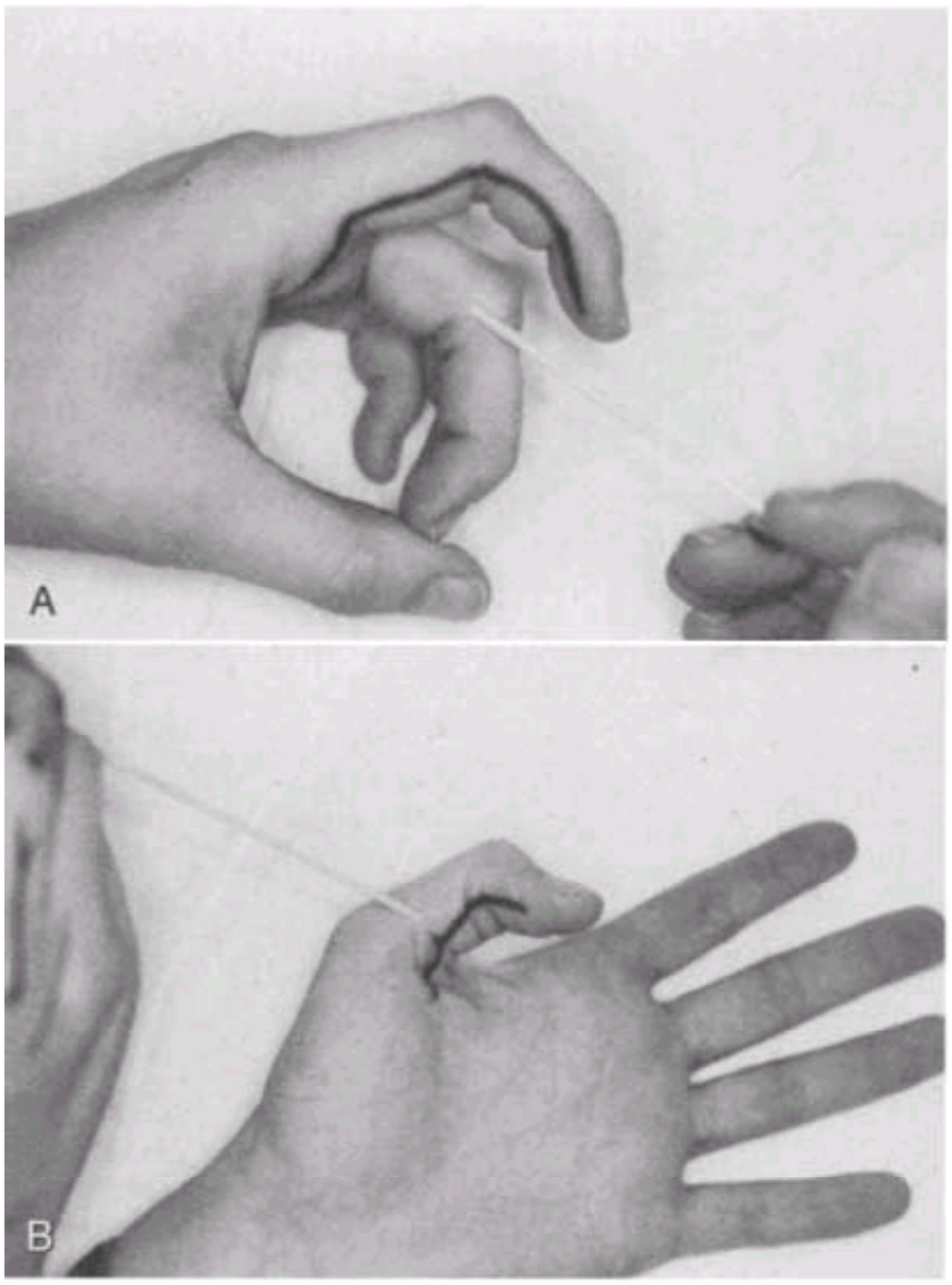


Figure 32-12a *A,* Each finger has four digital nerves: two palmar and two dorsal nerves. The palmar nerves travel in a line connecting the top of the skin creases made by flexing the proximal interphalangeal and distal interphalangeal joints *(black line)*. The nerves are more palmar than often appreciated, and are almost adjacent to the flexor tendon, so injecting the true lateral portion of the finger may miss the nerve. If the anesthesia needle is inserted at the tip of the skin creases (see probe) the nerve will be blocked. *B,* When performing a digital nerve block on the thumb, all four digital nerves must be blocked. As in the other fingers, the palmar nerve travels in a line made by connecting the top of the skin creases produced by flexing the digit *(black line)*. The anesthetizing needle (see probe) must be placed almost adjacent to the flexor tendon to block the nerve. *C,* Sites of digital nerve blocks at the base of the finger. *D,* Technique of anesthetizing the second injection site. *(From Adriani J: Labat's Regional Anesthesia: Techniques and Clinical Applications, 3rd ed. Philadelphia, WB Saunders, 1967, p 445. Reproduced with permission.)*



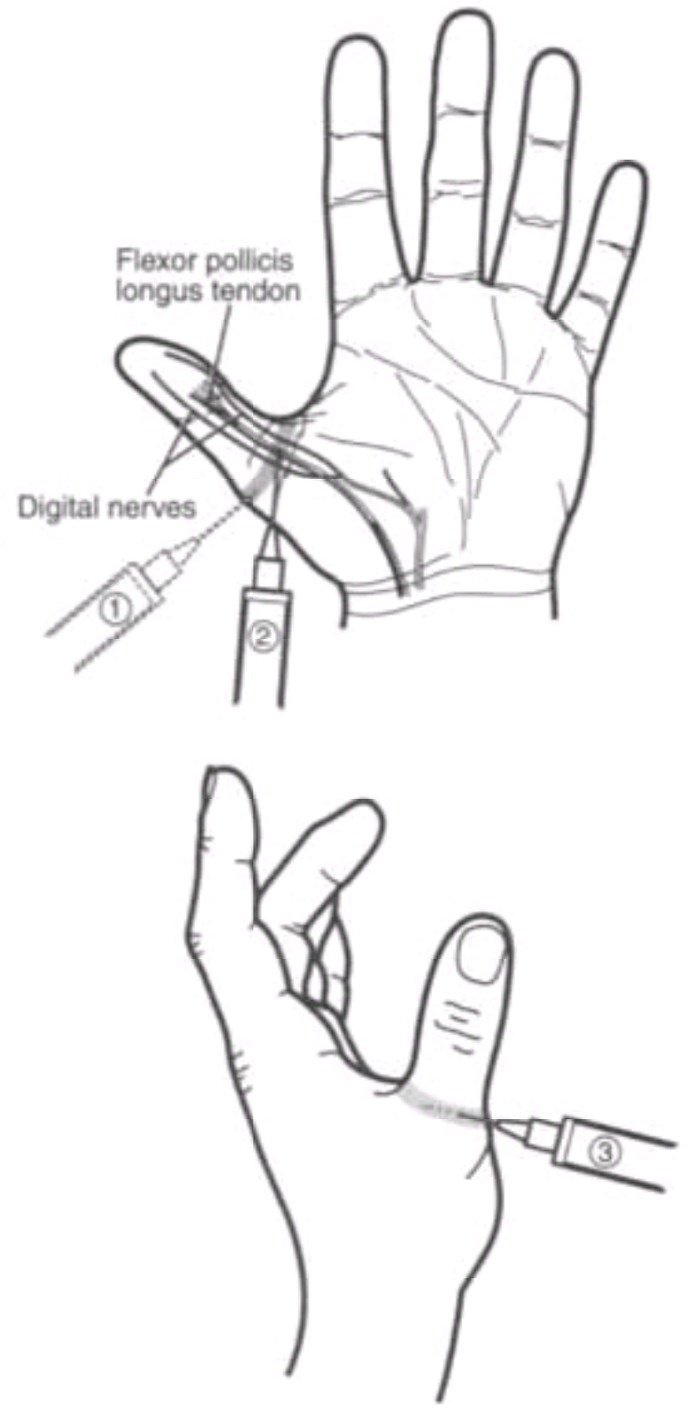


Figure 32-12b

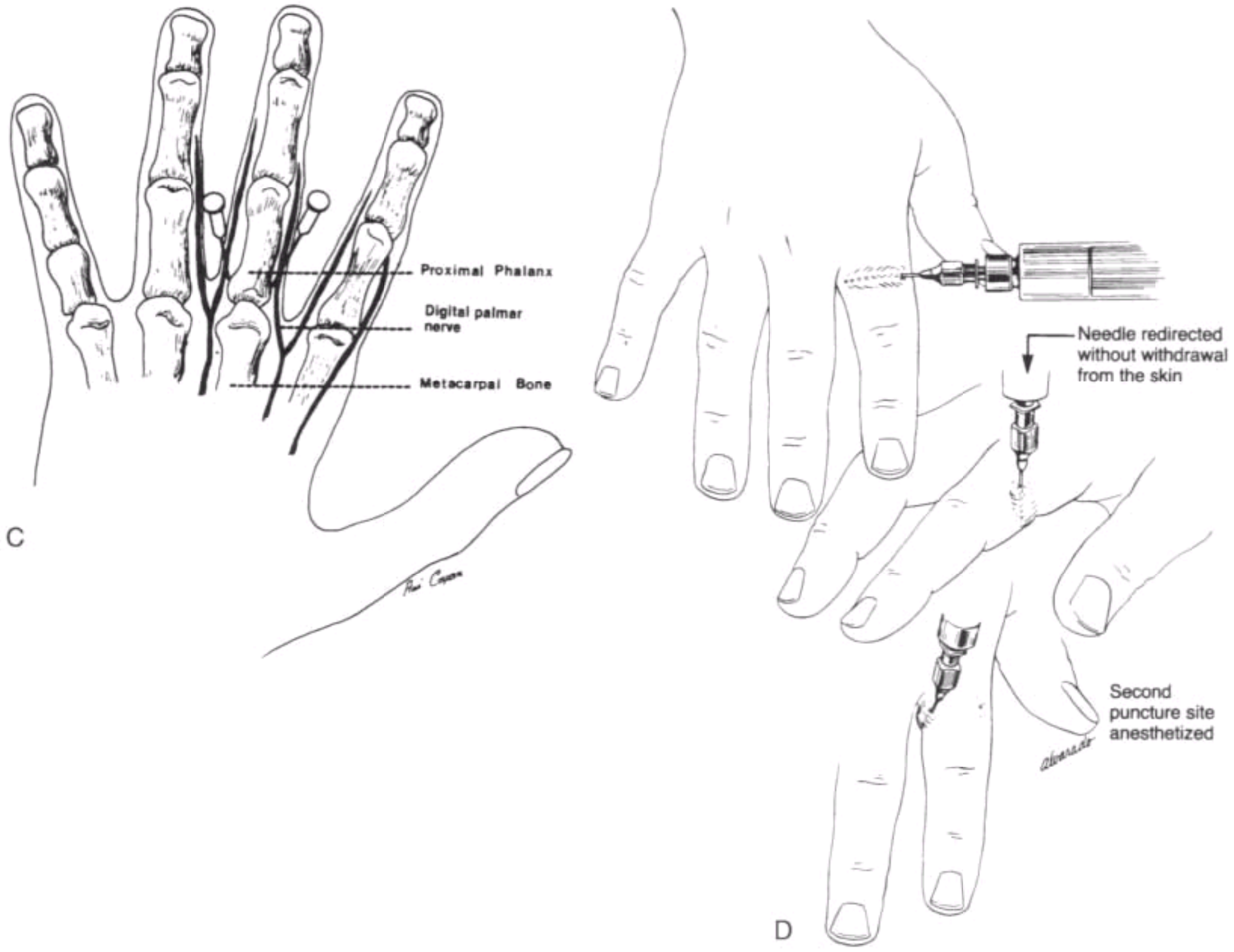


Figure 32-13 Palmar approach to the metacarpal head. After needle puncture in the midline, the needle is directed slightly to the right and left, and anesthetic is deposited along the course of both volar digital nerves. The needle is not withdrawn until both nerves are blocked. This technique blocks only the palmar nerves.

Figure 32-14 The palmar digital nerves (*N*) are adjacent to the head of the metacarpal (*MC*) in the palm. For a metacarpal head block the anesthetizing needle punctures the palm in the middle of the MC (see probe), and anesthetic is deposited both laterally and medially, blocking both nerves with a single puncture. Injection in this area is usually more painful than injecting in the web space, but it is almost foolproof for obtaining anesthesia.

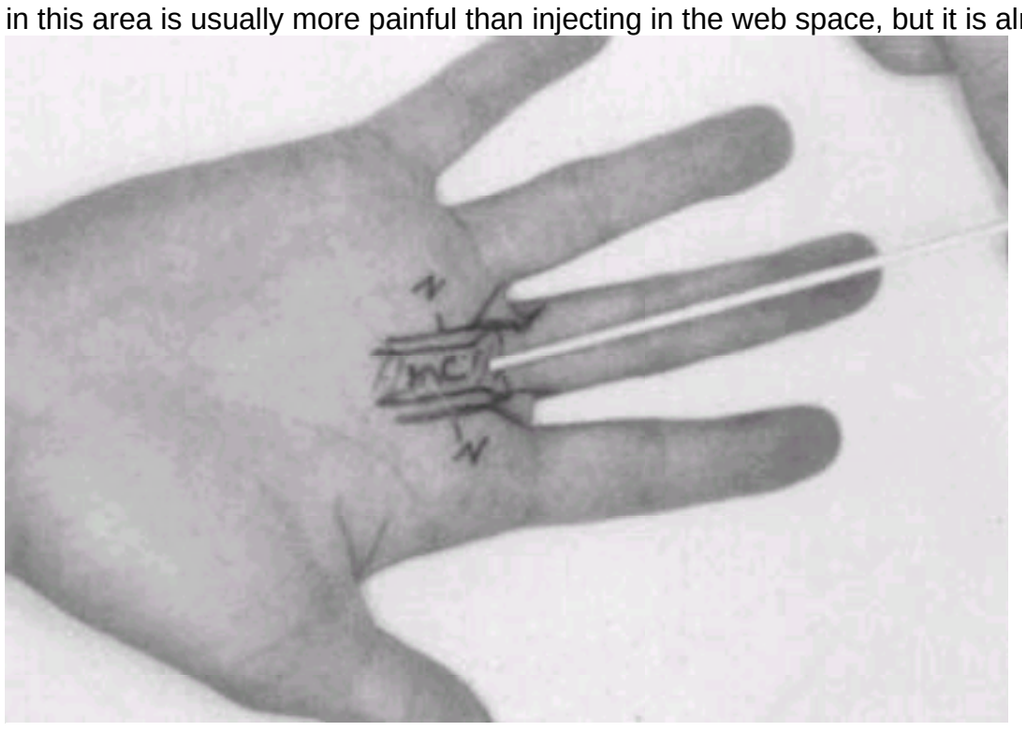
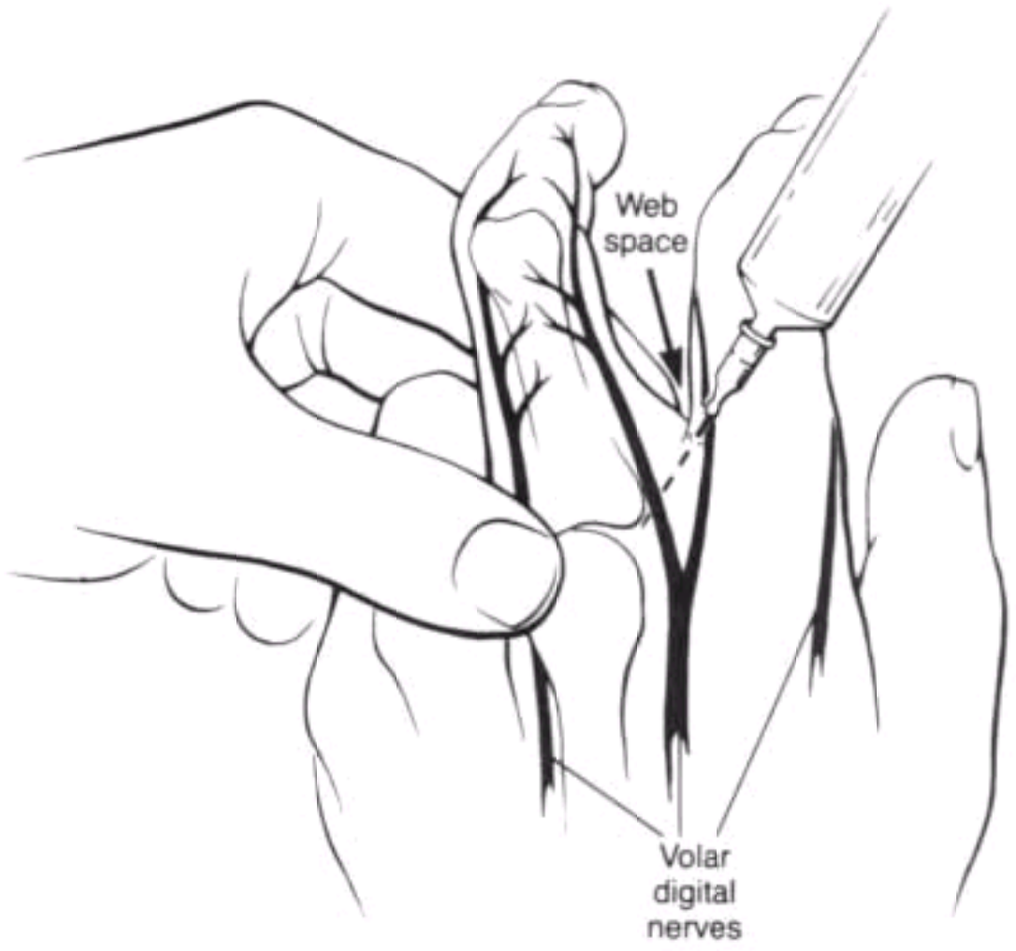


Figure 32-15 The palmer web space metacarpal head block allows two of the three inner fingers to be anesthetized with a single needle puncture. The clinician's index finger palpates the patient's metacarpal head on the patient's palm while the injured finger is supported and the web space is exposed. The index finger can feel the tissue distention by the anesthetic, but care must be taken to avoid passing the needle through the skin and puncturing the operator's finger. About 3 to 5 mL of anesthetic is deposited. To anesthetize the adjacent finger the needle is partly withdrawn and redirected to the other metacarpal head. Bending the needle to 30° allows easier access to the proper position without the syringe getting in the way.

Figure 32-16 Nerve block of the third digit. After injection of anesthetic into the web space skin, the needle is advanced to the digital nerve, where it passes just lateral to the volar metacarpal head. Anesthetic is injected, and the needle is advanced to the opposite digital nerve. This procedure requires about 3 to 5 mL of anesthetic. If the index finger also must be blocked, the needle is redirected without withdrawal from the skin. Thus, both fingers are blocked with a single needle puncture.



M

Figure 32-17 Transthecal block. The flexor tendon sheath is entered volarly just proximal to the metacarpophalangeal joint. With the use of a 25-ga needle on a 3-mL syringe the fluid should flow easily. Inject 2 mL of anesthetic and apply proximal tendon sheath pressure.

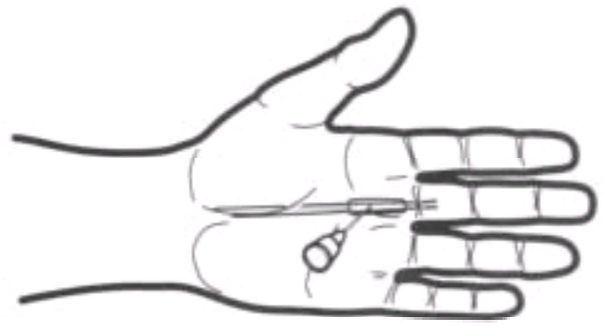


Figure 32-18 Cutaneous distribution of the nerves to the lower extremity. *(From Bridenbaugh PO: The lower extremity: Somatic blockade. In Cousins M, Bridenbaugh PO [eds]: Neural Blockade in Clinical Anesthesia and Management of Pain. 2nd ed. Philadelphia, JB Lippincott, 1988, p 425. Reproduced with permission.)*

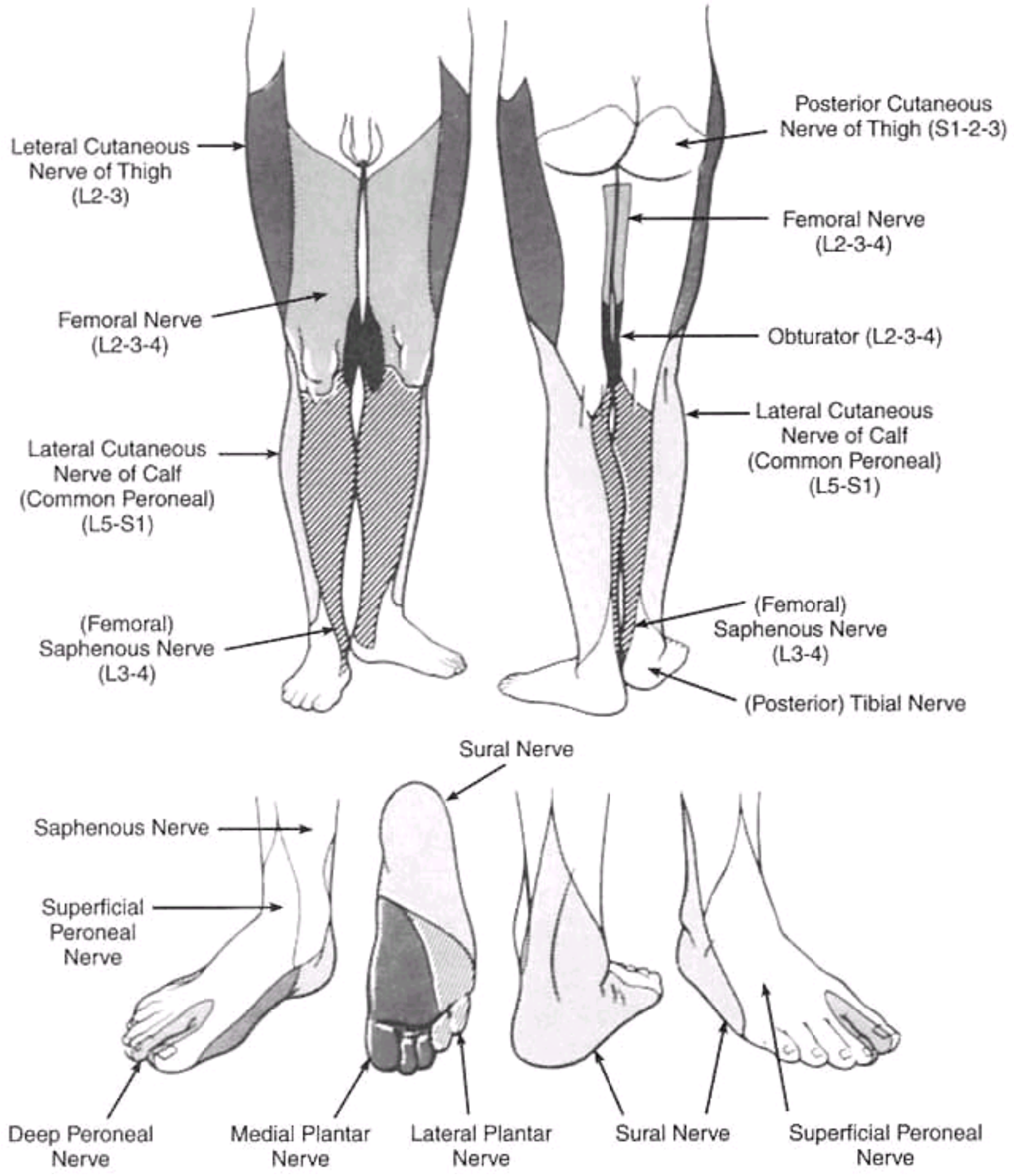


Figure 32-19 Anatomy of the femoral, obturator, and lateral femoral cutaneous nerves and the location of the injection site for the femoral and 3-in-1 nerve blocks. *(Adapted from Winnie AP, Ramamurthy S, Durrani Z: The inguinal paravascular technique of lumbar plexus anesthesia: The "3 in 1 block." Anesth Analg 52:989, 1973. Reproduced with permission.)*

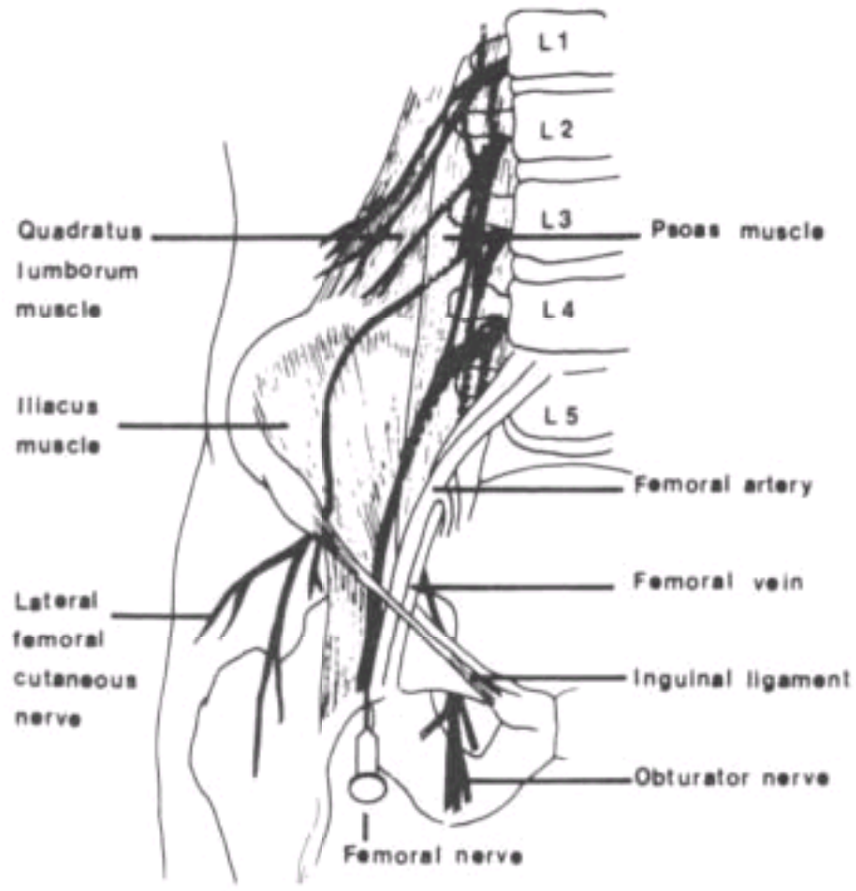
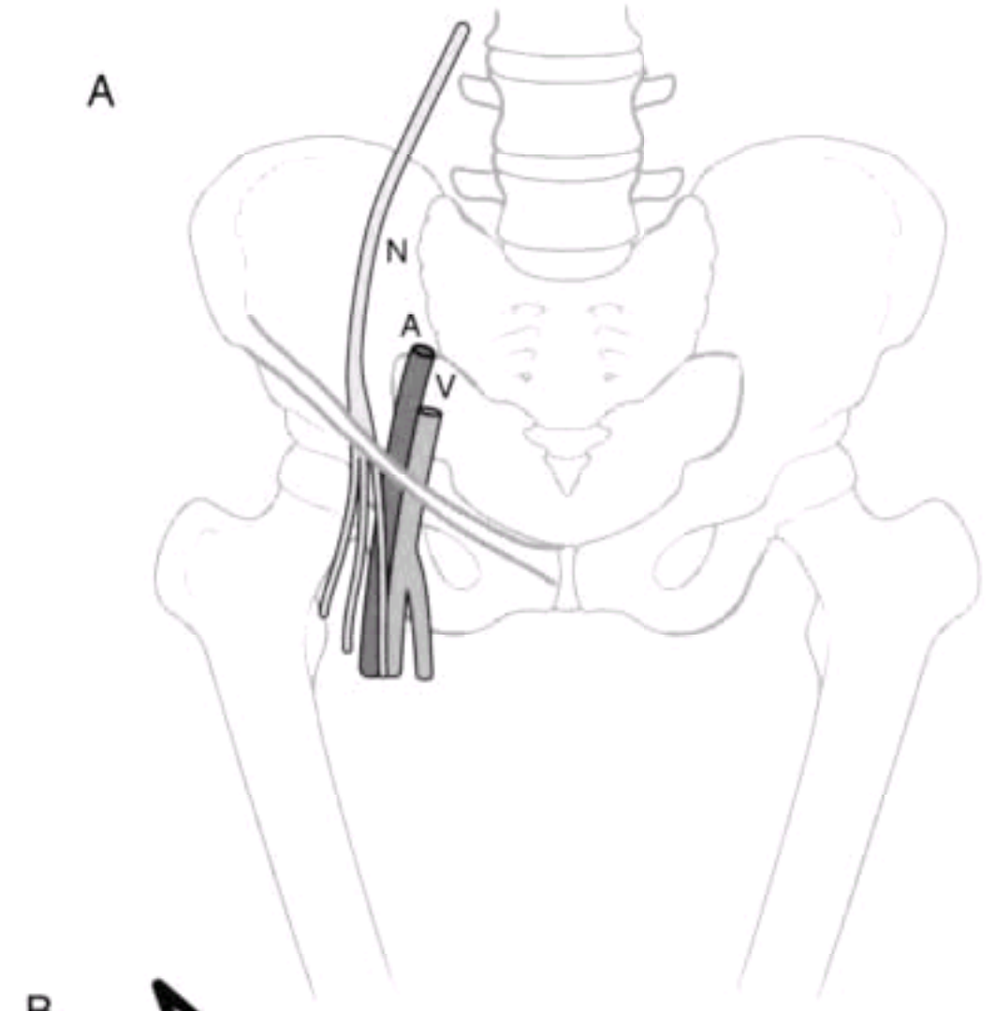


Figure 32-20 *A, B,* 3-in-1 block. See text for details.



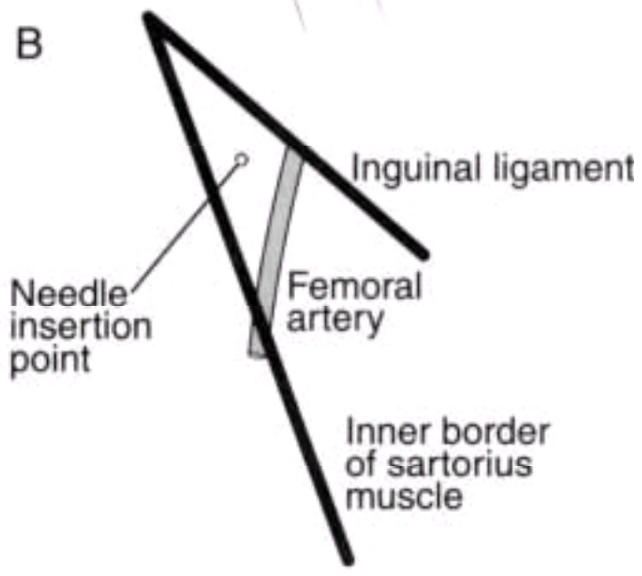


Figure 32-21 Sciatic nerve block (posterior approach). A line is drawn from the greater trochanter to the posterior-superior iliac spine (PSIS). The line is bisected and the needle will be inserted perpendicular to the line at a depth determined by a second line between the greater trochanter and the sacral hiatus.

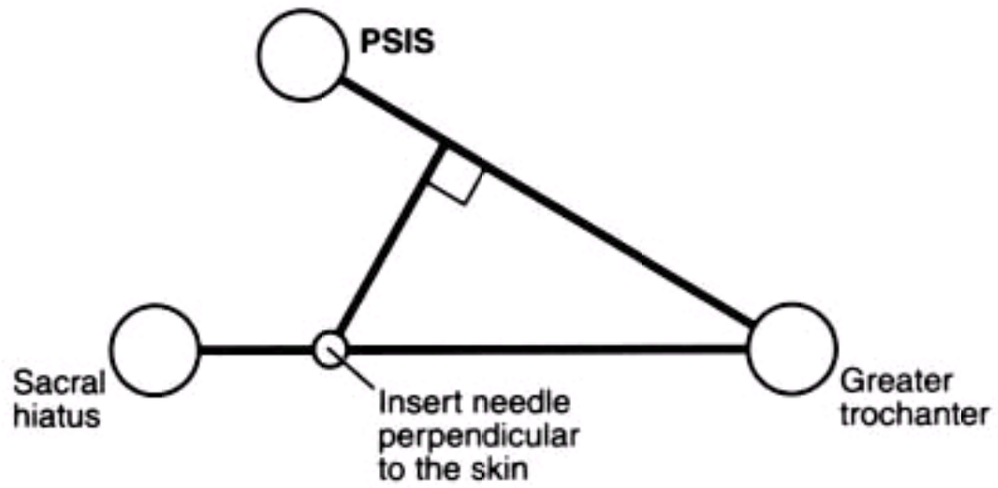
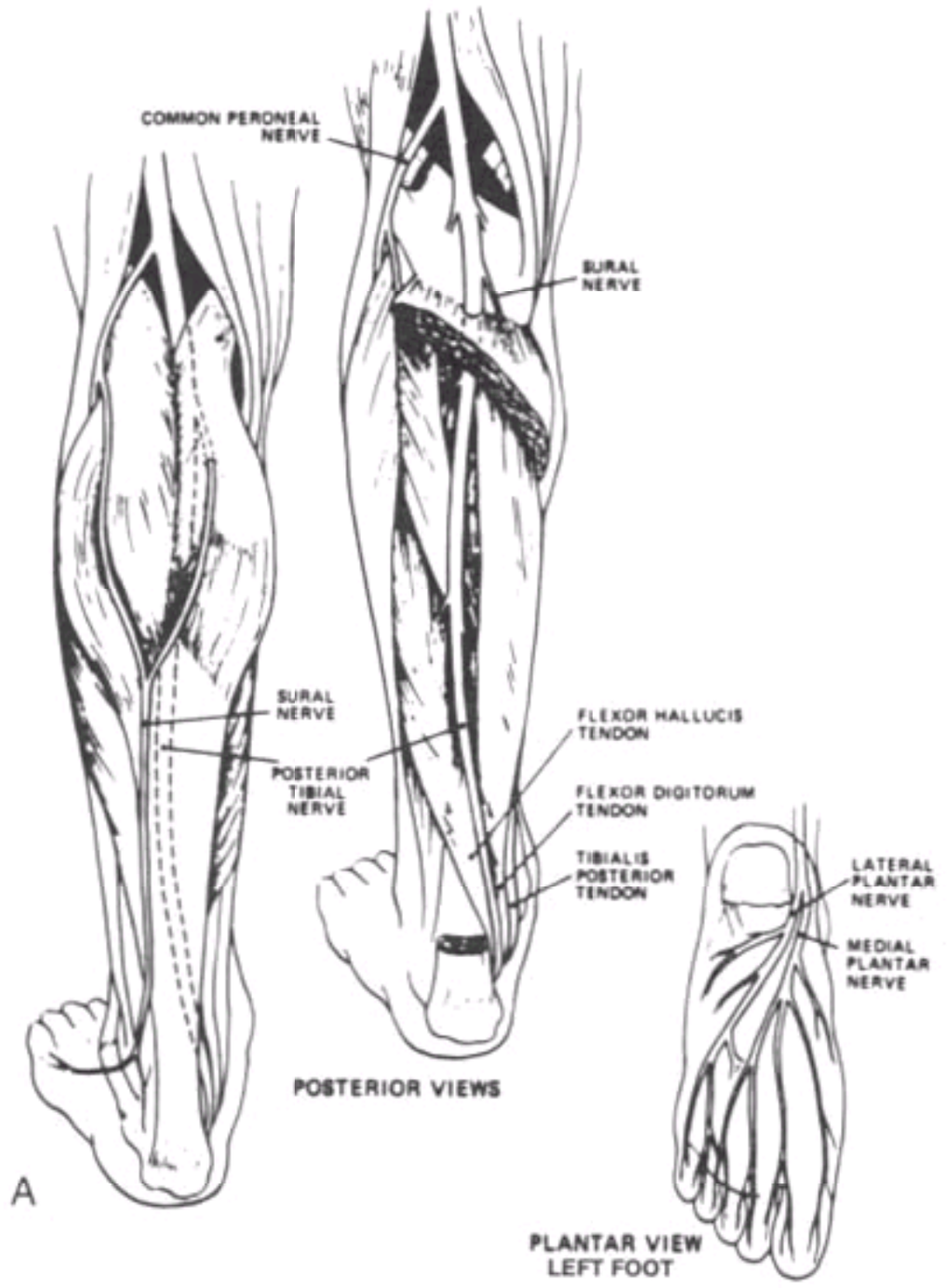


Figure 32-22 *A* and *B,* Anatomy and innervation of the left calf and foot. See Figure 32-18 for areas for innervation. *(From Shurman DH: Ankle-block anesthesia for foot surgery. Anesthesiology 44:348, 1976. Reproduced with permission.)*



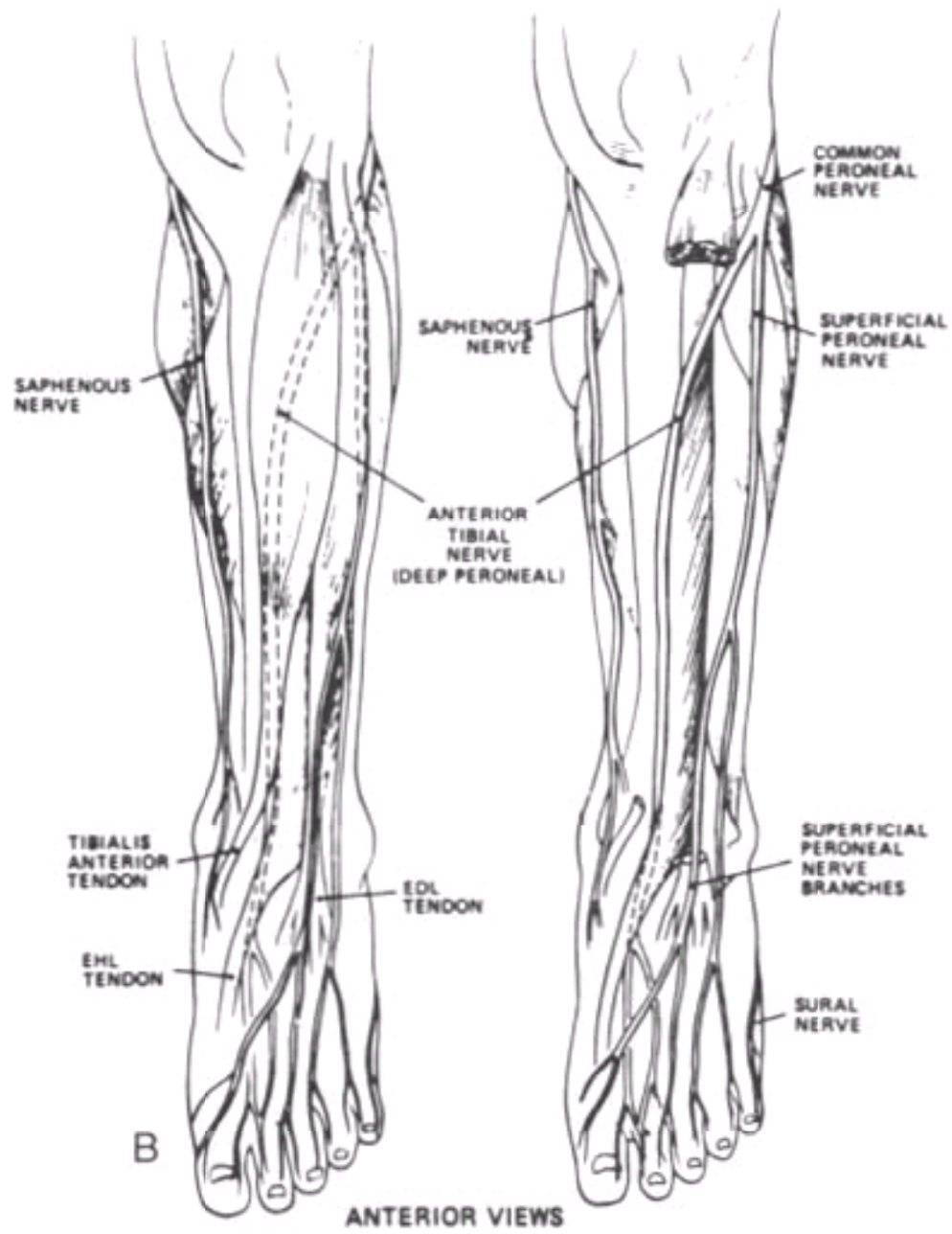


Figure 32-23 Anatomy and injection sites for nerve blocks at the ankle. *(From Bridenbaugh PO: The lower extremity: Somatic blockade. In Cousins M, Bridenbaugh PO [eds]: Neural Blockade in Clinical Anesthesia and Management of Pain, 2nd ed. Philadelphia, JB Lippincott, 1988, p 435. Reproduced with permission.)*

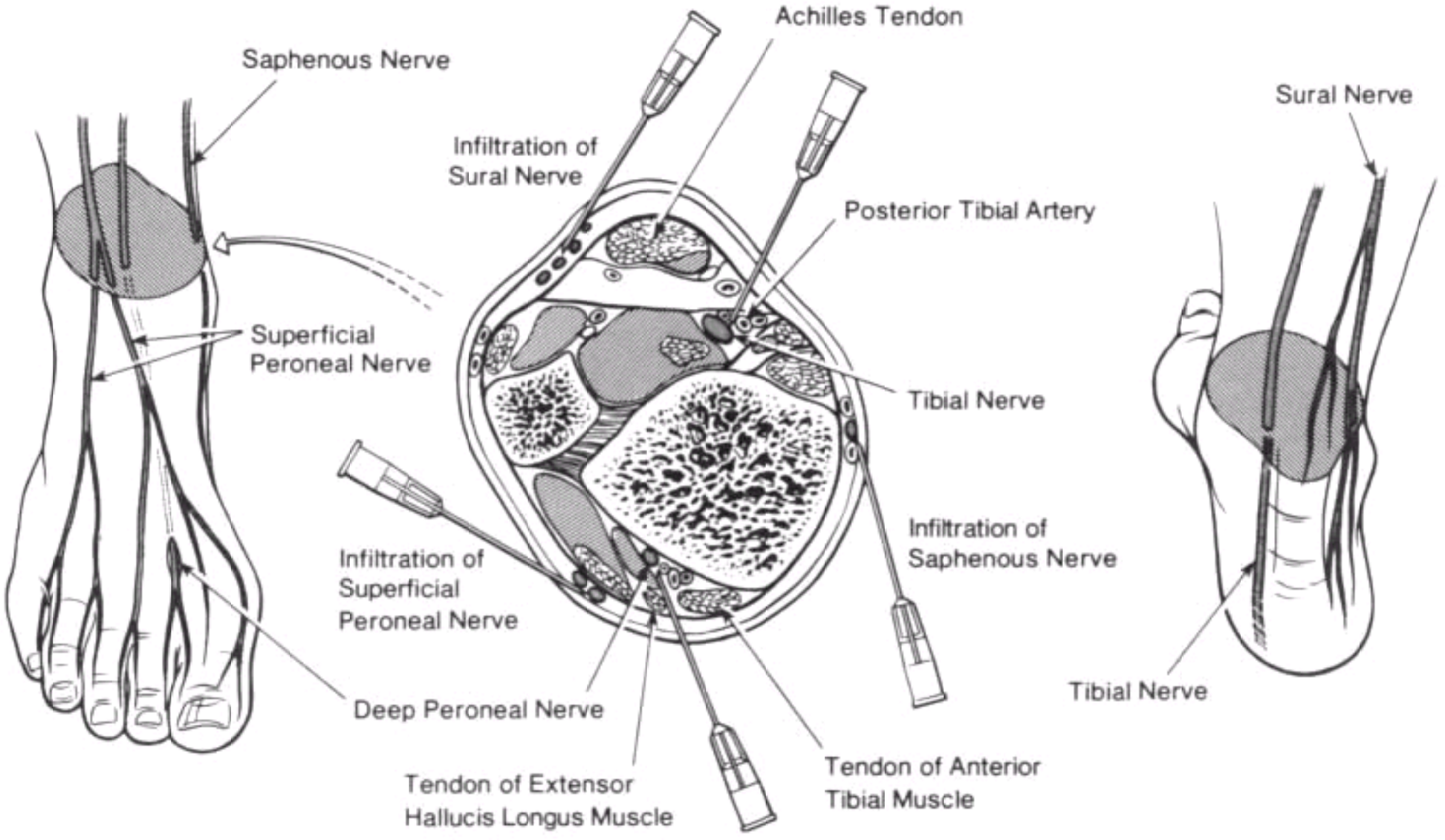


Figure 32-24 Anatomy and injection sites for nerve blocks at the ankle (lateral views). *(Adapted from Locke RK, Locke SE: Nerve blocks of the foot. JACEP 4:698, 1976.)*

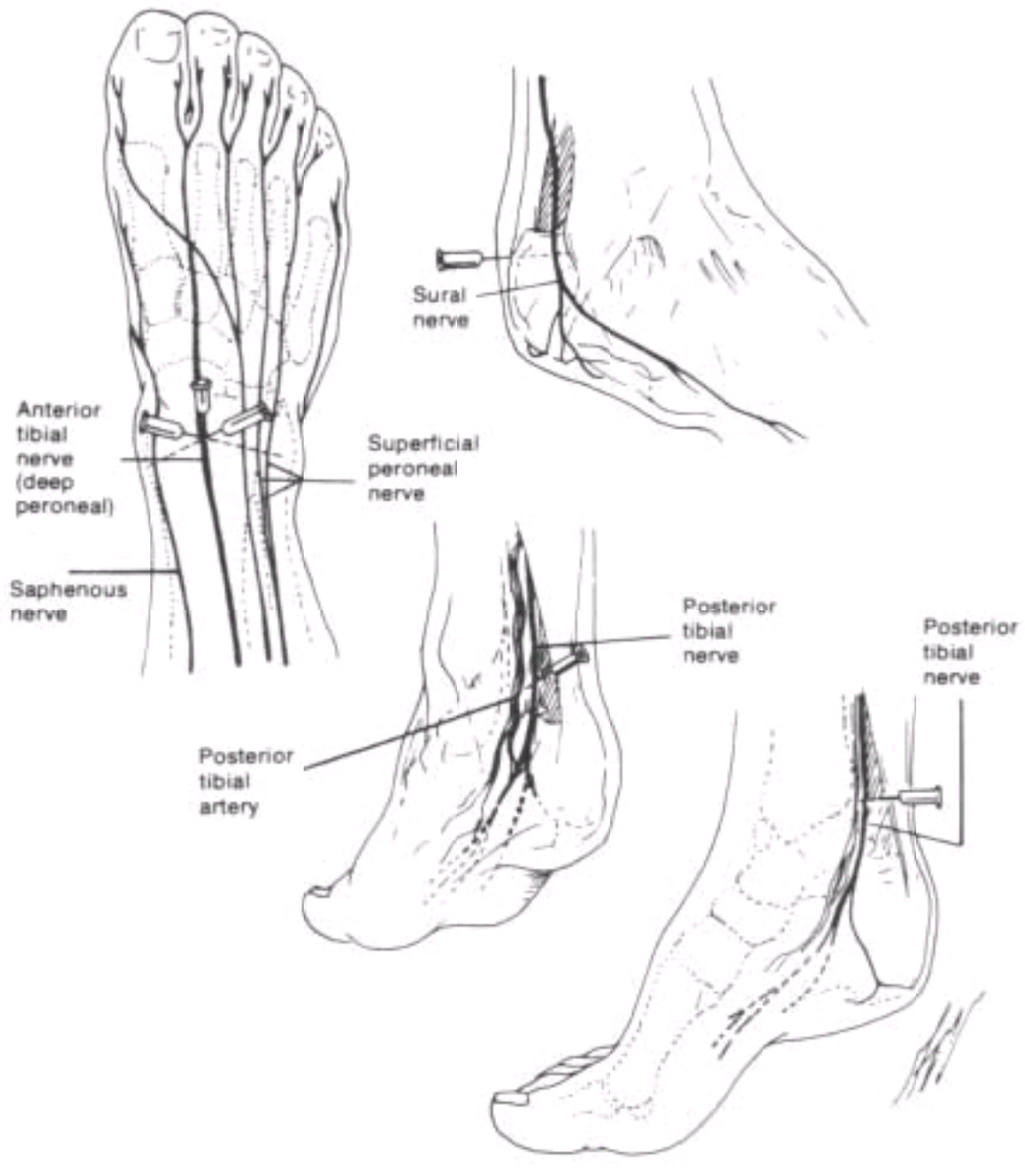


Figure 32-25 Anatomy and technique for digital nerve block at the metatarsals. *(From Bridenbaugh PO: The lower extremity: Somatic blockade. In Cousins M, Bridenbaugh PO [eds]: Neural Blockade in Clinical Anesthesia and Management of Pain, 2nd ed. Philadelphia, JB Lippincott, 1988, p 437. Reproduced with permission.)*

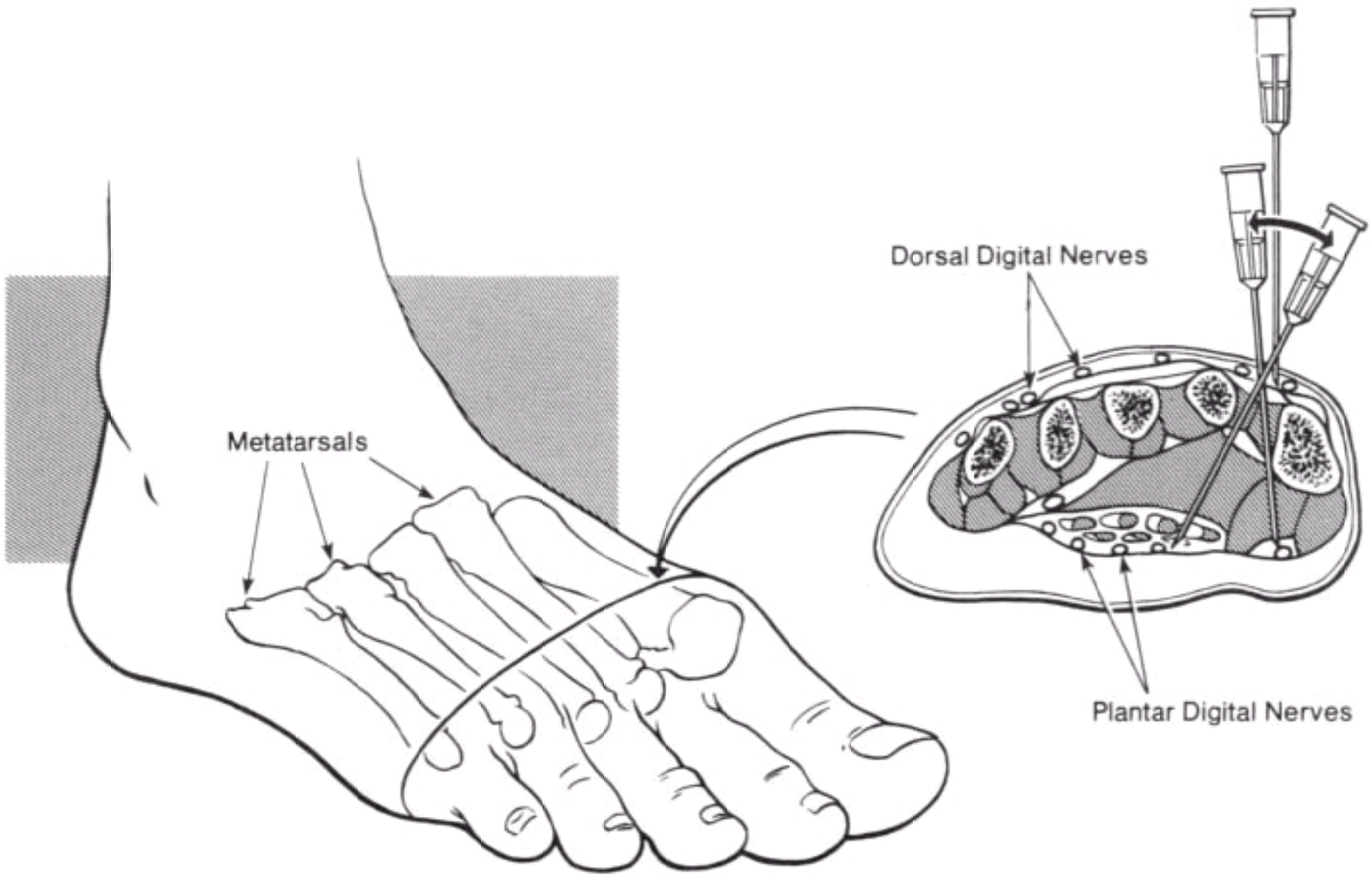


Figure 32-26 Technique of digital nerve blocks in the interdigital web spaces of the foot.

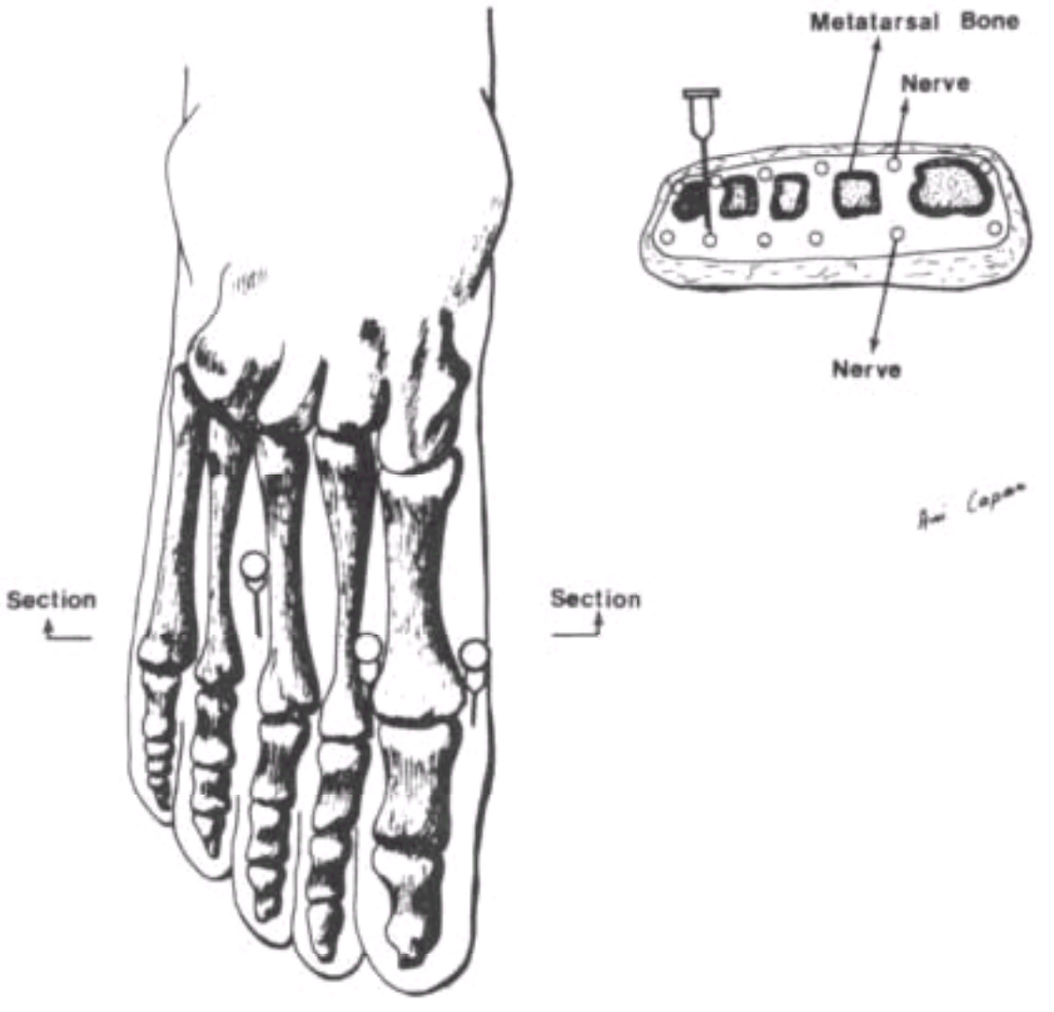


Figure 32-27 Alternative techniques of digital nerve blocks in the toes. *(From Locke RK, Locke SE: Nerve blocks of the foot. JACEP 4:698, 1976. Reproduced with permission.)*

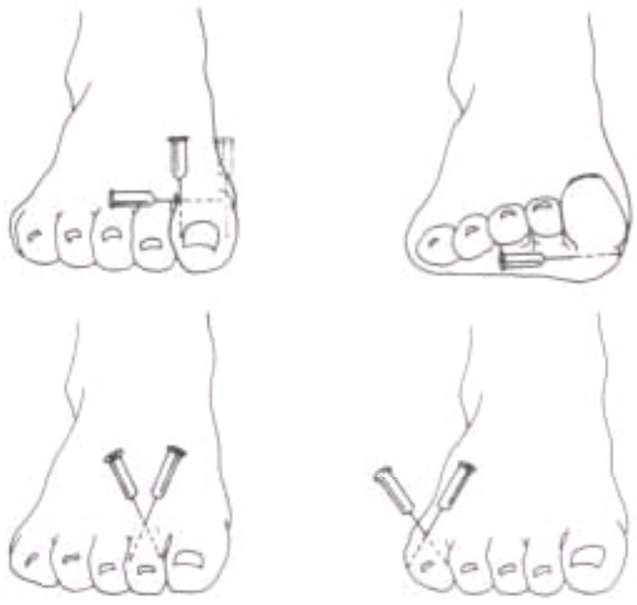


Figure 33-1 Preparation for induction of anesthesia in a patient with a fracture of the right radius. Note precautionary IV line and deflated tourniquet in place. The procedure has been explained, and preoperative sedation or analgesia has been given if required. *(From Roberts JR: Intravenous regional anesthesia. JACEP 6:263, 1977.)*

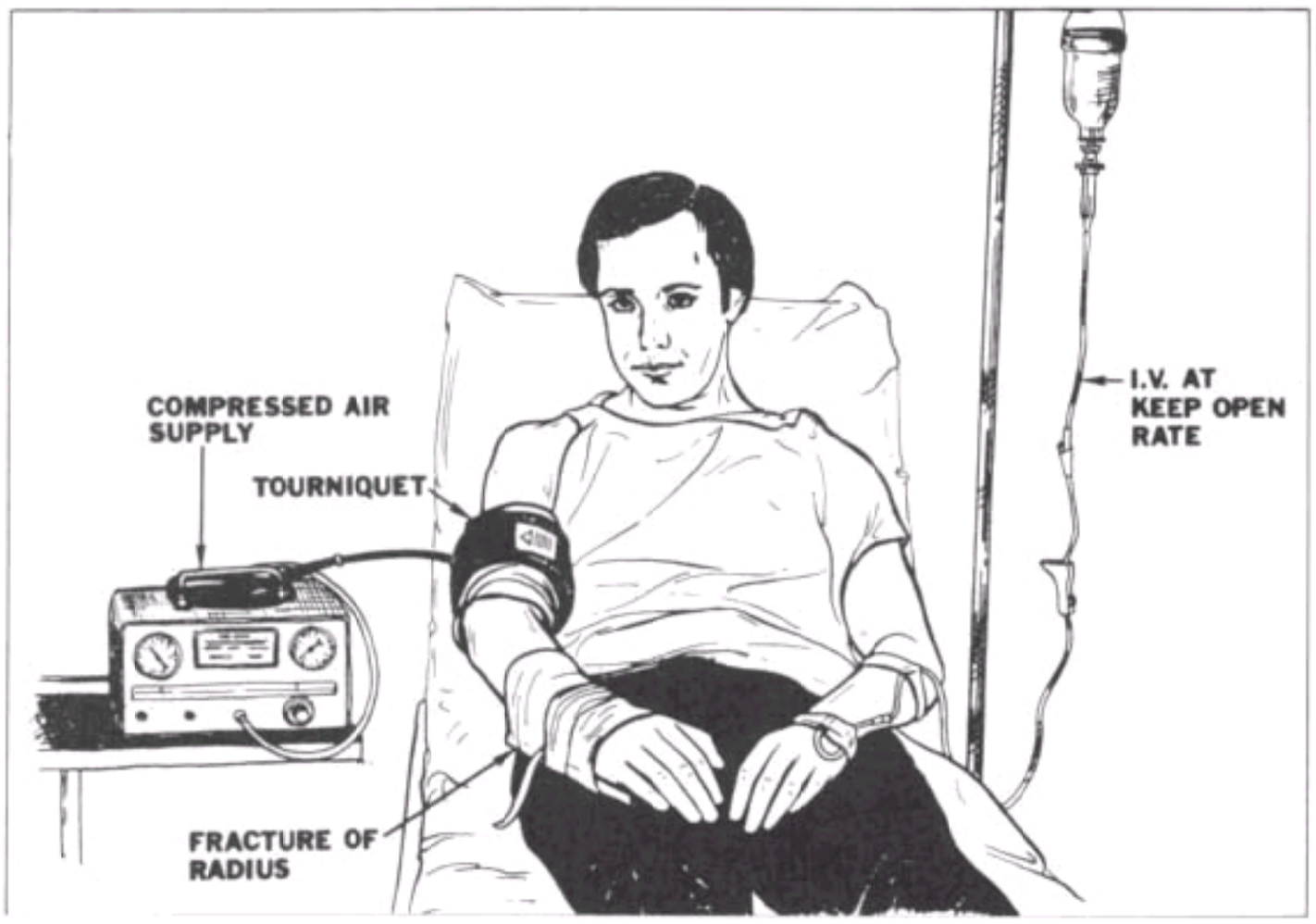


Figure 33-2 Double-cuff apparatus with two-way valves allows longer tourniquet time without pain. A standard blood pressure cuff should never be used. *(From Roberts JR: Intravenous regional anesthesia. JACEP 6:263, 1977.)*

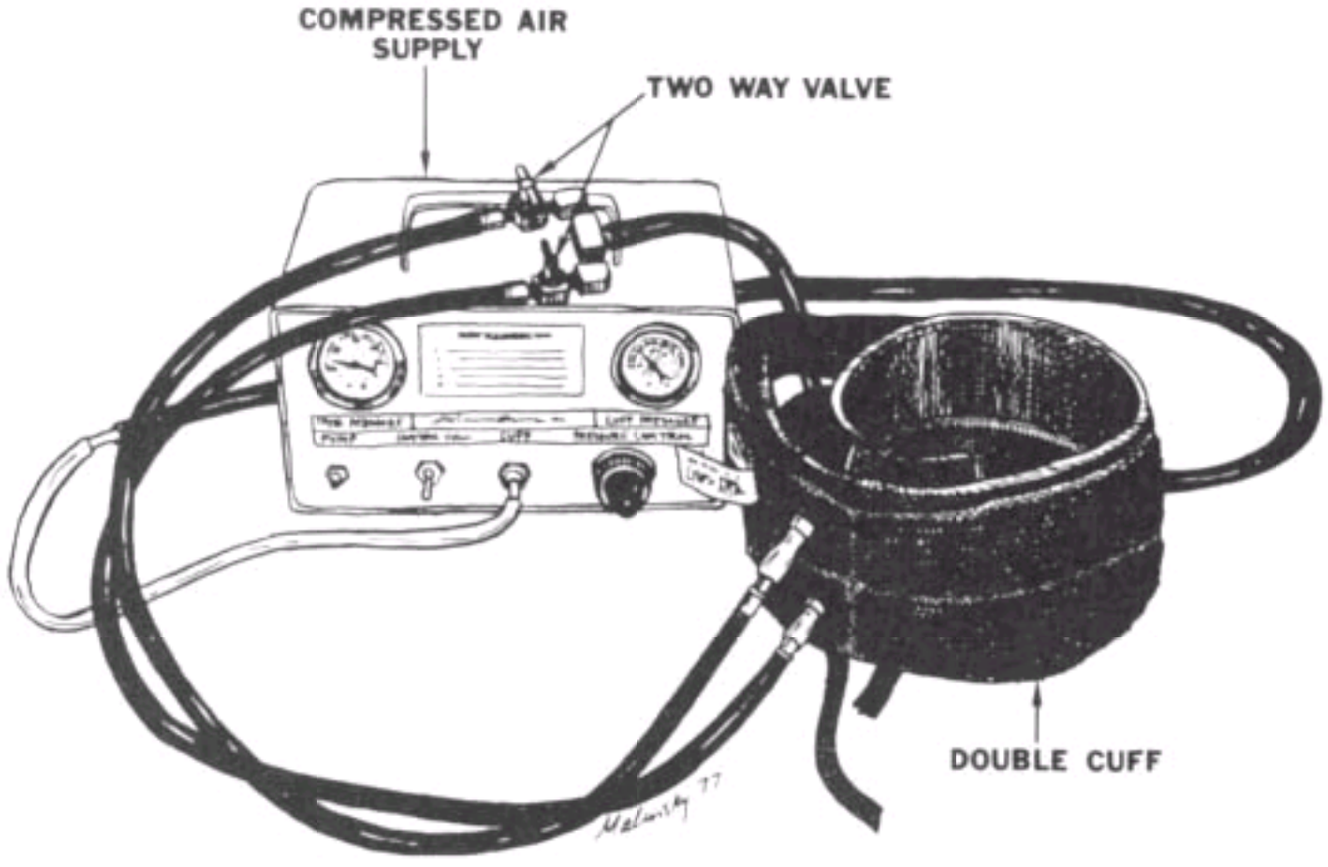


Figure 33-3 Infusion cannula securely taped in the dorsum of hand. A butterfly needle is shown here, but a plastic catheter with the hub attached may also be used. *(From Roberts JR: Intravenous regional anesthesia. JACEP 6:263, 1977.)*

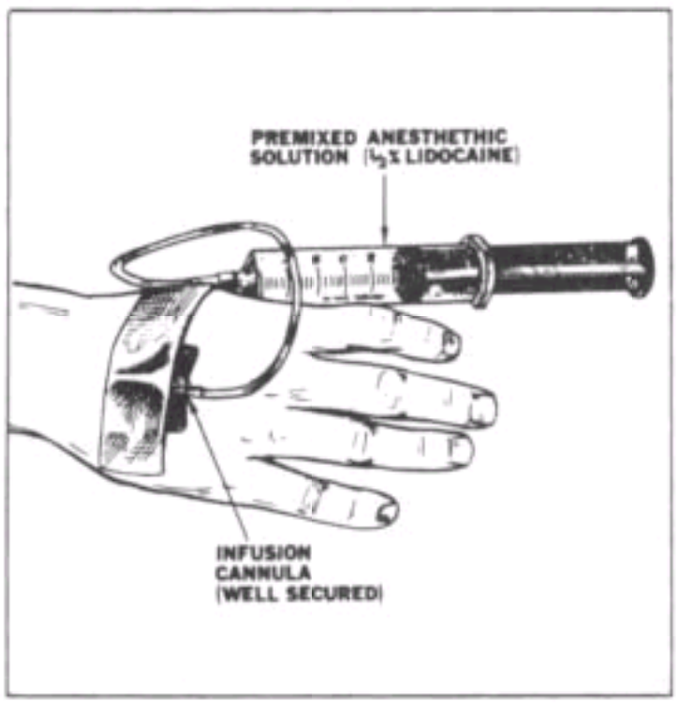


Figure 33-4 Exsanguination by elevation and elastic bandage. The tourniquet has yet to be inflated at this point. Care must be taken not to dislodge the infusion cannula. *(From Roberts JR: Intravenous regional anesthesia. JACEP 6:263, 1977.)*

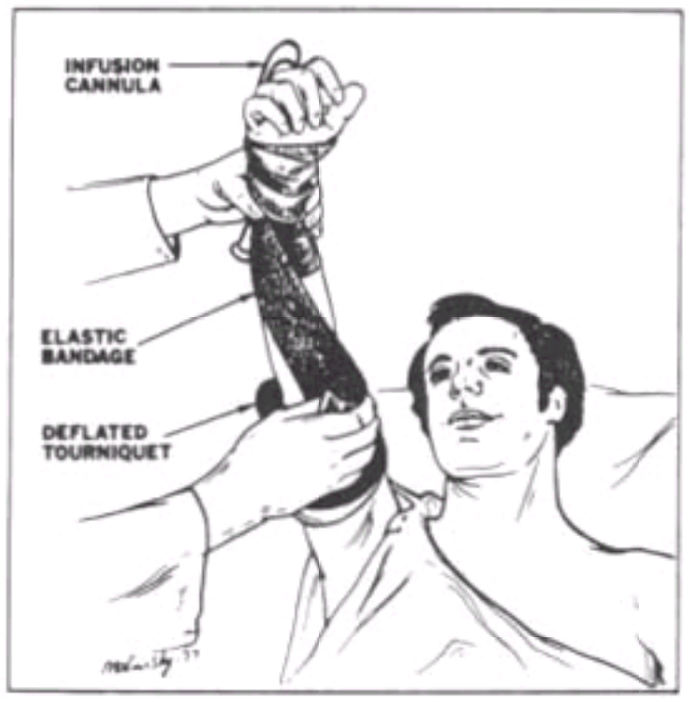


Figure 33-5 A cast is applied under anesthesia. Because the tourniquet is portable, postreduction radiographs may be obtained without losing anesthesia. *(From Roberts JR: Intravenous regional anesthesia. JACEP 6:263, 1977.)*

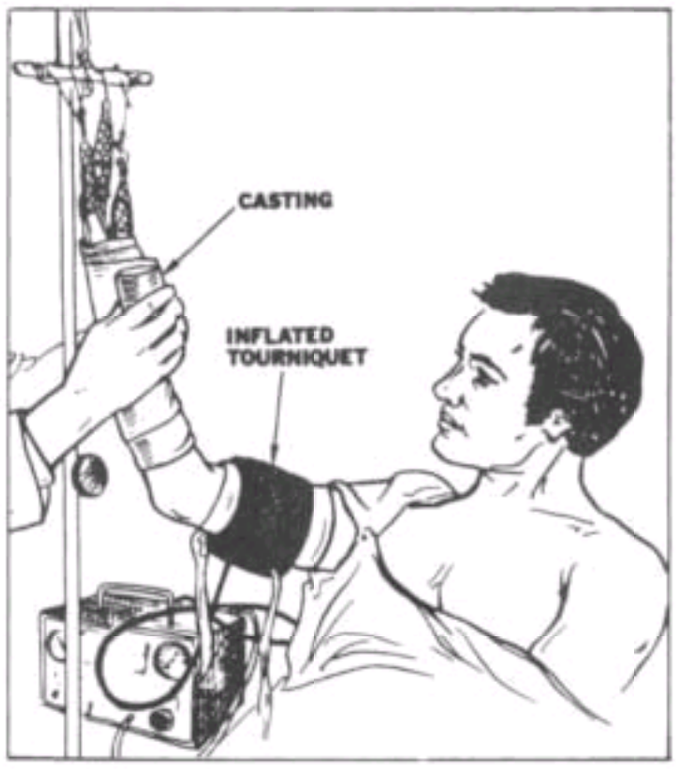


Figure 34-1 A child undergoing repair of a laceration while dissociated with ketamine. The blank stare is typical.

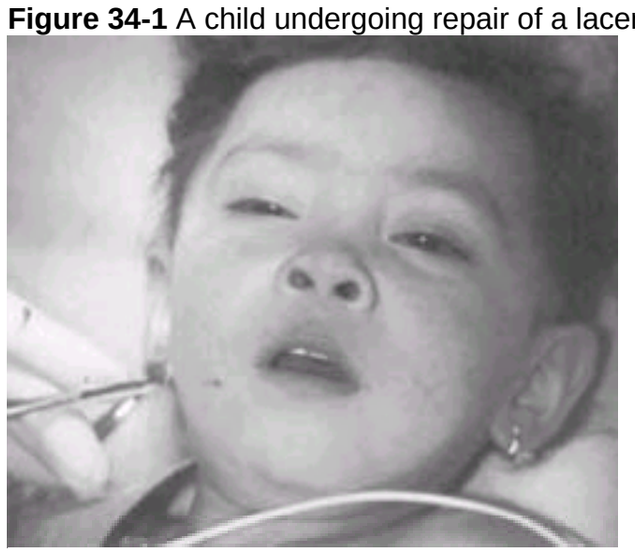
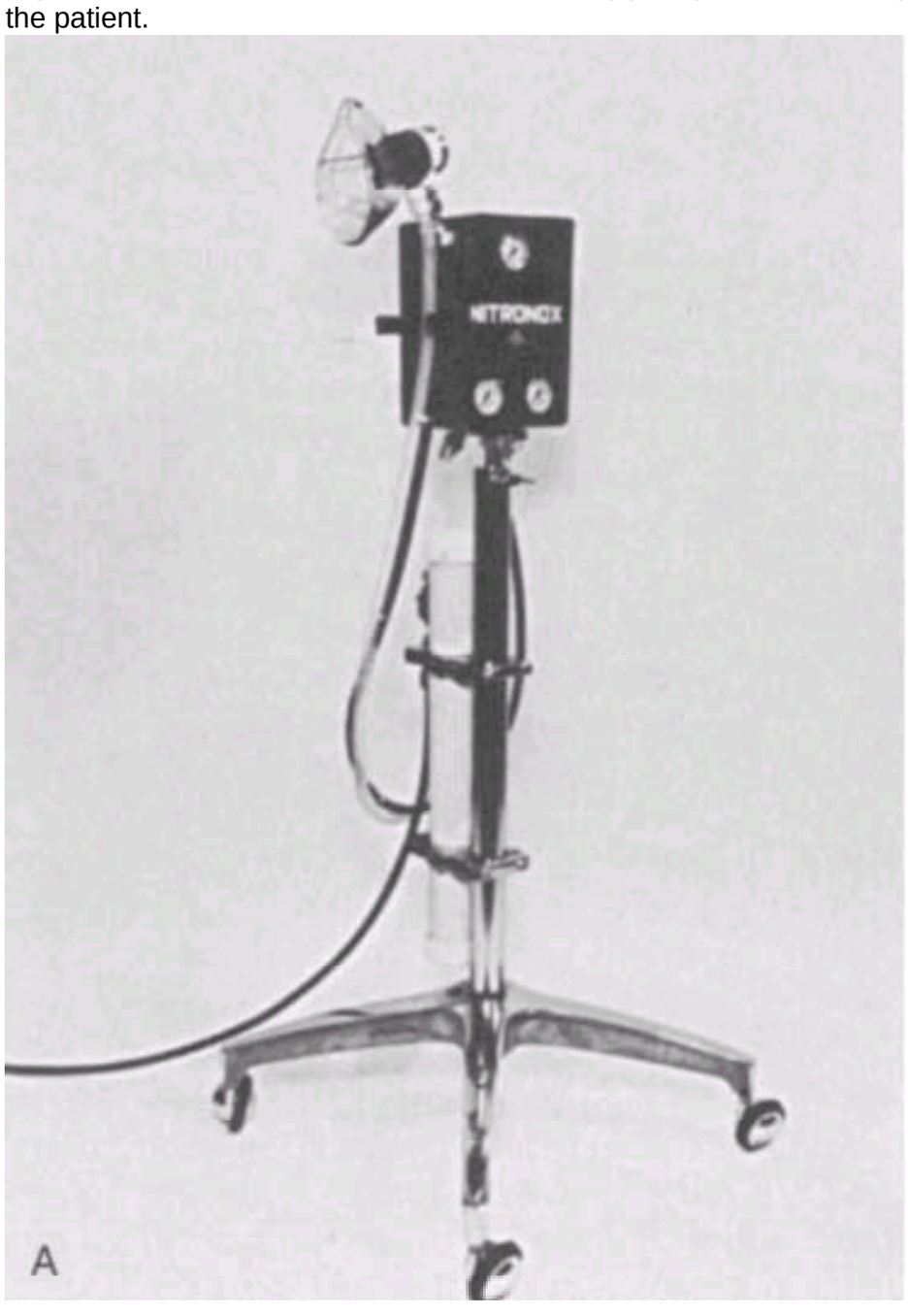


Figure 34-2 *A*, A demand-flow nitrous oxide/oxygen system. *B*, Example of a face mask for a nitrous oxide system. The mask must be held in contact with the face by the patient.



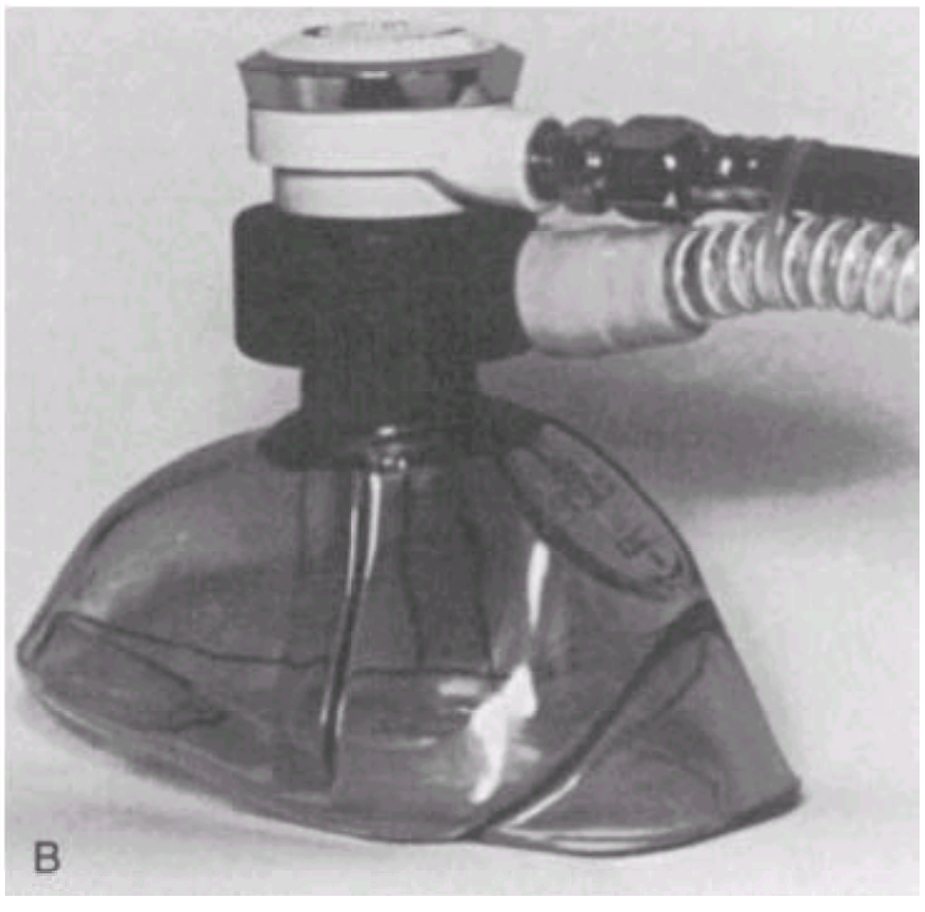


Figure 34-4 The downward spiral of fatal cocaine-induced delirium starts long before the chronic cocaine user is brought to the hospital, but physical restraints applied by police, security guards, and medical personnel only aggravate the situation. When it takes leather restraints and a bevy of personnel to subdue a delirious, violent, and totally out-of-control patient, it's time for aggressive and effective parenteral sedation and evaluation for and rapid correction of hyperthermia and acidosis. In the scenario shown here, it is impossible to even begin to adequately evaluate or treat this patient. The sagacious clinician knows, however, that this individual is an acute medical emergency.

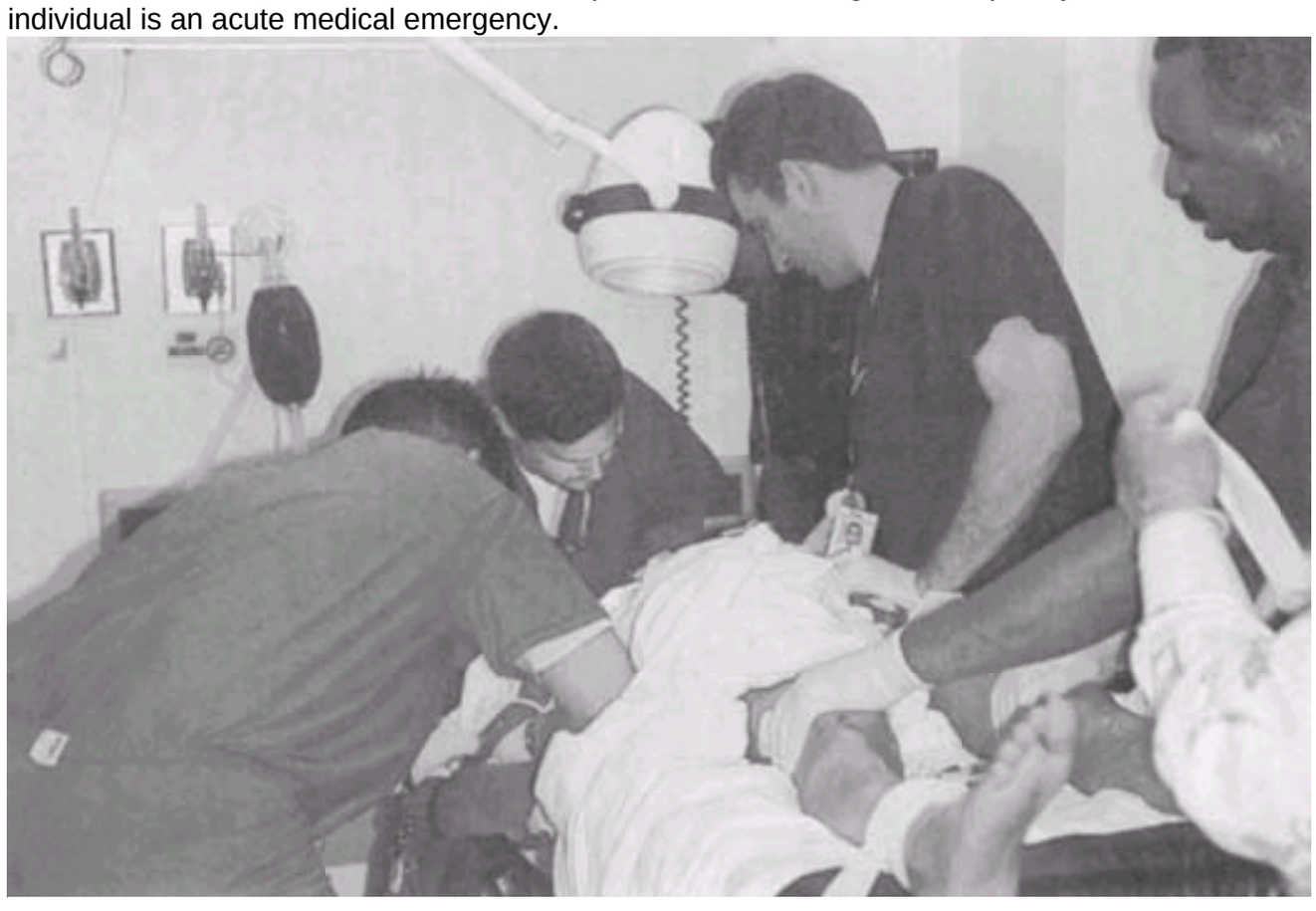


Figure 34-5 The postulated downward spiral of agitated delirium in a cocaine-intoxicated patient. An adverse outcome may theoretically be ameliorated with the aggressive use of chemical restraint.

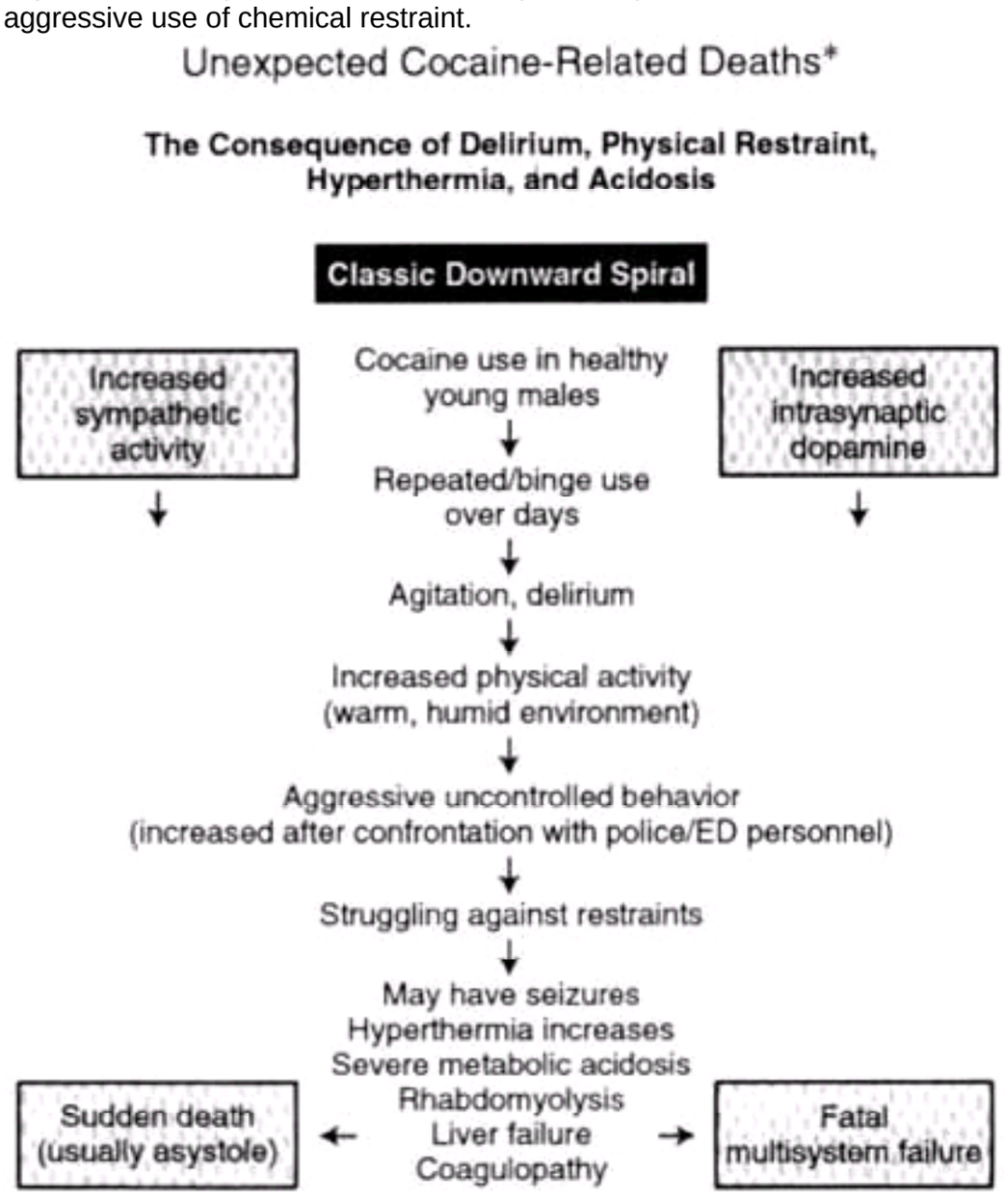


Figure 34-6 When an indwelling venous catheter cannot be obtained in the violent patient, the clinician may mainline the appropriate medications. By enlisting the help of security personnel, the arm or foot is immobilized by brute force, and medication is given by bolus injection into a large anticubital (*A*) or saphenous vein (*B*). Alternatively, two security guards lie across the upper thighs and lower stomach, and the clinician injects directly into the femoral vein (being careful to avoid an intra-arterial injection).

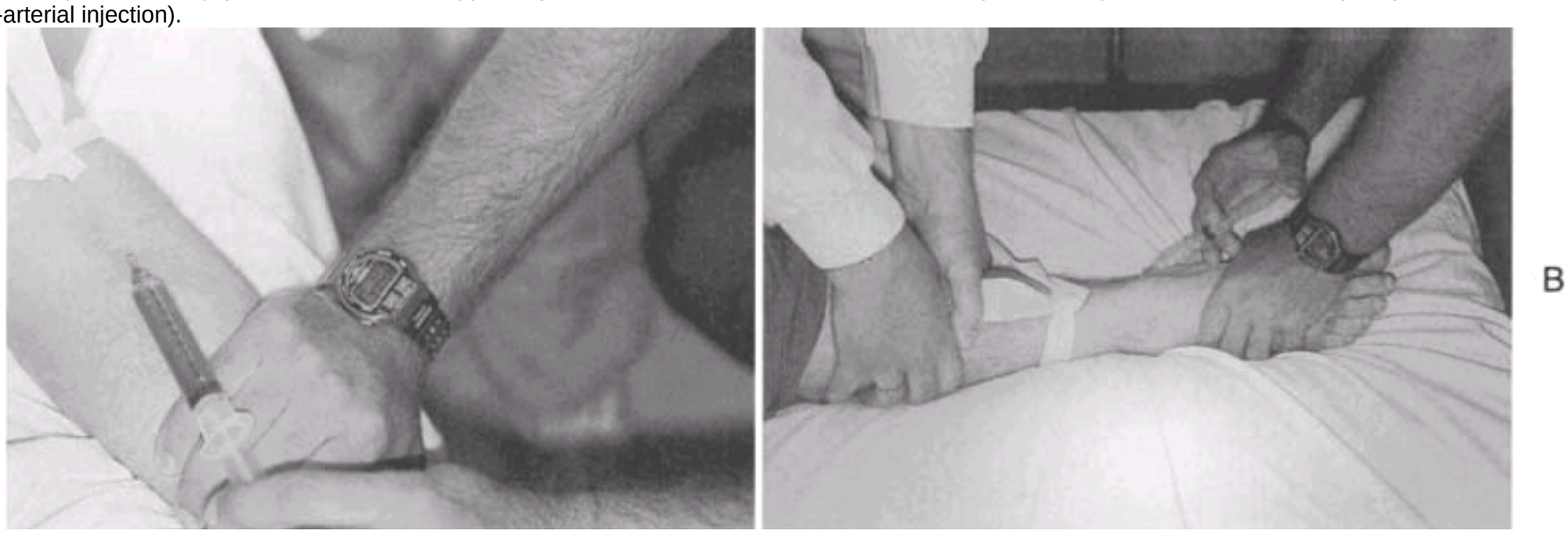


Figure 35-1 Graphic representation of the various phases of wound healing. Note that the tensile strength of scar tissue never reaches that of unwounded skin. Displayed values of tensile strength are approximate and demonstrate the general concept of wound healing.

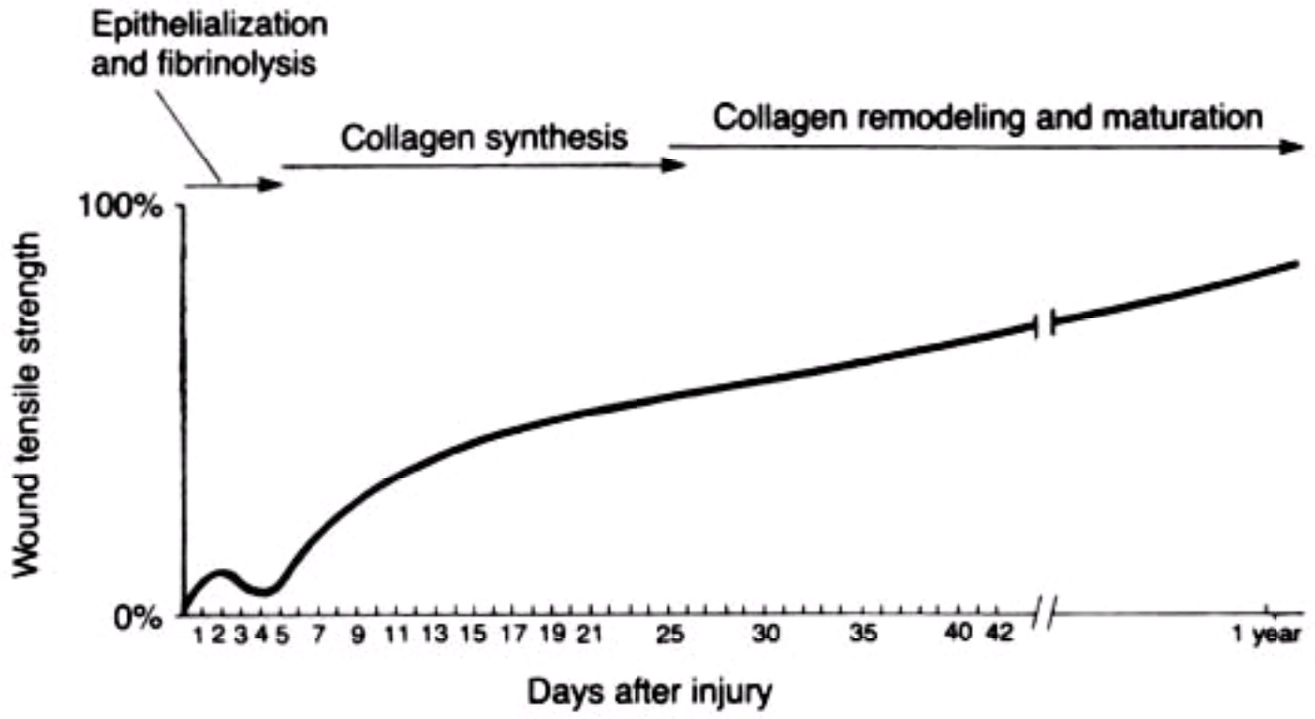


Figure 35-2 This laceration illustrates that there is no specific time frame (the so-called golden period) during which a laceration must be closed, or else relegated to an unsightly scar or a revision months later. *A*, This woman was punched in the face, suffered a laceration of the cheek, and presented to the ED 36 hours later. The wound was not infected, had contracted, and was beginning to heal by granulation. Under local anesthesia the wound was opened, irrigated, minimally debrided, and the skin edges were trimmed. *B*, Using a No. 15 blade, a 1-mm skin edge was incised. *C*, The trimmed edges were then cut by scissors. *D*, The wound was undermined to relieve tension on the skin. *E*, The wound was closed with 6-0 interrupted sutures that were removed in 5 days. No antibiotics were used and only a small linear scar resulted.

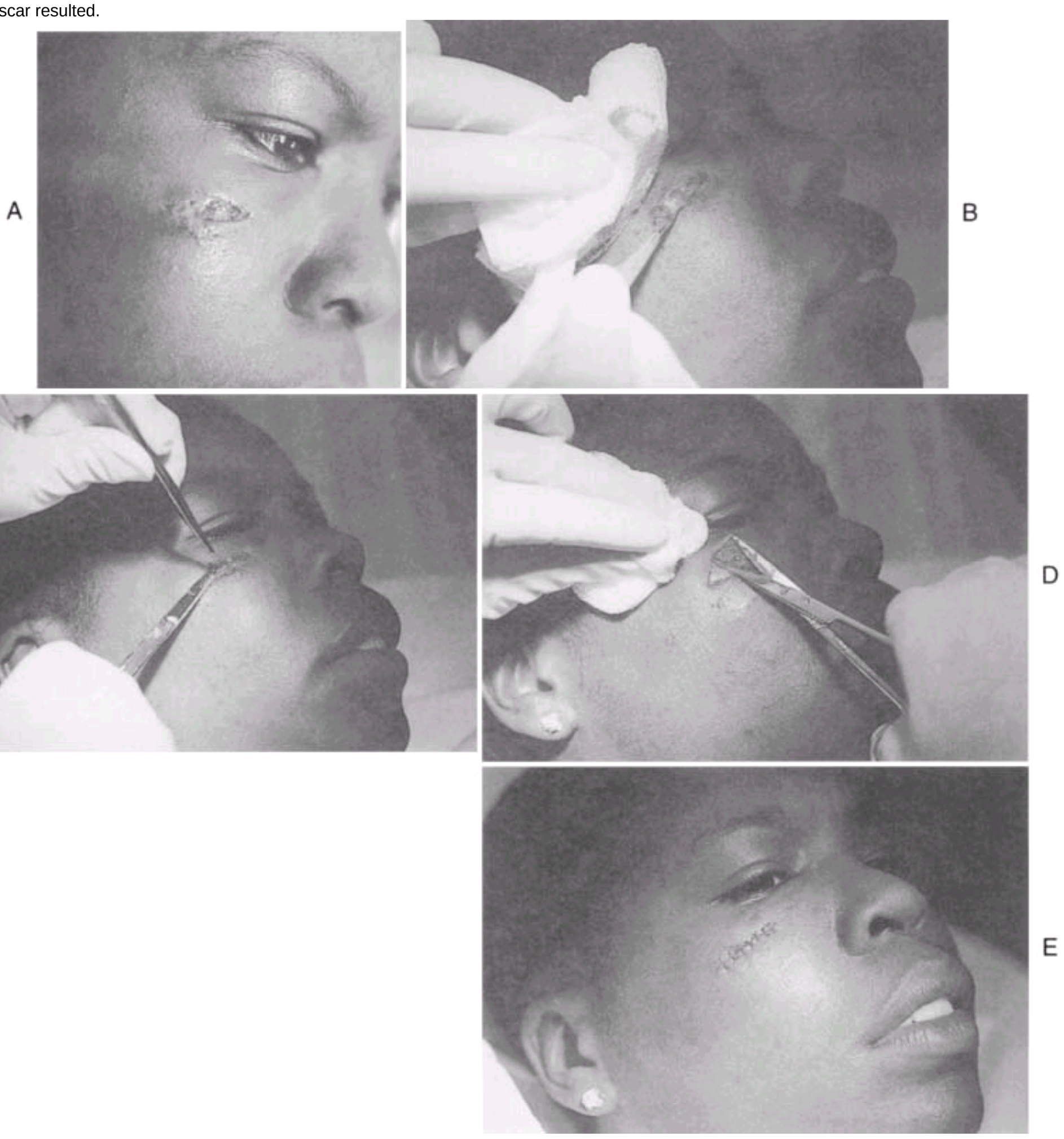


Figure 35-3 What's wrong with this picture? The patient is sitting up during laceration repair. Shortly after this picture was taken the patient fainted and nearly fell off the chair. Even the bravest patients should be *supine* for surgical procedures.

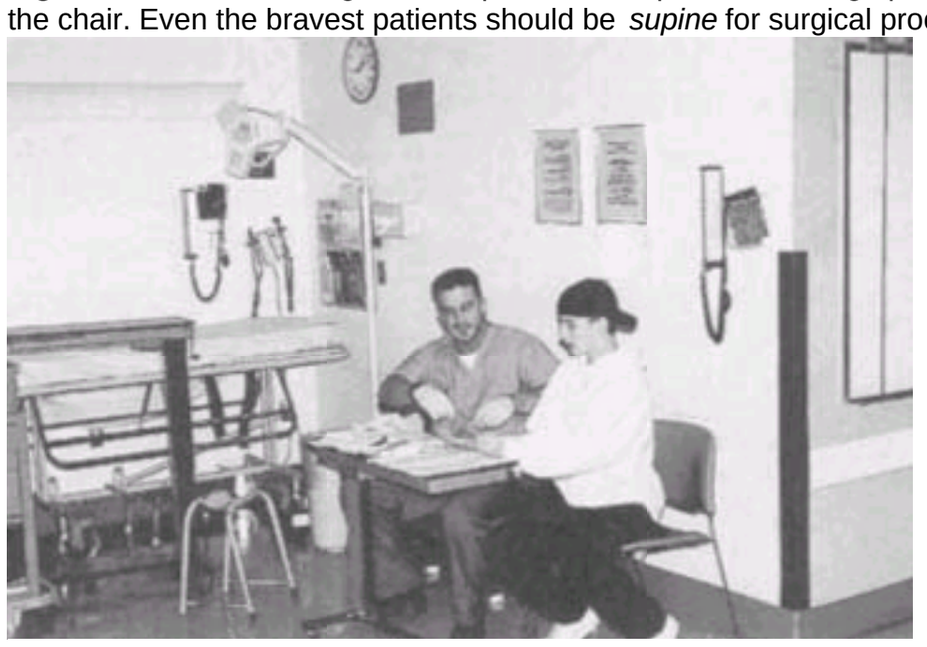


Figure 35-4 An easy method of high-pressure irrigation. IV tubing with an in-line, one-way valve is attached to a bottle of sterile 0.9% saline solution. The other end is connected to a stopcock. Saline solution is aspirated into the syringe. Maximal force is exerted on the plunger of the syringe, delivering the solution in a fine stream through an attached 19-ga needle held close to the wound. A splash shield may be used instead of a needle (see Fig. 35-6).

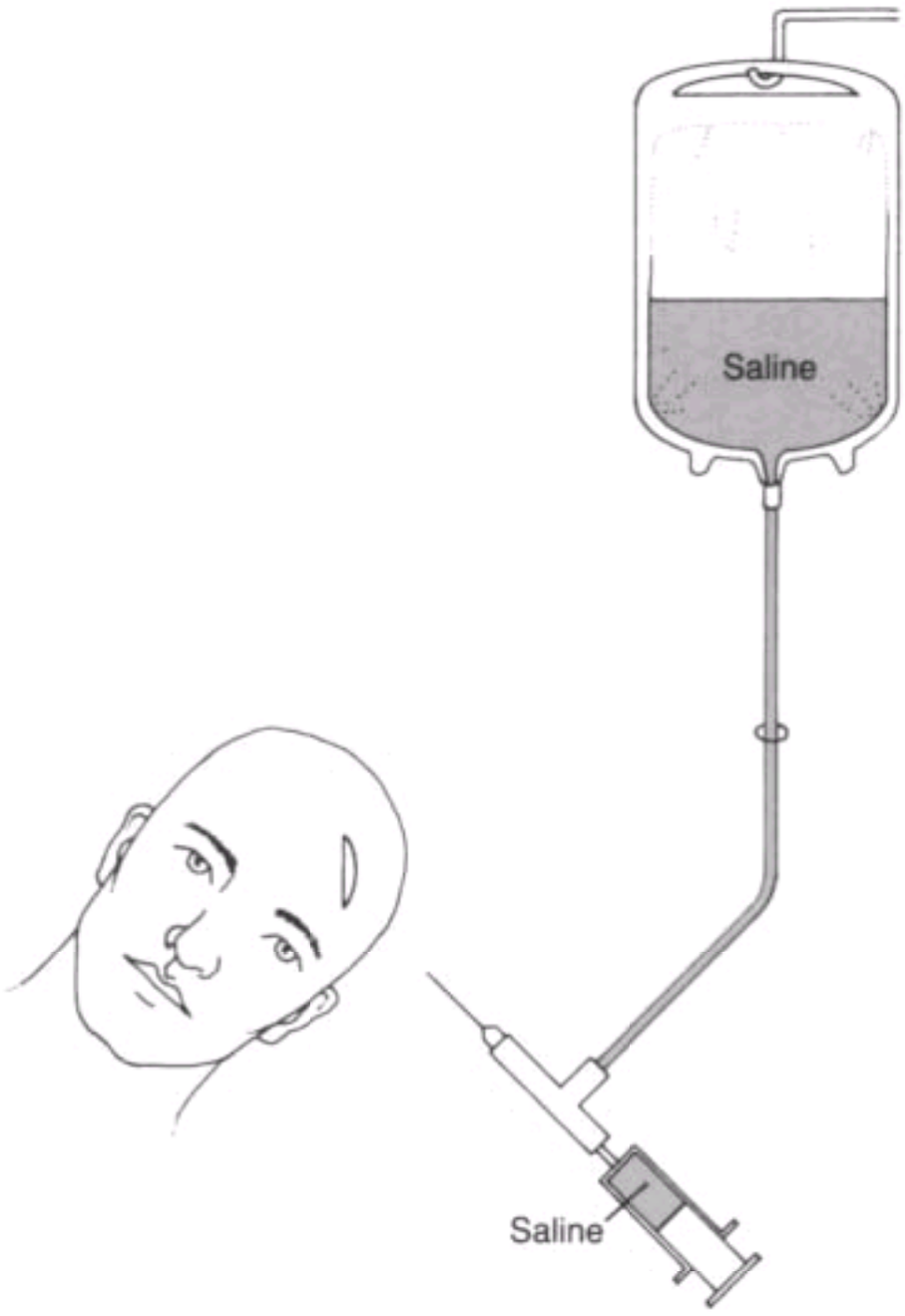


Figure 35-5 A spring-loaded irrigation device with ring handles speeds the somewhat tedious process of wound irrigation. *(Courtesy of Canyons International, Inc., Salt Lake City, UT.)*

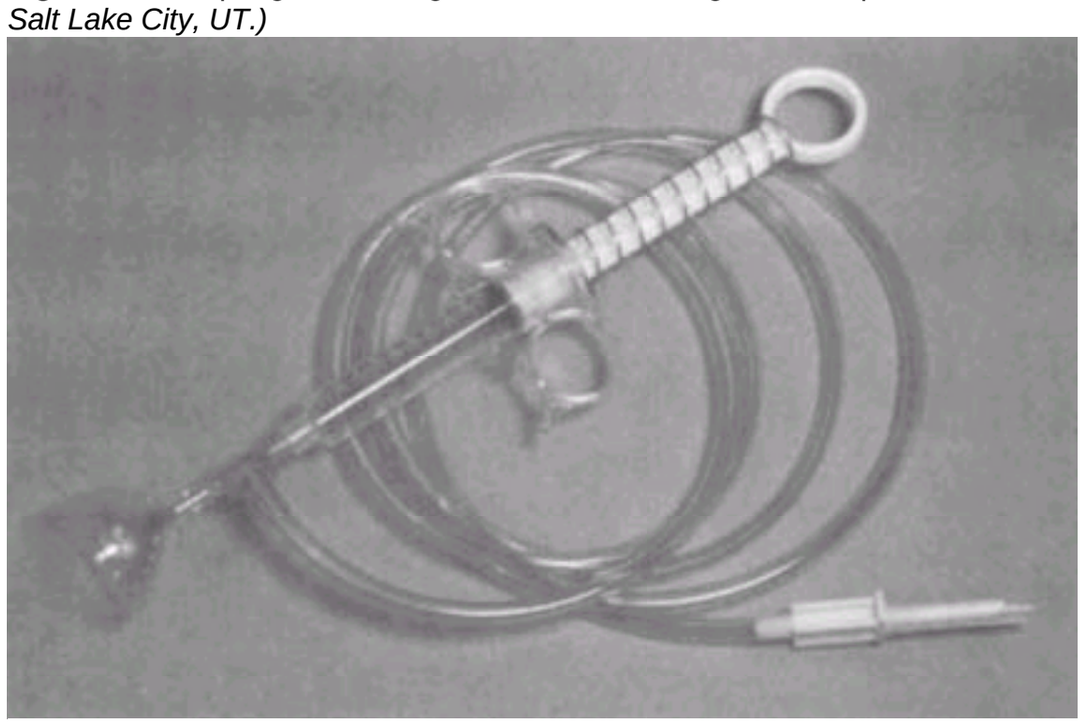
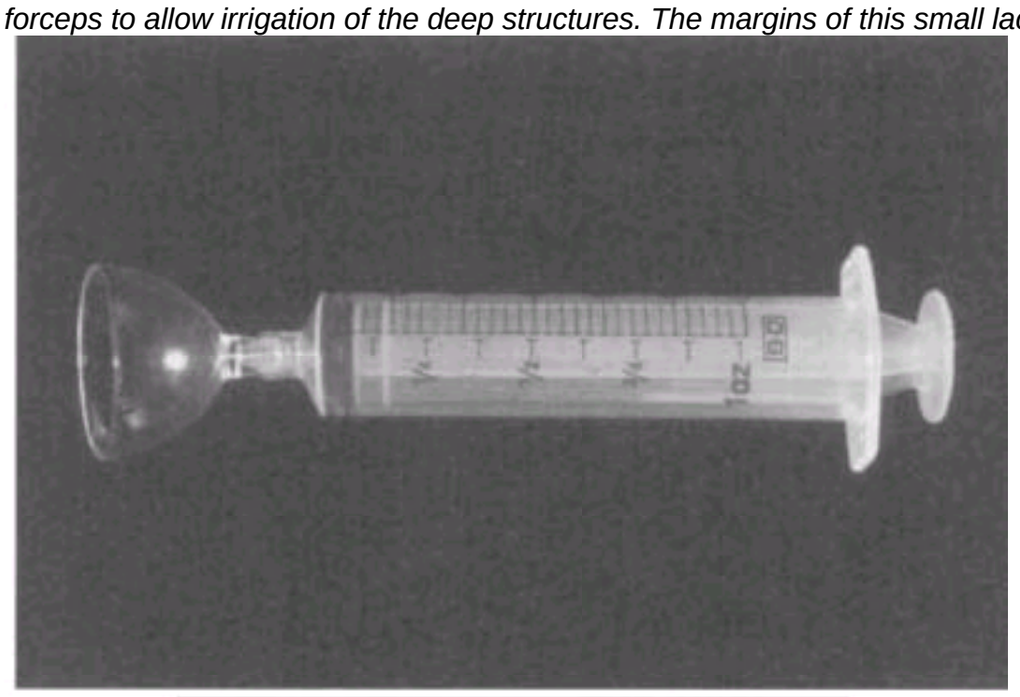


Figure 35-6 *A*, ZeroWet Splashield attached to end of a syringe. This device is used in lieu of a needle. The shield is held near the skin, and the tip of the syringe directs the irrigating solution. *B*, This protective shield allows forceful irrigation without splatter of infectious fluids. *Note that the clinician is holding open the laceration with forceps to allow irrigation of the deep structures. The margins of this small laceration were also extended to allow for better irrigation.*

A



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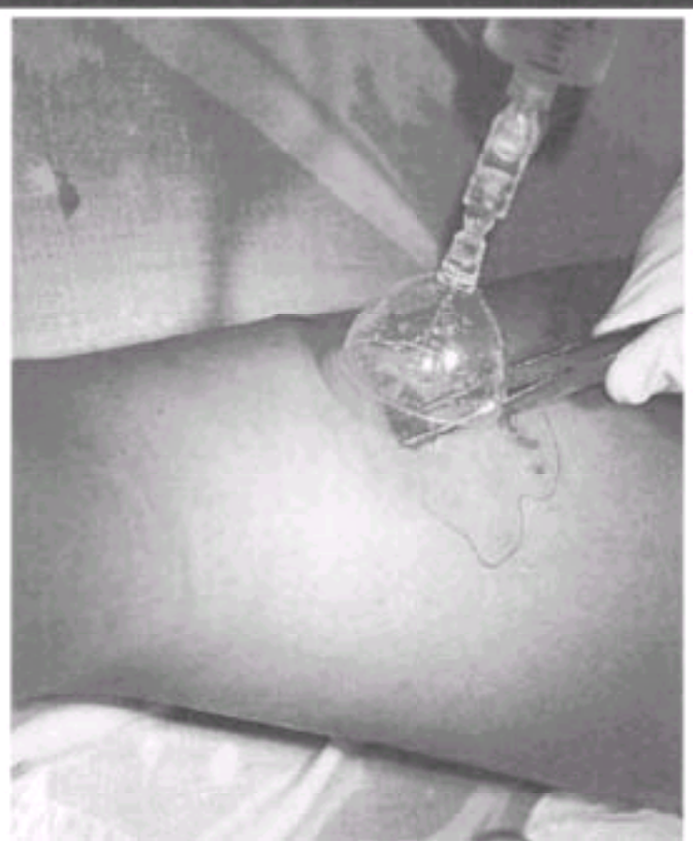


Figure 35-7 A sterile glove on the hand with the finger cut out, instead of an annoying drape, and a finger tourniquet to provide a bloodless field make examination and suturing of a wound easier.

Figure 35-8 *Do not* probe a wound with a finger looking for foreign bodies! If the clinician's skin is broken, the patient's blood may introduce HIV, hepatitis, viruses, and other infectious agents. Betters ways to find a foreign body are obtaining radiographs and exploring wounds with metal probes. Extending the wound edges and performing the examination under a bloodless field and with good lighting will find most foreign bodies.

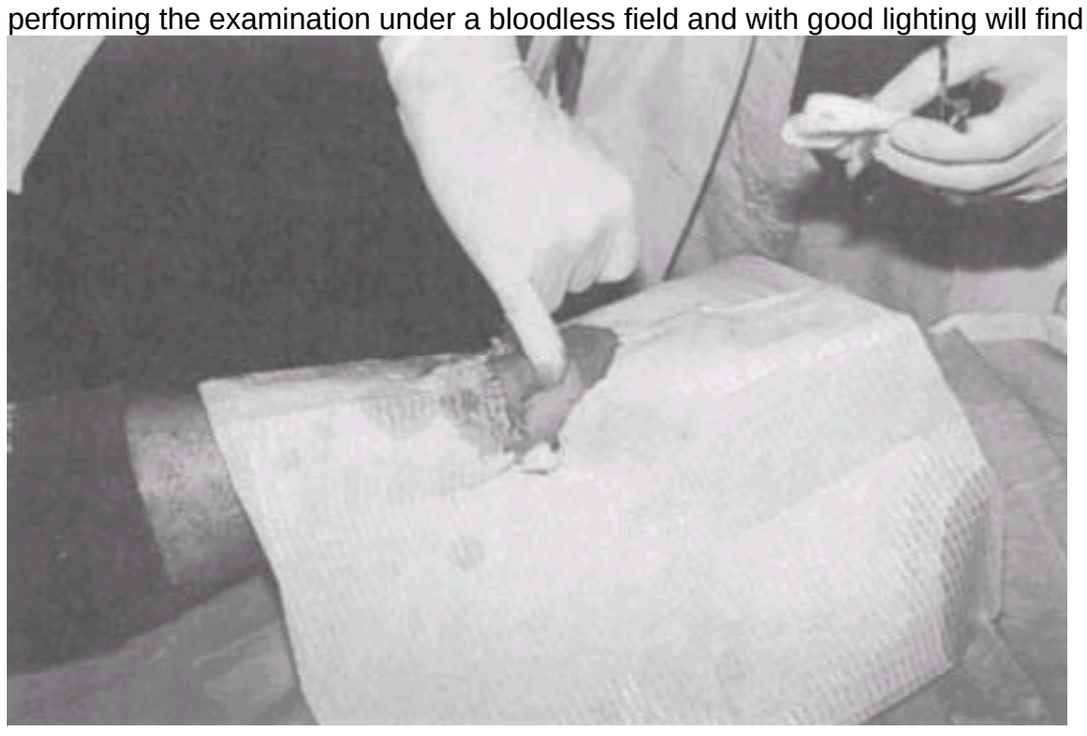


Figure 35-9 *A–C,* Complete excision of a wound. Grossly contaminated wounds may be excised and sutured primarily.

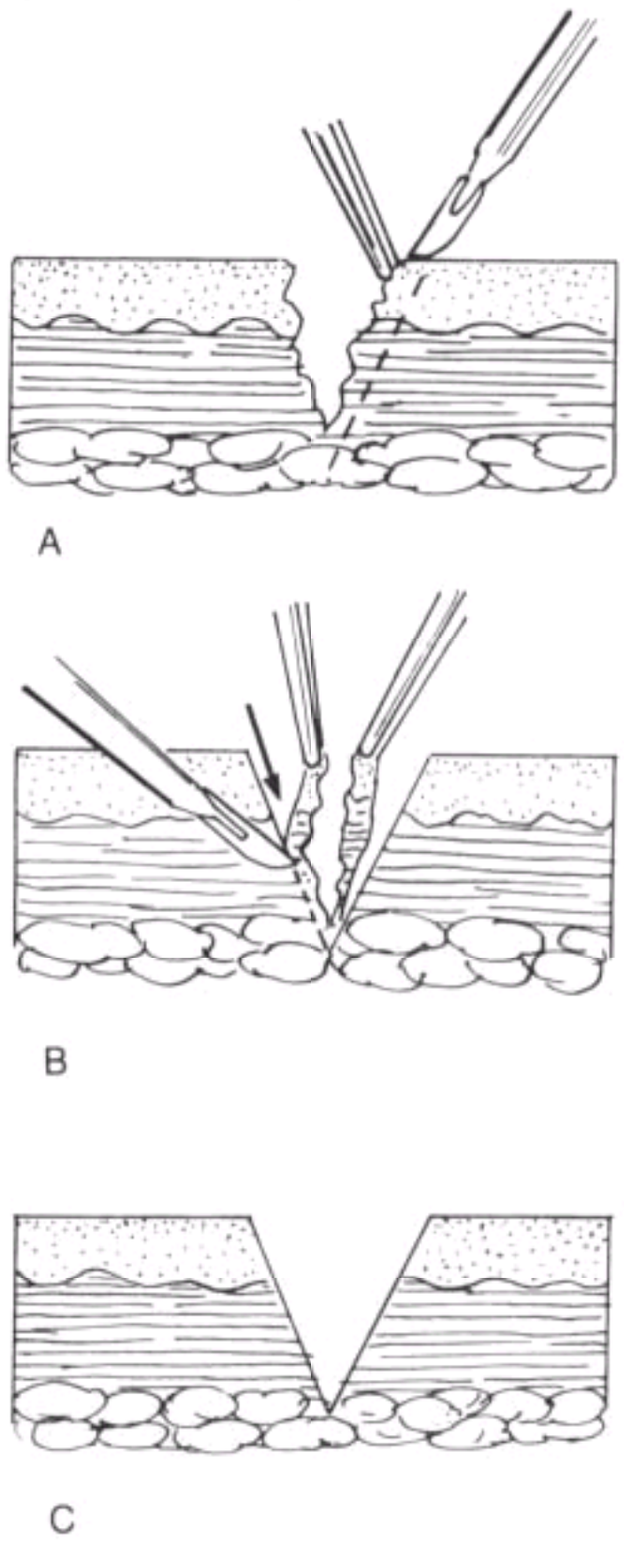


Figure 35-10 Excision through an eyebrow. Use an angled incision to remove tissue in the eyebrow, thus avoiding further injury of hair follicles. *(From Dushoff IM: A stitch in time. Emerg Med 5:2, 1973. Reproduced by permission.)*

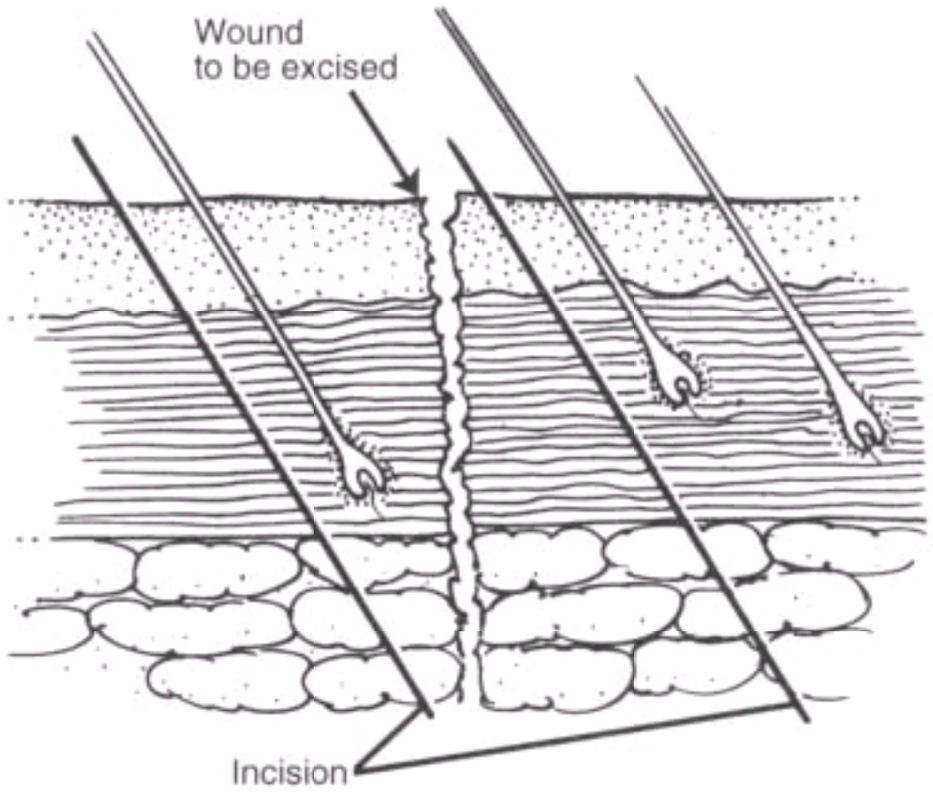


Figure 35-11 When one attempts to tie off a bleeding vessel, the tip of the hemostat should project beyond the clamped vessel.

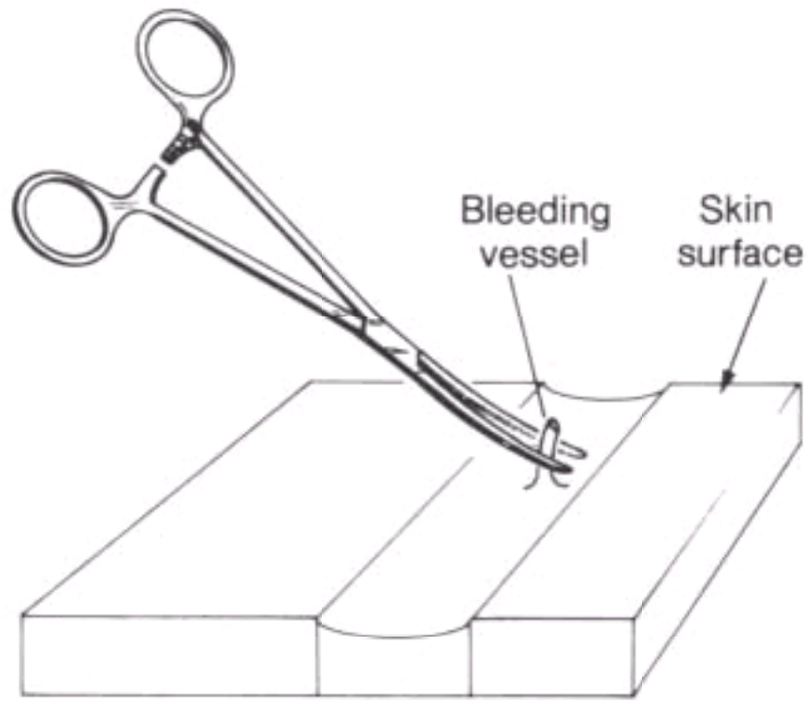
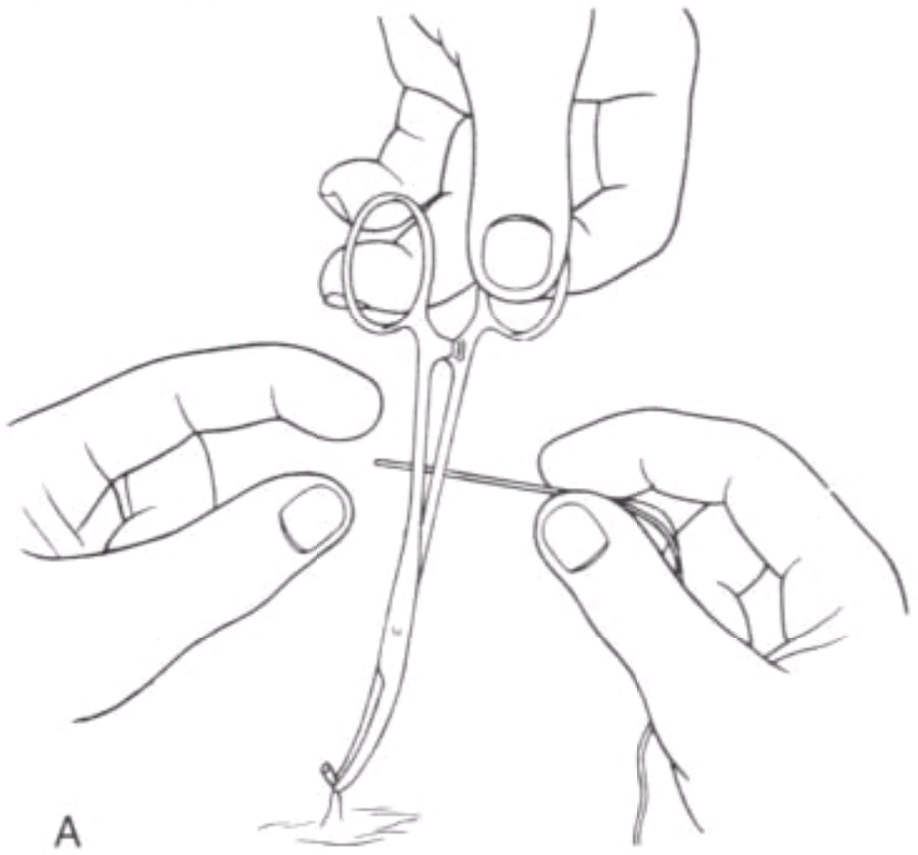


Figure 35-12 *A,* The handles of the hemostat are raised by an assistant as a ligature is passed under them. *B,* The ligature thread stretched between the index fingertips is carried under the projecting tips of the hemostat. *(Modified from Kirk RM: Basic Surgical Techniques. Edinburgh, Churchill Livingstone, 1978, p 50. Reproduced by permission.)*



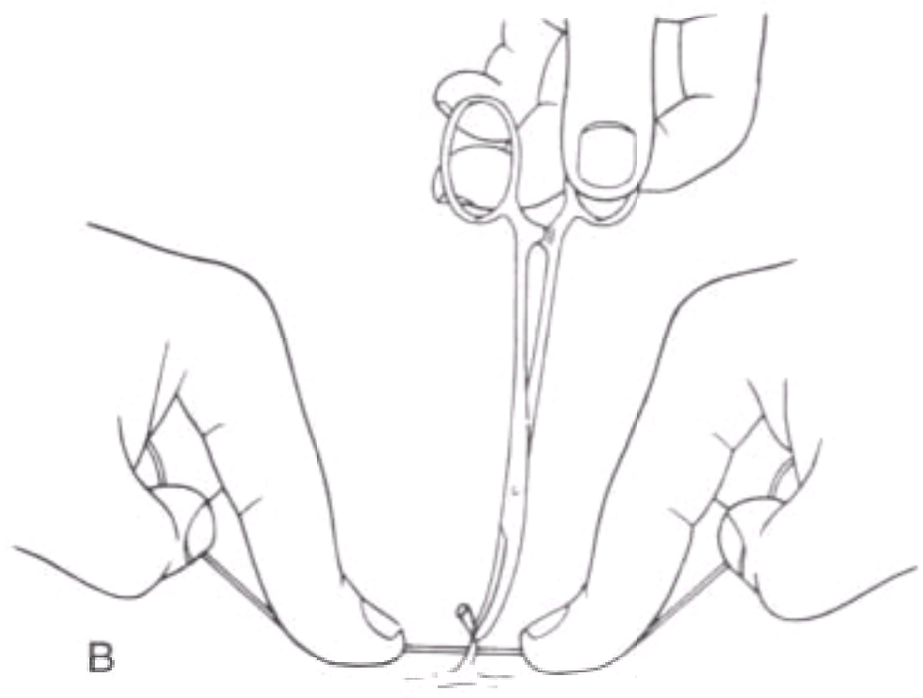


Figure 35-13 *A,* Ligature tension is maintained with one hand by grasping one tail of the suture with a clamp and keeping its base between the thumb and the forefinger. *B,* As the pedicle clamp is removed with the other hand, the ligature is tightened by extending the flexed thumb and index finger to the desired tension. Then the pedicle clamp is discarded from the right hand, and further knots are applied in the usual fashion. *(Modified from MacDonald RT: Maintenance of ligature tension by a single operator with simultaneous removal of a hemostatic clamp. Am J Surg 143:770, 1982. Reproduced by permission.)*

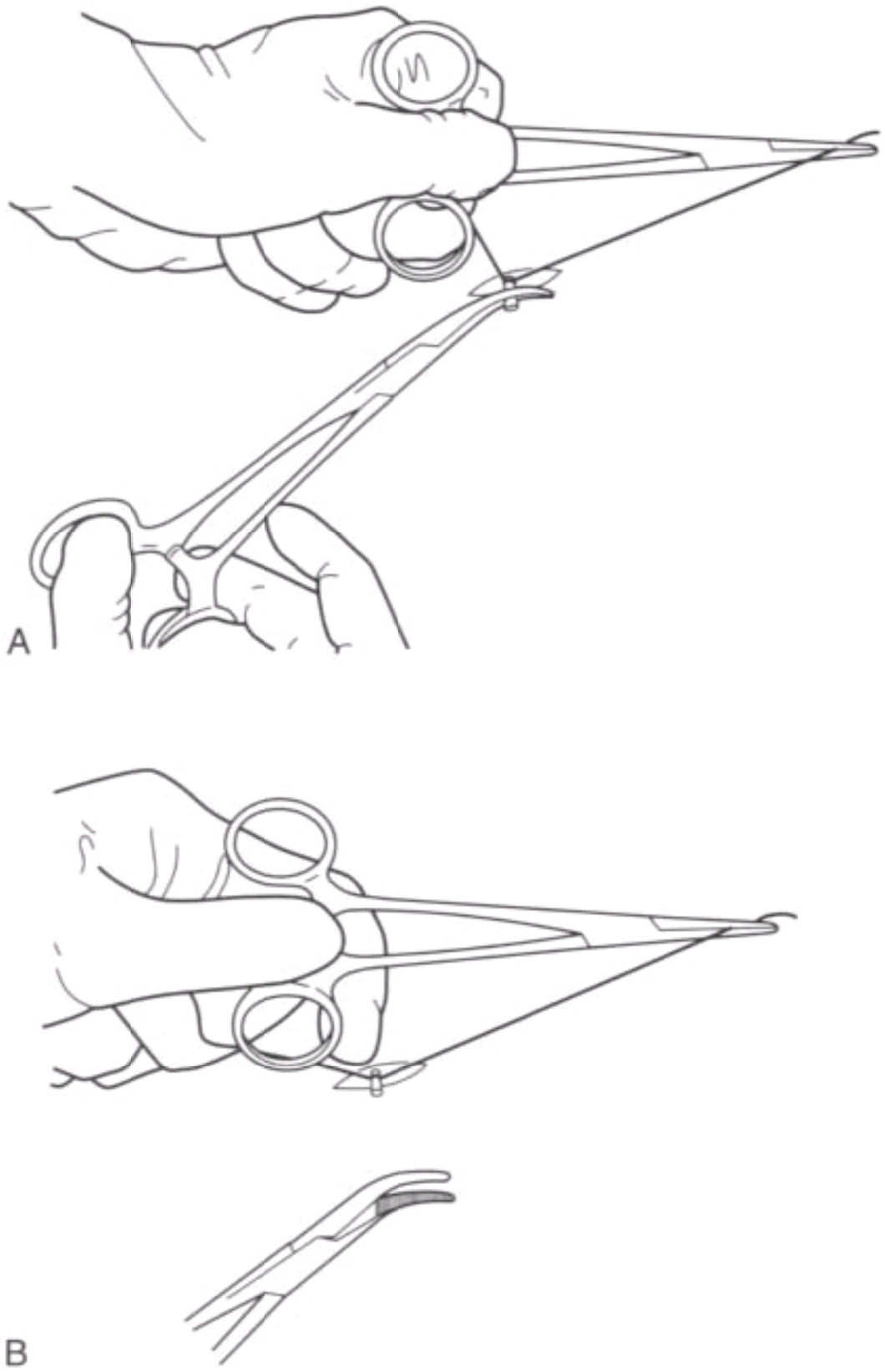


Figure 35-14 Ligation of a retracted, bleeding vessel. *A,* Horizontal mattress technique. *B,* Figure-of-eight technique.

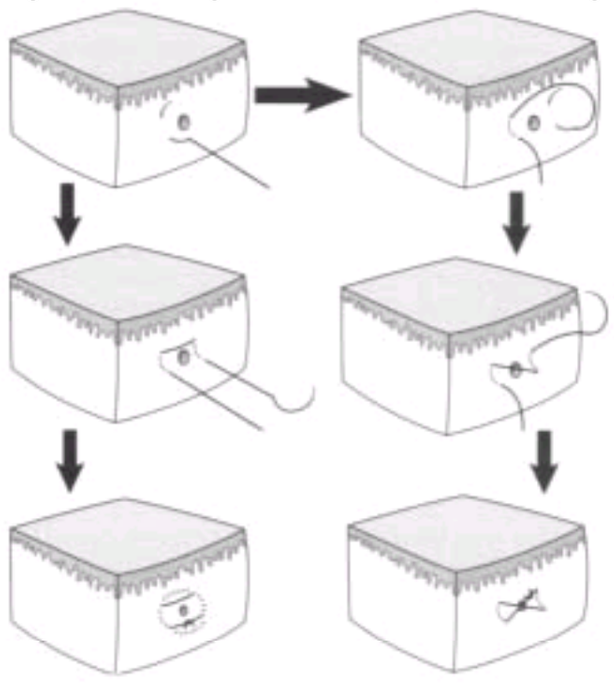


Figure 35-15 Battery-powered cautery.

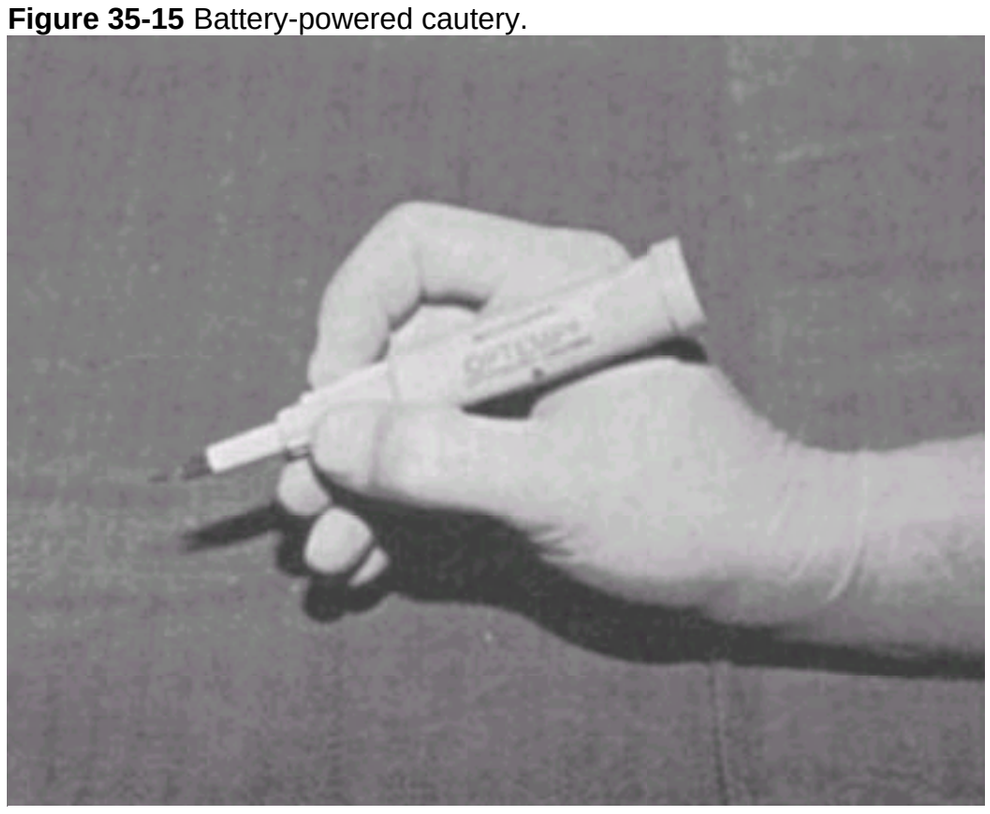


Figure 35-16 Use of Penrose drain for exsanguination *(A)* of a wounded digit. A second Penrose drain is applied *(B)* as a finger tourniquet, and the first drain is removed *(C)*. During actual patient care, the clinician would use sterile technique including gloves.

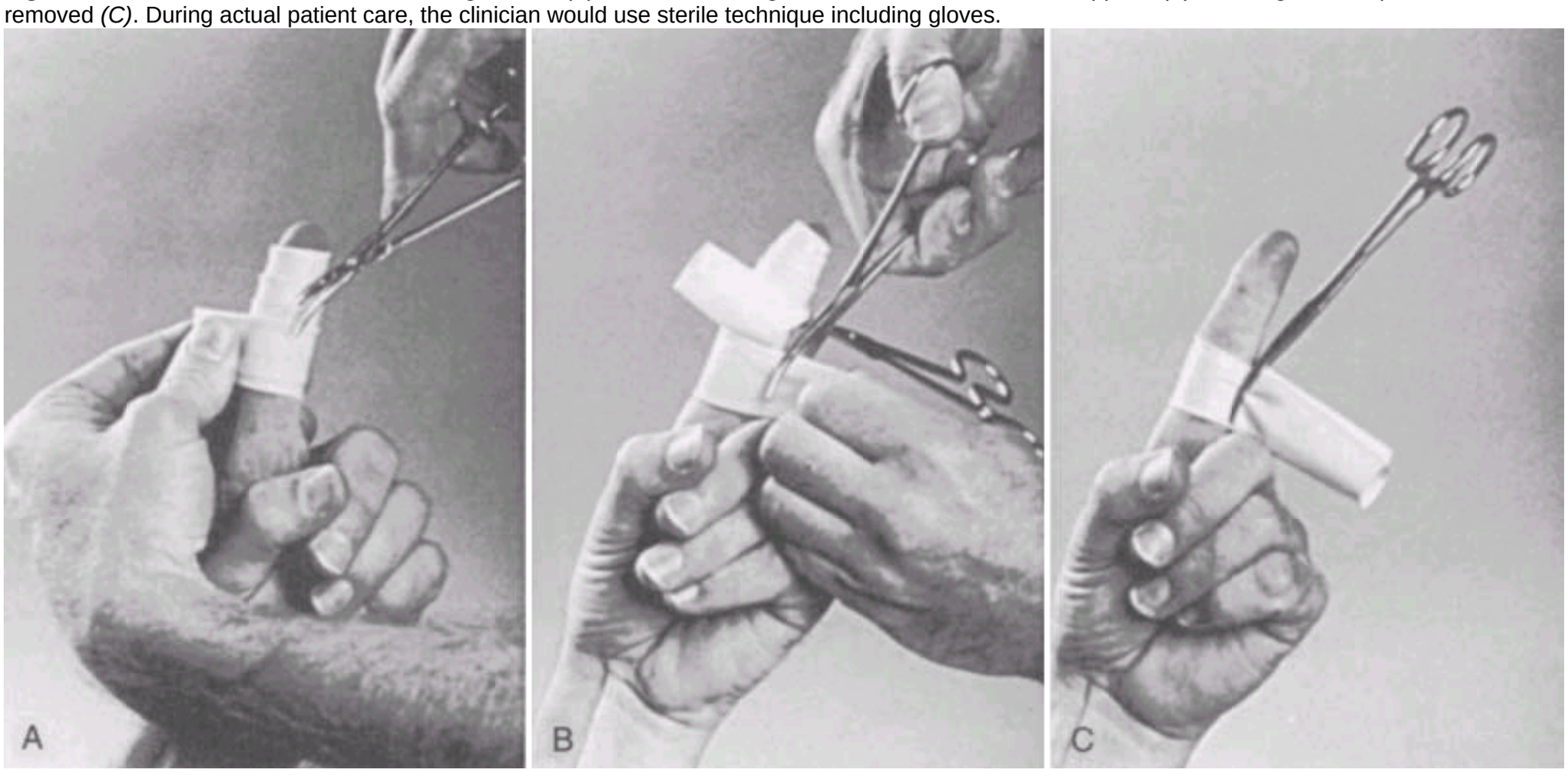


Figure 35-17 Use of a sterile glove to provide a clean field and serve as a finger tourniquet. The distal end of the glove is clipped *(A),* and the glove finger is rolled proximally over the digit *(B, C)*. During actual patient care, the clinician would use sterile technique including gloves.

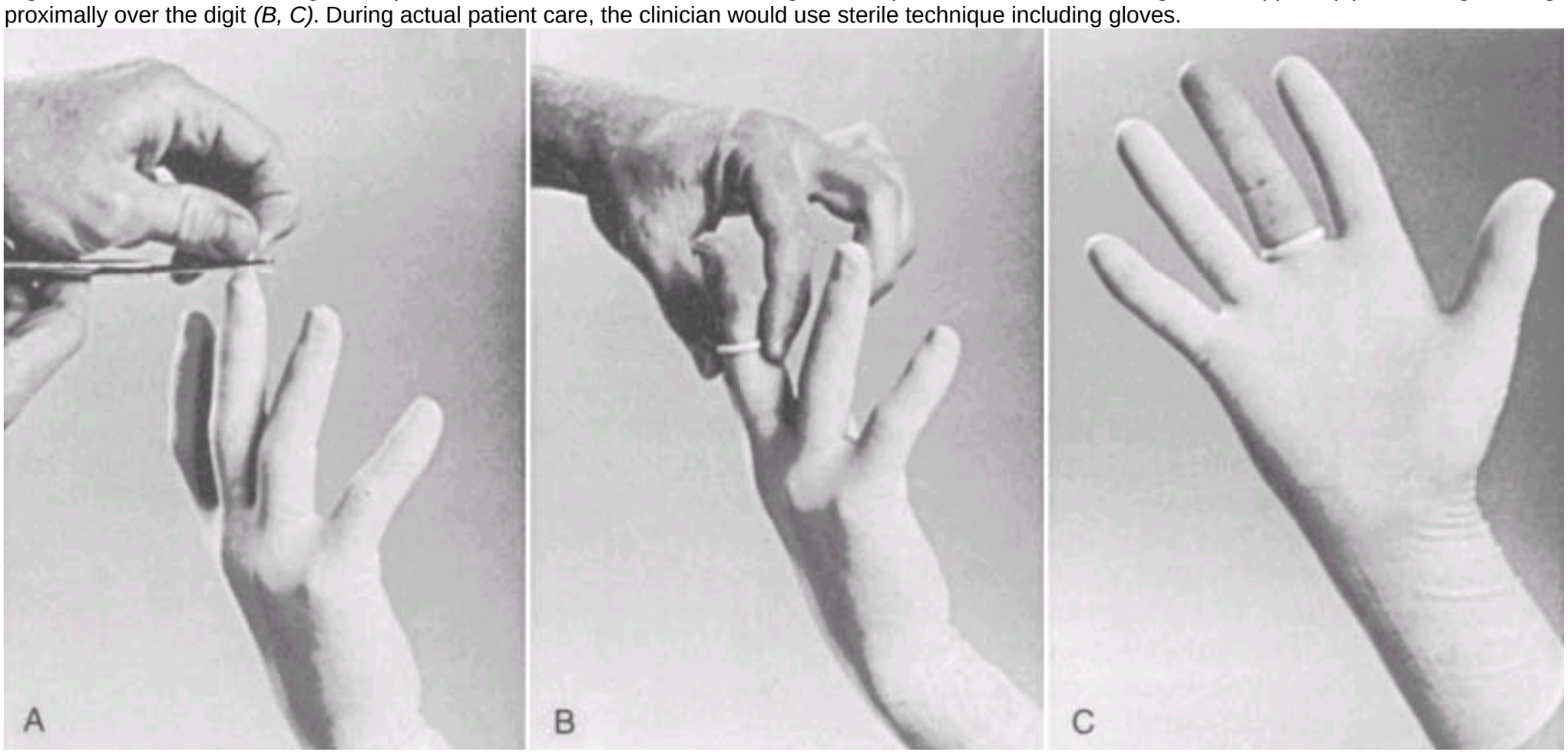


Figure 35-18 As in Figure 35-7 , a sterile glove has been used to provide a sterile field for the thumb. In addition, a commercial rubber ring tourniquet device has been applied to enhance hemostasis. The accompanying tag has been left exposed to remind the operator of the tourniquet device.

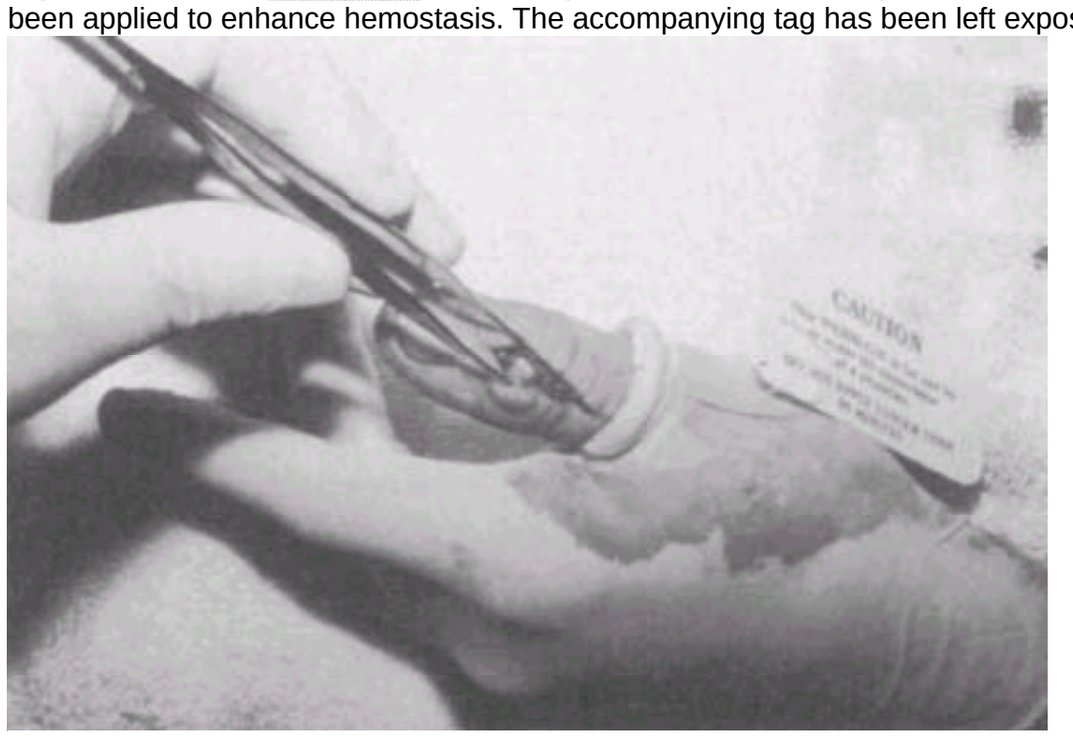


Figure 35-19 Incidence of wound infection over time when delayed closure is performed. Delayed closure is best accomplished on the fourth or fifth day to minimize the risk of infection. *(From Edlich RF, Thacker JG, Rodeheaver GT, et al: A Manual for Wound Closure. St. Paul, MN, 3M Medical Surgical Products, 1979. Reproduced by permission. © 1979 by Minnesota Mining and Manufacturing Company.)*

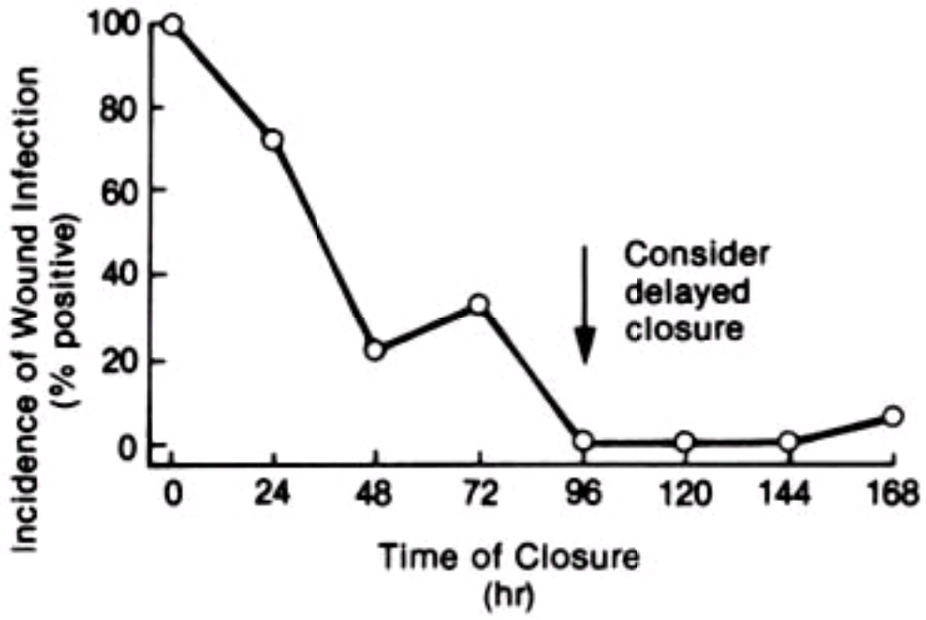
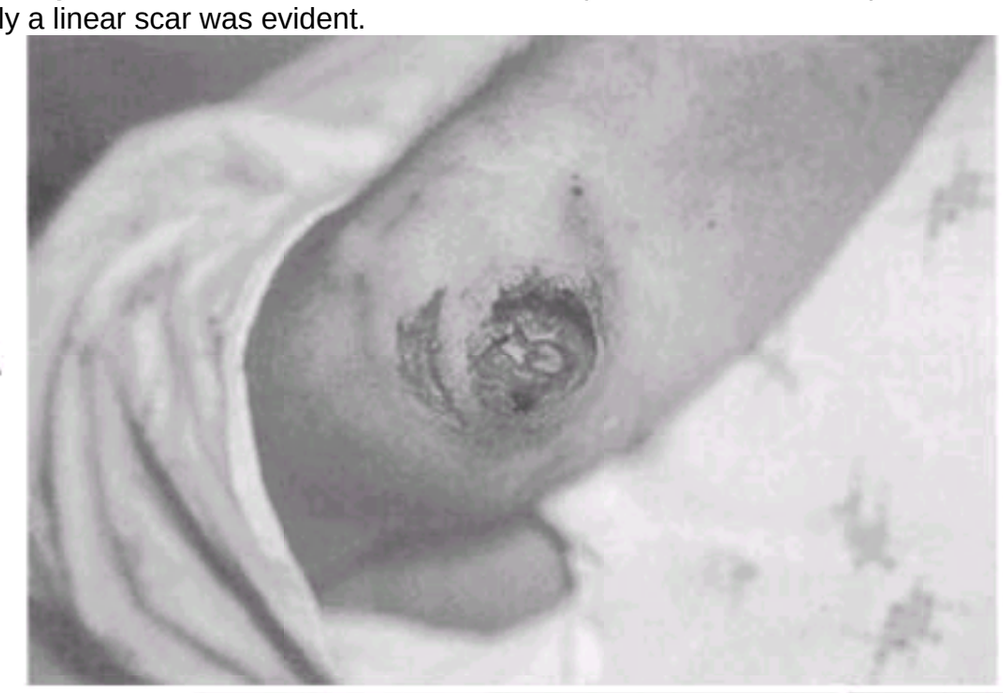
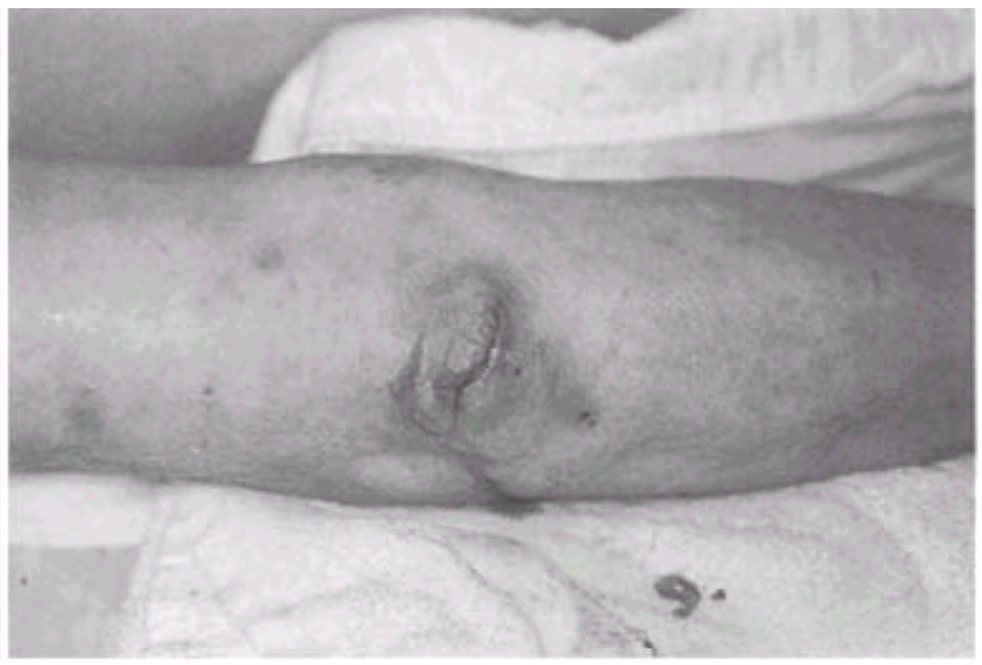


Figure 35-20 *A*, This dirty contused wound, now 18 hours old, is an ideal candidate for a delayed primary closure. *B*, At presentation the wound is anesthetized, scrubbed, irrigated, and debrided. *C*, The wound is packed with sterile gauze and covered by a dry dressing. No antibiotics were prescribed. *D*, Four days later the packing is removed, and the wound is minimally debrided. *E*, Interrupted sutures are placed as though this is a fresh, clean wound. At suture removal 10 days later, only a linear scar was evident.



A



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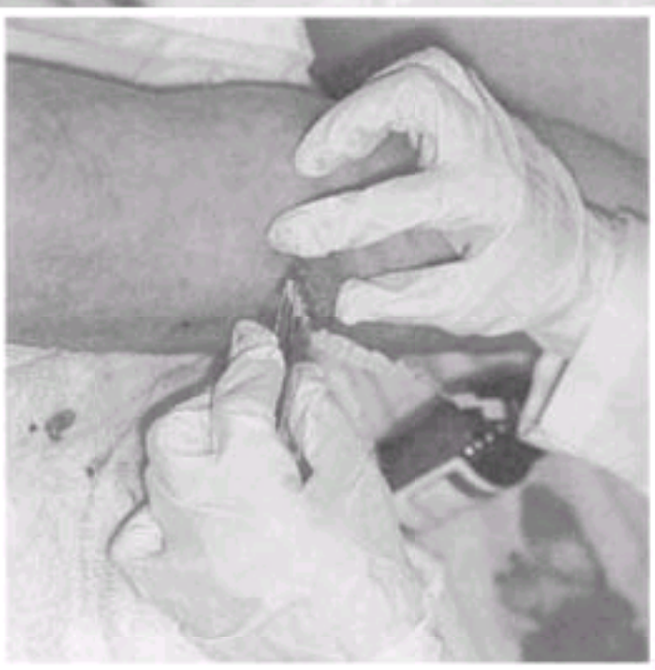

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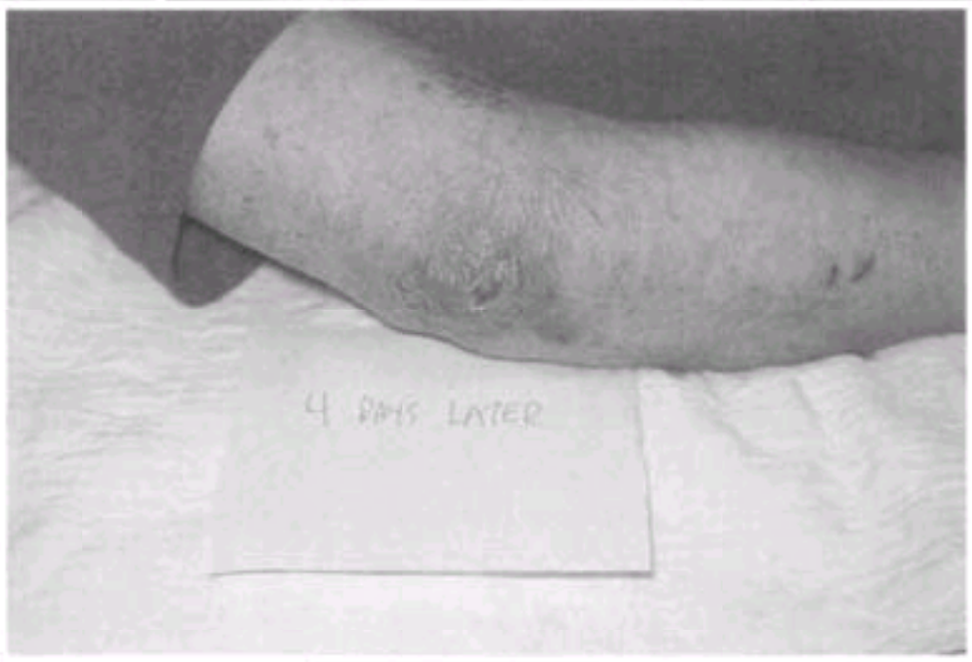

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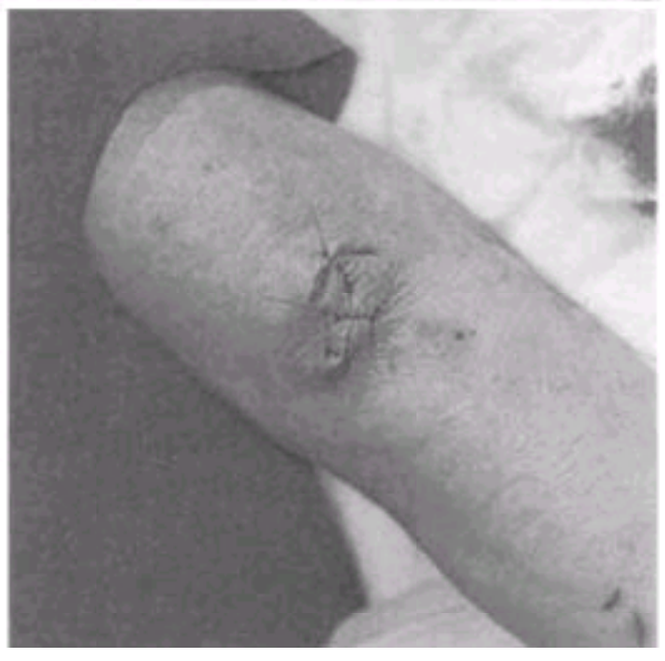


E

Figure 35-21 A common three-layer dressing, consisting of antibiotic ointment, Adaptic, and gauze.

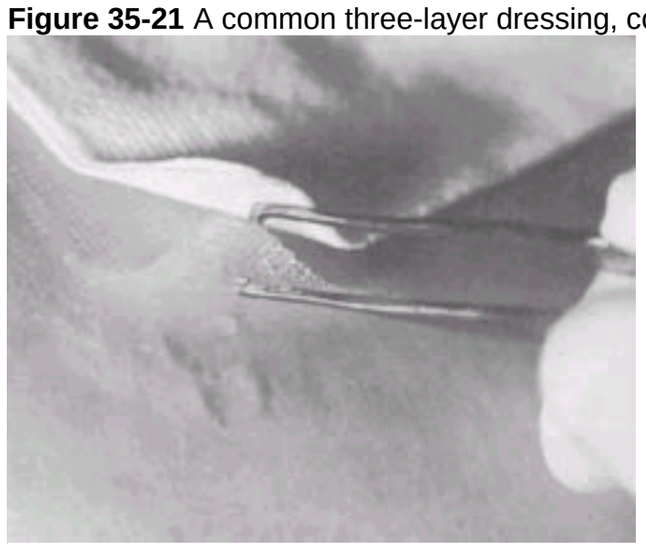


Figure 35-22 Snugness of the bandage is increased by 180° rotation of the bandage roll after each circular turn to create a reverse spiral. *(From Norton LW: Trauma. In Hill GJ II: Outpatient Surgery. Philadelphia, WB Saunders, 1980. Reproduced by permission.)*

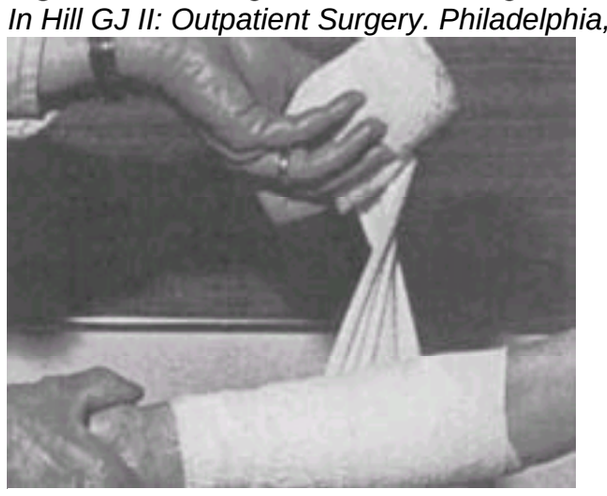


Figure 35-23 Finger dressing. *A,* The inner layer is nonadherent gauze or whatever is required for soft tissue care. The middle layer is 2 × 2 in. gauze sponges wrapped circumferentially and held in place with a small strip of tape. *B,* Begin No. 2 tube gauze at the base of the finger. It is useful to hold this end with one finger while the tube gauze applicator is pulled toward the fingertip. A twisting motion firms the wrap about the digit; generally about 90° is necessary. Excessive stretch or twisting can compromise circulation. *C,* When the fingertip is reached, make a 360° twist. *D,* Pass the applicator toward the finger base with an additional 90° twist. Repeat once more; thus, three layers are in place. *E,* Cut enough gauze to reach the base of the finger, and tape it there. As an alternative, pull the final layer beyond the tip, leaving it long enough to reach to and around the wrist (about three times the finger length). Split this gauze into two strands; bring them dorsally to the wrist, knot, and loosely wrap around the wrist. *(Redrawn from Kaplan EN, Hentz VR: Emergency Management of Skin and Soft Tissue Wounds: An Illustrated Guide. Boston, Little, Brown, 1984, p 86. Reproduced by permission.)*

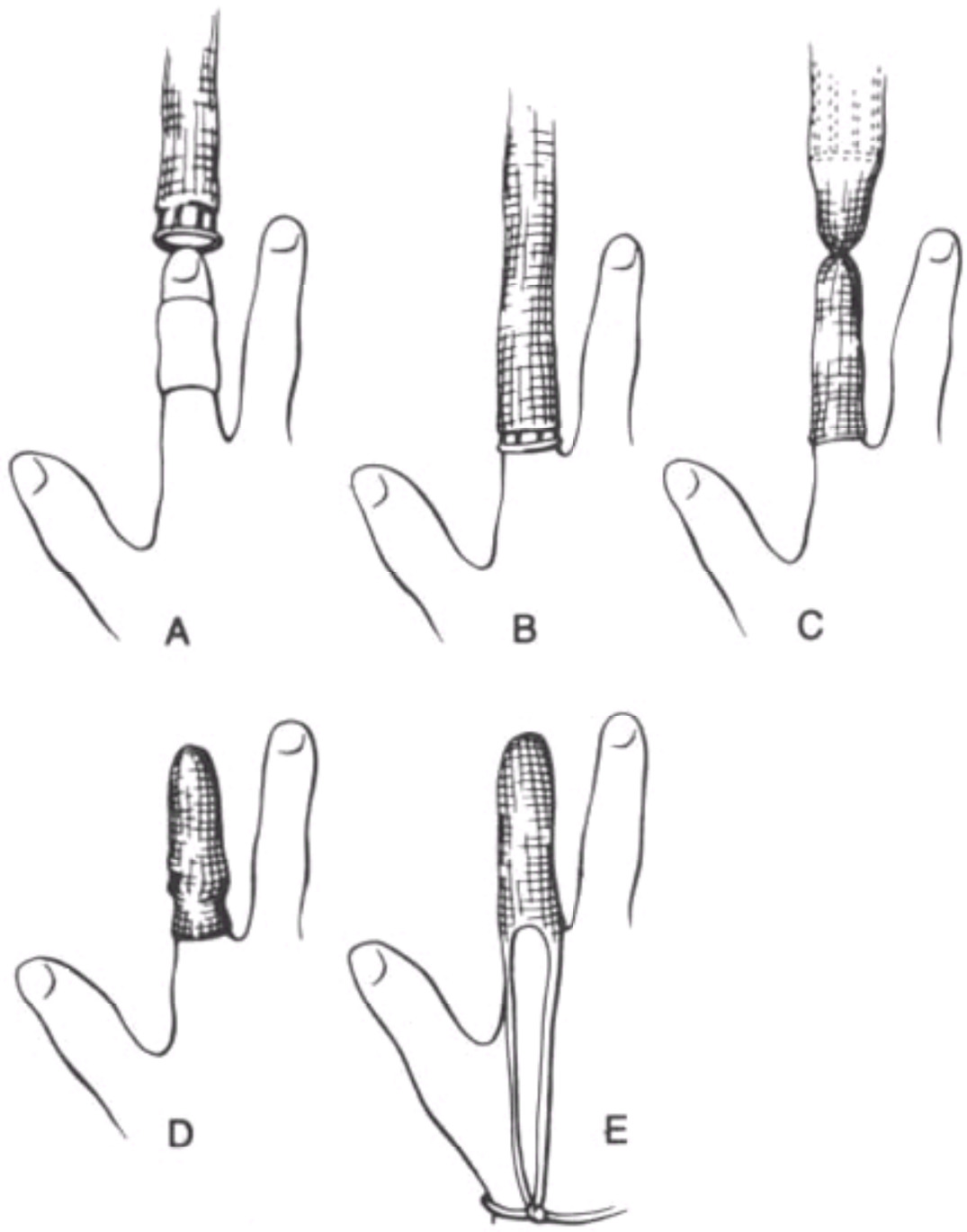


Figure 35-24 *A,* When a Kling or Kerlix wrap must be applied to an area such as the forearm, start by putting a strip of tape on opposite sides of the arm, leaving the ends free. *B,* Wrap the bandage around the arm, covering the portions of the tape that are attached to the skin. *C,* After completing one layer of wrapping, tuck the free ends of the tape down so that the nonadhesive side faces the first layer of wrapping and the sticky side faces out. Place another layer of wrapping around the arm. *D,* After completing a second layer of wrapping, the dressing will not slip because it is adhered to itself as well as to the skin. *(From Lazo J: Non-slip dressing technique. Res Staff Physician 22:103, 1976. Reproduced by permission.)*

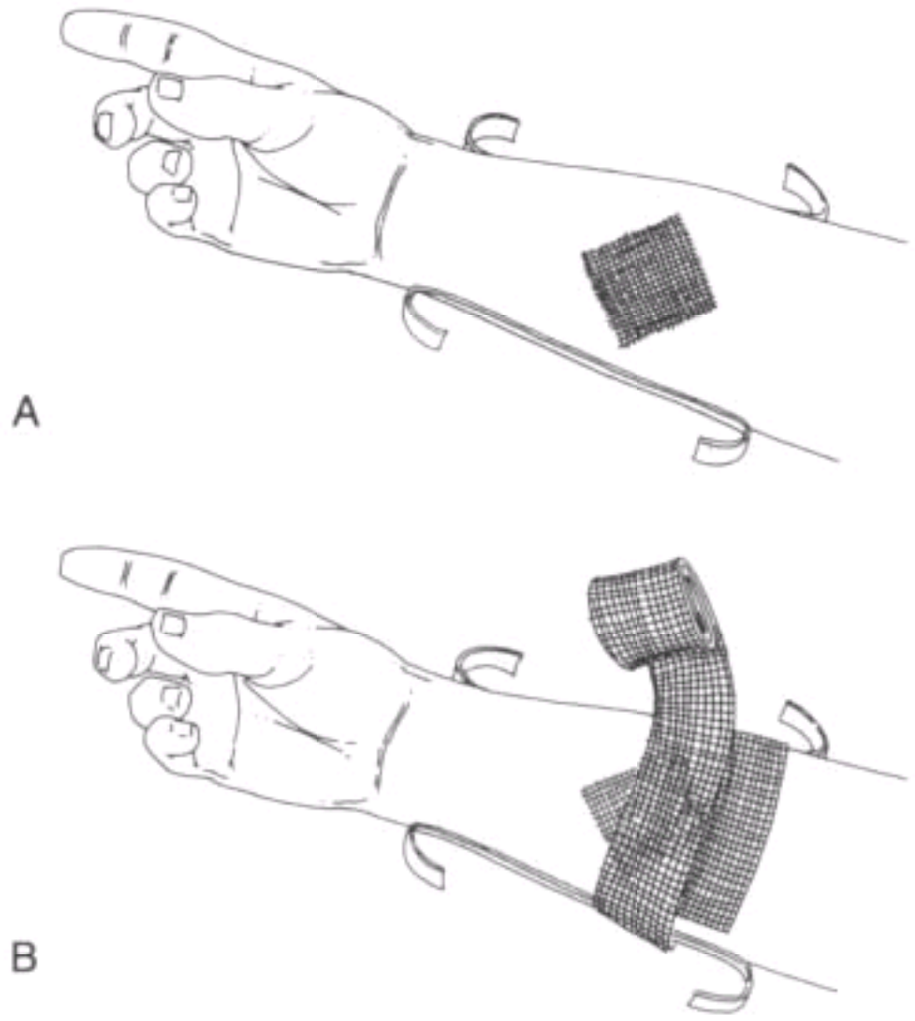


Figure 35-25 *A–E,* Technique for bandaging the head. A strip of bandage (the "tie strip") 3 inches wide and 3 feet long is placed over the head in the frontal plane (*A)*. While the patient maintains downward tension on the first strip of bandage, the clinician places a full-length gauze bandage at the forehead level in a horizontal plane, winding the bandage around the head (*B)*. (The "Kling"-type bandage is preferred.) The main bandage is stabilized with several turns, passing near the patient's ear, then wrapped around one side of the tie in a full turn (*C)*. The main bandage is then taken across the front of the head, wrapped full-turn around the other side of the tie bandage (*D)*. The main bandage is wrapped around the head, from front to back, overlapping with each pass. The dressing is secured in place by tying the ends of the tie strip under the chin (*E)*. This dressing can be removed easily by untying the chin straps and gently pulling both ends of the tie strip upward (*F)*.

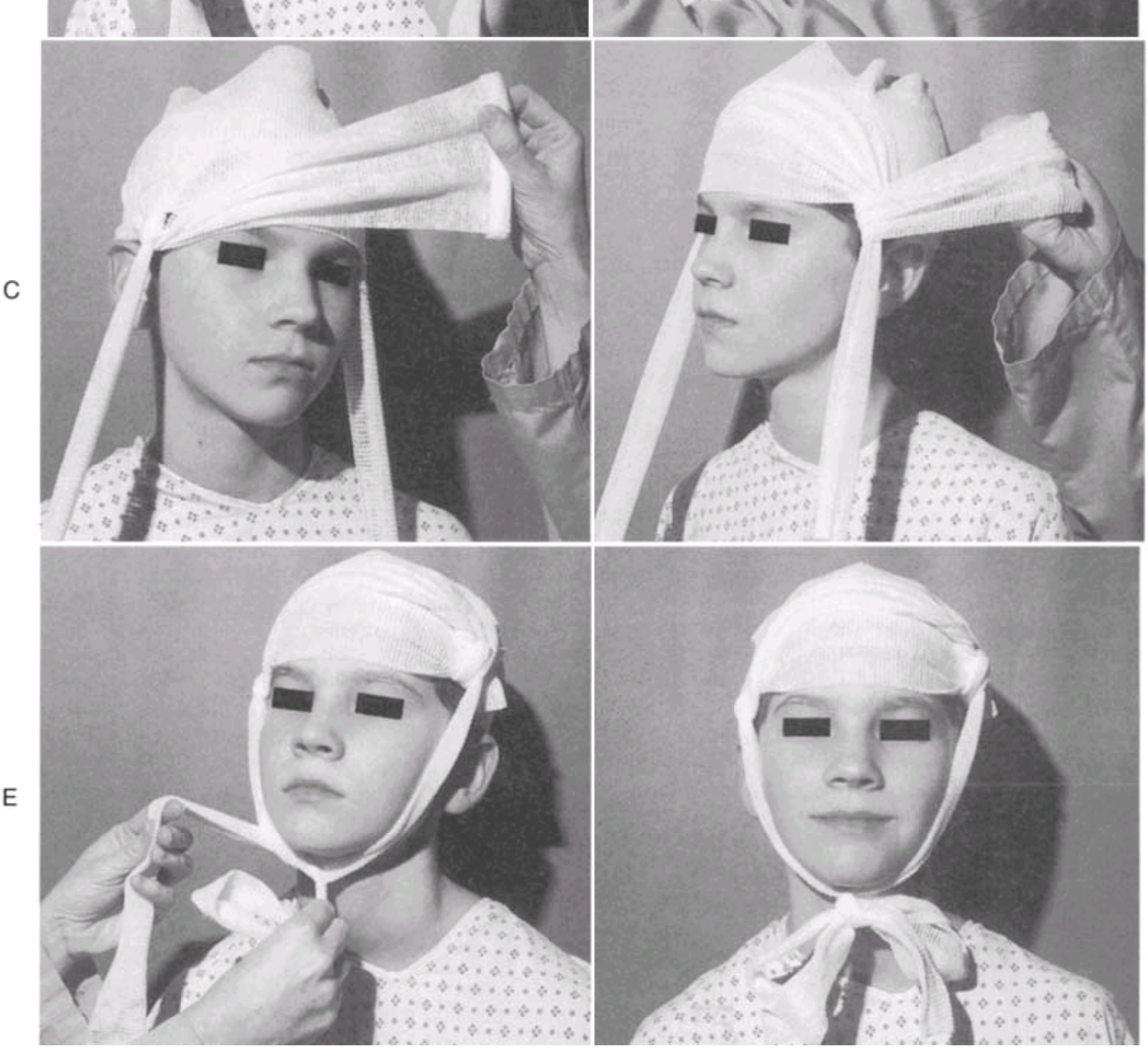


Figure 35-26 This patient used a neomycin-containing ointment on a minor wound, and developed redness, swelling, pruritis, and skin changes. The patient thought it was an infection but it was a contact dermatitis from the neomycin. Plain bacitracin ointment will not do this.

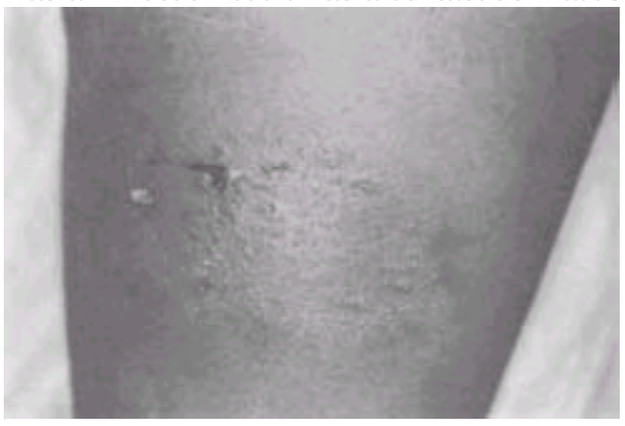


Figure 35-27 Tetanus immunization guidelines.

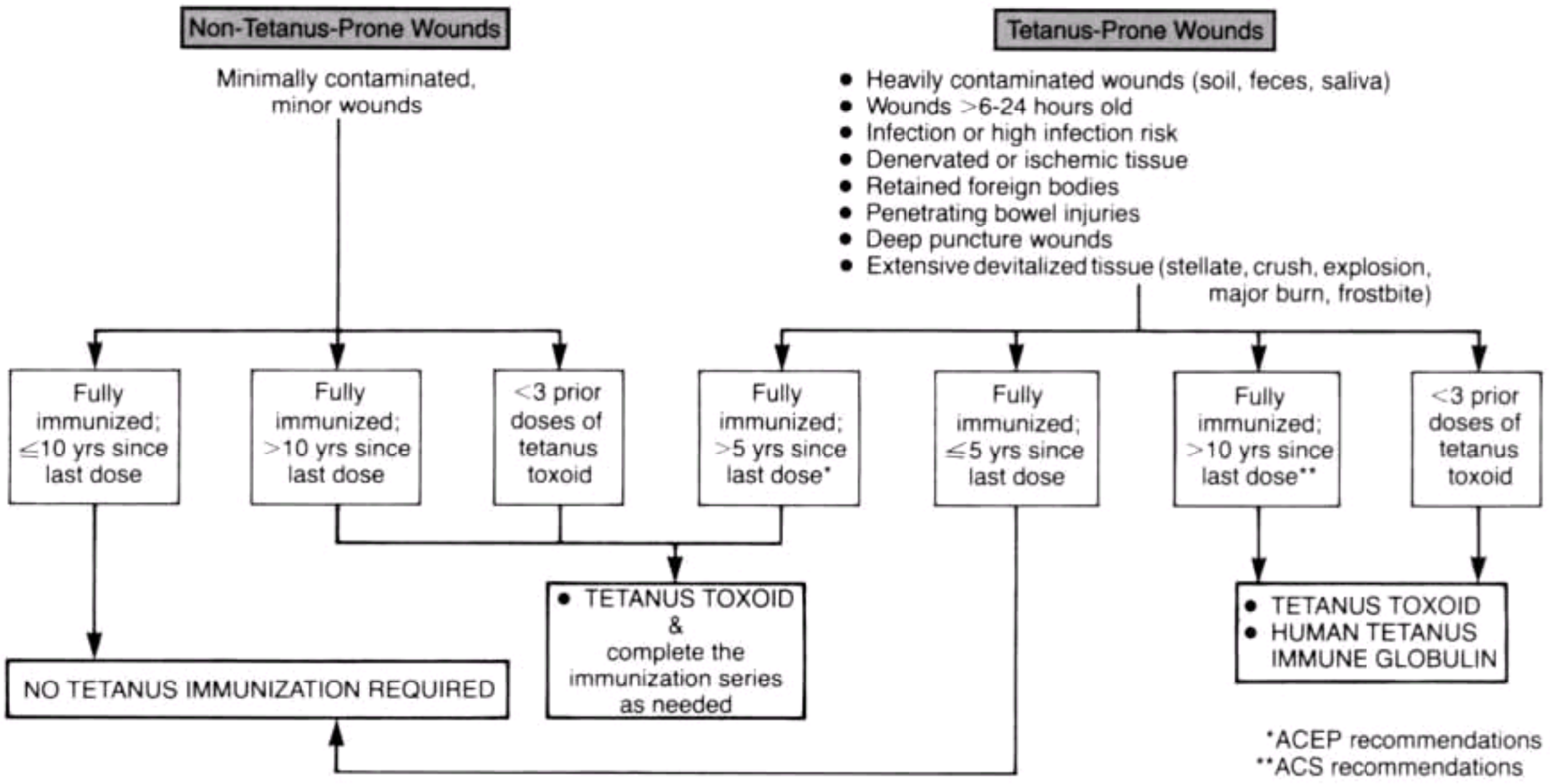


Figure 35-28 Technique for suture removal. Pull should be toward the wound line *(A)* rather than away from it *(B),* which causes the wound to tear apart. *(Modified from Stuzin J, Engrav LH, Buehler PK: Emergency treatment of facial lacerations. Postgrad Med 71:81, 1982.)*

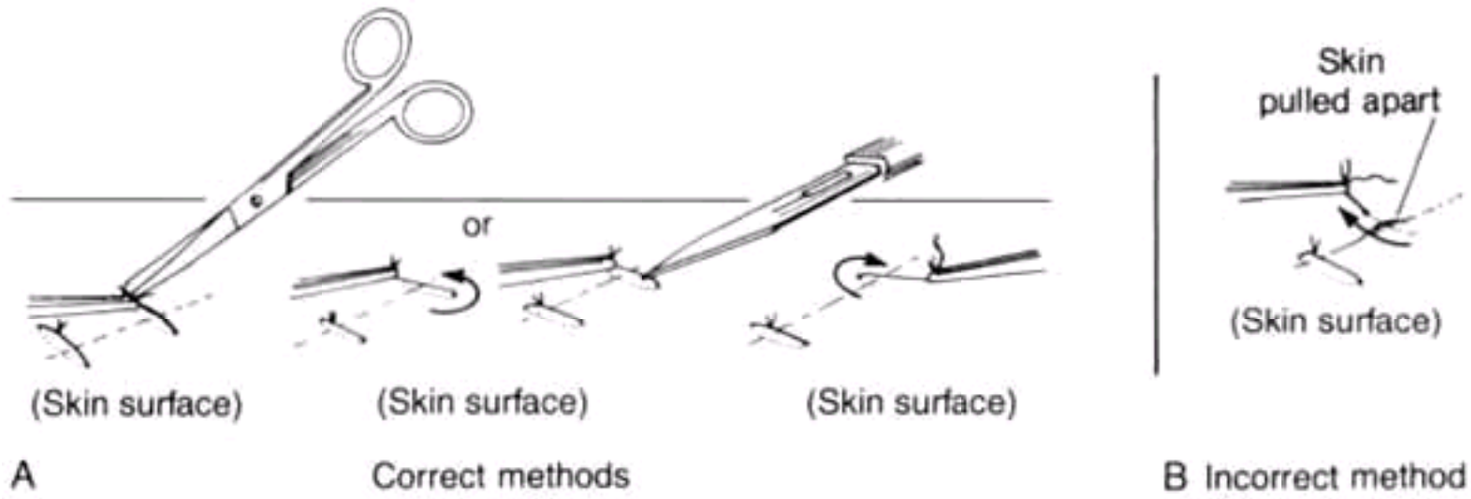


Figure 35-29 Support of the wound with surgical tape.

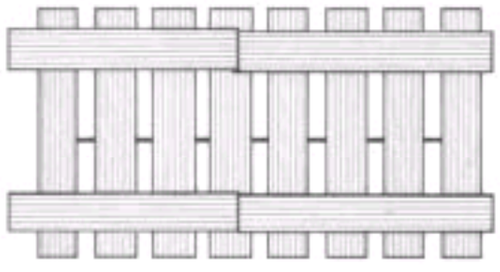


Figure 35-30 At the time of suture removal, the suture is cut at the midpoint of the relief *(A)*. The proximal portion is removed at the point of original entry into the skin *(B),* and the distal portion is removed through the original exit point *(C)*. *(From Grimes DW, Garner RW: "Reliefs" in intracuticular sutures. Surg Rounds 1:48, 1978. Reproduced by permission.)*

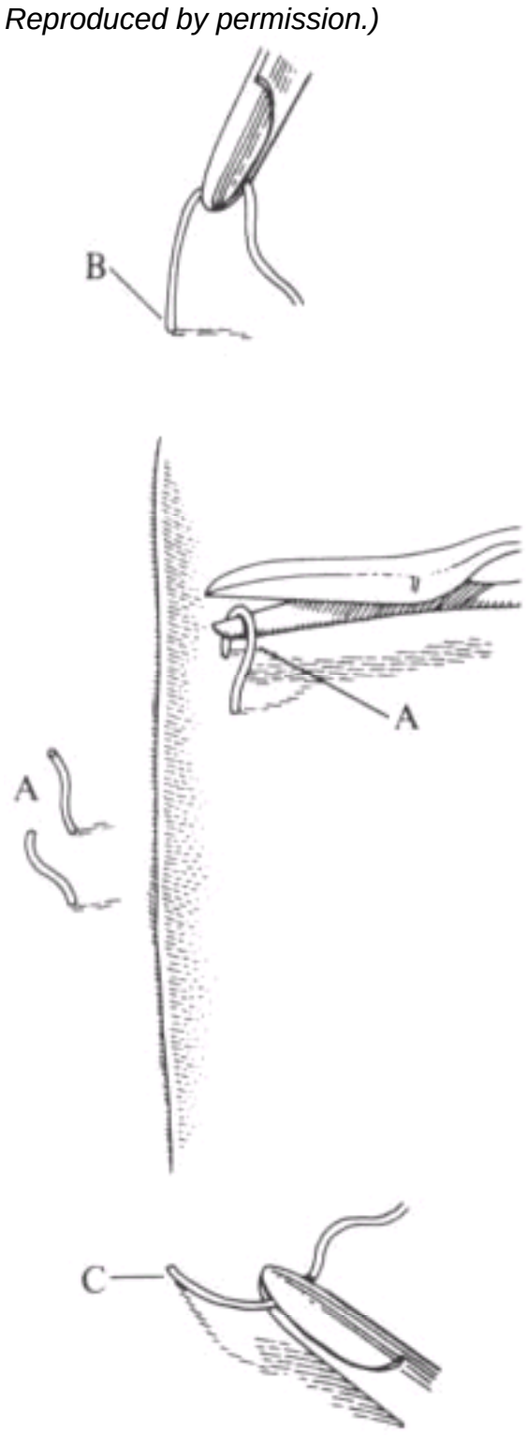


Figure 35-31 Minor gunshot wounds may be treated as outpatients, even when bullet fragments remain and there are *minor* fractures. *A* and *B*, This through-and-through injury transversed the hypothenar eminence. No bullet remained and no bones were involved. *C*, Usually it is impossible to irrigate a puncture wound, but in this case note the saline at the exit site. *D*, After the entrance wound is debrided of the powder burn, a hemostat is passed through the wound. *E*, The instrument grasps gauze packing and pulls it into the wound. The gauze was pulled back and forth to debride the wound tract, and then a clean gauze was left in place. No antibiotics were given, the pack was removed at wound check in 24 hours, and the patient did well. Many gunshot wounds cannot be irrigated to this extent but the treatment principles are similar.

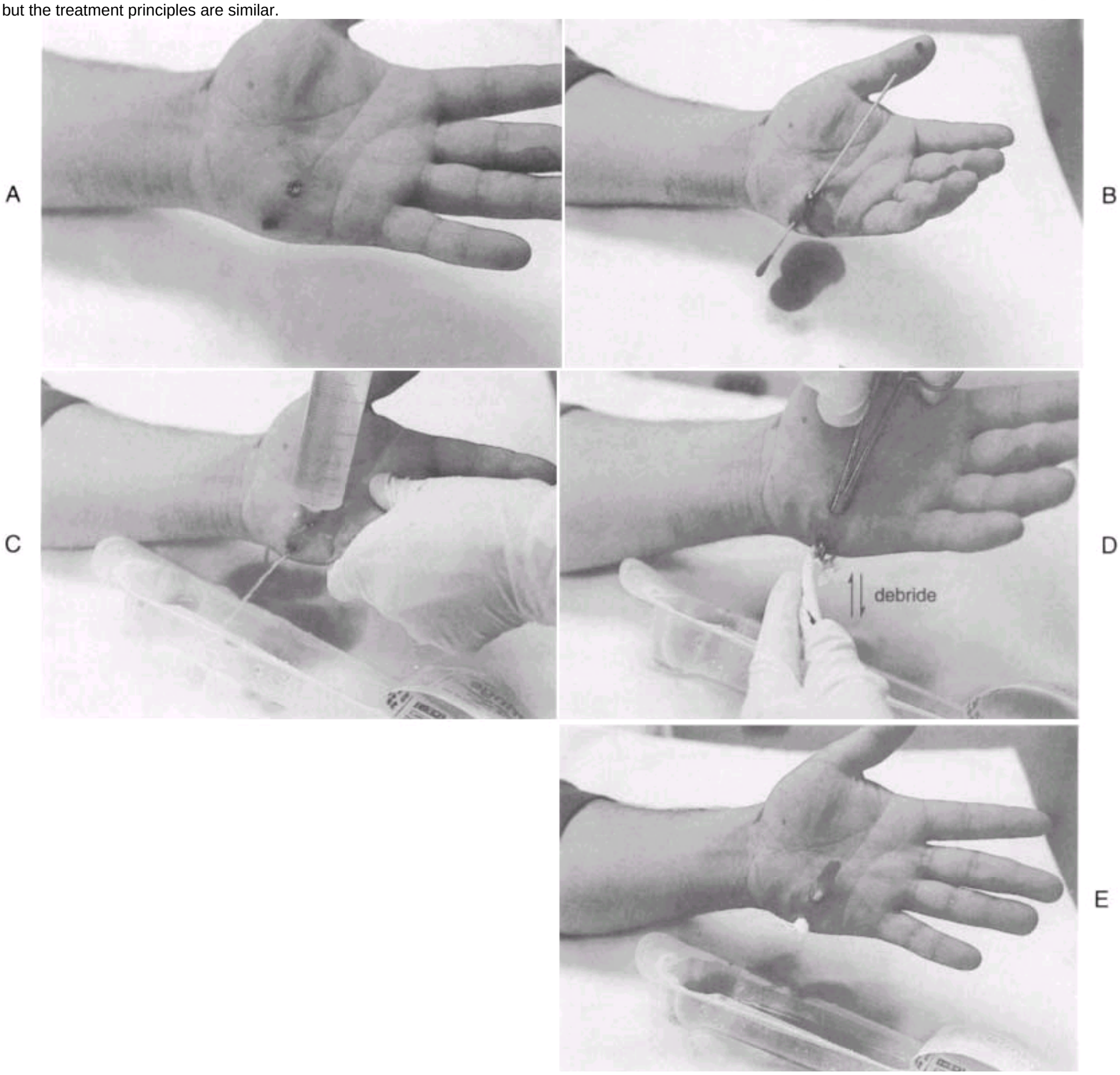


Figure 36-1 *A,* Hair on each side of a laceration is twisted to form "ropes" of hair. *B,* The "roped" strands are tied across the wound in a surgical knot along with additional throws to oppose the skin edges.

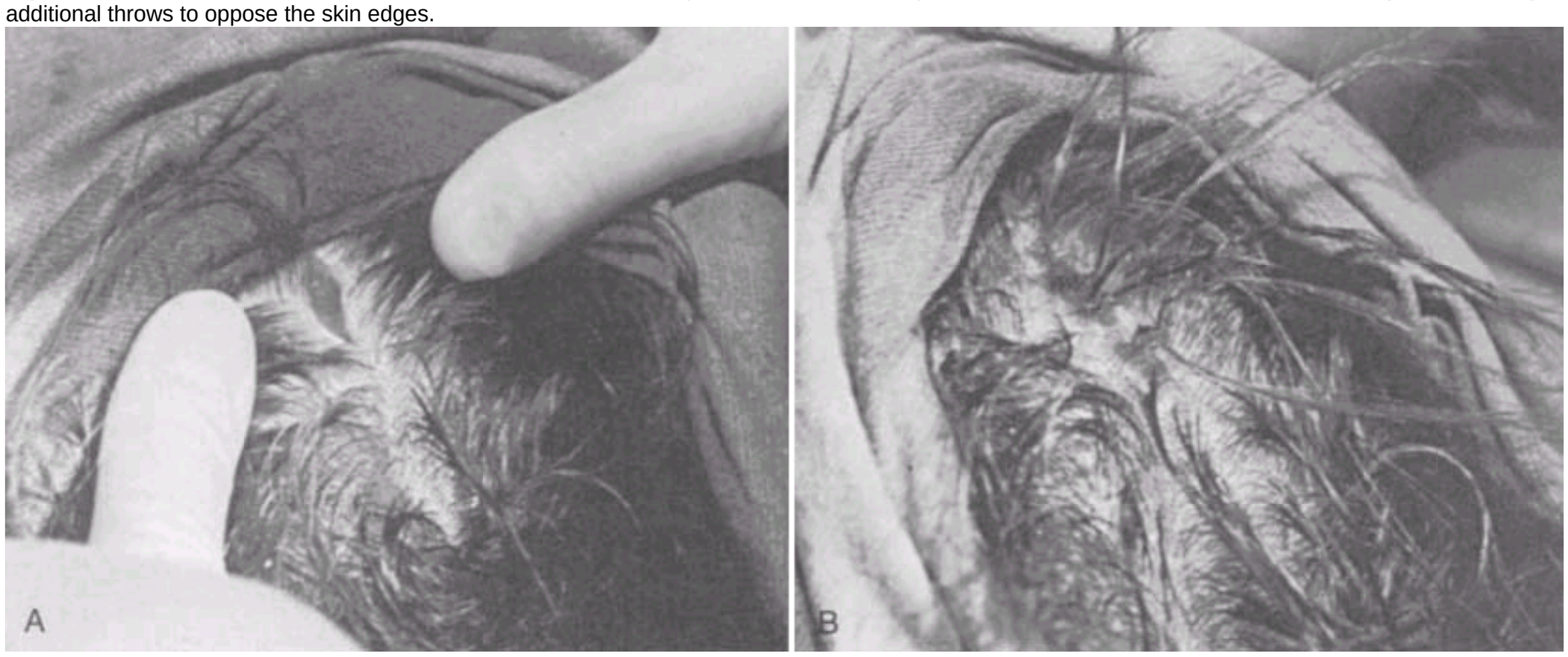


Figure 36-2 *A,* A skin avulsion is an ideal wound to close with closure tapes. An elderly woman who was on steroids had extremely thin skin and suffered an anterior tibial skin avulsion that could not be replaced with sutures. *B,* The skin edges are uncurled, stretched, and anatomically replaced. *C,* The wound should heal when closure tapes keep the skin in place. A compression dressing, such as an elastic bandage or a Dome paste (Unna) boot dressing, can be applied to minimize flap movement and decrease fluid buildup under the flap. *D,* Tape should be placed in a semicircular or spiral pattern on digits to avoid constriction.

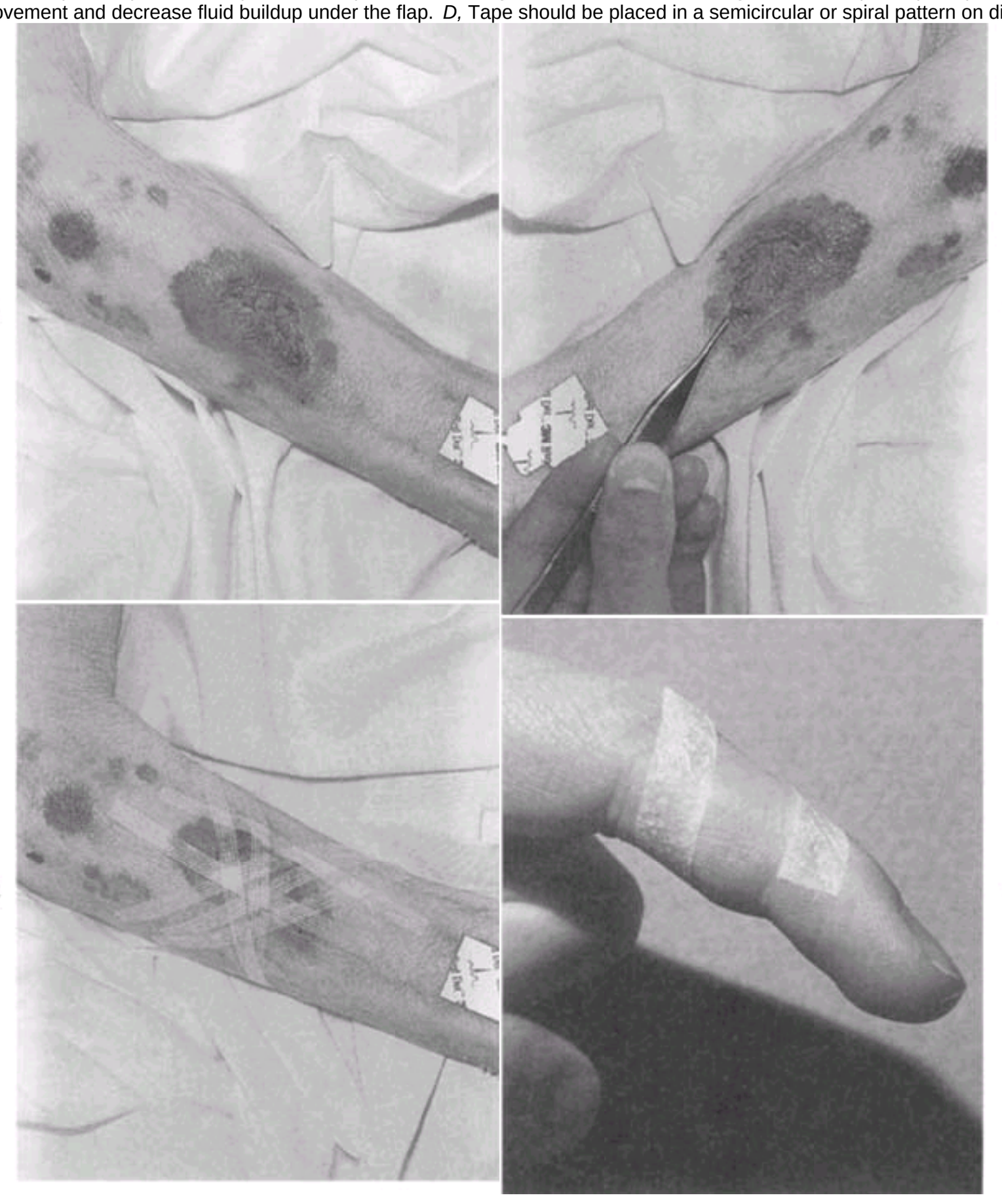
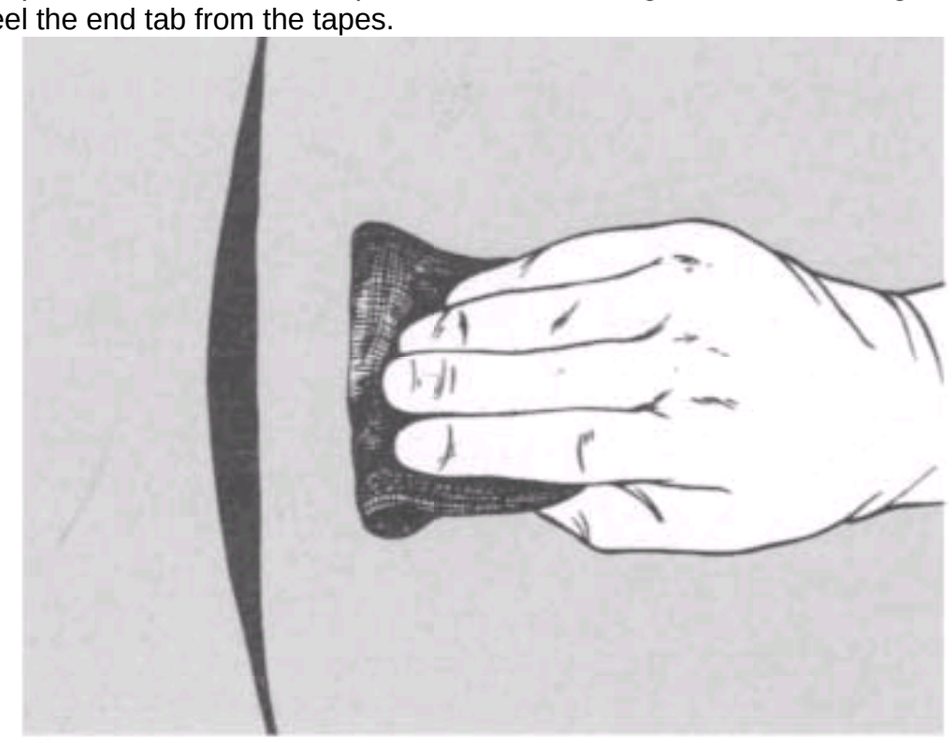
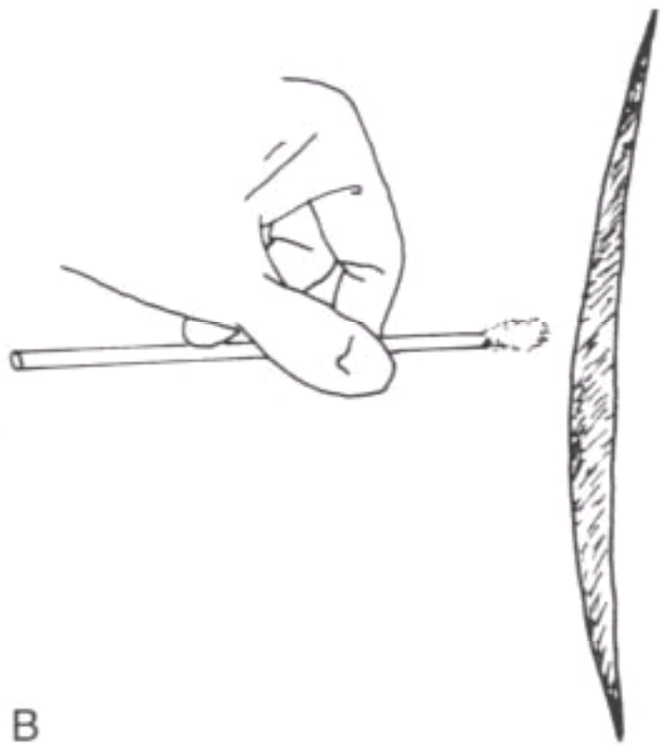


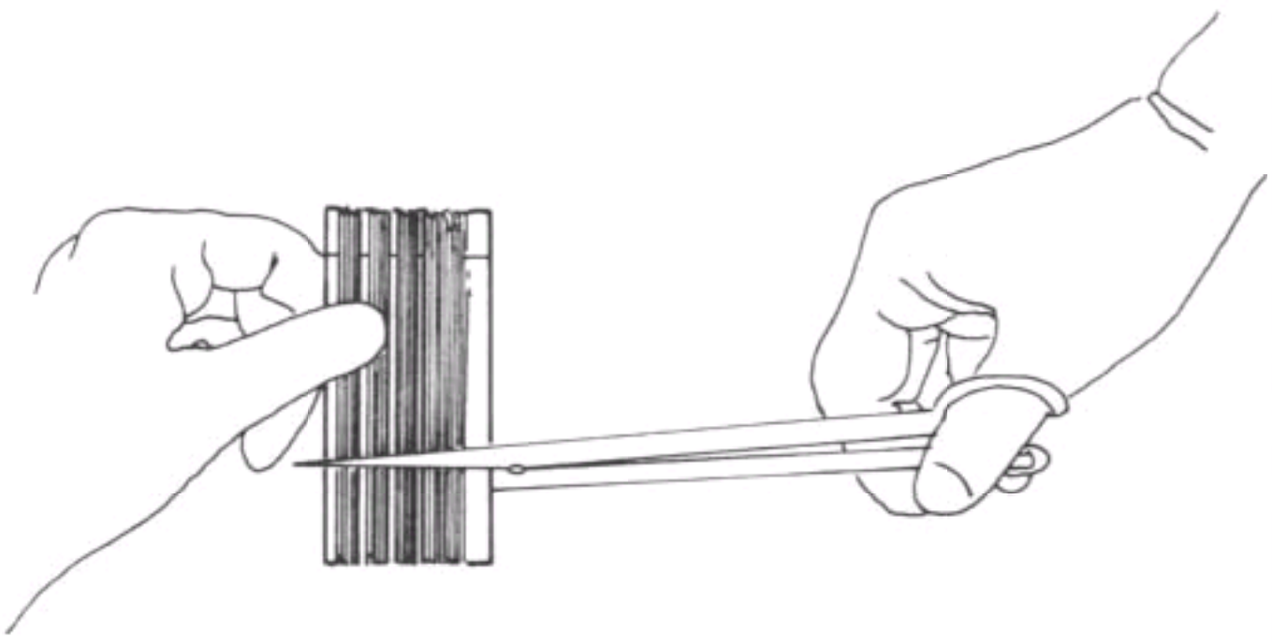
Figure 36-3a Proper technique for application of tapes for skin closure. *A,* After wound preparation (and placement of deep closures, if needed), dry the skin thoroughly at least 2 inches around the wound. Failure to dry the skin and failure to obtain perfect hemostasis are common causes of failure of tapes to stick to the skin. *B,* If desired, apply a thin coating of tincture of benzoin around the wound to enhance tape adhesiveness. Benzoin should not enter the eye, and it causes pain if it seeps into an open wound. *C,* Cut the tapes to the desired length before removing the backing. *D,* The tapes are attached to a card with perforated tabs on both ends. Gently peel the end tab from the tapes.



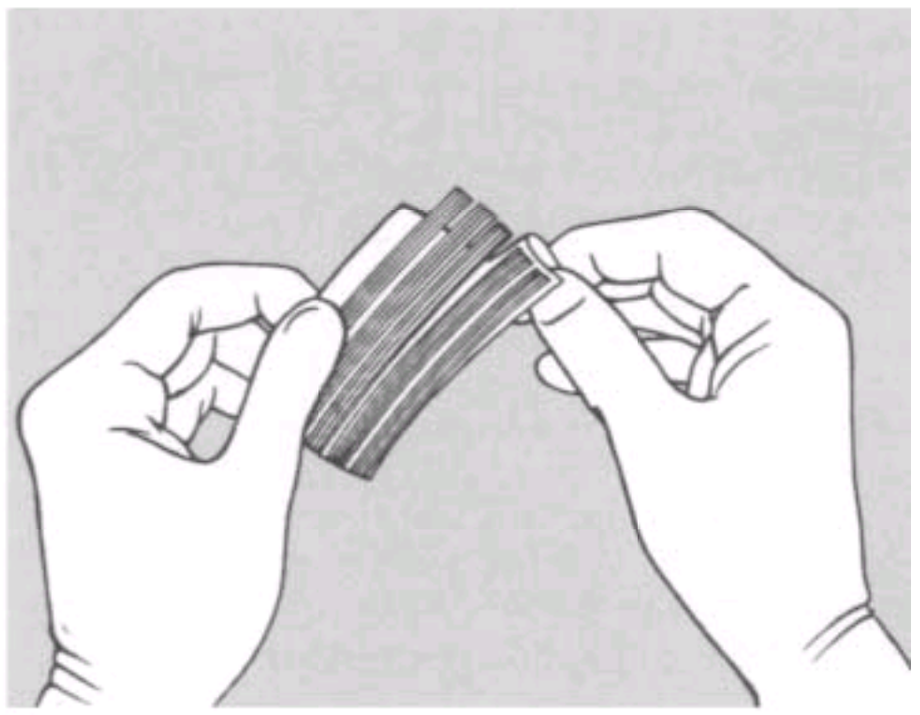
A



B

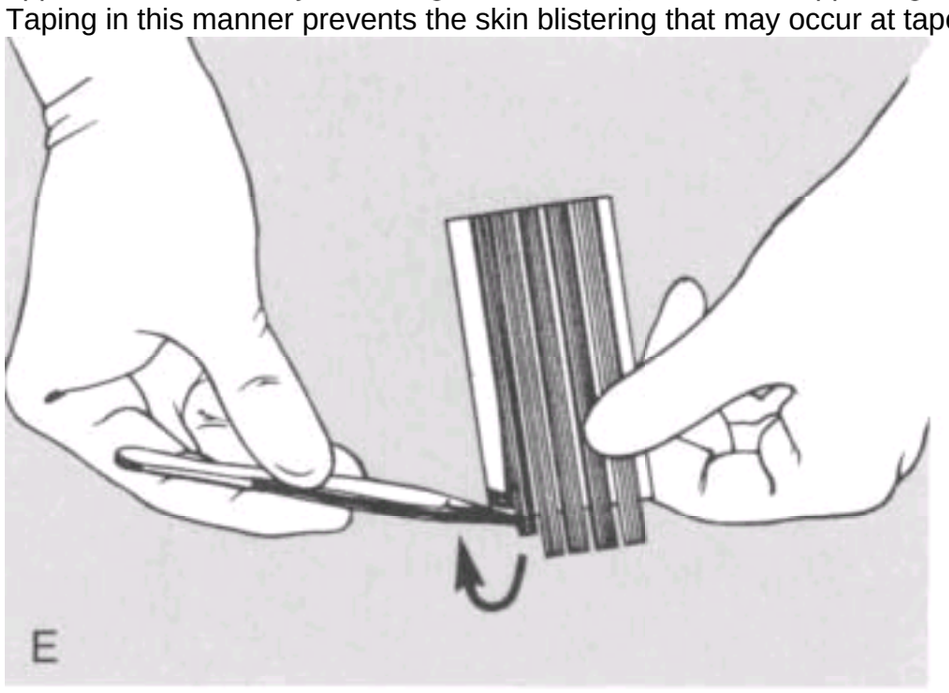


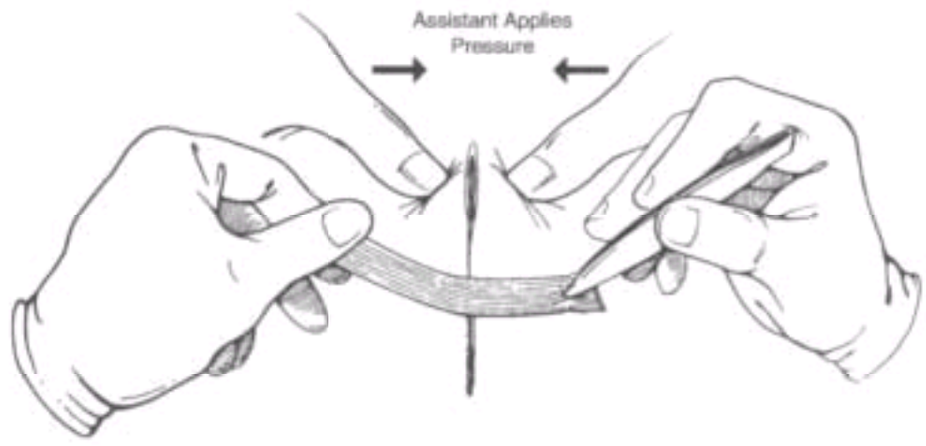
C

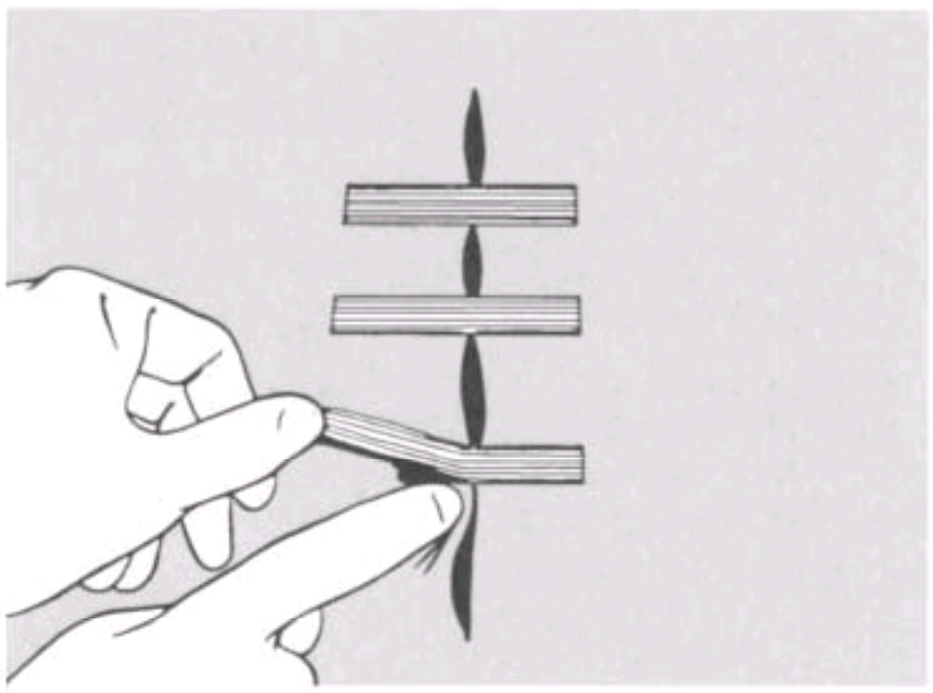


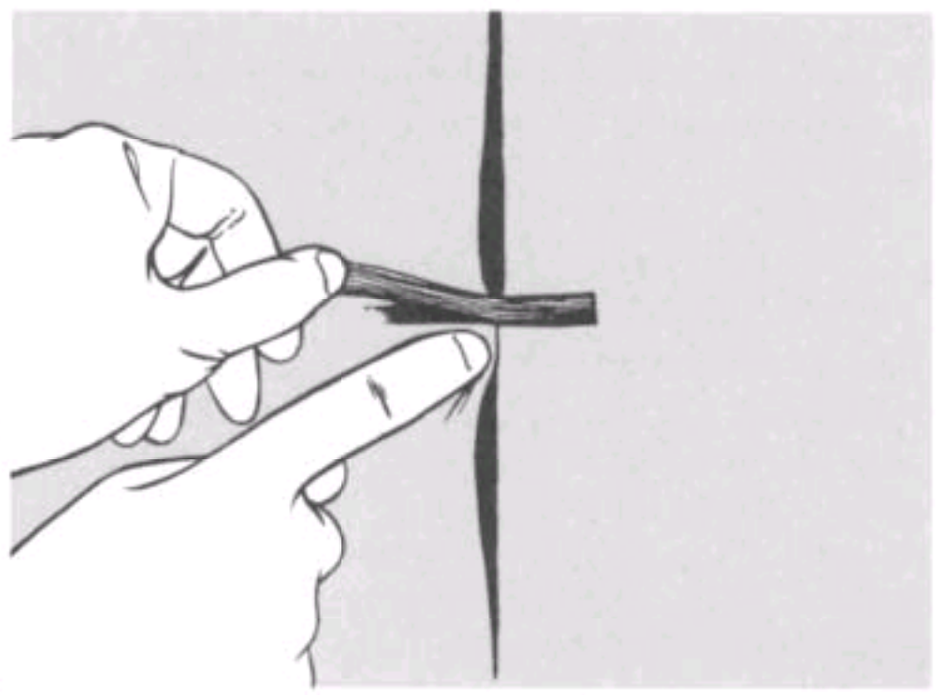
D

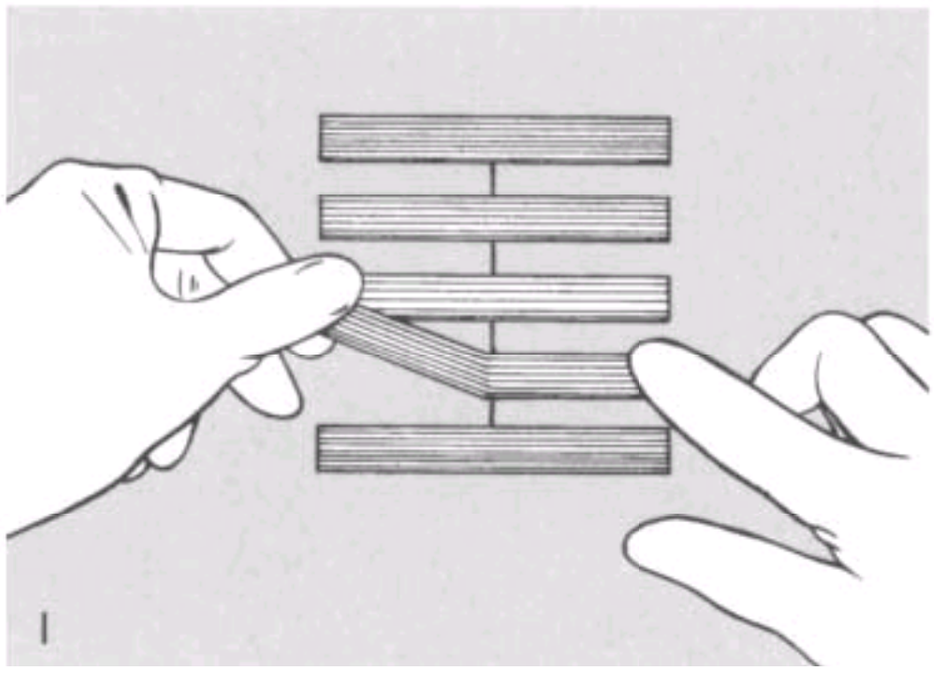
Figure 36-3b *E,* Use forceps to peel the tape off the card backing. Pull directly backward, not to the side. *F,* Place one half of the first tape at the midportion of the wound; secure firmly in place. *G,* Gently but firmly oppose the opposite side of the wound, using the free hand or forceps. If an assistant is not available, the operator can approximate the wound edges. *H,* The tape should be applied by bisecting the wound until the wound is closed satisfactorily. *I,* Wound margins are completely opposed without totally occluding the wound. *J,* Additional supporting tapes are placed approximately 2.5 cm from the wound and parallel to the wound direction. Taping in this manner prevents the skin blistering that may occur at tape ends.











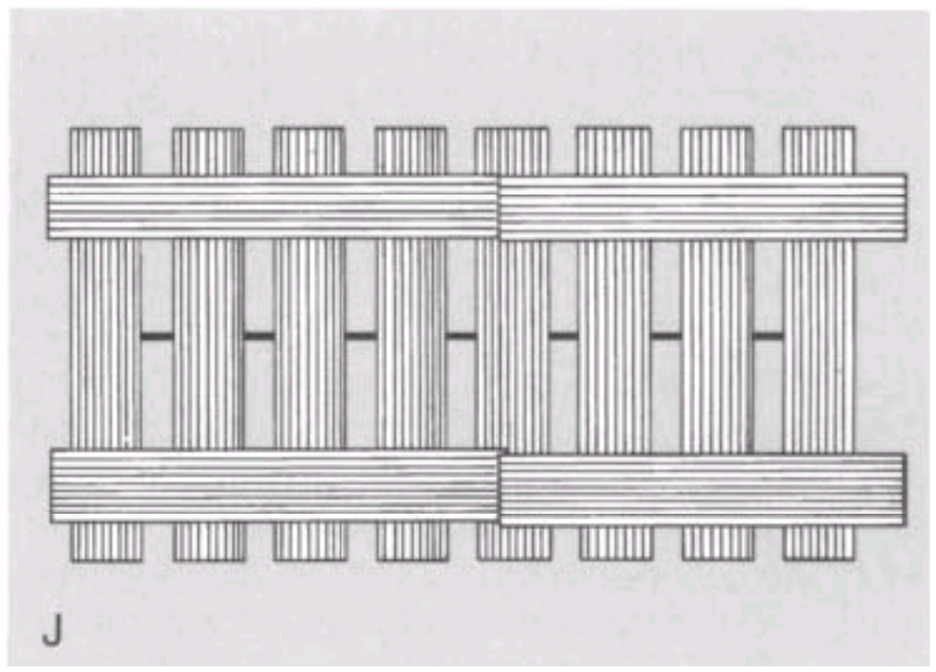


Figure 36-4 Tissue adhesive, 2-octyl cyanoacrylate in a commercially available application dispenser.

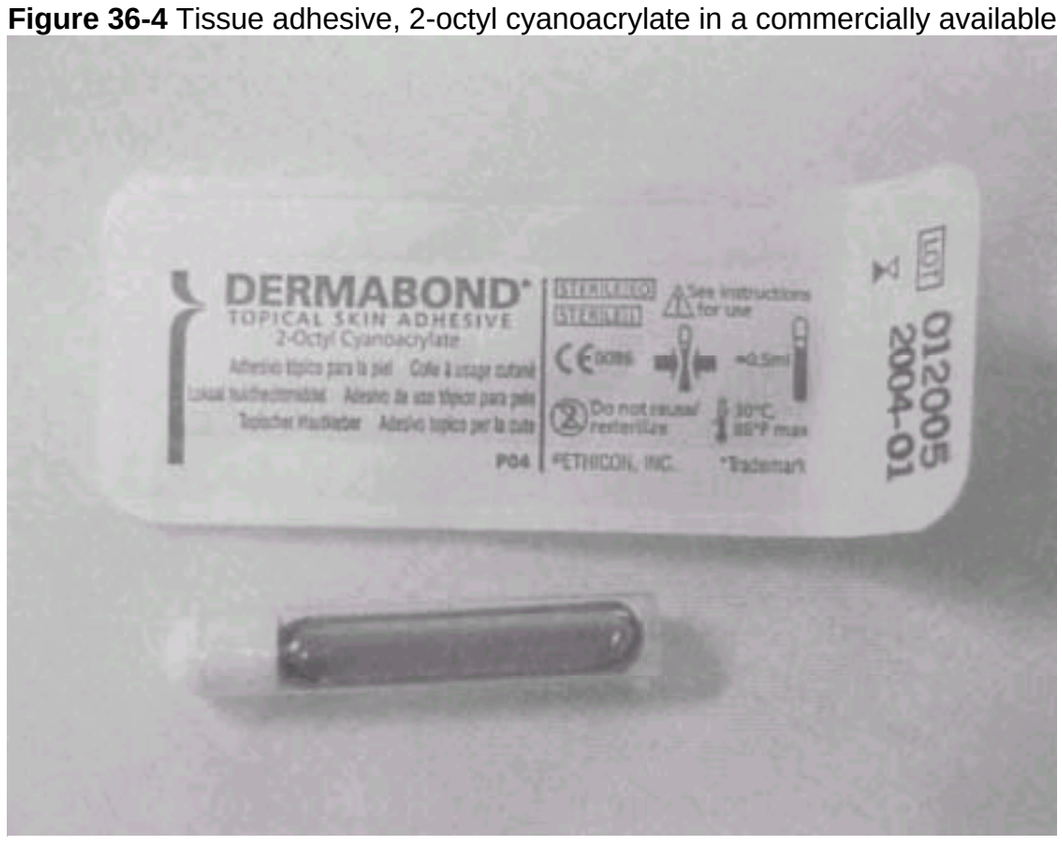


Figure 36-5 The skin edges must be approximated and everted by hand or with forceps before they are secured with staples. Failure to evert the wound edges is a common error that may cause an unacceptable result. *(Adapted with permission from Edlich RF: A Manual for Wound Closure. St. Paul, MN, 3M Medical-Surgical Products, 1979. Reproduced by permission.)*

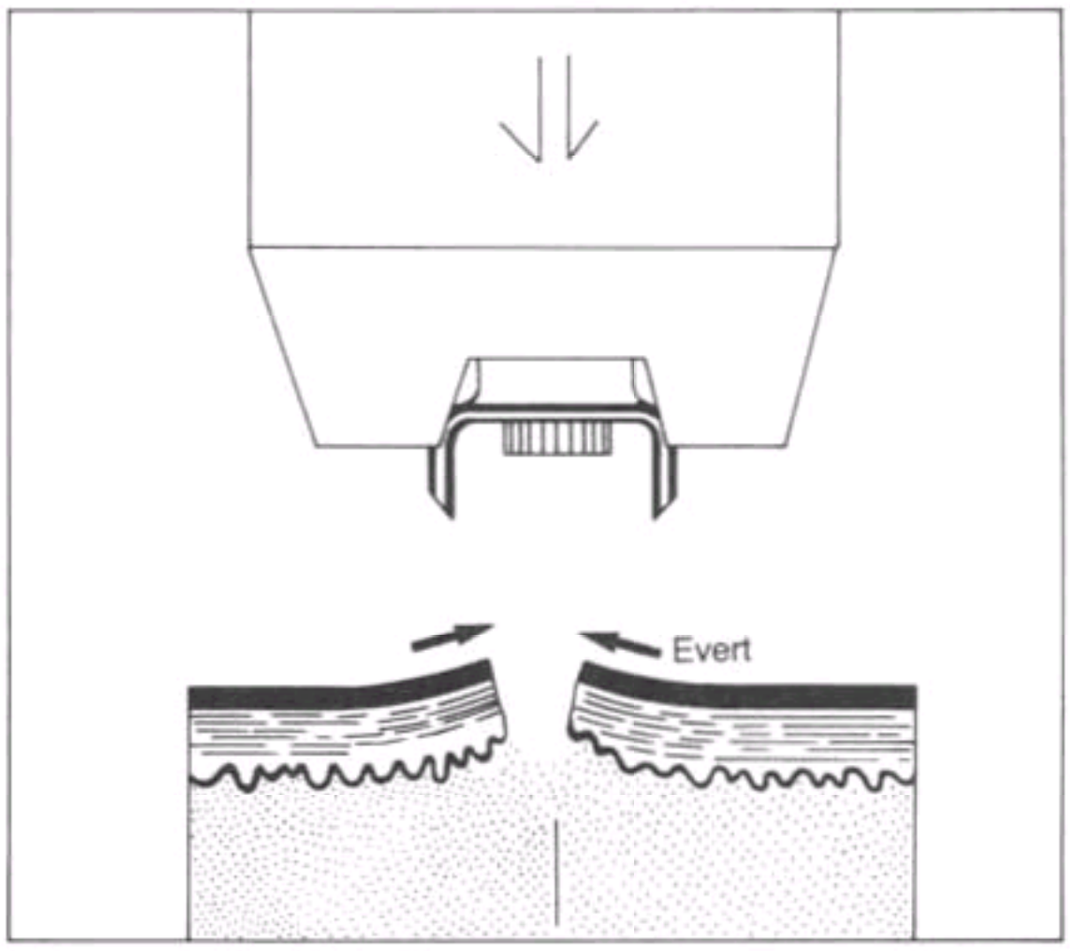


Figure 36-6 By squeezing the stapler handle, a plunger advances one staple into the wound margins. *(From Edlich RF: A Manual for Wound Closure. St. Paul, MN, 3M Medical-Surgical Products, 1979. Reproduced by permission.)*

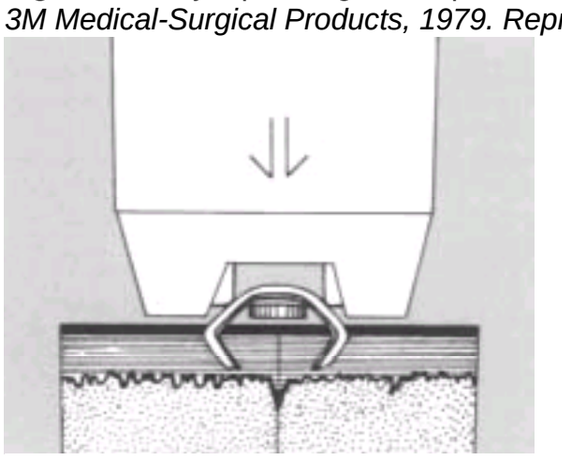


Figure 36-7 An anvil automatically bends the staple to the proper configuration. *(From Edlich RF: A Manual for Wound Closure. St. Paul, MN, 3M Medical-Surgical Products, 1979. Reproduced by permission.)*

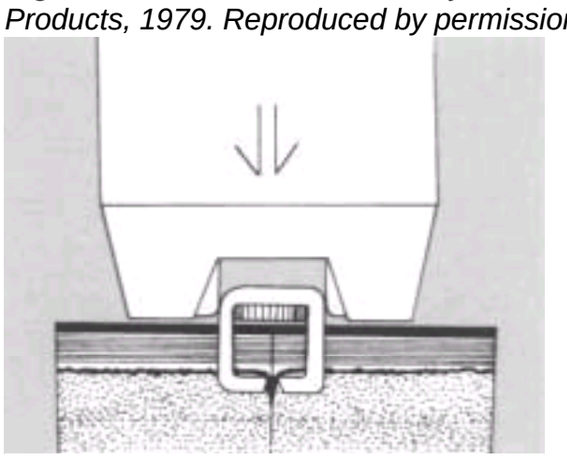


Figure 36-8 Care should be taken to ensure that a space remains between the skin and the crossbar of the staple. Excessive pressure created by placing the staple too deep causes wound edge ischemia, as well as pain on removal. Note that the staple bar is 2 to 3 mm above the skin line. *(From Edlich RF: A Manual for Wound Closure. St. Paul, MN, 3M Medical-Surgical Products, 1979. Reproduced by permission.)*

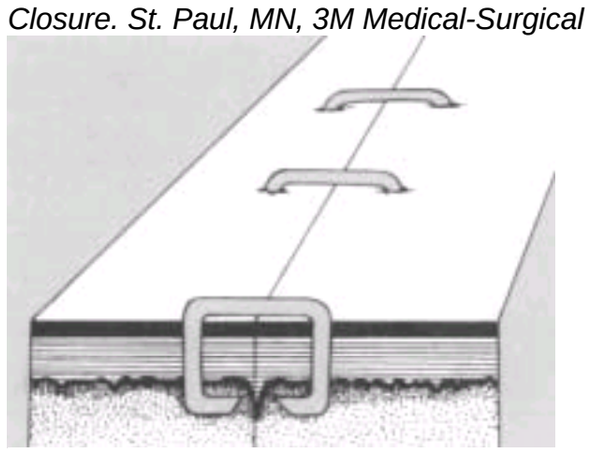


Figure 36-9 The lower jaw of the staple remover is placed under the crossbar of the staple. *(From Edlich RF: A Manual for Wound Closure. St. Paul, MN, 3M Medical-Surgical Products, 1979. Reproduced by permission.)*

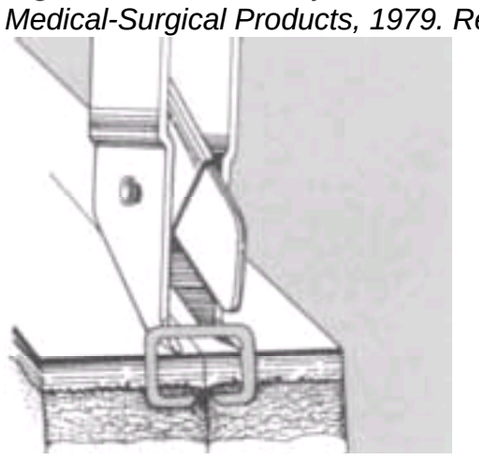


Figure 36-10 By squeezing the handle gently, the upper jaw compresses the staple and allows it to exit the skin. *(Adapted from Edlich RF: A Manual for Wound Closure. St. Paul, MN, 3M Medical-Surgical Products, 1979. Reproduced by permission.)*

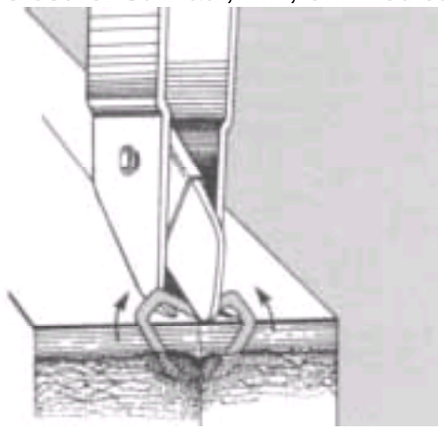


Figure 36-11 A very poor result occurred when staples (*some marked with arrows*) were used to close this deep scalp laceration (*A*). The wound edges were not everted (in fact, the skin overlapped significantly), and poor hemostasis was obtained because the galea was not closed by the superficial staples. Three days later during a wound check, the staples were removed, and the laceration was closed with 3-0 interrupted nylon sutures (*B*). The clinician should attempt to obtain a cosmetic closure on all scalp lacerations, because as patients lose their hair, a previously hidden, unsightly scar emerges. In general, staples should not be used to close full-thickness scalp lacerations, especially wounds that are actively bleeding.

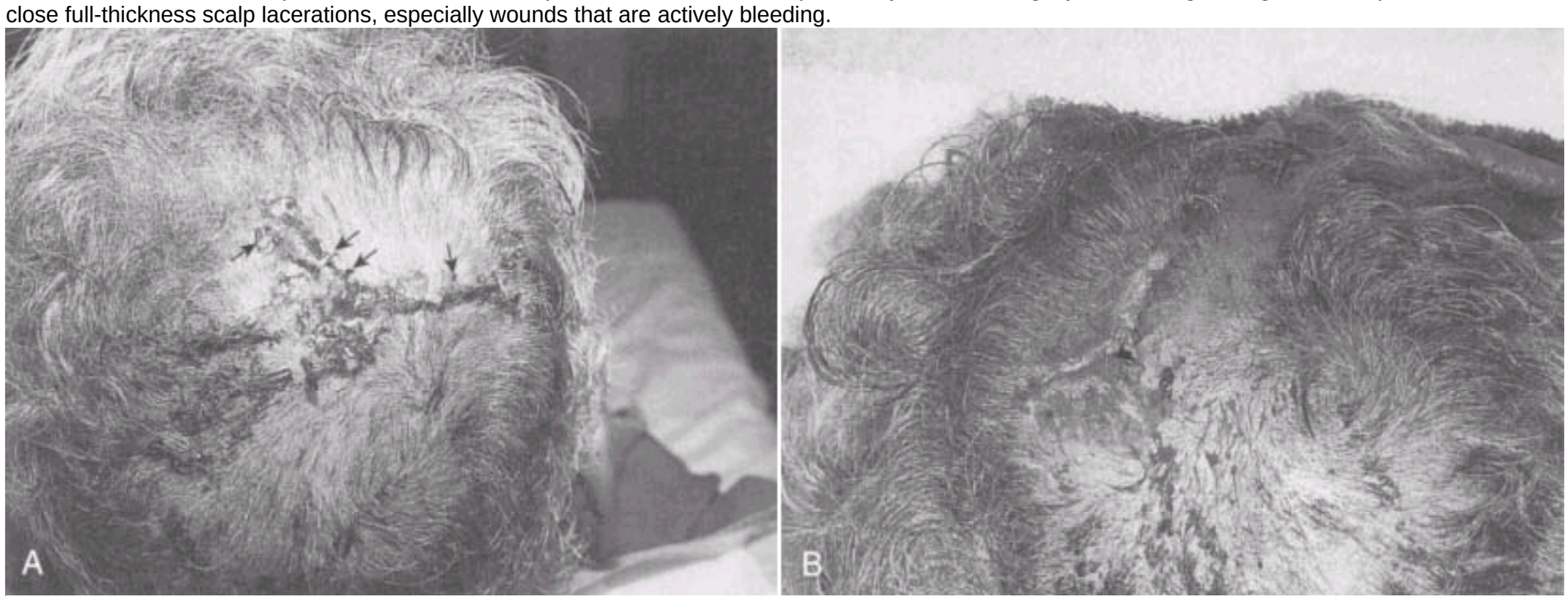


Figure 36-12 The eyeless, or "swaged," needle. *(From Suture Use Manual: Use and Handling of Sutures and Needles. Somerville, NJ, Ethicon, Inc., 1977, p 29. Reproduced by permission.)*

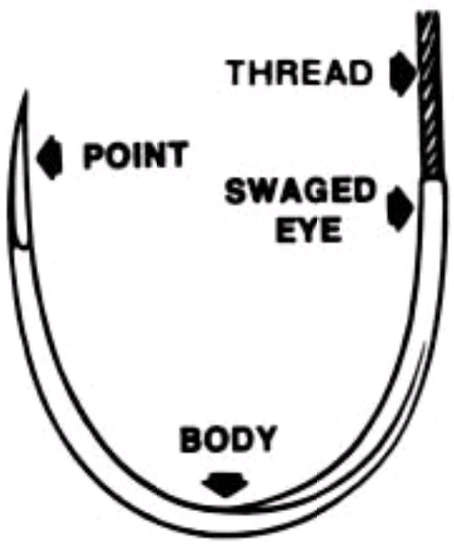


Figure 36-13 The needle should be large enough to pass through tissue and should exit far enough to enable the needle holder to be repositioned on the end of the needle at a safe distance from the point.

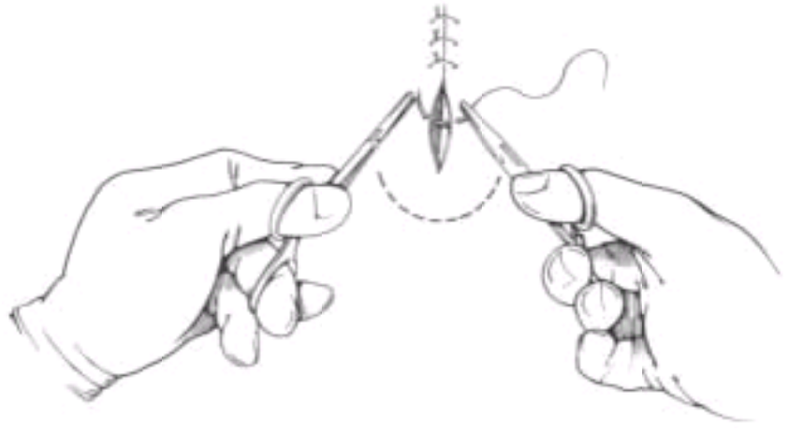


Figure 36-14 One-half and three-eighths circle needles, used for most traumatic wound closures.

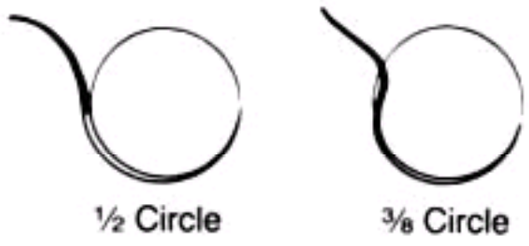


Figure 36-15 Types of needles. *A,* The conventional cutting needle has two opposing cutting edges, with a third edge on the inside curvature of the needle. The conventional cutting needle changes in cross section from a triangular cutting tip to a flattened body. *B,* The reverse cutting needle is used to cut through tough, difficult-to-penetrate tissues, such as fascia and skin. It has two opposing cutting edges, with the third cutting edge on the outer curvature of the needle. The reverse cutting needle is made with the triangular shape extending from the point to the swage area, with only the edges near the tip being sharpened. *(From Suture Use Manual: Use and Handling of Sutures and Needles. Somerville, NJ, Ethicon, Inc., 1977, p 31. Reproduced by permission.)*

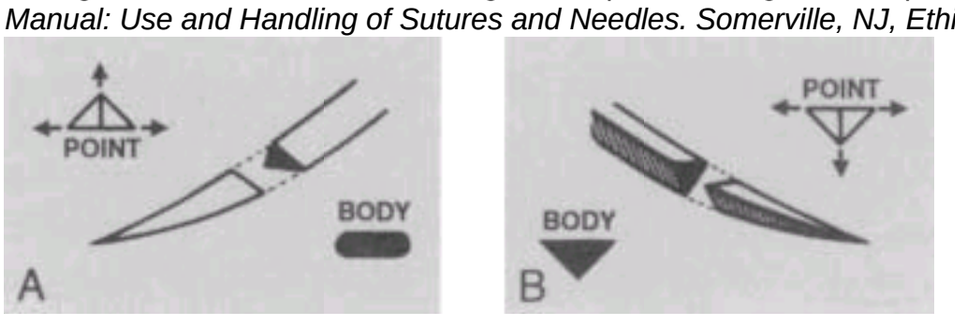


Figure 36-16 *A–D,* Technique for wrapping the scalp to keep stray hair from falling into the operating field. A scrub hat is an acceptable alternative.

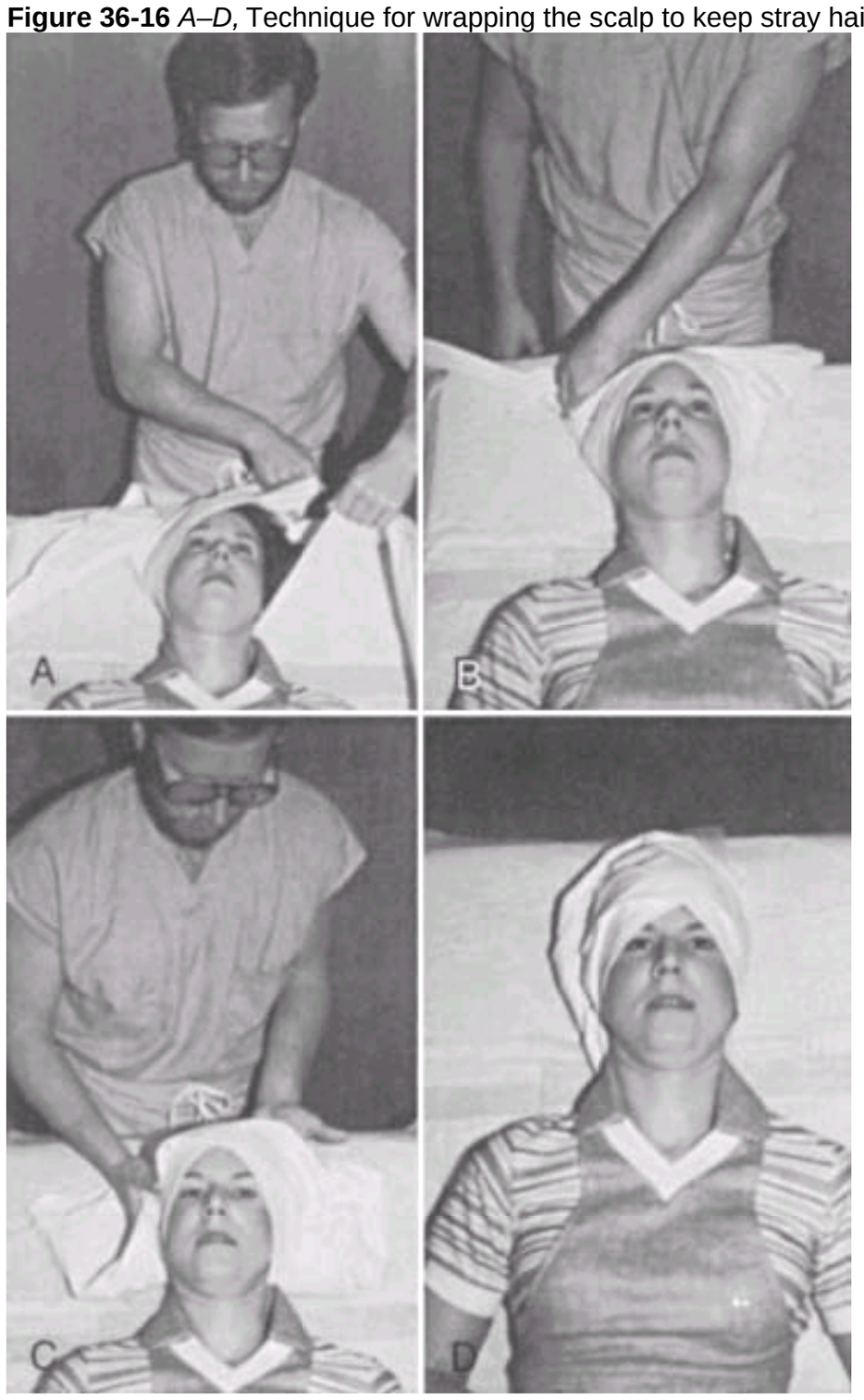


Figure 36-17 The technique of undermining. The scalpel is used to find an appropriate site; a natural plane often exists at the epidermis-dermis junction. Undermining relieves tension on the wound and renders a better cosmetic result. This technique is simple to master, but sometimes overlooked.

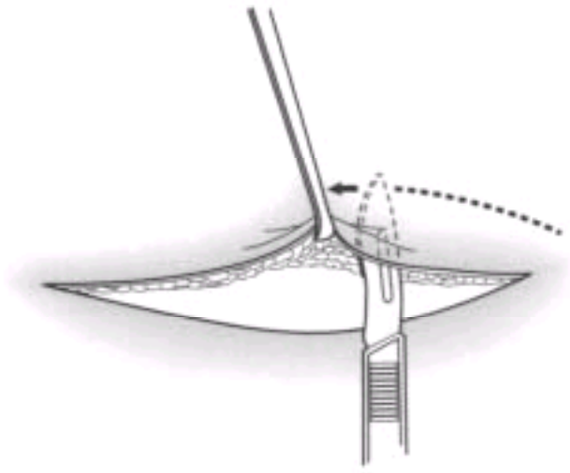
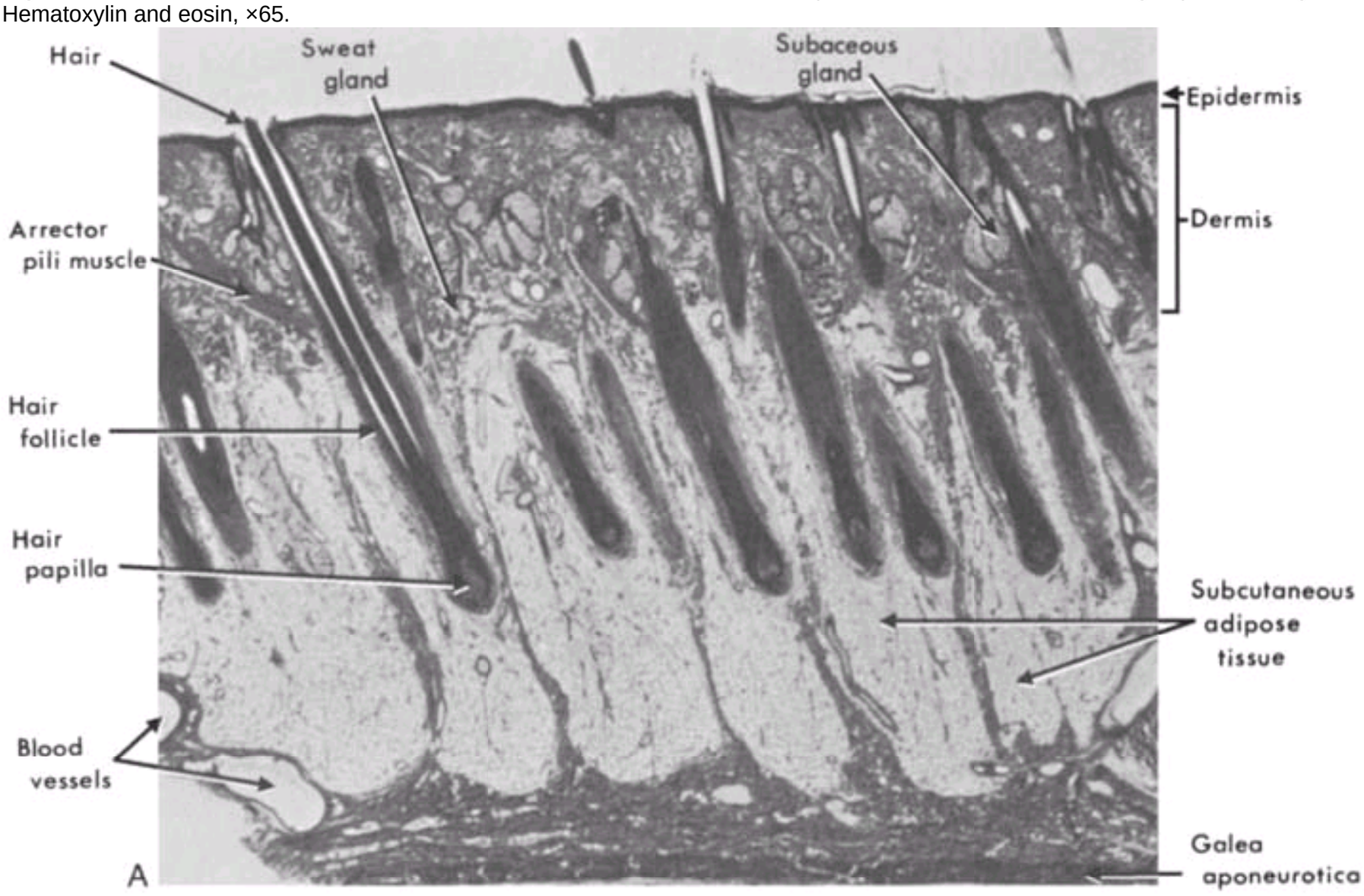


Figure 36-18a Variation in the structure of skin. *A,* Section of the skin of the scalp, ×15. *B,* Skin of the human fingertip, illustrating a very thick stratum corneum. Hematoxylin and eosin, ×65.



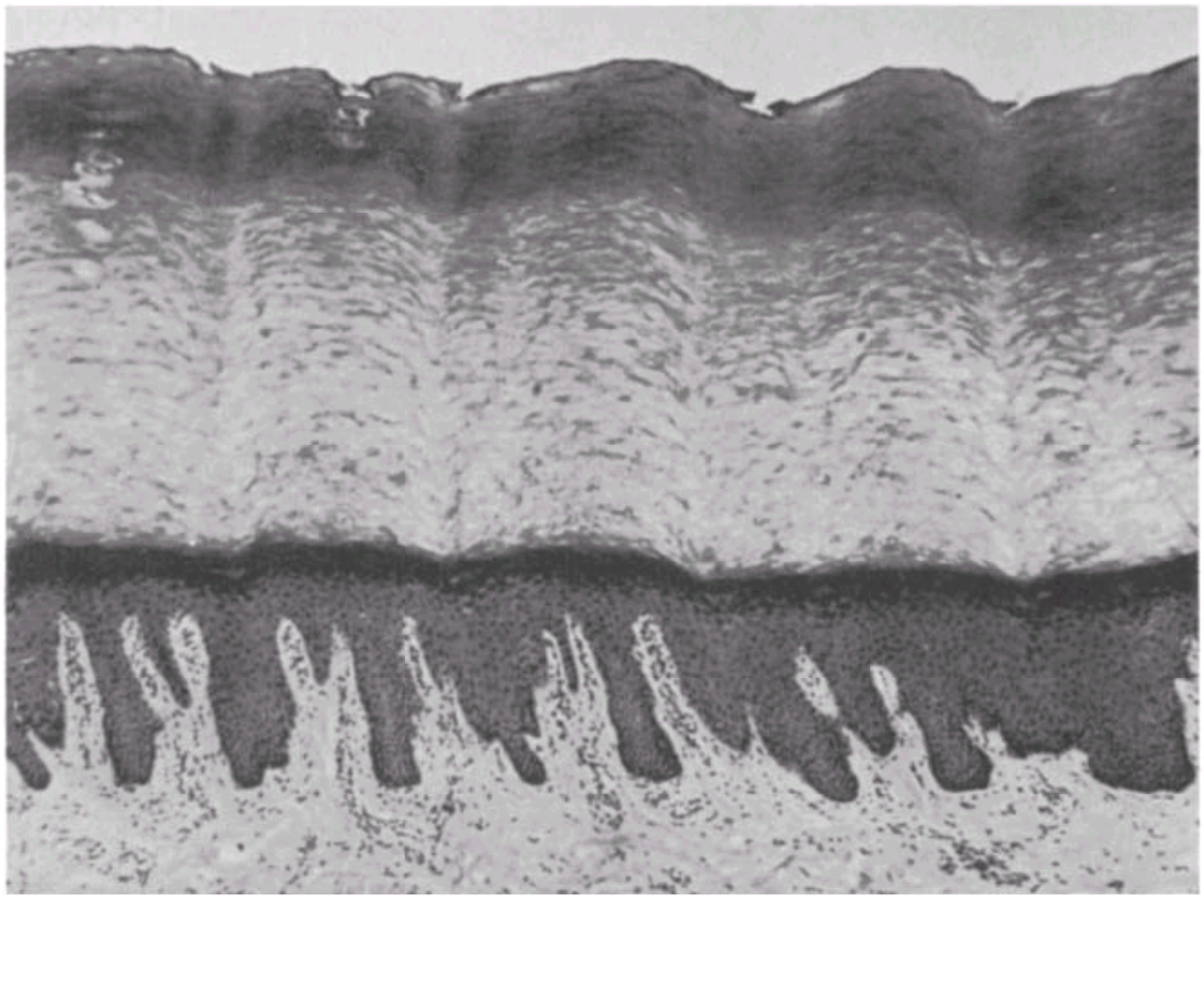
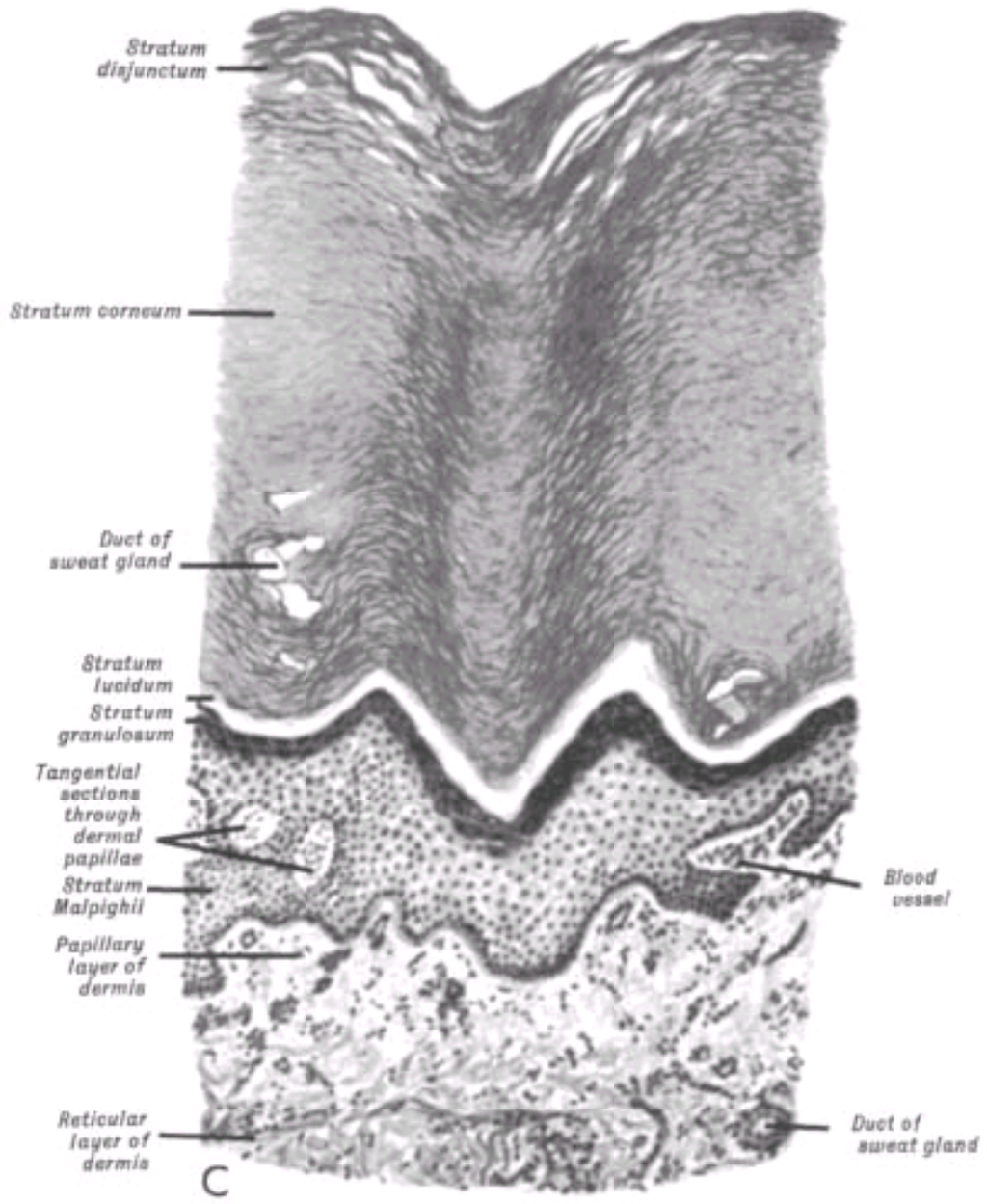


Figure 36-18b *C,* Section of human sole perpendicular to the free surface, ×100. *D,* Section through human thigh perpendicular to the surface of the skin. Blood vessels are injected and appear black. Low magnification. *(A Courtesy of H Mizoguchi. C and D after AA Maximow. From Bloom W, Fawcett DW: A Textbook of Histology, 10th ed. Philadelphia, WB Saunders, 1975. Reproduced by permission.)*



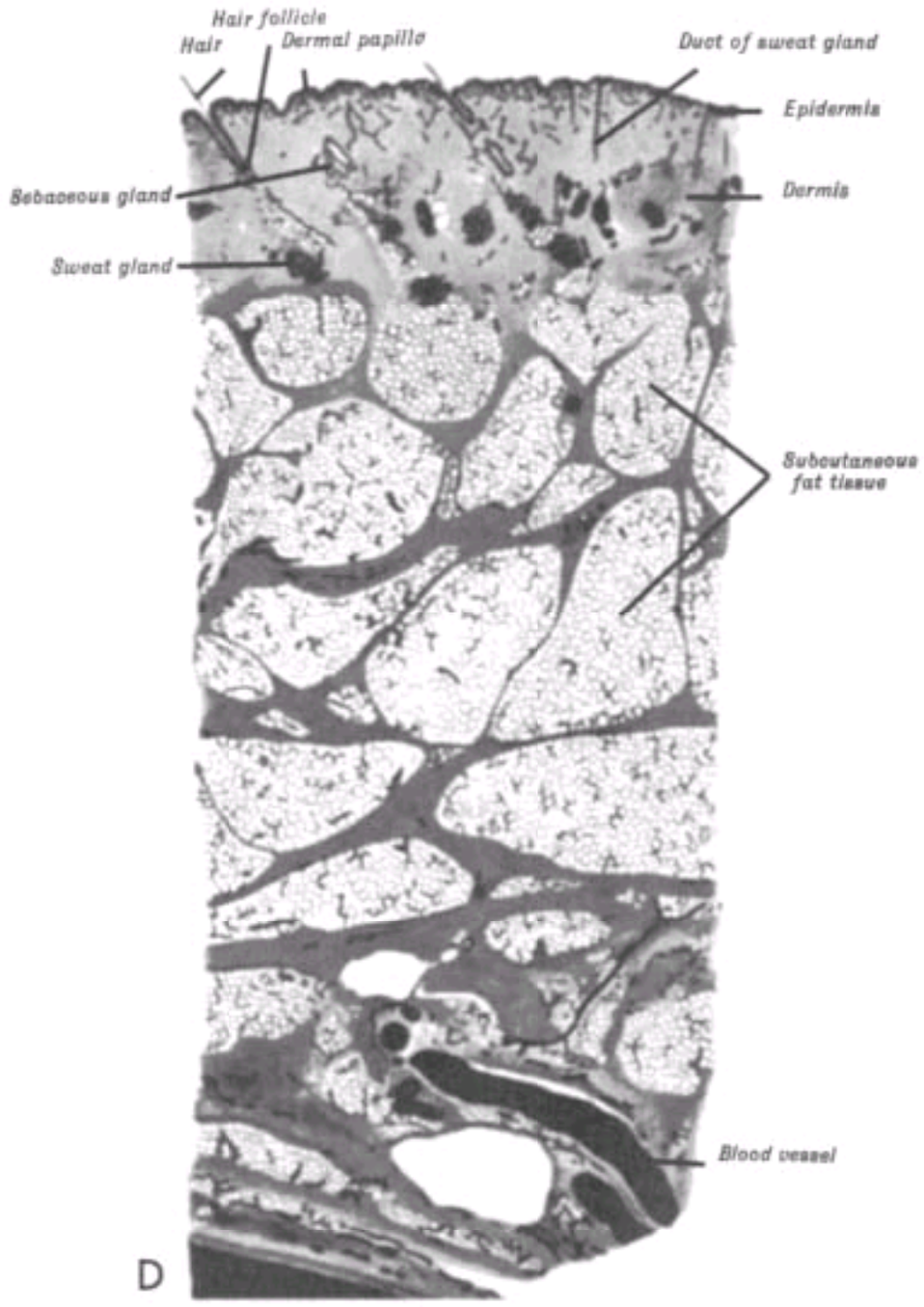


Figure 36-19 The thenar grip technique of handling the needle holder. The index finger is placed on the side of the needle holder, where it guides the placement of the needle. Neither the index nor the middle finger is placed in the ringlet hole. An alternate method (the thumb-ring finger grip) is shown in Figure 36-27 .

Figure 36-20 *A* and *B,* Inverted subcutaneous stitches.

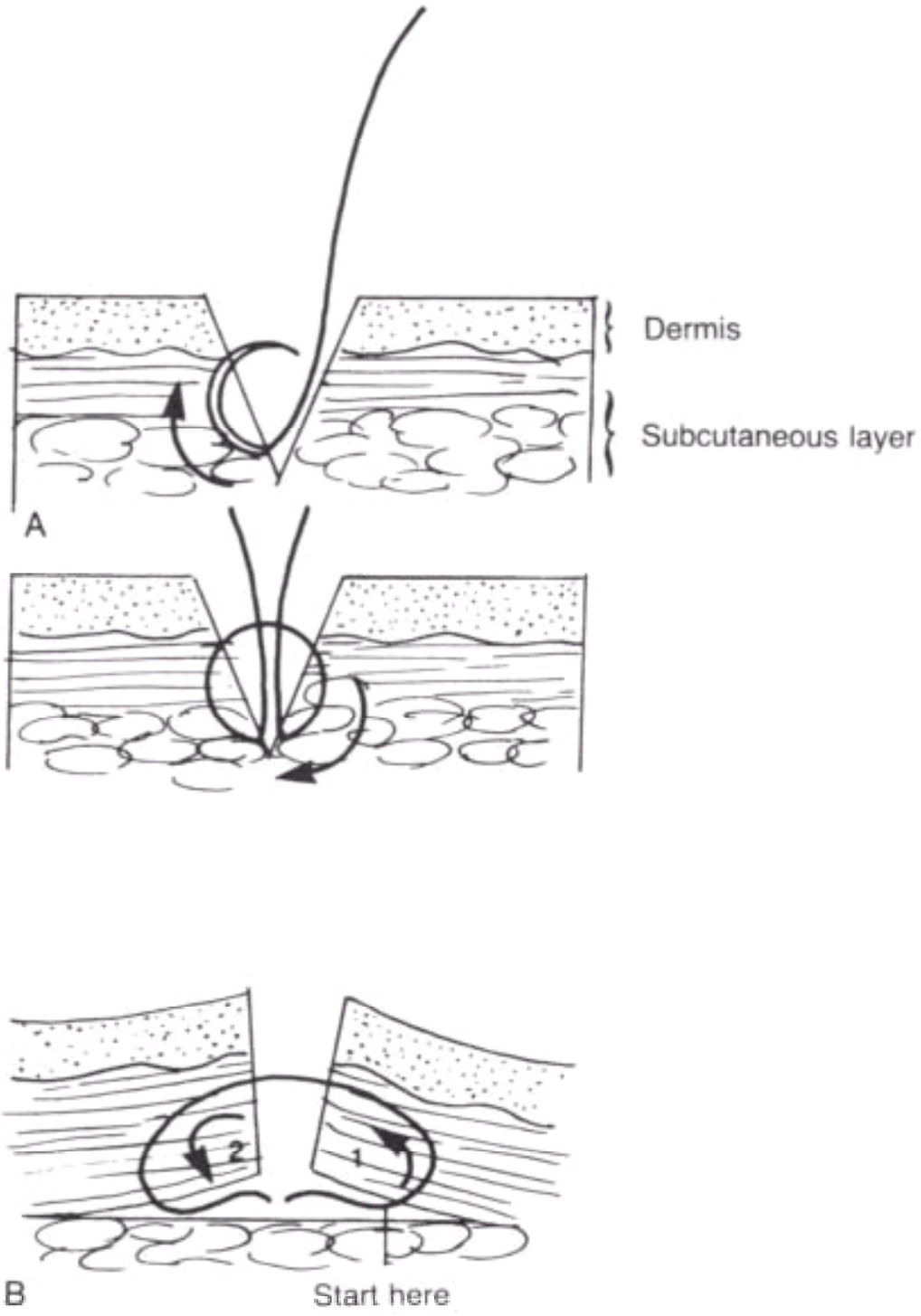


Figure 36-21 The two tails of the subcutaneous suture are pulled in the same direction, tightly opposing the edges of the wound.

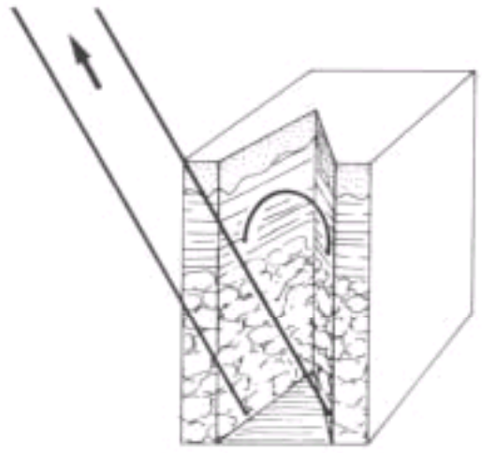


Figure 36-22 Cutting the tails of the subcutaneous suture. Note that the cutting blade is tilted away from the knot to avoid cutting it. *(Modified from Anderson CB: Basic surgical techniques. In Klippel AP, Anderson CB (eds): Manual of Outpatient and Emergency Surgical Techniques. Boston, Little, Brown, 1979. Reproduced by permission.)*

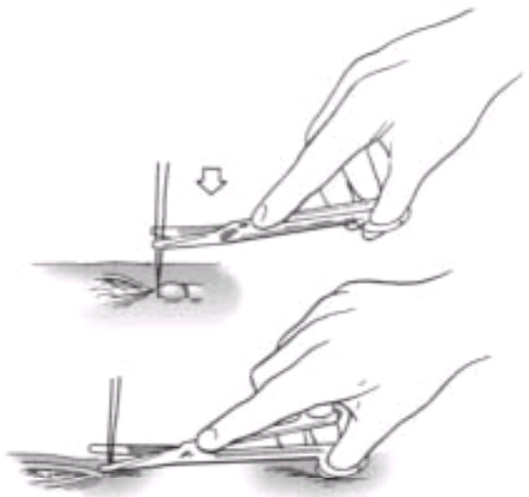


Figure 36-23 Horizontal dermal stitch. (A vertical suture also closes the deep tissue.)

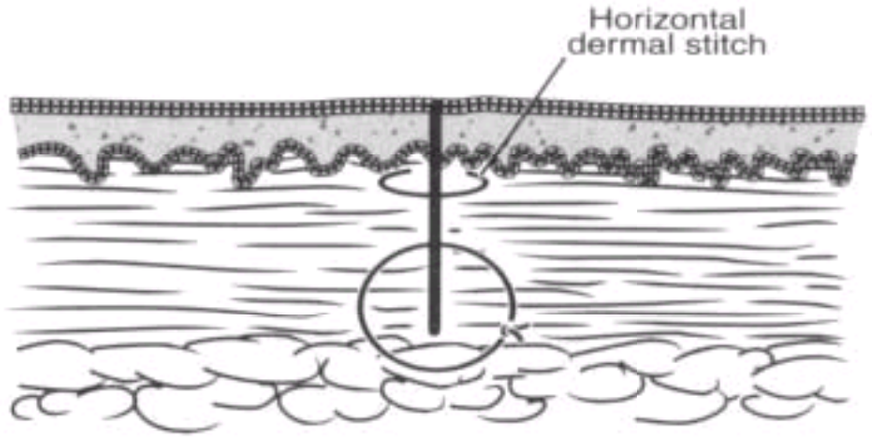


Figure 36-24 Closure of the surface of the wound in segments.

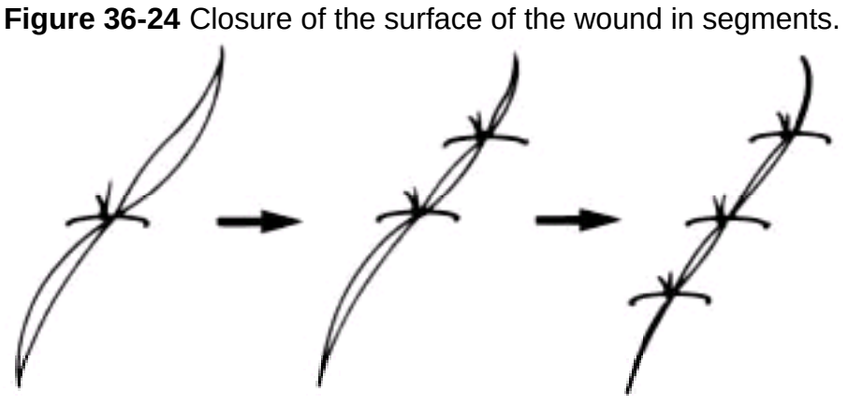


Figure 36-25 *A,* Too few stitches used. Note gaping between sutures. *B,* Too many stitches used. *C,* Correct number of stitches used for a wound under an average amount of tension.

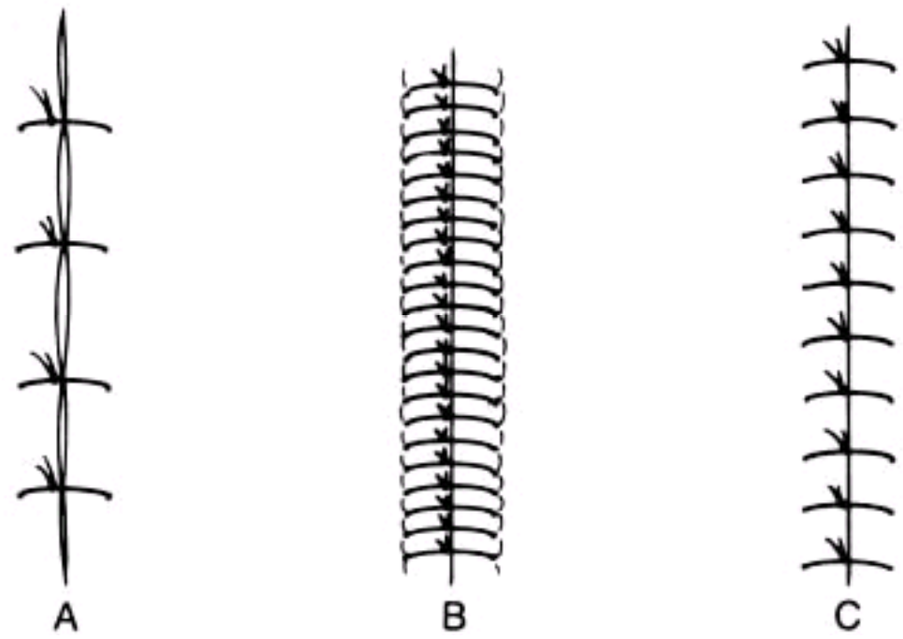


Figure 36-26 The simple suture. *A,* Hold the needle pointing down by excessively pronating the wrist so that the needle tip initially moves farther from the laceration as the needle penetrates deeper into the skin. Thus, there is more dermis in the depth of the wound than at the surface. Drive the needle tip downward and away from the cut edge, into the fat. *B,* Advance the needle into the laceration. The needle tip can be advanced directly into the opposite side. This can be achieved by rolling the needle holder as the needle enters the opposite side at the same level, and the arc pathway of the needle is controlled by retracting the skin edge. This causes more dermis to be incorporated into the depths than at the surface. As an alternative, if a small needle is used in thick skin or the distance across the wound is great, the needle can be removed from the first side, remounted on the needle holder, and advanced to the opposite side. *C,* Advance the needle upward toward the surface so that it exits at the same distance from the wound edge as on the contralateral side of the wound. Grasp the needle behind the tip and roll it out in the arc of the needle. *D,* The final position, with more tissue in the depth than the surface. The distance from each suture exit to the laceration is one half the depth of the dermis. *(Redrawn from Kaplan EN, Hentz VR: Emergency Management of Skin and Soft Tissue Wounds: An Illustrated Guide. Boston, Little, Brown, 1984, p 86. Reproduced by permission.)*

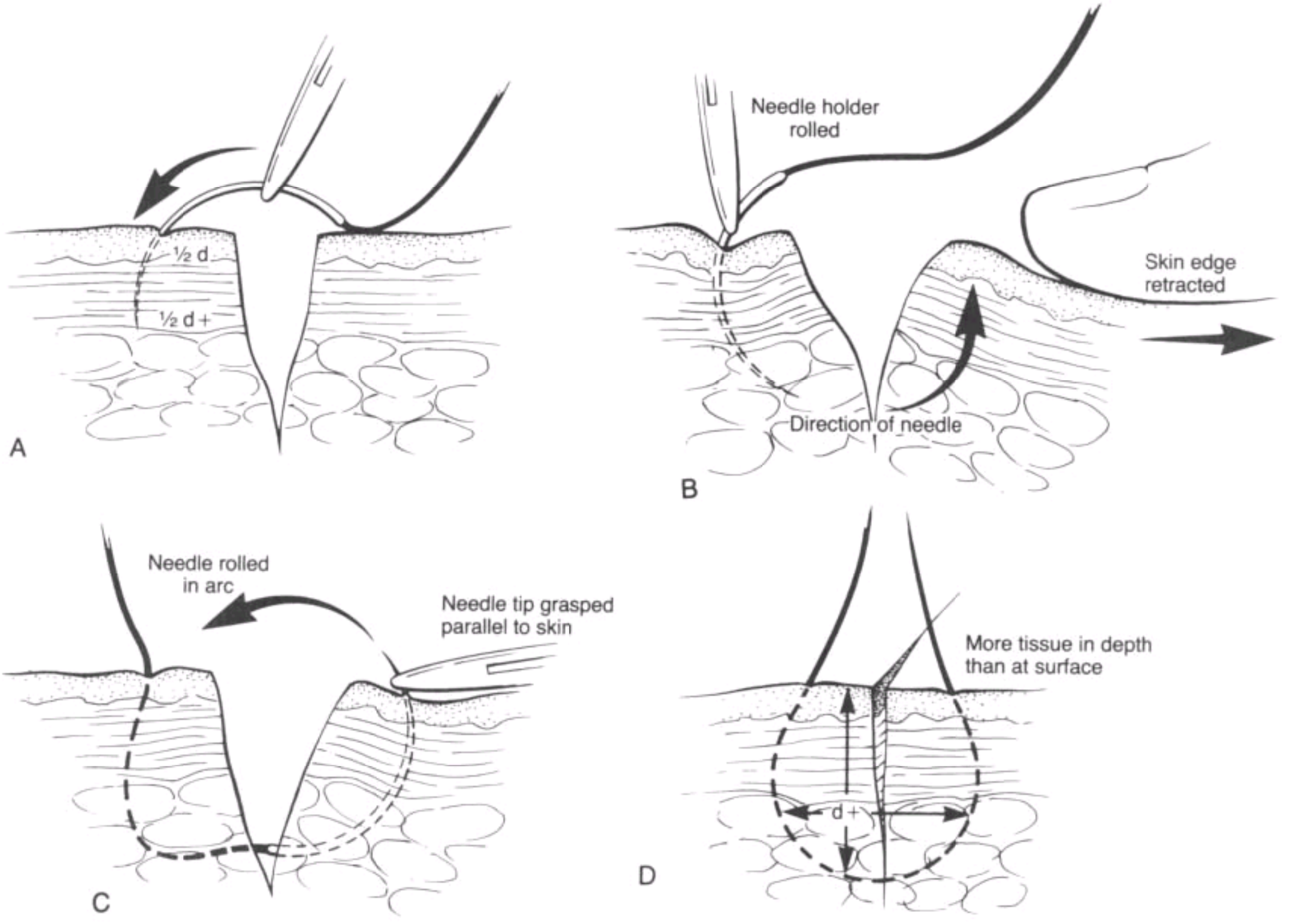


Figure 36-27 Motion of the needle holder. *(From Anderson CB: Basic surgical techniques. In Klippel AP, Anderson CB (eds): Manual of Outpatient and Emergency Surgical Techniques. Boston, Little, Brown, 1979. Reproduced by permission.)*

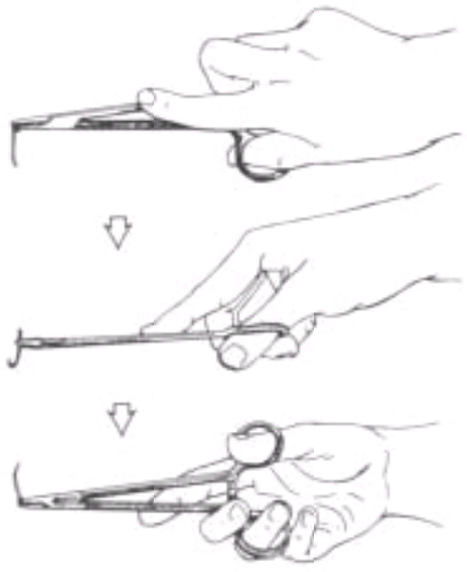


Figure 36-28 Skin edges that are everted will gradually flatten to produce a level wound surface. *(From Grabb WC: Basic technique of plastic surgery. In Grabb WC, Smith JW: Plastic Surgery: A Concise Guide to Clinical Practice. Boston, Little, Brown, 1979. Reproduced by permission.)*

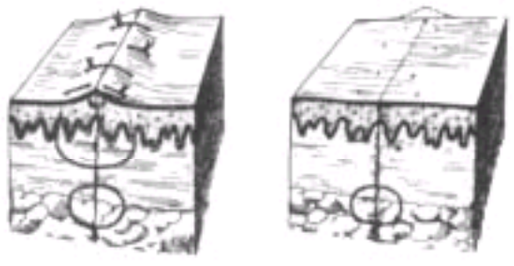


Figure 36-29 The use of a skin hook to evert the wound edge. This technique allows the operator to see the needle path, ensuring that the proper depth has been reached, and promotes eversion of the skin edges.

Figure 36-30 Eversion can often be obtained by slight thumb pressure. Care should be taken to avoid a needle stick, a common complication of this technique. *(From Converse JM: Introduction to plastic surgery. In Converse JM: Reconstructive Plastic Surgery: Principles and Procedures in Correction, Reconstruction, and Transplantation, vol 1, 2nd ed. Philadelphia, WB Saunders, 1977. Reproduced by permission.)*

Figure 36-31 The vertical mattress suture is the best technique for producing skin edge eversion. *A,* Usual type of mattress suture for approximating and everting wound edges. *B,* "Tacking" type of vertical mattress suture, extending into deep fascia to obliterate dead space under wound. Note that only a small bite of skin is included on the inner suture. *(Modified from Converse JM: Introduction to plastic surgery. In Converse JM: Reconstructive Plastic Surgery: Principles and Procedures in Correction, Reconstruction, and Transplantation, vol 1, 2nd ed. Philadelphia, WB Saunders, 1977. Reproduced by permission.)*

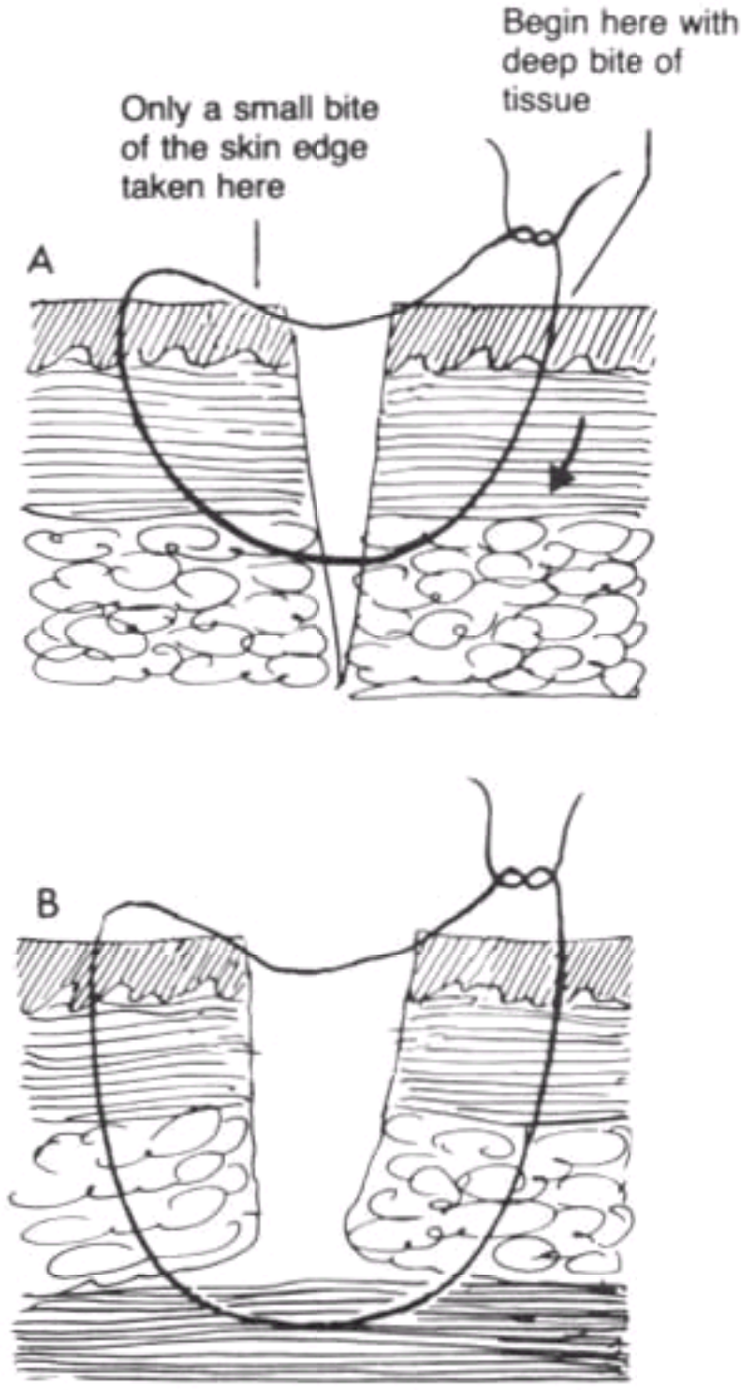


Figure 36-33 Simple interrupted stitch. Additional throws in a partially tied knot are not shown. *(From Grabb WC: Basic techniques of plastic surgery. In Grabb WC, Smith JW (eds): Plastic Surgery: A Concise Guide to Clinical Practice. Boston, Little, Brown, 1979. Reproduced by permission.)*

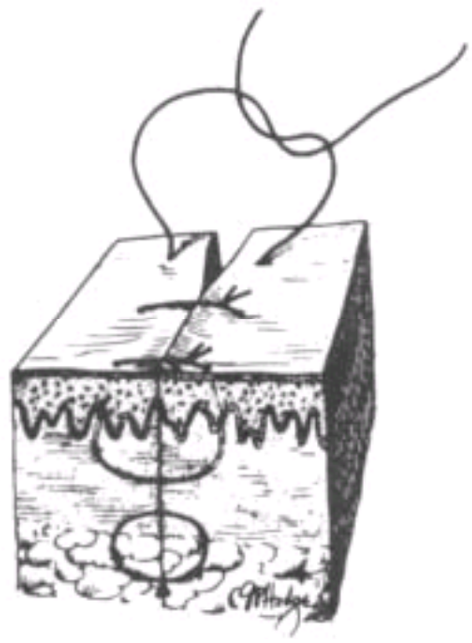


Figure 36-34 Simple continuous stitch. *(From Grabb WC: Basic techniques of plastic surgery. In Grabb WC, Smith JW (eds): Plastic Surgery: A Concise Guide to Clinical Practice. Boston, Little, Brown, 1979. Reproduced by permission.)*

Figure 36-35 Completing the simple continuous stitch. A series of square knots is tied, with the loop as one of the ties.

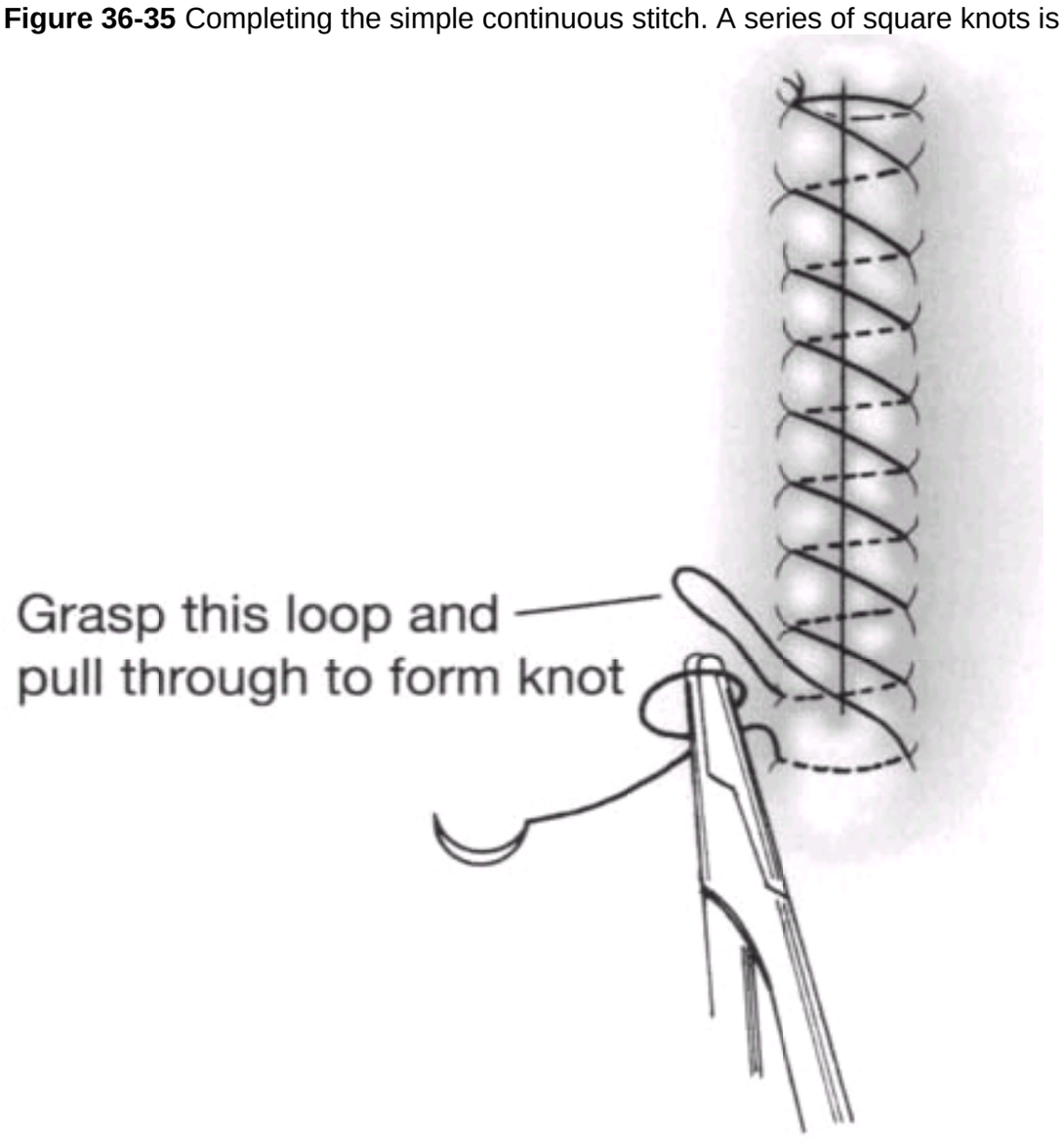


Figure 36-36 Continuous interlocking stitch. *(Modified from Suture Use Manual: Use and Handling of Sutures and Needles. Somerville, NJ, Ethicon, Inc, 1977. Reproduced by permission.)*

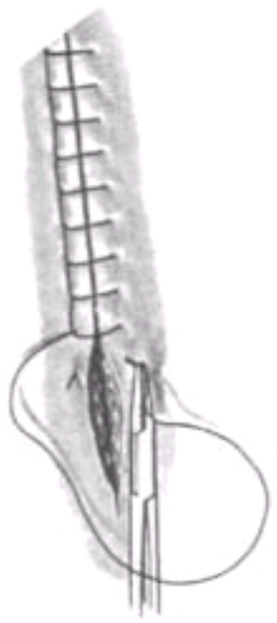


Figure 36-37 *A,* Pullout subcuticular stitch. The suture is introduced into the skin in line with the incision, approximately 1 to 2 cm away. *(From Grimes DW, Garner RW: "Reliefs" in intracuticular sutures. Surgical Rounds 1:46, 1978. Reproduced by permission.) B,* By backtracking each stitch slightly, one can produce a straight scar. *(From Grabb WC: Basic techniques of plastic surgery. In Grabb WC, Smith JW (eds): Plastic Surgery: A Concise Guide to Clinical Practice. Boston, Little, Brown, 1979. Reproduced by permission.)*

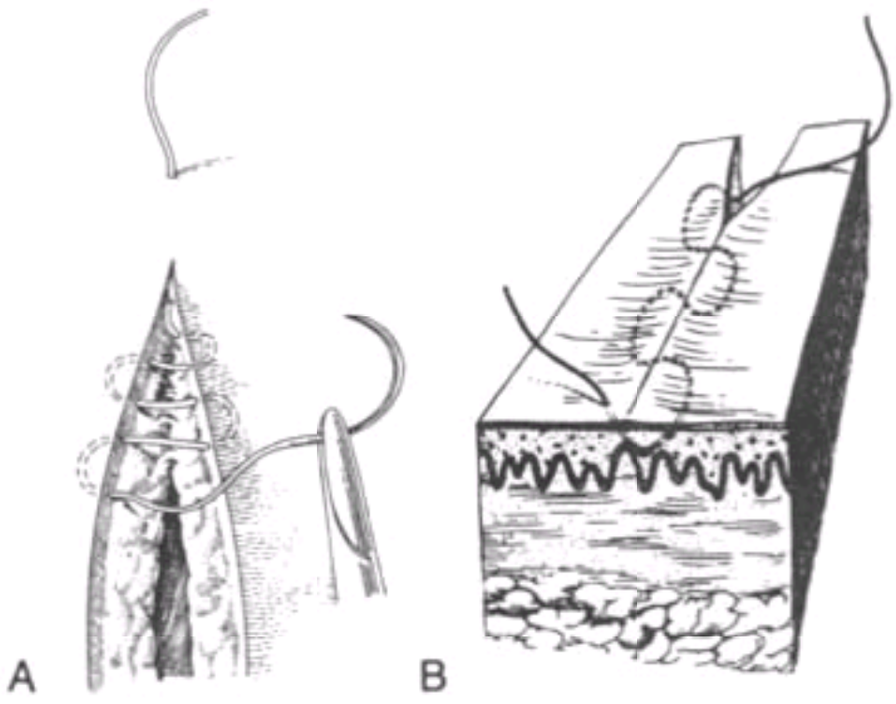


Figure 36-38 In constructing the relief to facilitate suture removal, the suture is crossed to the opposite side, going into the subcuticular area beneath the skin for approximately 2 cm before exiting *(A)*. The suture is then carried over the epidermis for approximately 2 cm *(B)* and then back under the dermis again *(C)*. Reentry is made into the wound area *(D)* at approximately the same location where the next "bite" would have been placed had the relief not been used. *(From Grimes DW, Garner RW: "Reliefs" in intracuticular sutures. Surgical Rounds 1:47, 1978. Reproduced by permission.)*

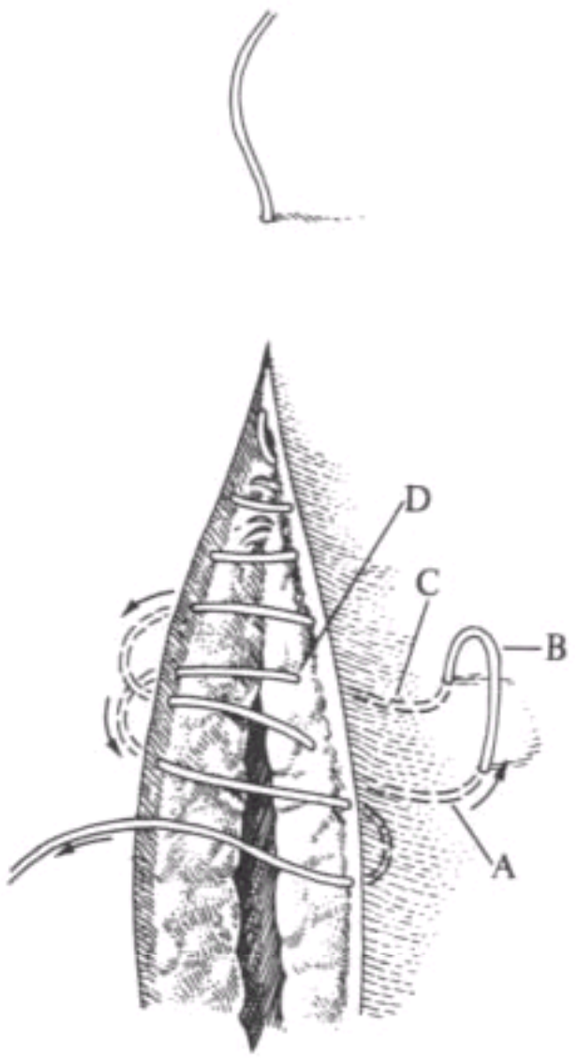


Figure 36-39 Subcuticular closure without epidermal penetration. *A,* The initial knot is secured in the dermal or subcutaneous tissue. *B,* The short strand is cut, and the needle is inserted into the dermis at the apex of the wound. *C,* The needle in the dermis, close to the corner of the wound and exiting the wound at the same horizontal level. *D,* After the subcuticular stitch has been completed, a knot is tied with the tail and the loop of the suture. *(Modified from Stillman RM: Wound closure: Choosing optimal materials and methods. ER Reports 2:43, 1981.)*

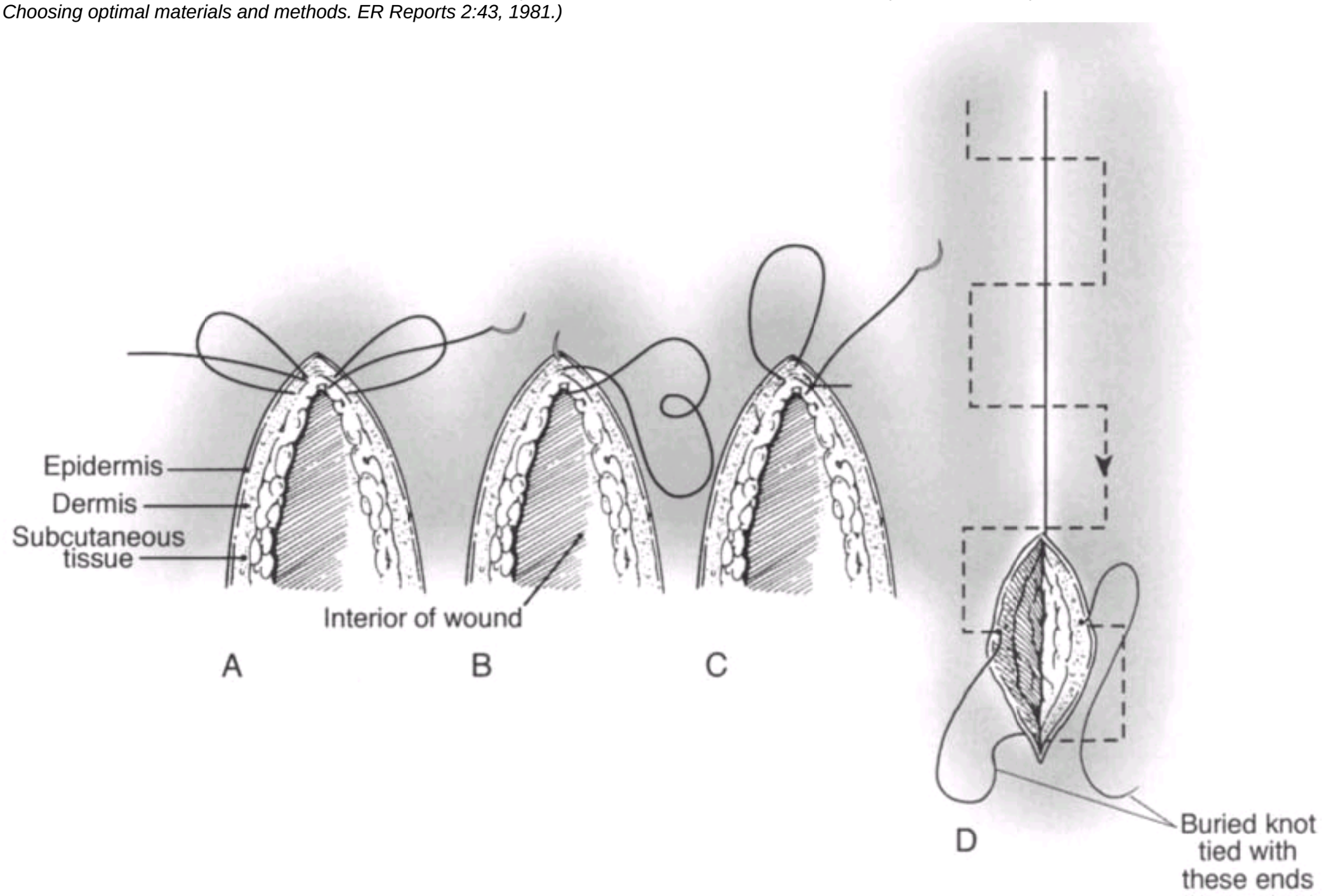


Figure 36-40 Interrupted subcuticular stitch (also called a *horizontal dermal stitch*). Absorbable sutures are used. A deep vertical suture is also shown.

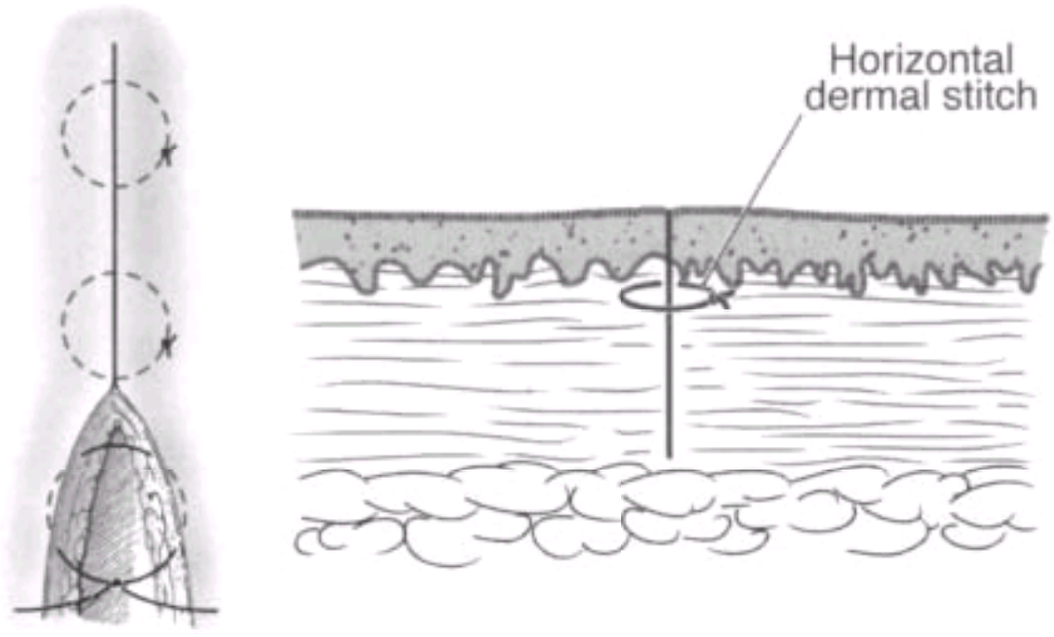


Figure 36-41 Vertical mattress stitch. The key to a tight closure is to place the inner sutures very close to the suture line (wound edge). *(From Grabb WC: Basic techniques of plastic surgery. In Grabb WC, Smith JW (eds): Plastic Surgery: A Concise Guide to Clinical Practice. Boston, Little, Brown, 1979. Reproduced by permission.)* See also Fig. 36-31 .

Figure 36-42 *A*, Horizontal mattress stitch. *B,* The dorsum of the hand, foot, or finger is an ideal place for a horizontal mattress suture to evert the wound edges. The relatively thin skin in these areas precludes the use of vertical mattress sutures. *(A from Grabb WC: Basic techniques of plastic surgery. Grabb WC, Smith JW (eds): Plastic Surgery: A Concise Guide to Clinical Practice. Boston, Little, Brown, 1979. Reproduced by permission.)*

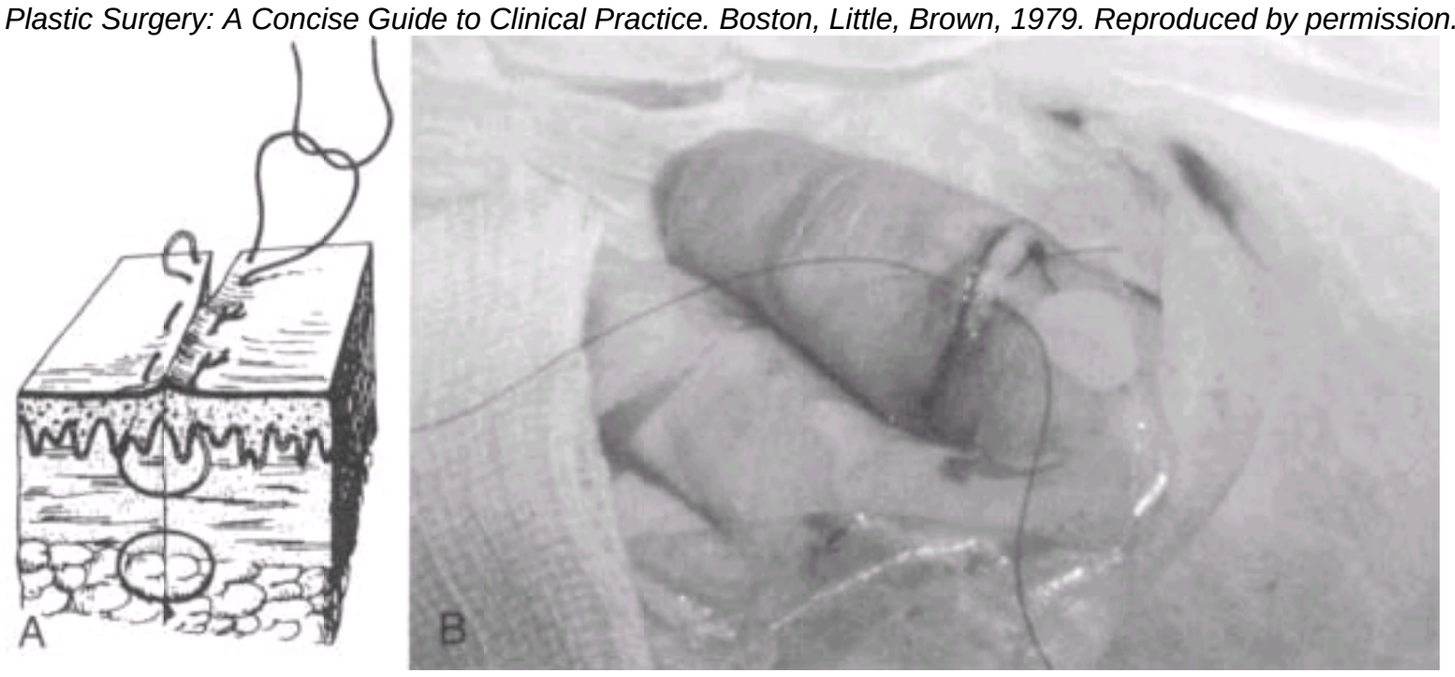


Figure 36-43 Half-buried horizontal mattress stitch. *(From Grabb WC: Basic techniques of plastic surgery. In Grabb WC, Smith JW: Plastic Surgery: A Concise Guide to Clinical Practice. Boston, Little, Brown, 1979. Reproduced by permission.)*

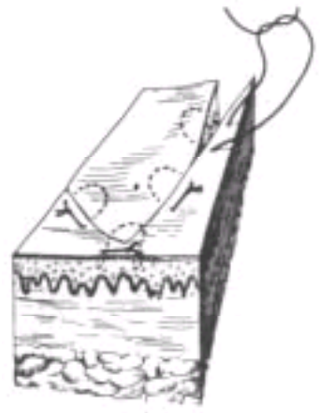


Figure 36-44 *A* and *B,* Approximation of a corner flap with a half-buried horizontal mattress stitch. Because of its applicability to this closure, the stitch is often called a *corner stitch*.

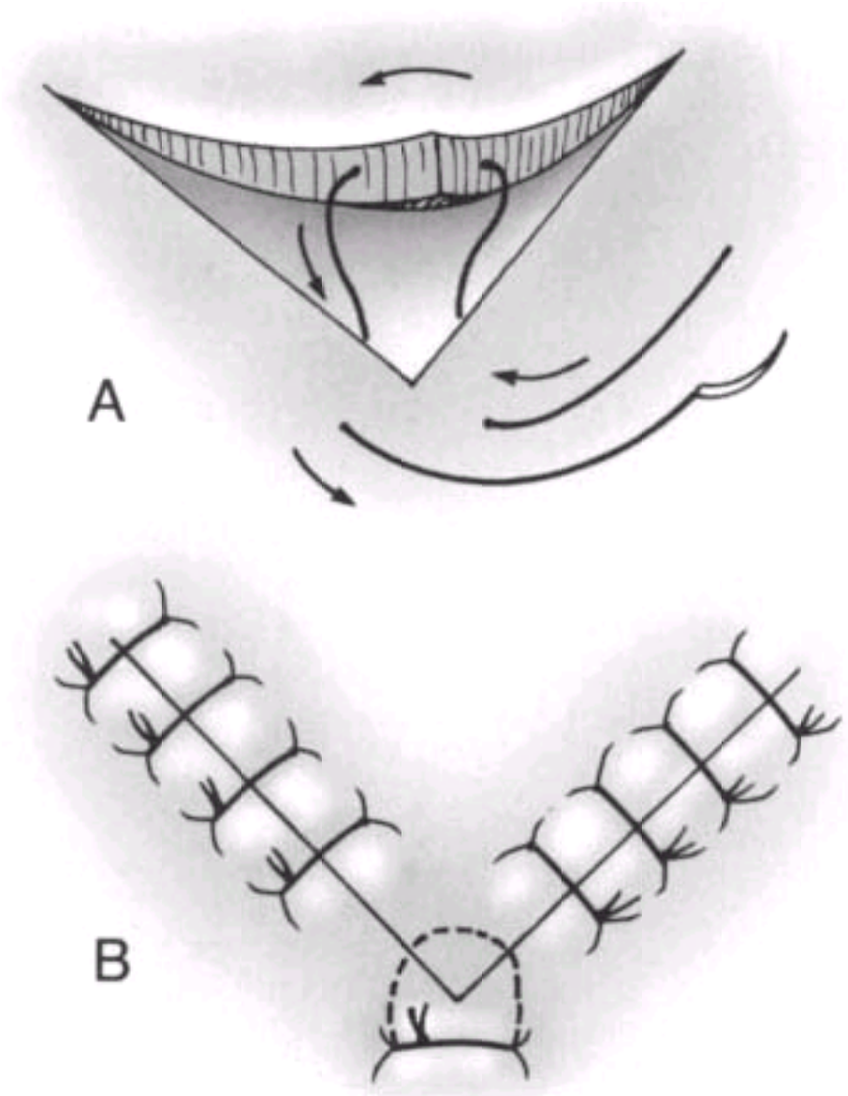


Figure 36-45 Figure-of-eight stitch-two methods. *(Modified from Dushoff IM: About face. Emerg Med 6:11:1974. Reproduced by permission.)*

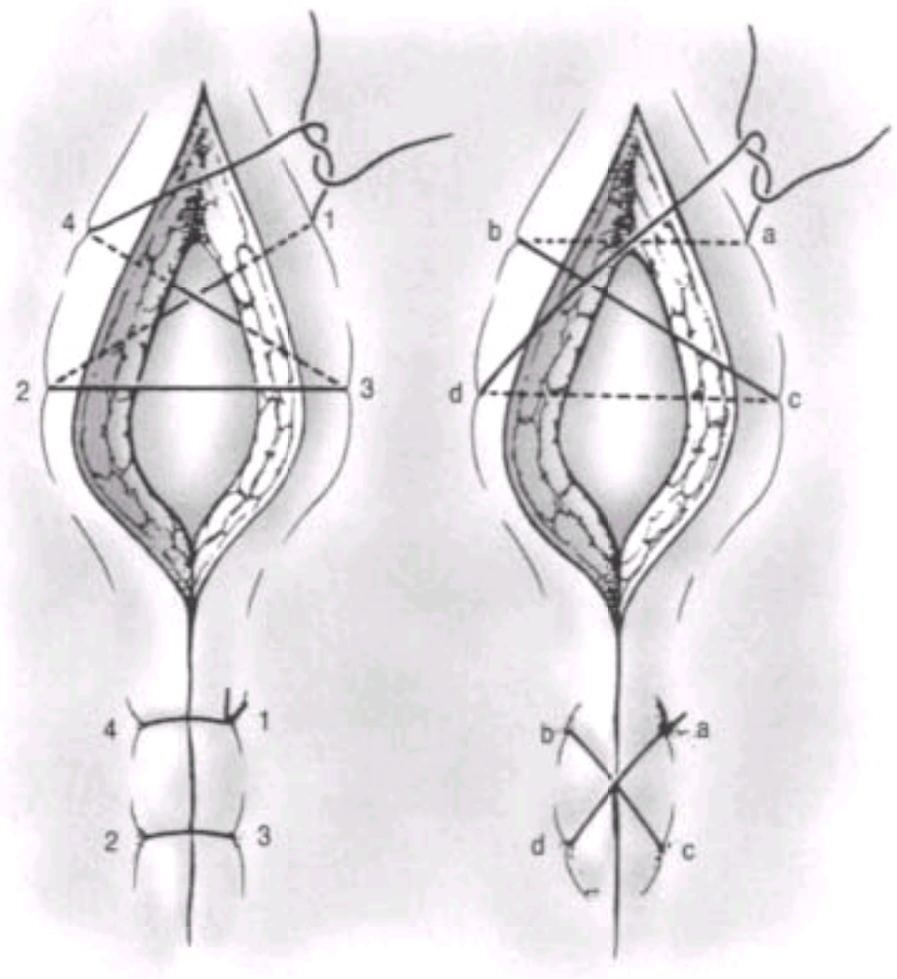


Figure 36-46 Vertical figure-of-eight suture technique. This can be used to close parallel lacerations. *(From Mitchell GC: Repair of parallel lacerations [letter]. Ann Emerg Med 16:924, 1987.)*

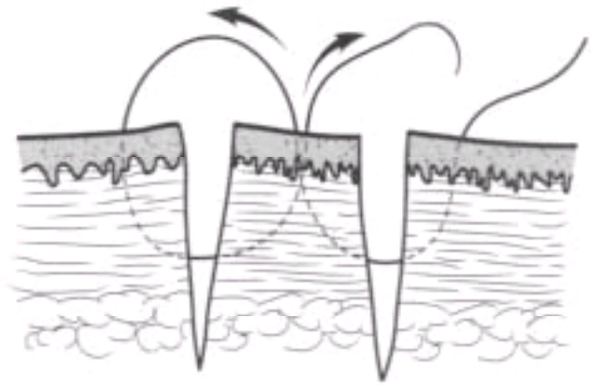


Figure 36-47 Techniques for closure of parallel lacerations. *A,* Central tissue island with intact base. *B,* Central tissue island shaved from base. *(Redrawn from Samo DG: A technique for parallel lacerations. Ann Emerg Med 17:297, 1988.)*

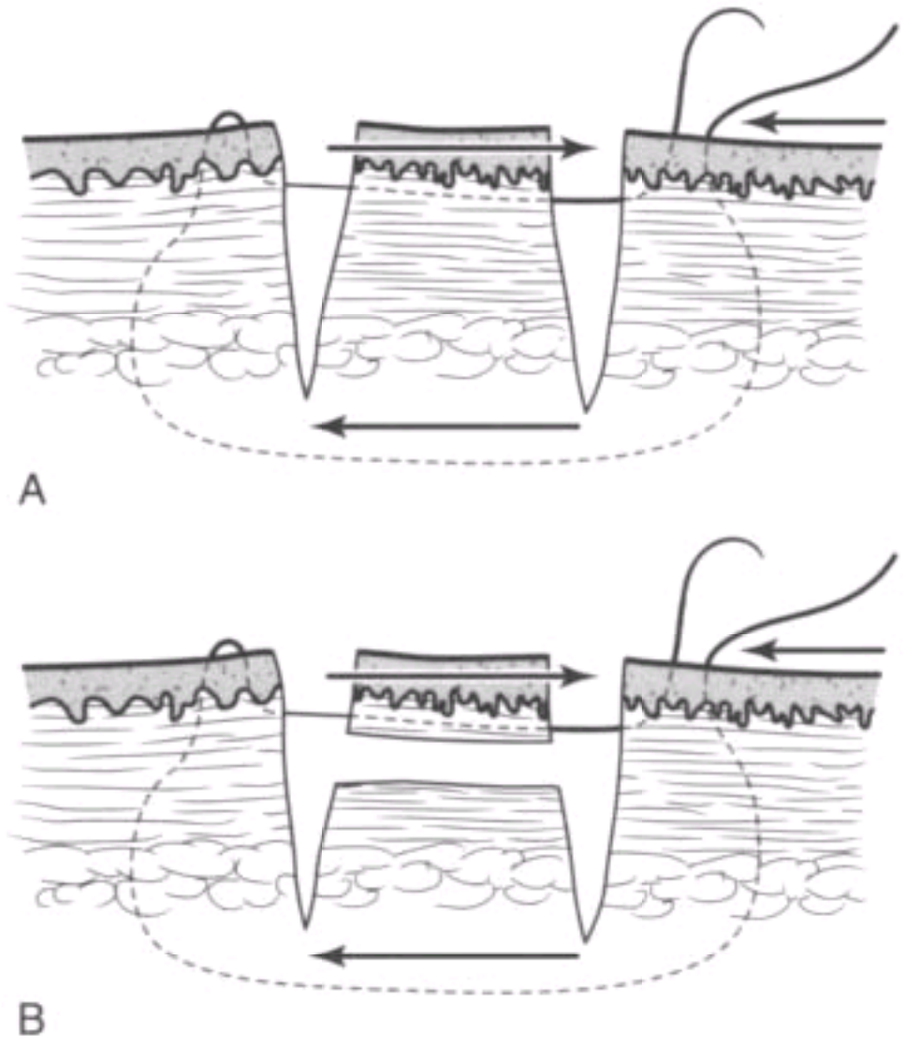
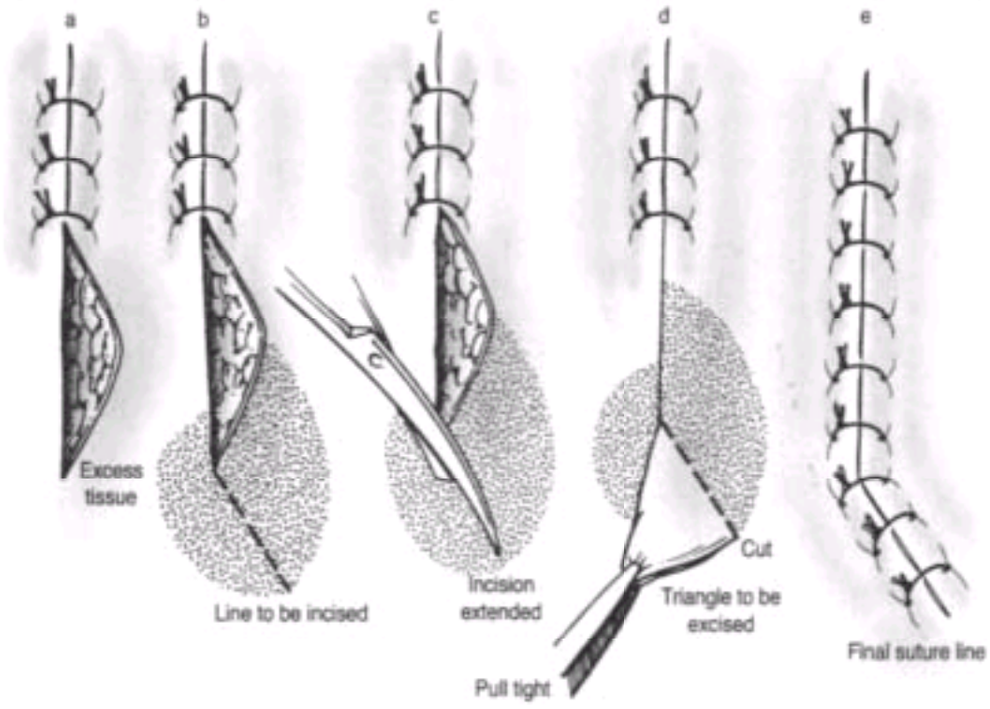
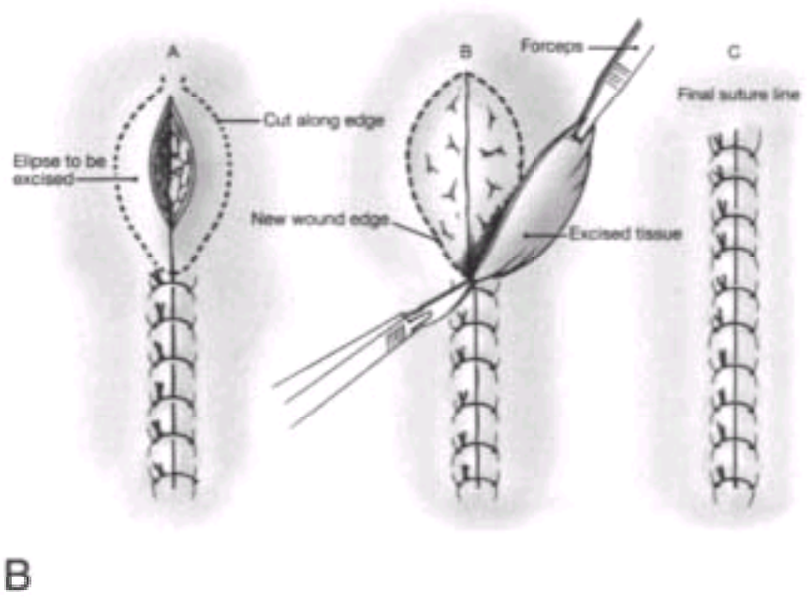


Figure 36-48 *A,* Correction of a dog-ear. *B,* Excision of bilateral dog-ears. *(A from Dushoff IM: A stitch in time. Emerg Med 5:1, 1973. Reproduced by permission.)*



A



B

Figure 36-49 *A* and *B,* V-Y advancement flap. *(From Rosen P, Sternbach G: Atlas of Emergency Medicine. Baltimore, Williams & Wilkins, 1979, p 132. Reproduced by permission.)*

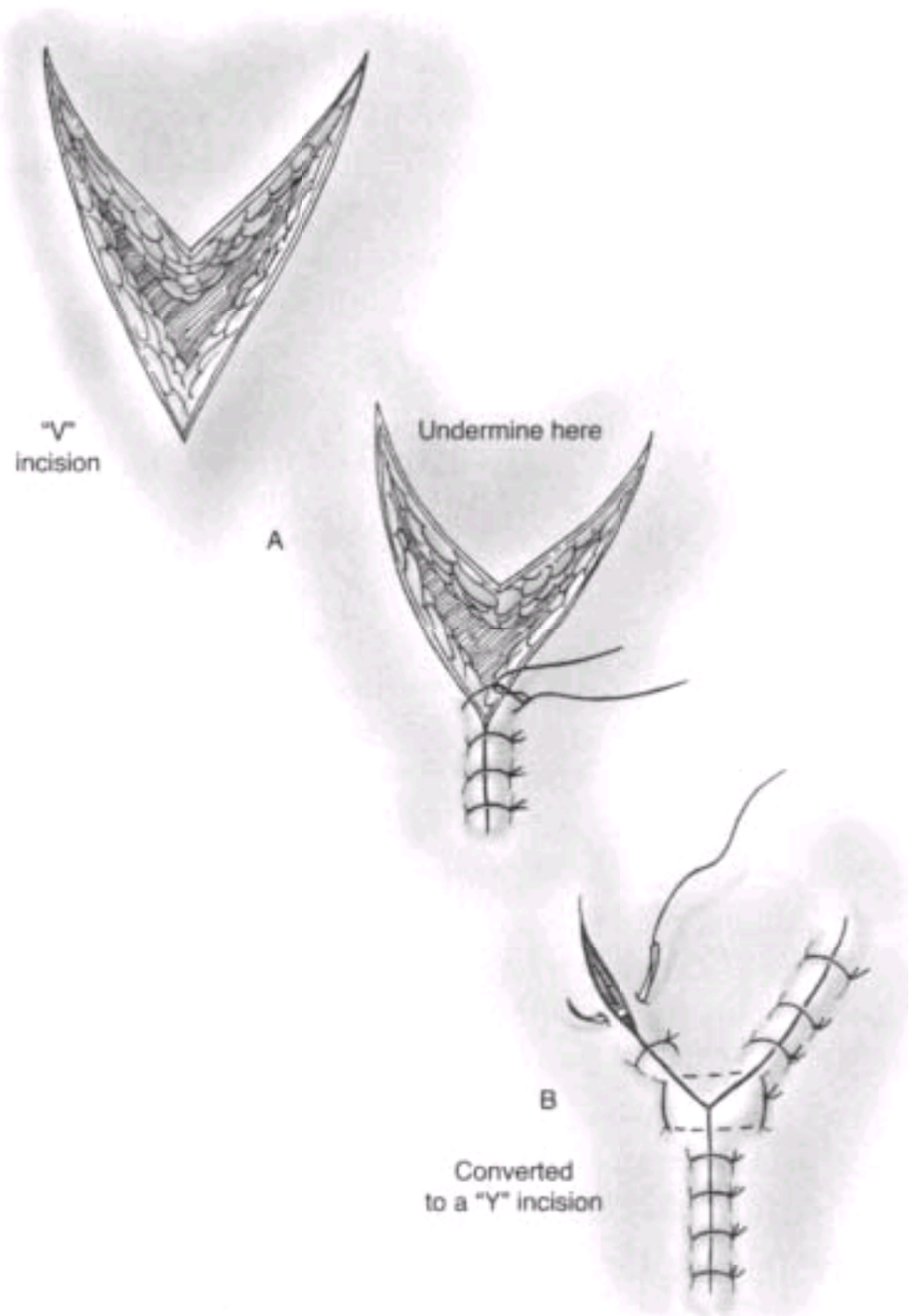


Figure 36-50 View from *above* stellate laceration, showing closure with half-buried mattress stitches.

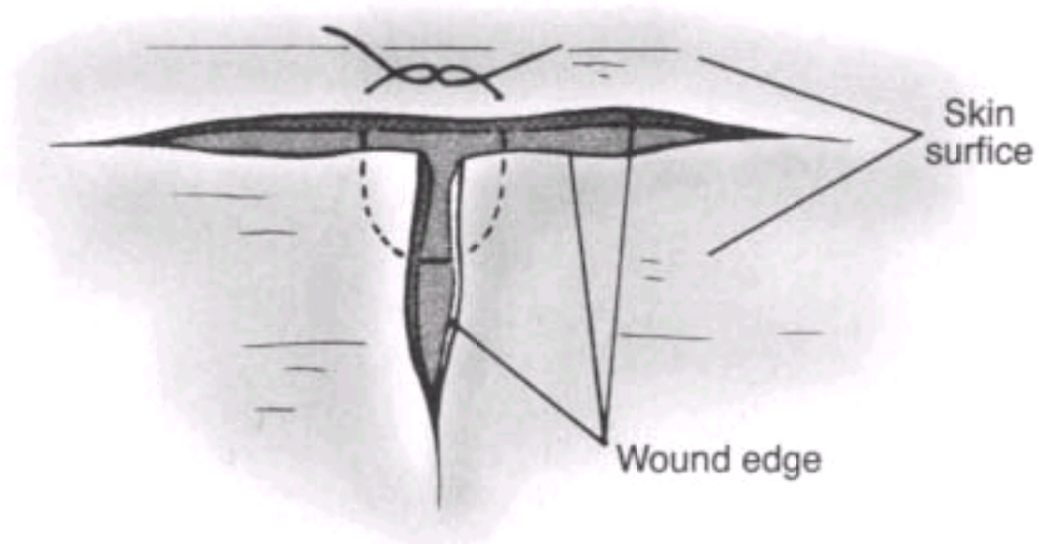


Figure 36-51 Lacerations following natural skin lines (shown here) heal with a less noticeable scar than those that are oblique or perpendicular to natural lines (or wrinkles).

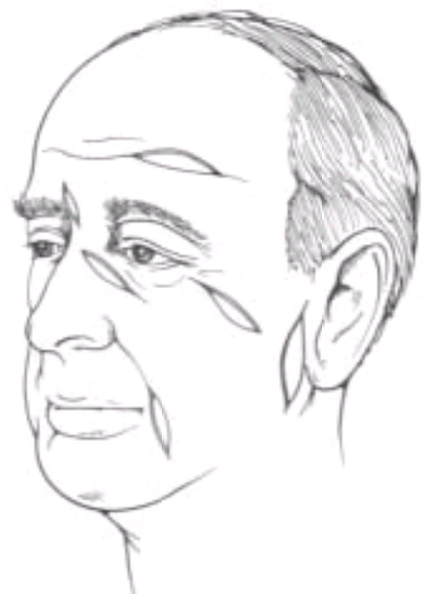


Figure 36-52 Wound closure tape can be used to provide additional support while sutures are in place and after they are removed. This may be especially useful in cosmetic areas, such as the face.

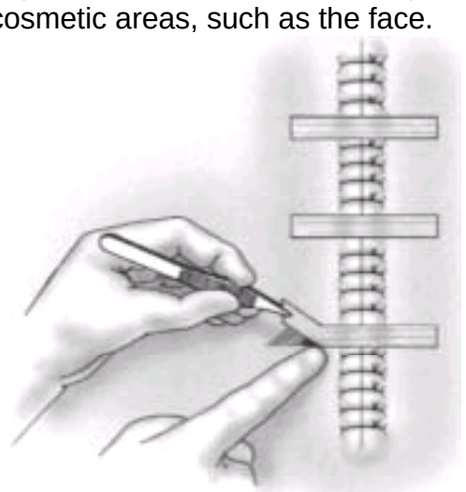
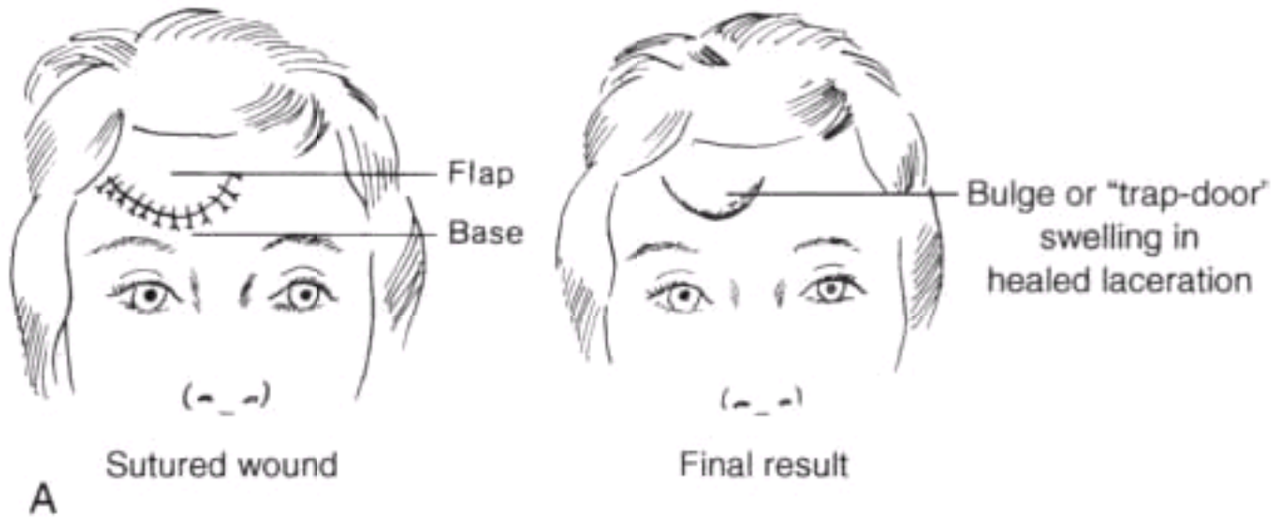


Figure 36-53 *A*, Elevation of a forehead flap. The "trap-door effect" is a natural healing process of elliptical or round lacerations. Patients should be advised of this phenomenon. *B,* This flap-type laceration of the knee will heal with a puffed-up center (trap door), even under the best of circumstances. *(A from Grabb WC, Kleinert HE: Technics in Surgery: Facial and Hand Injuries. Somerville, NJ, Ethicon, Inc., 1980. Reproduced by permission.)*



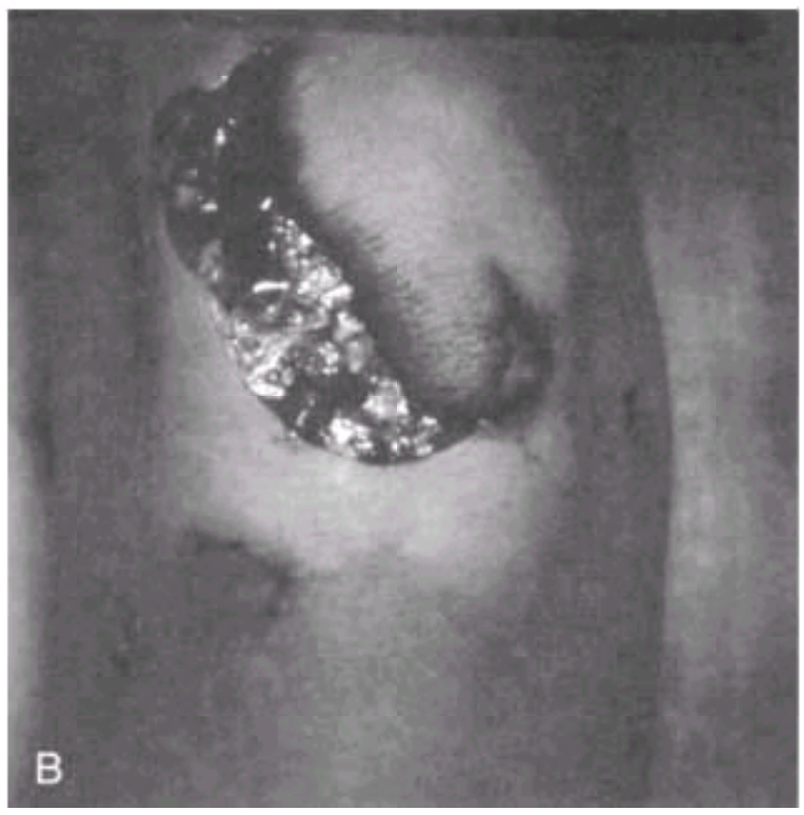


Figure 36-54 *A,* Repair of a U-shaped flap laceration with a superiorly oriented base to minimize the trap door effect. *A,* Excision of edges. *B,* undermining. *C,* Approximation of SQ tissue on the flap to SQ tissue at a deeper level on the base; *D,* skin closure. *B* and *C,* When a laceration in the thin skin of the forehead borders the thicker skin of the scalp, a horizontal mattress suture with an intradermal component can enhance healing by bringing tissues to the same plane. These figures show eversion of thinner skin to obtain adequate approximation with thicker scalp tissue. *(B from Converse JM: Introduction to plastic surgery. In Converse JM (ed): Reconstructive Plastic Surgery: Principles and Procedures in Correction, Reconstruction, and Transplantation, vol 1, 2nd ed. Philadelphia, WB Saunders, 1977. Reproduced by permission.)*

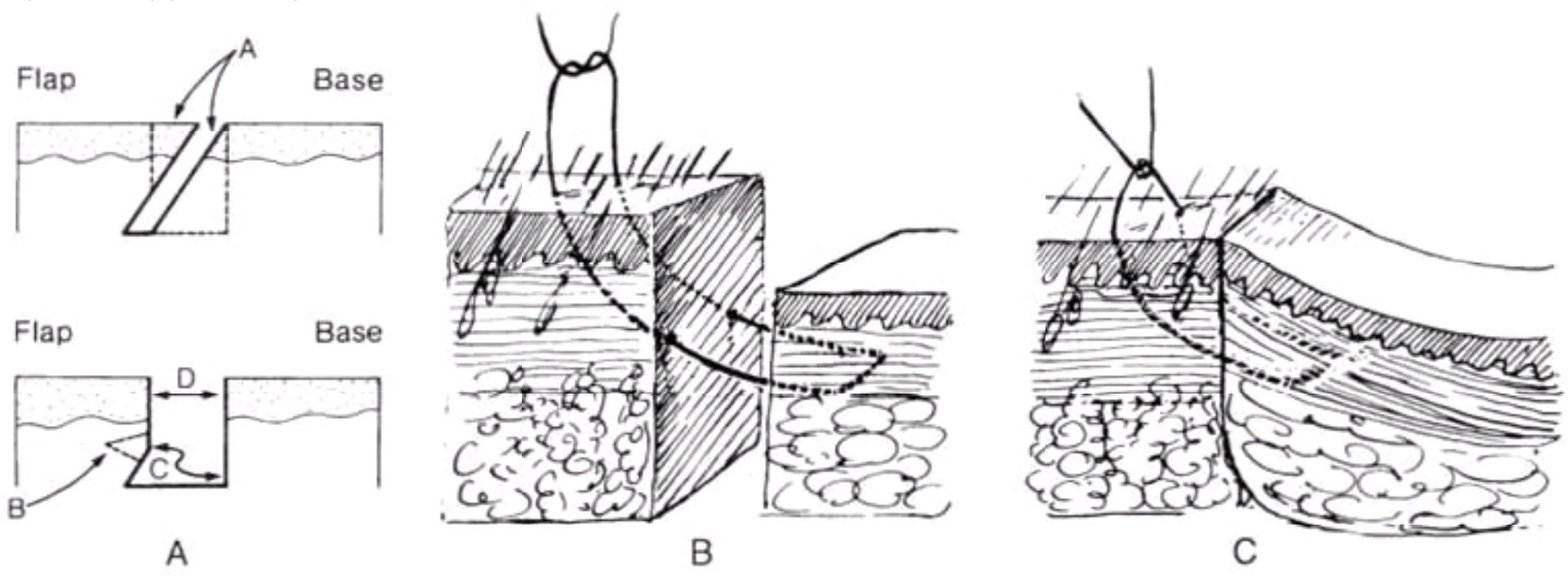
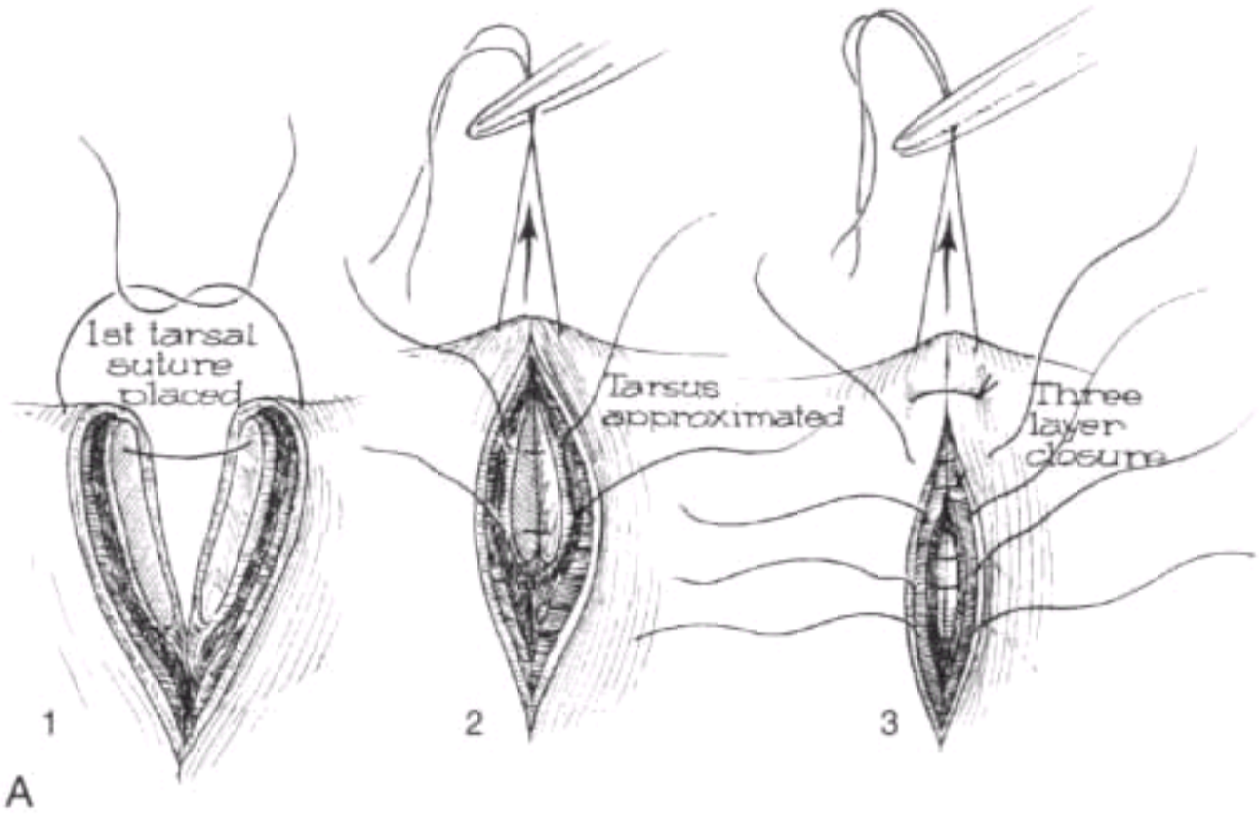
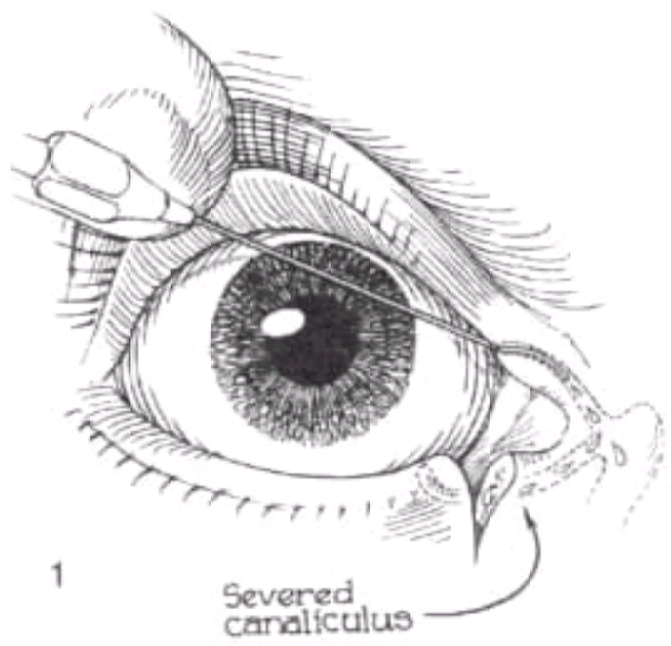
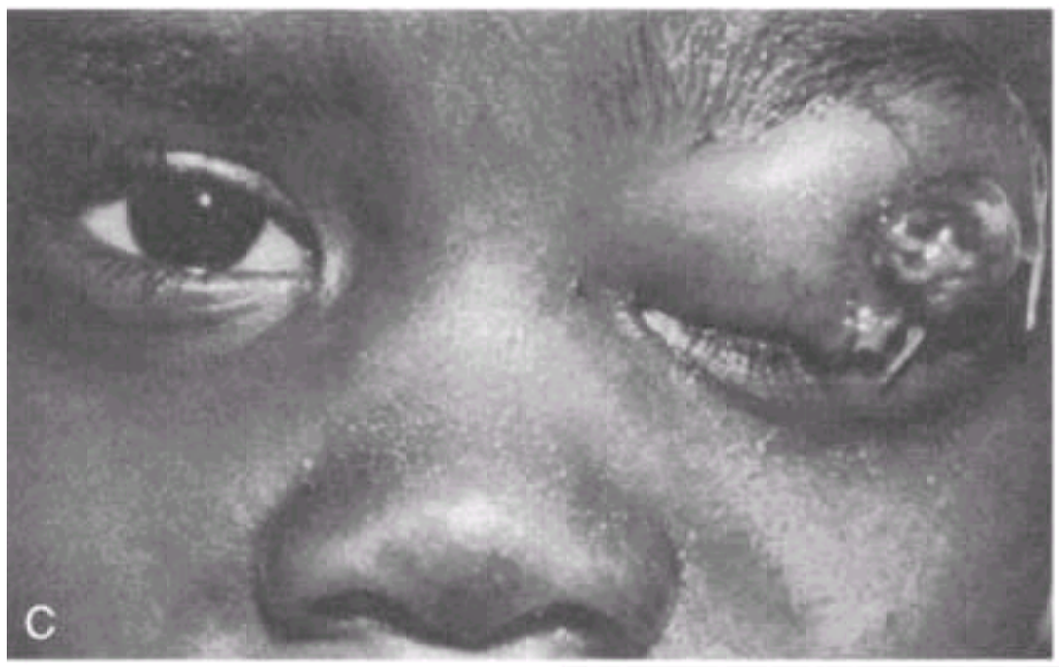
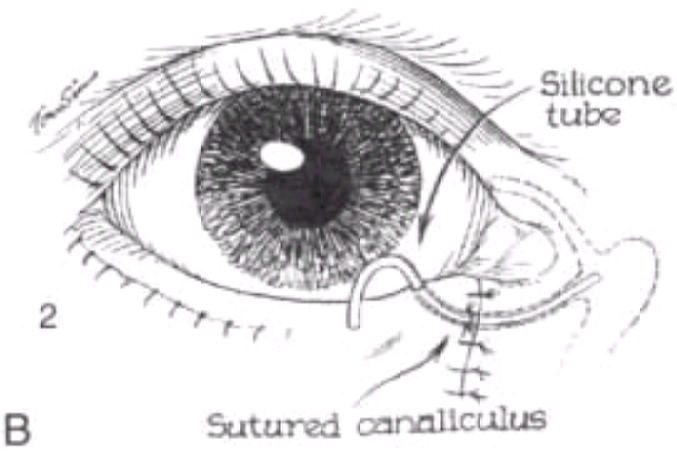


Figure 36-55 *A,* Repair of a simple lid laceration. The first sutures are placed at the lid margin so that the lid can be extended by traction with a hemostat. Interrupted absorbable sutures are used to close the tarsus (1), followed by separate closure of the muscle layer with absorbable sutures (2), and finally by closure of the skin with interrupted 8-0 black silk or synthetic sutures (3). Such a repair should not be performed by the novice. *B*, A method of identifying and repairing the canaliculus. This repair is best left to the ophthalmologist, but recognizing the potential for a canaliculus injury is the task at hand in the emergency department. *C,* Deep laceration of the left upper lid with herniation of orbital fat. For fat to prolapse, the orbital septum (and potentially the globe itself) must have been perforated. This is a wound requiring operating room exploration and repair. *D*, Lacerations of the ear require a special repair aimed at covering cartilage and preventing hematoma formation. With this through-and-through laceration of the margin of the pinna, the cartilage is trimmed just enough to allow the skin to be approximated to cover all exposed cartilage. The repair is easiest if the posterior pinna is sutured first.









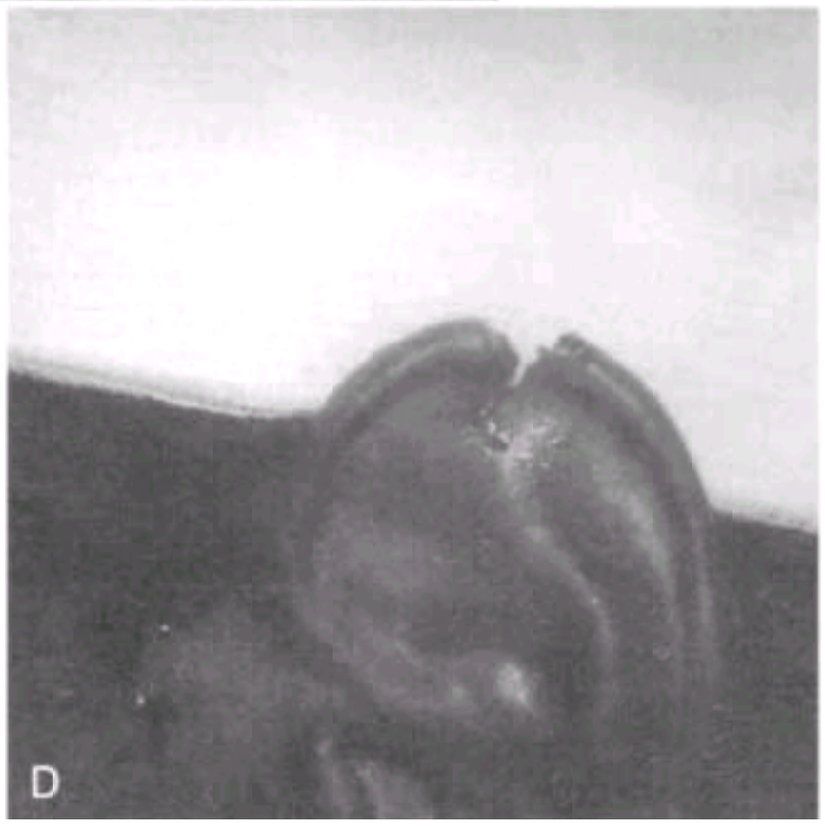


Figure 36-56 *A,* In the repair of lip lacerations, the first stitch should be placed at the vermilion-cutaneous border to obtain proper alignment. *(A from Grabb WC, Kleinert HE: Technics in Surgery: Facial and Hand Injuries. Somerville, NJ, Ethicon, Inc., 1980. Reproduced by permission.)*

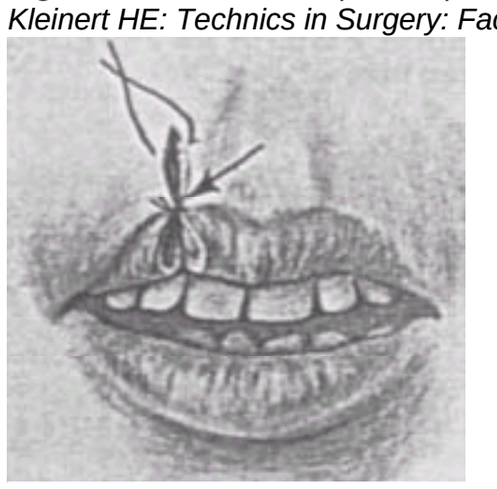


Figure 36-57 Through-and-through injuries and lacerations of the tongue margins require sutures to achieve anatomic healing. Dexon, Vicryl, or silk sutures are ideal for suturing the tongue surface. Bleeding is usually controlled with direct pressure and local infiltration of lidocaine with epinephrine. For through-and-through lacerations, the muscle layer should be closed separately (with absorbable sutures) to prevent hematoma formation. In general, buried sutures are better tolerated by the patient.

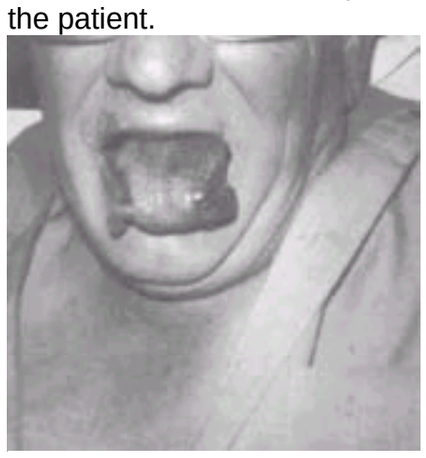


Figure 36-58 *A*, Anatomy of the scalp. Note that the skin, superficial fascia, and galea are adherent and constitute the outer layer. Blood vessels in the fascia are the major source of the blood loss noted in scalp lacerations. *B,* To temporarily control bleeding from vessels in the fascia, the galea can be everted to compress the fascia. *C,* The galea has been transected in wounds that gape open like this one, and to achieve hemostasis and obtain the best closure, the galea should be sutured. This is most easily accomplished with the use of a long needle, forceps, and 3-0 sutures that incorporate the skin, SQ tissue, and galea in a single bite *(D)*. In this figure, the needle is passing through the galea from the underside, having traversed all three layers on the other side of the laceration. If this technique is used, individual buried sutures in the galea are not required, and hemostasis is ensured. At the base of this wound is the periosteum, a tissue-like covering of the skull. In *C*, the galea is actually adherent to the avulsed flap; the anesthetic needle is touching the underside of the galea. A simple laceration that does not gape open *(E)* means the galea is intact. It can be easily closed with superficial sutures or staples.

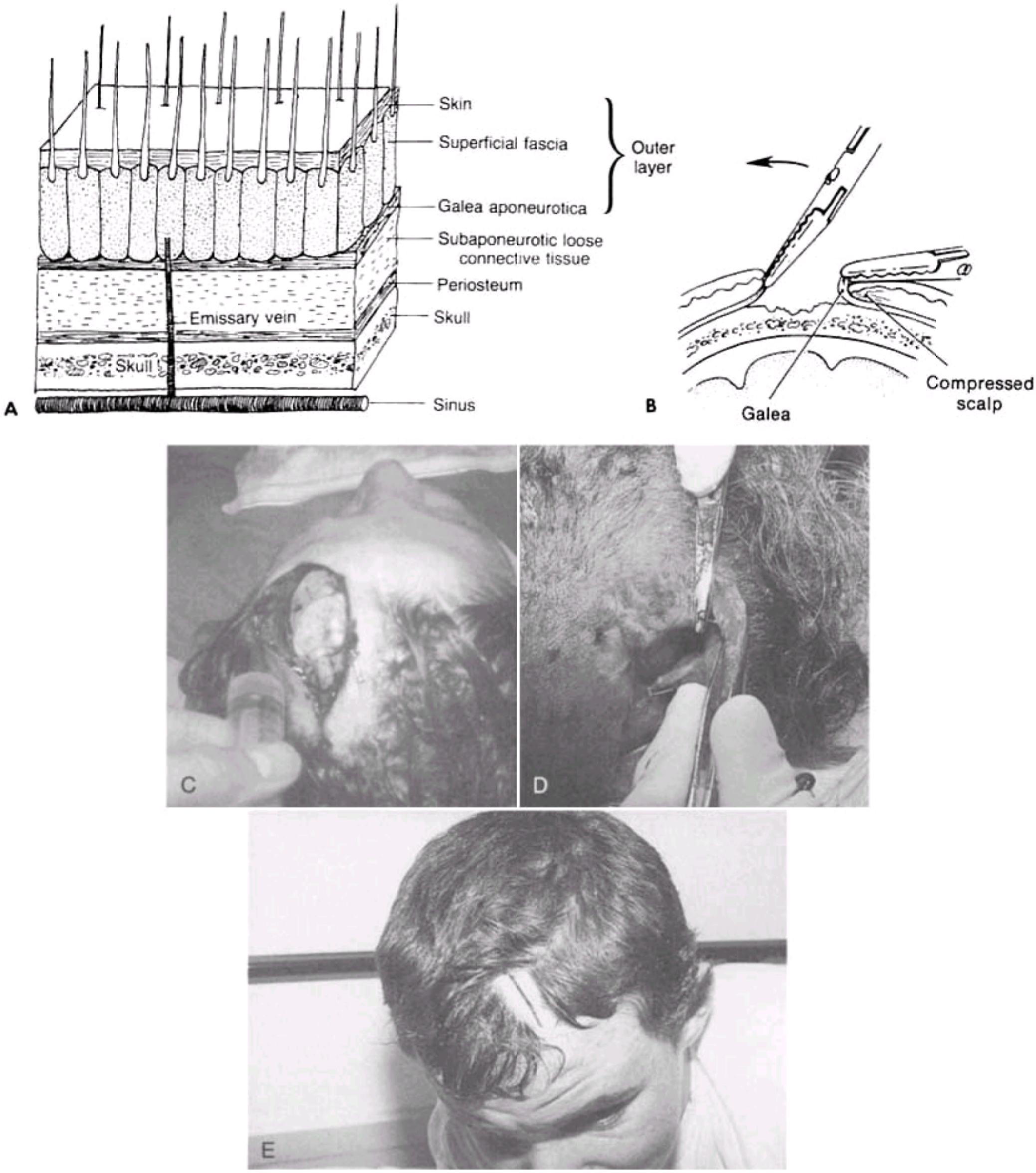


Figure 36-59 Large partial scalp avulsion.

Figure 36-60 *A,* To achieve hemostasis of a scalp laceration, a wide, tight, sterilized rubber band or Penrose drain may be placed around the forehead and occiput. This compresses the arterial supply to the scalp. *B,* Alternatively the wound margins can be temporarily clamped to control hemorrhage. Raney scalp clips and accompanying instrument for application to scalp wound edges are shown.

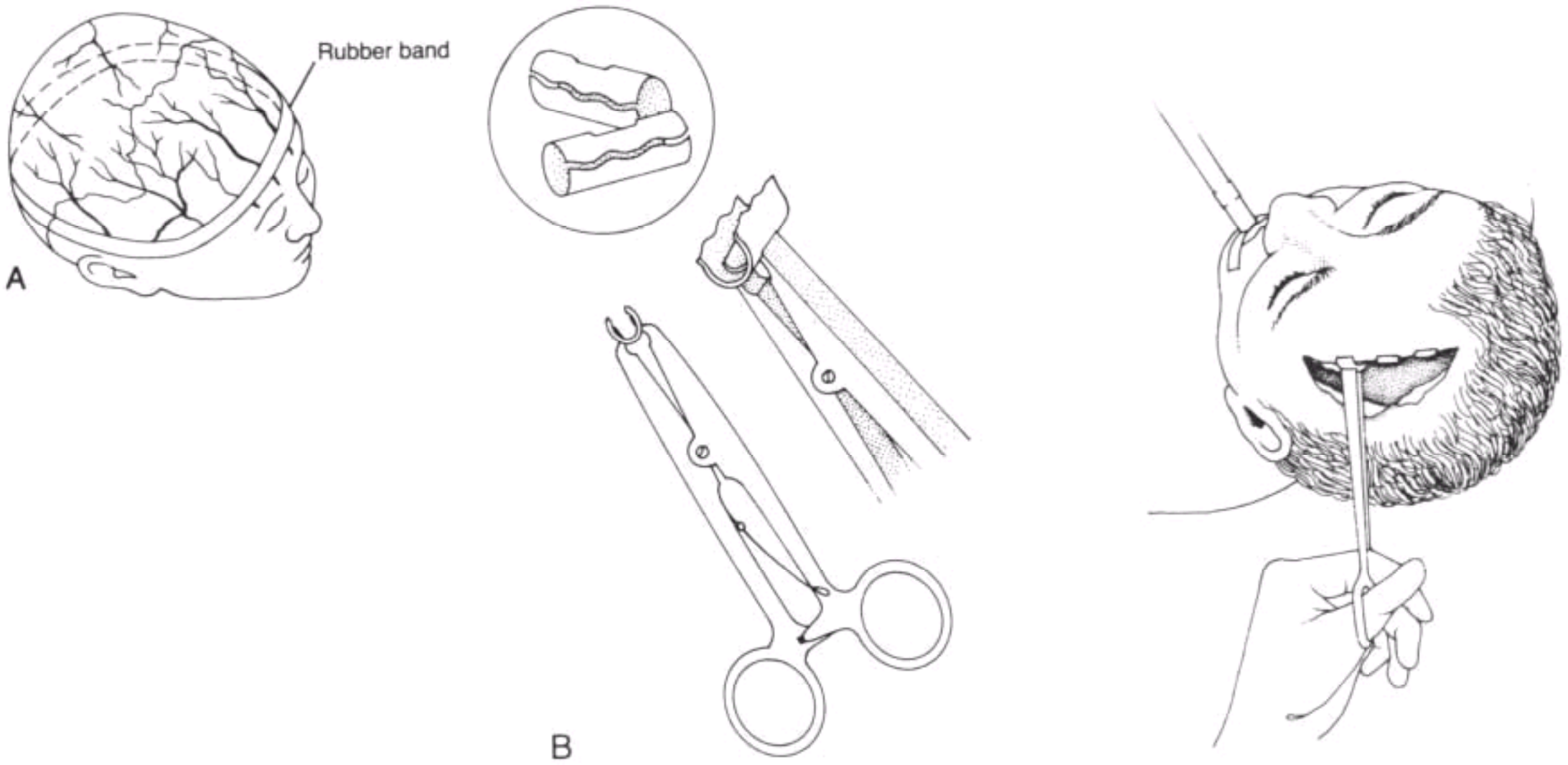


Figure 36-61 Anatomy of the fingernail. The fingernail rests on the nailbed, also termed the *matrix*. The distal nail covers the sterile matrix; the proximal nail arises from and covers the germinal matrix. The tissue adherent to the proximal dorsal nail is the eponychium (also termed the *cuticle*), and the potential space between the nail and the eponychium is the nail fold.

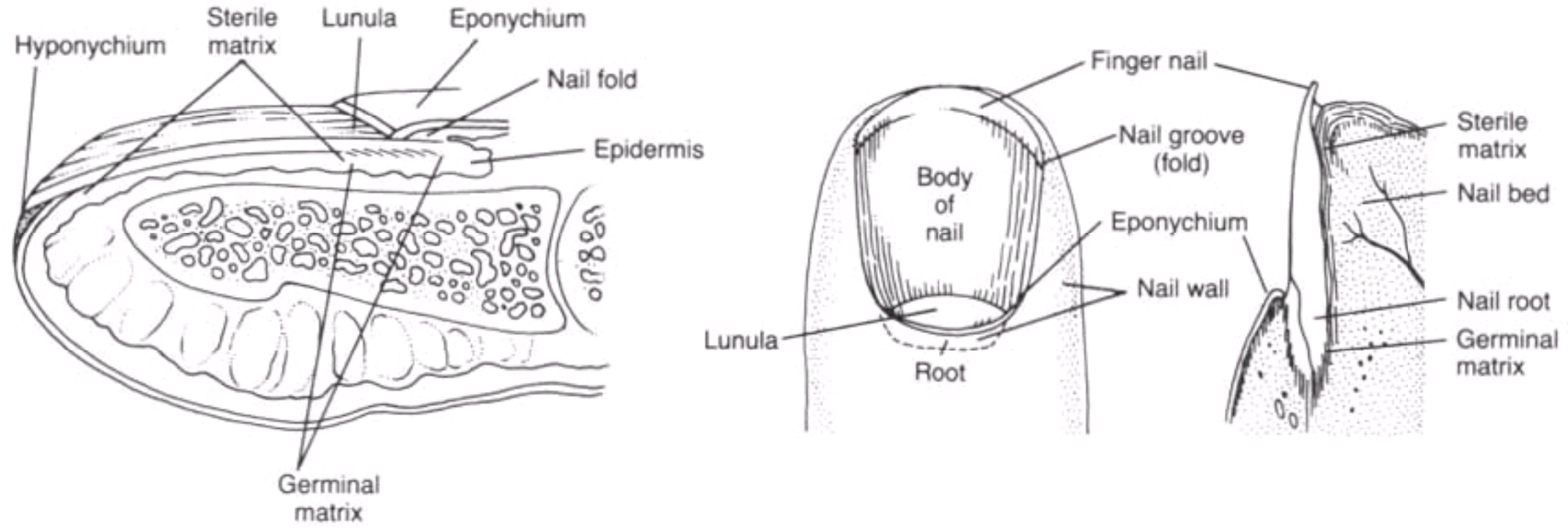


Figure 36-62 This subungual hematoma occupies about two thirds of the nail and should be drained by simple nail trephination. The injury does not require nailbed repair, because the nail is still firmly attached to the matrix. Even though there must be a nail matrix laceration (the source of the bleeding), the cosmetic result will be excellent. The presence of an underlying digital tuft fracture does not change management (see also description of nail trephination in Chapter 38).

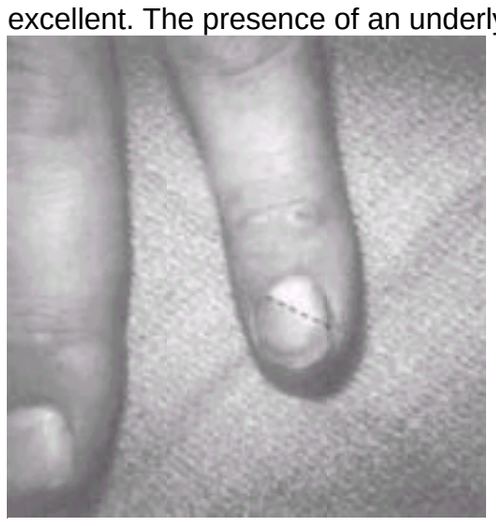


Figure 36-63 This fingernail was avulsed at the base *(A)*, a common result of having a door slam on the digit. Since the nail is mobile *(B)* and there is subungual bleeding, the nail can be removed and the nailbed inspected. Any large laceration should be meticulously repaired. Absorbable sutures size 6-0 or smaller should be used. After repair, the nail is replaced under the eponychium (cuticle). See Figure 36-68 for a simple technique for removal of the fingernail.

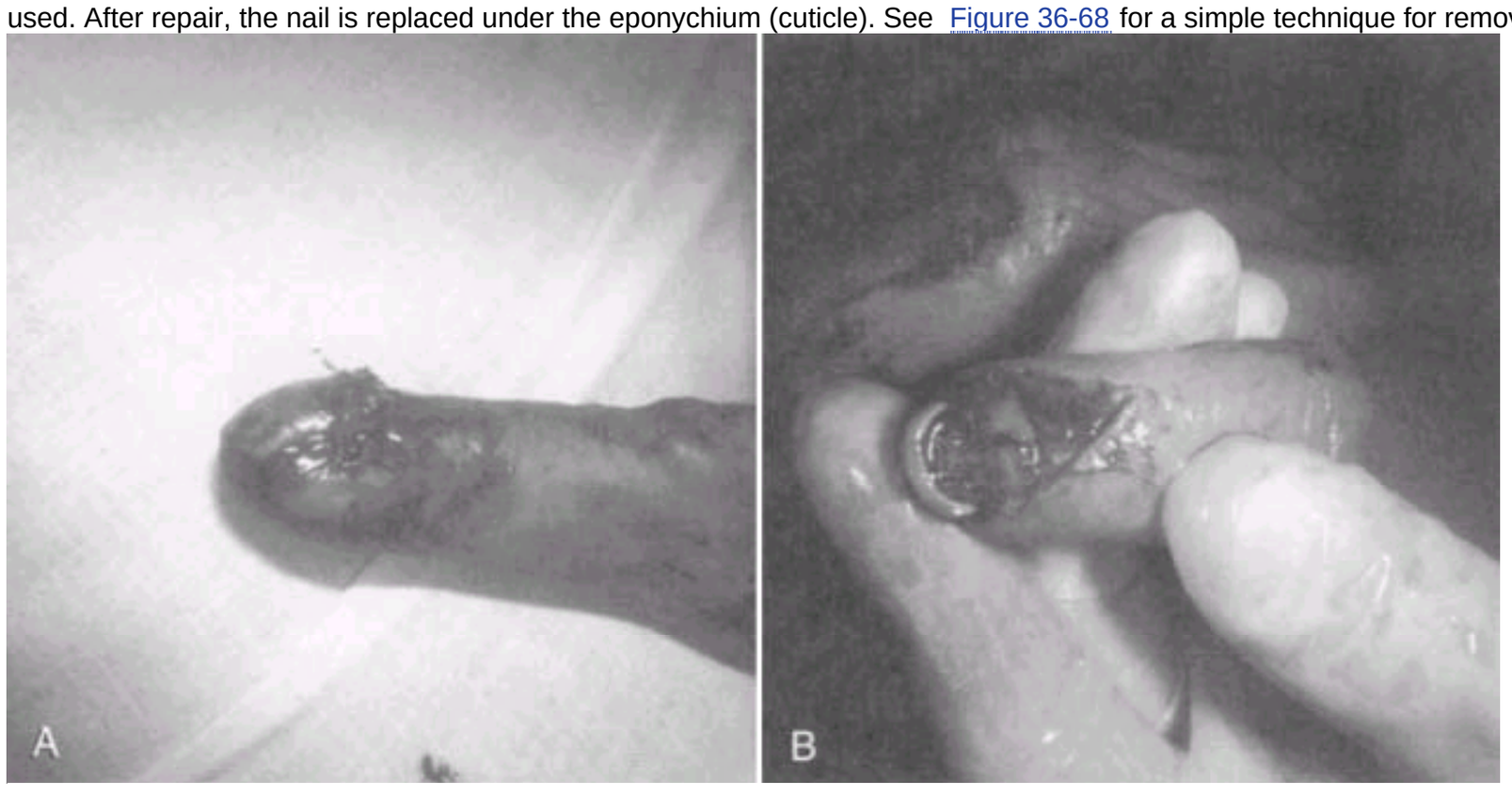


Figure 36-64 This nail is permanently deformed with ridges. Although crush injury to the nailbed is likely responsible for this deformity, nailbed repair is believed to minimize the resultant deformity.

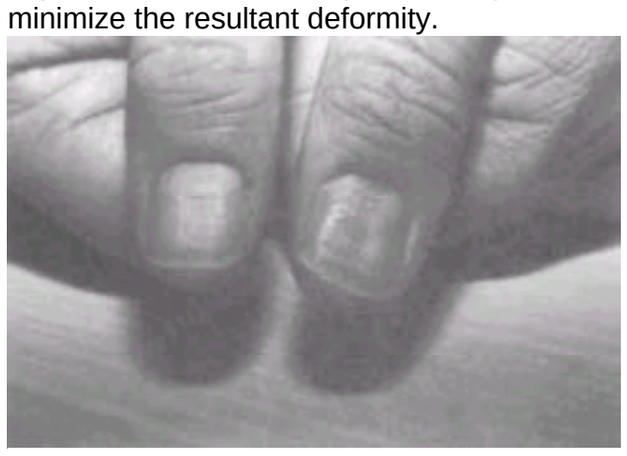


Figure 36-65 A laceration involving the nailbed, germinal matrix, and skinfold must be carefully approximated. First the nail is completely removed. (see Fig. 36-68). Fine, absorbable sutures are used to repair the nailbed under a bloodless field provided by a finger tourniquet. The avulsed nail (trimmed at the base) or a gauze pack is gently placed between the matrix and eponychium for 2 to 3 weeks to prevent scar formation *(A)*. If the original nail is replaced (the best option), it may be sutured or taped in place *(B)*. A large hole in the nail will allow drainage. The old nail is gradually pushed out by a new one. If the nail matrix is replaced quickly and atraumatically, the nail may act as a free graft and grow normally. Note: Only absorbable sutures are used to repair the nailbed.

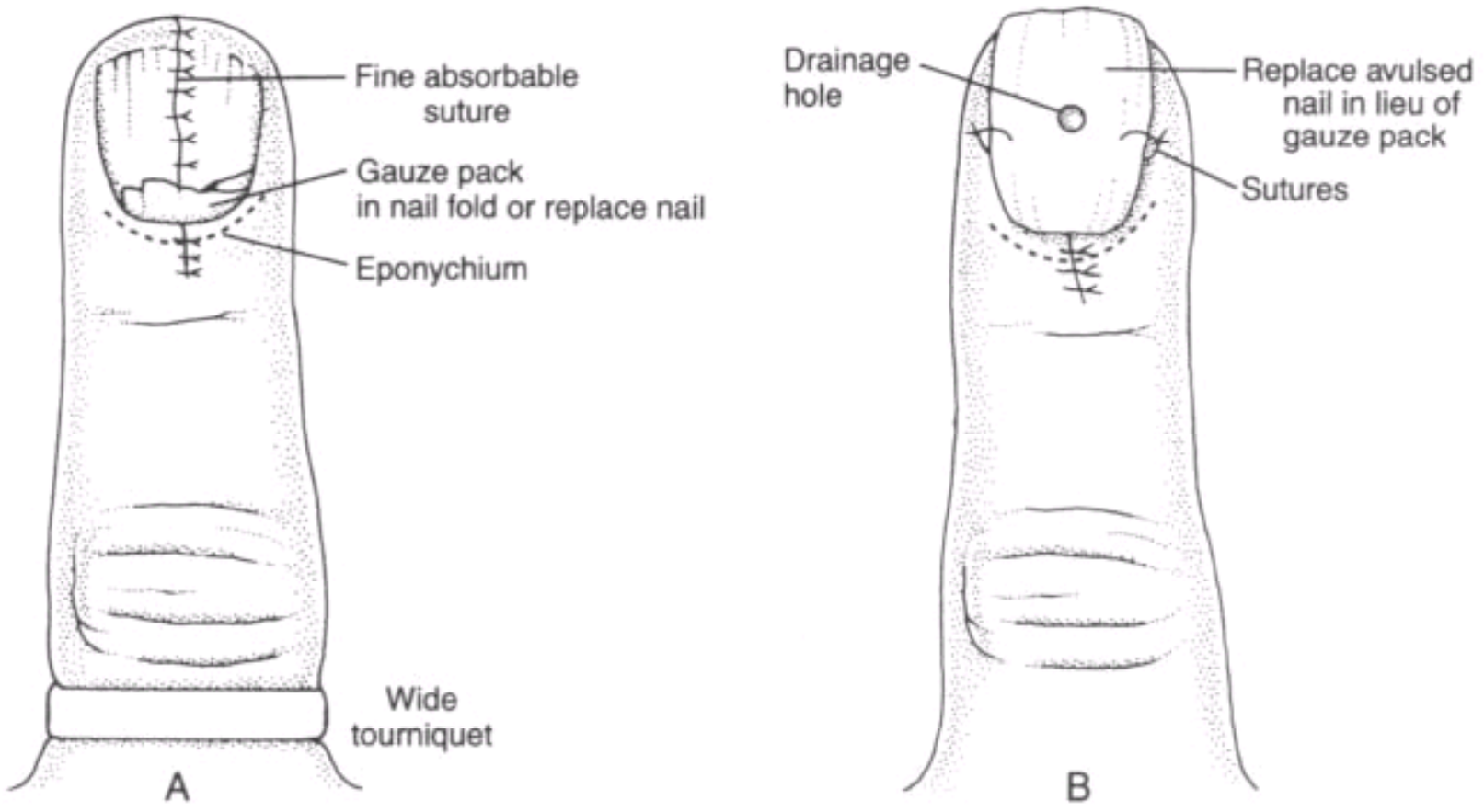


Figure 36-66 Avulsion of the nail, leaving the matrix intact, requires only a nonadherent dressing to separate the skinfold from the nailbed. If the germinal matrix is avulsed, as shown in this figure, it should be replaced to its original position under the eponychium with 6-0 plain absorbable sutures. *(From Grabb WC, Kleinert HE: Technics in Surgery: Facial and Hand Injuries. Somerville, NJ, Ethicon, Inc., 1980. Reproduced by permission.)*

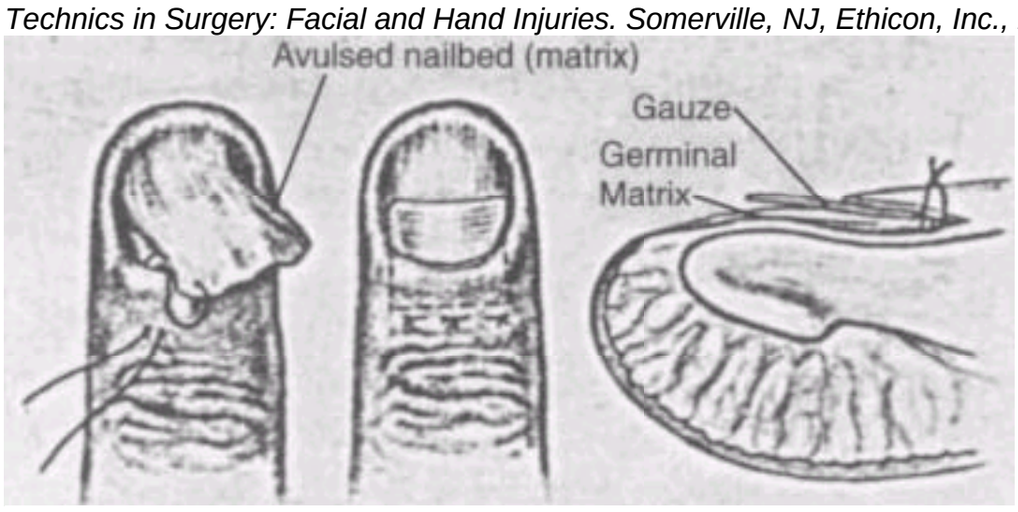


Figure 36-67 Repair of a distal finger laceration involving the nail and the onychial fold. In this case the nail is still adherent to the nail matrix and acts as a natural splint. If the nail is loose or completely transected, it is prudent to remove the entire nail and then carefully suture the nailbed under direct vision. *(From Dushoff IM: Handling the hand. Emerg Med 1976, p 111. Reproduced by permission.)*

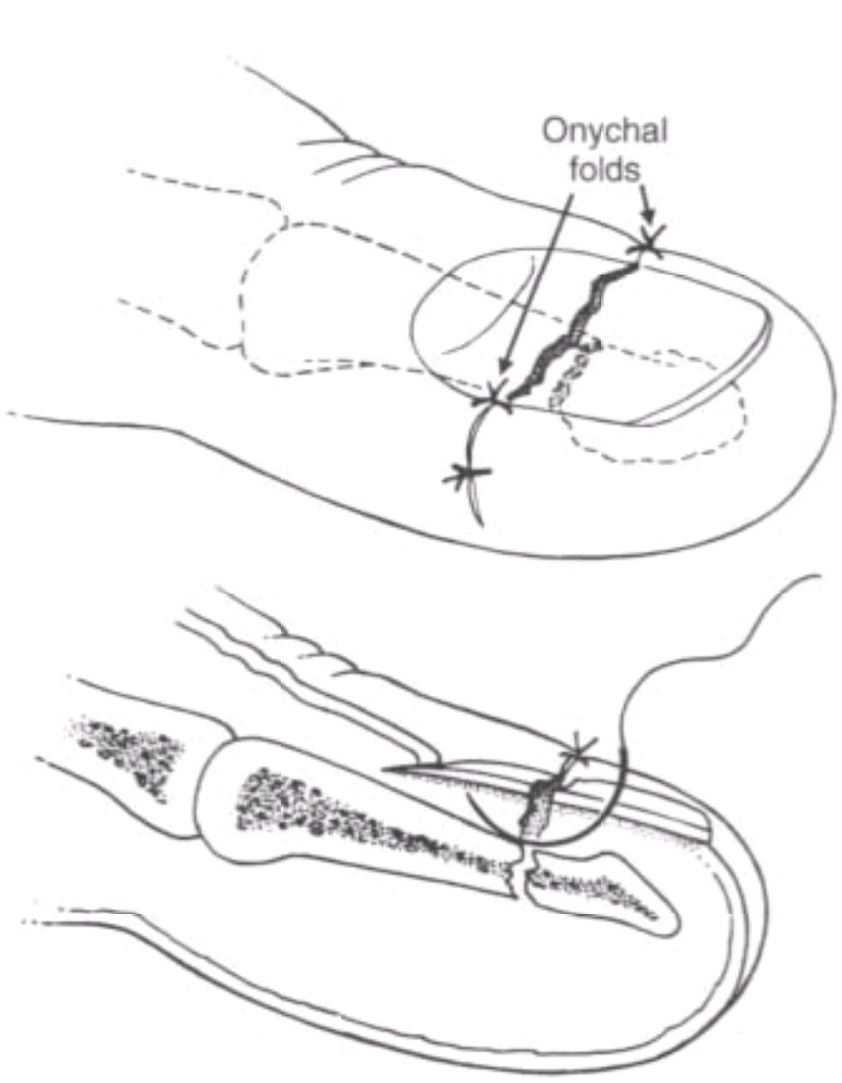


Figure 36-68 To remove a fingernail or toenail atraumatically, the blades of iris scissors are held parallel to the nailbed to avoid lacerating the matrix. A digital block is usually performed to make the procedure painless. The closed blades are slowly advanced in the plane between the nail and the nailbed *(A)* and then gently spread *(B)* to loosen the nail. The scissors are advanced and spread in stages until the base of the nail is reached and the entire nail is loose. The nail is grasped with a hemostat and pulled from the base *(C)*, exposing the nail matrix *(D)*. The nail can be replaced, if desired, once the nailbed laceration has been repaired.

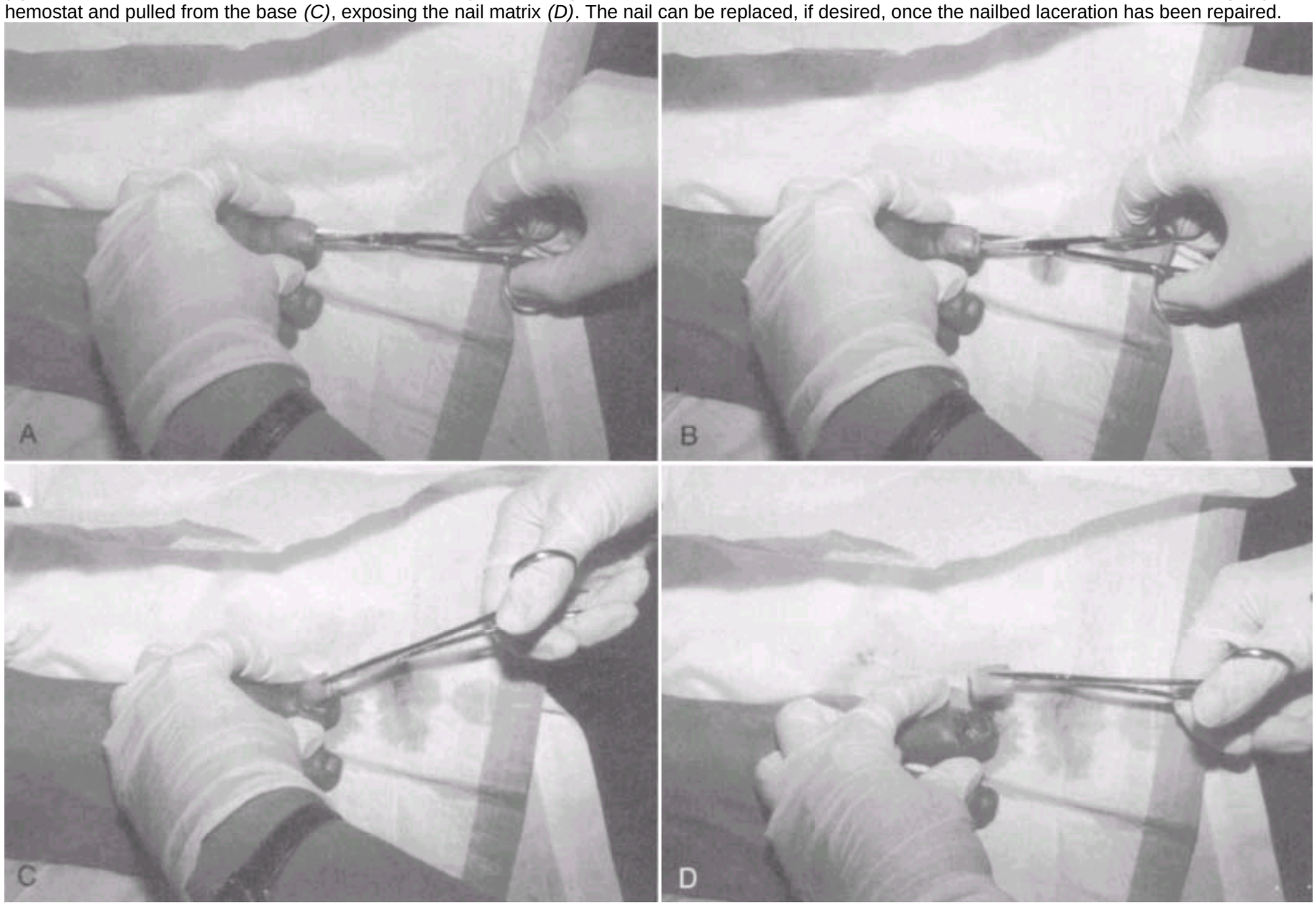


Figure 37-1 A common foreign body (FB) of the foot is a splinter, toothpick, pin, or needle that is impaled while walking barefoot on a carpet. This sewing needle was obvious, but some FBs may be mistaken for a heel spur, contusion, or tendinitis. Preoperative *(A)* and postoperative *(B)* radiographs demonstrate complete removal.

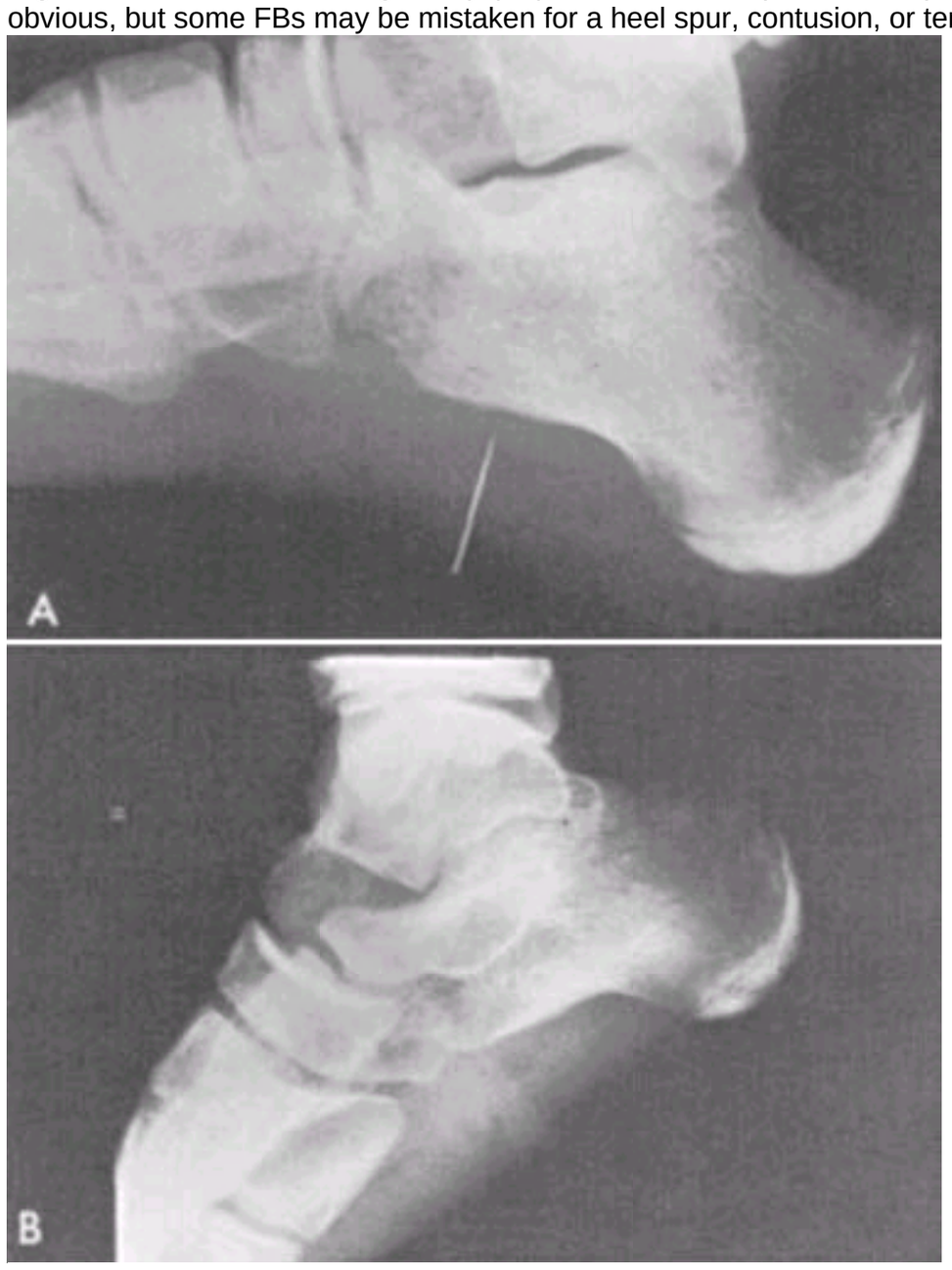


Figure 37-2 This patient fell, landed on a metal pipe, and suffered a laceration to the thigh. A radiograph was taken to rule out a fracture, and the key was seen but thought to be an artifact (i.e., an item left on the backboard). During the examination the key was found embedded in the wound. It had been in the patient's pants pocket and was forced into the wound by the pipe during the injury.

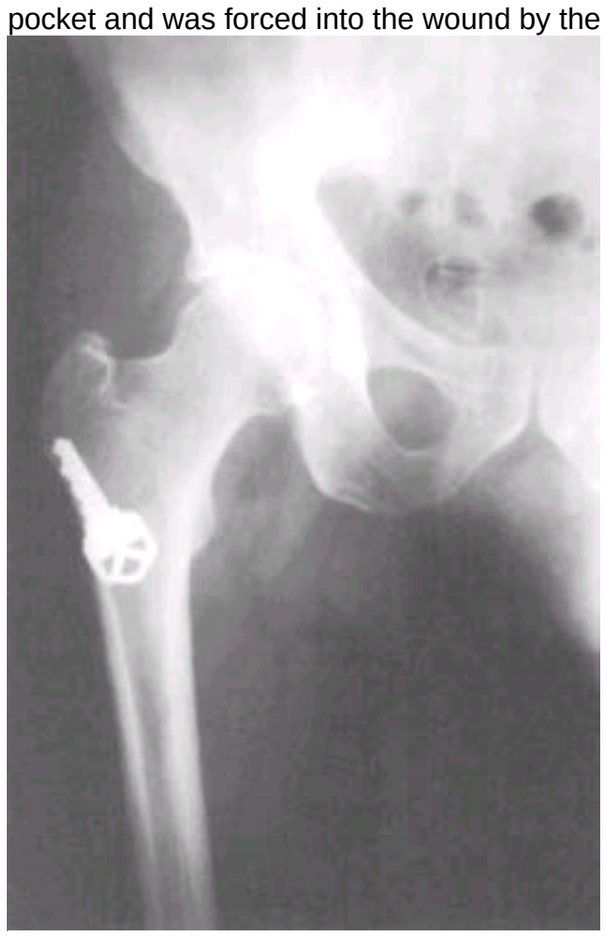


Figure 37-3 Most lead foreign bodies are well tolerated, but if a bullet is bathed in synovial, pleural, peritoneal, or cerebrospinal fluid, the lead may leach out over time and produce a significant elevation in blood lead levels. Symptoms are often vague, and the relation between the retained lead and the patient's clinical scenario may be difficult to sort out. *(From Schwartz DT, Goldfrank LR: Toxicologic imaging. In Goldfrank LR, Flomenbaum NE, Lewin NA, et al [eds]: Goldfrank's Toxicologic Emergencies. 5th ed. Norwalk, CT, Appleton & Lange, 1994, p 116. Reproduced with permission.)*

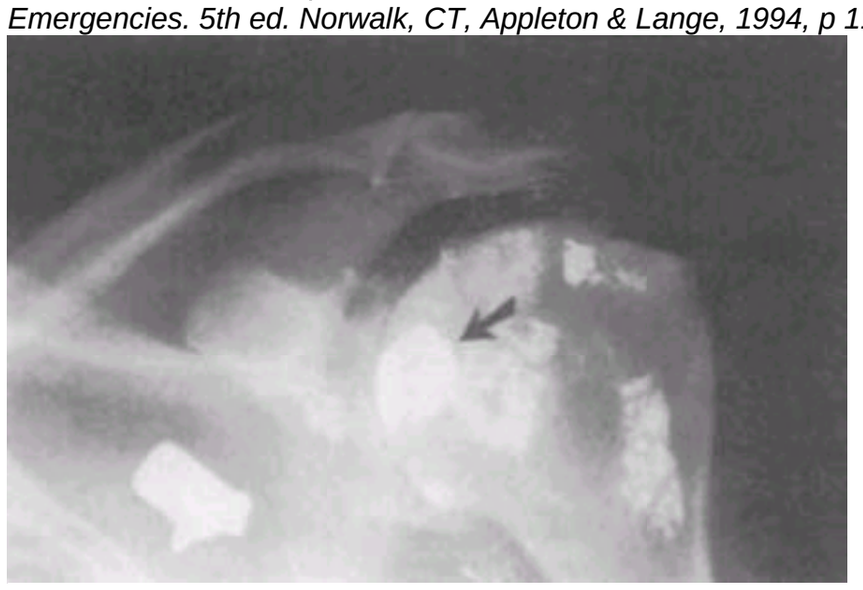


Figure 37-4 Comparison of plain radiography, computed tomography, and ultrasonography in imaging wooden, glass, and plastic foreign bodies (FBs) in an in vitro preparation. Computer manipulation of a digitized radiograph may aid in FB assessment. *(From Ginsburg MJ, Ellis GL, Flom LL: Detection of soft-tissue FBs by plain radiography, xerography, computed tomography, and ultrasonography. Ann Emerg Med 19:701, 1990. Reproduced with permission.).*

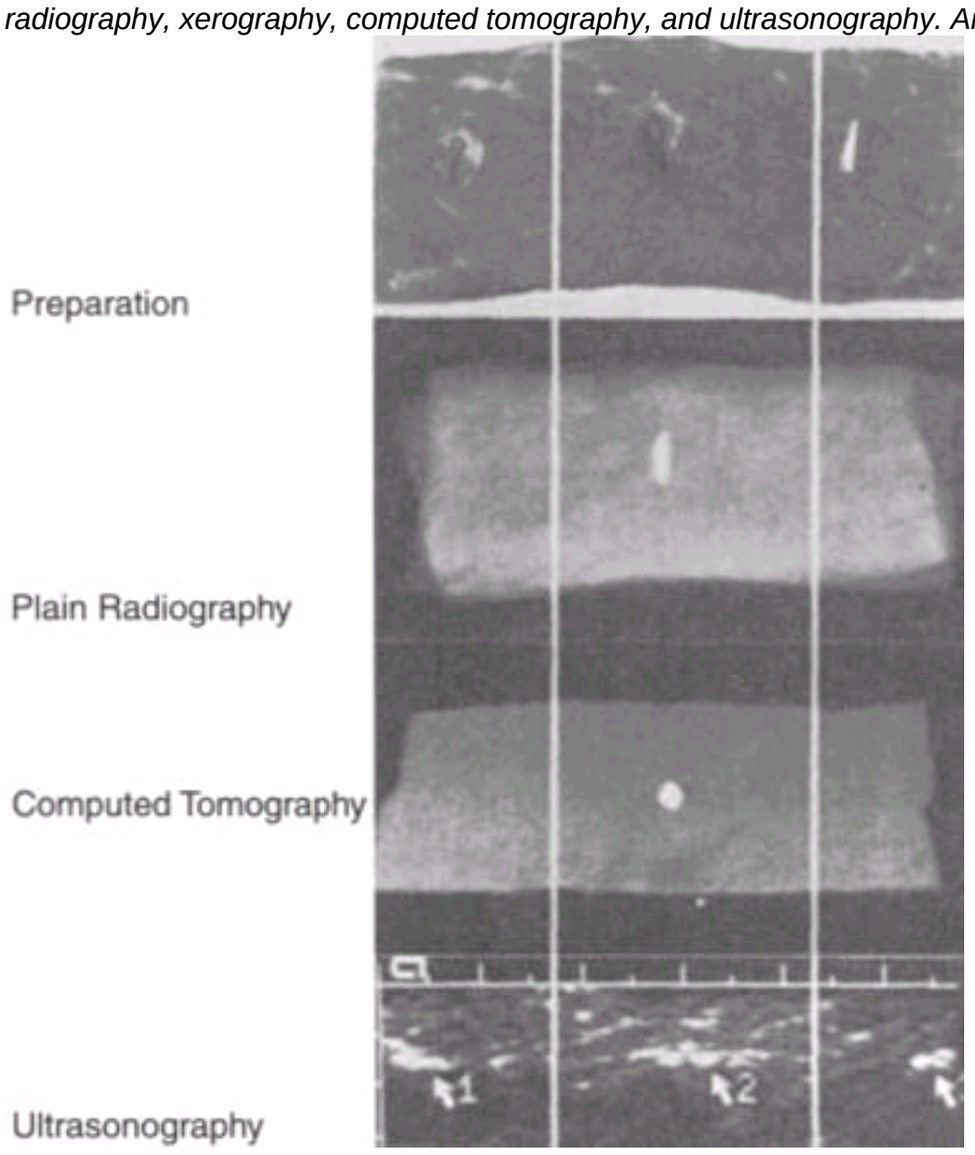


Figure 37-5 Almost all glass is visible on a plain radiograph. Small size is the major limiting factor, not the lead content. If glass is superimposed over bone, it may be obscured, so multiple projections are required. Samples of glass are superimposed on the abdomen and exposed to x-rays. *(Reproduced with permission from Zatzkin HR: The Roentgen Diagnosis of Trauma. Copyright © 1965 by Year Book Medical Publishers, Chicago. Reproduced with permission.)*

Figure 37-6 This foreign body (FB) granuloma developed after the FB was stable for 6 months in this laborer's hand. There was no gross infection, but the pain was quite bothersome. The FB and reactive tissue were excised under local anesthesia.

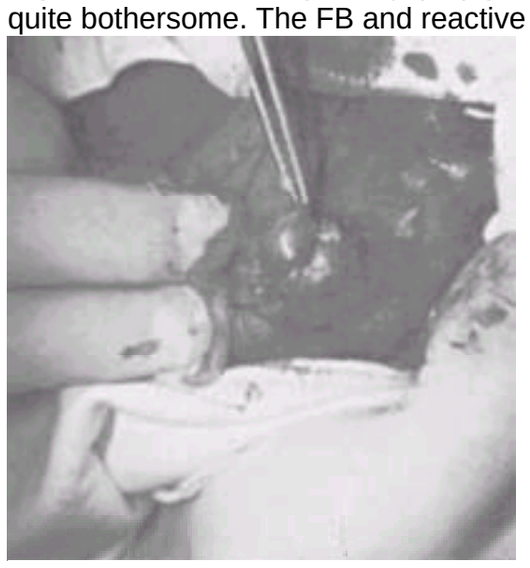


Figure 37-7 This intoxicated patient punched out a window and sustained seemingly minor puncture wounds *(A)*. He did not believe that glass was in the wound, there was little pain, and no FB could be palpated externally. A radiograph *(B)* revealed a large shard of glass deeply embedded in the wound *(C)*.

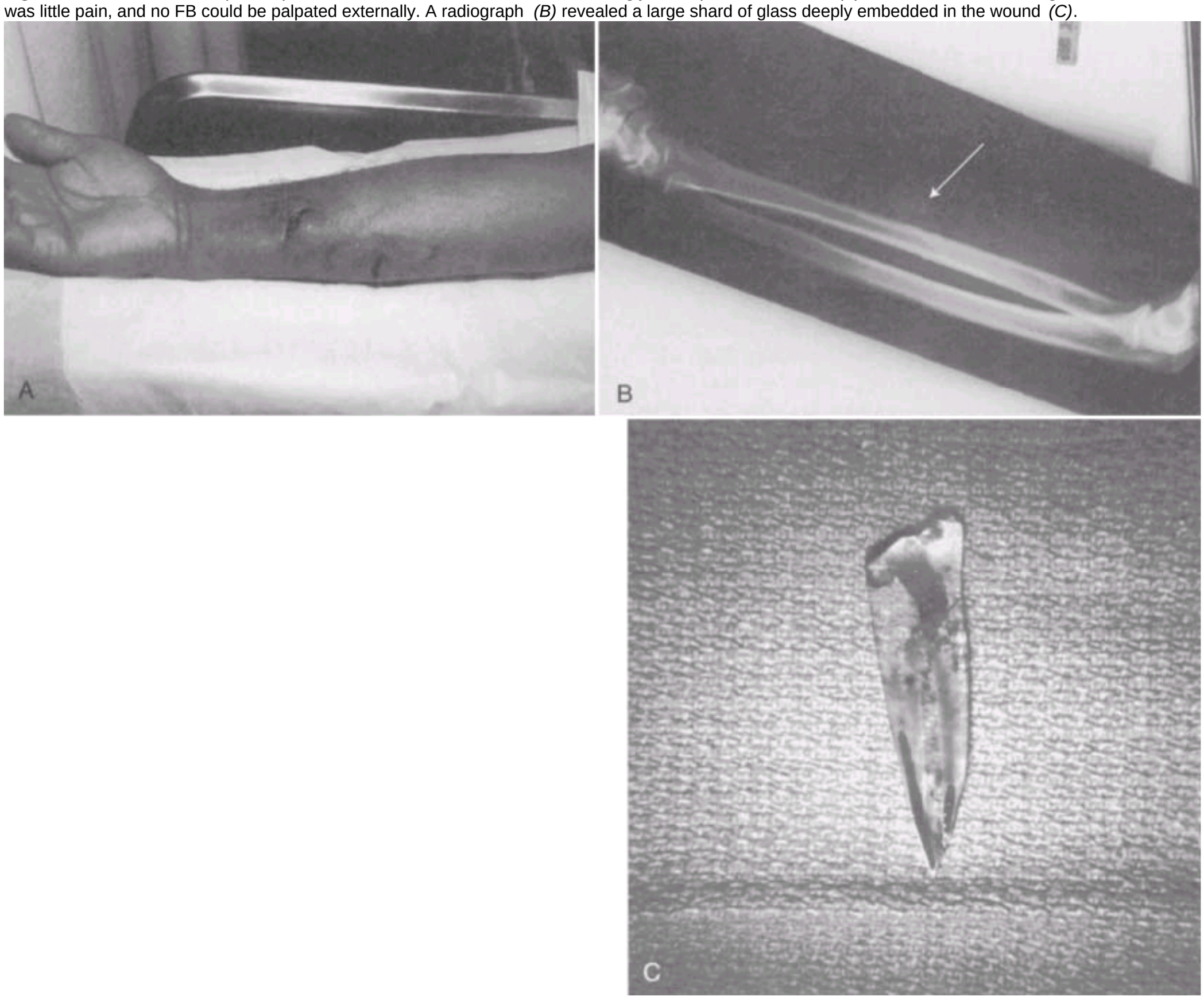


Figure 37-8 What's wrong with this picture? Although historically suggested as a useful technique to find FBs, probing the depths of a wound with a gloved finger may result in a puncture wound in the operator. The practice is strongly discouraged because of the prevalence of hepatitis and human immunodeficiency virus infections.

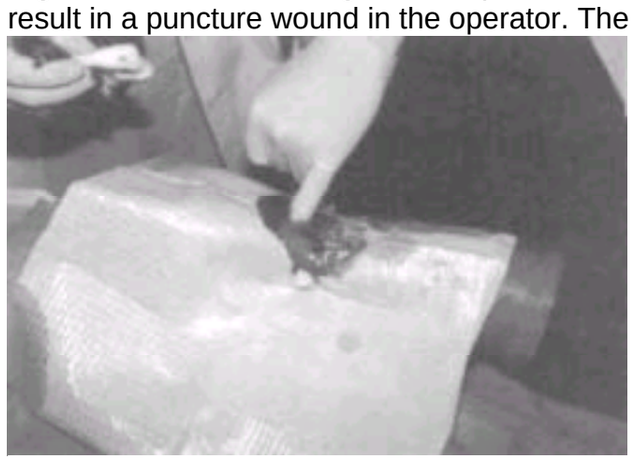


Figure 37-9 When a small entrance wound *(A)* is noted but the foreign body (FB) is not seen, noninvasive localization is preferable to blind probing. Metal markers taped to the skin or needles inserted close to the FB under local anesthesia *(B)* and radiographed at different angles provide a guide to FB localization and extraction. *(Reproduced from Hospital Medicine, © January 1981, with permission of Cahner's Publishing Co.)*

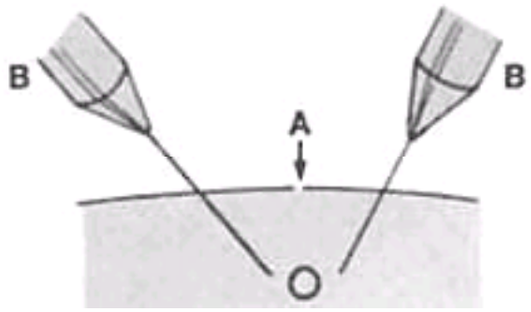


Figure 37-10 A sewing needle completely embedded below the surface *(A* and *B)* is easily located by a radiograph. Following local anesthesia, a small incision over the superficial end permits removal with a hemostatic forceps *(C)*. The hemostat is introduced through an adequate incision, spread to open the tissue, and used to "feel" the foreign body as the hemostat is advanced. *(Reproduced from Hospital Medicine, © January 1981, with permission of Cahner's Publishing Co.)*

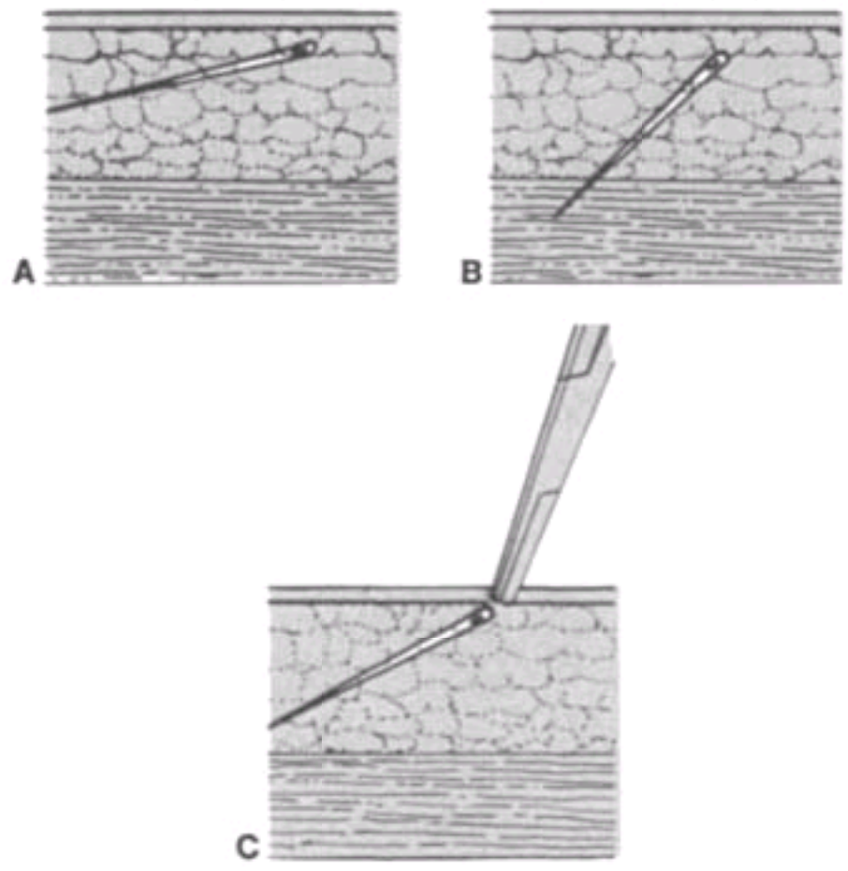


Figure 37-11 If a linear skin incision is used to locate a mobile foreign body (FB) that is perpendicular to the skin in the subcutaneous fat *(A),* the FB may be displaced *(B)*. A modified elliptical incision is made *(C),* and the skin edges are undermined, displacing the FB into the middle of the wound. Pressure with the thumbs may be applied to the skin to force the FB into view. *(From Rees CE: The removal of FBs: A modified incision. JAMA 113:35, 1939. Copyright 1939, American Medical Association. Reproduced with permission.)*

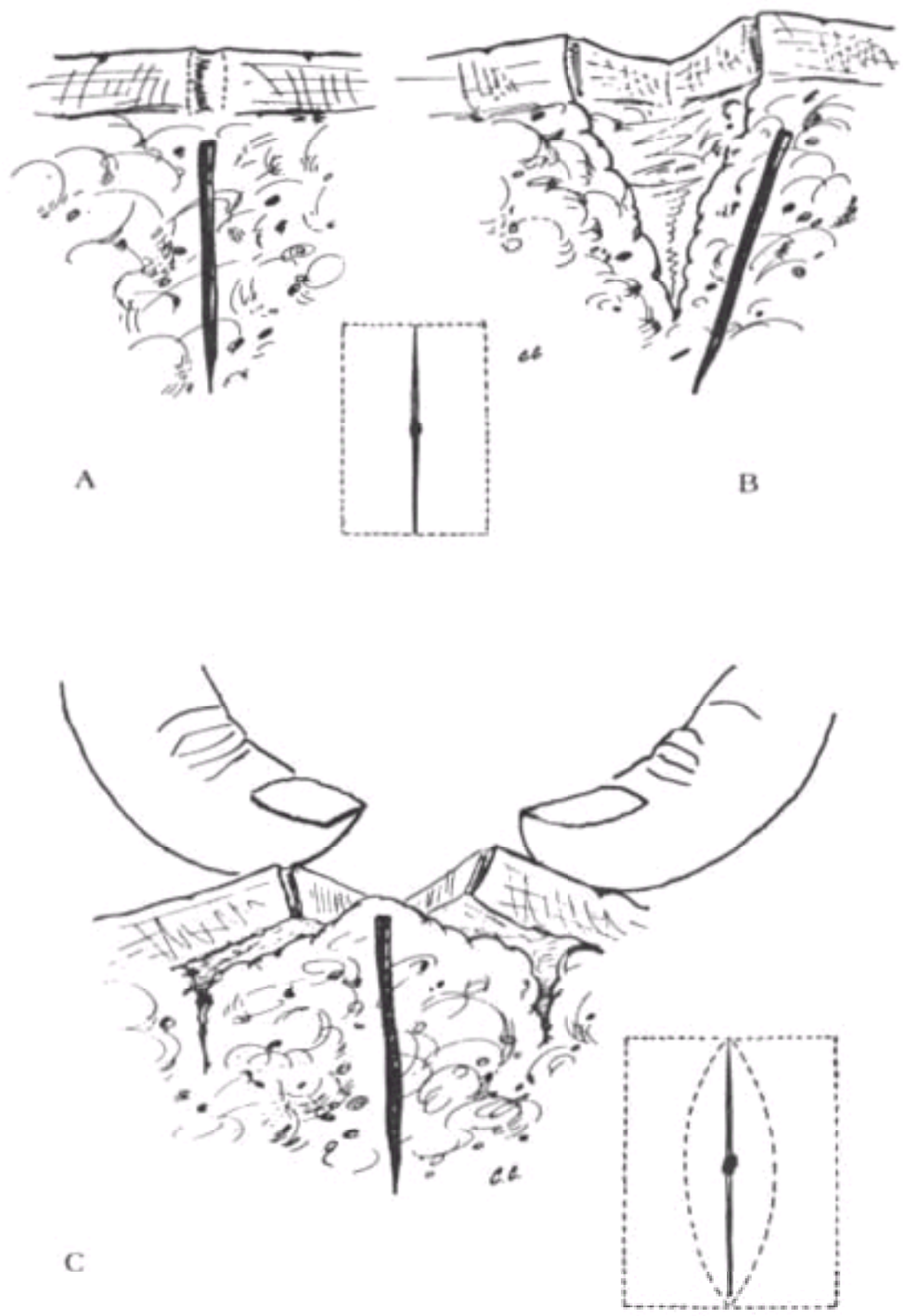


Figure 37-12 Foreign bodies (FBs) in deep fat *(A)* may be approached by a small elliptical incision around the entry point *(B)*. The incision is then laterally undercut and grasped (without pulling) with an Allis clamp *(C)*. The ellipse is then further undercut until contact with the FB is made *(D)*. The FB may be grasped and removed along with the entry tract and the soiled local fat *(E)*. *(Reproduced from Hospital Medicine, © January 1981, with permission of Cahner's Publishing Co.)*

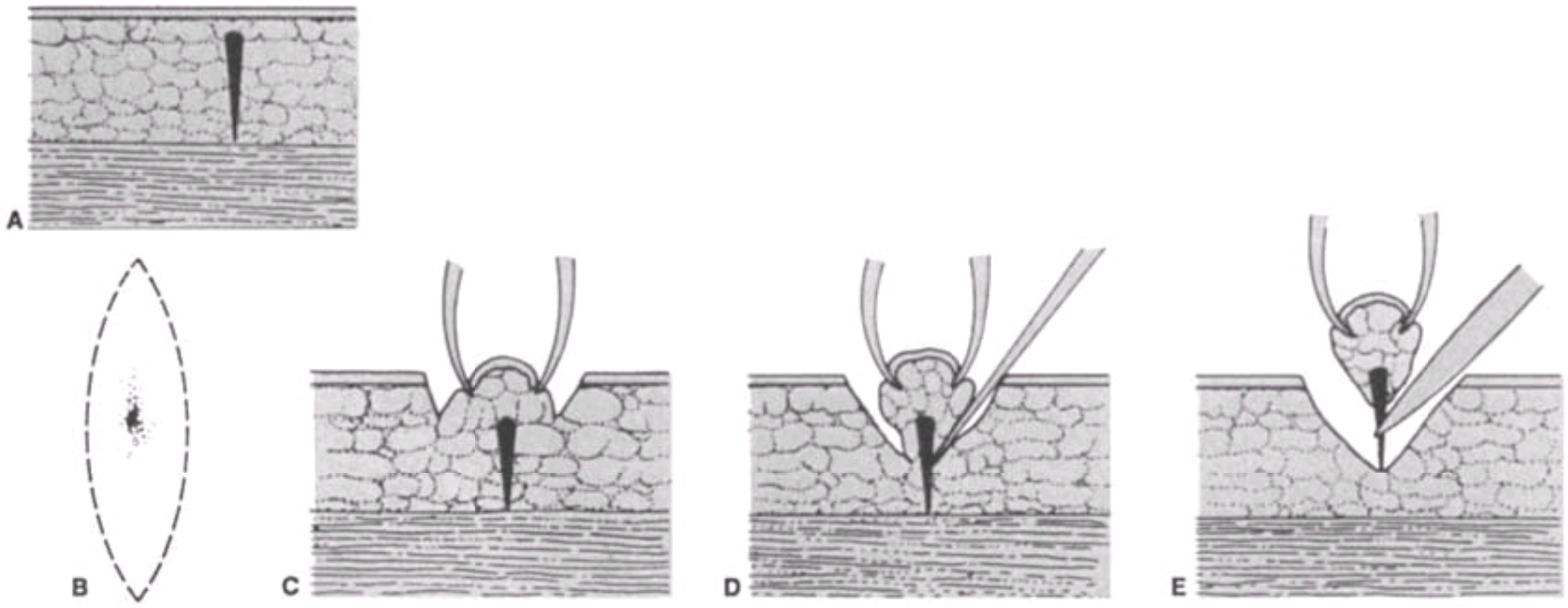


Figure 37-13 The vast majority of patients who step on a nail have no long-term consequences, but a few experience soft tissue infections or serious and debilitating osteomyelitis. The depths of a fresh puncture wound are not routinely explored, but key to avoid missing a superficial foreign body (FB) is to examine the patient under good lighting, supine, and with an arterial tourniquet if necessary (*A*). If the patient was wearing socks or tennis shoes, a small FB, like this piece of rubber, may be introduced into the wound. It may only be evident when the edges of the skin are trimmed and the wound is explored (*B*). Coring out the puncture wound tract is controversial and unproven, but it is one way to remove foreign material. In this case a No. 11 blade is inserted to the hilt and a 2 to 3 mm core of tissue is removed with the FB (*C*). This tract may be left open or a small packing applied. If a patient returns with an infection within a few days after a seemingly simple puncture wound (*D*), it is most likely a soft tissue infection, but persistent pain demands a search for osteomyelitis or a deep FB.

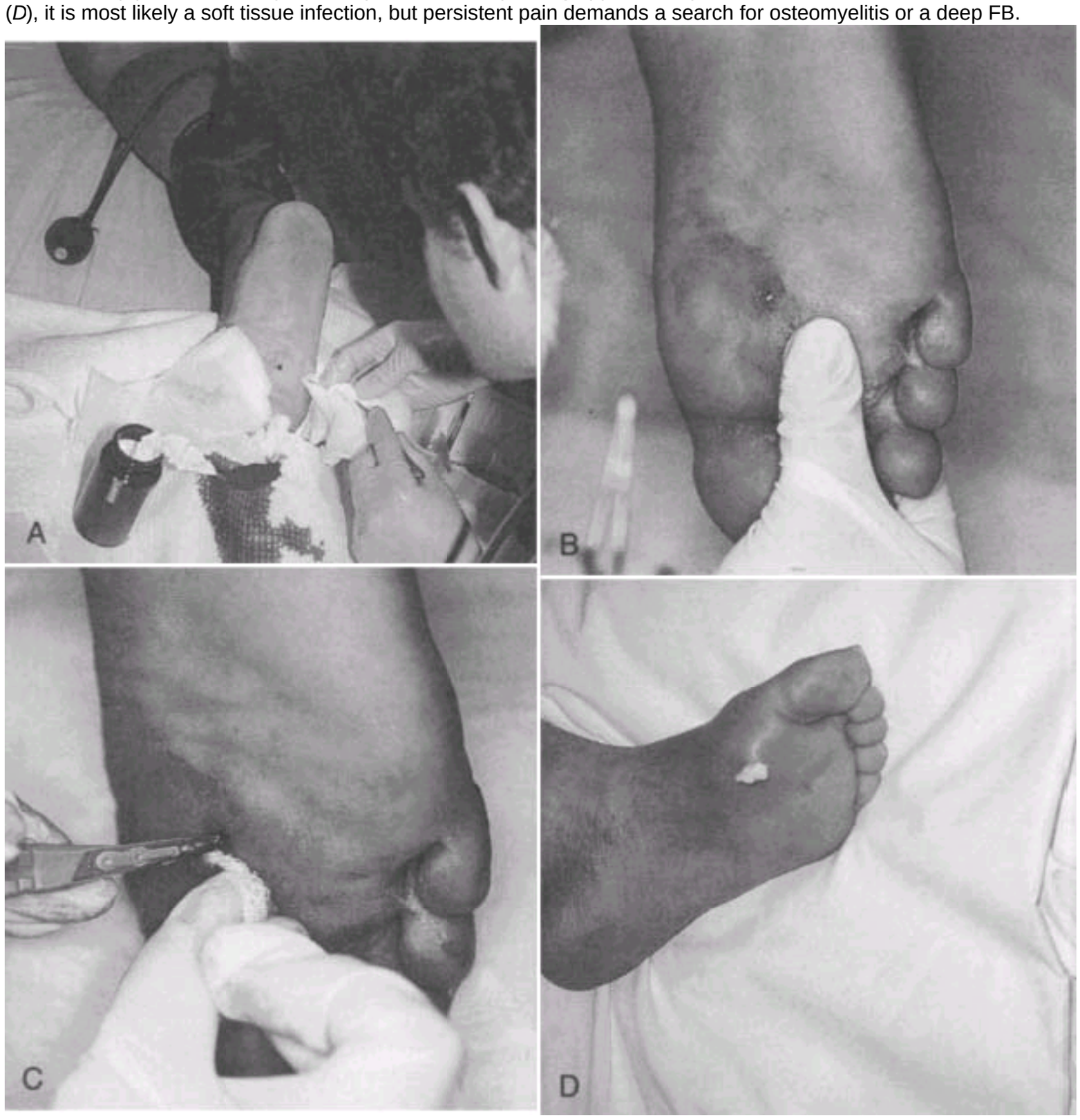


Figure 37-14 For an FB deep in the nailbed, take as small a wedge of nail as will allow access to the proximal end of the splinter, then extract the FB with splinter forceps. All wood particles should be removed. A digital nerve block is usually necessary. *(Reproduced from Hospital Medicine, © January 1981, with permission of Cahner's Publishing Co.)*

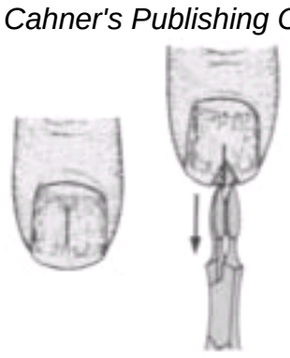


Figure 37-15 Method of removing an embedded fishhook when anesthesia is available and when the point of the fishhook is close to the skin. *A,* Obtain local anesthesia overlying the point of the hook. *B,* Force the point through the anesthetized skin. *C,* Clip off the barb. *D,* Remove the rest of the hook by reversing the direction of entry. *(Reproduced from Hospital Medicine, © July 1980, with permission of Cahner's Publishing Co.)*

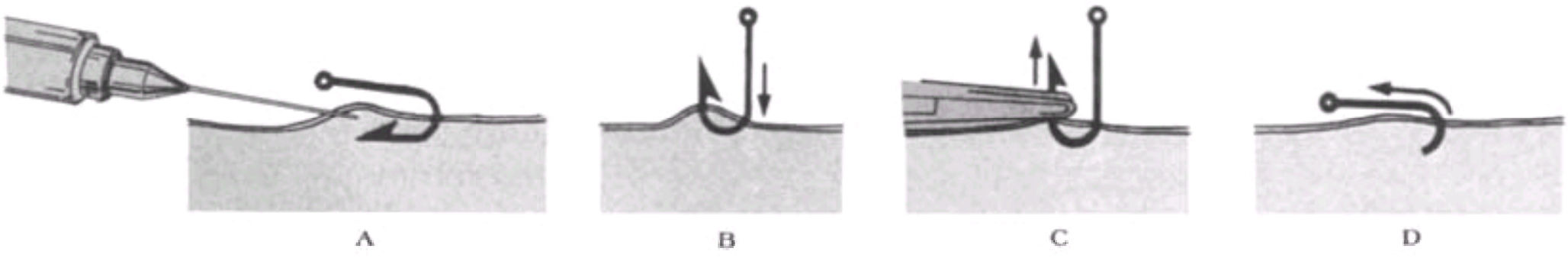


Figure 37-16 Method of removing an embedded fishhook when anesthesia is unavailable or when the barb of the fishhook lies too deep to force it out through a second wound without causing significant additional damage. Loop a piece of string (or thick suture material) around the belly of the hook and hold it down against the skin with the index finger of the left hand *(A)*. Depress the shaft of the hook against the skin with the middle finger and thumb while applying light downward pressure with the index finger of the left hand to disengage the barb from the subcutaneous tissue *(B)*, and pull *sharply* on the ends of the string with the right hand *(C)* to remove the hook through its entry wound. Bystanders should be out of the expected path of the hook. *(Reproduced from Hospital Medicine, © July 1980, with permission of Cahner's Publishing Co.)*

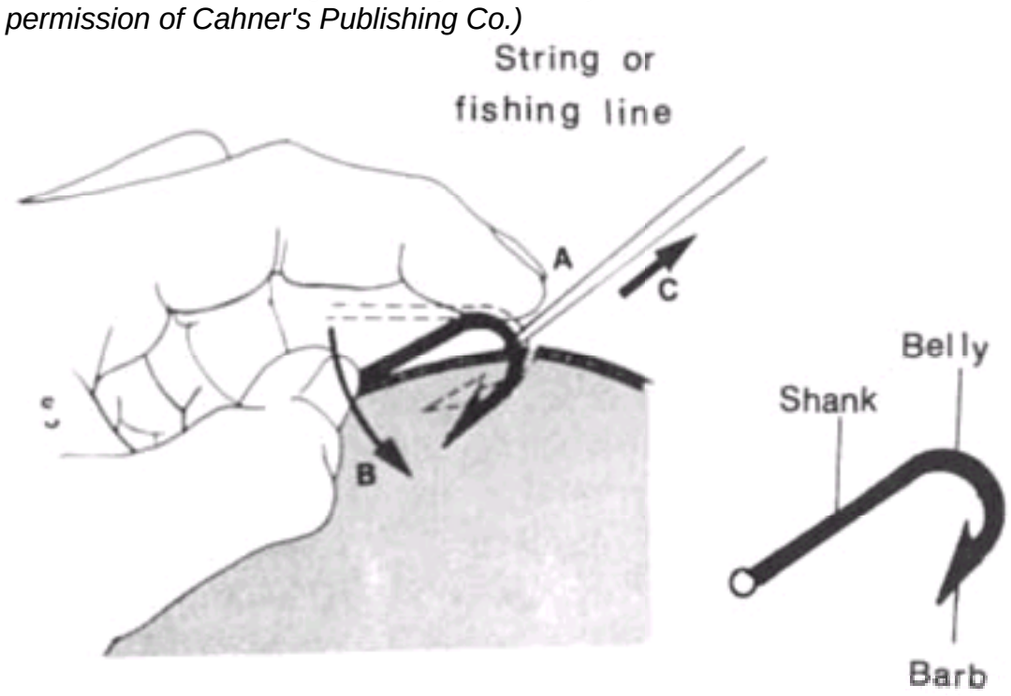


Figure 37-17 Method of removing an embedded fishhook using anesthesia when the hook is large and not too deep in the skin. After anesthetizing the area with 1% lidocaine *(A),* insert a short-bevel 18-ga needle through the entry wound of the hook and attempt to sheathe the barb of the hook within the needle *(B)*. If this is done correctly, the hook and needle may then be backed out together *(C)*. *(Reproduced from Hospital Medicine, © July 1980, with permission of Cahner's Publishing Co.)*

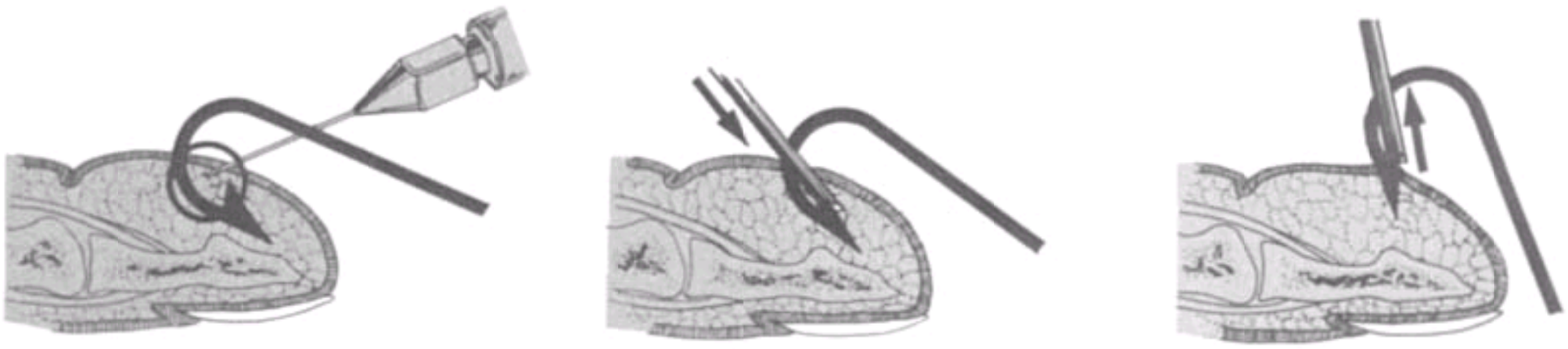
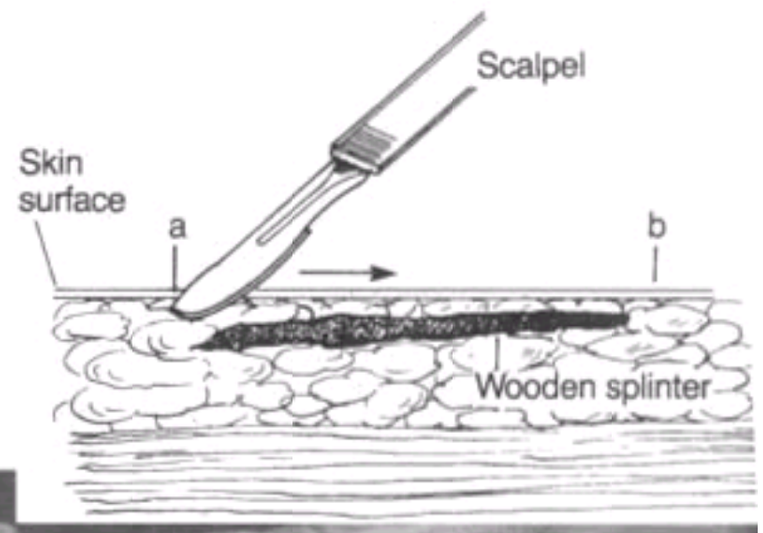
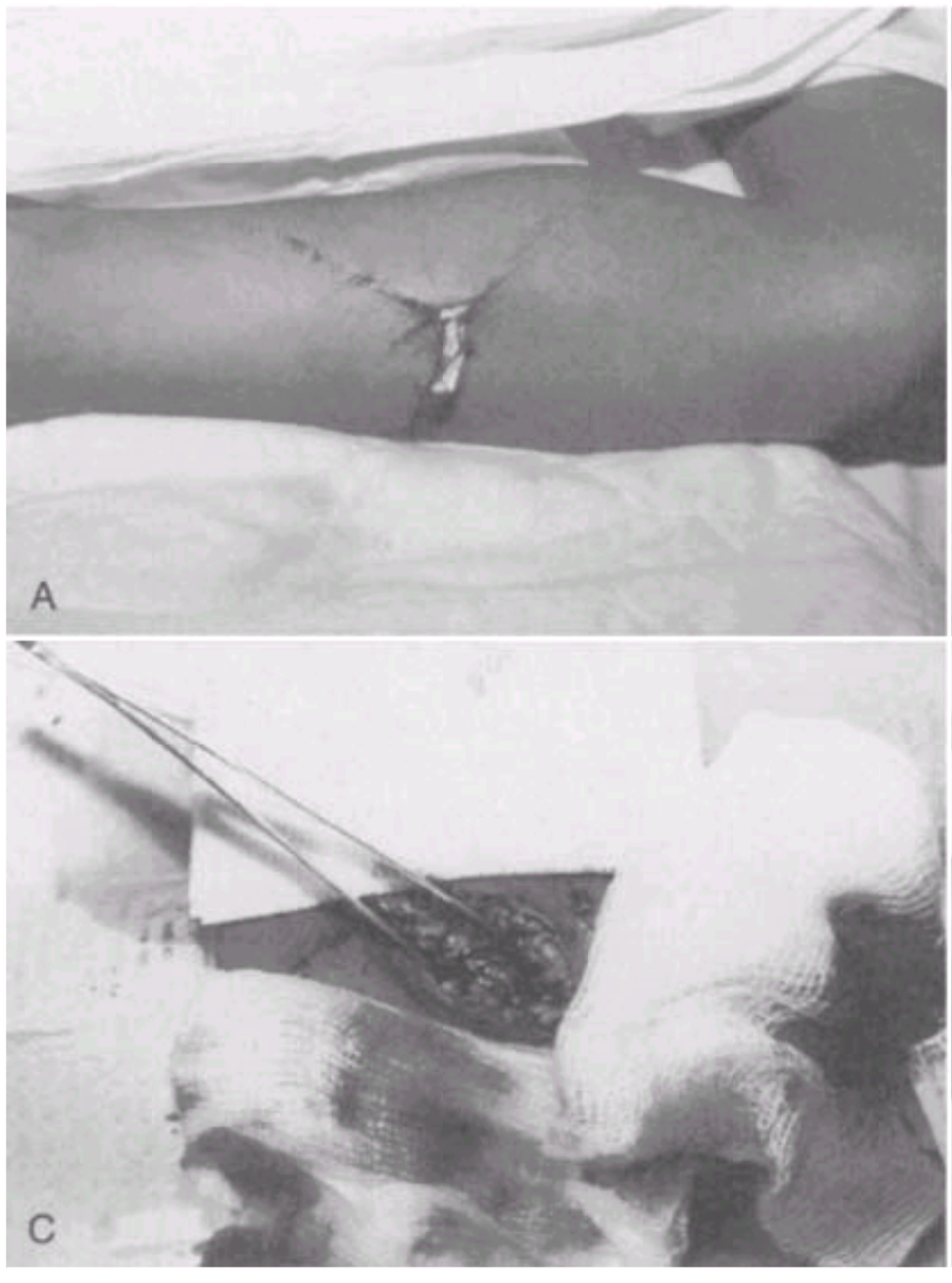


Figure 37-18a The course and depth of penetration of this large wood splinter of the leg *(A)* is uncertain, but it is axiomatic that all pieces of wood must be removed to prevent infection. To ensure complete FB removal, an incision is made over the entire course of the splinter *(B)* (*see inset*). All pieces of wood are carefully removed under direct vision *(C),* and the laceration is sutured primarily *(D)*. While it may be tempting to simply pull out the splinter and irrigate the puncture tract, such actions often lead to retained particles and complications.





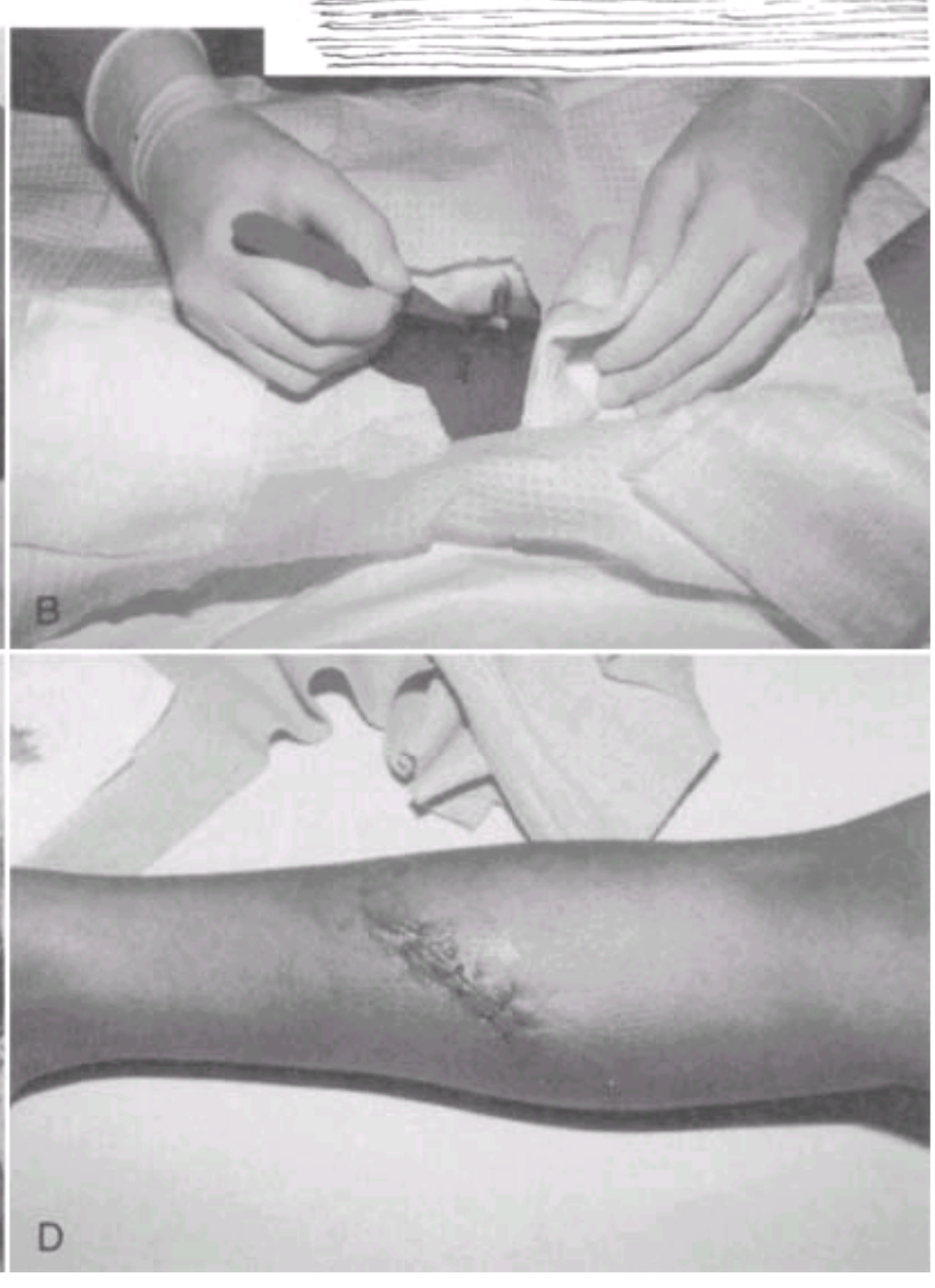


Figure 37-18b Here is another example of longitudinal incision of the overlying skin followed by withdrawal of a wood foreign body en mass to avoid retained fragments (*E*, *F*, *G*, and *H*.)

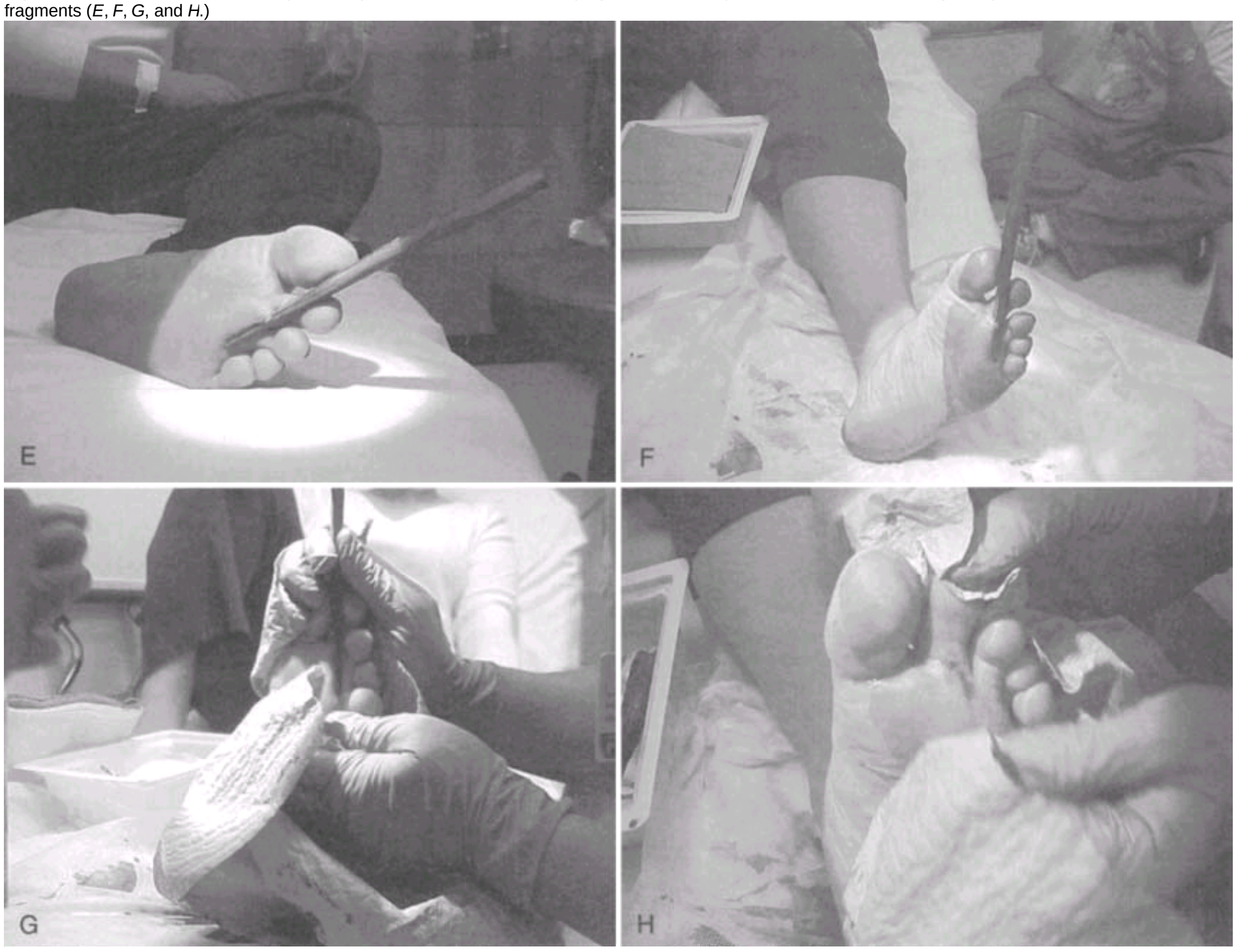


Figure 37-19 Magnified view of venom-containing nematocyst in its resting or "cocked" state *(A)*. After contact with the skin, a dart-like tail is extended and penetrates the dermis *(B),* and venom is injected *(C)*. Further stimulation of the attached nematocyst can expel more venom (see decontamination/removal technique in text).

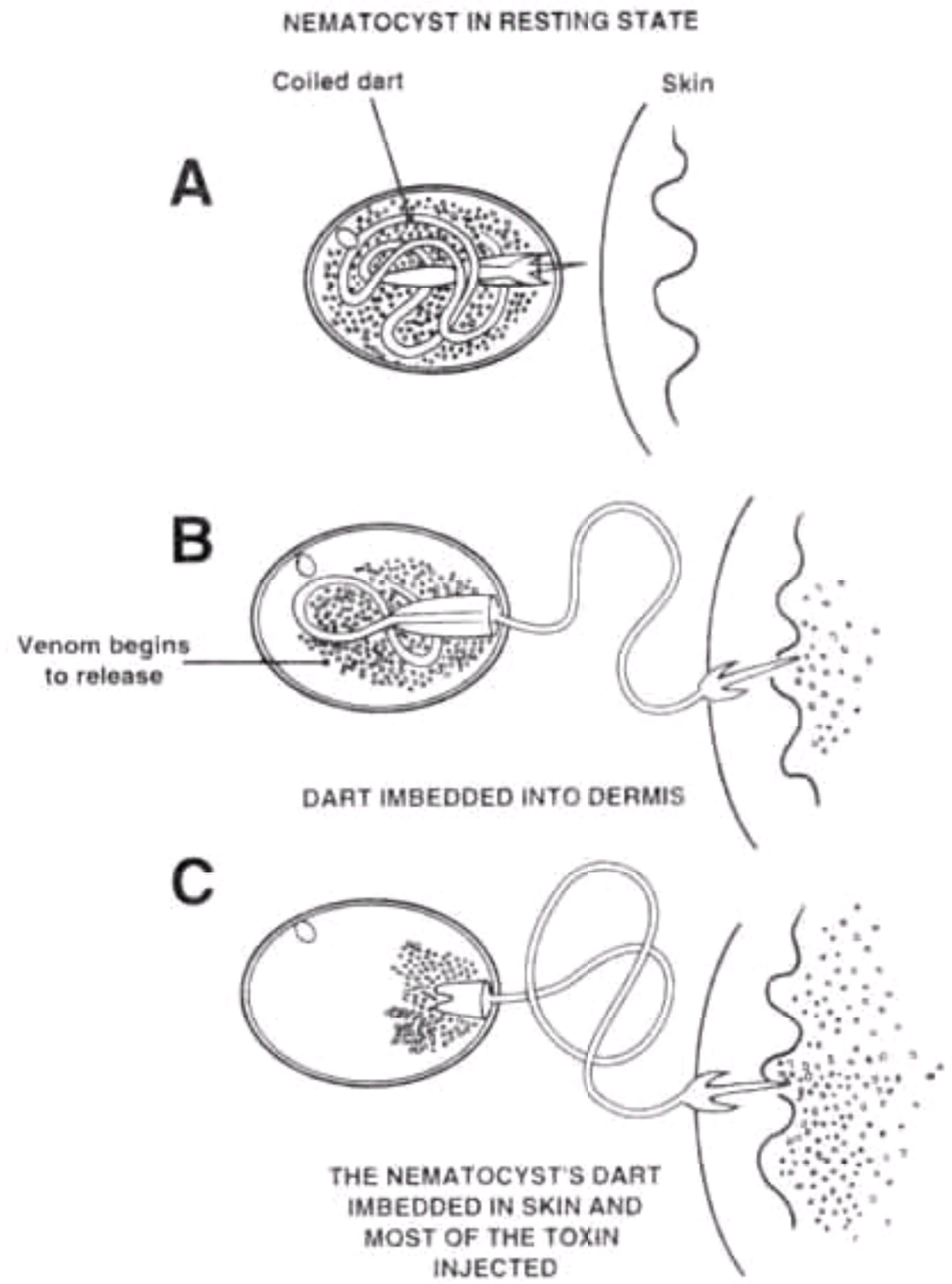


Figure 37-20 *A,* Stingray resting on the bottom of the ocean, usually covered by a layer of sand. An unsuspecting victim steps on the stingray, and the whiplike tail impales the foot (even through a heavy boot) with one or more spines *(B)*. The spine has backward-facing barbs covered by a sheath with venom-containing cells (*C*), causing a toxic envenomation and the potential for multiple FBs.

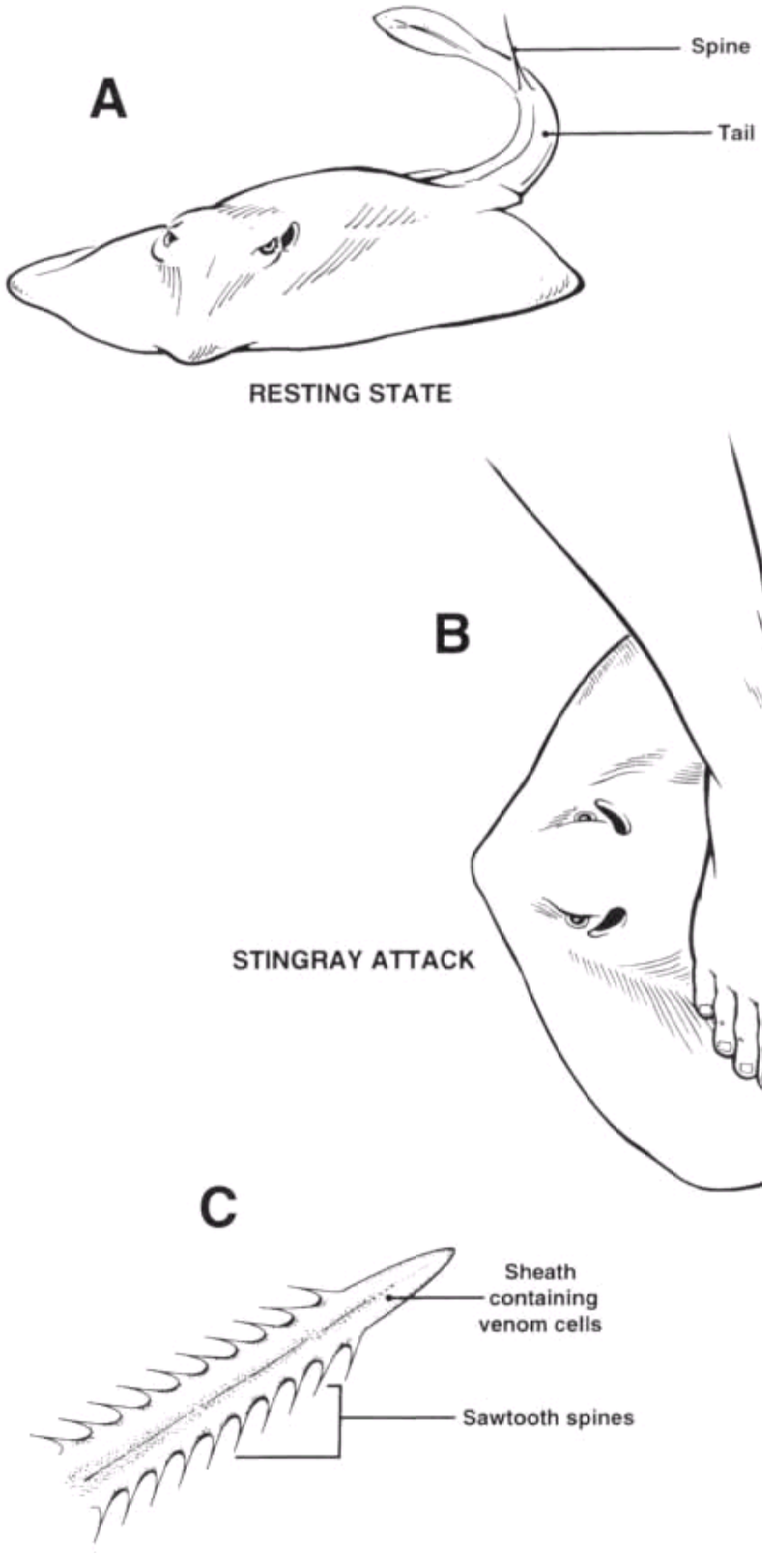


Figure 37-21 *A,* A thousand or more glochids affixed to the skin by contact with a single pad of polka dot cactus. *B,* The *professional* facial gel is spread with a fan brush. *C,* The gel is spread thin at the edges. *D,* The gel rollup is started by picking at the edge with the fingernails. *E,* When the *professional* gel is peeled off, all of the very small spines come with it. *(From Lindsey D, Lindsey WE: Cactus spine injuries. Am J Emerg Med 6:362, 1988. Reproduced by permission.)*

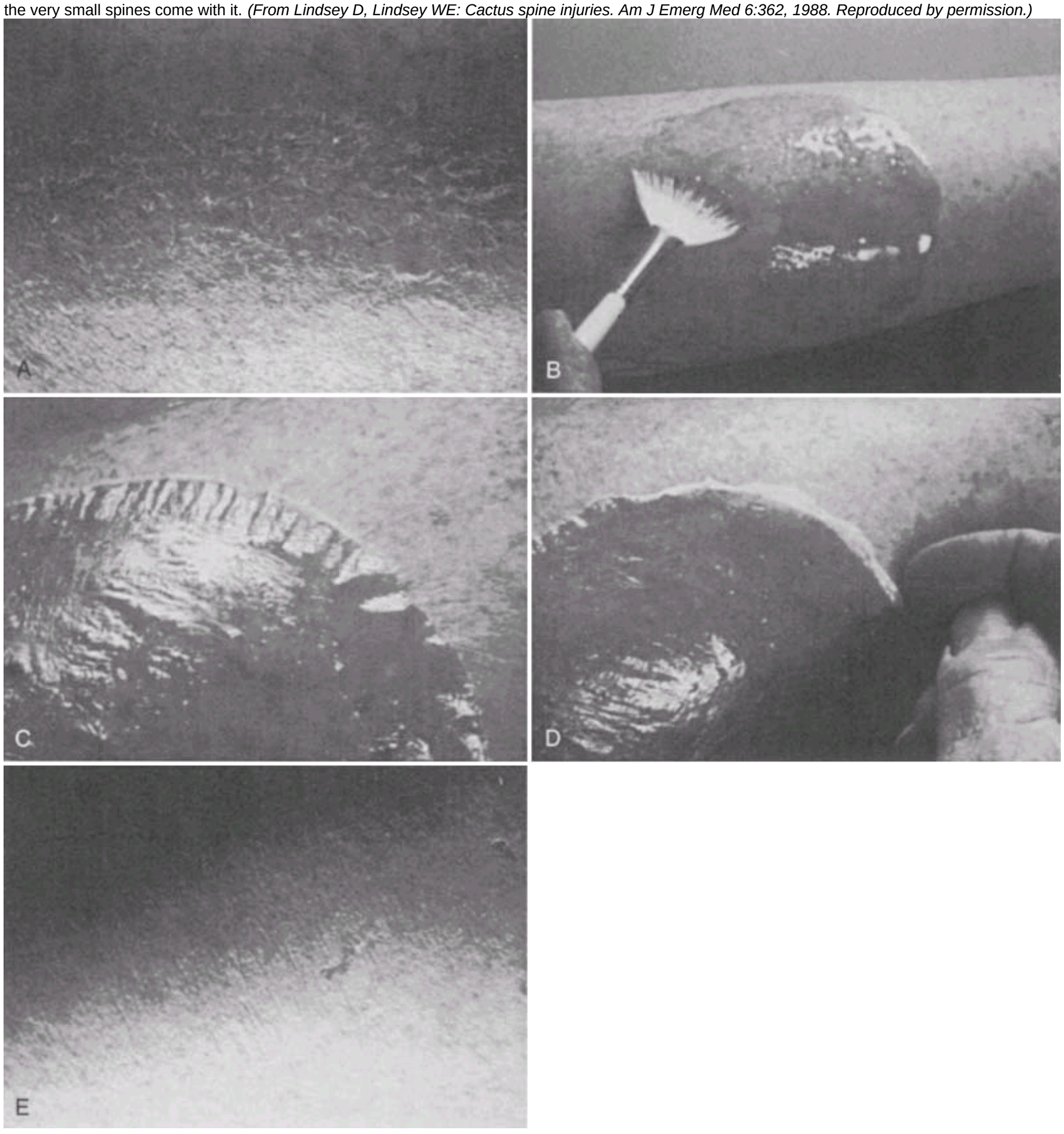


Figure 37-22a String removal technique for a tight ring. Note the absence of a laceration or underlying fracture *(A)*. A digital or metacarpal block is suggested. Edema is lessened by compression of the finger with a Penrose drain *(B)*. A long piece of umbilical tape is placed under the ring with the winding portion left long *(C)*.

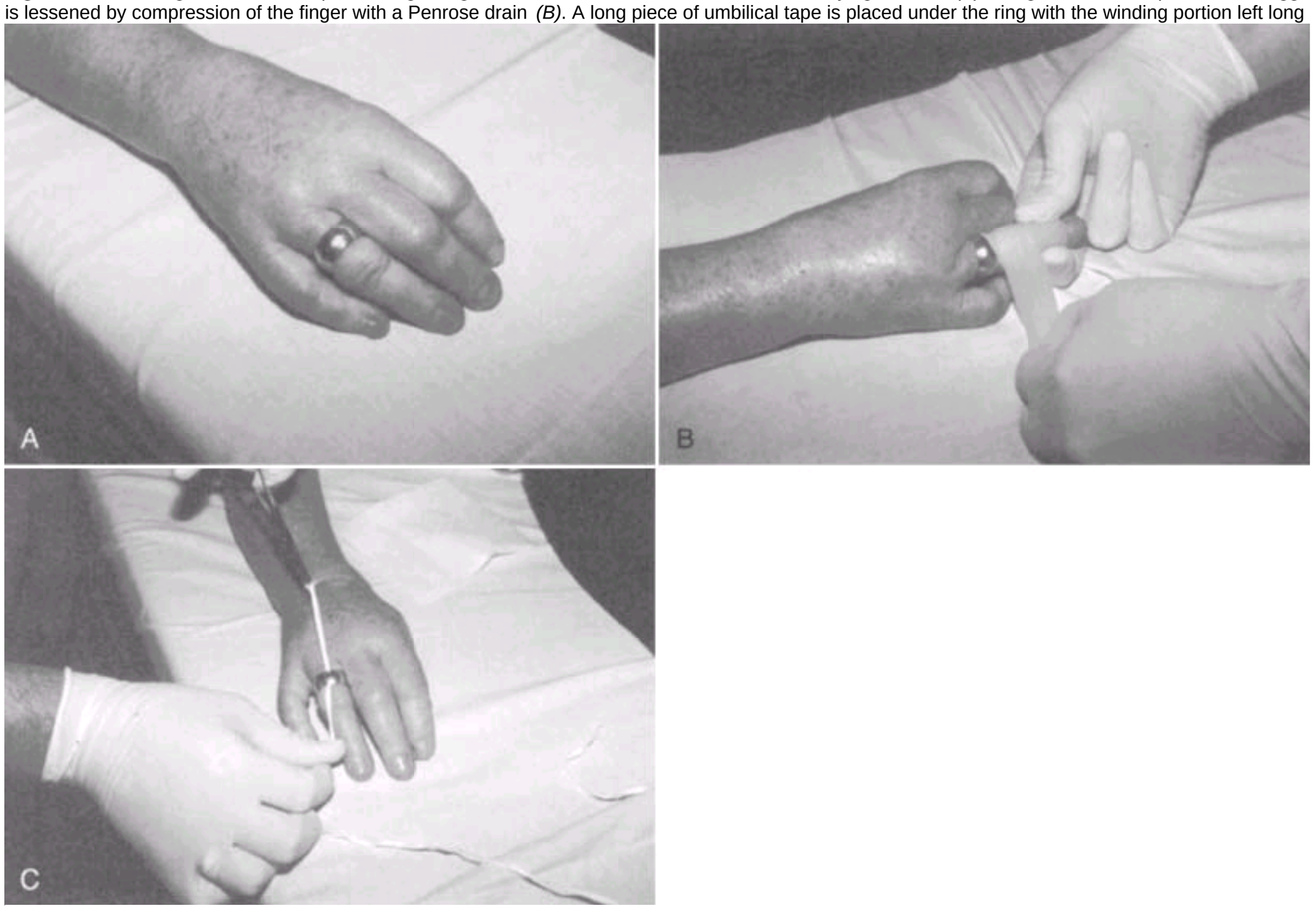
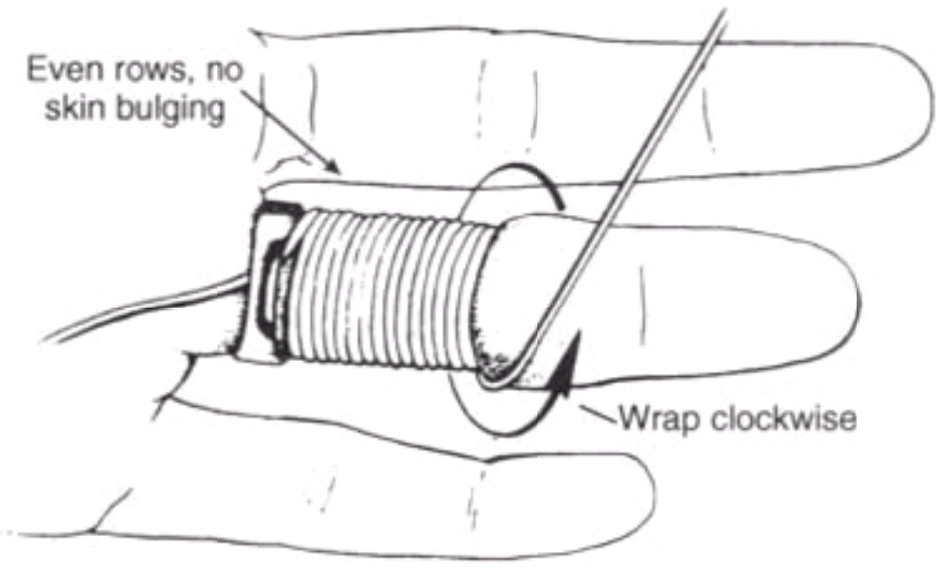
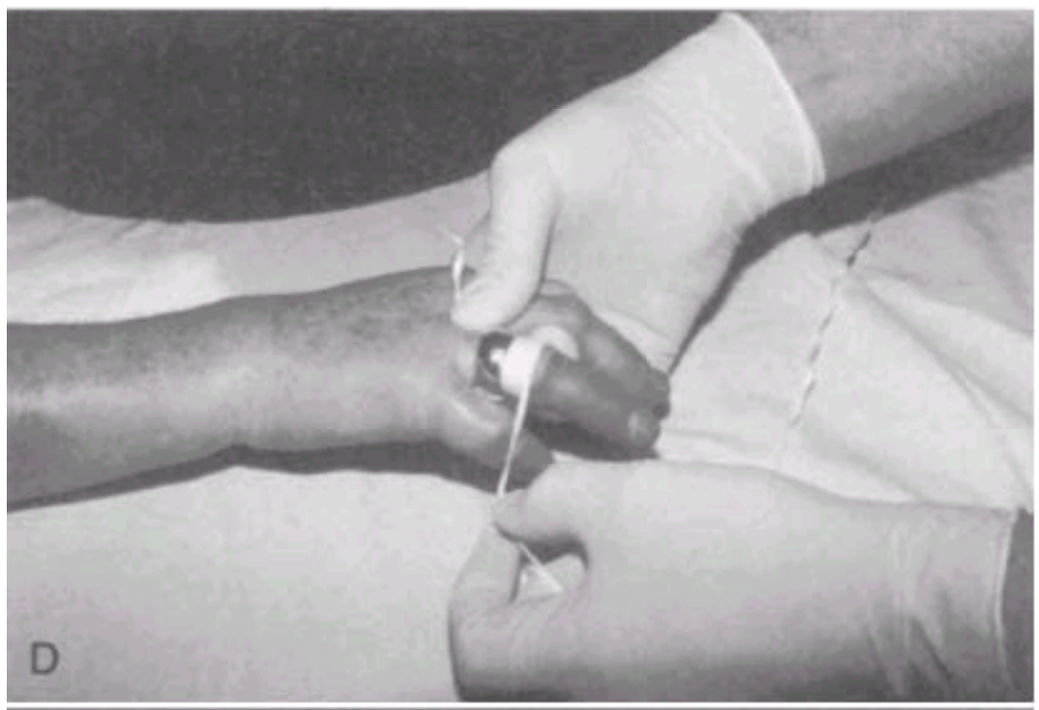
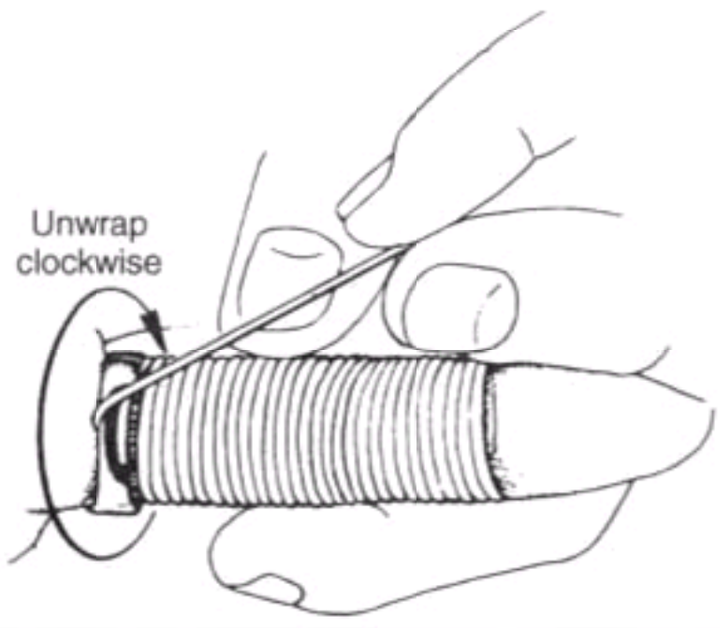


Figure 37-22b Begin winding either clockwise (*inset*) or counterclockwise (*photograph*) to compress the skin *(D) (see details in inset)*. The ring is removed by unwrapping the tape proximally and in the same direction it was wrapped *(E* and *F)*. The most difficult area to negotiate is the proximal interphalangeal joint. Note abrasion of skin after removal.







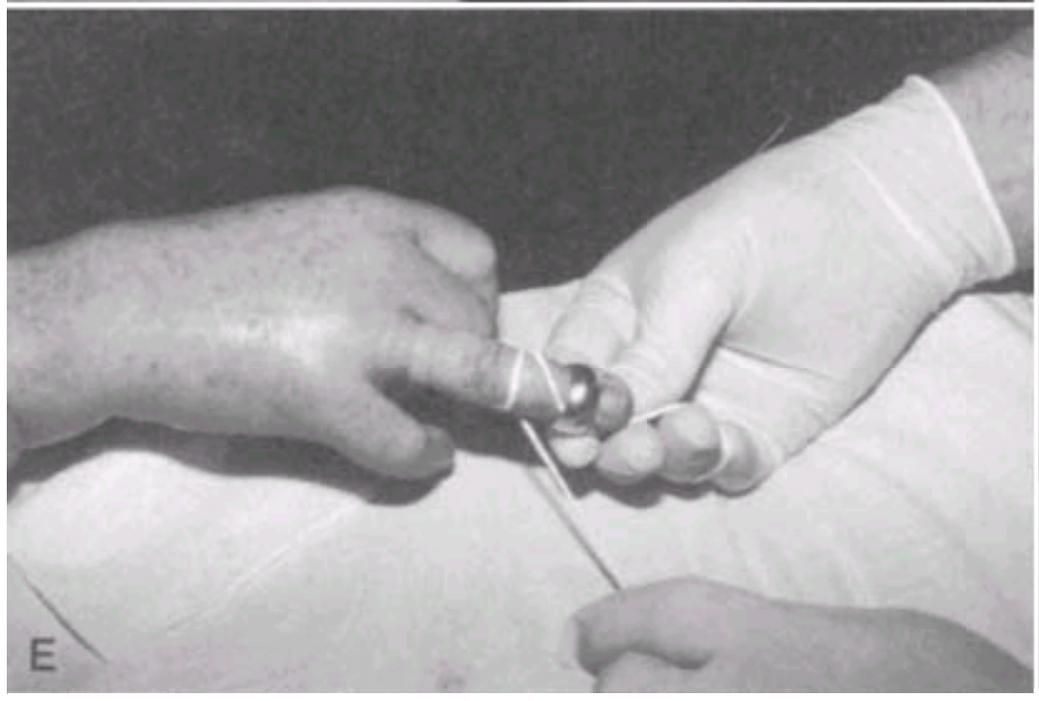


Figure 37-23 Ticks should be removed as soon as possible to minimize the transmission of tick-borne pathogens and to limit their fixation to the skin by a secreted cement compound. This engorged tick has been attached for about a day *(A)* and has burrowed under the skin. Most home remedies are worthless. A recommended approach is to grasp the tick with forceps near its head *(B)* where it enters the skin (avoid the soft body) and gently pull it out. Some advise twisting the head counterclockwise, but this has not objectively been found to be more effective. If pieces of the tick remain, they should be dug out.

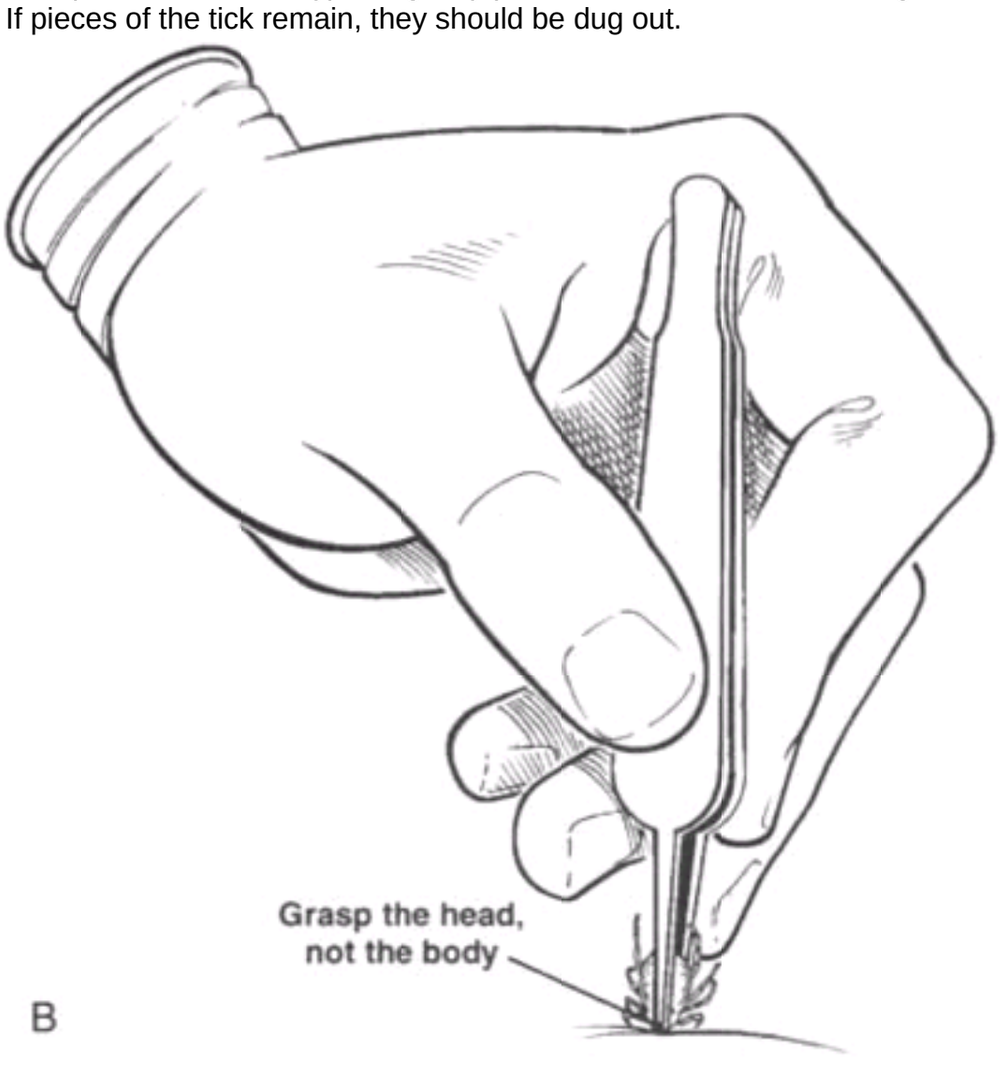


Figure 37-25 This child has multiple hairs compromising the circulation to two toes. The hairs are deeply embedded in the skin creases and cannot be visualized. The only way to ensure removal of the constriction is to cut the depth of the folds with a scalpel blade (using a dorsal incision to avoid the neurovascular bundle) and attempt to extricate individual fibers. Return of circulation should be obvious by temperature and color change in the affected digit(s), before it is assumed that all of the fibers have been cut.

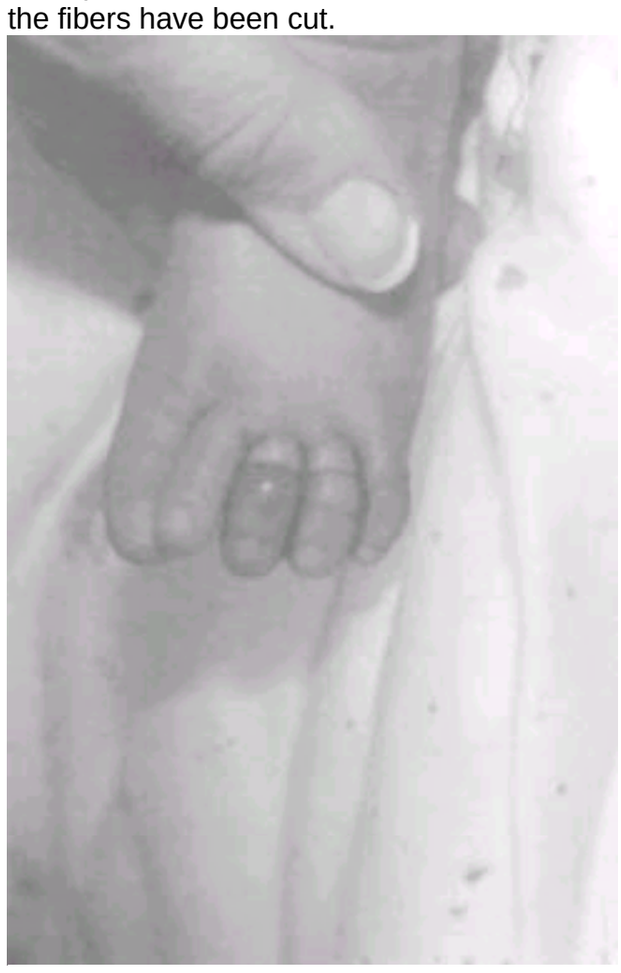


Figure 38-1 An abscess that appears in an atypical place or recurs after initial treatment is successful should raise the possibility of rare or underlying conditions. *A*, This IV drug user had an "abscess" of the chest wall drained in various EDs several times over a 2-month period, and it seemed to initially respond to drainage and antibiotics. He still manifested an area of cellulitis, minor fluctuance, and continued drainage near the center of the chest. This is an atypical place for a simple cutaneous abscess. Magnetic resonance imaging demonstrated an abscess of the sternolclavicular joint that was draining to the skin, simulating a recurrent cutaneous abscess. He required extensive surgical debridement and prolonged antibiotics. The etiologic organism was never ascertained, but pseudomonas is often present. *B*, This patient has a large "abscess" of the lateral chest wall that initially drained unusual gelatinous material, not frank pus. *C*, At follow-up 3 days later the abscess was much improved. The contents of the abscess had been sent for pathologic analysis since it had an unusual consistency, and it demonstrated a highly undifferentiated malignancy. The fluid was sterile. Normally, analyzing or culturing the contents of an abscess will not yield helpful information, but in this case the unusual consistency of the collection prompted further analysis. *D*, This patient had a sternotomy for bypass surgery a few months ago. She had been sporadically treated for a minor wound infection but then presented with a draining fluctuant mass at the inferior border of the sternum. This is the external manifestation of an extensive sternal osteomyelitis.

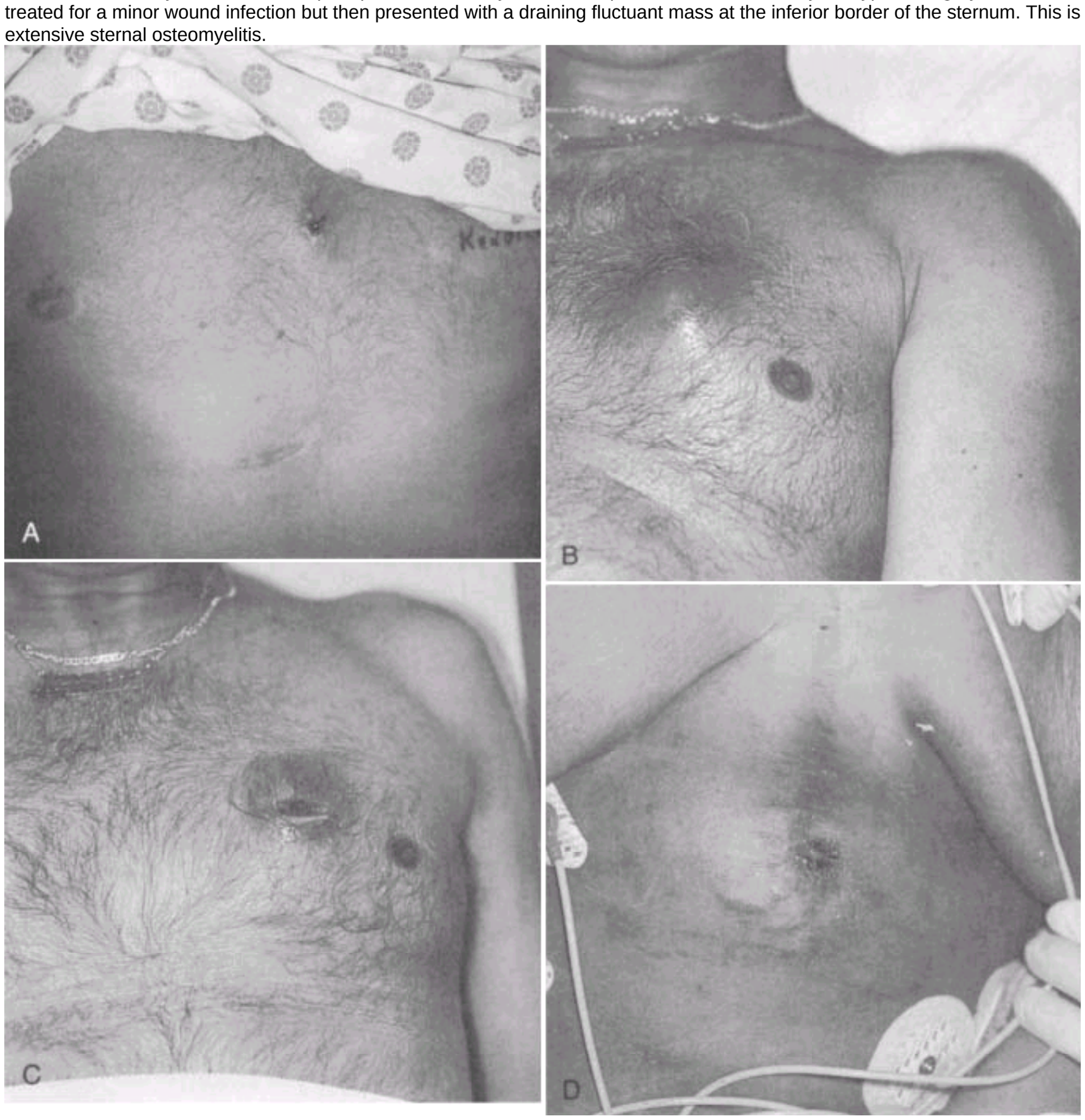


Figure 38-2 This patient presented with a large abscess of the deltoid area (arrow) and could offer no explanation for it *(A)*. This is a typical scenario for a drug user who injects directly into the skin, and the characteristic circular skin lesion from "skin popping" found on the legs *(B)* confirmed the clinical suspicion. Even though a drug screen was positive for opioids, the patient still denied drug use and attributed the leg lesions to frequent trauma on the job.

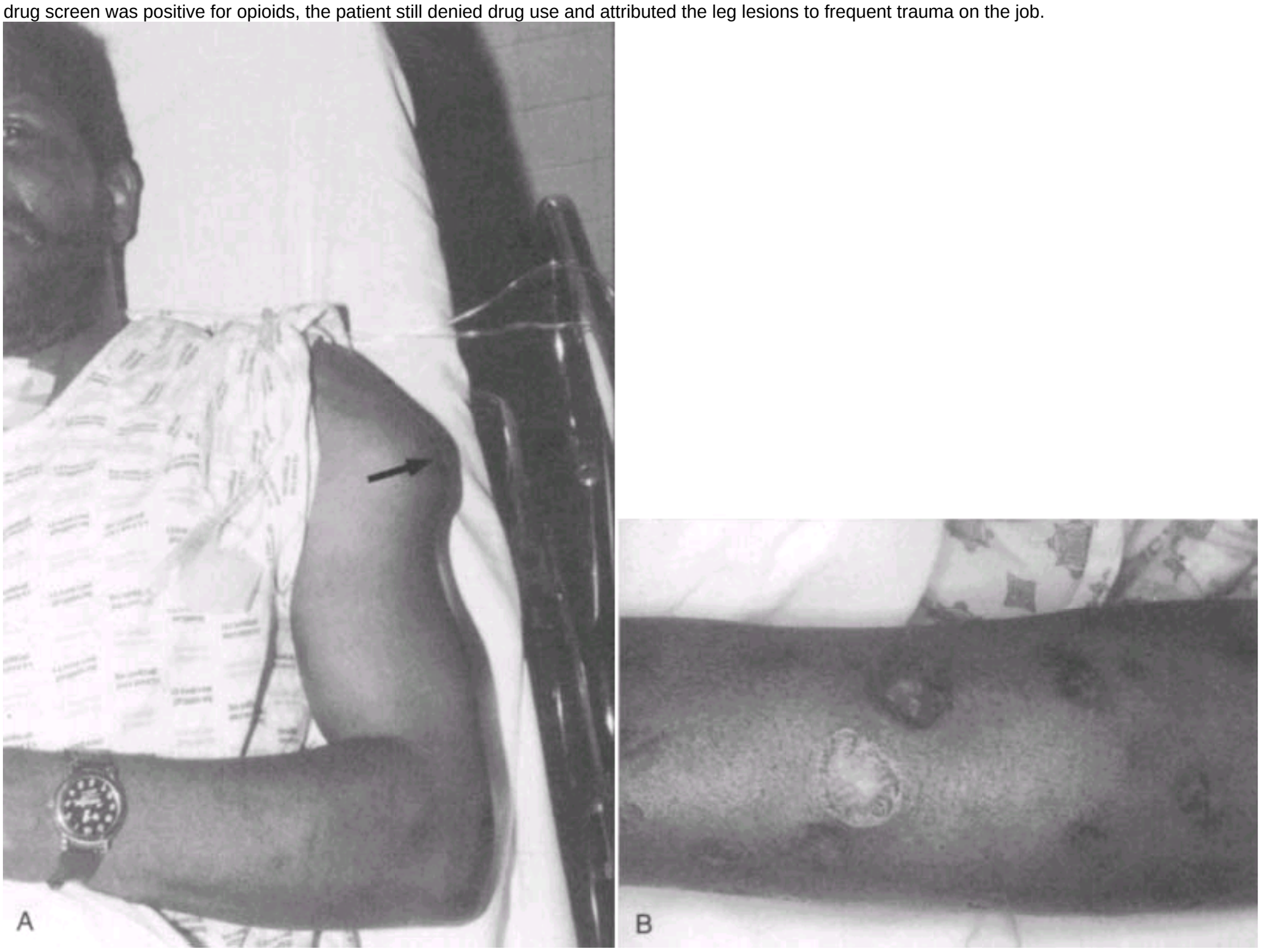
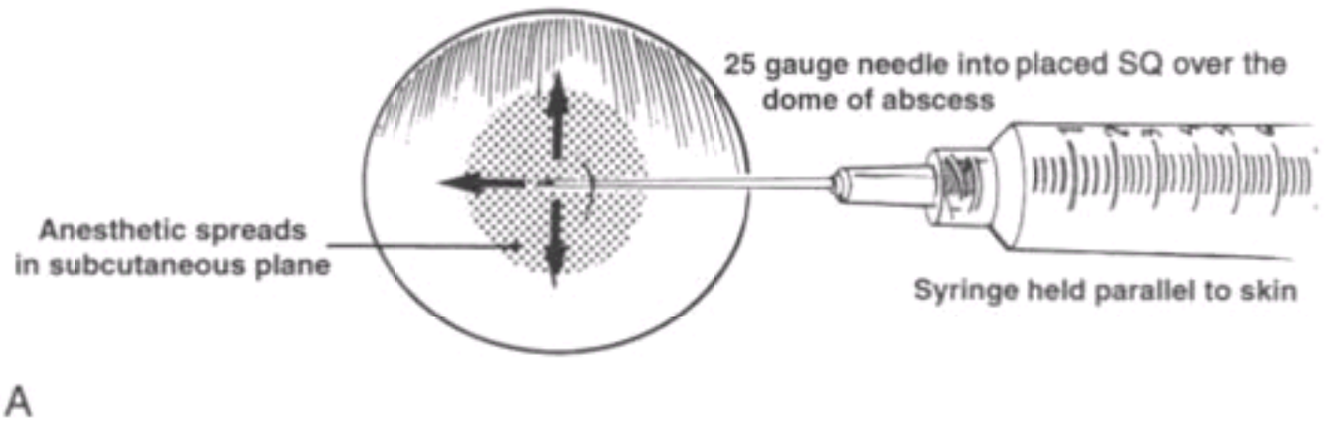


Figure 38-3 *A,* To anesthetize an abscess, hold the syringe parallel to the skin and insert a 25-ga needle into the middle of the dome of the abscess, so the needle is just slightly under the skin. The anesthetic will spread over the entire abscess without moving the needle position. *B*, Note the blanching of the skin that spreads to cover the entire abscess without repositioning the needle. *C,* The skin over the abscess is incised the full length of the abscess. A small stab incision does not allow for proper drainage. *D,* An instrument is inserted into the abscess cavity to spread the skin, drain the pus, and break up loculations. *E*, One way to break up loculations is to wrap a hemostat with gauze and, *F*, swirl it around the margins of the wound. *Do not use a gloved finger to break up loculations in an abscess that may contain a sharp or jagged foreign body.*



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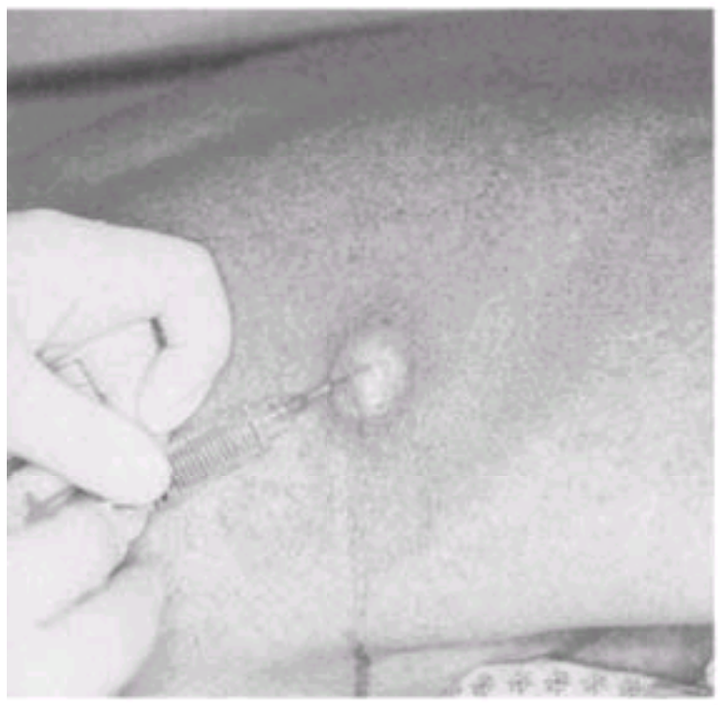

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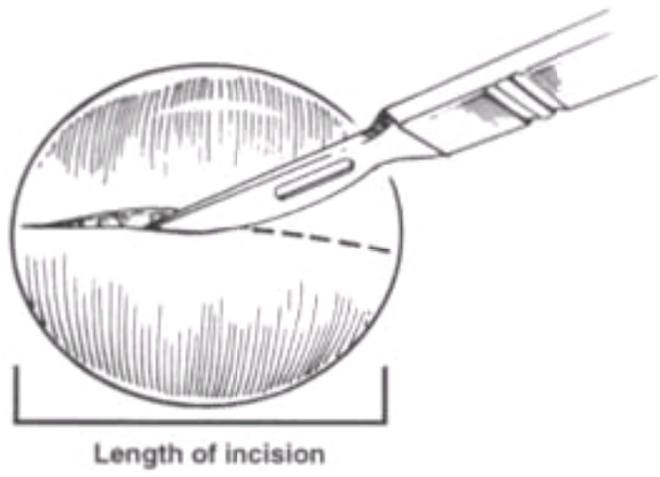

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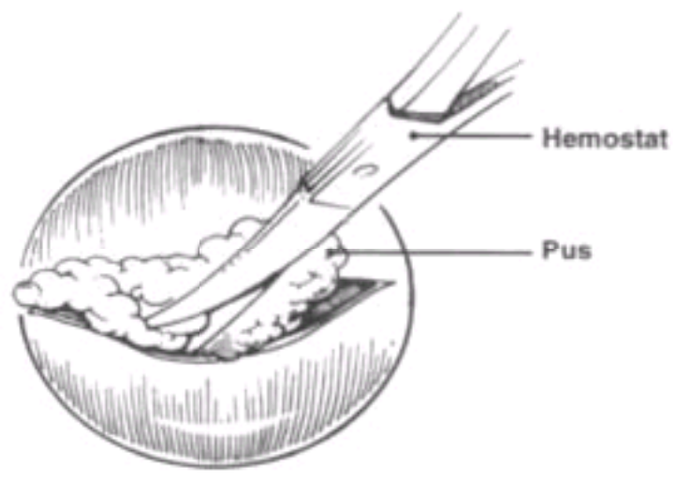

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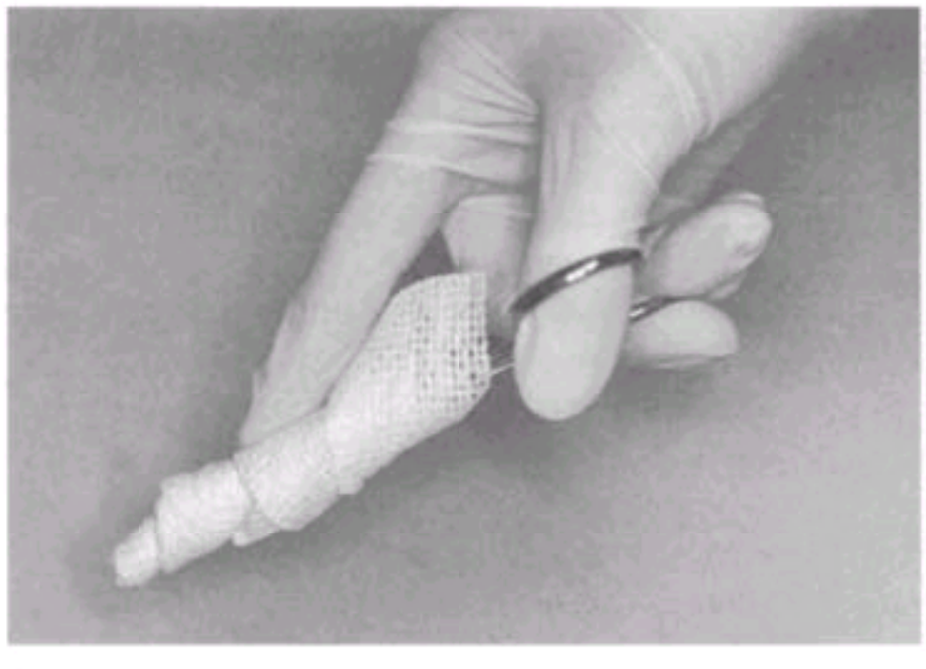

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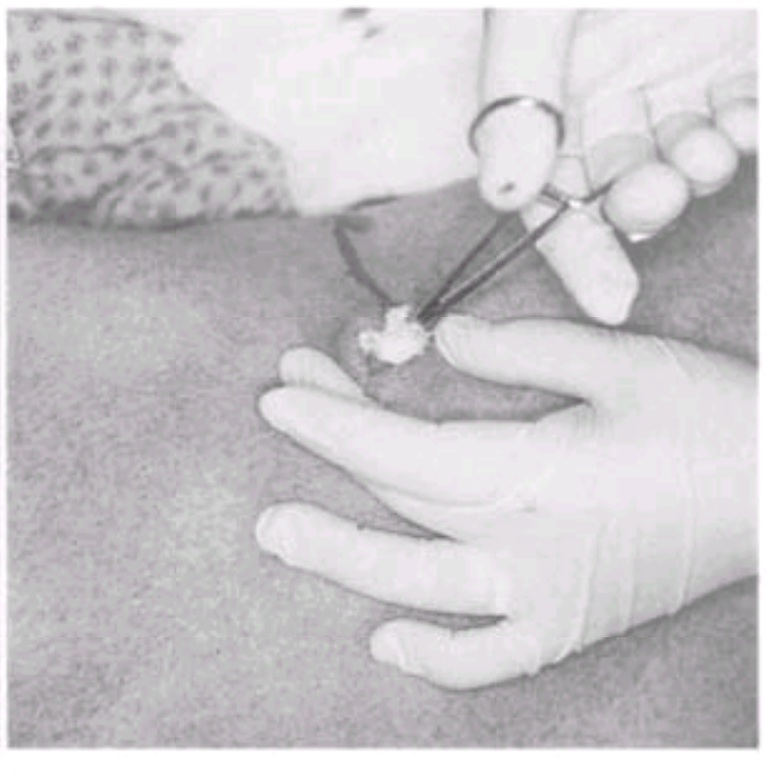


F

Figure 38-4 The relation of the elective lines of tension in the face to the underlying mimetic musculature. Only in the lower eyelid are these lines not perpendicular to the muscles. The left side of the drawing shows the use of this principle when common facial lesions are excised or a facial abscess is drained. *(From Schwartz SI, Lillehei RC: Principles of Surgery, 2nd ed. New York, McGraw-Hill, 1974. Reproduced with permission.)*

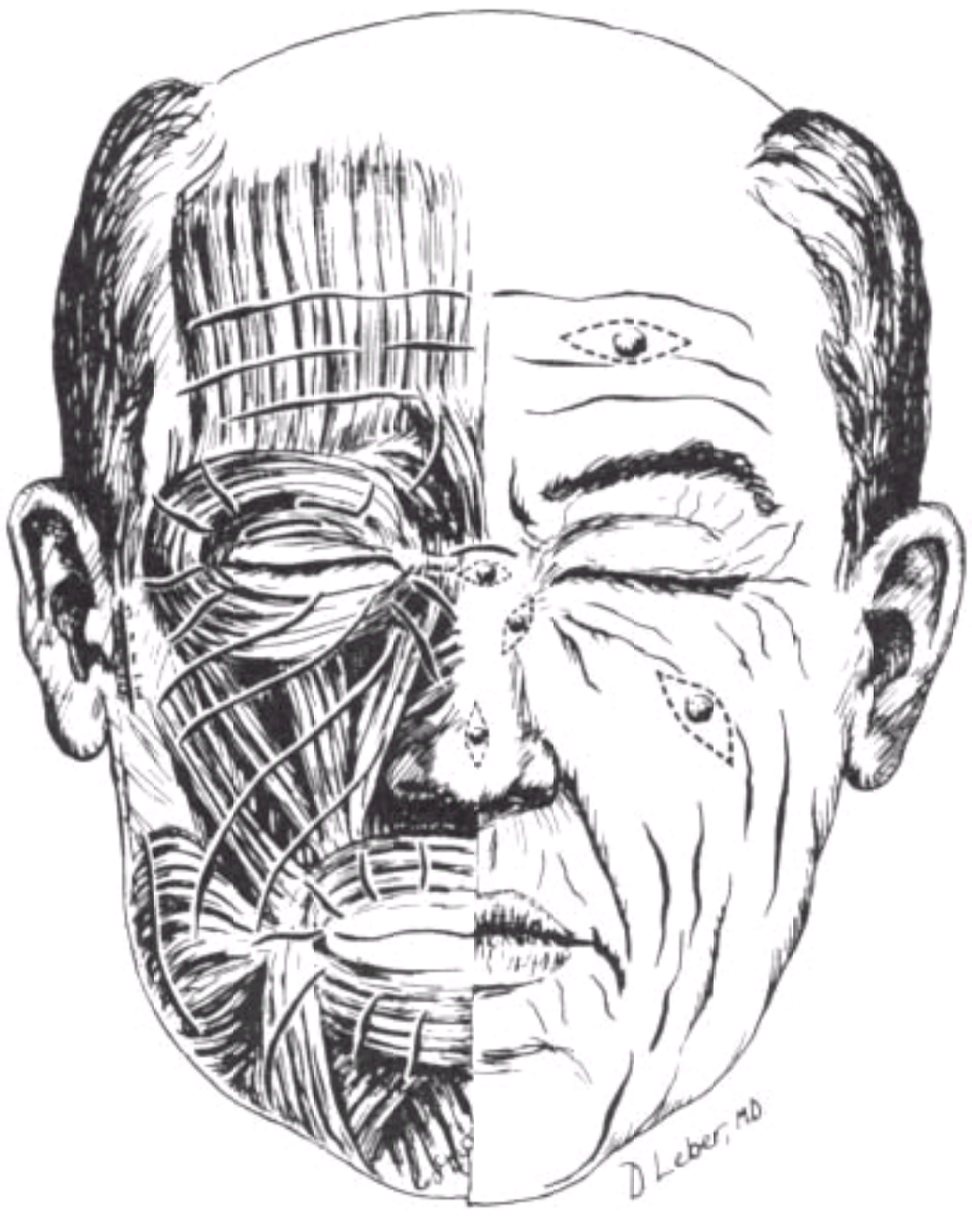


Figure 38-5 A simple linear incision is preferred over an "X" or crosshatched incision. In this case, a cutaneous post-surgical scalp abscess was drained by an "X" incision and the tips of the flap necrosed, leaving a slowly healing full thickness wound.

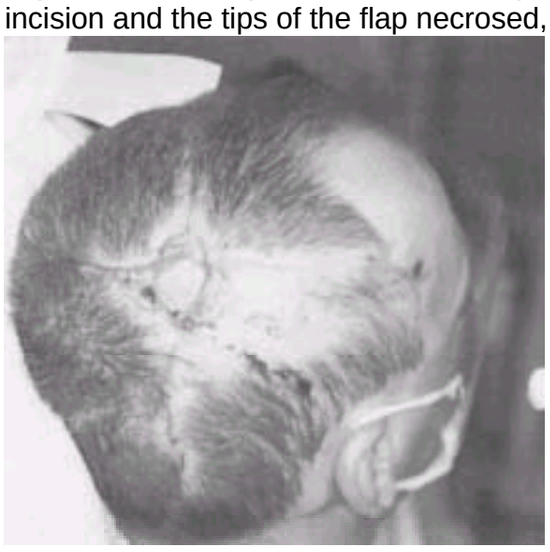


Figure 38-6 *A,* This intravenous drug user presented with an abscess *(arrow)* of the antecubital fossa. Following incision, the clinician attempted to break up the loculations with his finger. When a radiograph was obtained, three needle fragments (arrows) were found embedded in the wound *(B)*. The patient was HIV-positive and claimed no knowledge of the presence of the needles. Instead of using a finger, loculations should be broken up with an instrument.

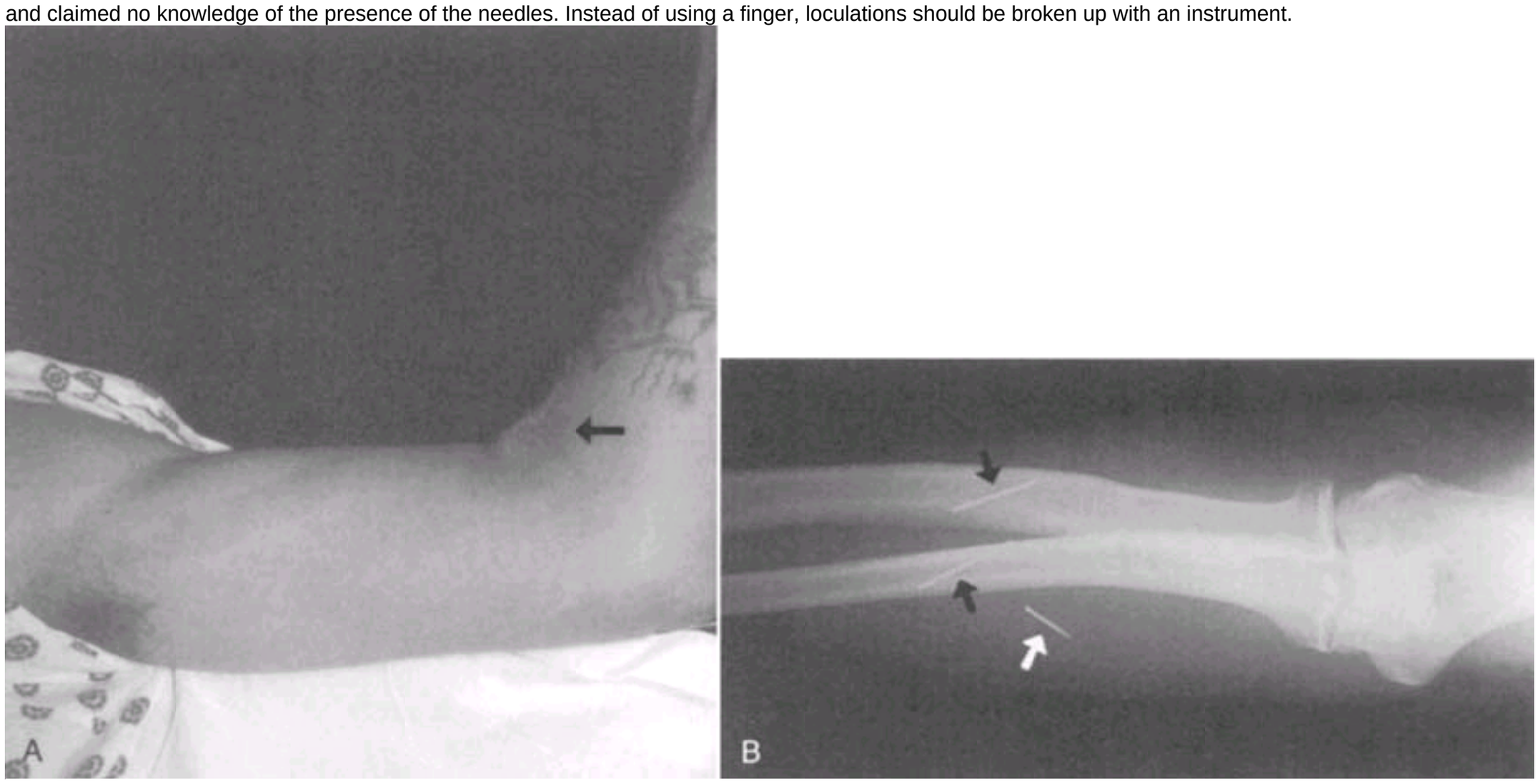


Figure 38-7 Packing change is usually performed by a health care professional. In the compliant and motivated patient, home care may replace frequent visits. Cotton-tipped applicators dipped in peroxide are used to swirl around the cavity to remove debris and the cavity is held open for tap water irrigation.

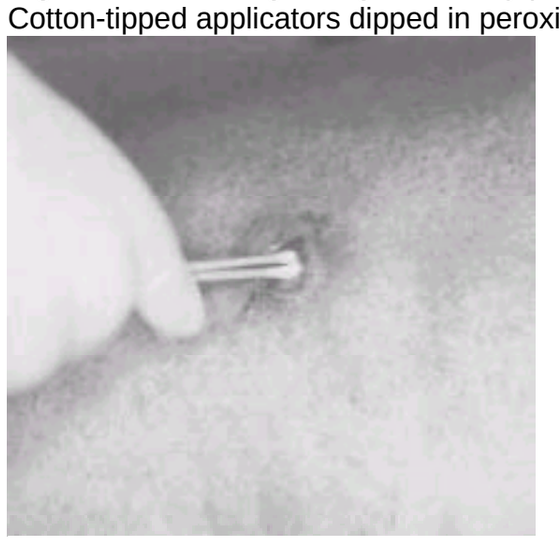


Figure 38-8 A carbuncle is a complicated abscess on the nape of the neck. It is very common in diabetics *(A)*. Because of many crypts and loculations, simple incision and drainage are often not readily curative. This patient had multiple incisions yet was not cured *(B)*. Antibiotics may augment healing of this abscess, but wide surgical excision may be required.

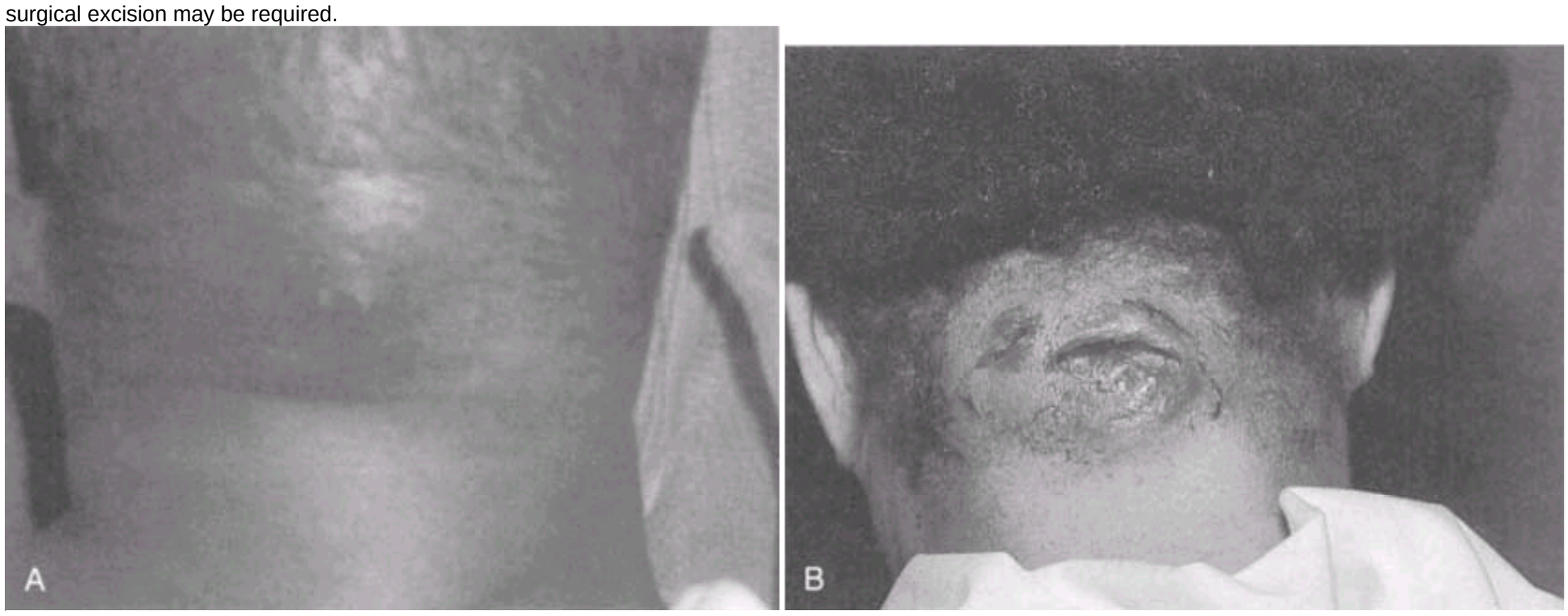


Figure 38-9 Hidradenitis suppurativa of the groin or axilla is a complicated series of abscesses that may not be amenable to simple incision and drainage. In the case of involvement with the groin, extensive surgery was required to excise recurrent infection.

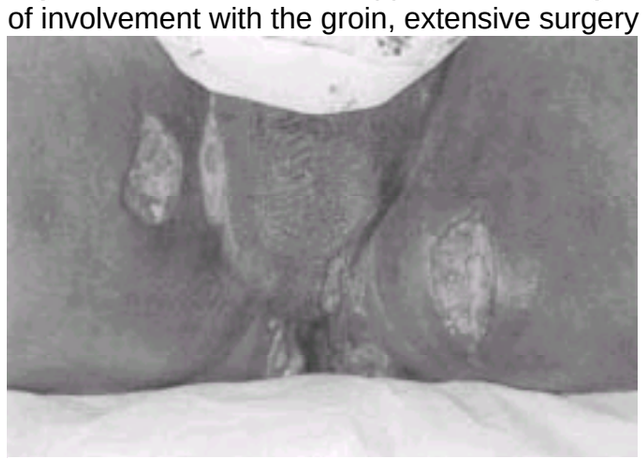


Figure 38-10 *A,* A superficial breast abscess may be drained with a linear incision that radiates from the nipple. *B* and *C,* Diagrams of intramammary abscess *(B)* and retromammary abscess *(C)*. Both require drainage under general anesthesia. The abscess itself may not be fully appreciated if it is deep-seated, and the mistaken diagnosis of cellulitis may be made. *(Redrawn from Wolcott MW: Ferguson's Surgery of the Ambulatory Patient, 5th ed. Philadelphia, JB Lippincott, 1974. Reprinted with permission.)*

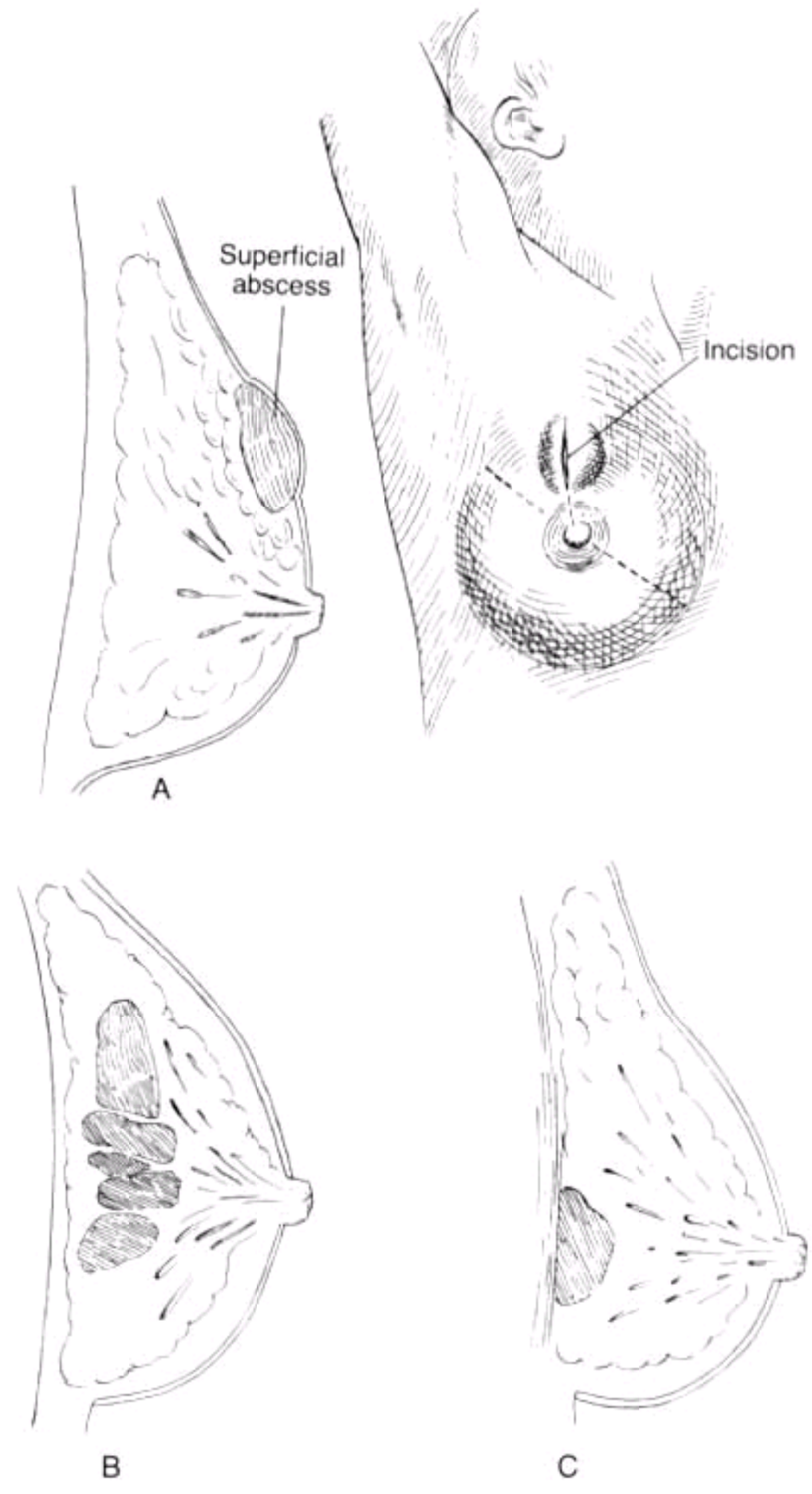


Figure 38-11 Use of the Word catheter for outpatient drainage of a Bartholin gland abscess. This is a fistulization procedure rather than a standard incision and drainage. A stab incision is made on the mucosal surface *(A)*. A catheter is inserted into the cyst cavity *(B)* and filled with 3 to 4 mL of water *(C)*. *D,* Inflatable bulb-tipped catheter. *Left,* Uninflated. *Right,* Inflated with 4 mL water. *(A through C from Word B: Office treatment of cyst and abscess of Bartholin gland. JAMA 190:777, 1964. D from Word B: Office treatment of cyst and abscess of Bartholin gland duct. South Med J 61:514, 1968. Reproduced with permission.)*

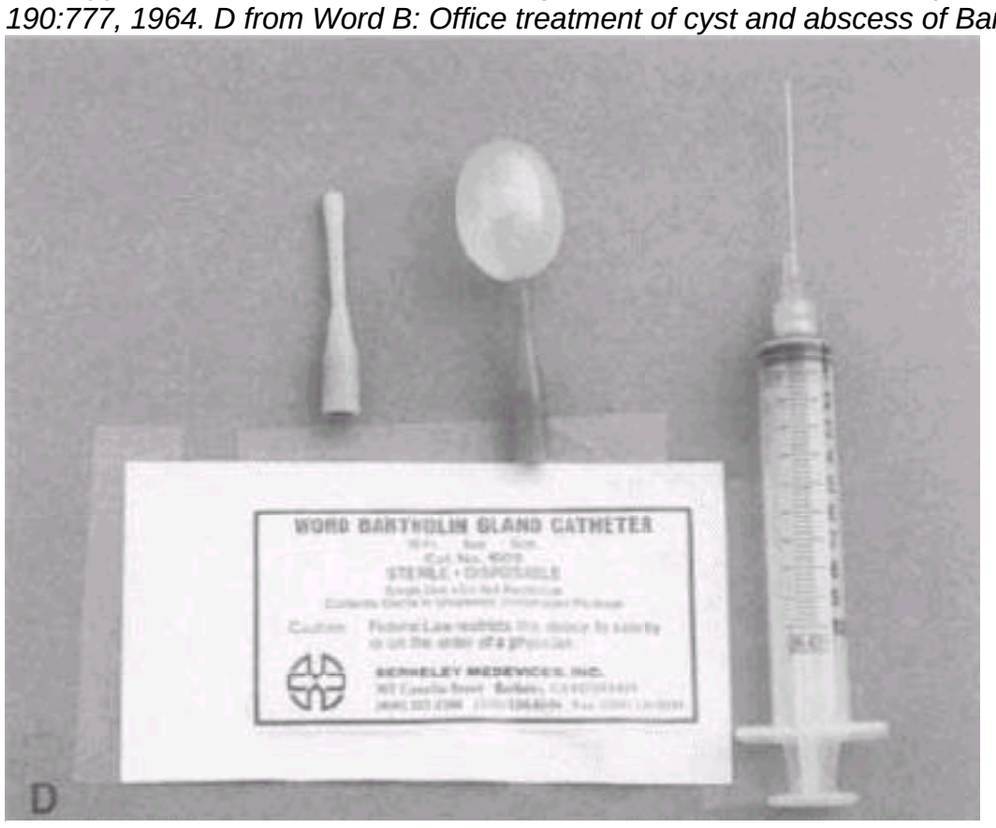


Figure 38-12a *A,* An alternative to formal incision, drainage, and packing for the treatment of this Bartholin abscess is to place a Word catheter. *B*, The abscess is stabilized with the thumb and index finger, and a local anesthetic is injected into the mucosal (not skin) surface. *C*, A stab incision is made with a scalpel. *D*, The abscess is punctured with a hemostat. Deep abscesses may be difficult to puncture.

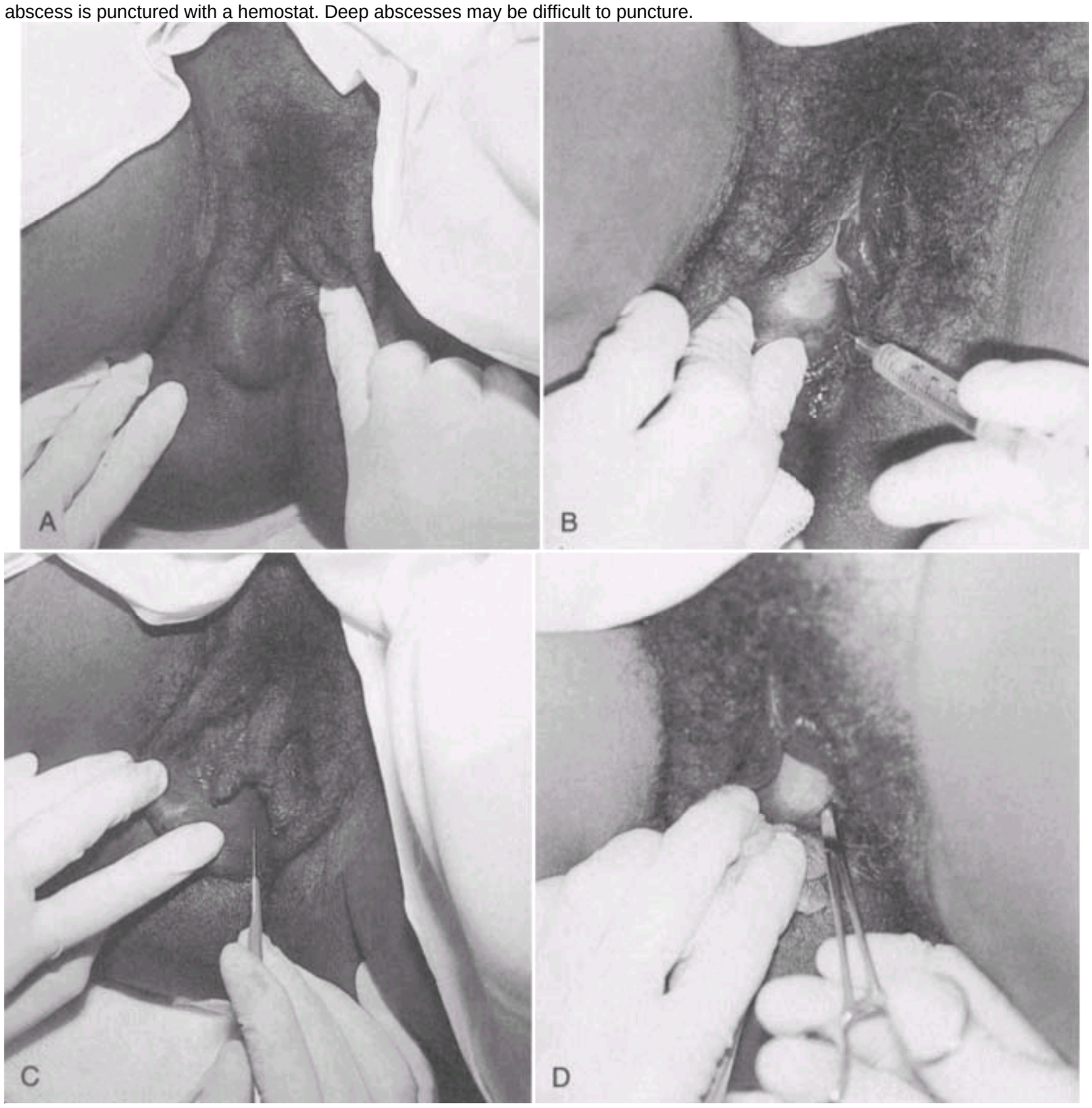
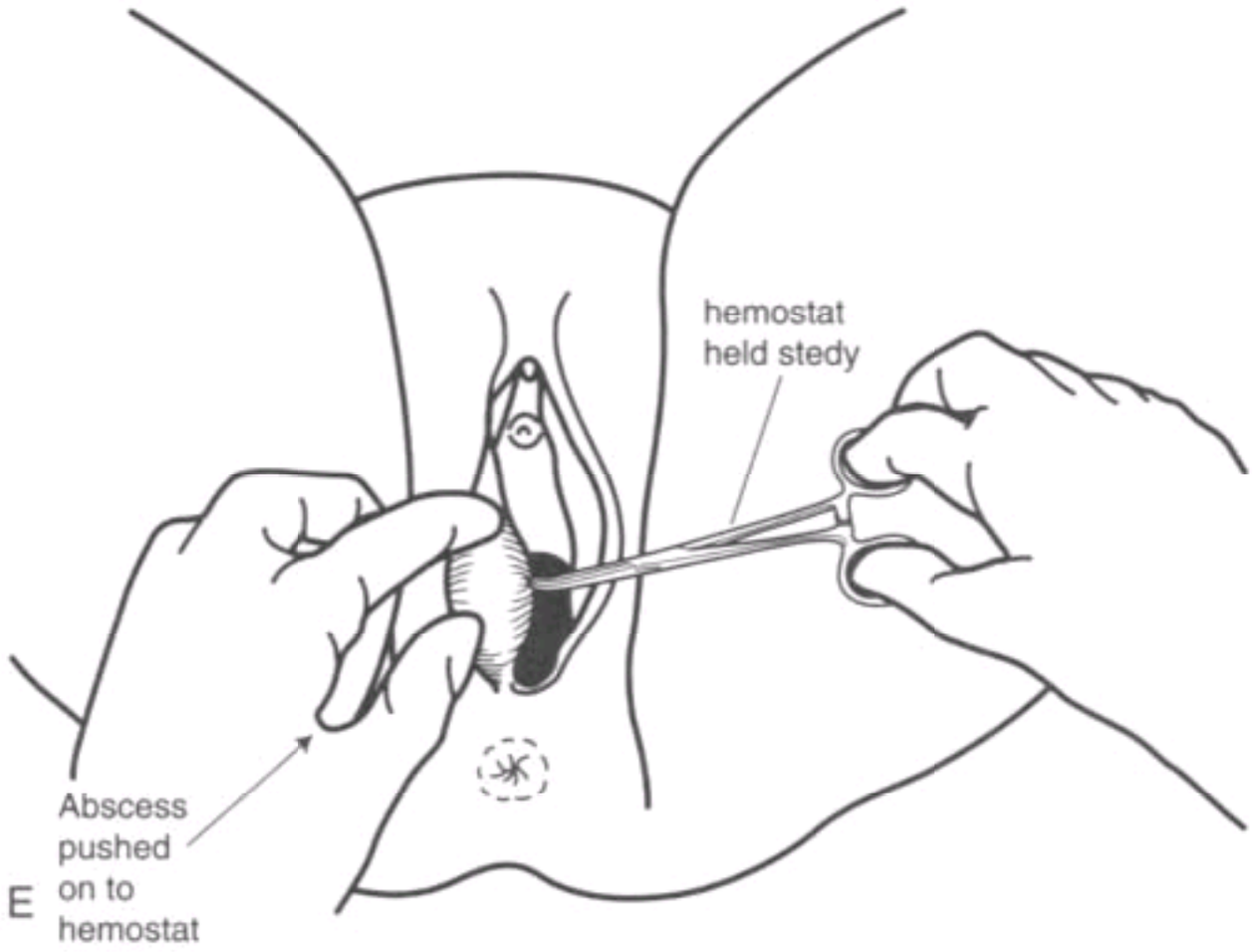
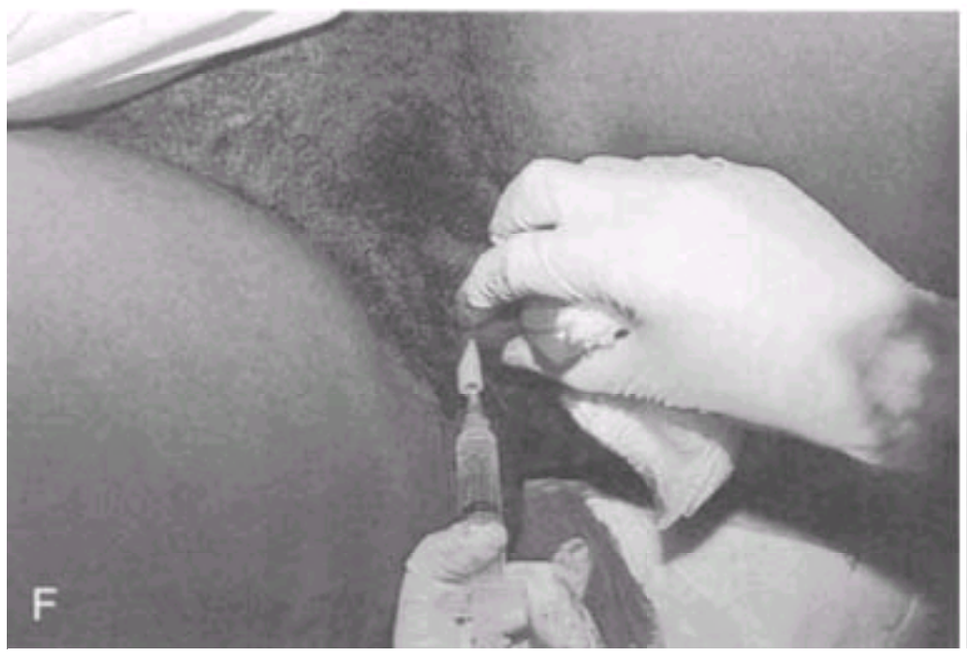


Figure 38-12b *E*, It is technically easier to enter the Bartholin gland abscess cavity if the hemostat is held steady and the abscess, held with the thumb and index finger, is skewered onto the hemostat. Attempting to puncture the deep immobilized abscess by stabbing with the hemostat may be more difficult. A palpable pop when entering the abscess or drainage of frank pus is expected. *F*, The catheter is placed to its hilt into the abscess cavity, and the balloon is filled with saline. Use a 25-ga needle to fill balloon. *G*, The catheter is left in place for 2 to 4 weeks to form a fistula.





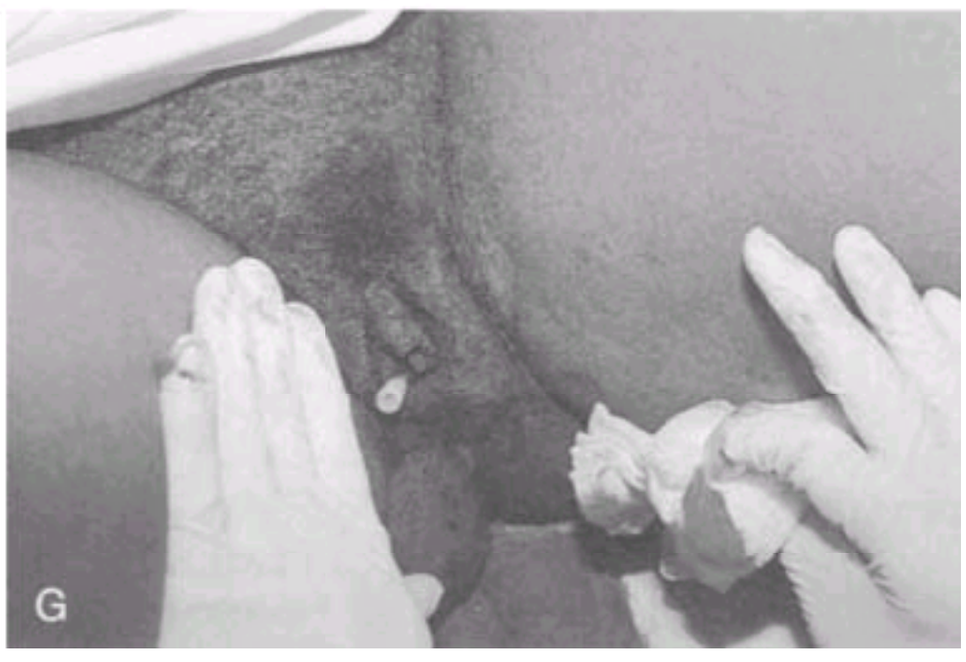


Figure 38-13 Pilonidal sinus. *A*, Sinuses occur in the midline some 5 cm above the anus in the natal cleft. *B*, Longitudinal section showing sinuses and pits. *(From Hill GJ II: Outpatient Surgery, 3rd ed. Philadelphia, WB Saunders, 1988. Reproduced with permission.)*

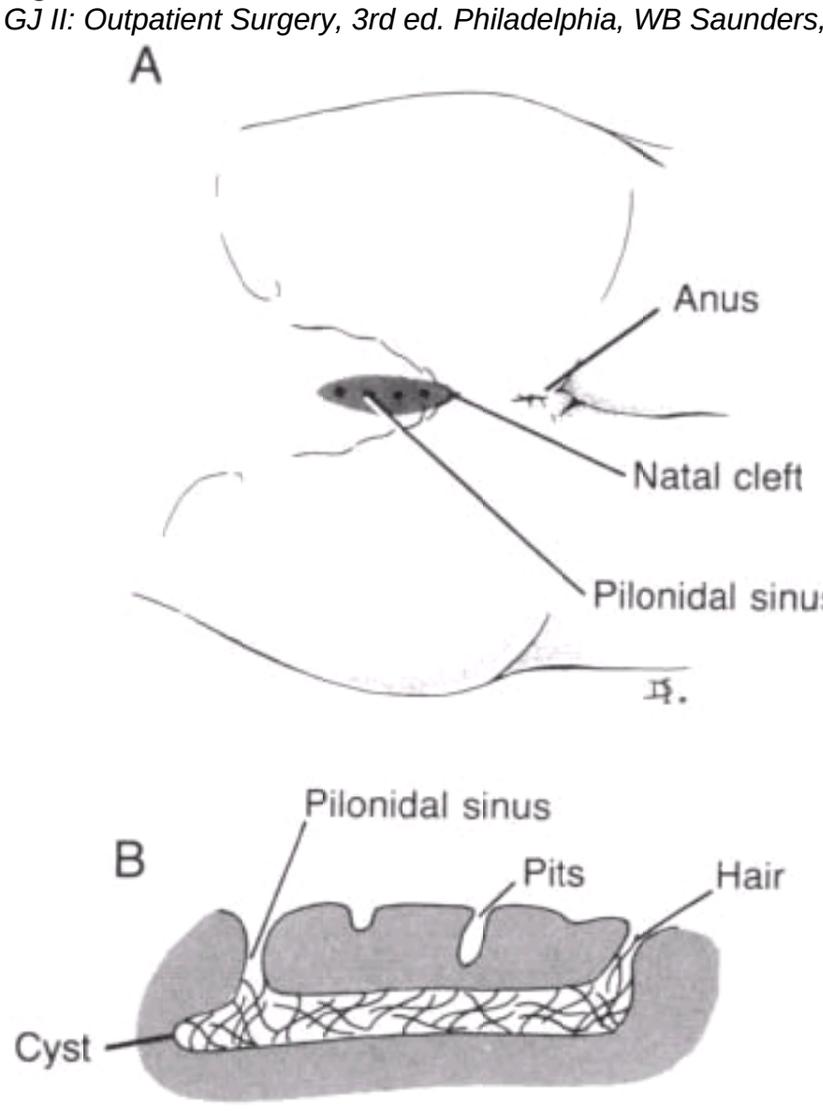


Figure 38-14 *A*, A pilonidal abscess can be quite extensive (*induration outlined by marker*) but it may be difficult to feel fluctuance because of the depth of the infection. *B*, Local anesthesia and conscious sedation make the initial ED incision and drainage successful. *C*, A deep incision yields copious pus, and occasionally other debris, such as hair. *D*, The abscess cavity is packed open. *Not shown*: When infection and inflammation have subsided, definitive treatment may include wide excision of the sinus and its lateral tracks.

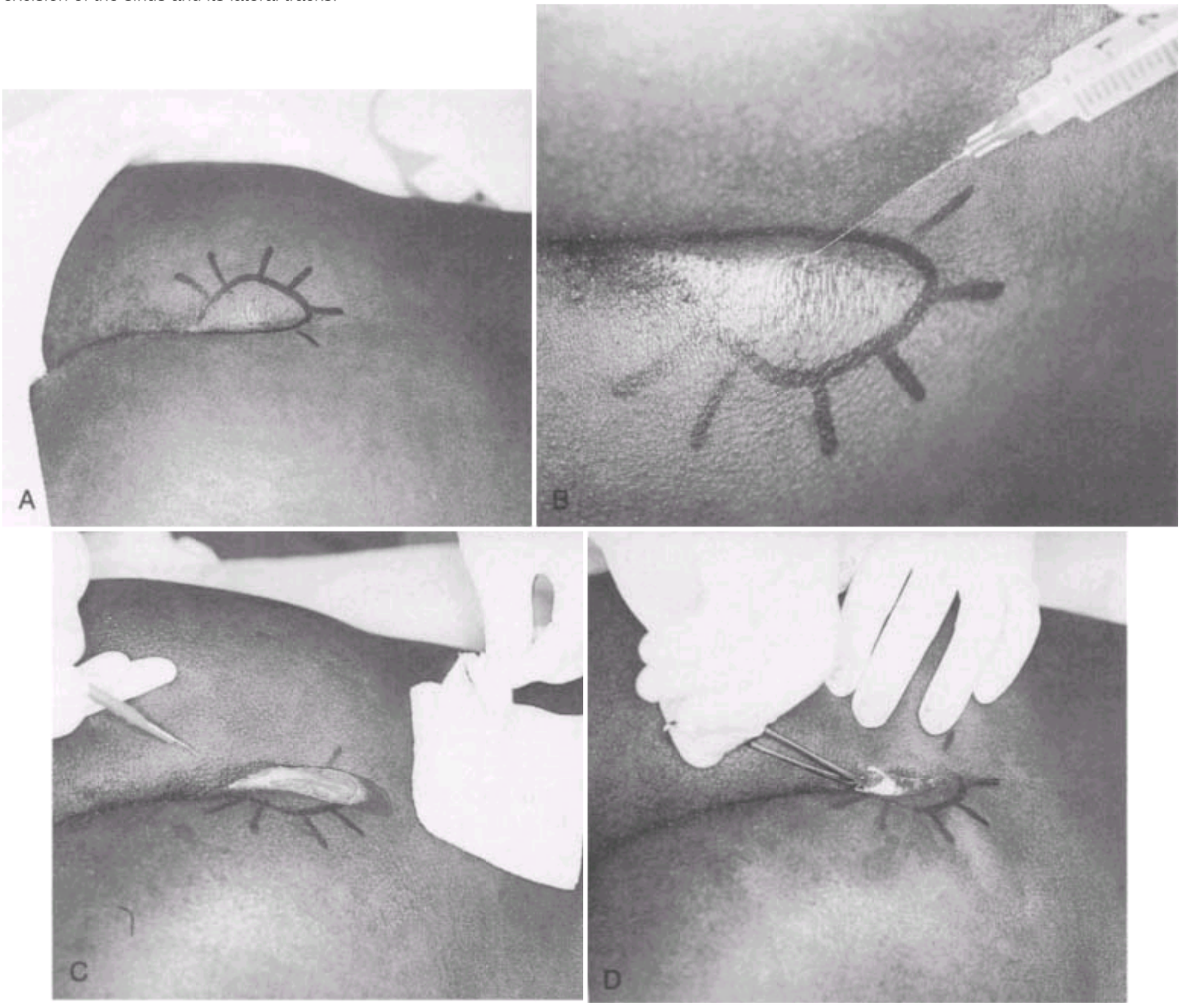
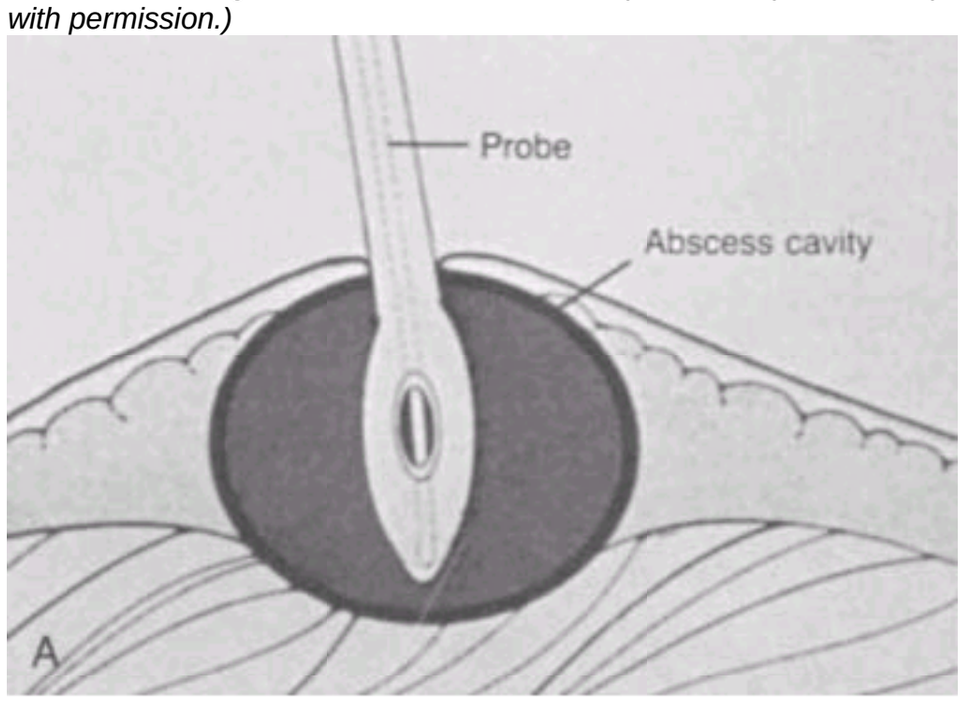


Figure 38-15 A method of prolonged drainage of a pilonidal abscess with a flared-end de Pezzer catheter. Following a stab incision, a stretched catheter (probe inside lumen) is inserted into the abscess cavity *(A)*. When the probe is removed, the head of the catheter expands and remains in the abscess cavity *(B)*. Drainage is continuous through the lumen of the catheter. *(From Phillip RS: A simplified method for the incision and drainage of abscesses. Am J Surg 135:721, 1978. Reproduced with permission.)*



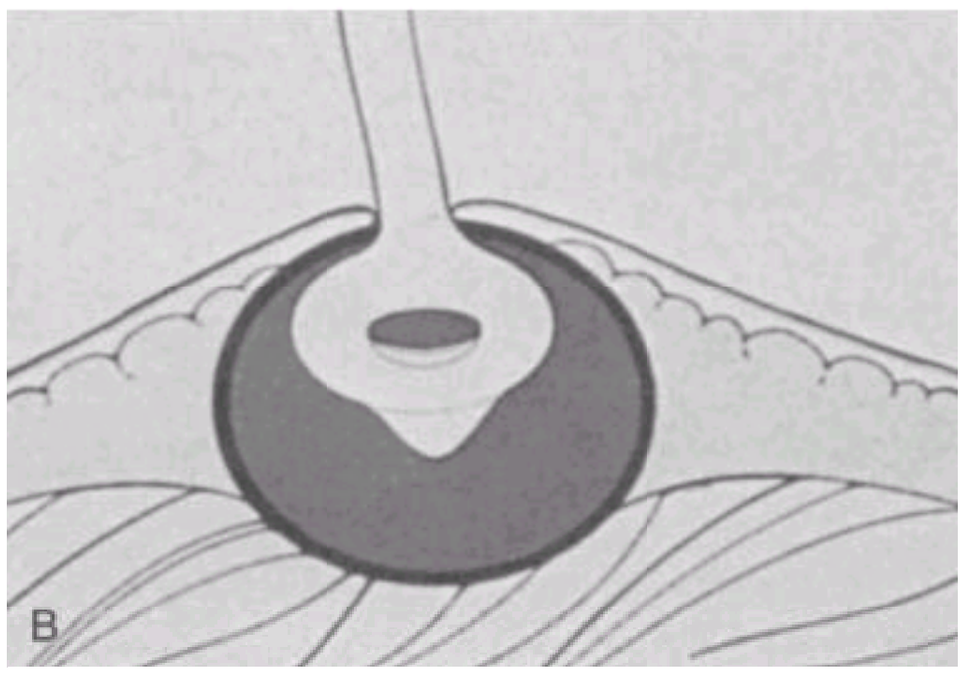


Figure 38-16 *A*, If a perirectal abscess spontaneously ruptures and drains, a formal incision, drainage, and packing should still be performed. *B*, A deep, poorly localized perirectal abscess of this size simply cannot be adequately drained in the ED. Arrows outline the area of induration. This patient requires extensive drainage under general anesthesia. A computed tomography scan may further evaluate the location of this abscess.

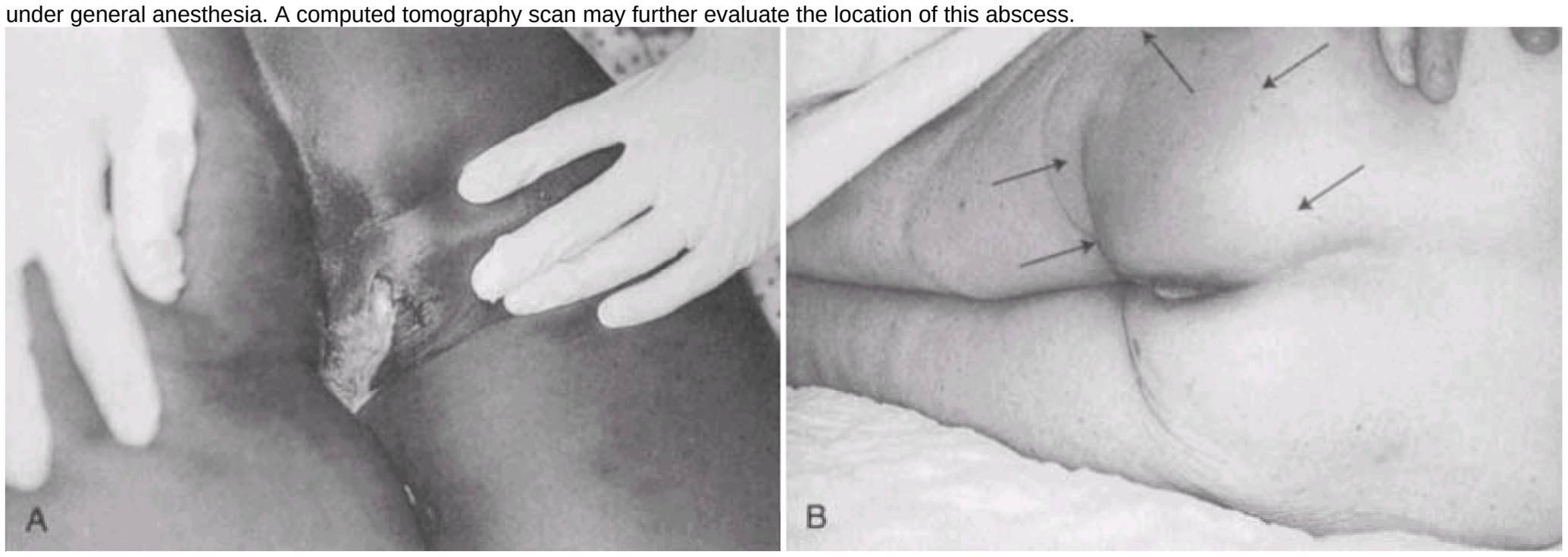


Figure 38-17 Schematic coronal section of the anal canal and the rectum. *(From Schwartz SI, Lillehei RC: Principles of Surgery, 2nd ed. New York, McGraw-Hill, 1974. Reproduced with permission.)*

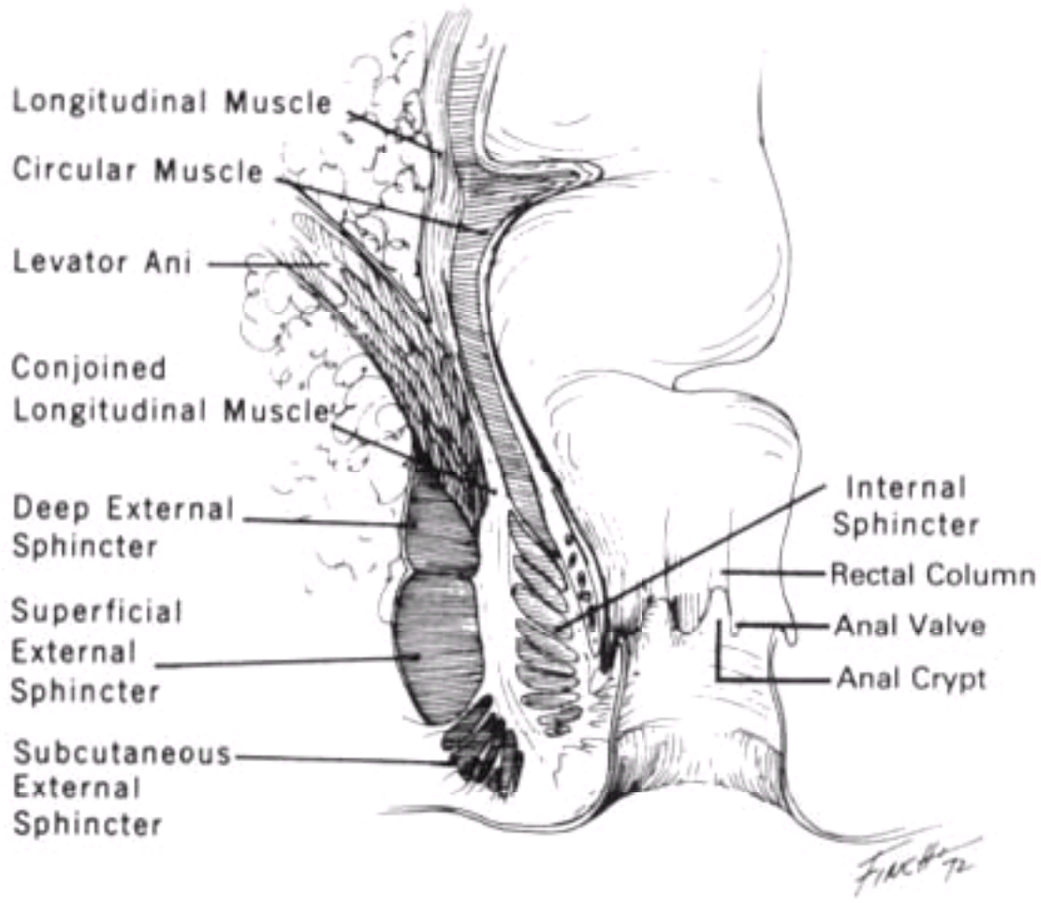


Figure 38-18 Classification of perirectal abscesses: 1, Perianal. 2, Ischiorectal. 3, Intersphincteric. 4, High intramuscular. 5, Pelvirectal. *(From Hill GJ II: Outpatient Surgery, 3rd ed. Philadelphia, WB Saunders, 1988. Reproduced by permission.)*

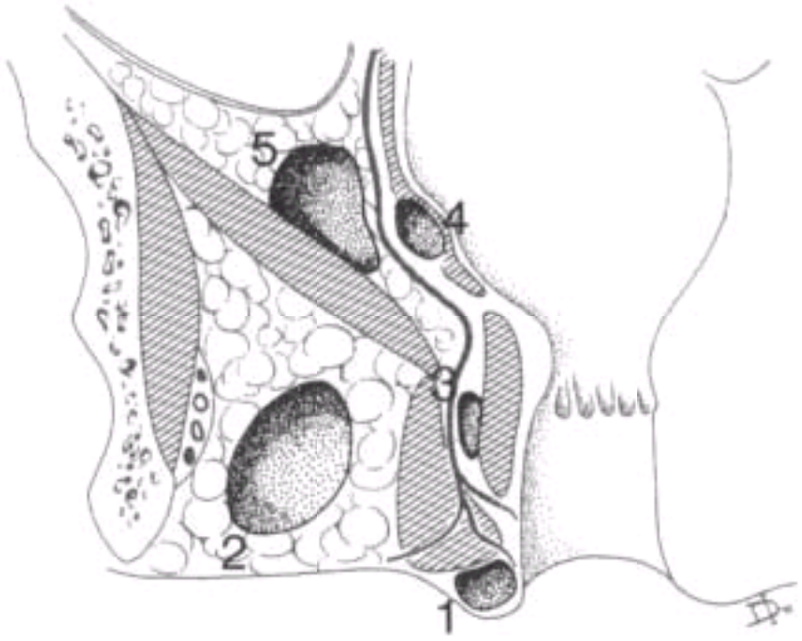


Figure 38-19 The thick, cheesy sebaceous material of a sebaceous cyst must be expressed after incision *(A)*. The shiny capsule of this infected sebaceous cyst is easily visible *(B)* and was removed in its entirety on the initial visit *(C)*.

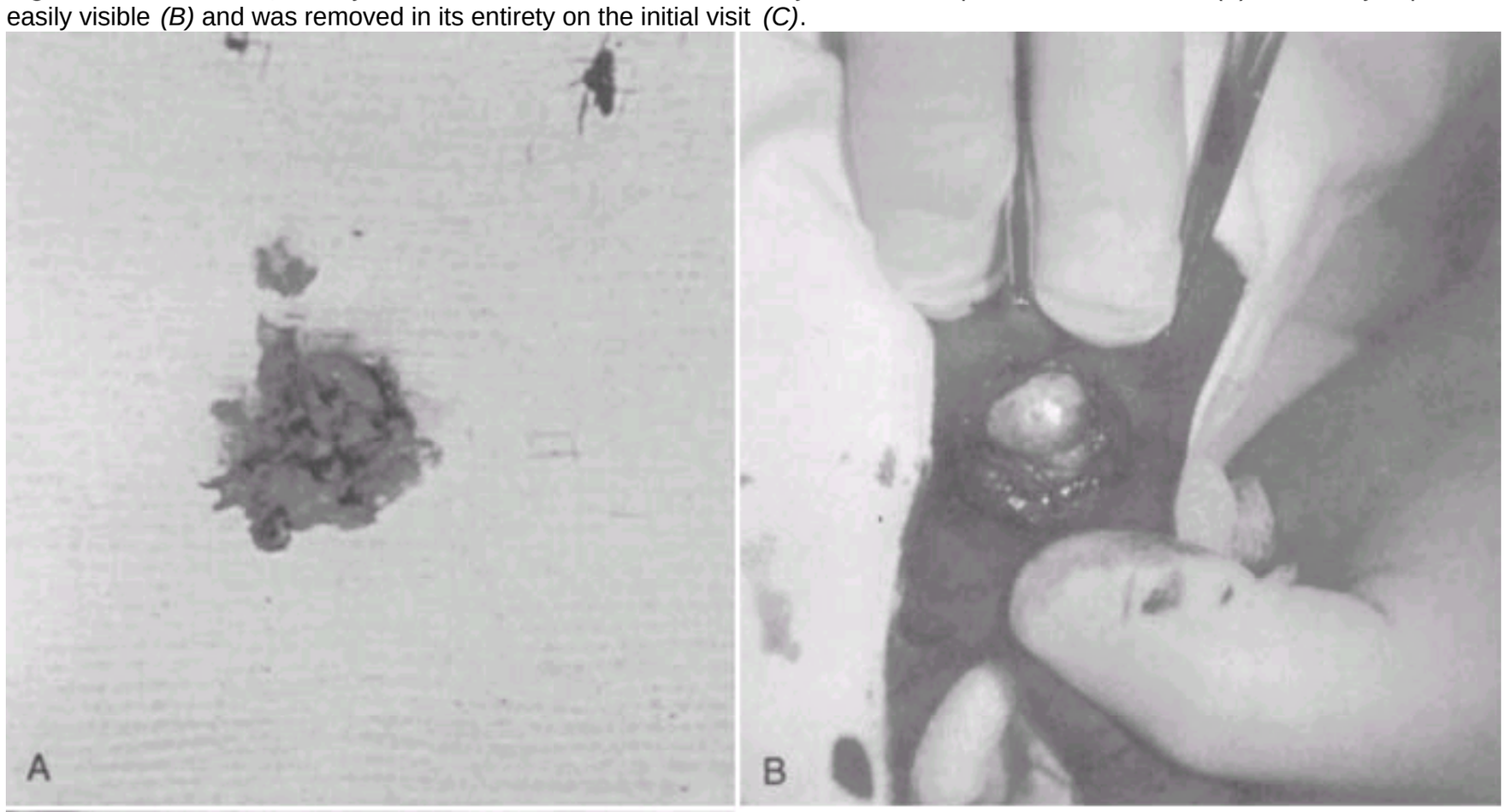


Figure 38-20 Paronychia. *1*, The site of the abscess at the side of the nail. *2*, The infection has extended around the base of the nail. It has raised the eponychium but has not penetrated under the nail. *3*, End stage of paronychia with a subeponychial and subungual abscess. *(From Wolcott MW: Ferguson's Surgery of the Ambulatory Patient, 5th ed. Philadelphia, JB Lippincott, 1974. Reproduced with permission.)*

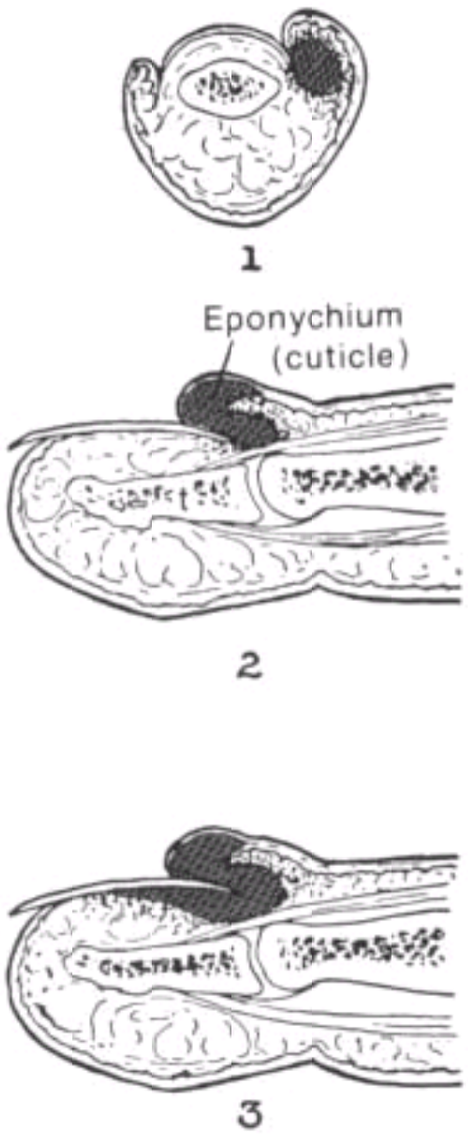


Figure 38-21 A paronychia may occur with obvious pus localization between the eponychium (cuticle) and the nail. *A*, Actual pus may be seen or, *B*, the area is swollen and tender. *C*, A paronychia can also occur in the toe. This is not a true cutaneous abscess but rather a collection of pus in a potential space. Actual skin incision is not required if eponychial elevation results in adequate drainage.

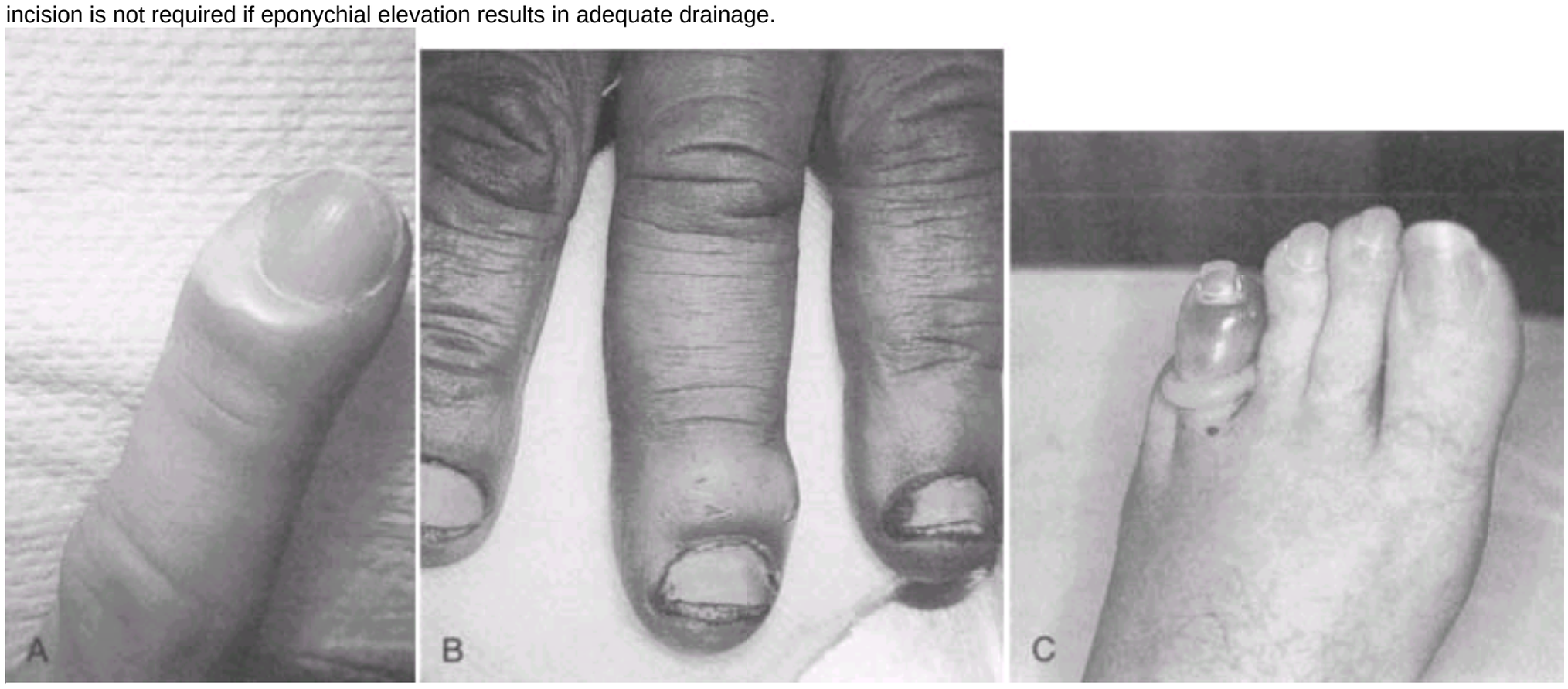


Figure 38-22 An initial treatment method for a well-localized paronychia. Following a digital nerve block, the eponychium (cuticle) is elevated at the area of greatest fluctuance, and no actual skin incision is made. A tourniquet can be used to limit bleeding and facilitate drainage. *A* and *B*, A sharp instrument (scissors or a No. 11 blade) is held parallel to the nail and advanced until pus is drained. *C* and *D*, A hemostat or scissors is placed into the base and all margins of the pus collection and spread in a fan-like fashion to open up the abscess. The entire pocket is opened to break up loculations and create a cavity. *E*, A small packing is placed for 2 days. At recheck a new pack may be placed or the cavity left open to heal. *Actual incision of tissue or removal of the nail is reserved for complicated or resistant infections and is not first-line therapy.*

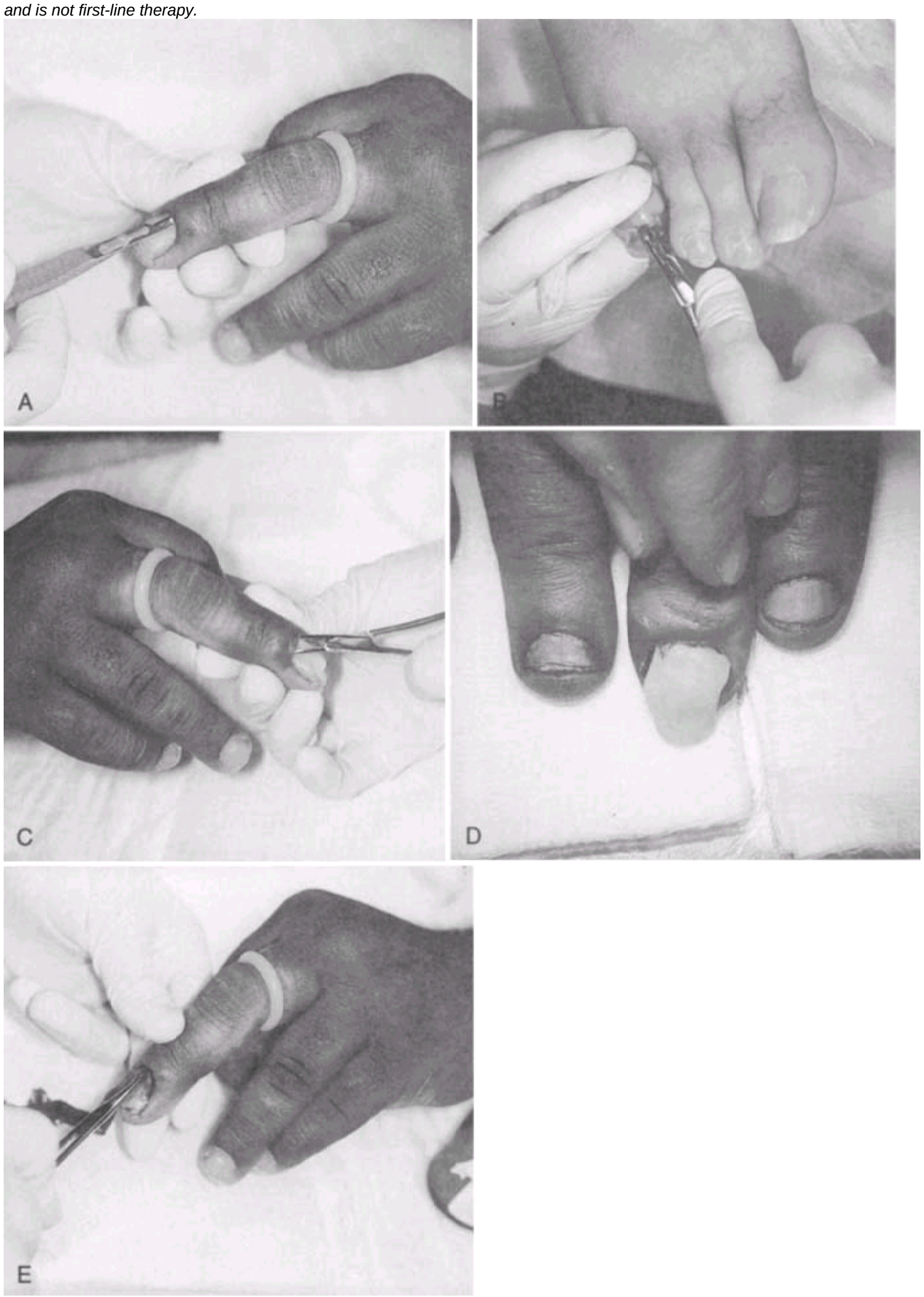
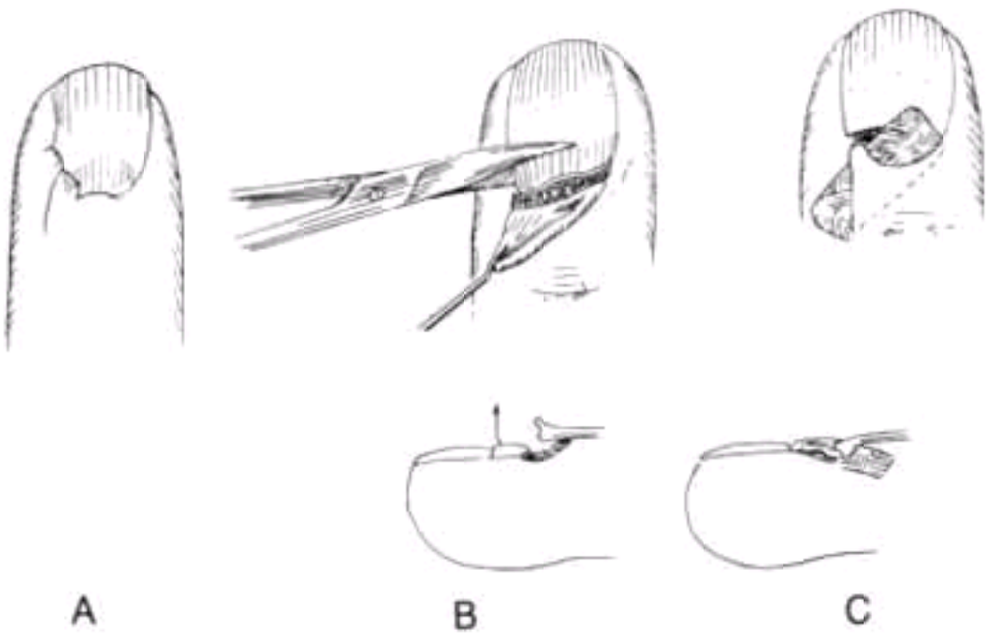


Figure 38-23 *A–C*, Aggressive treatment of recurrent paronychia or subungual abscess includes removal of a portion of the proximal nail and incision of the eponychium. *D,* Some physicians prefer to use a semilunar incision proximal to the eponychium rather than directly incising and potentially injuring the cuticle permanently. *These aggressive therapies are seldom required.*



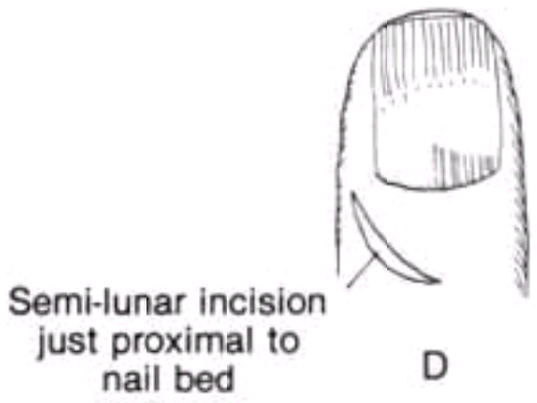


Figure 38-24 A well-developed felon. In this advanced case, the patient had little pain at the time of presentation, and the distal phalanx was almost completely resorbed owing to the extensive pressure and inflammation. This infection is extensive and warrants consultation with a hand specialist.

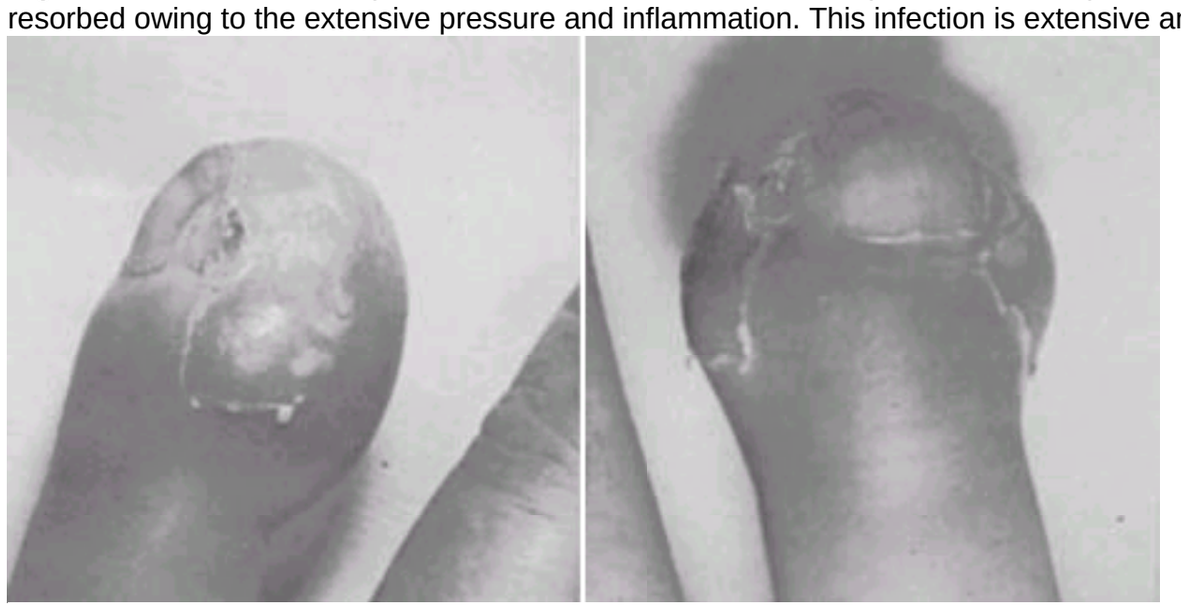


Figure 38-25 The preferred initial incision for draining a felon is one made directly into the area of most fluctuance. More aggressive incisions should be reserved for complicated cases, since they have a greater morbidity and require more complicated wound care. The unilateral longitudinal approach is a good first choice *(A)*. Fat pad incisions are acceptable but may be associated with a painful scar in an area that is often traumatized. The transverse fat pad incision should not injure digital nerves *(B),* and the longitudinal fat pad incision should avoid the flexor tendon *(C)*.

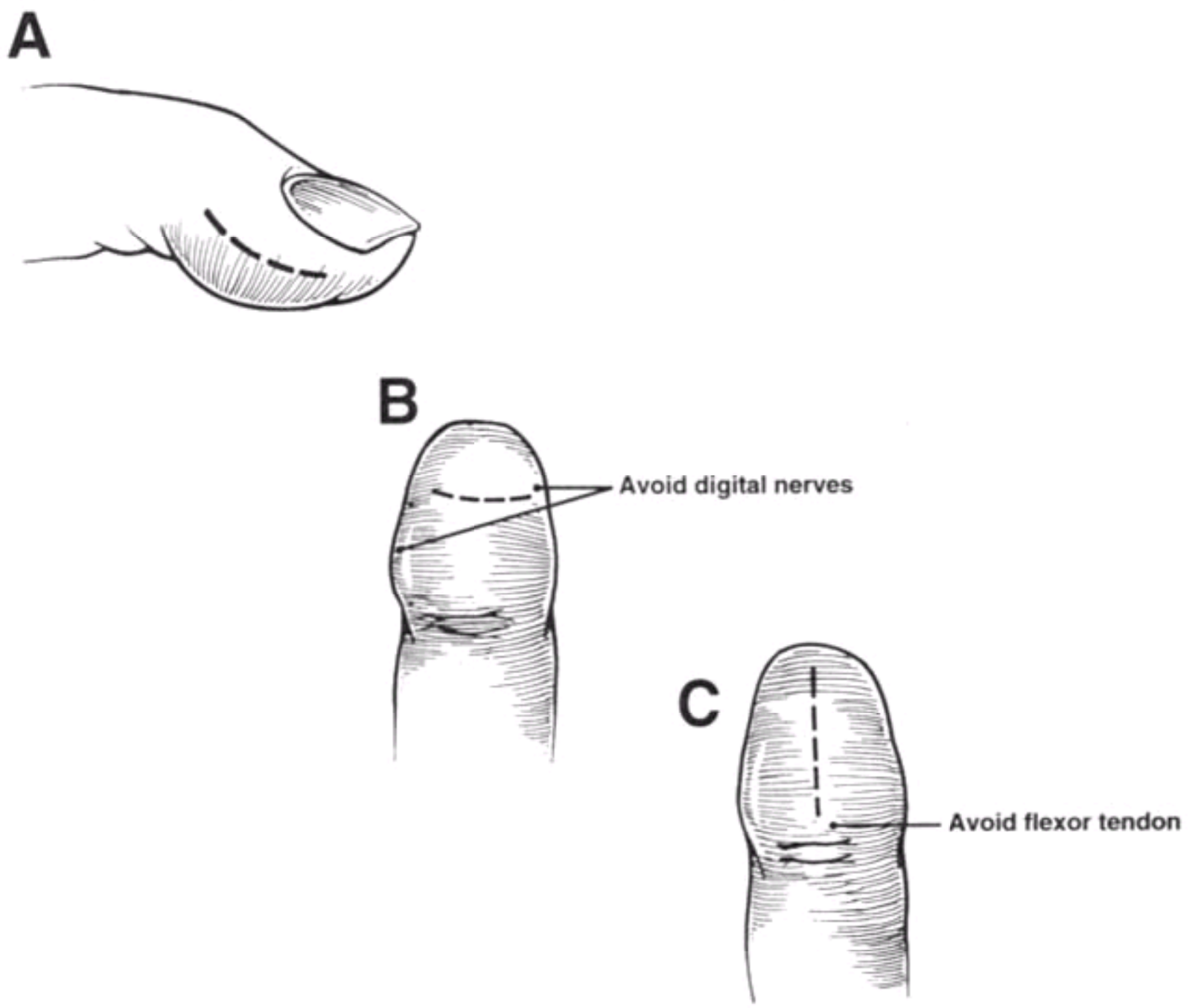


Figure 38-26 Hockey-stick incision for the drainage of a felon. *Note:* An incision on the ulnar side of the index, middle, and ring fingers is appropriate. The little finger is best incised on the radial side. The site of the incision on the thumb is also preferably on the radial side, but this may depend on the occupation of the patient. *(From Chase RA: Atlas of Hand Surgery. Philadelphia, WB Saunders, 1973. Reproduced with permission.)*

Figure 38-27 Through-and-through incision for a felon. A Penrose drain is placed for a few days to promote the withdrawal of fluid. This is an alternative to the hockey-stick incision.

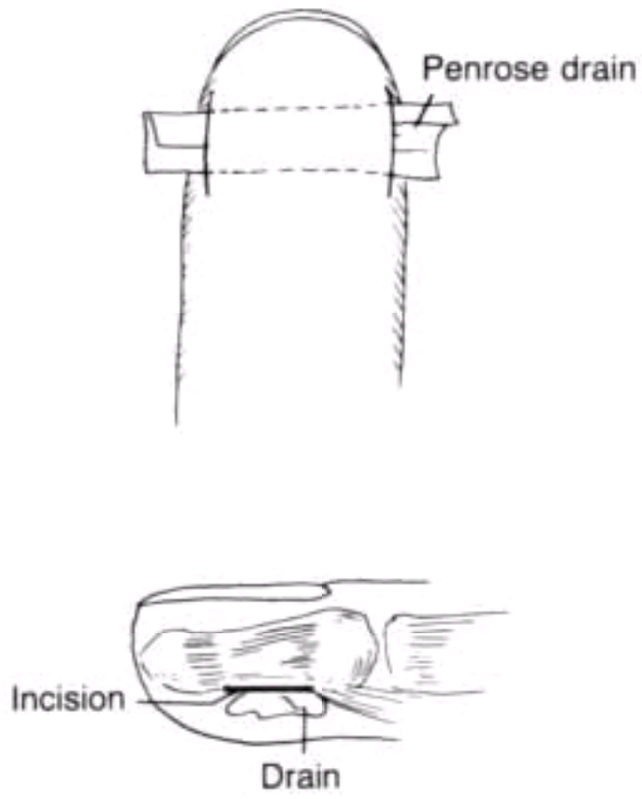


Figure 38-28 *A–C,* Fishmouth incision. This is a rather radical incision that is best reserved for resistant cases. Its advantage is that it allows for complete drainage and visualization of the infection in complicated cases. This incision takes longer to heal than others and may leave a large and sensitive scar. The fingertip may also be left unstable as a result of the lysis of all septa. The incision is seldom used on an outpatient basis for these reasons.

A

B

C

Figure 38-29 *A*, This patient slammed his finger in a car door, sustaining an acute flexion of the distal phalynx and a crush injury. It appears to be a simple subungual hematoma, but note the blood in the paronychial space. There is a communication between the nail bed laceration and the eponychium. *B*, All of the blood did not drain when the nail was trephinated. *C*, Blood accumulated in this area because the base of the fingernail has been avulsed, and lies between the eponychial fold and the skin of the cuticle. This is appreciated when the skin is debrided. The closed nature of the injury causes the confusion. With the nail removed, the nail bed laceration can be appreciated and repaired. *D*, The old trephined nail can now be replaced to its original position to keep open the eponychial space, or that space is packed with gauze for a few weeks to discourage scar formation and a subsequent nail deformity.

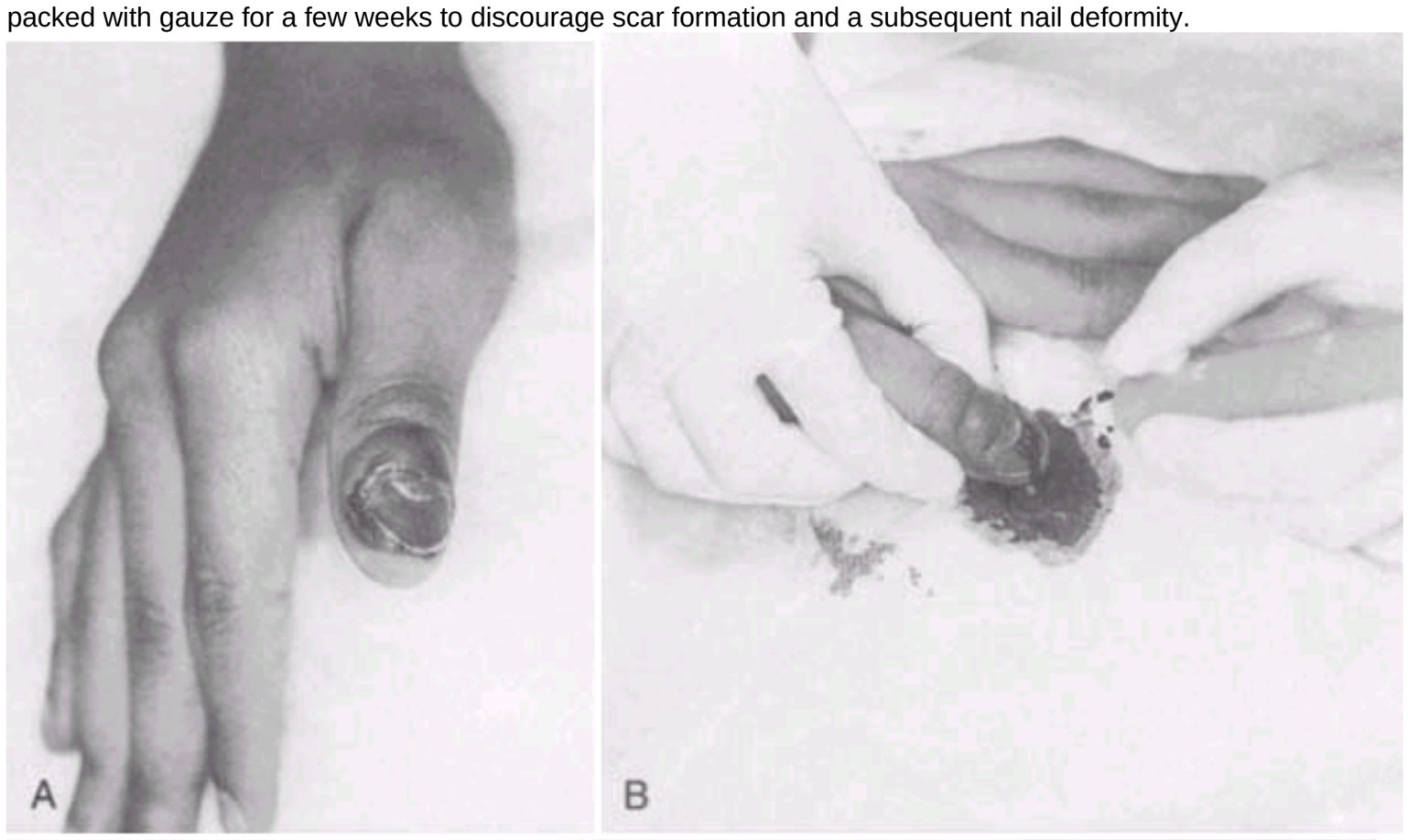


Figure 38-30 *A,* Subungual hematoma with a blackened nailbed. *B,* Following trephination, blood flows freely from the puncture site. An adequately sized drainage hole may be placed in the nail with a heated paper clip. Small holes, which tend to clog and inhibit drainage, should not be made. The hole should be large enough to allow continued drainage of blood. Note that in this example, the blood only accumulates under the nail, not in any paronychial areas.

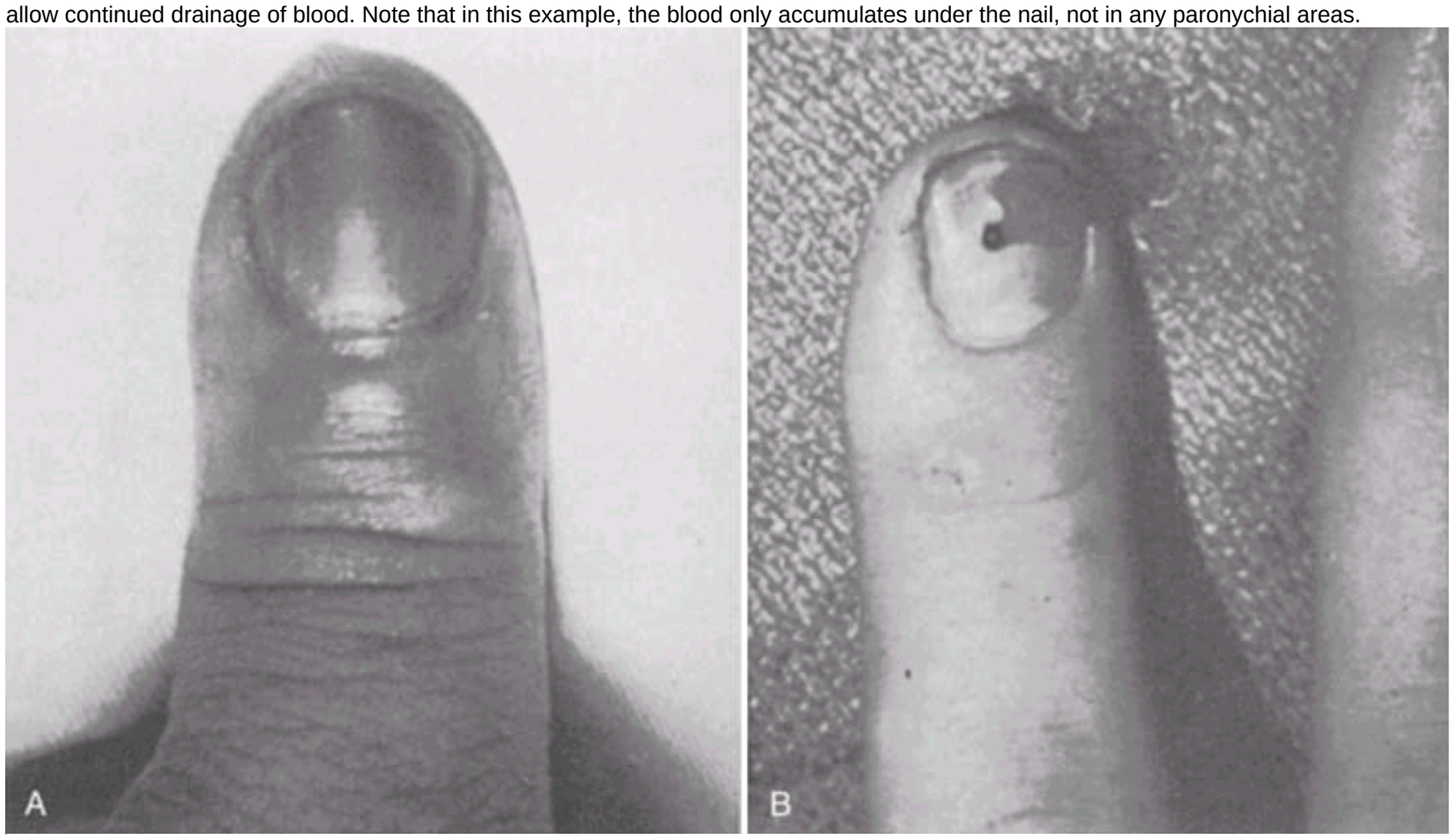


Figure 39-1 The "rule of nines" for estimating percentage of area burned. (As a rough guide, the area covered by the individual's palm is approximately 1.25% the total body surface area [TBSA].) The rule of nines is a rough estimate of the TBSA burned. Note that adults and children are different. This formula frequently *overestimates* the extent of a burn in clinical practice. See Figure 39-2 for a more accurate method of determining the TBSA burned for children.

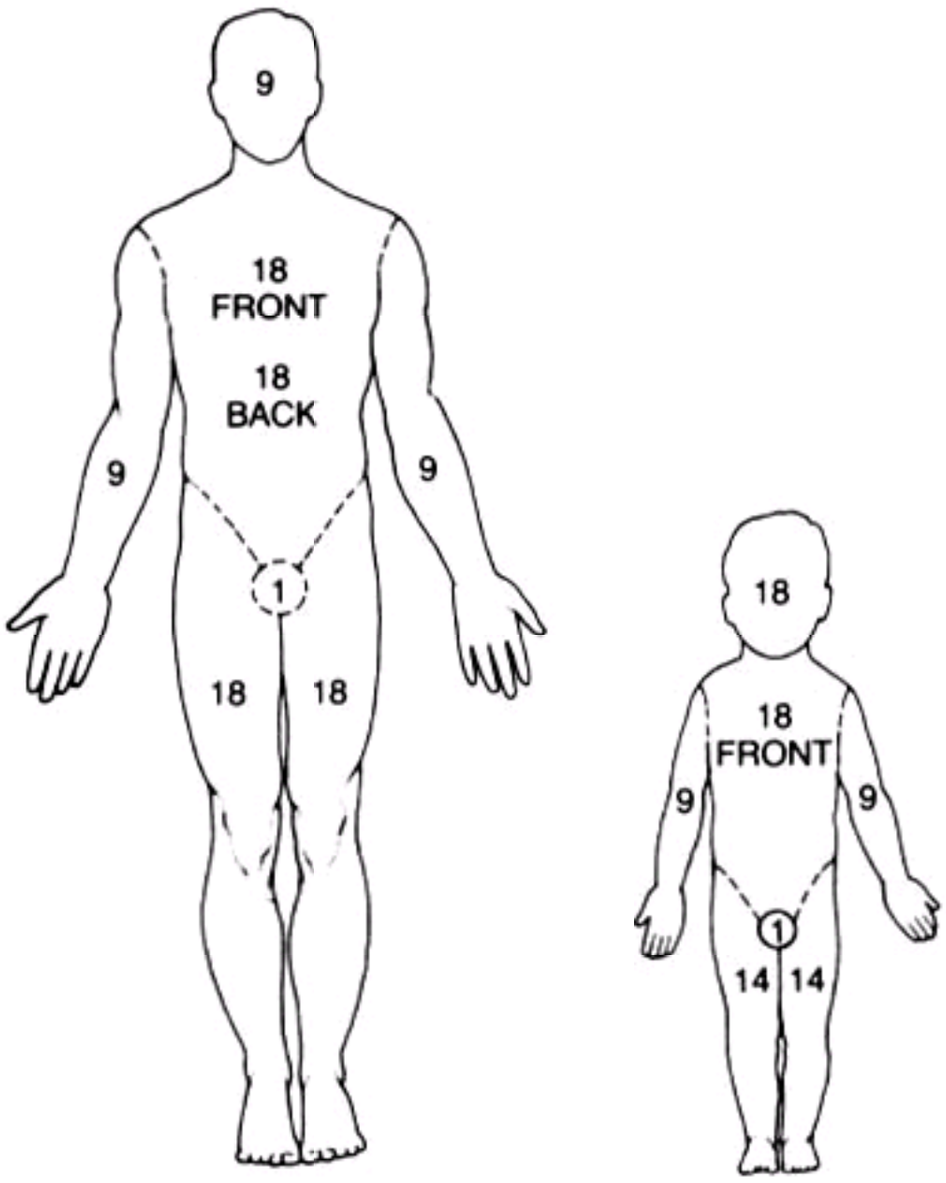
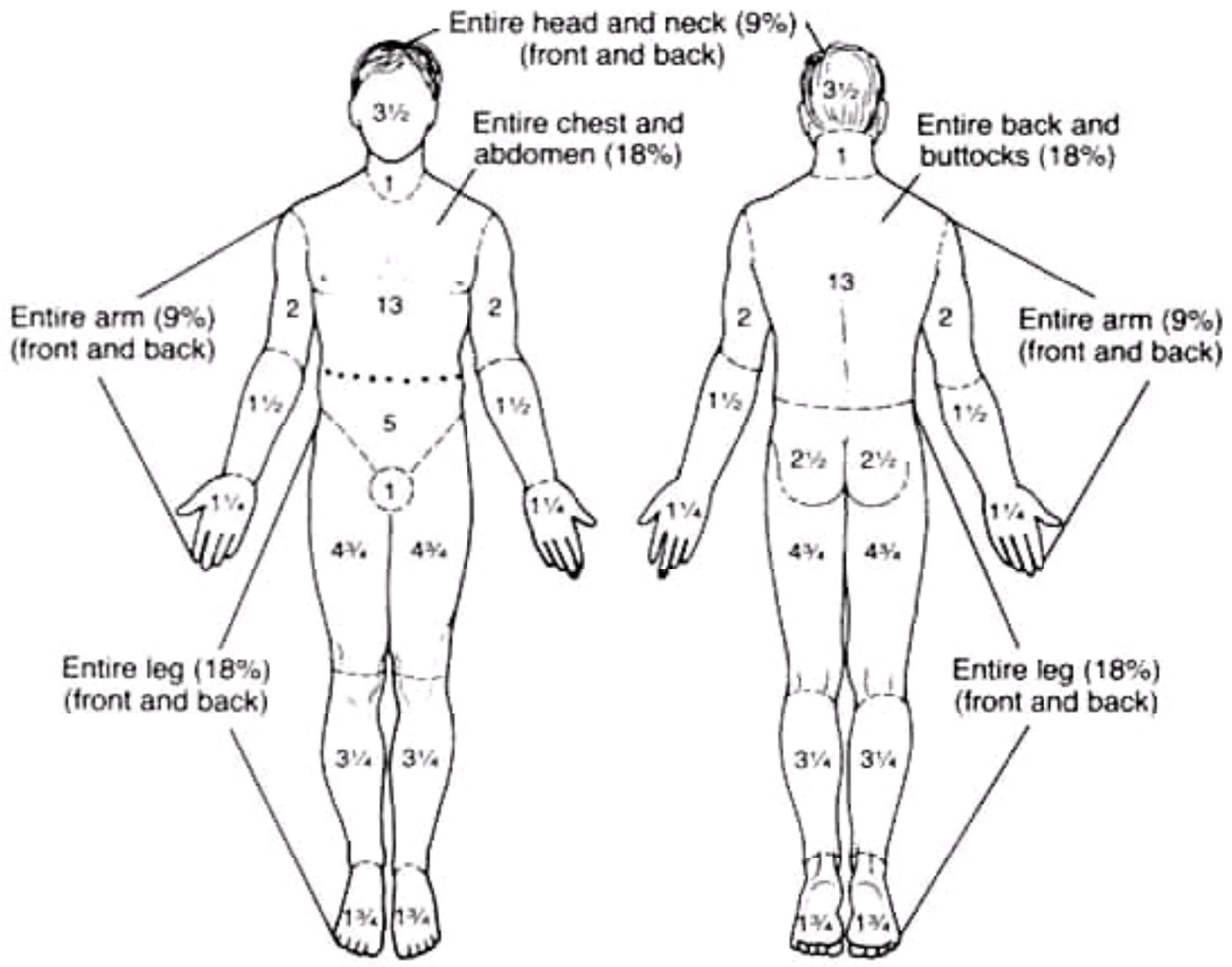
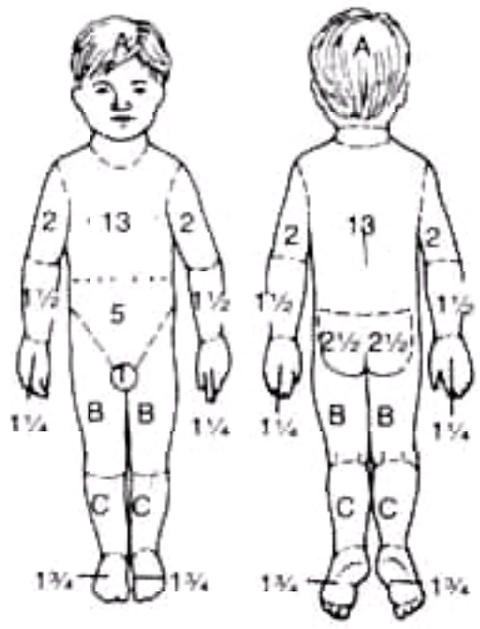


Figure 39-2 *A,* The Lund and Browder charts are somewhat more accurate than the rule of nines in estimating the TBSA burned. *B,* The proportion of TBSA of individual areas, according to age. Compared with adults, children have larger heads and smaller legs. Other areas are relatively equivalent throughout life. The rule of nines is not accurate in determining the percentage of TBSA burned in children.





A

AGE	Birth–1 Yr	1–4 yr	5–9 yr	10–14 yr	15 yr	Adult
Head	19	17	13	11	9	7
Neck	2	.....				
Ant trunk	13	.....				
Post trunk	13	.....				
R buttock	2½	.....				
L buttock	2½	.....				
Genitalia	1	.....				
R U arm	4	.....				
L U arm	4	.....				
R L arm	3	.....				
L L arm	3	.....				
R hand	2½	.....				
L hand	2½	.....				
R thigh	5½	6½	8	8½	9	9½
L thigh	5½	6½	8	8½	9	9½
R leg	5	5	5½	6	6½	7
L leg	5	5	5½	6	6½	7
R foot	3½	.....				
L foot	3½	.....				

B BODY AREA

Figure 39-3 It may be difficult to accurately assess the depth or severity of a burn on the first visit. *A*, This is a full-thickness burn that will not heal without a skin graft. *B*, This blistered hot water burn is likely second degree, but full-thickness burns can develop under blisters.

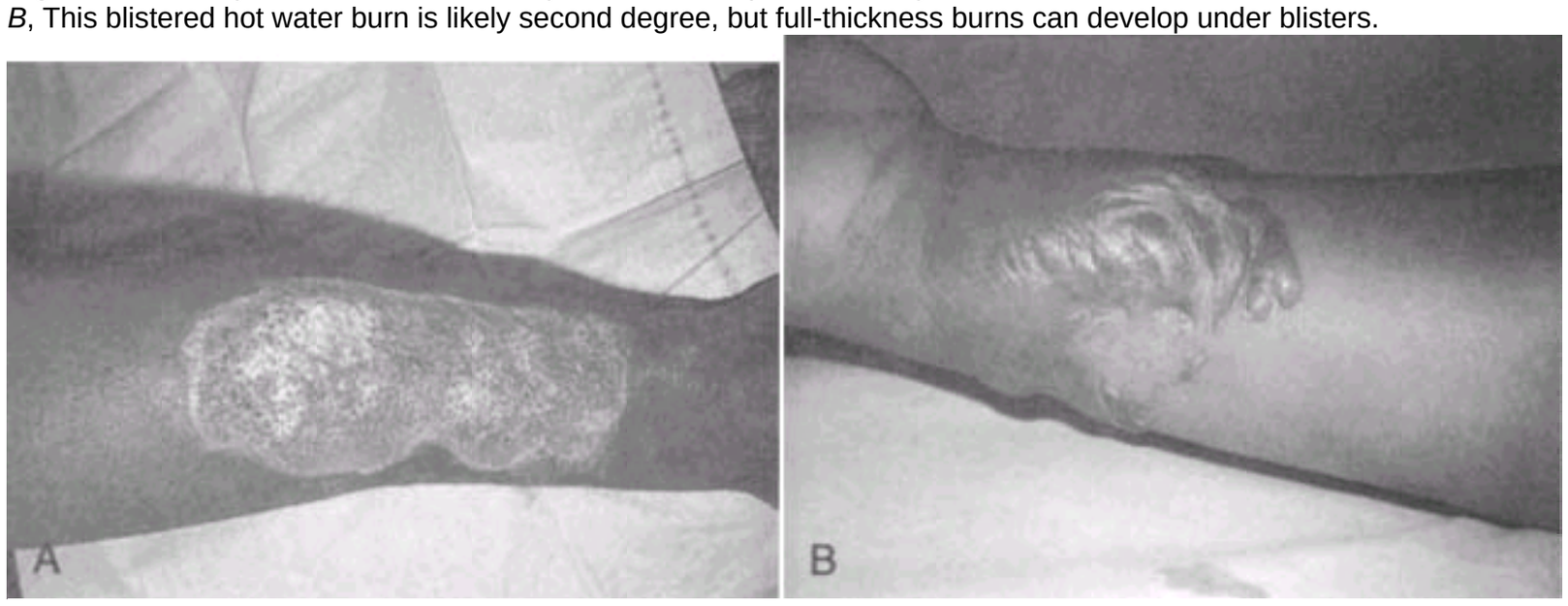


Figure 39-4 To cool a burn that cannot be easily immersed in water, cover the area with unfolded gauze pads that have been soaked in room-temperature saline. Continue to frequently soak the gauze with cool saline or tap water drawn up in a syringe. Adding a few ice chips to the liquid is helpful, but do not cover the burn with ice. Towels are generally too bulky for this procedure.

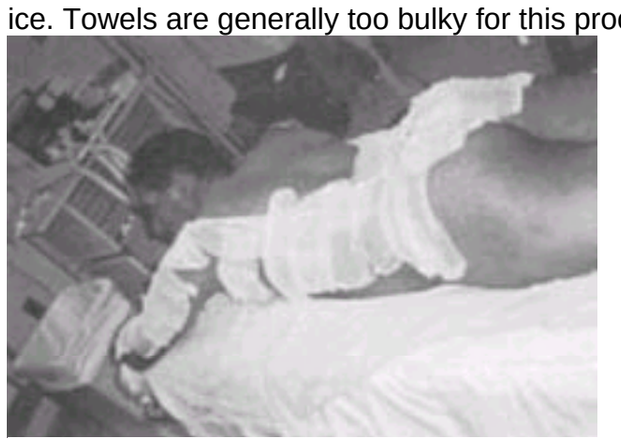
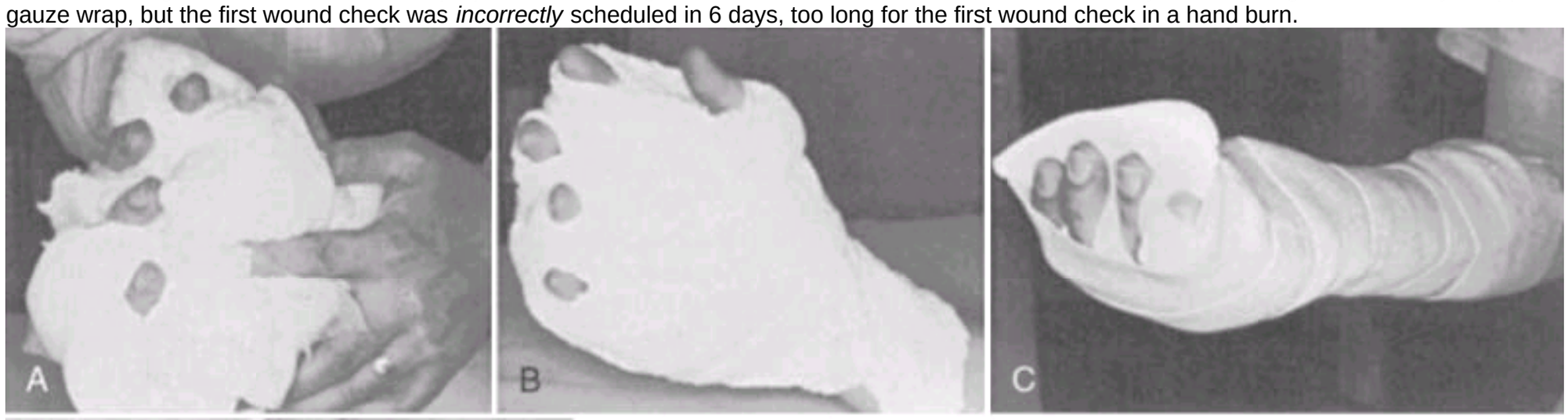


Figure 39-5 Outpatient burn dressing of the hand. Persons with serious hand burns should be admitted to the hospital, but persons with minor burns can be treated in the outpatient setting. Following application of an antibiotic ointment or a dry, nonadherent dressing, the fingers are separated with fluffs in the web spaces *(A),* and the entire hand is enclosed in a position of function *(B)* (here with the help of a roll of Kerlix). If the wrist is involved, a removable plaster splint may be applied over the dressing *(C)*. *D,* The result of a minor burn to the hand when the fingers were not wrapped individually. Initially there were only a few blisters, but this patient now has second-degree skin loss due to an improper burn dressing that caused maceration of normal skin. Not only were the fingers *incorrectly* wrapped together in one gauze wrap, but the first wound check was *incorrectly* scheduled in 6 days, too long for the first wound check in a hand burn.



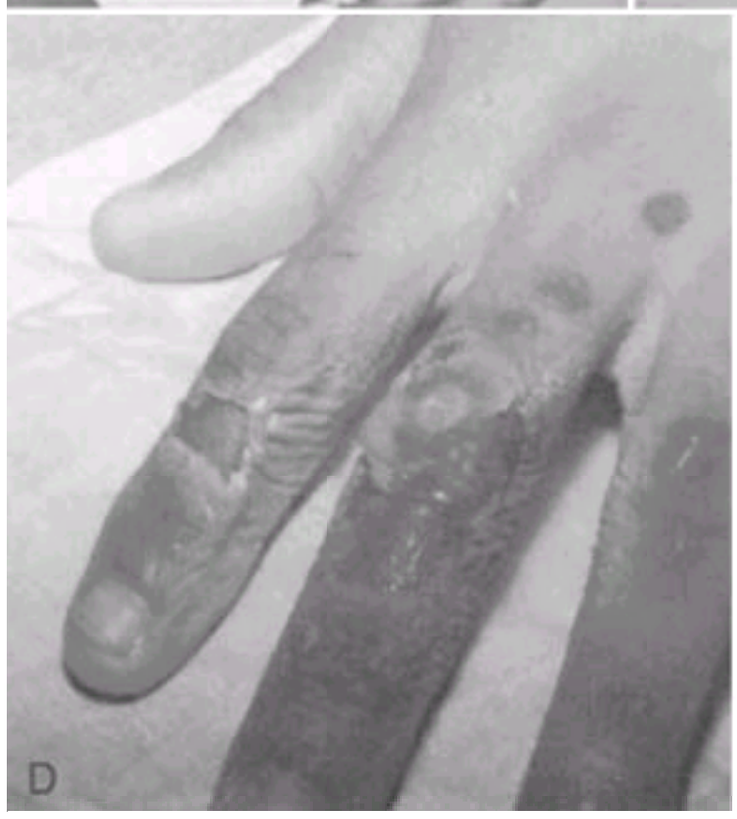


Figure 39-6 It is difficult to do anything wrong with minor burn blisters, and many regimens are acceptable. Eventually, however, blisters will have to be debrided. An expeditious and relatively painless way to debride a burn is to use a dry gauze pad to grasp the dead skin *(A)* and peel it off *(B)*. Meticulous instrument debridement is often time consuming and stressful to the patient. Be aware that pain occurs when air comes in contact with the debrided skin and prophylactic analgesia should be provided.

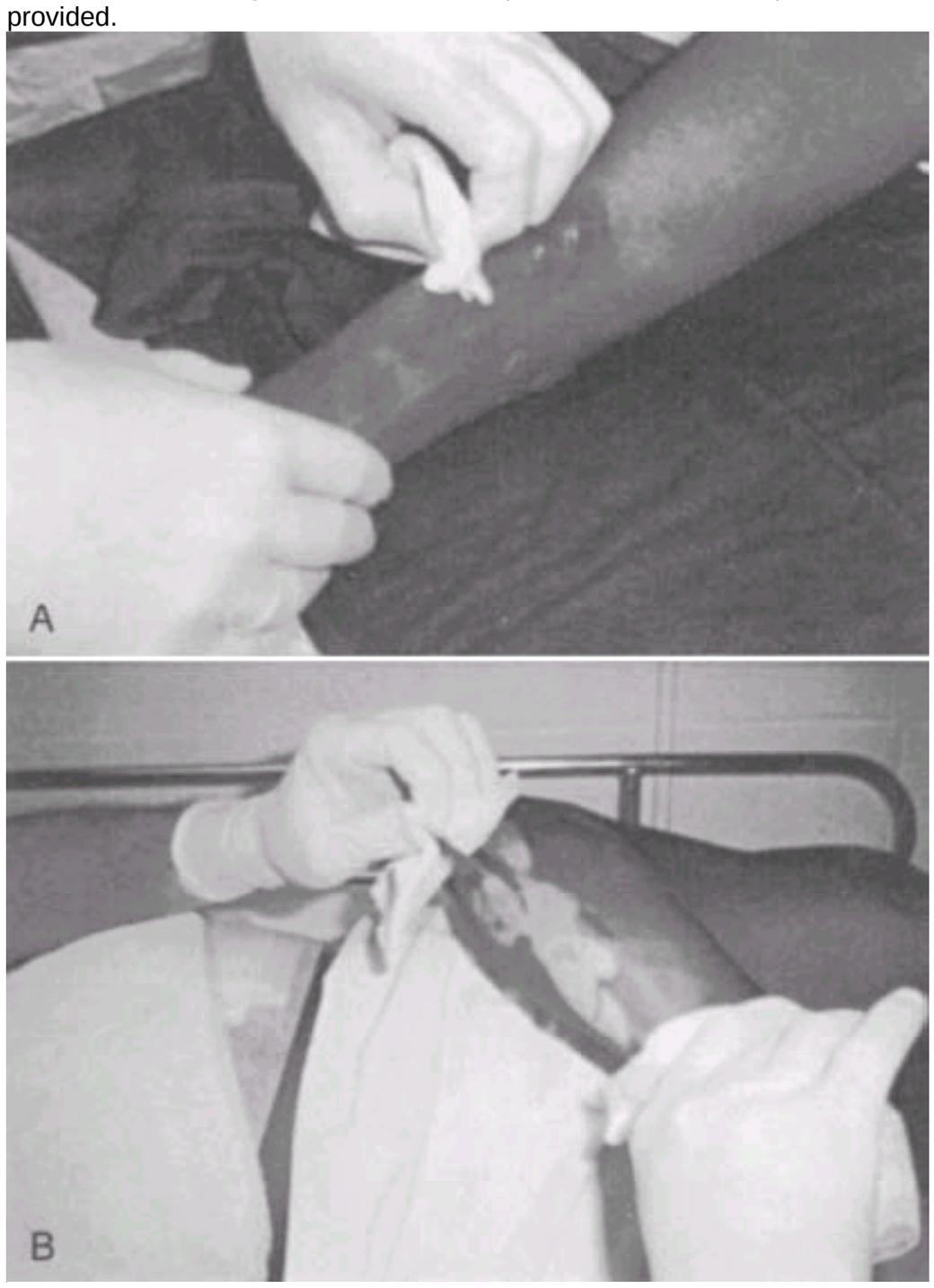


Figure 39-7 Elevation of a burned hand should begin in the ED. After a properly applied hand dressing is applied, the arm is suspended from an IV pole with stockinette.

Figure 39-8 *A*, The most popular topical burn preparation is Silvadene cream. While commonly used on minor burns, it likely has little beneficial effect on healing, and minor burns rarely become infected. Nonetheless, Silvadene is a standard intervention that at least causes the patient to look at the burn and become involved in dressing changes. *B*, Many burn specialists suggest inexpensive topical antibiotic ointments (such as bacitracin and neosporin) for all outpatient burns. They are commonly used on face and neck burns. Bacitracin is preferred since a contact dermatitis, such as noted in this abrasion, can occur from the neomycin portion of some topicals.

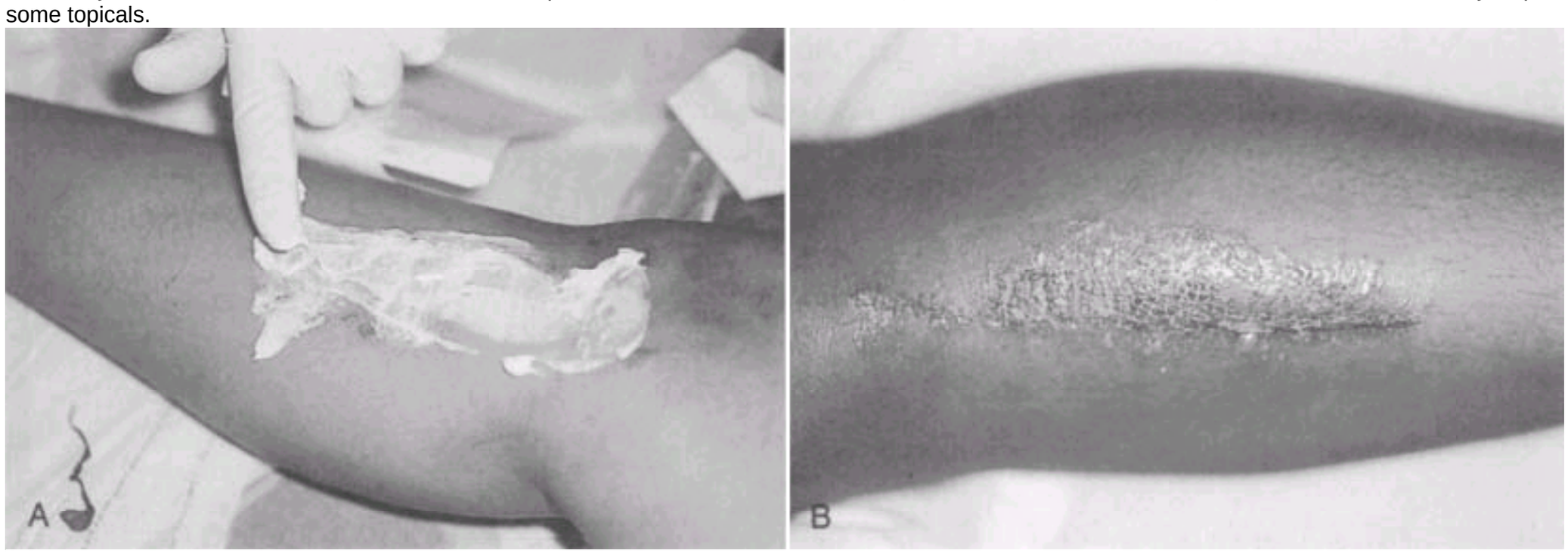


Figure 39-9 Burns of the feet are specialized burns that require a careful evaluation and an individualized treatment plan, even if the burn surface area is relatively small. It is difficult for most patients to provide ideal burn care at home when the feet are involved. *A*, It is tempting to initially treat this seemingly minor superficial second-degree foot burn in an outpatient setting, but the patient's compliance and social situation must be ideal for a successful outcome. Hospitalization until home health care can be established is prudent. *B*, An example of a foot burn that is a potential disaster, in this case due to a late presentation in a diabetic.

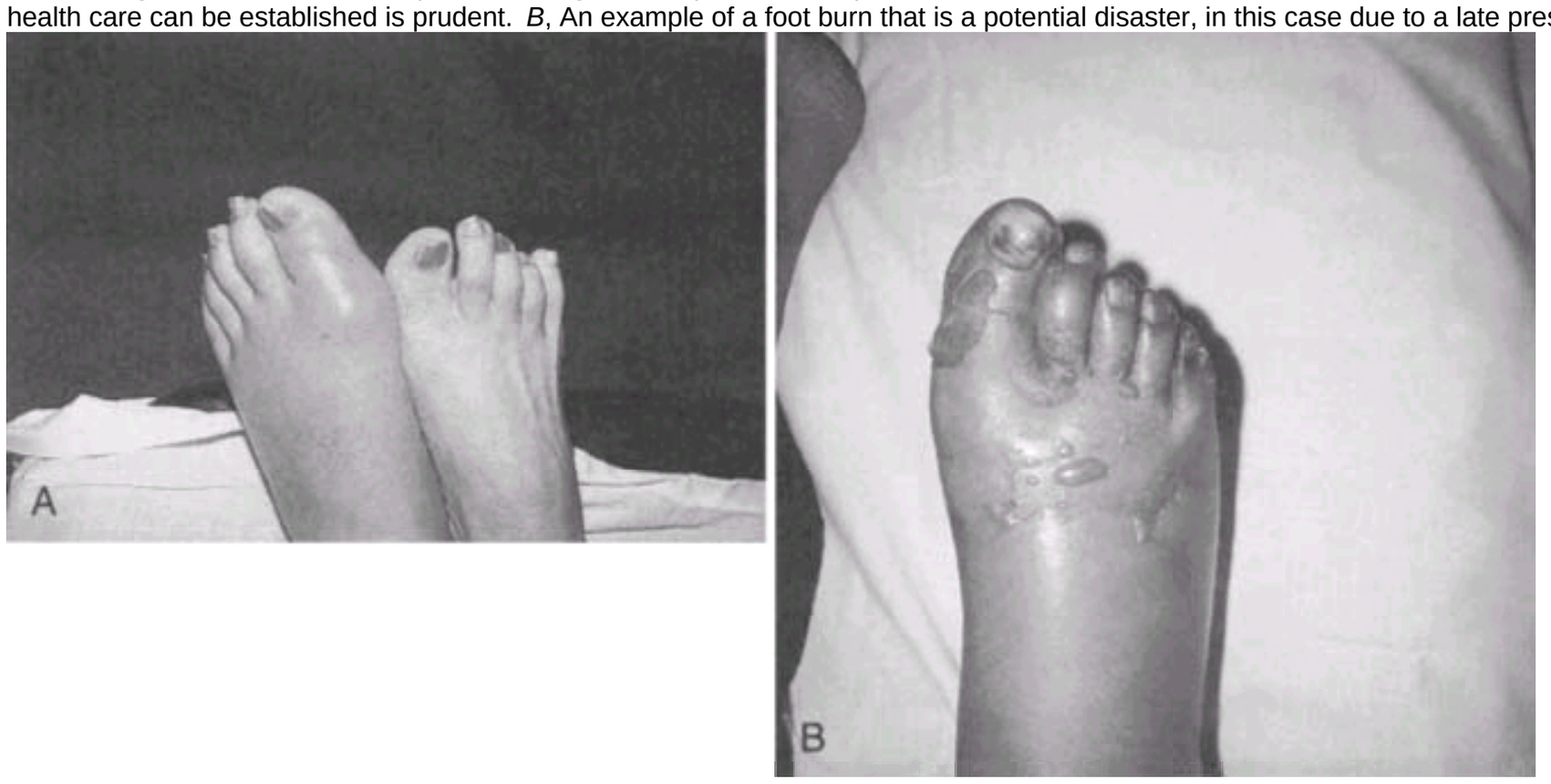


Figure 39-10 This badly burned hand requires referral to a burn center and should not be handled as an outpatient.

Figure 39-11 *A*, Flash burns to the face from lighting a gas stove. These burns are painful and may cause edema, but usually they do well. Note the singed facial hair. The eyes are usually protected by rapid reflex blinking and carbon monoxide poisoning and pulmonary burns are not an issue. Most can be handled in the outpatient setting with bacitracin ointment and no dressing. Pain control may be problematic unless opioids are prescribed. *B*, Facial and neck burns when a radiator cap was removed and the victim was sprayed with steam and hot antifreeze.

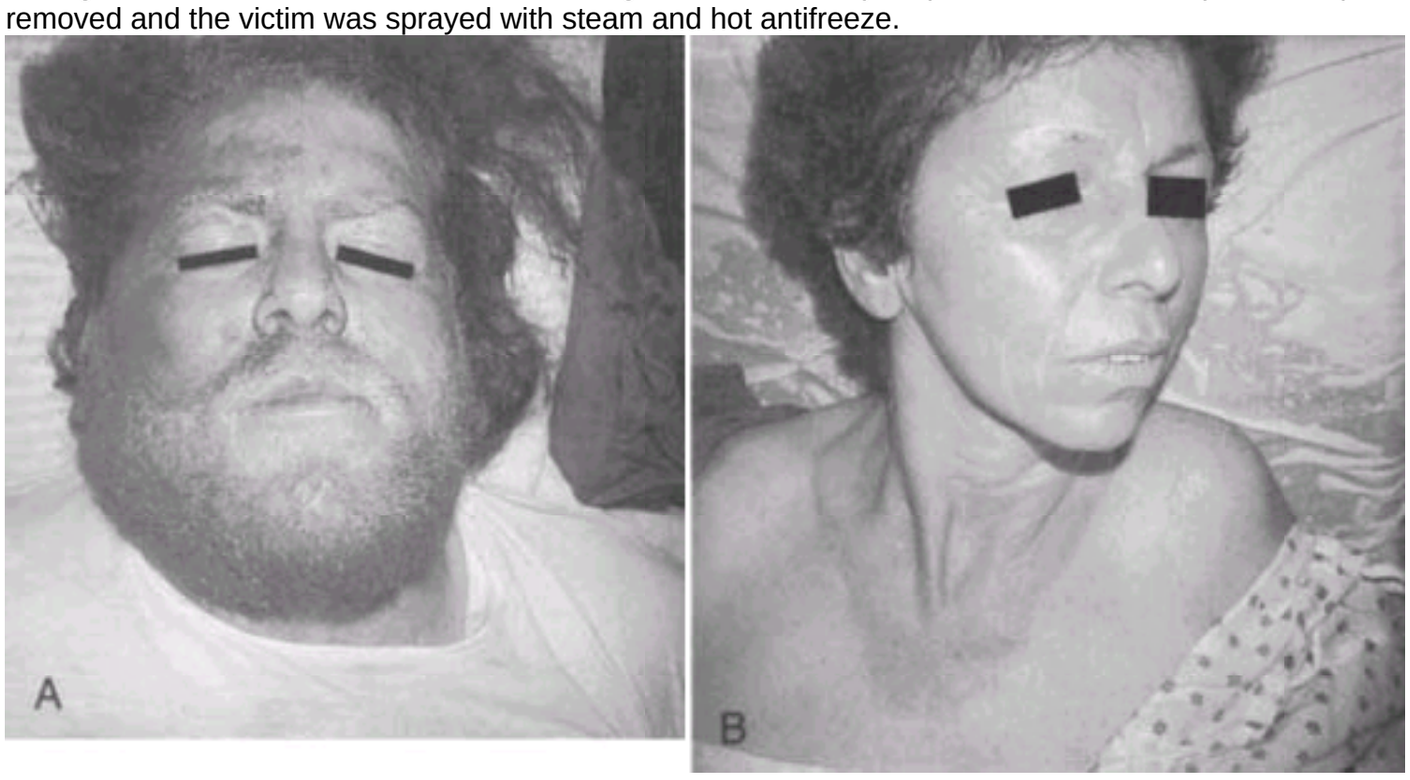
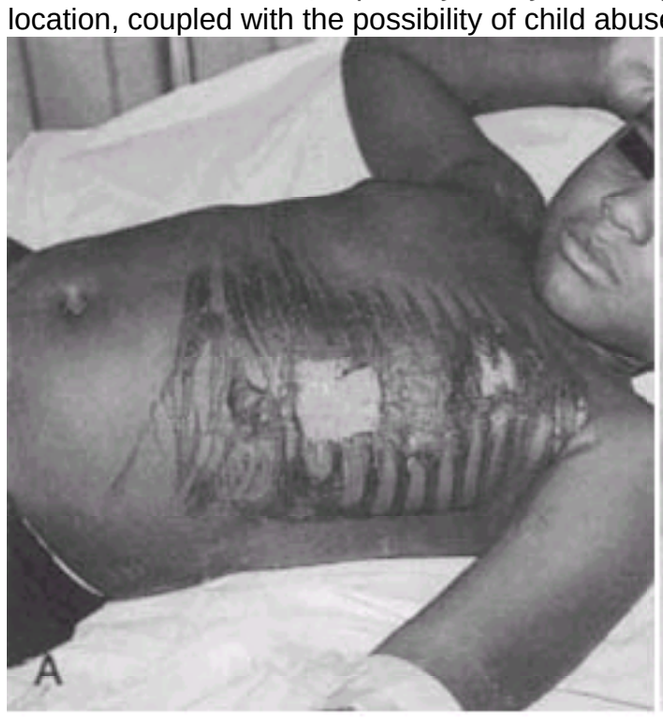
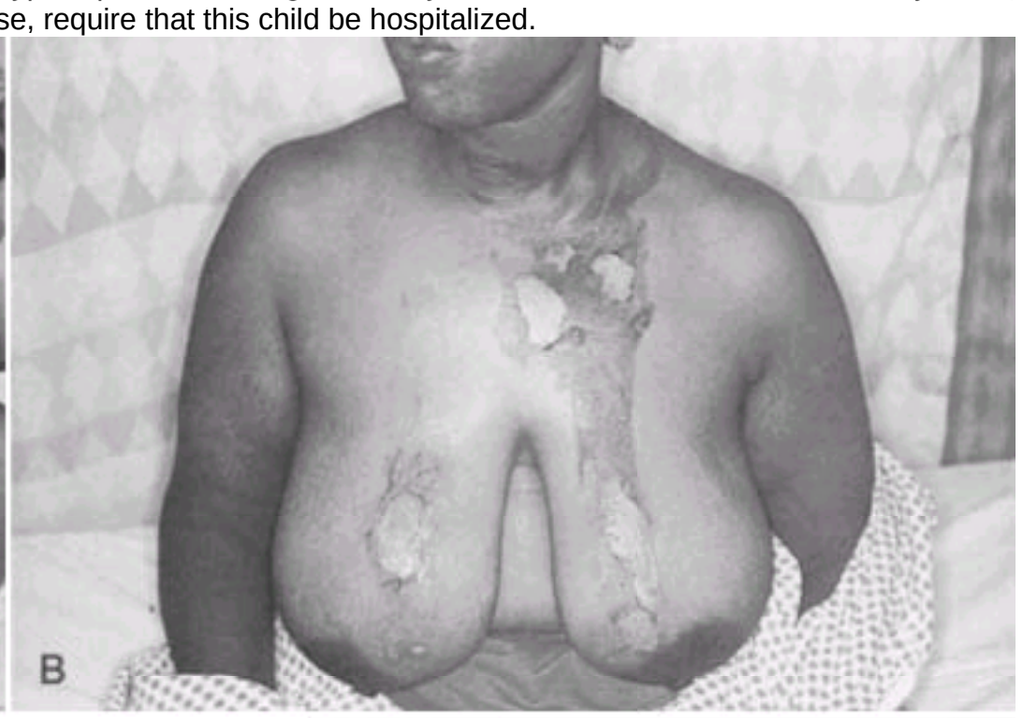


Figure 39-12 Burns can be a manifestation of child abuse, spouse abuse, or abuse of the elderly. *A*, Abuse burns from contact with a hot metal grate, from a child allegedly falling. *B*, This burn was the result of spouse abuse, caused by throwing hot soup during an argument. The delayed presentation to the hospital was a clue. *C*, Burns of the face and neck are common when a toddler pulls hot liquid from a stove. This case was never proven to be child abuse, but burns in young children often are due to abuse, especially if they are in atypical places. Although the body surface area of this burn is relatively small, the patient's age and the burn's location, coupled with the possibility of child abuse, require that this child be hospitalized.





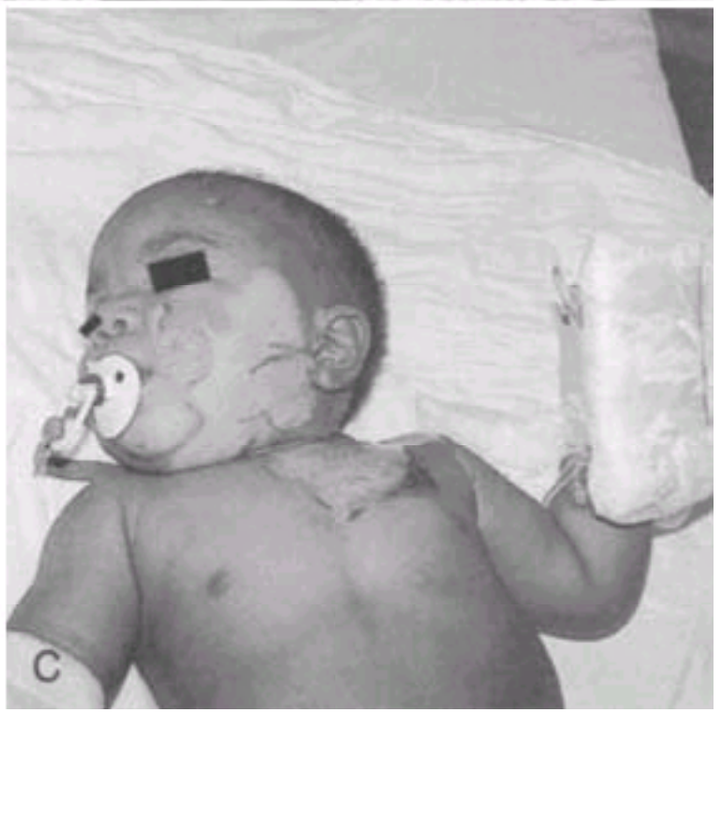


Figure 39-13 There is no compelling reason to remove all tar on the first visit. Physical removal of cooled tar usually results in avulsion of the underlying skin. Skin that is obviously loose should be debrided, but adherent tar is best liquefied with an emulsifying agent. Neomycin cream, not ointment, is a suggested emulsifier, but others are acceptable (see text). Final removal may be delayed for several days to permit loosening of the tar. Frequent dressing changes using an emulsifying agent can be performed by the patient, removing the tar over a few days.

Figure 39-14 Tar stuck to the face *(A)* can be emulsified with various agents and a lot of patience and persistence *(B)*. Fortunately, tar burns are usually not full-thickness burns.

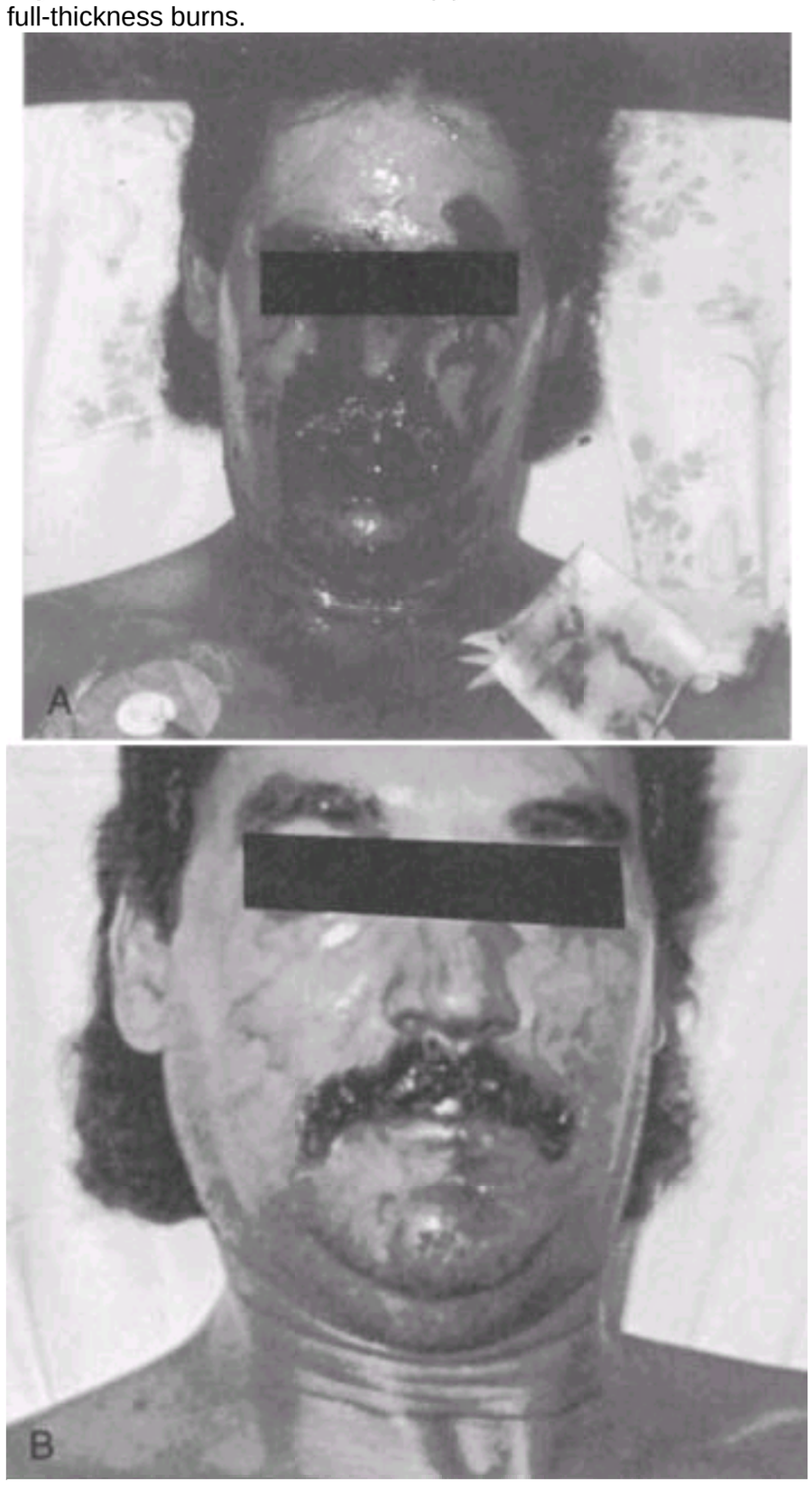


Figure 39-15 There is no need to remove all the tar on the first visit *(A)*. This extremity was covered with an emulsifying agent and with gauze, and the residual tar was washed off easily 36 hours later *(B)*.

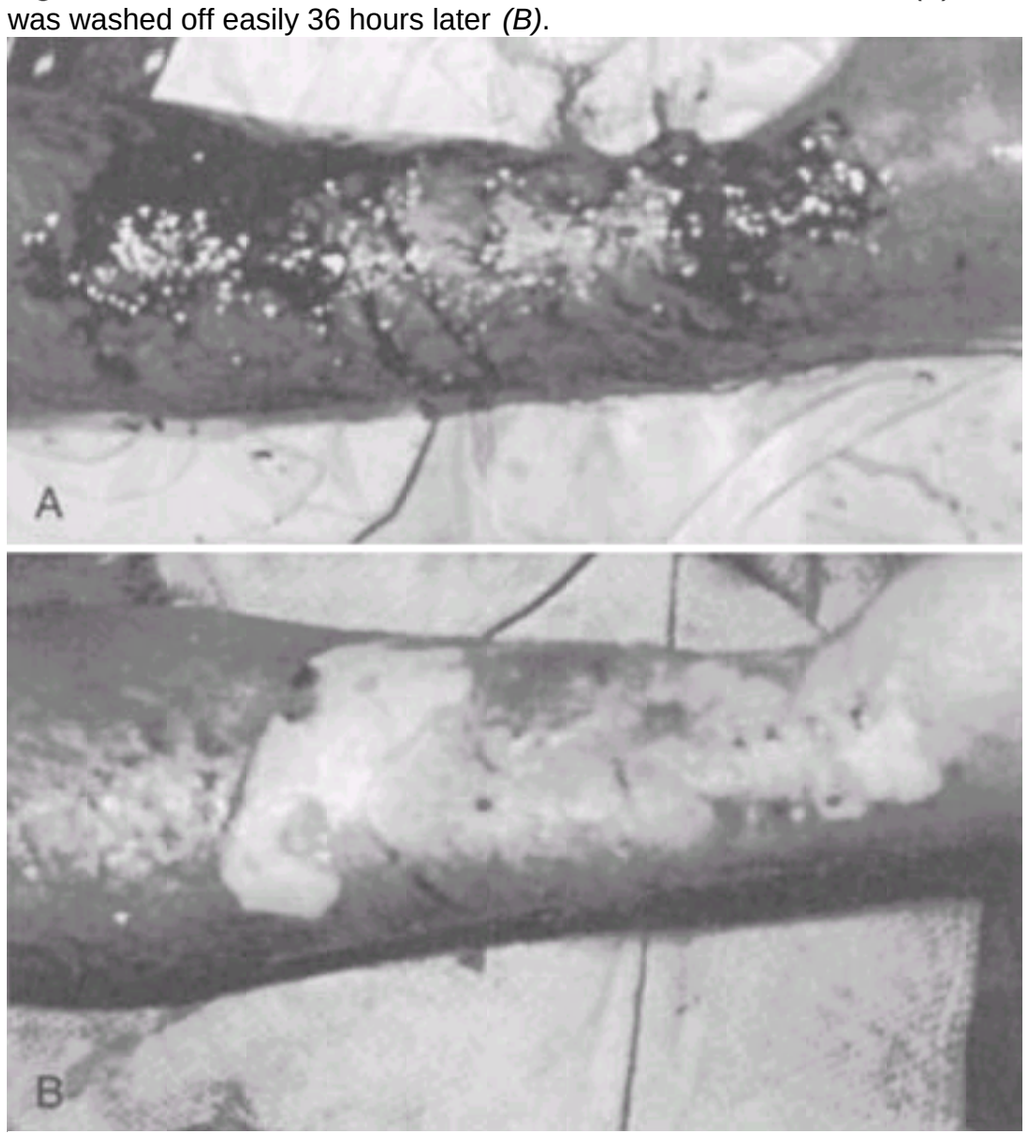


Figure 39-16 Alkali burns from wet cement develop insidiously, are extremely painful, and are frequently full-thickness injuries. They are most common on the feet when cement leaks over the top of the boots *(A)* or from kneeling in wet cement while working *(B)*. The alkali can penetrate clothing.

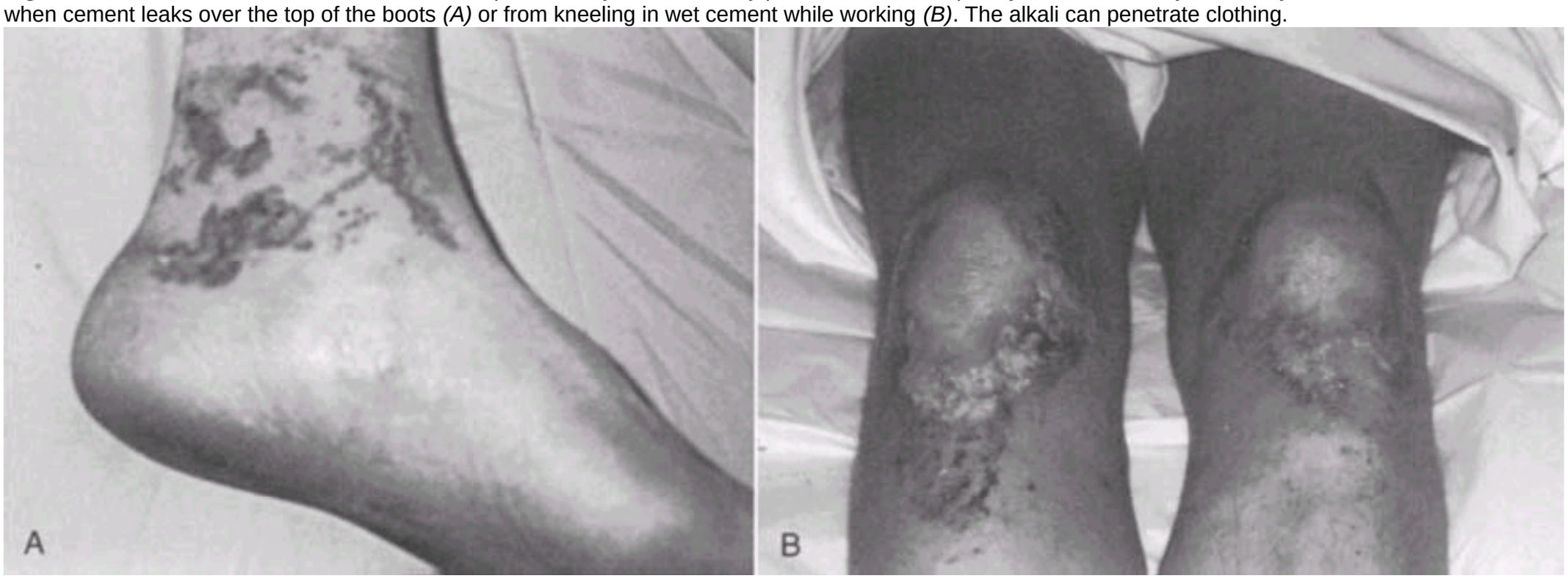


Figure 39-17 *A*, Initially this very painful hydrofluoric acid burn of the thenar and hypothenar emminence appeared minimal. *B*, Despite infiltration with calcium gluconate, a deep burn developed 3 days later.

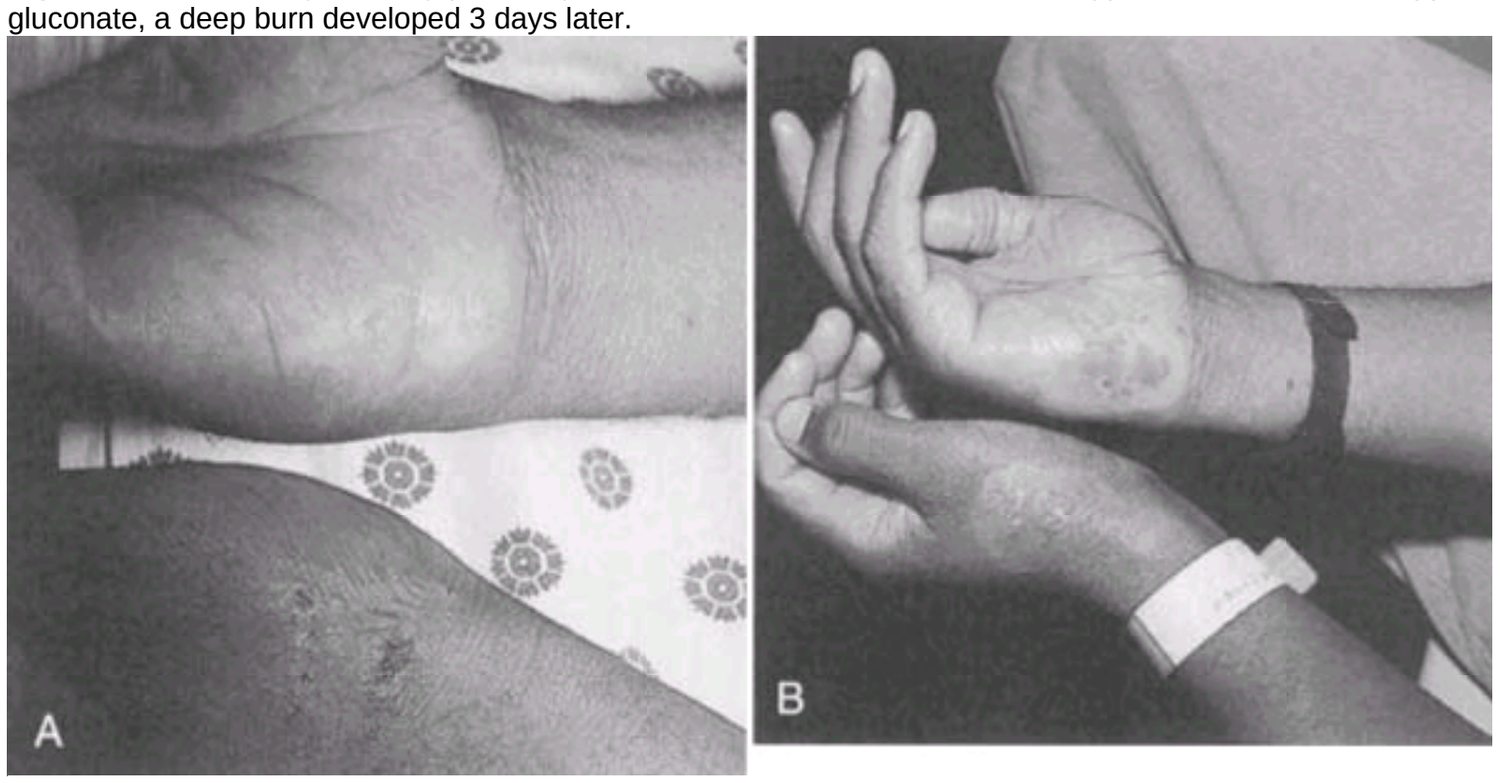


Figure 39-18 Hydrofluoric acid burns to the fingertips are extremely painful, despite minimal clinical findings. In this case the pain was excruciating, but the patient manifested only hyperemia and minor ecchymosis. *A,* Note that the acid has penetrated the intact fingernail, producing a significant injury to the nailbed. *B,* Prior to performing digital block anesthesia in order to painlessly infiltrate the fingertips with calcium gluconate, the patient outlines the painful areas with a felt-tip marker to ensure accurate placement of the antidote. *C,* In the treatment of hydrofluoric acid burns, topical therapy is often ineffective. Calcium gluconate may be injected subcutaneously with a 25- to 27-ga needle into the nailbed via the fat pad under a digital nerve block. *Fingernails should not be removed routinely* if burns are mild, such as those seen with household products containing <10% concentration of the acid. Intra-arterial calcium infusions are often quite successful in relieving pain and limiting necrosis.

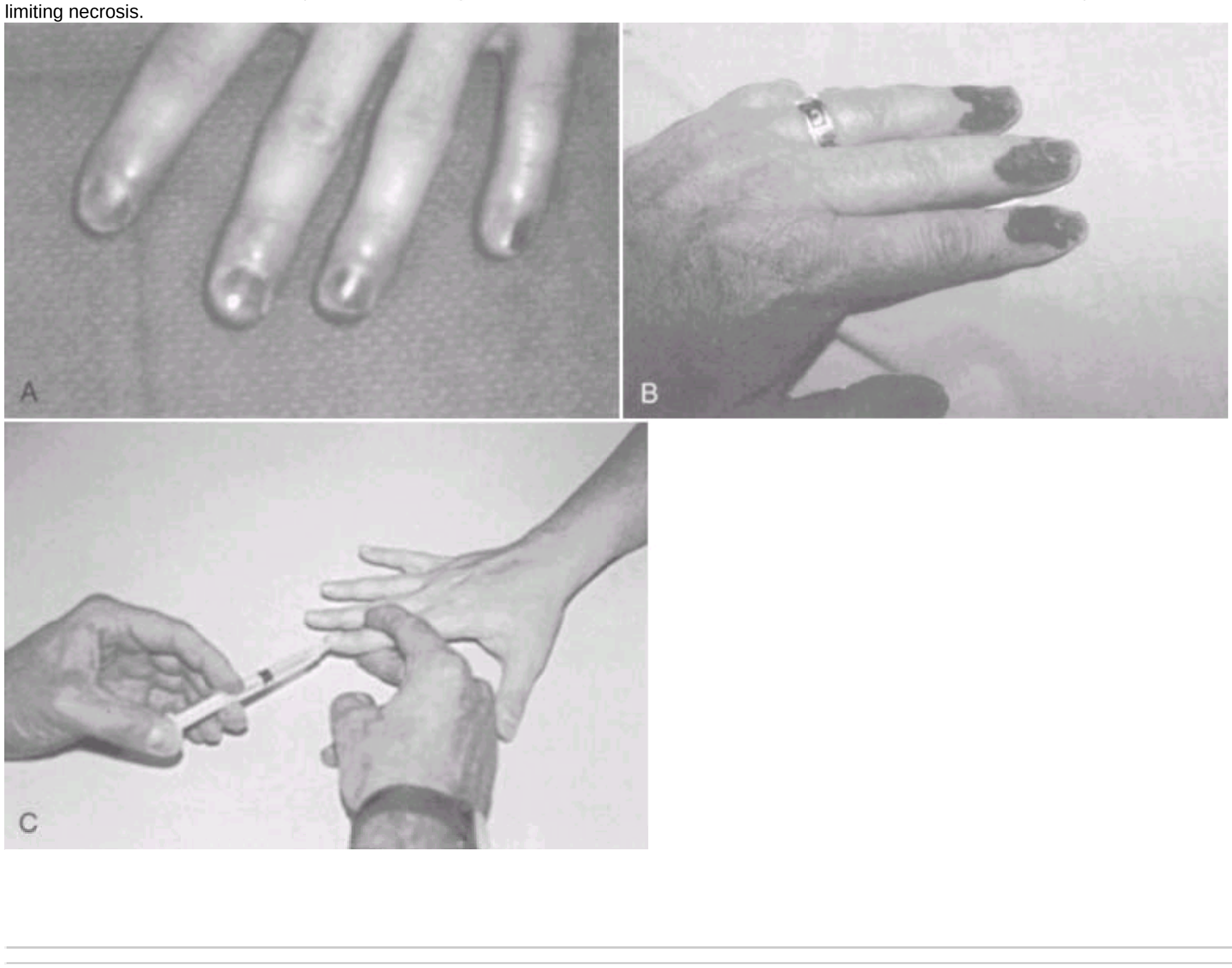


Figure 39-19 The circulatory embarrassment caused by edema beneath the encircling full-thickness burns of the legs of this patient was relieved by escharotomy incisions placed in the midmedial line of each limb. The restriction of the ventilatory excursion of the chest wall caused by the encircling full-thickness truncal burns was relieved by escharotomies placed in both anterior axillary lines and by a costal margin escharotomy. Compression of the abdominal contents and restriction of diaphragmatic excursion by the constricting deep abdominal wall burns were relieved by placement of escharotomy incisions in the lateral abdominal wall bilaterally. *(From Davis JH, Drucker WR, Foster RS, et al: Clinical Surgery. St. Louis, CV Mosby, 1987.)*

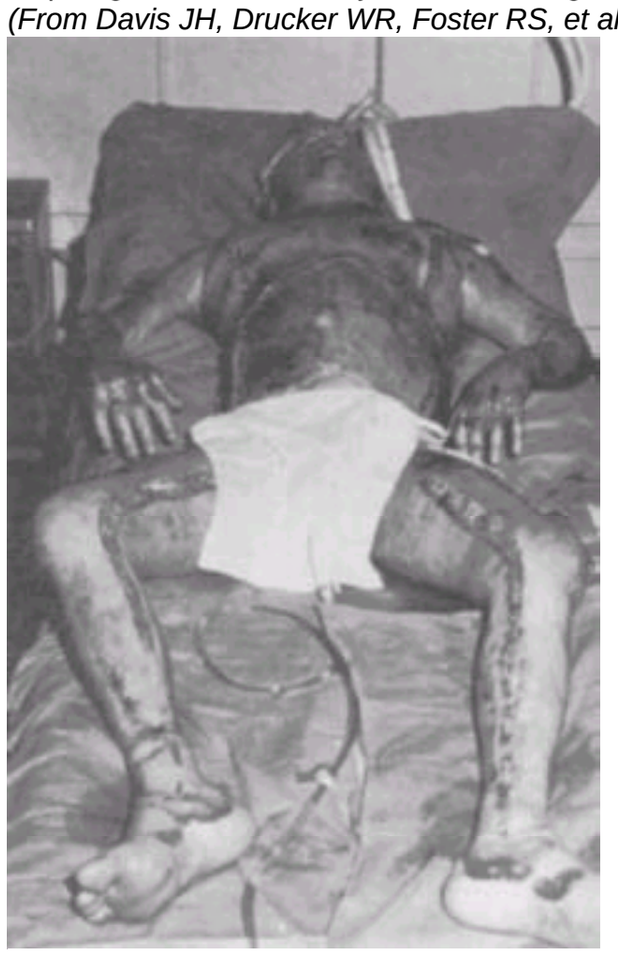


Figure 39-20 Diagram showing preferred sites for escharotomy incisions. *Dotted lines* indicate the escharotomy sites. *Bold lines* indicate areas where caution is required since vascular structures and nerves may be damaged by escharotomy incisions. *(From Davis JH, Drucker WR, Foster RS, et al: Clinical Surgery. St. Louis, CV Mosby, 1987.)*

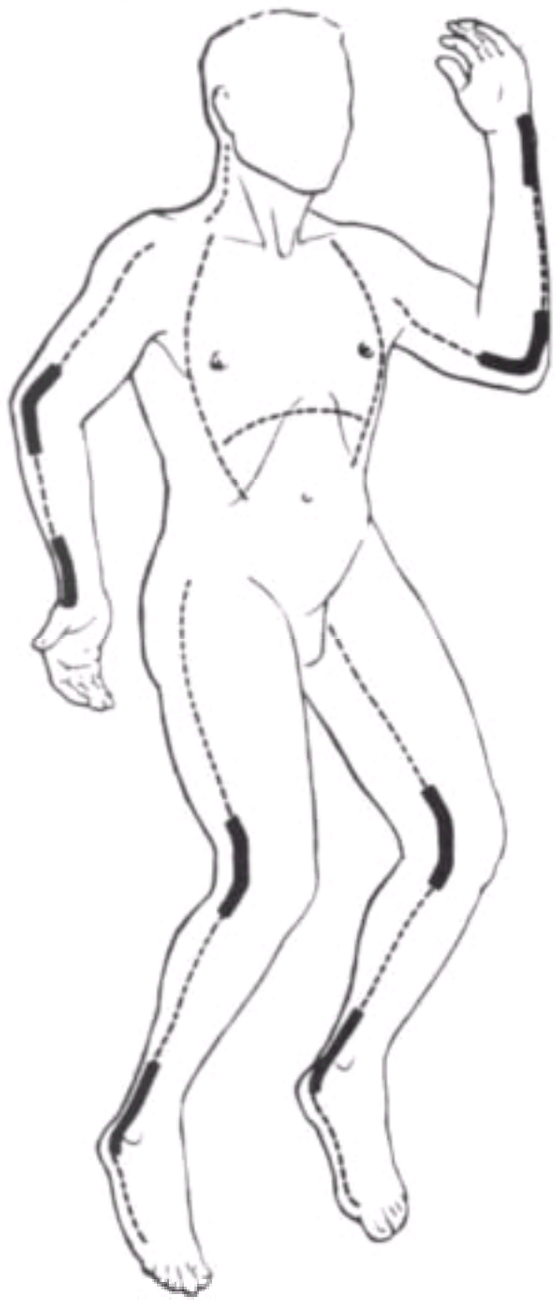


Figure 40-1 Blunt esophageal foreign bodies are most commonly lodged at one of three anatomical areas of narrowing: the cricopharyngeus muscle; the level of the aortic crossover; and the lower esophageal sphincter.

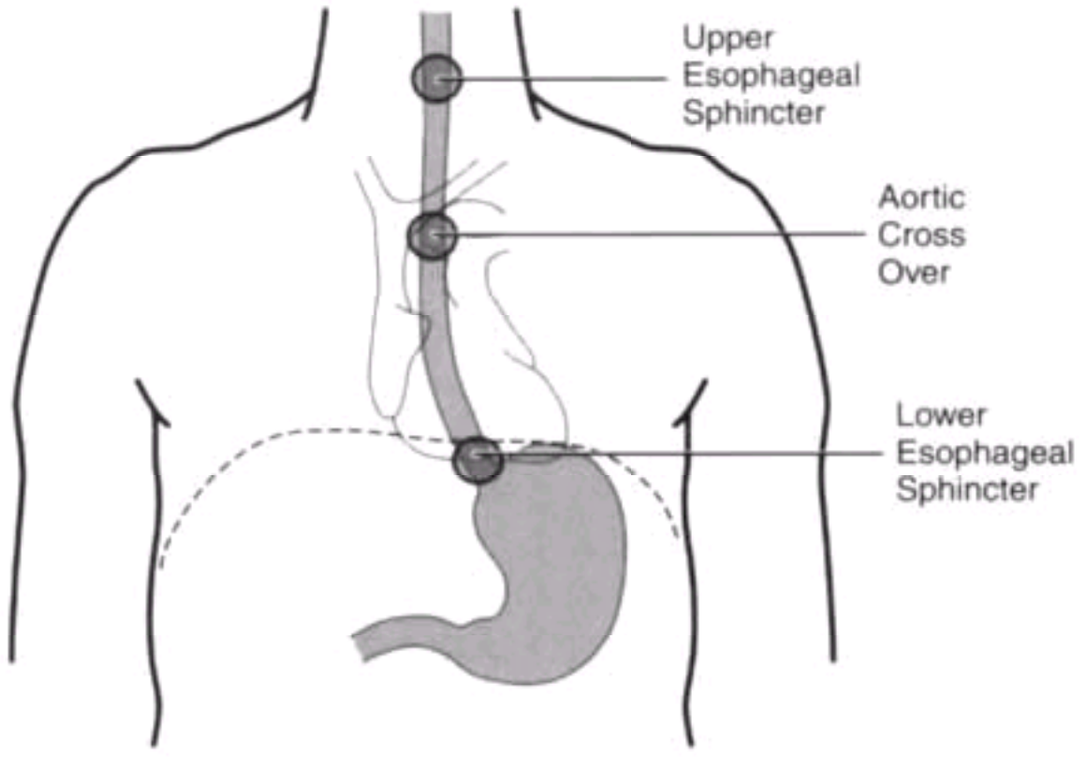


Figure 40-2 PA radiograph of an esophageal foreign body (coin) lodged at the level of the cricopharyngeus muscle. This is the most common area of the esophagus to harbor a coin in children.

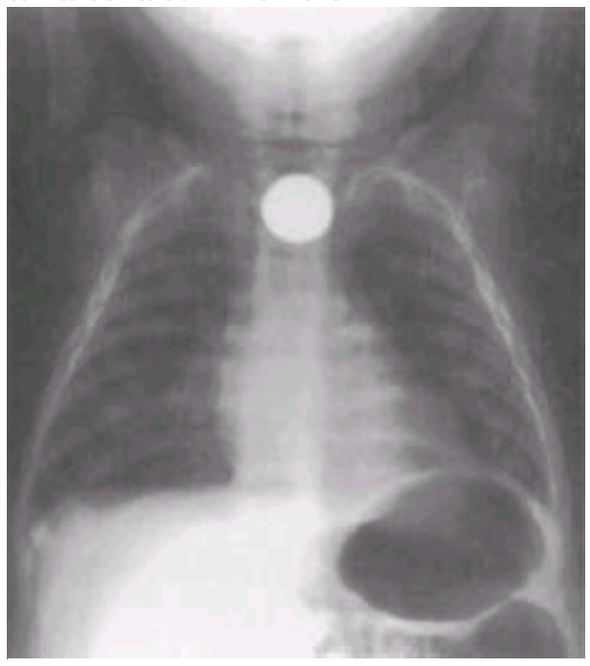


Figure 40-3 Posteroanterior radiograph of an esophageal foreign body (coin) lodged at the level of the aortic cross-over.

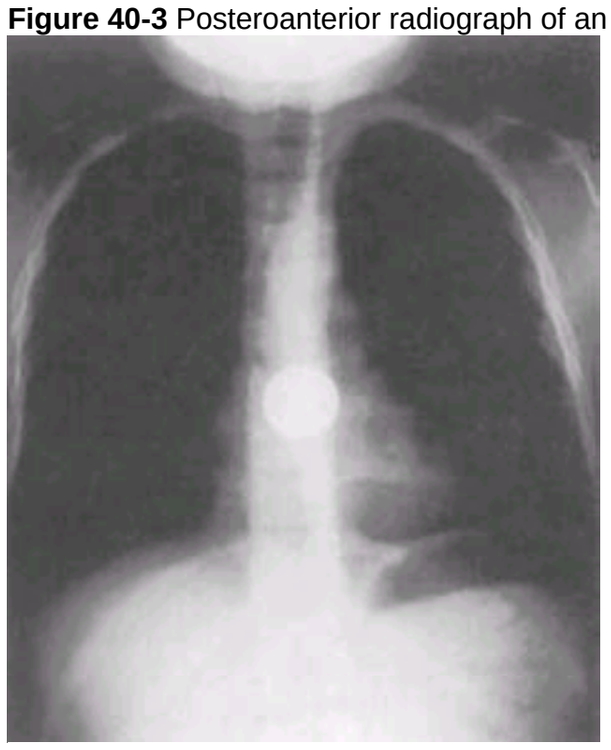


Figure 40-4 A posteroanterior radiograph of an esophageal foreign body (coin) lodged at the level of the lower esophageal sphincter (LES).

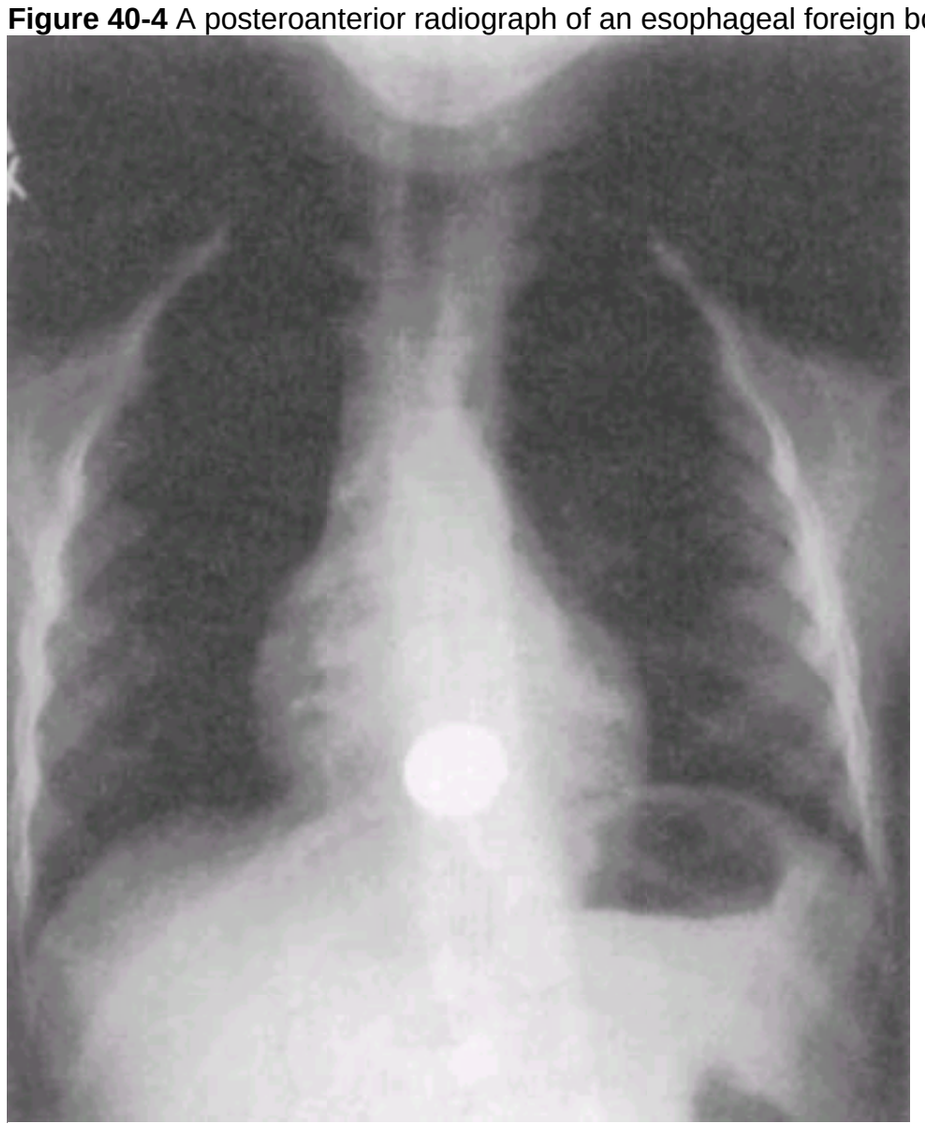


Figure 40-5 A lateral neck radiograph showing a chicken bone lodged in the pharynx with associated soft tissue swelling. The arrow points to the bone. Plain radiographs have poor diagnostic accuracy for detecting bones in the esophagus and they are often eschewed in favor of a CT scan if radiographic evaluation is deemed necessary.

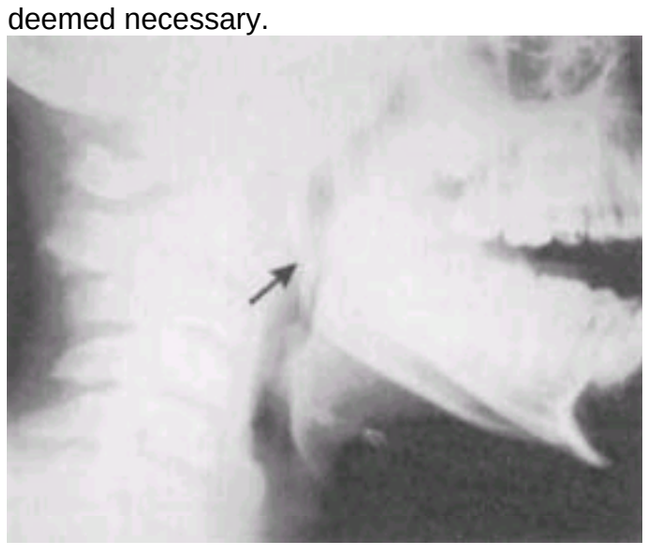


Figure 40-6 A barium swallow demonstrating a complete esophageal obstruction in the proximal to mid-esophagus.

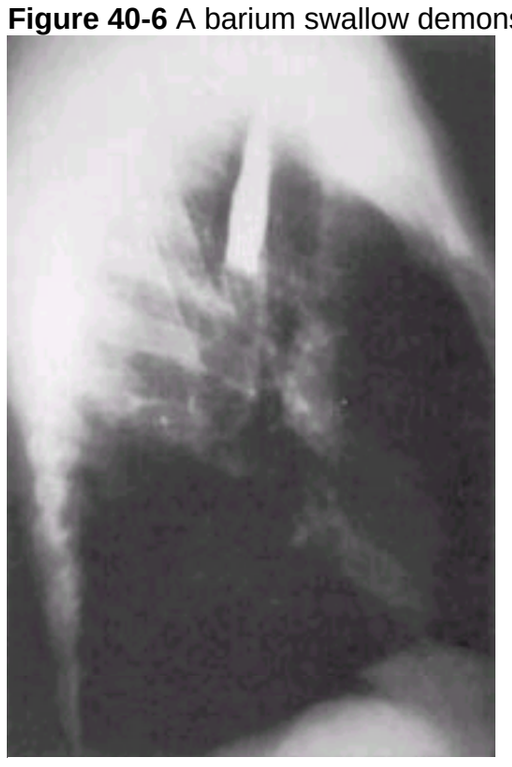


Figure 40-7 Computed tomography demonstrating an esophageal foreign body.

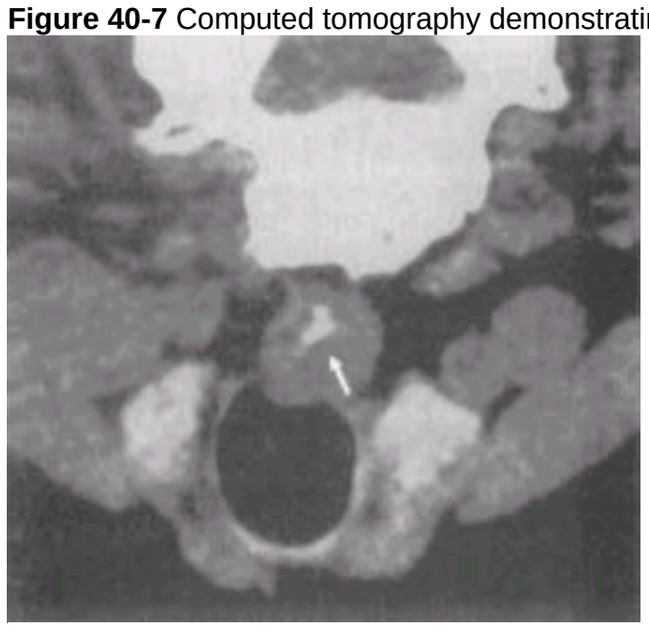


Figure 40-8 Esophageal foreign body seen on nasopharyngoscopy.

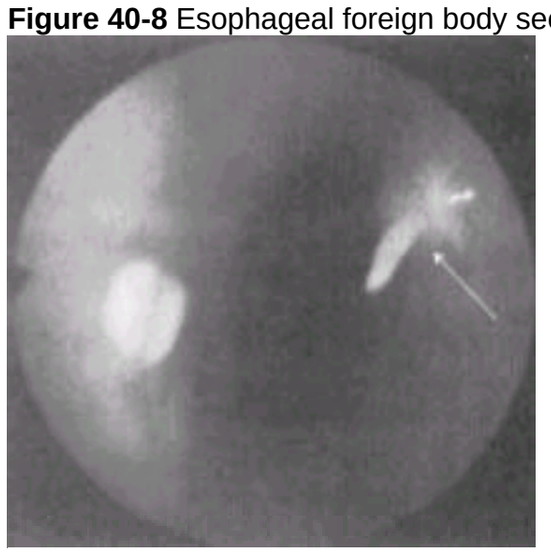


Figure 40-9 Technique of Foley catheter extraction. In children, this procedure is best done with the patient restrained on a papoose board, or consciously sedated, with the head lowered. Some operators place the patient in a prone position or roll the patient to the prone position after catheter insertion to enhance oral expulsion of the foreign body. *A,* A catheter is inserted orally into the esophagus distal to the coin. A bite block may be used to assist oral passage. *B,* The balloon is inflated. Gentle traction moves the coin proximally through the esophagus. *C,* The coin is moved steadily past the glottis. *D,* The coin is present in the mouth to be expectorated or grasped. *(Adapted from McSwain N: Esophageal foreign body. Emerg Med 21:85, 1989.)*

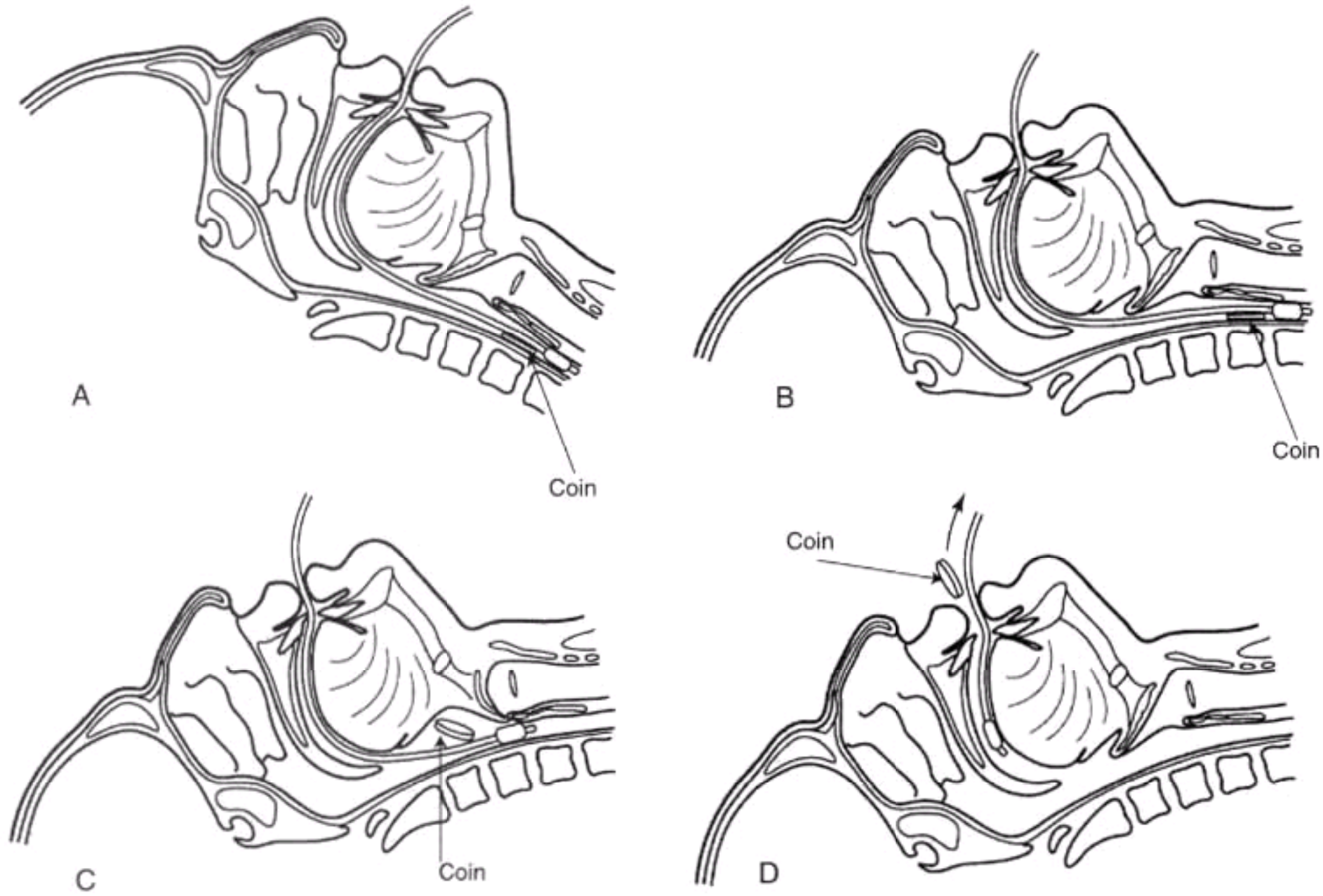


Figure 40-10 *A*, Lateral radiograph of the child shown in Figure 40-2 . This lateral view shows four coins at the same location. Multiple swallowed coins are common in children. It is important to obtain both posteroanterior and lateral films to ascertain the exact number and location of swallowed coins. *B*, A single coin was seen on the PA chest film, but this lateral film suggests three coins. However, they do not seem to be stacked directly on top of each other. This digital radiograph was accidentally exposed three times, and actually only one coin was swallowed.

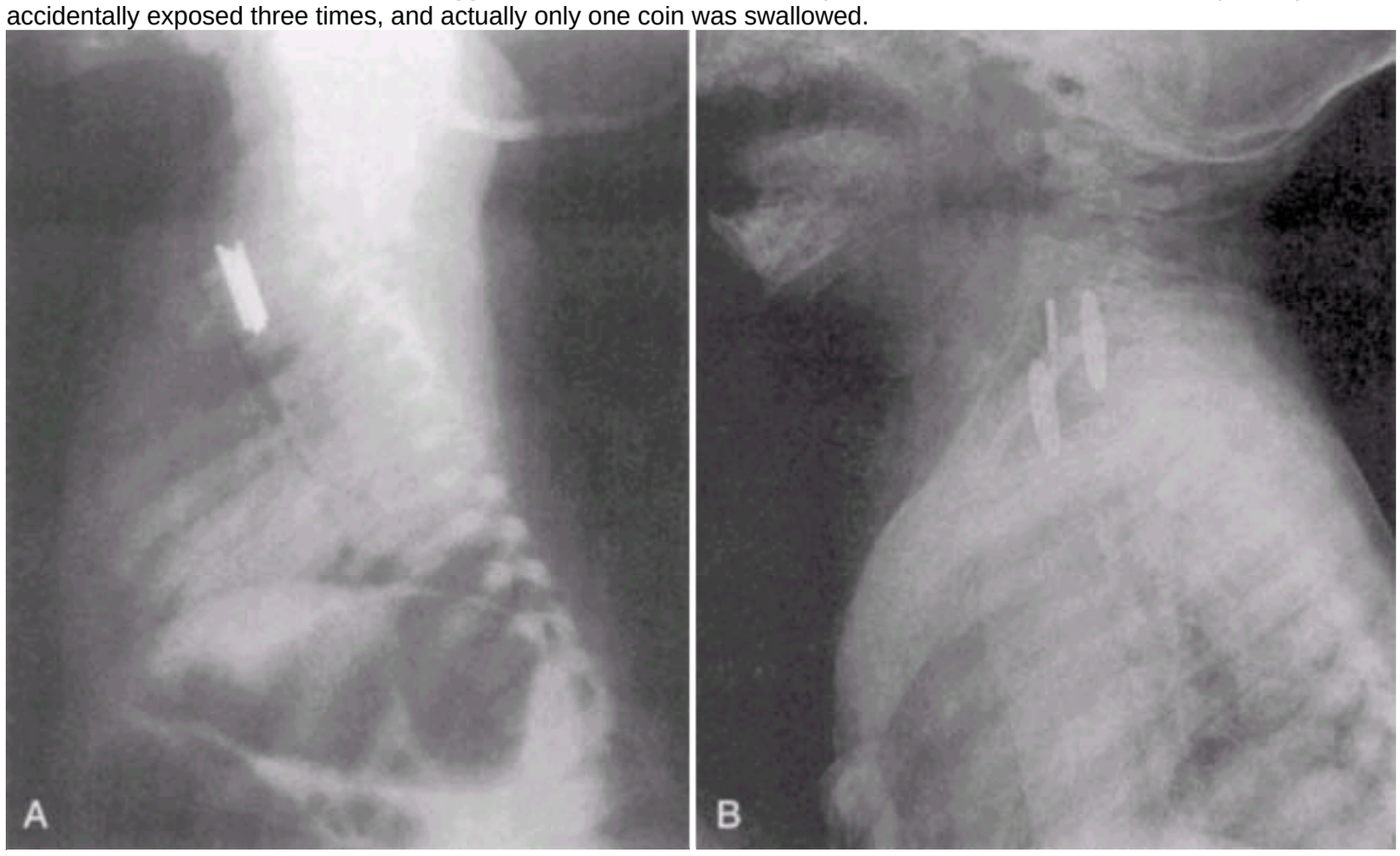


Figure 40-11 Flow diagram outlining an approach to the management of swallowed coins.

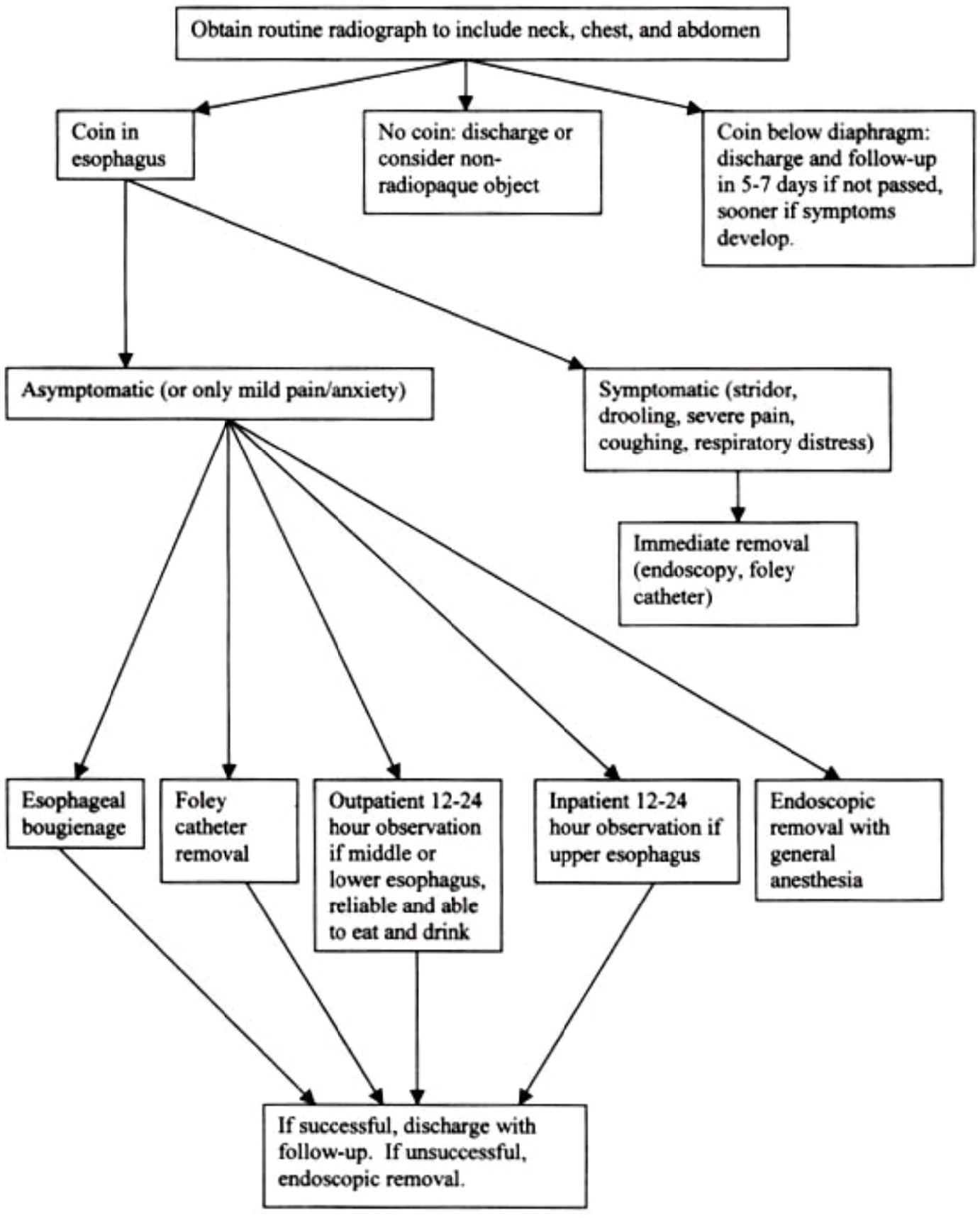


Figure 40-12 Many fish bones become impaled in the soft tissues of the upper digestive tract. This woman felt a bone catch in her throat while eating fish. As is often the case, she was able to consistently localize the FB to the submandibular area (*A*). With only a tongue blade, local anesthetic spray, and good lighting, a fish bone was found embedded in the tonsil and was easily removed with forceps (*B*). Strands of saliva can mimic a fish bone, so be careful when probing and grasping.

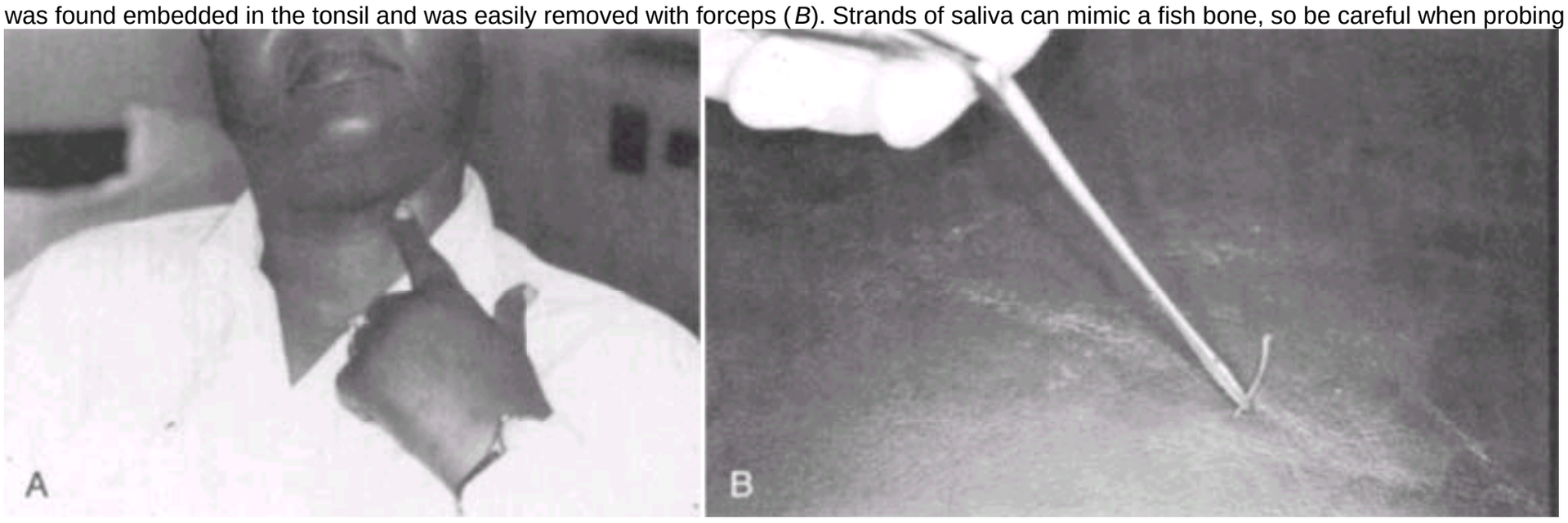


Figure 40-13 *A*, Posteroanterior radiograph of an open safety pin lodged in the upper esophagus. Sharp foreign bodies in the esophagus are best removed with endoscopic visualization. *B*, This 10-year-old child came to the ED with severe chest pain. No history of a foreign body was given. Even when the radiograph demonstrated this metallic object in the esophagus, it remained a mystery how it got there. Objects such as this are removed under anesthesia with an endoscope, and no ED intervention, except for pain relief, is indicated.

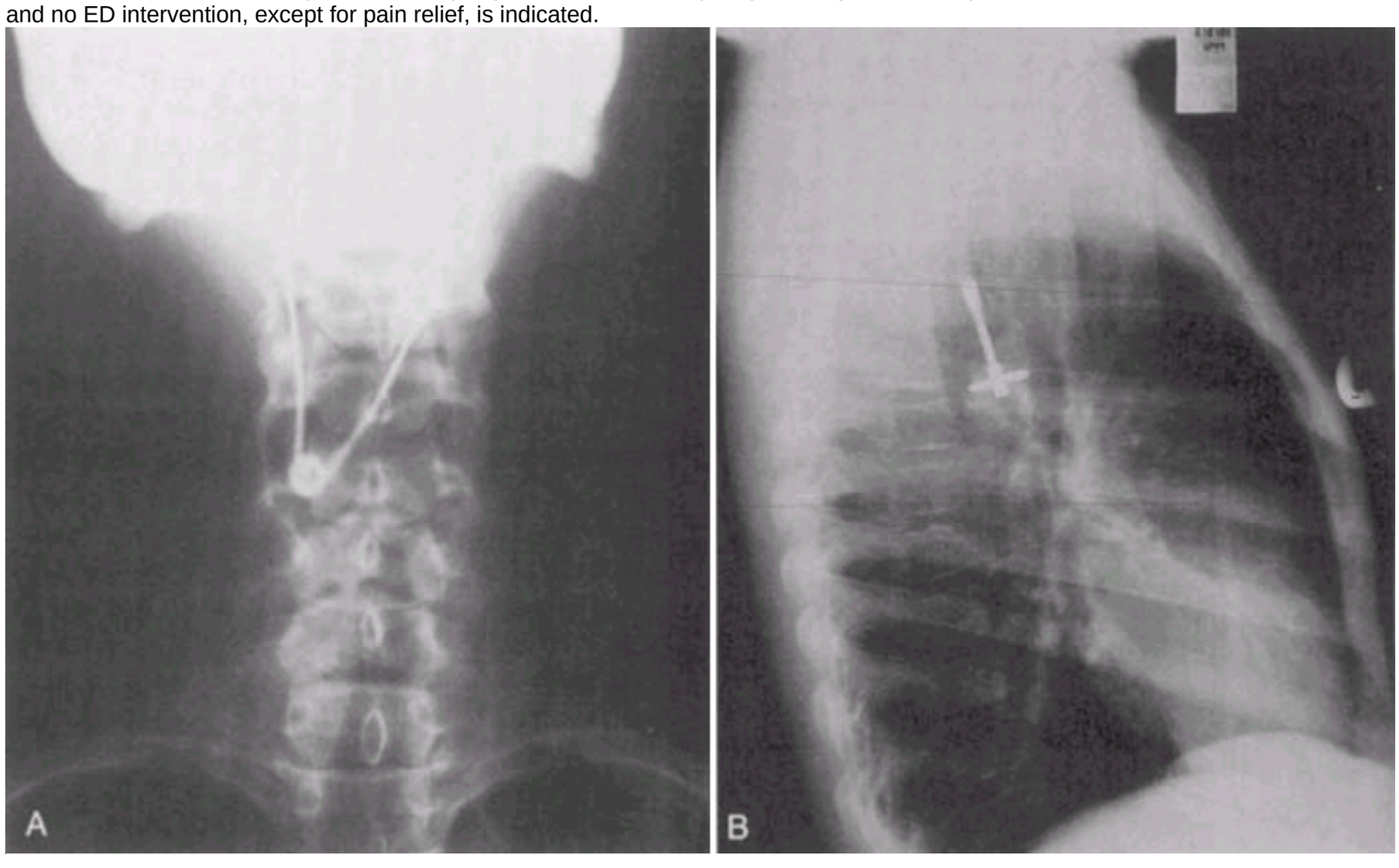


Figure 40-14 Button batteries. These batteries have a wide range of sizes and can mimic coins on radiographs.

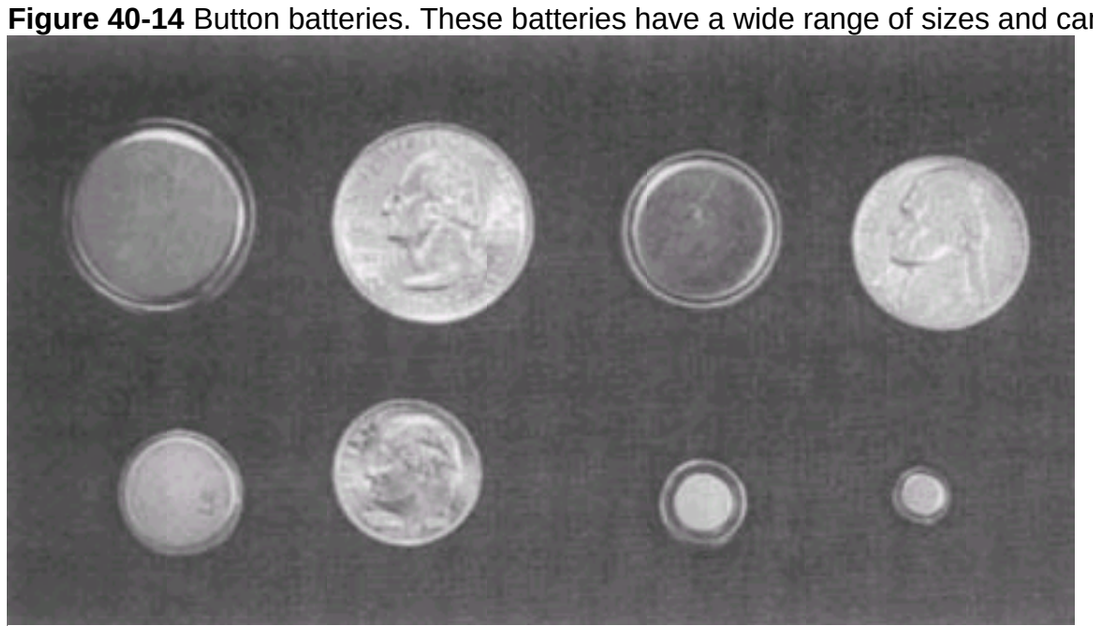


Figure 40-15 Posteroanterior radiograph of a button battery lodged in the esophagus, which can easily be confused with a coin. Button batteries can cause esophageal wall necrosis within 2 hours and must be removed expeditiously. *(From Kost KM, Shapior RS: Button battery ingestion: A case report and review of the literature. J Otolaryngol 16:4, 1987.)*

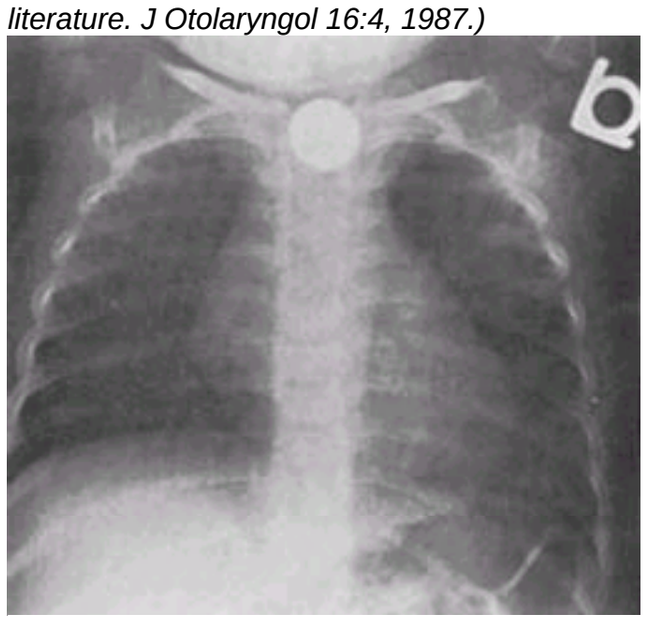


Figure 41-1 Standard nasogastric tubes. *A,* Standard Levin tube. This tube has a single lumen. It is sufficient for instilling material into the stomach or for diagnostic stomach aspiration. *B,* Salem sump tube. This tube contains a second lumen that allows venting during continuous suction. *1,* Gastric end with suction eyes. *2,* Pigtail extension (blue) of the air vent lumen. *3,* Connector for attachment of suction lumen to vacuum line.

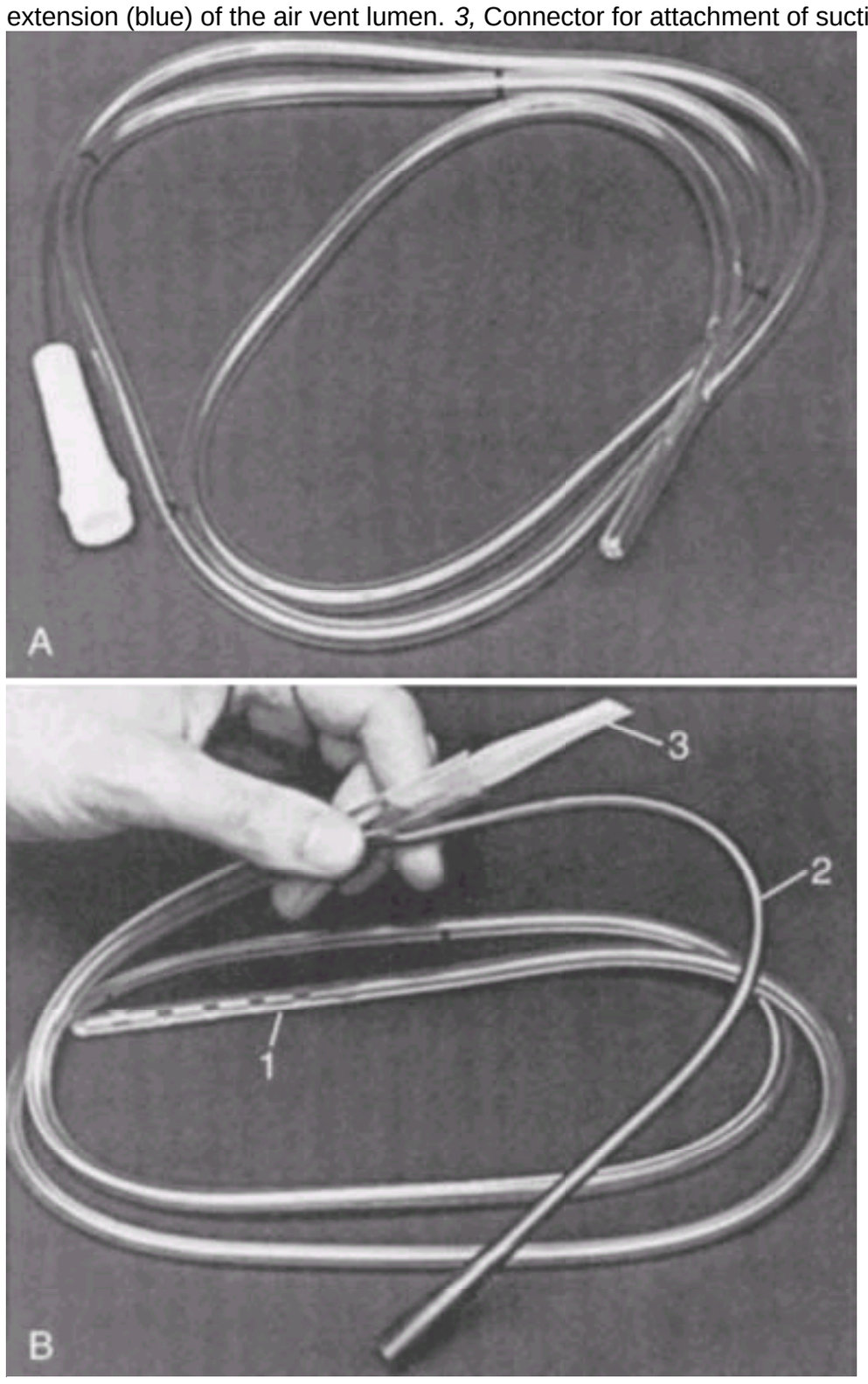
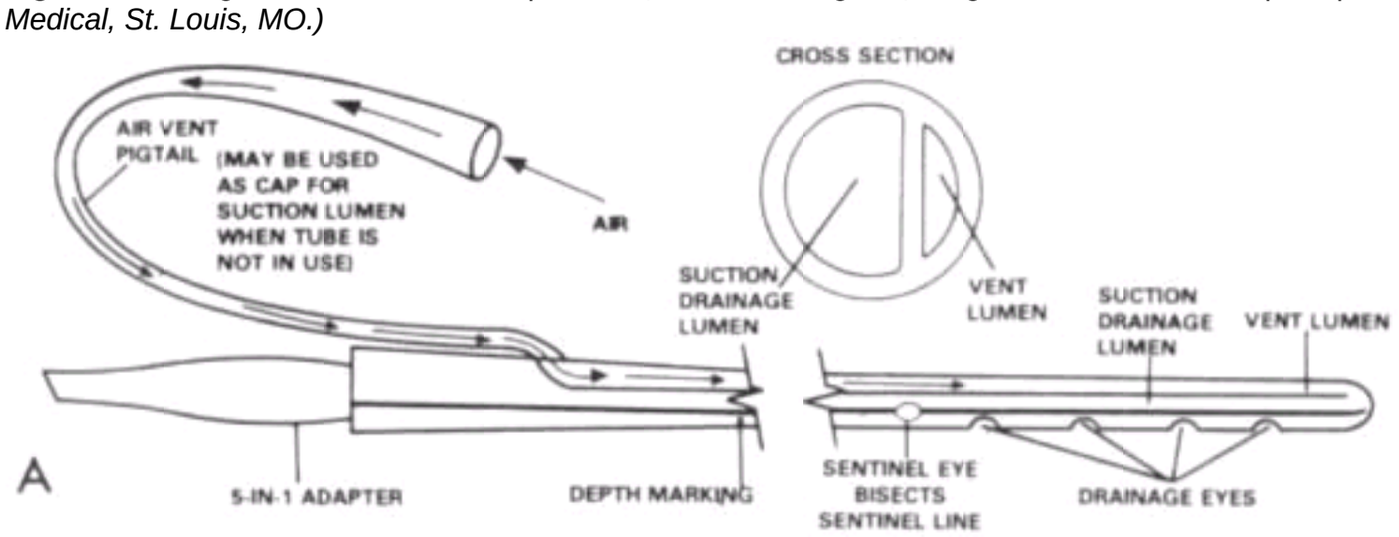


Figure 41-2 Diagram of the Salem sump tube. *A,* General design. *B,* Diagram of double-lumen principle for suction. *(Courtesy of the Argyle Division of Sherwood Medical, St. Louis, MO.)*



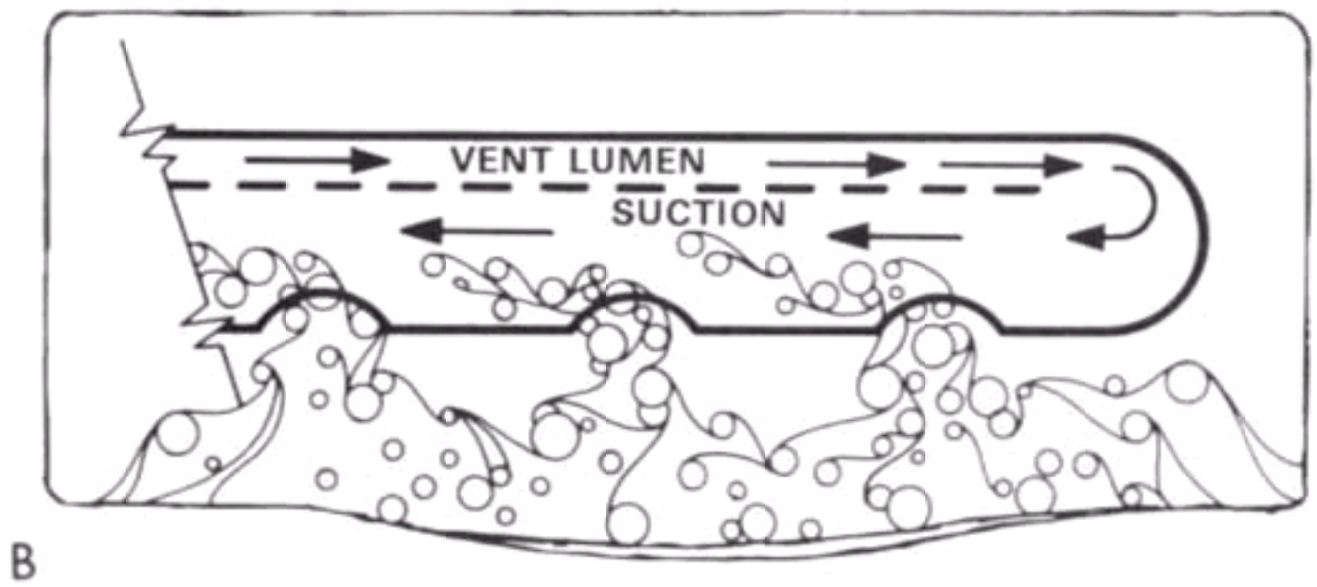


Figure 41-3 An NG tube may enter the cranium or facial soft tissues in patients with severe head or facial trauma. Those with a coagulopathy may experience significant bleeding from nasal or pharyngeal trauma during passage of an NG tube. In such cases, a standard NG tube inserted through the mouth may be a better alternative.

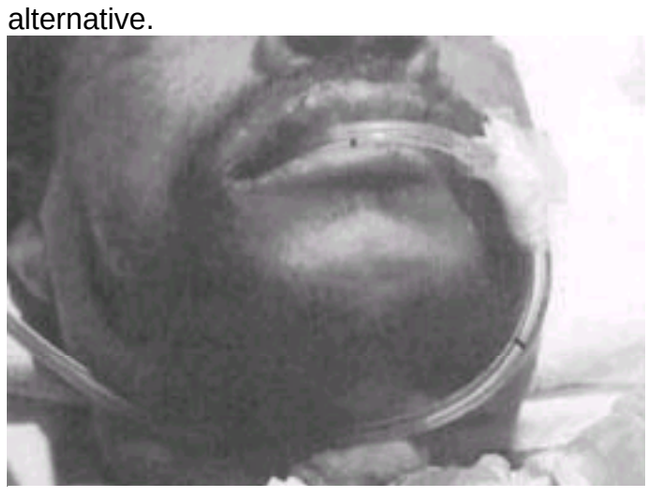


Figure 41-4 Estimation of tube insertion depth. Before inserting the nasogastric tube, the clinician should estimate the length of tubing that will be required to ensure intragastric positioning without excess coiling. Holding the tube against the patient's body, measure the distance from the tip of the xiphoid to the earlobe *(A)*. Add the distance from the earlobe to the tip of the nose *(B)*. Then add another 15 cm *(C)*. Note the total distance using markers on the tube, or attach a piece of tape to the tube.

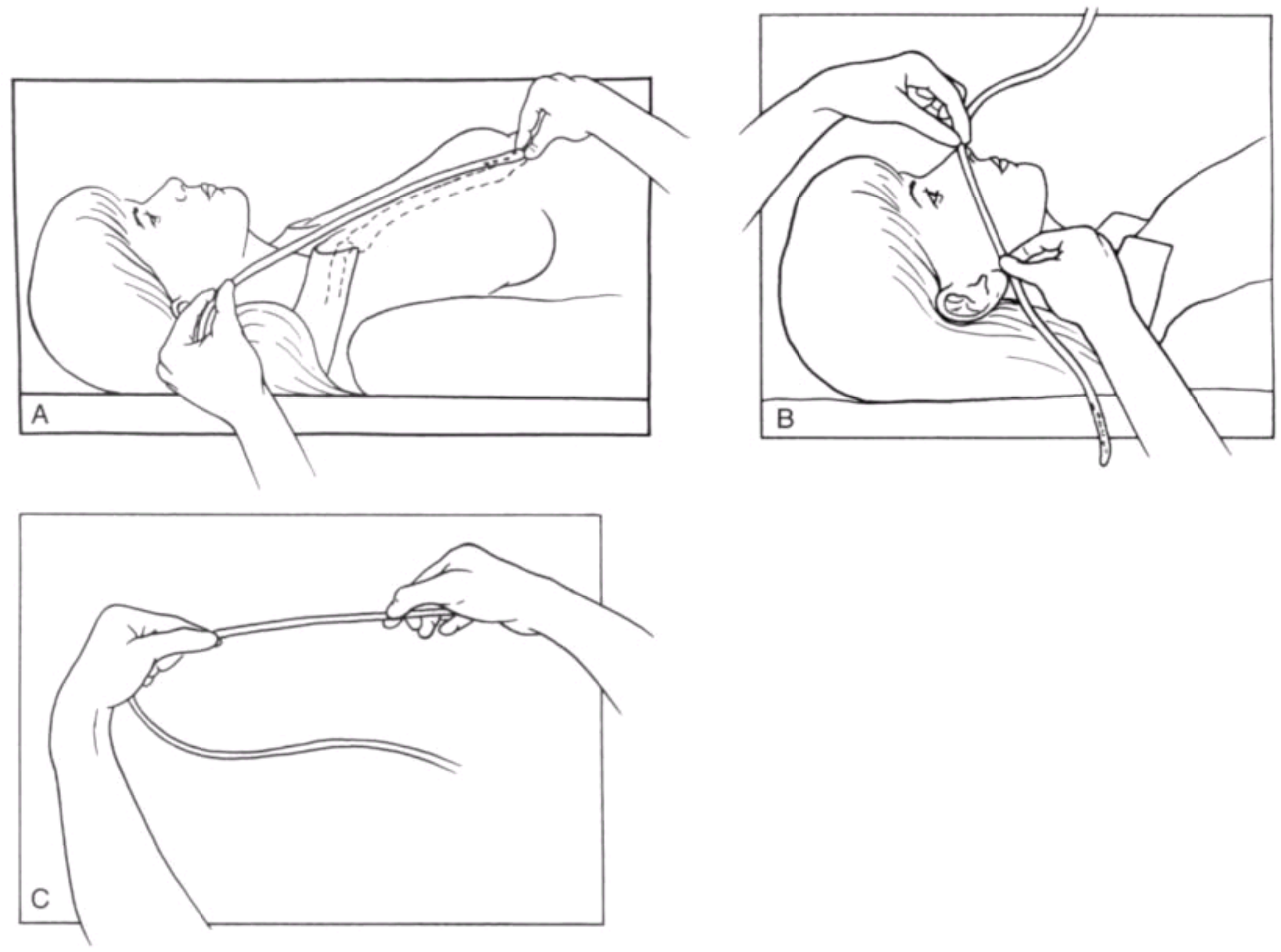


Figure 41-5 If time and situation permit, administration of nebulized lidocaine (2.5 mL of 4% solution) via a face mask and standard nebulizer used to deliver asthma medications, can decrease pain and gagging without increasing aberrant placement. The patient is instructed to breathe through the mouth and nose to anesthetize the nose and pharynx.

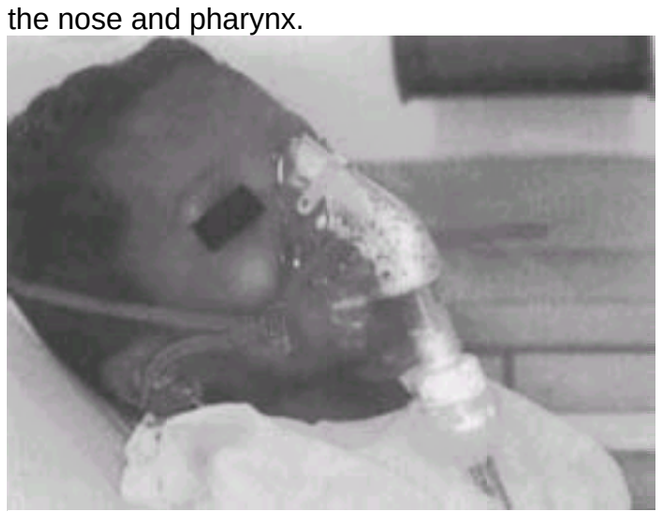


Figure 41-6 Whenever possible, some form of topical anesthesia should be used before passing a nasogastric tube. The method pictured can be used in addition to atomized anesthetic, and will be more effective than treating only the nasal opening. Fill a syringe barrel with 5 mL of 2% viscous lidocaine. Without using a needle, squirt the solution along the floor of the nose and allow it to drip into the nasopharynx and be swallowed. This method works best with the patient supine. Wait 5 minutes for the anesthetic to take effect.

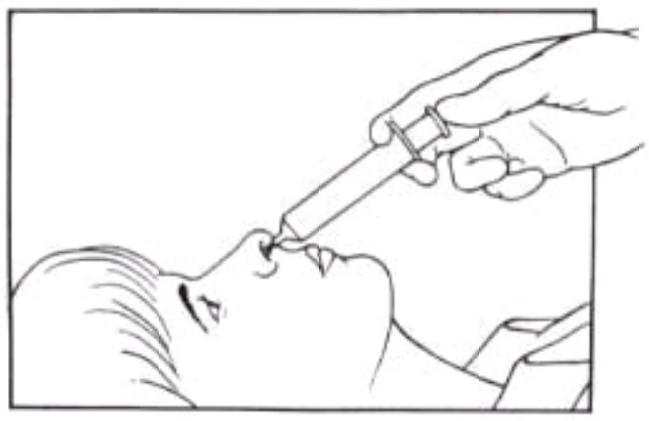


Figure 41-7 Tube location in the nostril. The nasogastric tube is directed along the *floor* of the nose, not toward the nasal bridge. The tube often slides through the tunnel beneath the inferior turbinate.

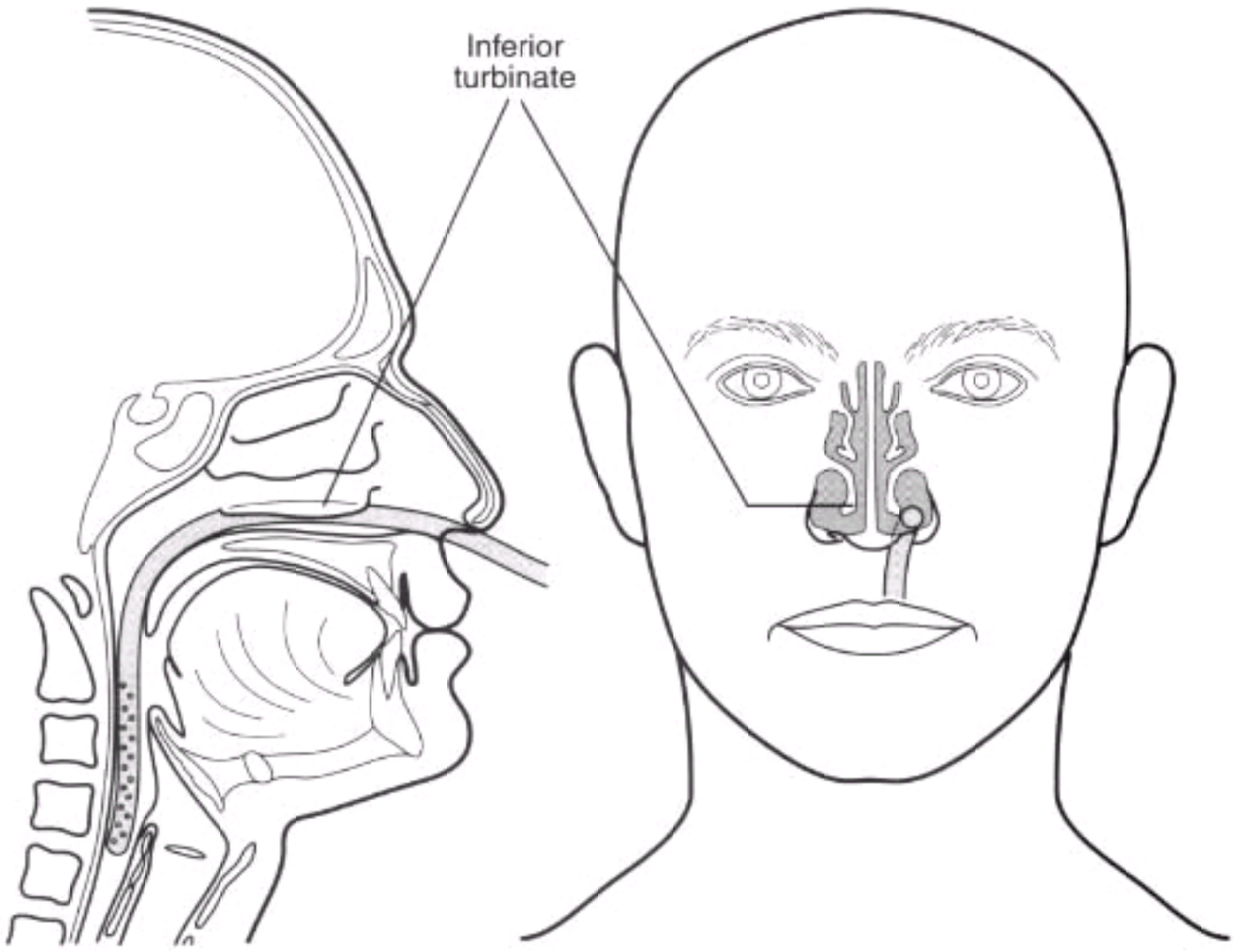


Figure 41-8 Attachment of tube to gown. *A,* Attach the nasogastric tube to the patient's gown using a rubber band and a safety pin as shown, so that the first tug on the tube pulls the gown and not the tape holding the tube in the patient's nose. *B,* Close-up view of attachment site.

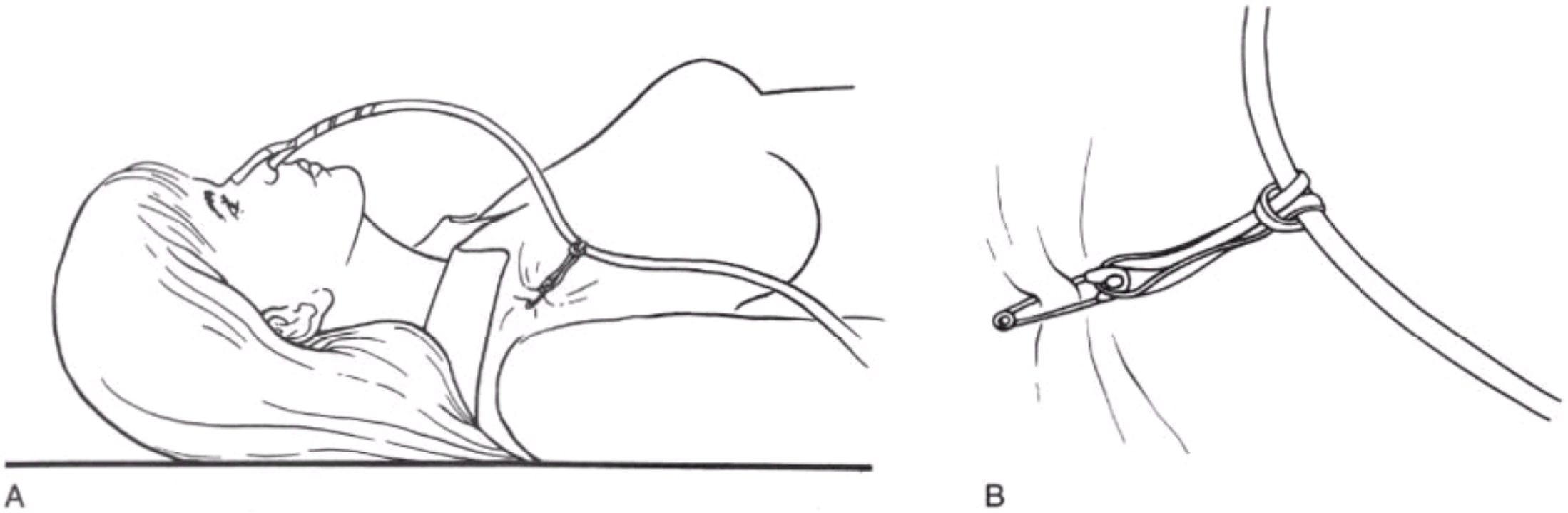


Figure 41-9 Diagrammatic representation of the separation of the nasogastric tube from the guiding endotracheal (ET) tube through the slit in the guiding ET tube. The nasogastric tube has first been passed through the nose and is pulled out through the mouth. The tip of the tube is then threaded into the guiding ET tube to ensure passage down the esophagus. The guiding ET tube is removed from the esophagus before separation from the nasogastric tube. Note the prior placement of another endotracheal tube in the trachea (partially shown) to avert passage of the guiding ET tube into the trachea. *(From Sprague DH, Carter SR: An alternate method for nasogastric tube insertion. Anesthesiology 53:436, 1980.)*

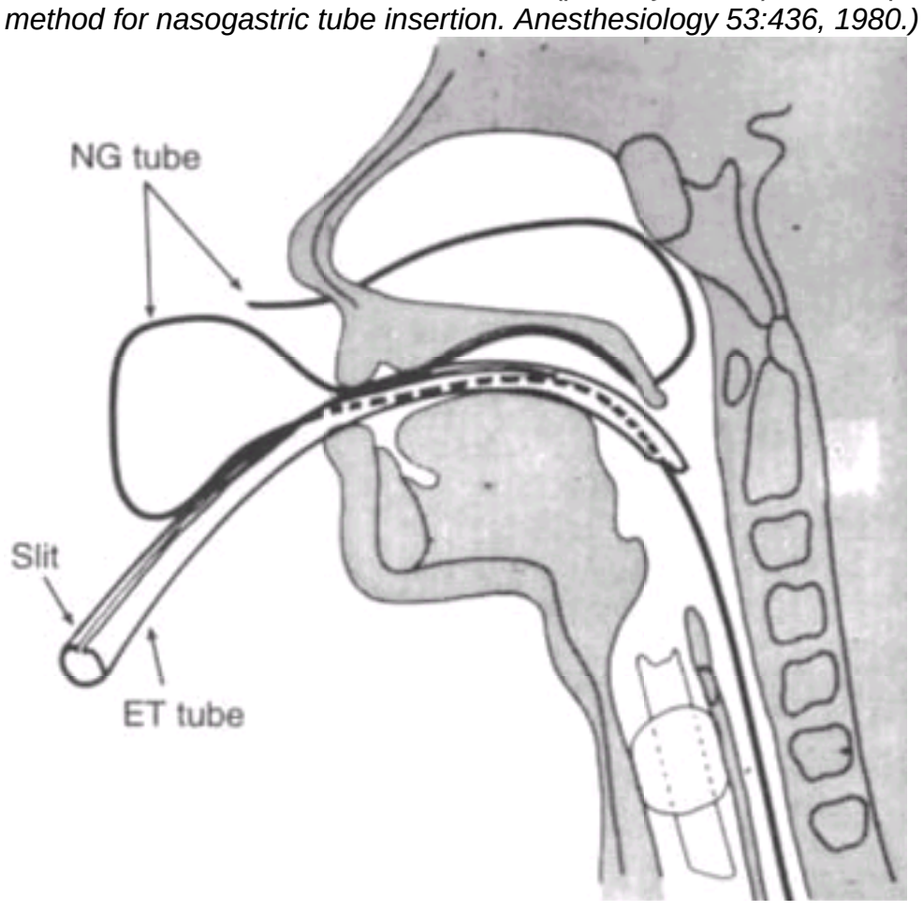


Figure 41-10 The passage of an nasogastric tube through the nose of an intubated patient. An endotracheal tube is in the tracheal via the mouth. The second and third fingers are placed into the posterior pharynx. Fingers depress the tongue. The nasogastric tube is guided down the esophagus by passing it through the second and third fingers that are in the posterior pharynx. The thumb is under the jaw and pulls the jaw forward.

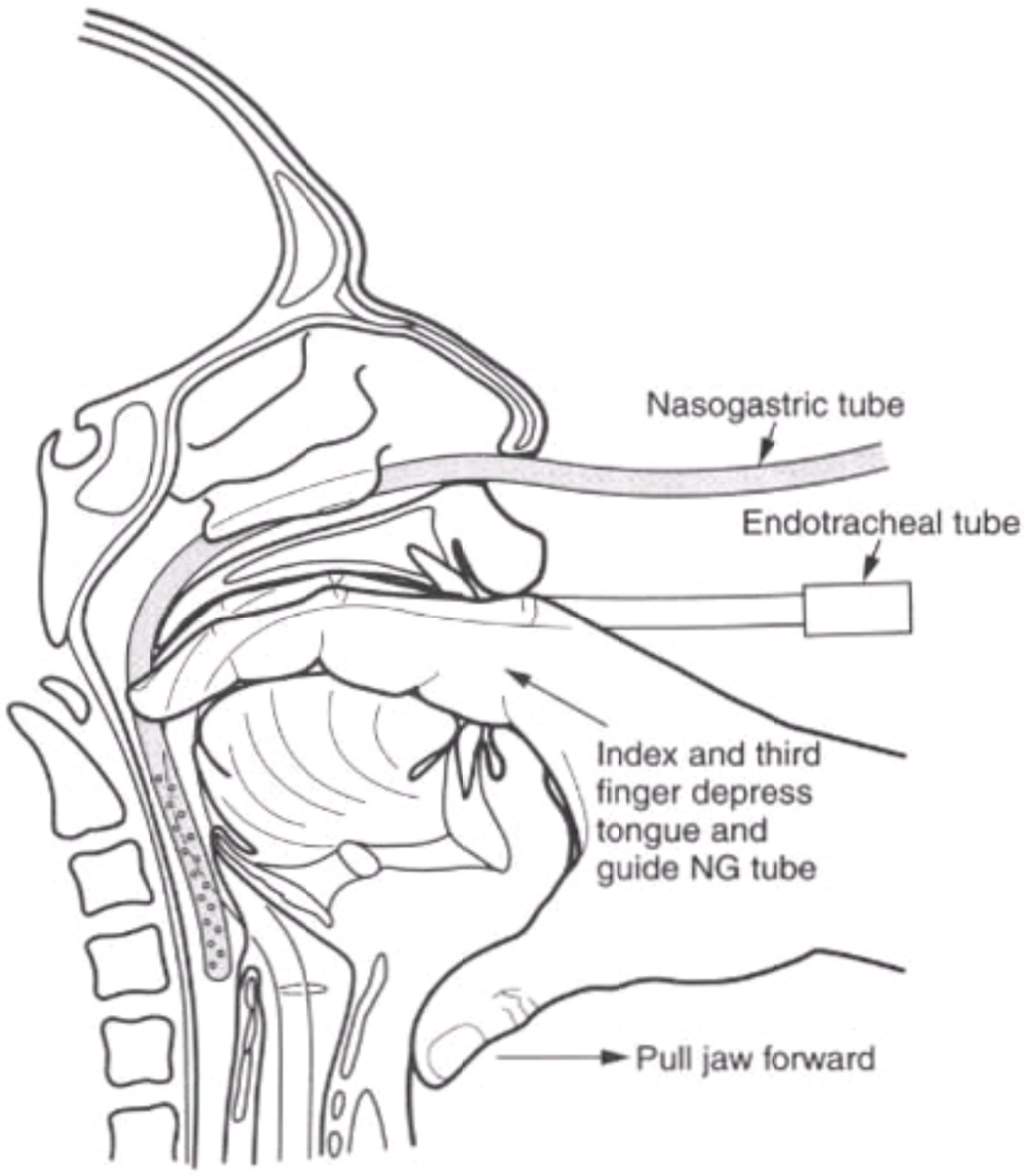


Figure 41-11 Levin tube inadvertently placed in the right main stem bronchus; an alveolar infiltrate consistent with early pneumonia is also shown. *(From Johnson JC: Letter to the editor: Back to basics for morbidity-free nasogastric intubation. JACEP 8:289, 1979.)*

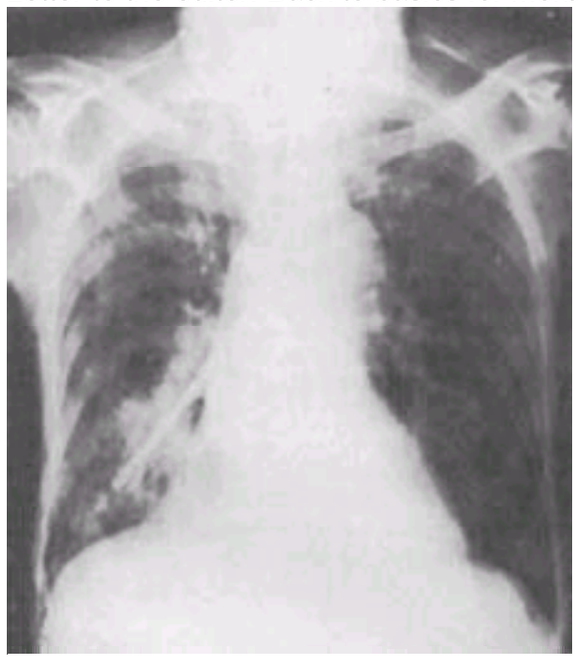


Figure 41-12 Anteroposterior and lateral skull radiographs demonstrating intracranial insertion of a nasogastric tube in a patient with multiple skull fractures. *(From Johnson JC: Letter to the editor: Back to basics for morbidity-free nasogastric intubation. JACEP 8:289, 1979.)*

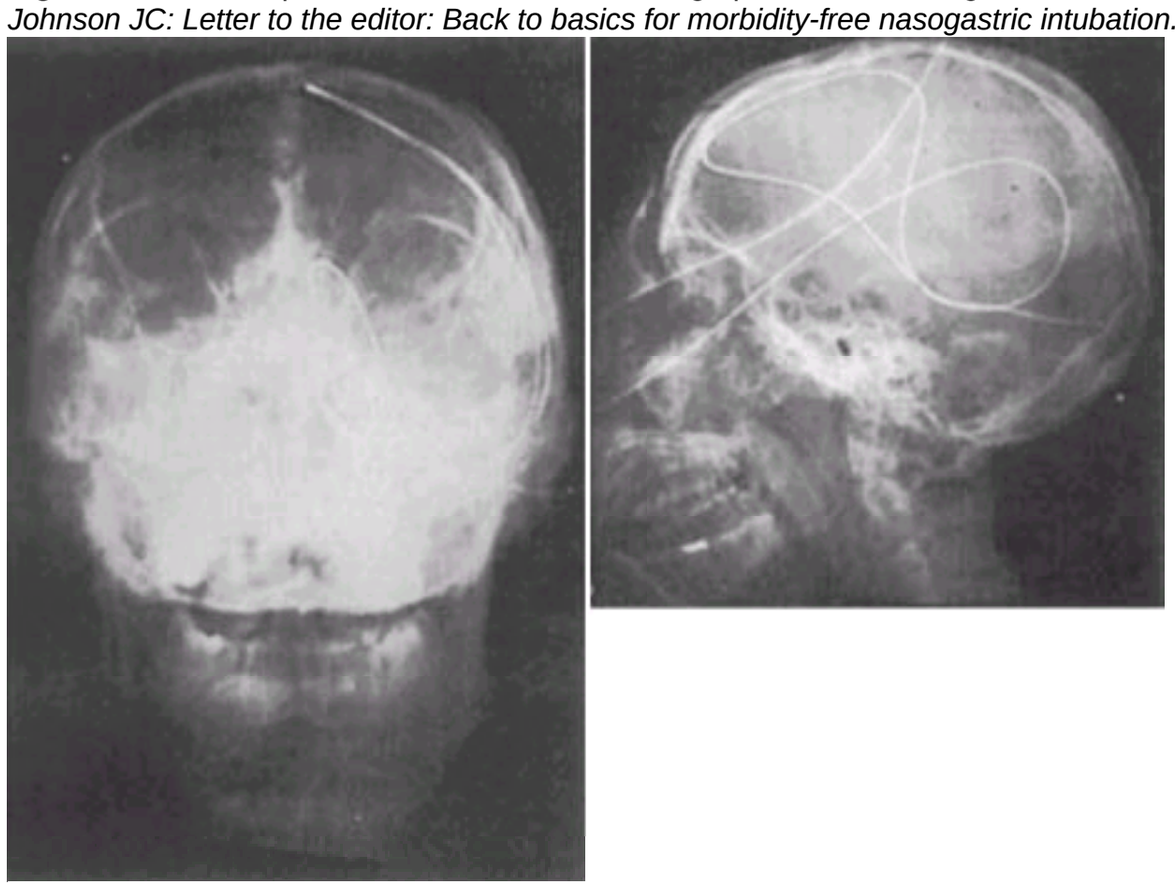


Figure 41-13 To facilitate the passage of a soft or very pliable plastic feeding tube, the tip of the feeding tube is joined to the tip of a stiffer nasogastric tube (such as a Salem sump) with the aid of a gelatin capsule. The capsule dissolves in the stomach in a few minutes, and the companion nasogastric tube separates and is withdrawn, leaving the feeding tube in place.

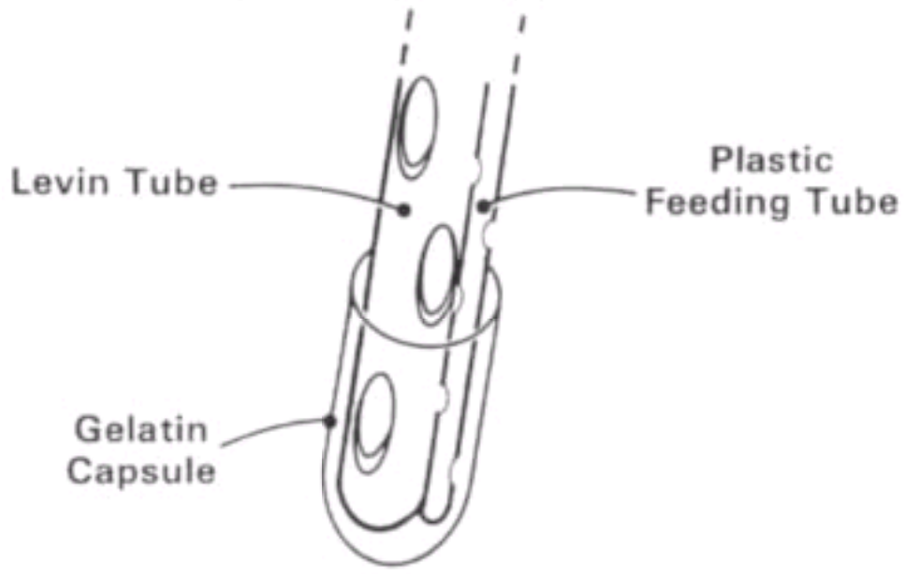
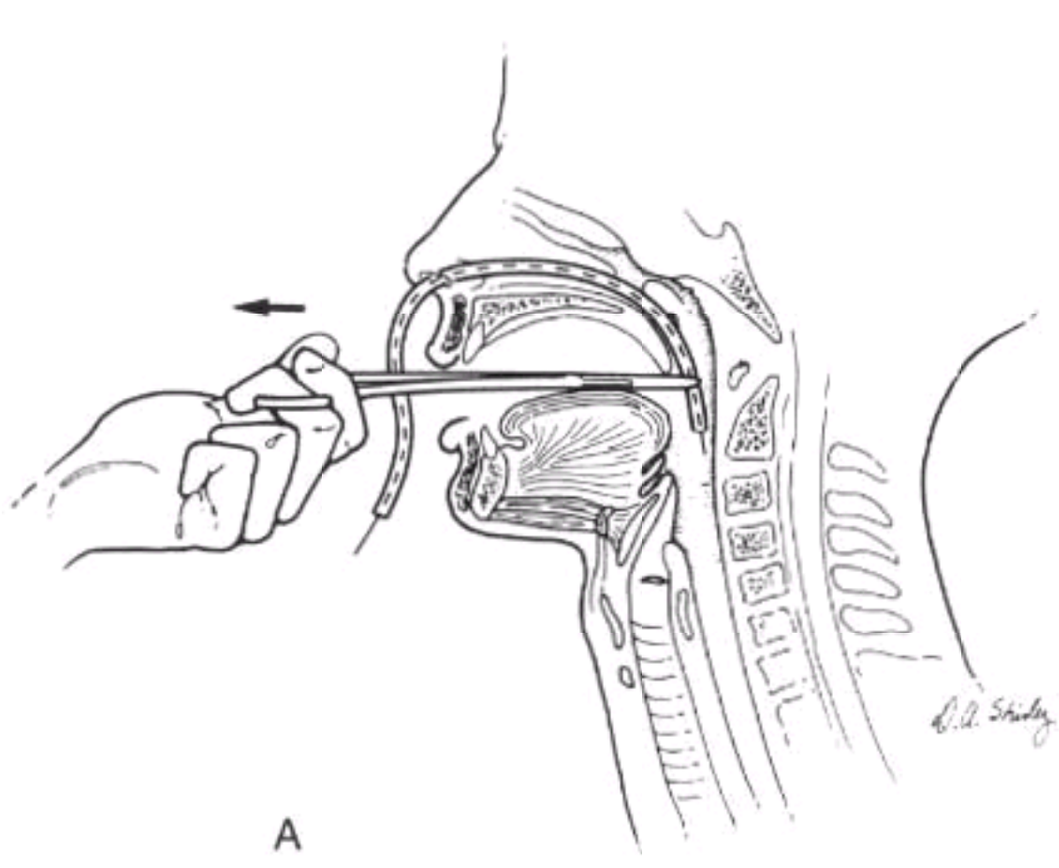
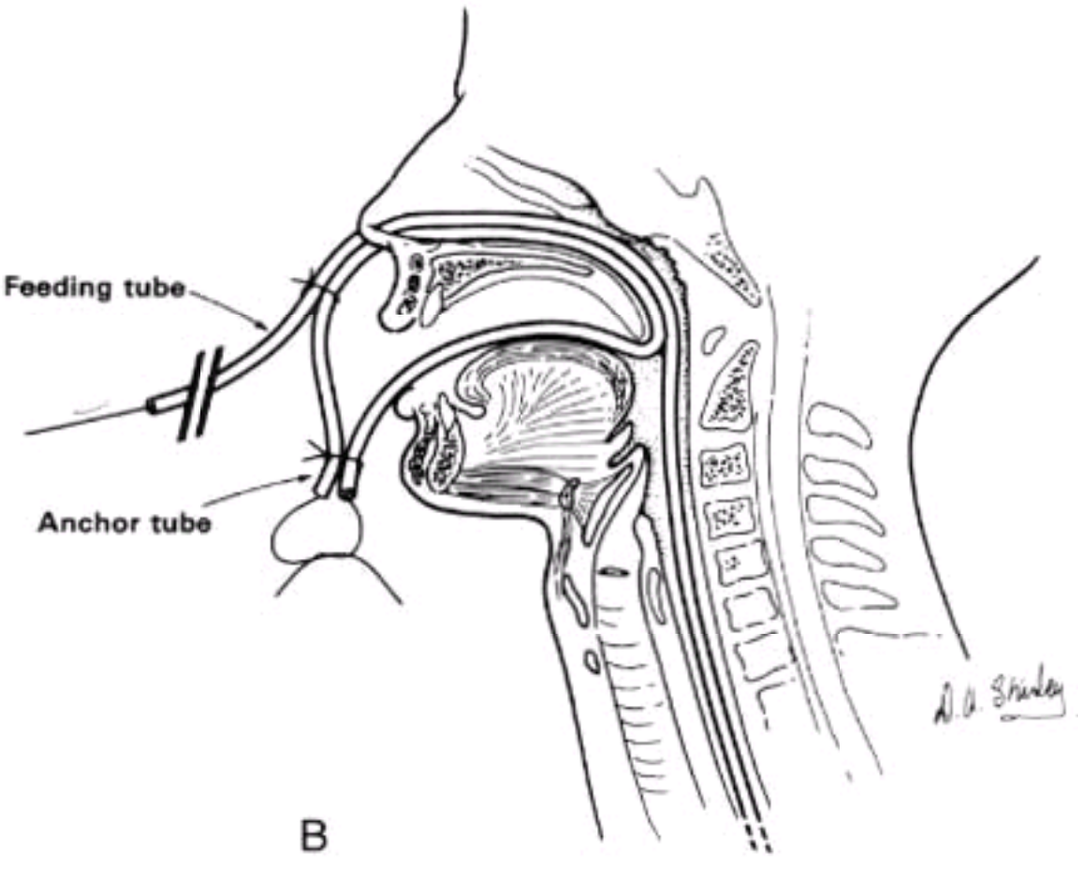


Figure 41-14 Placement of a nasogastric tube anchor to secure a companion nasogastric or feeding tube in an uncooperative patient who repeatedly pulls out the feeding tube. *A*, Forceps grasp the tube in the pharynx and pull it out through the mouth. This will serve as an anchor tube. *B*, The ends of the short anchor tube are tied together to form a loop, and the companion nasogastric or feeding tube is tied to the anchor loop.



A



B

Figure 41-15 Pharyngostomy feeding tube.

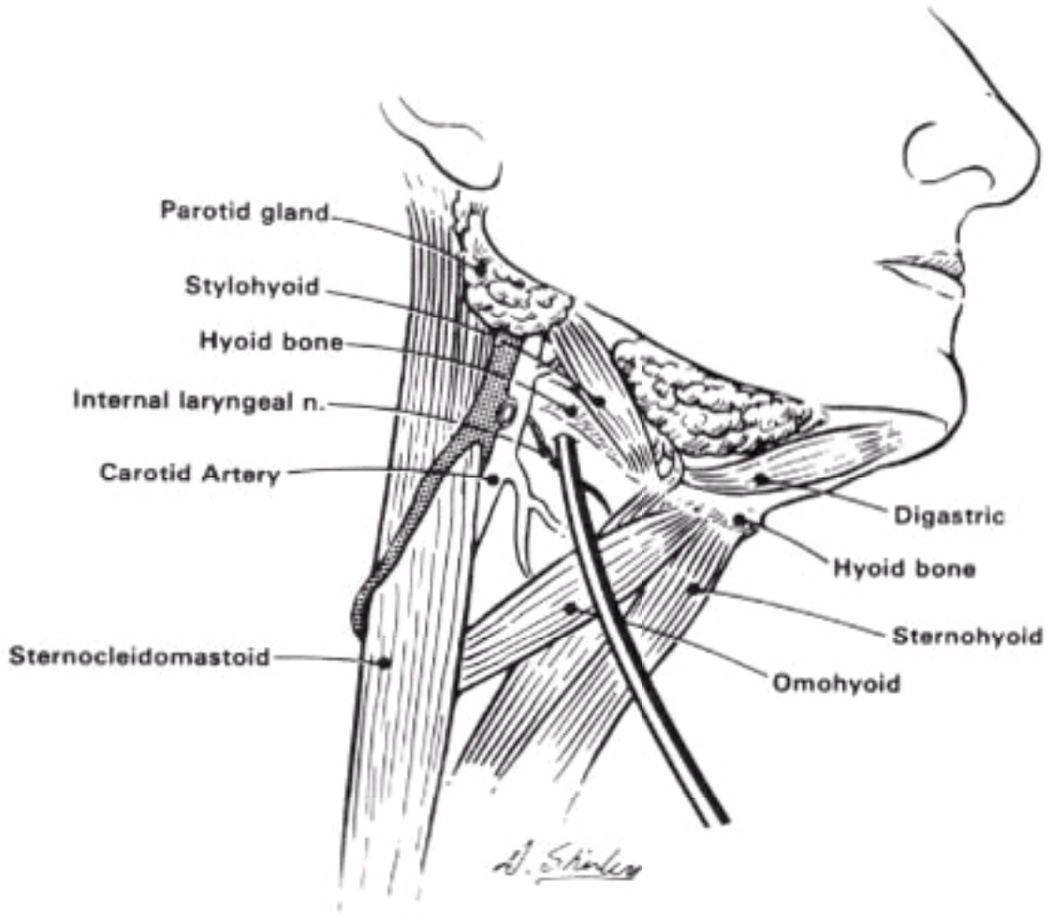


Figure 41-16 Proper path for an esophagostomy or a pharyngostomy tube.

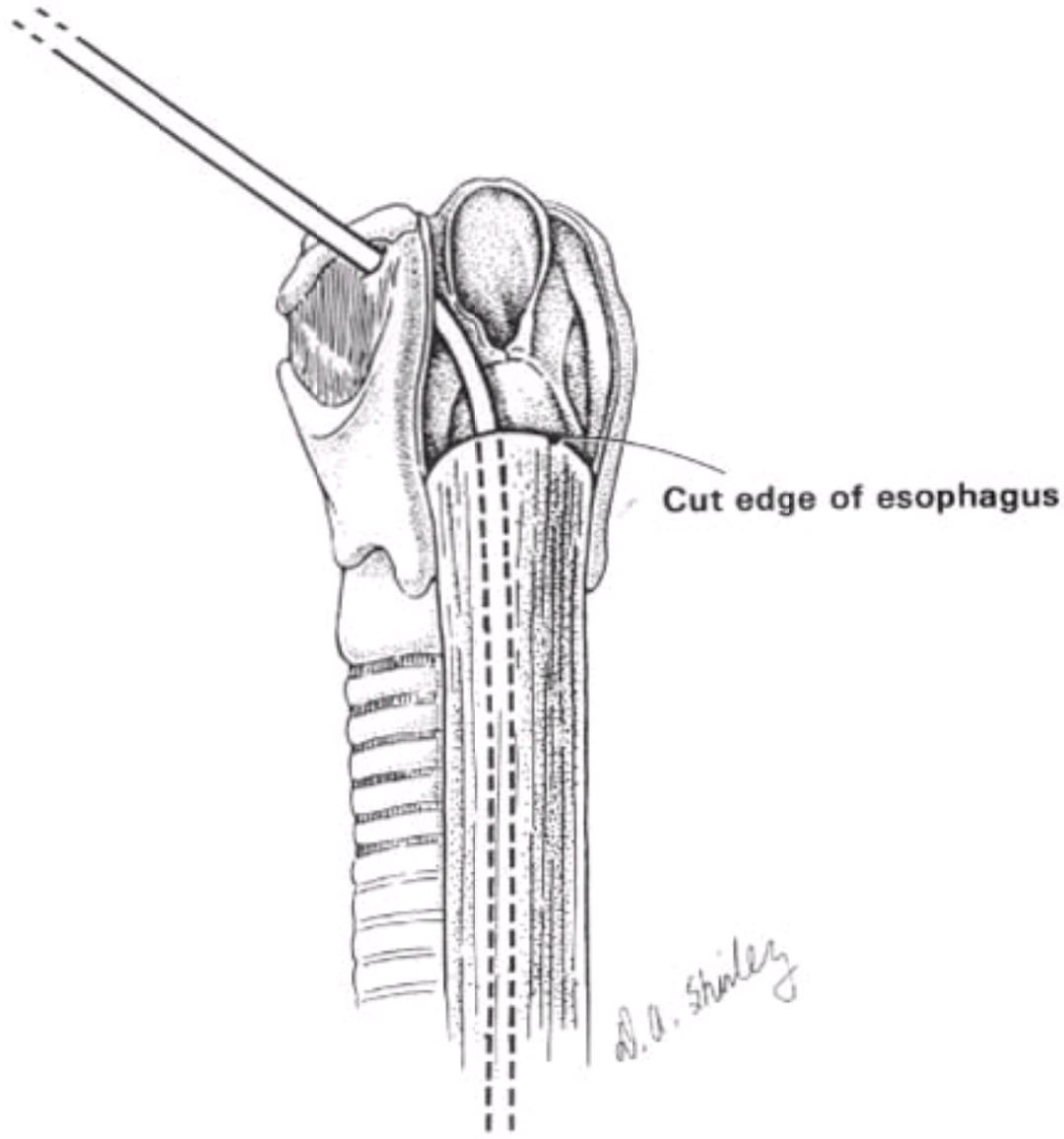


Figure 41-17 *A,* Formation of the Witzel tunnel. *B,* Final catheter placement. *(From Wiedeman JE, Smith VC: Use of the Hickman catheter for jejunal feedings in children. Surg Gynecol Obstet 162:69, 1986.)*

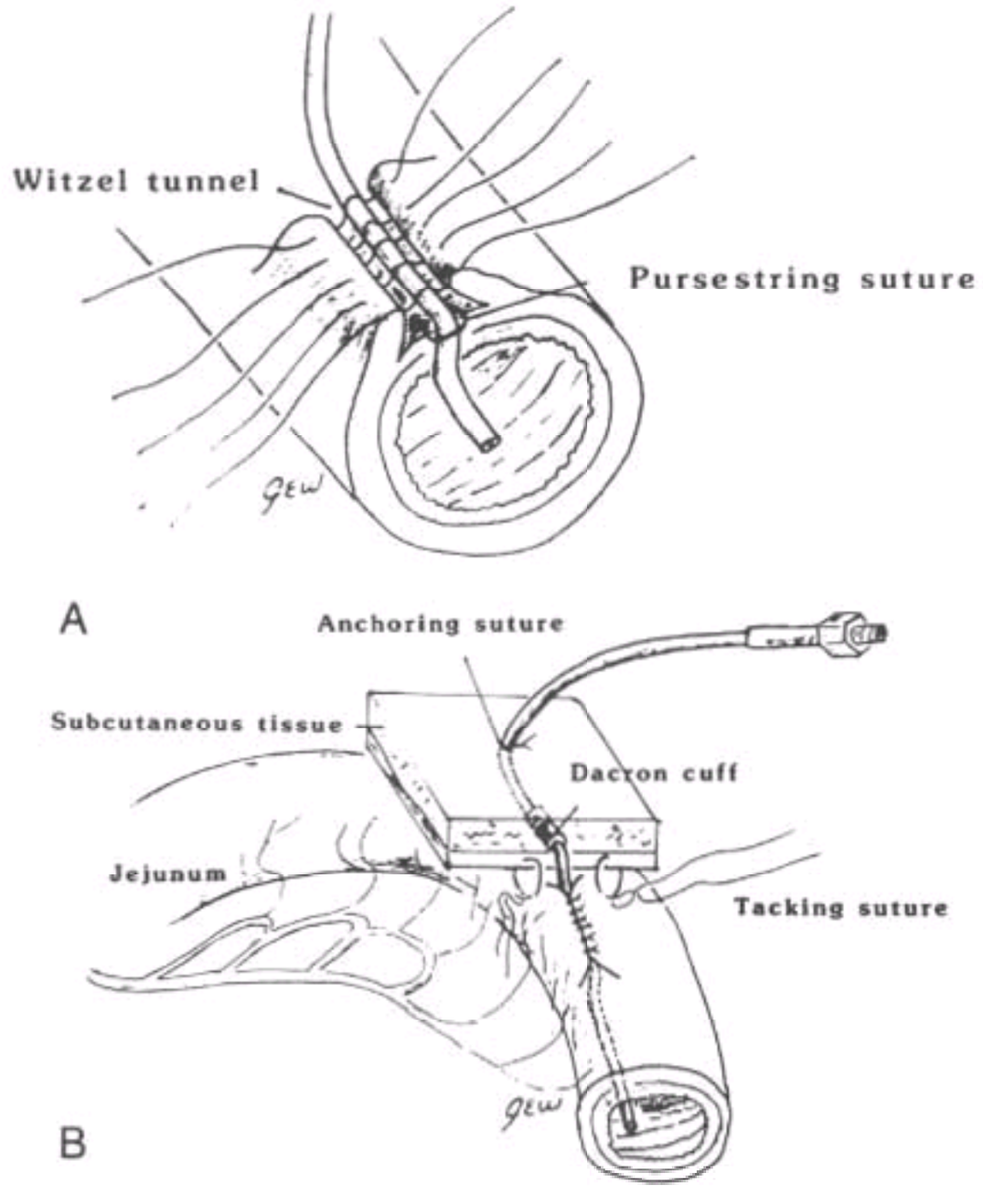
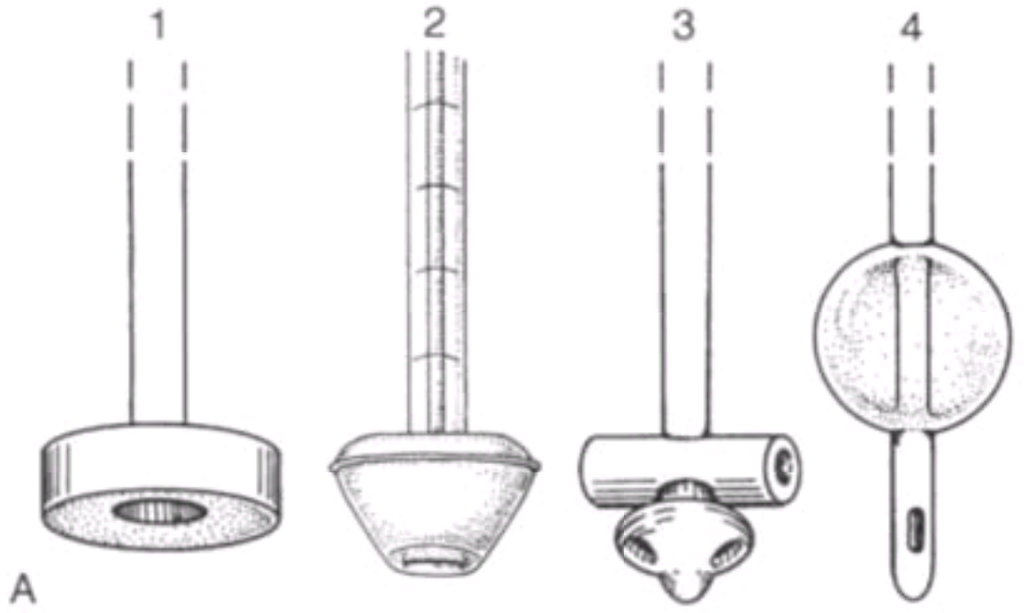
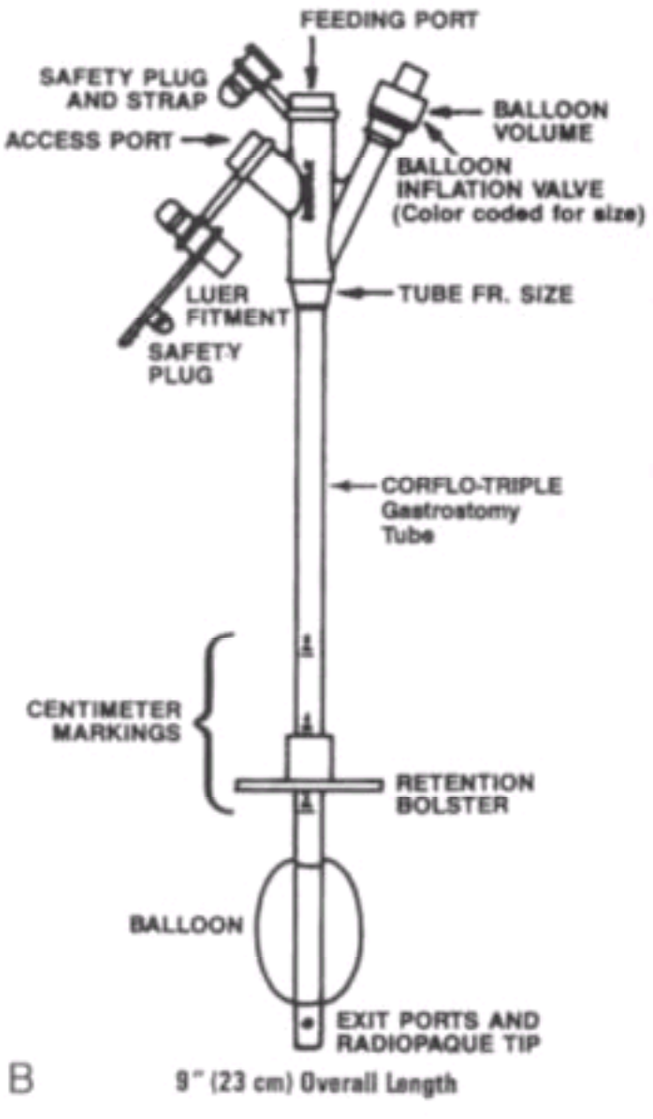
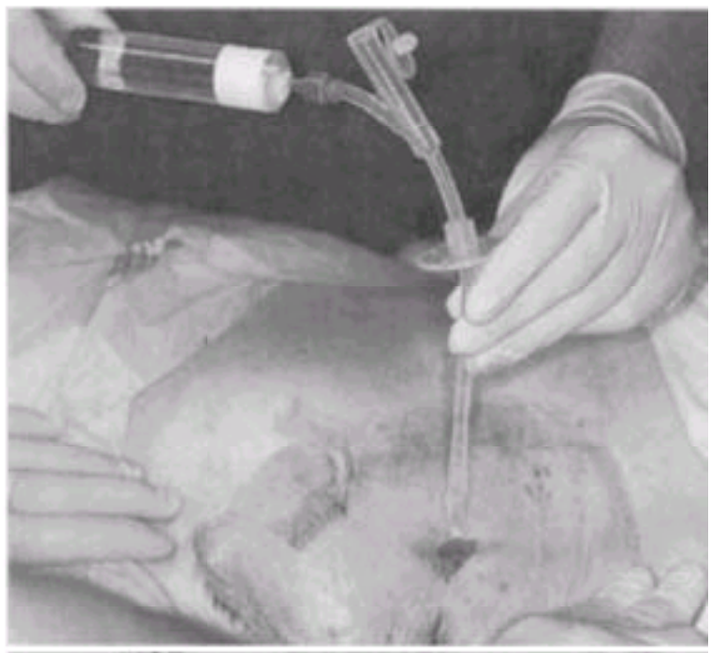
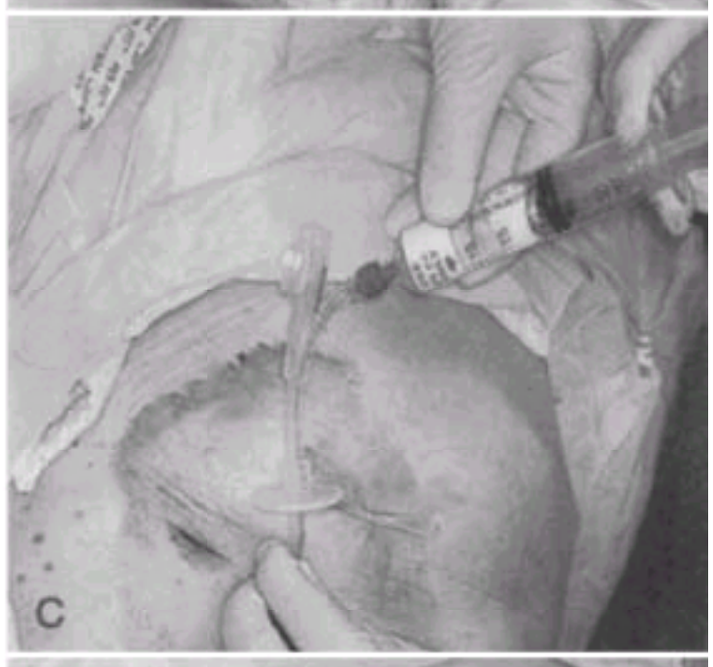


Figure 41-18 *A,* Various types of gastrostomy tubes. *1,* Polyurethane catheter with collapsible foam flange (CORPAK MedSystems of Kentec Medical Inc, Irvine, CA). *2,* Silicone catheter (American Endoscopy [Bard Interventional Products, Billerica, MA]). *3,* Latex catheter with a movable external bolster and an internal mushroom or de Pezzer-type flange on the end (American Endoscopy [Bard Interventional Products, Billerica, MA]). *4,* Balloon (Foley) catheter (Wilson-Cook Co., Winston-Salem, NC). *B,* A user-friendly gastrostomy tube is supplied by CORPAK MedSystems (Wheeling, IL). The tube is packaged with lubricant, a prefilled syringe for inflating the balloon, and an extension set. The color-coded inflation valve indicates tube size (12–24 Fr). The silicone tube uses a retention balloon and a movable bolster, similar in design to a Foley catheter. Note that the retention bolster is designed to prevent inward migration of the tube and is not to be an anchoring device sutured to the skin. *C,* Placement of the CORPACK feeding tube and inflation of the balloon. Note that the bolster on the external tube is advanced to the skin to secure the tube. *D,* A standard Foley catheter may serve as a more specialized feeding tube replacement. The latex balloon may deflate or be weakened by gastric acid; so, this catheter is not ideal for long-term use. In addition, unless the Foley catheter is secured to the skin, it may migrate and the balloon may cause a pseudo obstruction.









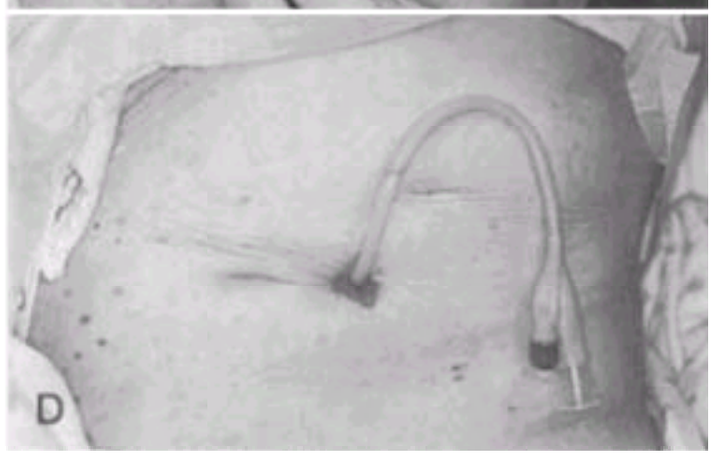


Figure 41-19 *A,* An endotracheal tube stylet used to distend the flange of the de Pezzer catheter. *B,* A lubricated wooden cotton-tipped swab can also be used as a makeshift stylet.

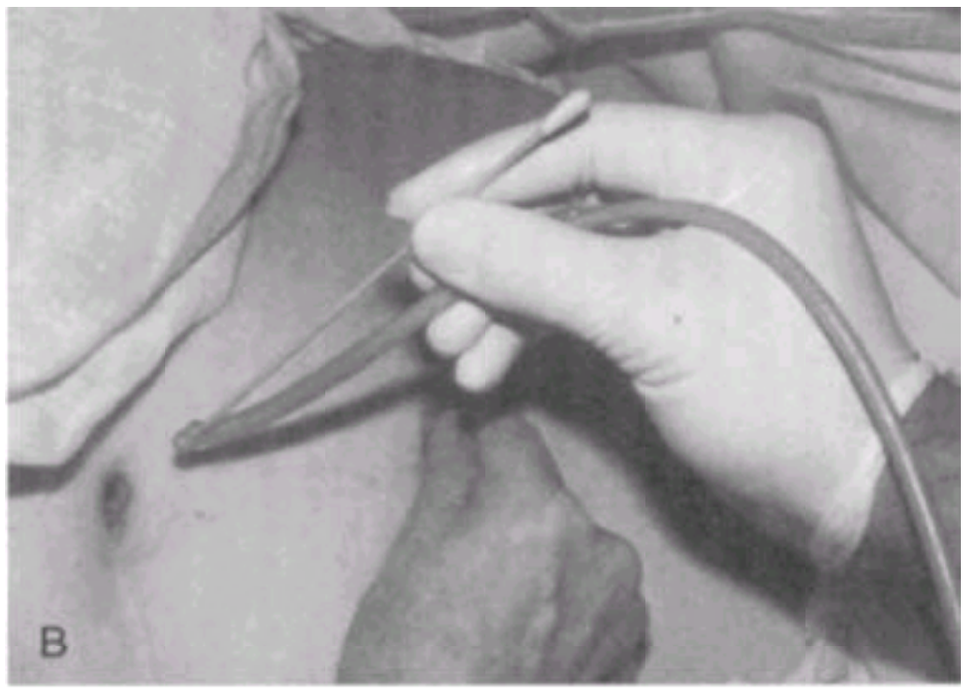


Figure 41-20 A modified pediatric chest tube inserter used to distend the flange of the de Pezzer catheter. The sharpened end of the inserter (trocar) has been rounded.

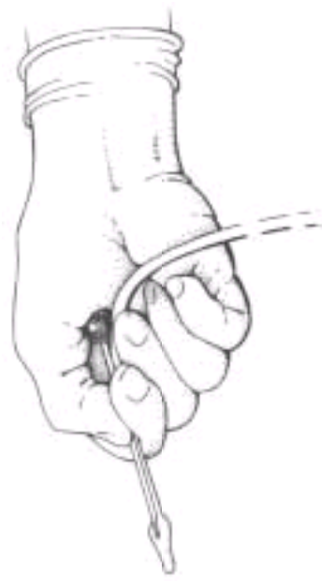


Figure 41-21 Gentle, firm traction *(A),* using the flat part of the opposite hand for countertraction, may remove most percutaneous endoscopic gastrostomy tubes, even those with internal mushroom bumpers *(B)*.

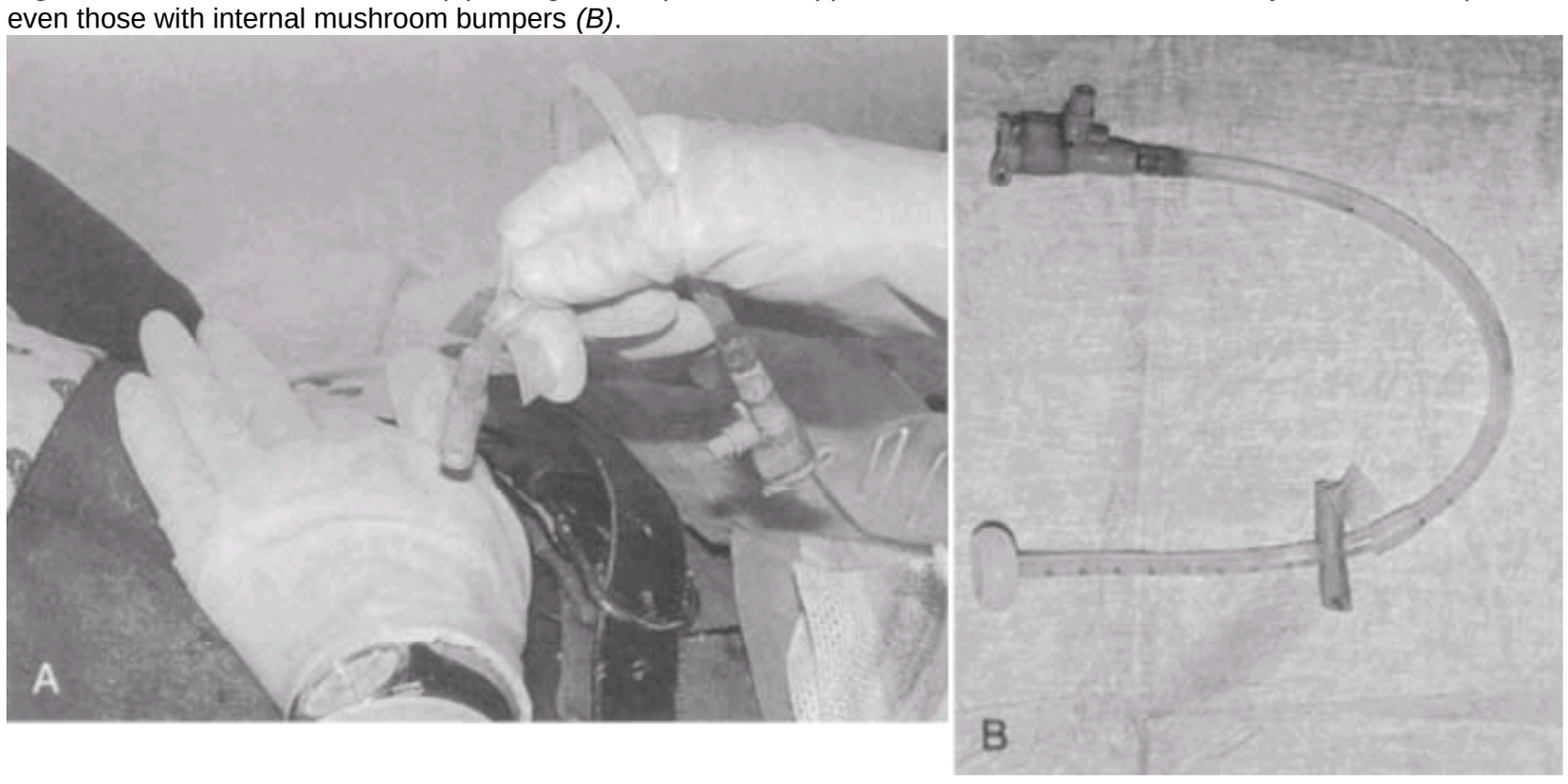


Figure 41-22 The tract of a feeding tube may close or become narrowed within a few hours after removal. When replacing a tube that has been removed, gentle probing of the tract of the previously placed feeding tube gives the clinician an idea of the patency and direction of the tract. In this case, a sterile cotton swab is gently advanced, being careful not to produce a false tract.

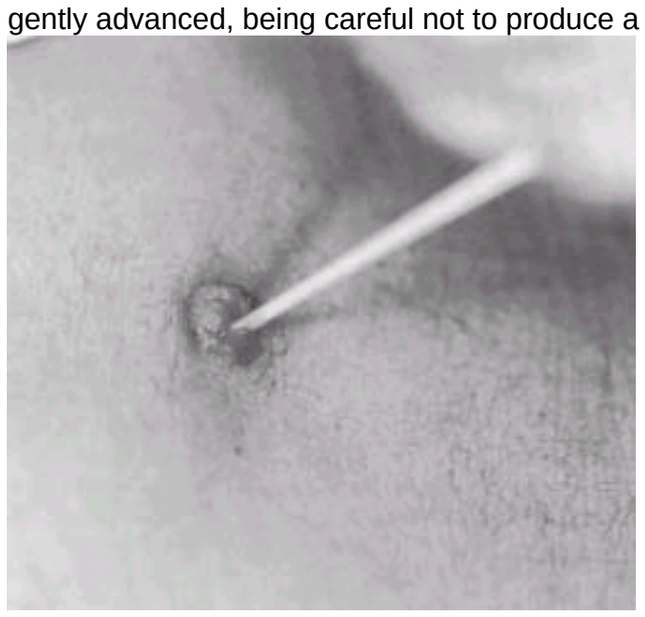


Figure 41-23 *A,* A 3-cm segment of thick latex tubing is cut from the proximal segment of a catheter. This segment is used to make an external bolster for a feeding tube and anchors the feeding tube, preventing unwanted ingress of the tube into the patient. *B,* A 3-cm segment of latex tubing is bent in half and cut to create a hole on each side of the segment.

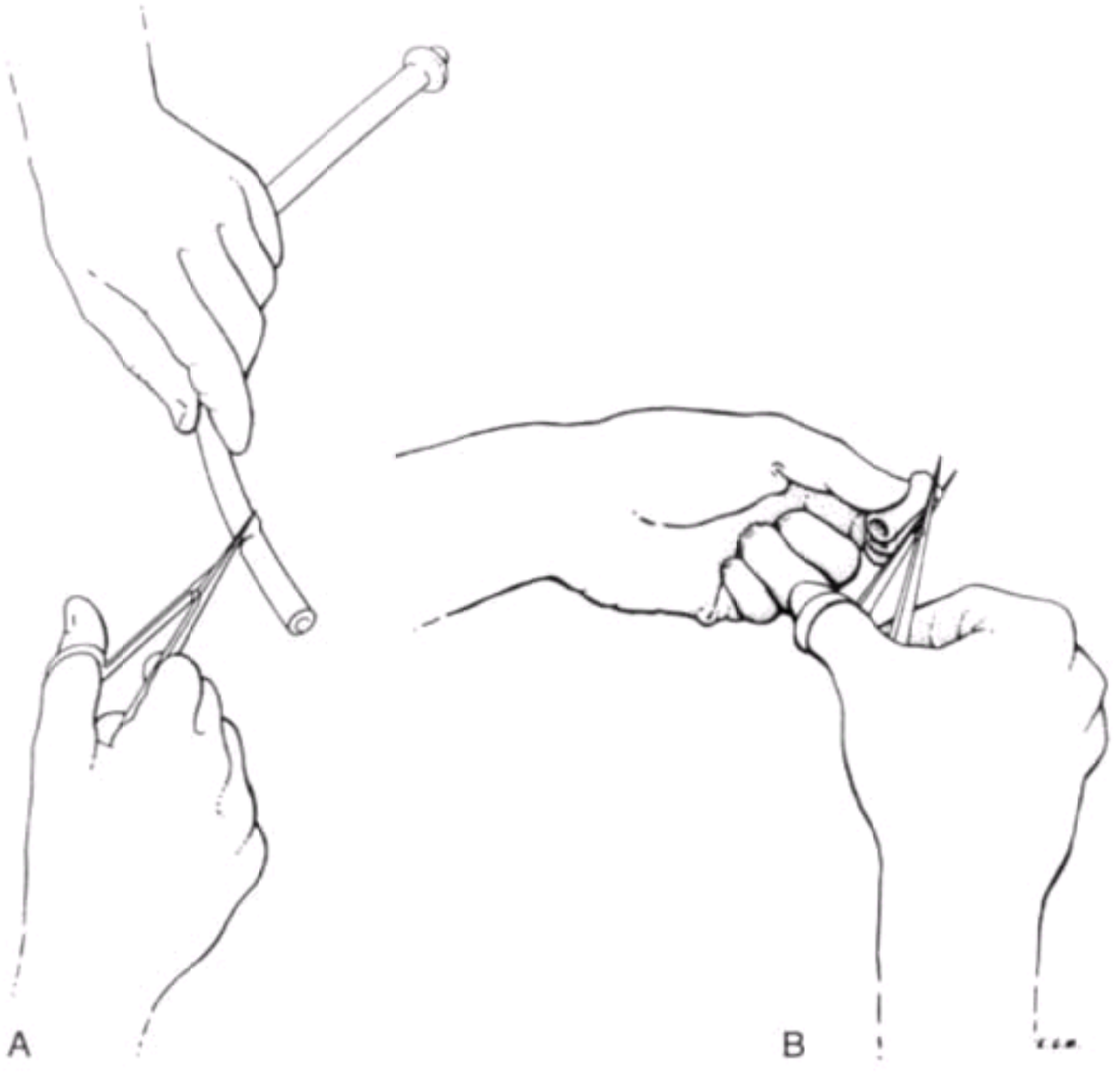


Figure 41-24 A hemostat is inserted through the holes in the completed bolster and grasps the feeding tube. The end of the feeding tube has been folded to reduce its external diameter.

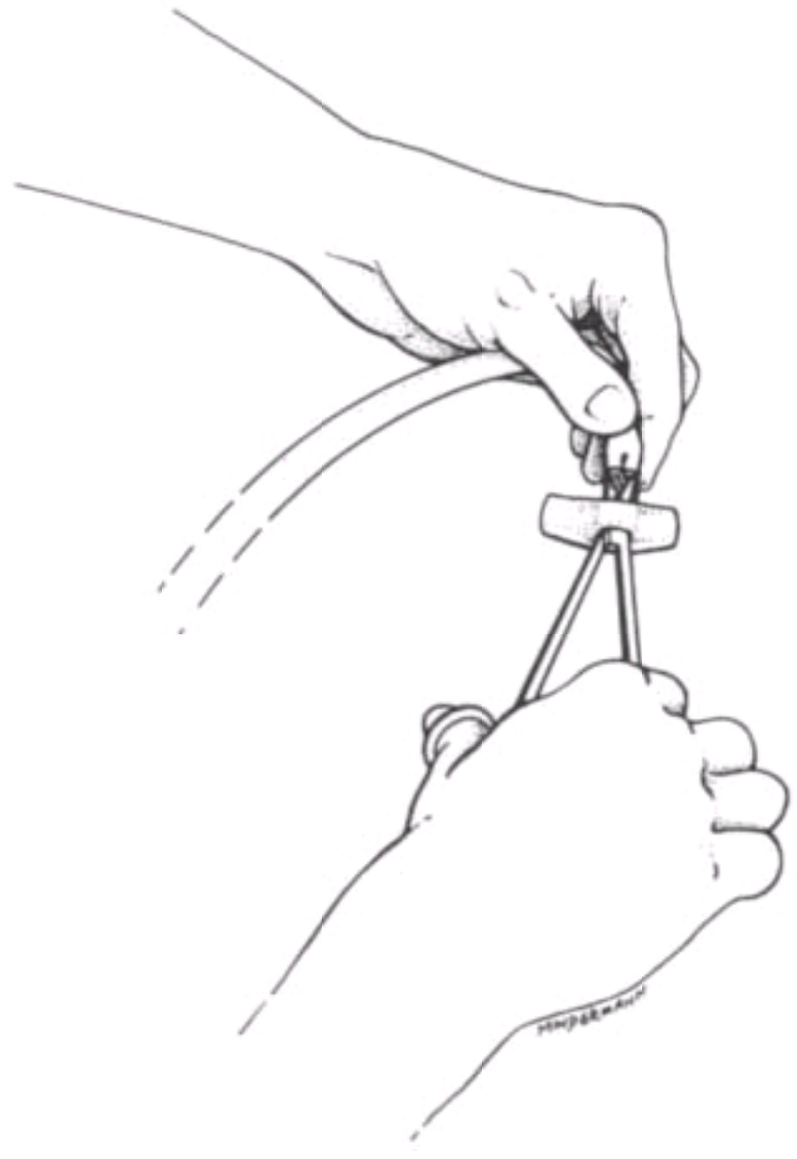


Figure 41-25 The feeding tube is pulled through the bolster. The bolster is advanced to 1 cm above the skin of the external abdomen.

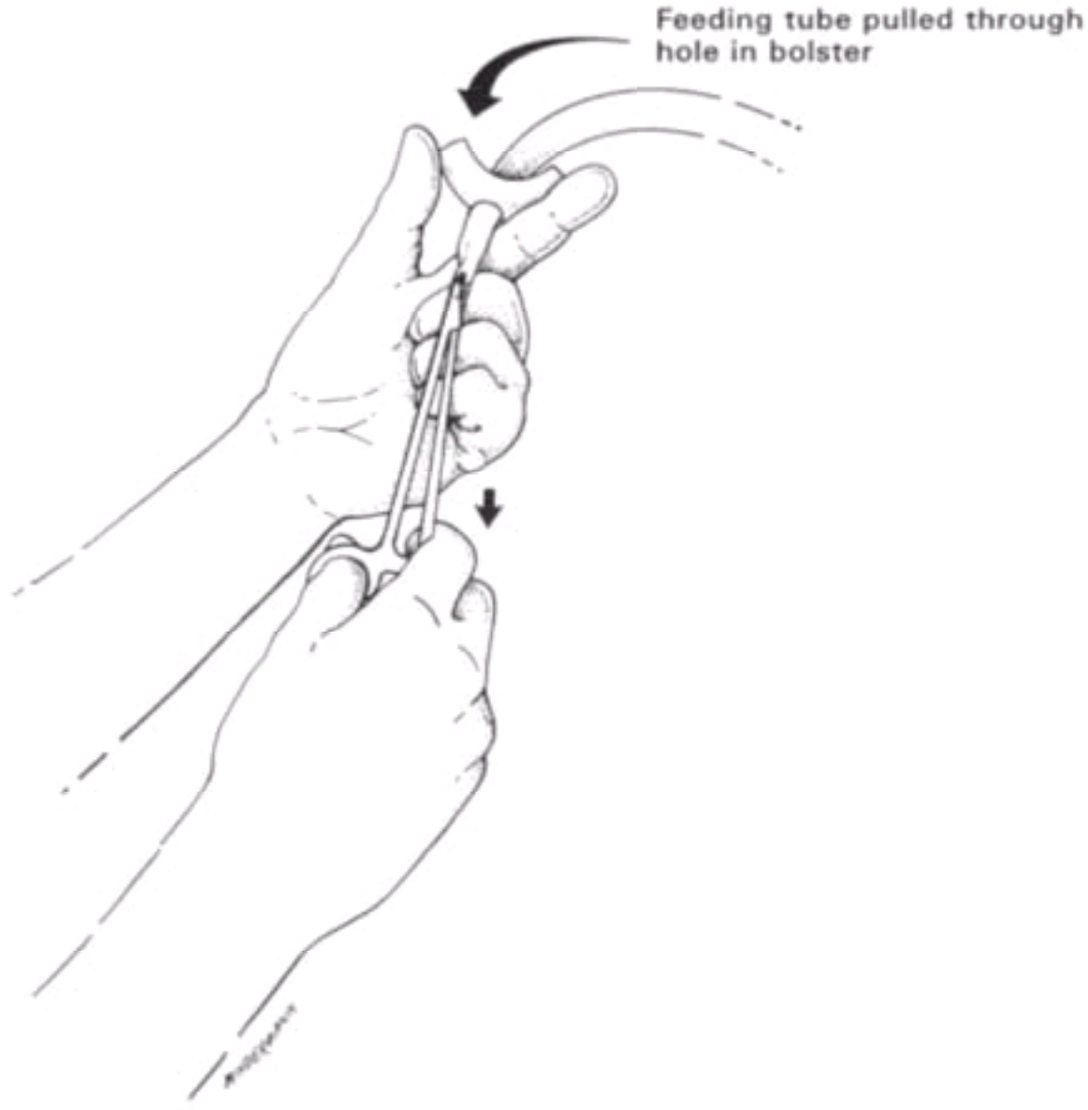


Figure 41-26 A Foley catheter can always be used as a temporary feeding tube replacement *(A)*. The thin-walled Foley balloon usually only remains inflated for a month or so, and a specialized feeding tube, such as the CORPAK (see Fig. 41-18) is preferred for long-term use. A bolster can be made to prevent inward migration of the tube. In this case the previously removed Foley catheter was used to make the bolster for the new one *(B)*.

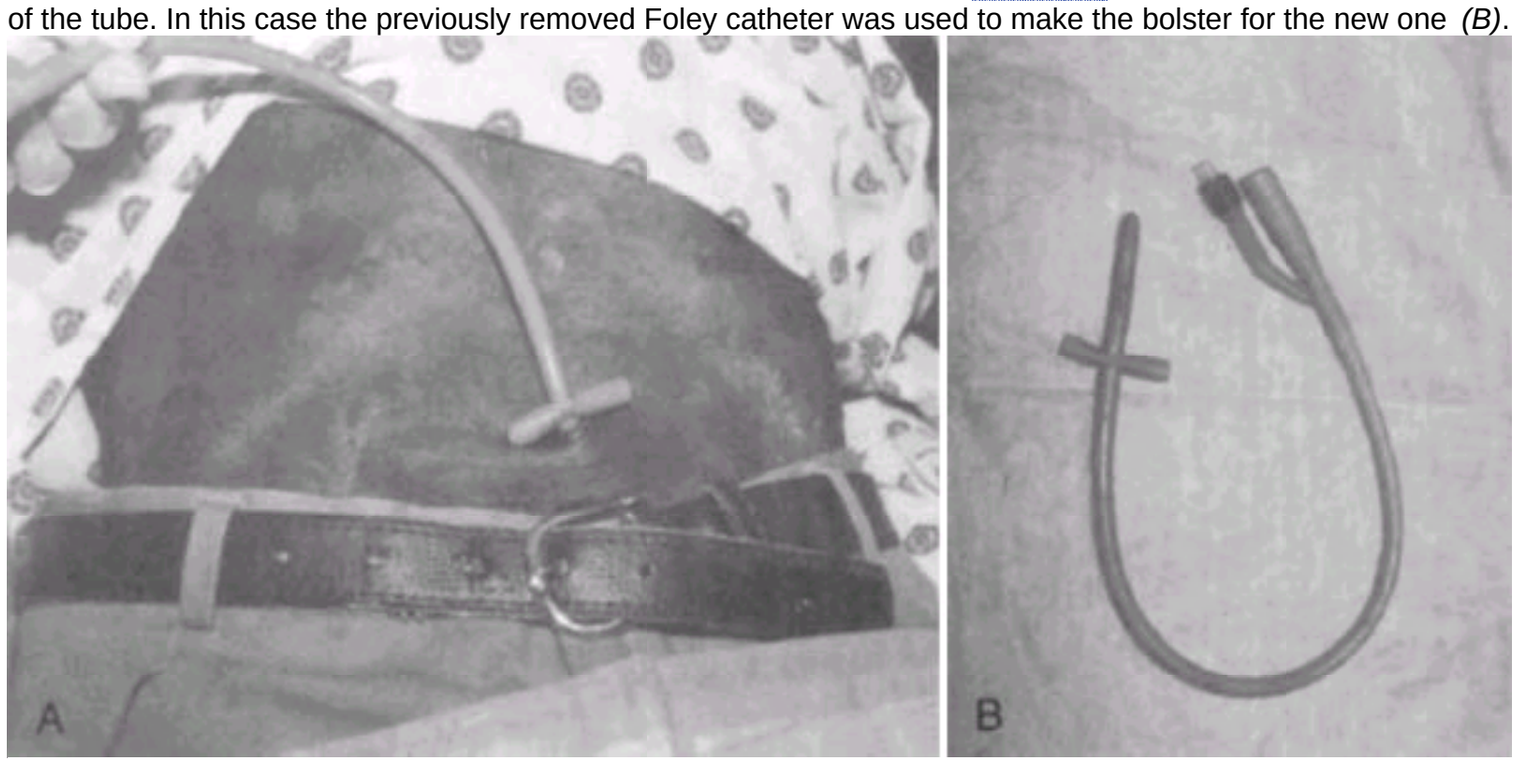


Figure 41-27 Contrast study demonstrating correct placement of a feeding tube. Note the outline of the gastric rugae and the characteristic mucosal folds of the small intestine.

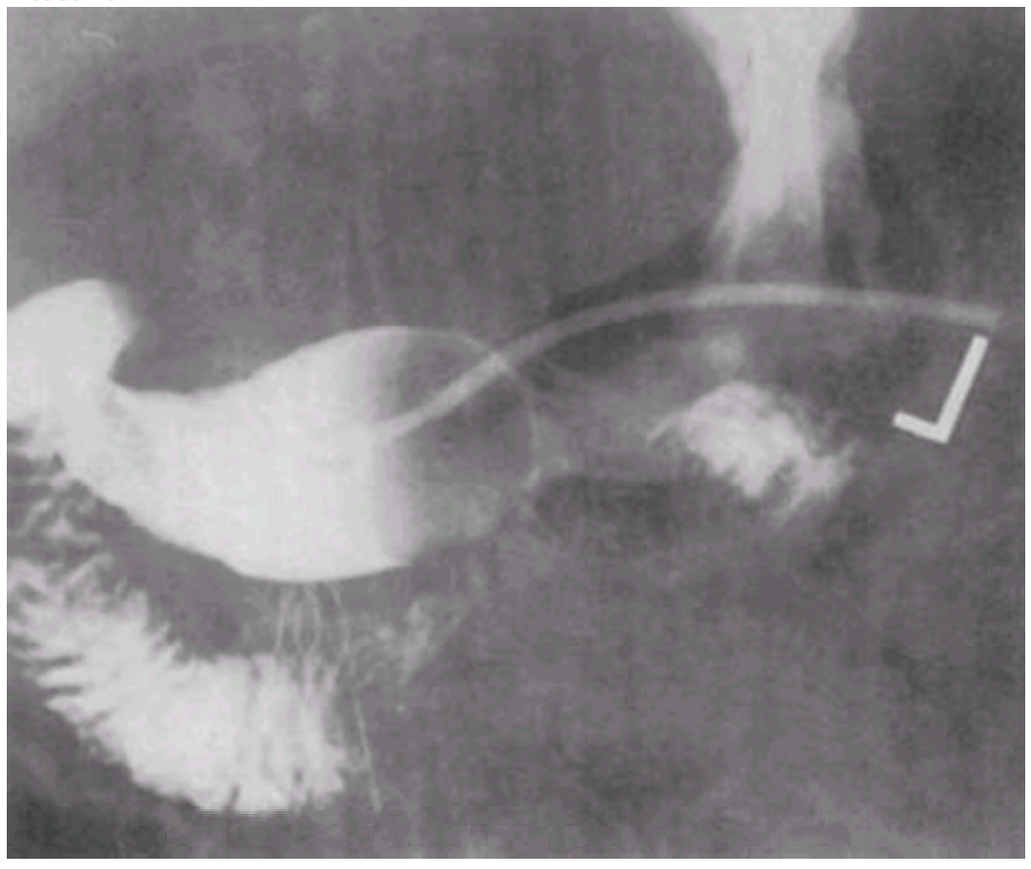


Figure 41-28 Although this feeding tube seemed to be easily replaced in an uncommunicative nursing home patient, gastric contents could not be aspirated; therefore, a contrast study was performed. Note the free flow of contrast material throughout the abdomen, especially outlining the liver *(arrows)*. This film indicates placement of the feeding tube into the peritoneum. Placing food through this tube could be disastrous.

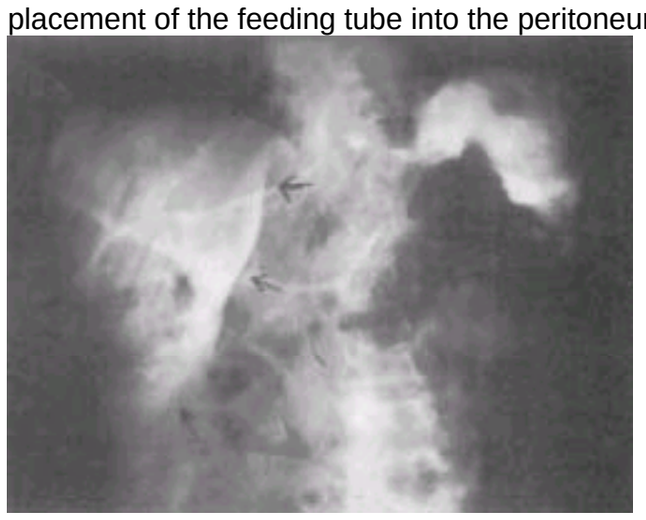
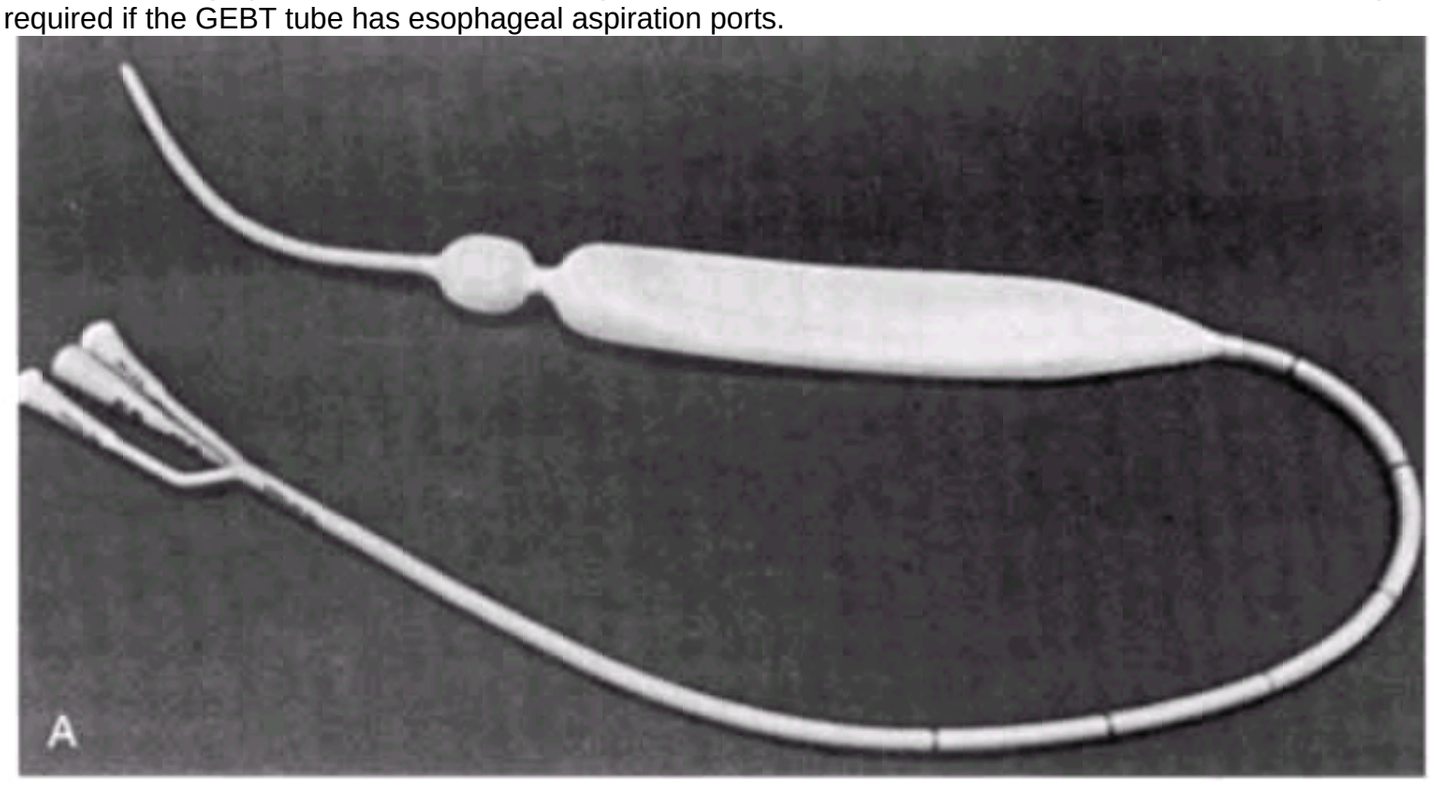


Figure 42-1 *A,* Photograph of the three-lumen Sengstaken-Blakemore tube with the balloons deflated. *B,* Diagram of GEBT tube with nasogastric tube attached. This is not required if the GEBT tube has esophageal aspiration ports.



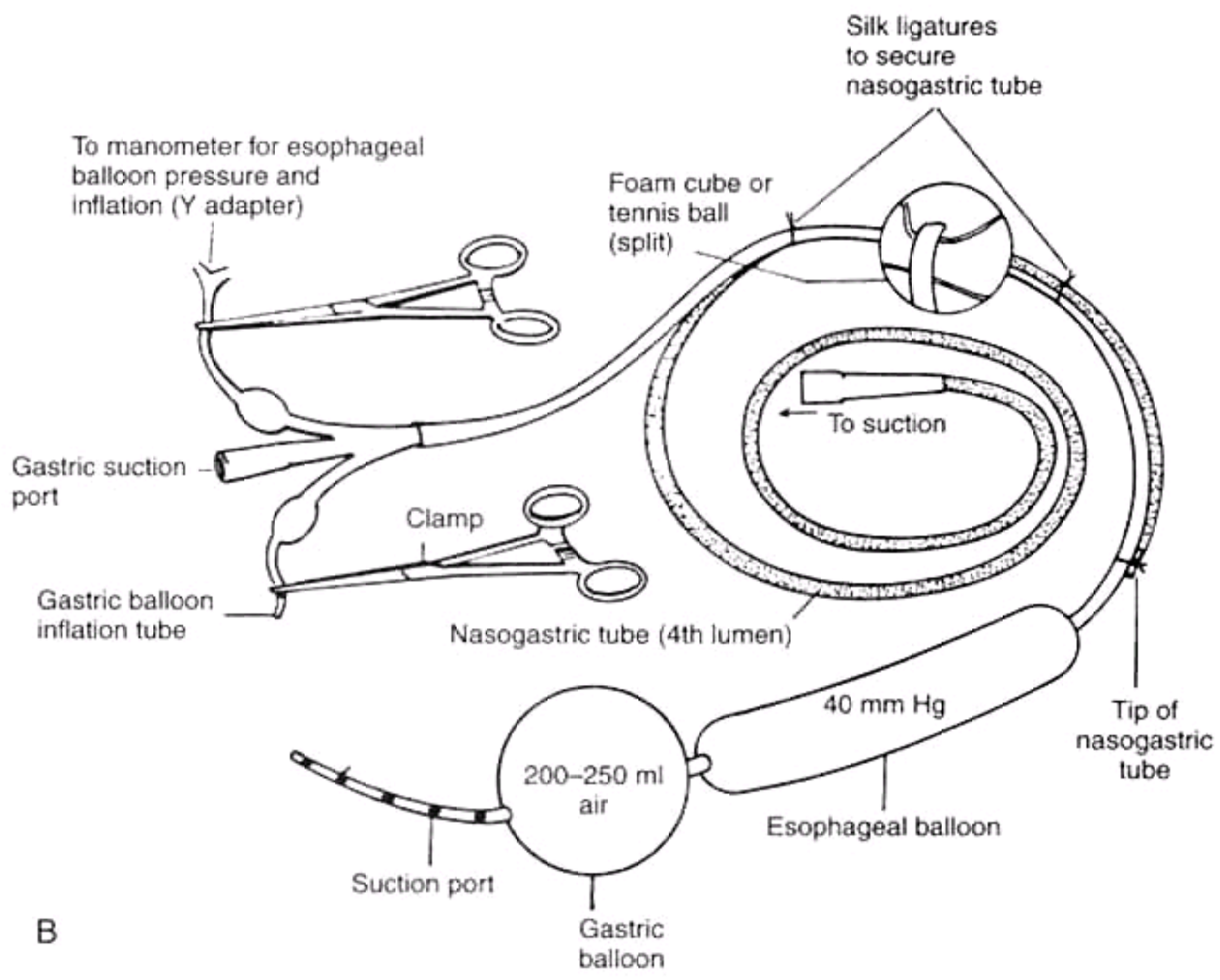


Figure 42-2 Diagrammatic representation of the four-lumen Minnesota tube with the balloons properly positioned and inflated. Note manometer attached to esophageal balloon port to measure pressures foam rubber cuff at nose as a fixation device, and the esophageal aspiration lumen.

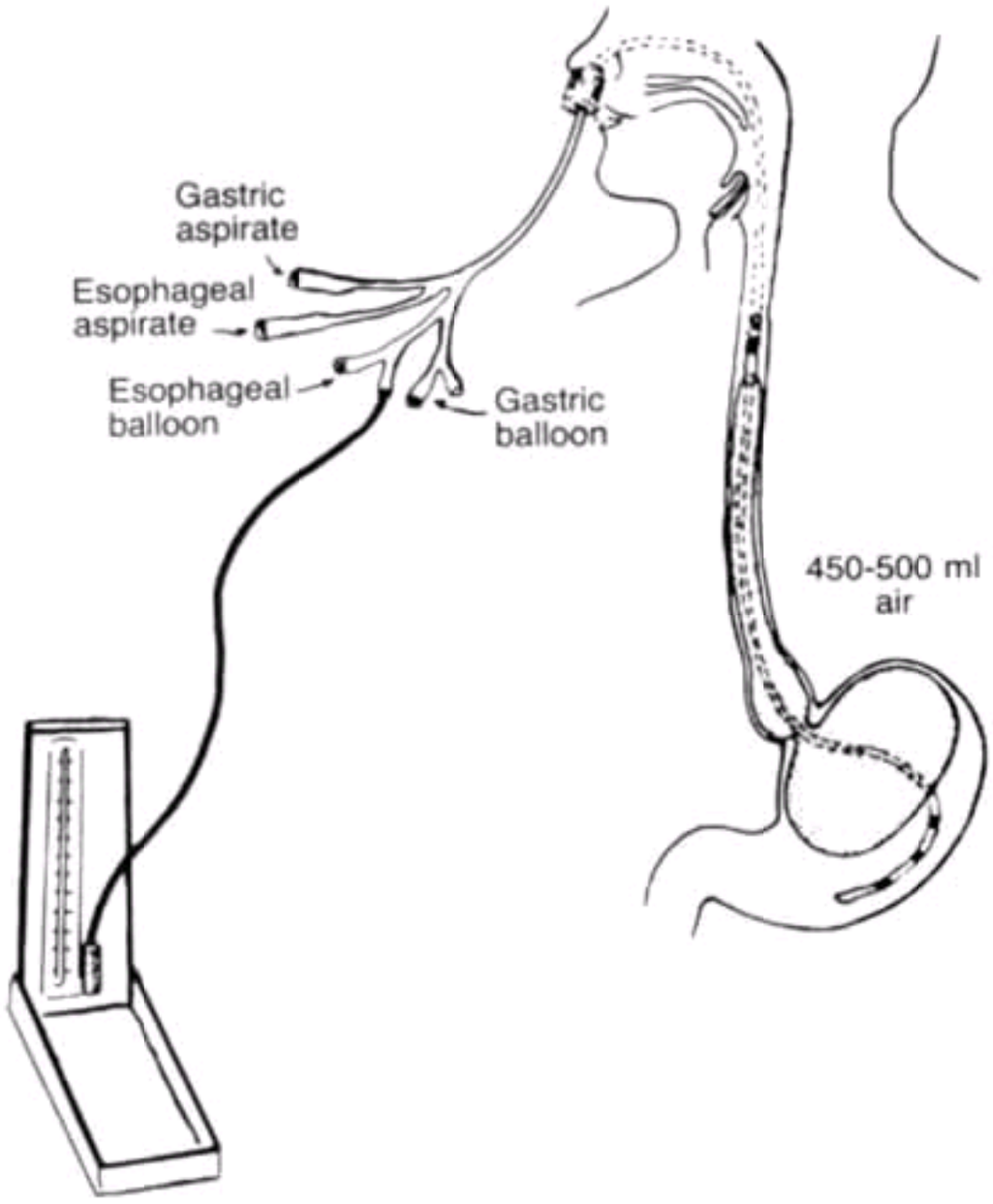


Figure 42-3 Algorithm for management of acute variceal hemorrhage, showing the role for balloon tamponade. After endoscopy banding and/or lauage have been used. Note that balloon tamponade can also be used after failed sclerotherapy as a temporizing measure pending emergency surgery, or between repeat attempts at sclerotherapy.

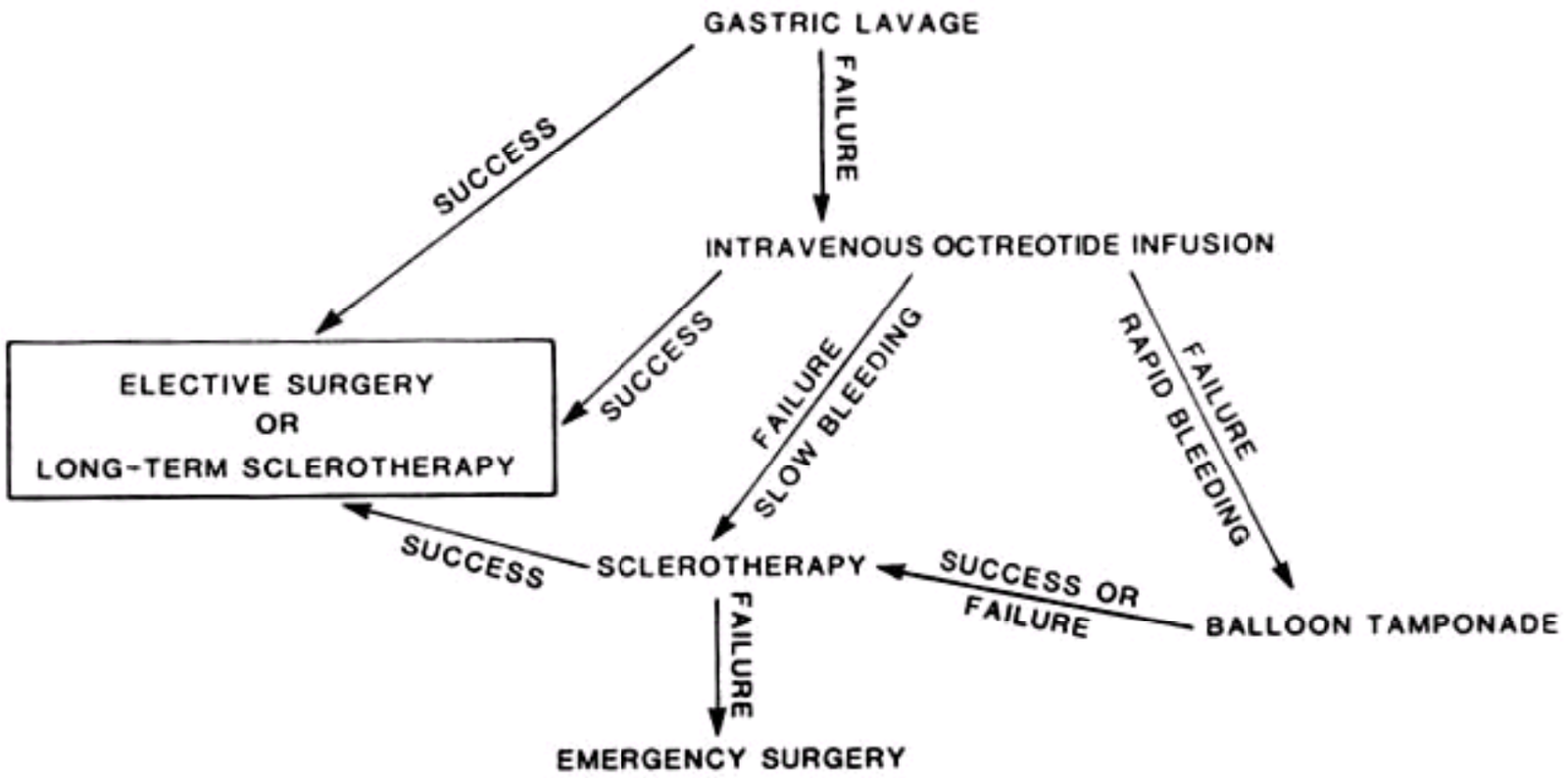


Figure 42-4 Esophageal balloon pressure measurement in the Sengstaken-Blakemore tube using a manometer. To inflate the esophageal balloon, the operator inserts one end of a Y connector to the esophageal balloon port, one end to a manometer, and one end to a bulb. At this point, no clamps are closed. The bulb is compressed until the manometer reads the desired pressure, and then the esophageal balloon port is clamped. Periodic readings to check the esophageal balloon pressure are subsequently taken by clamping the bulb port and unclamping the esophageal balloon port. The pressure in the esophageal balloon may vary with respirations and spasms, but the baseline pressure should not exceed 45 mm Hg. Most bleeding is controlled with pressures well below the maximum safe pressure. Note that standard toothed clamps are to be avoided (see text).

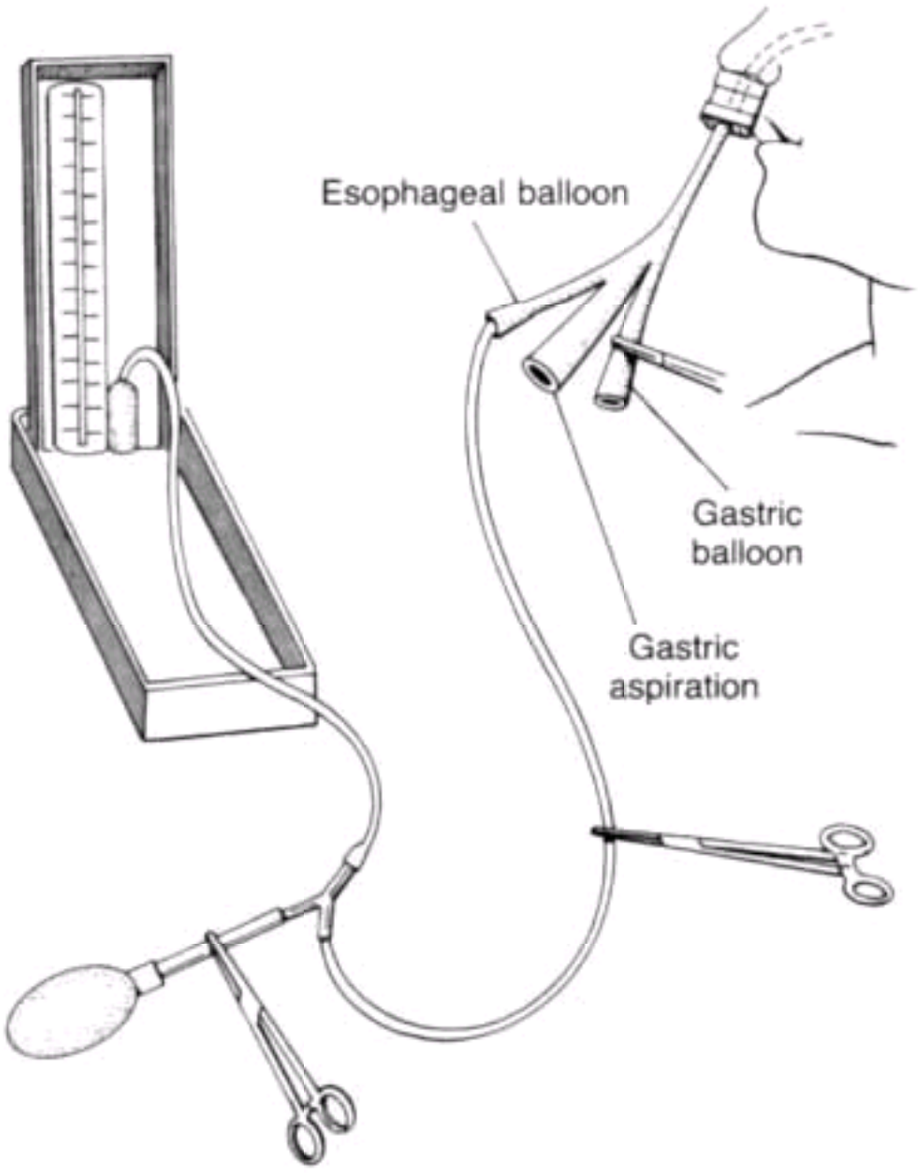
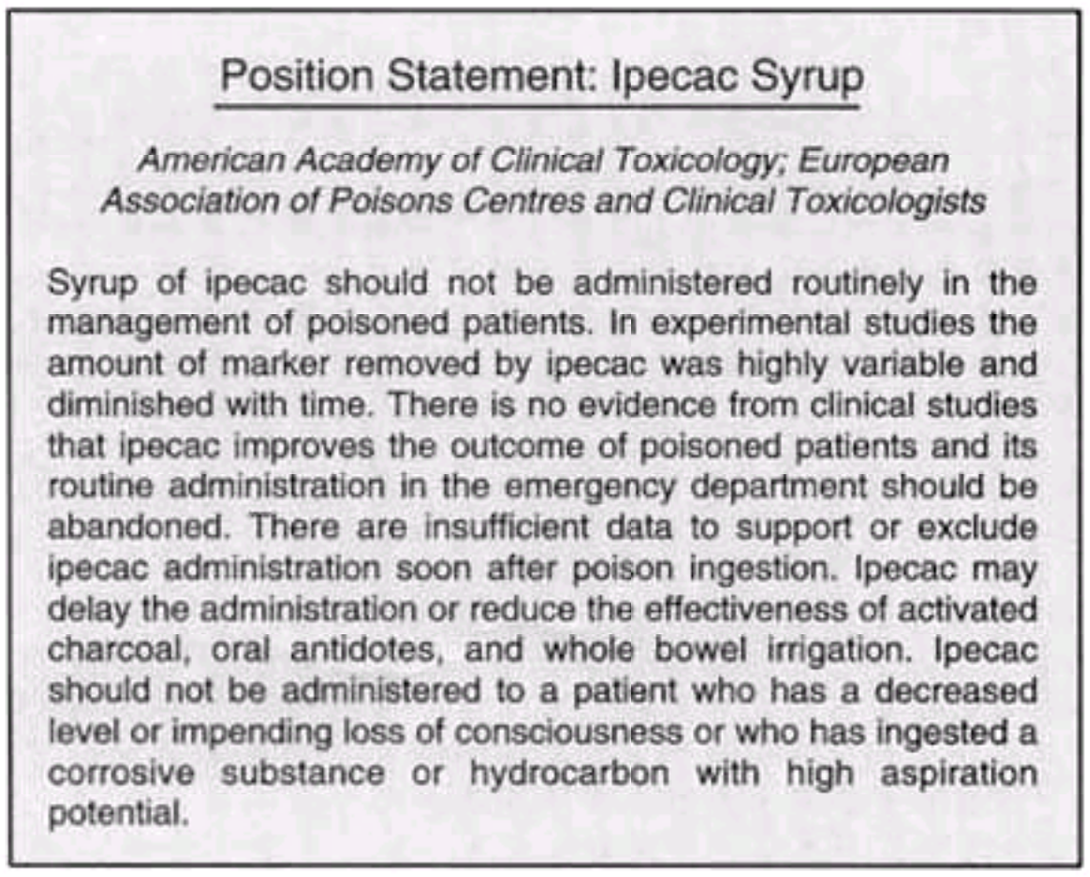


Figure 43-1 Position statement: ipecac syrup. *(From the American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Published in Clin Toxicol 35:699, 1997.)*

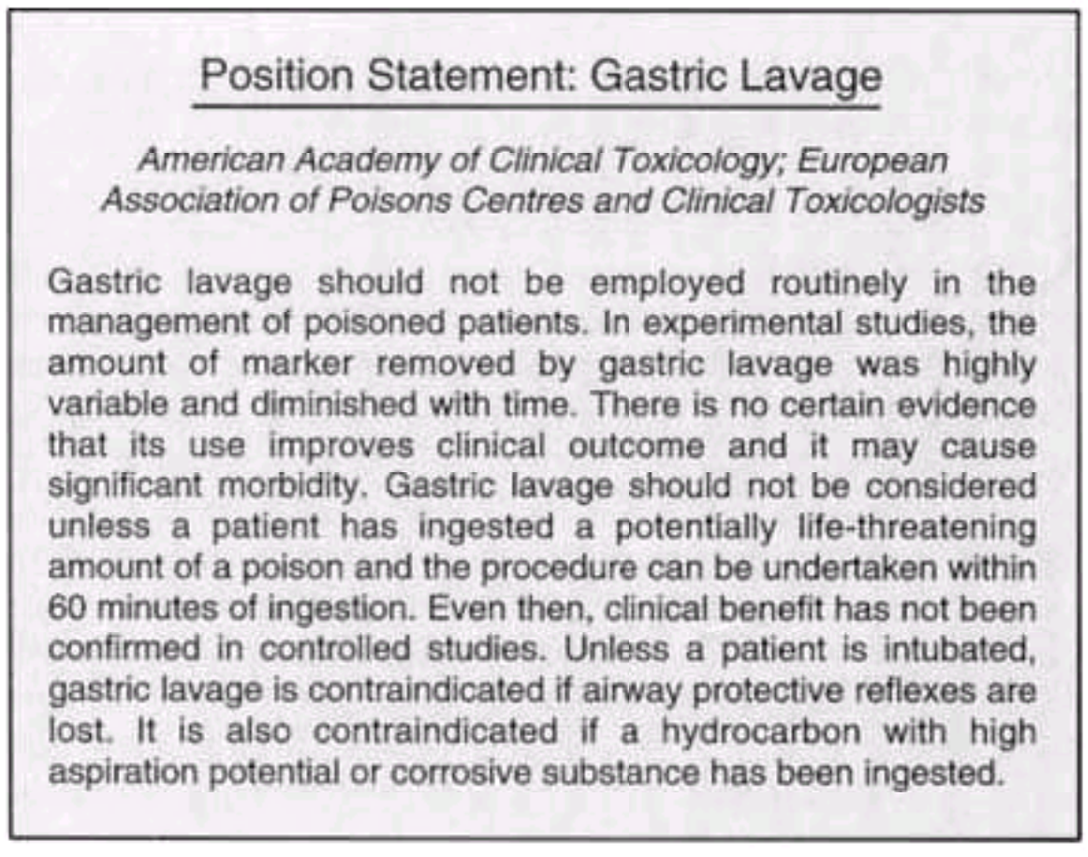


Position Statement: Ipecac Syrup

American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists

Syrup of ipecac should not be administered routinely in the management of poisoned patients. In experimental studies the amount of marker removed by ipecac was highly variable and diminished with time. There is no evidence from clinical studies that ipecac improves the outcome of poisoned patients and its routine administration in the emergency department should be abandoned. There are insufficient data to support or exclude ipecac administration soon after poison ingestion. Ipecac may delay the administration or reduce the effectiveness of activated charcoal, oral antidotes, and whole bowel irrigation. Ipecac should not be administered to a patient who has a decreased level or impending loss of consciousness or who has ingested a corrosive substance or hydrocarbon with high aspiration potential.

Figure 43-2 Position statement: gastric lavage. *(From the American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Published in Clin Toxicol 35:711, 1997.)*



Position Statement: Gastric Lavage

American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists

Gastric lavage should not be employed routinely in the management of poisoned patients. In experimental studies, the amount of marker removed by gastric lavage was highly variable and diminished with time. There is no certain evidence that its use improves clinical outcome and it may cause significant morbidity. Gastric lavage should not be considered unless a patient has ingested a potentially life-threatening amount of a poison and the procedure can be undertaken within 60 minutes of ingestion. Even then, clinical benefit has not been confirmed in controlled studies. Unless a patient is intubated, gastric lavage is contraindicated if airway protective reflexes are lost. It is also contraindicated if a hydrocarbon with high aspiration potential or corrosive substance has been ingested.

Figure 43-3 The effect of patient positioning on lavage. The left lateral decubitus position is preferred.

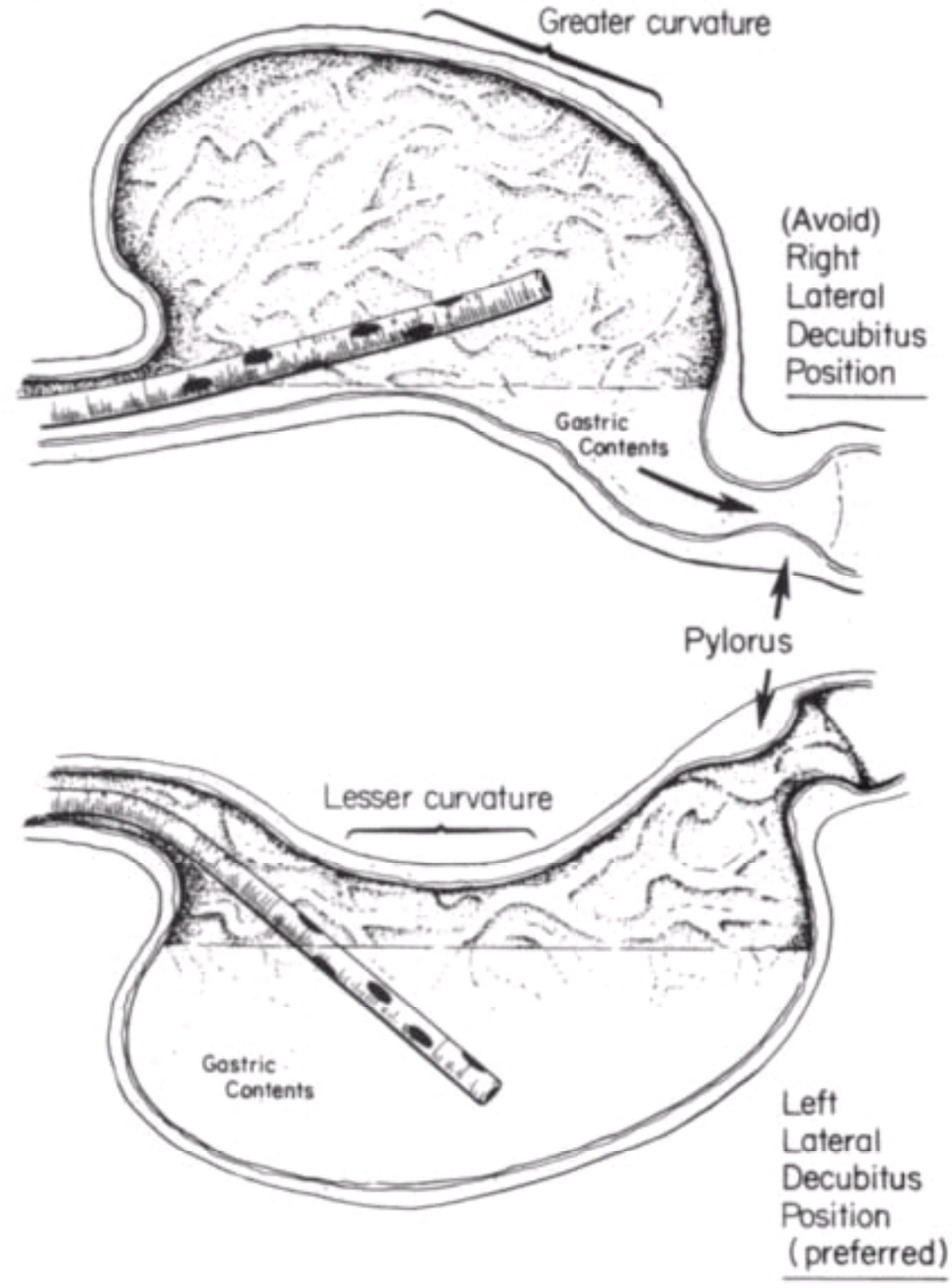


Figure 43-4 A large-diameter gastric tube. Note the extra side holes that have been cut near the tip. This is a theoretical advantage over a standard tube or a nasogastric tube. For small pill particles and liquids, a large-bore nasogastric tube may suffice.

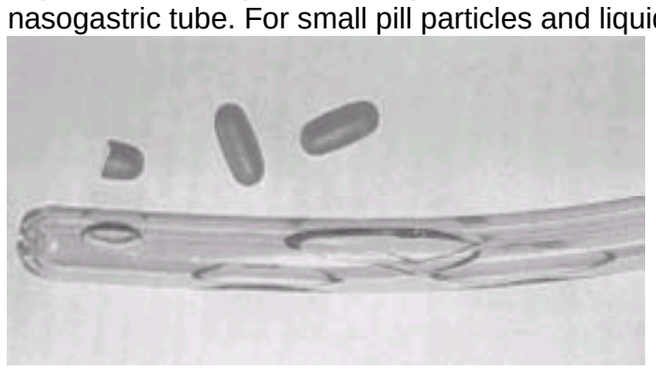


Figure 43-5 Gastric lavage in a child is always problematic. Obviously, an adult-sized large-bore oral gastric tube cannot be used, but a nasogastric (NG) tube may suffice. Some pediatric textbooks recommend a 24 Fr oral gastric tube for toddlers, and a 36 Fr tube for adolescents. In this case, a child was found with an open bottle of digoxin, and it could not be determined if ingestion had occurred. She would not drink charcoal. The 18 Fr NG tube was used to attempt to aspirate digoxin from the stomach (none was recovered) and to instill charcoal. Some would suggest the oral route for this tube, but it was passed rather easily through the nose. An NG tube is not ideal for some ingestants (iron, sustained-release products), but most pills quickly dissolve in the stomach and the small particles can easily be removed with an NG tube. Although lavage may have been reasonable in this scenario, a potent and safe antidote for digoxin does exist. The common routine practice of passing an NG tube in a child who is unwilling to drink charcoal is controversial and likely done far too often for benign ingestions. *(Reprinted with permission from Elsevier [The Lancet, vol 338 (8778), 1991, pp 1313–1315].)*

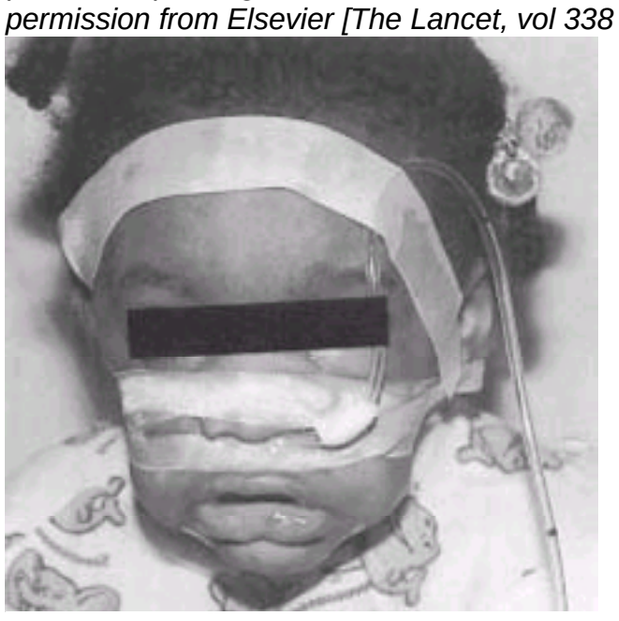


Figure 43-6 Failure to premeasure a lavage tube before passage is a common error. Here a piece of tape marks the depth of proper passage to ensure that the tip is in the stomach without excess tubing that may hinder fluid egress.

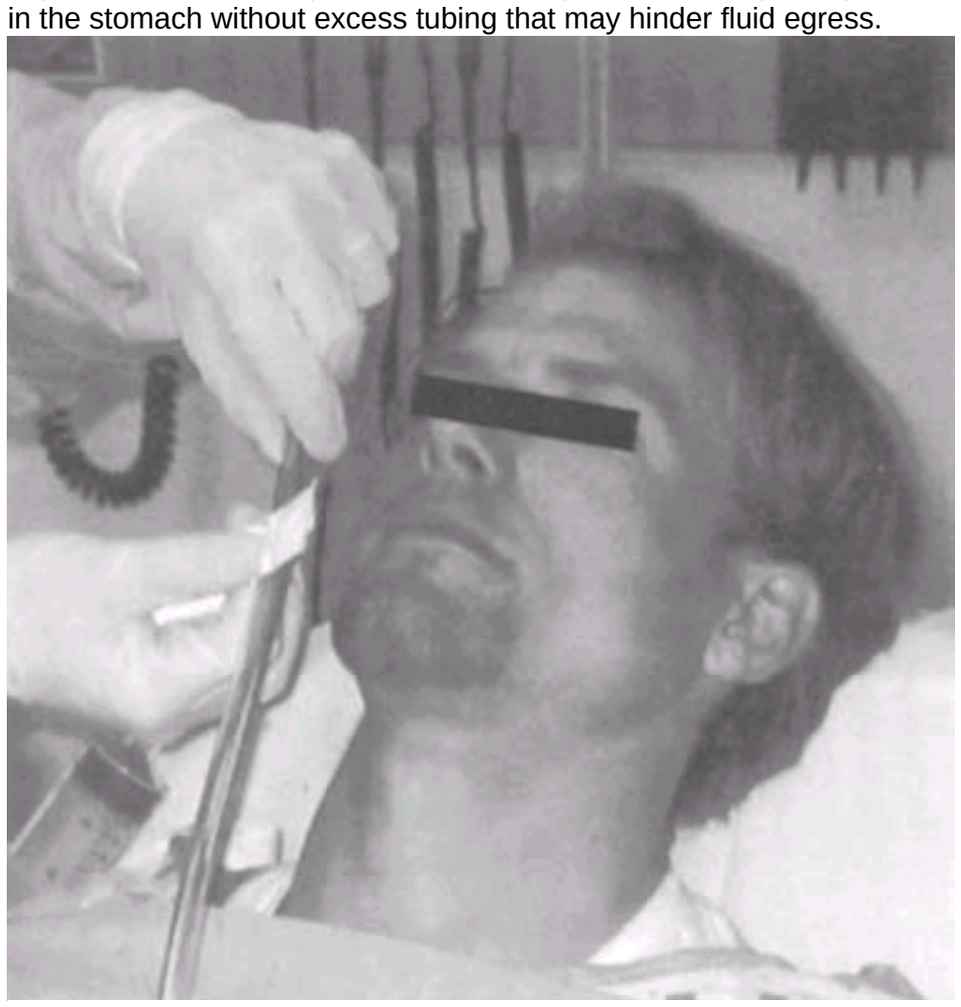


Figure 43-7 Positioning the patient's chin on the chest can facilitate passage of the tube into the esophagus once the pharynx has been entered. Once the tube is positioned, lavage is performed in the left lateral decubitus position. If the patient begins to vomit, the tube is immediately withdrawn.

Figure 43-8 An example of the Y connector closed system with the patient in a left lateral decubitus position. Patients on a ventilator, or those who are intubated may be lavaged in the supine position due to logistic issues, but an awake nonintubated patient is never lavaged in the restrained supine position.

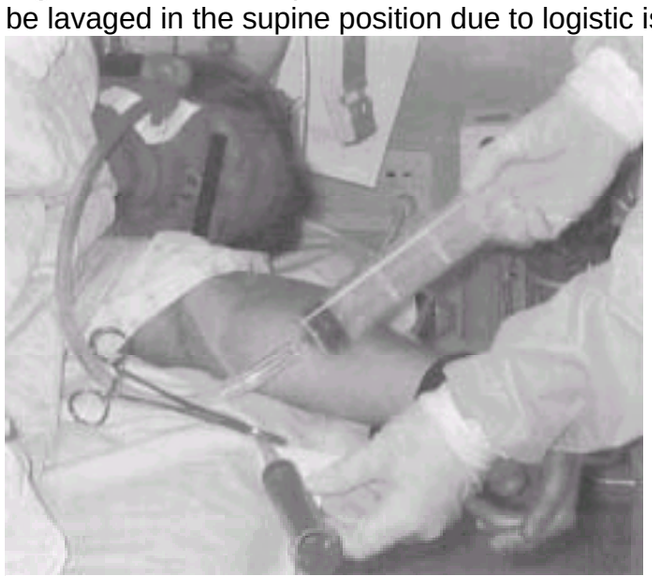
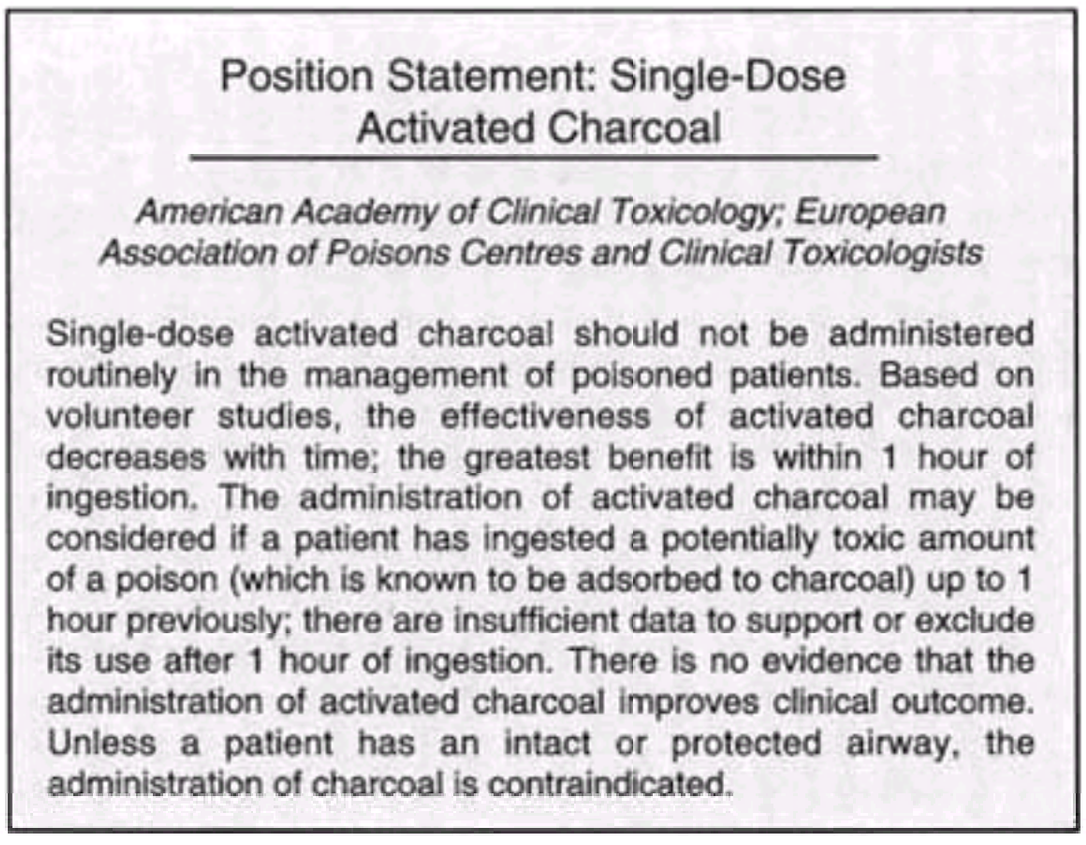


Figure 43-9 Position statement: single-dose activated charcoal. *(From the American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Published in Clin Toxicol 35:721, 1997.)*



Position Statement: Single-Dose Activated Charcoal

American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists

Single-dose activated charcoal should not be administered routinely in the management of poisoned patients. Based on volunteer studies, the effectiveness of activated charcoal decreases with time; the greatest benefit is within 1 hour of ingestion. The administration of activated charcoal may be considered if a patient has ingested a potentially toxic amount of a poison (which is known to be adsorbed to charcoal) up to 1 hour previously; there are insufficient data to support or exclude its use after 1 hour of ingestion. There is no evidence that the administration of activated charcoal improves clinical outcome. Unless a patient has an intact or protected airway, the administration of charcoal is contraindicated.

Figure 43-10 Radiographic confirmation of nasogastric tube placement before lavage or instillation of charcoal. Tracheal placement of a lavage tube is usually readily evident. Vomiting during lavage suggests that the tube has curved back into the esophagus. A confirmatory radiograph is suggested in the obtunded patient if gastric placement is questioned. Tracheal intubation precludes passage of a tube into the lungs, but it does not ensure proper gastric placement.

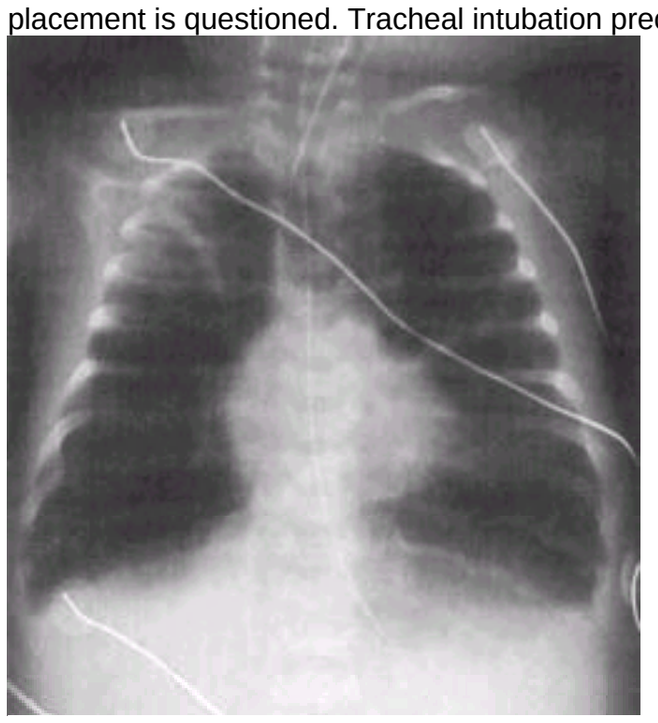


Figure 43-11 If the overdose patient will voluntarily drink charcoal, there are few reasons to withhold it, even though a definite clinical benefit in the routine case cannot be proved. If a patient will not drink charcoal, patient management becomes controversial. Passing a nasogastric tube in a struggling patient or in a recalcitrant child merely to instill the unproven, but theoretically useful, antidote is not supported by scientific data. Nonetheless, it remains a common procedure. Although not always easy or pleasant, such an intervention is usually safe. Pulmonary aspiration, even in the awake patient, is the major downside. *Restrained supine patients are at greatest risk for aspiration, and that position should be avoided, even in the initially awake patient.*

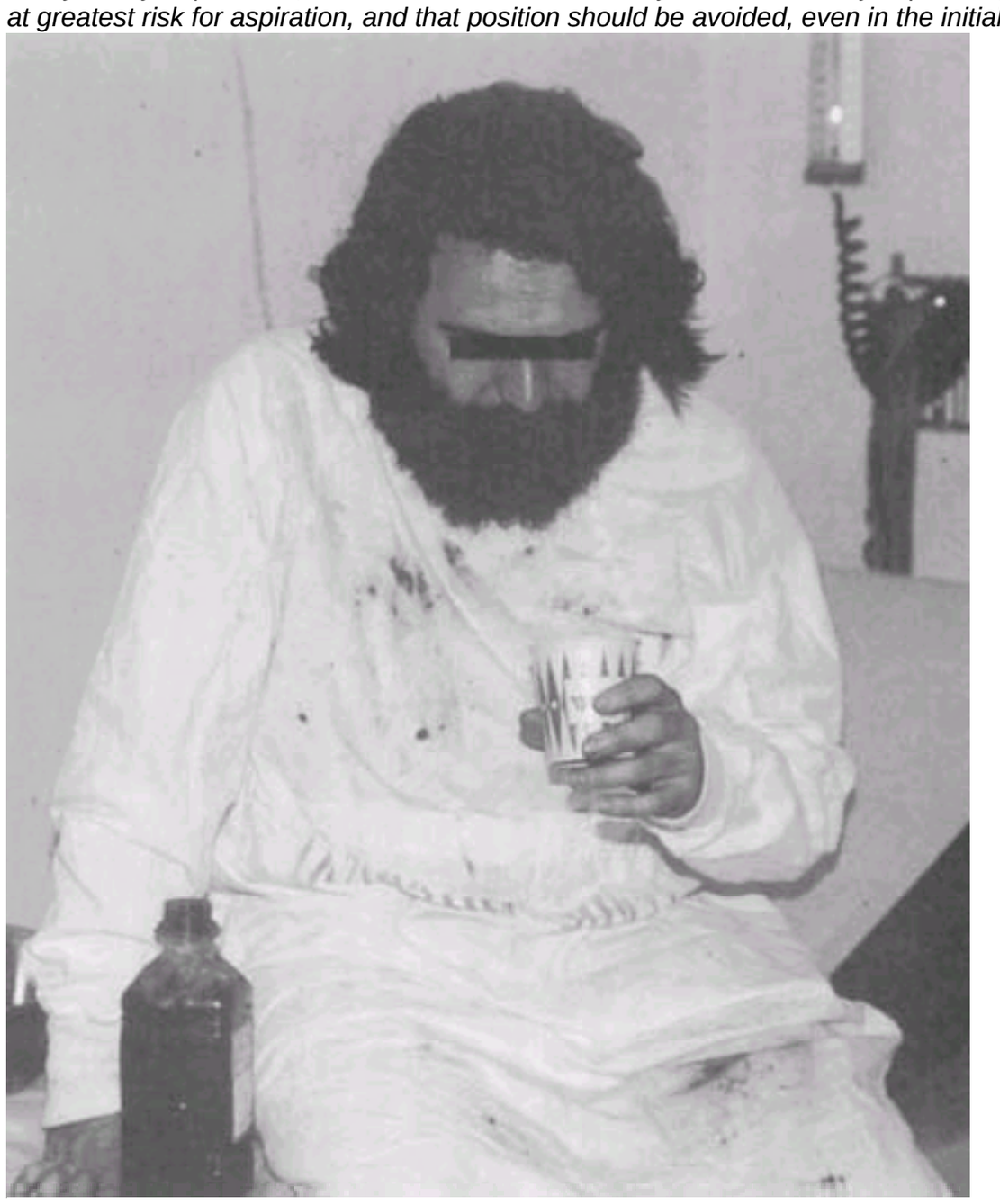


Figure 43-12 Charcoal that is voluntarily swallowed or instilled via an oral-gastric lavage tube or nasogastric tube can induce emesis. This occurs in both the obtunded and awake patient. In this instance, the patient was unconscious from the overdose and the airway was protected with prior tracheal intubation. Although the intubation procedure does not totally exclude pulmonary aspiration, and it carries some morbidity in its own right, it is recommended prior to charcoal use in the patient who is not able to fully protect the airway. Patients who initially are asymptomatic or minimally affected but have ingested drugs that have the potential to produce rapid deterioration, seizures, or loss of airway protection make decisions on the use of charcoal difficult for the clinician. In borderline cases, some experienced clinicians avoid the use of charcoal altogether.

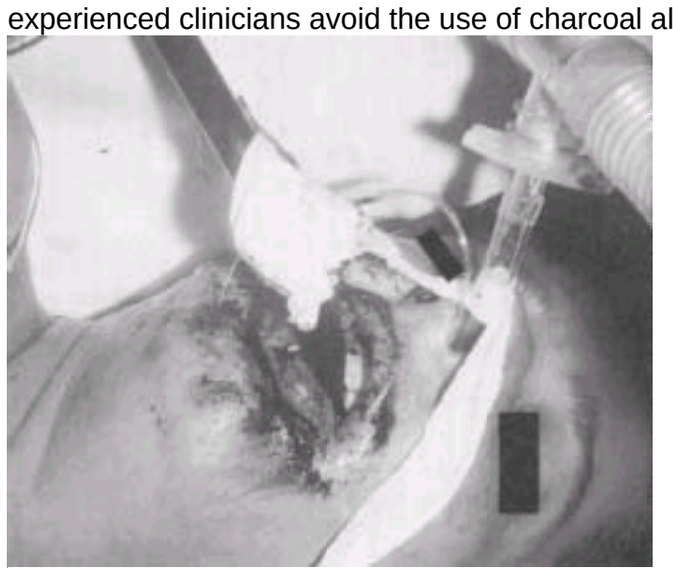


Figure 43-13 Position statement and practice guidelines on the use of multi-dose activated charcoal in the treatment of acute poisoning. *(From the American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Published in Clin Toxicol 37:731, 1999.)*

Position Statement: Position Statement and Practice Guidelines on the Use of Multi-Dose Activated Charcoal in the Treatment of Acute Poisoning

American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists

Although many studies in animals and volunteers have demonstrated that multiple-dose activated charcoal increases drug elimination significantly, this therapy has not yet been shown in a controlled study in poisoned patients to reduce morbidity and mortality. Further studies are required to establish its role and the optimal dosage regimen of charcoal to be administered.

Based on experimental and clinical studies, multiple-dose activated charcoal should be considered only if a patient has ingested a life-threatening amount of carbamazepine, dapsone, Phenobarbital, quinine, or theophylline. With all of these drugs there are data to confirm enhanced elimination, though no controlled studies have demonstrated clinical benefit.

Although volunteer studies have demonstrated that multiple-dose activated charcoal increases the elimination of amitriptyline, dextropropoxyphene, digitoxin, digoxin, disopyramide, nadolol, phenylbutazone, phenytoin, piroxicam, and sotalol, there are insufficient clinical data to support or exclude the use of this therapy.

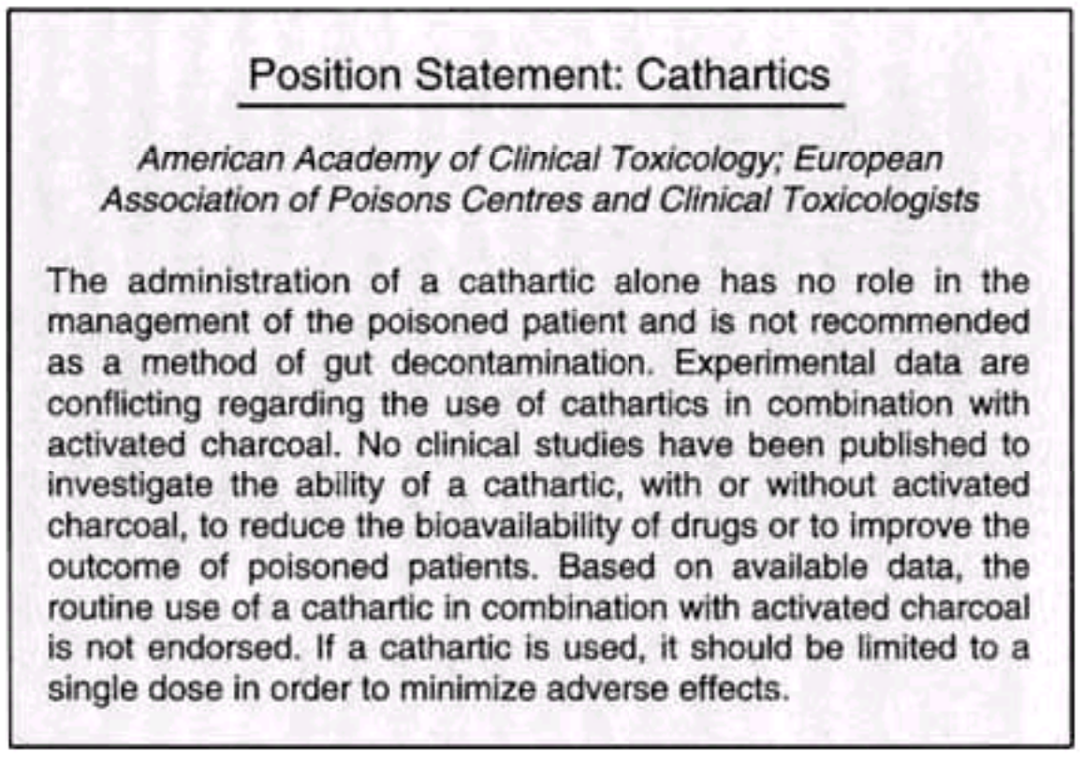
The use of multiple-dose charcoal in salicylate poisoning is controversial. One animal study and 2 of 4 volunteer studies did not demonstrate increased salicylate clearance with multiple-dose charcoal therapy. Data in poisoned patients are insufficient presently to recommend the use of multiple-dose charcoal therapy for salicylate poisoning.

Multiple-dose activated charcoal did not increase the elimination of astemizole, chlorpropamide, doxepin, imipramine, meprobamate, methotrexate, phenytoin, sodium valproate, tobramycin, and vancomycin in experimental and/or clinical studies.

Unless a patient has an intact or protected airway, the administration of multiple-dose activated charcoal is contraindicated. It should not be used in the presence of an intestinal obstruction. The need for concurrent administration of cathartics remains unproven and is not recommended. In particular, cathartics should not be administered to young children because of the propensity of laxatives to cause fluid and electrolyte imbalance.

In conclusion, based on experimental and clinical studies, multiple-dose activated charcoal should be considered only if a patient has ingested a life-threatening amount of carbamazepine, dapsone, Phenobarbital, quinine, or theophylline.

Figure 43-14 Position statement: cathartics. *(From the American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Published in Clin Toxicol 35:743, 1999.)*



Position Statement: Cathartics

American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists

The administration of a cathartic alone has no role in the management of the poisoned patient and is not recommended as a method of gut decontamination. Experimental data are conflicting regarding the use of cathartics in combination with activated charcoal. No clinical studies have been published to investigate the ability of a cathartic, with or without activated charcoal, to reduce the bioavailability of drugs or to improve the outcome of poisoned patients. Based on available data, the routine use of a cathartic in combination with activated charcoal is not endorsed. If a cathartic is used, it should be limited to a single dose in order to minimize adverse effects.

Figure 43-15 This "body packer" *(A)* attempted to smuggle more than 50 packets of heroin. All packets were passed intact after 12 hours of whole-bowel irrigation. Note the integrity of the carefully wrapped packets that were passed *(B)*.

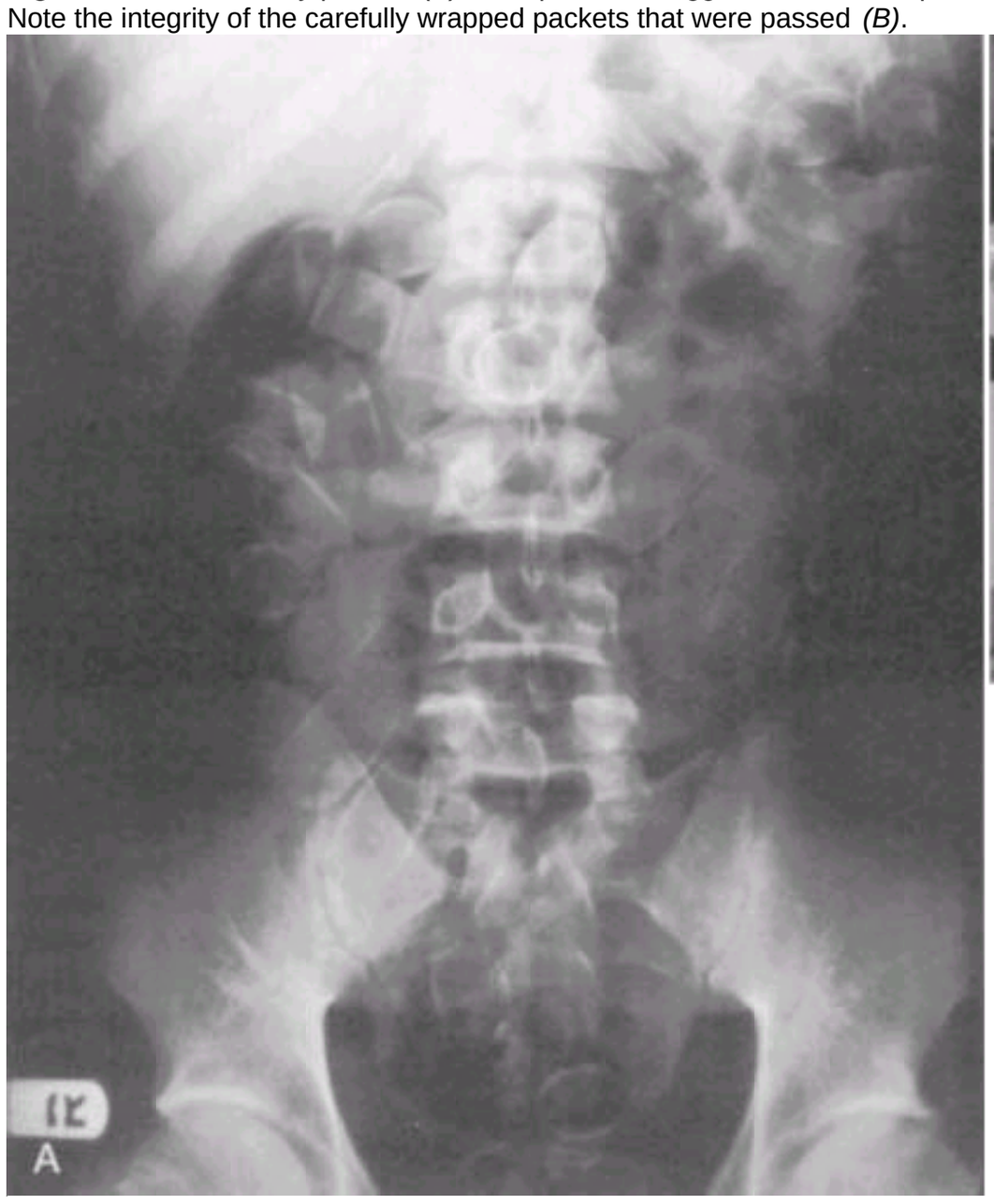
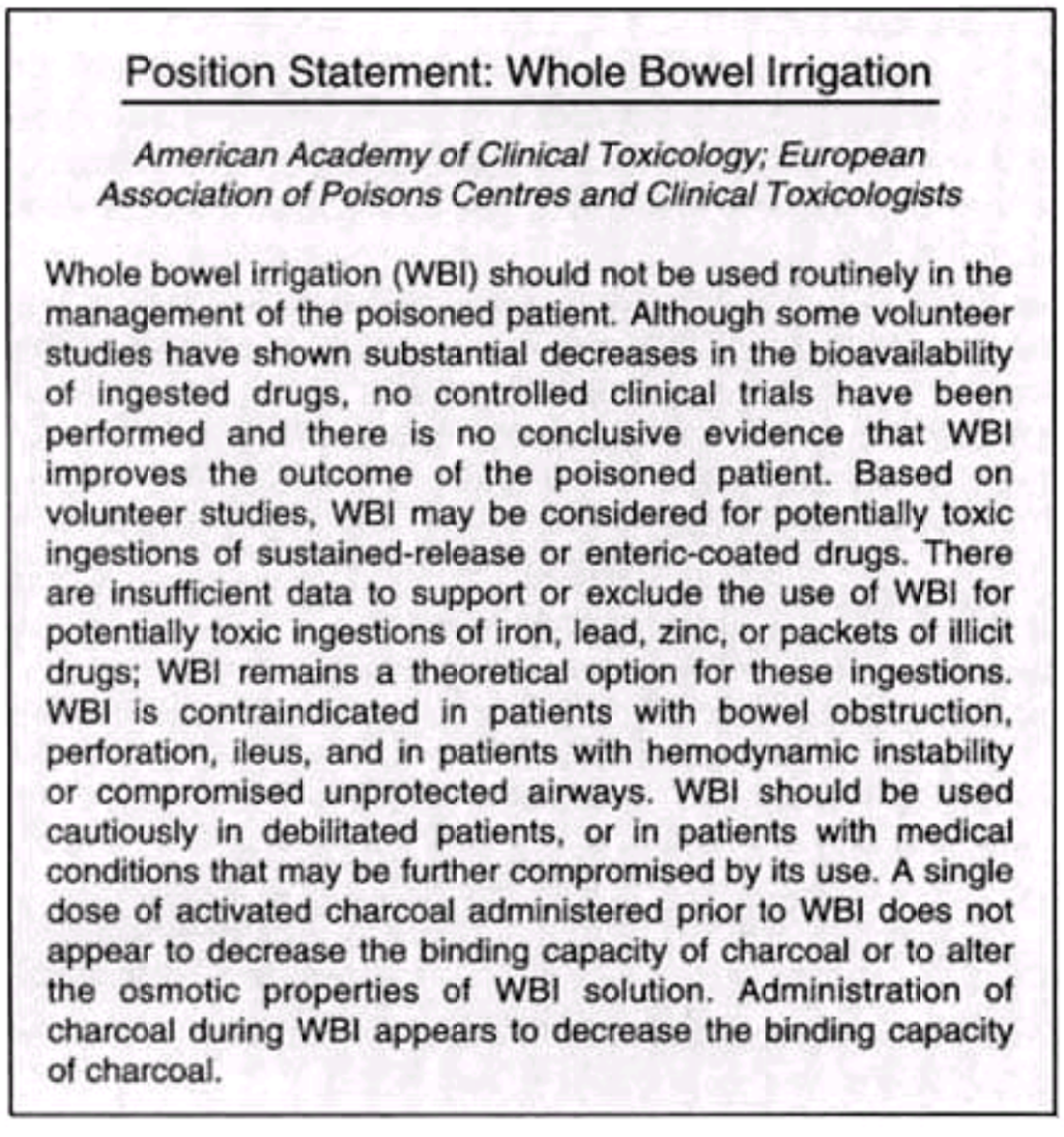


Figure 43-16 Position statement: whole-bowel irrigation. *(From the American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Published in Clin Toxicol 35:753, 1997.)*



Position Statement: Whole Bowel Irrigation

American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists

Whole bowel irrigation (WBI) should not be used routinely in the management of the poisoned patient. Although some volunteer studies have shown substantial decreases in the bioavailability of ingested drugs, no controlled clinical trials have been performed and there is no conclusive evidence that WBI improves the outcome of the poisoned patient. Based on volunteer studies, WBI may be considered for potentially toxic ingestions of sustained-release or enteric-coated drugs. There are insufficient data to support or exclude the use of WBI for potentially toxic ingestions of iron, lead, zinc, or packets of illicit drugs; WBI remains a theoretical option for these ingestions. WBI is contraindicated in patients with bowel obstruction, perforation, ileus, and in patients with hemodynamic instability or compromised unprotected airways. WBI should be used cautiously in debilitated patients, or in patients with medical conditions that may be further compromised by its use. A single dose of activated charcoal administered prior to WBI does not appear to decrease the binding capacity of charcoal or to alter the osmotic properties of WBI solution. Administration of charcoal during WBI appears to decrease the binding capacity of charcoal.

Figure 43-17 Whole-bowel irrigation is commonly recommended for the treatment of iron ingestion. These radiographs depict the effect of 5 hours of whole-bowel irrigation. Note the marked decrease of radiopaque pills in the gastrointestinal tract. Intact pills were recovered in the rectal effluent.

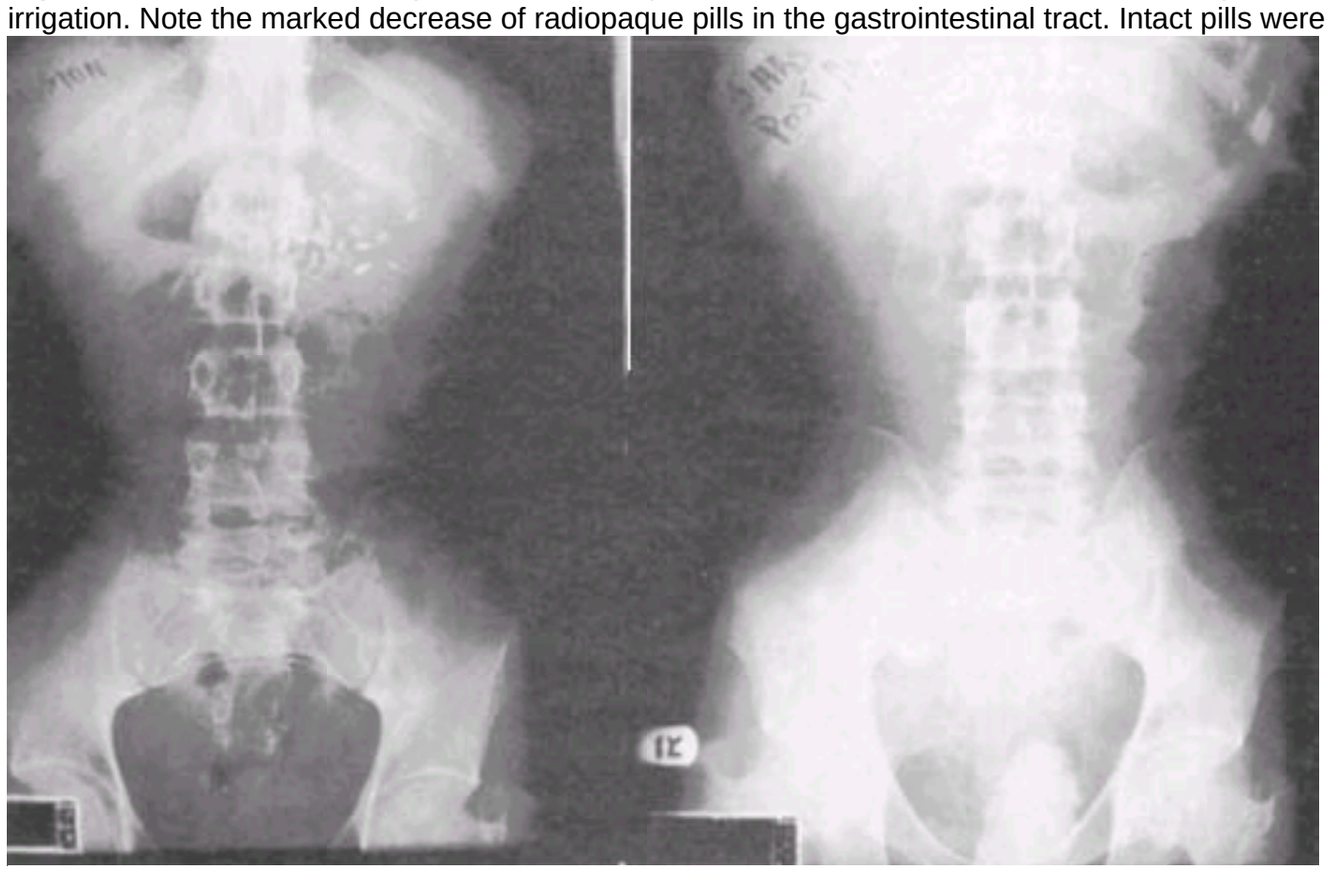


Figure 43-18 It is very difficult for even the most motivated patient to drink an effective volume of whole-bowel irrigation solution. To enhance compliance and to decrease vomiting, PEG-ES may be slowly and continuously administered via a nasogastric tube. An empty bag of saline is hung on an IV pole, the corner of the bag is removed, and the PEG-ES is poured into the bag. Standard IV tubing is connected to the proximal end of a nasogastric tube and the solution is infused continuously. In this picture, charcoal has been added to the whole-bowel irrigation solution. Metoclopramide was coadministered to reduce nausea.

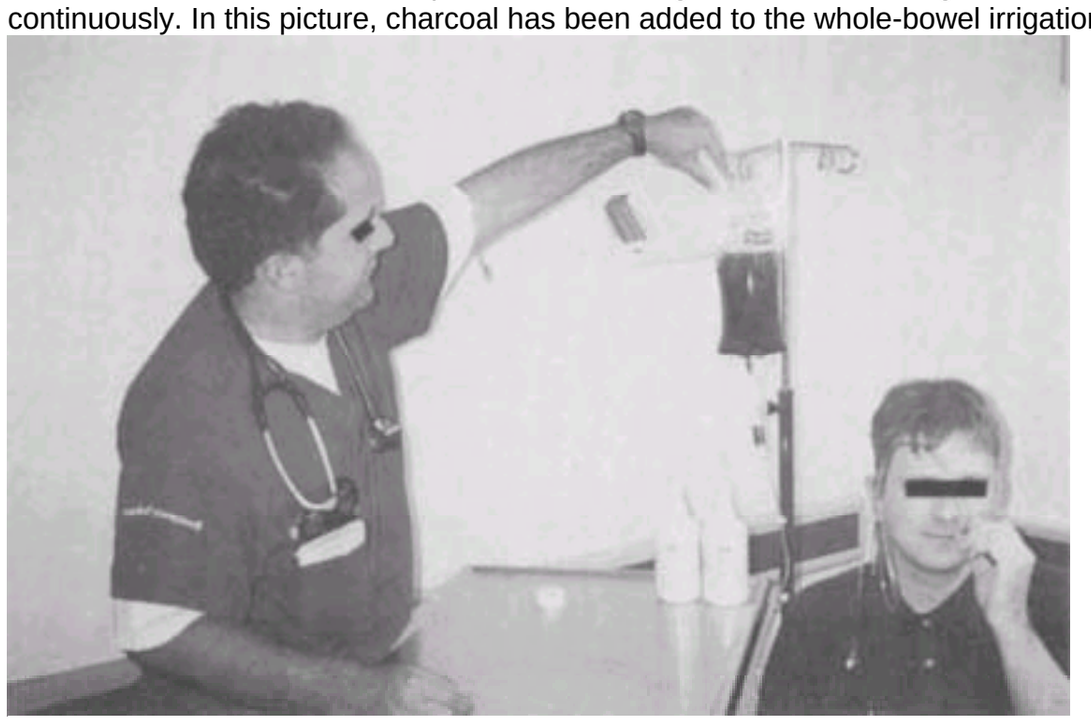


Figure 43-19 Decontamination of personnel using copious water irrigation.

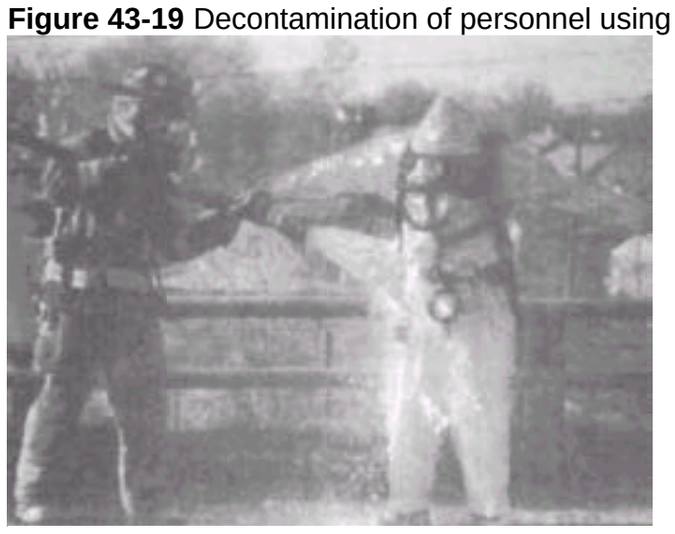


Figure 44-1 Blunt abdominal trauma algorithm. BAT, blunt abdominal trauma; CT, computed tomography; D/C, discharge; DPA, diagnostic peritoneal aspiration; DPL, diagnostic peritoneal lavage; IP, intraperitoneal; IPH, intraperitoneal hemorrhage; LAP, laparotomy; SPE, serial physical examinations; US, ultrasound. *Determined by unequivocal free intraperitoneal fluid on ultrasound or positive peritoneal aspiration on diagnostic peritoneal lavage. †Can be unreliable because of closed head injury, intoxicants, distracting injury, or spinal cord injury. ‡One or more studies may be indicated. §Need for laparotomy is based on clinical scenario, diagnostic studies, and institutional resources. *(From Marx J: Abdominal Trauma. In Marx JA, Hockberger RS, Walls RM, et al. [eds]: Rosen's Emergency Medicine Concepts and Clinical Practice, 5th ed. St. Louis, CV Mosby, 2002, p 432.)*

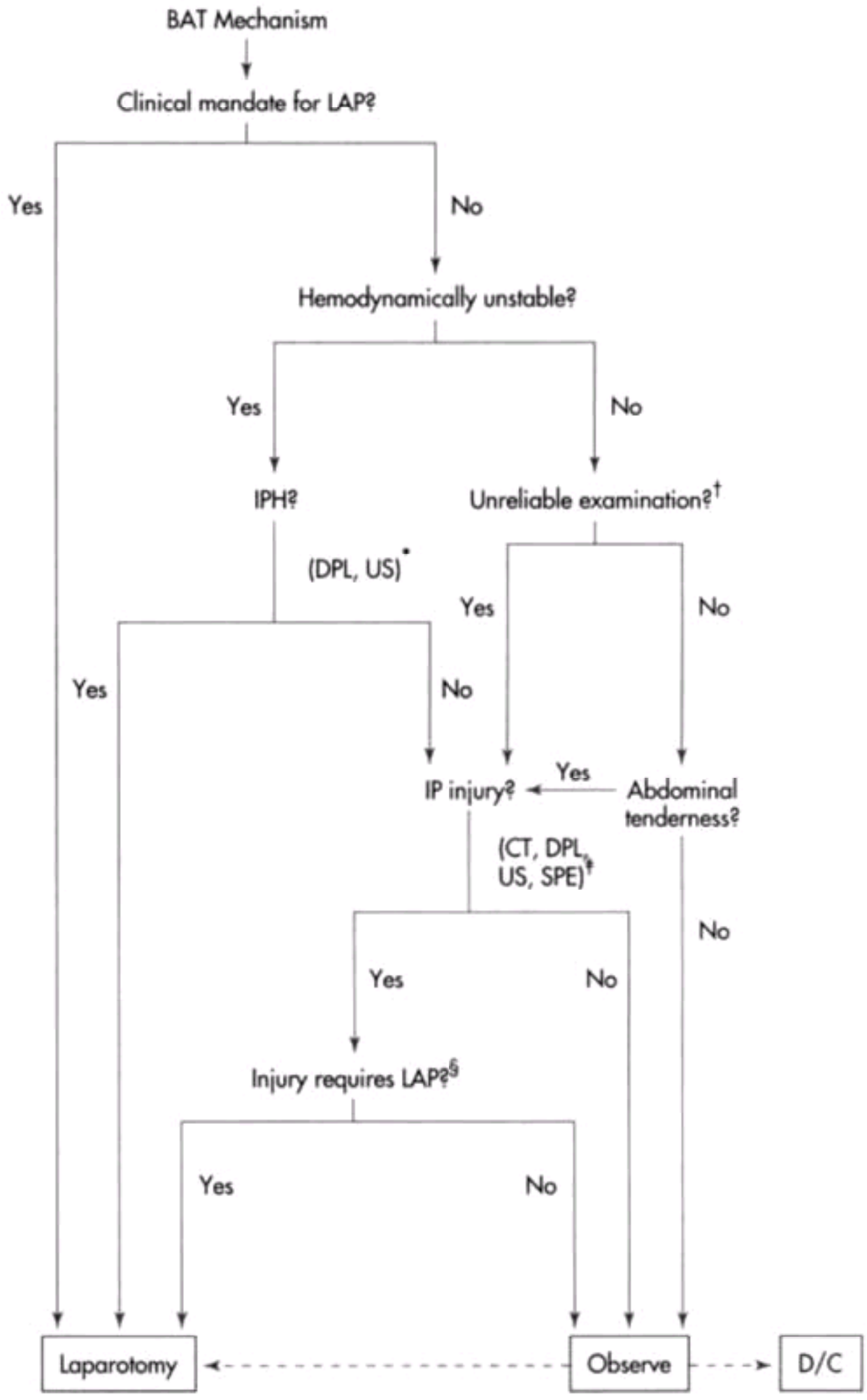


Figure 44-2 Anterior abdomen stab wound algorithm. *Plain films, ultrasound, laparoscopy, and computed tomography (CT) can assess peritoneal entry. †Laparoscopy or CT can complement or replace diagnostic peritoneal lavage. ‡Expectant management of injuries is infrequently attempted. D/C, discharge; DPL, diagnostic peritoneal lavage; LAP, laparotomy; LWE, local wound exploration; PEx, physical examination. *(From Marx J: Abdominal Trauma. In Marx JA, Hockberger RS, Walls RM, et al. [eds]: Rosen's Emergency Medicine Concepts and Clinical Practice, 5th ed. St. Louis, CV Mosby, 2002, p 427.)*

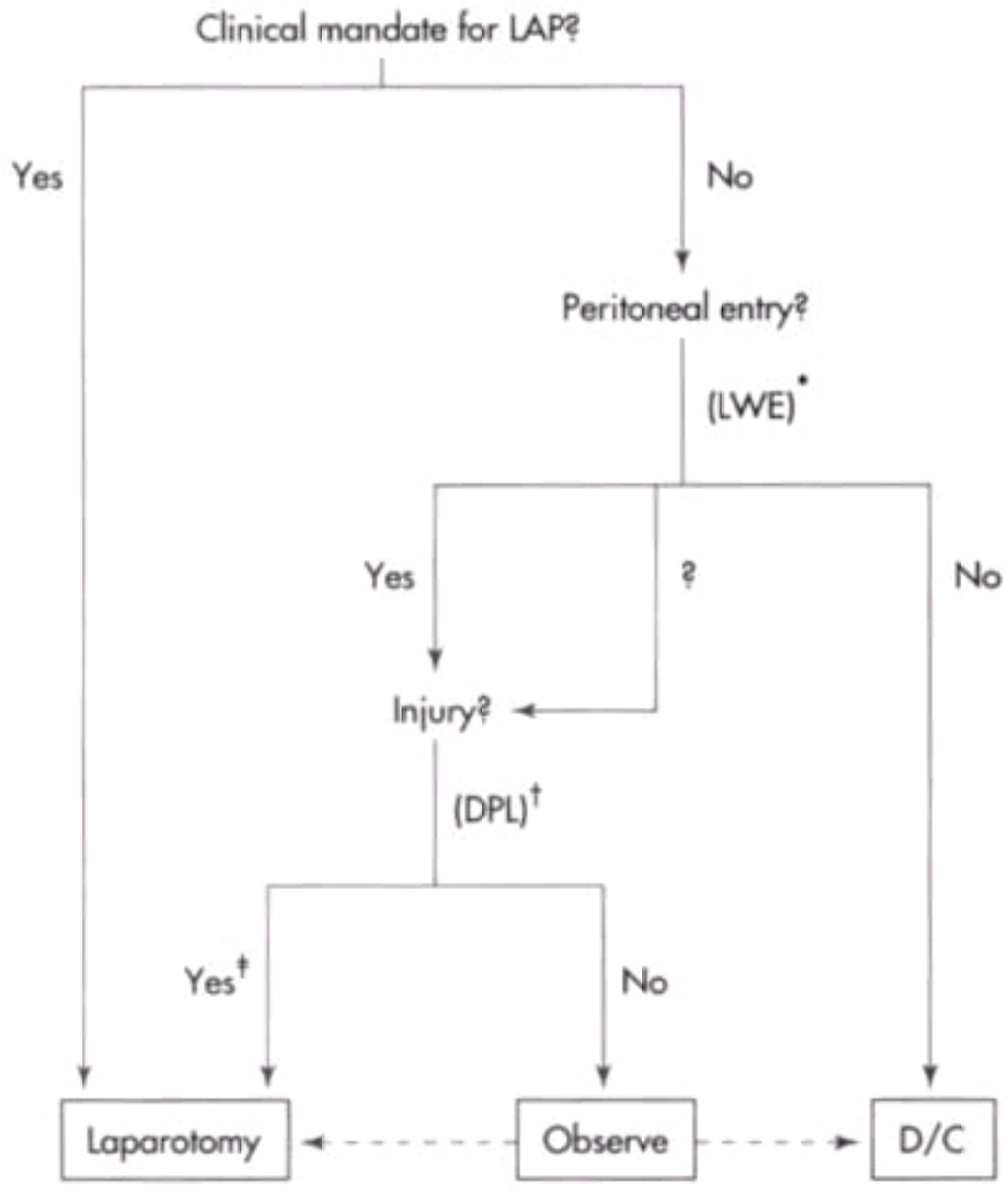


Figure 44-3 Gunshot wound algorithm. D/C, discharge; DPL, diagnostic peritoneal lavage; LAP, laparotomy. *Can be assessed by missile path, plain films, local wound exploration, ultrasound, and laparoscopy. †Most centers proceed to laparotomy if peritoneal entry is suspected. ‡Patients with documented superficial and low-velocity injuries can be discharged; unknown depth or high-velocity injuries require further tests or observation. §Serial examinations, laparoscopy, or a computed tomography scan can complement or replace diagnostic peritoneal lavage. ¶Expectant management of injuries caused by gunshot wounds is rarely attempted. *(From Marx J: Abdominal Trauma. In Marx JA, Hockberger RS, Walls RM, et al. [eds]: Rosen's Emergency Medicine Concepts and Clinical Practice, 5th ed. St. Louis, CV Mosby, 2002, p 430.)*

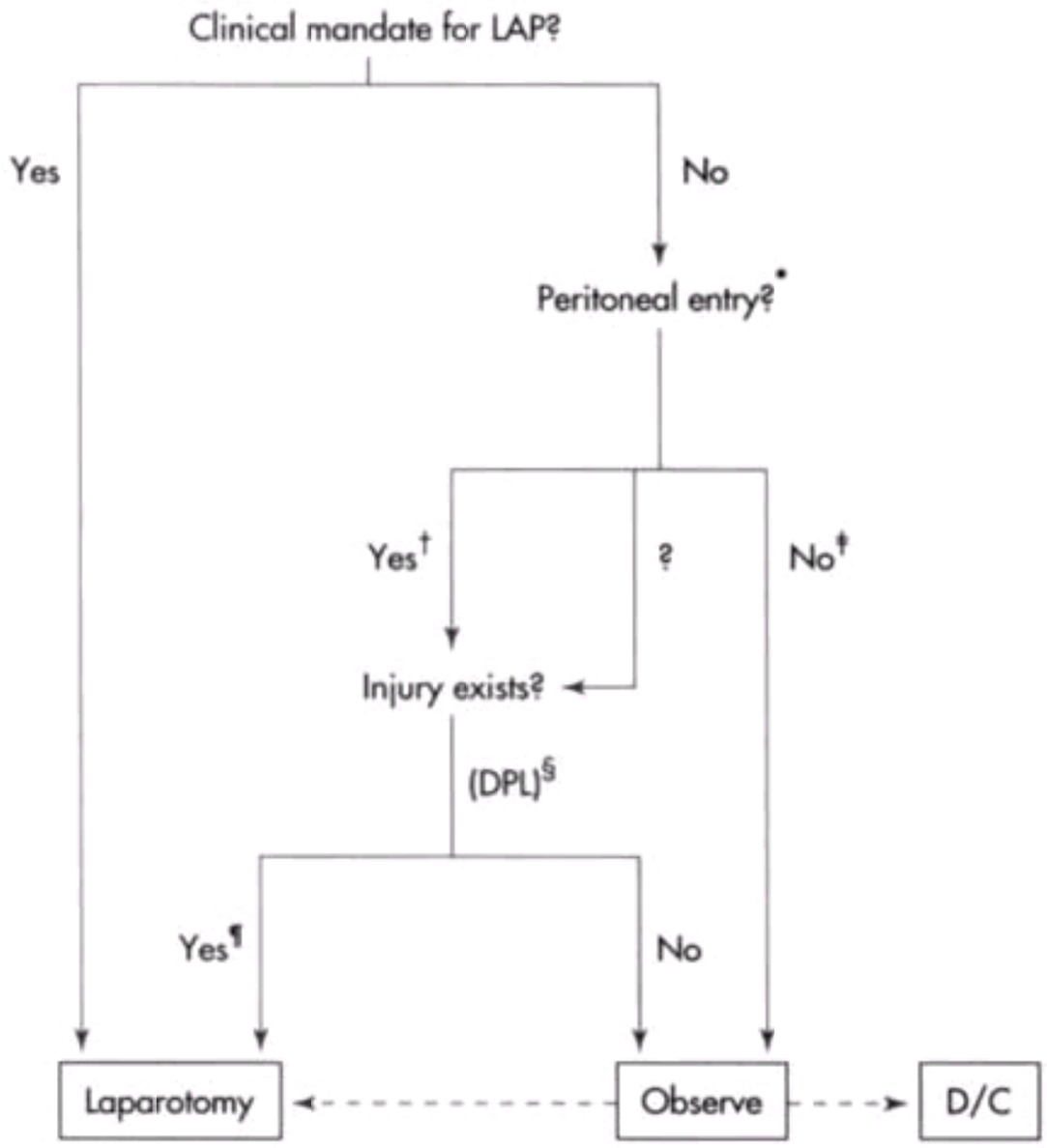


Figure 44-4 Local anesthesia is introduced at incision or puncture site. The patient is supine with the head of the bed elevated slightly.

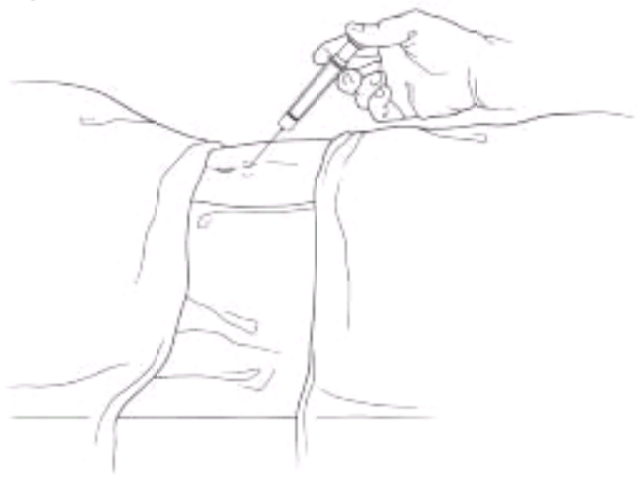


Figure 44-5 Standard equipment used for open lavage technique.

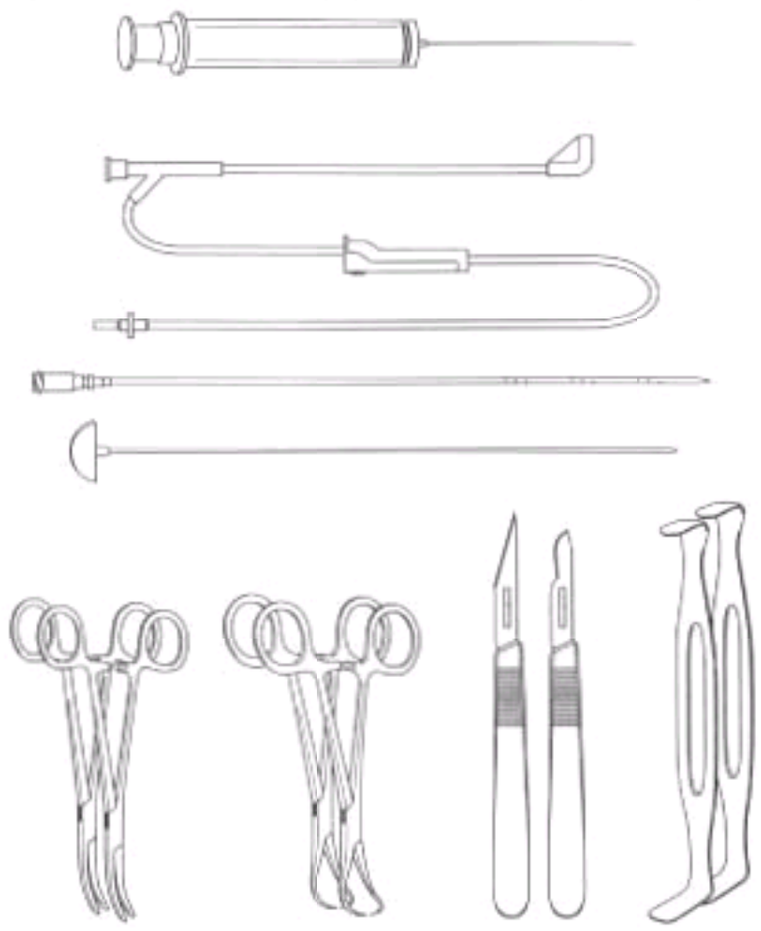
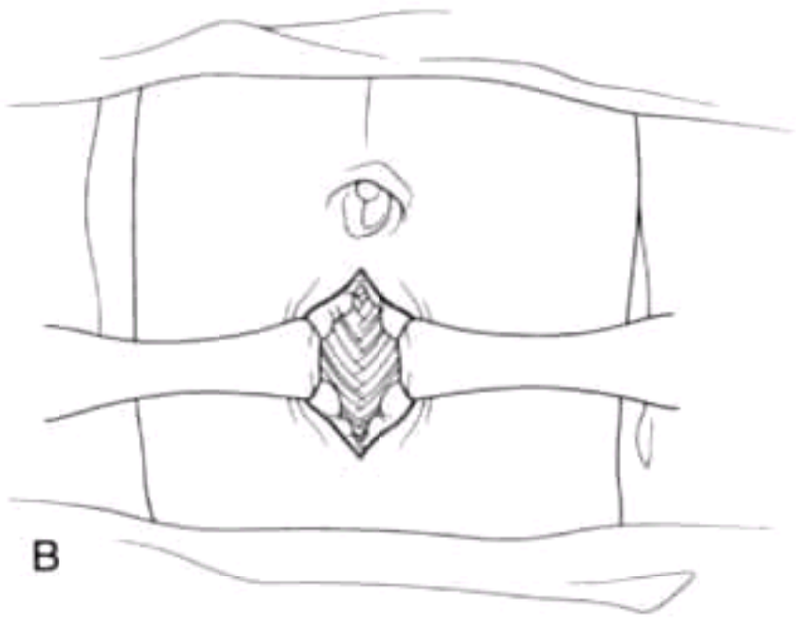
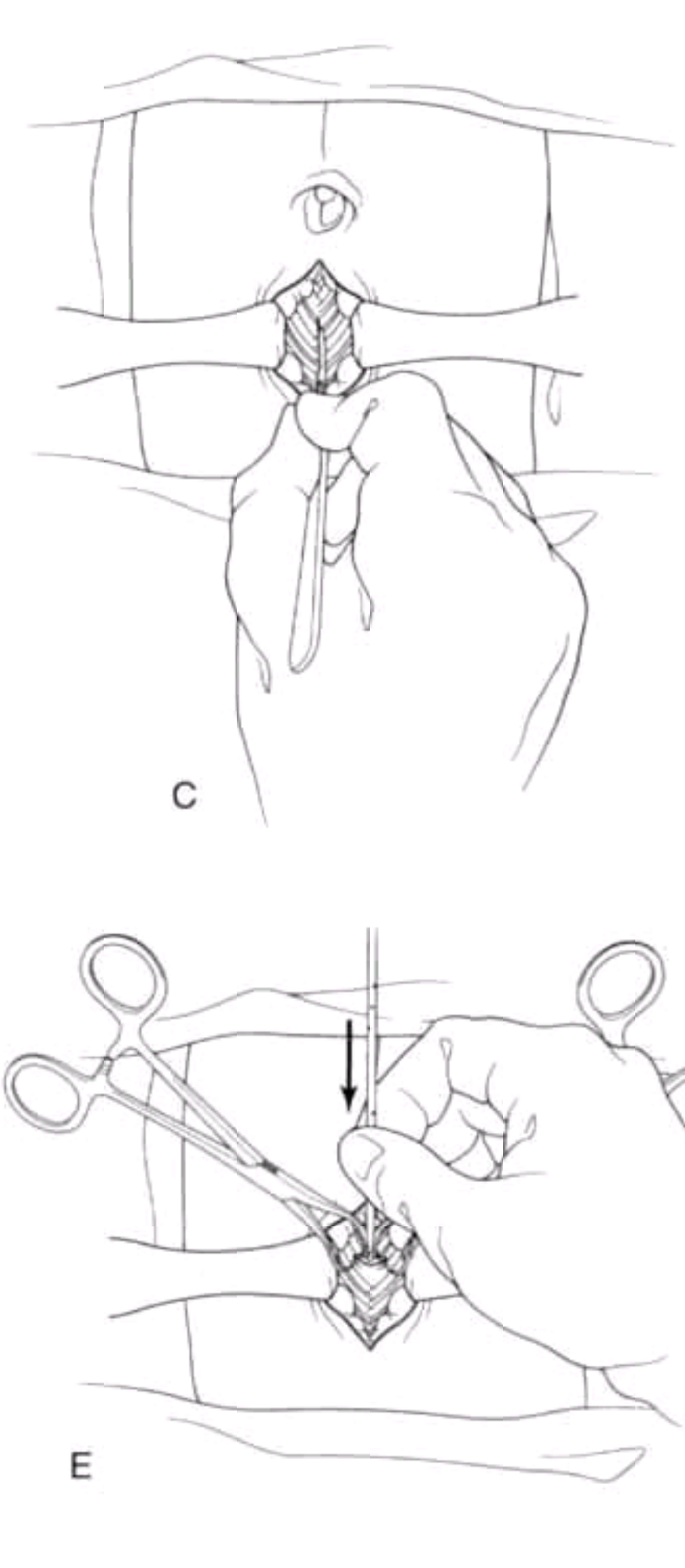
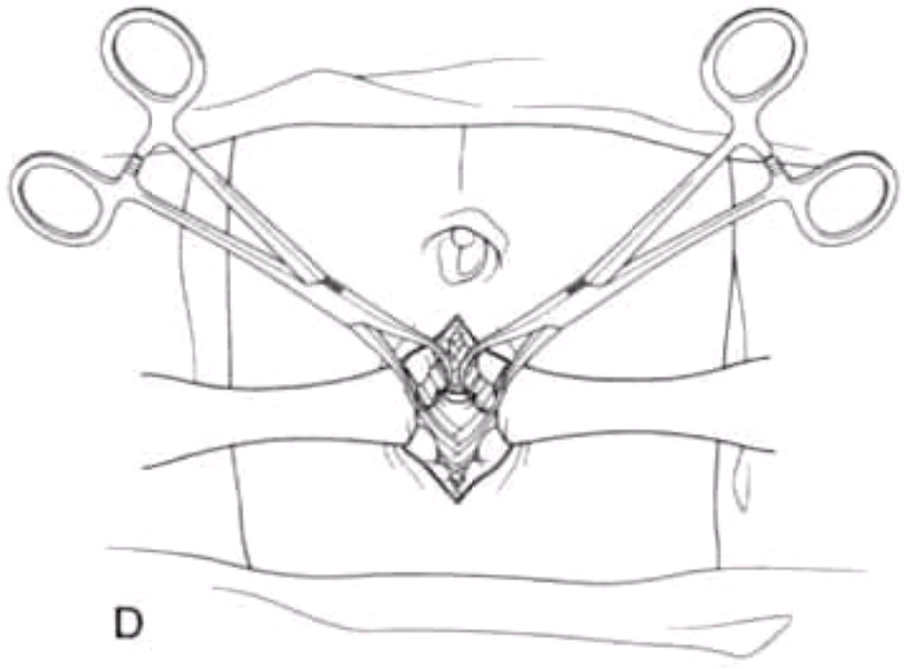
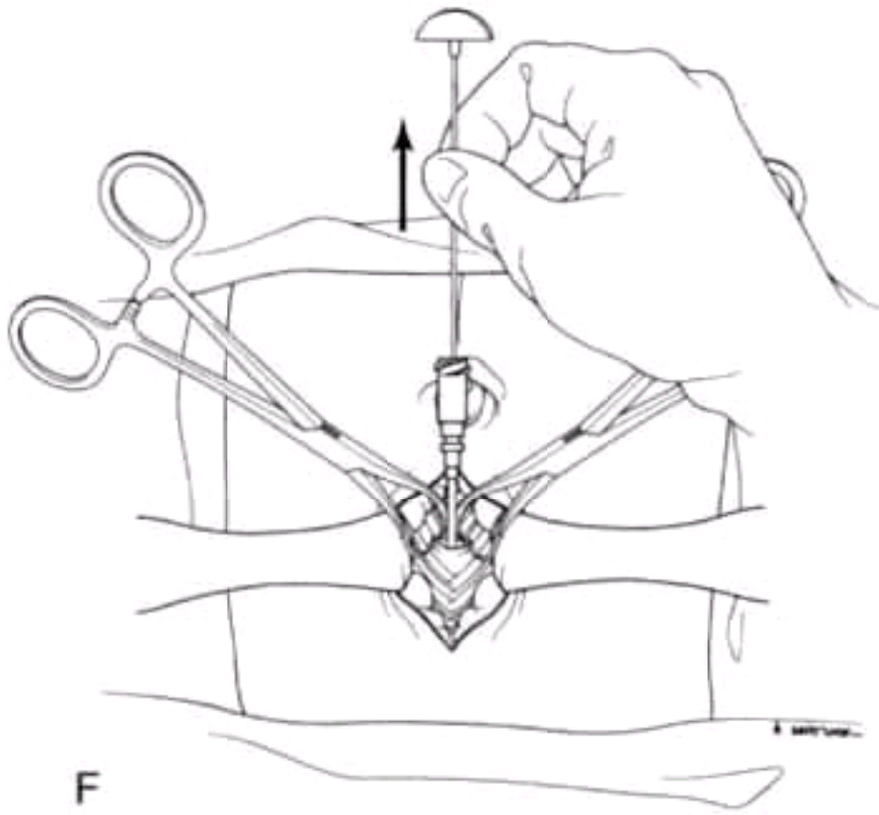


Figure 44-6 *A,* After bladder decompression (generally by Foley catheter placement), a 4- to 6-cm long vertical infraumbilical incision is made with a No. 11 scalpel. *B,* Blunt dissection using Army-Navy retractors is carried down to the *rectus fascia*. Crossing bands of crural fibers may be seen. *C,* A 2- to 3-mm incision is made through the *rectus fascia* in the midline (*linea alba*) with a No. 15 scalpel. *D,* Towel clips grasp each side of the *rectus fascia*, which is lifted prior to insertion of the trocar and diagnostic peritoneal lavage (DPL) catheter. *E,* The trocar with DPL catheter is passed at a 45° caudad angle into the fascial opening and through the peritoneum. Note that in the fully open method, the incision in the *rectus fascia* is extended, the peritoneum is directly visualized and incised, and the catheter alone is placed into the peritoneal cavity. *F,* As soon as the peritoneum has been entered, only the catheter is gently advanced into the peritoneal cavity while the trocar is withdrawn. It is often helpful to advance the catheter with a slight twisting motion and to direct it toward either the right or left pelvic gutter.





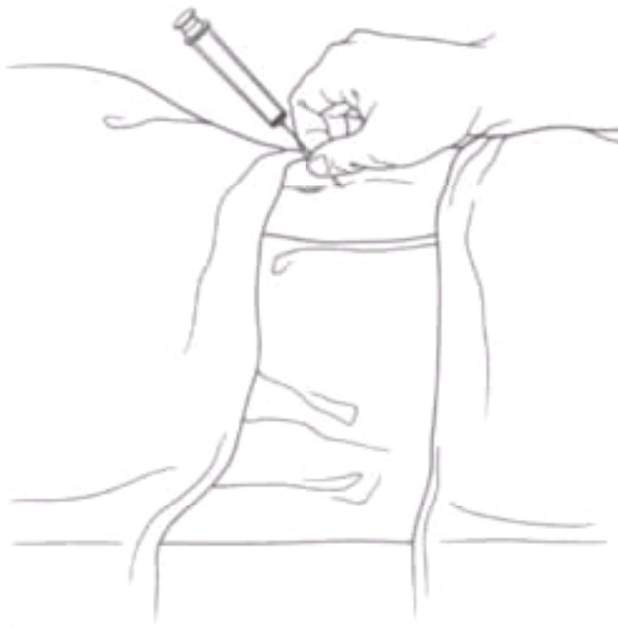




E

F

Figure 44-7 *A,* For the closed diagnostic peritoneal lavage (DPL) method using a guide wire (Seldinger technique), the needle is inserted into the peritoneal cavity in the midline just below the umbilicus and aimed slightly caudad. *B,* The flexible guide wire is passed through the needle and into the peritoneal cavity. Ideally, the wire should be directed toward the right or left pelvic gutter. The needle is withdrawn while the wire is stabilized with the operator's free hand at all times. *C,* A stab incision is made with a No. 11 scalpel immediately below the wire to permit easier passage of the DPL catheter. *D,* The DPL catheter is directed over the wire and into the peritoneal cavity using a slight twisting motion. The wire is stabilized by the operator at all times and removed after catheter placement. The catheter should be directed toward the right or left pelvic gutter when advanced.



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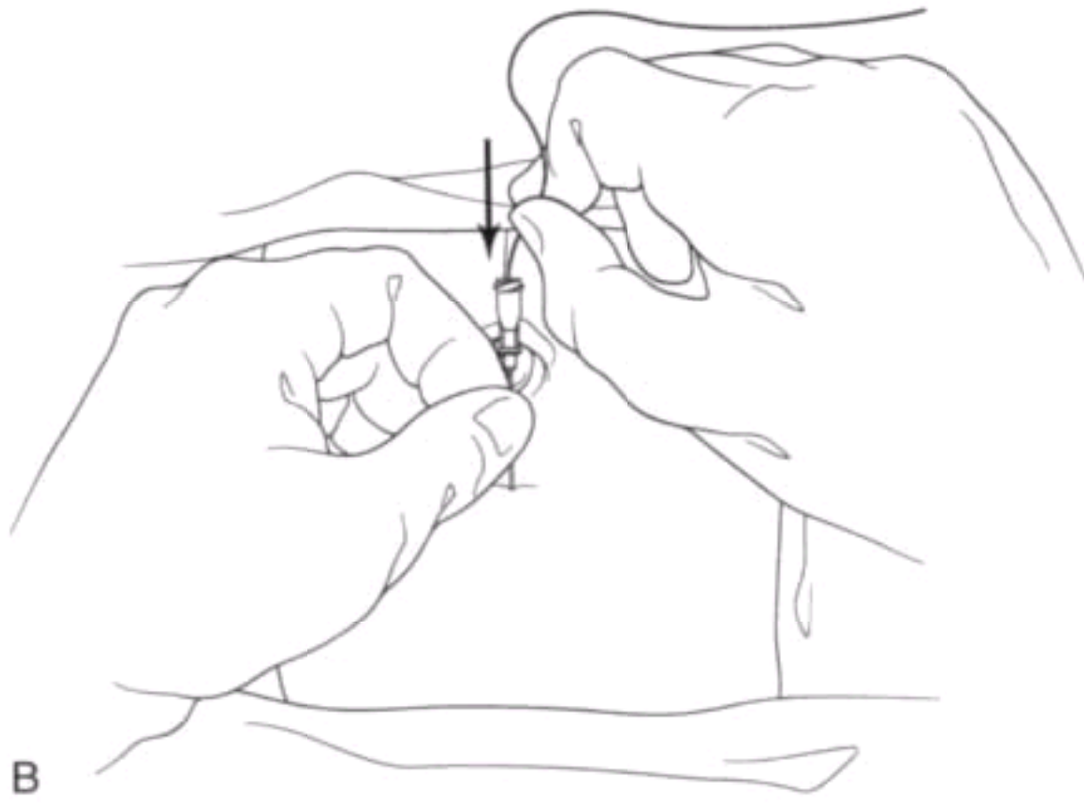

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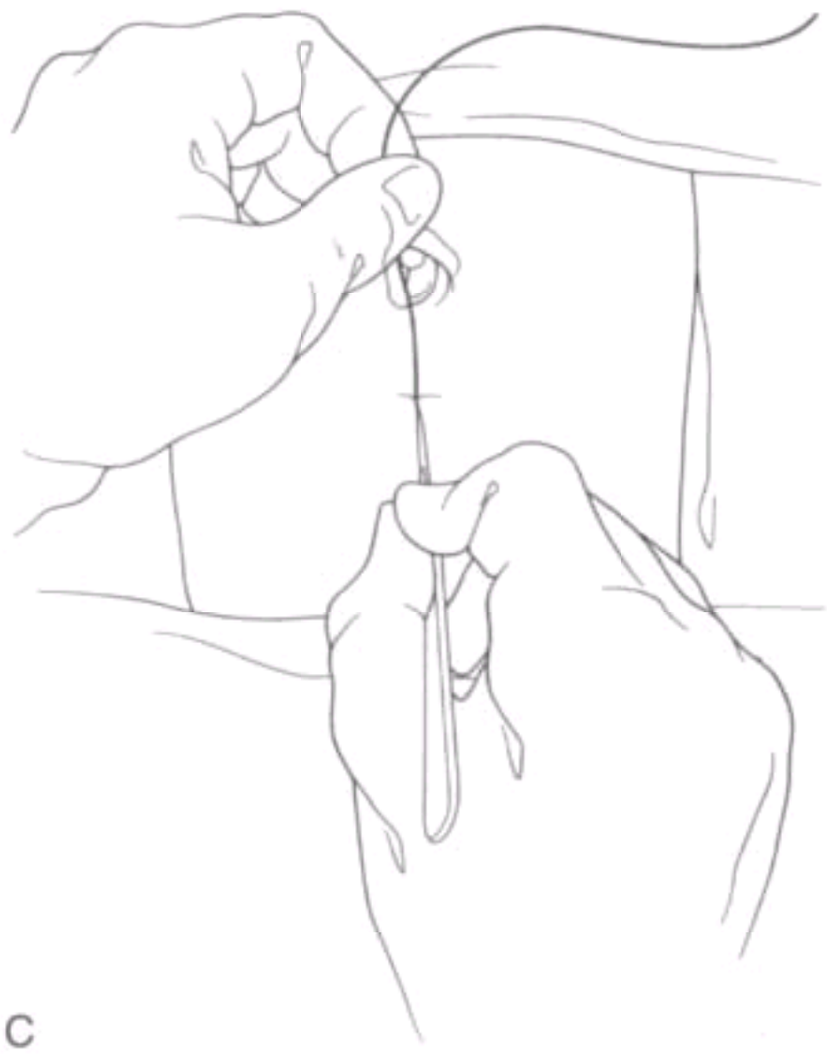

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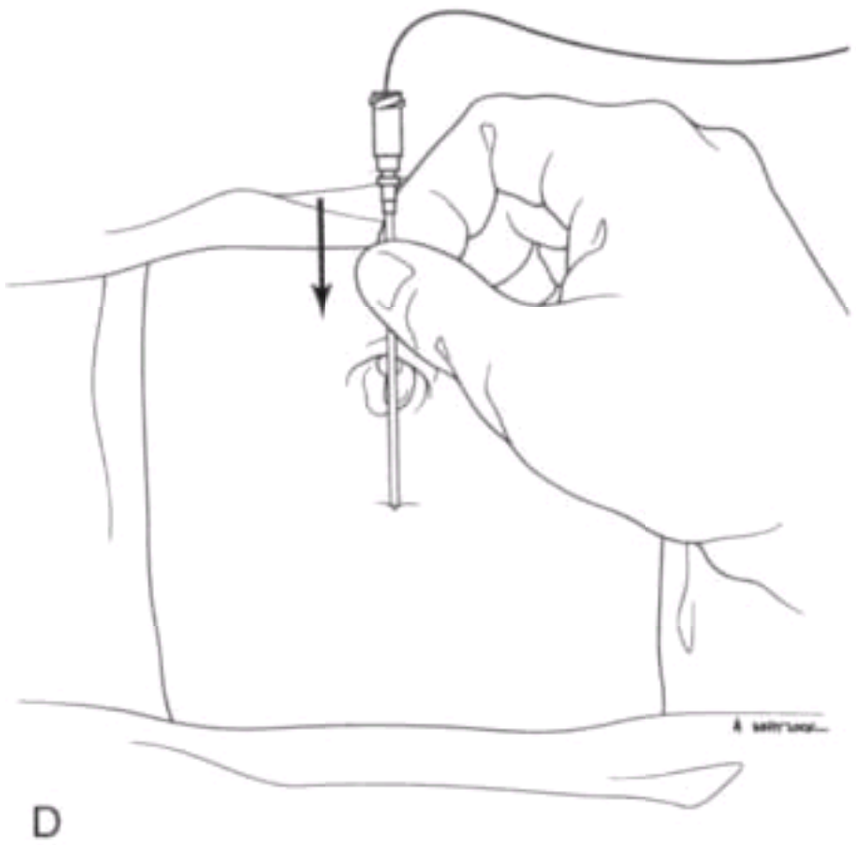


D

Figure 44-8 After attachment of the right angle connector and extension tubing, aspiration of the peritoneum is attempted.

Figure 44-9 If the aspiration is negative, normal saline or Ringer's lactate solution is instilled through the catheter. *The IV tubing should have no valves in place.* After infusion, the fluid bag is placed on the floor and allowed to fill with peritoneal effluent via gravity.

Figure 44-10 Pelvic fracture algorithm. CT, computed tomography; D/C, discharge; DPA, diagnostic peritoneal aspiration; DPL, diagnostic peritoneal lavage; IP, intraperitoneal; IPH, intraperitoneal hemorrhage; LAP, laparotomy; Pelvic Fx, pelvic fracture; US, ultrasound. *Determined by unequivocal free intraperitoneal fluid on ultrasound or positive peritoneal aspiration on diagnostic peritoneal lavage. †One or more studies may be indicated. ‡Need for laparotomy is based on clinical scenario, diagnostic studies, and institutional resources. *(From Marx J: Abdominal Trauma. In Marx JA, Hockberger RS, Walls RM, et al [eds]: Rosen's Emergency Medicine Concepts and Clinical Practice, 5th ed. St. Louis, CV Mosby, 2002, p 434.*

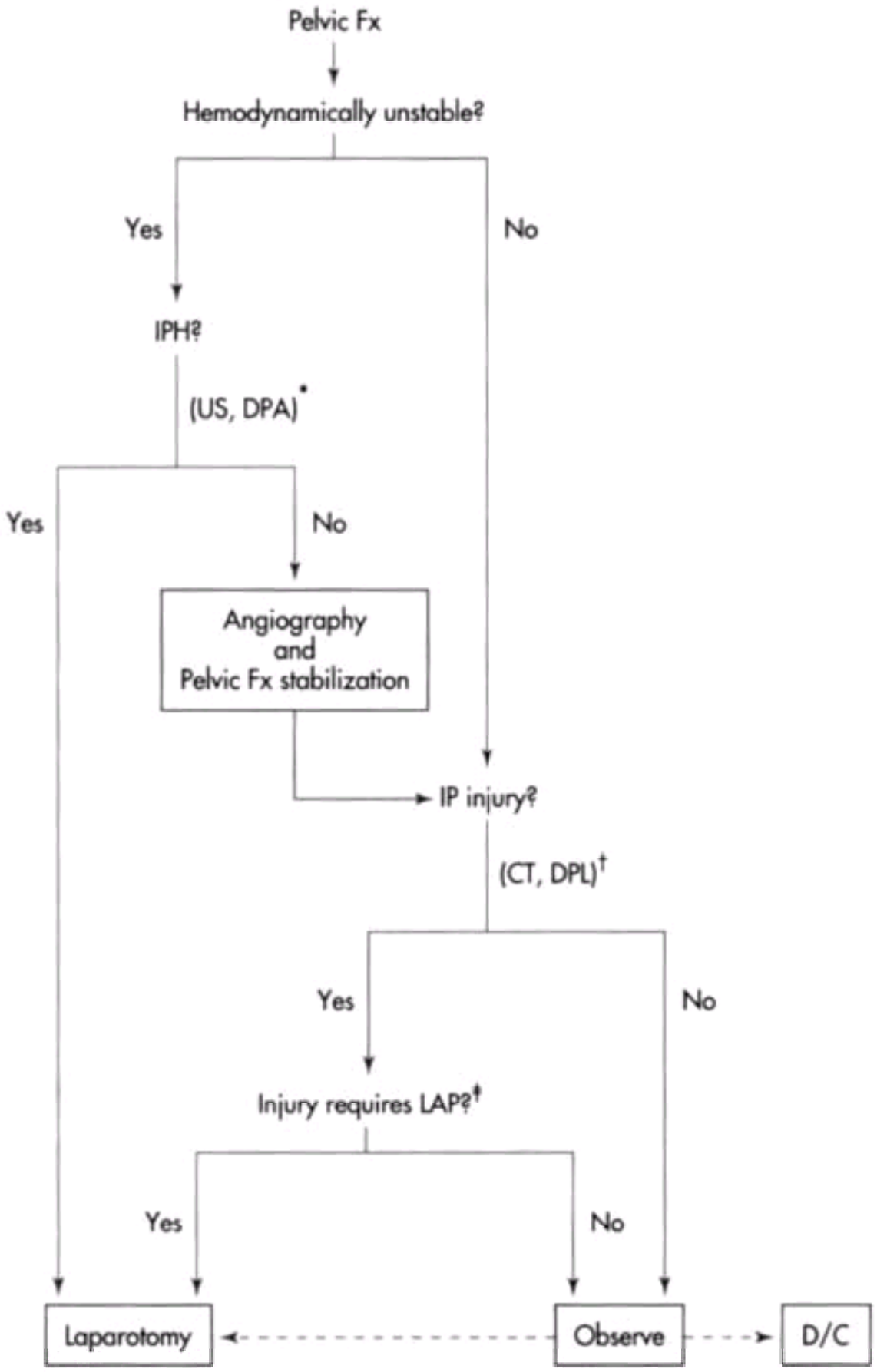


Figure 44-11 Preferred sites for paracentesis: *1,* Primary site is infraumbilical in midline through *linea alba*. *2,* Preferred alternate (lateral rectus) site is in either lower quadrant, approximately 4 to 5 cm cephalad and medial to the anterior superior iliac spine.

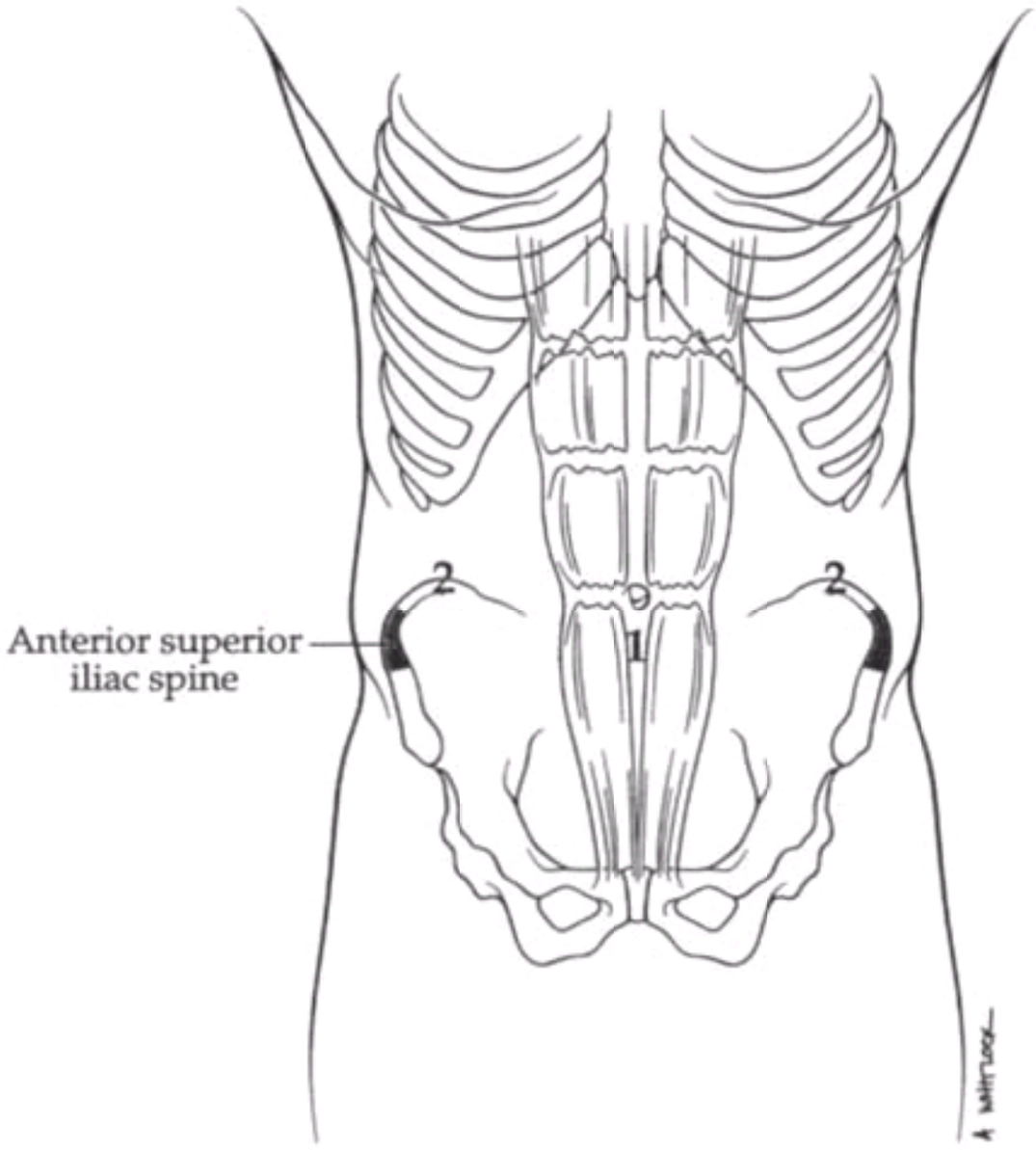


Figure 44-12 An alternative to the sitting or supine position for needle paracentesis is to place the patient in the lateral decubitus position. In this example the midline is aspirated, although the lateral rectus sites may also be used. Some prefer the lateral decubitus position routinely, because the bowel tends to float upward and out of the path of the needle.

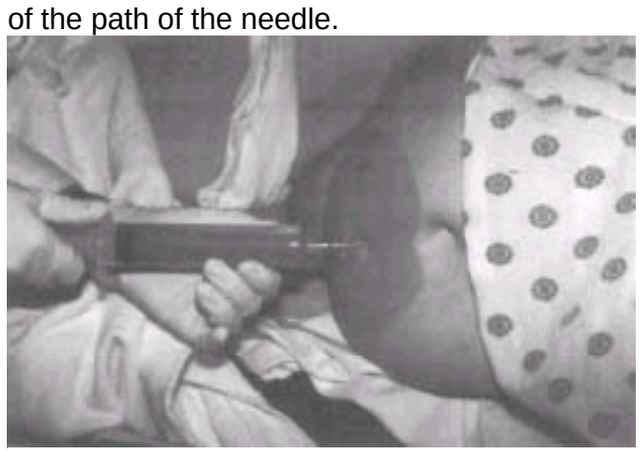


Figure 44-13 *A,* Z-tract method of paracentesis. The skin is pulled approximately 2 cm caudad in relation to the deep abdominal wall by the non-needle-bearing hand while the paracentesis needle is slowly being inserted directly perpendicular to the skin. *B,* After penetrating the peritoneum and obtaining fluid return, the skin is released. Note that the needle is now angulated caudally.

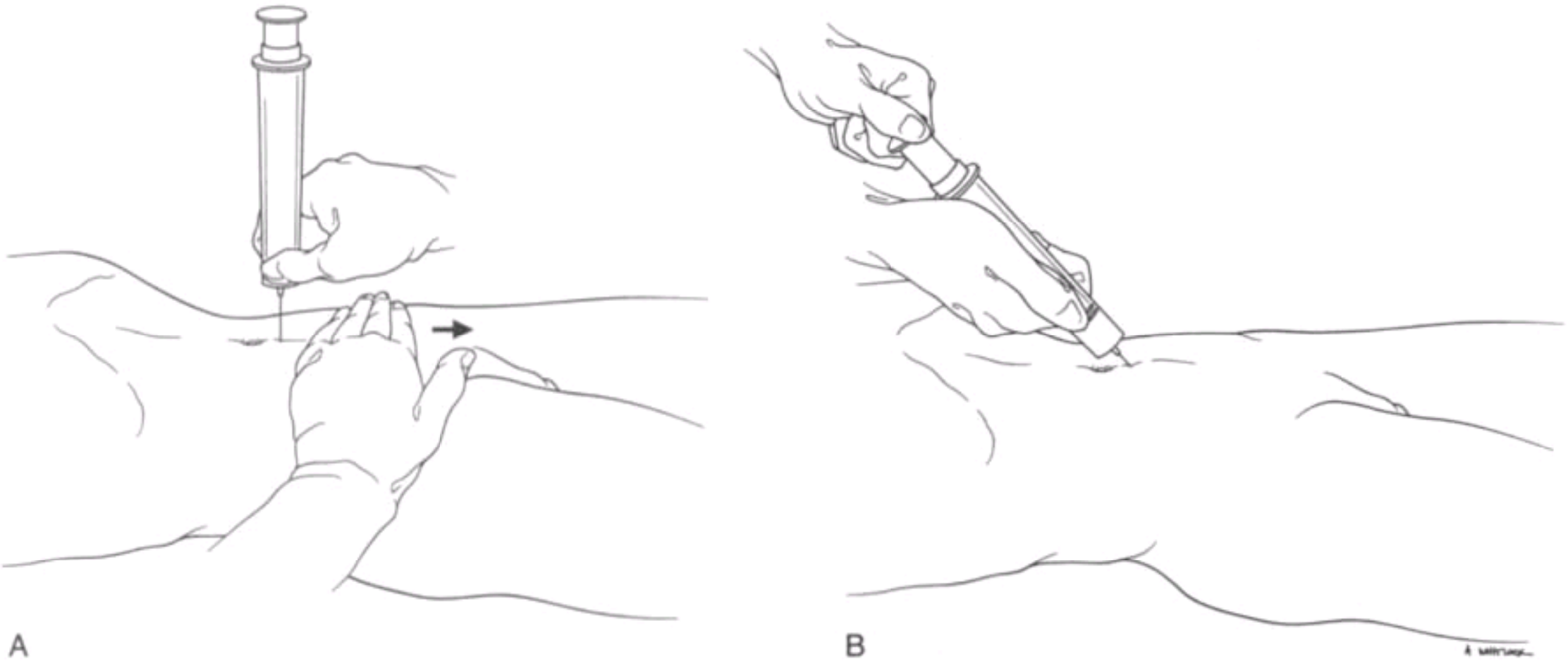


Figure 45-1 Indirect inguinal hernia.

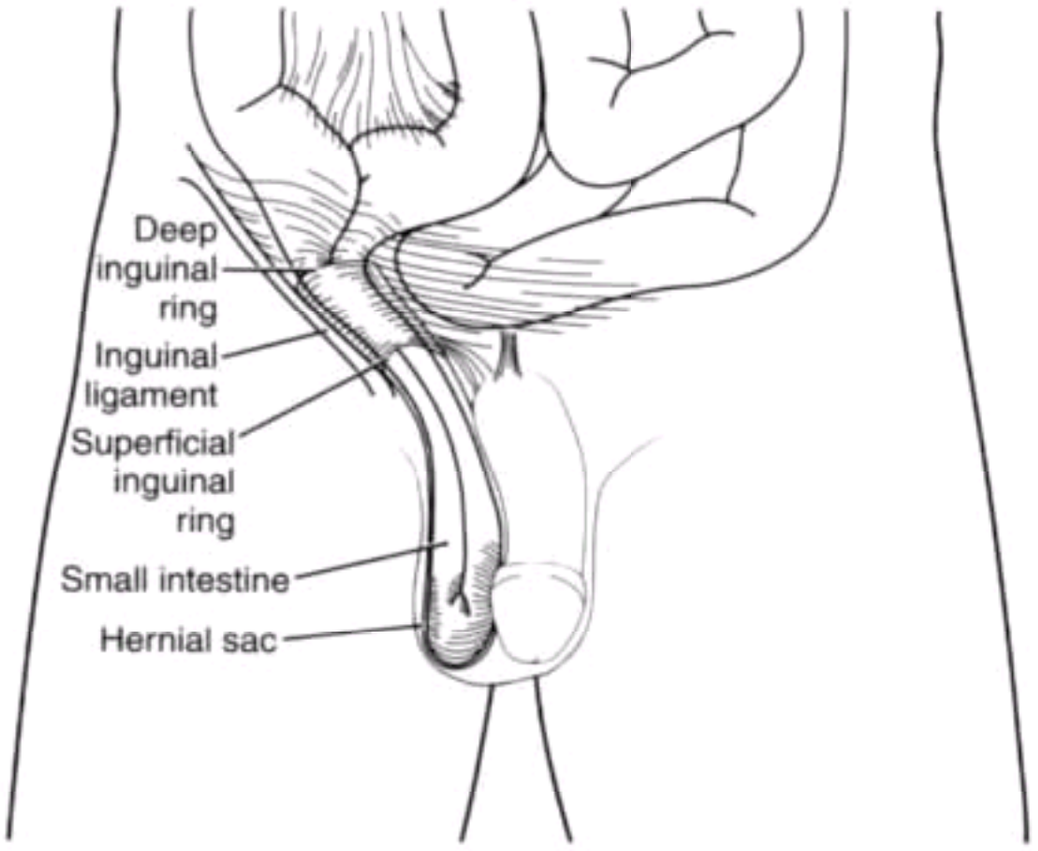


Figure 45-2 Direct inguinal hernia.

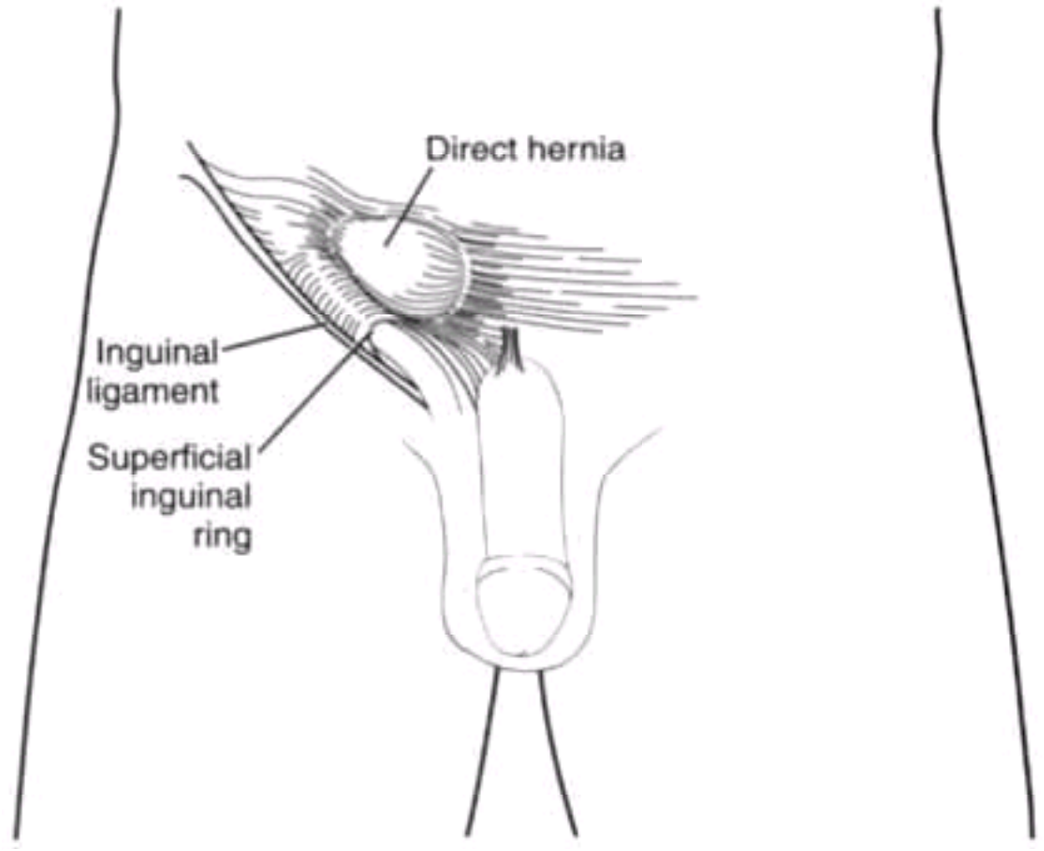


Figure 45-3 Femoral hernia.

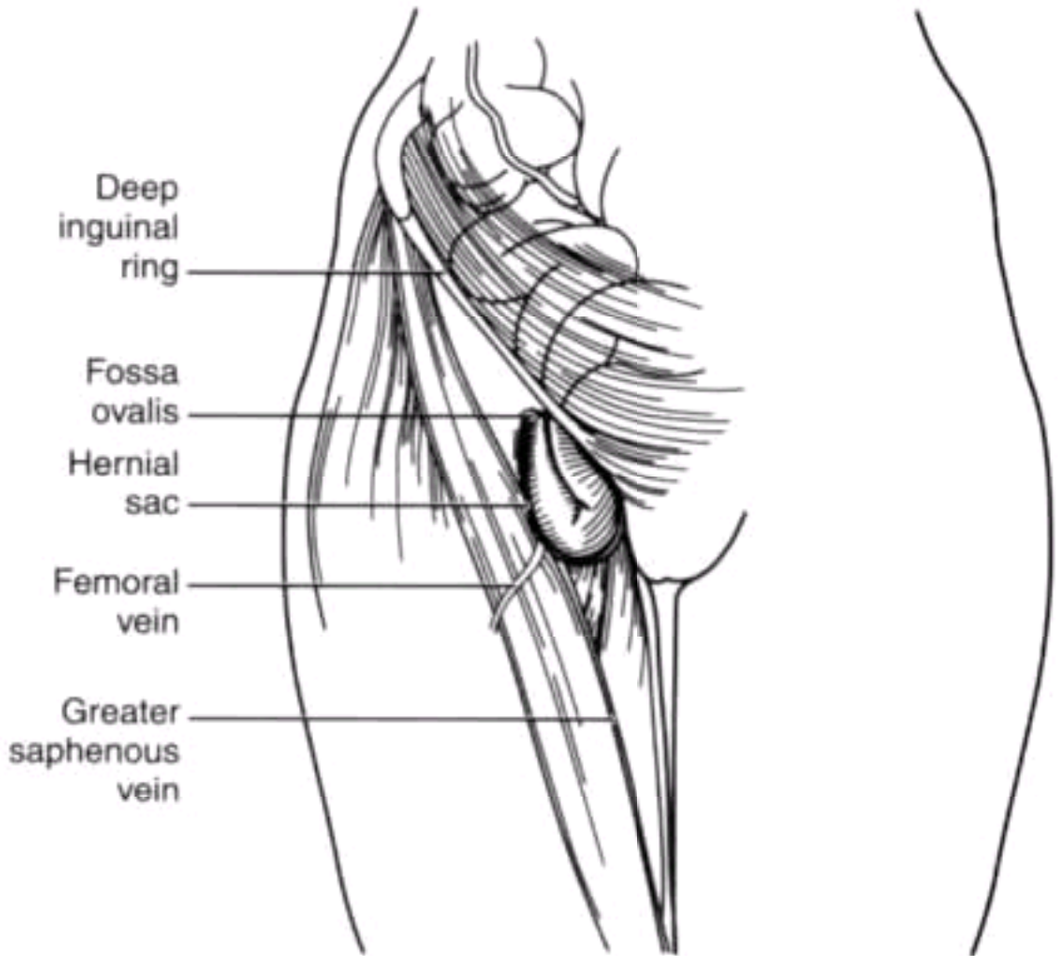


Figure 45-4 Ventral hernias. *A,* Incisional hernia. *B,* Umbilical hernia. *C,* Epigastric hernia. *D,* Spigelian hernia.

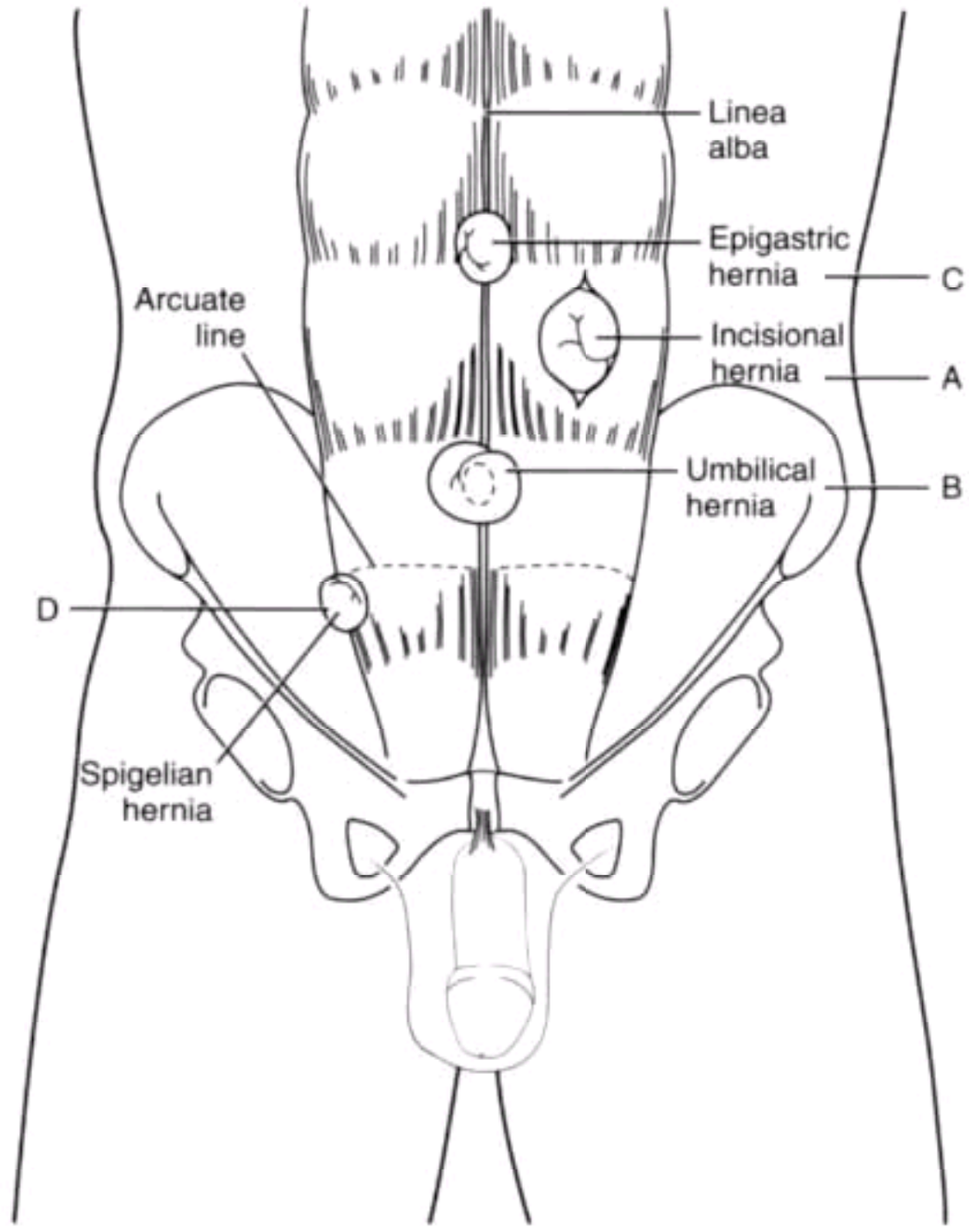


Figure 45-5 En masse reduction. *A,* When a hernia forms, it projects from the fascia into the subcutaneous fat. The object of reduction is to replace the hernia into the peritoneal cavity. *B,* If the hernia sac is partially reduced into the subcutaneous fat of an obese patient, it may appear reduced and may not be palpable owing to the patient's body habitus. However, the hernia is still susceptible to incarceration or ischemia, as it has not been returned to the peritoneal sac.

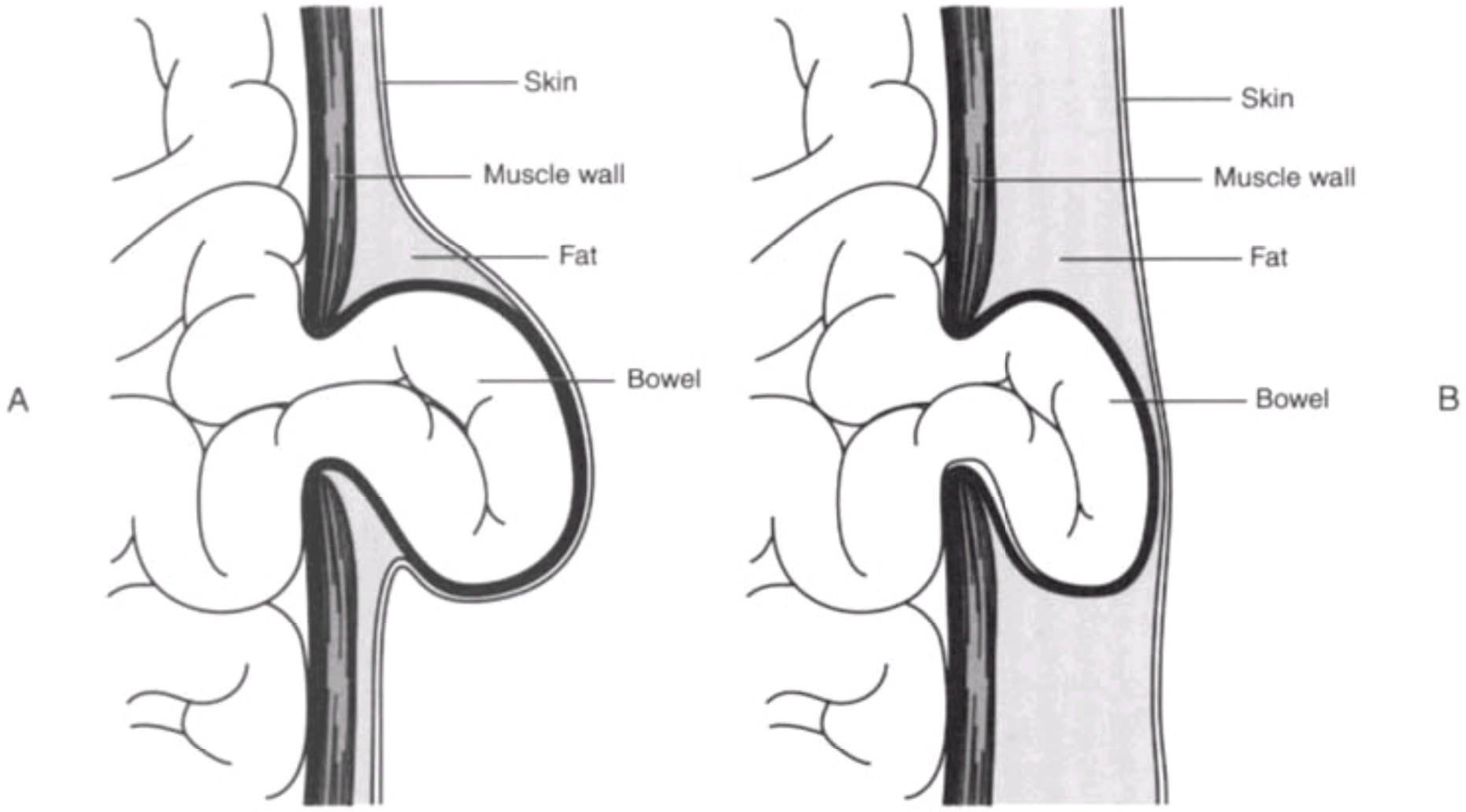


Figure 45-6 Hydrocele versus hernia. *A,* normal anatomy; *B,* noncommunicating hydrocele that may be confused with hernia; *C,* indirect hernia that can be palpated from inguinal ring to testicle.

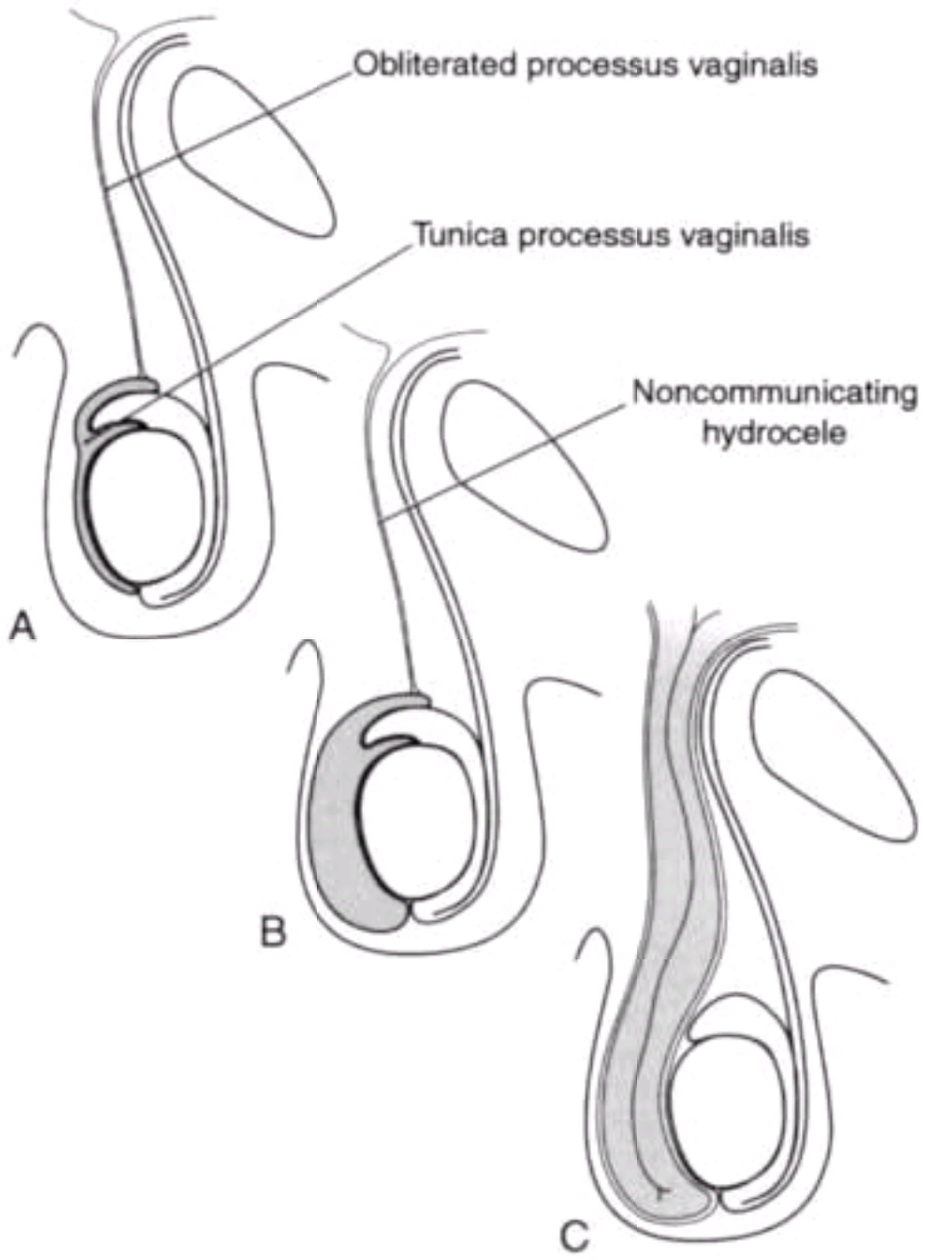


Figure 45-7 Ballooning of contents. *A,* When reducing a hernia, the contents may ride up over the edge of the fascial defect. This leads to ballooning of the contents around the neck of the hernia, which hinders the reduction. *B,* By placing fingers along the edge of the hernia neck, one can direct the contents into instead of over the fascial defect.

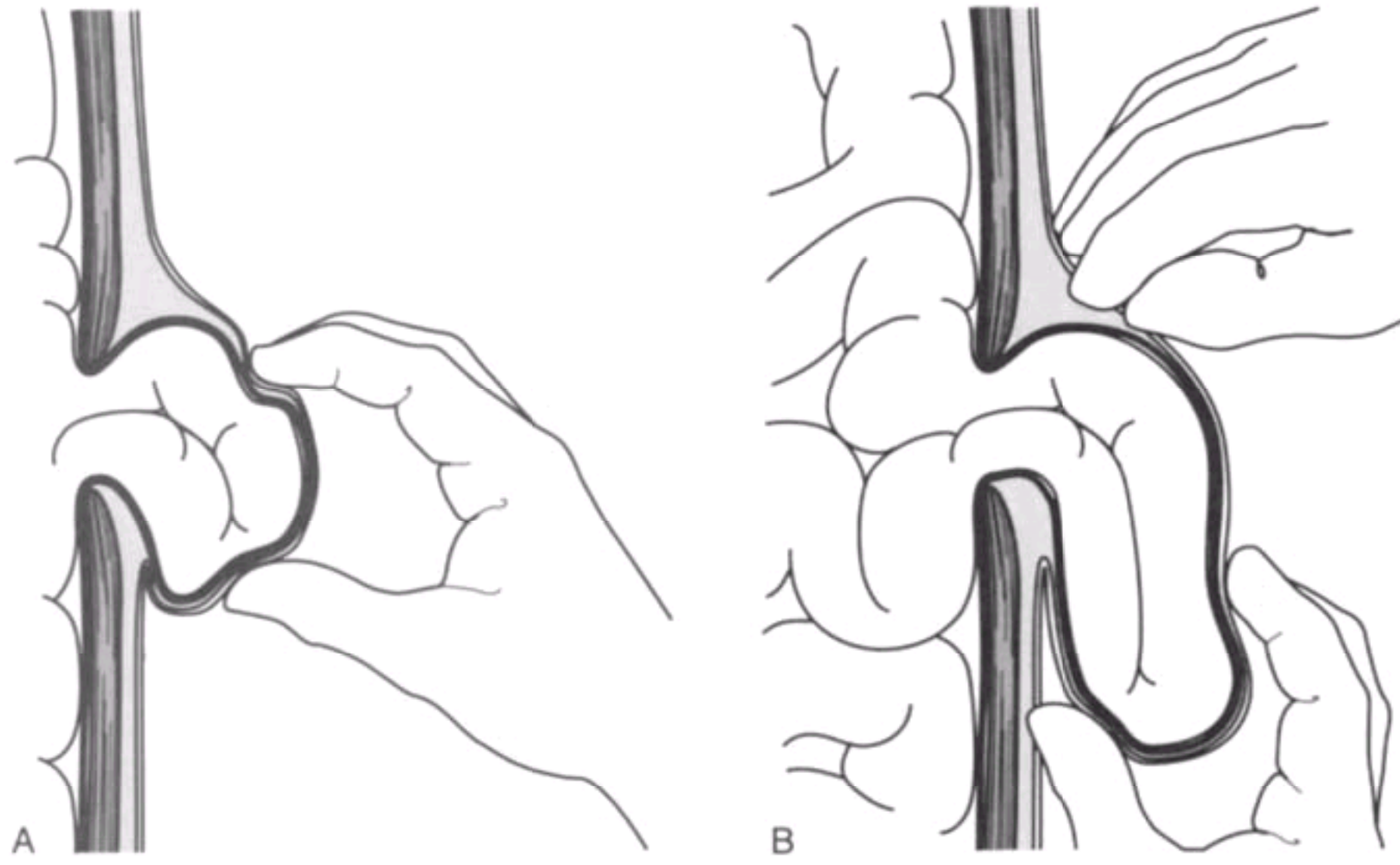
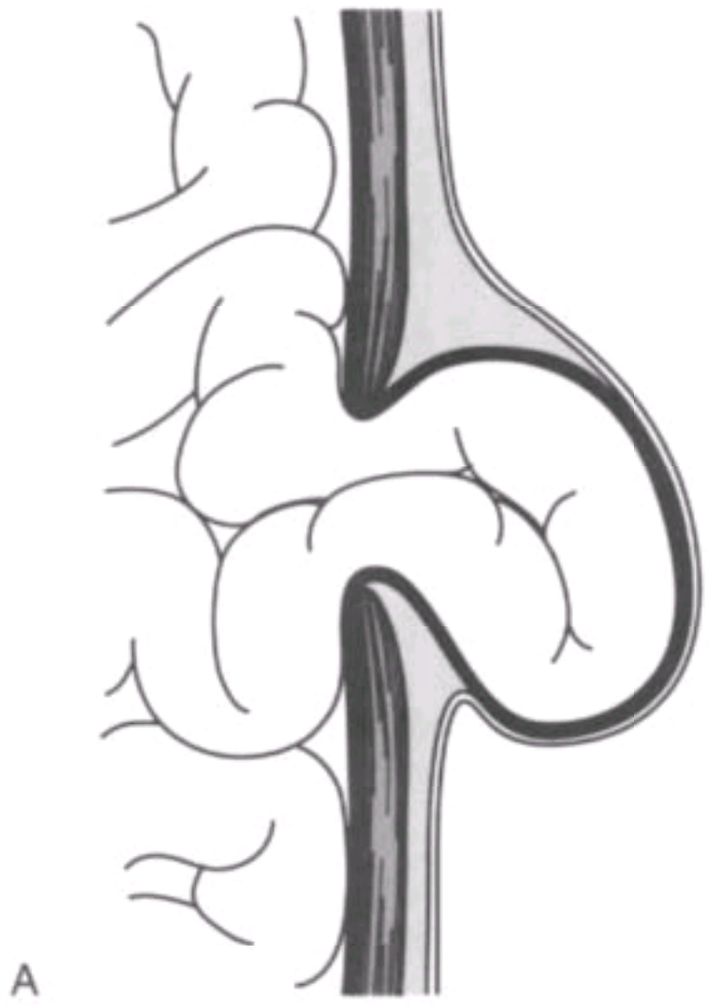


Figure 45-8 Guiding hernia into place. A, The hernia can be divided into several sections. The proximal portion is closest to the neck or fascial defect, through which the hernia protrudes. The distal portion is farthest from the neck. B, When attempting to reduce the hernia, be careful not to invaginate the distal portion first or the proximal portion may obstruct the opening as it is pushed over the sides. C, The last portion of the bowel that exits into the hernia sac should be the first returned to the peritoneal cavity.



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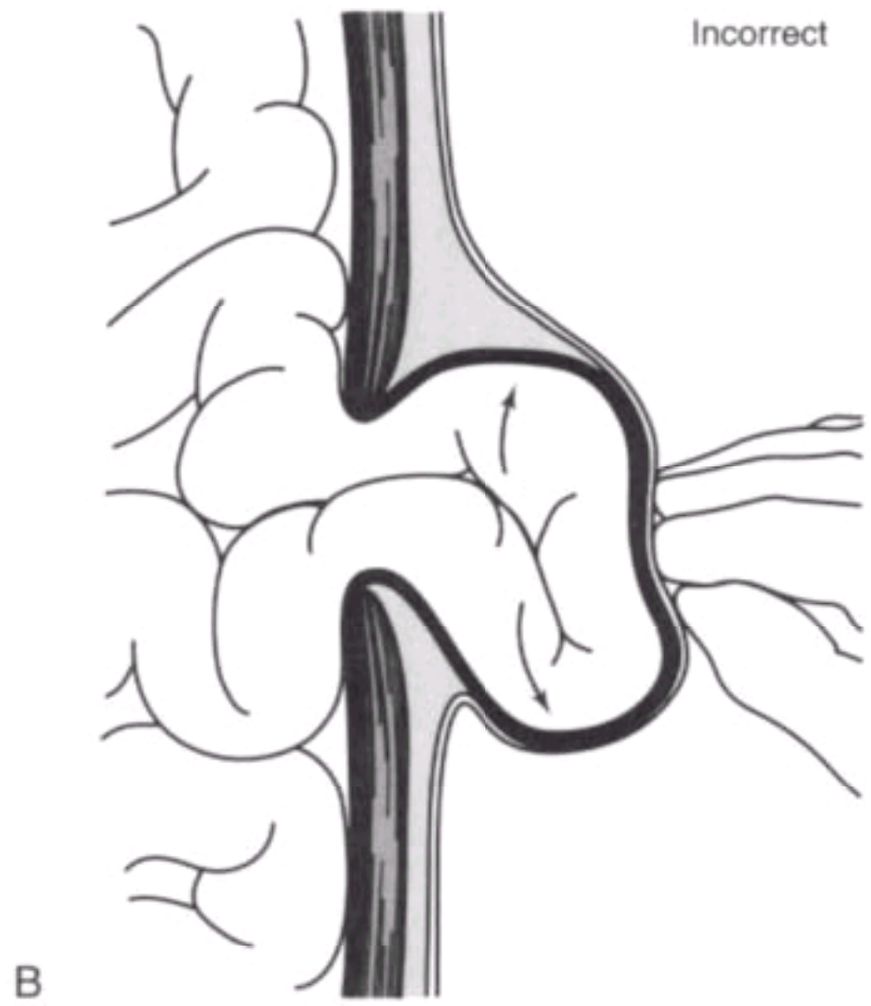

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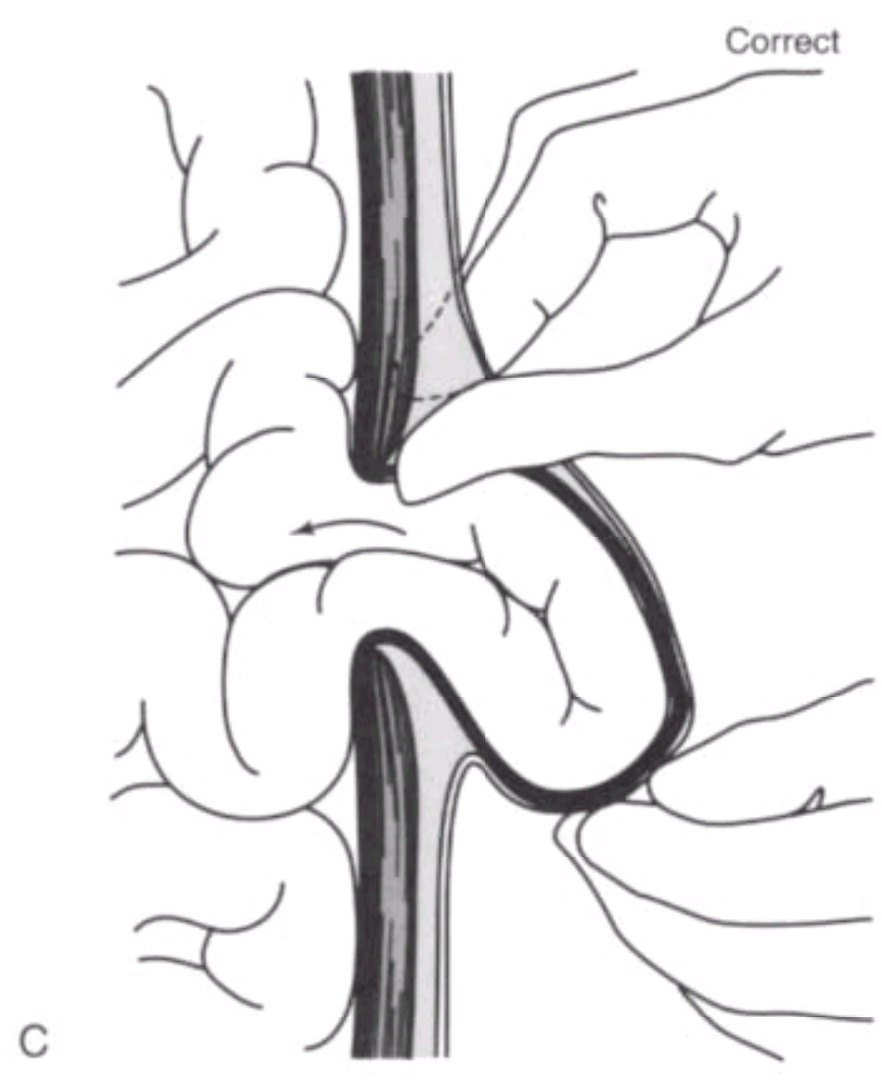


C

Figure 45-9 *A,* This patient presented with signs and symptoms of a bowel obstruction. It was not until the surgery consultant removed the patient's pants that an inguinal hernia was found to be the cause of the obstruction. *B,* After intravenous analgesia/sedation, the inguinal ring is stabilized with the left hand. *C,* Slow gentle, yet persistent pressure is applied to reduce the hernia. It required 5 minutes. The hernia was reduced. Although this was a reducible hernia, because of the bowel obstruction this patient should be admitted to the hospital for observation and next day surgery.

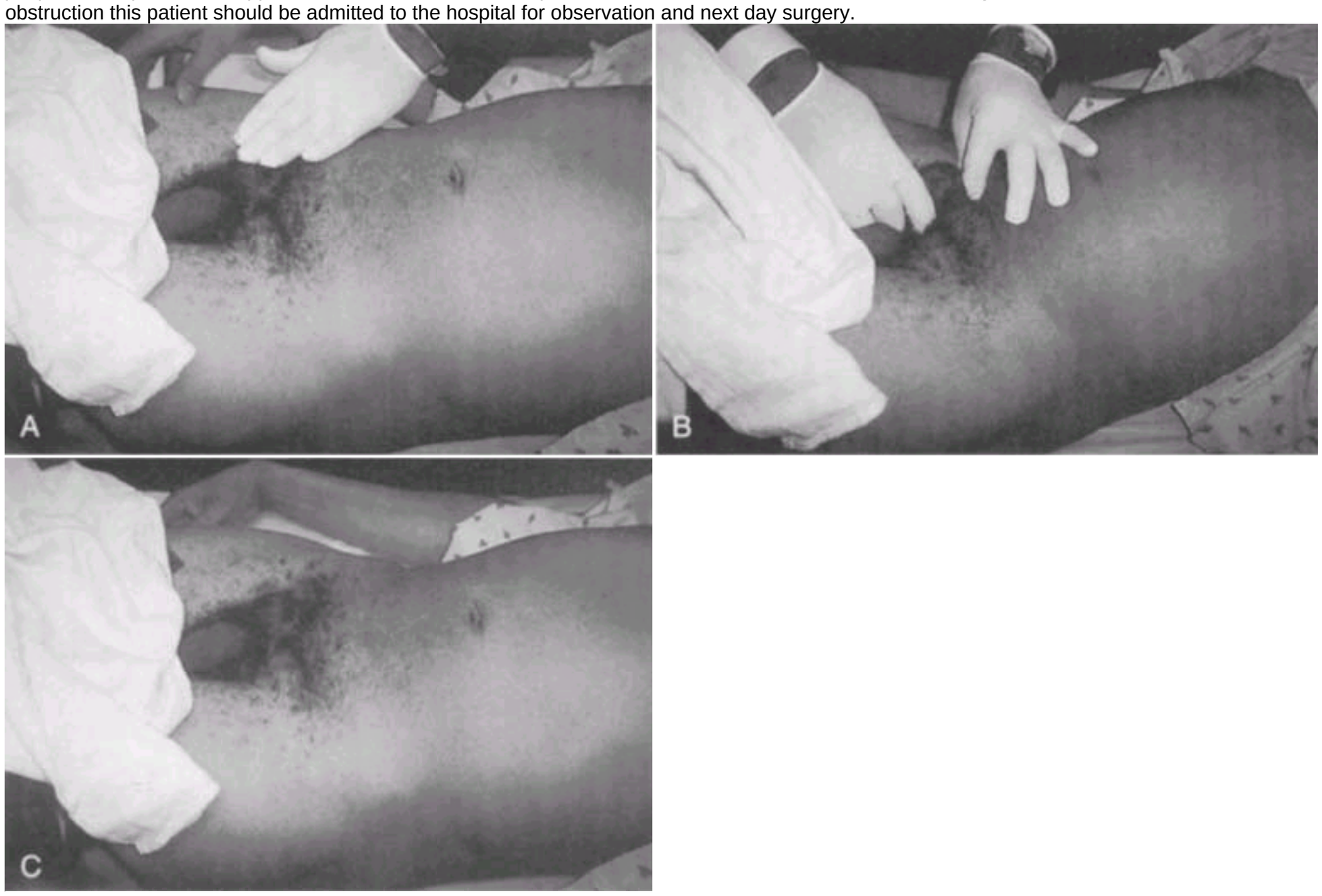


Figure 45-10 Frog leg technique.

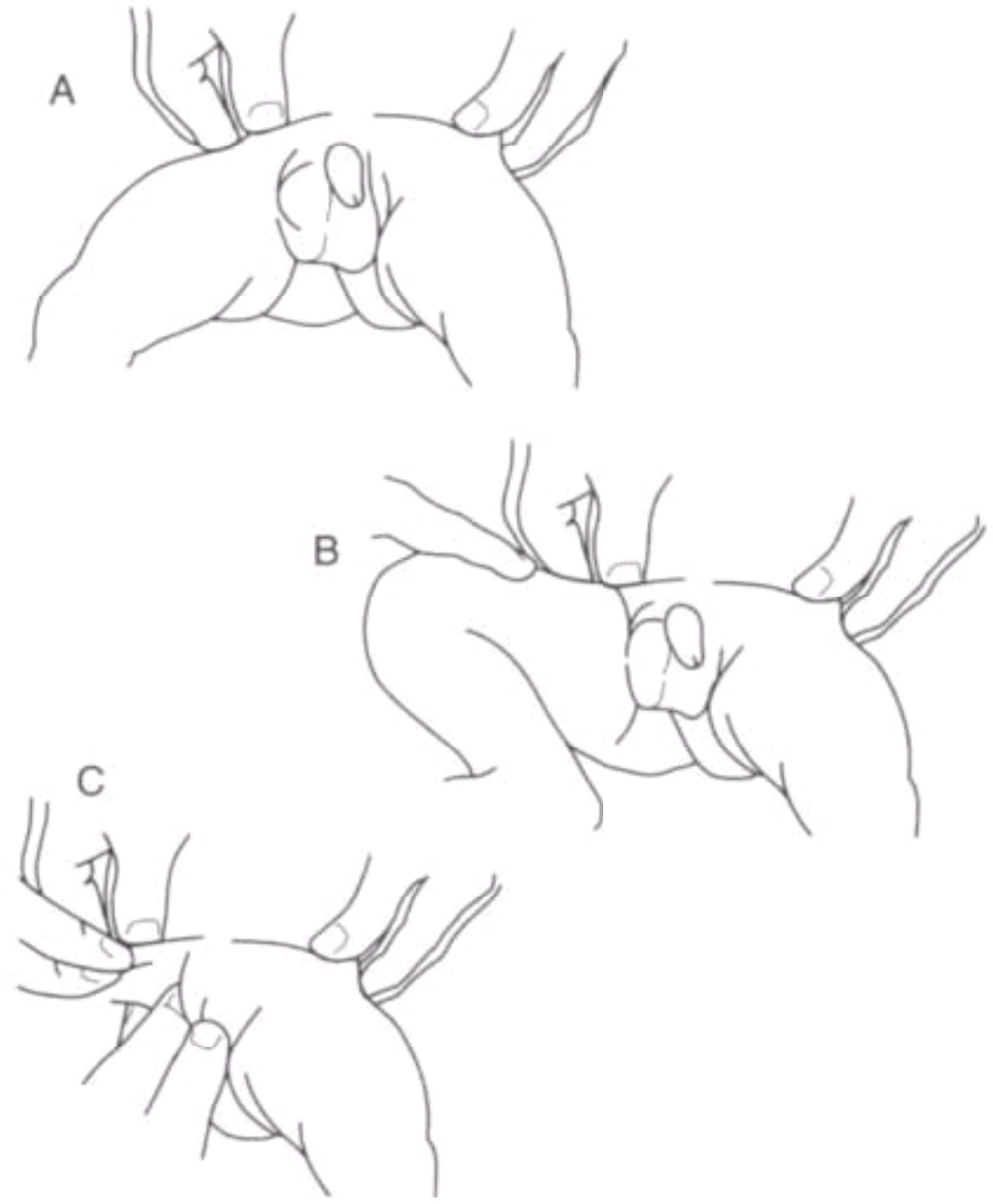


Figure 46-1 Anatomy of the terminal gastrointestinal tract. *(Redrawn from Abrahams PH, Webb PJ: Clinical Anatomy of Practical Procedures. London, Pitman, 1975.)*

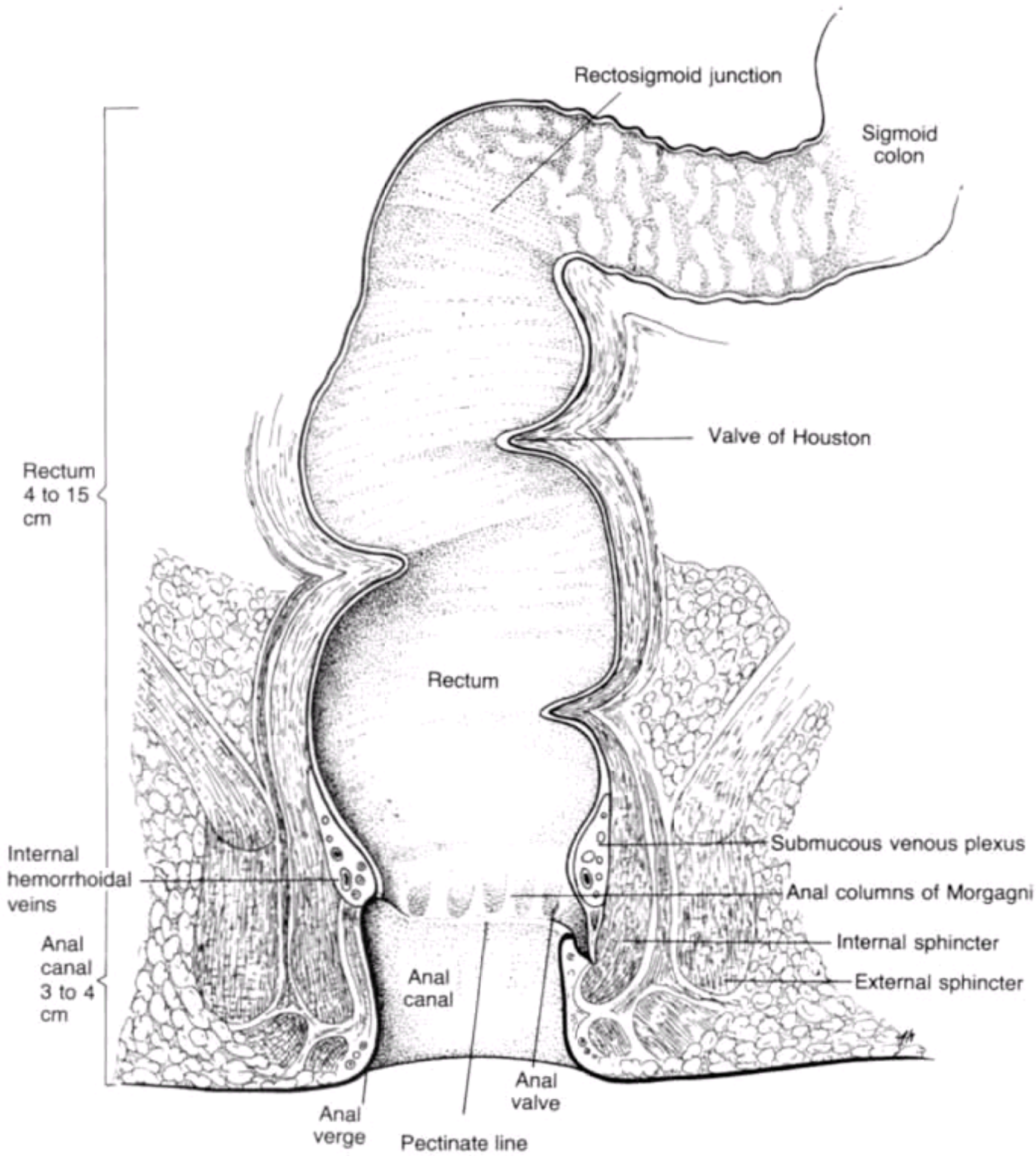


Figure 46-2 Stainless steel anoscope. *A,* Anoscope with obturator. *B,* Anoscope prepared for insertion.

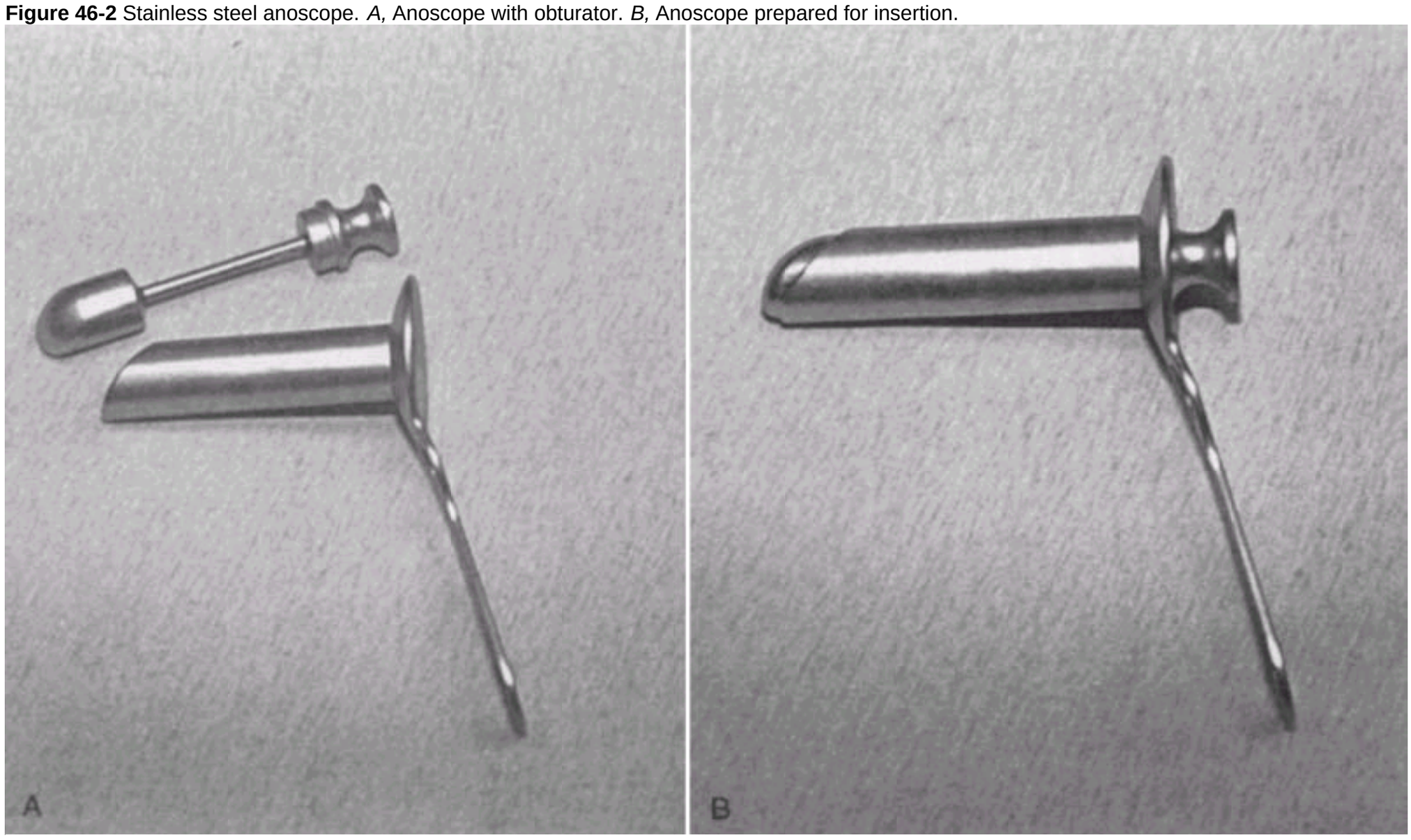


Figure 46-3 Anoscope with integrated light source. *A,* Scope with obturator in place for insertion. *B,* Scope attached to light source, also with obturator in place. Generally the scope is inserted before attaching it to the light source.

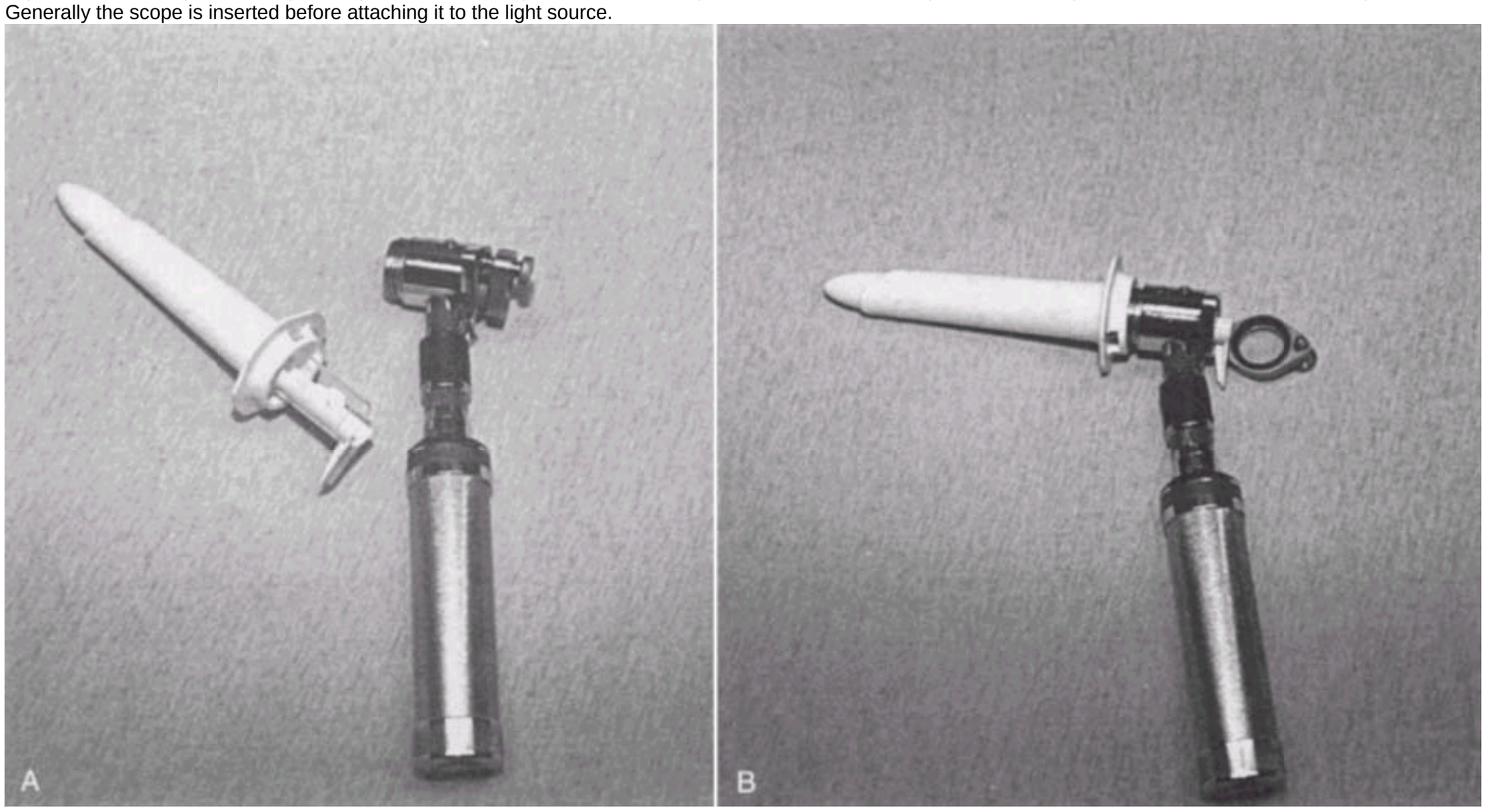


Figure 46-4 Positions for performing sigmoidoscopy or anoscopy. *(From Hill GJ II: Outpatient Surgery, 3rd ed. Philadelphia, WB Saunders, 1988.)*

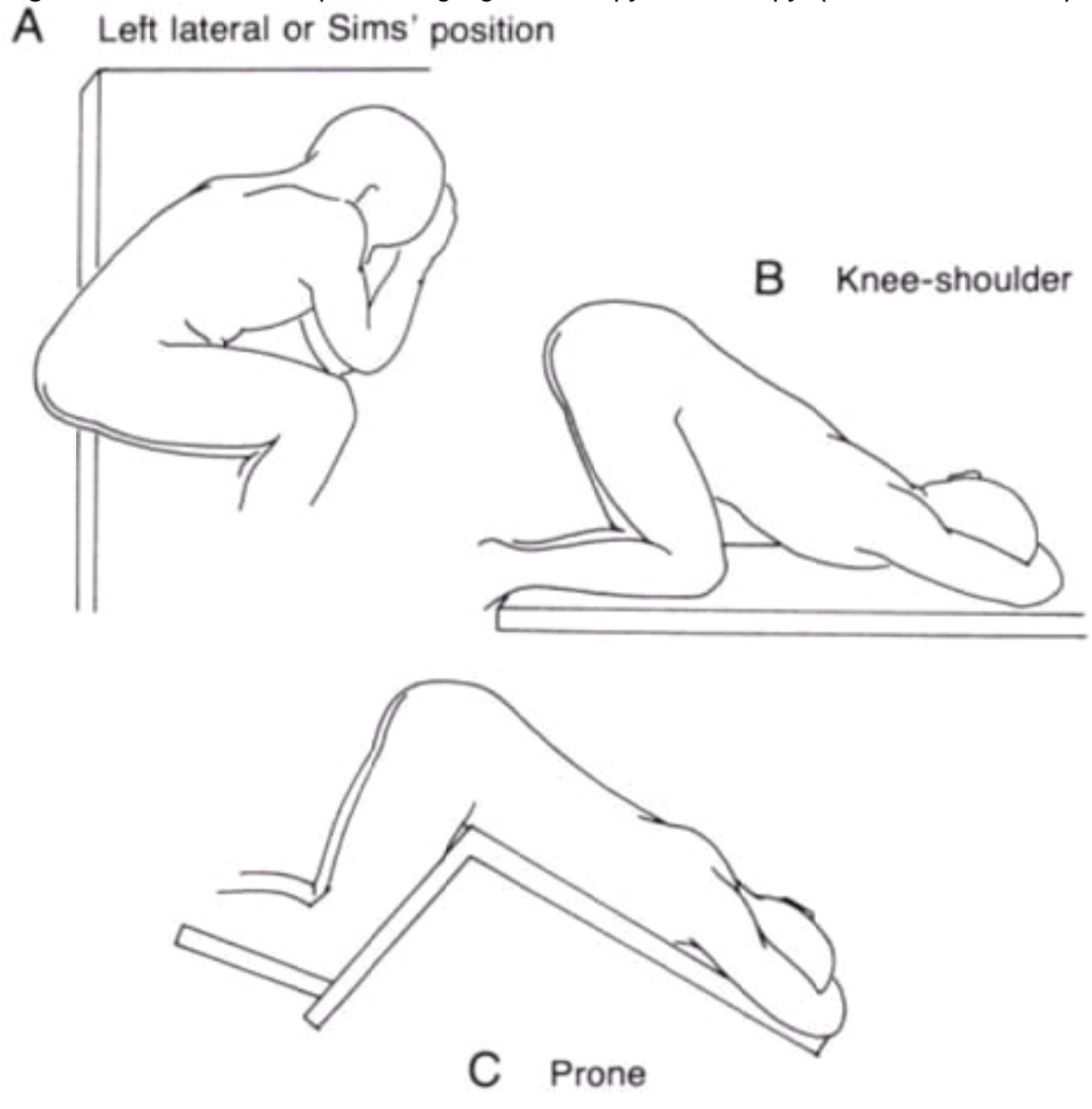


Figure 46-5 Anoscopy. Initially, with the thumb used to keep the obturator fully inserted during passage of the instruments, the anoscope is directed toward the umbilicus, and the obturator is not withdrawn until the scope has been passed to the hilt. Detailed examination is performed on withdrawal of the instrument. A penlight is used in this illustration for illumination of the deep anus.

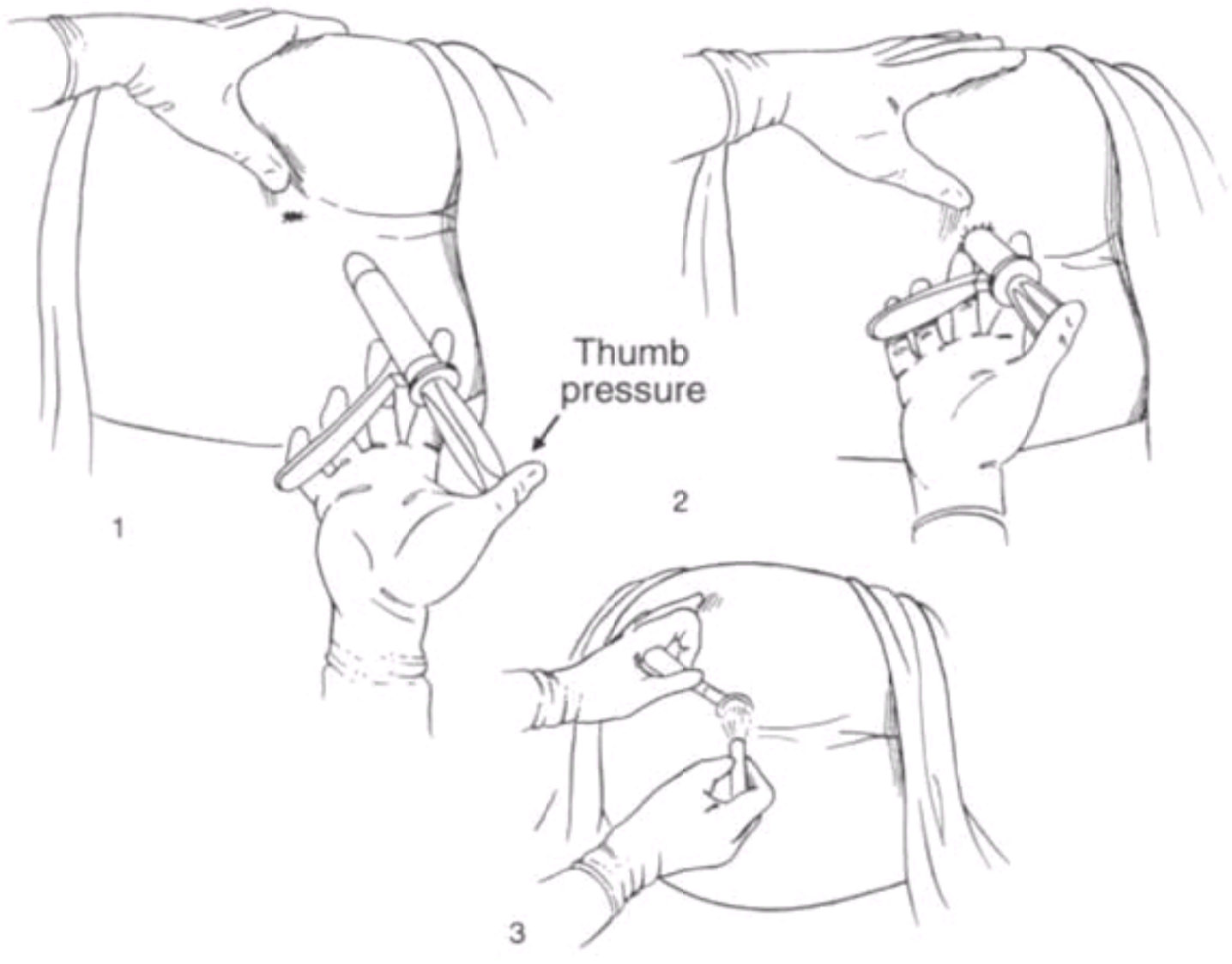
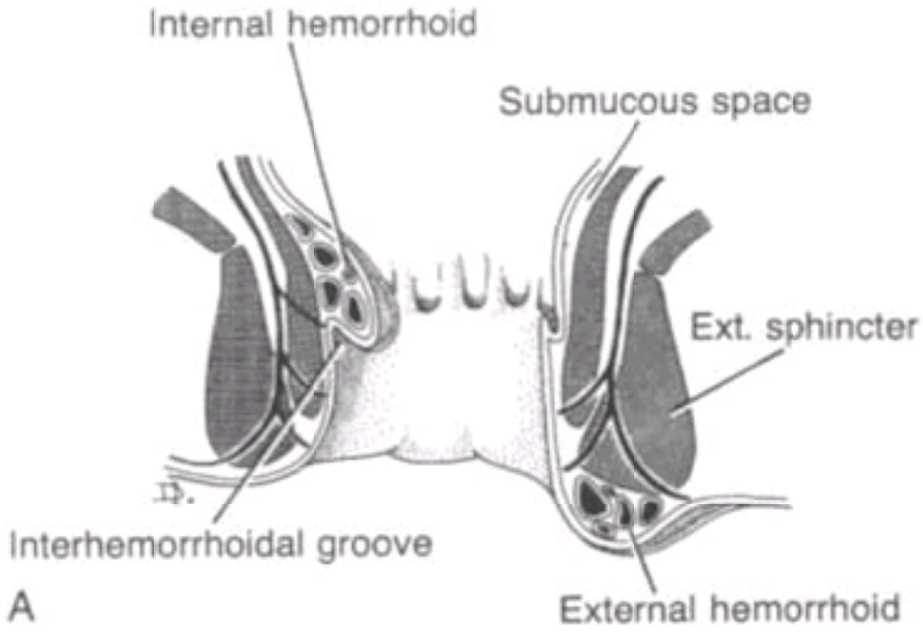
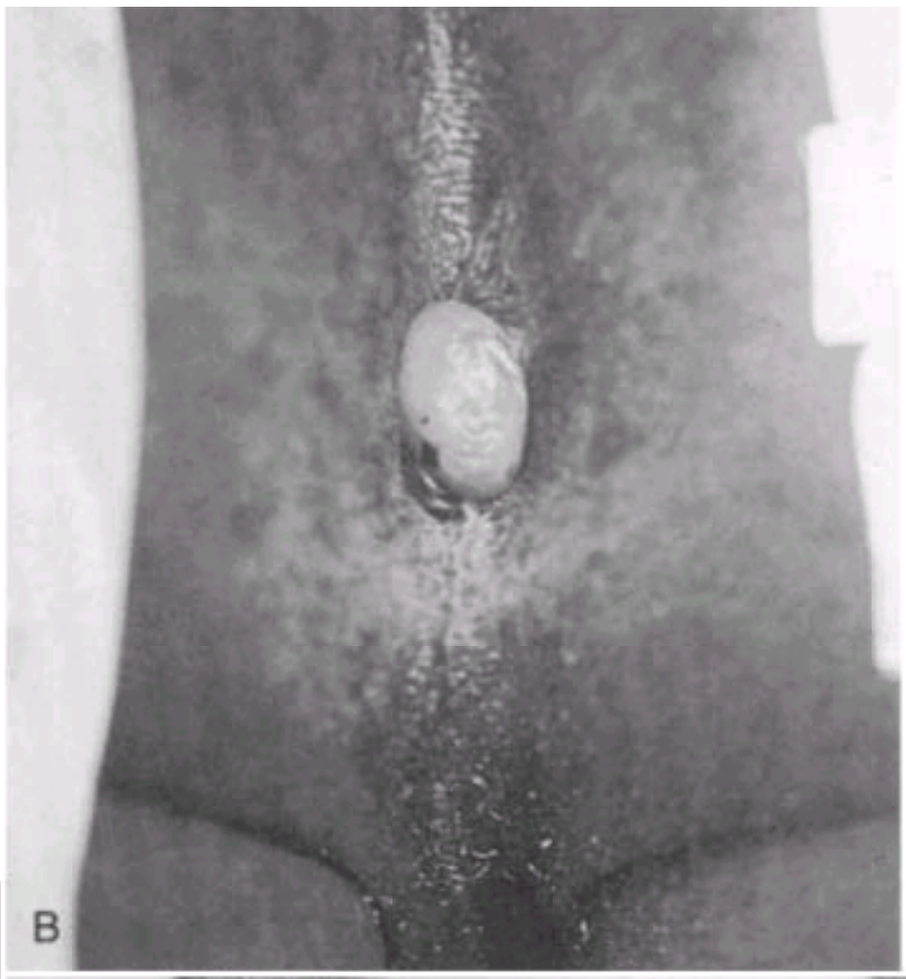
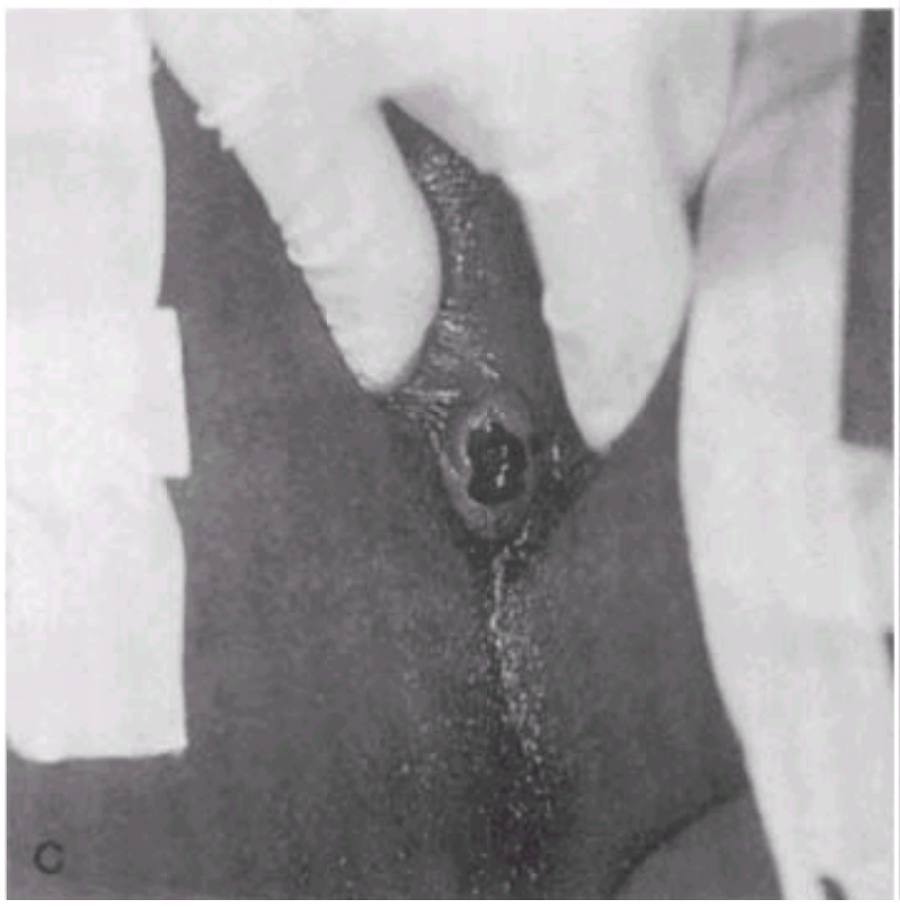


Figure 46-6 *A*, Anatomic location of internal and external hemorrhoids. *B*, Large thrombosed external hemorrhoid. *C*, This thrombosed external hemorrhoid spontaneously ruptured. This demonstrates the concept that a thrombosed hemorrhoid is actually a hematoma into soft tissues. *D*, Thrombosed prolapsed internal hemorrhoids. These hemorrhoids cannot be reduced and are quite painful. They should not be incised in the ED, a formal hemorrhoidectomy is required if conservative measures are not successful. They are often mistaken for a partial "rectal prolapse". *(A from Hill GJ II: Outpatient Surgery. 2nd ed. Philadelphia, WB Saunders, 1980. Reproduced by permission.)*







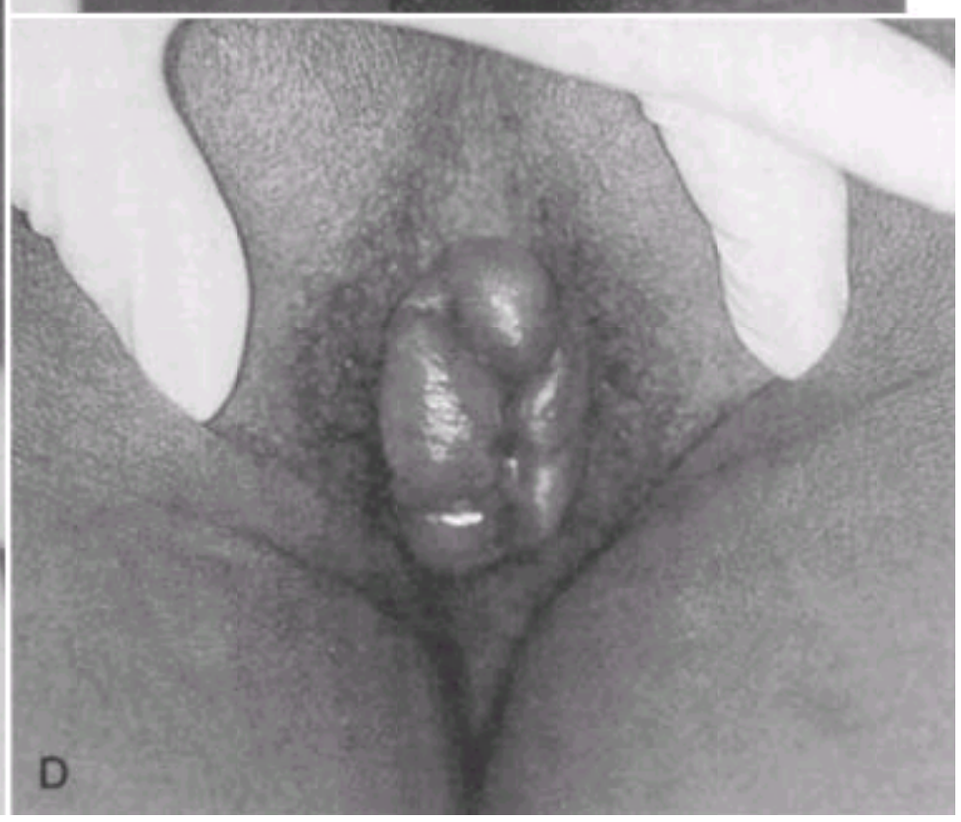


Figure 46-7 Taping the buttocks to gain exposure.

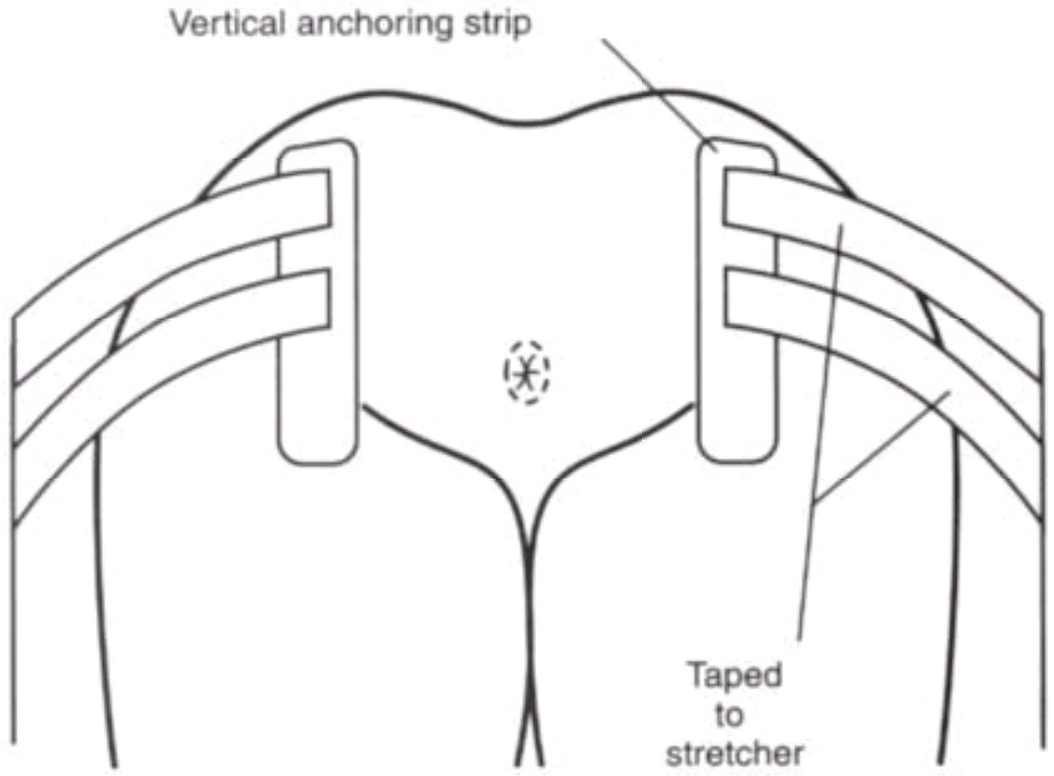


Figure 46-8 Very adequate anesthesia often can be obtained by a single injection of long-acting bupivacaine. *A,* A 25-ga needle is inserted in the middle of the swollen hemorrhoid, just below the skin surface. *B,* With the injection, the anesthetic spreads over the surface of the dome and into the surrounding tissue. A field block about the hemorrhoid can also be used, but it is much more painful, usually not required, and not advised as a first attempt at anesthesia.

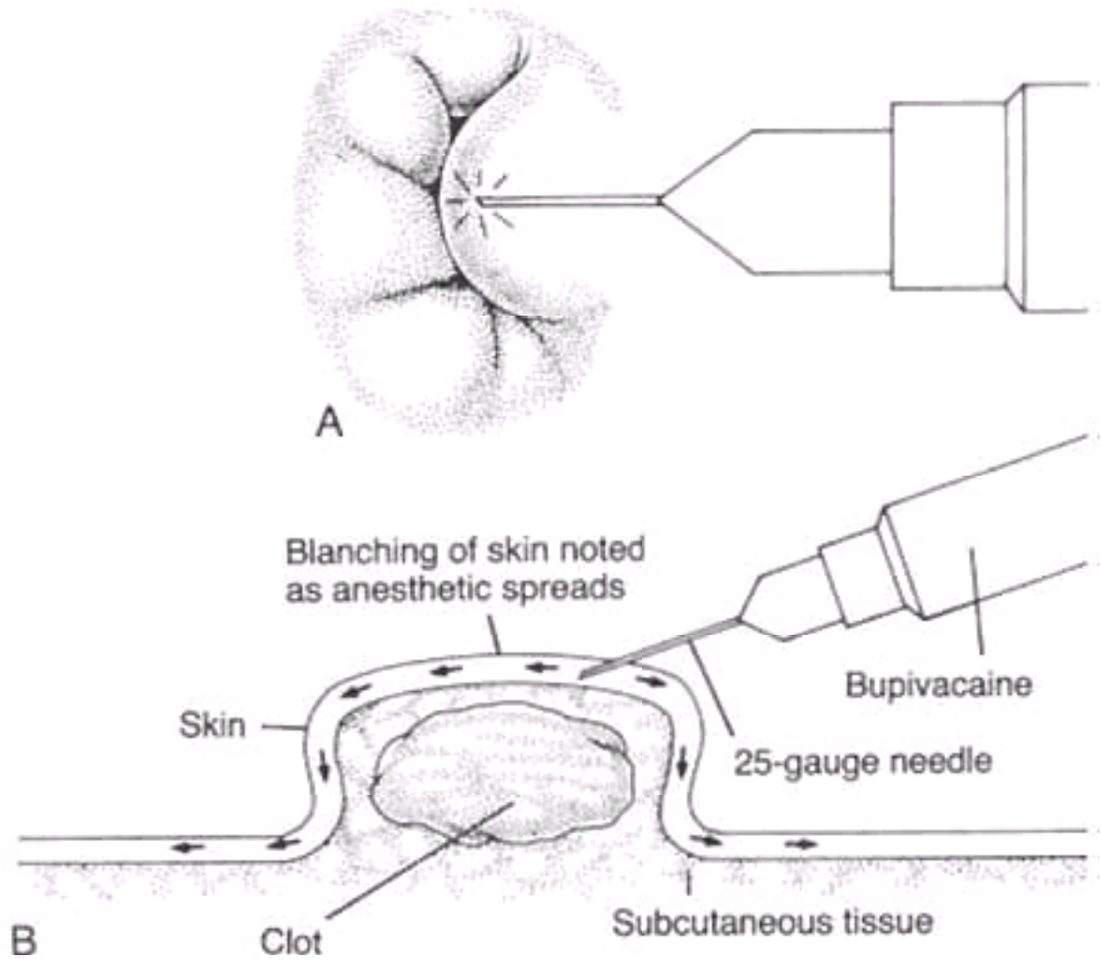


Figure 46-9 Subcutaneous injection of a long-acting local anesthetic using a field block will provide complete anesthesia during a procedure, but is rarely required.

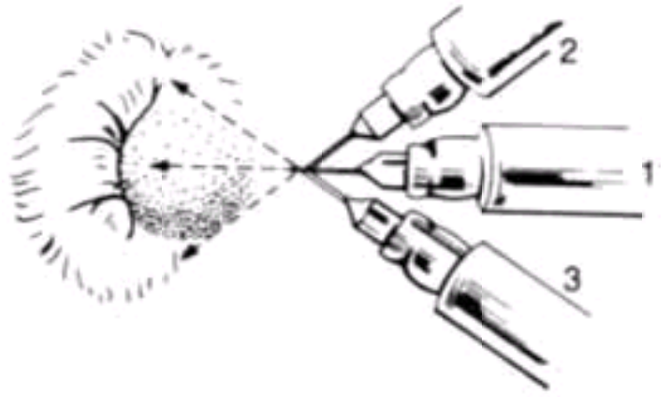


Figure 46-10 An unroofing technique uses an elliptic or triangular incision that removes a piece of the overlying skin. A simple linear incision should not be used.

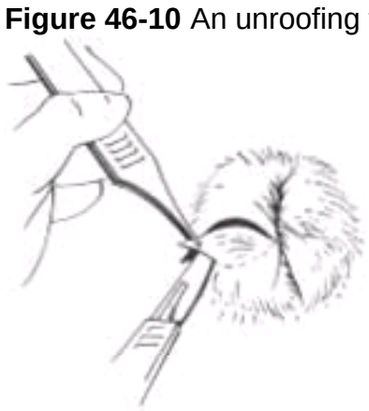


Figure 46-11 Blood clots are removed with forceps *(A)* or expressed with the fingers *(B)*. Often multiple clots are present, and all should be removed. *C,* After the initial clot is removed, an assistant spreads the incision to expose the base of the hemorrhoid and allow other smaller clots to be removed individually with forceps or with a small hemostat.

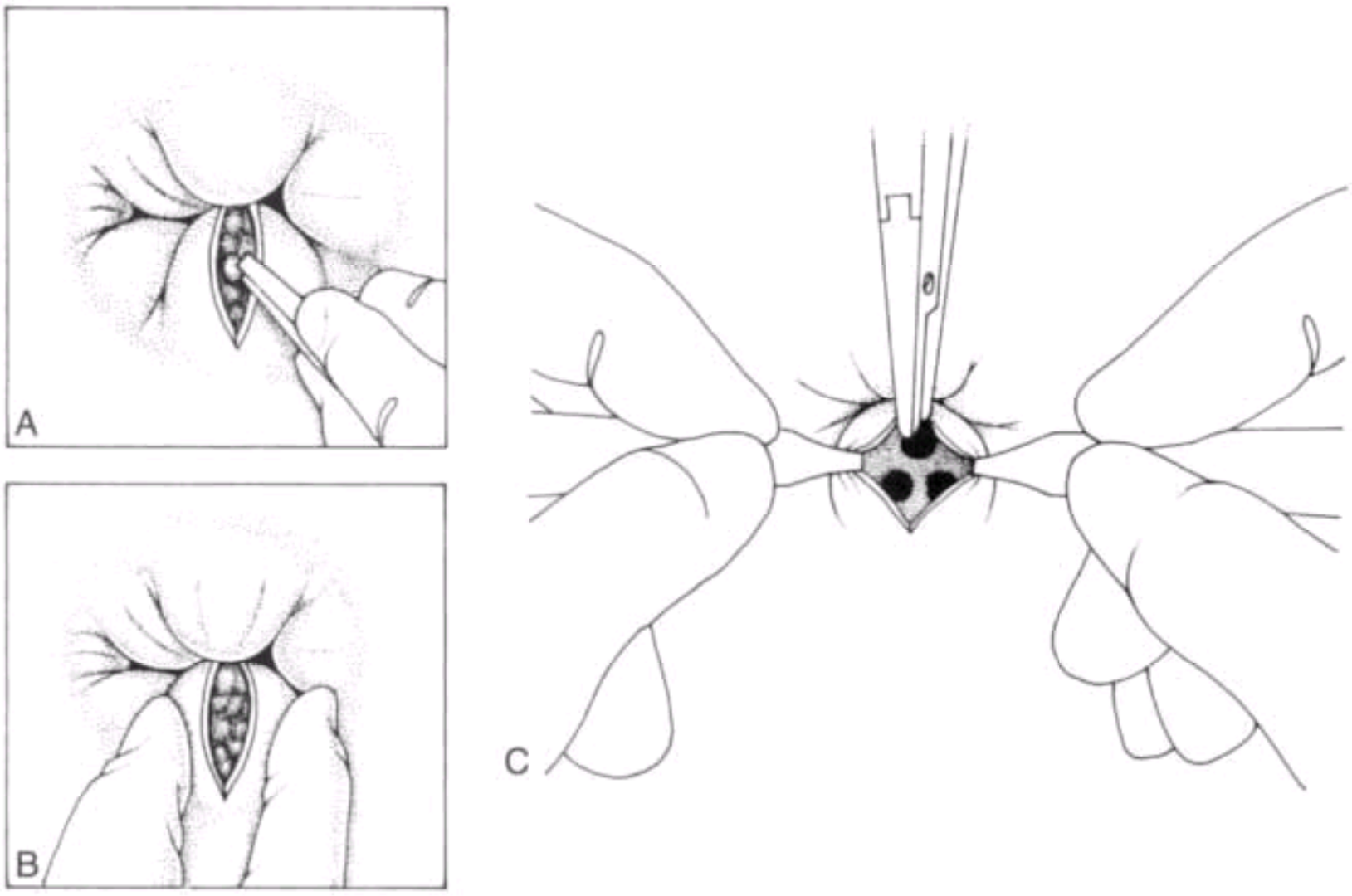


Figure 46-12 Emergency approach to the removal of rectal foreign bodies. Foreign bodies that are fragile or are associated with rectal spasm are generally managed with regional or general anesthesia. The use of supplemental analgesic, anxiolytic, and local anesthetic medications is highly recommended.

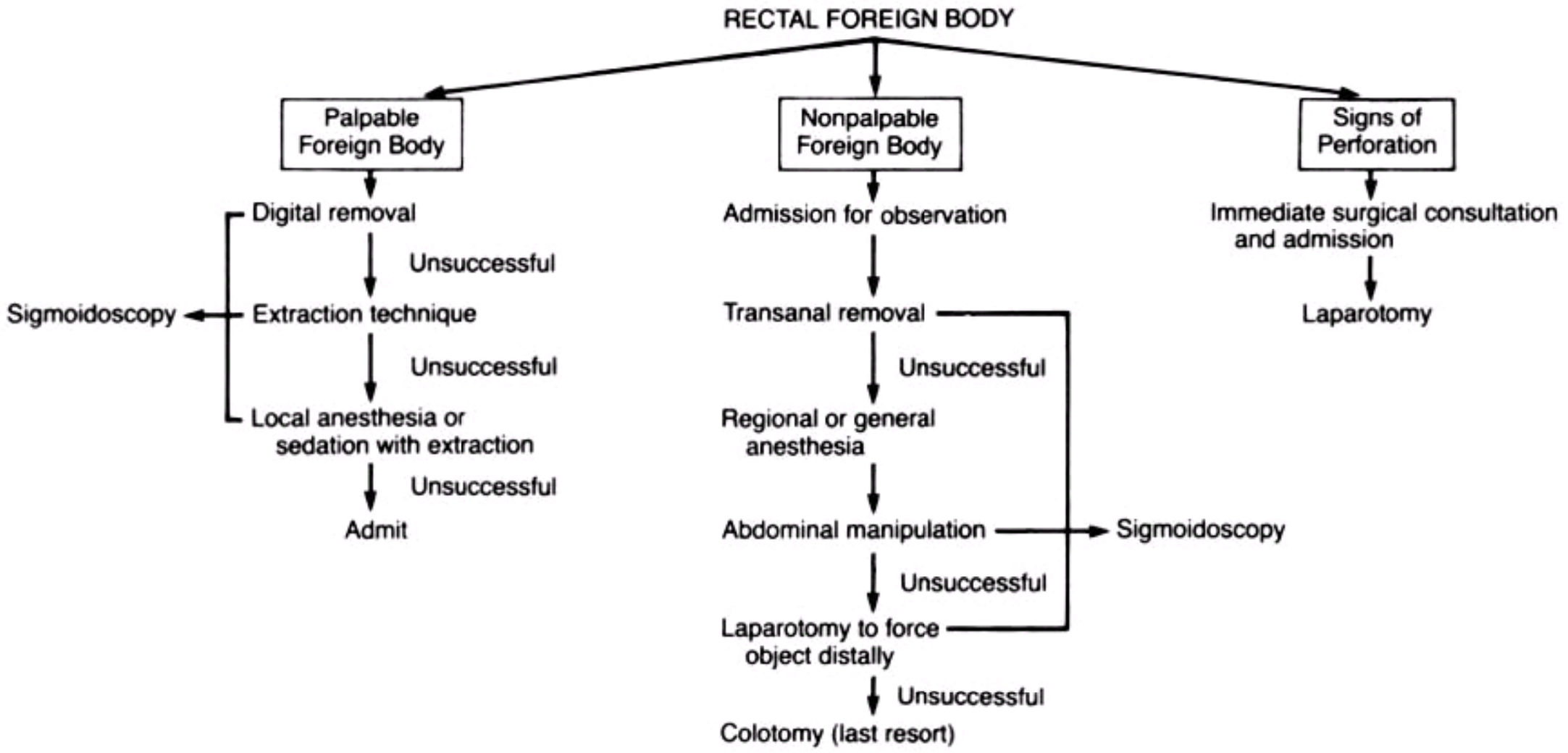
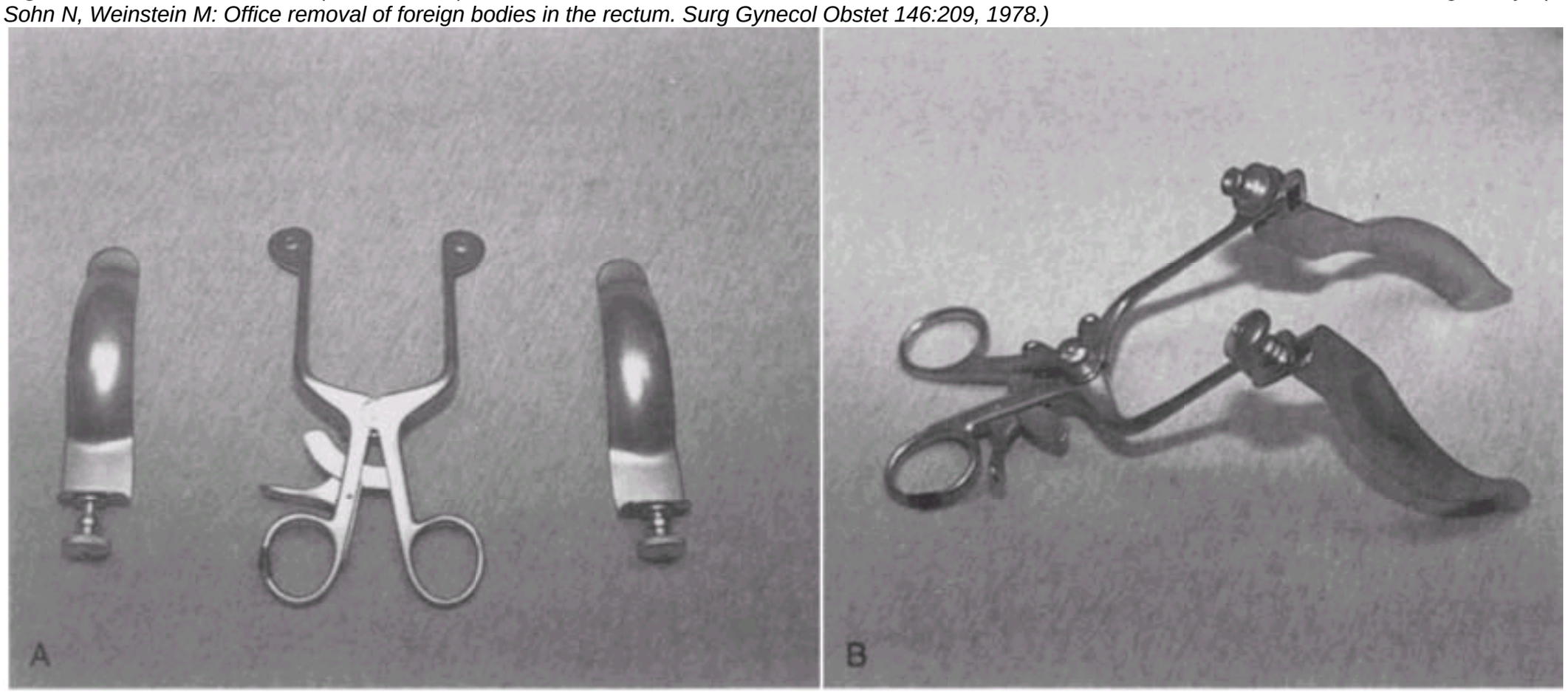


Figure 46-13 *A,* Parks retractor (unassembled). *B,* Assembled Parks retractor. *C,* A Parks retractor inserted into the anal canal to visualize the foreign body. *(C from Sohn N, Weinstein M: Office removal of foreign bodies in the rectum. Surg Gynecol Obstet 146:209, 1978.)*



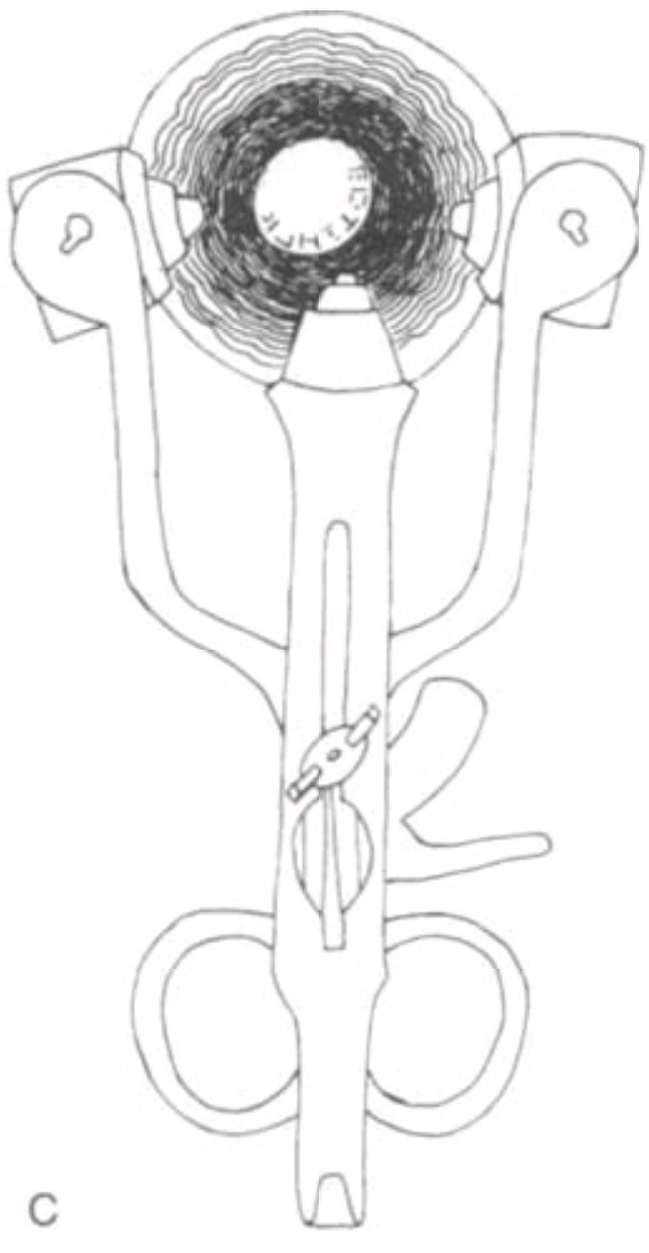


Figure 46-14 Use of Foley catheters to remove a rectal foreign body made of glass. *(From Eftaiha M, Hambrick E, Abcarian H: Principles of management of colorectal foreign bodies. Arch Surg 112:693, 1977.)*

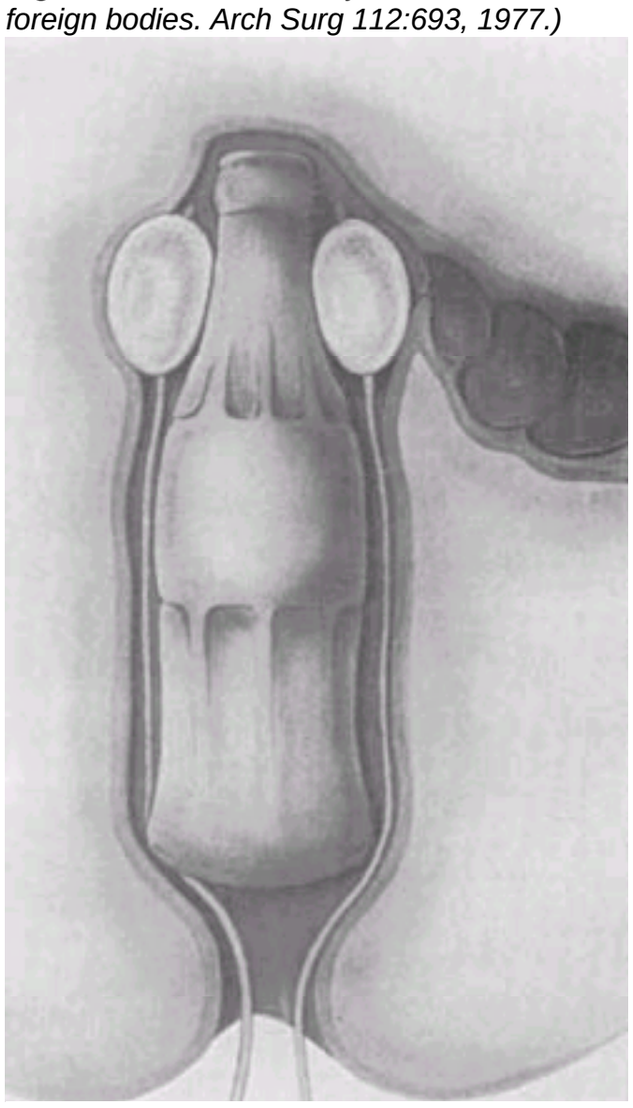


Figure 46-16 *A,* Type I procidentia (rectal prolapse). *B,* Intussusception of sigmoid colon beyond the anus. *(From Kratzer GL, Demarest RJ: Office Management of Colon and Rectal Disease. Philadelphia, WB Saunders, 1985, pp 221–333.)*

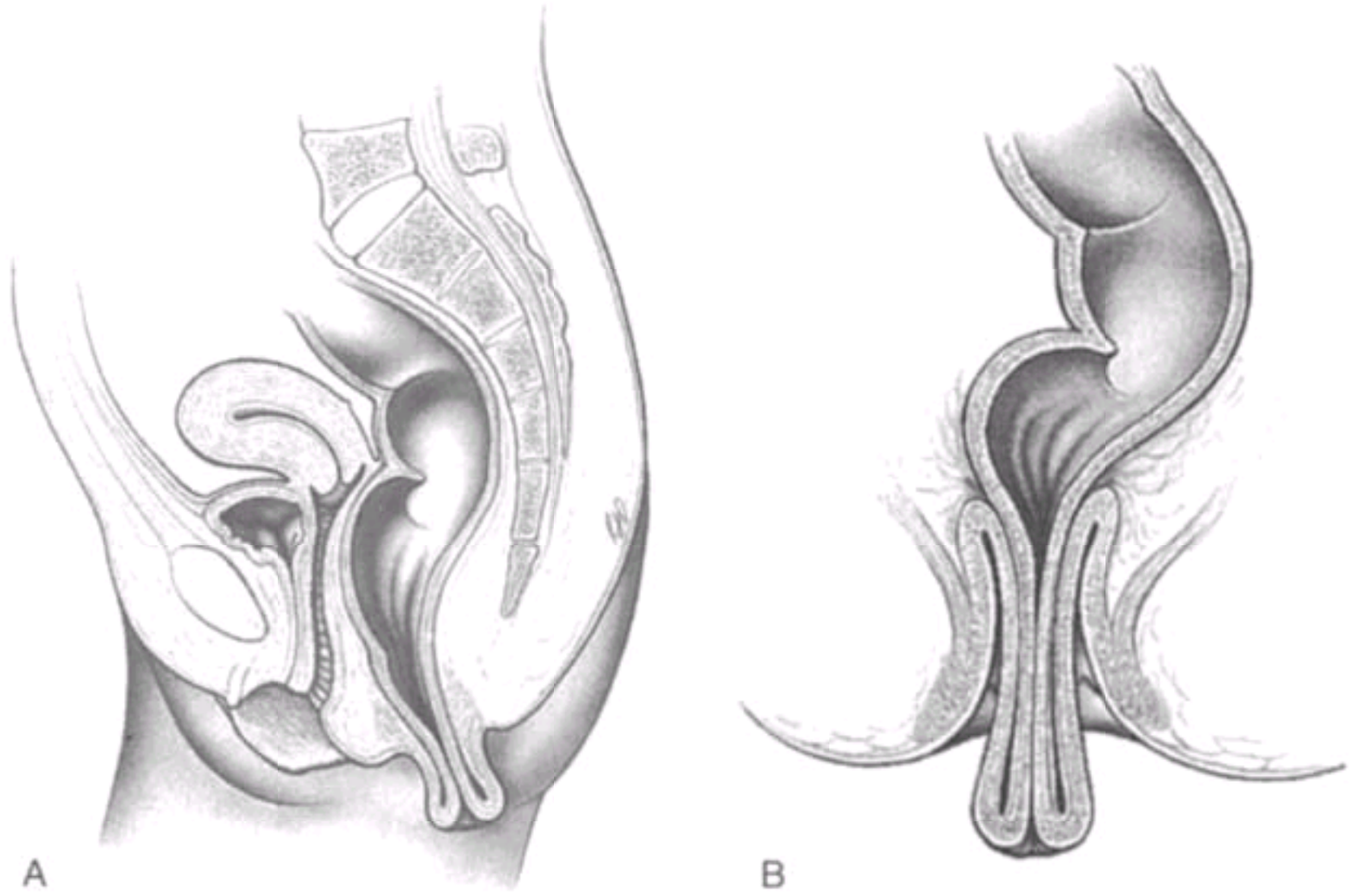
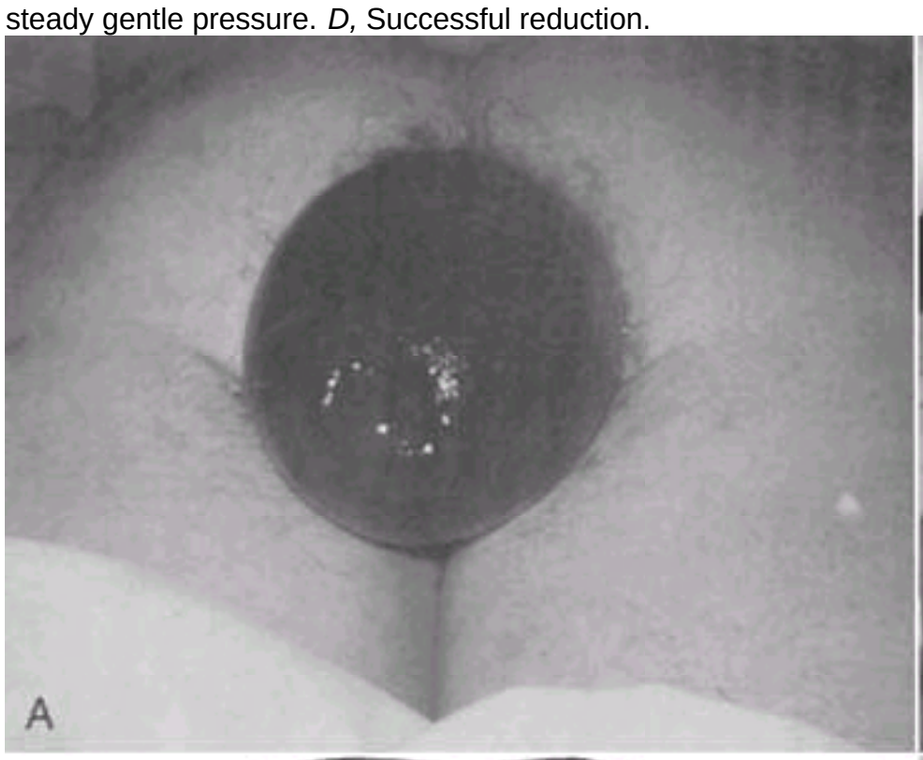
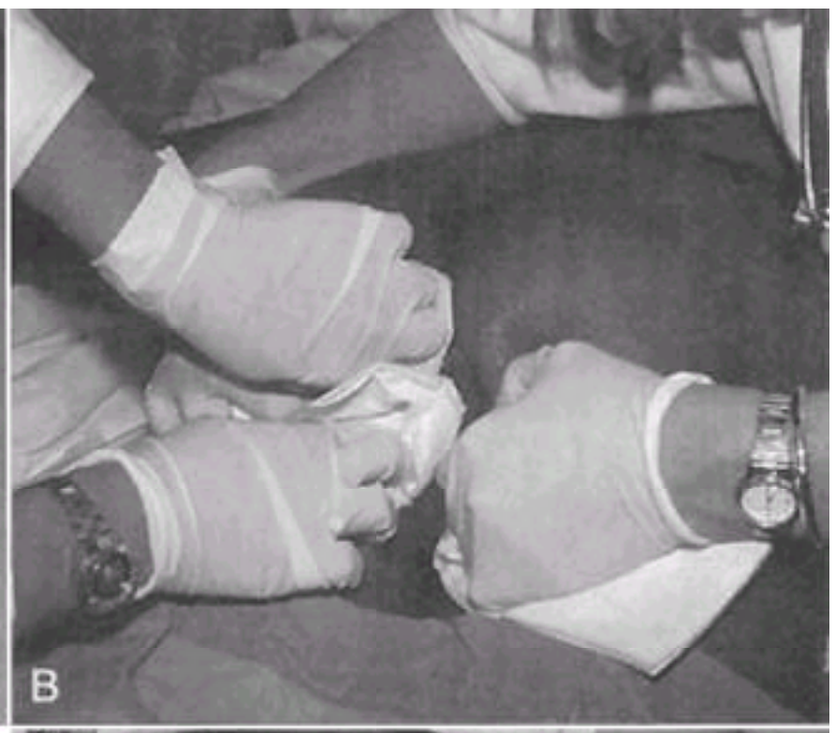


Figure 46-17 *A,* Complete (recurrent) rectal prolapse in a nursing home patient. This can be very distressful and painful, or relatively asymptomatic. *B and C,* To reduce the prolapse, an assistant spreads the buttocks and the clinician's hands encircle the prolapse. The key to success is adequate sedation/analgesia and a slow steady gentle pressure. *D,* Successful reduction.





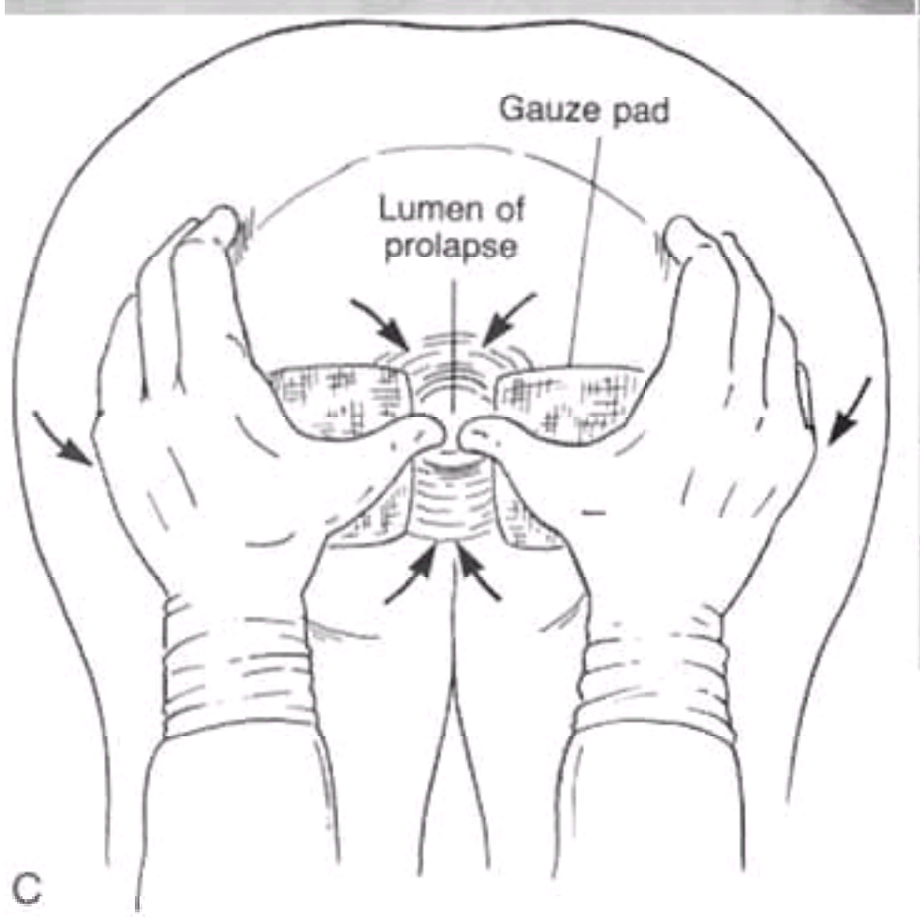


Figure 47-1 Philadelphia collar. This is a two-piece, high-type collar that comes in four sizes. The collar supports the head in a dish-shaped contour that is formed when the front and rear halves are joined by Velcro fasteners. When properly sized for a patient, this collar provides excellent support. When applied too tightly, it tends to force the mandible backward and can cause thyroid compression in some patients. It is extremely comfortable. *(Courtesy of Philadelphia Cervical Collar Company, Westville, NJ.)*

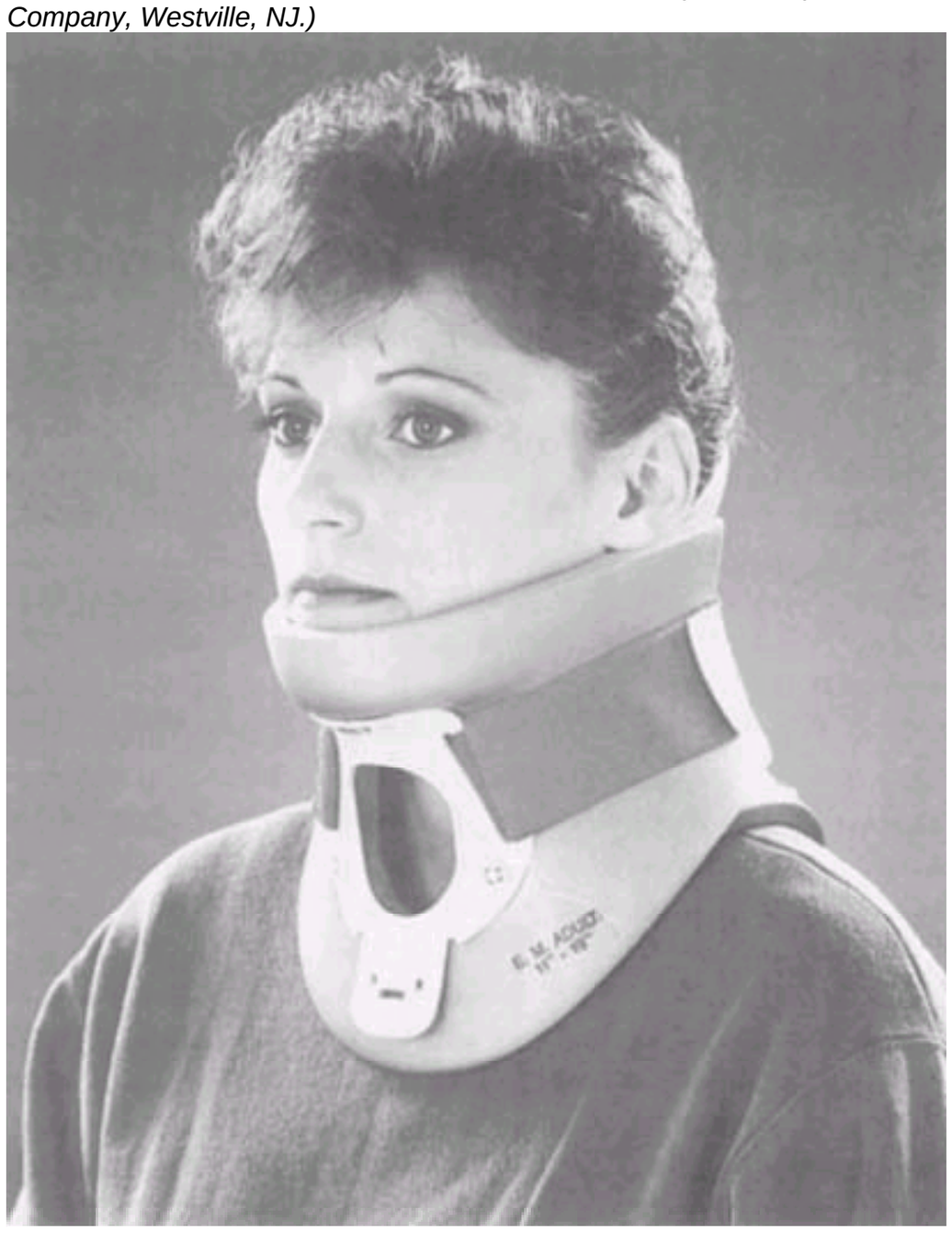


Figure 47-2 Stifneck collar. This collar is made of high-density polyethylene (a hard material) and padded with semiflexible foam margins. Note the low-reaching anterior panel, which contacts the sternum for additional support. *(Courtesy of Laerdal Medical Corporation, Wappingers Falls, NY.)*

Figure 47-3 *A,* Young child immobilized on a standard backboard; note how the large head forces the neck into flexion. Backboards can be modified by an occiput cutout (*B*) or a double mattress pad (*C*) to raise the chest. *(Adapted from Herzenberg JE, Hensinger RN, Dedrick DK, et al: Emergency transport and positioning of young children who have an injury of the cervical spine. J Bone Joint Surg Am 71:15, 1989.)*

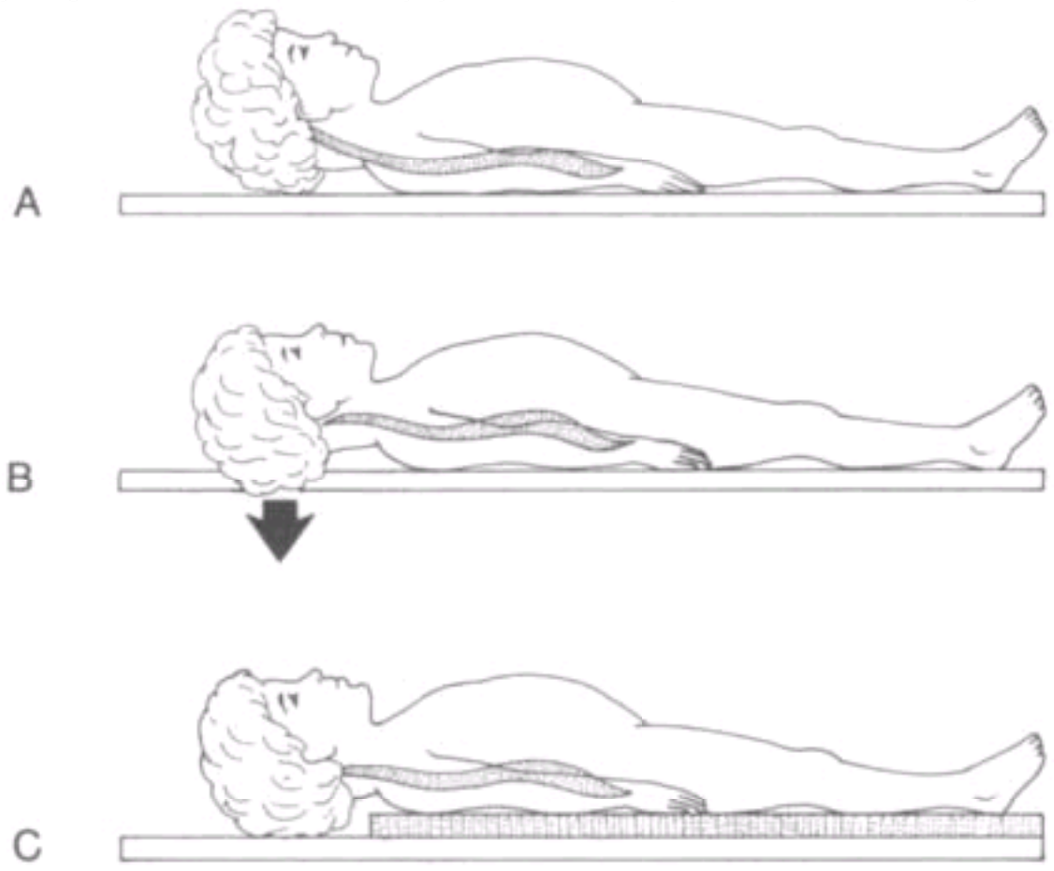


Figure 47-4 Pediatric high-cut extrication collar. *(Courtesy of Laerdal Medical Corporation, Wappingers Falls, NY.)*

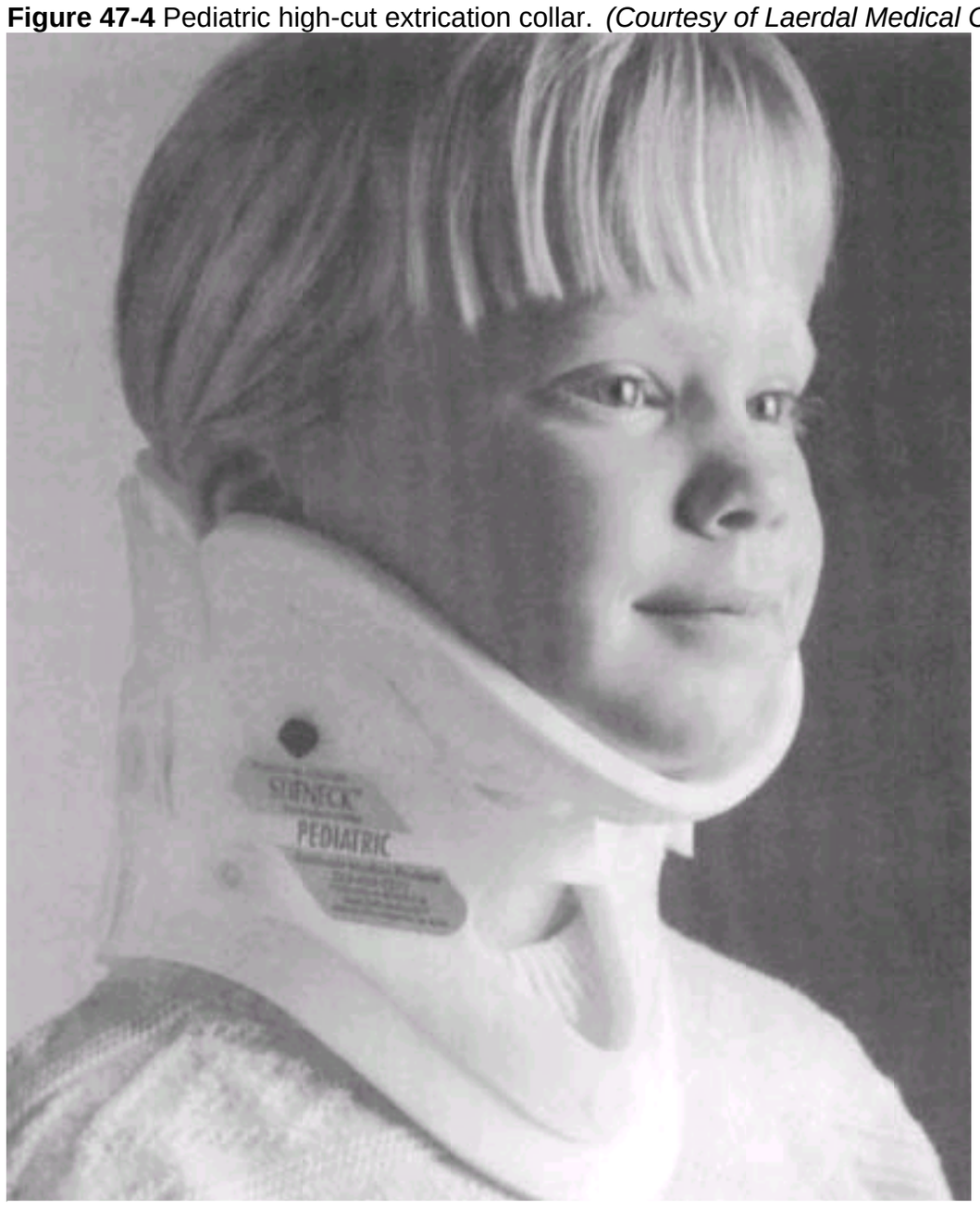
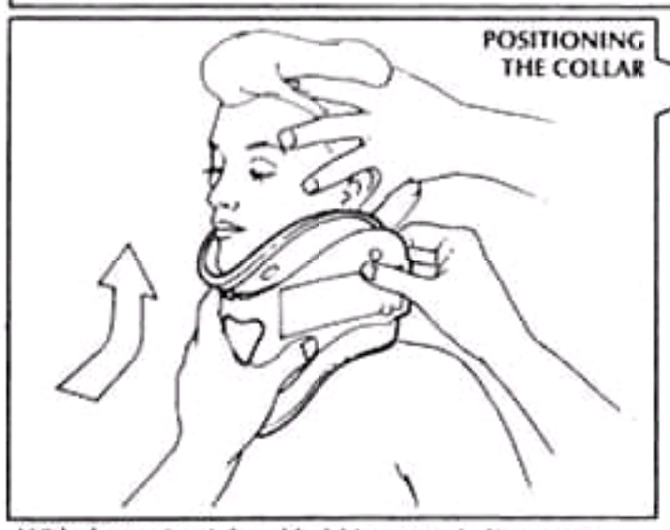


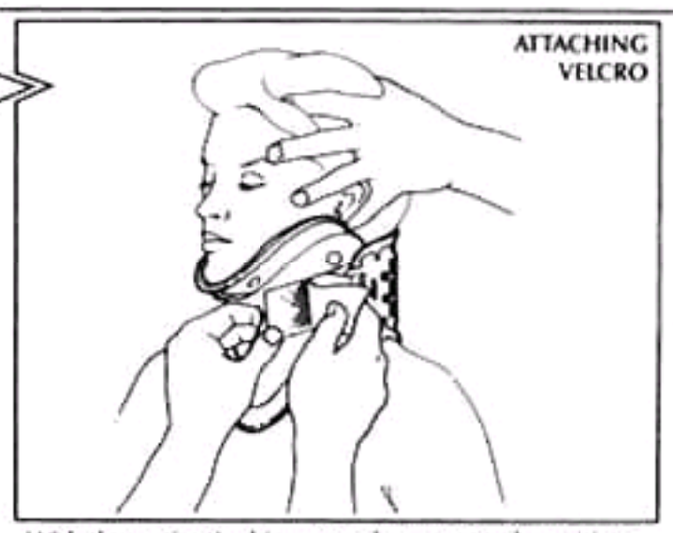
Figure 47-5 LSP pediatric immobilization board with cervical collar. *(Courtesy of Allmed, St. Louis.)*

Figure 47-6 Technique for the application of an extrication collar. *(Courtesy of Laerdal Medical Corporation, Wappingers Falls, NY.)*

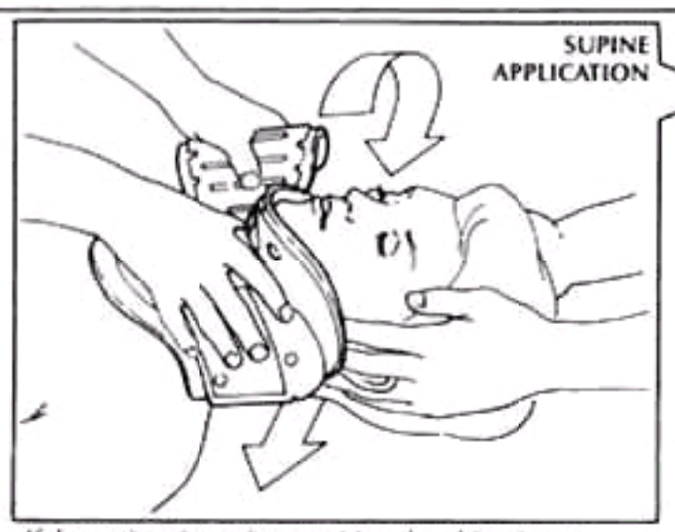
APPLICATION



With the patient's head held in neutral alignment, position the chin piece by sliding the collar up the chest wall. Be sure that the chin is well supported by the chin piece and that the chin extends far enough onto the chin piece to at least cover the central fastener. Difficulty in positioning the chin piece may indicate the need for a shorter collar.



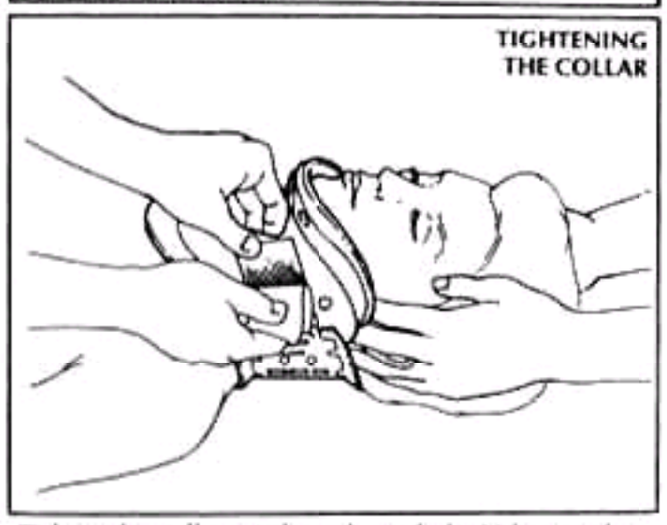
With the patient's chin properly supported, position the back of the collar around the neck and attach the loop velcro so that it mates with, and is parallel to, the hook velcro. Use the trach hole to keep the collar in proper position as you tighten the velcro. BE SURE TO MAINTAIN NEUTRAL ALIGNMENT THROUGHOUT THIS PROCEDURE.



If the patient is supine, position the chin piece as previously described and slide the back portion of the collar behind the patient's neck. Be sure to fold the loop velcro inward on top of the foam padding to prevent it from collecting debris which could limit its gripping ability.

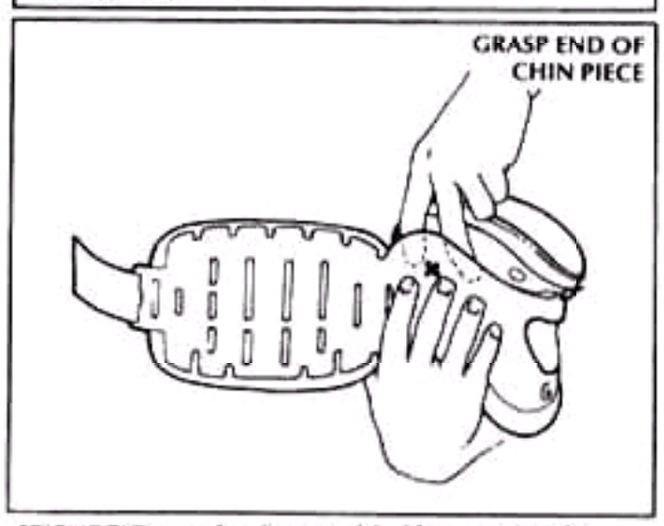
An alternative is to begi
of the collar behind the
velcro is again visible,
positioning the chin pie
described.

APPLICATION



Tighten the collar gently and attach the Velcro so that the two pieces are parallel. Re-check the position of the patient's head and collar for proper alignment. Tighten the collar further until proper support is obtained.

DISASSEMBLY



STIFNECK™ may be disassembled by grasping the end of the chin piece, as shown, with the black fastener between the fingers and working this fastener out of its hole. Do not try to pull the white fastener apart.

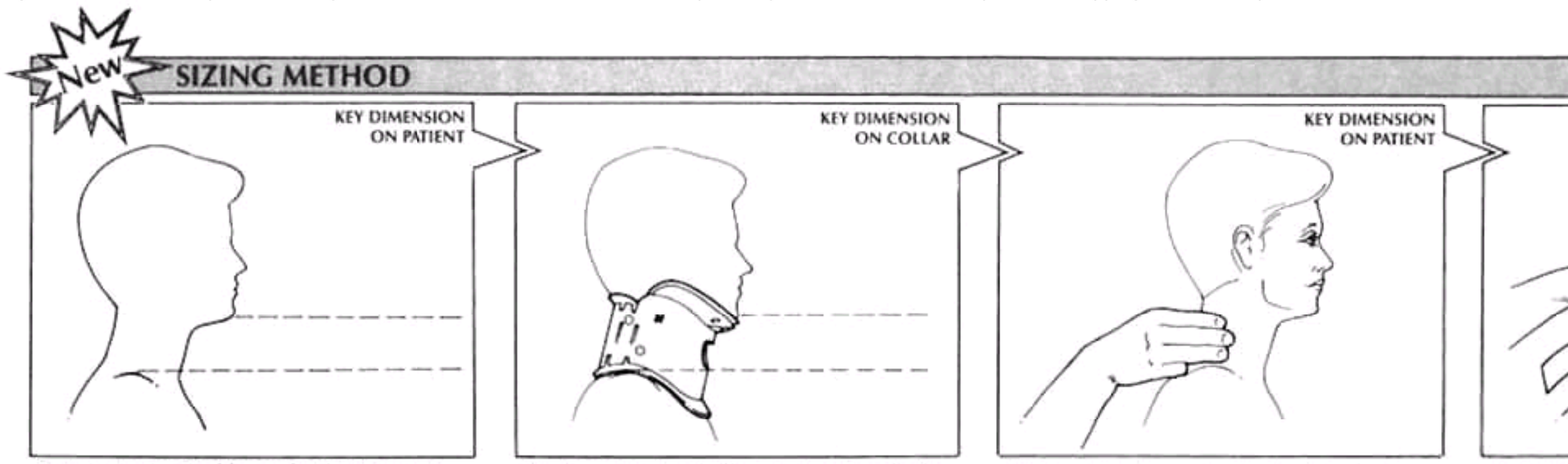
DO'S

1. DO follow the directions of your local EMS authorities for the approved use of extrication collars.
2. DO use all appropriate cervical spine immobilization techniques.
3. DO refer to your EMT paramedic training manual regarding the use of extrication collars and spinal immobilization techniques.

DON'TS

1. DO NOT rely on any cervical collar to adequately immobilize a patient's cervical spine. Collars are tools to aid in immobilization, but no collar by itself provides sufficient immobilization.
2. DO NOT use an improperly sized collar. Too large a collar may hyperextend a patient's cervical spine; too small a collar may not provide appropriate stability.
3. DO NOT hesitate to contact your local EMS dealer or local EMS authority with questions regarding the use of extrication collars.

Figure 47-7 Method for predetermining the correct size for an extrication collar. *(Courtesy of Laerdal Medical Corporation, Wappingers Falls, NY.)*



Proper sizing is critical for good patient care. Too short a collar may not provide enough support. Too tall a collar may hyperextend. Use the tallest collar that does not hyperextend. The key dimension is the distance between an imaginary line drawn across the top of the shoulders, where the collar will sit, and the bottom of the patient's chin.

This dimension is easily determined on the collar by measuring the distance between this black fastener and the lower edge of the rigid plastic encircling band (not the foam padding).

Use your fingers to visualize the shoulder to chin distance on the patient.

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matches t

Figure 47-8 No-Neck extrication collar. Designed for individuals with extremely short necks. *(Courtesy of Laerdal Medical Corporation, Wappingers Falls, NY.)*

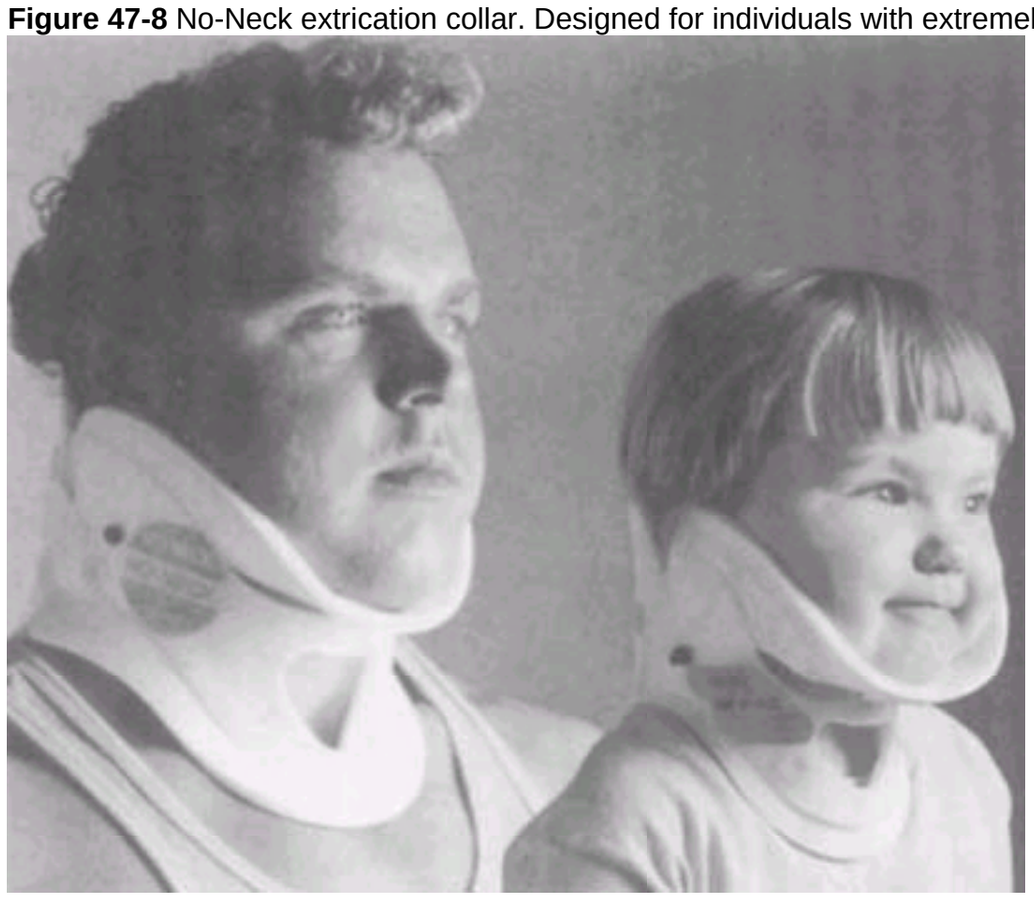


Figure 47-9 Horse collar. Most extrication collars are available in three to five factory sizes. If a collar is not sized properly to fit a particular patient, it performs no function. Patients with extremely long necks or especially short ones can be immobilized by means of a horse collar fashioned from a bulky rescue blanket. The blanket is rolled to the thickness desired and slid under the patient's neck while a bystander applies manual stabilization; the ends of the blanket are then brought across the patient's anterior chest. The patient's forearms are fastened as shown to stabilize the "tails" of the collar. *(From Dick T: Tricks of the trade: Horse sense, immobilizing necks that don't fit collars. J Emerg Med Serv 7:23, 1982. Reproduced with permission.)*

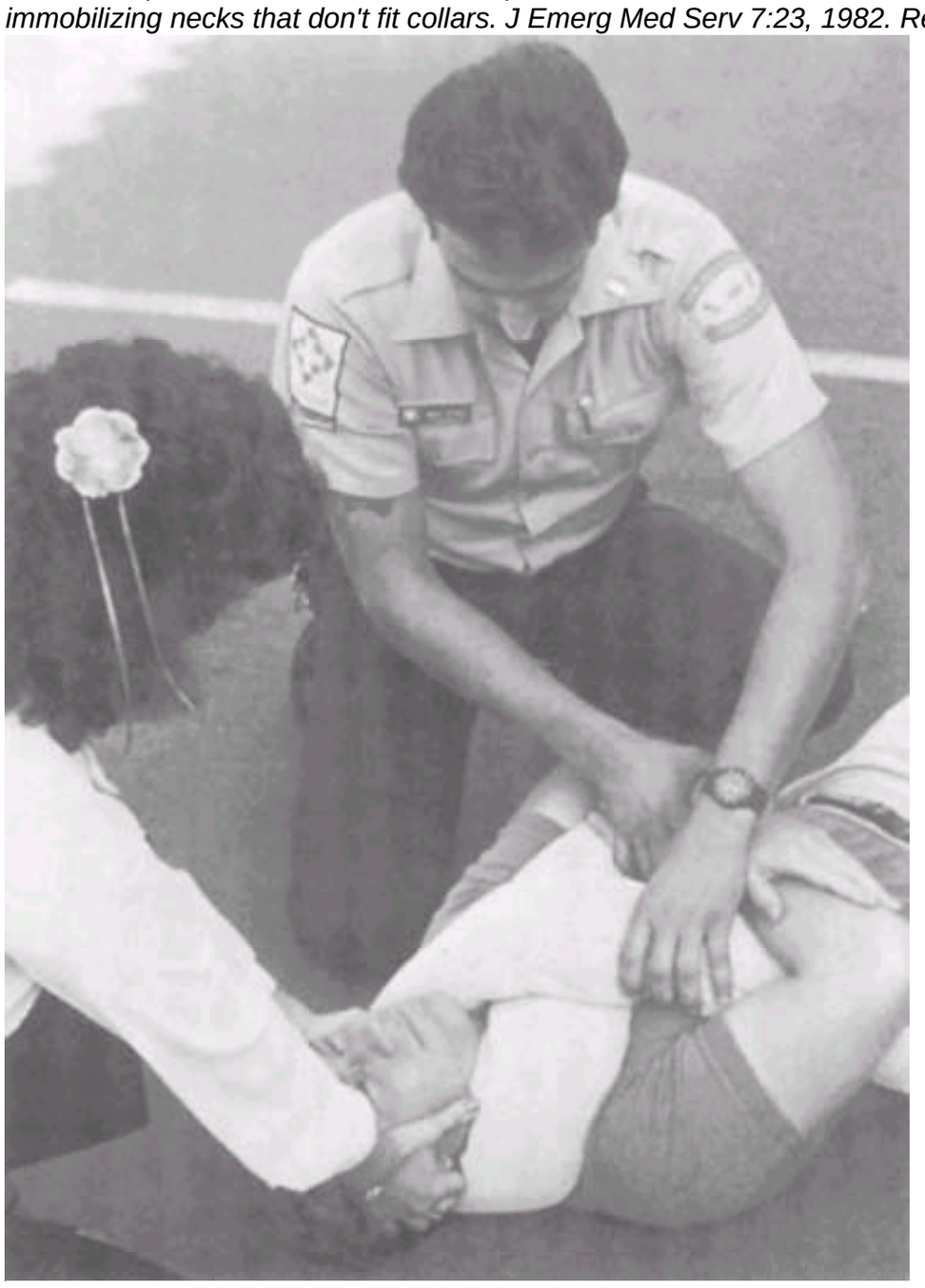


Figure 47-10 Extrication collar combined with a cervical immobilization device. *(Courtesy of Laerdal Medical Corporation, Wappingers Falls, NY.)*

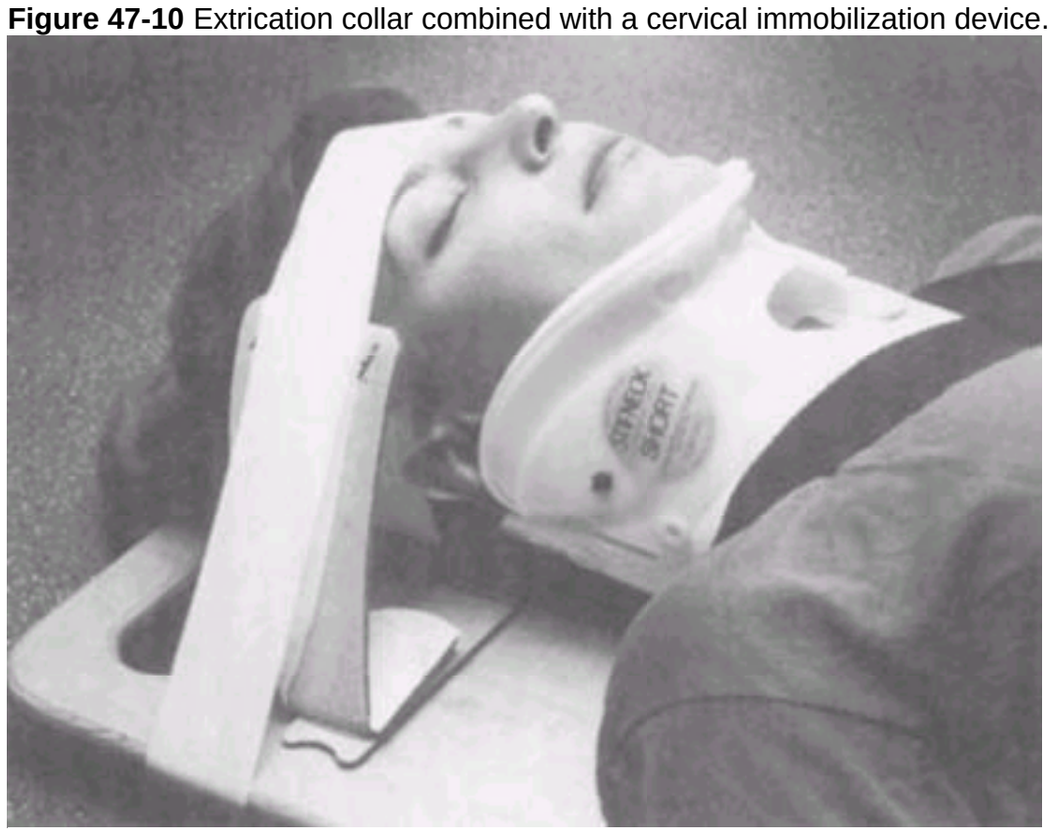


Figure 47-11 The Evac-U-Splint mattress. *(Courtesy of Hartwell Medical, Carlsbad, CA.)*

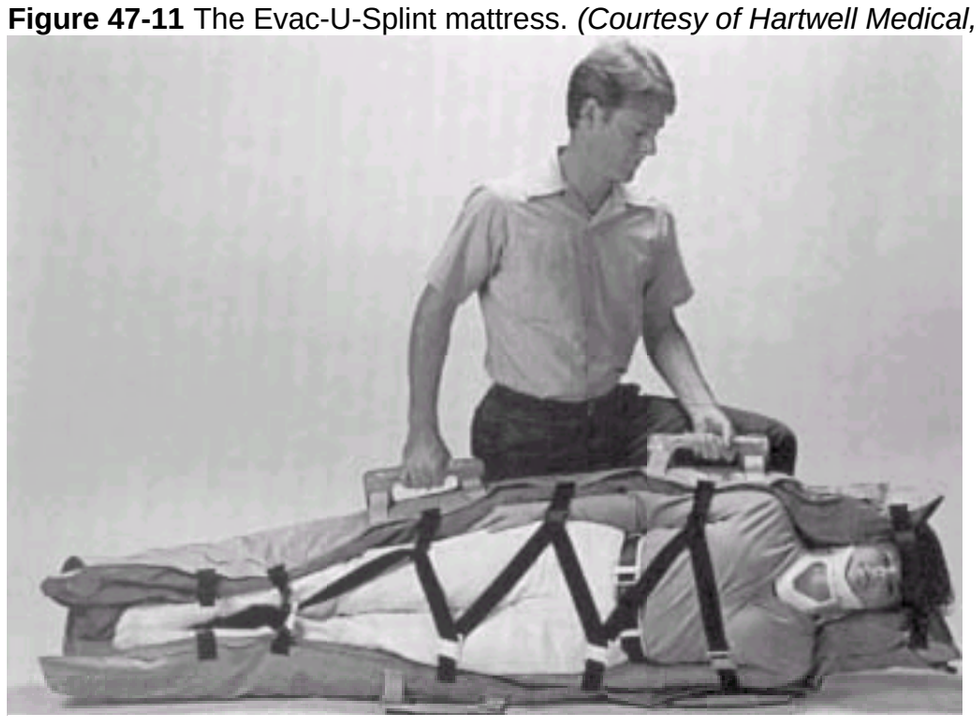


Figure 47-12 Rigid short boards. *(Courtesy of Ferno-Washington, Inc., Wilmington, OH.)*

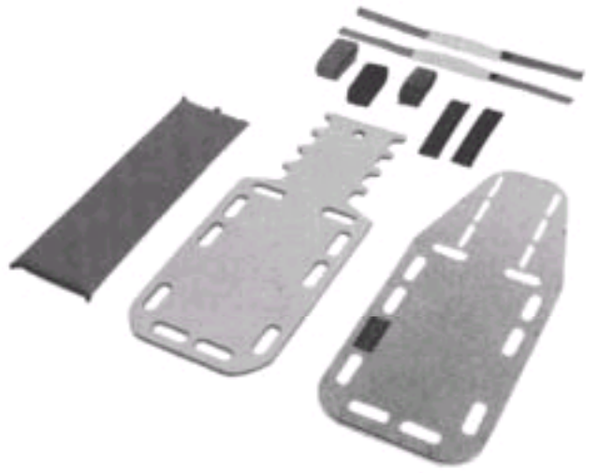


Figure 47-13 The Kendrick extrication device (KED). Note the presence of a cervical collar, applied before the KED. *(Courtesy of MediXchoice, El Cajon, CA.)*

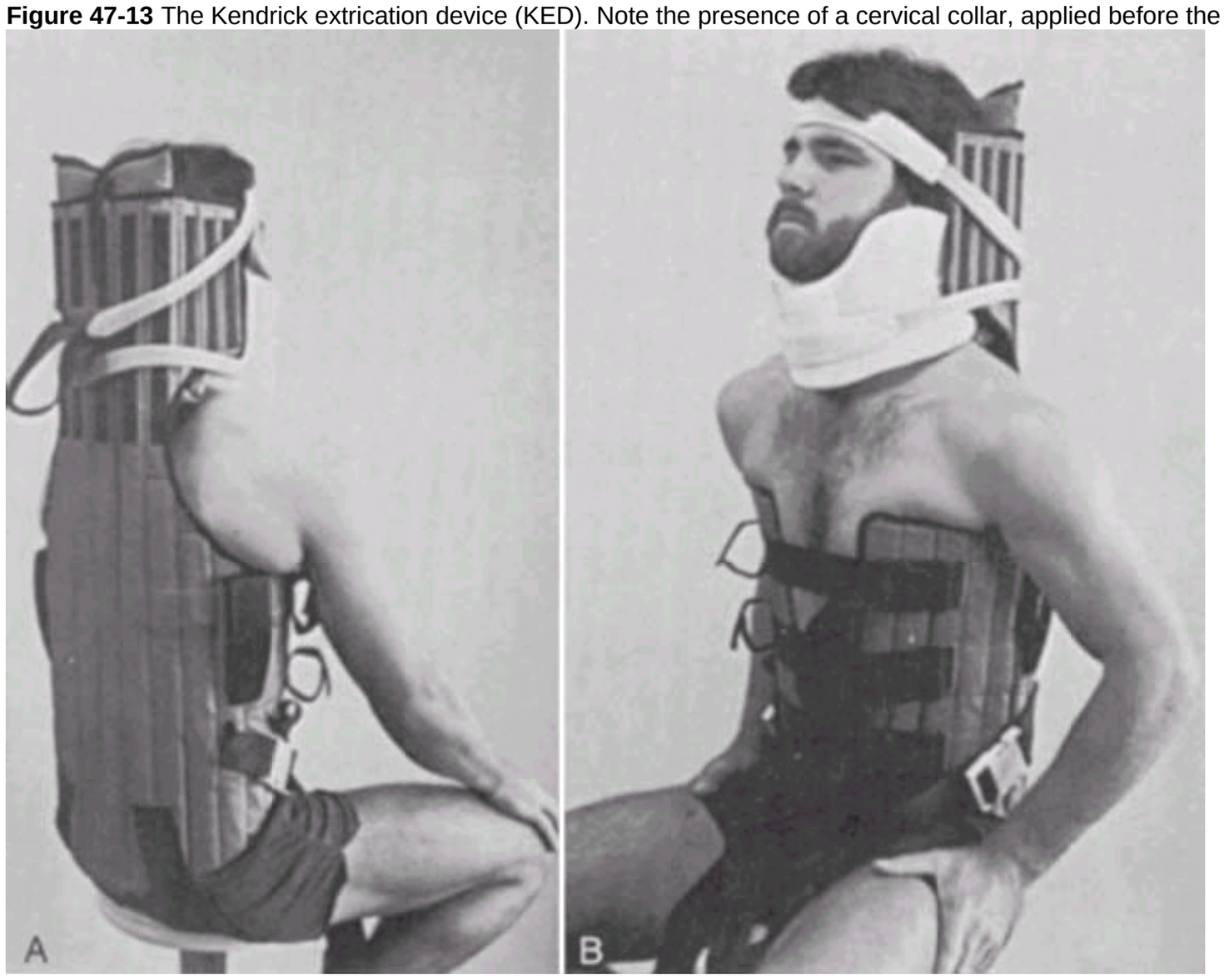


Figure 47-14 The Bound Tree spine board. An example of a commercial plywood backboard. *(Courtesy of Bound Tree ALS Medical Products Corporation, Phoenix, AZ.)*

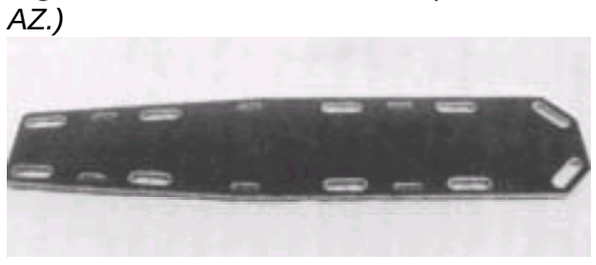


Figure 47-15 The Ferno-Washington model 65 orthopedic (scoop) stretcher. *(Courtesy of Ferno-Washington, Inc., Wilmington, OH.)*

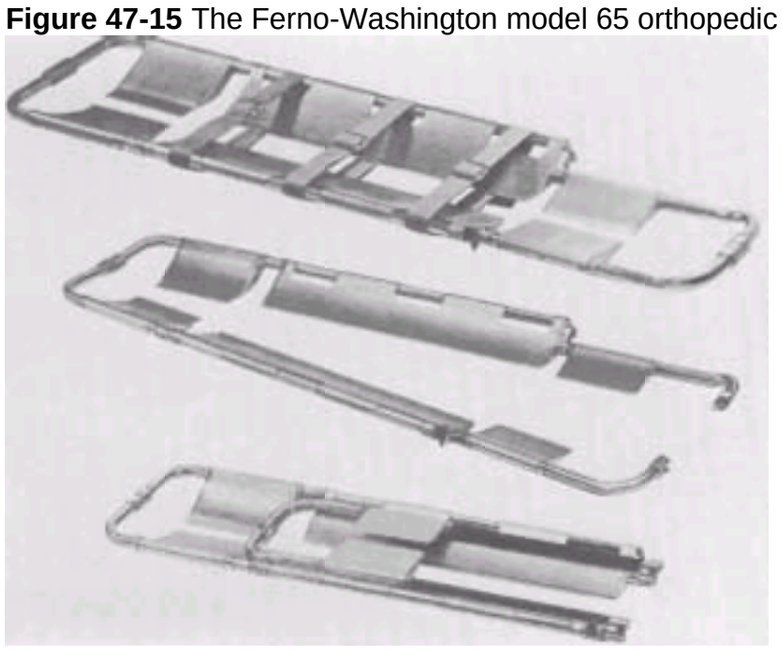


Figure 47-16 *A*, CombiCarrier. *(Courtesy of Hartwell Medical, Carlsbad, CA.) B*, Scoop EXL. *(Courtesy of Ferno-Washington, Inc., Wilmington, OH.)*

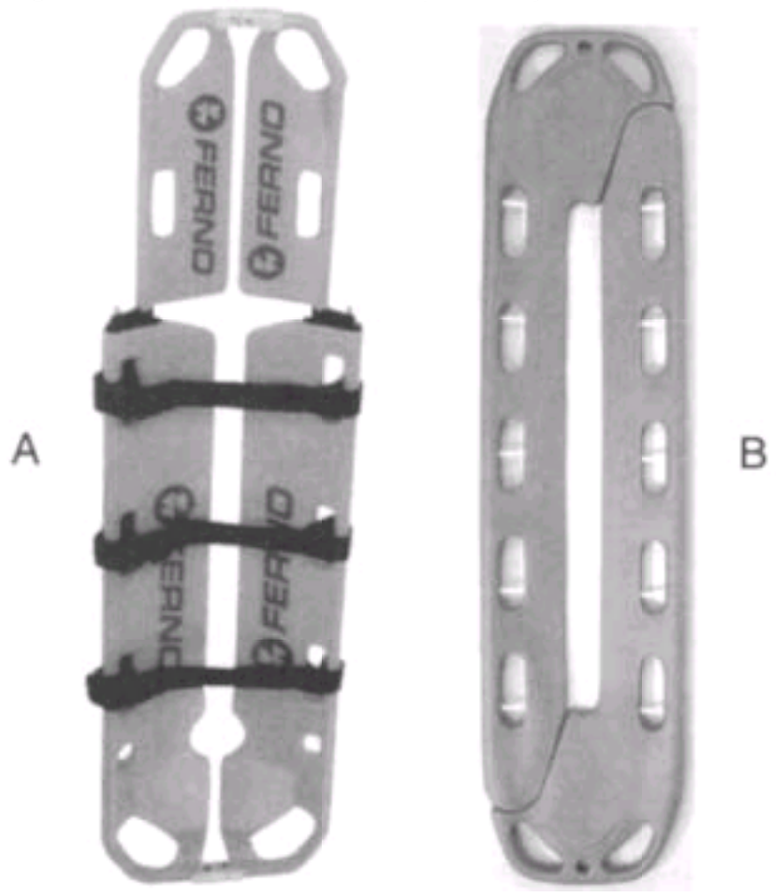


Figure 47-17 The Miller body splint. *(Courtesy of Life Support Products, Inc., Irvine, CA.)*

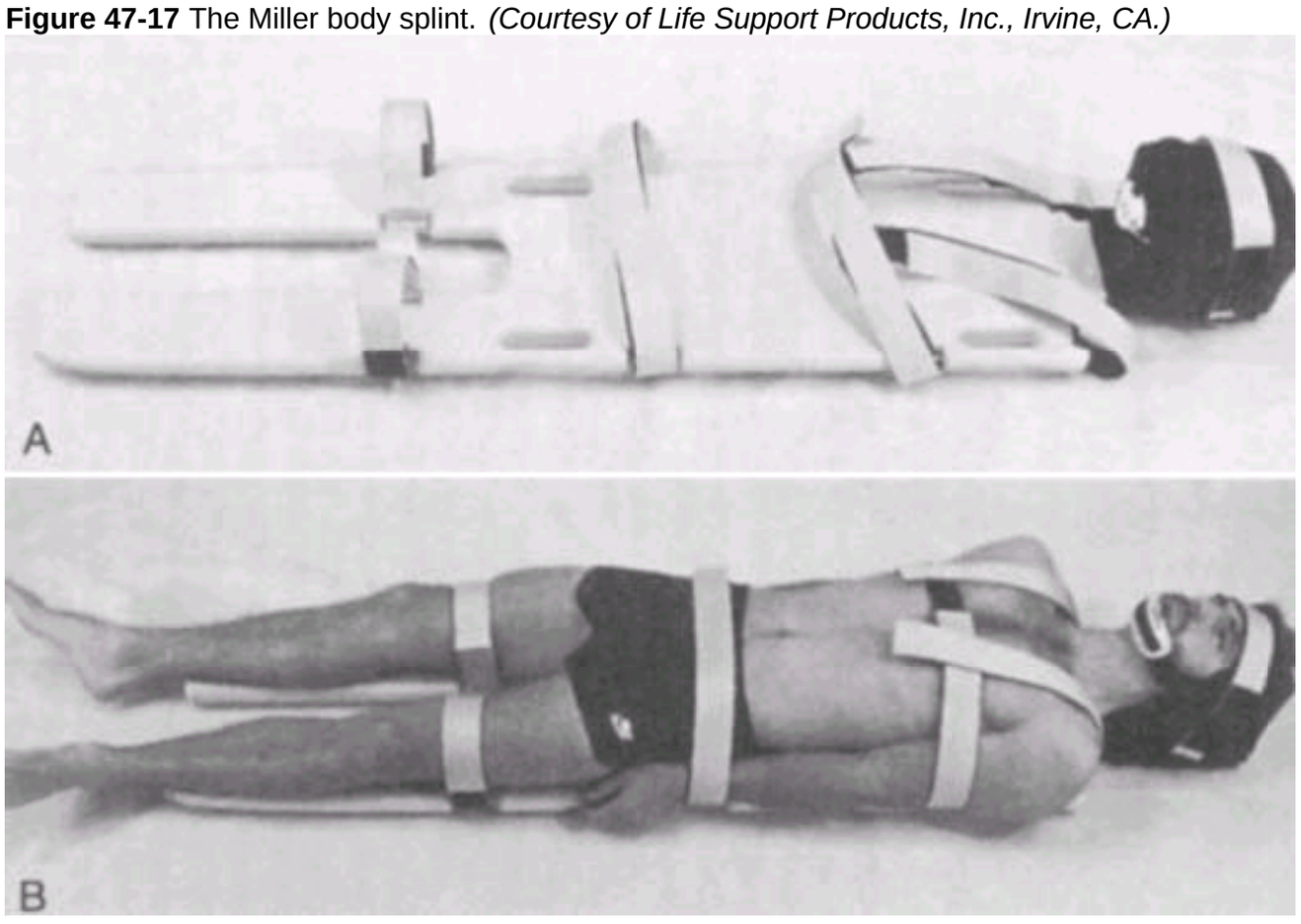


Figure 47-18 The HeadBed, a cervical immobilization device made of a water-resistant corrugated board. *(Courtesy of Laerdal Medical Corporation, Wappingers Falls, NY.)*

Figure 47-19 The Bashaw cervical immobilization device (CID). *(Courtesy of Bashaw Medical, Inc., Pensacola, FL.)*

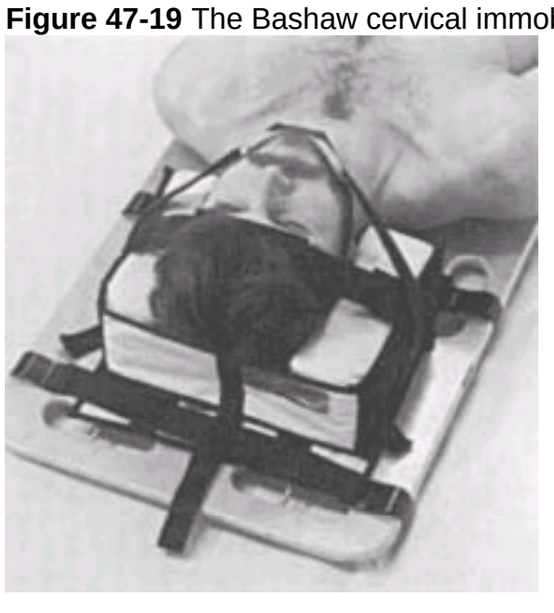


Figure 47-20 Ferno Universal Head Immobilizer. *(Courtesy of Ferno-Washington, Inc., Wilmington, OH.)*

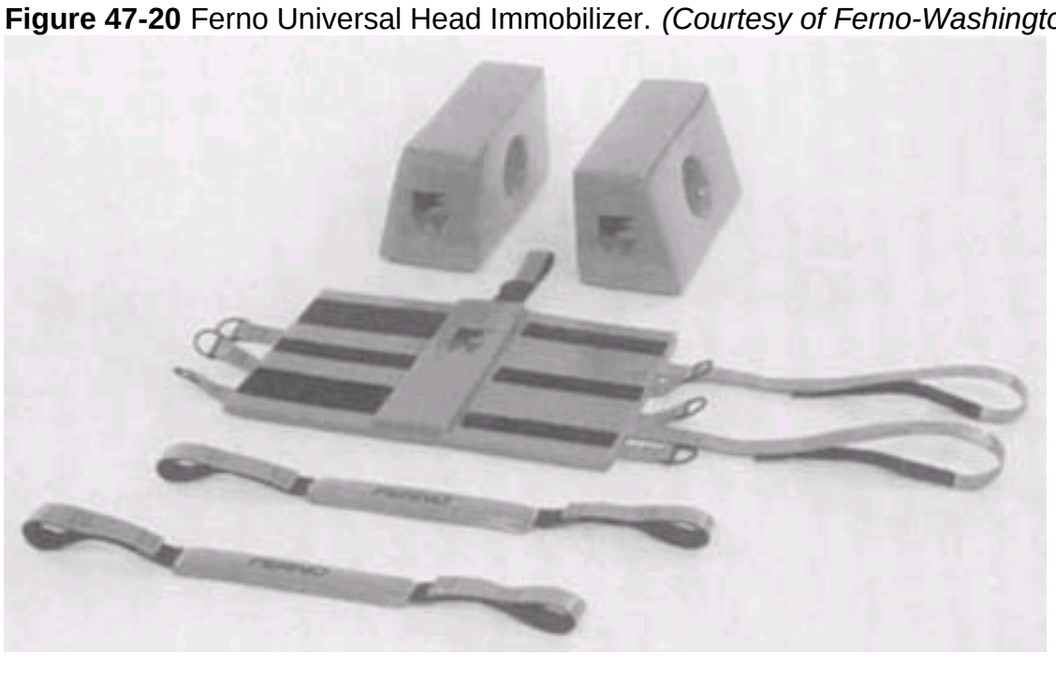


Figure 47-21 Logroll maneuver. *(Courtesy of Albert Einstein Medical Center Emergency Medical Services Education, Philadelphia.)*

Figure 47-22 Backboarding the standing patient. *A,* Step 1: Manual stabilization. *B,* Step 2: Apply a rigid collar. *C,* Step 3: Insert a long backboard. *D,* Step 4: Center the backboard. *E,* Step 5: Emergency medical technicians grasp the board, using a handle higher than the patient's armpit *(F)*. *G,* Step 6: Slowly lower the patient. *H,* Step 7: Fully immobilize the torso, then the head and neck. *(From Elling R, Politis J: Backboarding the standing patient. J Emerg Med Serv 12:9, 1987. Reproduced with permission.)*

Figure 47-23 Upper extremity vacuum splint. *(Courtesy of Hartwell Medical, Carlsbad, CA.)*

Figure 47-24 Hand/wrist pillow splint.

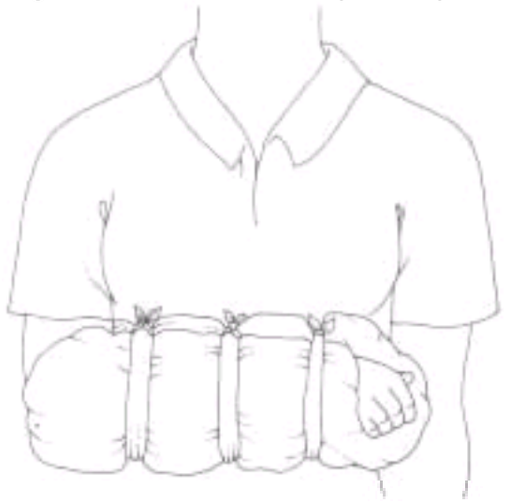


Figure 47-25 Examples of rigid splints.

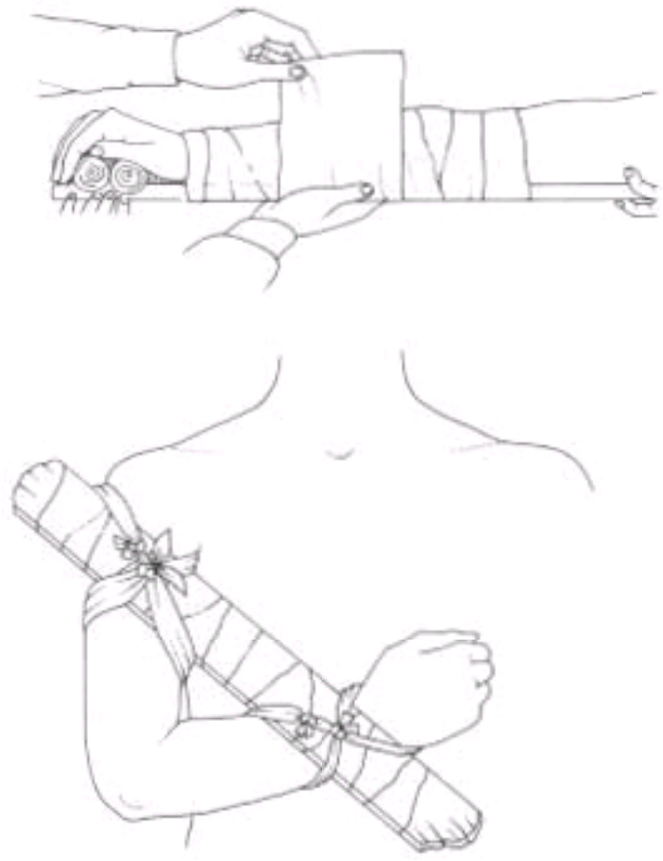


Figure 47-26 Application of an air splint. *A*, The rescuer supports the injured extremity with one hand and places the air splint on the other arm. *B*, An assistant slides the splint onto the patient's arm. *C*, The air splint is inflated until finger pressure makes a slight dent.

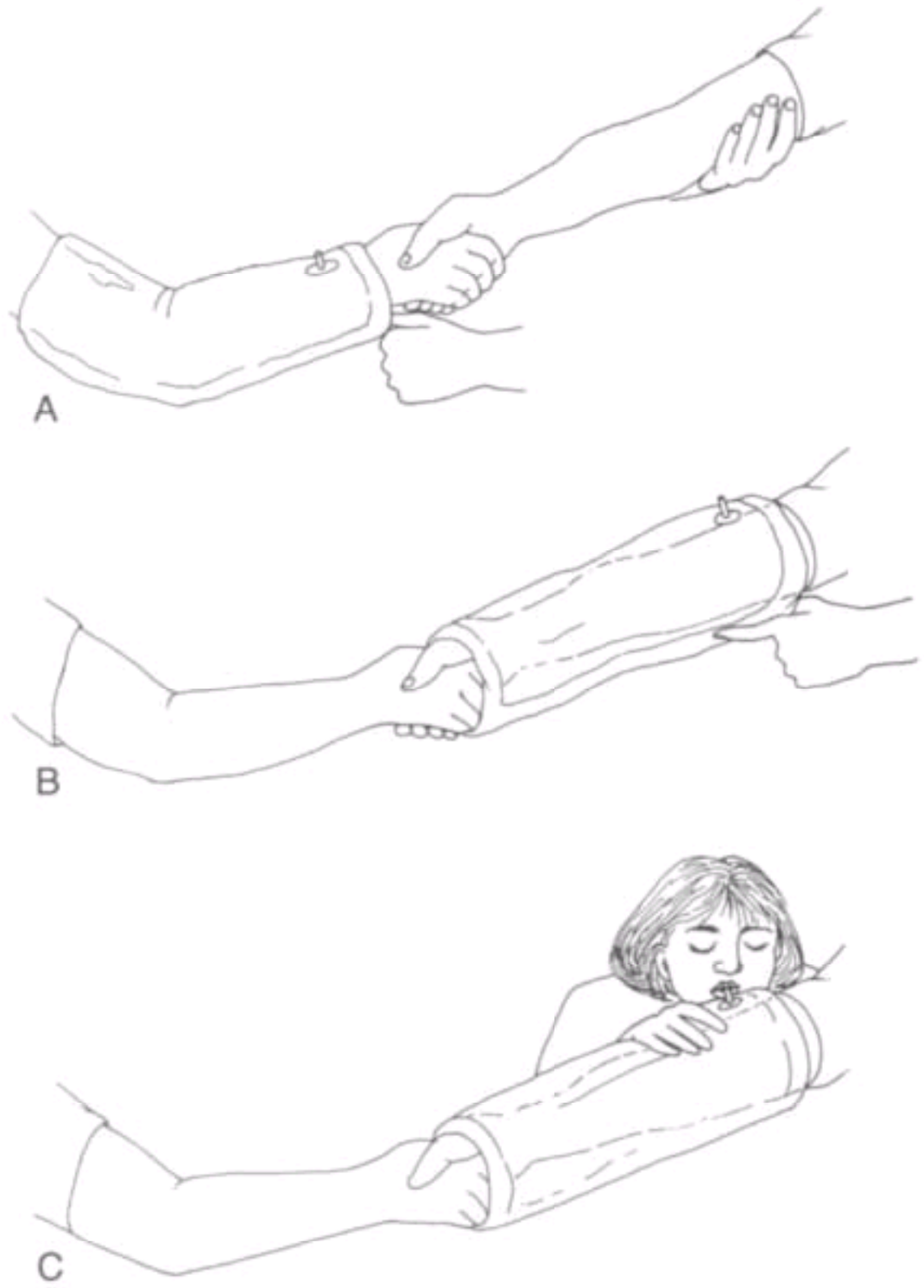


Figure 47-27 Stepwise application of a triangular bandage. 1, Place tip A over the uninjured shoulder. 2, Bring tip B over the injured shoulder to enclose the arm. 3, Draw tip C around the front and pin.

Figure 47-28 Completed triangular bandage.

Figure 47-29 Sling with swath.

Figure 47-30 Lower leg pillow splint.

Figure 47-31 Application of the Ferno traction splint. *A–B*, Applying the ankle wrap. *C–E*, Applying the splint. *(Reproduced and modified with permission. Ferno-Washington, Inc., Wilmington, OH.)*

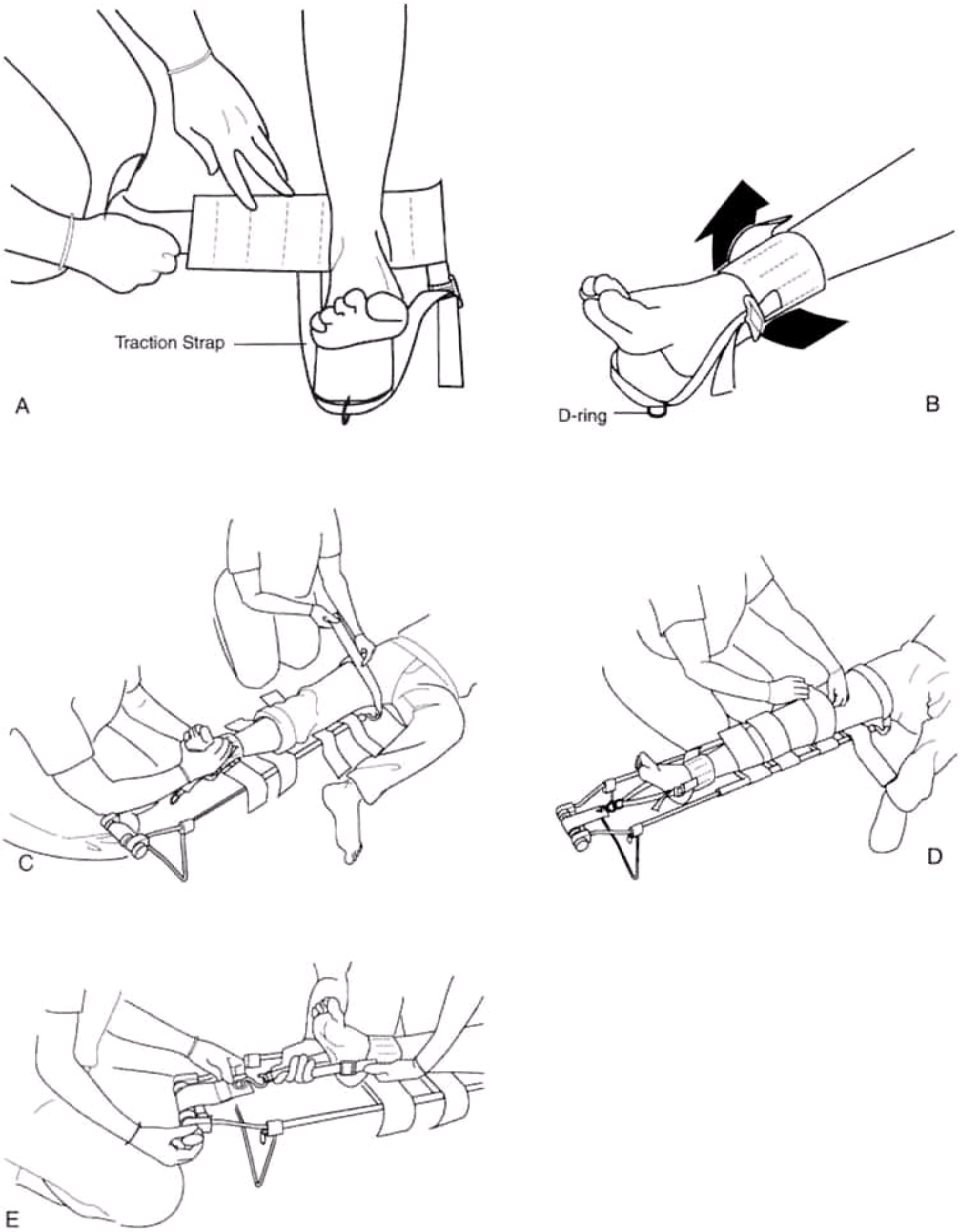
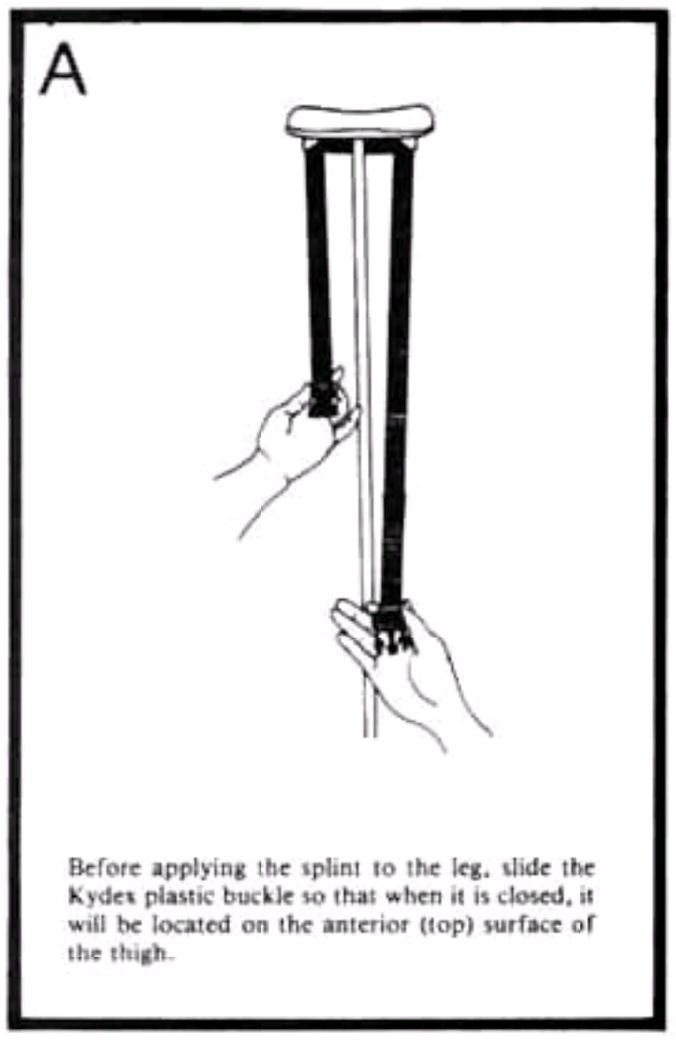
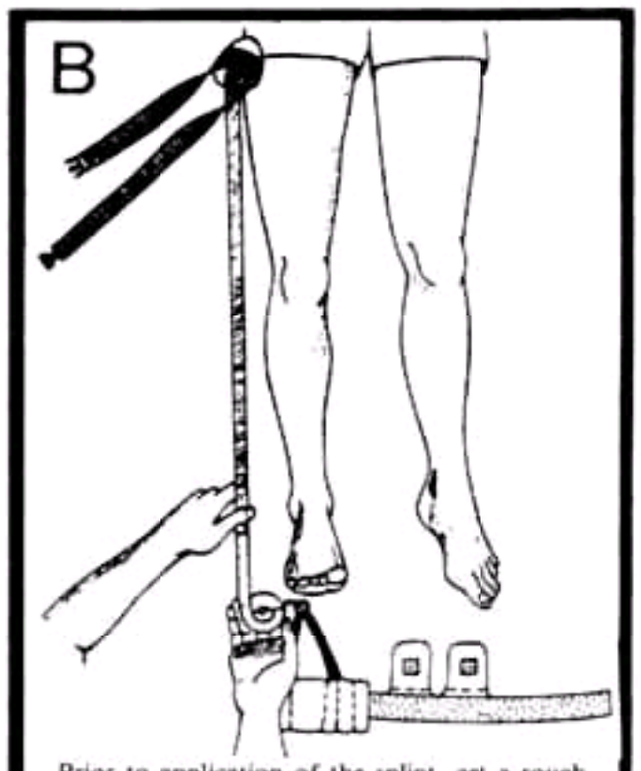


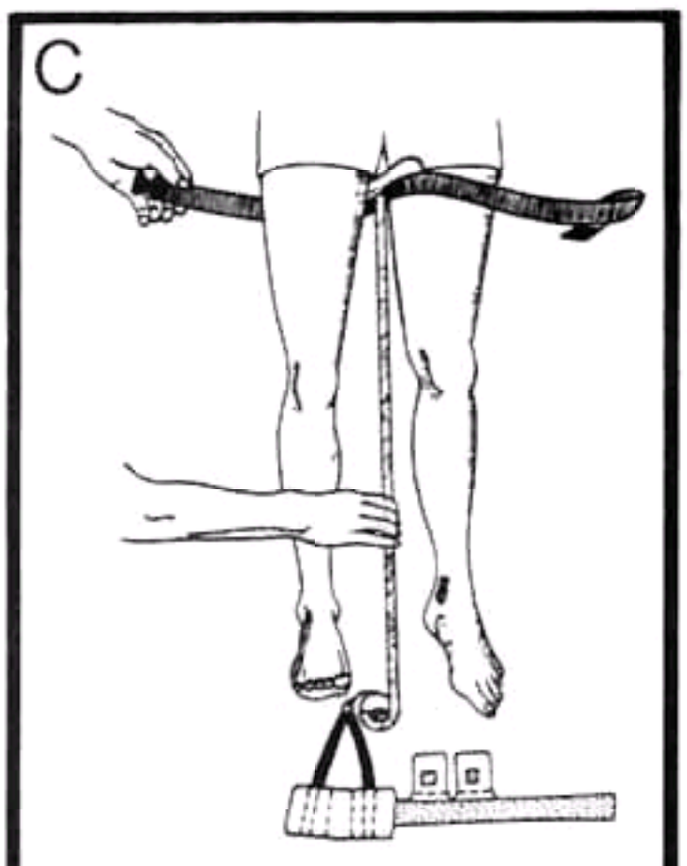
Figure 47-32a Application of the Sager emergency traction splint. *A–J*, Standard application.



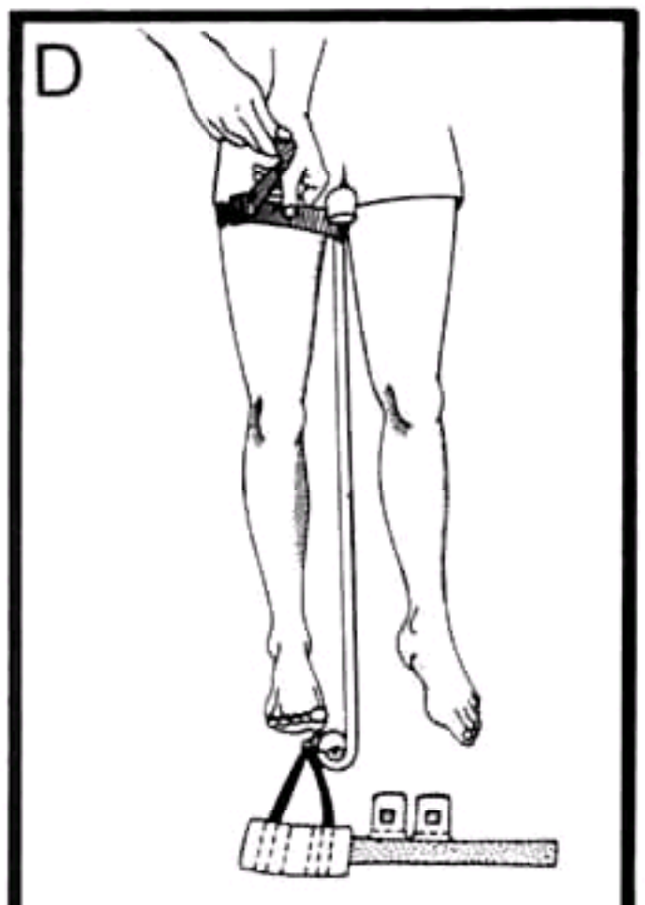
Before applying the splint to the leg, slide the Kydex plastic buckle so that when it is closed, it will be located on the anterior (top) surface of the thigh.



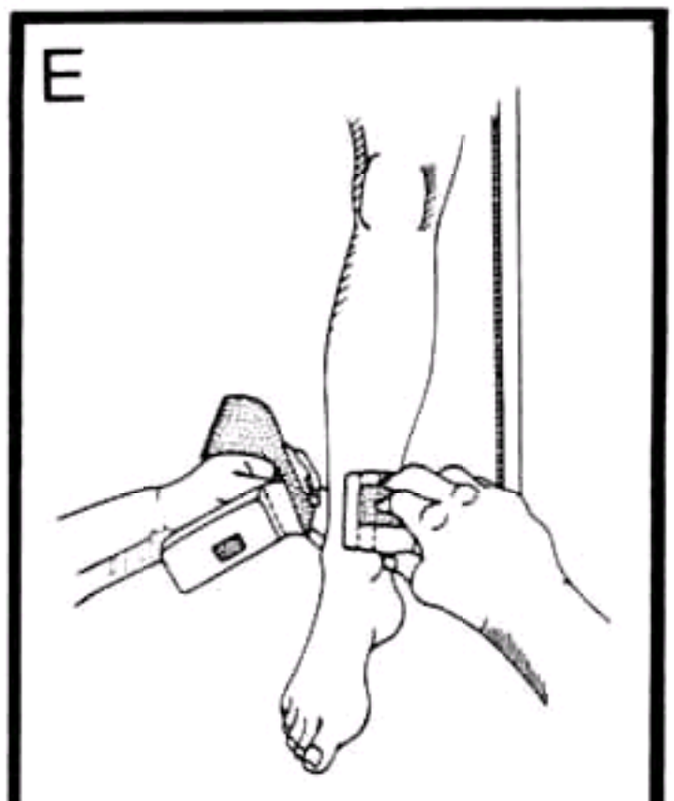
Prior to application of the splint, get a rough measure of length of splint needed. Extend the splint so that the wheel is at the heel. NOTE: Patients wearing tight jeans or underclothing, especially males, will find the splint uncomfortable to wear unless clothing is removed or cut open, which, of course, should be done as part of patient secondary evaluation prior to application of splint.



Grasp the Kydex buckle and slide the thigh strap up under the leg so that the perineal cushion is snug against the perineum and ischial tuberosity.

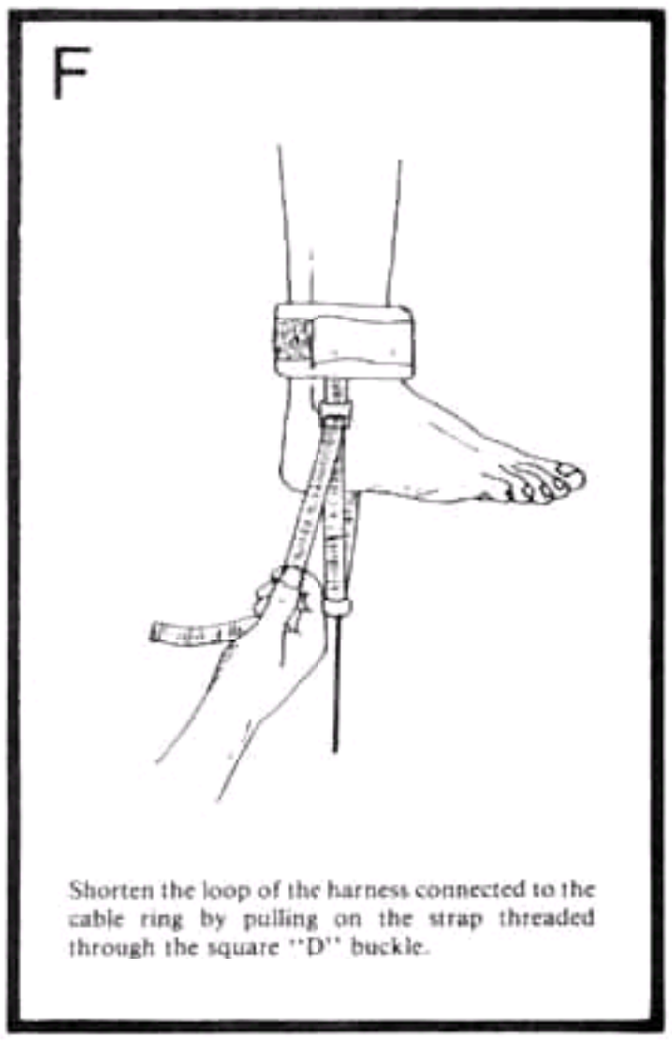


Tighten the Kydex buckle thigh strap, drawing the perineal-ischial pad to the lateral portion of the crotch.

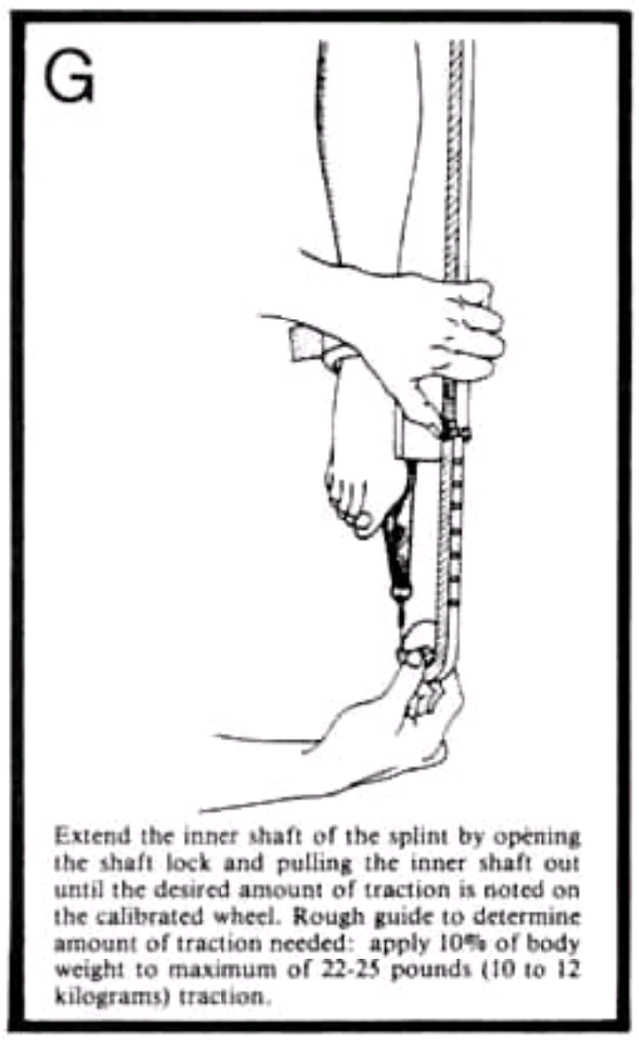


Apply the ankle harness tightly around the ankle above the medial and lateral malleoli of the ankle. Check posterior tibial and dorsalis pedis pulses before hitch application and after traction is established.

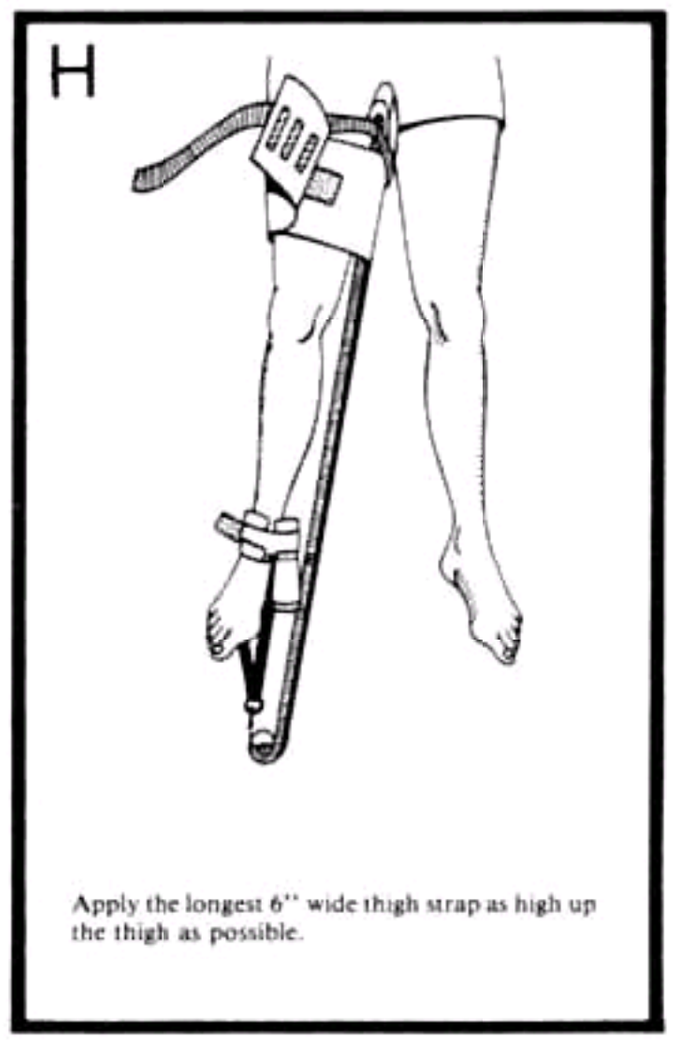
Figure 47-32b



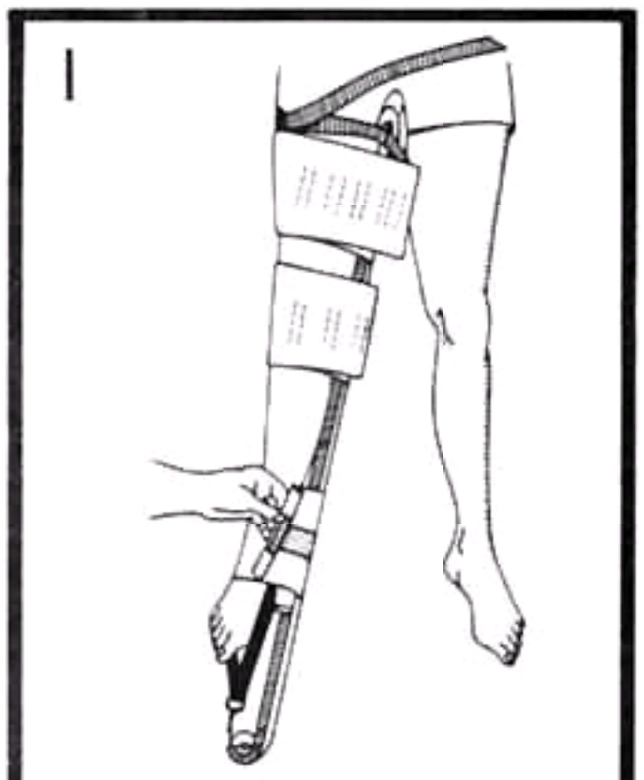
Shorten the loop of the harness connected to the cable ring by pulling on the strap threaded through the square "D" buckle.



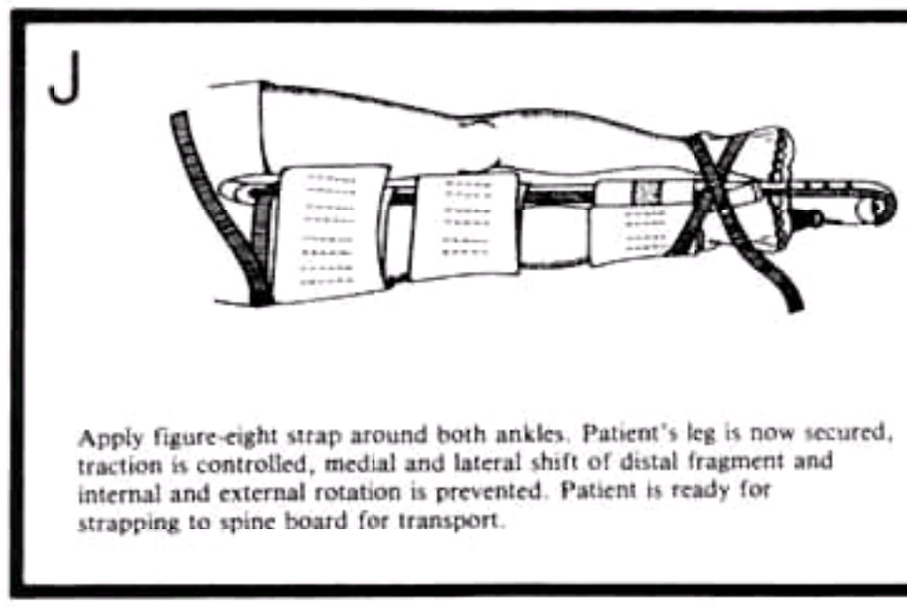
Extend the inner shaft of the splint by opening the shaft lock and pulling the inner shaft out until the desired amount of traction is noted on the calibrated wheel. Rough guide to determine amount of traction needed: apply 10% of body weight to maximum of 22-25 pounds (10 to 12 kilograms) traction.



Apply the longest 6" wide thigh strap as high up the thigh as possible.

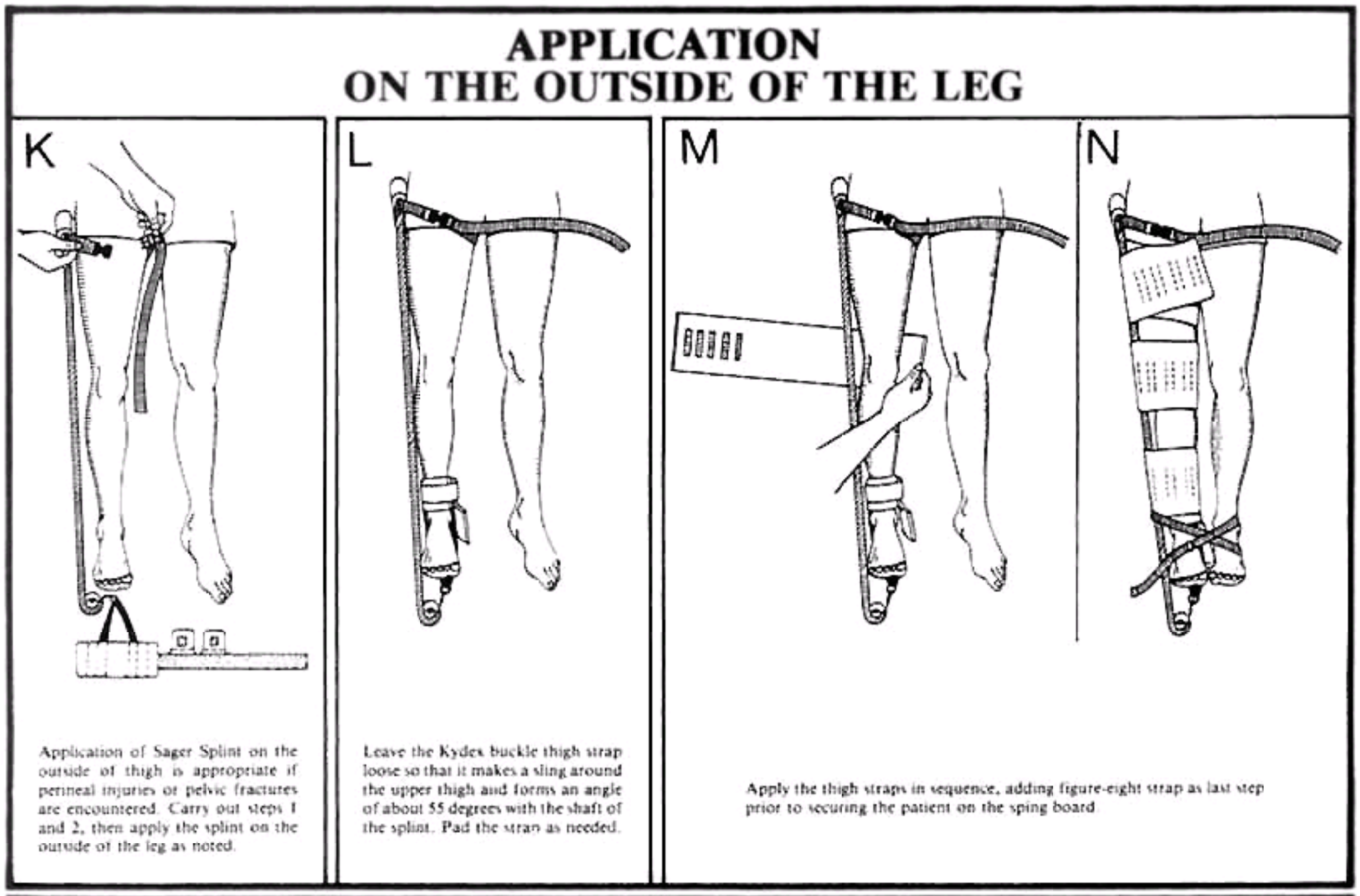


Apply the second longest thigh strap around the knee. Use padding as needed. Next, apply the shortest 6-inch wide strap over the ankle harness and lower the leg.



Apply figure-eight strap around both ankles. Patient's leg is now secured, traction is controlled, medial and lateral shift of distal fragment and internal and external rotation is prevented. Patient is ready for strapping to spine board for transport.

Figure 47-32c *K–N*, Application on the outside of the leg. *O–Q*, Application of the bilateral splint.



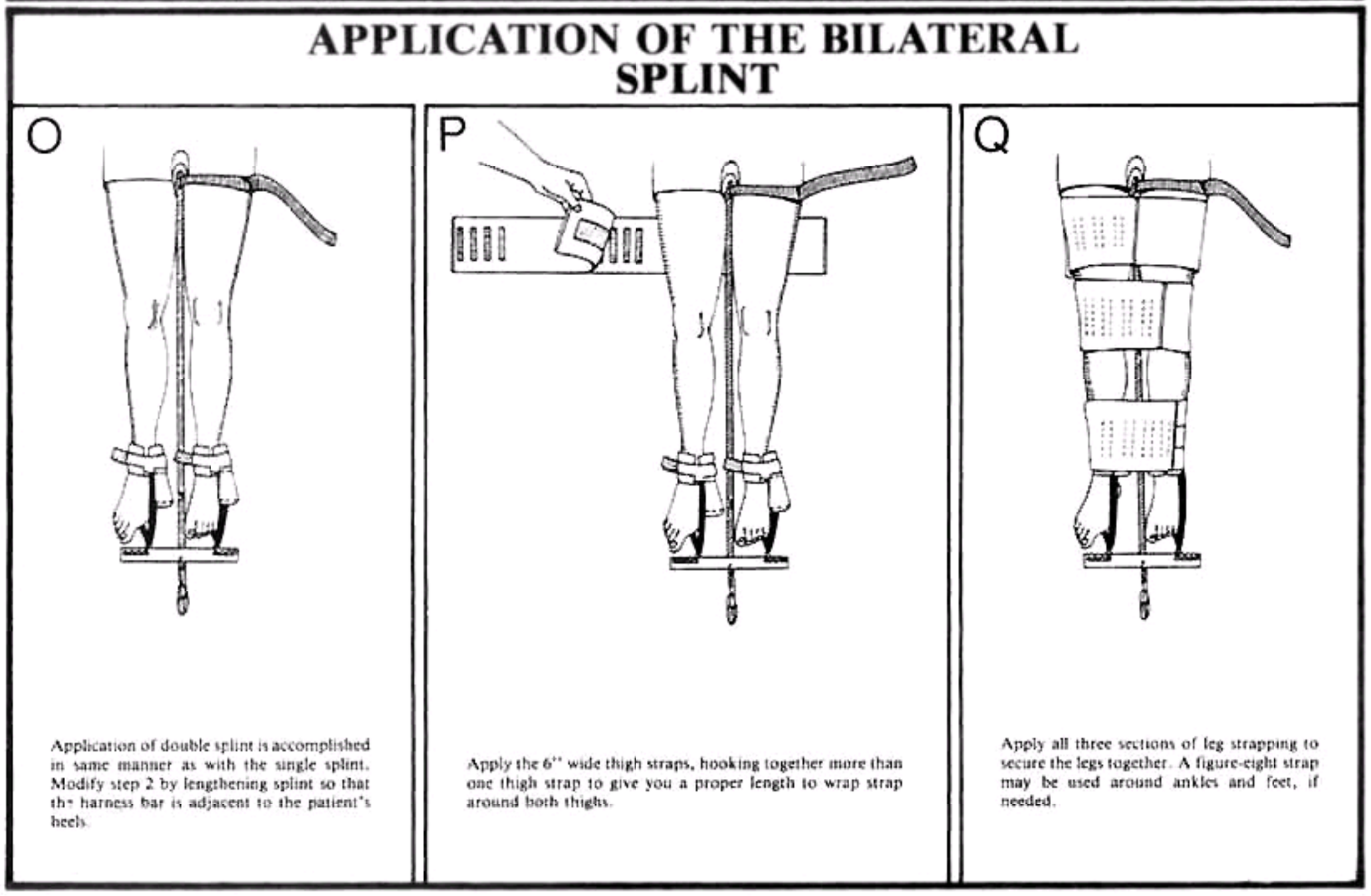


Figure 47-33 Assembled pneumatic anti-shock garment and inflation pump. *(Courtesy of the American College of Surgeons, Committee on Trauma.)*

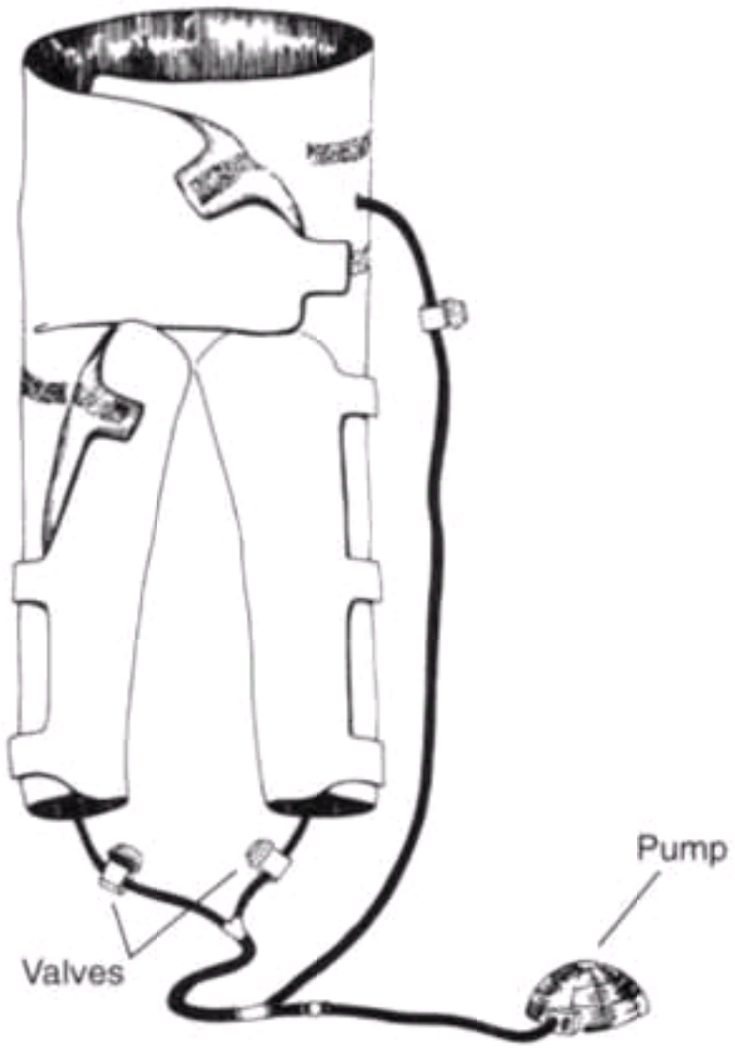


Figure 47-34 Pneumatic anti-shock garment applied to a patient and inflated. *(Courtesy of the American College of Surgeons, Committee on Trauma.)*

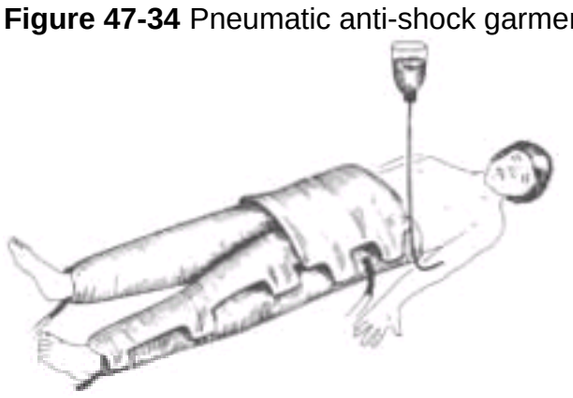


Figure 47-35 Pneumatic anti-shock garment application. *(Courtesy of the American College of Surgeons, Committee on Trauma.)*

Figure 47-36 Stabilization of a pelvic fracture using a sheet.

Figure 47-37 Helmet removal technique. *(Modified from McSwain N: Techniques of helmet removal from injured patients. Bull Am Coll Surg 65:20, 1980.)*

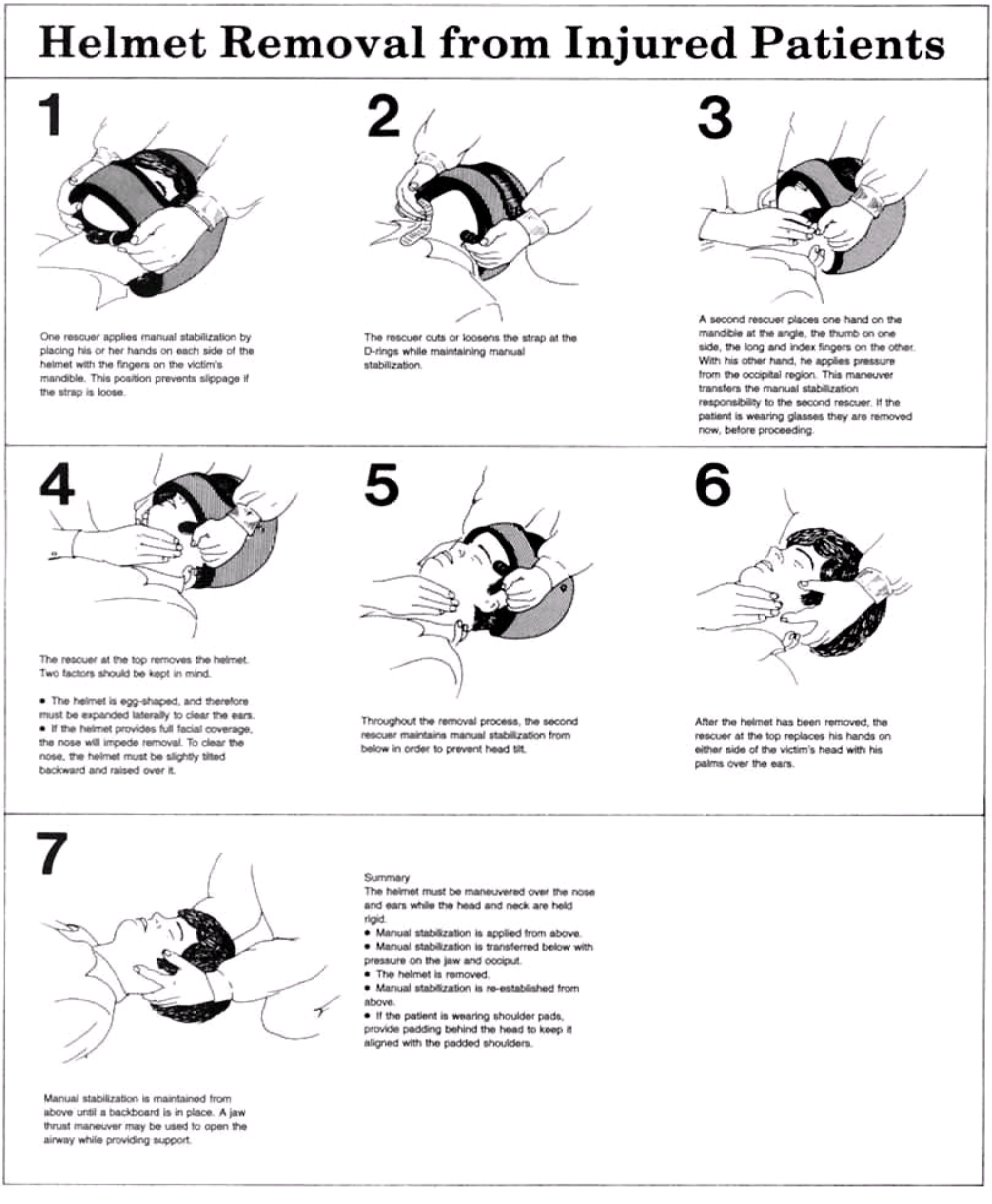


Figure 48-1 Evaluate the patient's condition to ensure that resuscitation is not necessary before transfer. *A,* The wound should be rinsed with saline solution. *Do not scrub or apply antiseptic solution to the wound.* Apply saline moistened sterile dressing, wrap in Kling or Kerlix for pressure, and elevate. *B,* The amputated part should be rinsed with saline. *Do not scrub or apply antiseptic solution to the amputated part.* Wrap it in moist sterile gauze or a towel, depending on its size, and place it in a plastic bag or plastic container. *Do not place the amputated part directly in saline. C,* The part is then put in a container, preferably Styrofoam, and cooled by separate plastic bags containing ice or in a container of ice water. Do not pack the bagged, injured part in ice, but it can be immersed in half water, half ice. *(From Hand Trauma: Emergency Care. Baltimore, MD, Emergency Services.)*

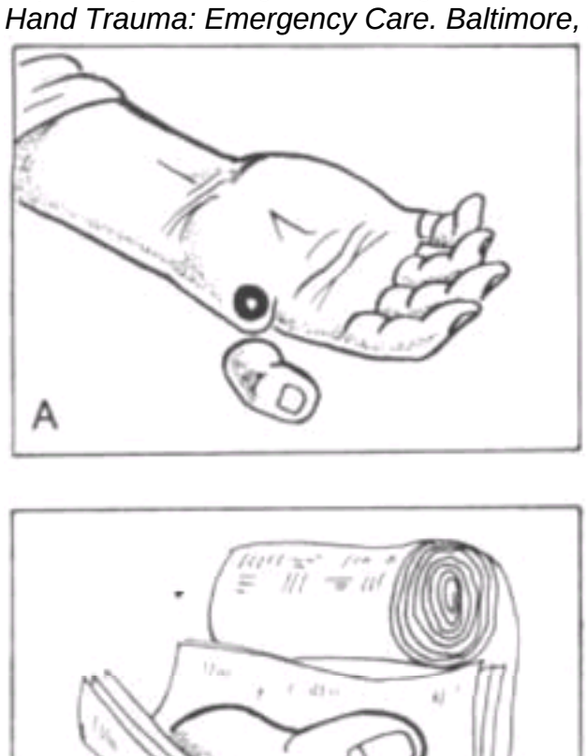


Figure 48-2 For a *partial amputation*, rinse with saline (*A*); then place part(s) in a functional position, apply a saline moistened sterile dressing, and splint and elevate *(B)*. Apply coolant bags to the *outside* of the dressing *(C)*. *Do not scrub or apply antiseptic solution to the wound.* Control any bleeding with pressure. If a tourniquet is necessary, place it close to the amputation site. *(From Hand Trauma: Emergency Care. Baltimore, MD, Emergency Services.)*

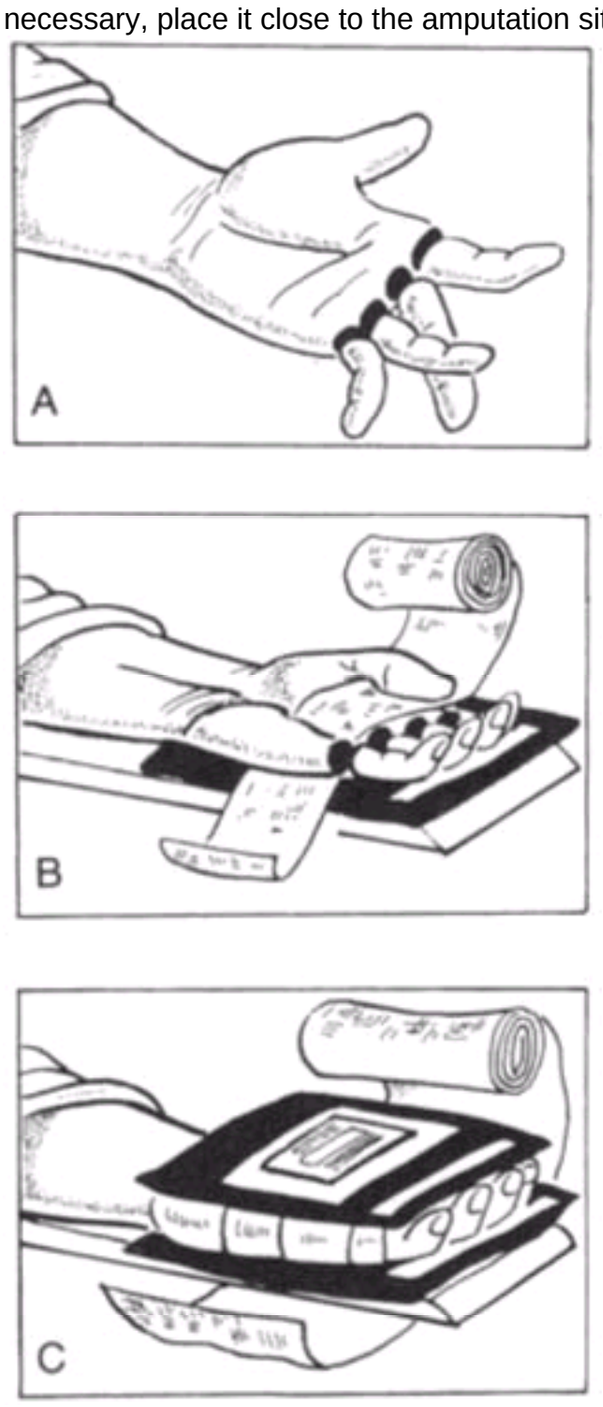


Figure 49-1 Extensor muscles and tendons of the RIGHT forearm and wrist. *A,* superficial layer. *B,* deep layer *(Modified from Pansky B: Upper extremity. In Review of Gross Anatomy, 4th ed. New York, Macmillan Publishing, 1979, pp 219 and 243.)*

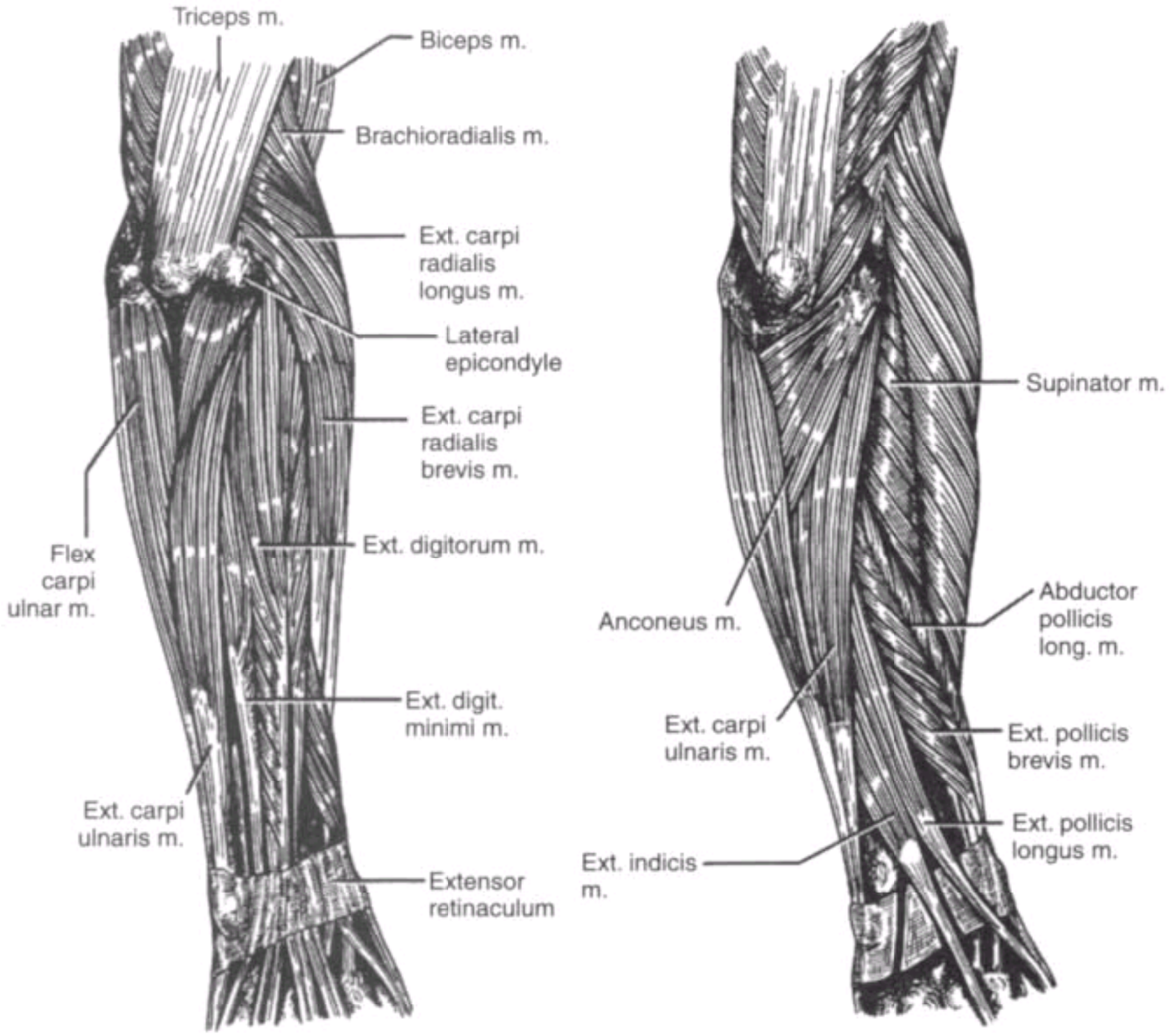


Figure 49-2 *A,* The extensor mechanism at the wrist and dorsum of the right hand. The six extensor compartments at the wrist contain *(1)* the abductor pollicis longus (APL) and extensor pollicis brevis (EPB); *(2)* the extensor carpi radialis longus (ECRL) and brevis (ECRB); *(3)* the extensor pollicis longus (EPL); *(4)* the extensor digitorum communis (EDC) II–V and extensor indicis proprius (EIP); *(5)* the extensor digiti quinti (EDQ); and *(6)* the extensor carpi ulnaris (ECU). An important anatomic detail is the presence of a synovial sheath around each tendon unit within each fibro-osseous canal. *(From Thomas JS, Peimer CA: Extensor tendon injuries: Acute repair and late reconstruction. In Chapman MW [ed]: Operative Orthopaedics, 3rd ed. Philadelphia, JB Lippincott, 2001, p 1497.) B,* Cadaver model demonstrating the dorsal retinaculum of the left wrist with a probe passing through the fourth dorsal compartment. Note that the EDQ is also called the extensor digiti minimi (EDM) by some authors.

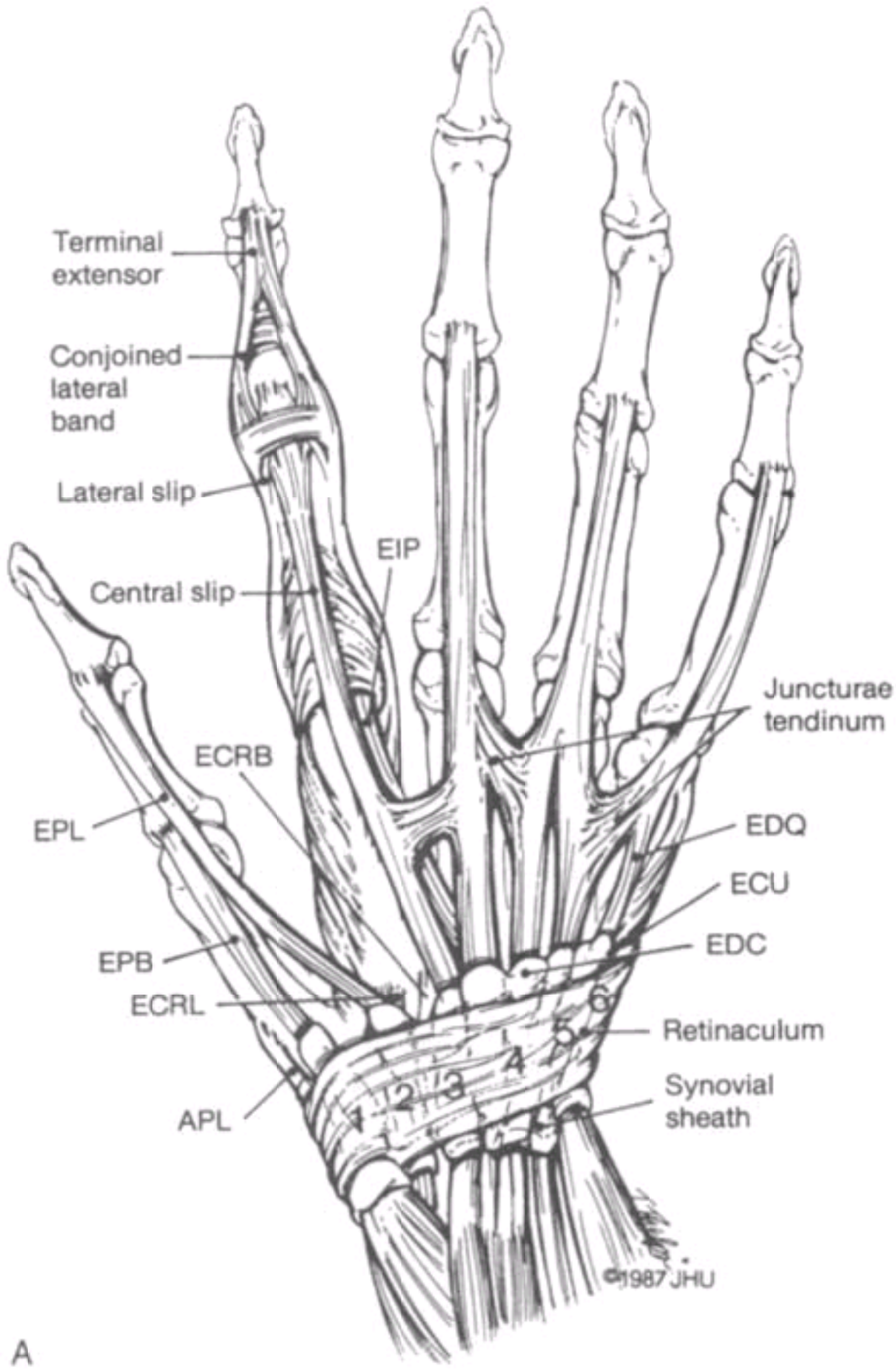


Figure 49-3 Digital extensor mechanism. *A,* Dorsal view. *B,* Lateral view. ORL, oblique retinacular ligament. See text and Figure 49-1 legend for other abbreviations. *(From Thomas JS, Peimer CA: Extensor tendon injuries: Acute repair and late reconstruction. In Chapman MW [ed]: Operative Orthopaedics, 3rd ed. Philadelphia, JB Lippincott, 2001, p 1498.)*

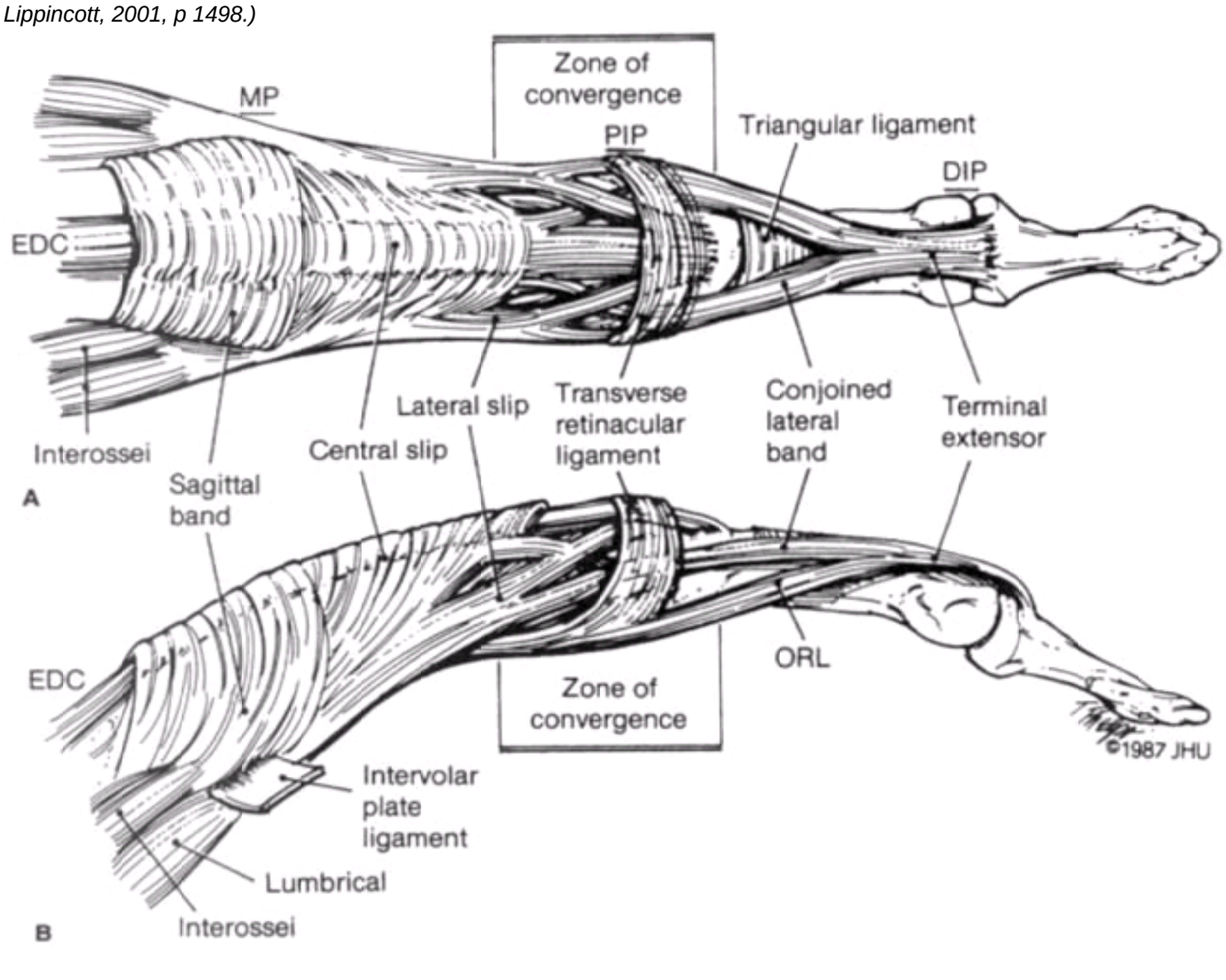


Figure 49-4 The zone of convergence of the digital extensor mechanism, which begins at about the midportion of the proximal phalanx and ends at the level of the central slip insertion into the dorsal base of the middle phalanx. Proximal to the zone of convergence, the extrinsic and intrinsic components of the extensor mechanism are separate: the central slip is extrinsic, whereas the lateral slips are intrinsic. Within the zone of convergence there is complete reciprocal crossover of fibers from the central slip and lateral slips. The products of the completed convergence are the central slip insertion and the conjoined lateral bands, both of which have dual muscular activity. PIP, proximal interphalangeal joint; TRL, transverse retinacular ligament; ORL, oblique retinacular ligament; E, extrinsic contribution to conjoined lateral bands; I, intrinsic contribution to central slip insertion. *(From Thomas JS, Peimer CA: Extensor tendon injuries: Acute repair and late reconstruction. In Chapman MW [ed]: Operative Orthopaedics, 3rd ed. Philadelphia, JB Lippincott, 2001, p 1500.)*

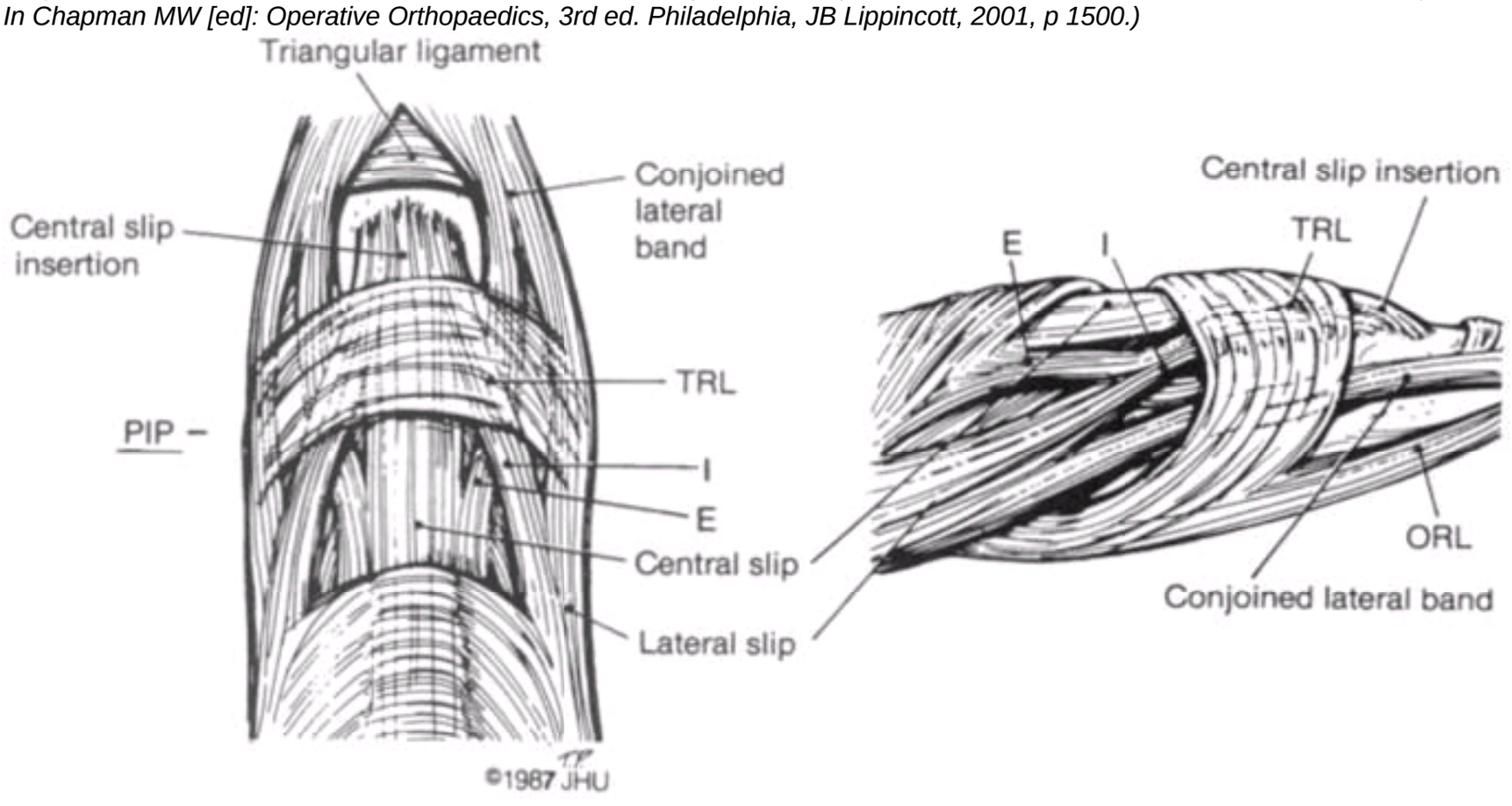


Figure 49-5 The extensor mechanism on the dorsum of a finger. *Arrows* point to the radial and ulnar lateral band portions of the extensor mechanism, and the probe lifts the entire structure up off the phalanx.

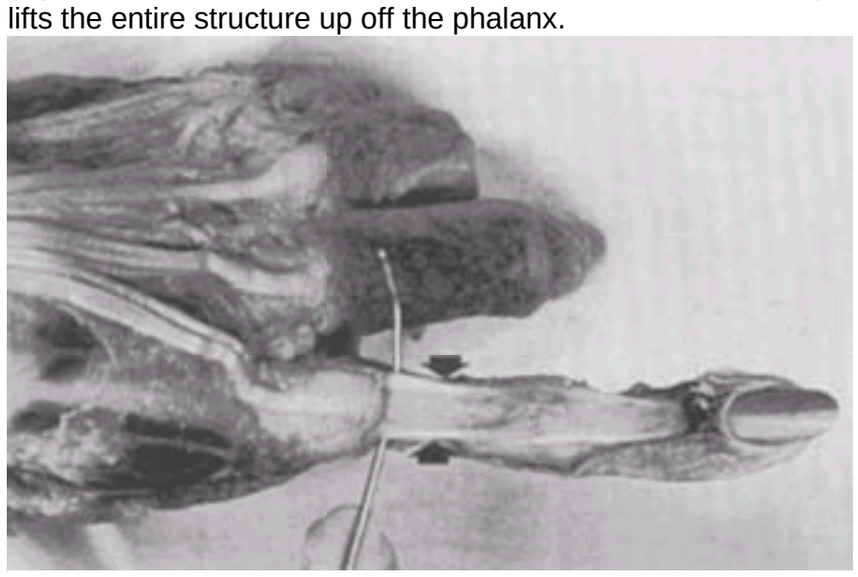


Figure 49-6 The terminal extensor mechanism.

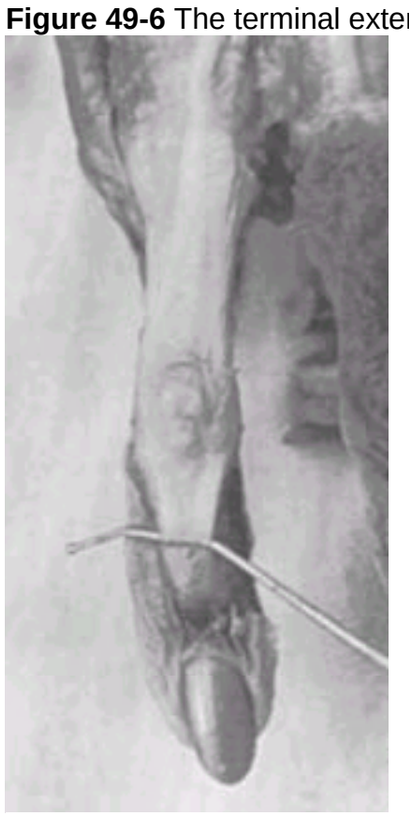


Figure 49-7 Because of their superficial location, it is difficult to avoid at least partial injury to extensor tendons in even superficial lacerations of the dorsum of the wrist, hand, or fingers. In some cases *(A)* the injury is obvious. In other cases, a partial tendon laceration is not appreciated, but becomes obvious when complete rupture occurs *(B)*. In this case the patient seemingly had full tendon function during the initial ED evaluation, but the entire tendon could not be visualized due to an uncooperative patient. An unappreciated partial laceration progressed to complete rupture by the time of suture removal. The injury was diagnosed and an expeditious delayed primary repair resulted in a good outcome. Note: Inability to extend index finger is evident in both photos.

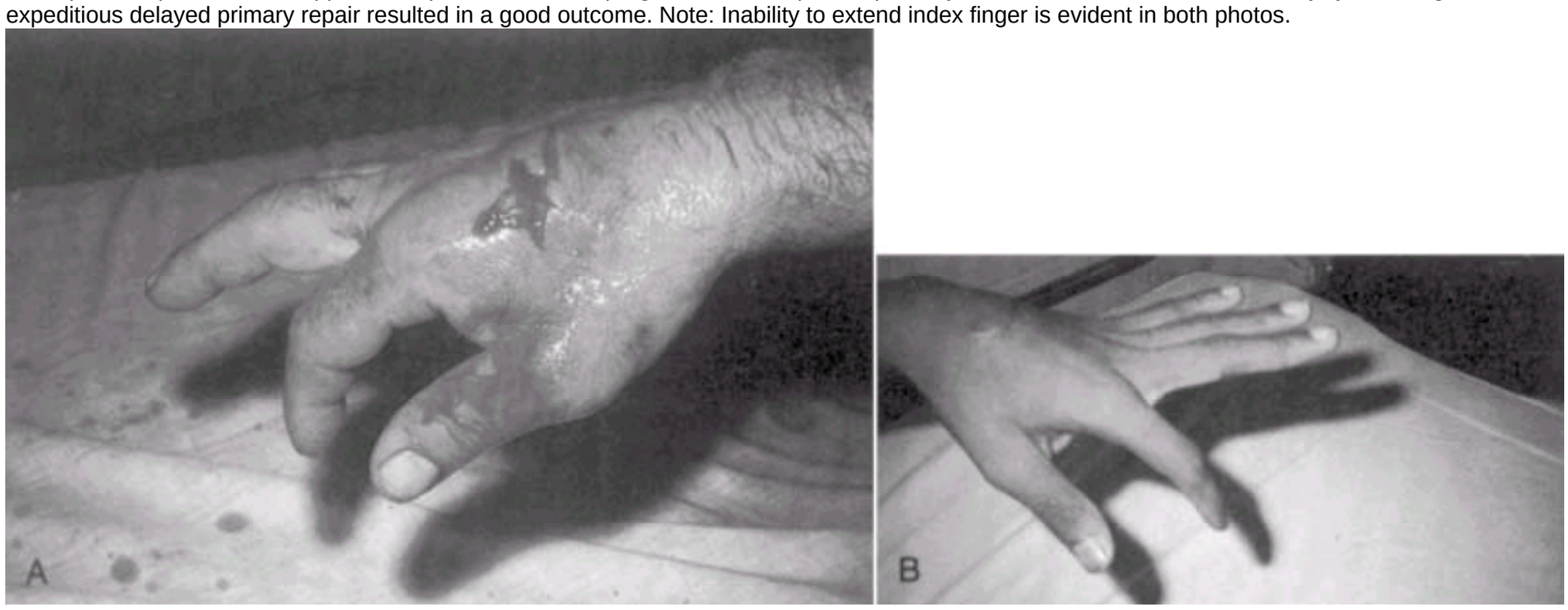


Figure 49-8 The location and depth of this laceration suggests an extensor tendon injury. On examination, the patient had full extension. Note that an intact tendon is visualized when the laceration is examined with the fingers in extension *(A)*. The injury occurred with a closed fist, and when the fingers were fully flexed, an 80% laceration of the extensor tendon could be viewed in the depths of the wound *(B)*. Note: Contrary to this figure, gloves should be worn by the examiner.

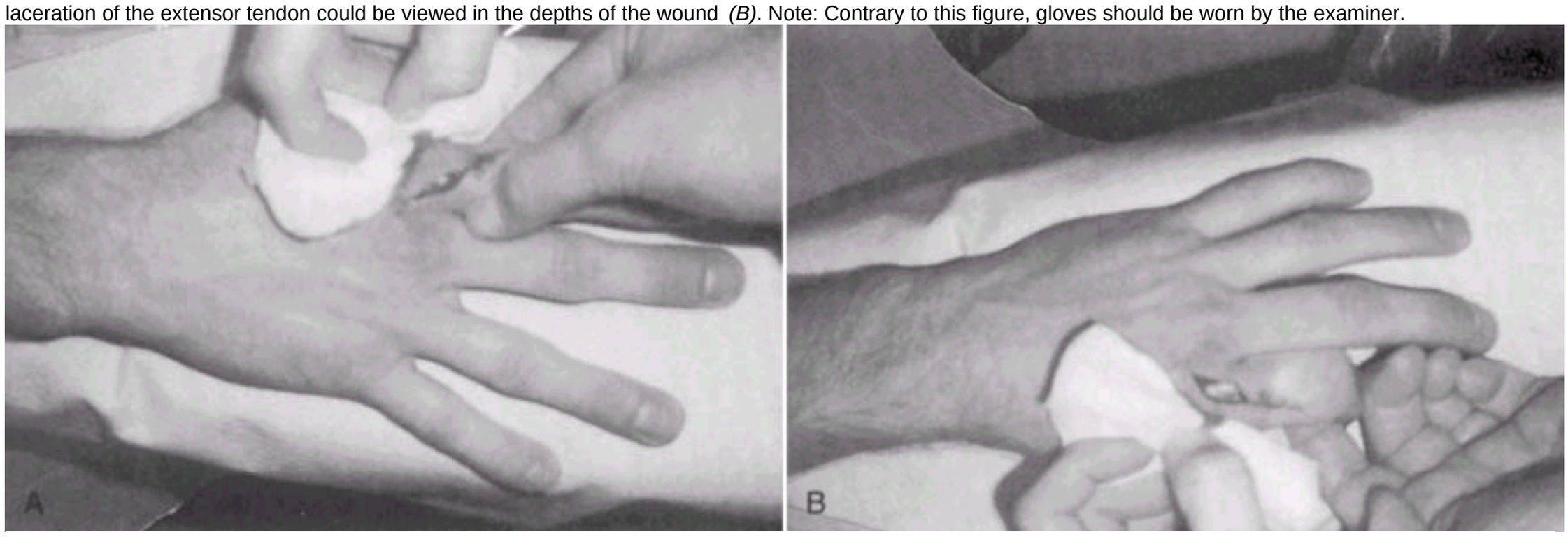


Figure 49-9 With this injury on the dorsum of the hand, the clinician should expect to find an extensor tendon injury. The proper way to examine this wound includes a bloodless field (often with the use of a blood pressure cuff tourniquet), good light, a cooperative patient, excellent anesthesia, and extending the skin laceration to visualize the depths of the laceration. Note that the tendon is visualized with the fingers manipulated from full extension to full flexion.

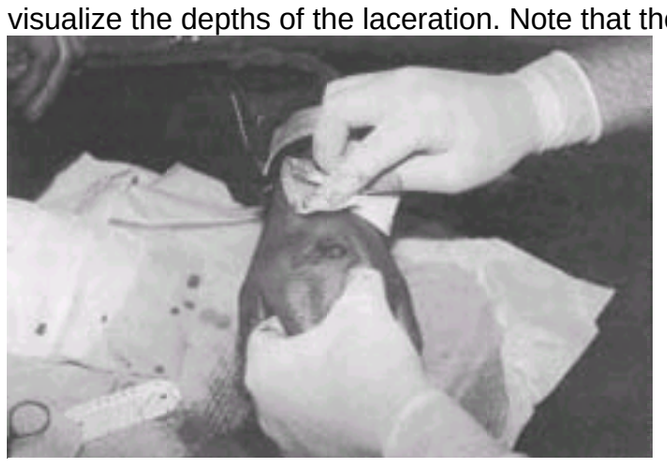


Figure 49-10 Dorsum of left hand. The injury classification system recommended by Verdan includes eight anatomically based zones. *(From Blair WF, Steyers CM: Extensor tendon injuries. Orthop Clin North Am 23:142, 1992.)*

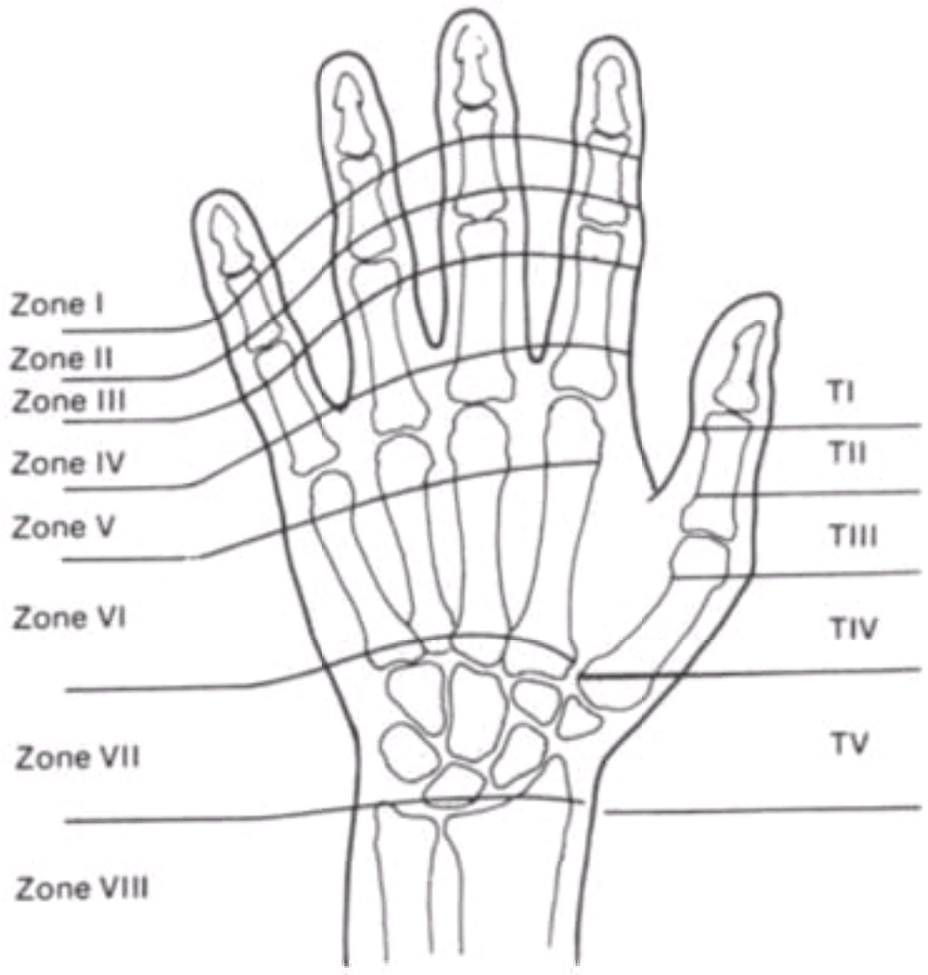


Figure 49-11 An effective way to fully immobilize a finger with a tendon laceration is to incorporate an aluminum foam splint into the middle of a standard dorsal plaster/fiberglass splint.

Figure 49-12 Suture techniques used in extensor tendon repair. *(From Newport ML, Williams CD: Biomechanical characteristics of extensor tendon suture techniques. J Hand Surg 17:1117, 1992.)*

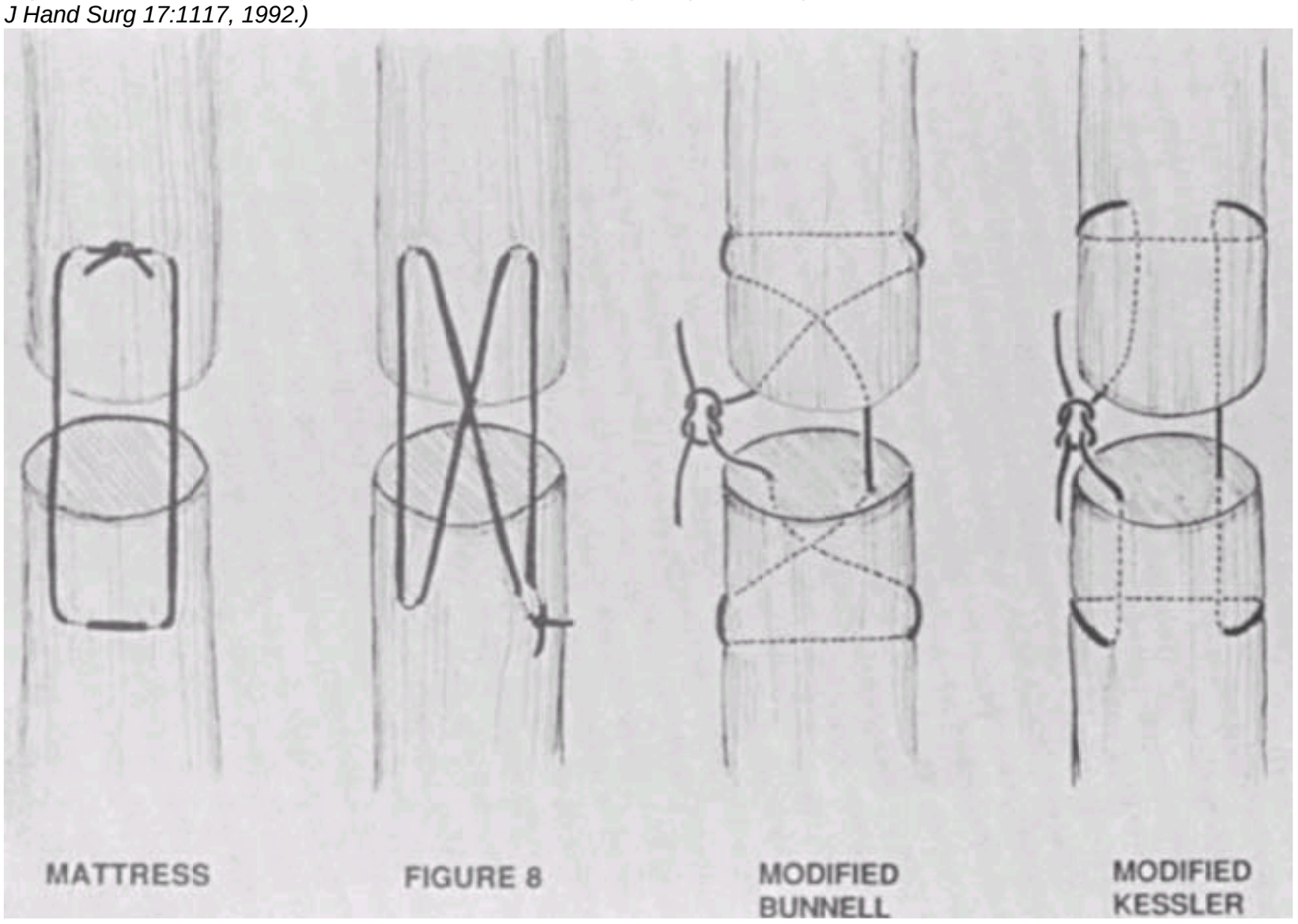


Figure 49-13 Four-strand suture techniques used in extensor tendon repair. *(Adapted from Howard RF, Ondrovic L, Greenwald DP: Biomechanical analysis of four-strand extensor tendon repair techniques. J Hand Surg 22:839, 1997.)*

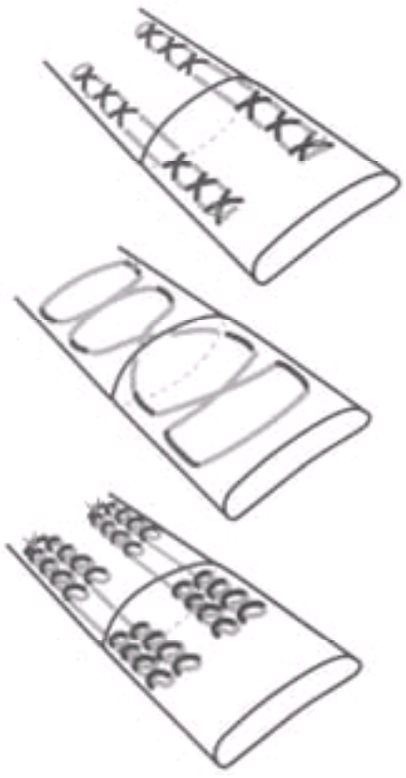


Figure 49-14 *A,* Regardless of this patient's history, this wound is highly suggestive of a human bite injury. Human bites cause extensor tendon injuries, fractures, and joint capsule injuries, and can harbor foreign bodies. *B,* This patient stated that he cut his hand on a piece of metal at work (expecting a Workers Compensation claim), but was unable to explain the piece of tooth that was found in the wound on exploration.

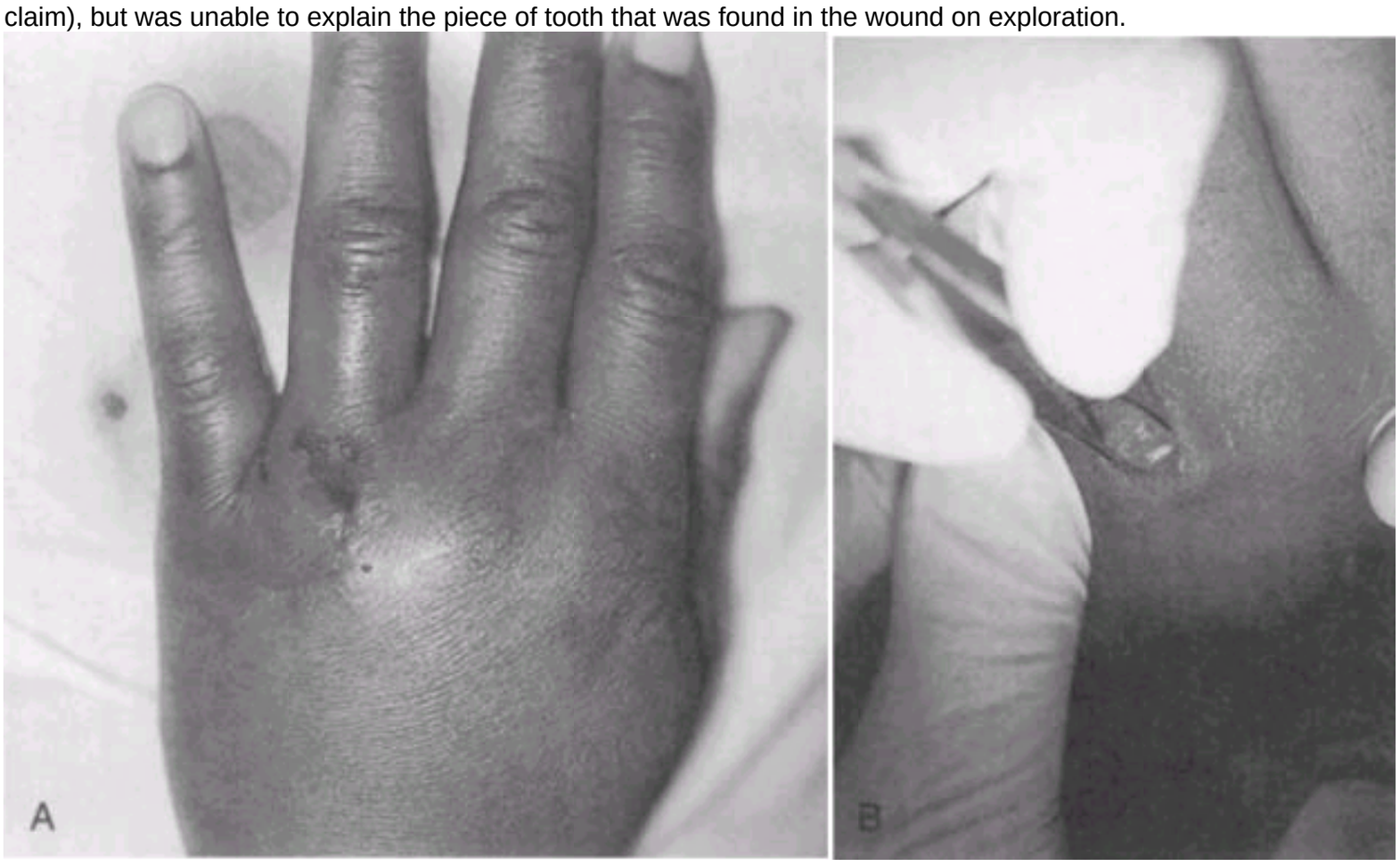


Figure 49-15 Ulnar dislocation of the extensor tendon of metacarpophalangeal joint of the middle finger. *(From Leddy JP, Dennis TR: Tendon injuries. In Strickland JW, Rettic AC [eds]: Hand Injuries in Athletes. Philadelphia, WB Saunders, 1992, p 196.)*

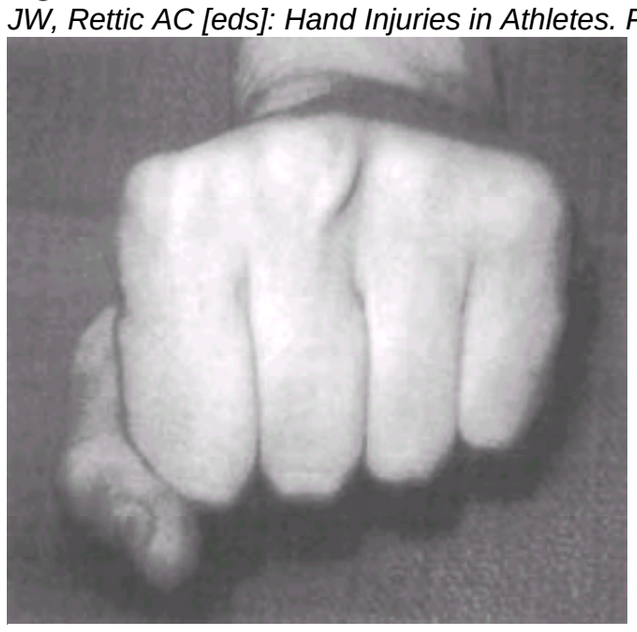


Figure 49-16 Boutonnière deformity. This can be an open or closed injury. Note flexion of the PIP joint and extension of the DIP joint, from a laceration of the central slip mechanism.

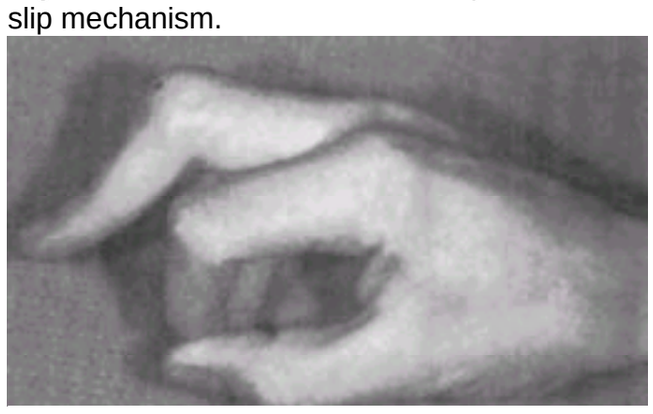


Figure 49-18 *A,* Boutonnière splint. *B,* This splint allows active flexion at the metacarpophalangeal and distal interphalangeal joints.

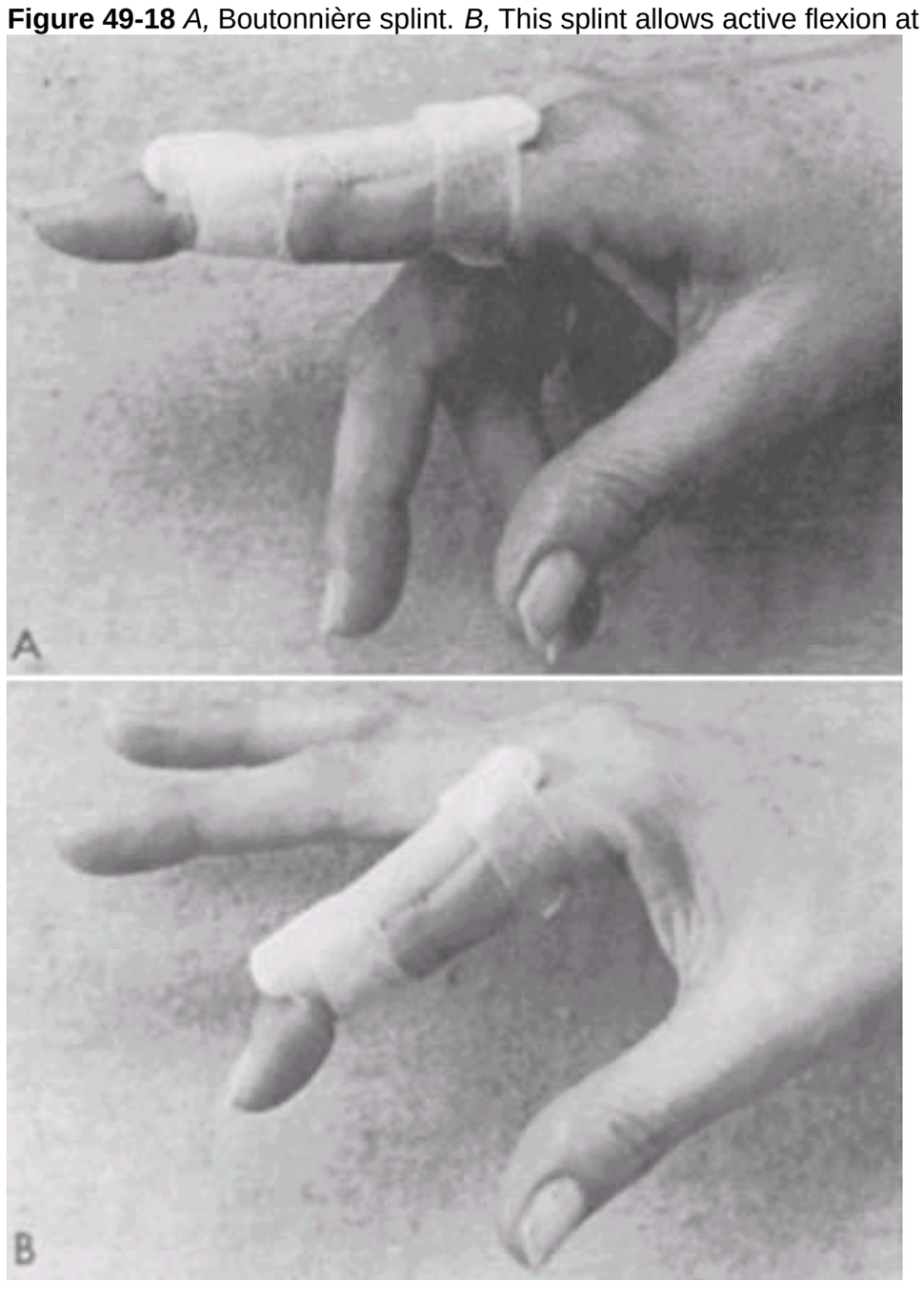


Figure 49-19 Mallet finger deformity. *(From Leddy JP, Dennis TR: Tendon injuries. In Strickland JW, Rettic AC [eds]: Hand Injuries in Athletes. Philadelphia, WB Saunders, 1992, p 180.)*

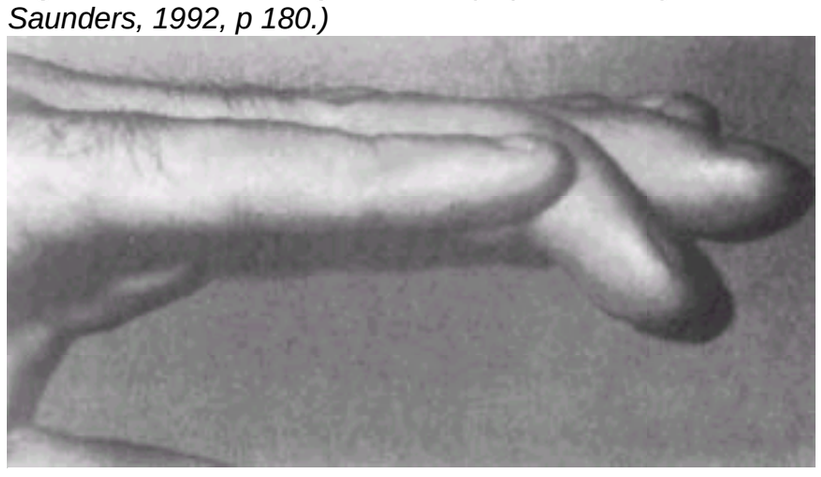


Figure 49-20 Dermatotenodesis technique for zone I extensor tendon repair. *A,* Fresh lacerations of the extensor mechanism over the distal joint with mallet finger deformity are repaired by a running-type suture, which simultaneously approximates the skin and tendon *(B and C)*. A small dressing is applied along with a splint, which maintains the joint in full extension. The sutures are removed at 10 to 12 days, but the splint is continued for a total of 6 weeks. *(Adapted from Doyle JR: Extensor tendons: Acute injuries. In Green DP [ed]: Operative Hand Surgery, 4th ed. New York, Churchill Livingstone, 1999, p 1968.)*

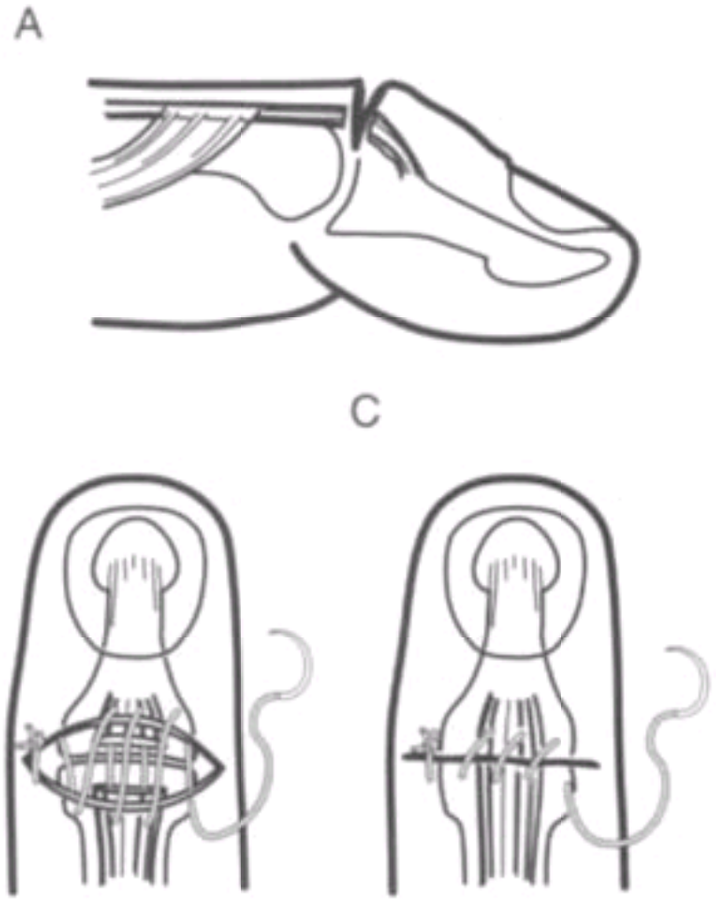


Figure 49-21 Repair of zone II extensor tendon lacerations *(A)*. Zone II lacerations may be repaired with a running stitch *(B)* near the edge of the laceration, followed by the cross-stitch *(C)*. The extensor tendon in this zone is comparatively thin, and this dual-suture technique has sufficient grasping capability to restore and maintain the normal length of the extensor. The repair is protected by 6 weeks of DIP joint splinting, as for zone I injuries. *(Adapted from Doyle JR: Extensor tendons—Acute injuries. In Green DP [ed]: Operative Hand Surgery, 4th ed. New York, Churchill Livingstone, 1999, p 1971.)*

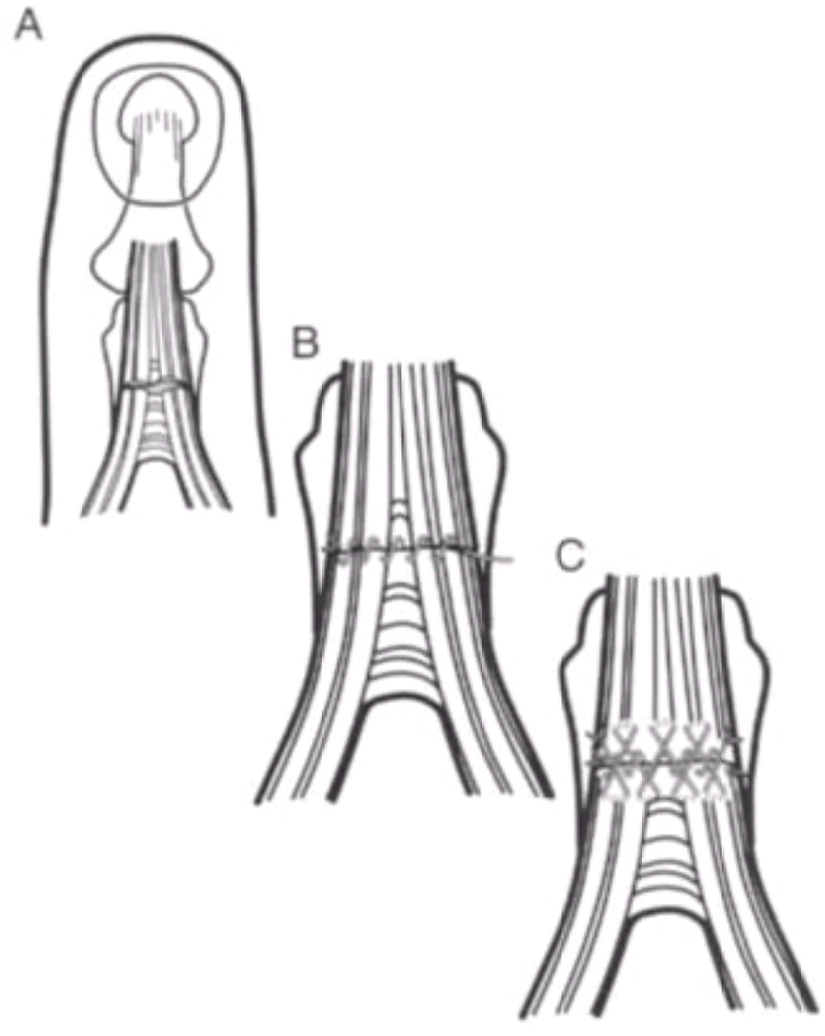


Figure 49-22 A proper mallet finger splint. Either a commercially available volar plastic splint (Stack mallet finger splint) *(A–C),* or a volar or dorsal aluminum foam splint may be used *(D and E)*. The splint should allow easy motion of the proximal interphalangeal joint. *(Adapted from Doyle JR: Extensor tendons—Acute injuries. In Green DP [ed]: Operative Hand Surgery, 4th ed. New York, Churchill Livingstone, 1999, p 1967.)*

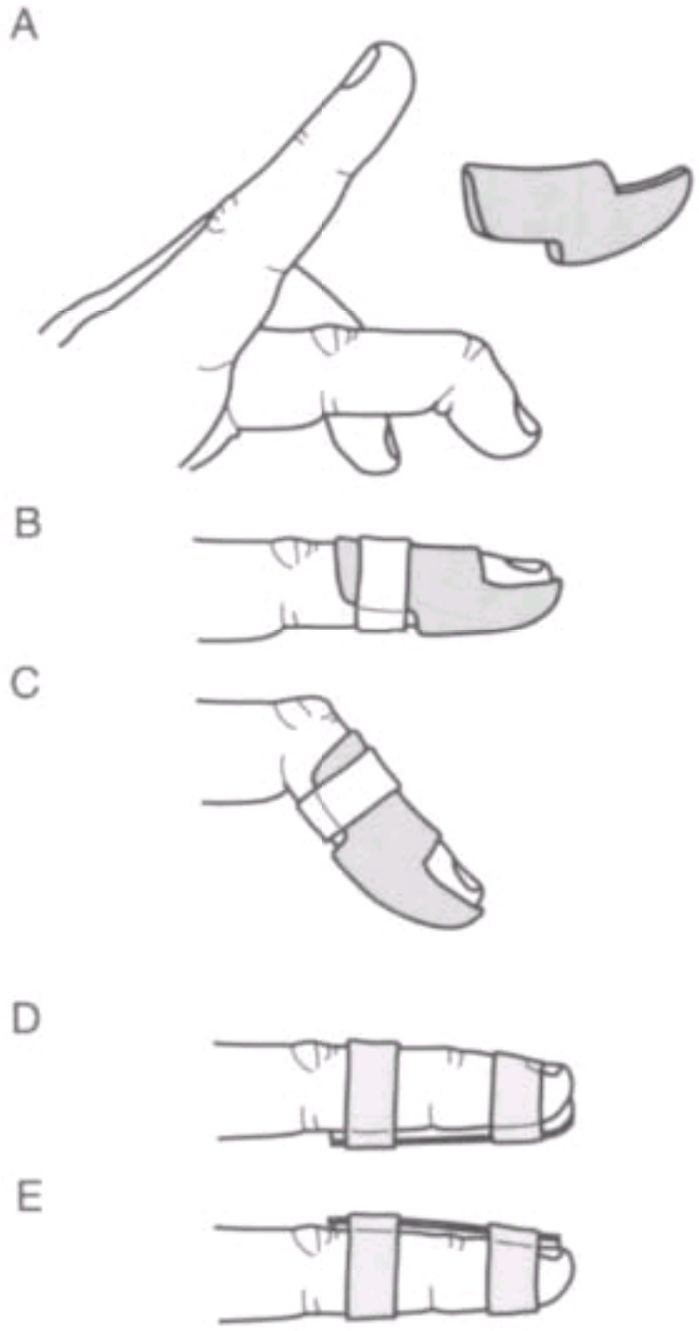


Figure 49-23 A mallet fracture with volar subluxation of the distal phalanx.

Figure 49-24 Swan neck deformity. *(Courtesy of Raymond G. Hart and Joseph E. Kutz.)*

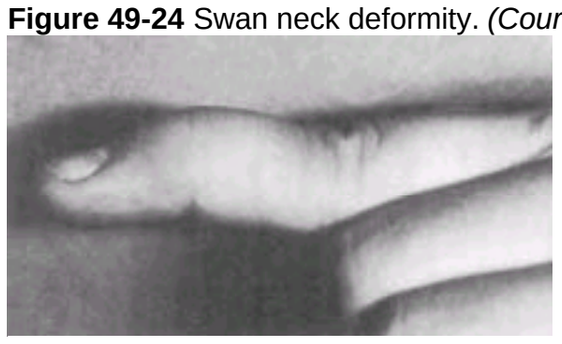


Figure 49-25 A dynamic extension splint allows for early motion following extensor tendon repair. Elastic retraction maintains the fingers in extension. Excursion of the repaired tendon is achieved by active flexion. *(Adapted from Doyle JR: Extensor tendons—Acute injuries. In Green DP [ed]: Operative Hand Surgery, 4th ed. New York, Churchill Livingstone, 1999, p 1957.)*

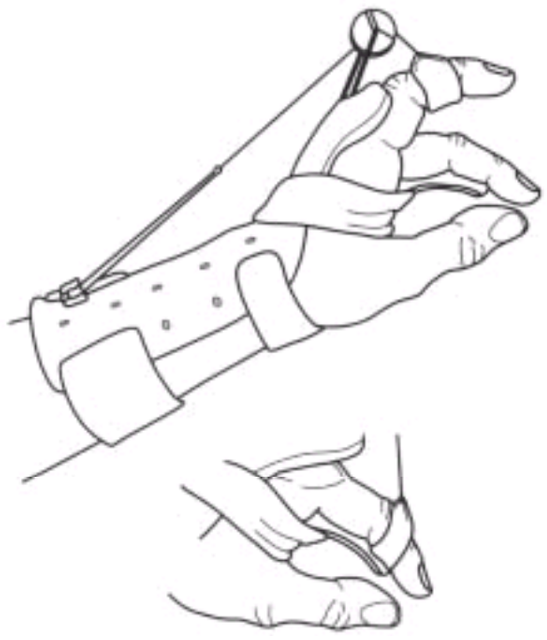


Figure 49-26 First layer of the dorsum of the foot and ankle. *1,* anterior tibial artery; *2,* anterior medial malleolar artery; *3,* anterior lateral malleolar artery; *4,* dorsalis pedis artery; *5,* first dorsal metatarsal artery; *6,* arcuate artery; *7,* dorsal metatarsal arteries; *8,* medial tarsal artery; *9, 10,* deep peroneal nerve; *11,* motor nerve branch to extensor digitorum brevis; *12,* inferior extensor retinaculum; *13,* superomedial band of inferior extensor retinaculum; *14,* inferomedial band of inferior extensor retinaculum; *15,* superolateral band of inferior extensor retinaculum; *16,* superior extensor retinaculum; *17,* tibialis anterior tendon; *18,* extensor hallucis longus tendon; *19,* extensor digitorum longus tendon; *20,* extensor digitorum brevis muscle to toes; *21,* extensor hallucis brevis muscle. *(From Sarrafian SK: Functional Anatomy of the Foot and Ankle: Descriptive, Topographic, Functional, 2nd ed. Philadelphia, JB Lippincott, p 423.)*

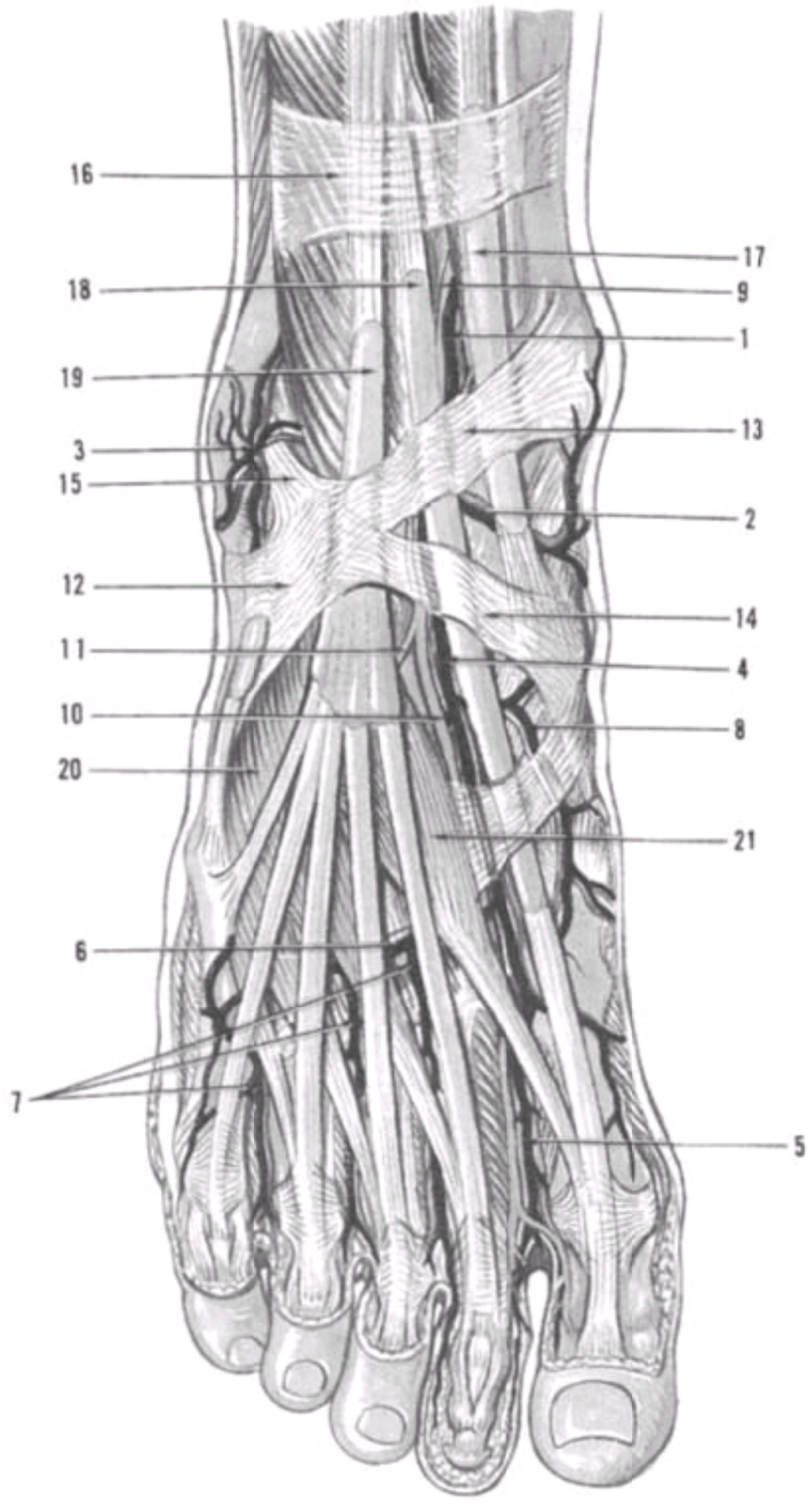
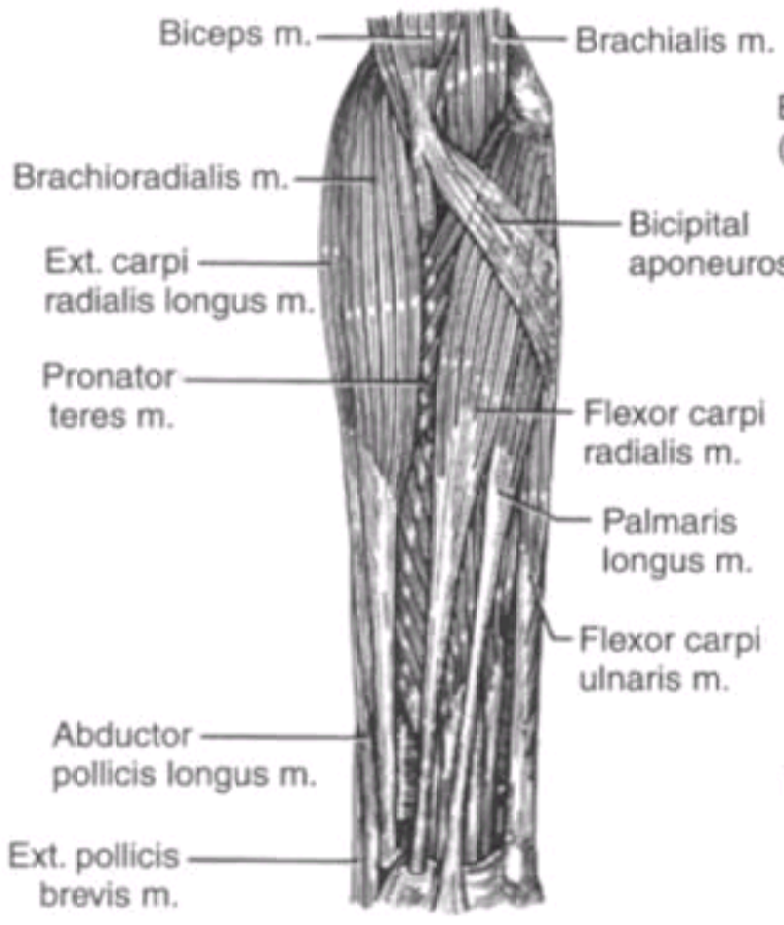


Figure 49-27 Flexor tendons of the right forearm, wrist, and hand. *A,* Superficial forearm layer. *B,* Deep forearm layer. *C,* Left wrist and palm. Proper function of the flexor tendons requires defect-free tendons and smooth tendon sheaths for easy gliding, and an intact pulley system; hence, flexor tendon lacerations are seldom repaired in the ED. *(Modified from Pansky B: Upper extremity. In Pansky B [ed]: Review of Gross Anatomy, 4th ed. New York, Macmillan Publishing, 1979, pp 219, 243.)*



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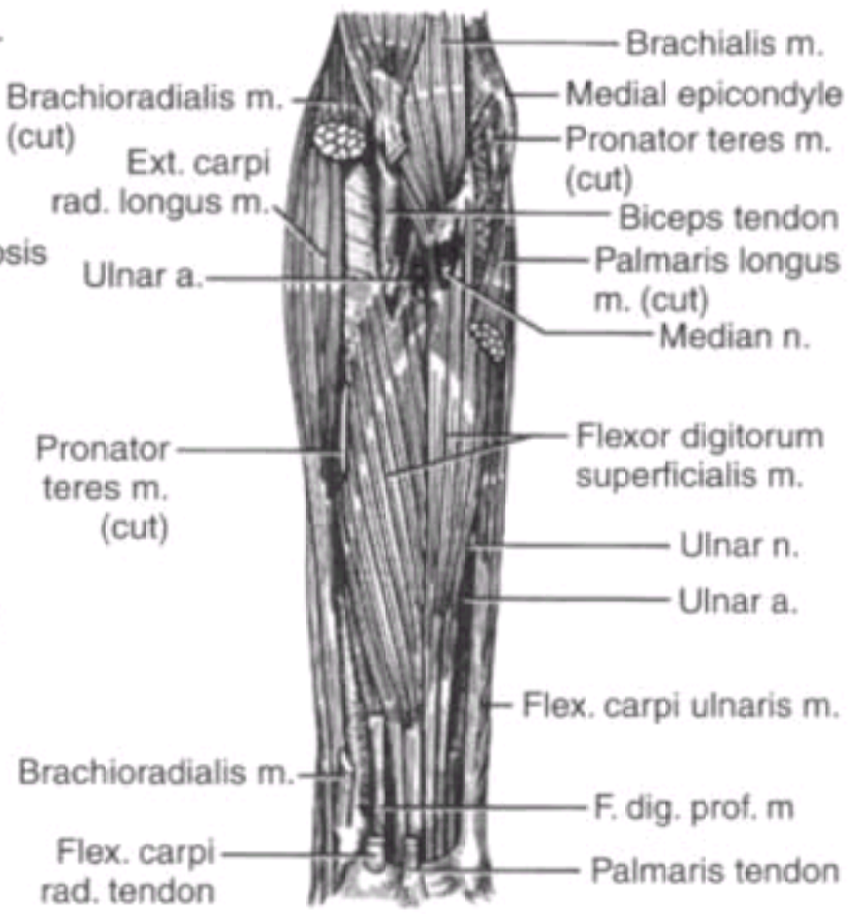

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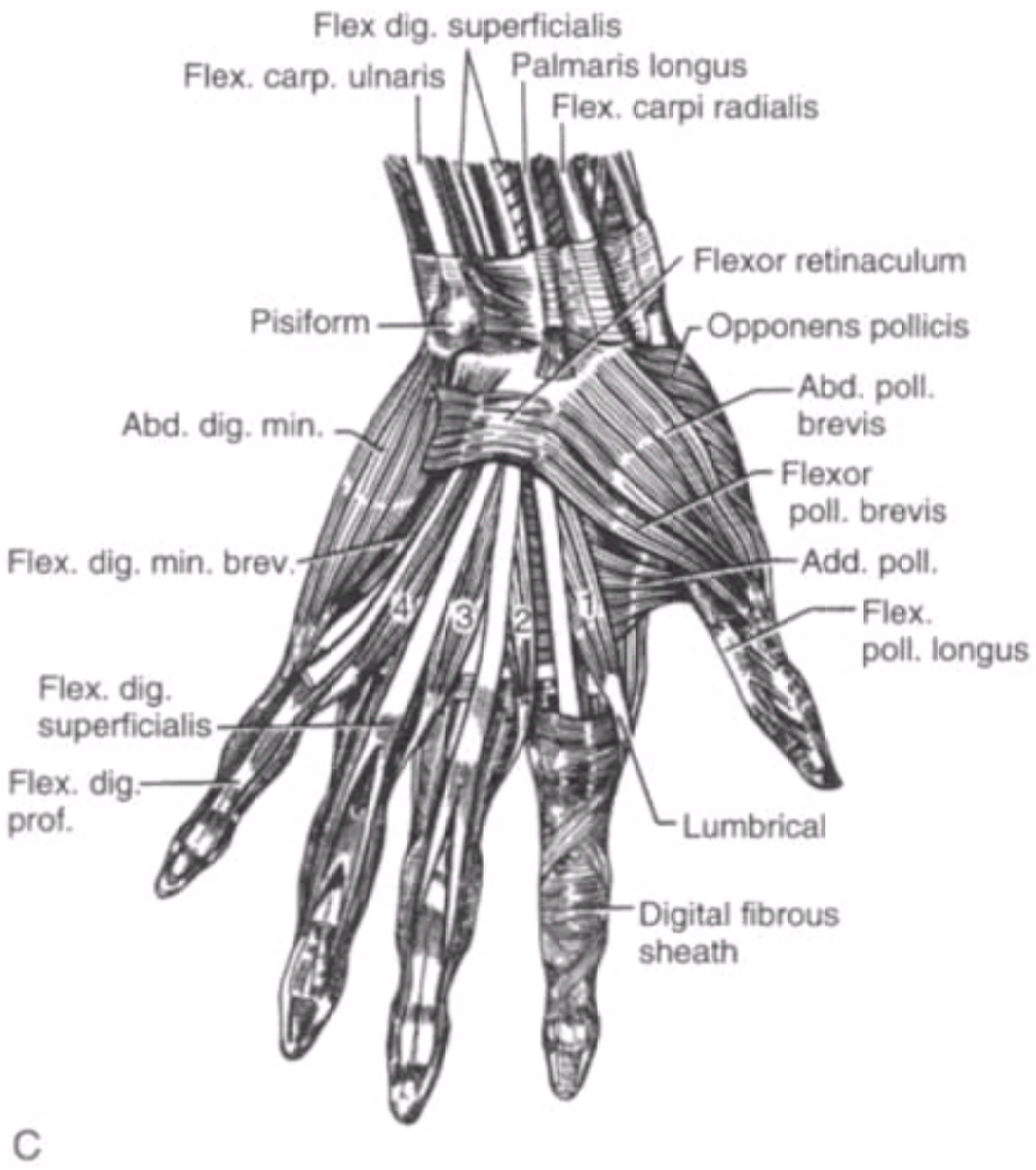


C

Figure 49-28 Deep puncture wounds of the palm may injure flexor tendons. The depth of this wound precludes extensive exploration to visualize the tendon. Partial tendon lacerations may still initially allow full function. Clues to a partial tendon laceration include weakness of flexion or pain with attempts at flexion against resistance, but many partial lacerations are clinically silent. Despite full function this wound's location and depth suggest the possibility of at least a partial tendon injury. The prudent course would include meticulous wound care, splinting, skin closure, and contact with a hand specialist to arrange re-examination in a few days, while cautioning the patient that further care may be required.

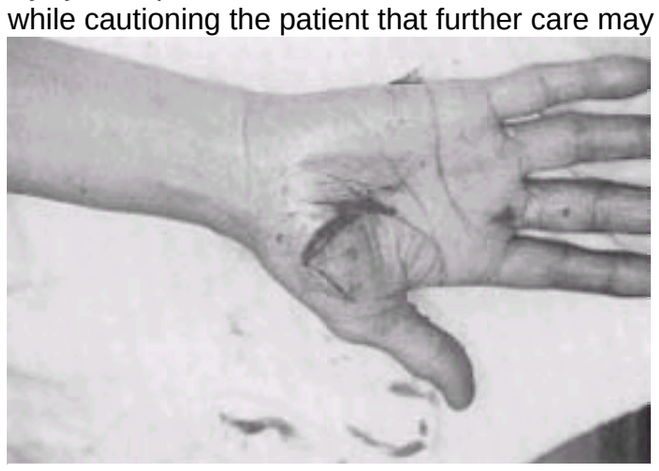


Figure 49-29 *A,* The flexor digitorum profundus tendon is examined by immobilizing the digit in question and asking the patient to flex the distal interphalangeal joint against resistance. *B,* The flexor digitorum superficialis tendon is examined by immobilizing the digits not being tested and asking the patient to flex the proximal interphalangeal joint against resistance. Pain and weakness associated with flexion against resistance may suggest a partial tendon laceration, but this is often a very subtle or inaccurate evaluation that must be repeated when pain and swelling have subsided.

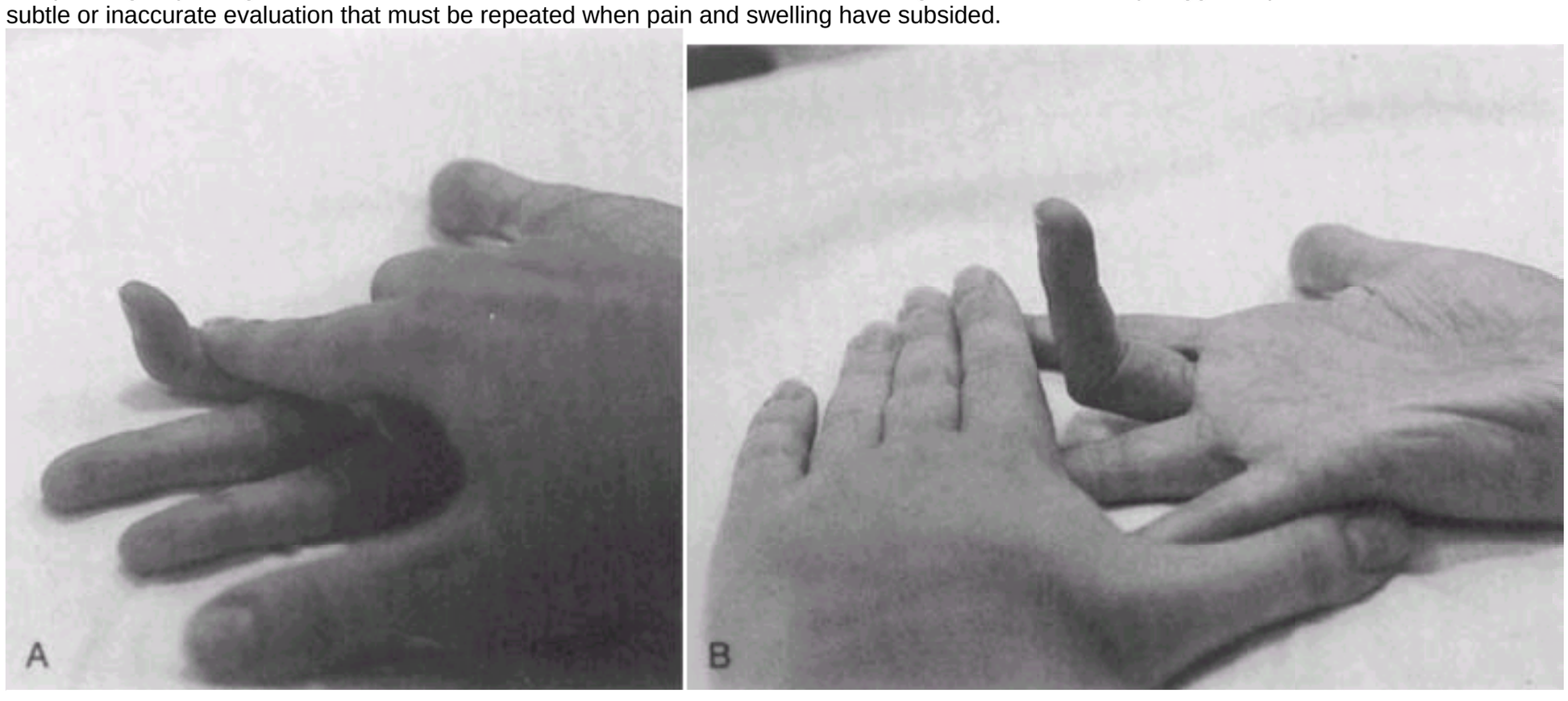


Figure 50-1 Significant vascular injuries from dislocations, such as the knee, are usually obvious, but some reduction in distal circulation may be subtle due to partial vascular compromise that heralds subsequent ischemia. The standard techniques to assess vascular injury are assessing the strength of the pulse and capillary refill, but other techniques may be helpful. While these procedures are neither well studied nor quantified, taking the blood pressure distal to the injury with a cuff and Doppler (shown here) or applying a pulse oximeter distal to the injury and comparing the results to the uninjured extremity may give some helpful clues to underlying vascular injuries. Calculating a brachial/ankle blood pressure index (see Chapter 1) is also more accurate than simple palpation.

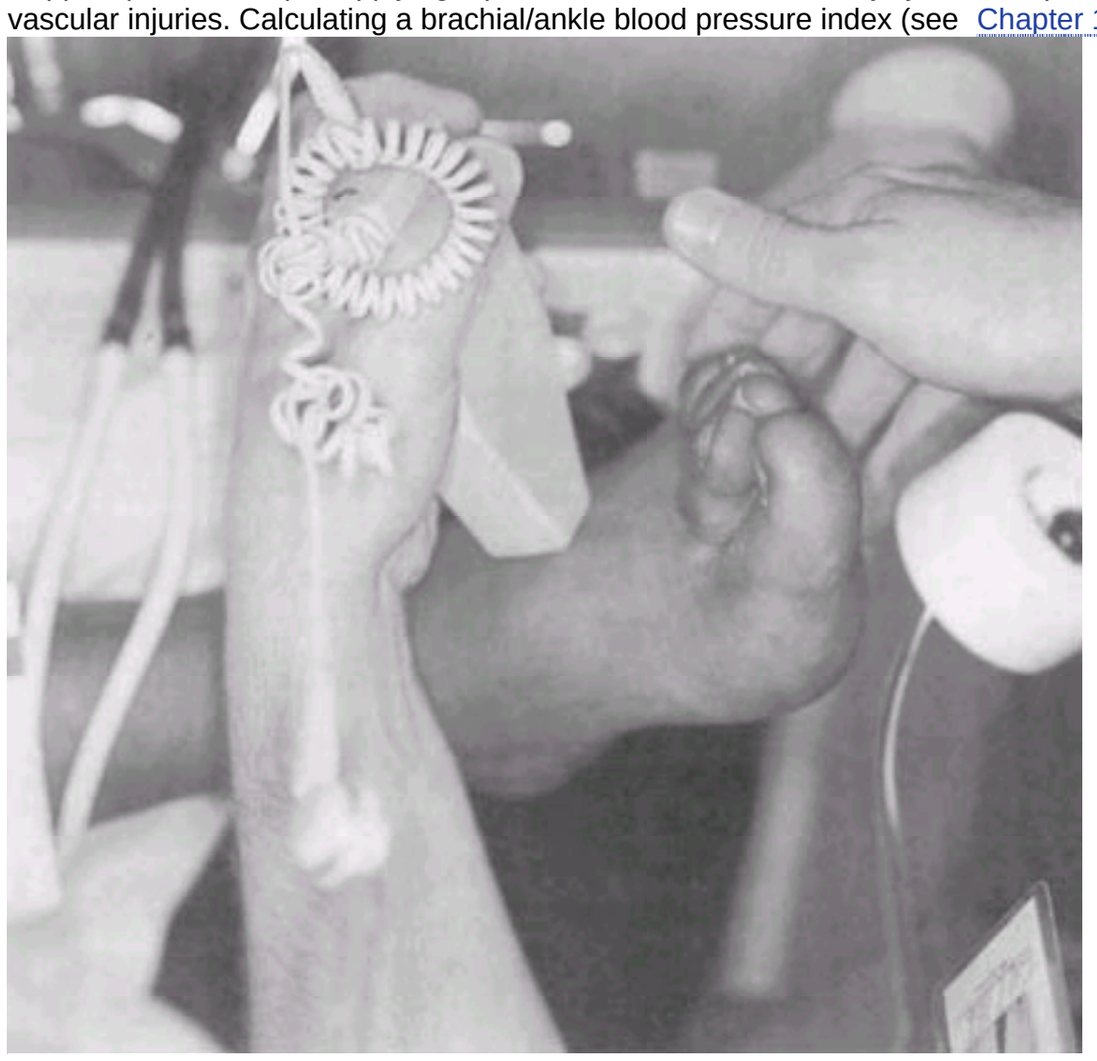
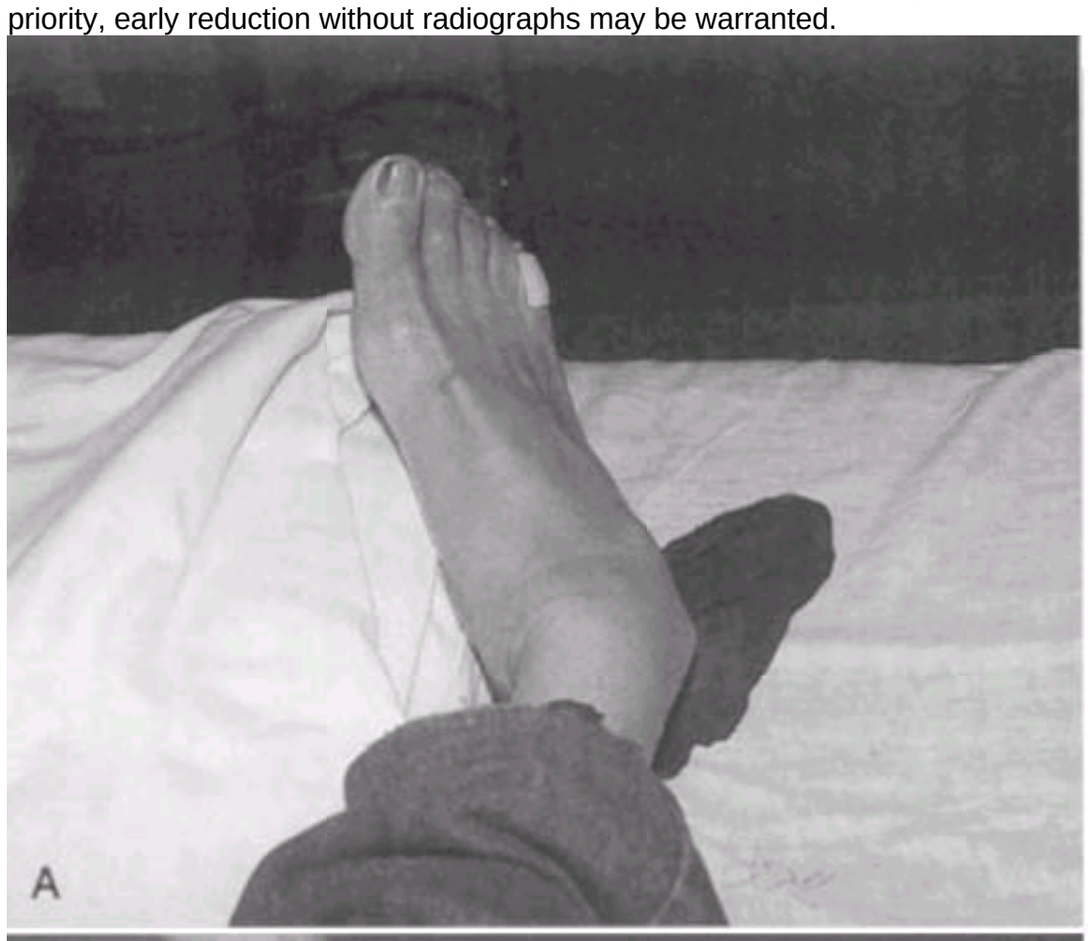
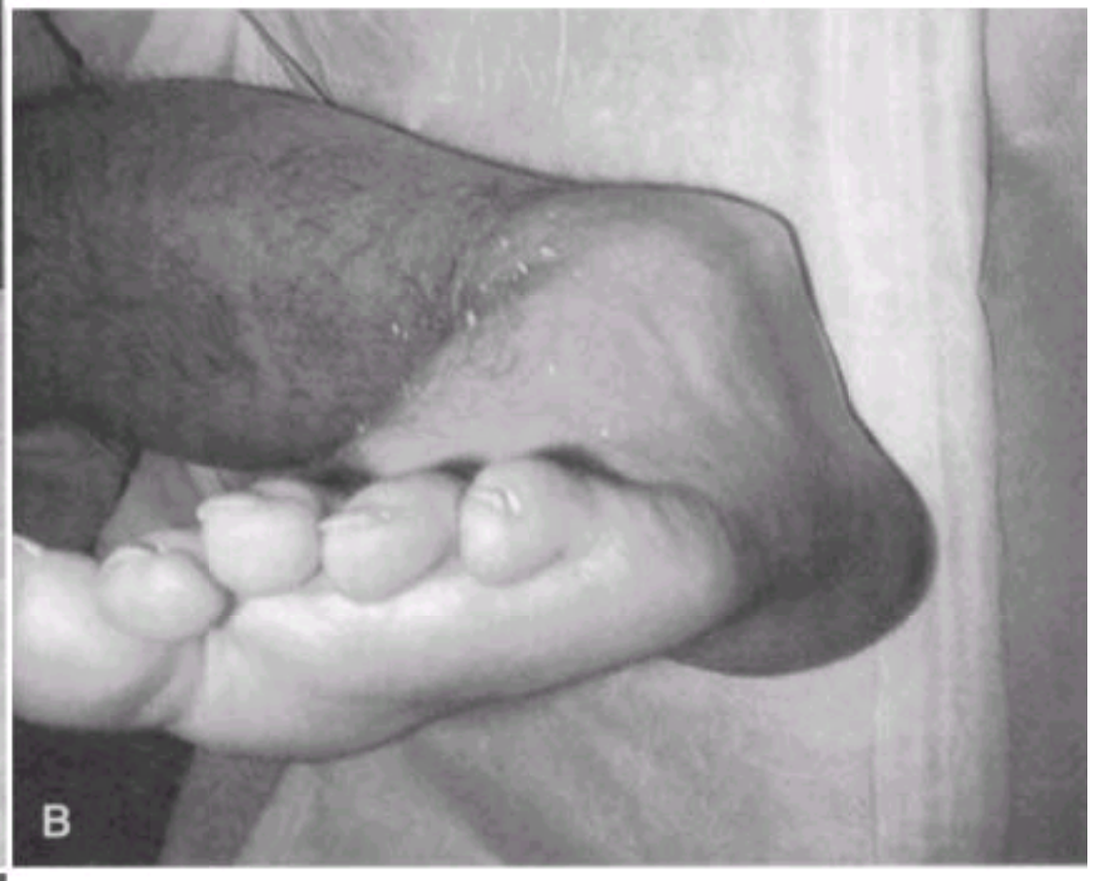


Figure 50-2 *A* and *B*, Because the distal pulse is weak and the toes are numb, it may be tempting to immediately reduce these obvious dislocations while the patient is still on the ambulance stretcher. Some clinicians prefer to first obtain proper analgesia/sedation to allow a less traumatic reduction. *C*, Prereduction radiographs (even limited-view portable films are acceptable) can be helpful since once the reduction is accomplished, the specific initial injury may be impossible to reconstruct from the physical examination alone. The few minutes required to properly prepare the patient for reduction and to document the initial injury should not result in a more adverse outcome than has been prognosticated by the initial injury. However, when the patient has sustained multiple trauma and extremity films are a low priority, early reduction without radiographs may be warranted.





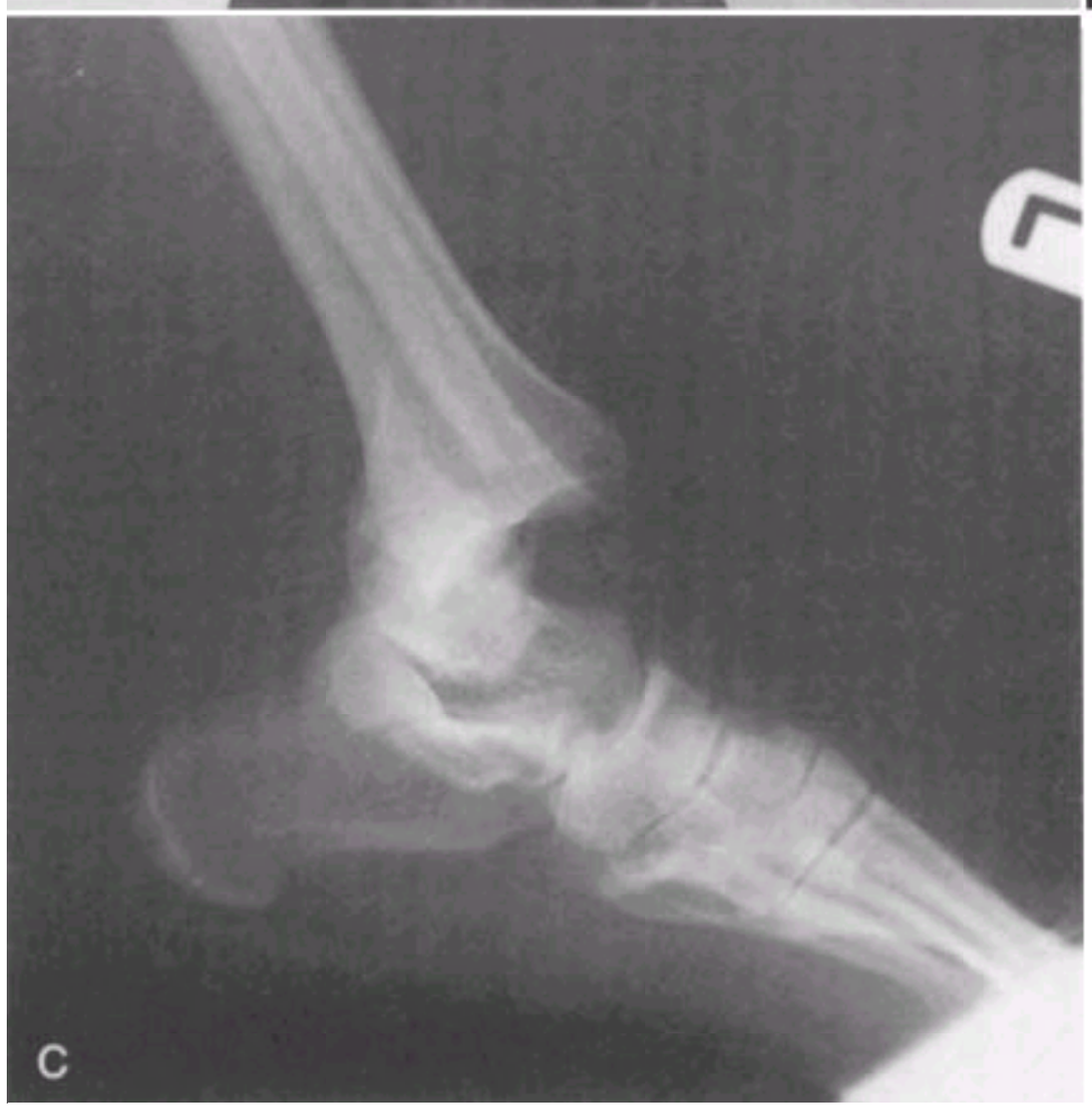


Figure 50-3 Types of anterior dislocations. These types of anterior dislocations should receive the same treatment. *A,* Subglenoid dislocation (rare type). *B,* Subcoracoid dislocation (most common type). *C,* Subclavicular dislocation (rare type). *(From DePalma AF: Management of Fractures and Dislocations: An Atlas. Philadelphia, WB Saunders, 1970, p 617. Reproduced by permission.)*

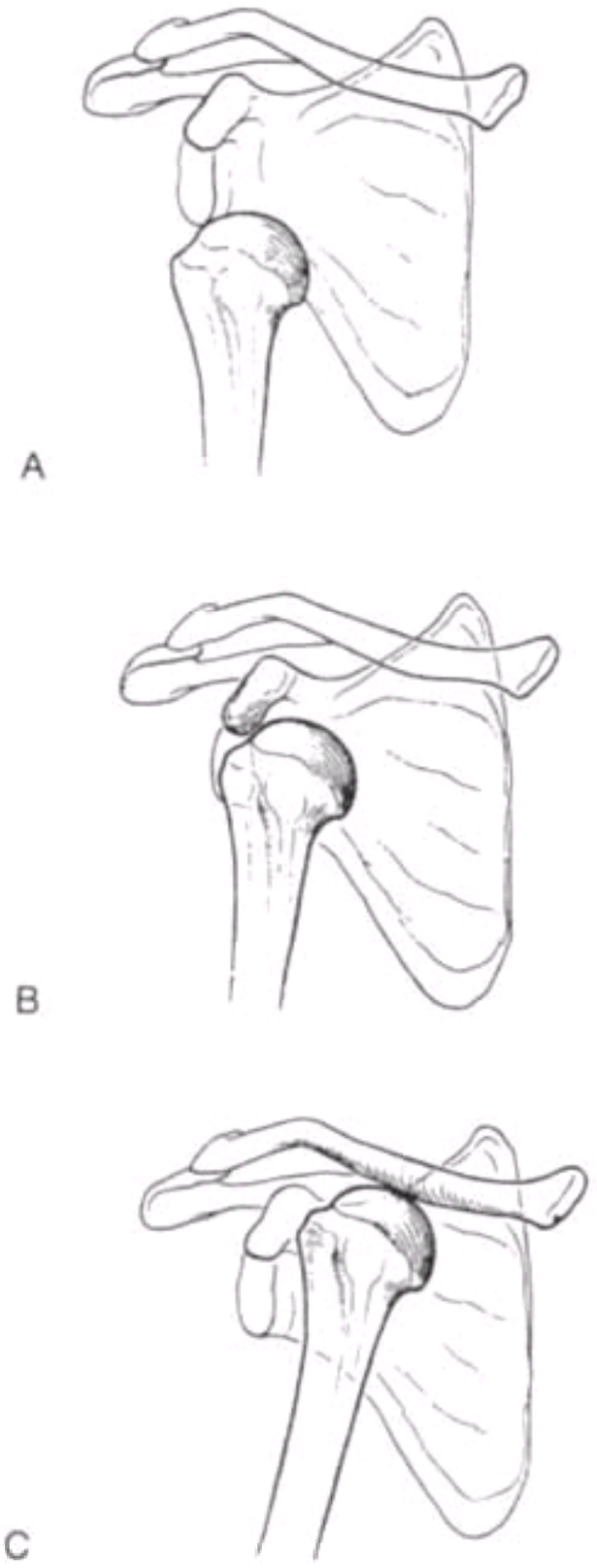


Figure 50-4 *A*, Typical presentation of an anterior right shoulder dislocation. The shoulder is very painful; thus, the patient resists movement. The outer round contour of the shoulder is flattened, and the displaced humeral head may be appreciated in the subcoracoid area. Often the patient abducts the arm slightly, bends the torso toward the injured side, and supports the flexed elbow on the injured side with the other hand. *B*, Another example of an obvious left shoulder dislocation. This chronic dislocation occurred frequently with minimal trauma, in this case from rolling over in bed.

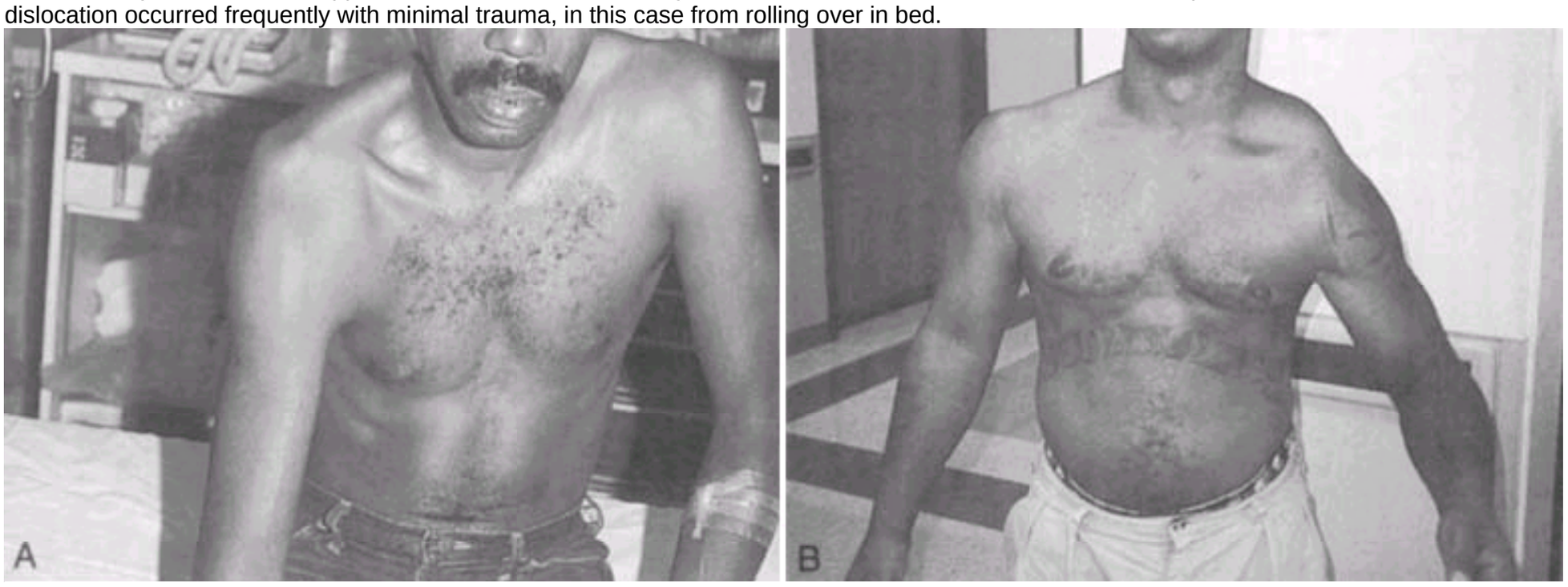


Figure 50-5 Evaluation of the upper extremity with a shoulder dislocation. Axillary (circumflex) nerve palsy is the most common neurologic complication. The axillary nerve has a sensory and motor function. Test the integrity of the nerve by assessing sensation to pin prick (*1)* in its distribution over the "regimental badge" area. (The shoulder is usually too painful to allow assessment of deltoid activity with certainty.) Look for other (rare) involvement of the radial portion of the posterior cord (*2)* and involvement of the axillary artery (*3)*. *(From McRae R: Practical Fracture Treatment. Edinburgh, Churchill Livingstone, 1981, p 84. Reproduced by permission.)*

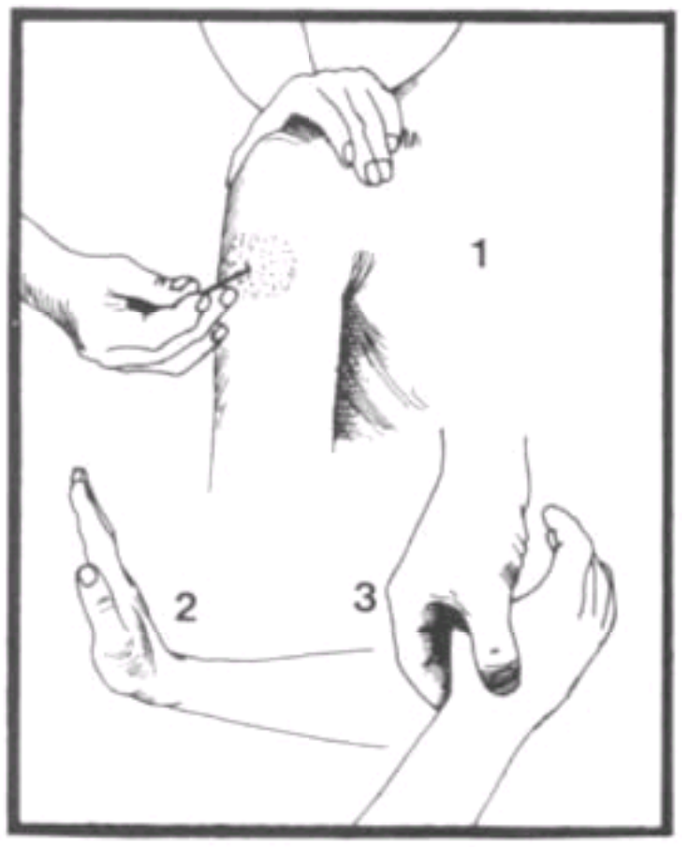


Figure 50-6 With repeated anterior shoulder dislocations, a Hill-Sachs lesion may form. During the dislocation the humeral head is damaged by the sharp anterior rim of the glenoid *(2)*. With repeated dislocation the lesion, called the "hatchet sign" develops *(3)*. On the reduction film the lesion is apparent *(4)*.

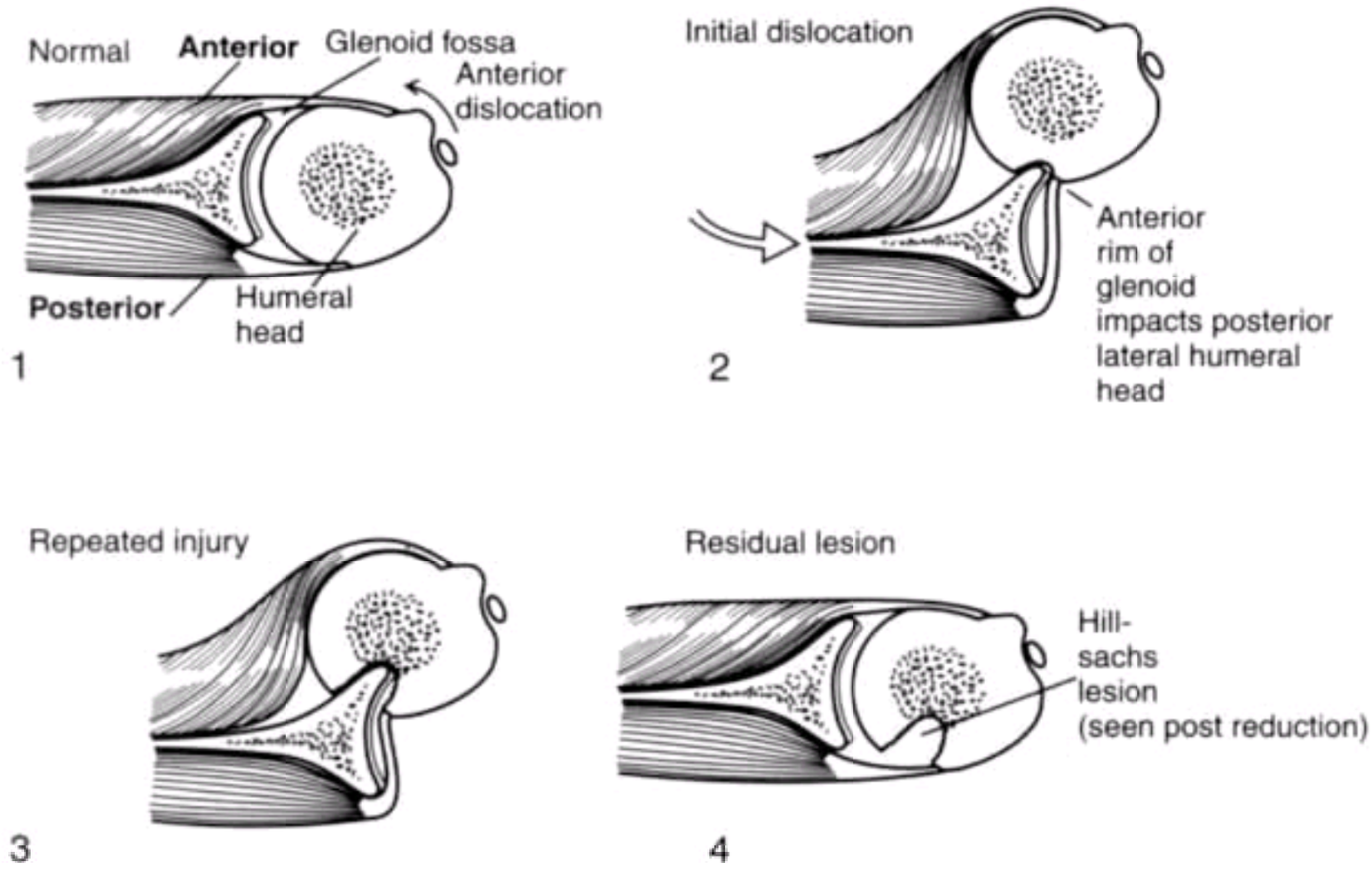


Figure 50-7 Trauma series includes two views of the shoulder made perpendicular and parallel to the scapular plane. This provides an anteroposterior (*A*) and a scapular Y (*B*) view. The advantage is that roentgenograms may be obtained without moving the patient or removing the arm from the sling. *(From Heppenstall RB: Fracture Treatment and Healing. Philadelphia, WB Saunders, 1980, p 374. Reproduced by permission.)*

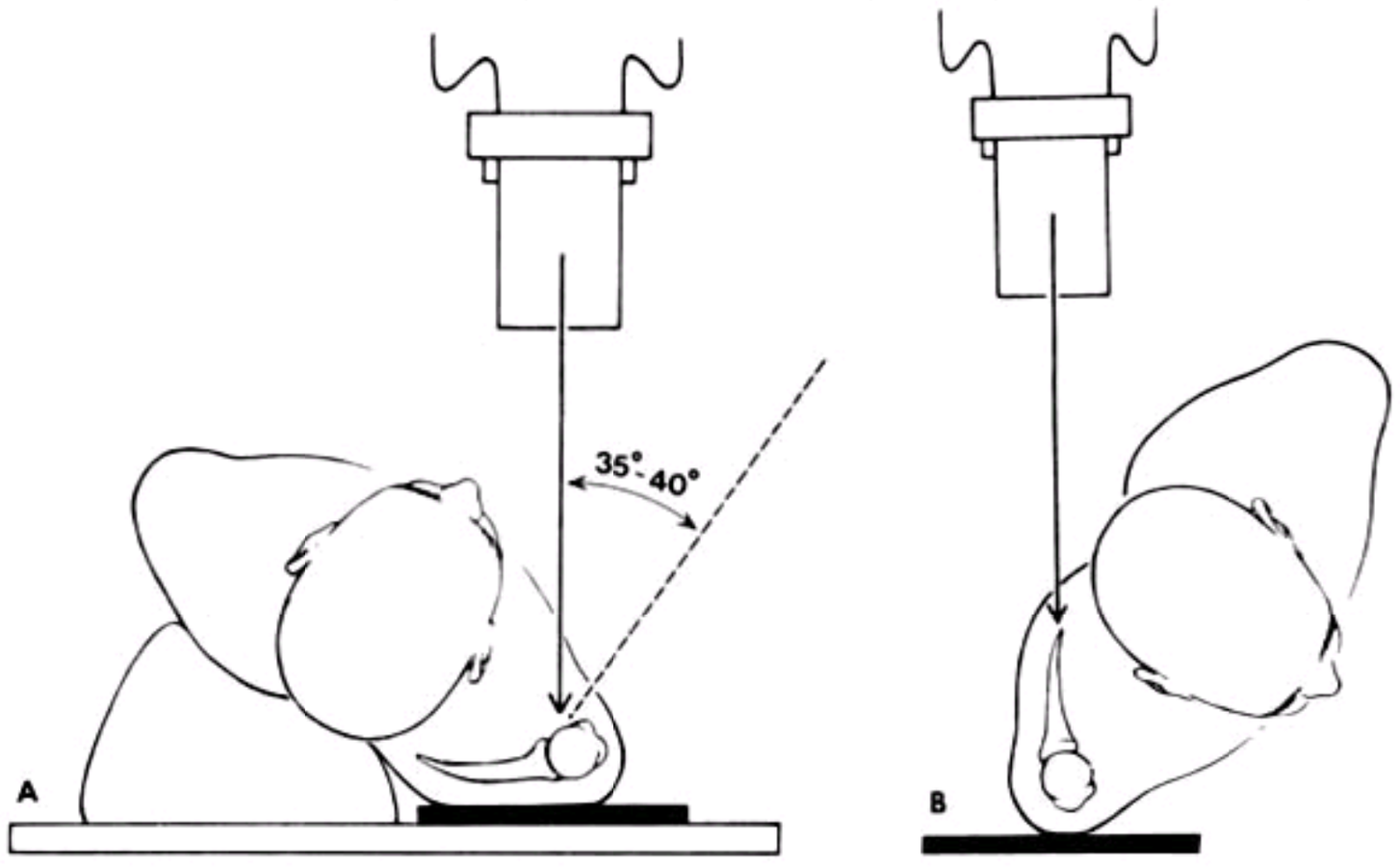


Figure 50-8 In the trauma series, a lateral view of the scapula (also called scapular Y view) demonstrates the head of the humerus displaced inferiorly and medially, the most common position for an anterior dislocation *(A)*. An anterior dislocation is shown on the anteroposterior projection *(B)*. A posterior dislocation: Transscapular projection showing the dislocated humeral head, posterior in relationship to the intersecting limbs of the Y *(C)*. *(A and B from Heppenstall RB: Fracture Treatment and Healing. Philadelphia, WB Saunders, 1980, p 392; C from Greenbaum E (ed): Radiology of the Emergency Patient. New York, John Wiley & Sons, 1982, p 512.)*

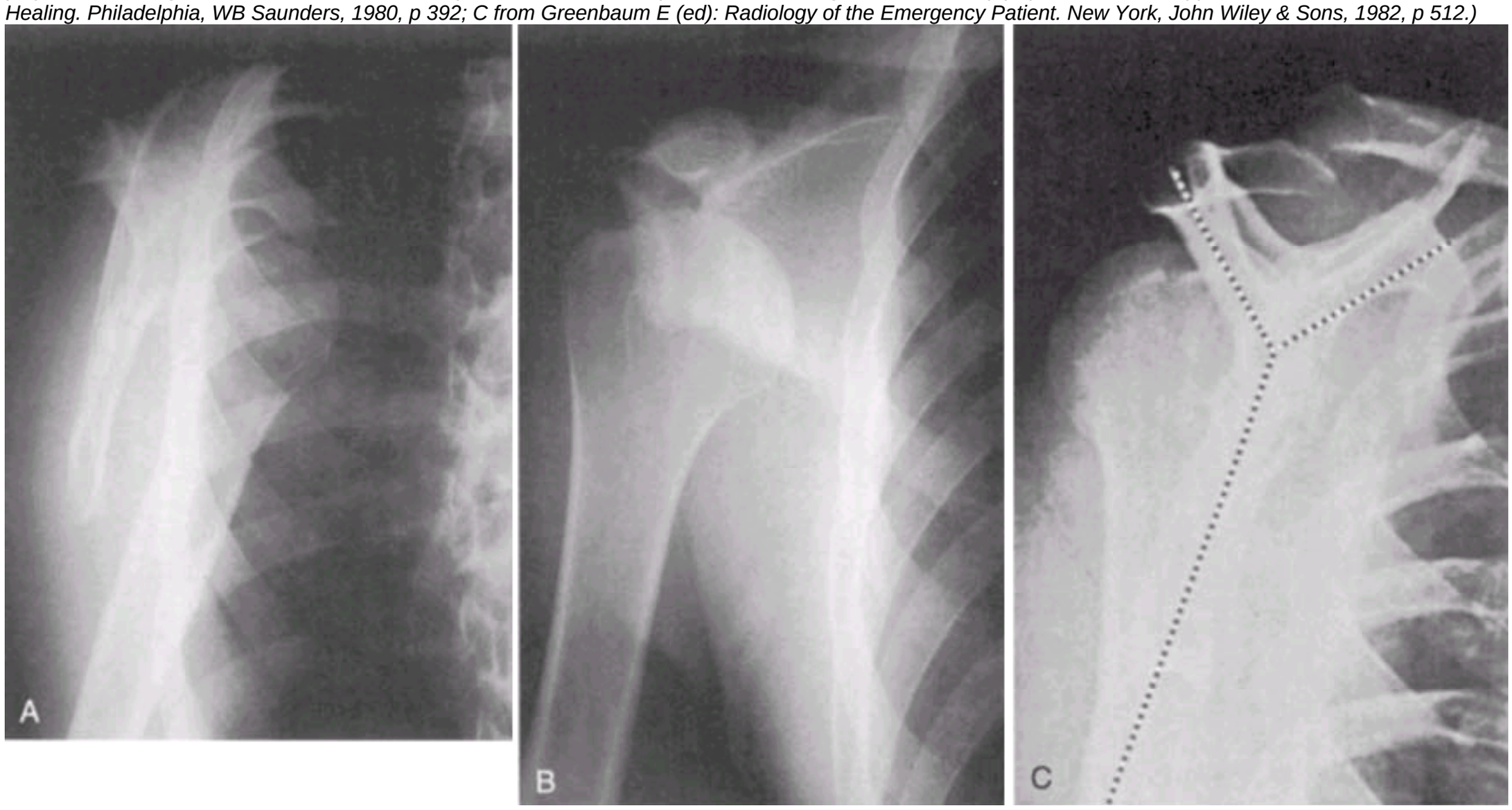


Figure 50-9 *A,* Positioning for apical oblique view. The affected shoulder is placed at a 45° oblique position and the central ray is angled 45° caudad. The affected arm is adducted. *B,* Normal apical oblique view. *(A and B from Heppenstall RB: Fracture Treatment and Healing. Philadelphia, WB Saunders, 1980, p 392. Reproduced by permission.)*

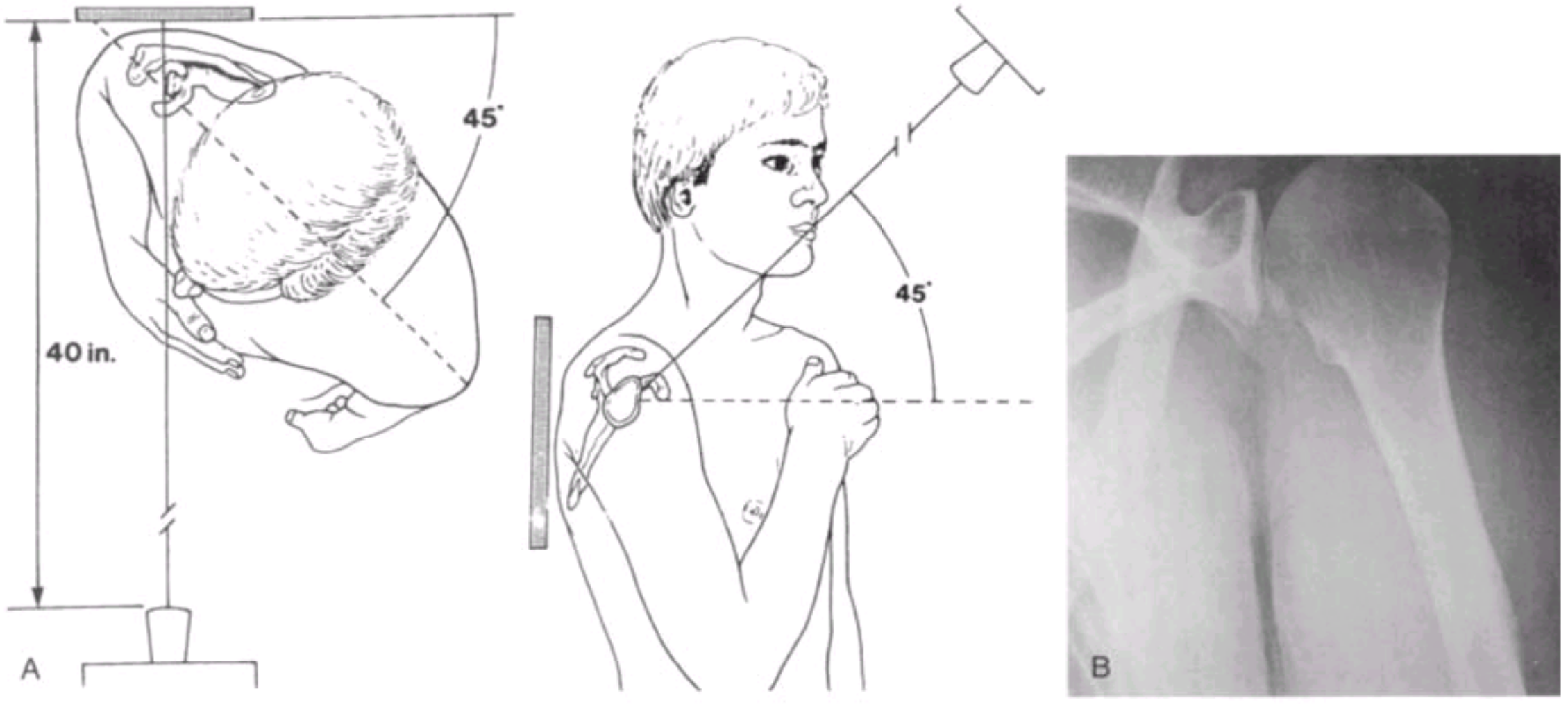


Figure 50-10 Intra-articular injection for the reduction of an acute anterior shoulder reduction. *A,* After aspirating blood from the joint, 10 to 20 mL of 1% plain lidocaine is slowly injected through the lateral sulcus, aiming slightly caudad. *B,* Anterior view. Allow 15 to 20 minutes for the lidocaine to take effect. *(From Matthews DE, Roberts T: Intraarticular lidocaine versus intravenous analgesic for reduction of acute anterior shoulder dislocations. Am J Sports Med 23:54, 1995. Reproduced by permission).*

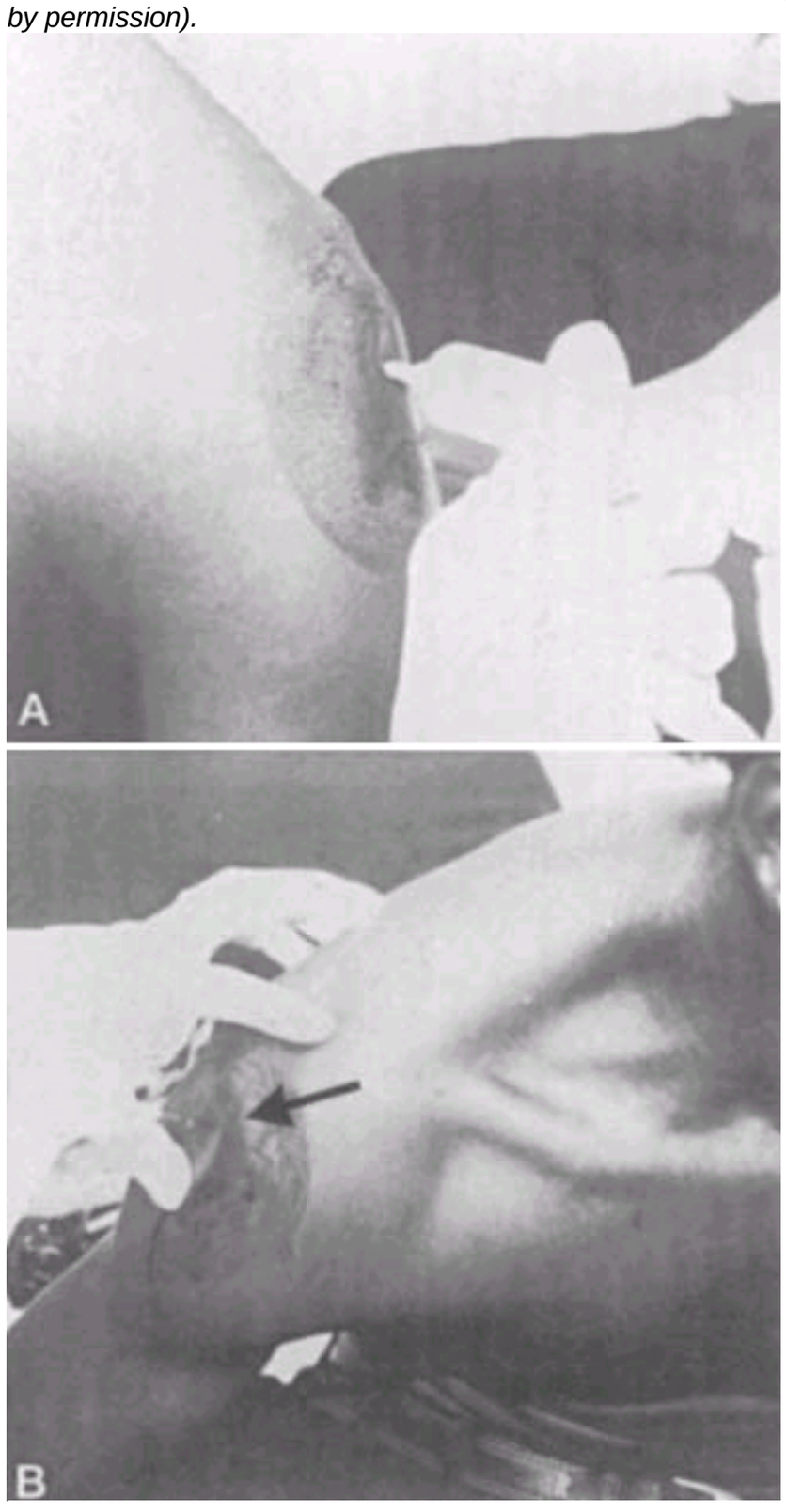


Figure 50-11 Stimson technique. This technique is often tried first, because it is the least traumatic if the patient can relax the shoulder muscles. *1,* The patient is lying prone on the edge of the table. One must be careful that the sedated or intoxicated patient does not fall off the table. Belts or sheets can be used to secure the patient to the stretcher. *2,* 5-kg weights are attached to the arm, and the patient maintains this position for 20 to 30 minutes, if necessary. *3,* Occasionally, gentle external and internal rotation of the shoulder with manual traction aids reduction. *(From DePalma AF: Management of Fractures and Dislocations: An Atlas. Philadelphia, WB Saunders, 1970, p 618. Reproduced by permission.)*

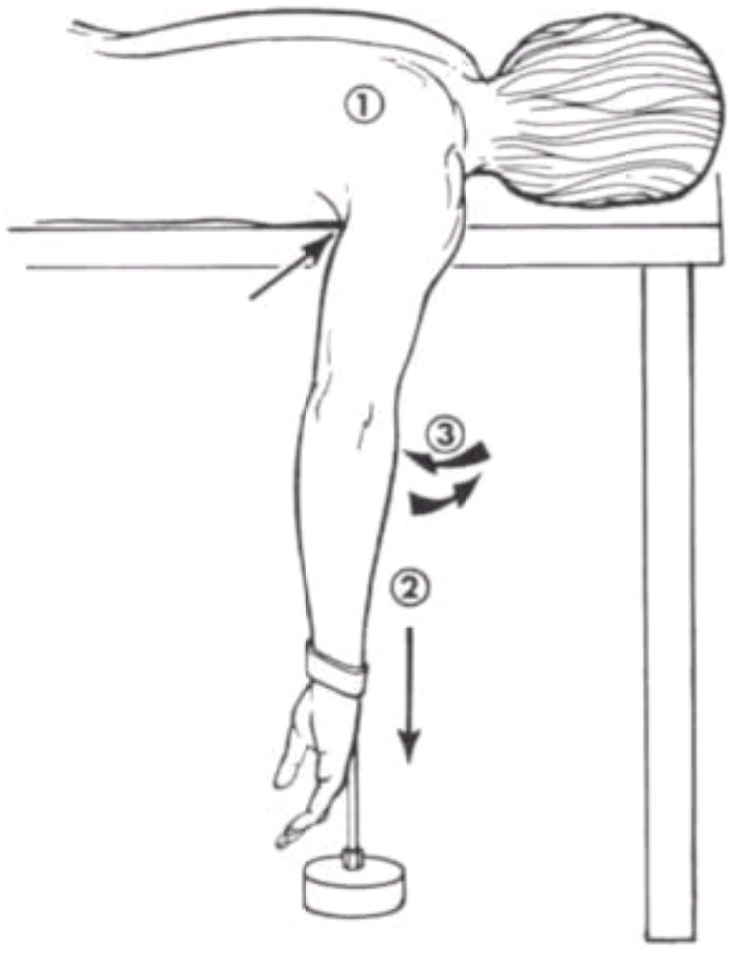


Figure 50-12 Scapular manipulation technique. *A,* The inferior tip of the scapula is pushed medially and dorsally with the thumbs while the superior aspect of the scapula is stabilized with the fingers of the superior hand. Weights may be attached to the hand to apply hanging traction. *B,* While the patient is seated, the operator applies traction with one hand and countertraction with the other, while an assistant rotates the scapula in the same manner as in *A*. *(From McNamara RM: Reduction of anterior shoulder dislocations by scapular manipulation. Ann Emerg Med 22:1140, 1995. Reproduced by permission.)*

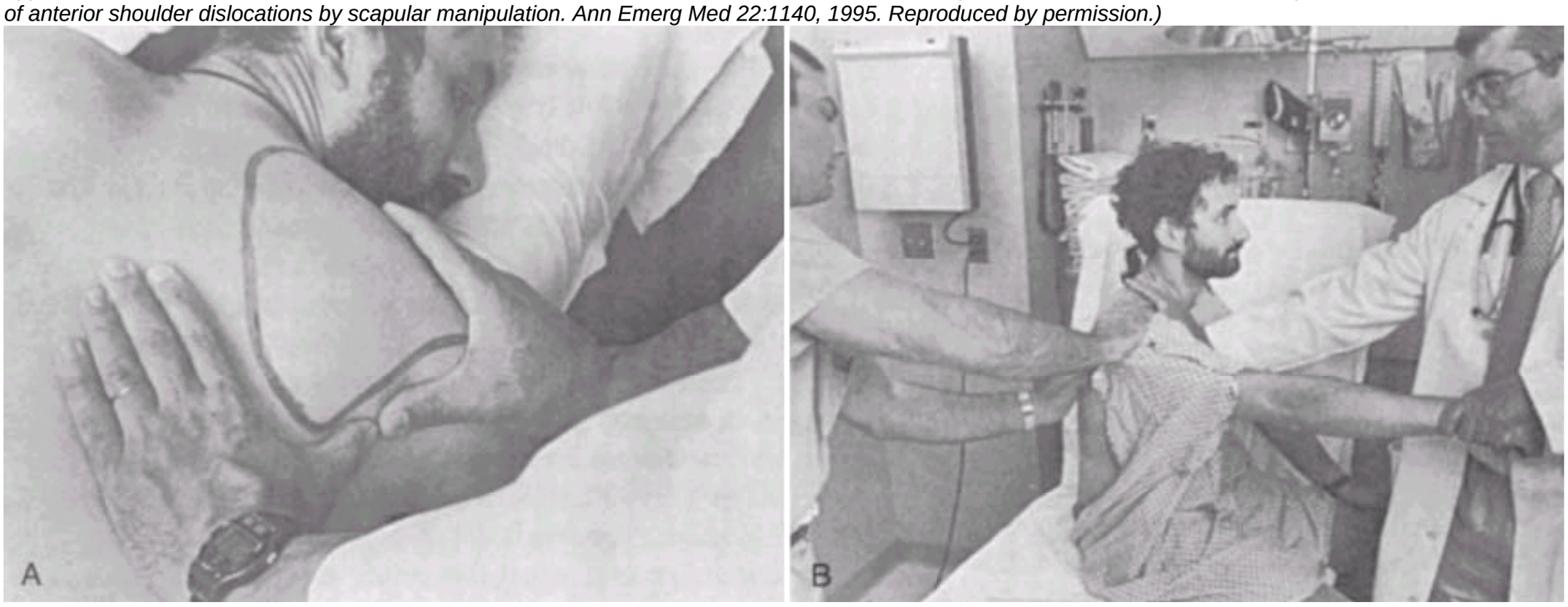


Figure 50-13 External rotation method. No traction is applied and a slow, gentle approach is essential. *A,* Arm is adducted to the patient's side. In one hand, the elbow is held flexed at 90° while the other hand grasps the wrist. *B,* Slowly and gently, the forearm is used as a lever to rotate the arm externally. *C,* Usually by the time the forearm has reached the coronal plane, the shoulder will have been reduced. *(From Mirick MJ, Clinton J, Ruiz E: External rotation method of shoulder dislocation reduction. JACEP 8:529, 1979. Reproduced by permission.)*

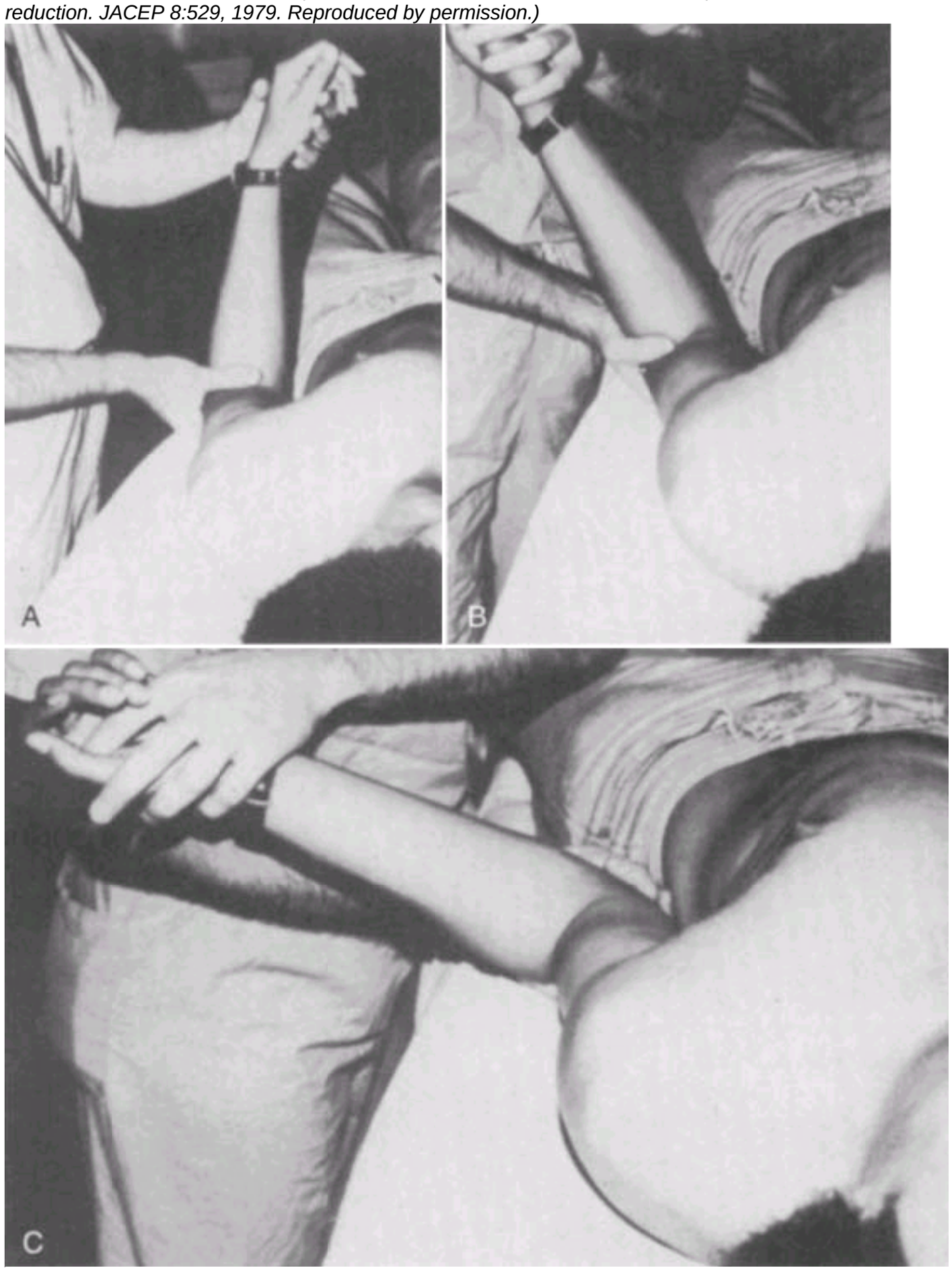
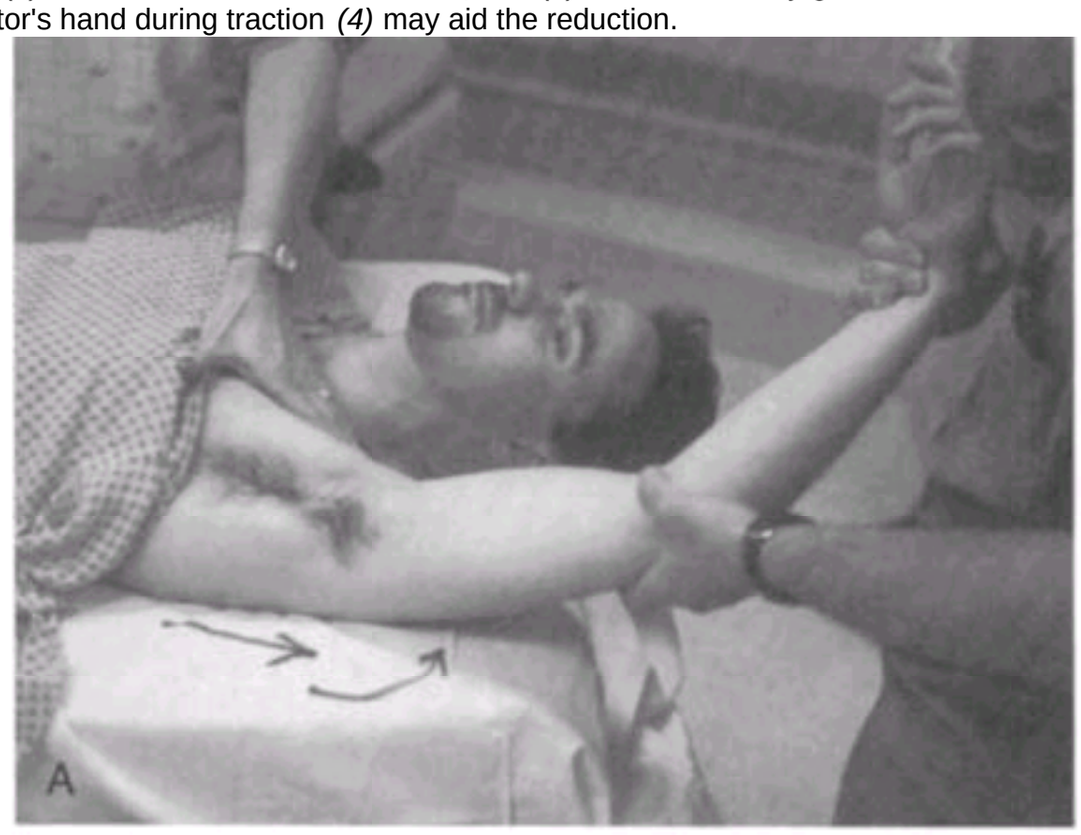


Figure 50-14 *A*, Milch technique. Slow, steady abduction with overhead-traction, external rotation (not shown), and direct pressure over the humeral head are the steps of the Milch technique. The procedure may take 3 to 4 minutes to complete, and the operator should avoid sudden, jerky manipulations. It may help to ask the patient to make a motion as if he or she is reaching up and picking an apple from a tree. *B*, The Milch method diagrammed: reduction of an anterior shoulder dislocation includes *(1)* abduction and external rotation, and *(2)* slow and steady gentle traction. When reduced, the arm is adducted *(3)*. Pressure to the humeral head with the operator's hand during traction *(4)* may aid the reduction.



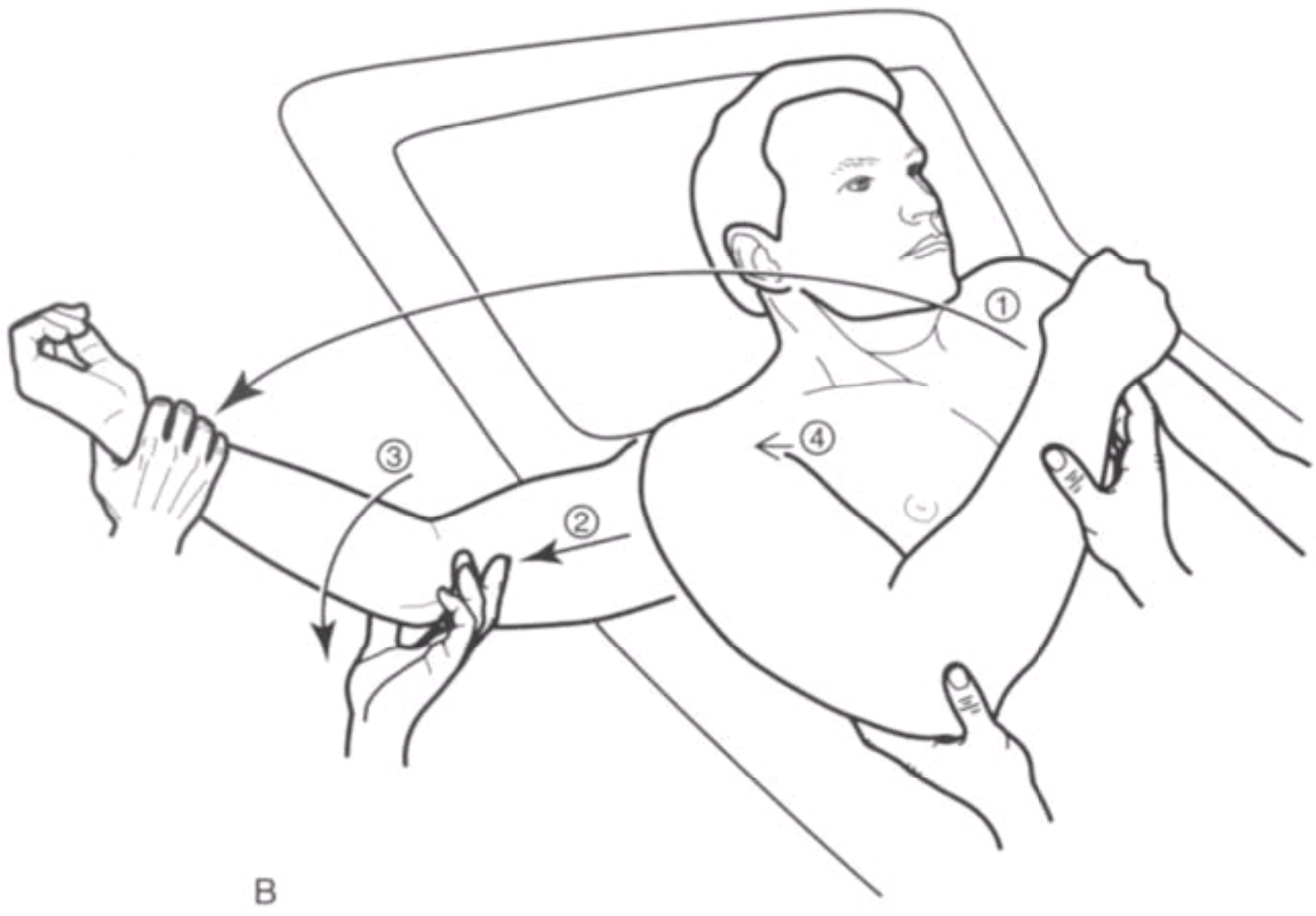
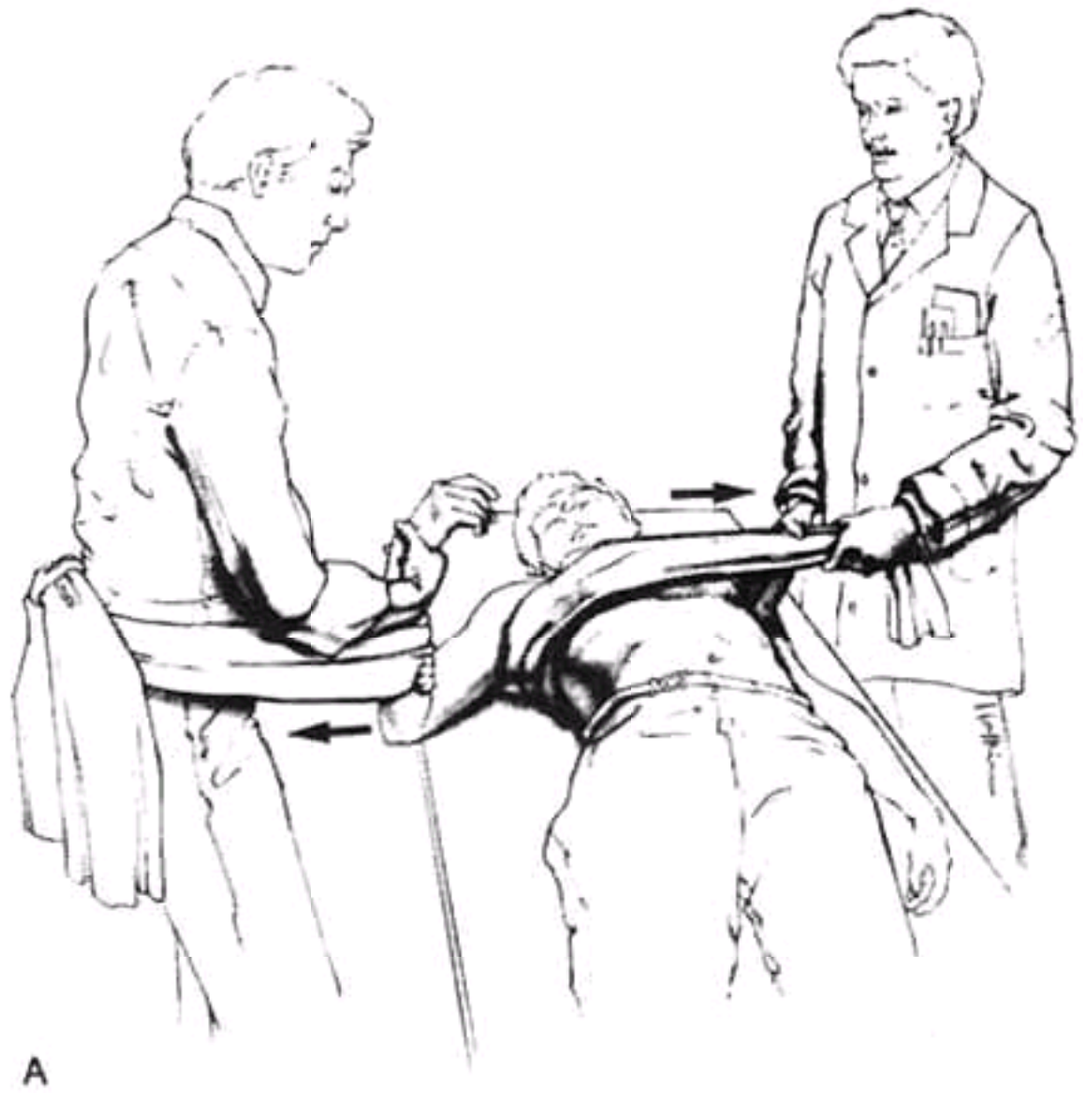


Figure 50-15 Traction-countertraction method. This simple technique for reducing the dislocated shoulder applies gradual and steady traction along the axis of the dislocated limb. A bedsheet, wrapped around the supine patient's upper chest wall and over the unaffected shoulder, is either tied or held by an assistant and acts as a fixed counterforce. A second bedsheet is placed around the patient's flexed forearm, just distal to the flexed elbow, and securely tied behind the operator's back. Note that a significant skin avulsion or friction burn may occur if there is excessive motion of the sheets, especially in the elderly patient with thin, delicate skin. With the patient's forearm held in a neutral rotation and the hand in a vertical position, the operator applies traction by leaning back, rather than using the biceps to apply traction. *(From Respet PB: A practical technique for reducing shoulder dislocations. J Musculoskel Med 5:29, 1988.)*



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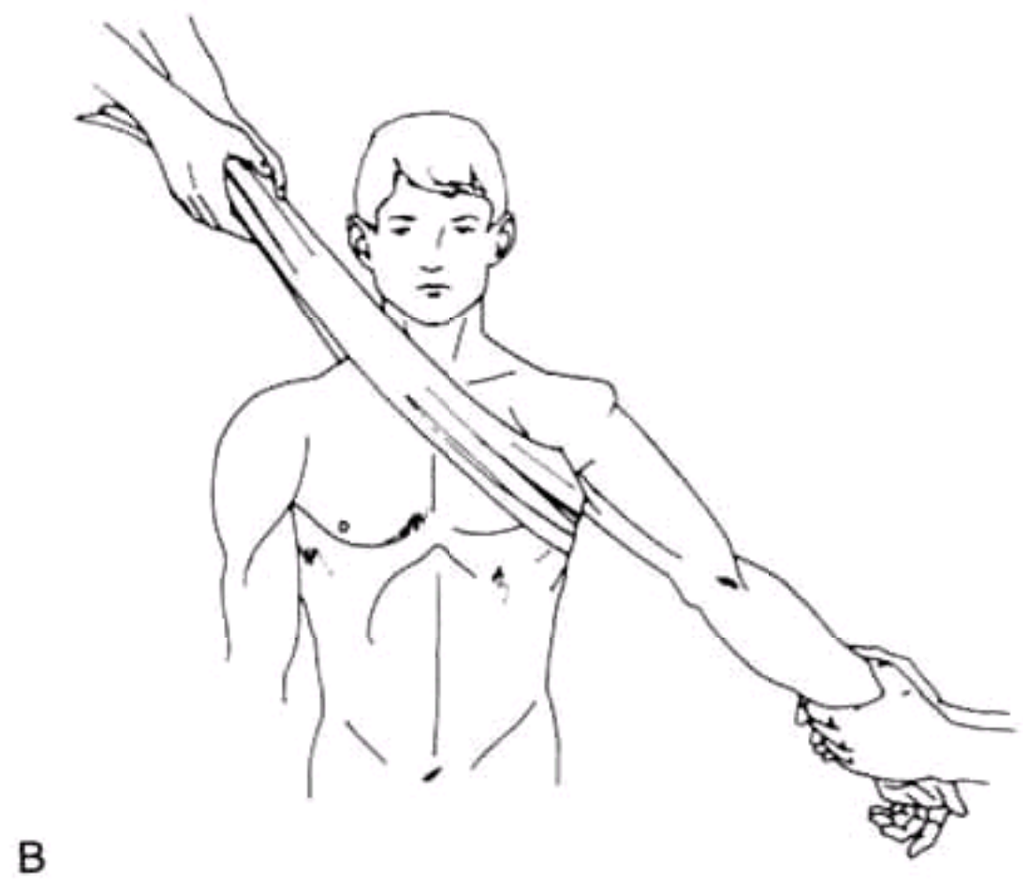

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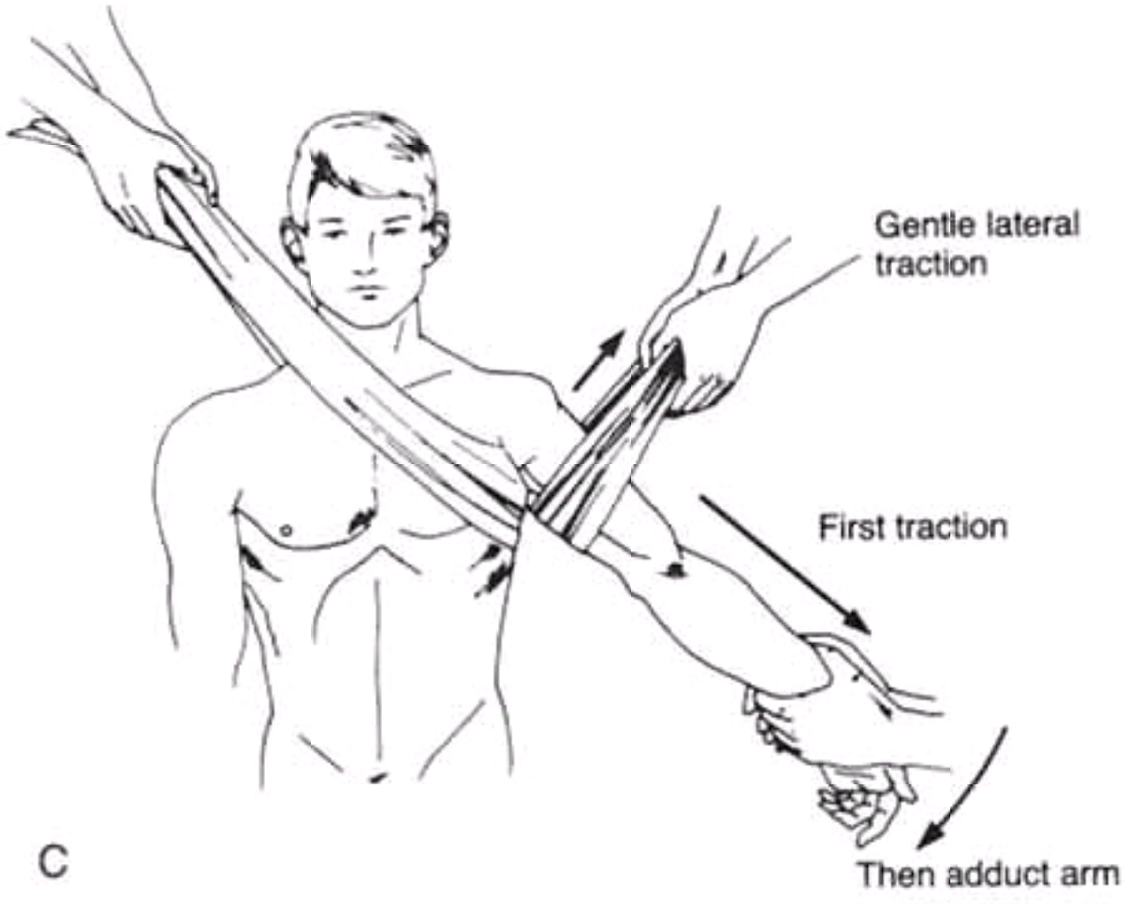


C

Figure 50-16 Spaso technique. While maintaining gentle vertical traction, the affected arm is externally rotated by grasping the wrist or forearm. Reduction may be subtle.

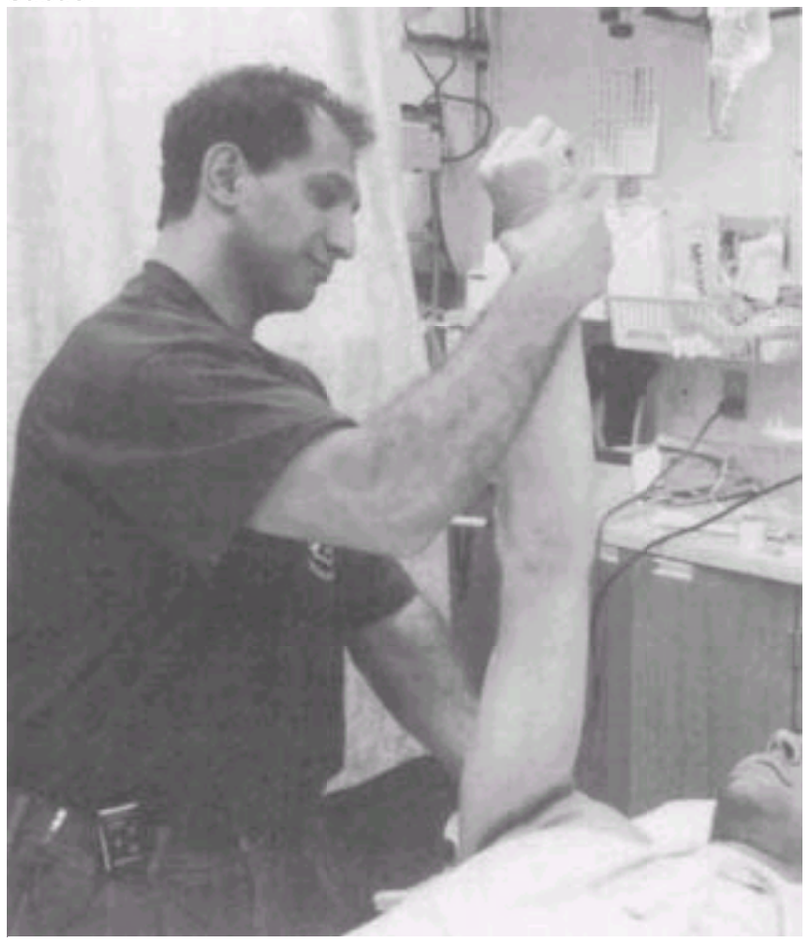


Figure 50-17 If a patient with a shoulder injury can place the palm of the injured arm on top of the contralateral shoulder, it is unlikely that a shoulder dislocation is present. Alternatively, completion of this maneuver after a reduction attempt provides strong evidence that the reduction was successful.

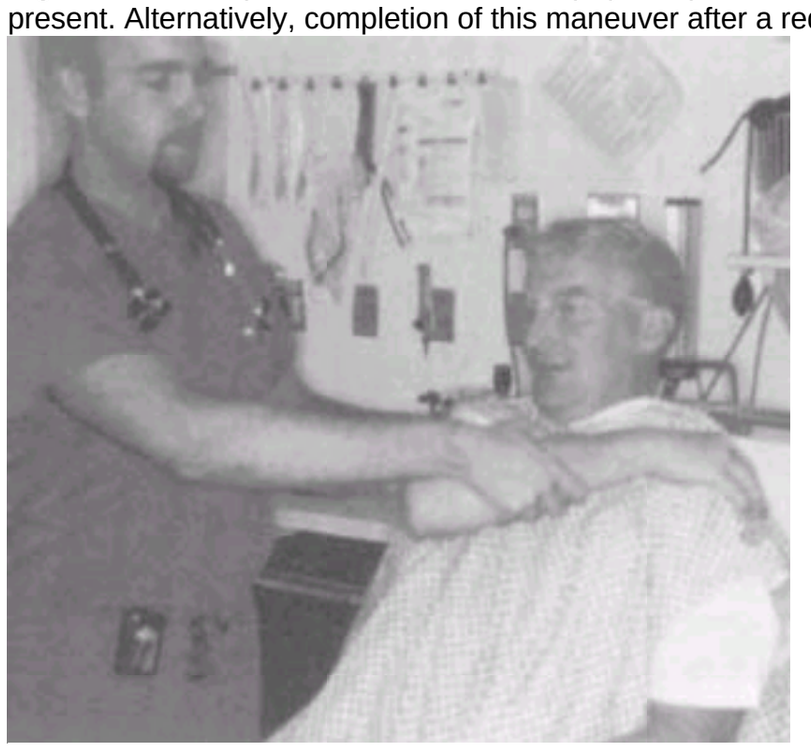


Figure 50-18 *A,* A clue to a posterior shoulder dislocation is the arm locked in adduction and internal rotation, with patient's inability to rotate the shoulder externally with the elbow flexed at 90°. *B,* Note that extension of the elbow with supination of the forearm may obscure loss of the external rotation.

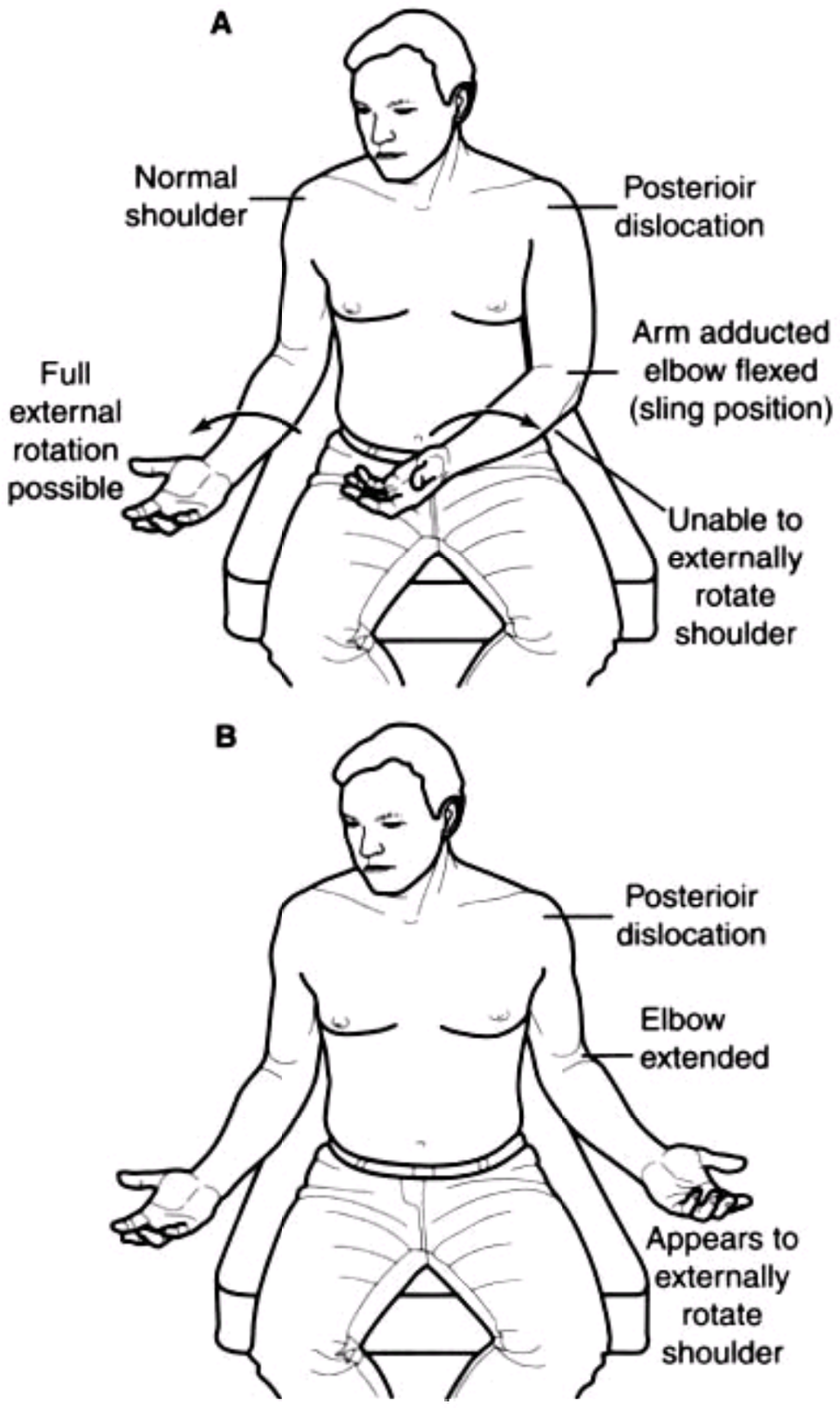


Figure 50-19 *A,* This patient has a posterior dislocation of the humerus. Because the dislocation is directly posterior, there is no superior or inferior displacement of the humeral head. On superficial observation, the head of the humerus appears to maintain a normal relationship with the glenoid fossa and the acromion process. However, definite abnormalities exist in this film. The space between the humeral head and the glenoid fossa is abnormally wide, and because of the extreme internal rotation of the humerus, the head and neck are seen end on. In this projection, the humeral head resembles a light bulb. Compare this film with the same patient's normal opposite shoulder (note that the film is reversed for illustrative purposes) *(B)*. *C,* The normal axillary view of the shoulder. The *asterisk* indicates the glenoid process. The *open arrow* indicates the coracoid process of the scapula and the *closed arrow* indicates the acromion process of the scapula. *D,* Axillary view of a posterior shoulder dislocation with an impression fracture of the humeral head. *(From Harris JH, Harris WH (eds): The Radiology of Emergency Medicine. Baltimore, Williams & Wilkins, 1971.)*

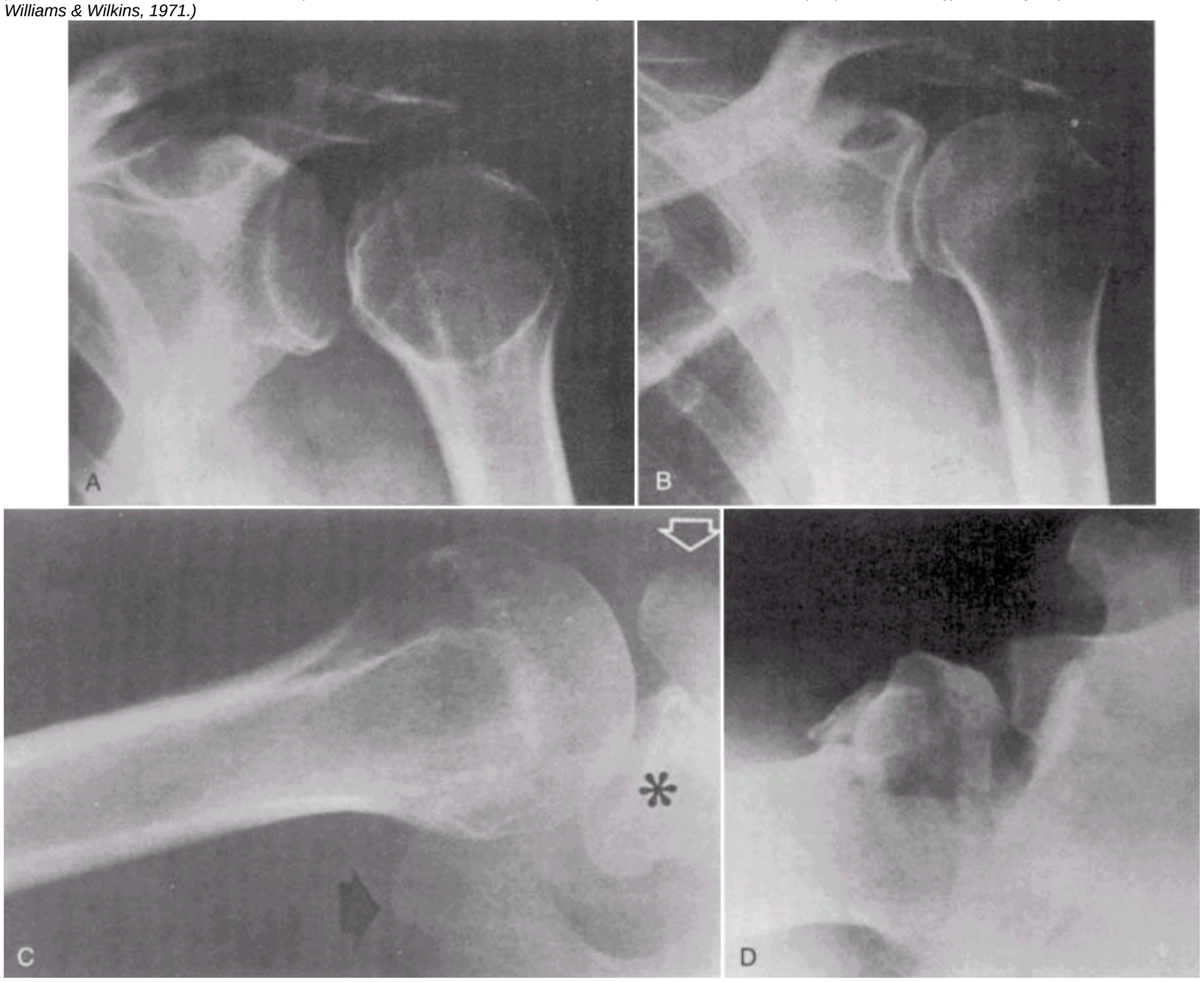


Figure 50-20 *A,* Note the normal elliptical pattern of overlap produced by the head of the humerus and the glenoid fossa. *B,* In the patient with a posterior dislocation, this pattern is lost, and internal rotation of the greater tuberosity is also noted. *(From Simon R, Koenigsknecht S: Orthopedics in Emergency Medicine. New York, Appleton-Century-Crofts, 1982, p 344. Reproduced by permission.)*

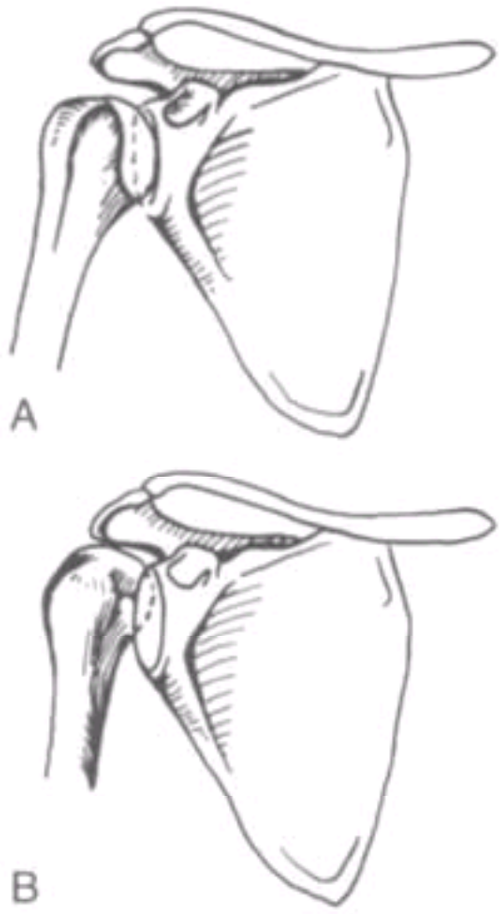


Figure 50-21 Posterior dislocation of the humeral head is a subtle, easily missed finding on the anteroposterior view. A key finding is an abnormal overlap of the humeral head with the glenoid fossa, but additional views are usually needed to confirm the dislocation. Comparison with the normal shoulder may also help. *A,* This AP film showing a posterior dislocation was initially read as normal, but the dislocation was obvious on an axillary view (see Fig. 50-19). *B,* "Light-bulb" appearance of the humeral head in a posterior dislocation. Posterior dislocation should be suspected in patients with significant pain and dysfunction after trauma, especially following seizures and electrical shock injuries. Occasionally the dislocations are bilateral. *(From Riddervold HO: Easily Missed Fractures and Corner Signs in Radiology. Mt Kisco, NY, Futura, 1991.)*

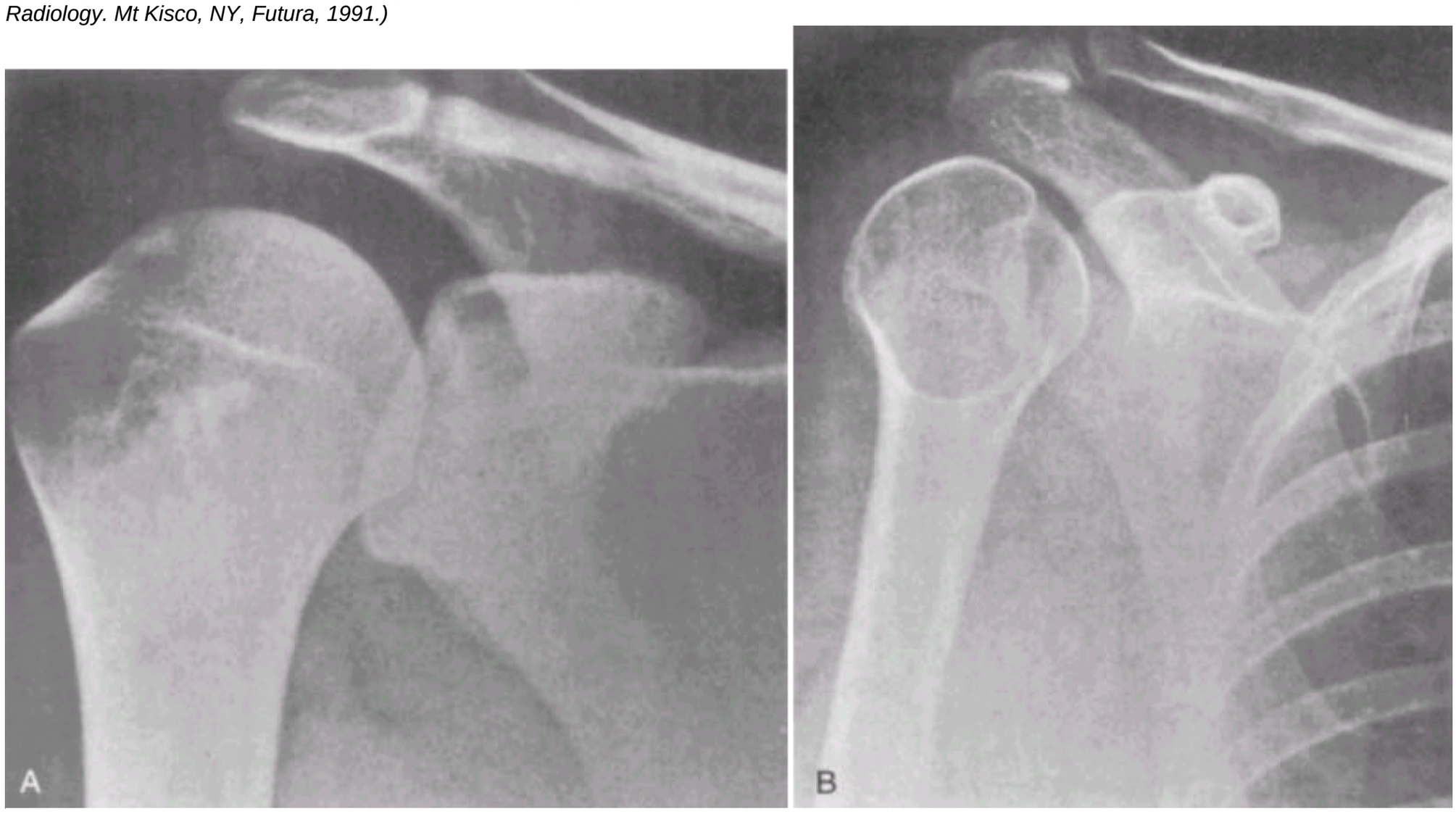


Figure 50-22 Reduction of posterior shoulder dislocation—with countertraction being applied, traction on the internally rotated and adducted arm is combined with posterior pressure on the humeral head to effect reduction.

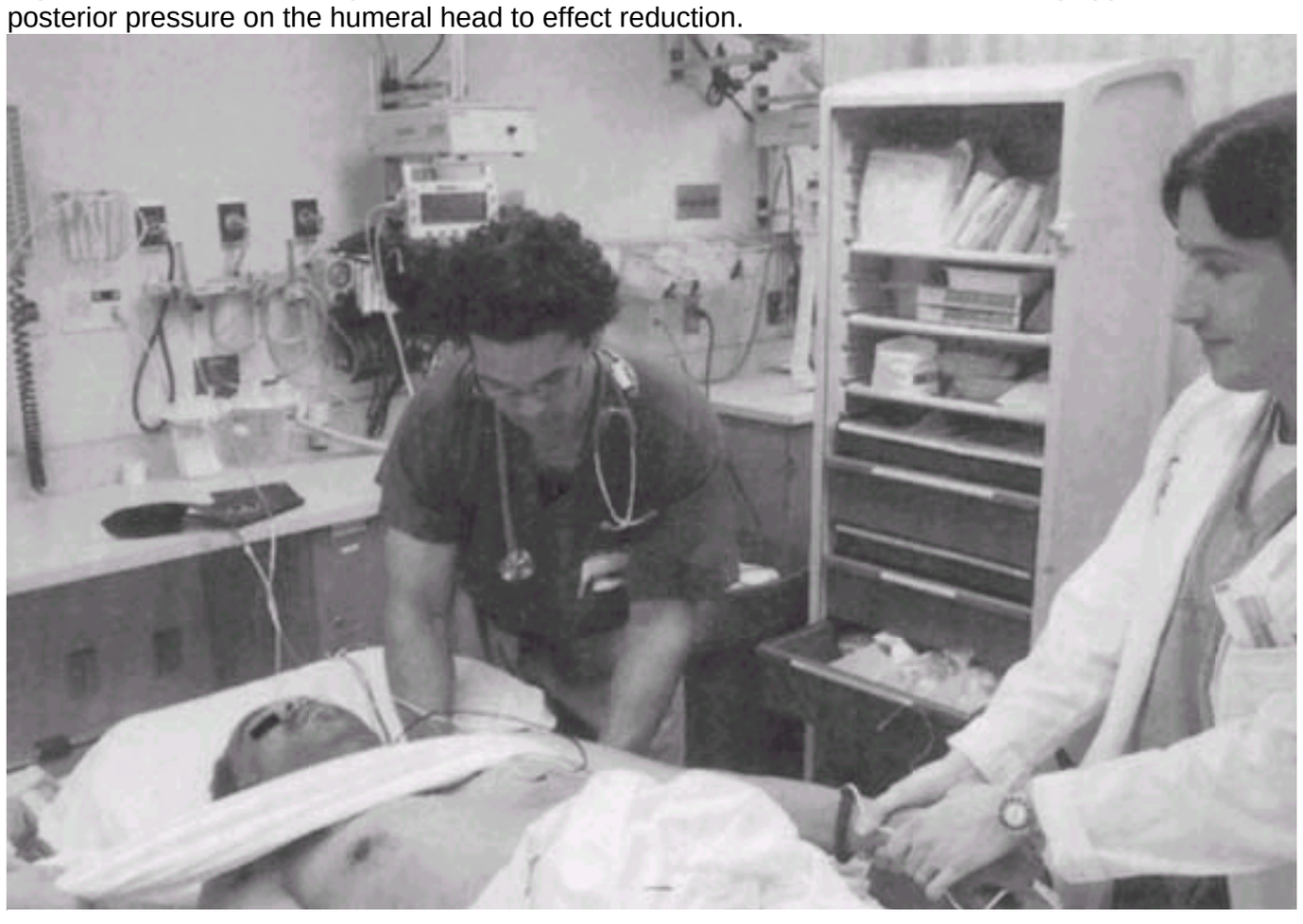


Figure 50-23 Handshake cast—after successful reduction of an acute posterior shoulder dislocation, this cast is applied in neutral rotation, slight extension, and 15 to 20 degrees of abduction. *(From Rockwood CA, Wirth MA: Subluxations and Dislocations about the Glenohumeral Joint. In Rockwood CA, Green DP, Bucholz RW, et al (eds): Rockwood and Green's Fractures in Adults, vol 2, 4th ed. Philadelphia, Lippincott-Raven, 1996, p 1291. Reproduced by permission.)*

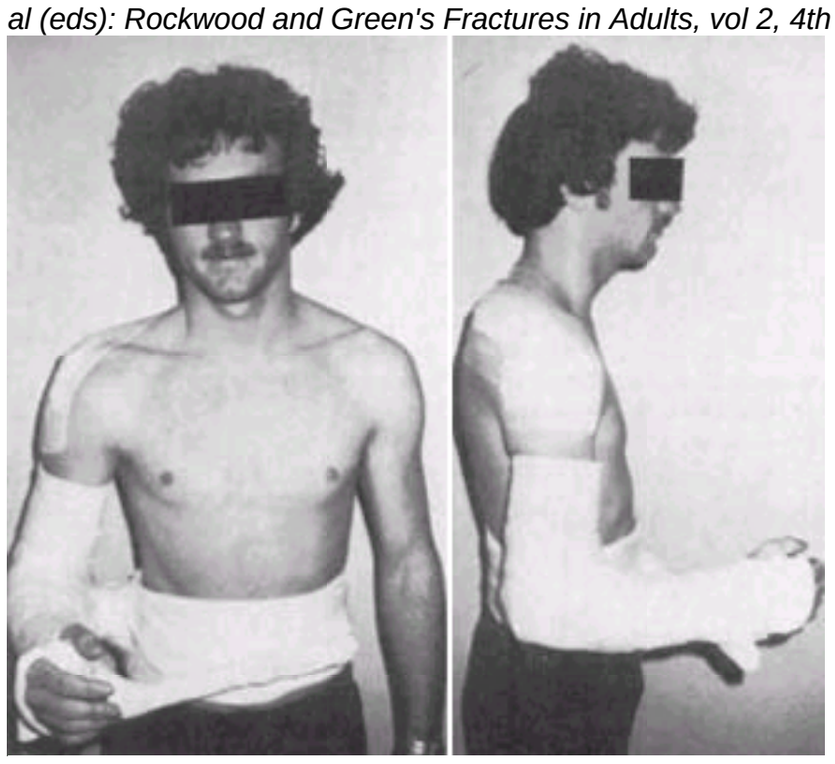


Figure 50-24 Grades I to III of acromioclavicular separation. (See text for description.) *(From Heppenstall RB: Fractures and dislocations of the distal clavicle. Orthop Clin North Am 6:480, 1975.)*

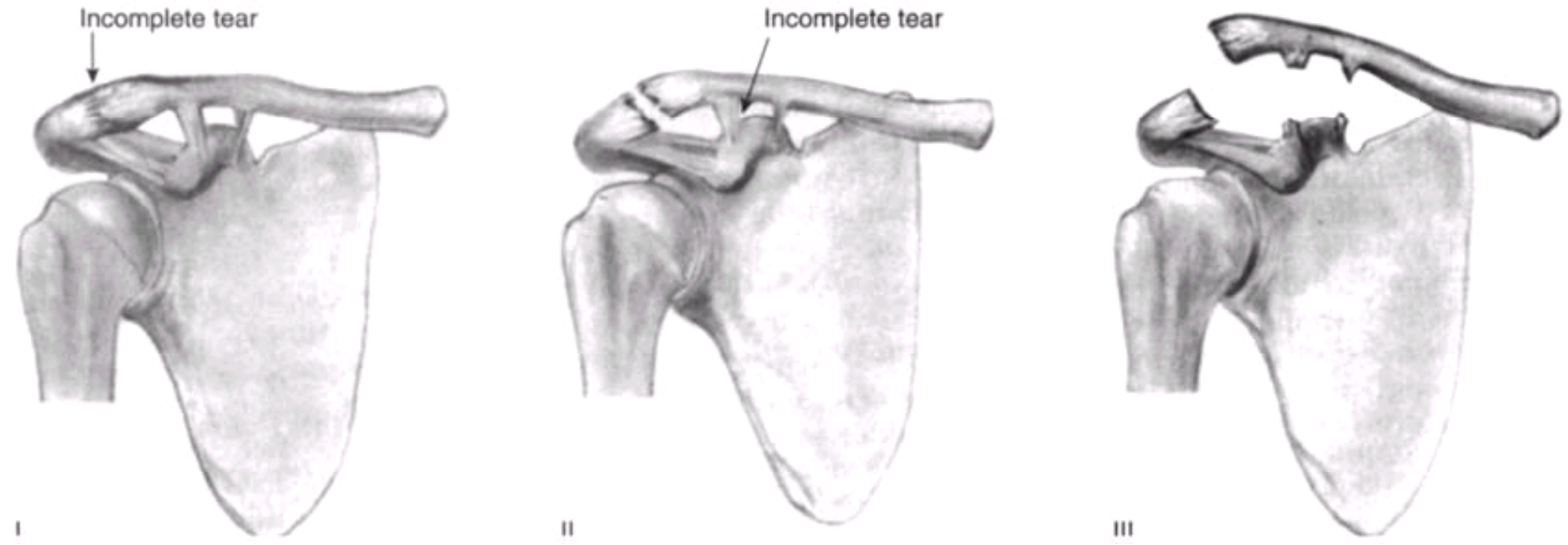


Figure 50-25 Classification of elbow dislocations. *(From Simon R, Koenigsknecht S: Orthopedics in Emergency Medicine. New York, Appleton-Century-Crofts, 1982, p 333. Reproduced by permission.)*

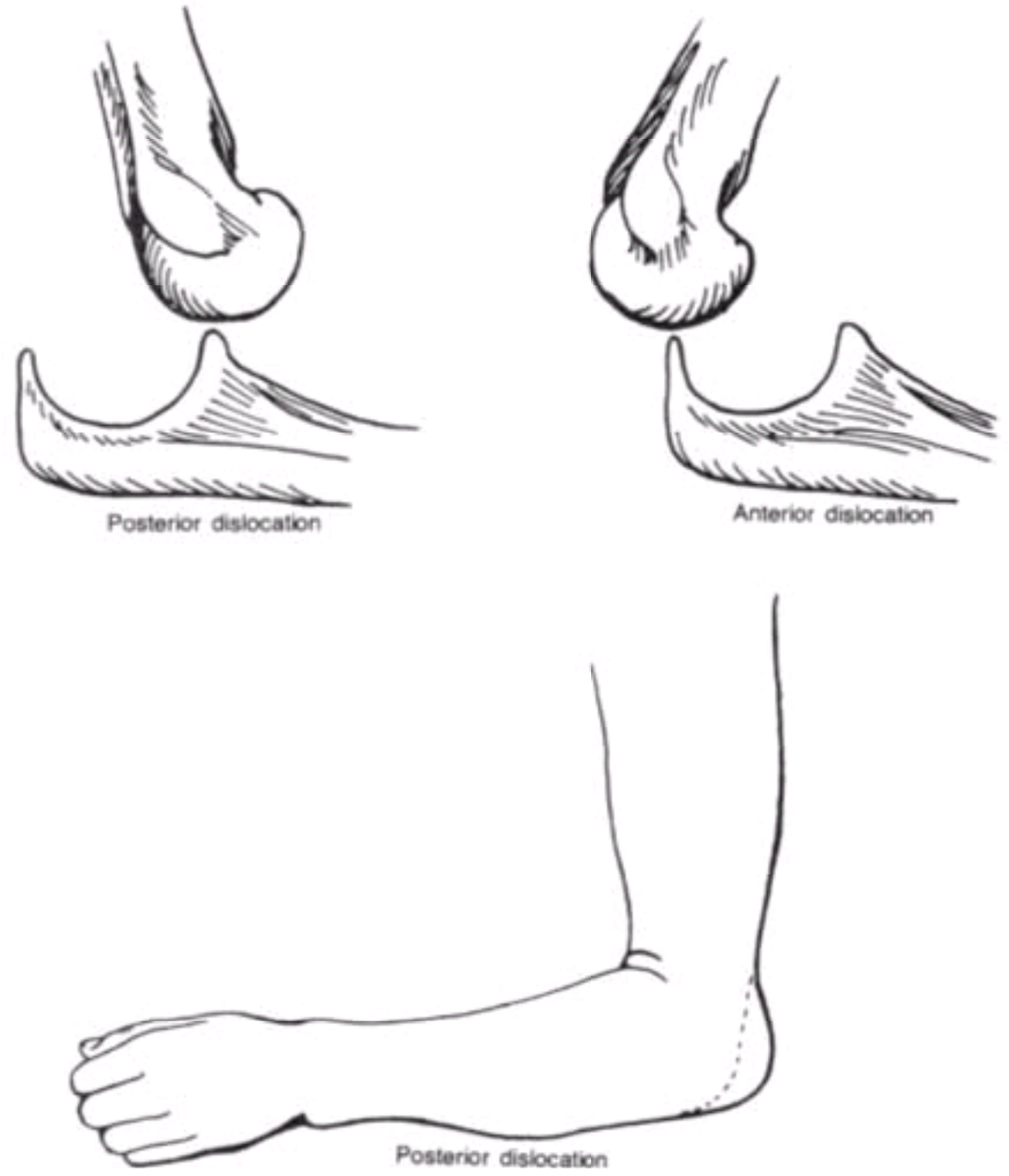


Figure 50-26 Manipulative reduction of posterior elbow dislocation. While an assistant holds the arm and makes steady countertraction *(1),* grasp the wrist with one hand and apply steady traction on the forearm in the position in which it lies *(2)*. While traction is maintained, correct any lateral displacement with the other hand *(3)*. While traction is maintained *(4),* gently flex the forearm *(5)*. Note that with reduction, a clunk is usually felt and heard as the olecranon engages the articular surface of the humerus. *(From DePalma AF: Management of Fractures and Dislocations: An Atlas. Philadelphia, WB Saunders, 1970, pp 793 and 794. Reproduced by permission.)*

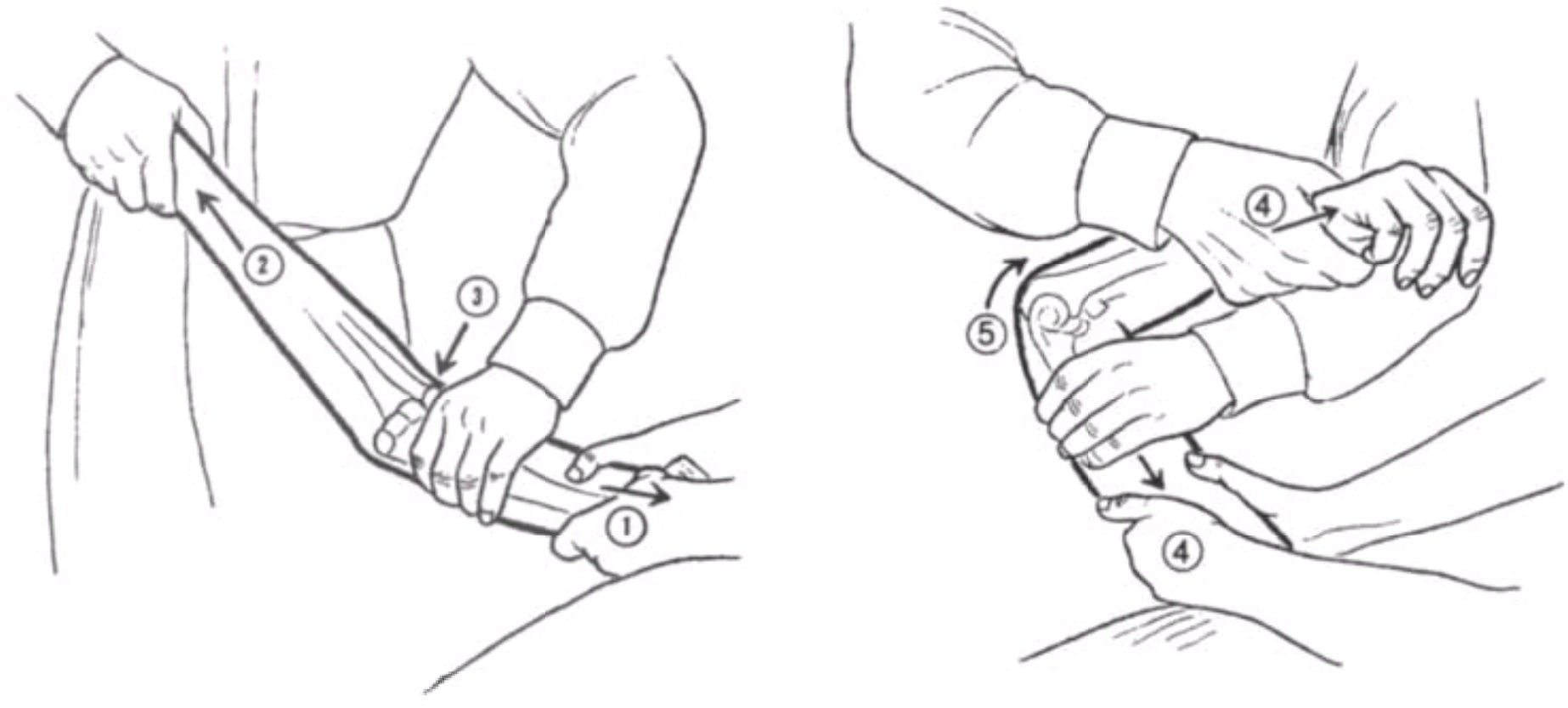
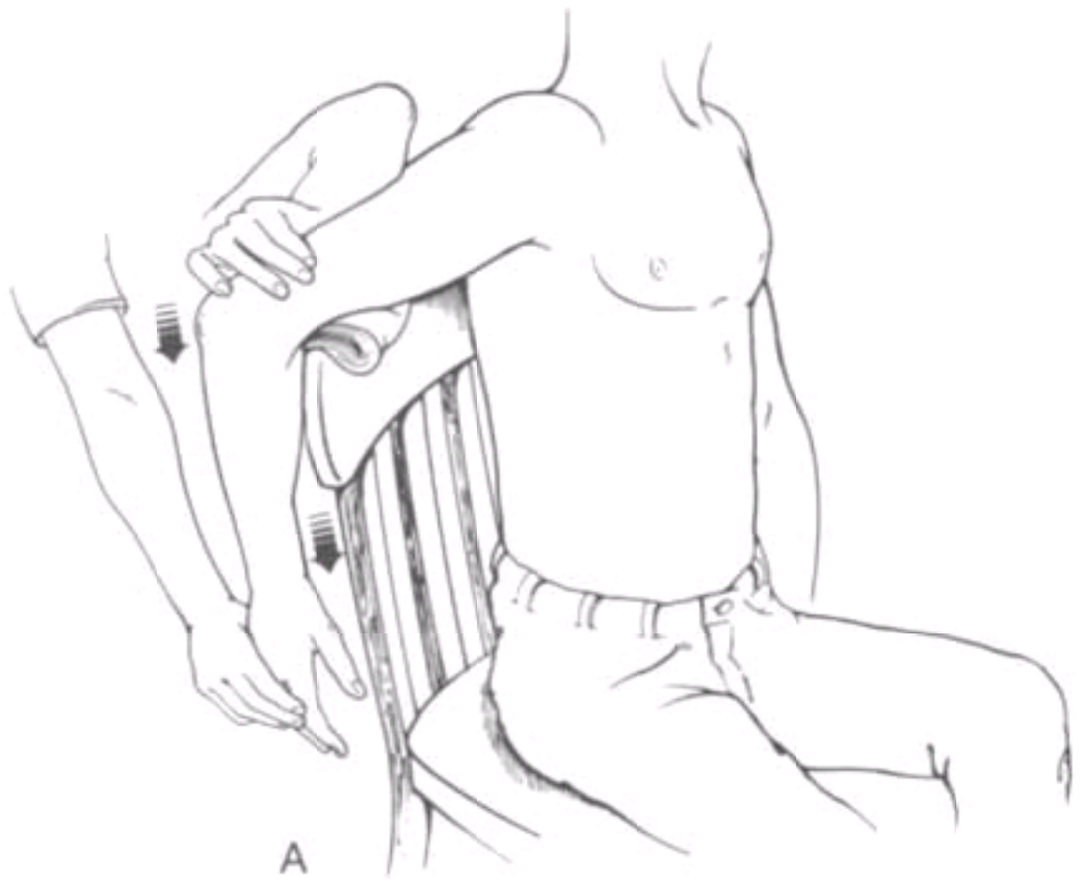


Figure 50-27 *A,* Hanging method of elbow dislocation reduction. Downward traction is applied to the hand while the olecranon is guided into place. *(From Lavine LS: A simple method of reducing dislocations of the elbow joint. J Bone Joint Surg Am 35:785, 1953.) B,* Reduction of posterior elbow dislocation. An assistant encircles the distal humerus and applies pressure to the olecranon. Traction is applied with the elbow in slight flexion to relax the triceps. The reduction can be performed with the patient reclining *supine* as shown. Having the patient recline *prone* with the upper arm supported by the stretcher can often enhance muscle relaxation.



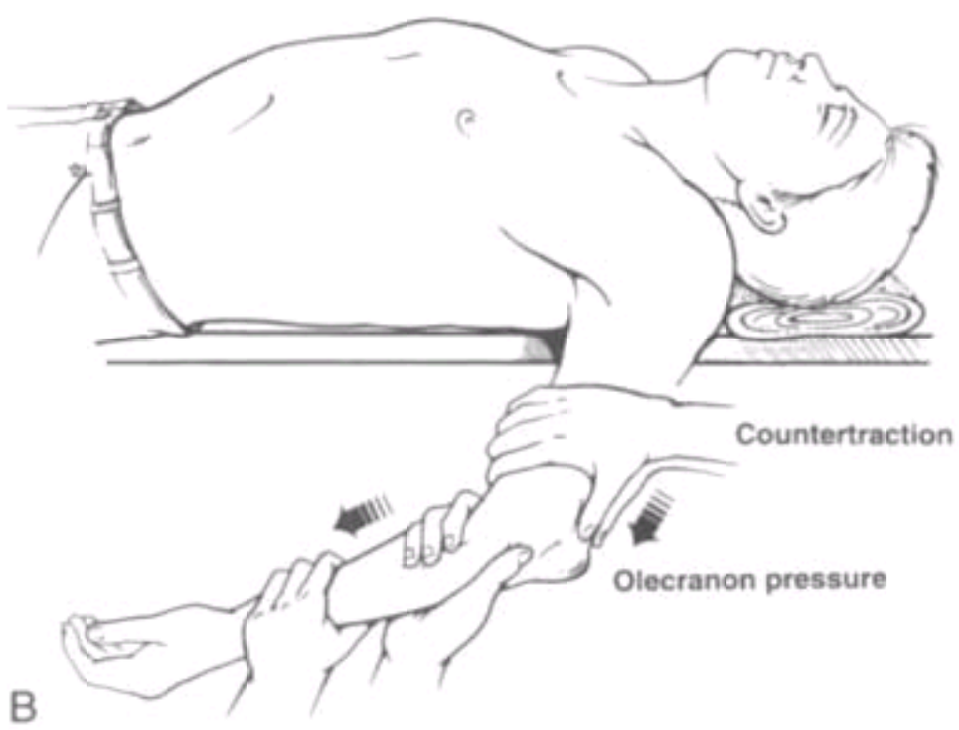


Figure 50-28 Manipulative reduction of anterior elbow dislocation. Reduction is performed with the patient under local or general anesthesia. *1*, An assistant grasps the arm and provides countertraction. *2*, The operator grasps the wrist with one hand and applies traction in the line of the arm, and with the other hand applies firm, steady pressure downward and backward on the upper end of the forearm *(3)*. A clunk usually indicates that reduction is achieved. *4*, The arm is flexed to 45° beyond a right angle. *(From DePalma AF: Management of Fractures and Dislocations: An Atlas. Philadelphia, WB Saunders, 1970, p 796. Reproduced by permission.)*

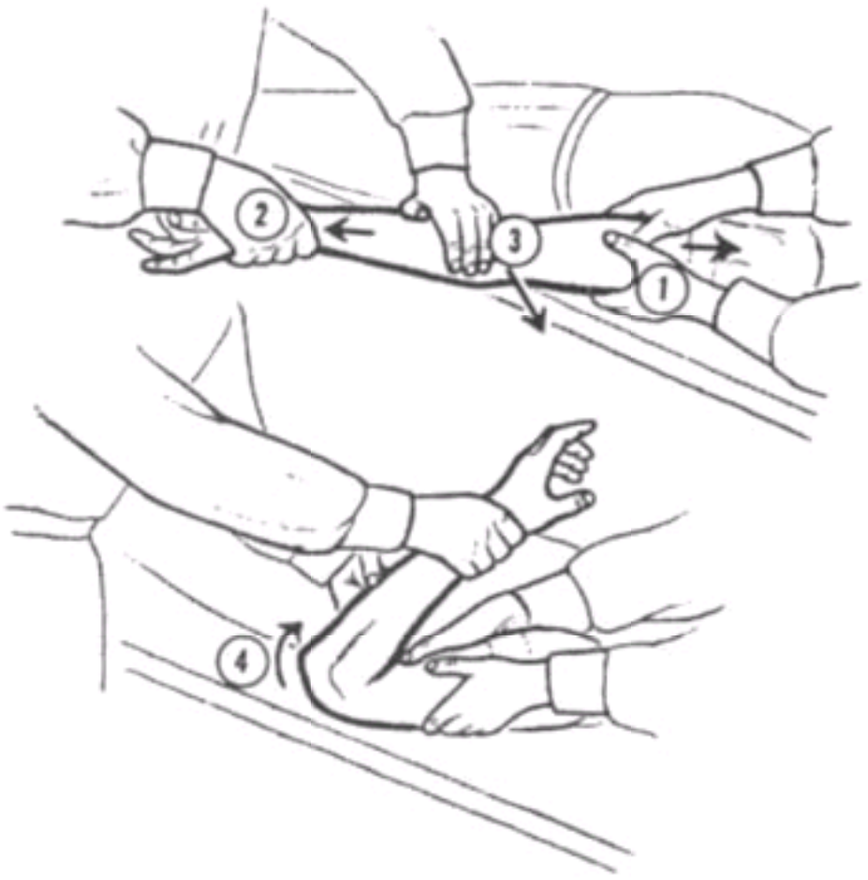


Figure 50-29 Typical presentation of a child with a subluxation of the radial head (nursemaid's elbow). It may be difficult to determine exactly where the pathology exists, and often the wrist is thought to be the culprit. This child will not use the injured arm but has minimal discomfort as long as the elbow is not manipulated. *A*, The affected arm hangs down at the side, slightly flexed and pronated. *B*, Once reduced, full activity is generally regained in a matter of minutes.

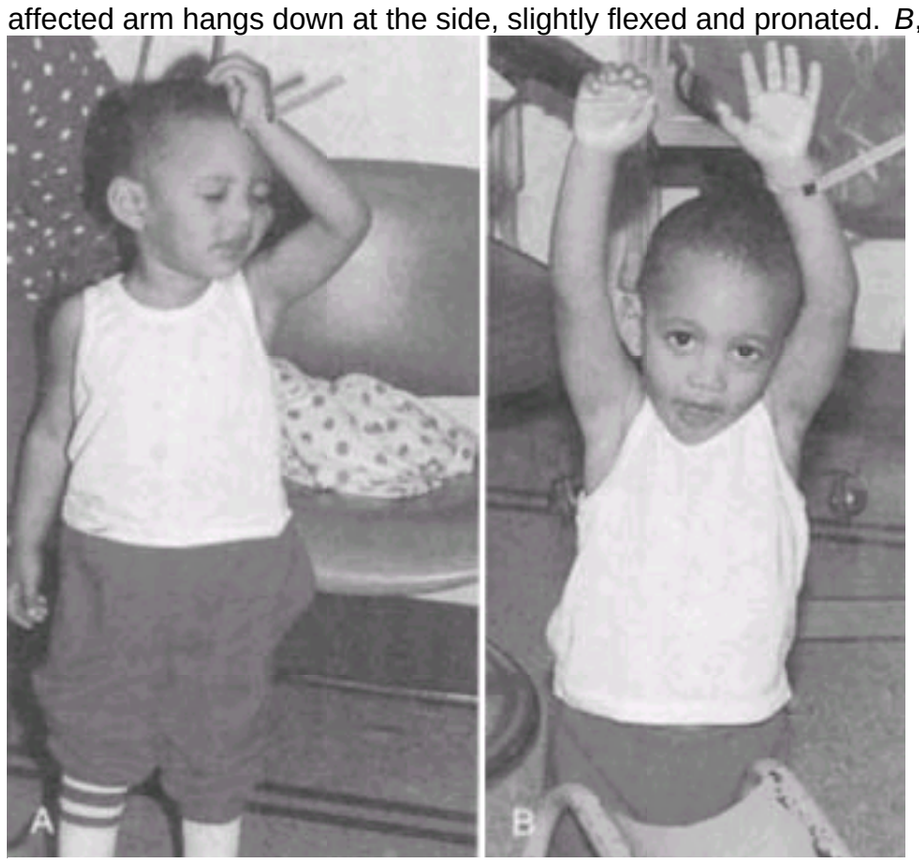


Figure 50-30 Radial head subluxation. *A*, Anatomically, this injury represents interposition of the torn annular ligament between the radial head and the capitellum. *B*, The supination method of reduction is performed by grasping the arm about the wrist and placing the other hand about the elbow with the thumb over the radial head. The forearm is then supinated *(C)* and then the arm is flexed *(D)* in one continuous motion. *(From Fleisher GR, Ludwig S: Textbook of Pediatric Emergency Medicine. Baltimore, Williams & Wilkins, 1988, p 1322. Reproduced by permission).*

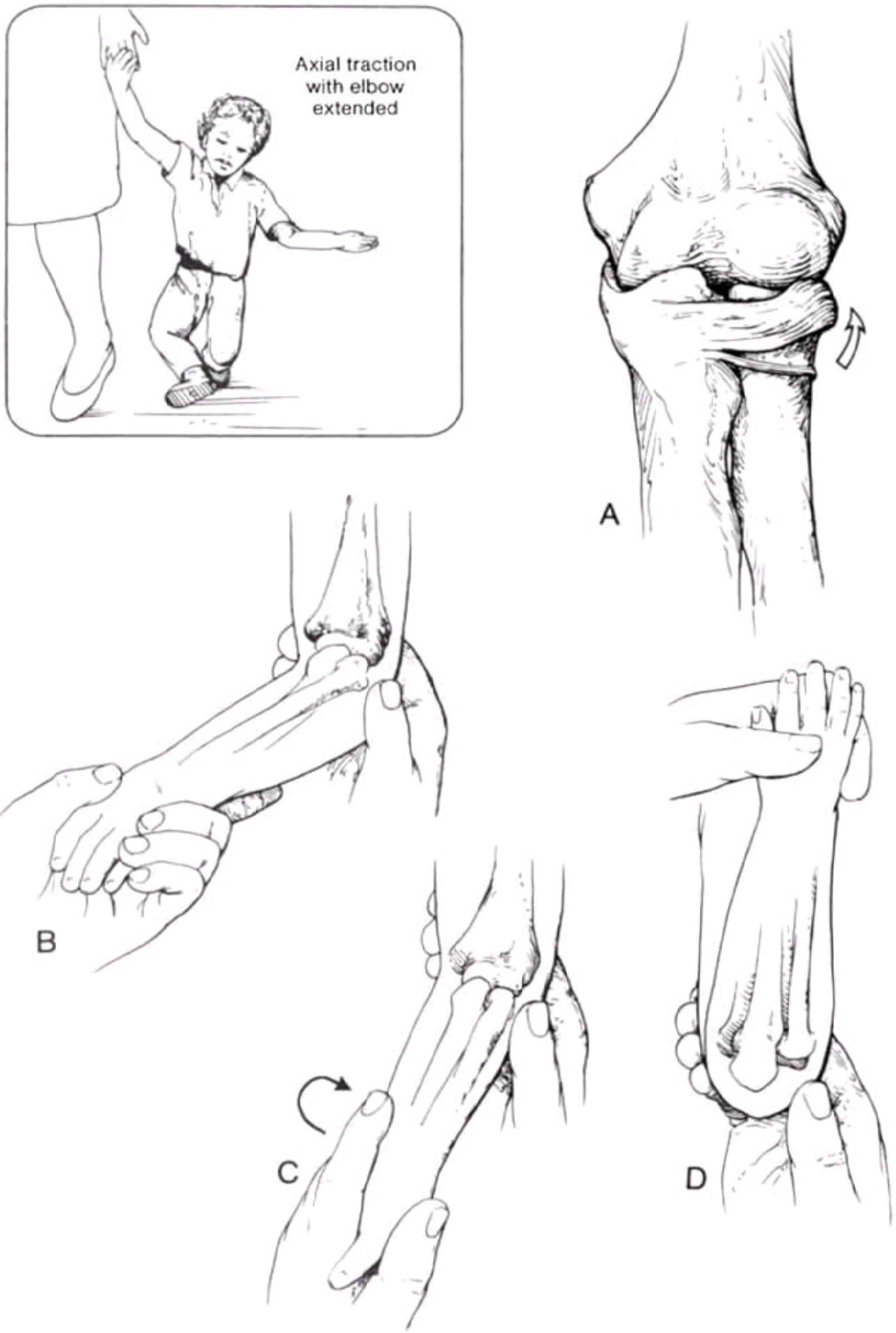


Figure 50-31 *A* and *B,* The collateral ligament-volar plate relationship. The metacarpophalangeal (MCP) and interphalangeal (IP) joints derive their strength from a combination of the two collateral ligaments and the volar plate. Dislocations of these joints require tearing of at least two parts of this three-part structure. *(From Carter P [ed]: Common Hand Injuries and Infections. Philadelphia, WB Saunders, 1983, p 114. Reproduced by permission.) C,* Lateral view demonstrating collateral ligament-volar plate relationship. *(Redrawn from Eaton RG: Joint Injuries of the Hand. Springfield, IL, Charles C Thomas, 1972.)*

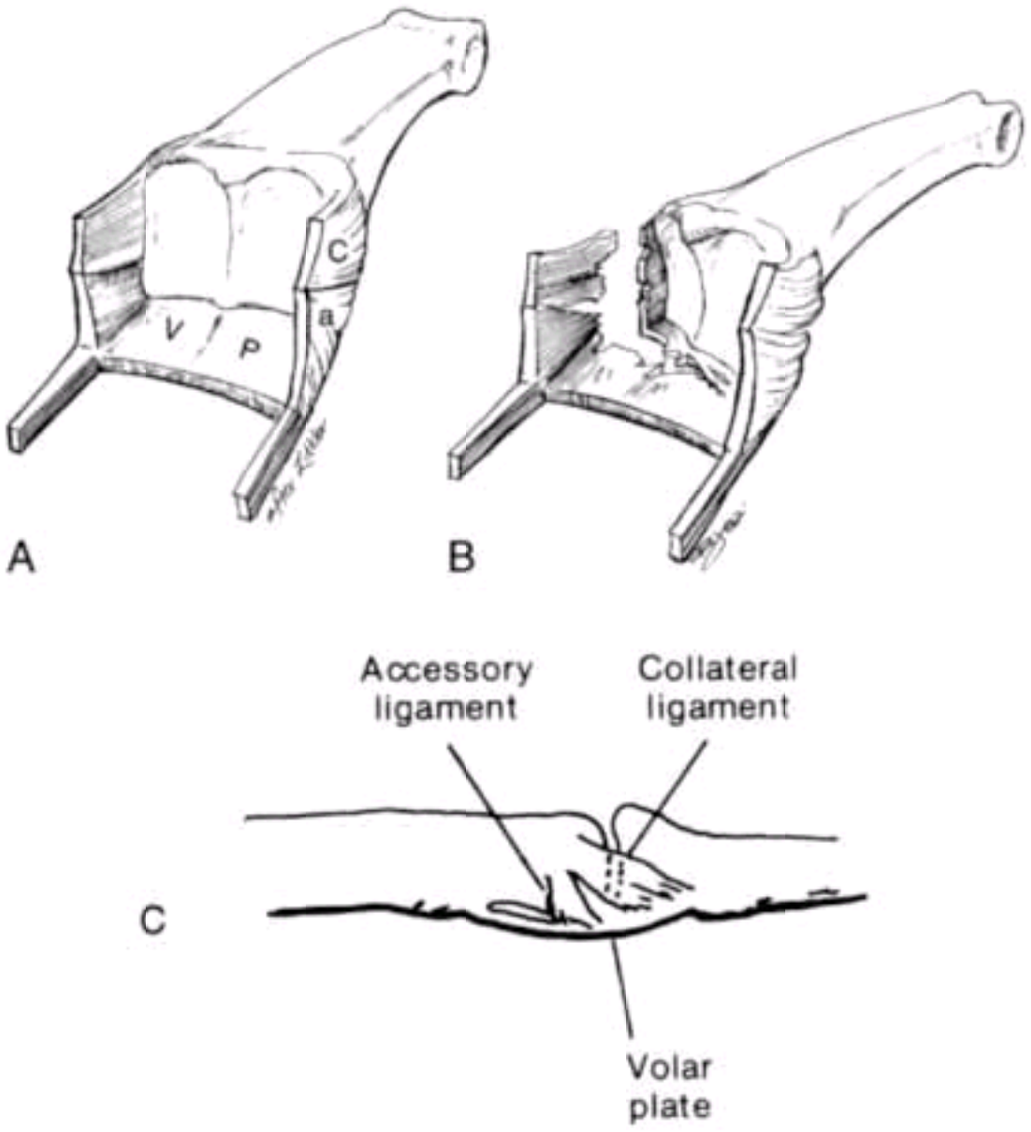


Figure 50-32 *A–D,* Traction method of joint reduction. Complete anesthesia using a regional block should precede reduction attempts. *E–H,* Exaggeration of existing deformity method. First, exaggerate the deformity that is present. Then, in addition to steady traction, push the joint back into position. *(From Carter P [ed]: Common Hand Injuries and Infections. Philadelphia, WB Saunders, 1983, pp 109 and 110. Reproduced by permission.)*

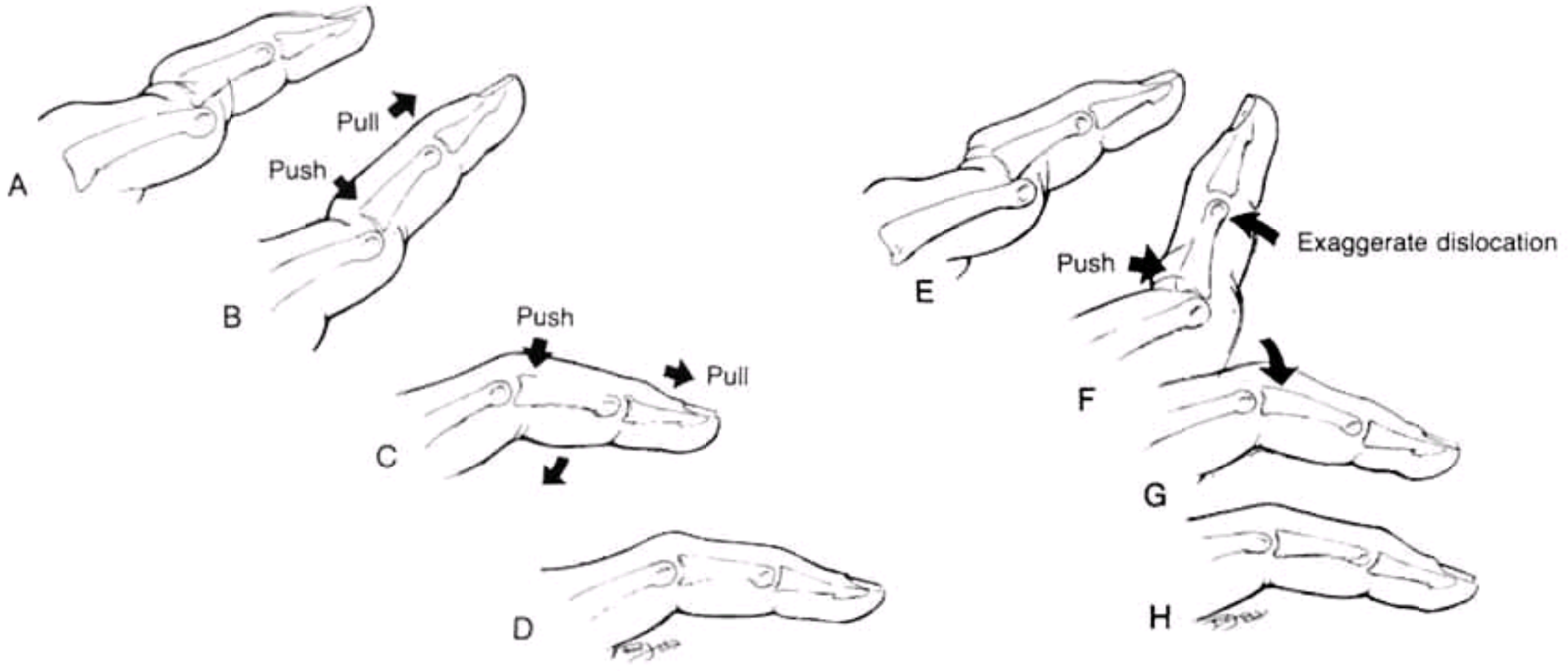


Figure 50-33 Complete dorsal dislocation at the metacarpophalangeal (MCP) joint of the thumb. There is neither associated fracture nor epiphyseal separation. *(From Harris JH Jr, Harris WH: Radiology of Emergency Medicine. 2nd ed. Baltimore, Williams & Wilkins, 1981, p 239. Reproduced by permission.)*

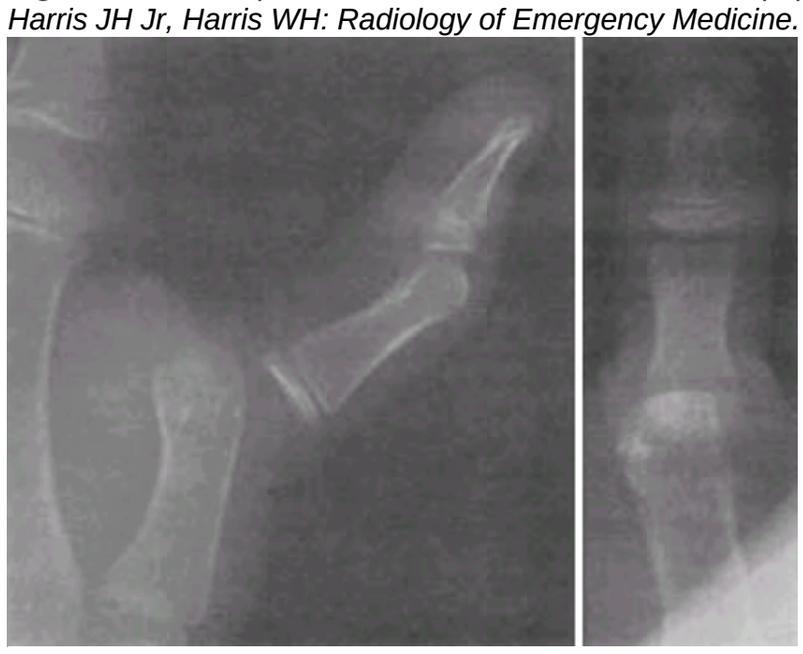


Figure 50-34 *A,* In a simple dorsal metacarpophalangeal (MCP) joint dislocation (note right angle between phalanx and metacarpal), the volar plate remains in front of the metacarpal head, although it is detached from its weaker metacarpal insertion. *B,* In a complex dislocation (note more parallel alignment between phalanx and metacarpal), the volar plate becomes entrapped in the joint and results in an irreducible reduction by closed methods. *(From DePalma AF: Management of Fractures and Dislocations: An Atlas. Philadelphia, WB Saunders, 1970, p 1177. Reproduced by permission.)*

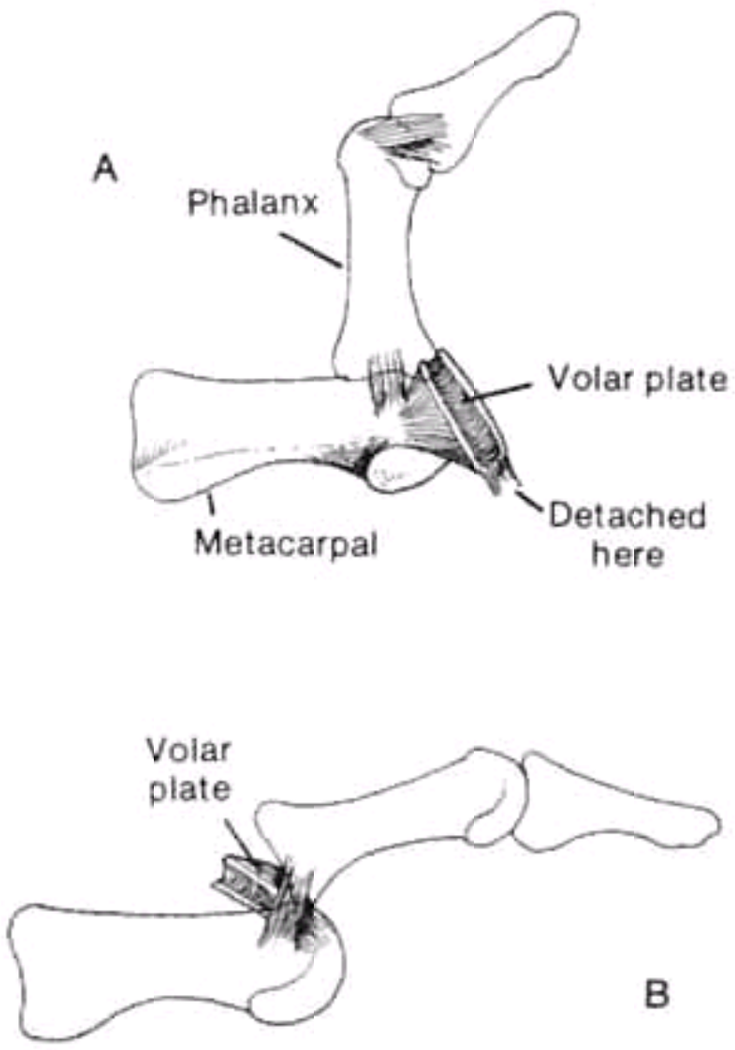


Figure 50-35 Irreducible metacarpophalangeal (MCP) joint dislocation of the thumb. Note the sesamoid bone *(arrow)*, indicating volar plate interposition between the two bone ends, which may prevent closed reduction. *(From Carter P [ed]: Common Hand Injuries and Infections. Philadelphia, WB Saunders, 1983, p 115. Reproduced by permission.)*

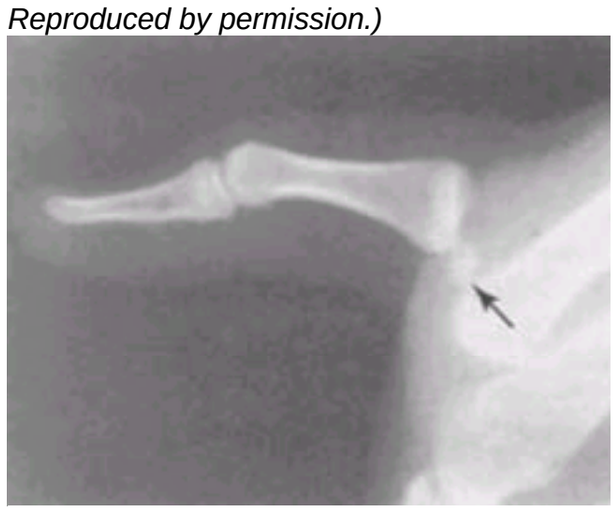


Figure 50-36 If a simple thumb metacarpophalangeal (MCP) dislocation is treated with traction alone, the forces will often interpose the volar plate and result in an irreducible complex dislocation. The proper technique for reduction includes *(1)* a good hold on the patient's distal thumb; *(2)* initial hyperextension of the dislocated phalanx; *(3)* pushing the base of the dislocated phalanx, rather than using traction alone; and *(4)* flexing the thumb. *(From DePalma AF: Management of Fractures and Dislocations: An Atlas. Philadelphia, WB Saunders, 1970, p 1178. Reproduced by permission.)*

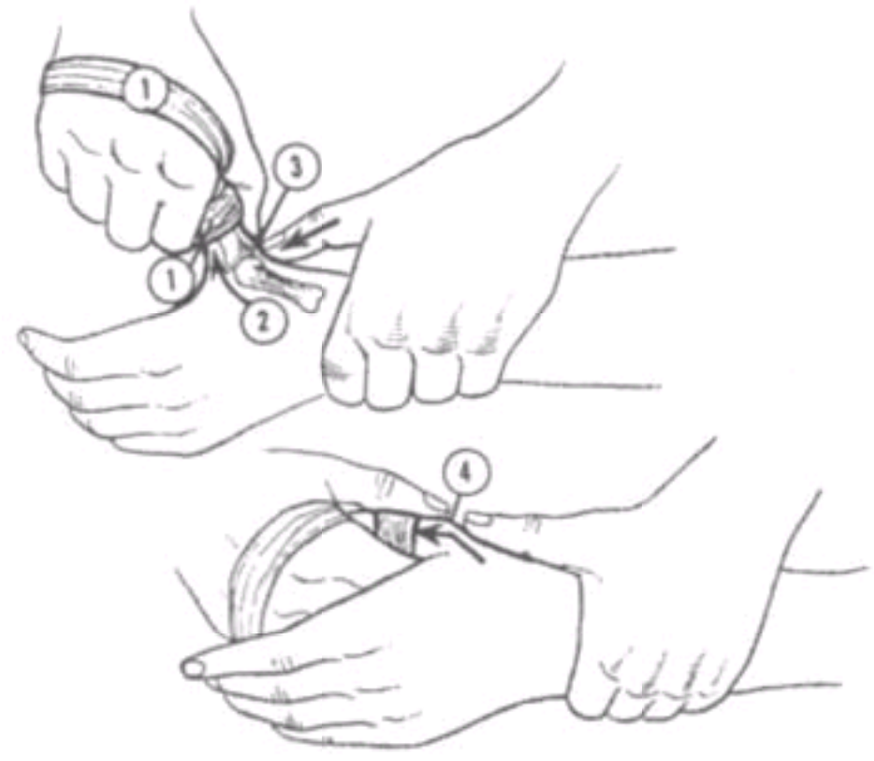


Figure 50-37 Rupture of the ulnar collateral ligament (gamekeeper's thumb). *1,* This injury is caused by forcible abduction. If unrecognized and untreated, progressive metacarpophalangeal (MCP) subluxation may occur *(2)* with interference during grasp, causing significant permanent disability. Suspect this injury when there is a complaint of pain in this region. Look for tenderness on the medial side of the MCP joint. *(From McRae R: Practical Fracture Treatment. Edinburgh, Churchill Livingstone, 1981, p 162. Reproduced by permission.)*

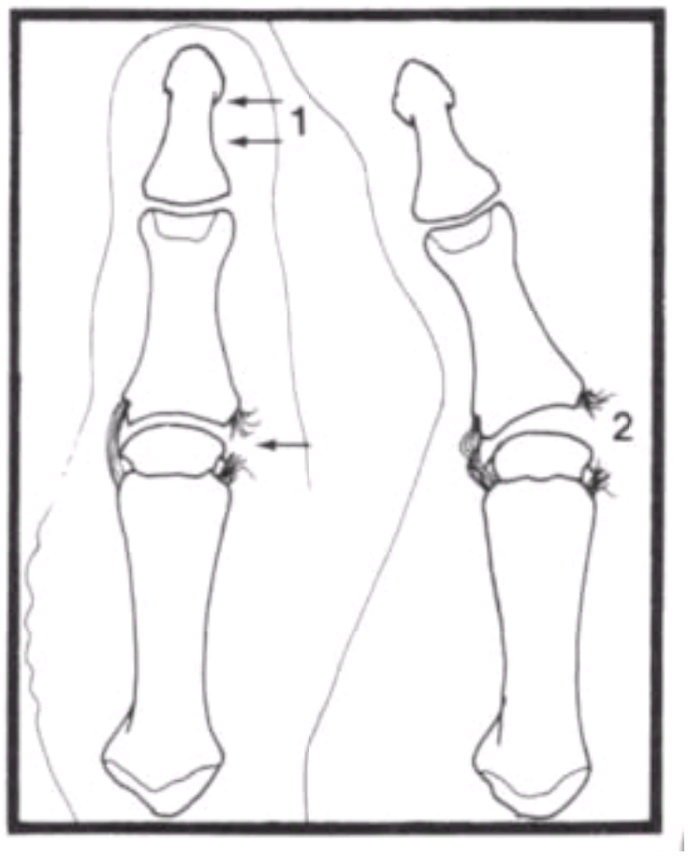


Figure 50-38 Stress testing of the ulnar collateral ligament of the thumb. This is done both clinically and with an anteroposterior radiograph. *A*, A line is drawn on the skin with a pen. The line is along the long axis of the metacarpal and the proximal phalanx. *B*, Deviation of the straight line during stress indicates instability. The metacarpal is fixed with the operator's other hand.

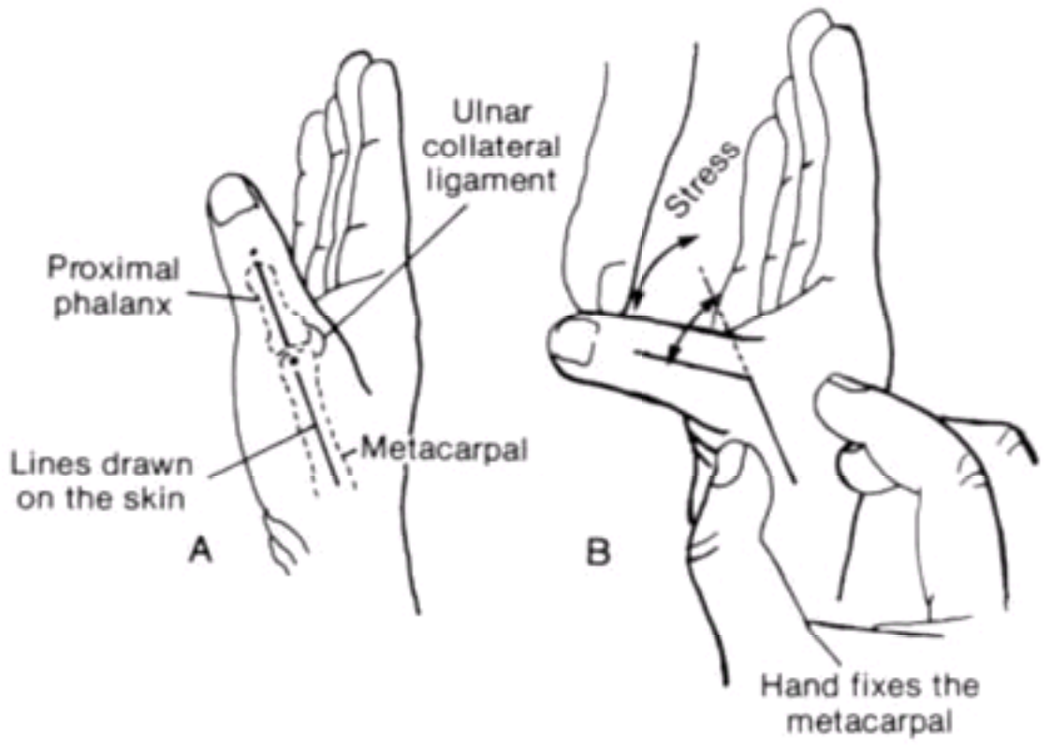


Figure 50-39 *A*, This fracture-dislocation is only fully appreciated on the true lateral film. *B*, A small fragment of bone was avulsed with the volar plate. This frequently is appreciated only with the postreduction film in a true lateral projection. *(From Carter P [ed]: Common Hand Injuries and Infections. Philadelphia, WB Saunders, 1983, p 113. Reproduced by permission.)*

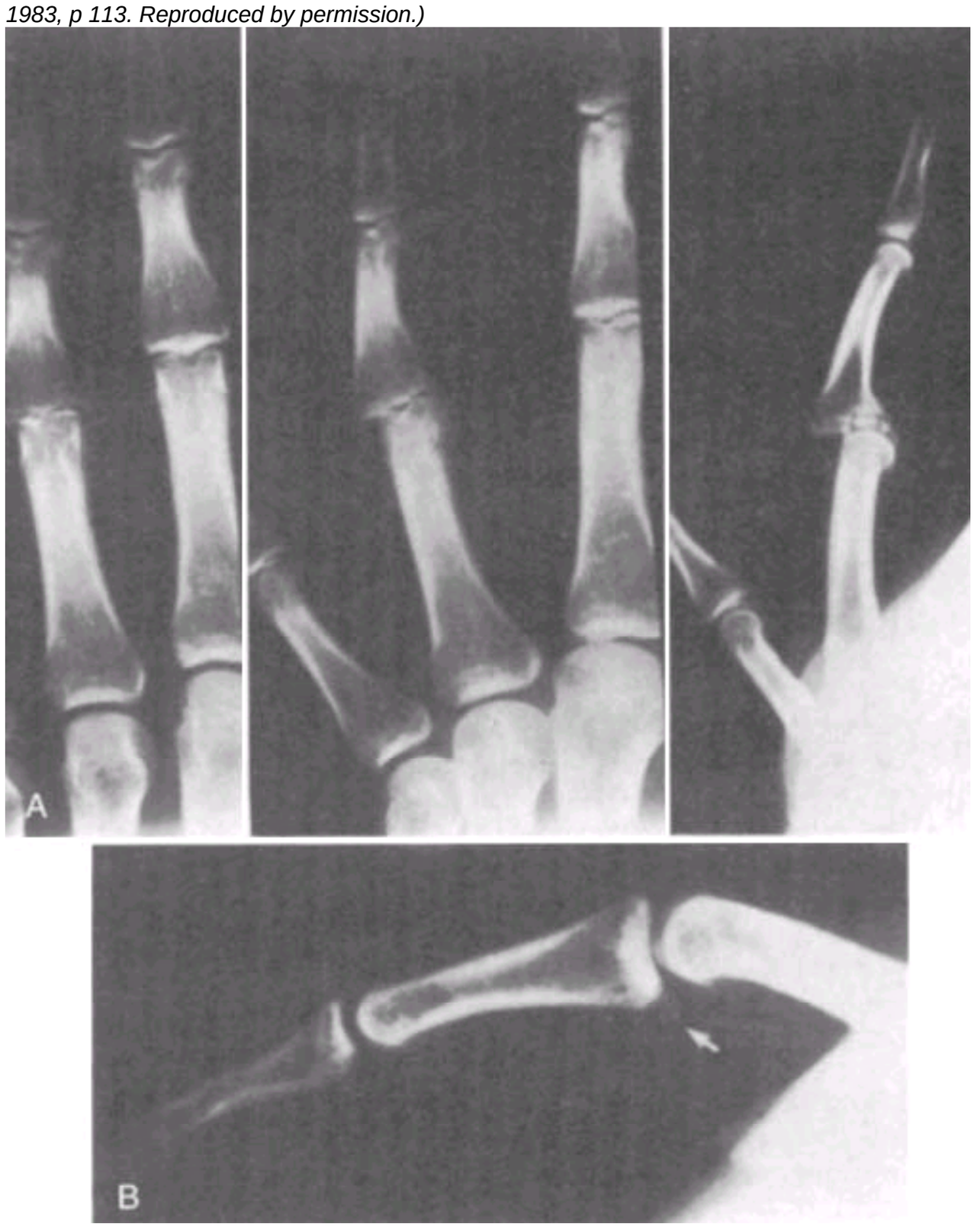


Figure 50-40 Postreduction stress of proximal interphalangeal dislocation. *A,* If the volar plate has been completely disrupted, the proximal interphalangeal joint will hyperextend with both passive and active motion. *B,* An inability to actively extend the proximal interphalangeal joint indicates a rupture of the central slip of the extensor tendon. *C,* Passive lateral stress is performed to check integrity of collateral ligaments. *(From DePalma AF: Management of Fractures and Dislocations: An Atlas. Philadelphia, WB Saunders 1970, pp 1203 and 1204. Reproduced by permission.)*

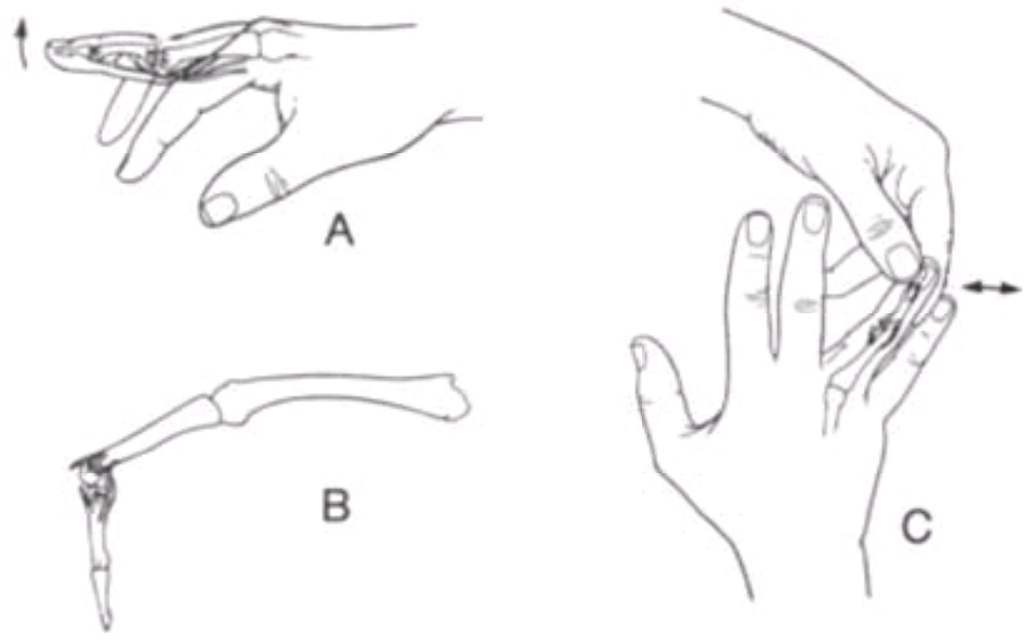


Figure 50-41 A dorsal proximal interphalangeal joint dislocation may involve rupture of the volar plate itself *(A)* or may involve an avulsion of varying amounts of bone from the middle phalanx *(B)*. If a large fragment of bone is avulsed from the base of the phalanx, the dislocation is unstable after reduction. The collateral ligaments will tear in varying degrees and should be assessed with stress testing following reduction.

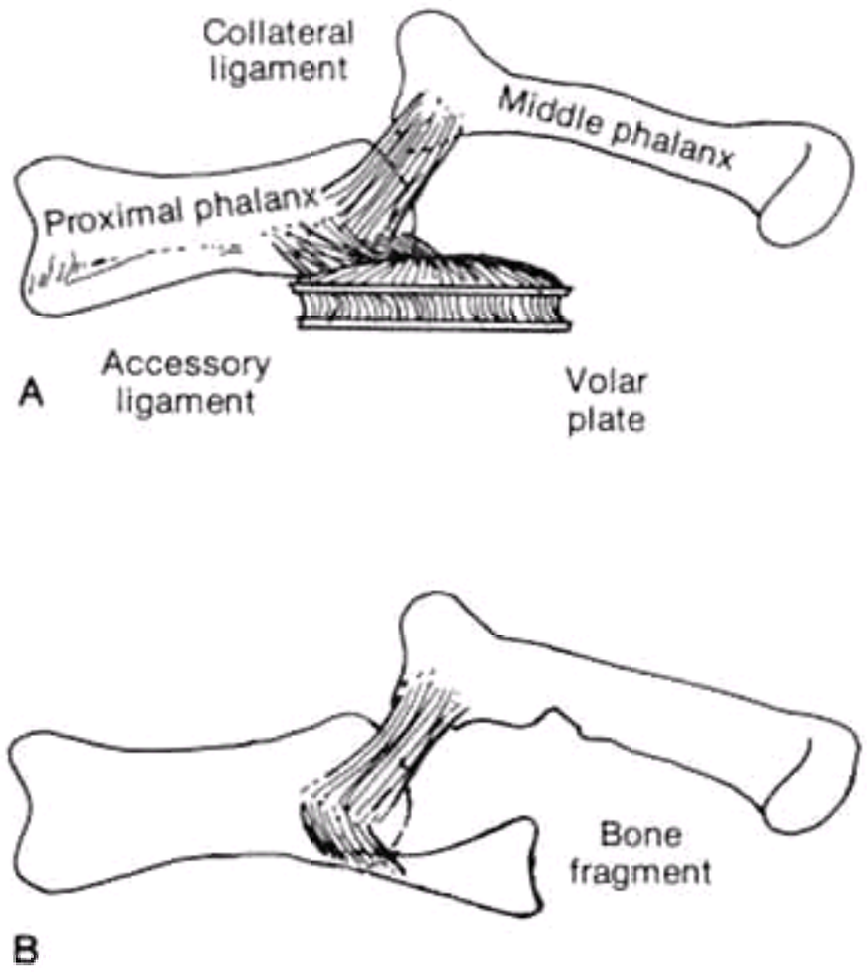


Figure 50-42 *A*, The mallet finger injury is not a dislocation; it is a rupture of the extensor tendon to the distal phalanx. *B*, This mallet deformity was caused by a baseball striking the fingertip end on, producing acute flexion of the joint.

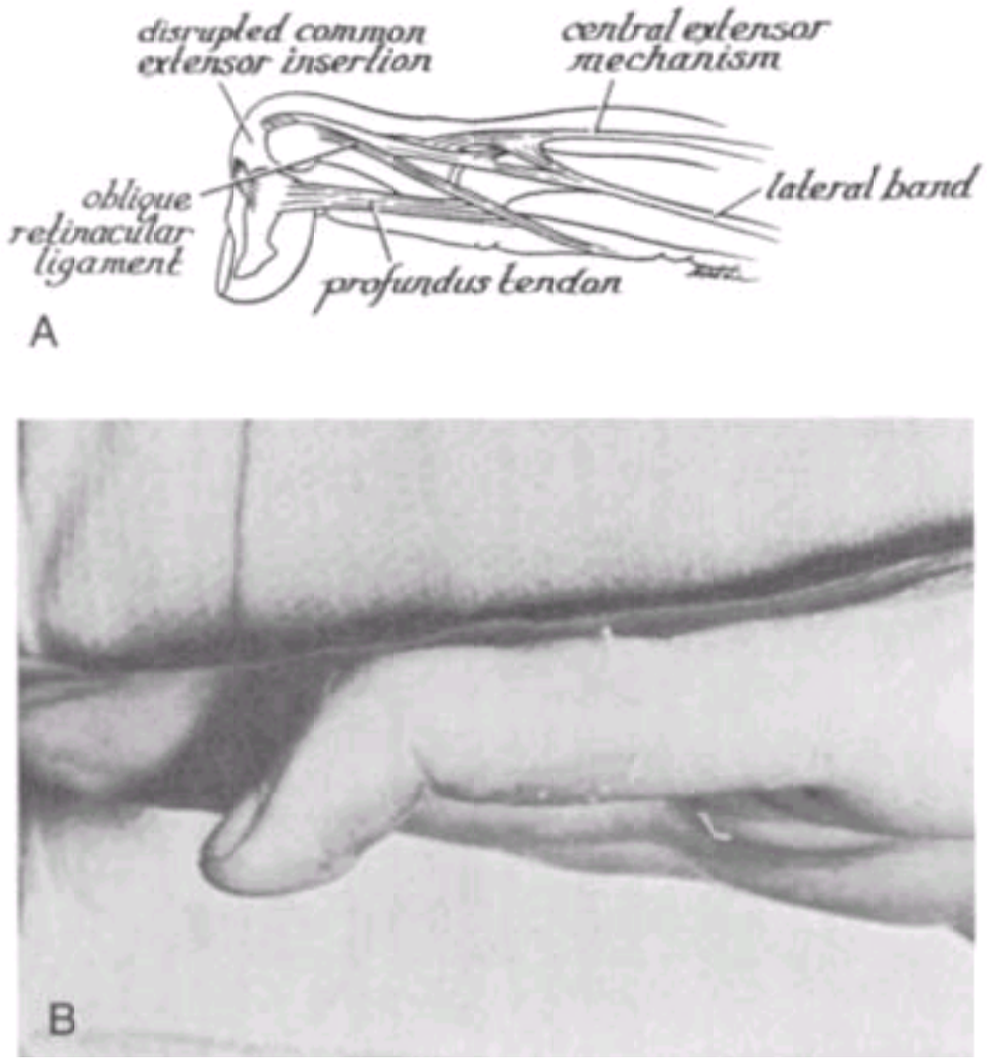
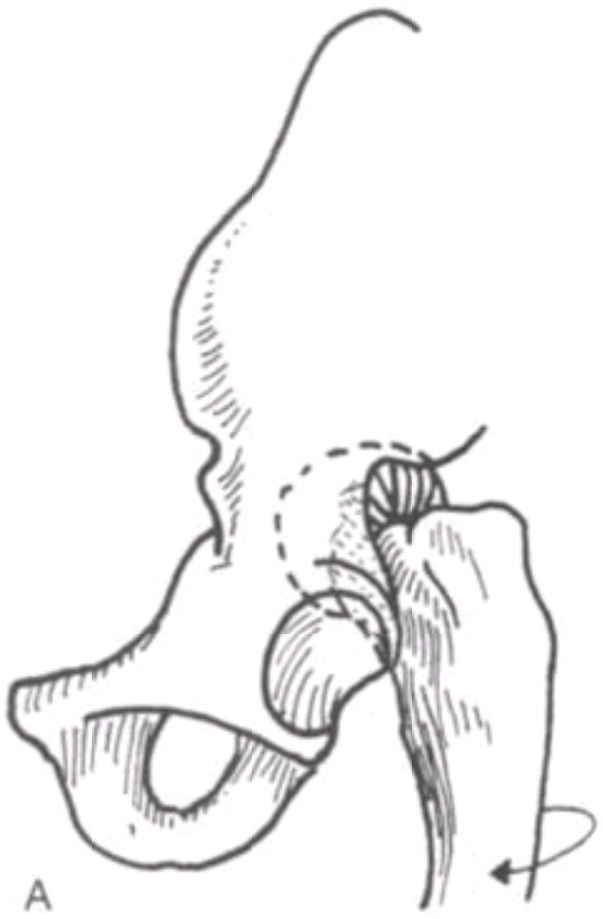


Figure 50-43 *A*, Posterior dislocation of the hip. *B,* In a severely injured patient, a hip dislocation may be initially missed while more pressing or obvious injuries are managed. In this posterior dislocation, the femur is adducted, internally rotated, and superiorly displaced, and an associated posterior acetabular fracture is present. A Malgaigne fracture of the pelvis is also present, with diastasis of the public symphysis and left sacroiliac joint. *(A from Simon R, Koenigsknecht S: Orthopedics in Emergency Medicine. New York, Appleton-Century-Crofts, 1982, p 366. B from Greenbaum E: Radiology of the Emergency Patient. New York, John Wiley & Sons, 1982, p 563. Reproduced by permission.)*



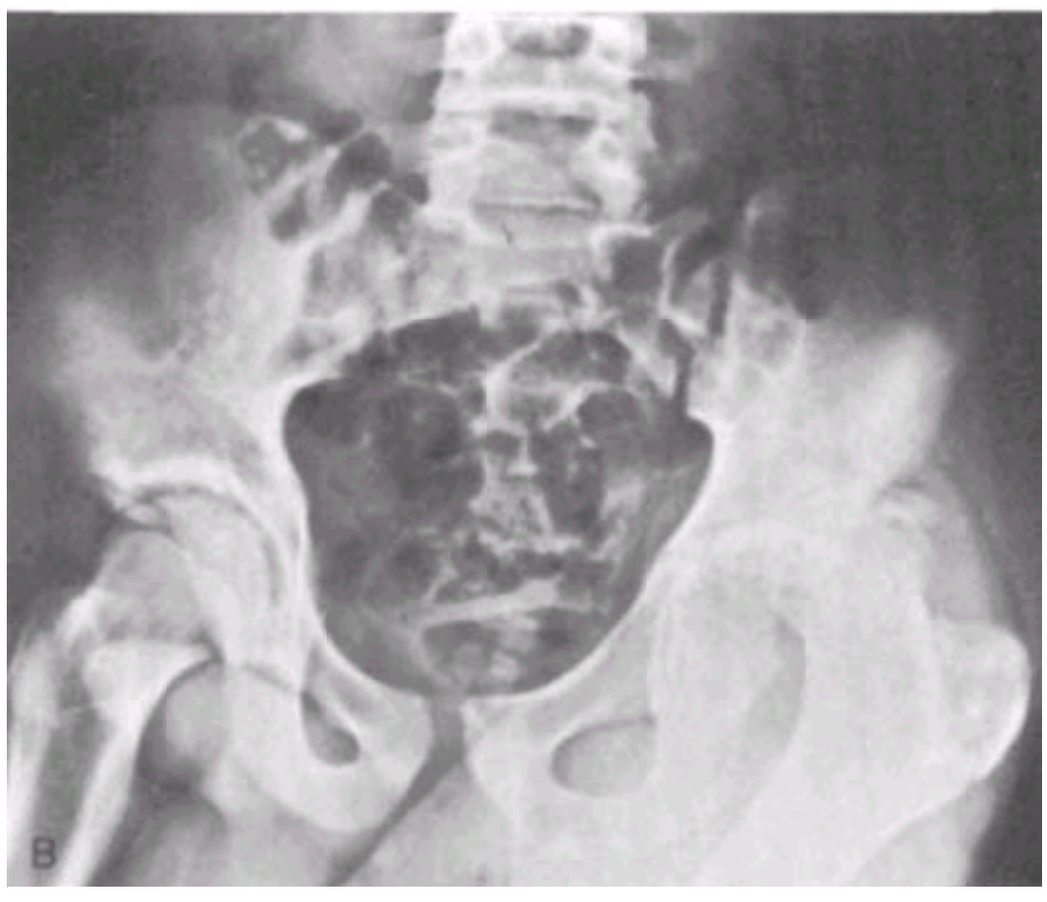


Figure 50-44 Stimson method of reduction for posterior dislocation of the hip (see text for description). *(From DeLee JC: Fractures and dislocations of the hip. In Rockwood CA, Green DP [eds]: Fractures in Adults, vol 2. Philadelphia, JB Lippincott, 1991, p 1588. Reproduced by permission.)*

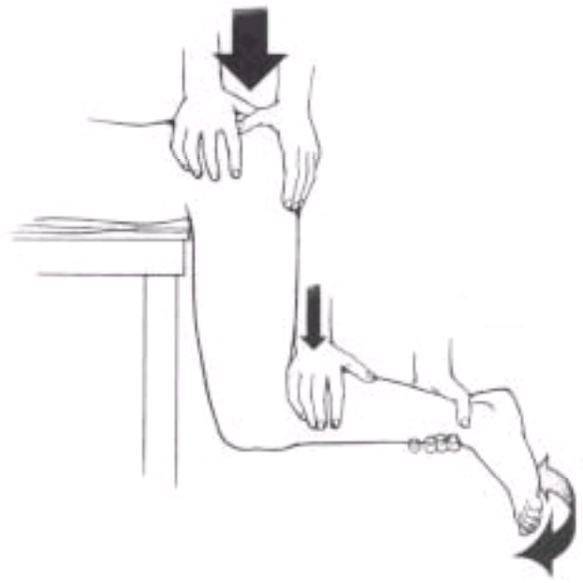


Figure 50-45 Usually a hip dislocation is the result of significant trauma. *A*, An anterior dislocation of the hip occurred in this elderly patient with a hip prosthesis from minimal trauma (rolling over in bed). *B*, Note the position of the left leg, with the hip abducted, externally rotated, and extended. This is the same position assumed by a patient with an intertrochanteric hip fracture. The more common posterior dislocation positions the hip as adducted and internally rotated. *C* and *D*, Some clinicians prefer to climb on the bed to effect reduction. The assistant may lie over the pelvis or hold the pelvis for countertraction. Care must be taken not to disrupt the prosthesis by using excessive force. Also note that osteoporotic bones can fracture during a forceful reduction. *E*, Following relocation, a knee immobilizer usually prevents the motions required to produce a recurrent dislocation.

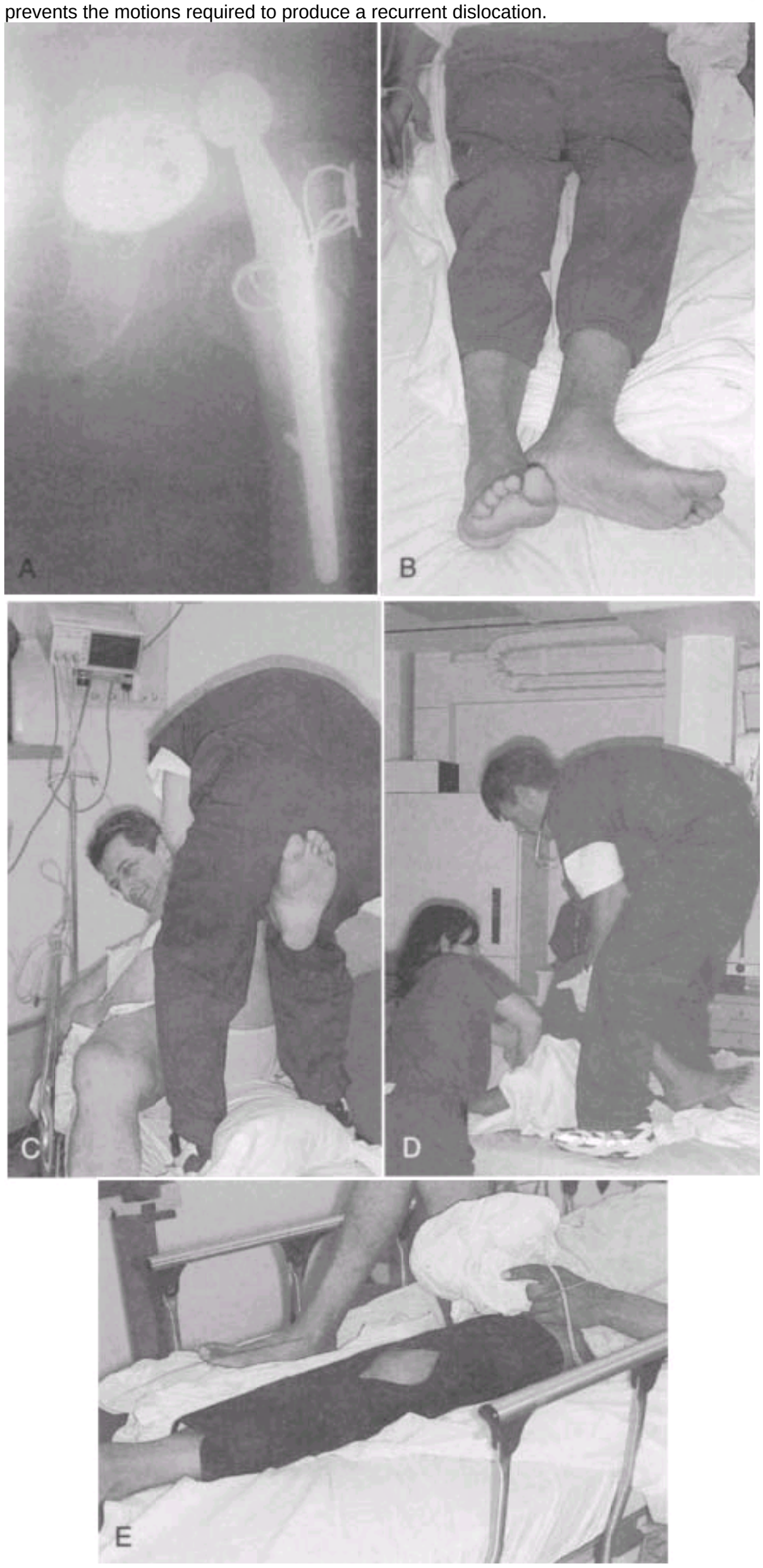


Figure 50-46 Allis method of reducing posterior dislocation of the hip (see text for description). *(From DeLee JC: Fractures and dislocations of the hip. In Rockwood CA, Green DP [eds]: Fractures in Adults, vol 2. Philadelphia, JB Lippincott, 1991, p 1594. Reproduced by permission.)*

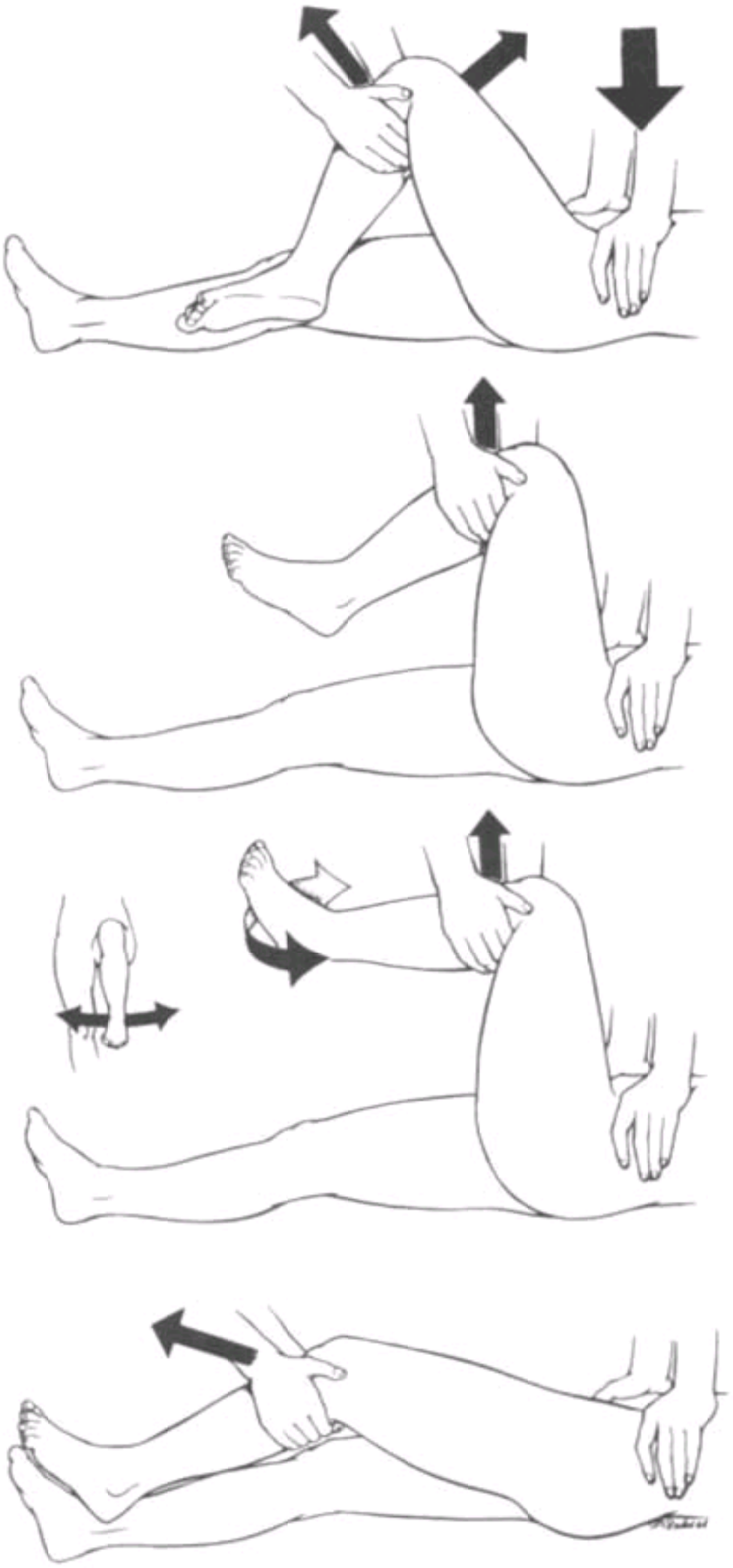


Figure 50-47 Whistler technique of reducing posterior hip dislocation. The operator stabilizes the affected ankle with one hand while placing the other arm under the affected knee to grasp the unaffected knee. While an assistant stabilizes the pelvis, the operator raises the shoulder, elevating the knee to reduce the dislocation.

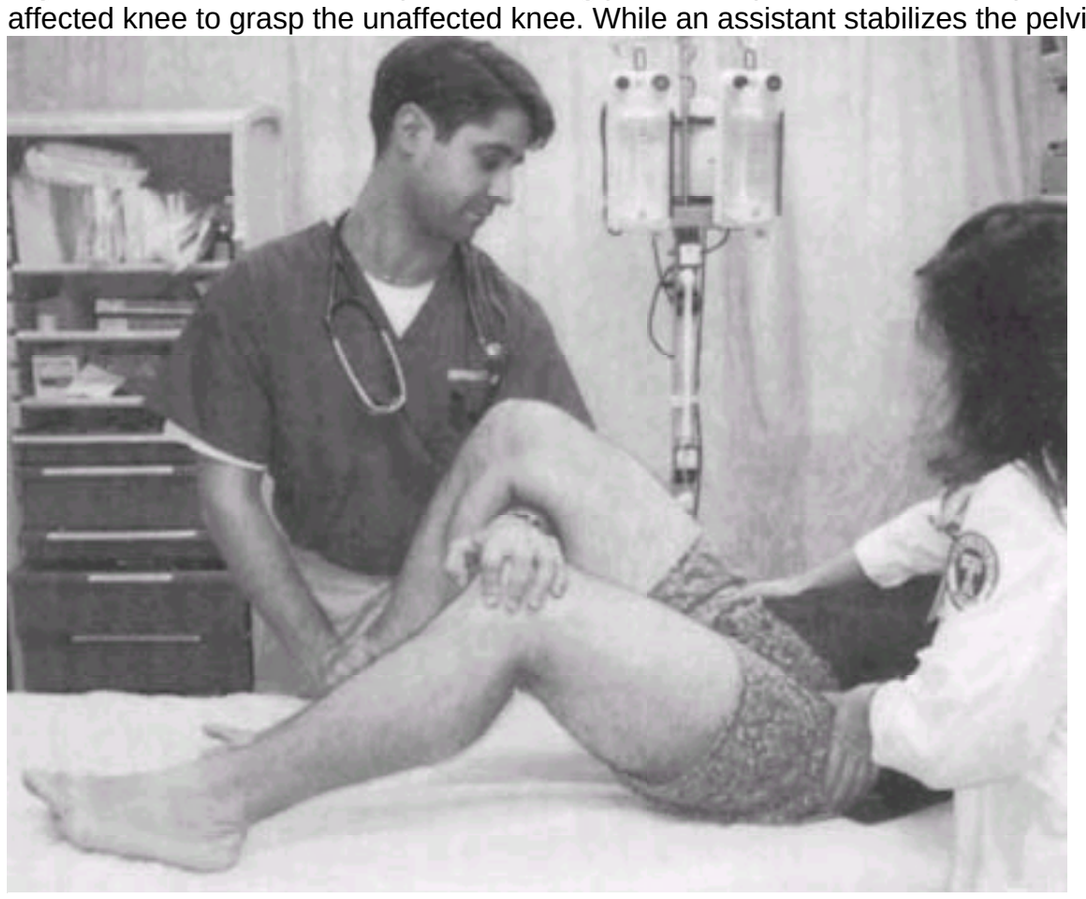


Figure 50-48 Anterior dislocations of the hip: obturator, pubic, and iliac. *(From Simon R, Koenigsknecht S: Orthopedics in Emergency Medicine. New York, Appleton-Century-Crofts, 1982, p 367. Reproduced by permission.)*

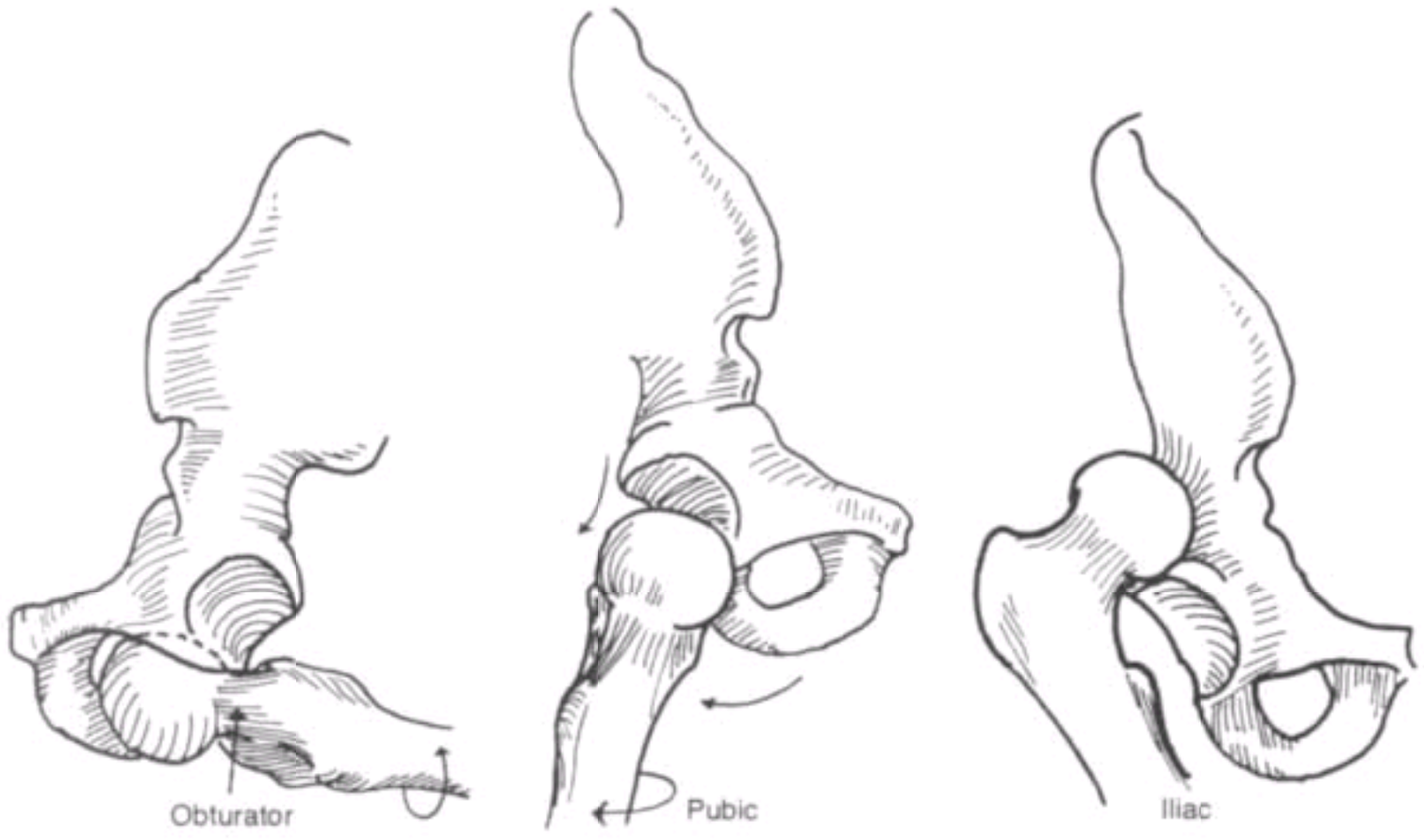


Figure 50-49 Modified Allis maneuver for reduction of anterior hip dislocation (see text for description). *(From DeLee JC: Fractures and dislocations of the hip. In Rockwood CA, Green DP (eds): Fractures in Adults, vol 2. Philadelphia, JB Lippincott, 1991, p 1588. Reproduced by permission.)*

Figure 50-50 *A*, In an obese patient a dislocated knee may not be obvious on initial inspection. This patient stated that she stepped into a hole and twisted the knee (a classic mechanism for dislocation), causing the clinician to suspect only a sprain. *B*, An x-ray demonstrated the seriousness of this seemingly benign injury. *If a spontaneous reduction occurs prior to ED evaluation, this diagnosis may not even be considered. C*, The dislocation is usually readily reduced by traction-countertraction. Tibial manipulation concurrent with traction-countertraction is often helpful.

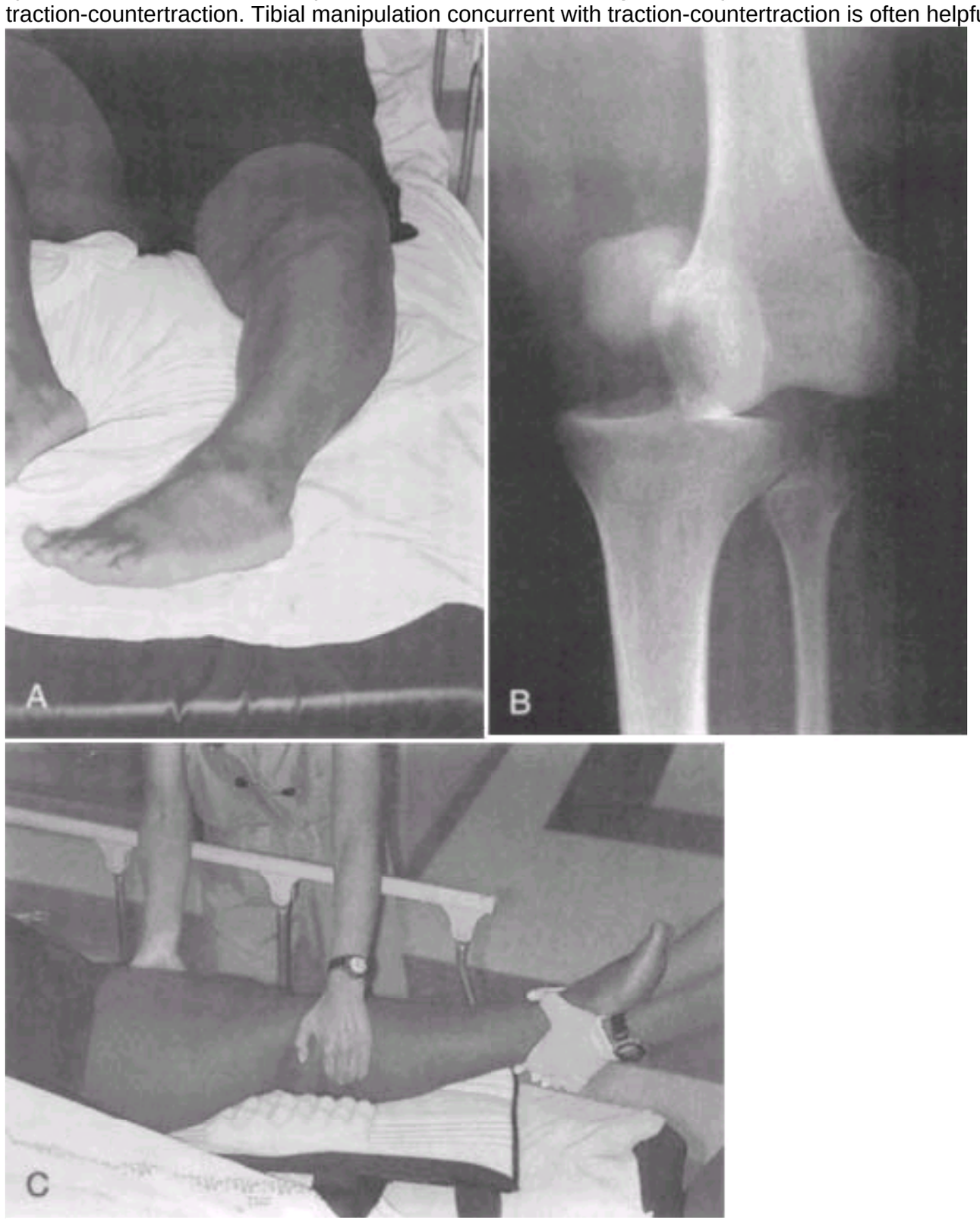


Figure 50-51 Types of knee dislocations. Anterior (*1*), posterior (*2*), and lateral (*3*). *(From DePalma AF: Management of Fractures and Dislocations: An Atlas. Philadelphia, WB Saunders, 1970, p 1621. Reproduced by permission.)*

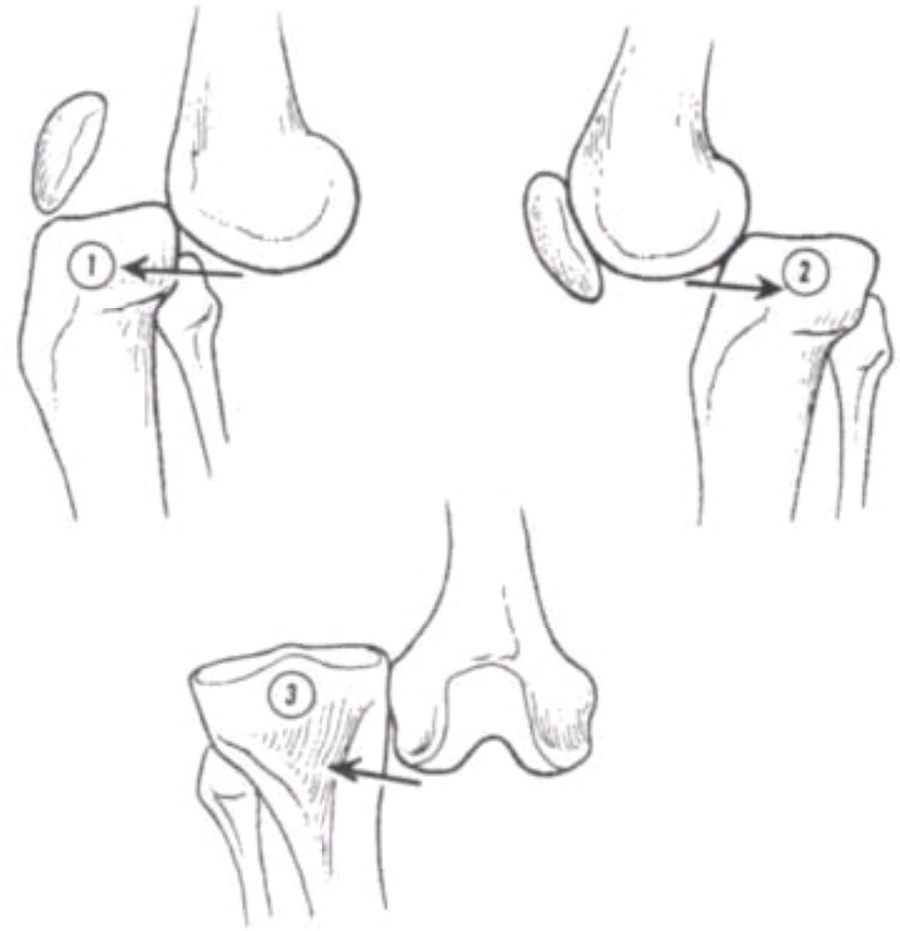


Figure 50-52 Manipulative reduction of a knee dislocation. *1,* An assistant fixes and provides countertraction on the thigh. *2,* Another assistant provides straight traction on the leg (this usually reduces the dislocation). *3,* The operator puts direct pressure over the displaced bones. *(From DePalma AF: Management of Fractures and Dislocations: An Atlas. Philadelphia, WB Saunders, 1970, p 1623. Reproduced by permission.)*

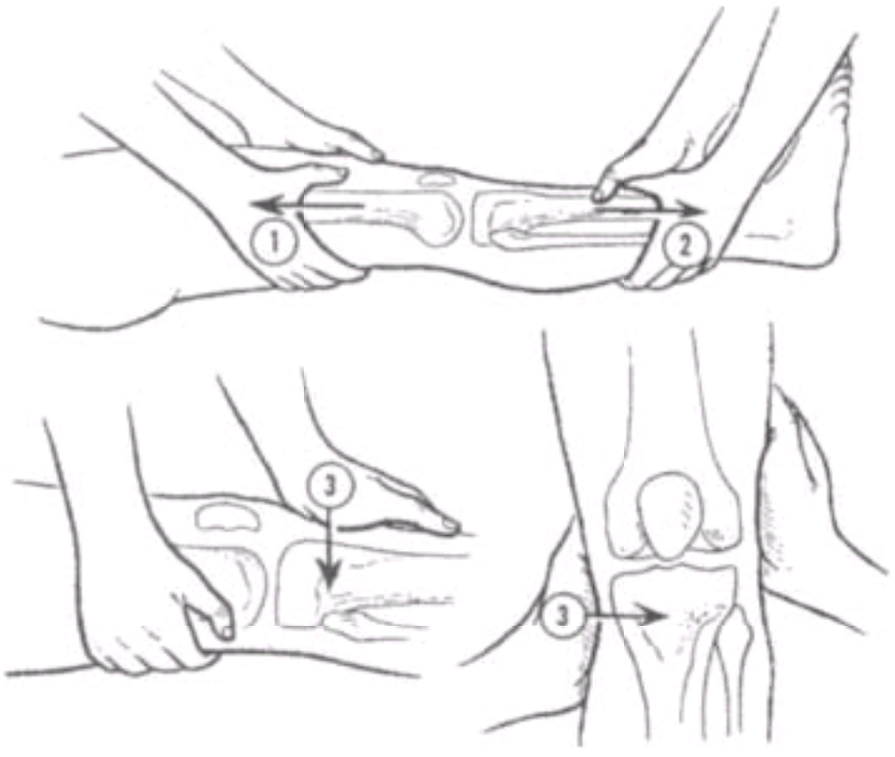


Figure 50-53 Anterolateral fibular head dislocation compared to the normal knee. The interosseous distance is widened and the proximal fibula is displaced laterally. *A*, normal anteroposterior projection of knee. *B*, lateral displacement of proximal fibula. *C*, use of bilateral comparison views to highlight fibular displacement.

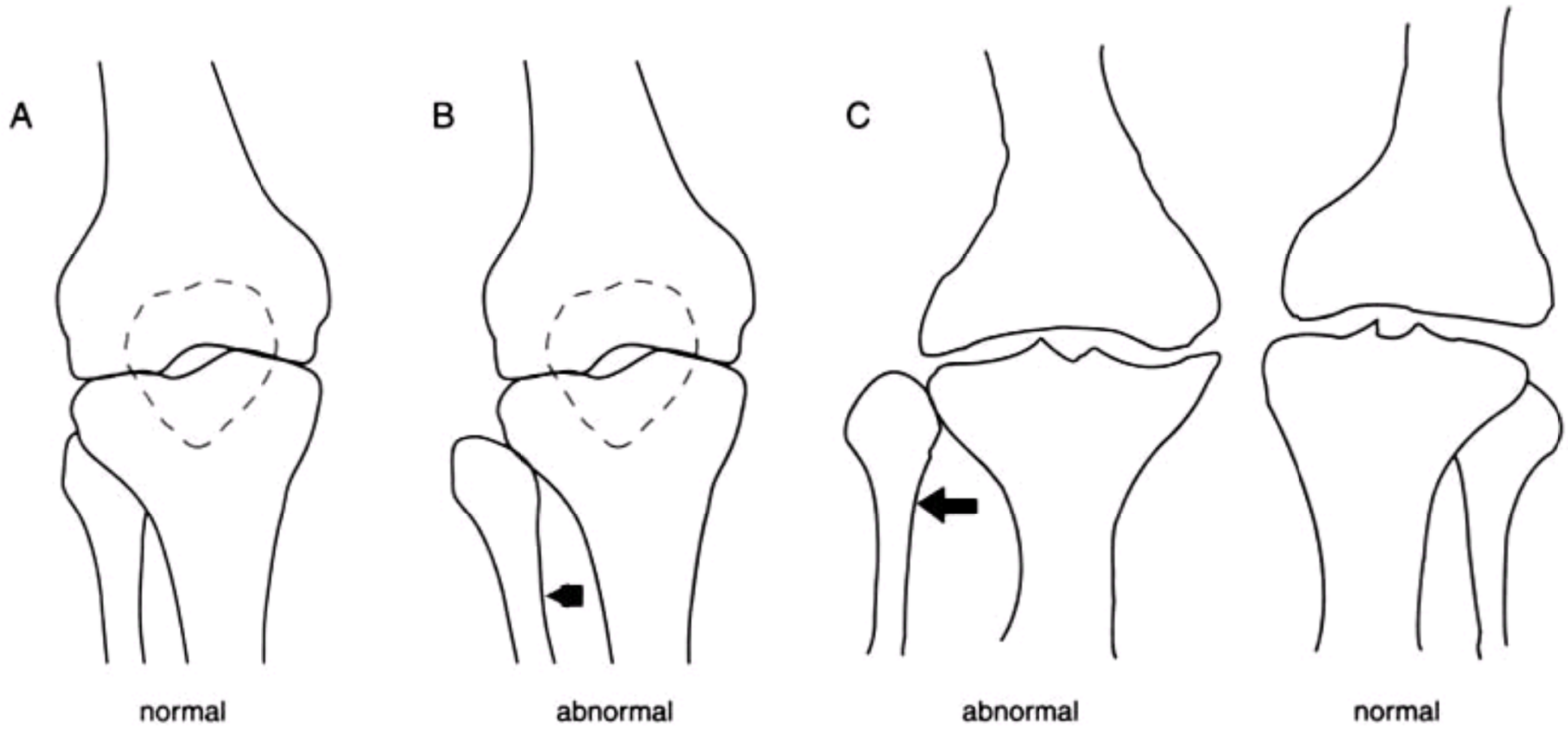


Figure 50-54 Various types of patellar dislocation. The lateral dislocation is the most common.

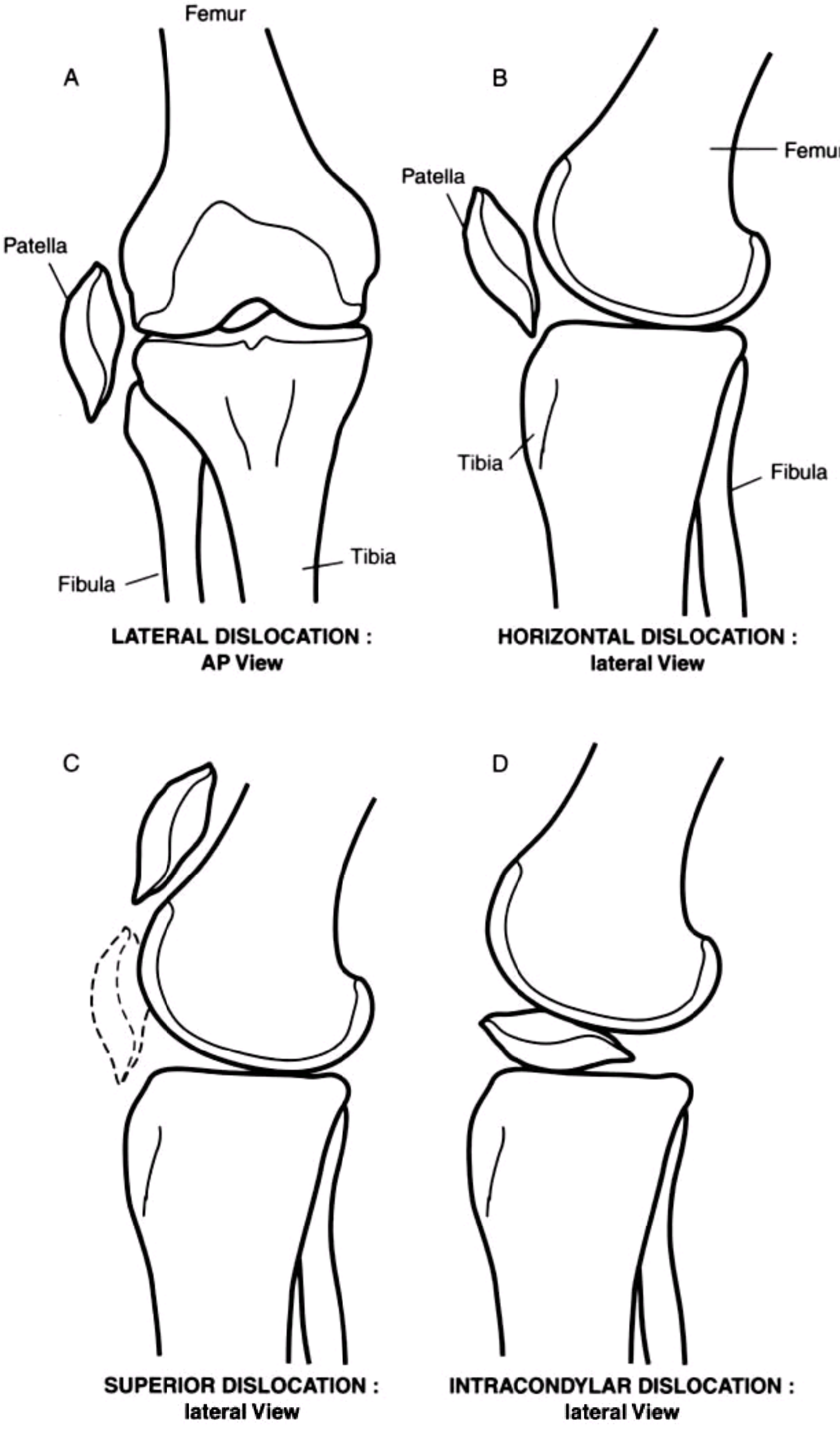


Figure 50-55 *A*, Lateral dislocation of the patella. *B*, To reduce a lateral dislocation, the knee is extended while the patella is directed medially using slight anteriorly directed elevation.

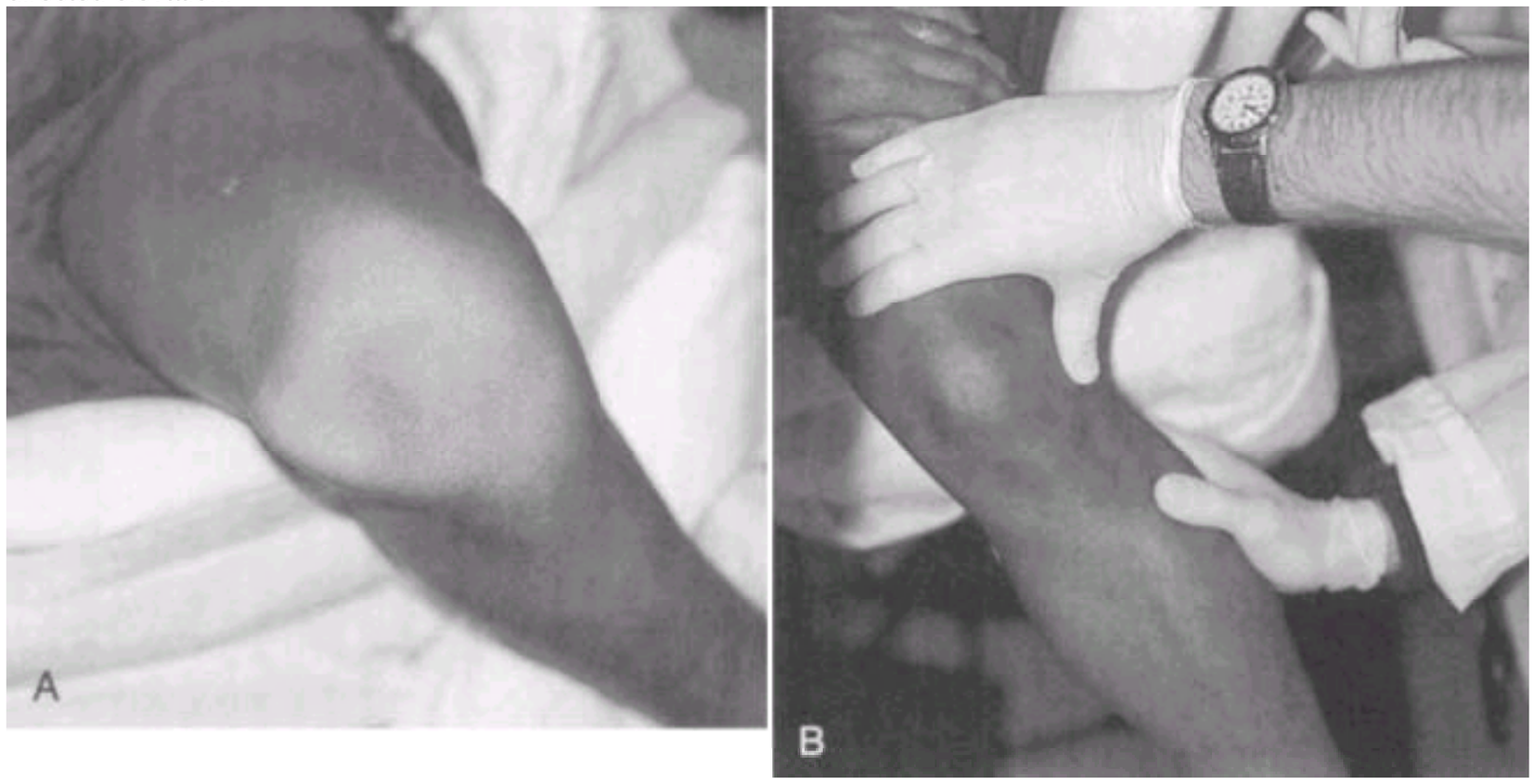


Figure 50-56 Manipulative reduction of a lateral patellar dislocation. Extend the knee gradually (*1*) while applying medially directed pressure on the patella *(2)*, pushing it over the lateral femoral condyle. *(From DePalma AF: Management of Fractures and Dislocations. Philadelphia, WB Saunders, 1970, p 1665. Reproduced by permission.)*

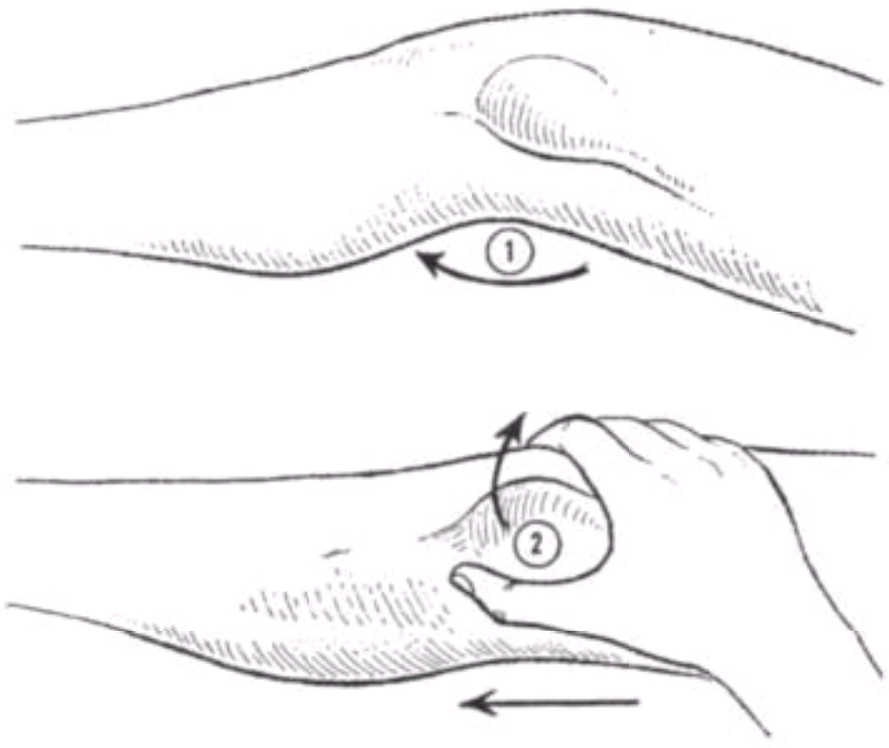


Figure 50-57 The types of dislocations of the ankle. *(From Simon R, Koenigsknecht S: Orthopedics in Emergency Medicine. New York, Appleton-Century-Crofts, 1982, p 419. Reproduced by permission.)*

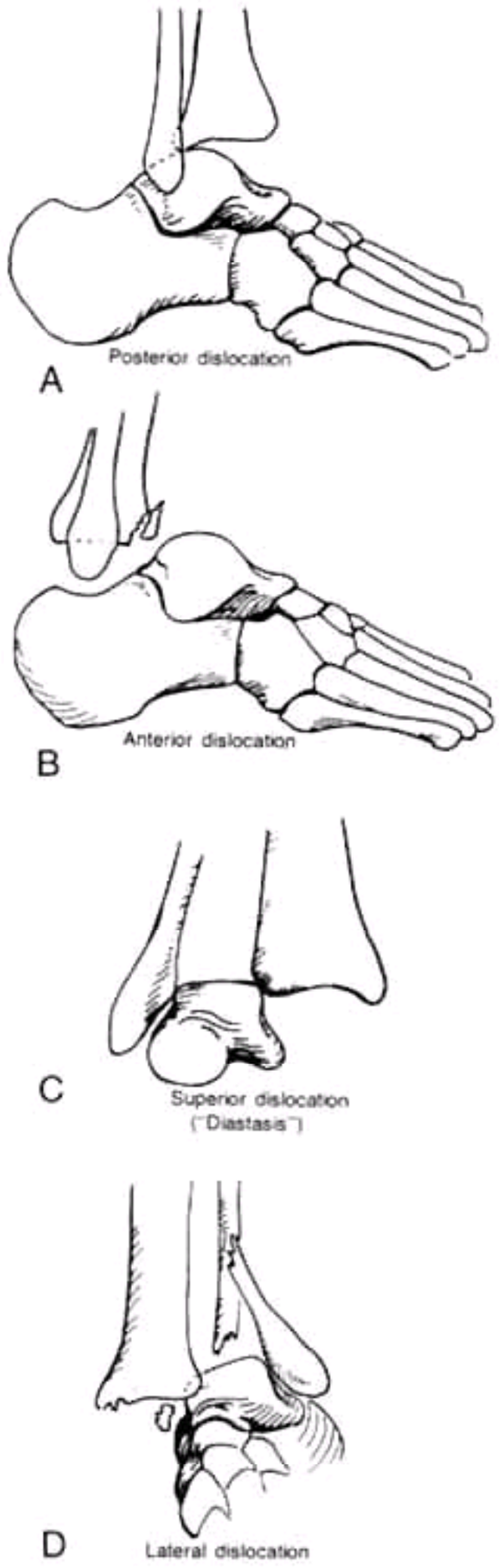


Figure 50-58 Isolated posterior tibial lip fracture *(open arrow),* seen after reduction of posterior ankle dislocation. *(From Harris JH Jr, Harris WH: Radiology of Emergency Medicine, 2nd ed. Baltimore, Williams & Wilkins, 1981, p 629. Reproduced by permission.)*

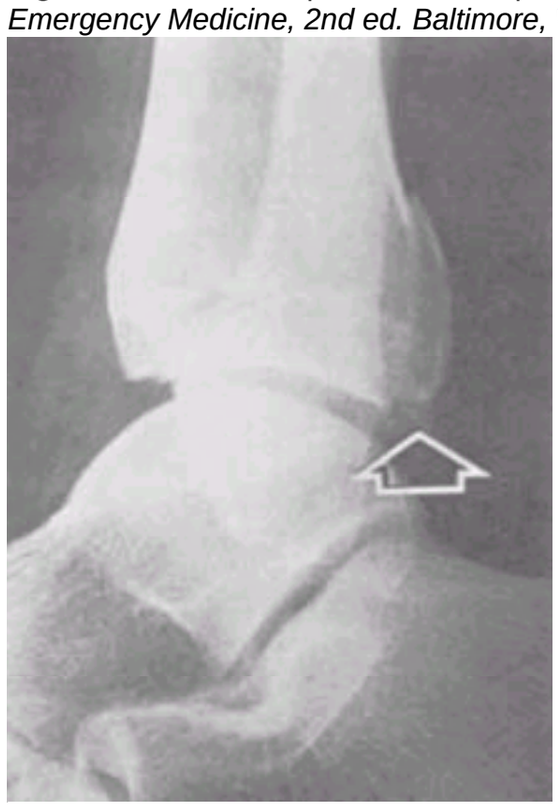


Figure 50-59 Manipulative reduction of a posterior ankle dislocation. *1,* The knee is flexed. *2,* The assistant provides countertraction on the leg. *3,* The forefoot is grasped with one hand and the heel with the other hand. *4,* The foot is slightly plantar flexed. *5,* Apply straight downward traction on the plantar flexed foot, then pull the foot forward *(6)* while a second assistant provides counterpressure on the front of the lower leg *(7)*. *(From DePalma AF: Management of Fractures and Dislocations. Philadelphia, WB Saunders, 1970, pp 1916 and 1917. Reproduced by permission.)*

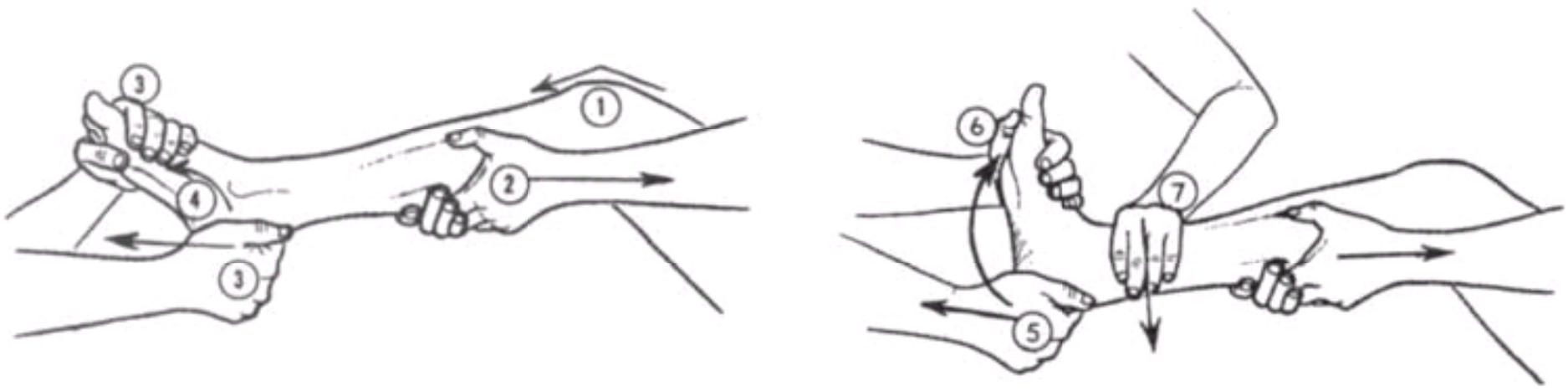


Figure 50-60 Manipulative reduction of anterior ankle dislocation. *1,* The knee is flexed. *2,* The operator grasps the forefoot with one hand and the heel with the other hand. *3,* Dorsiflexion of the foot is slightly increased (to disengage the talus). *4,* An assistant provides countertraction on the leg. *5,* Straight longitudinal traction is applied, then the foot is pushed directly backward *(6)* while a second assistant provides countertraction on the back of the lower leg *(7)*. *(From DePalma AF: Management of Fractures and Dislocations. Philadelphia, WB Saunders, 1970, pp 1918 and 1919. Reproduced by permission.)*

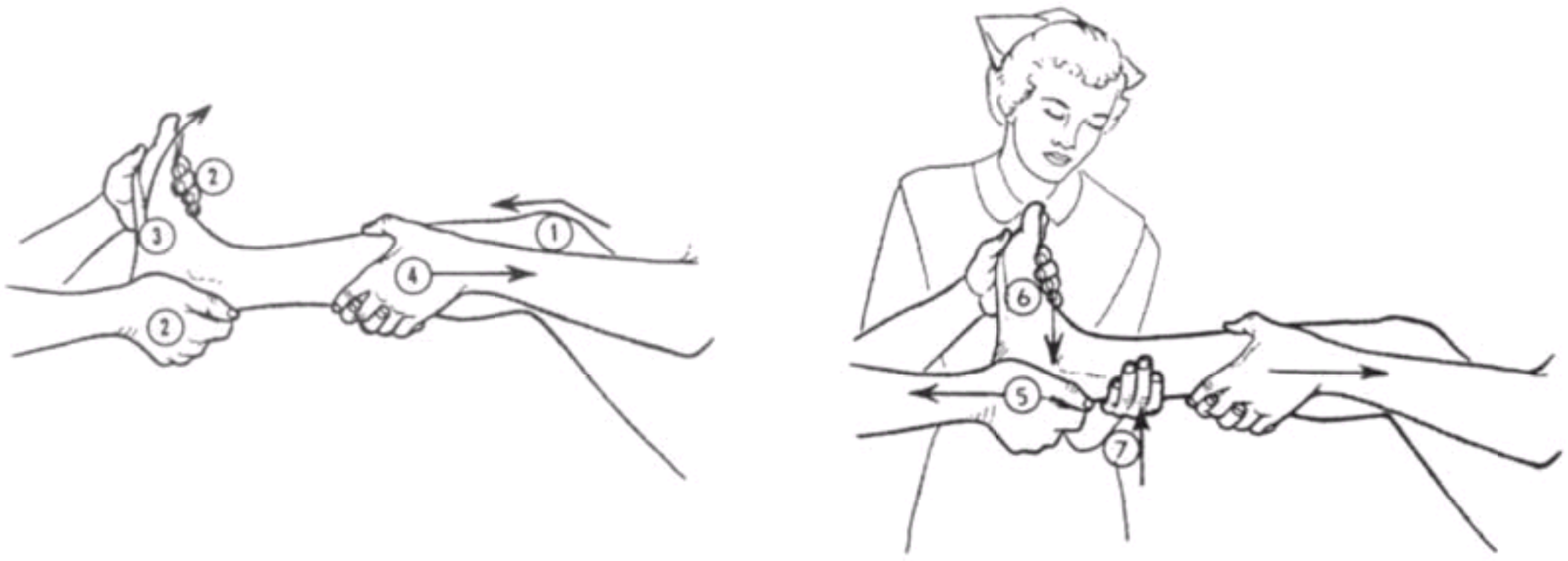


Figure 51-1 Some clinicians prefer to use premade plaster splints for temporary immobilization (e.g., OCL, J-Splint). There is less latitude for custom fitting with these products compared with the technique demonstrated in Figure 51-3 . *A,* Measure the appropriate length. (Note that plain plaster slabs shrink several centimeters when wet. Cut the material slightly long, since the excess may be folded back on itself if necessary). *B,* Submerge the plaster until bubbling stops (do not oversoak). *C,* Roll and squeeze excess water from the premade splint. *Inset,* If using plain plaster slabs, allow the water to drip off. *D,* Smooth the sheets to remove wrinkles and mix the plaster throughout the layers. *E,* Apply the splint and secure it with an elastic bandage. *F,* Mold the splint to fit the contour of the extremity—an important step. *(Courtesy of Johnson & Johnson Products, Inc., New Brunswick, NJ.)*

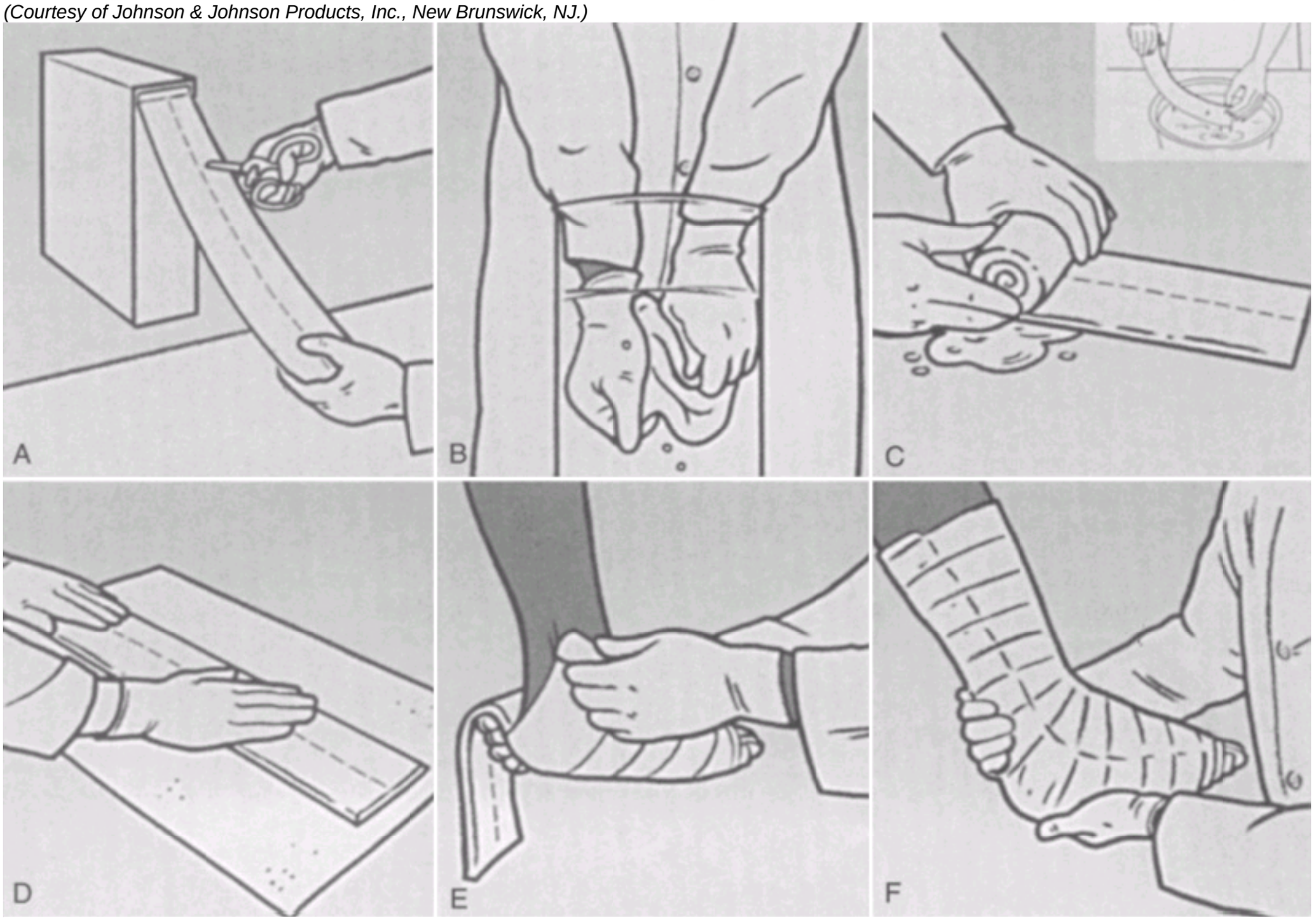


Figure 51-2 Premade fiberglass splints are also popular for temporary immobilization. Fiberglass splints set and cure more rapidly, have no messy residue (i.e., can be hydrated in a conventional sink without a special trap), can be washed and reapplied, and are lighter and stronger than plaster splints. *A*, Open the pouch and cut the splint to the desired length, stretching the padding to cover the exposed edge. Dip into cool water and squeeze three or four times (Note: Prefabricated fiberglass splints that do not contain water-repellant felt are hydrated in a different manner—Always follow the manufacturer's instructions). Remove the splint from the bucket and squeeze to remove any excess water (this allows a curing time of 3 to 4 minutes). *B*, Place the splint on an absorbent towel to remove excess water. *C,* Apply the splint and secure it with an elastic bandage. When applying the splint at a right angle (e.g., posterior ankle splint), pinch and fold over any extra splinting material). *(Adapted, courtesy of 3M Medical Division, 3M Health Care Markets, St. Paul, MN.)*

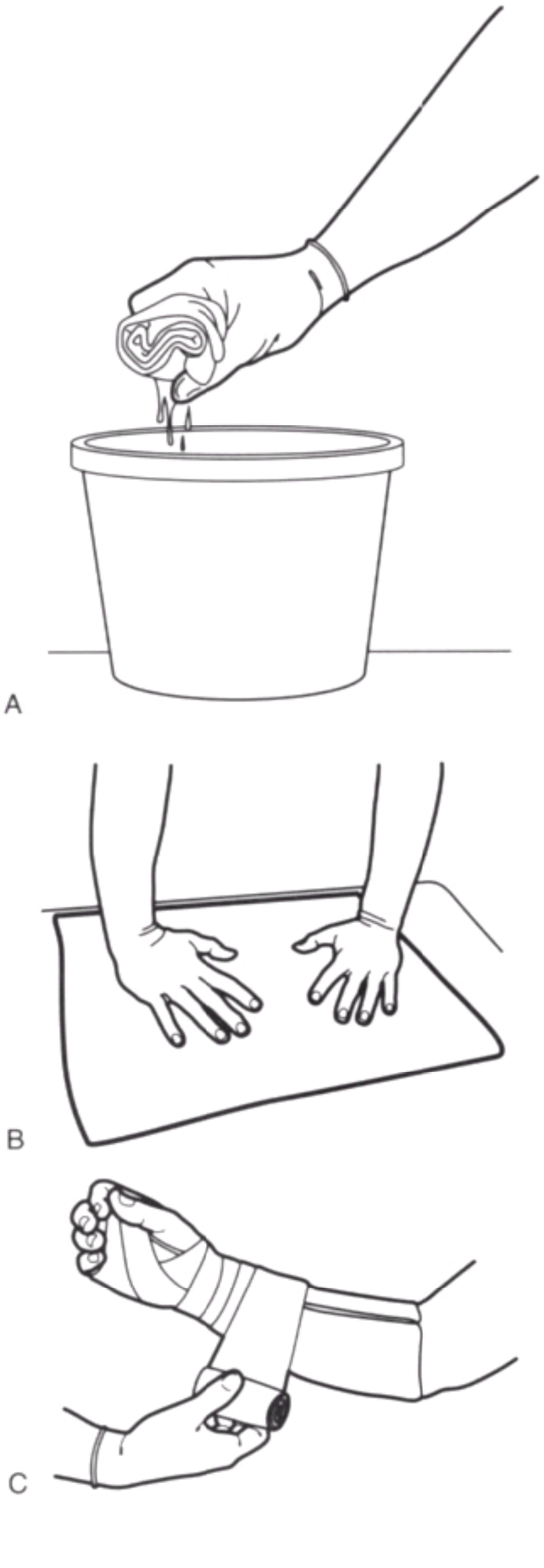


Figure 51-3 Principles of custom splint application. *A*, Stockinette is applied to extend 2 or 3 in beyond the plaster. *B*, Two to three layers of Webril are evenly and smoothly applied over the area to be splinted. *C*, The plaster slab is positioned over the area to be immobilized and the stockinette and Webril are folded back to help secure the slab in place and to form smooth, rounded ends. *D*, The elastic bandage is applied to secure the splint. *E*, While still wet, the plaster is molded to conform to the shape of the extremity. This is an important step that is often overlooked.

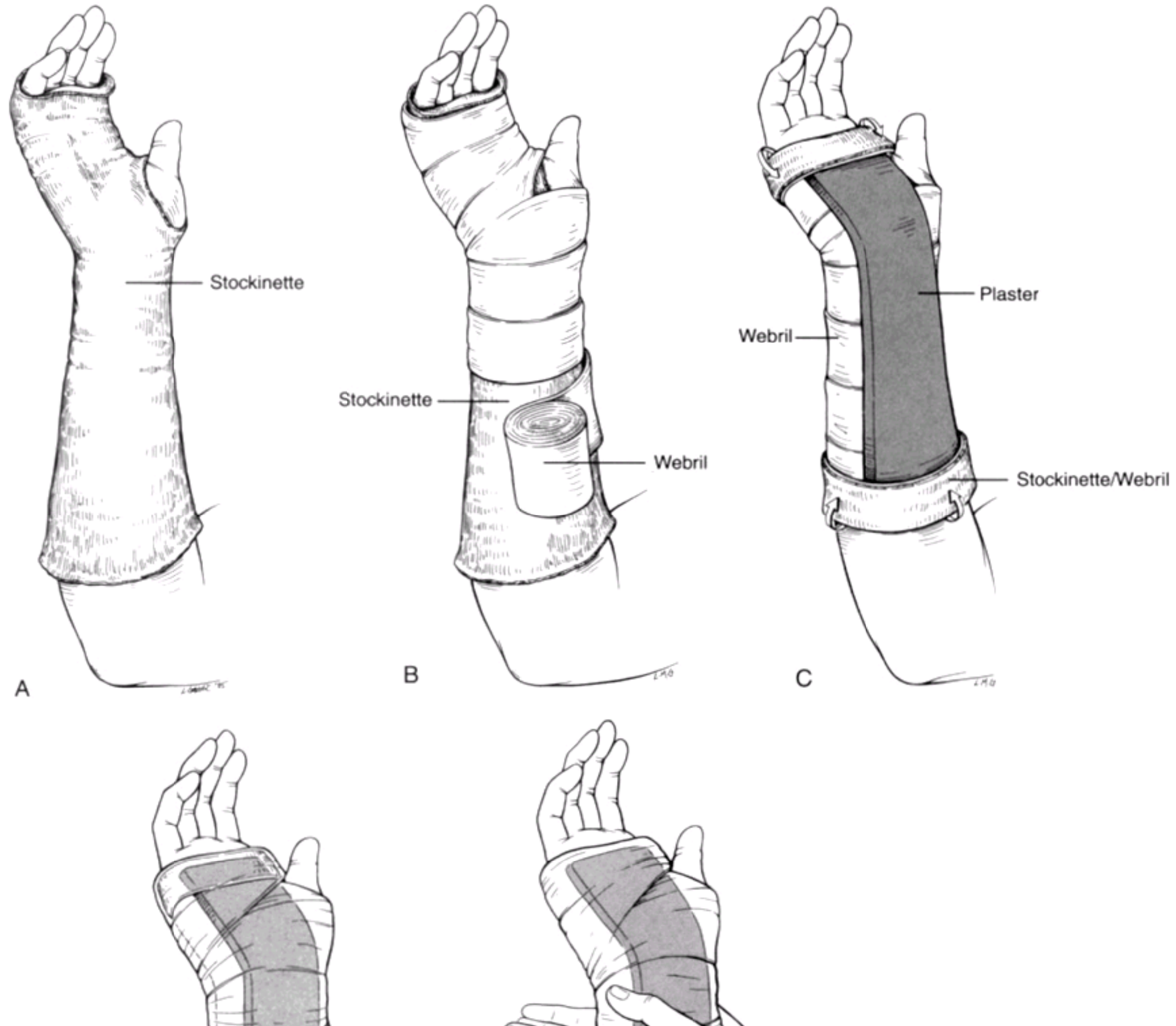


Figure 51-4 Alternative method of Webril application. If significant swelling is anticipated, Webril (the same diameter as the plaster) may be placed directly over the wet plaster, rather than wrapping it around the extremity. The Webril-lined splint is then positioned over the area to be immobilized and secured with an elastic bandage. Sandwiching the plaster strips with Webril will also minimize rigid adherence of the plaster to the elastic wrap.

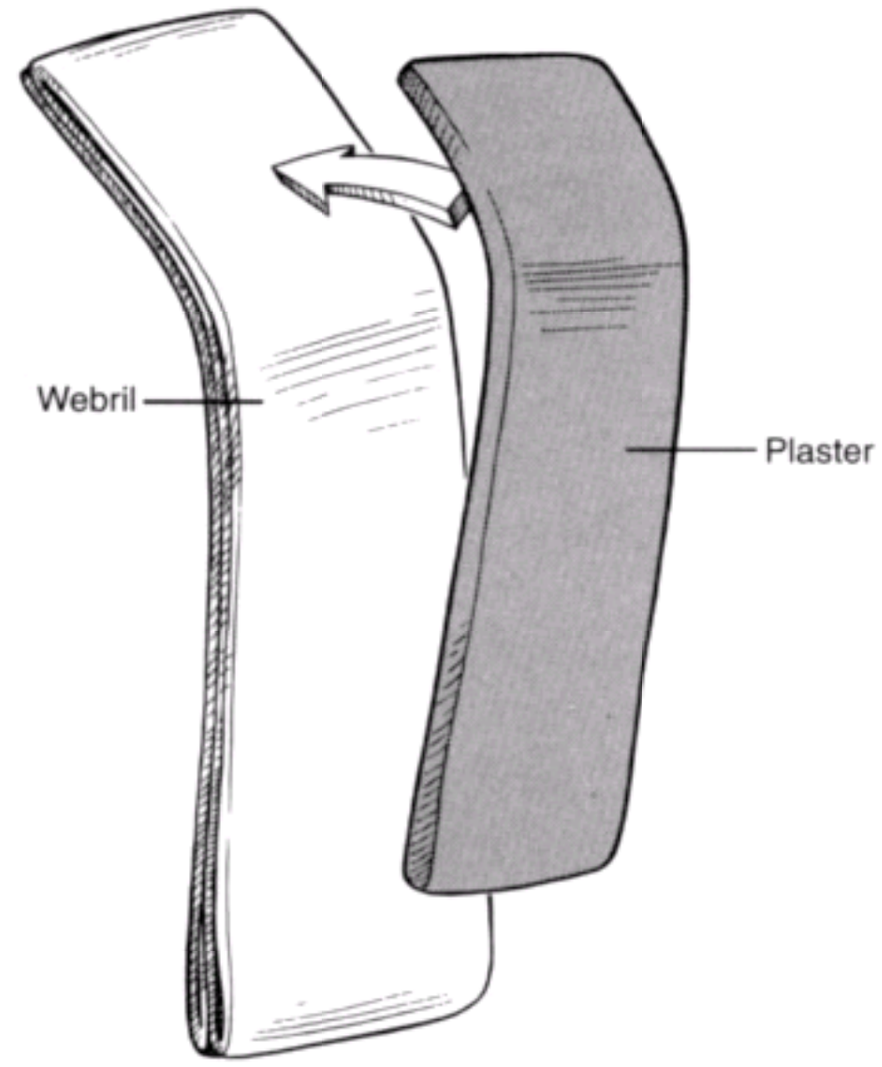


Figure 51-5 Application of a long arm posterior splint. *A*, The posterior portion of the splint begins on the posterior aspect of the proximal humerus. It runs down the arm to the elbow and then continues along the ulnar aspect of the forearm and hand to the distal metacarpals. The elbow is flexed at a 90° angle, the forearm is in the neutral (thumb up) position, and the wrist is in a neutral position or slightly (10°–20°) extended. *B*, Adding an anterior splint. The anterior splint mirrors the posterior splint by running down the anterior aspect of the arm to the antecubital fossa, where it continues along the radial aspect of the forearm and hand to the distal radius. The anterior splint is never used alone, but rather as an adjunct to the long arm posterior splint. It improves immobilization by increasing stability and preventing pronation and supination of the forearm.

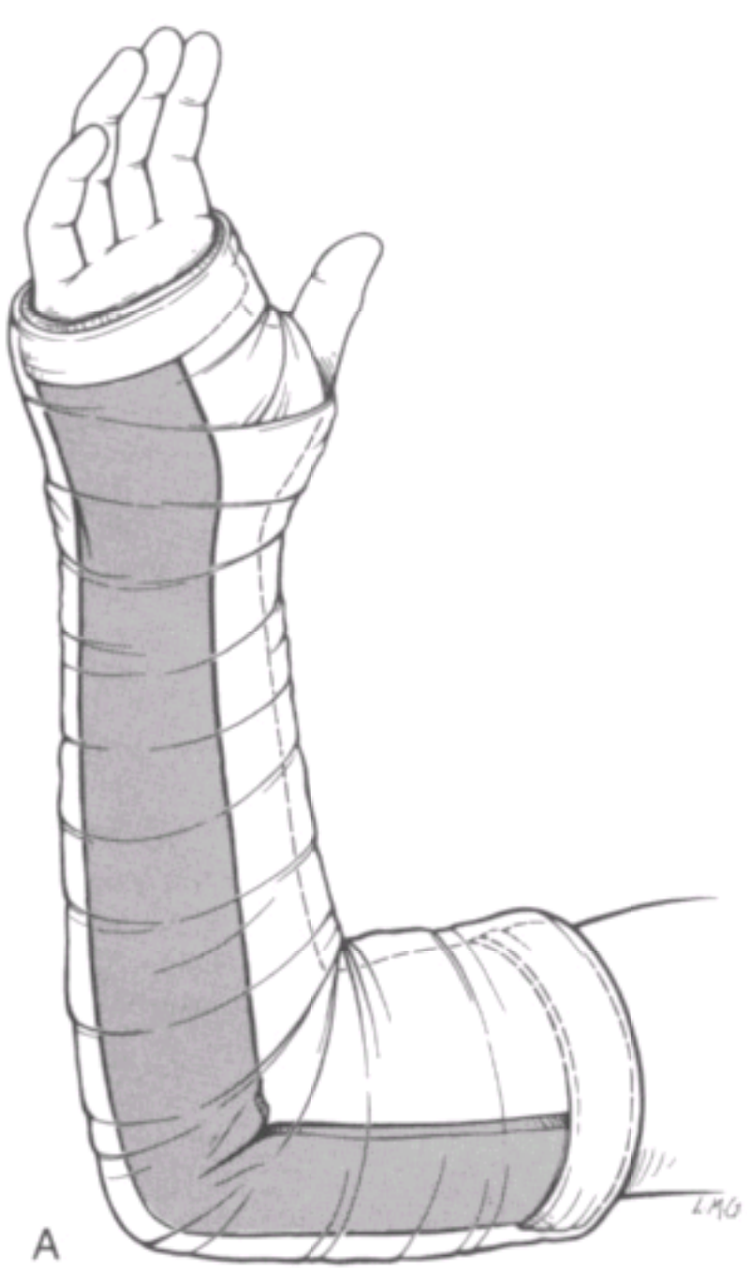


Figure 51-6 An alternative to the long arm posterior splint is a double sugar-tong splint. This splint immobilizes the elbow and prevents pronation and supination of the forearm. The splint consists of two separate pieces of 4-in plaster, a forearm splint, and an arm splint. The elbow is flexed at a 90° angle, the forearm is in the neutral (thumb up) position, and the wrist is in a neutral position or slightly (10°–20°) extended. The forearm portion of the splint is applied first. It runs from the metacarpal heads on the dorsum of the hand, along the dorsal surface of the forearm, and around the elbow. It continues along the volar surface of the forearm, stopping at the level of the metacarpophalangeal joints. The arm portion of this splint begins on the anterior aspect of the proximal arm. It runs down the arm over the forearm splint and around the elbow. It then continues up the posterior aspect of the arm, once again going over the forearm splint until it reaches the starting point. The fingers and thumb should remain free to avoid stiffness.

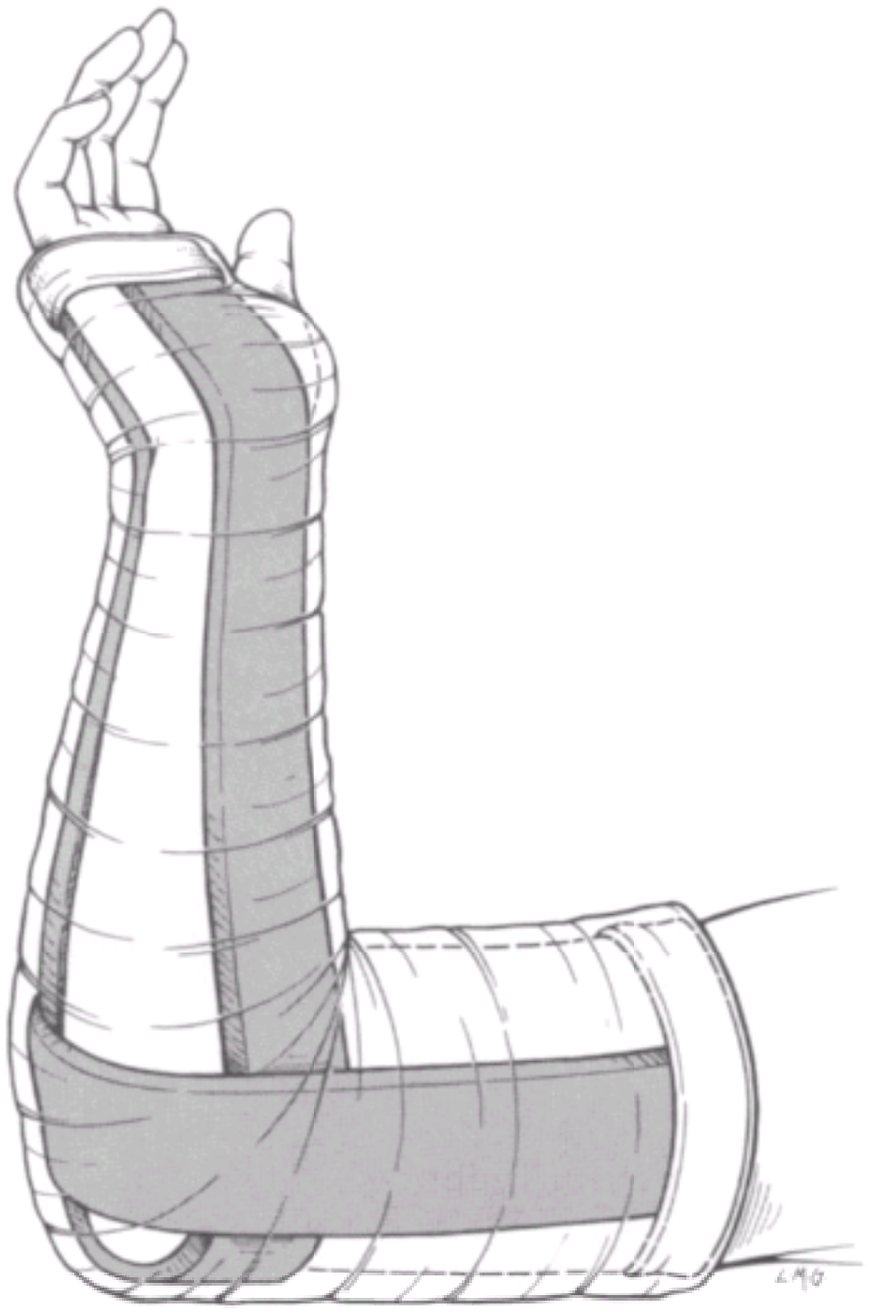


Figure 51-7 Application of a volar splint. *A*, The splint begins in the palm at the metacarpal heads and extends along the volar surface of the forearm to a point just proximal to the elbow. If any of the fingers are injured, the splint may be extended to incorporate the involved digits. The forearm is placed in the neutral position (thumb upward) with the wrist slightly (10°–20°) extended. Wrist flexion should be avoided. *B*, For more serious injuries, an additional dorsal slab may be used to create a bivalve splint.

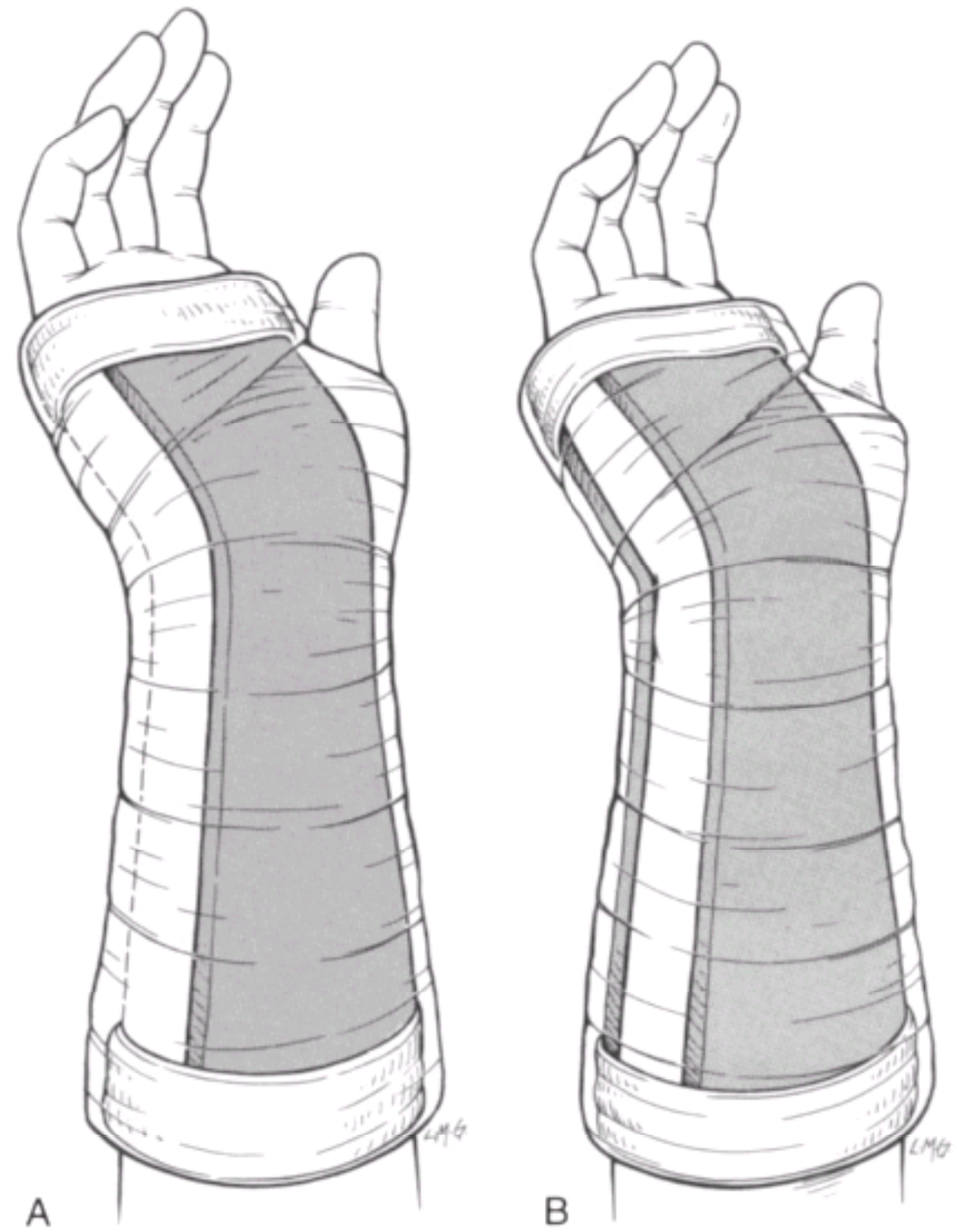


Figure 51-8 Application of a forearm sugar-tong splint. The splint runs from the metacarpal heads on the dorsum of the hand, along the dorsal surface of the forearm, and around the elbow. It continues along the volar surface of the forearm stopping at the level of the metacarpophalangeal joints. The elbow is flexed at a 90° angle, the forearm is in the neutral (thumb up) position, and the wrist is in a neutral position or slightly (10°–20°) extended. The advantage of this splint over the volar splint is immobilization of the elbow and prevention of pronation and supination of the forearm.

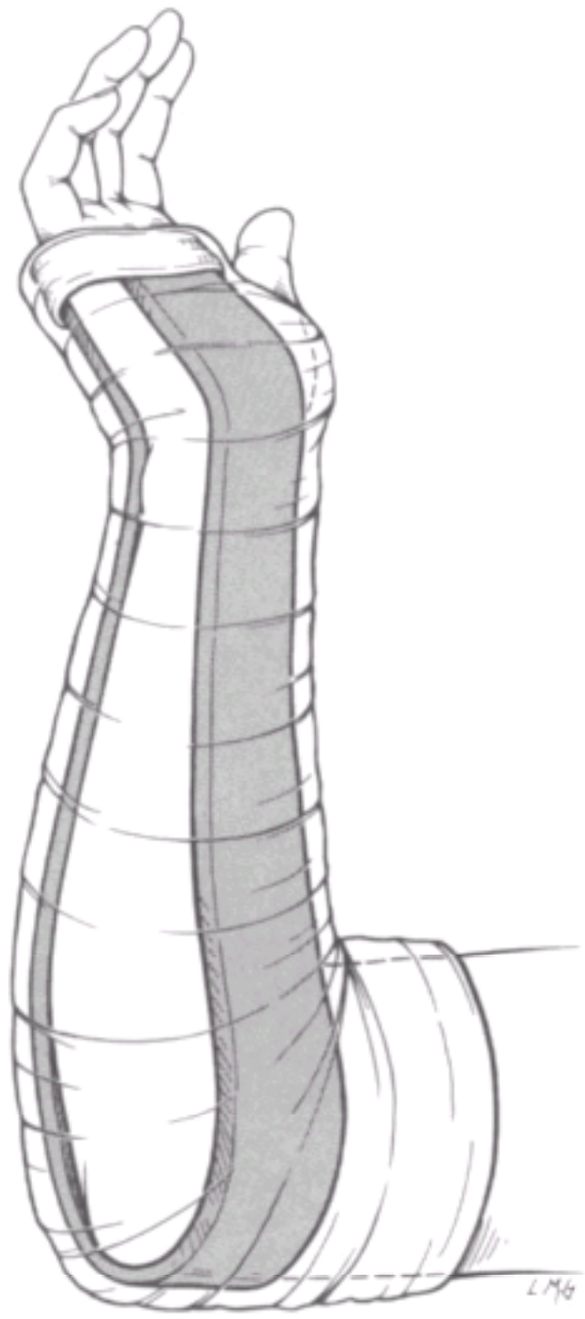


Figure 51-9 Application of a thumb spica splint. The splint extends from just distal to the interphalangeal joint of the thumb to the mid-forearm. The forearm is placed in the neutral position with the wrist extended 25° and the thumb in the wine glass position (see Fig. 51-10). *Inset, A* small (1- to 2-cm) perpendicular cut is made 1 cm distal to the first metacarpophalangeal joint on each edge of the plaster to allow molding of the splint around the thumb without creating a buckle in the plaster.

Figure 51-10 The wine glass position, which is a safe splint position for the hand. The wrist should allow alignment of the thumb with the forearm, the metacarpophalangeal joint should be moderately flexed, and the interphalangeal joints should be only slightly flexed. The thumb should be abducted away from the palm.

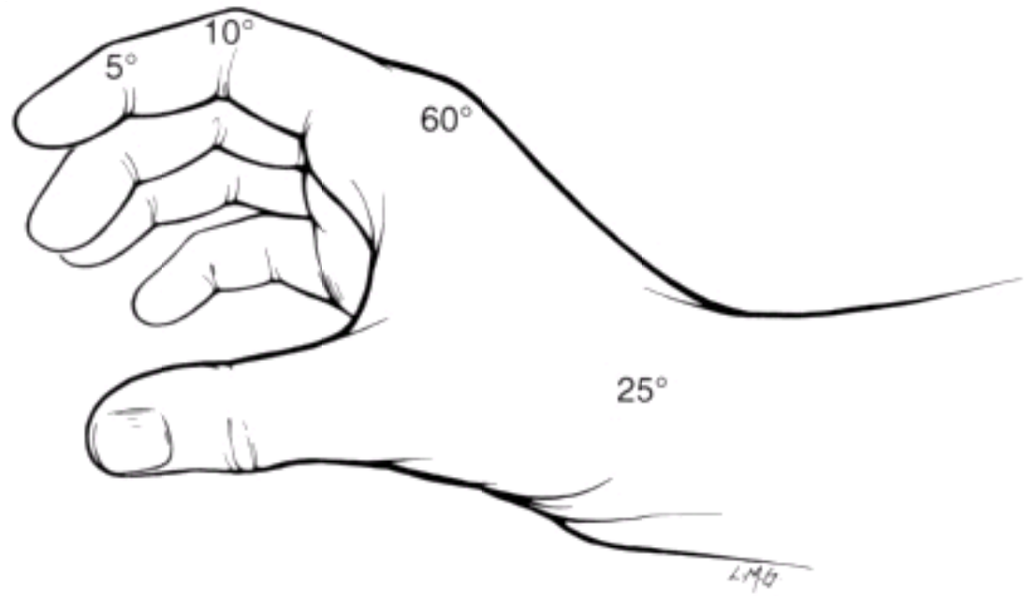


Figure 51-11 Application of an ulnar gutter splint. The ulnar gutter splint incorporates both the little and ring fingers. Webril or gauze should be placed between the digits to prevent maceration of the skin. The splint runs along the ulnar aspect of the forearm from just beyond the distal interphalangeal joint of the little finger to the mid-forearm. The forearm is in the neutral position with the wrist in slight extension (10°–20°), the metacarpophalangeal joint in 50° of flexion, and the proximal and distal interphalangeal joint in slight (10°–15°) flexion. When immobilizing a metacarpal neck fracture, the metacarpophalangeal joint should be flexed to 90°.

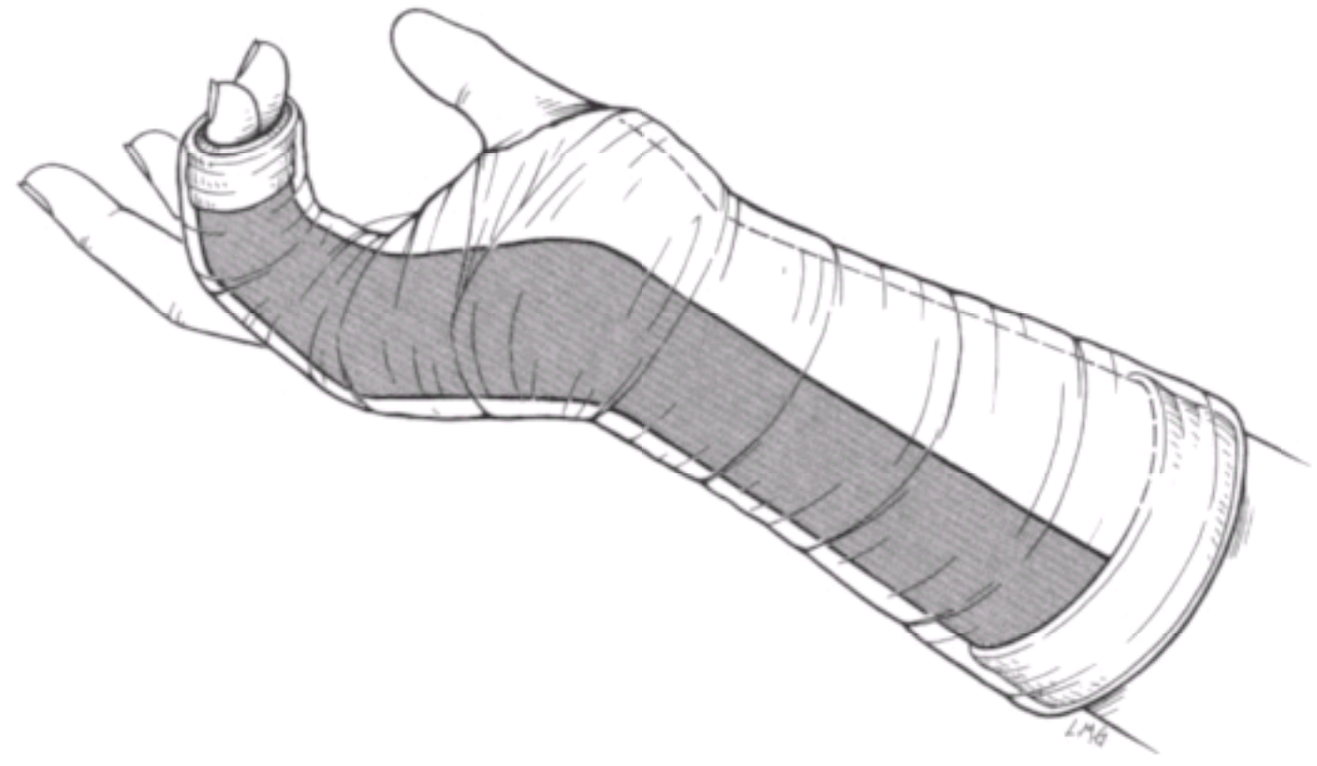


Figure 51-12 Application of a radial gutter splint. The radial gutter splint incorporates both the index and long fingers. Webril or gauze should be placed between the digits to prevent maceration of the skin. The splint runs along the radial aspect of the forearm from just beyond the distal interphalangeal joint of the index finger to the mid-forearm. The forearm is in the neutral position with the wrist in slight extension (10°–20°), the metacarpophalangeal joint in 50° of flexion, and the proximal and distal interphalangeal joint in slight (10°–15°) flexion. When immobilizing a metacarpal neck fracture, the metacarpophalangeal joint should be flexed to 90°.

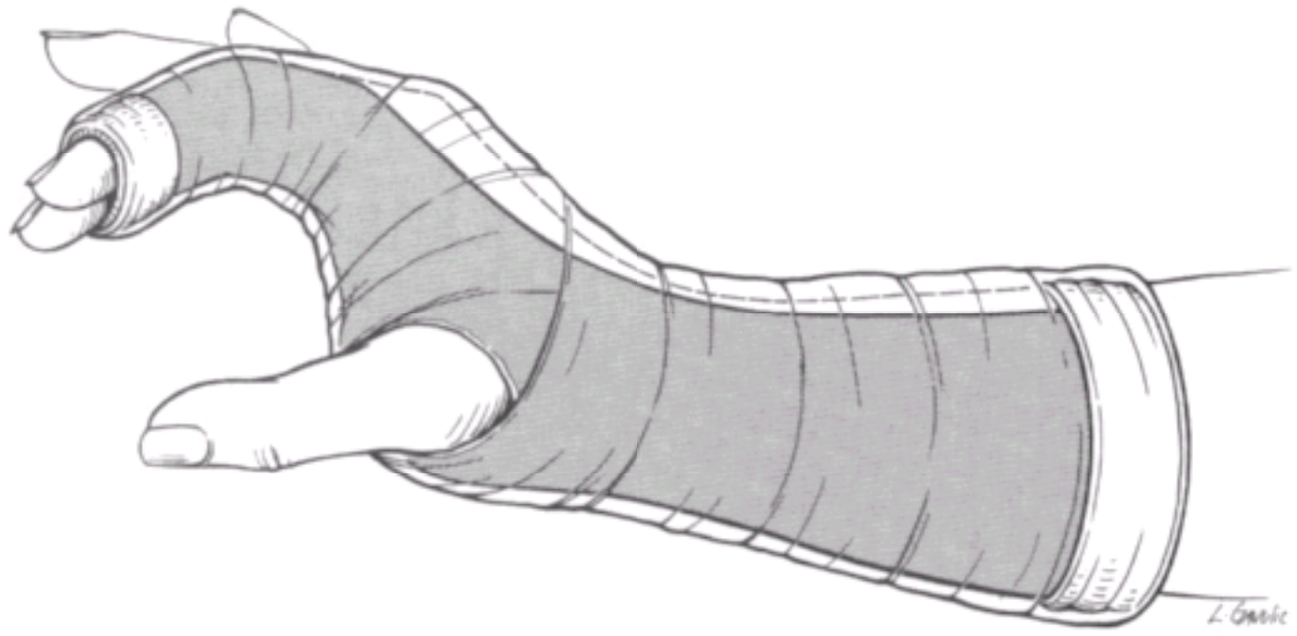


Figure 51-13 "Buddy tape" technique. Taping between the digital joints allows the normal adjacent finger to protect the collateral ligament of its injured neighbor. Webril should be placed between the digits to prevent maceration of the skin.

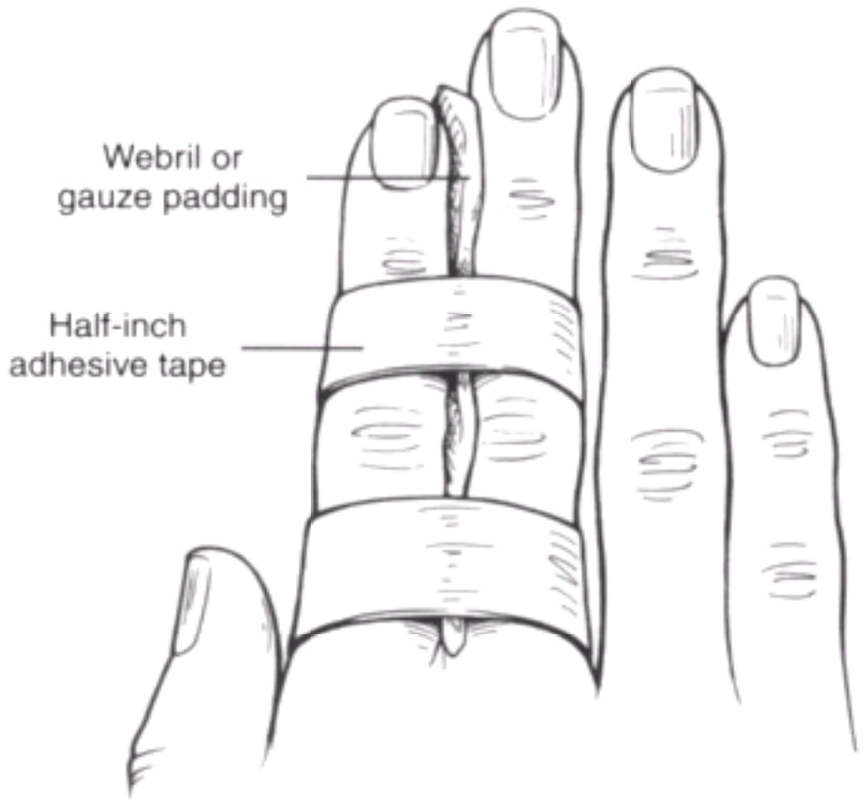


Figure 51-14 Dorsal aluminum foam splint. The bone is subcutaneous dorsally, and splints here afford better immobilization of the digit. The dorsal splint also allows preservation and use of tactile sense, which encourages function and better splint acceptance on the part of the patient.

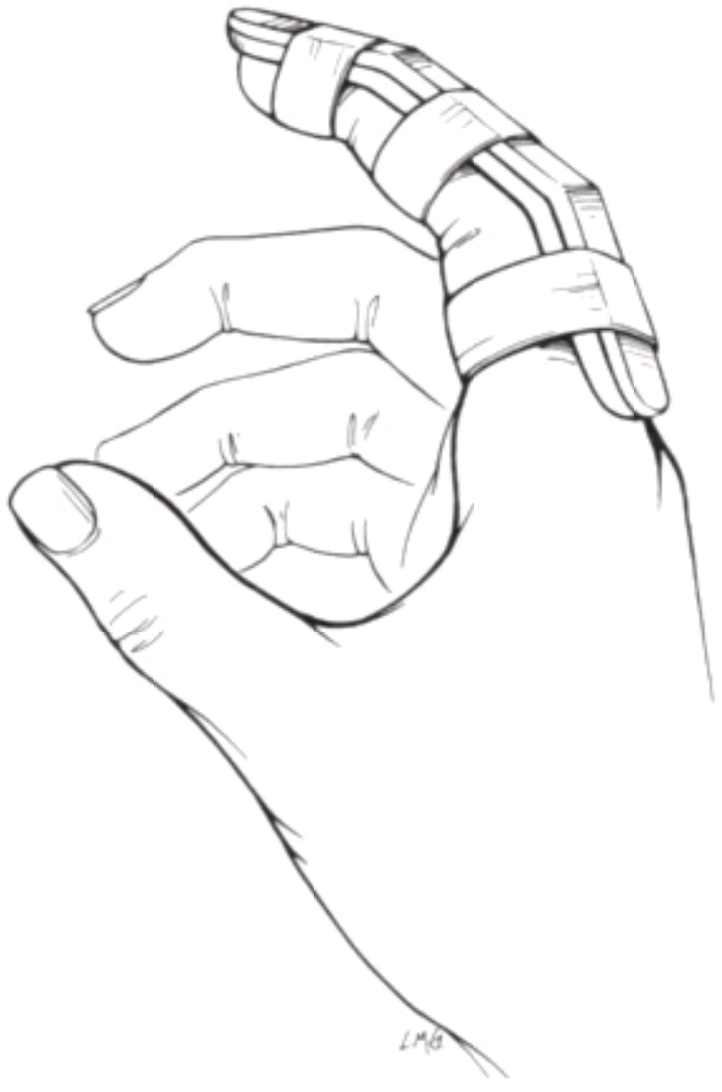


Figure 51-15 Splinting a mallet finger. The dorsal splint immobilizes only the distal interphalangeal joint. This allows use of the finger. Hyperextension of this joint predisposes to skin sloughing and should be avoided. The patient should be advised not to flex the joint during splint changes.

Figure 51-16 "Outrigger" finger splint for complete immobilization of the finger. A padded aluminum splint is incorporated into the middle of a plaster splint, forming an outrigger configuration. The plaster splint is applied to the dorsum of the hand and wrist with an elastic bandage; the finger is then taped to the aluminum splint.

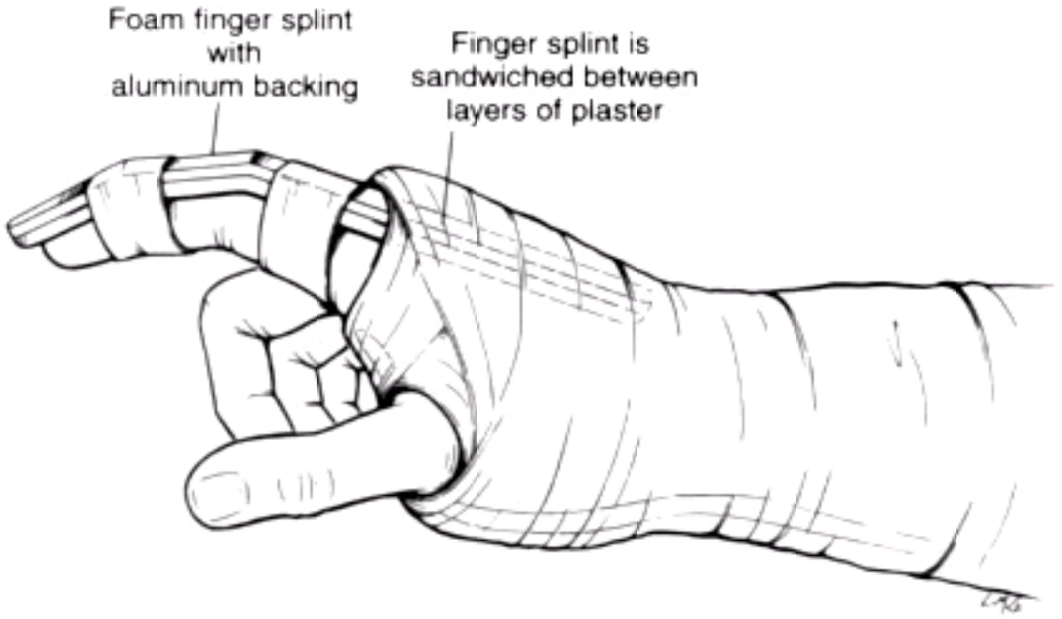


Figure 51-17 *A*, Step-wise application of a triangular muslin sling. (1) Place tip X over the uninjured shoulder. (2) Bring tip Y over the injured shoulder to enclose the arm. (3) Draw tip Z around the front and pin. *B*, Completed triangular muslin sling (Note: When applying a sling, it is important to have adequate support of the wrist and hand. A sling that is too short will allow the wrist and hand to hang down [ulnar deviate] and can result in ulnar nerve injury).

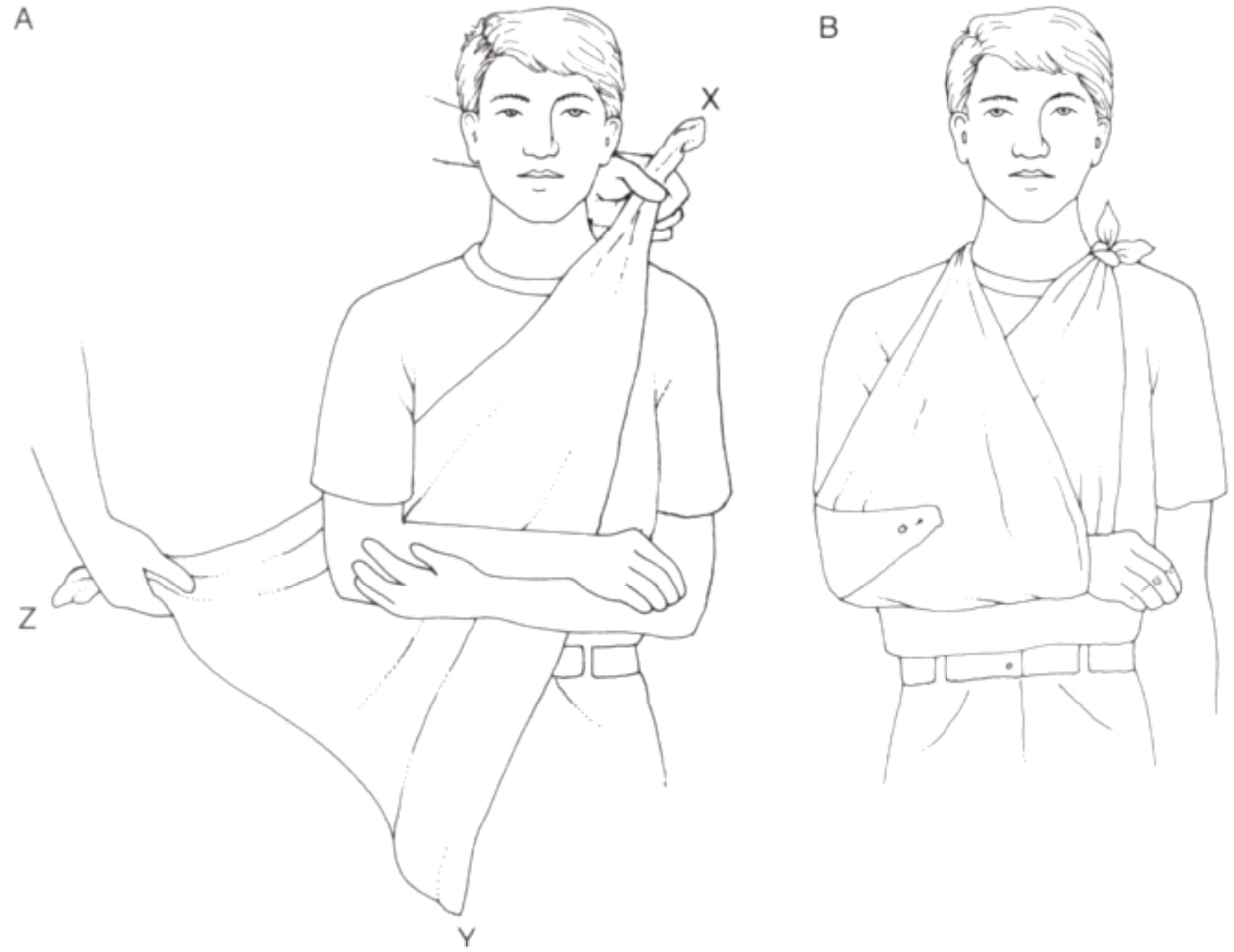


Figure 51-18 The shoulder immobilizer is used for most proximal humerus fractures and shoulder injuries. It may be removed for showering and range-of-motion exercises and is easily reapplied by the patient.

Figure 51-19 The Velcro strap bulky knee immobilizer is easily removed and readily applied by the patient. It can be worn over clothes.

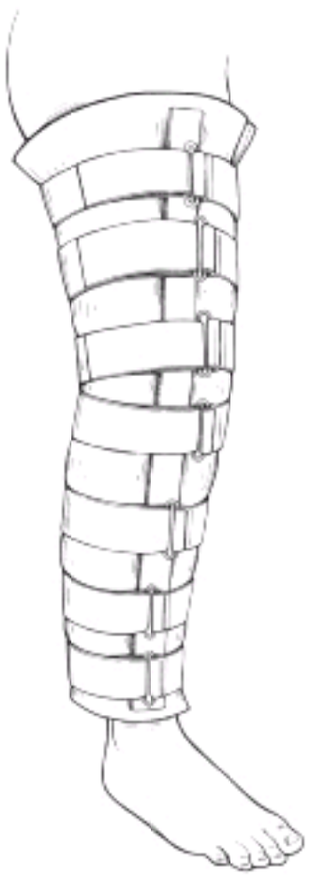


Figure 51-20 Application of a posterior knee splint. *A*, The posterior knee splint runs from just below the buttocks crease to approximately 2 to 3 cm above the malleoli. *B*, Alternatively, two parallel splints can be placed along each side of the leg and foreleg, creating a bivalve effect.

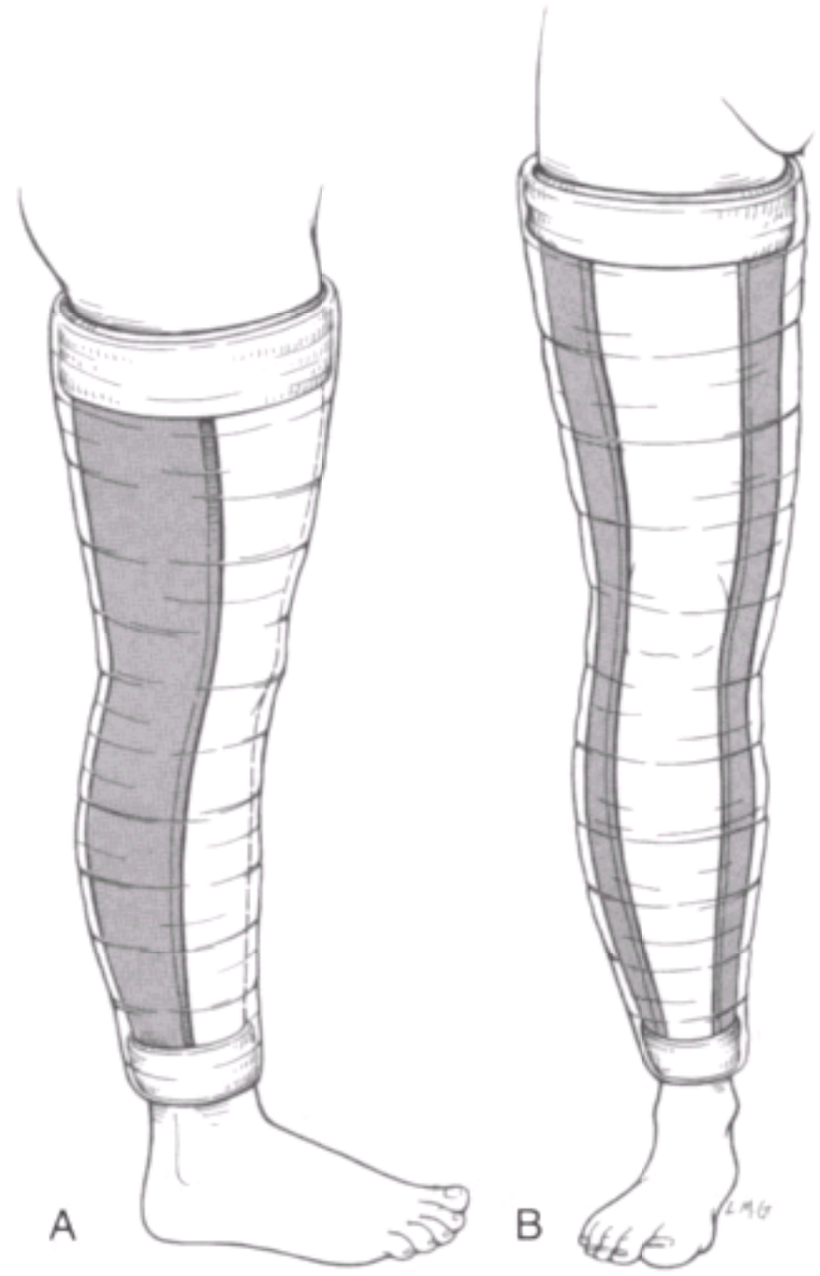
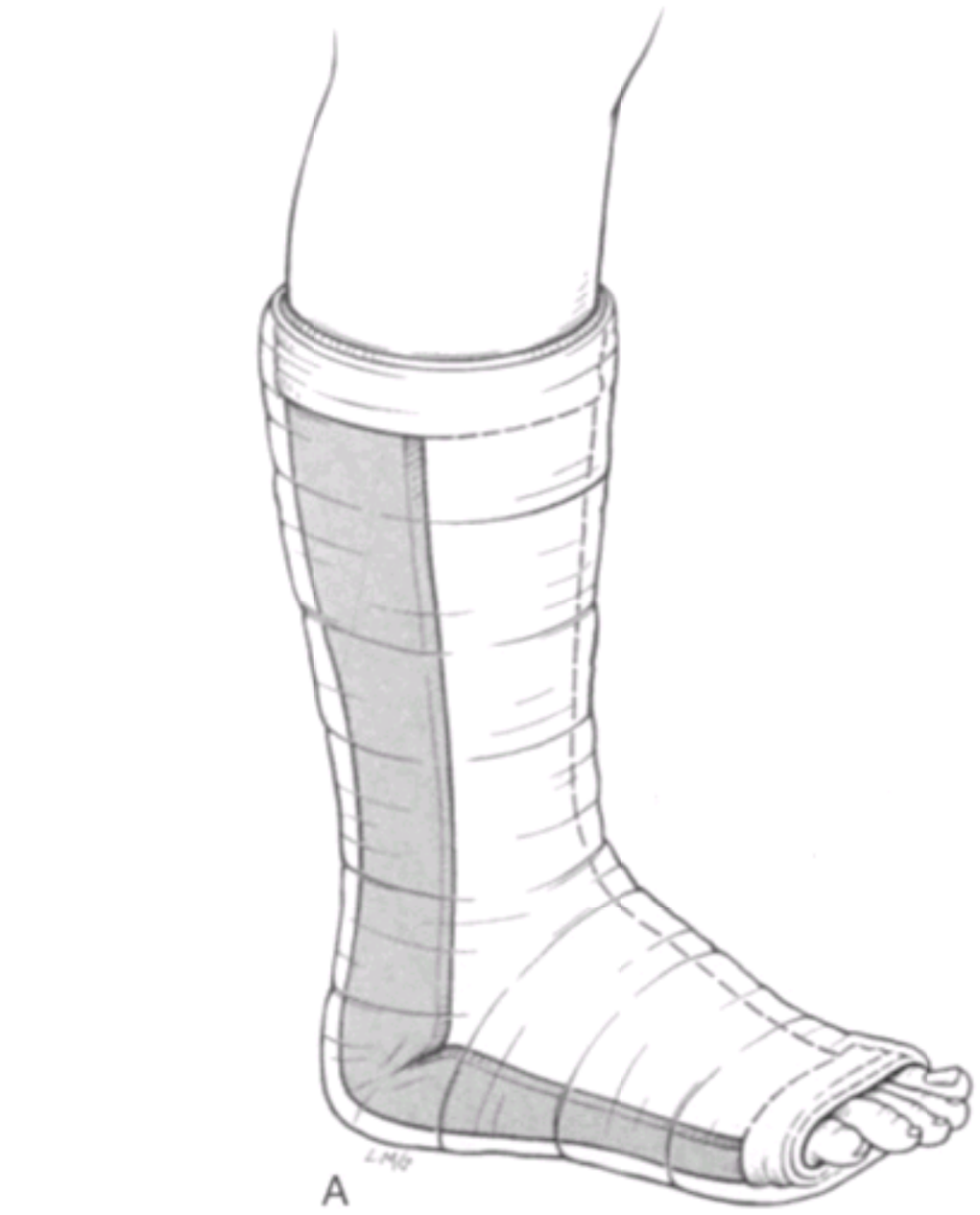
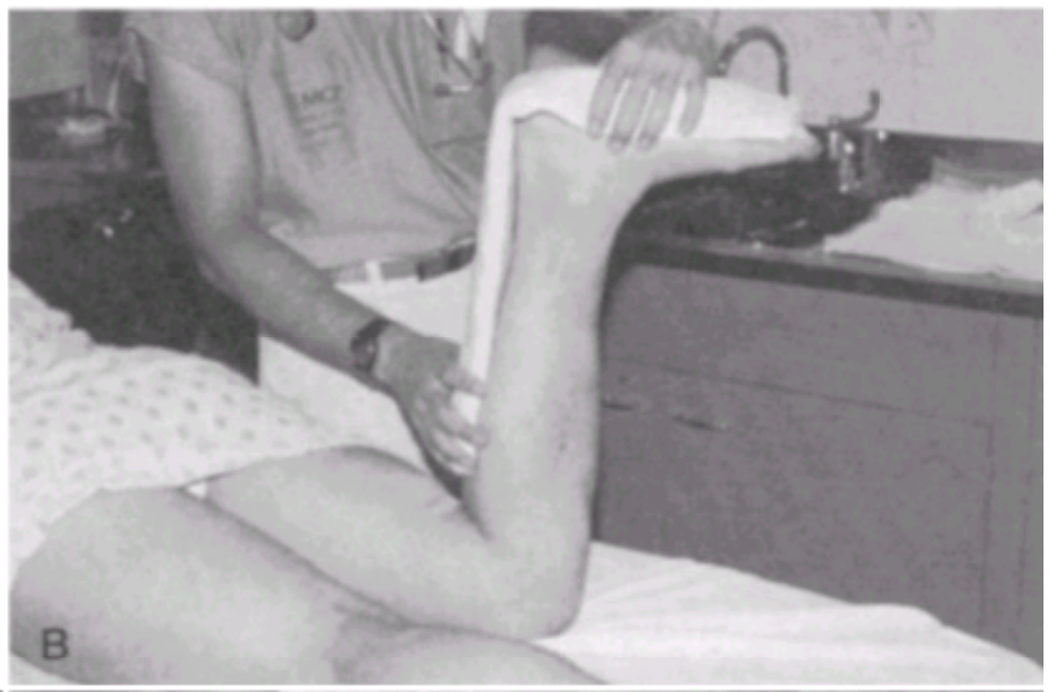
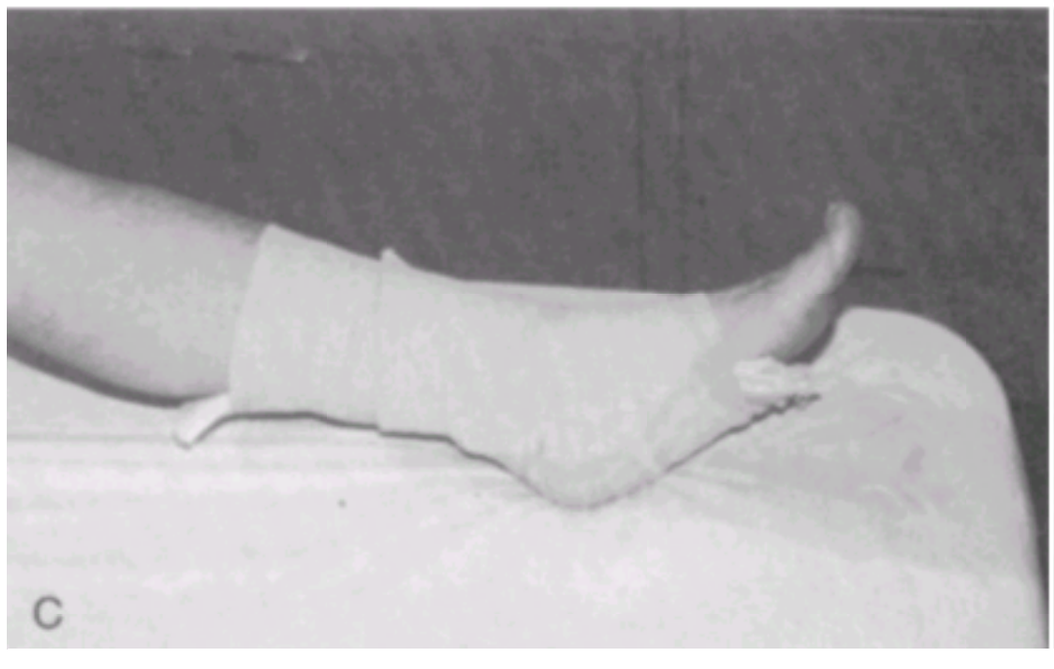


Figure 51-21 Posterior ankle pitfalls. *A*, Proper application of a posterior ankle splint. This splint extends from the plantar surface of the great toe (or metatarsal heads) along the posterior surface of the foreleg to the level of the fibular head. The ankle should be at a 90° angle. *B,* The most convenient way to apply an ankle splint is to have the patient lie prone and bend the knee to a 90° angle, thereby relaxing the calf muscles. The ankle should be at a 90° angle so that the foot is flat for partial weight bearing. *C, Incorrect splint application.* There are three things wrong with this posterior ankle splint: (1) It does not extend distally enough to support the entire foot. (2) The ankle is not maintained at a 90° angle. (3) The edges and ankle area are not molded. Overall the splint is sloppy and ineffective. *D*, The problem with this splint is that it was intended to be used for only a few days, but the patient wore it for 3 weeks. Note the resultant full-thickness skin loss. No padding was used under the premade splint. Skin grafting was eventually required.







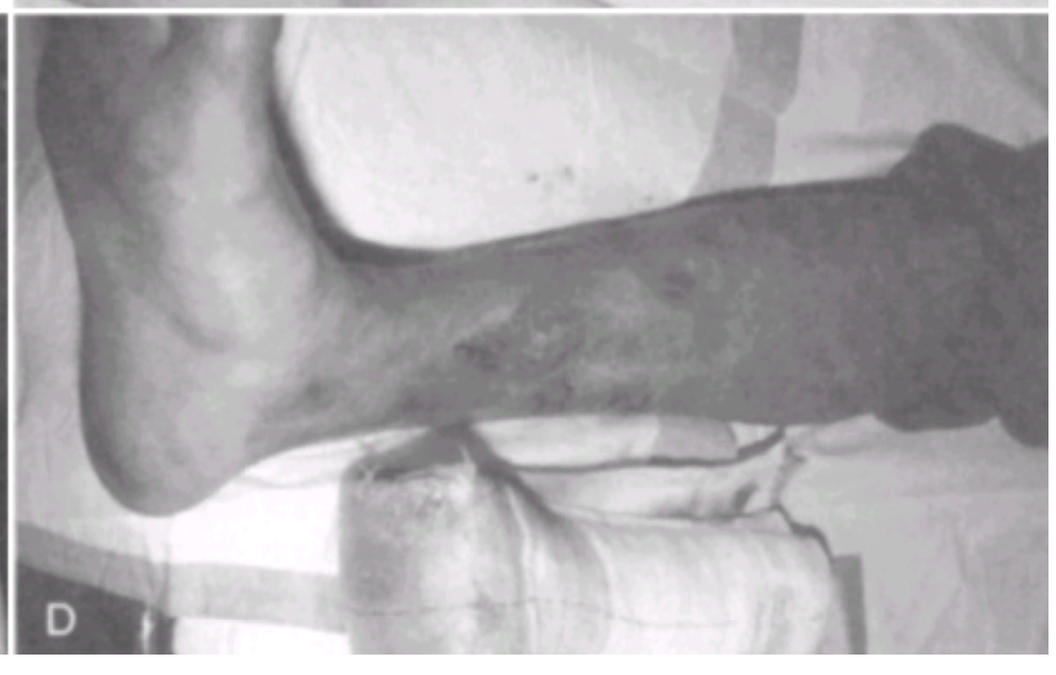


Figure 51-22 Application of an anterior-posterior ankle splint. In particularly severe or unstable injuries, an anterior splint may be added to a posterior ankle splint to provide extra immobilization resembling that of a formal cast. The anterior splint is never used by itself, but it can augment a posterior splint, creating a bivalve effect. The ankle should be at a 90° angle.

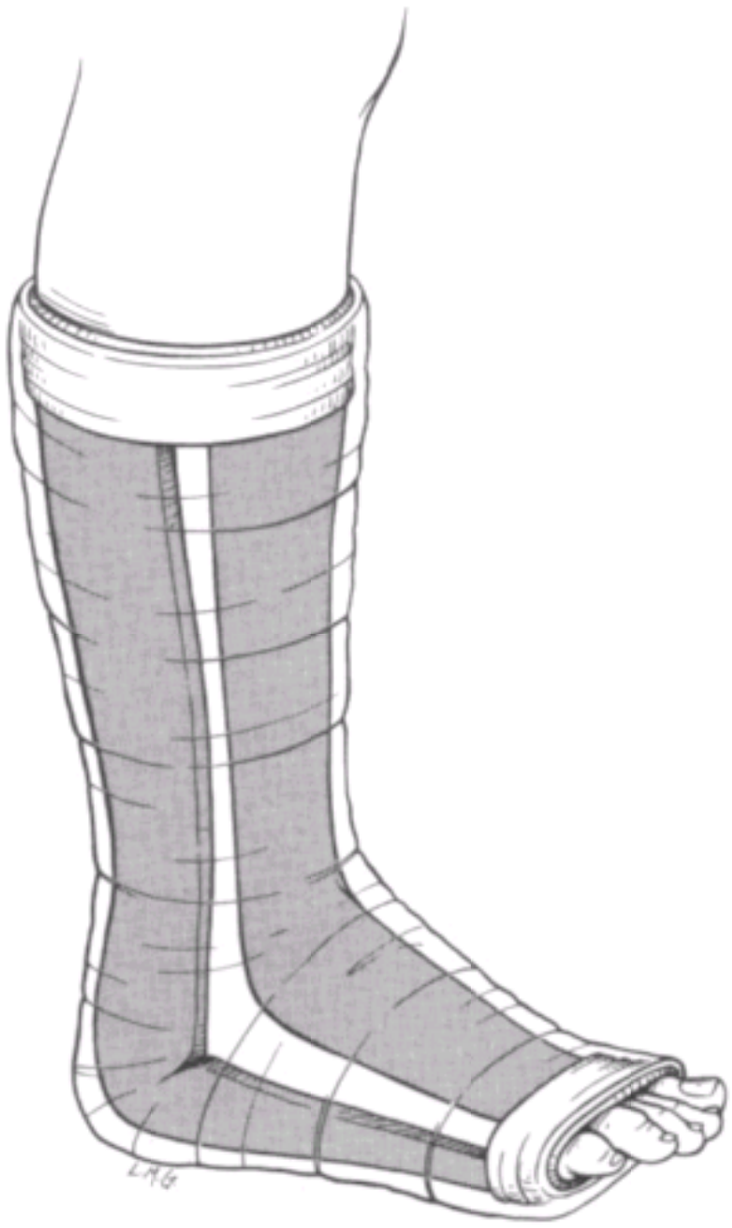


Figure 51-23 The U-splint (AKA sugar-tong or stirrup splint) is also used primarily for injuries to the ankle. The splint passes under the plantar surface of the foot, extending up the medial and lateral sides of the foreleg to just below the level of the fibular head. The ankle should be at a 90° angle. For immobilization of the knee, the sides of the splint may be extended proximally to the groin, creating a long leg splint.

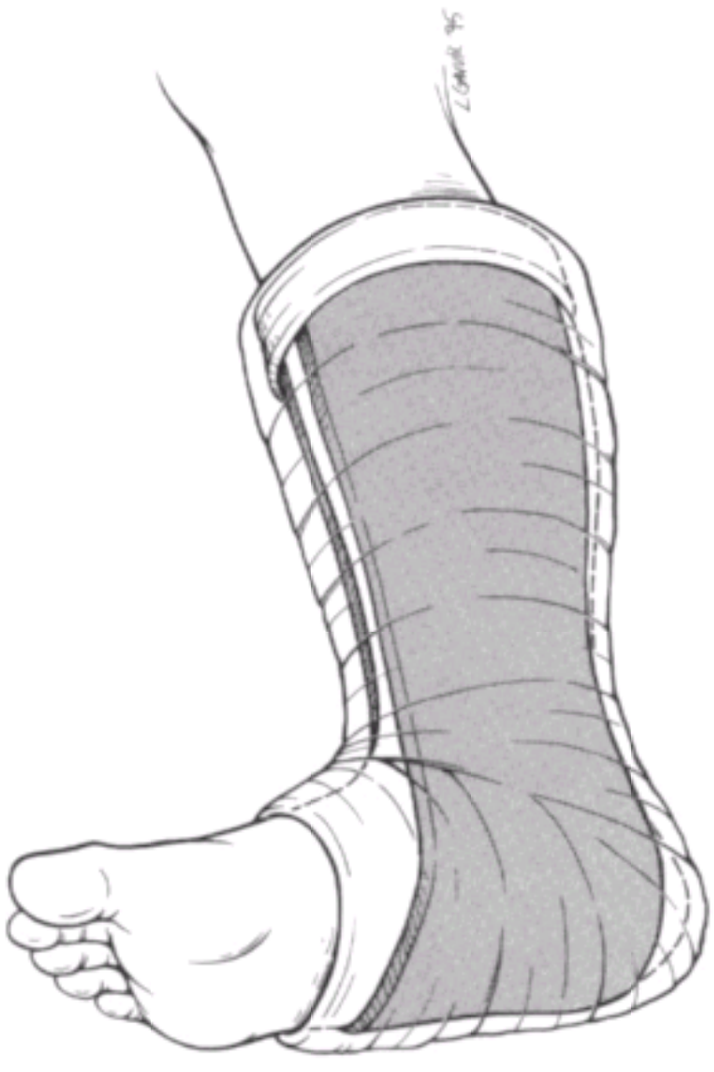


Figure 51-24 The Unna boot provides effective immobilization of an ankle soft tissue injury. It is applied from a semisolid paste roll. The wrap is then covered with gauze or an elastic bandage. The entire dressing can be cut off by the patient at home. For similar short-term immobilization without plaster, a modified Jones dressing can be used. Webril is wrapped around the ankle and it is covered with an elastic bandage.

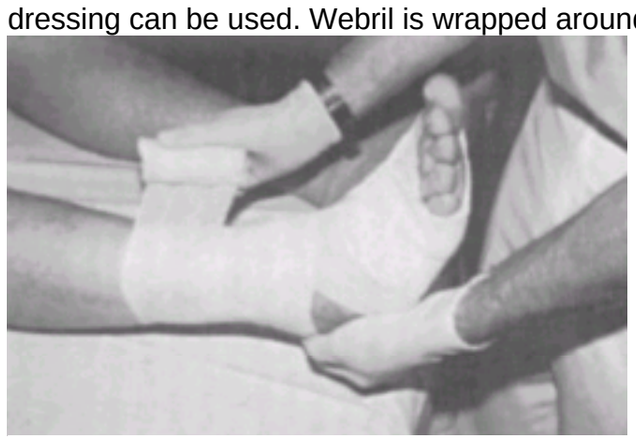
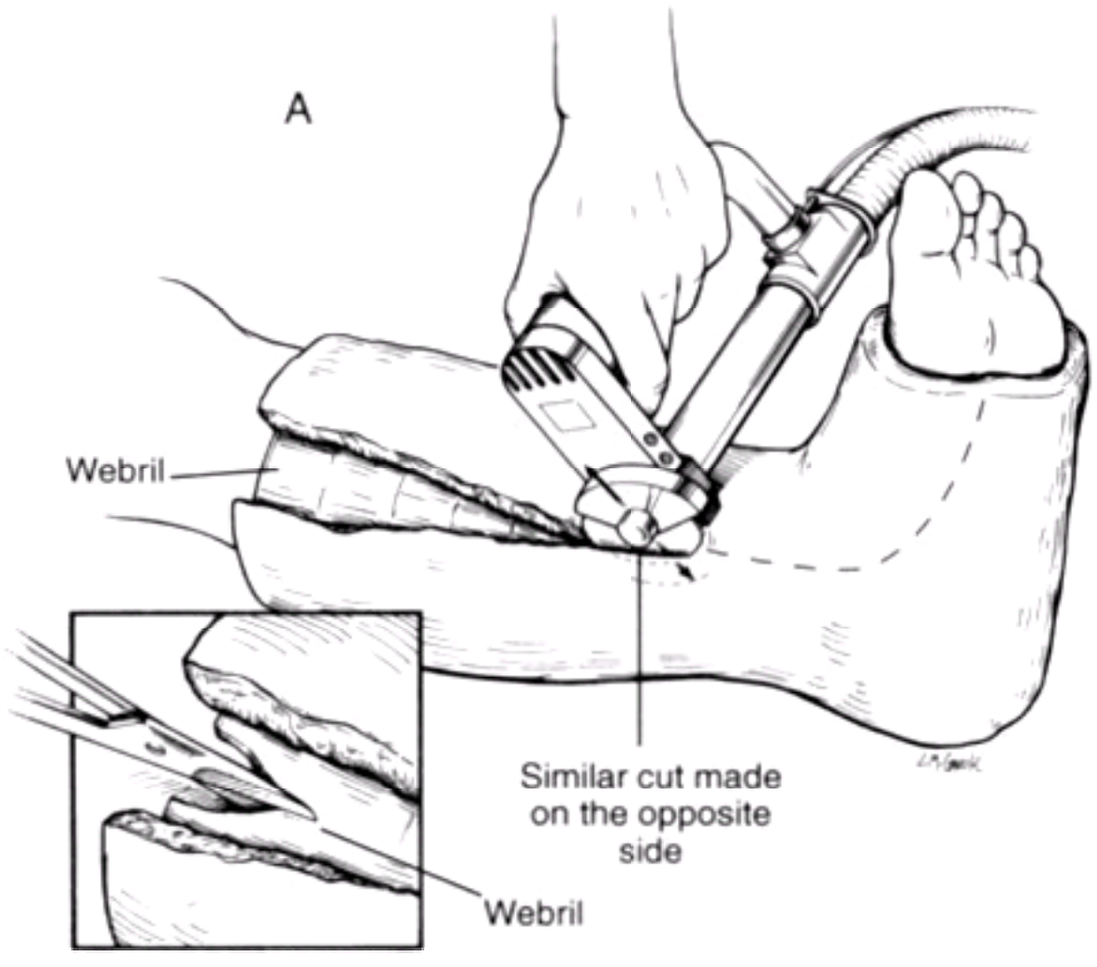


Figure 51-25 *A,* The cast saw vibrates; it does not rotate. The blade is controlled by placing the thumb on the splint and lowering the saw to the plaster. The blade is raised and lowered for each cut; it is *not* drawn across the plaster like a knife. *B,* This cast was too tight, and it was therefore bivalved from cast to forefoot with a cast saw. After separation of the edges of the cut cast, the anterior and posterior components were secured in place with an elastic bandage. Note that the underlying Webril padding was cut to relieve pressure but was not removed *(inset).* A bivalved cast provides temporary immobilization equal to that of an intact cast.



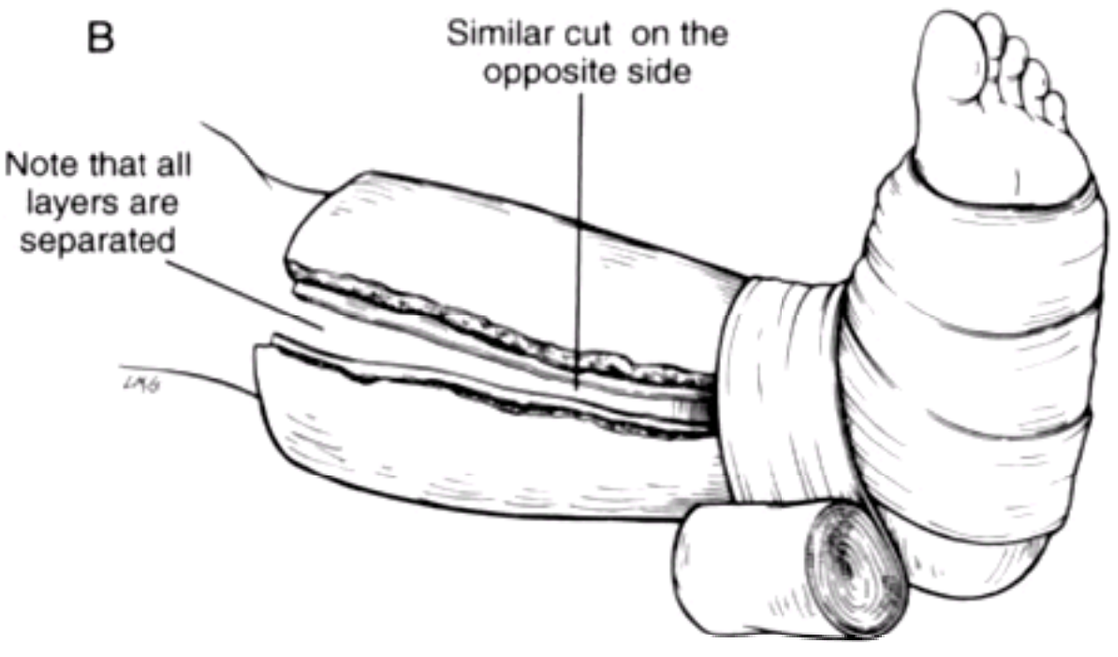


Figure 52-1 Use of aperture pads to redistribute pressure from painful areas to surrounding structures. *(Courtesy of Kenneth R. Walker, DPM.)*

Figure 52-2 Use of a self-adherent bunion pad. *(Courtesy of Kenneth R. Walker, DPM.)*

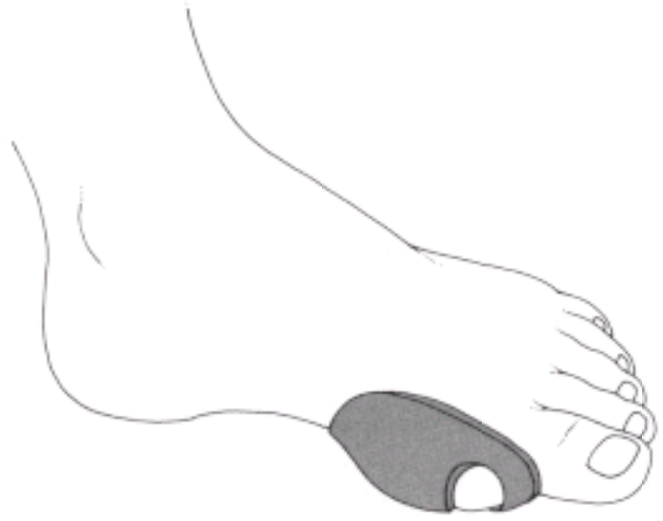


Figure 52-3 "Buddy taping" of a fractured lesser toe. *A,* a pad is placed between the injured toe and an adjacent toe. *B,* the toes are secured together with tape or self-adherent wrap. *(Courtesy of Kenneth R. Walker, DPM.)*

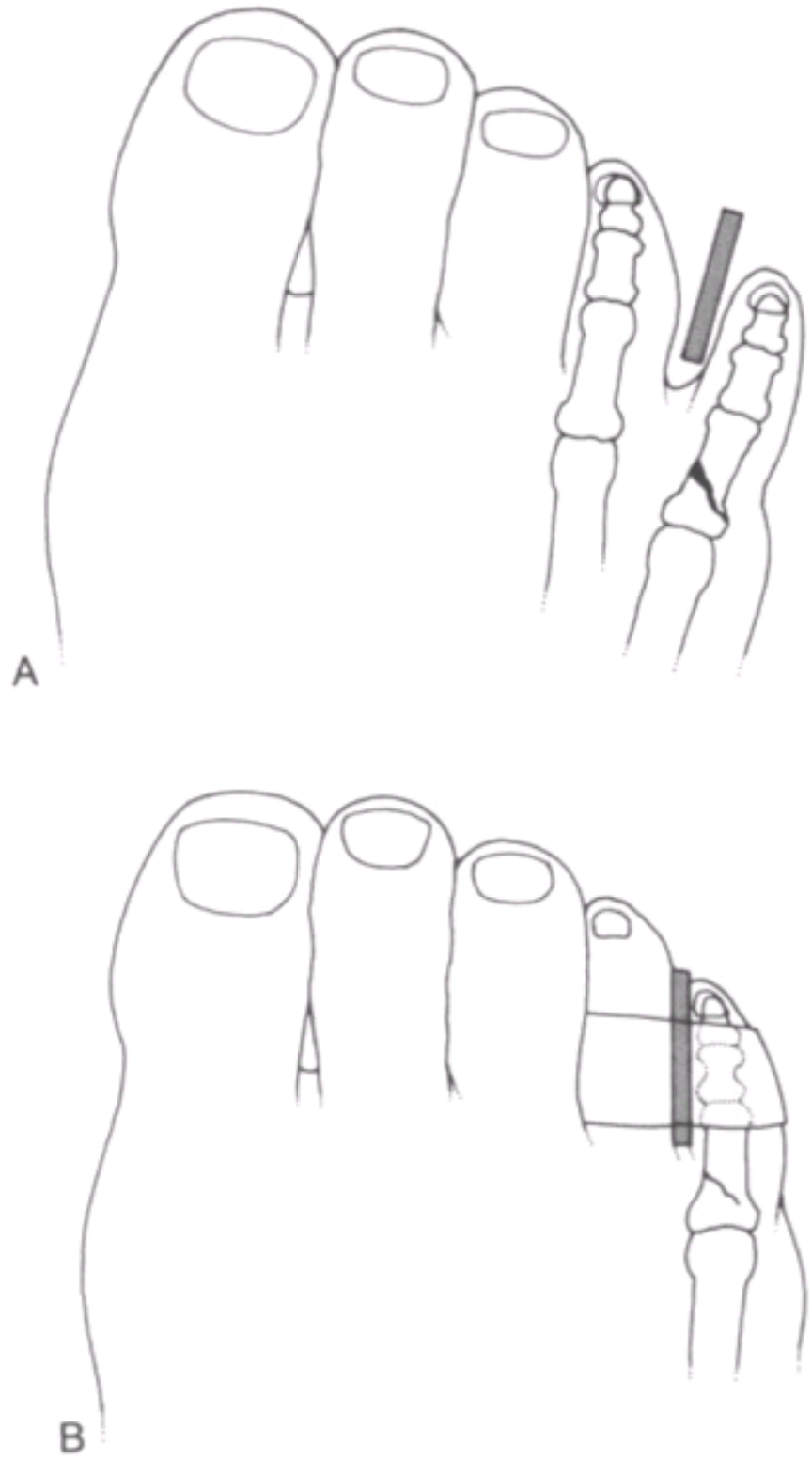


Figure 52-4 Bunion shields redistribute pressure away from fractured sesamoid bones. *(Courtesy of Kenneth R. Walker, DPM.)*

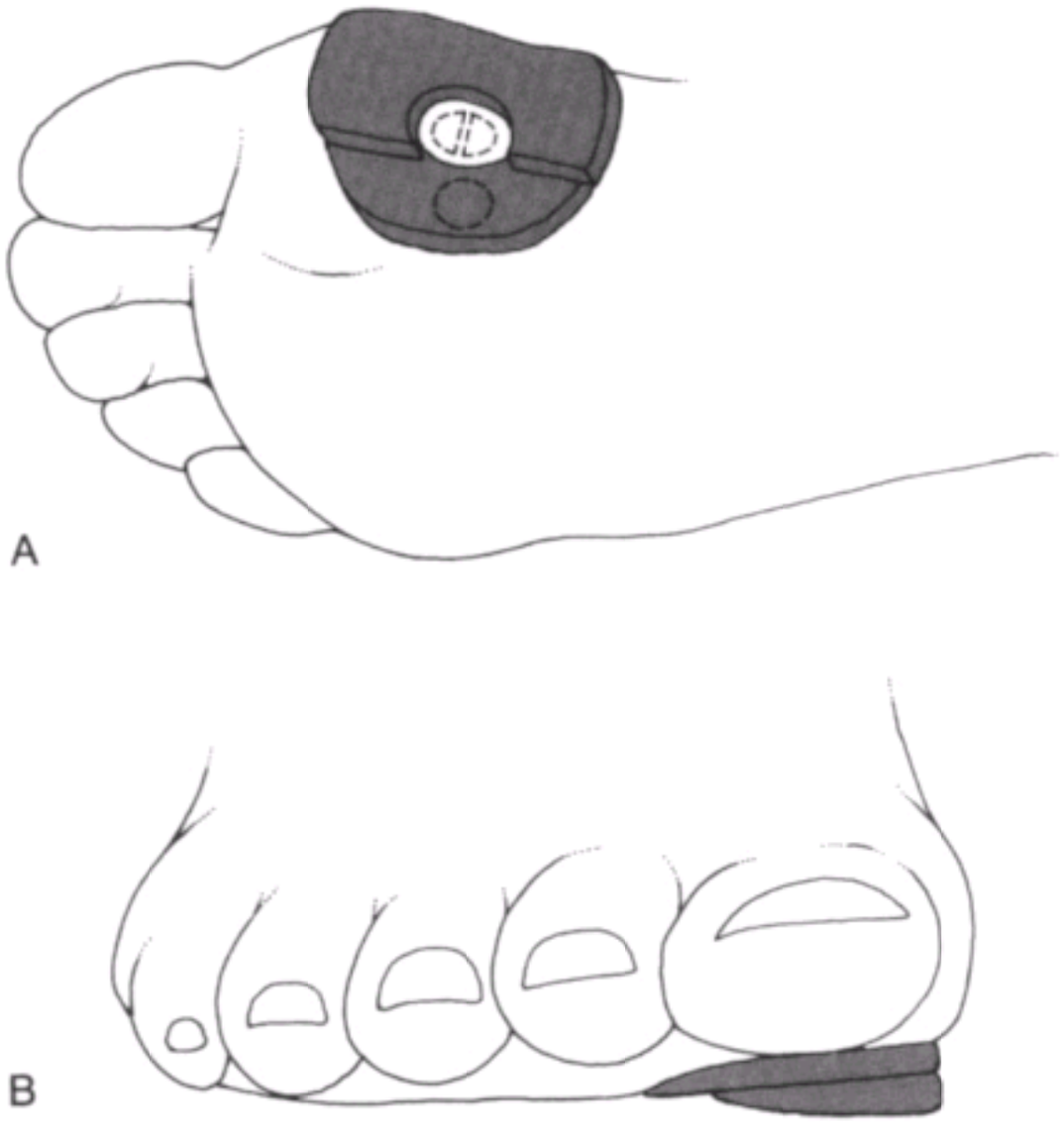


Figure 52-5 *A,* An ingrown toenail of this degree requires removal of a portion of the nail and debridement of inflamed tissue. *B,* Two days later, there is marked improvement. *C,* To remove a toenail, small scissors are held parallel to the nail bed, advanced, and spread. This will produce the least damage to the nail bed.

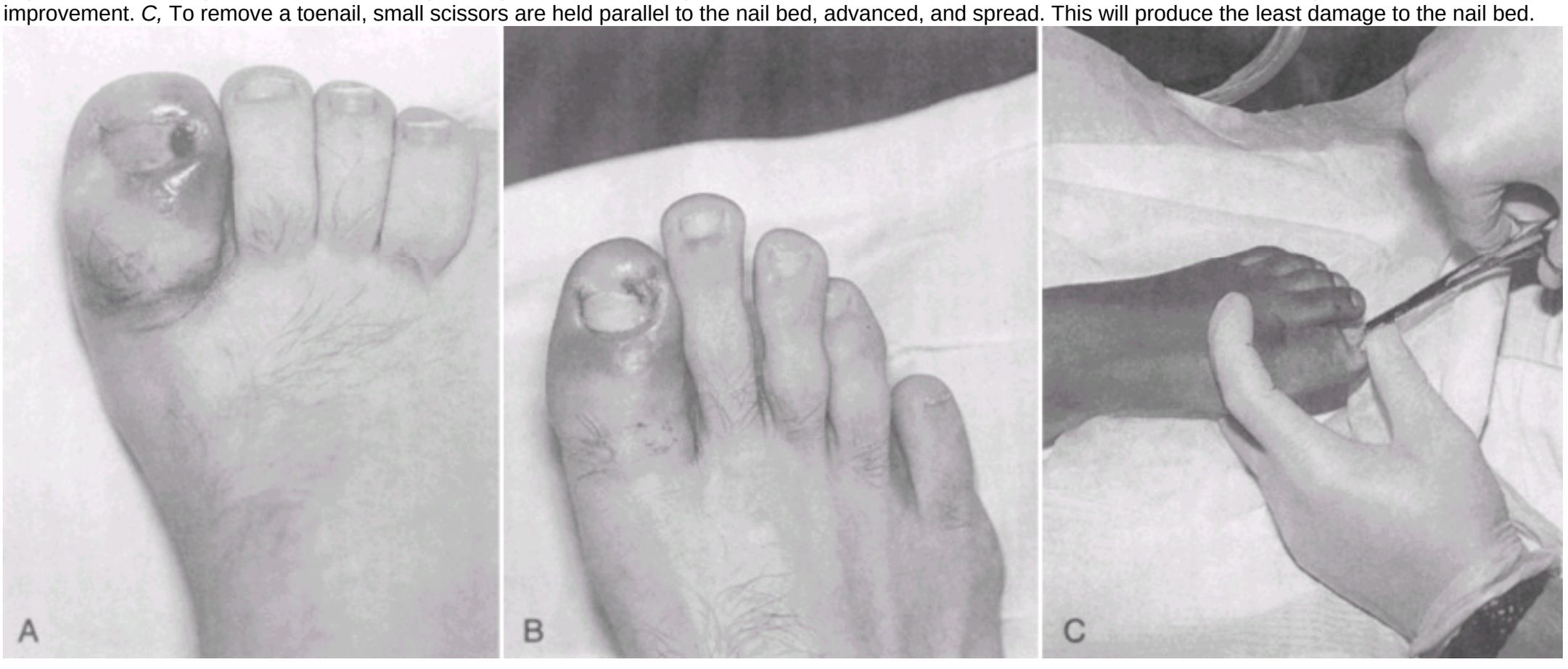


Figure 52-6 *A,* The best way to examine a puncture wound of the foot is to place the patient prone on a stretcher and obtain good lighting and a bloodless field (in this case, a blood pressure cuff was applied to the lower leg). *B,* When the overlying skin was unroofed, a small piece of rubber from the patient's sneaker was found imbedded in the wound. It was removed by a coring technique and packed.

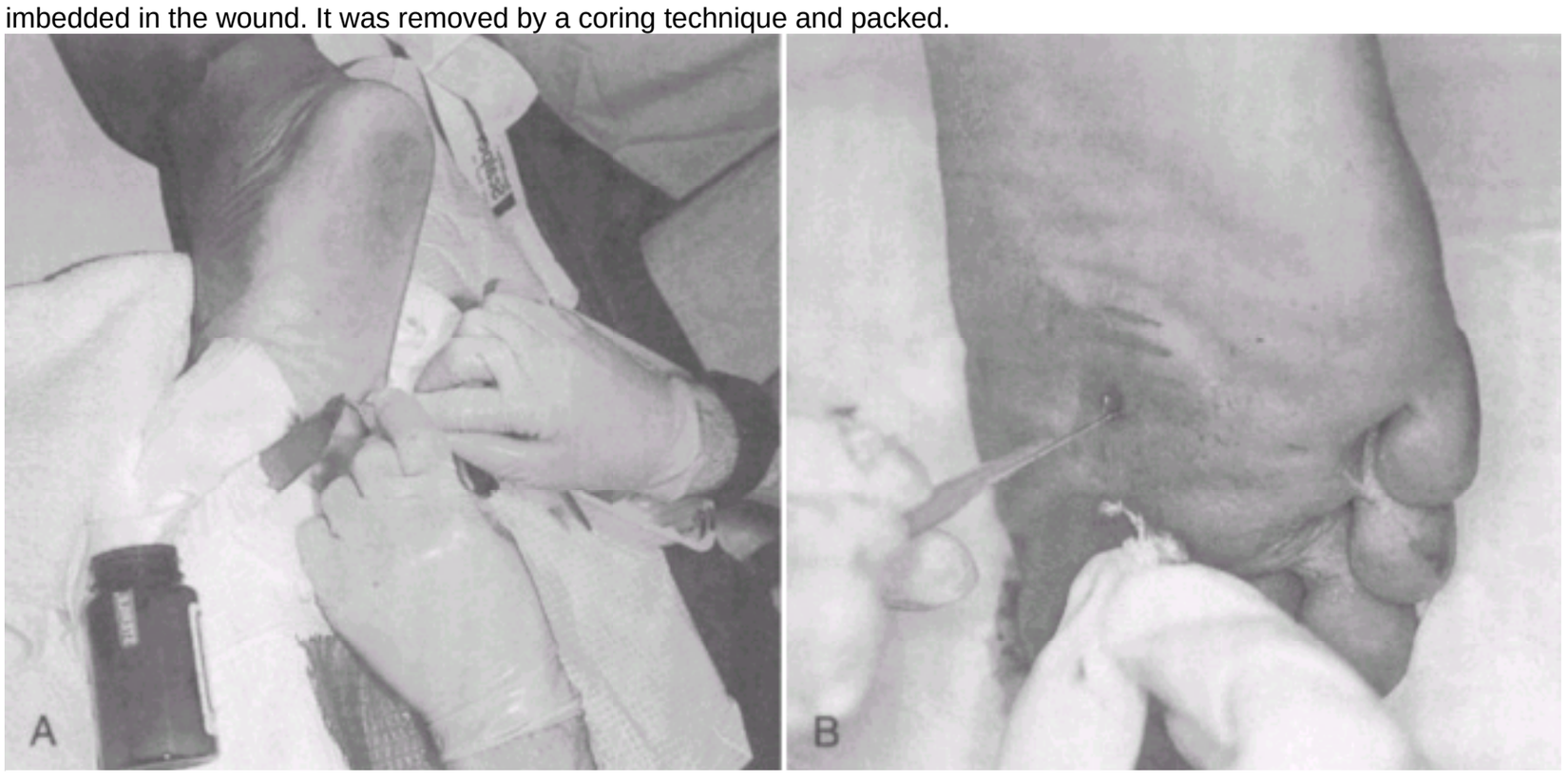


Figure 52-7 *A,* Patients who present with lymphangitis, obvious cellulitis, or both within a few days of stepping on a nail usually have a gram-positive soft tissue infection. *B,* In this case, the puncture wound was excised by a coring technique, yielding a few bits of foreign material. A pack was placed in the wound, oral antibiotics were given, and the patient did well.

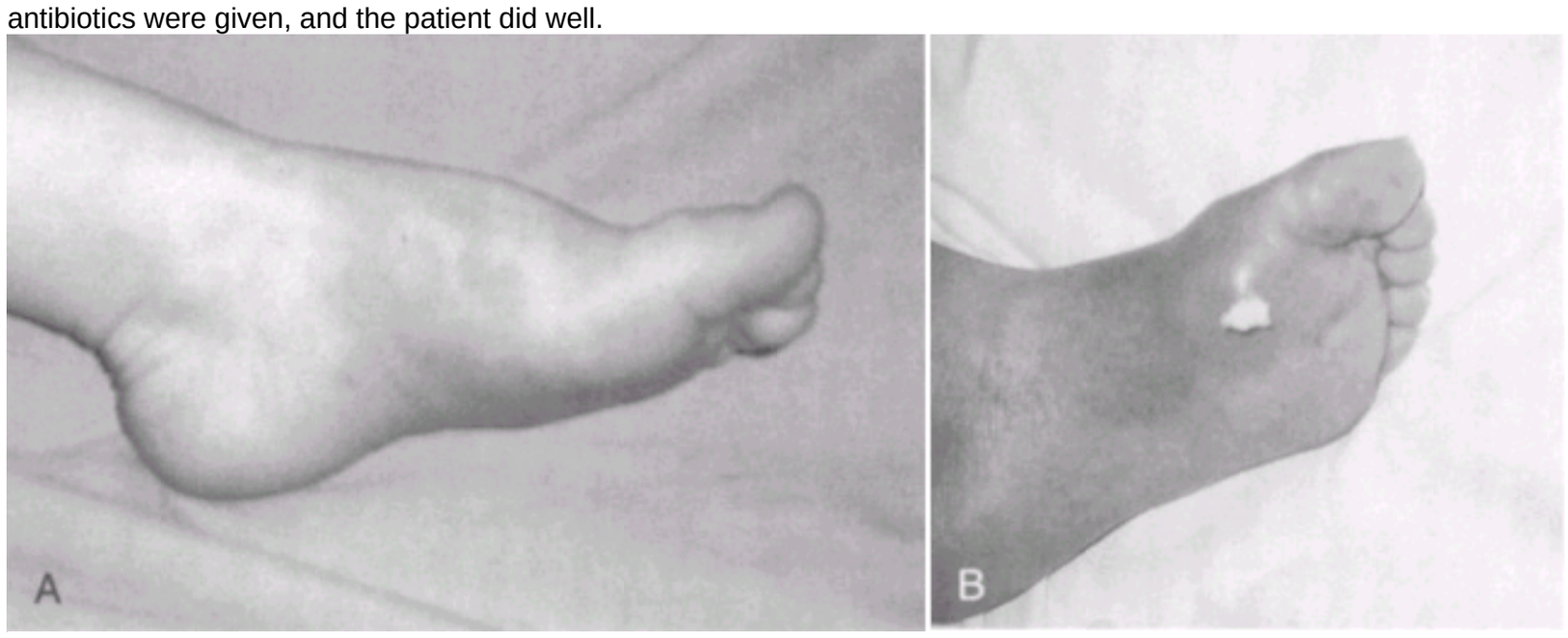


Figure 52-8 This 4-day-old inflamed puncture wound tract likely harbors a foreign body. Excision of a core of the tract found a few pieces of the patient's sock embedded in the wound.

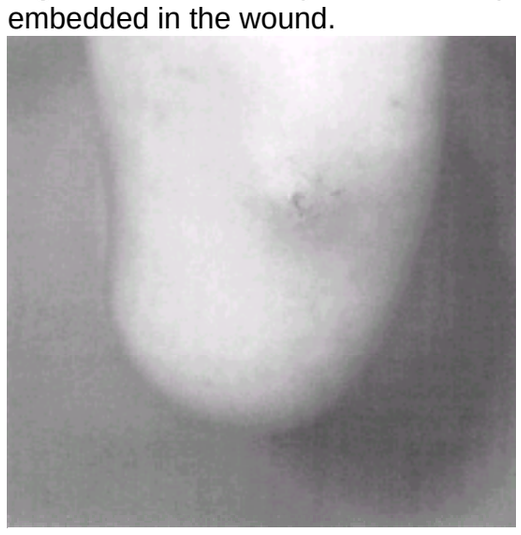


Figure 52-9 A patient stepped on a nail while wearing this shoe. An initial ED evaluation 3 weeks previous found no foreign body or infection. A week previous, the patient began taking antibiotics, but did not improve. The physical examination was quite benign, but the continued aching pain and minimal swelling suggested a deep infection. The complete blood count, sedimentation rate, and plain film were negative. A magnetic resonance imaging scan demonstrated osteomyelitis. *Pseudomonas* is often the offending organism in this scenario.

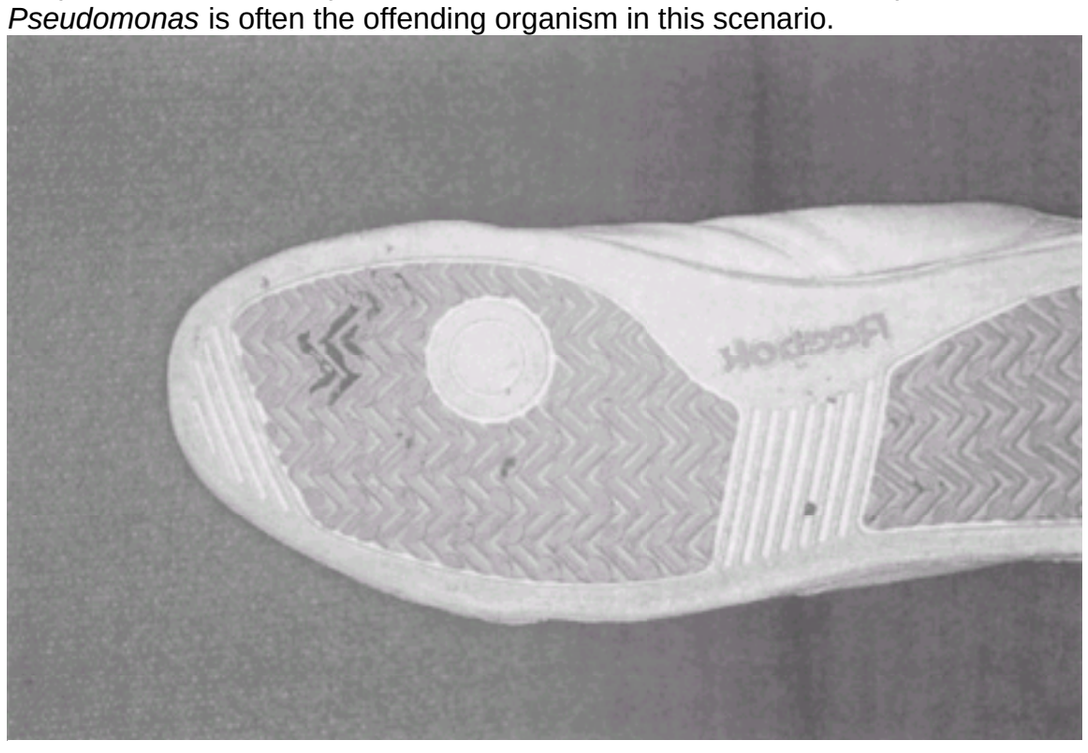


Figure 52-10 Plantar puncture wound management. *A,* Management within 24 hours of wounding. *B,* Management after 72 hours. *(From Chisolm CD, Schlesser JF: Plantar puncture wounds: Controversies and treatment recommendations. Ann Emerg Med 18:1352, 1989.)*

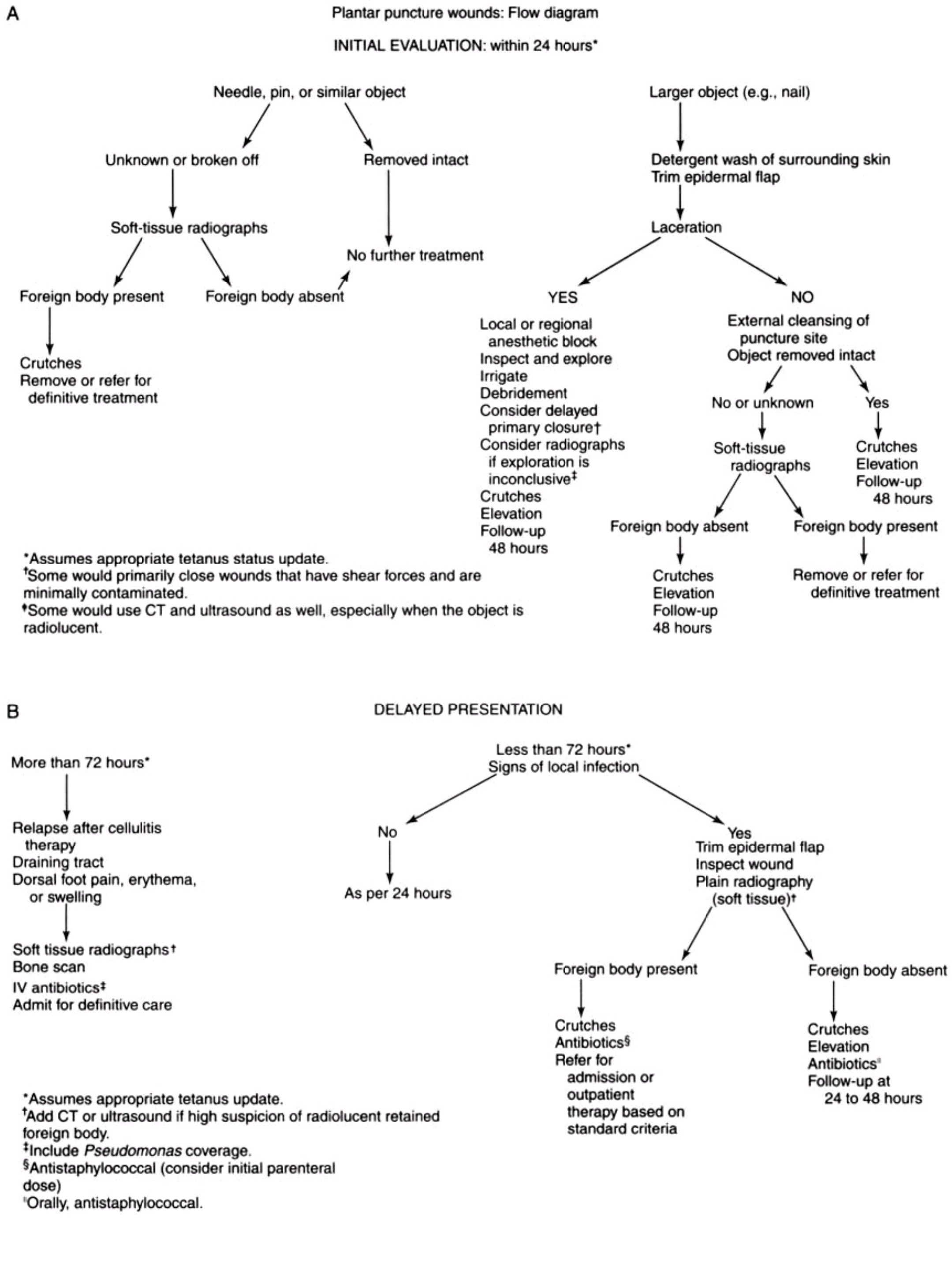


Figure 52-11 Pathology of an ingrown toenail. The normal free space at the nail margin is obliterated by inflammation and granulation tissue, which is caused by improper nail trimming, trauma to the matrix, and faulty footwear. *(From Hill GL II: Outpatient Surgery, 3rd ed. Philadelphia, WB Saunders, 1988. Reproduced by permission.)*

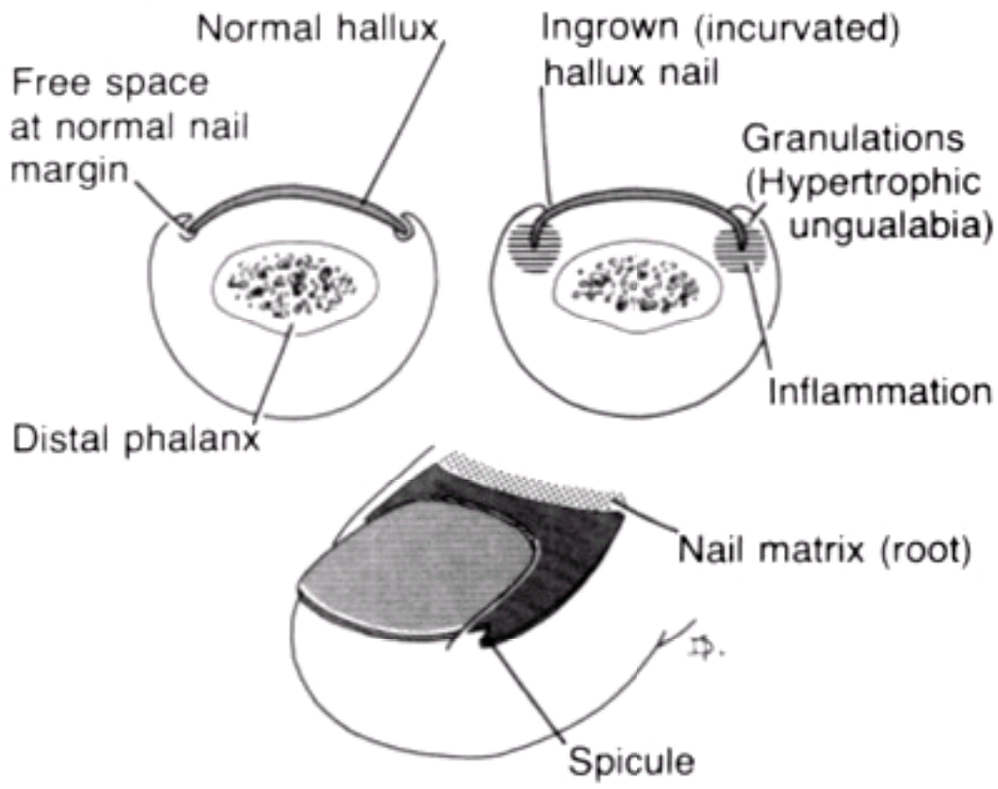


Figure 52-12 An oblique wedge of nail is trimmed from the lateral margin of the nail to free it from the hyperkeratotic area. *(Courtesy of Kenneth R. Walker, DPM.)*

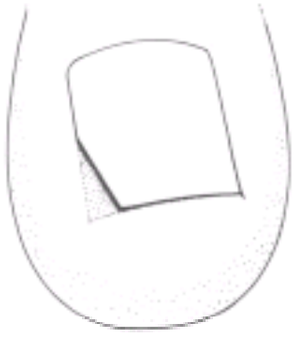


Figure 52-13 English nail anvil used to divide the nail. *(Courtesy of Gill Podiatry Supply Company, Middleburg Heights, OH.)*

Figure 52-14 The nail ablation technique for treatment of an ingrown toenail. The lateral portion of the nail is cut and removed (*A*), exposing the nailbed. Granulation tissue is curetted (*B* and *C*) and the nail matrix is cauterized with absolute phenol (*D*) (see text).

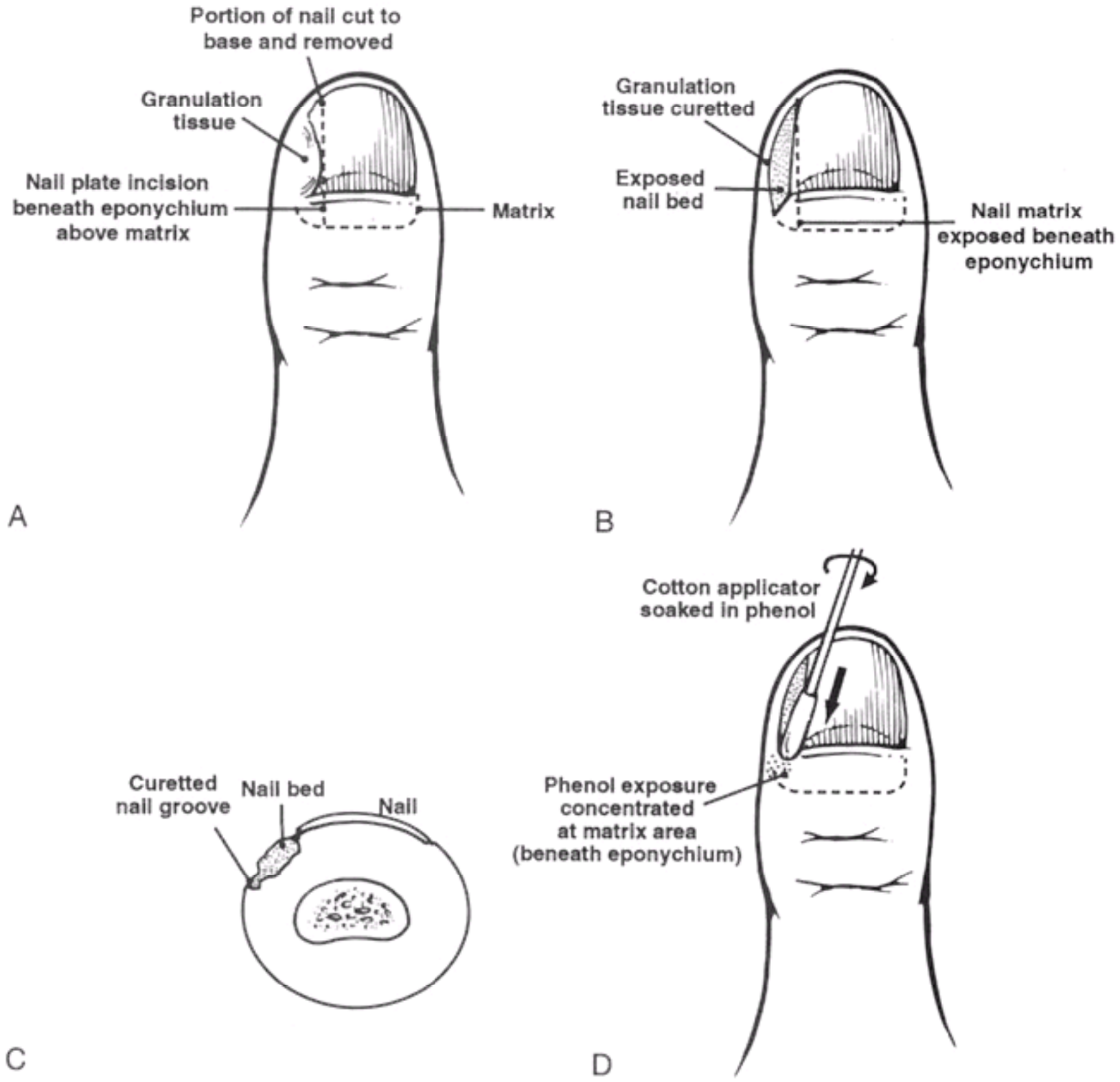


Figure 53-4 *A,* Anatomic site for injection into the bicipital groove for treating bicipital tendinitis. The anesthetic is infiltrated around the biceps tendon sheath in a fan-wise distribution, avoiding intratendinous injection. *B,* Actual peritendon injection of bicipital tendinitis: The patient is seated with the forearm and hand resting in the lap and externally rotated.

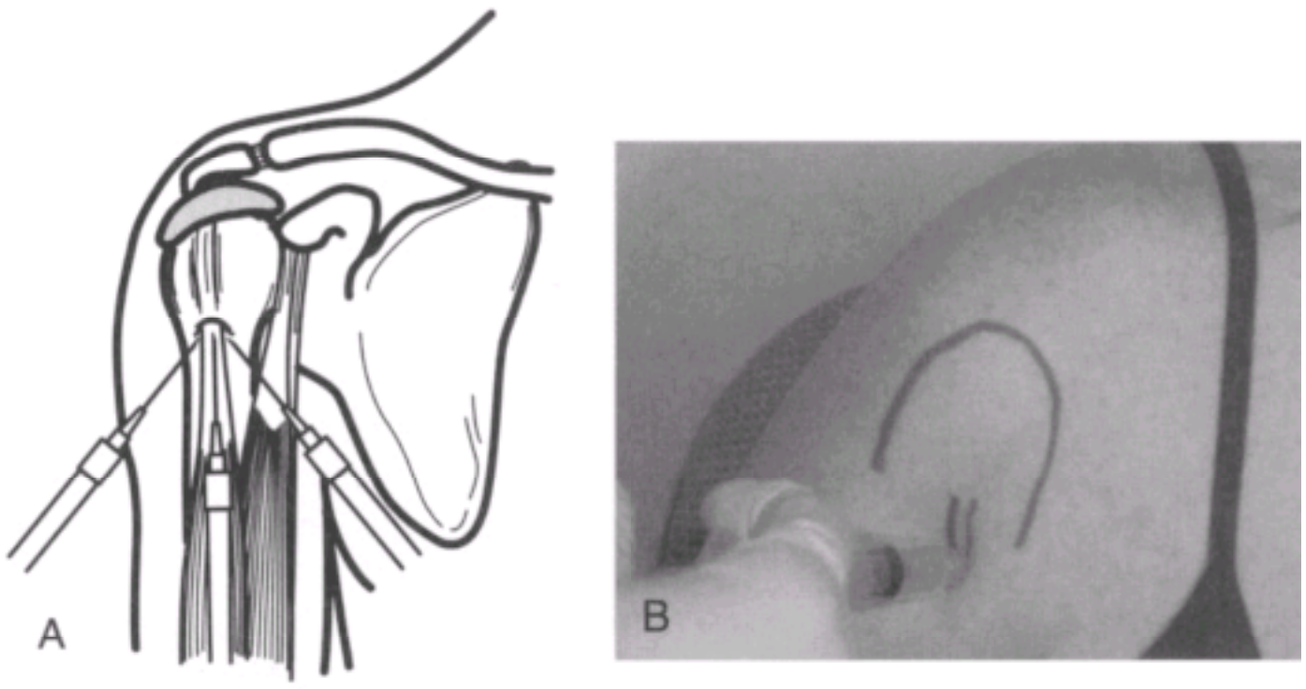


Figure 53-5 The "critical zone." Note the close relationship of the supraspinatus tendon and subacromial bursa to the humeral head and acromion.

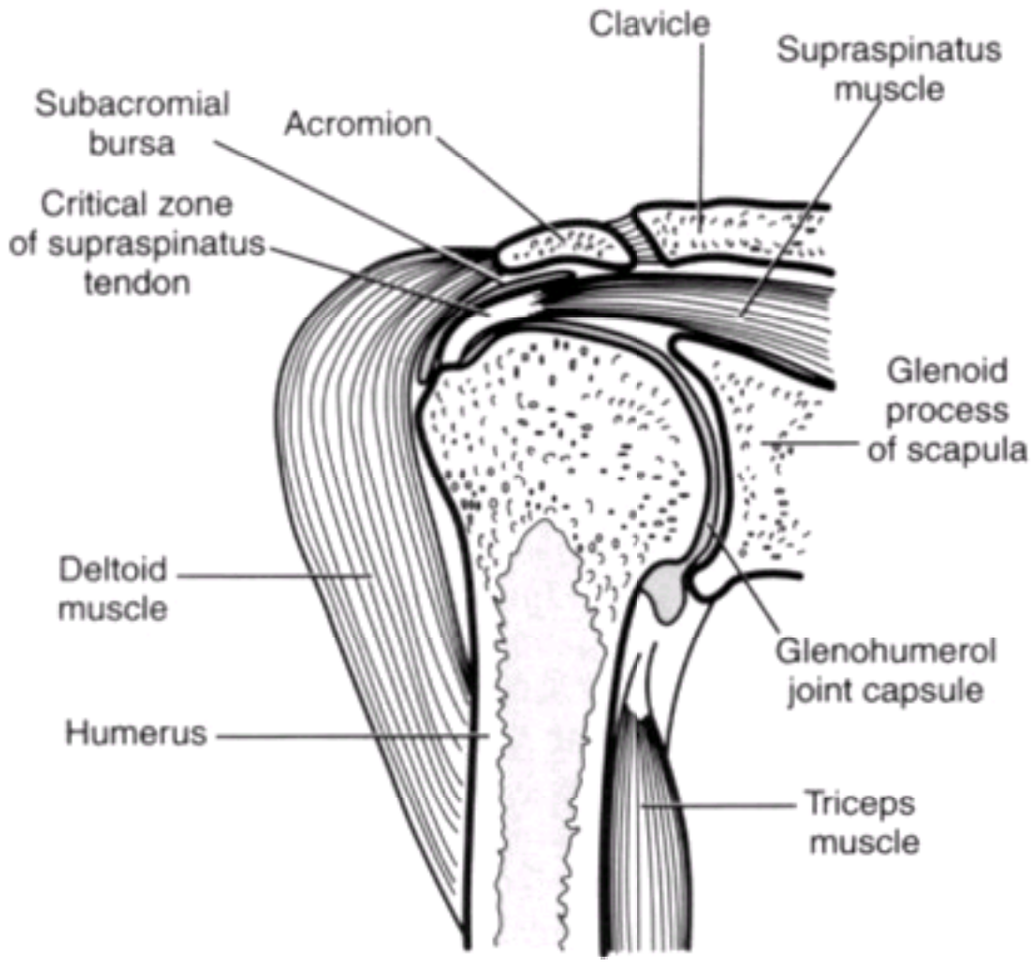


Figure 53-6 Abnormal calcific deposits in calcareous tendinitis of the shoulder are usually demonstrated roentgenographically in the suprahumeral region or adjacent to the greater tuberosity. When bursal calcifications appear dense (as in this x-ray film), they are frequently asymptomatic, whereas lightening at the margins of the calcium deposit is compatible with the presence of inflammatory edema in the rotator cuff, which produces pain and tenderness in the shoulder region. The location of the calcific deposit in the radiograph may be a useful guide for the point of entry of aspiration and injection. The needle is directed to the calcareous deposit, aspiration is carried out, and a portion of the steroid medication is deposited there. The calcium may subsequently disappear. Not infrequently, it does so spontaneously, without a needling procedure.

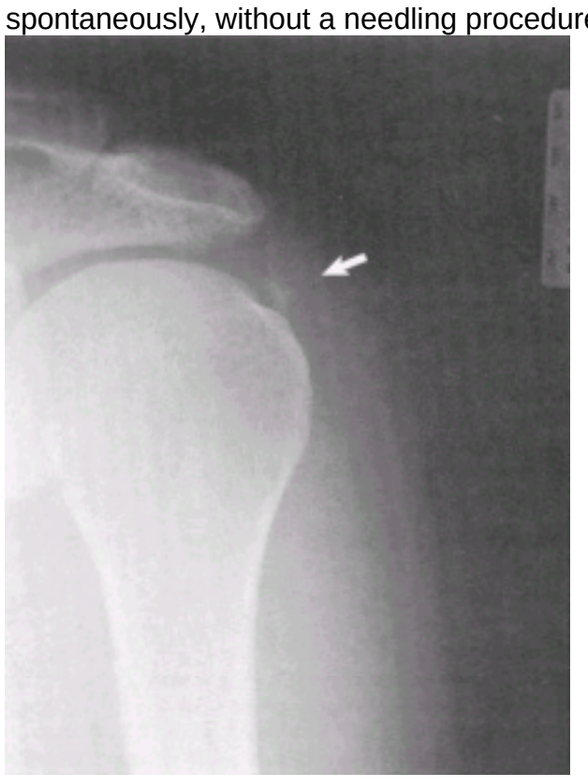


Figure 53-7 *A,* Anterior approach to the subacromial bursa or supraspinatus tendon. The needle is inserted into the depression located inferolateral to the coracoid process and medial to the humeral head.

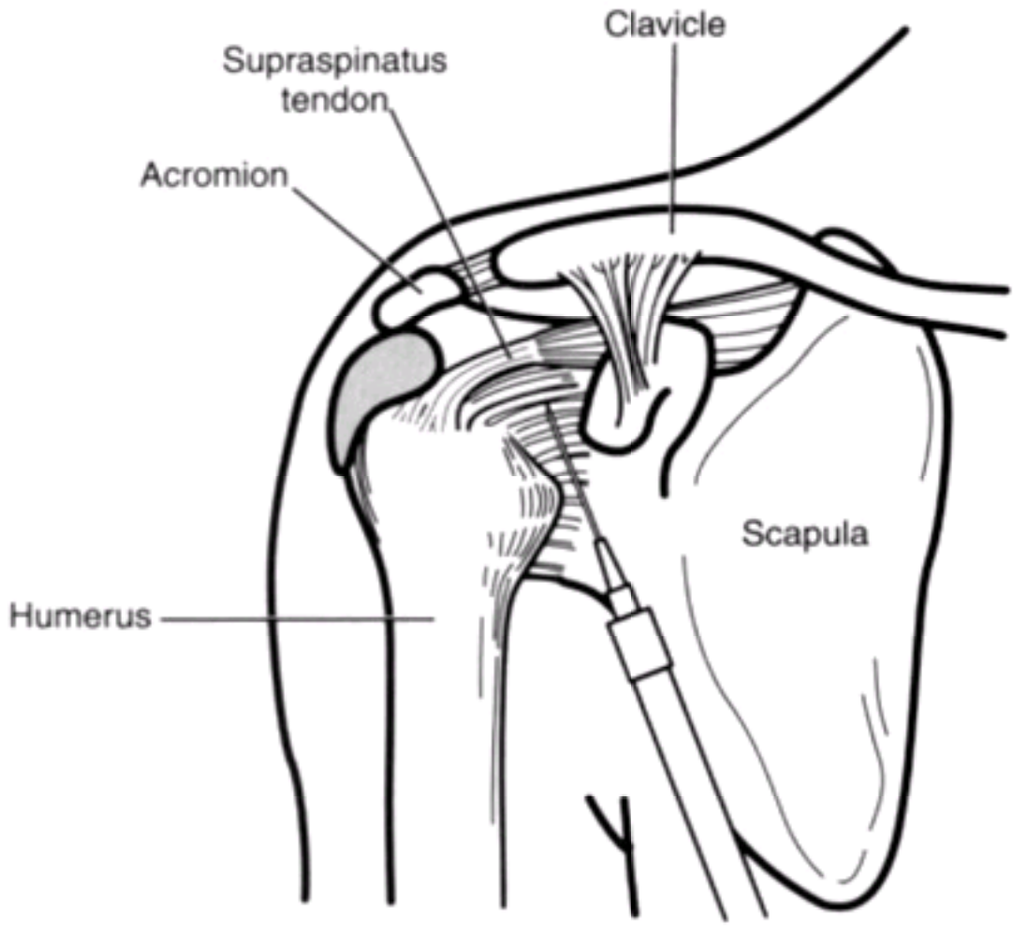


Figure 53-8 Posterolateral approach. *A,* The needle is inserted in the depression just inferior to the posterolateral tip of the acromion and superior to the head of the humerus. *B,* Injection using the posterolateral approach. The angle of entry should be approximately 15°.

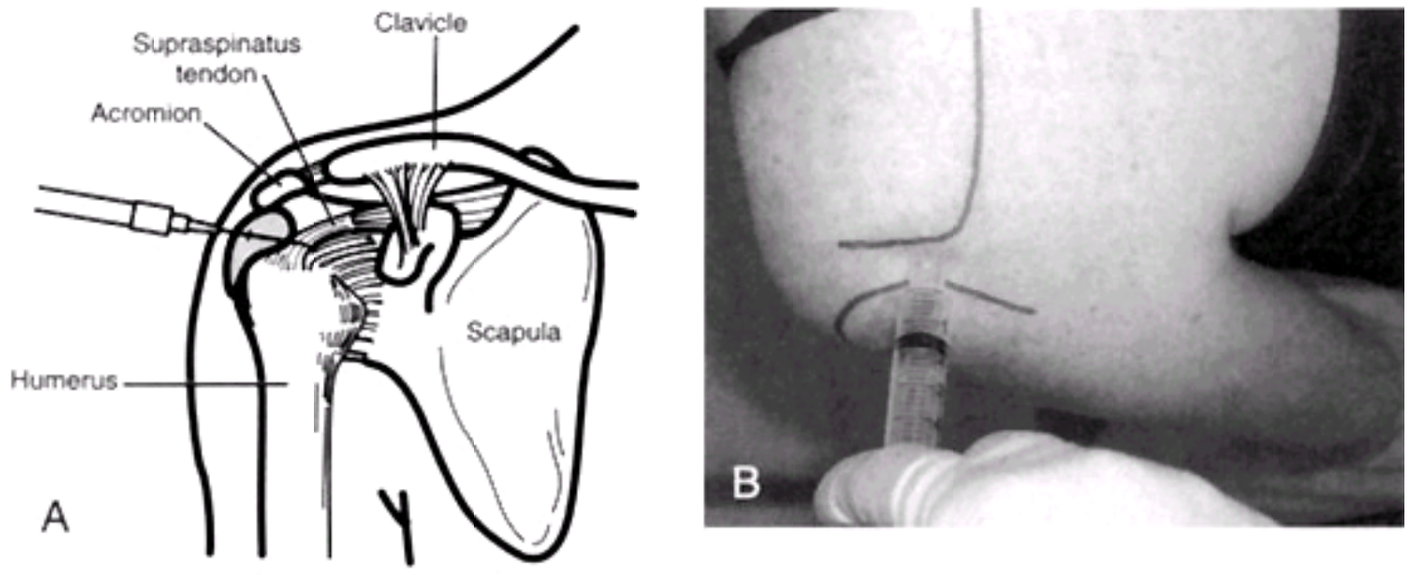


Figure 53-9 Injection of the acromioclavicular joint. Entry is made at the "V"-shaped depression at the most lateral aspect of the clavicle.

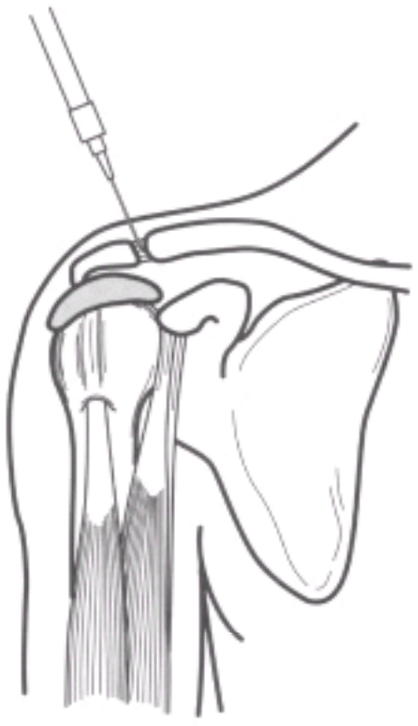
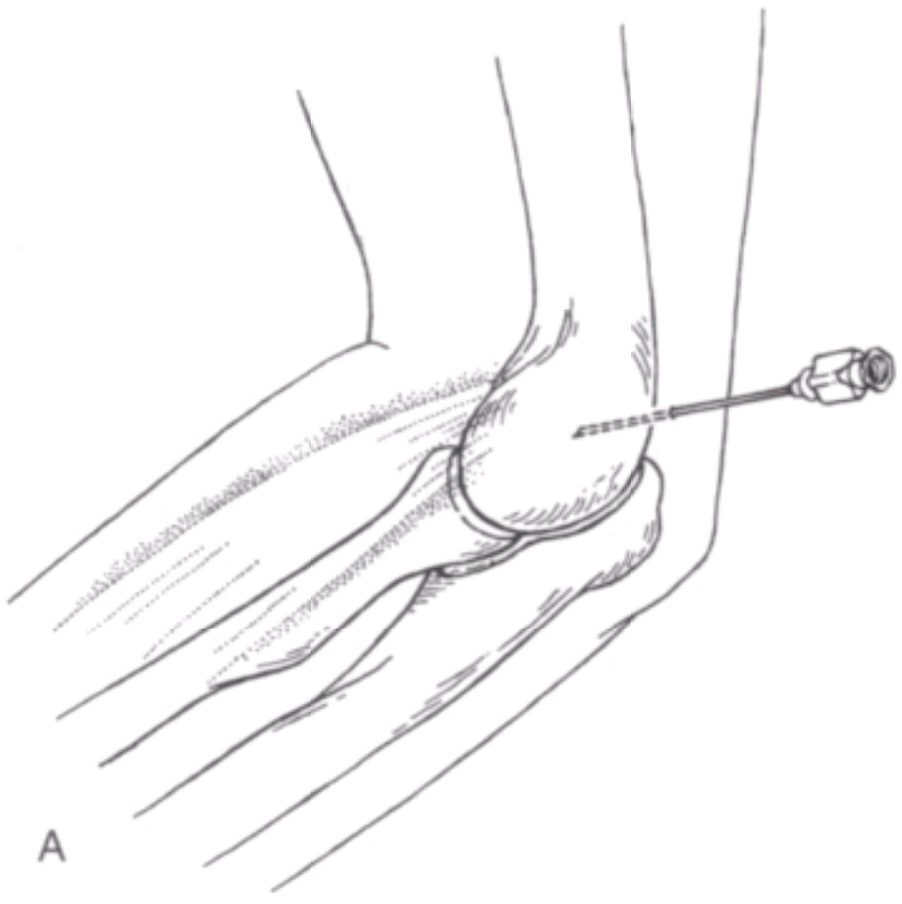


Figure 53-11 Clinical test for lateral epicondylitis. The patient extends the wrist against resistance, while the examiner localizes the site of maximal tenderness at the lateral epicondyle.

Figure 53-12 *A,* Injection at lateral epicondyle for treatment of "tennis elbow." *(Redrawn from steinbrocker O, Neustadt DH: Aspiration and Injection Therapy in rthritis and Musculoskeletal Disorders: A Handbook on Technique and Management. Hagerstown, MD, Harper & Row, 1972. Reproduced with permission.) B,* The needle is inserted in the area of maximal tenderness, avoiding injection directly into the tendon.



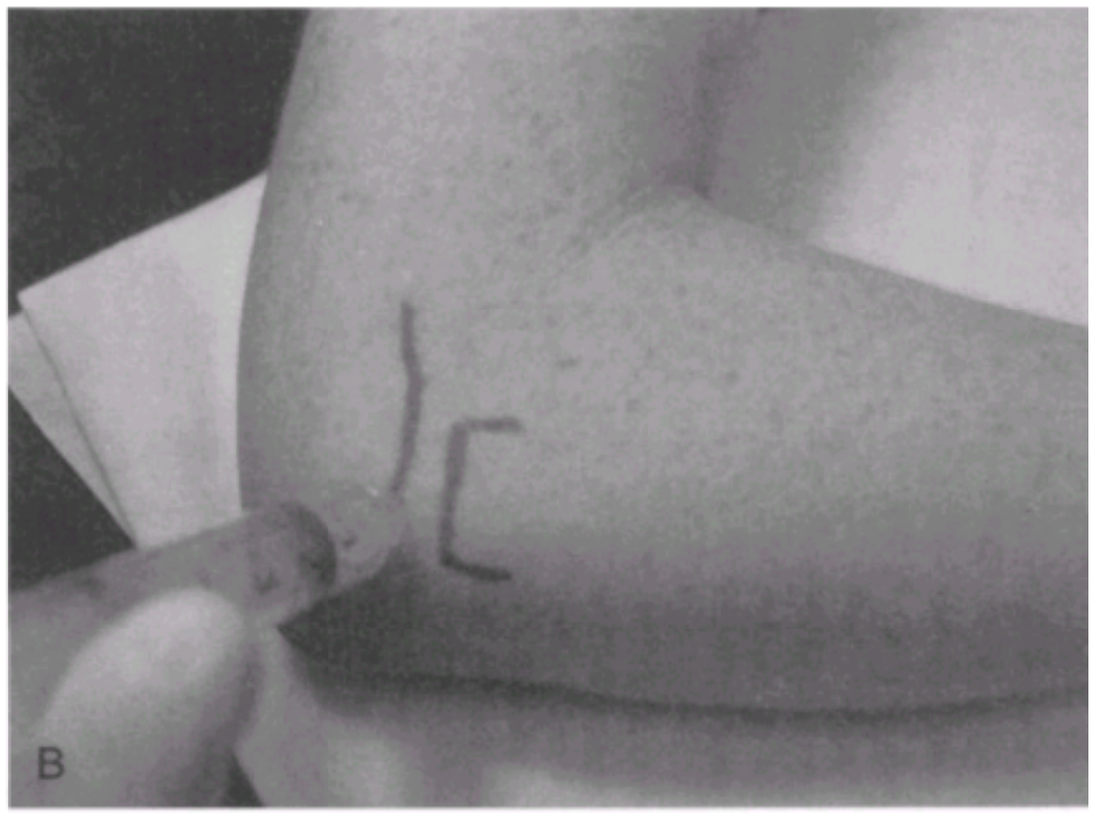
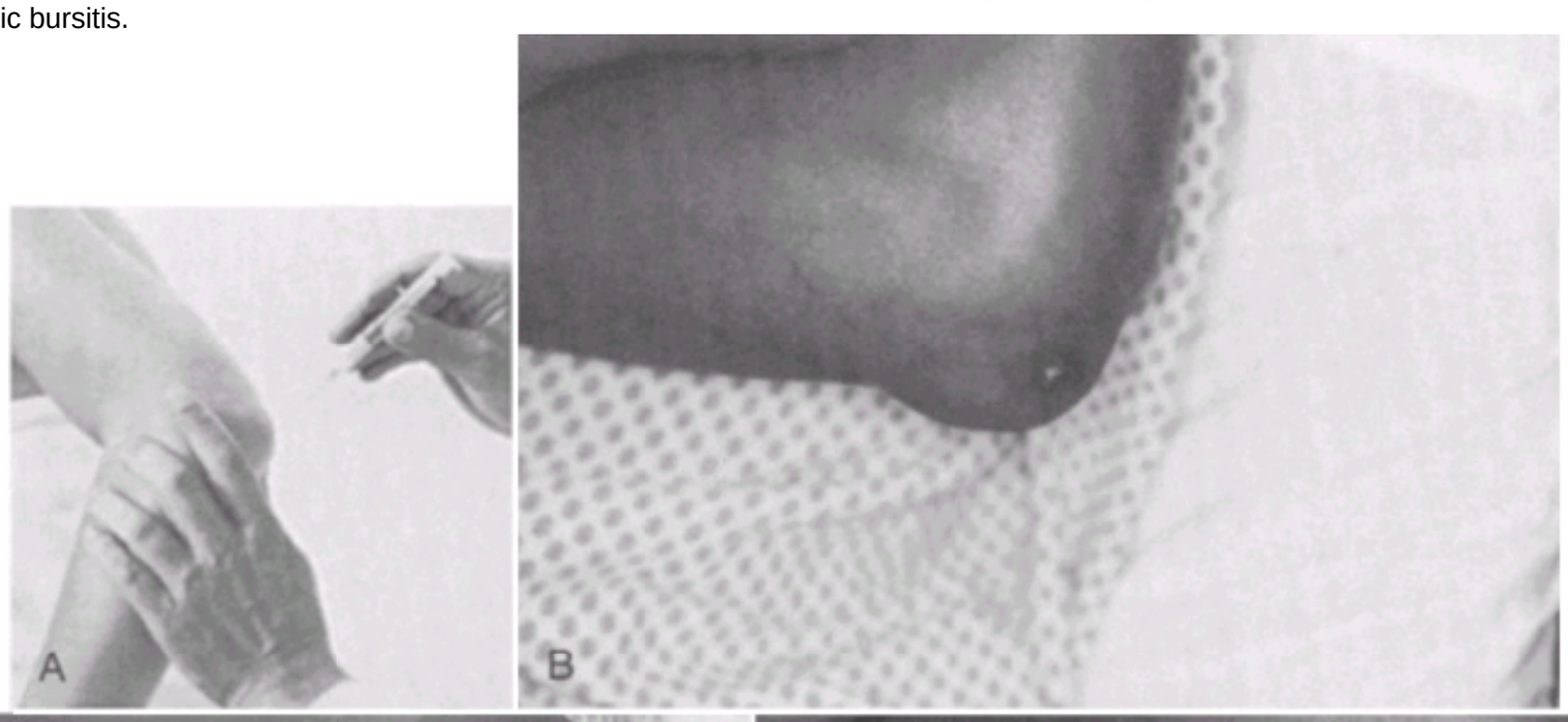
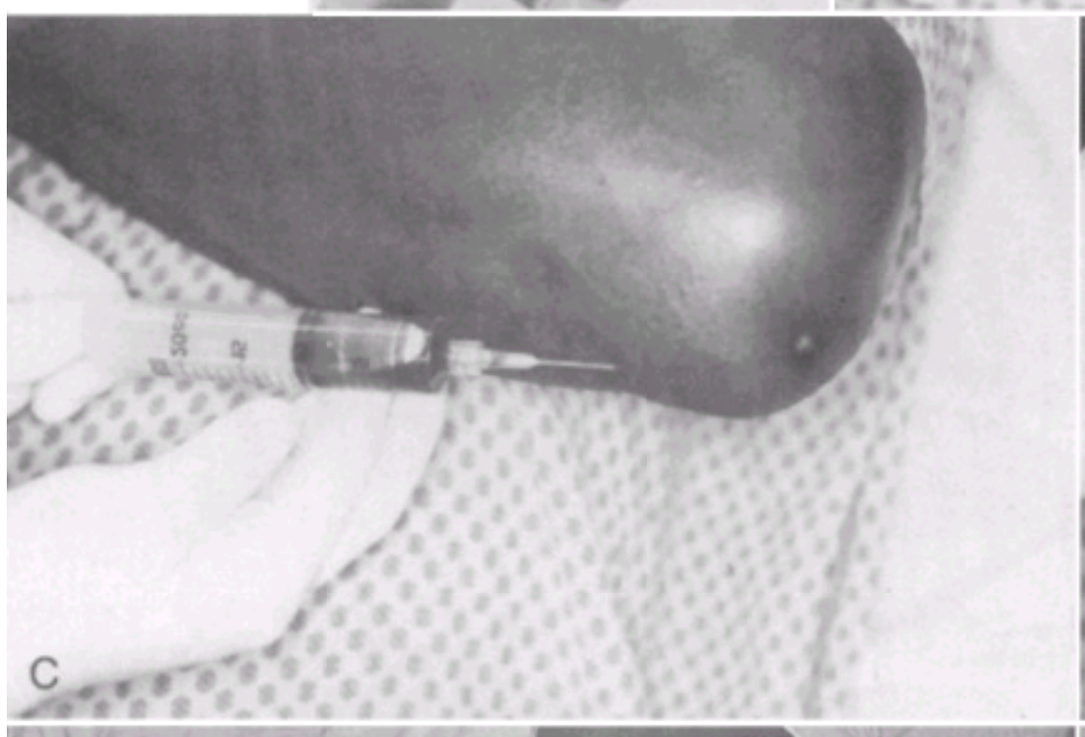
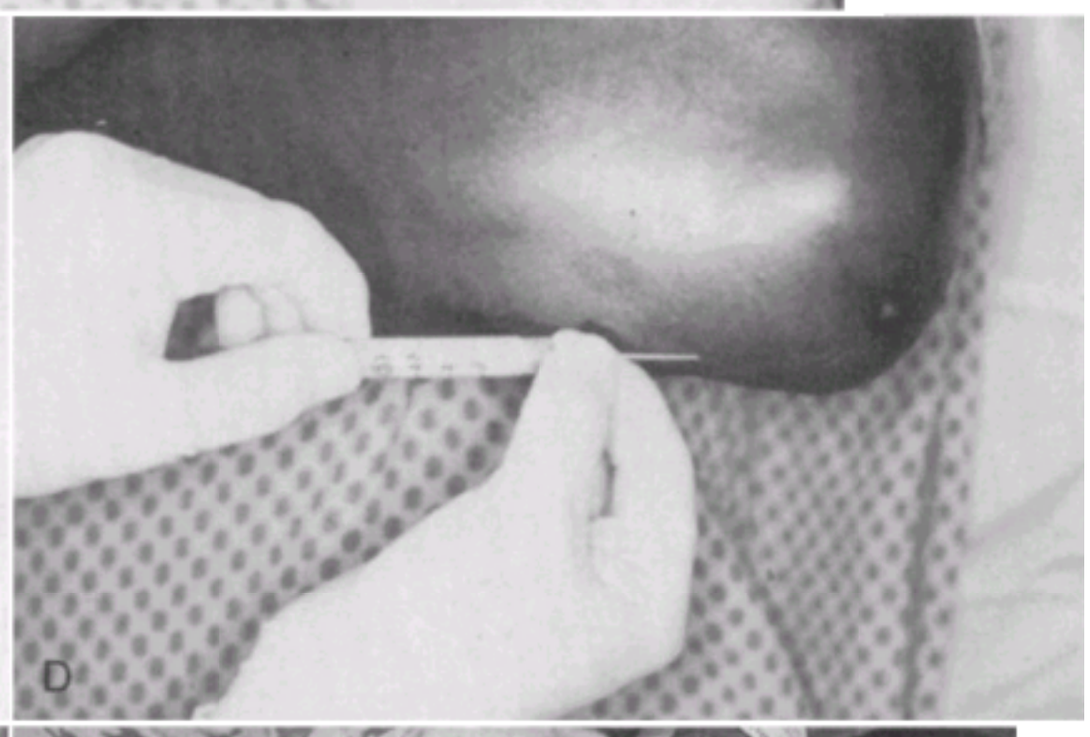
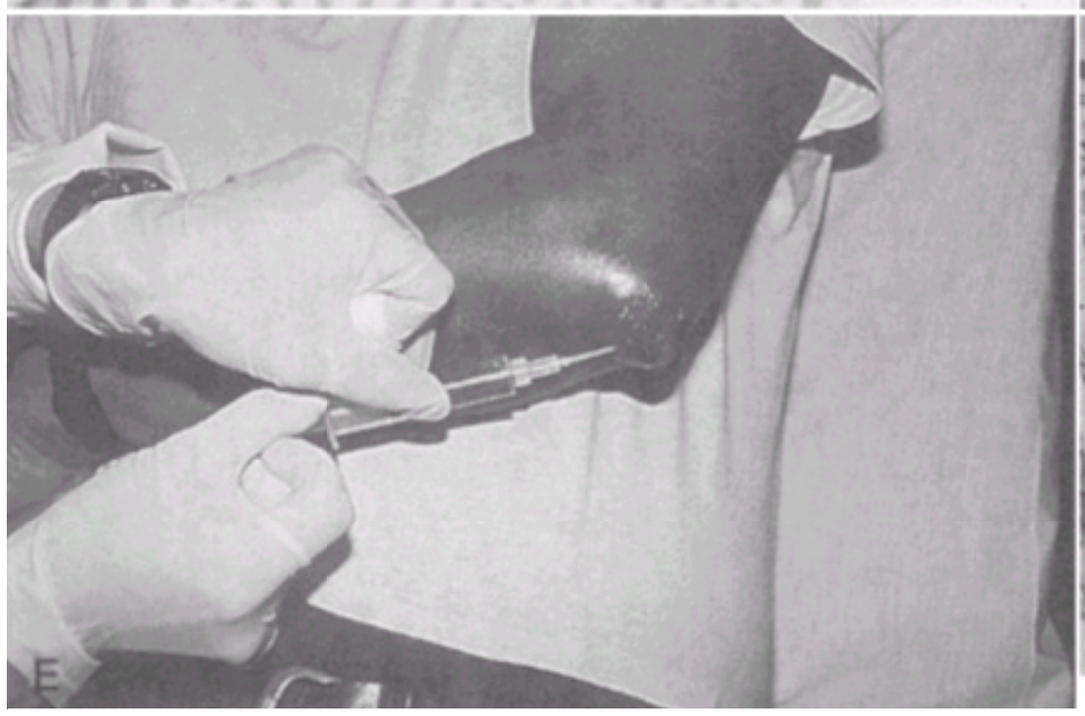


Figure 53-13 *A,* Anatomy showing location of the olecranon bursa superficial to the ulnar olecranon. *B,* traumatic painless swelling over the posterior elbow is characteristic of nonseptic olecranon bursitis. The mass is soft, movable, and fluctuant and is usually filled with serosanguineous fluid. *C,* Using sterile preparation, a 20-ga needle on a 10-mL syringe is advanced parallel to the forearm, and the bursal fluid is completely aspirated. *D,* The aspirating syringe is changed while the needle remains in the bursal sac, and a long-acting steroid preparation (such as 40 mg of methylprednisolone) is injected. An elastic bandage provides compression for 12 hours. *E,* An alternative posterior approach to the bursa. *F,* An extreme case of tophaceous gout involving the olecranon bursa. Most cases of acute gout bursitis resemble septic bursitis.









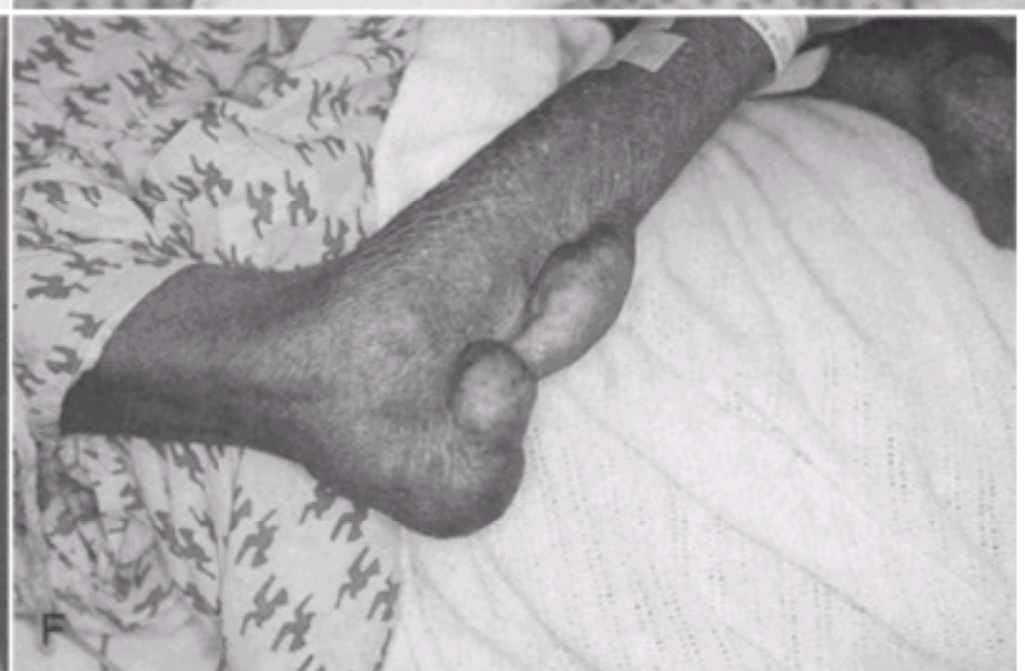


Figure 53-14 Fully developed septic olecranon bursitis is easily distinguished from nonseptic bursitis by clinical parameters. In the septic variety, there is diffuse swelling, as opposed to a discrete mass; the area is red, warm, and quite tender. In addition, bursal fluid leukocytosis is present.

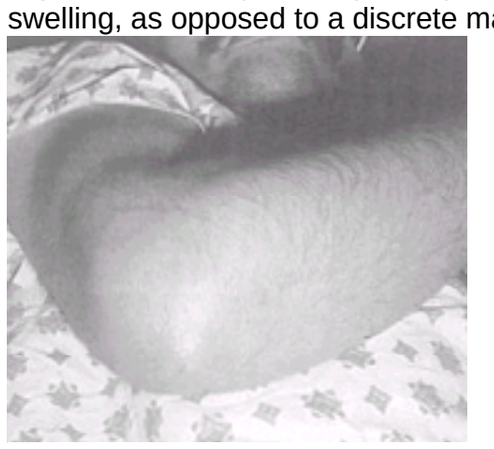
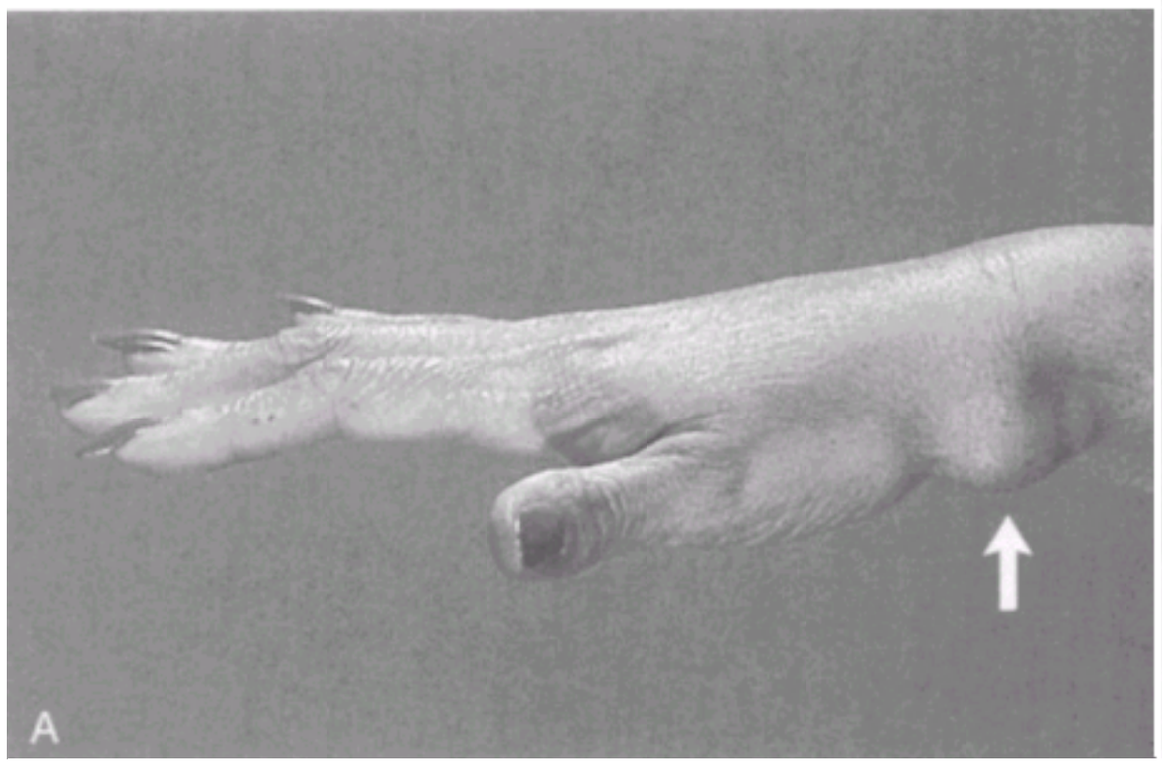


Figure 53-15 A typical volar ganglion cyst of the wrist (*A*) and dorsum of the foot (*B*).



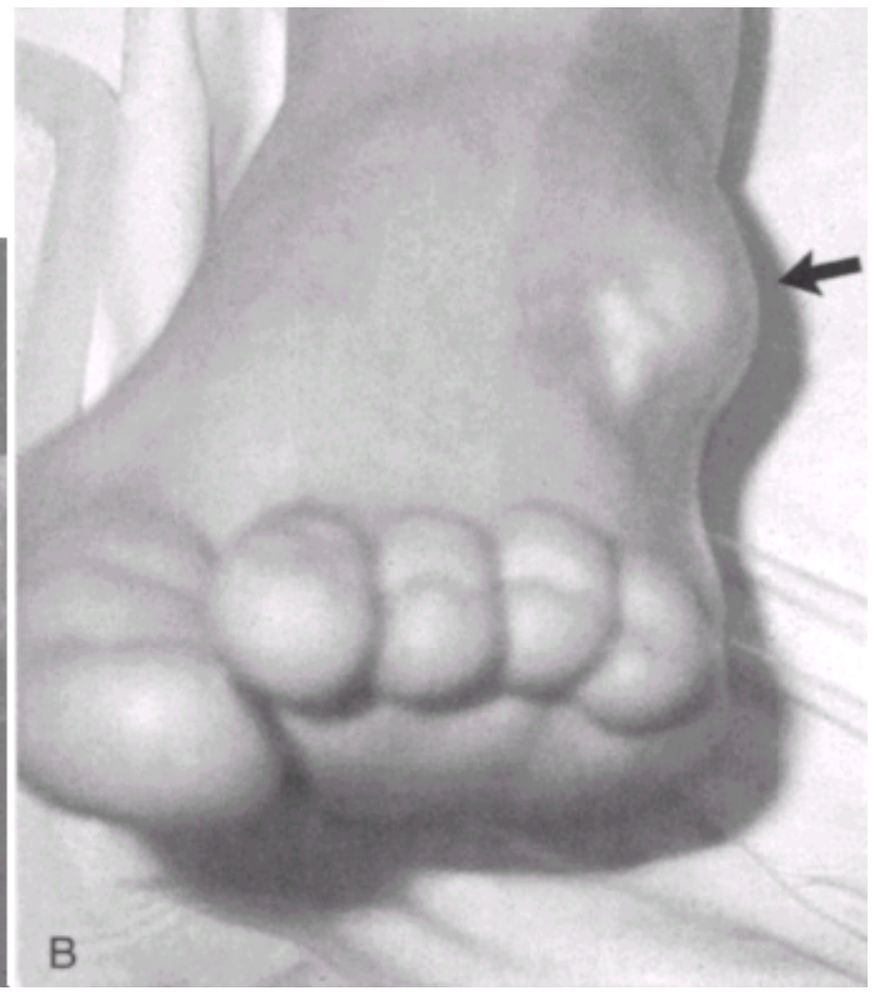


Figure 53-17 *A,* Site of de Quervain disease at the *extensor pollicis brevis* (EPB) and *abductor pollicus longus* (APL) tendons. *B,* Site of intersection syndrome at the *extensor carpi radialis longus* (ECRL) and *extensor carpi radialis brevis* (ECRB) tendons.

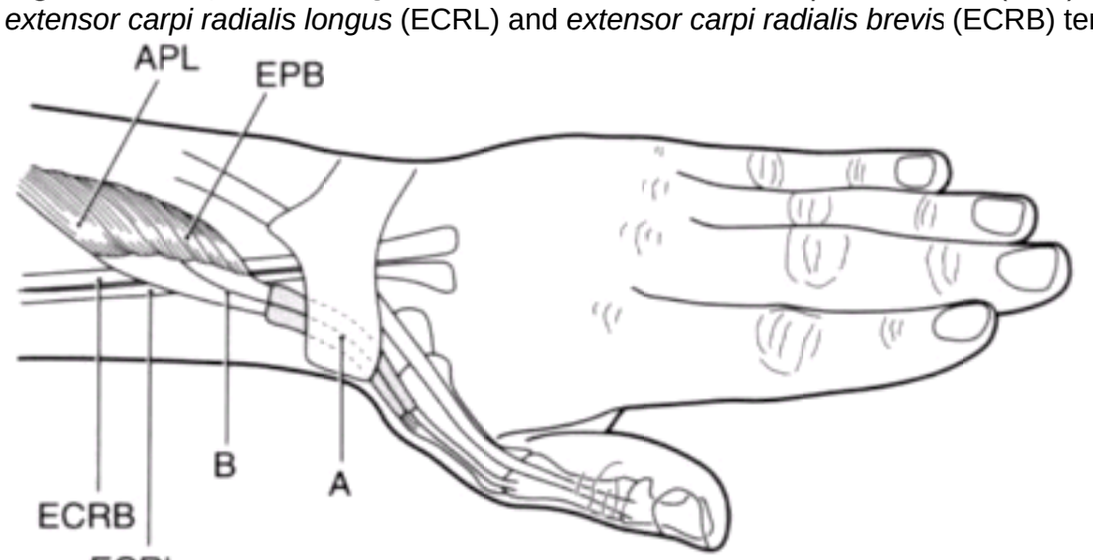


Figure 53-18 Photograph of a patient's wrist with intersection syndrome. Note that the swelling (arrow) is several centimeters *proximal* to the radial styloid, overlying the compartment of the common wrist extensors (*extensor carpi radialis longus* and *extensor carpi radialis brevis*). *(From Hanlon DP, Luellen JR: Intersection Syndrome: A case report and review of the literature. J Emerg Med 17:970, 1999. Reproduced with permission.)*

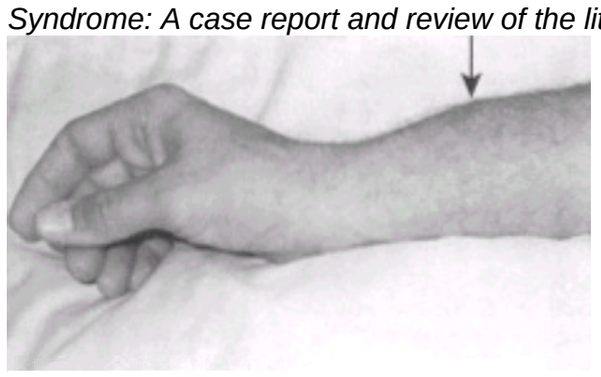


Figure 53-19 Injection of abductor tendon sheath (de Quervain disease). When giving an injection for de Quervain disease, it is desirable to avoid direct tendon injection and to instead aim for a peritendinous infiltration (*A*). If 3 to 4 mL of a local anesthetic is added to the steroid, generous infiltration over a diffuse area can compensate for a less than perfect localization (*B*). *(A from Steinbrocker O, Neustadt DH: Aspiration and Injection Therapy in Arthritis and Musculoskeletal Disorders: A Handbook on Technique and Management. Hagerstown, MD, Harper & Row, 1972. B from Zingas C, Failla JM, Holsbeeck MV: Injection accuracy and clinical relief of de Quervain tendinitis. J Hand Surg 23a:89, 1998. Reproduced with permission.)*

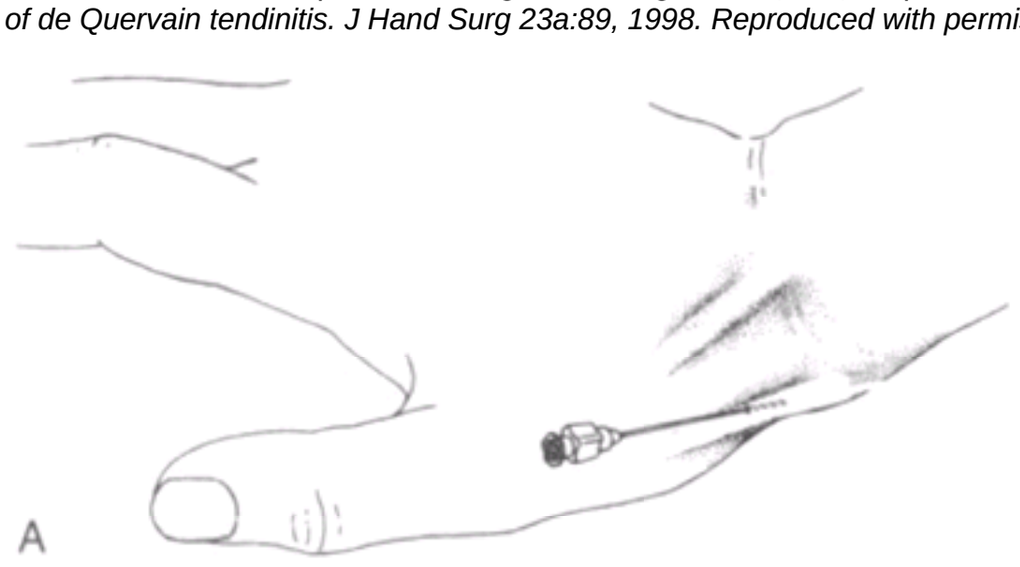


Figure 53-20 *A,* Injection of the median carpal tunnel with the wrist dorsiflexed over a rolled towel. To avoid direct injection of the median nerve, the needle is introduced just medial (ulnar) to the *palmaris longus* tendon. *B,* A palpable bulge at the distal edge of this ligament indicates correct placement of the injection. Direct injection into the median or ulnar nerve is to be avoided. *(A redrawn from Steinbrocker O, Neustadt DH: Aspiration and Injection Therapy in Arthritis and Musculoskeletal Disorders: A Handbook on Technique and Management. Hagerstown, MD, Harper & Row, 1972. B from Dehaan MR, Wilson RL: Diagnosis and management of carpal tunnel syndrome. J Musculoskel Med 6:47, 1989. Reproduced with permission.)*

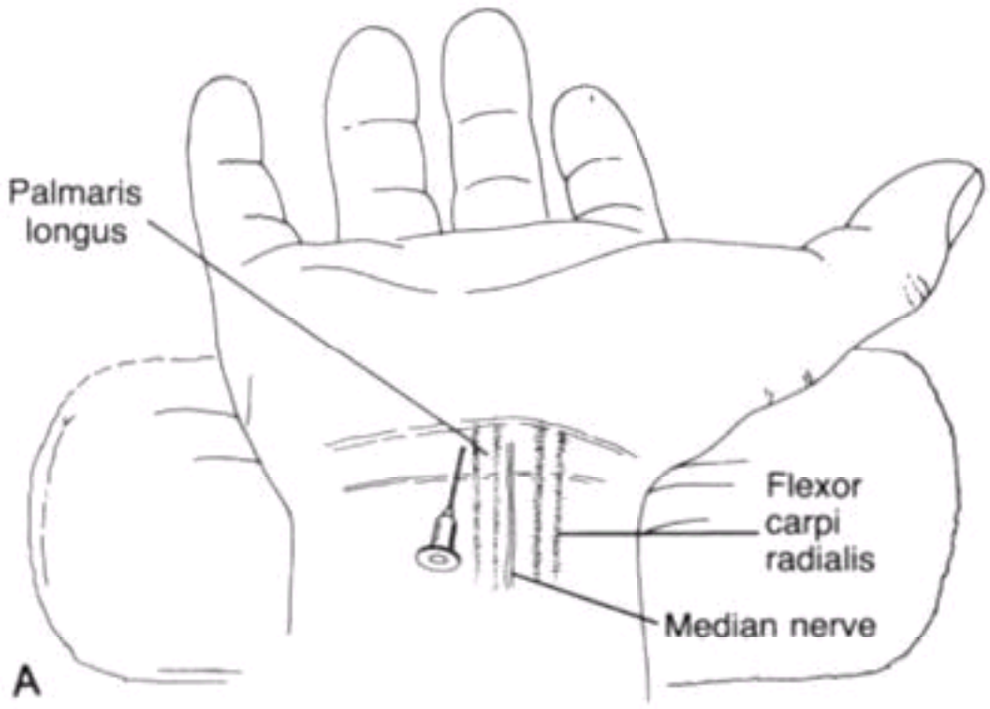


Figure 53-22 Injection of flexor tendon sheath in digital tenosynovitis.

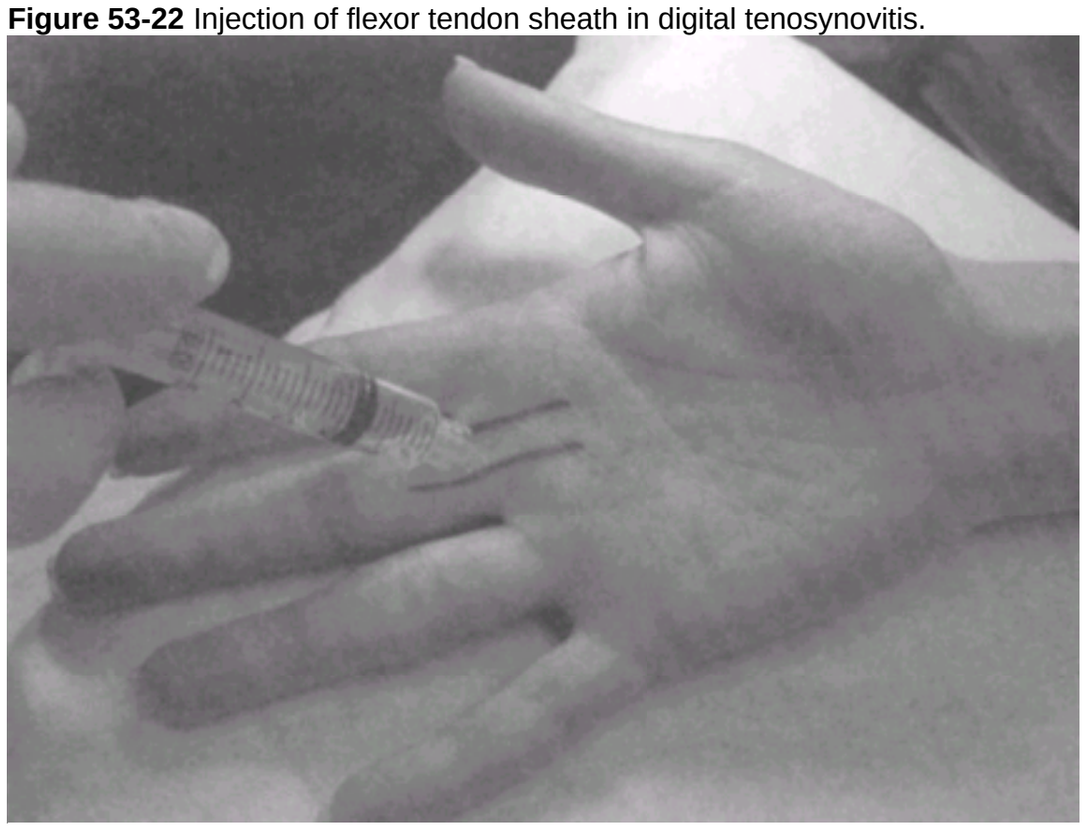


Figure 53-23 Trochanteric bursitis. The principal bursa lies between the gluteus maximus and the greater trochanter, although other bursae may also be involved.

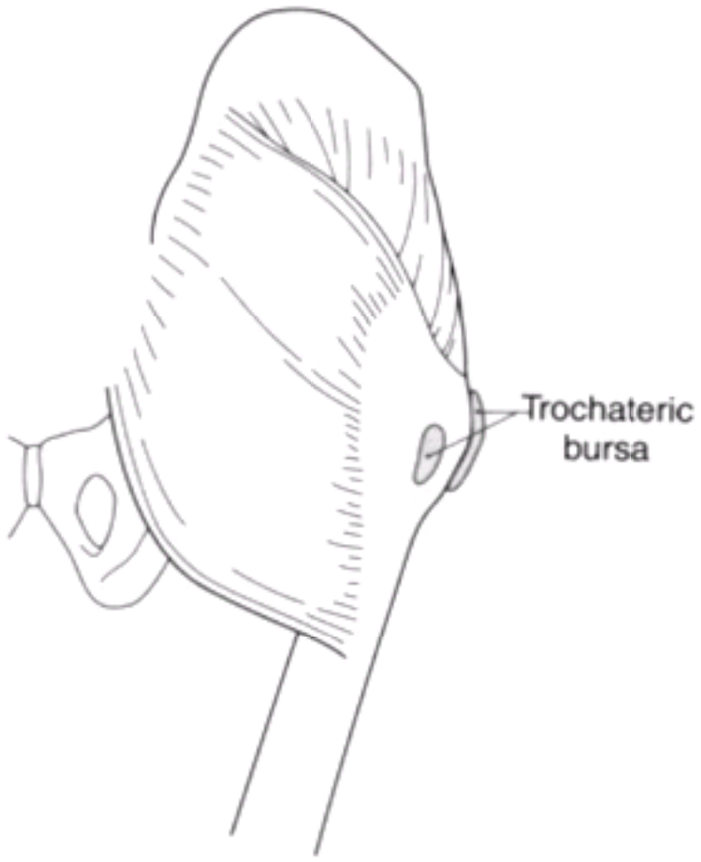
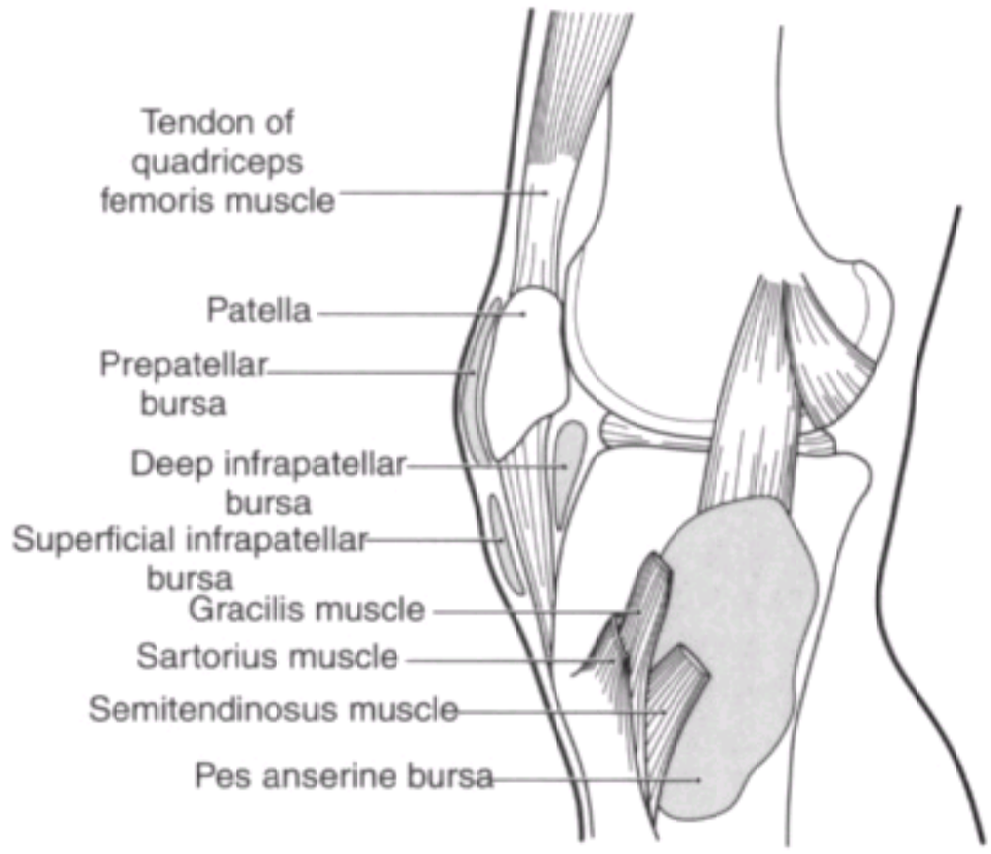


Figure 53-24 Prepatellar bursitis and *pes anserine* bursitis. The prepatellar bursa lies superior to the patella, while the *pes anserine* bursa lies deep to the insertion of the sartorius, gracilis, and semitendinosus tendons.



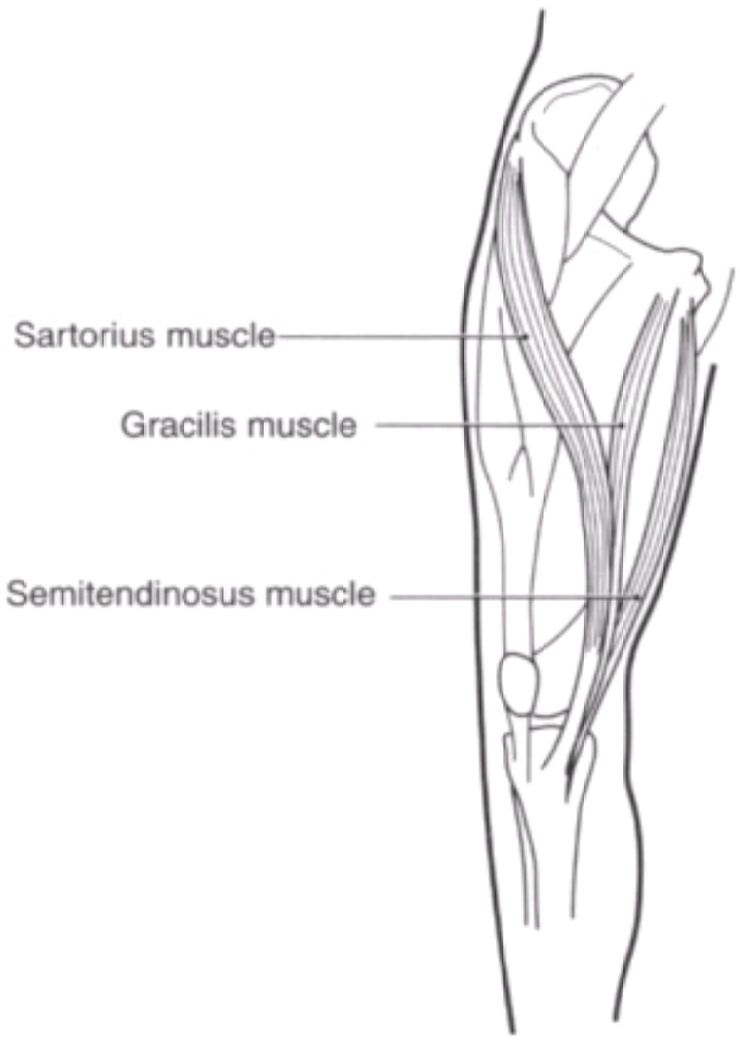


Figure 53-25 Injection of the anserine bursa of the knee. Note that the point of entry, which is the point of maximal tenderness, is inferior to the patella and medial to the tibial tuberosity.

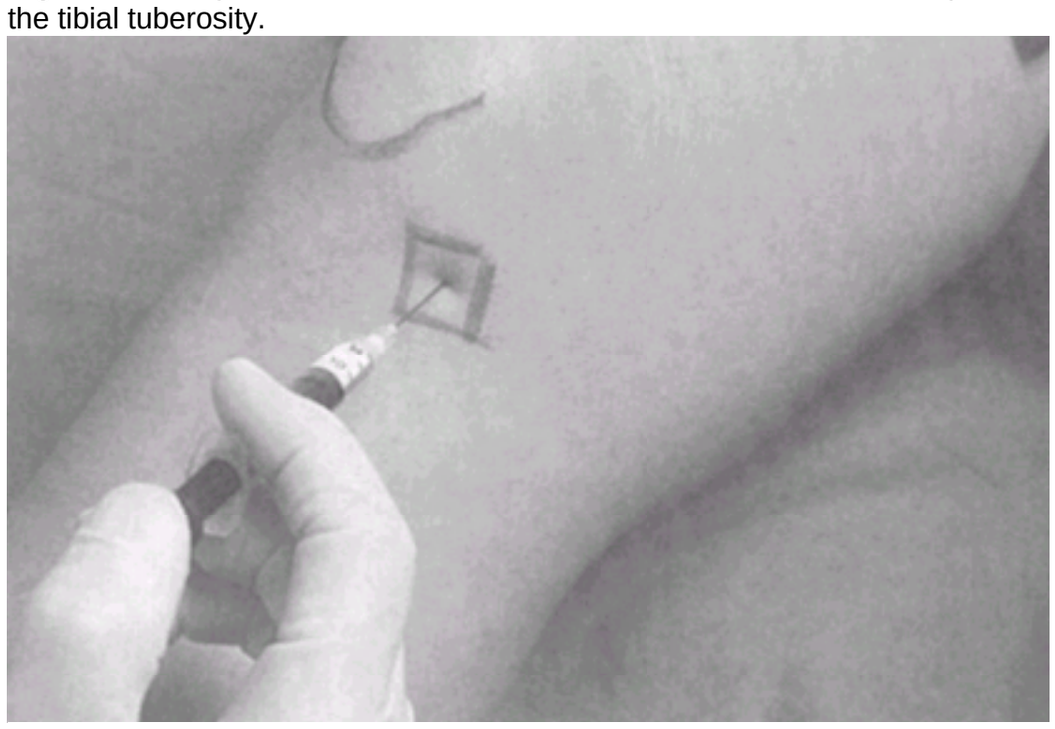


Figure 53-26 Heel pain. Talalgia may involve the tendons, bursa, or fascia around the heel.

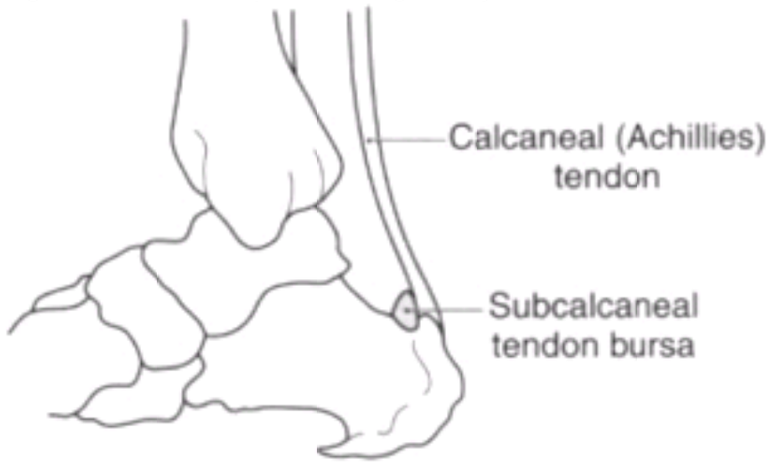


Figure 53-27 Injection of calcaneal bursitis with heel spur. *(Redrawn from Steinbrocker O, Neustadt DH: Aspiration and Injection Therapy in Arthritis and Musculoskeletal Disorders: A Handbook on Technique and Management. Hagerstown, MD, Harper & Row, 1972.)*

Figure 54-1 Periarticular problems may mimic an intra-articular process. *A*, This patient had trauma to the knee and presented with anterior soft tissue swelling and fluctuance, representing a hematoma of the prepatellar bursa. *B*, While an assistant (gloves should be worn) compresses the fluid, the bursa is aspirated of all blood.

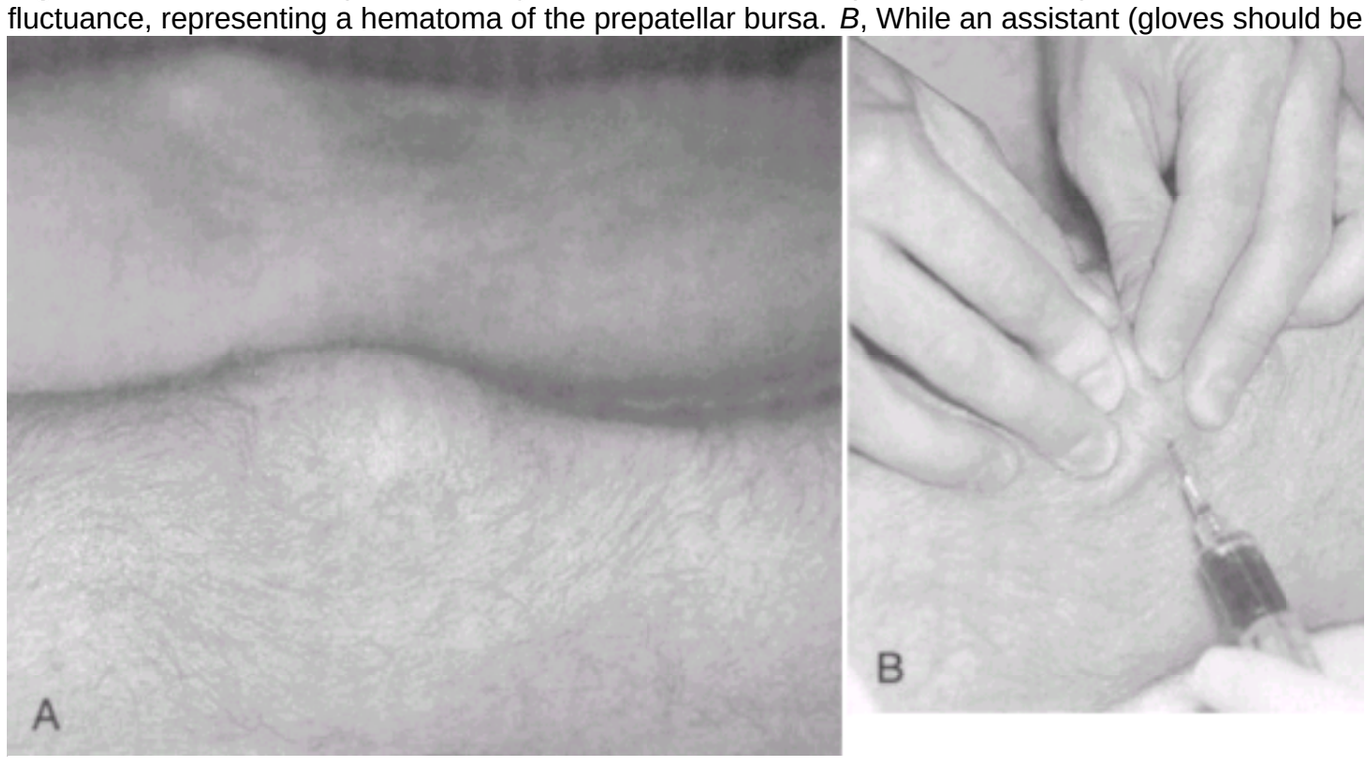


Figure 54-2 *A*, *B*, and *C*, Dramatic representation of tophaceous gout. These nodules are painless and full of uric acid crystals. An acutely swollen and painful joint in this patient is most likely acute gouty arthritis. *D*, This markedly swollen wrist is secondary to acute gouty arthritis. The patient had a fever and leukocytosis, and arthrocentesis with joint fluid analysis was the only way to differentiate gout from a septic joint.

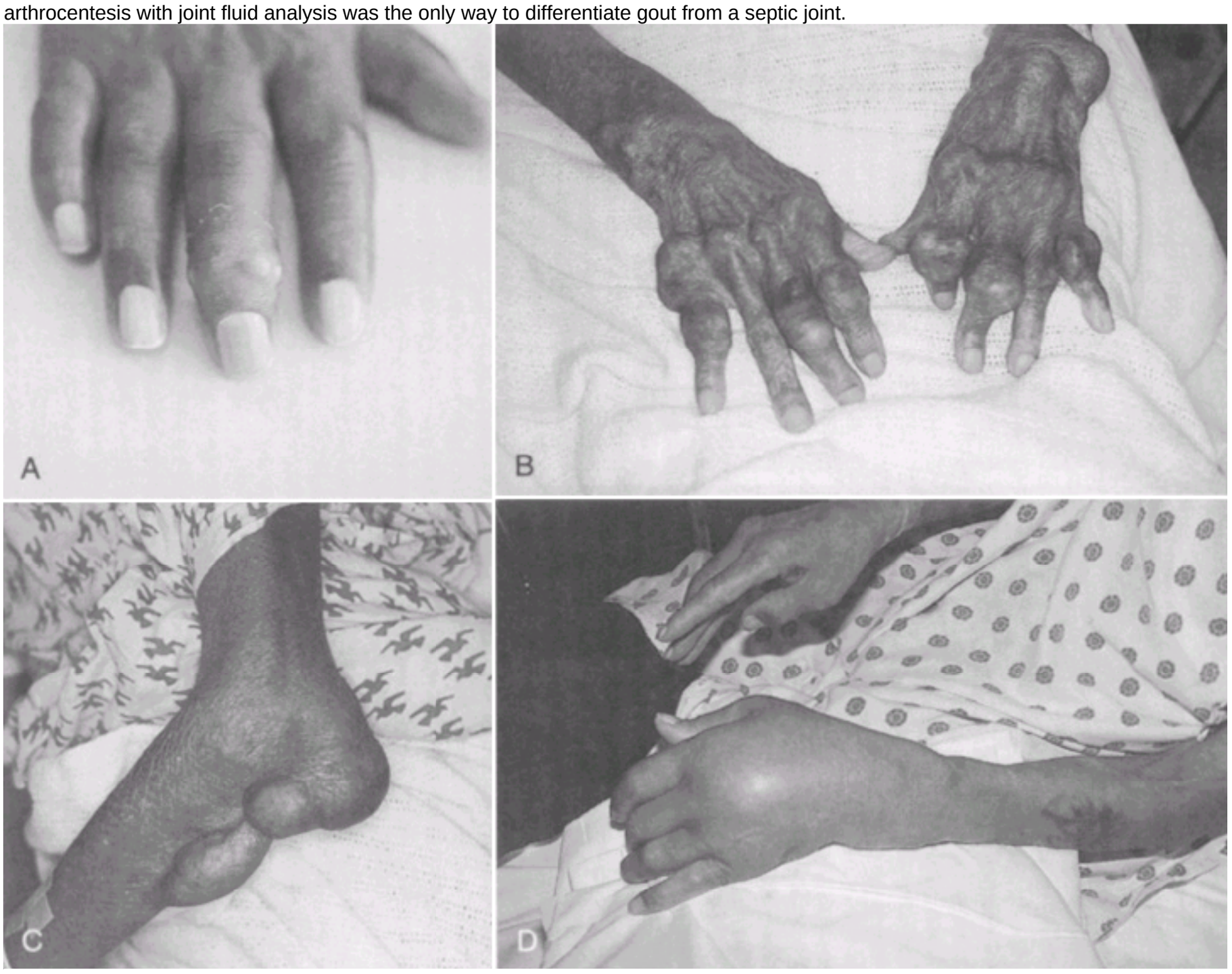


Figure 54-3 *A*, Skin lesions, ranging single or multiple petechiae to pustules may be seen in patients with acutely swollen joints infected with *Neisseria gonorrhoeae*. Skin lesions are usually found on the extremities, especially the feet and hands, and may be present before a large joint effusion accumulates, or with gonococcal tenosynovitis.

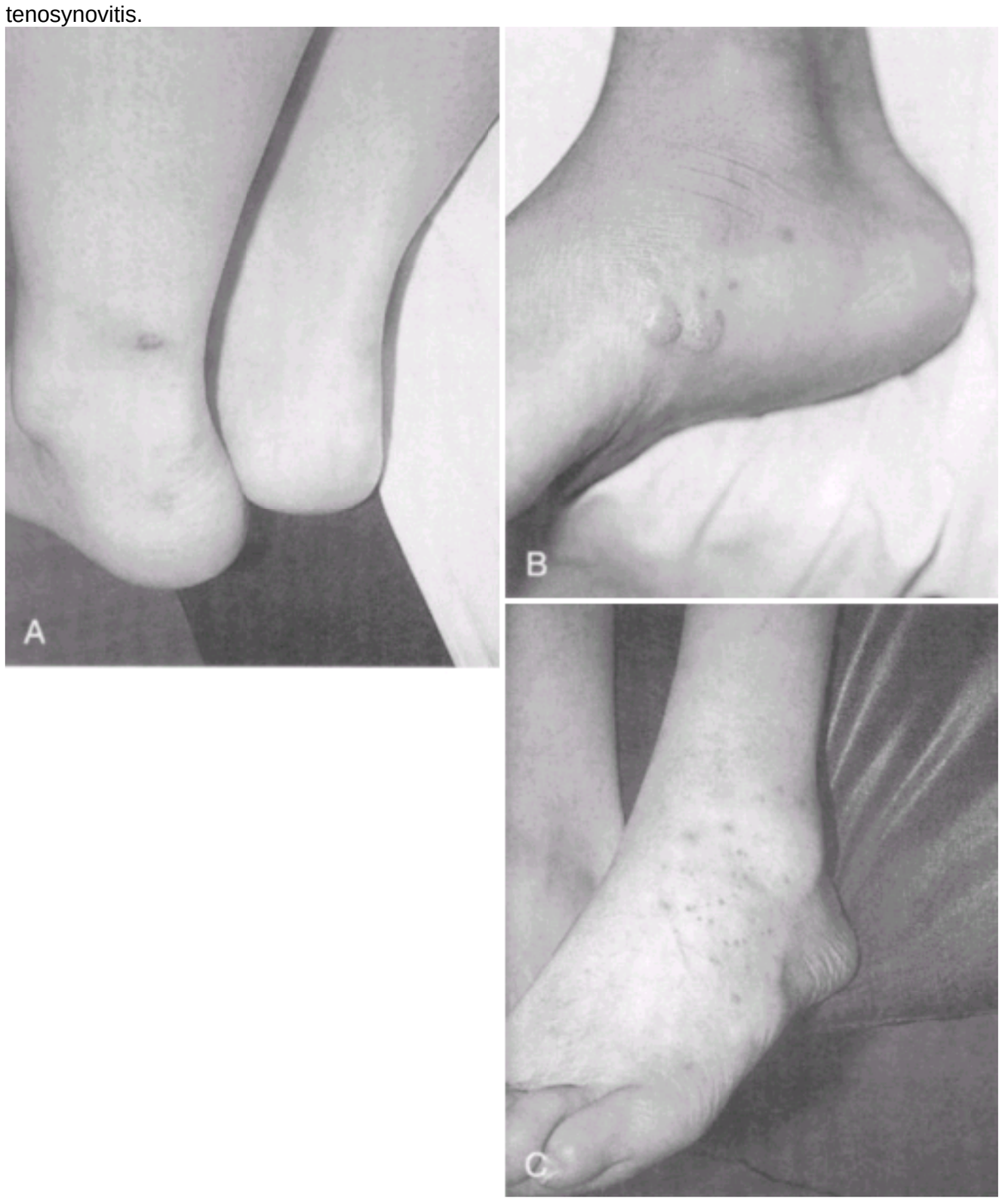


Figure 54-4 *A*, A chronic miniscus tear can produce a large effusion of clear yellow fluid. The miniscus has no blood supply, and a hemarthrosis suggests another or additional diagnosis. *B*, If the syringe is too small to accommodate all of the fluid, grasp the needle hub with a hemostat and remove the syringe without disturbing the correct position of the needle. A stopcock will negate the need to change the syringe. *C*, With the needle still held in place, use another syringe to instill a steroid when the aspirate is a clear yellow. *D*, Cloudy fluid such as this is from an inflammatory source, such as gout or a septic joint. Intra-articular steroids should not be administered in the setting of a cloudy aspirate.

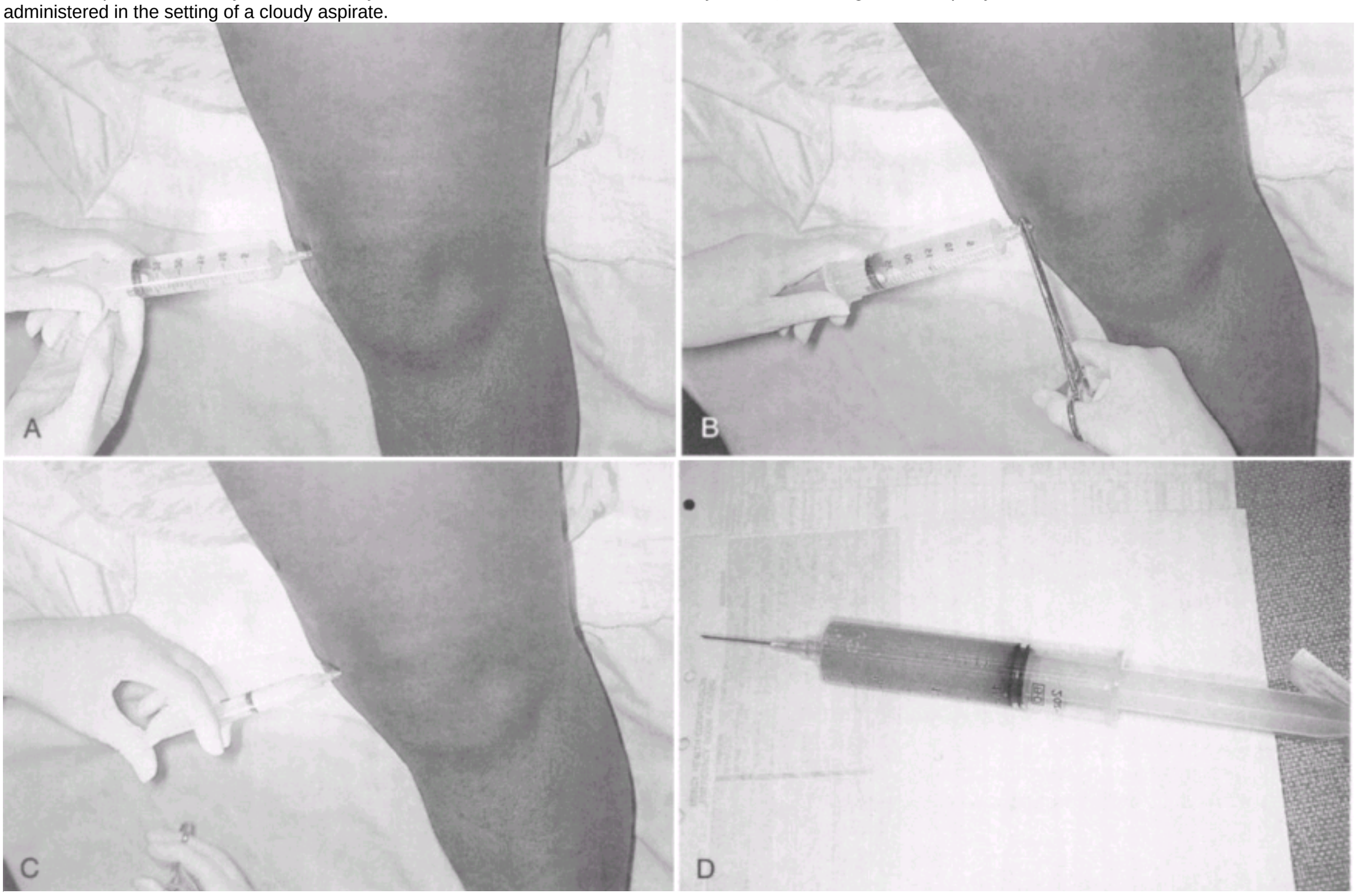


Figure 54-5 Landmarks for arthrocentesis of first carpometacarpal joint (see text). *(From Akins CM: Aspiration and injection of joints, bursae, and tendons. In Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

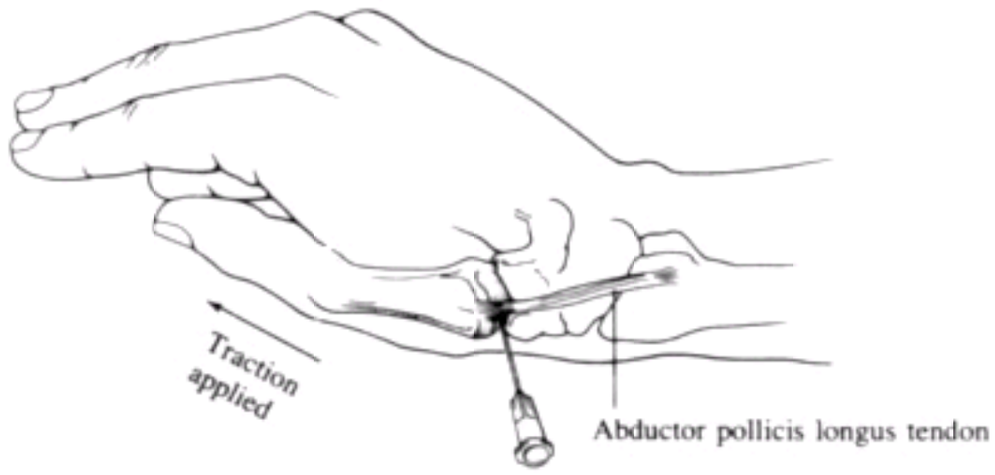


Figure 54-6 Landmarks for arthrocentesis of interphalangeal and metacarpophalangeal joints (see text). *(From Akins CM: Aspiration and injection of joints, bursae, and tendons. In Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

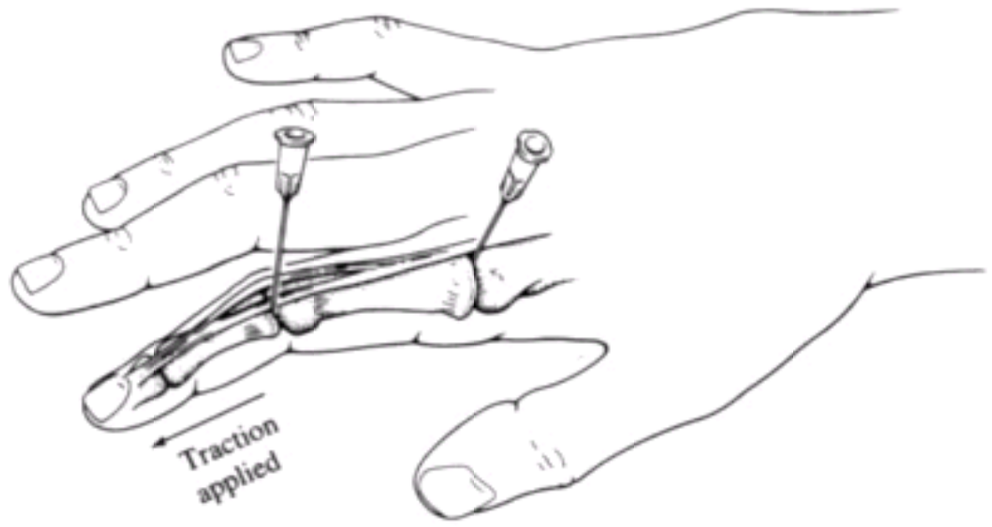


Figure 54-7 Landmarks for arthrocentesis of the radiocarpal joint (see text). Traction is applied with slight flexion. Contrary to the figure, *no* radial deviation is applied. *(From Akins CM: Aspiration and injection of joints, bursae, and tendons. In Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

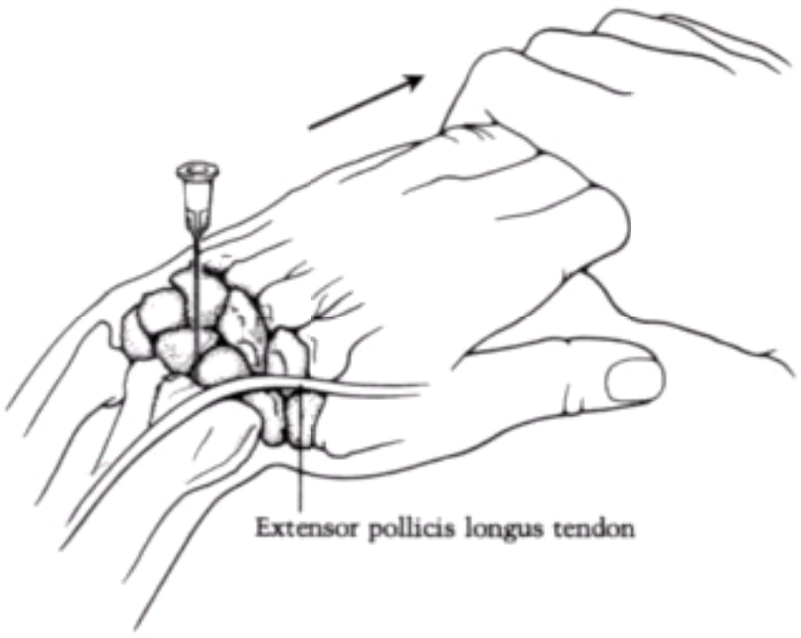
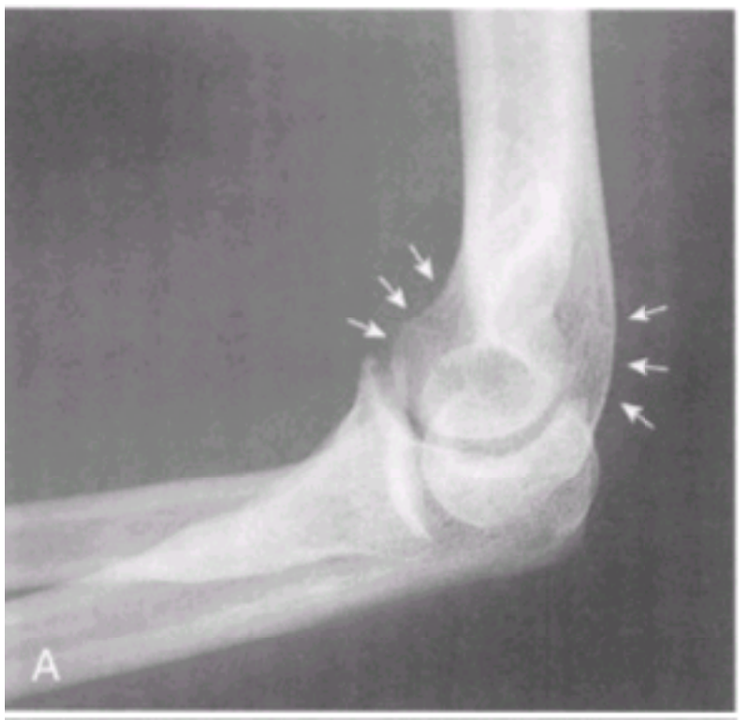
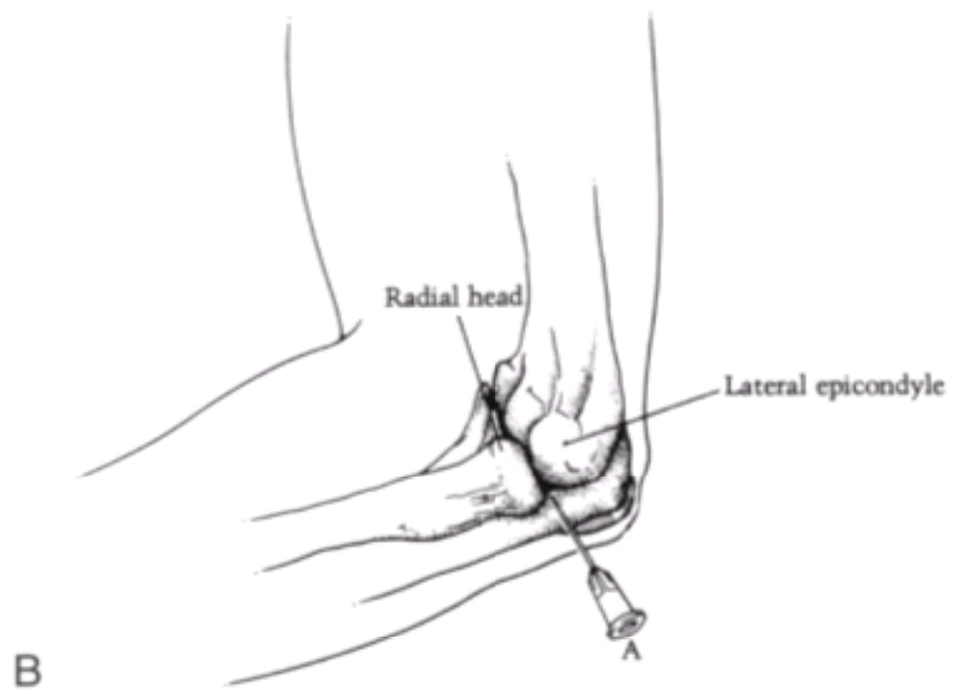
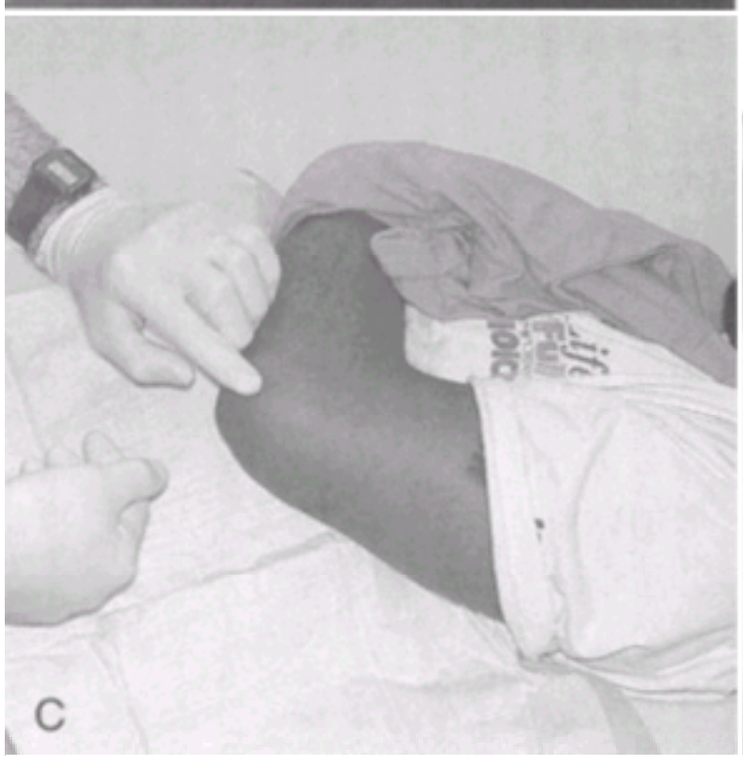


Figure 54-8 *A*, On a lateral elbow radiograph, displacement of the anterior fat pad (arrows) or the presence of a posterior fat pad (arrows) indicate blood, pus, or fluid in the joint. *B*, Landmarks for arthrocentesis of the radiohumeral joint (see text). *(From Akins CM: Aspiration and injection of joints, bursae, and tendons. In Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.) C*, An effusion in the elbow joint can usually be readily palpated. The index finger is placed over the lateral epicondyle and slid posteriorly toward the olecranon. Usually a depression is felt as the finger leaves the epicondyle, but a bulge is appreciated if there is a joint effusion. *D*, Removal of only a few milliliters of blood will reduce pain and hasten recovery of the range of motion. The most common pathology following trauma with an x-ray negative for fracture, but positive for a hemarthosis, is a nondisplaced radial head fracture.







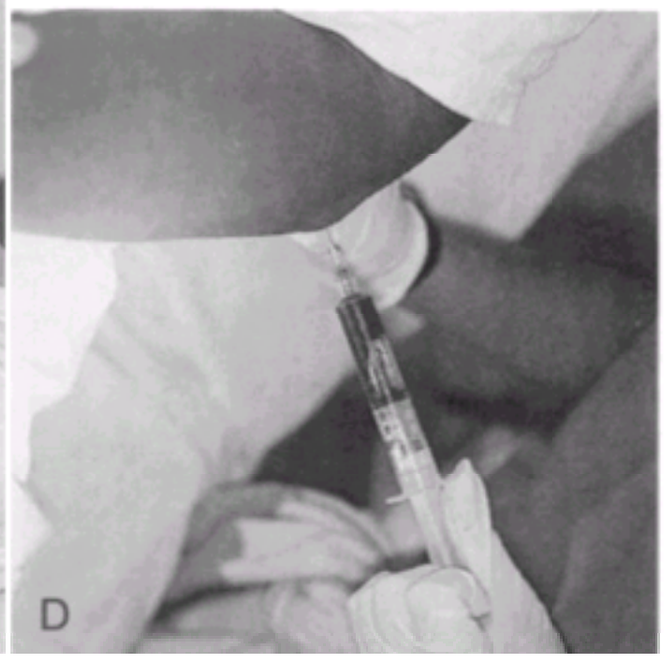


Figure 54-9 Landmarks for arthrocentesis of glenohumeral joint (see text). *(From Akins CM: Aspiration and injection of joints, bursae, and tendons. In Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

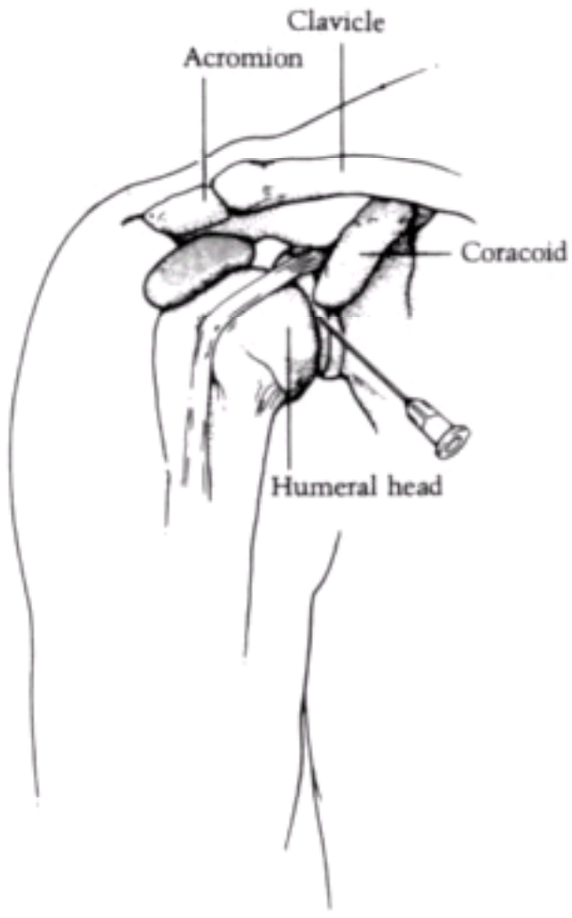
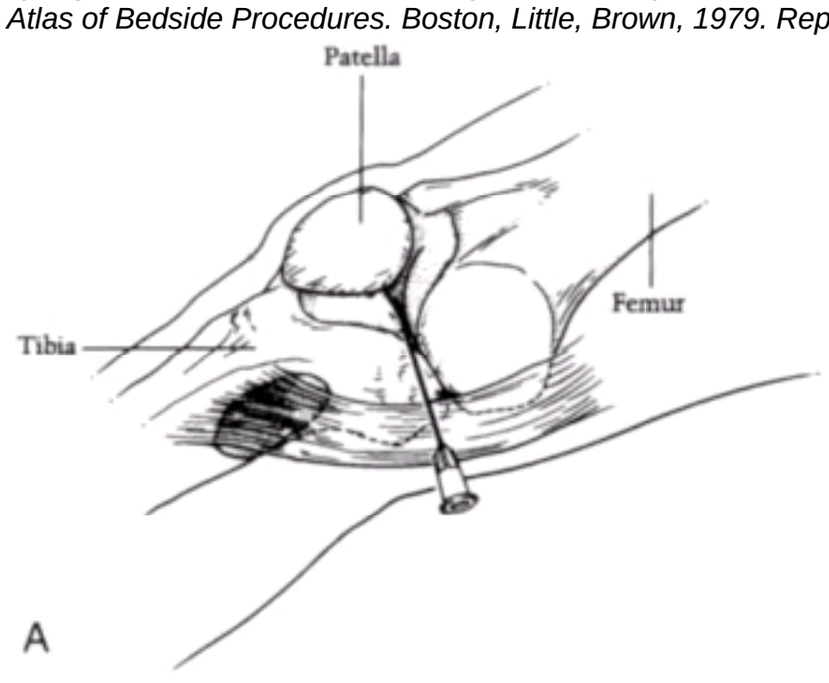


Figure 54-10 *A*, Landmarks for arthrocentesis of the knee joint, medial approach (see text). *B*, Note the use of a stopcock on the syringe to allow complete drainage without repositioning the needle. Compression of the suprapatellar region by the operator or an assistant will facilitate complete aspiration. For the knee, a 60 mL syringe should be used to drain large effusions. *(From Akins CM: Aspiration and injection of joints, bursae, and tendons. In Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*



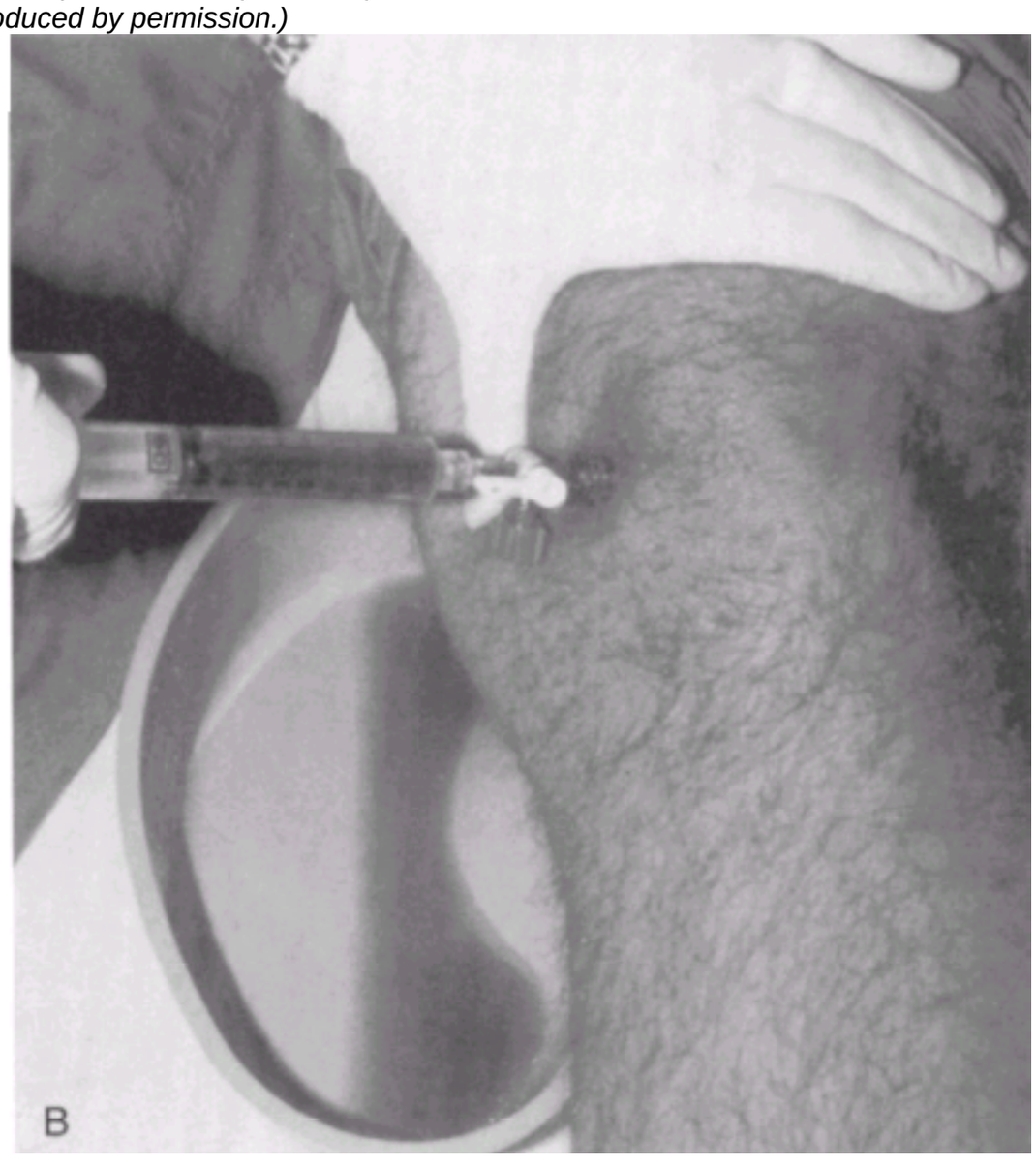


Figure 54-11 Landmarks for arthrocentesis of the tibiotalar joint (see text). *(From Akins CM: Aspiration and injection of joints, bursae, and tendons. In Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

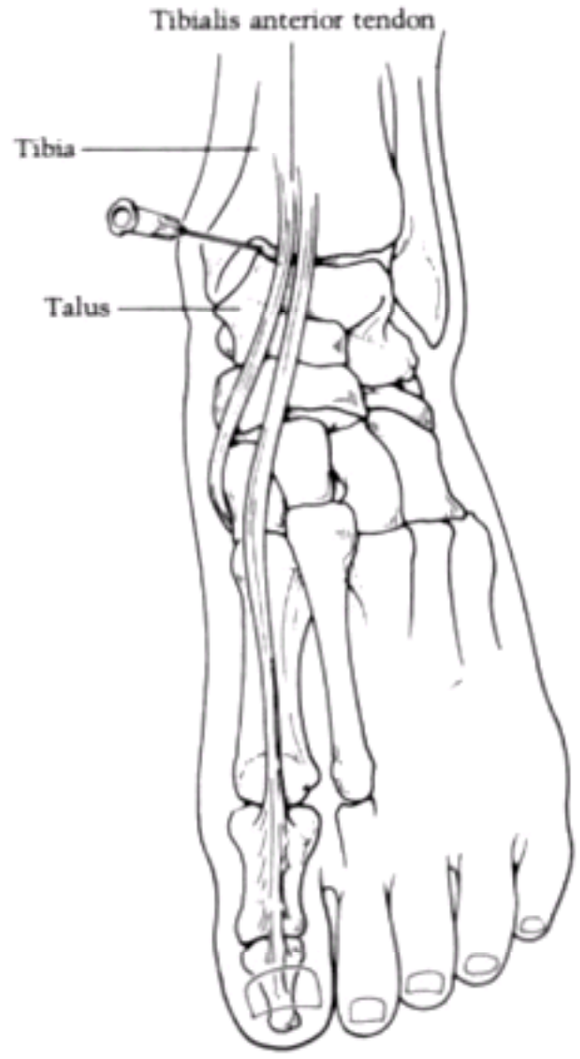


Figure 54-12 Landmarks for metatarsophalangeal and interphalangeal joints (see text). *(From Akins CM: Aspiration and injection of joints, bursae, and tendons. In Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. Reproduced by permission.)*

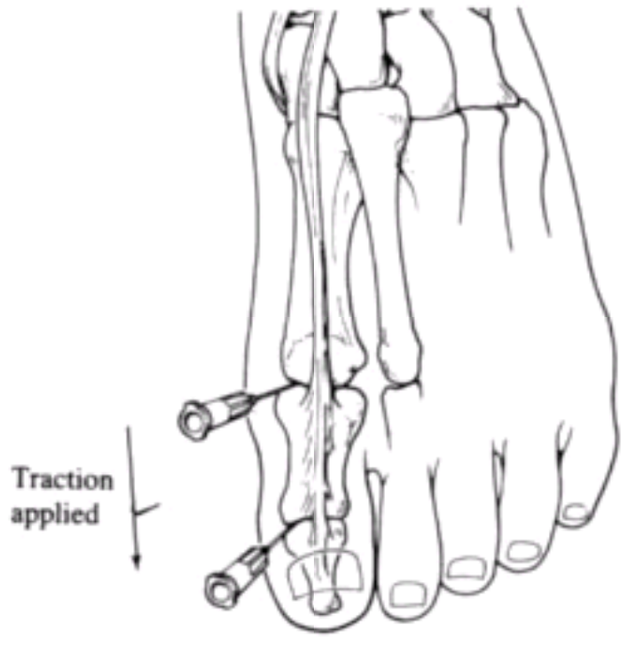
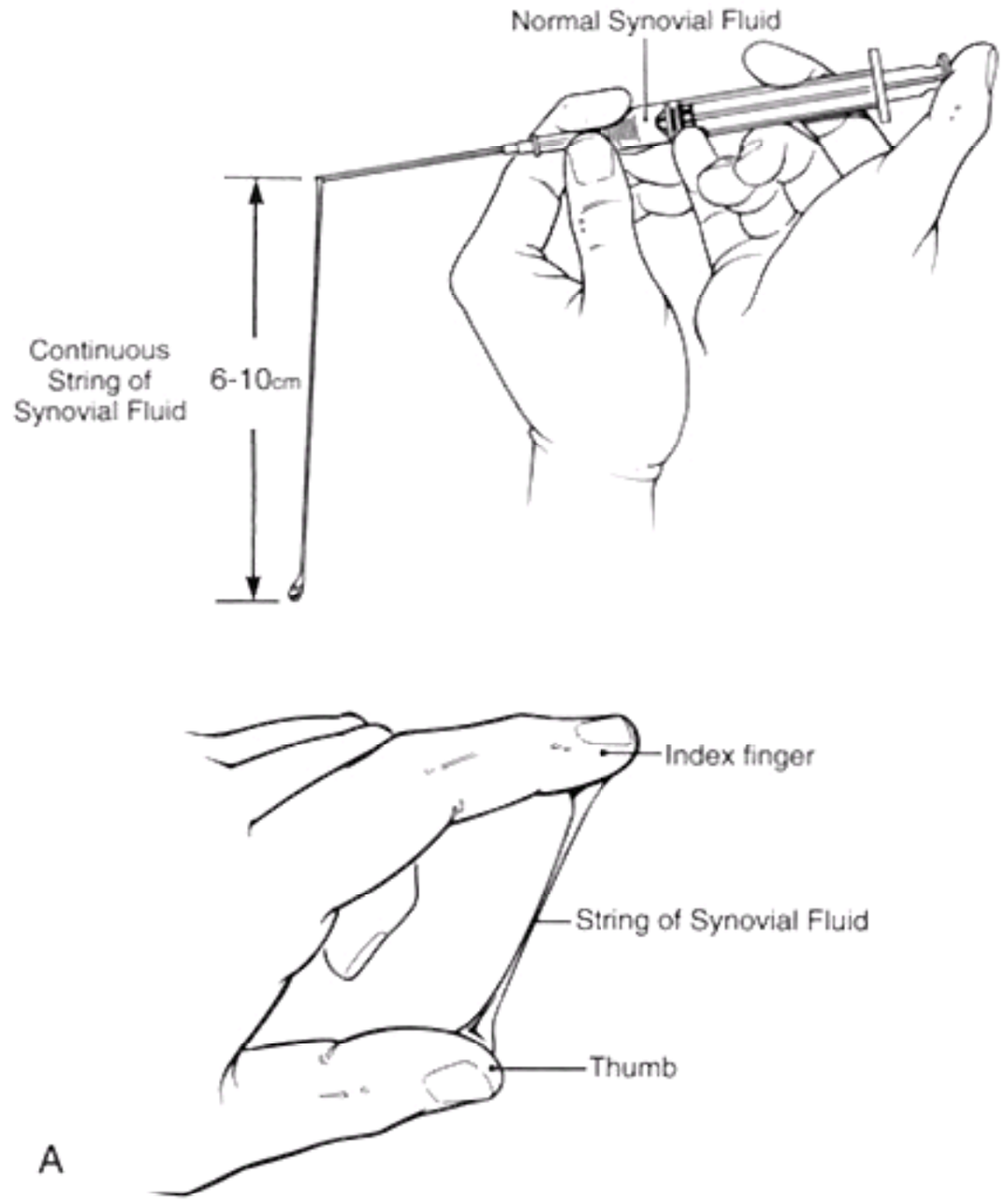


Figure 54-13 *A*, Ability of normal synovial fluid to form a long tenacious string. (Note: Gloves should be worn during procedure.) *B*, Bloody joint fluid from recent trauma forms a normal string sign.



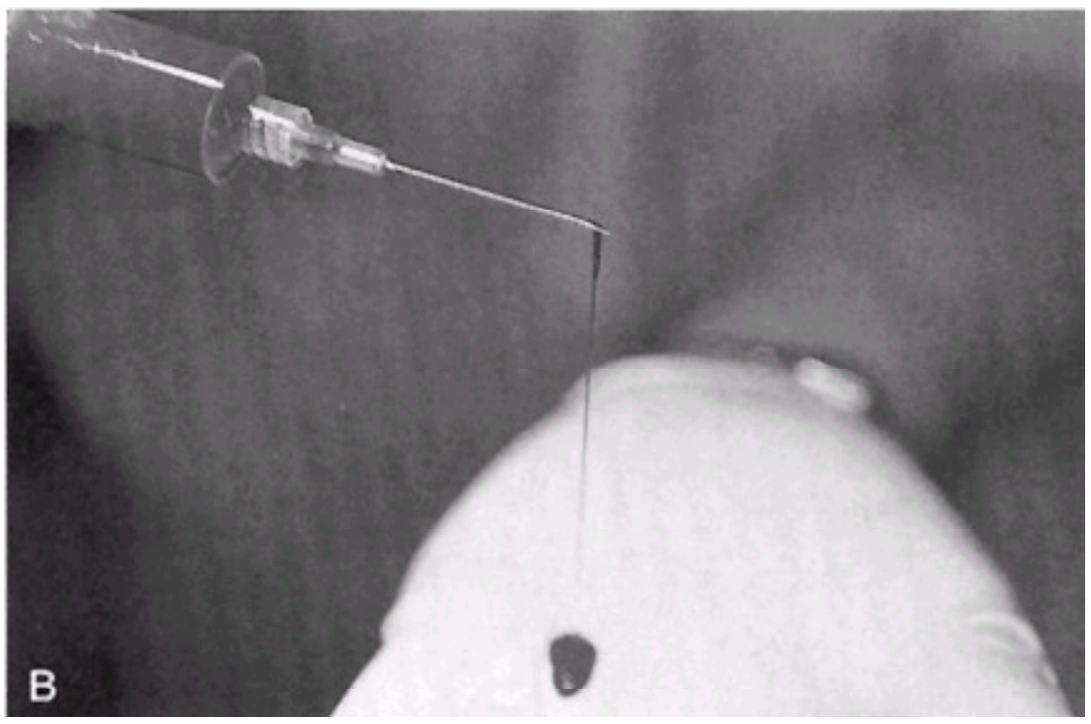


Figure 54-15 Synovial fluid with needle-shaped uric acid crystals *(arrows)*. Many crystals are characteristically engulfed by leukocytes. *(From Schumacher HR, Finkinson CA, Weiss JJ: Guidelines for obtaining and analyzing synovial fluid. ER Reports 4:40, 1983. Reproduced by permission.)*

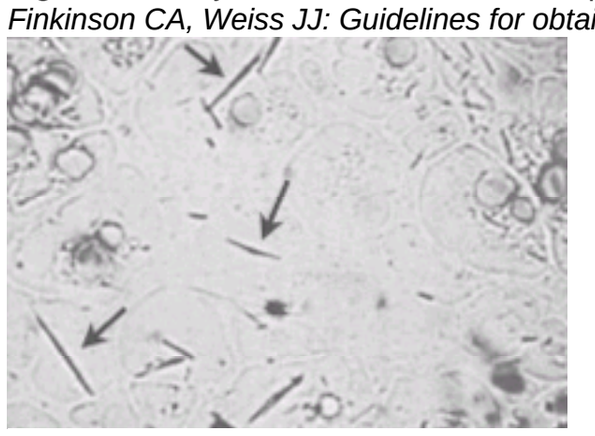


Figure 54-16 Synovial fluid with calcium pyrophosphate crystals. *(From Schumacher HR, Finkinson CA, Weiss JJ: Guidelines for obtaining and analyzing synovial fluid. ER Reports 4:40, 1983. Reproduced by permission.)*

Figure 54-17 Appearance of cartilage fragments. *(From Schumacher HR, Finkinson CA, Weiss JJ: Guidelines for obtaining and analyzing synovial fluid. ER Reports 4:40, 1983. Reproduced by permission.)*

Figure 54-18 Appearance of cholesterol crystals. *(From Schumacher HR, Finkinson CA, Weiss JJ: Guidelines for obtaining and analyzing synovial fluid. ER Reports 4:40, 1983. Reproduced by permission.)*

Figure 54-19 Microscopic and gross appearance of fat globules in synovial fluid. This finding suggests a fracture extending into the joint. *(From Schumacher HR, Finkinson CA, Weiss JJ: Guidelines for obtaining and analyzing synovial fluid. ER Reports 4:40, 1983. Reproduced by permission.)*

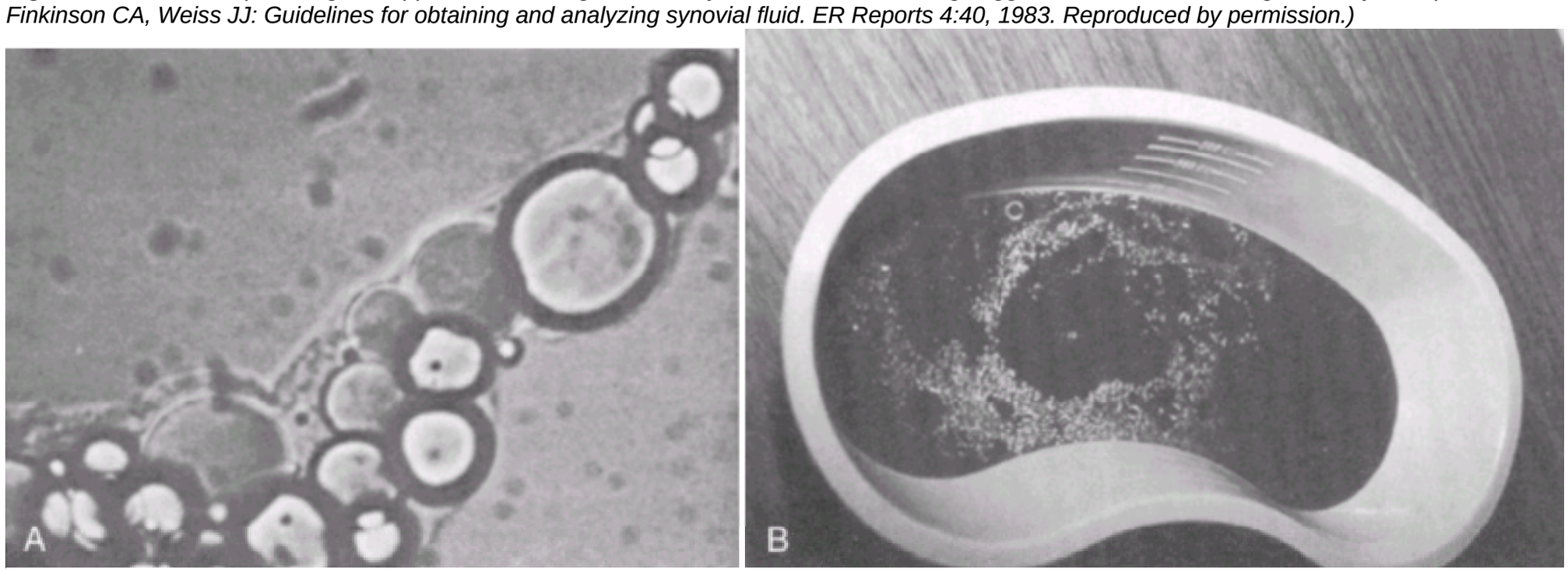


Figure 55-1 The Stryker 295 intracompartmental pressure monitor system. *(Courtesy of Stryker Instruments, Kalamazoo, MI.)*

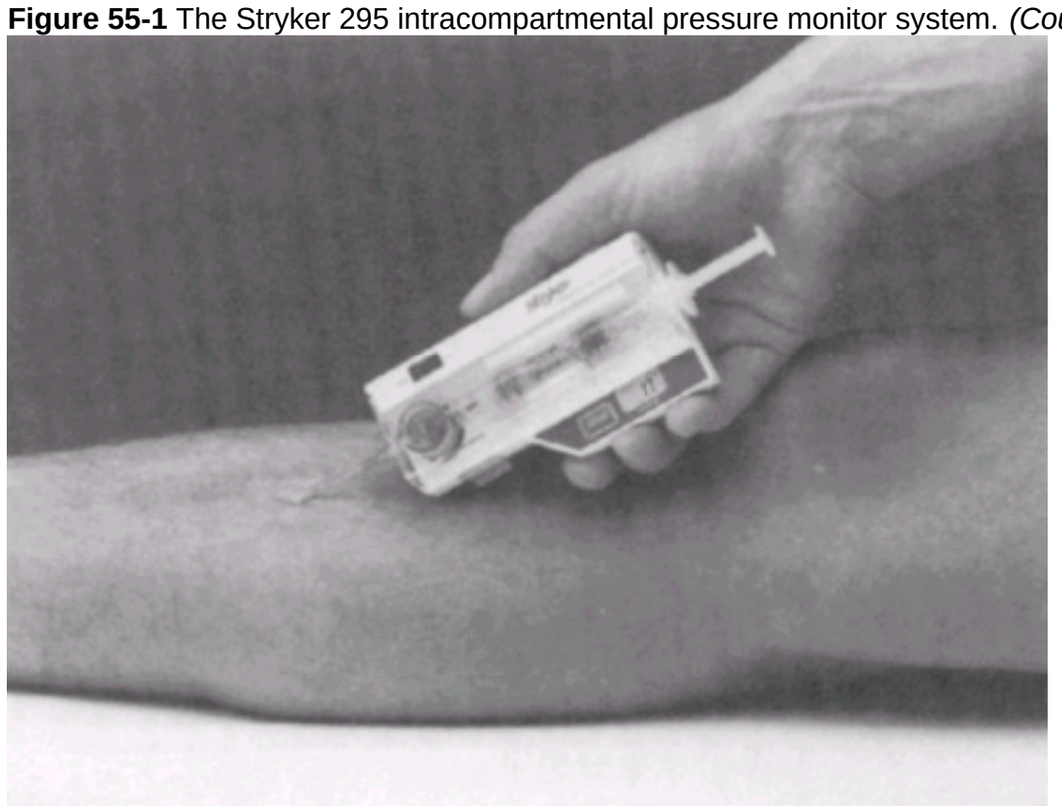


Figure 55-2 The HANDY electronic transducer-tipped catheter system *(Courtesy of Mammendorfer Institute for Physics and Medicine LTD. Munich, Germany.)*

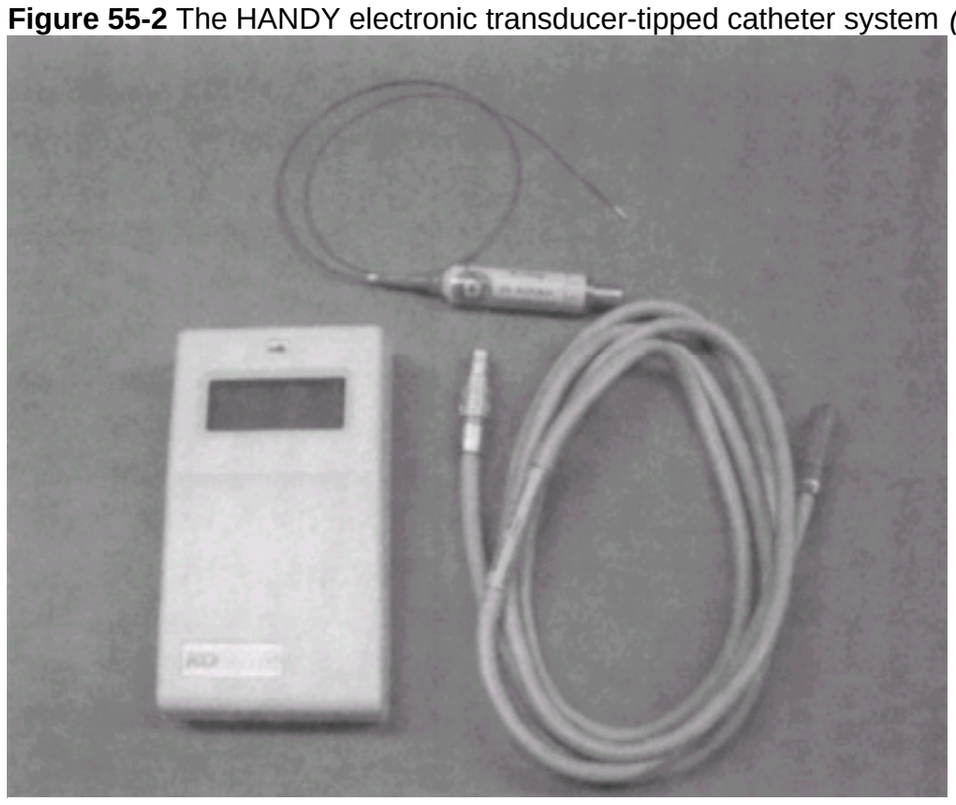


Figure 55-3 The diagnosis of a compartment syndrome is not always straightforward. This young man presented in coma from a drug overdose and had been lying on his arm for a number of hours. The entire arm was swollen and rhabdomyolysis was correctly suspected and treated *(A)*. Because of the coma he was unable to voice any complaint of pain. When he awakened 20 hours later the pain was severe, and compartment pressures demonstrated the need for fasciotomy *(B)*.

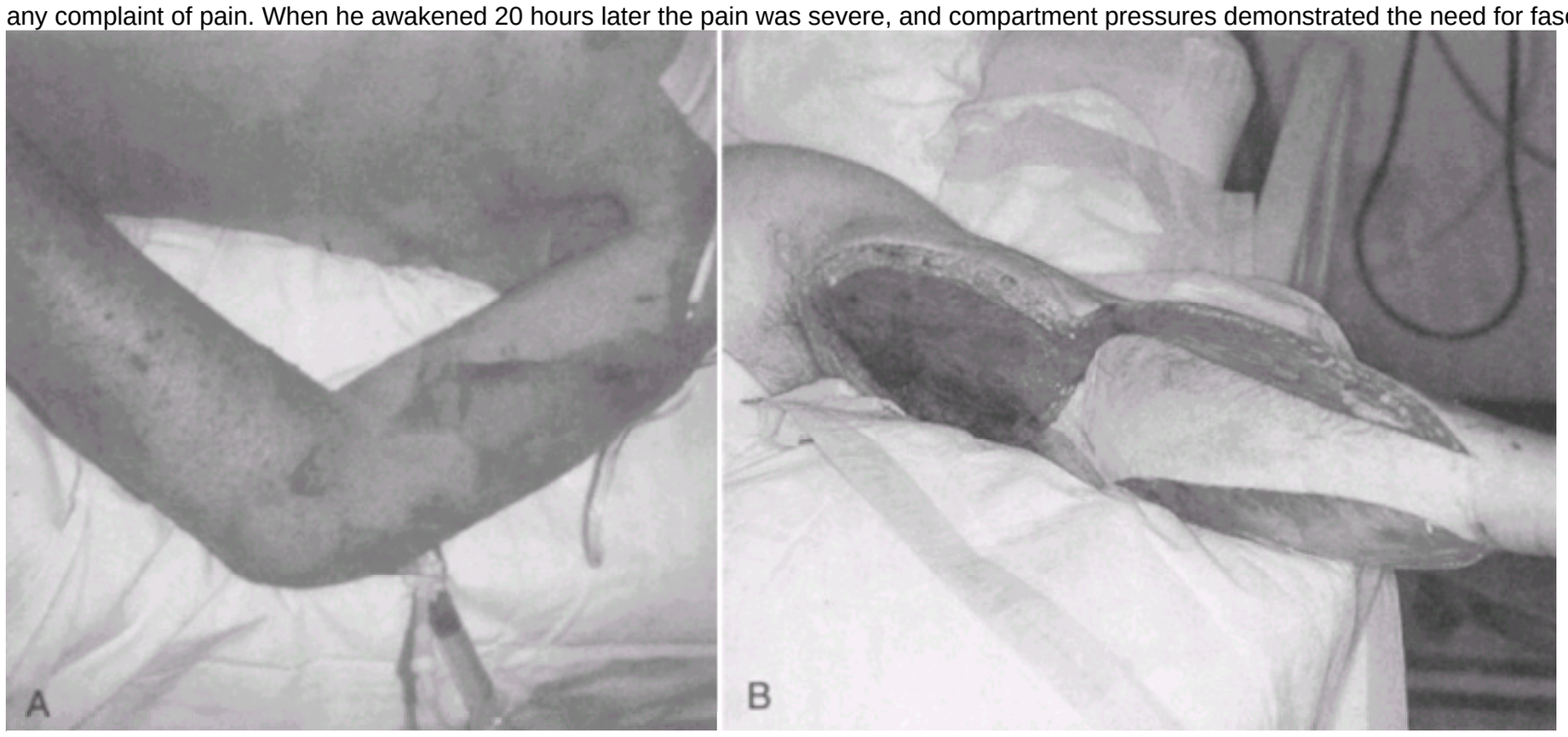


Figure 55-4 Algorithm for management of a patient with suspected compartment syndrome. Pressure thresholds (*) are based on published case series. *Clinical correlation is paramount to proper interpretation of compartment pressure measurements. (From Rorabeck CH: Compartment syndromes. In Browner BD, Jupiter JB, Levine AM, Trafton PG (eds): Skeletal Trauma: Fractures, Dislocations, Ligamentous Injuries, vol 1, 2nd ed. Philadelphia, WB Saunders, 1992, p 290.)*

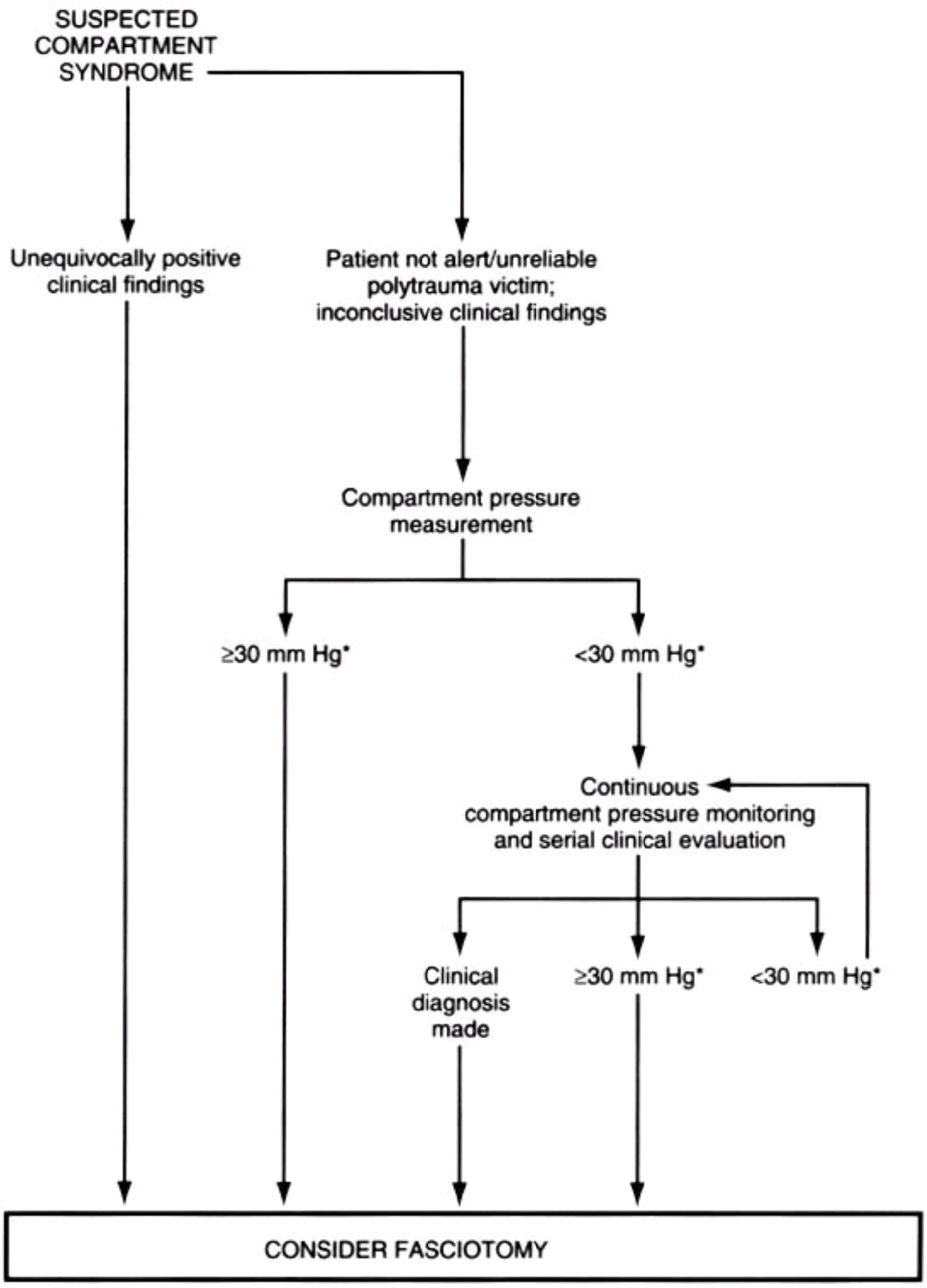


Figure 55-5 *A* and *B*, Mercury monitor technique for compartmental pressure monitoring. *(From Whitesides TE, Haney TC, Morimoto K, et al: Tissue pressure measurements as a determinant for the need of fasciotomy. Clin Orthop 113:43, 1975.)*

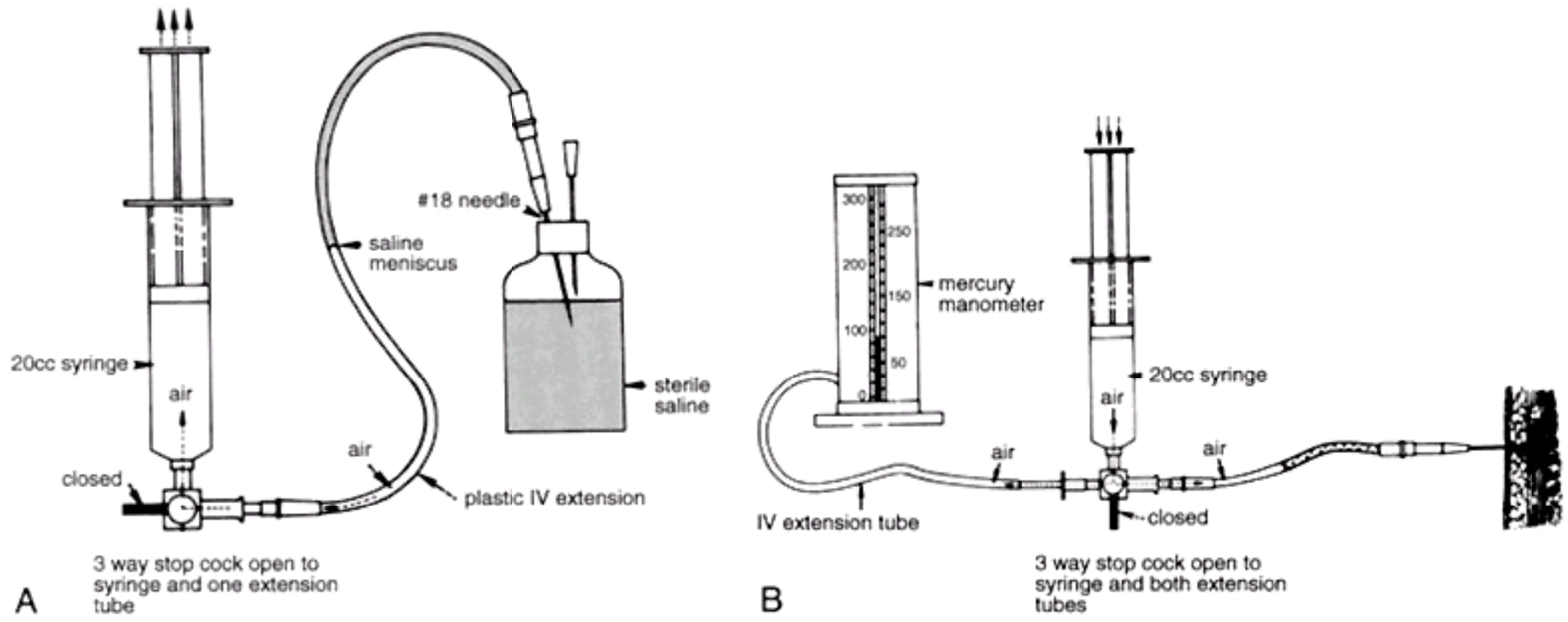


Figure 55-6 The arterial line system for compartmental pressure measurement. *(From Rorabeck CH: Compartment syndromes. In Browner BD, Jupiter JB, Levine AM, Trafton PG (eds): Skeletal Trauma: Fractures, Dislocations, Ligamentous Injuries, vol 1, 2nd ed. Philadelphia, WB Saunders, 1992, p 290.)*

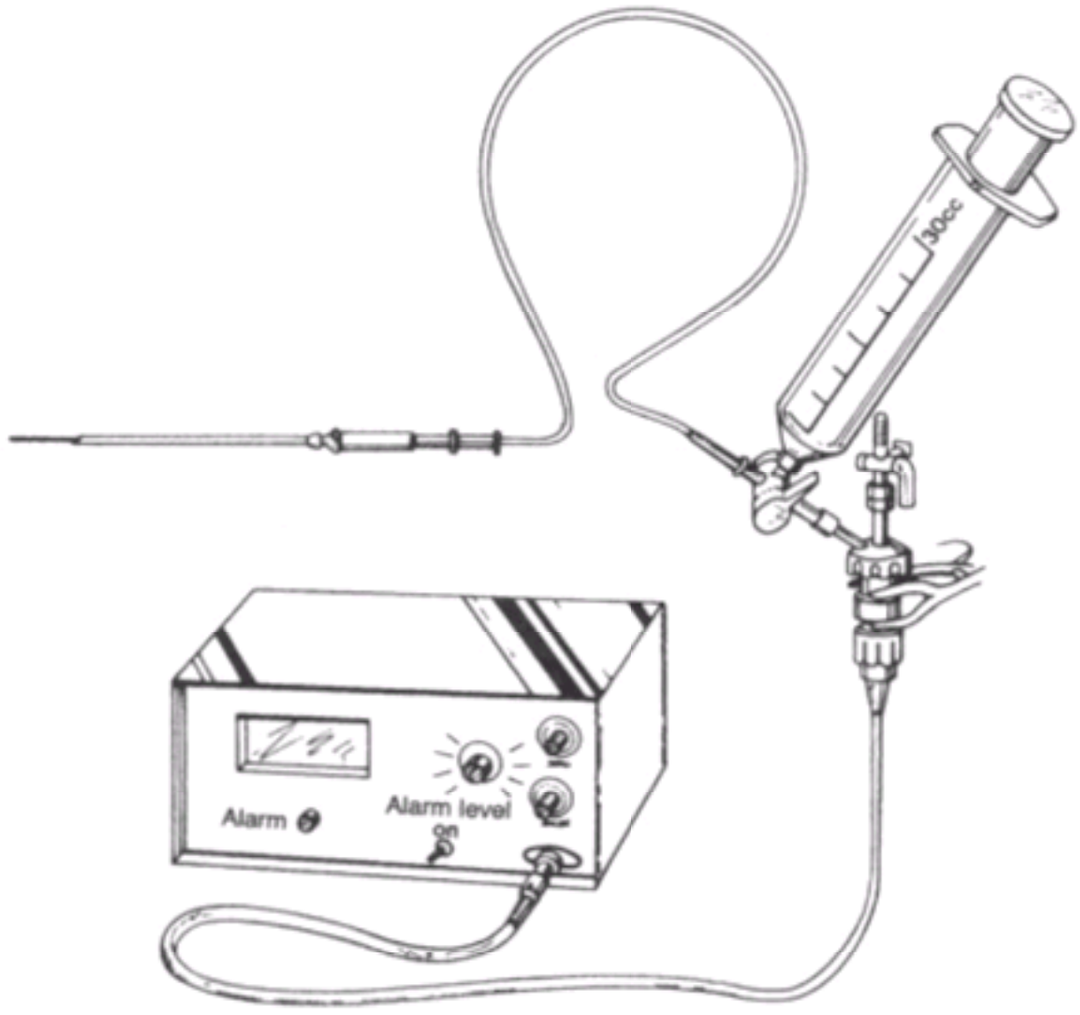
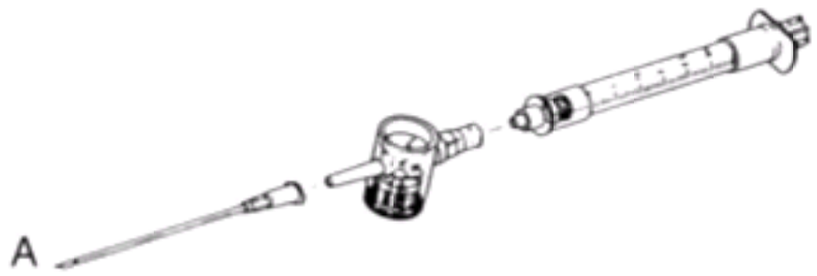
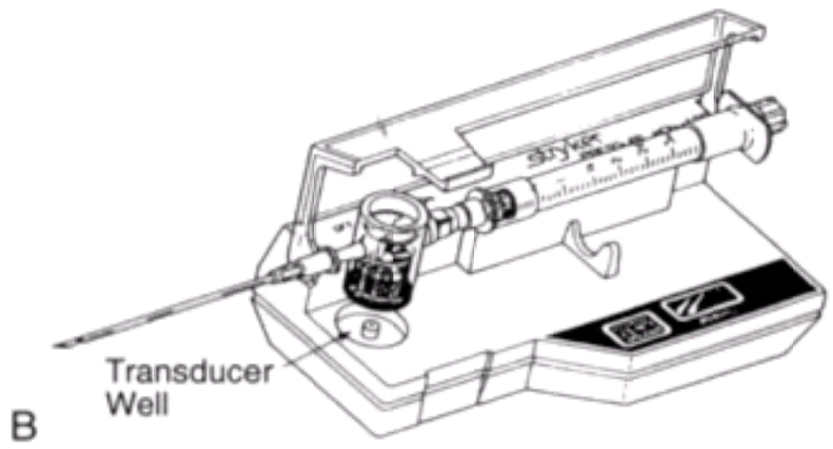


Figure 55-7 *A–C*, The Stryker 295 intracompartmental pressure monitor system assembly. *(Courtesy of Stryker Instruments, Kalamazoo, MI.)*





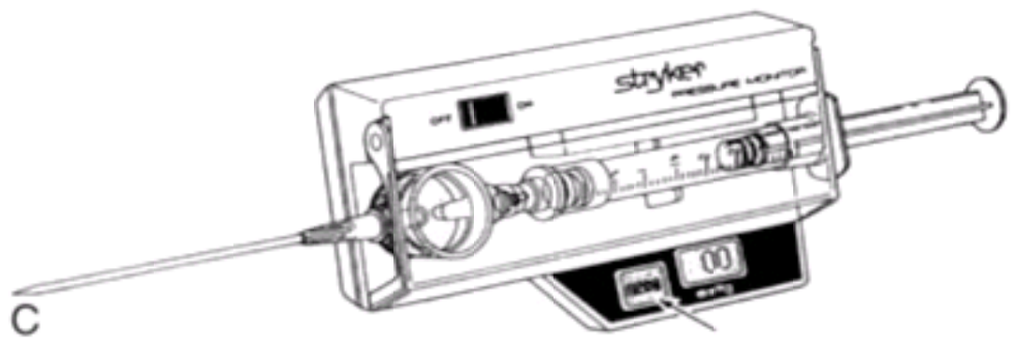


Figure 55-8 Fascial compartments of the lower leg with enclosed muscle groups (insert upper left): (1) anterior; (2) lateral; (3) superficial posterior; and (4) deep posterior compartments.

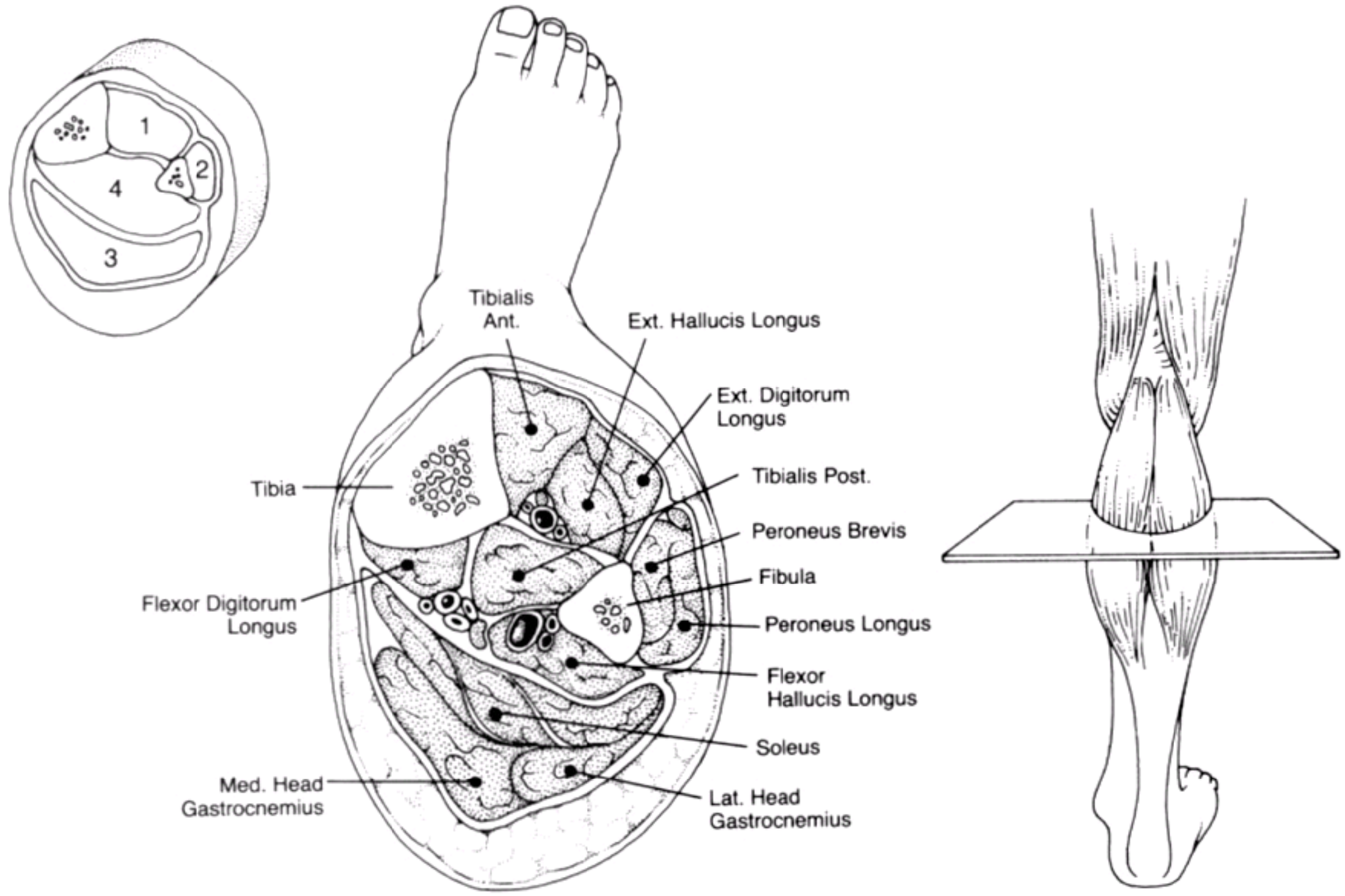


Figure 55-9 Anterior compartment syndrome of the lower leg. Suggested needle entry point is indicated by the small circle *(A)*. The needle should be inserted (*) to a depth of 1 to 3 cm *(B)*. *(Modified with permission from Matsen FA (ed): Compartmental Syndromes. New York, Grune & Stratton, 1980, p 91.)*

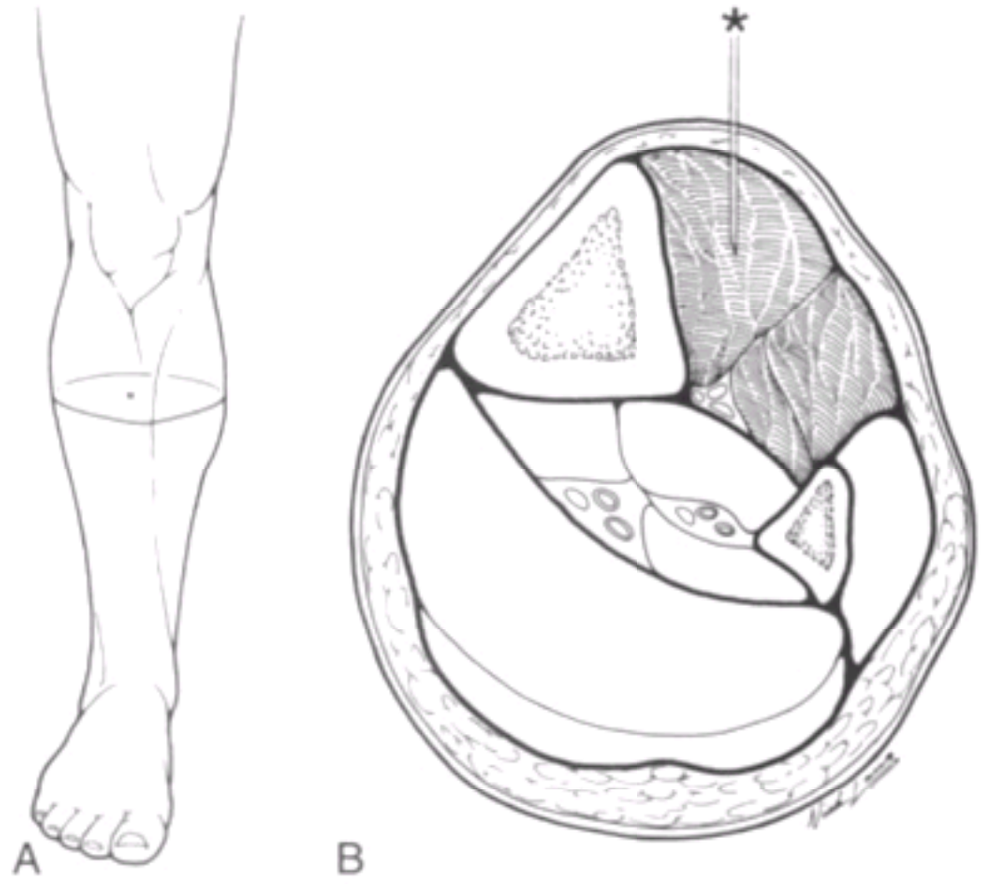


Figure 55-10 Deep posterior compartment syndrome of the lower leg. Suggested needle entry point indicated by the small circle *(A)*. The needle should be inserted (*) to a depth of 2 to 4 cm *(B)*. *(Modified with permission from Matsen FA (ed): Compartmental Syndromes. New York, Grune & Stratton, 1980, p 92.)*

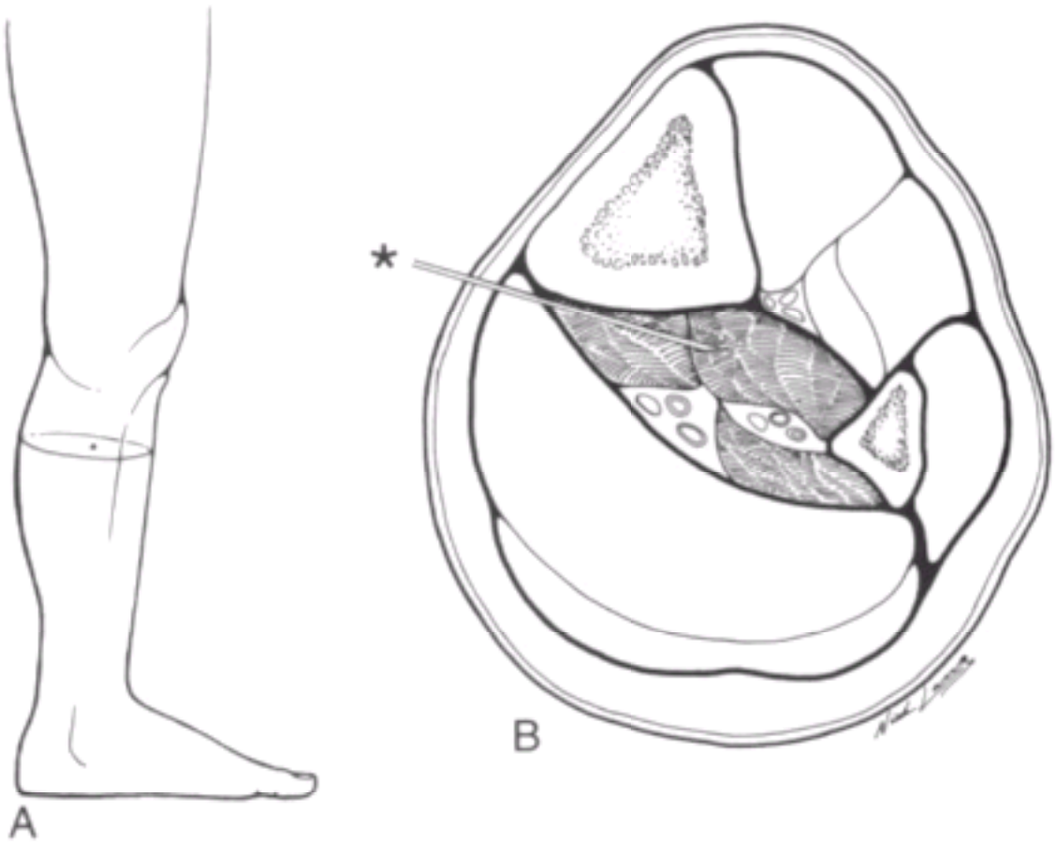


Figure 55-11 Lateral compartment syndrome of the lower leg. Suggested needle entry point indicated by the small circle *(A)*. The needle should be inserted (*) to a depth of 1 to 1.5 cm *(B)*. *(Modified with permission from Matsen FA (ed): Compartmental Syndromes. New York, Grune & Stratton, 1980, p 92.)*

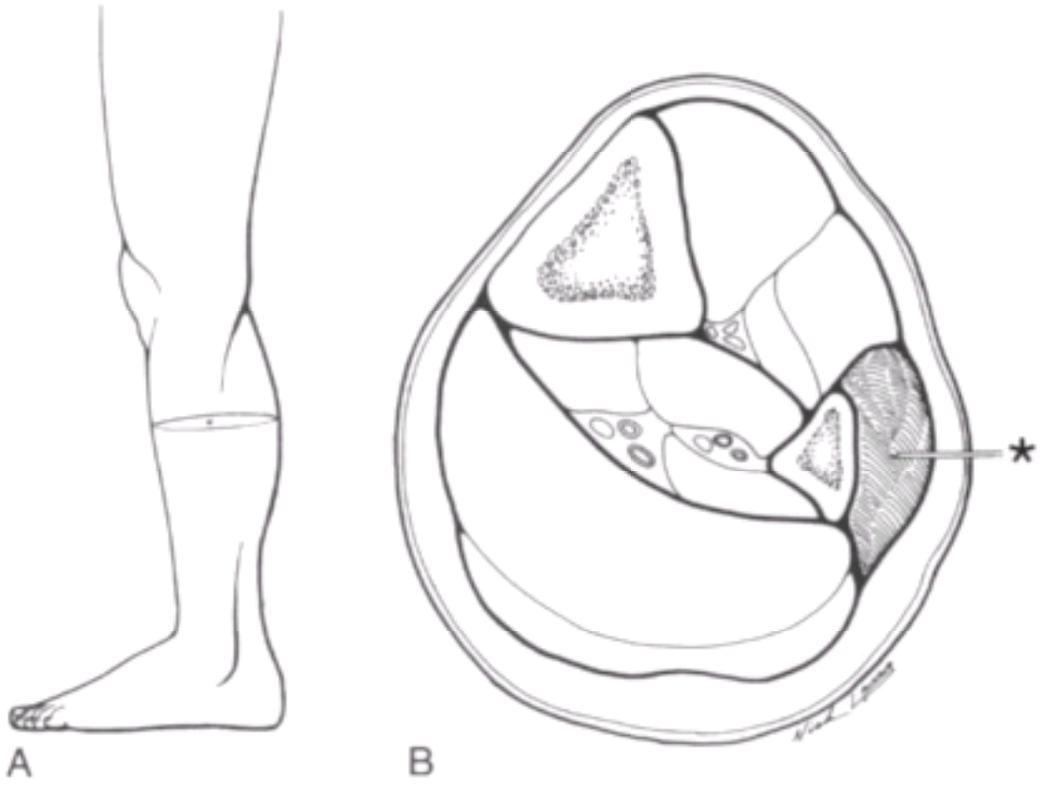


Figure 55-12 Superficial posterior compartment syndrome of the lower leg. Suggested needle entry point indicated by the small circle *(A)*. The needle should be inserted (*) to a depth of 2 to 4 cm *(B)*. *(Modified with permission from Matsen FA (ed): Compartmental Syndromes. New York, Grune & Stratton, 1980, p 92.)*

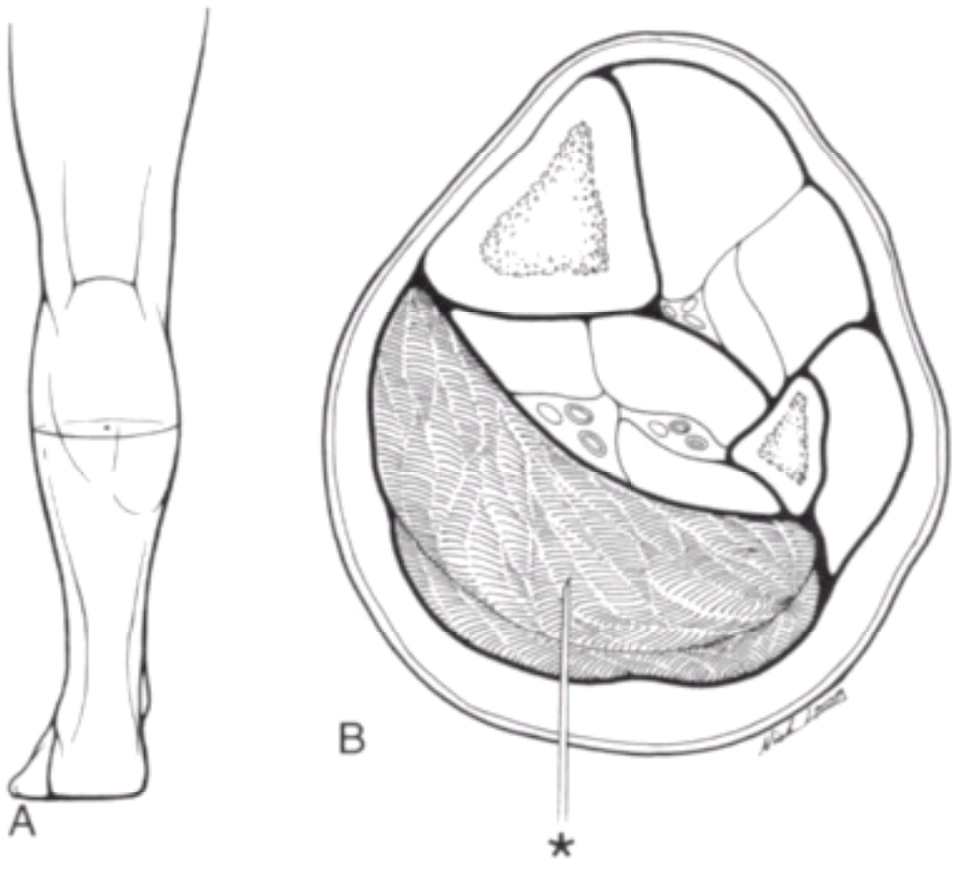


Figure 55-13 Level of needle insertion of the forearm *(A)* with cross section through the upper third of the forearm *(B)* demonstrating the three forearm compartments (volar, dorsal, mobile wad). *(With permission from Green DP (ed): Operative Hand Surgery. New York, Churchill Livingstone, 1982.)*

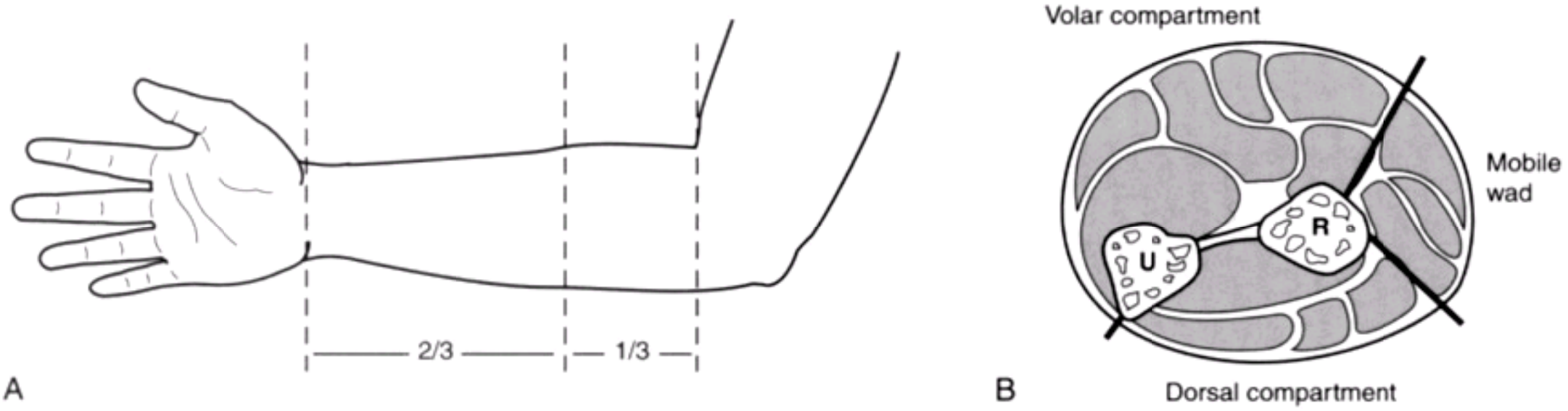


Figure 55-14 Volar compartment syndrome of the forearm. Suggested needle entry point indicated by the small circle on the volar forearm *(A)*. The needle should be inserted (*) to a depth of 1 to 2 cm *(B)*. *(Modified with permission from Matsen FA (ed): Compartmental Syndromes. New York, Grune & Stratton, 1980, p 93.)*

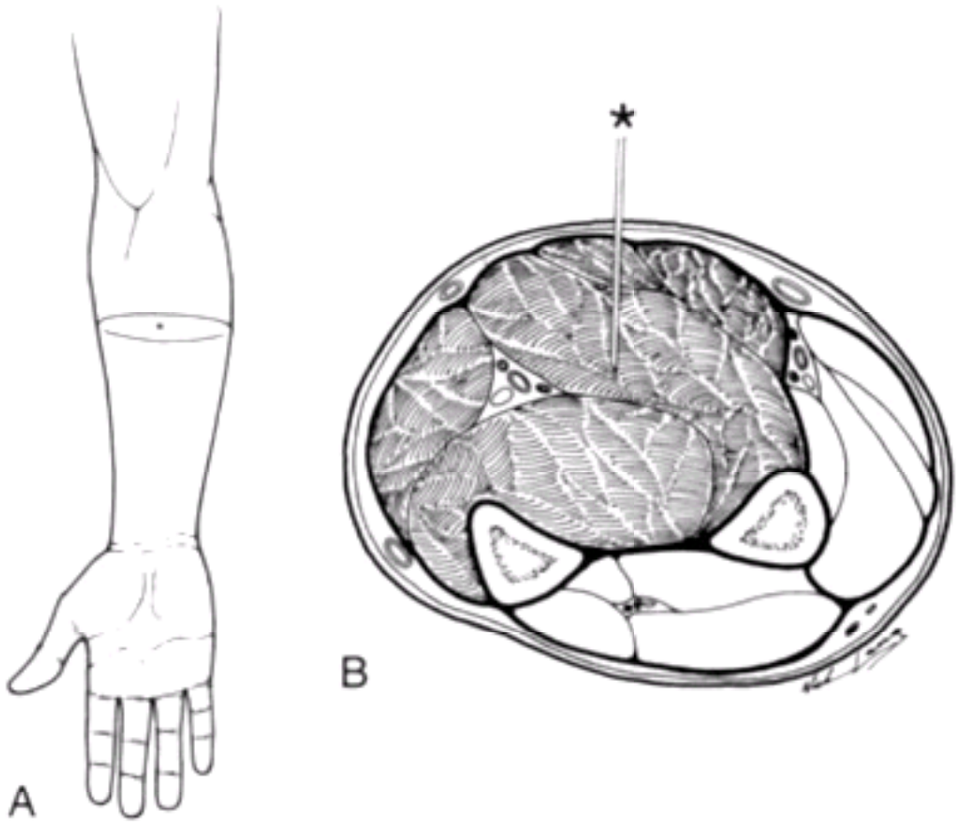


Figure 55-15 Dorsal compartment syndrome of the forearm. Suggested needle entry point indicated by the small circle *(A)*. The needle should be inserted (*) to a depth of 1 to 2 cm *(B)*. *(Modified with permission from Matsen FA (ed): Compartmental Syndromes. New York, Grune & Stratton, 1980, p 93.)*

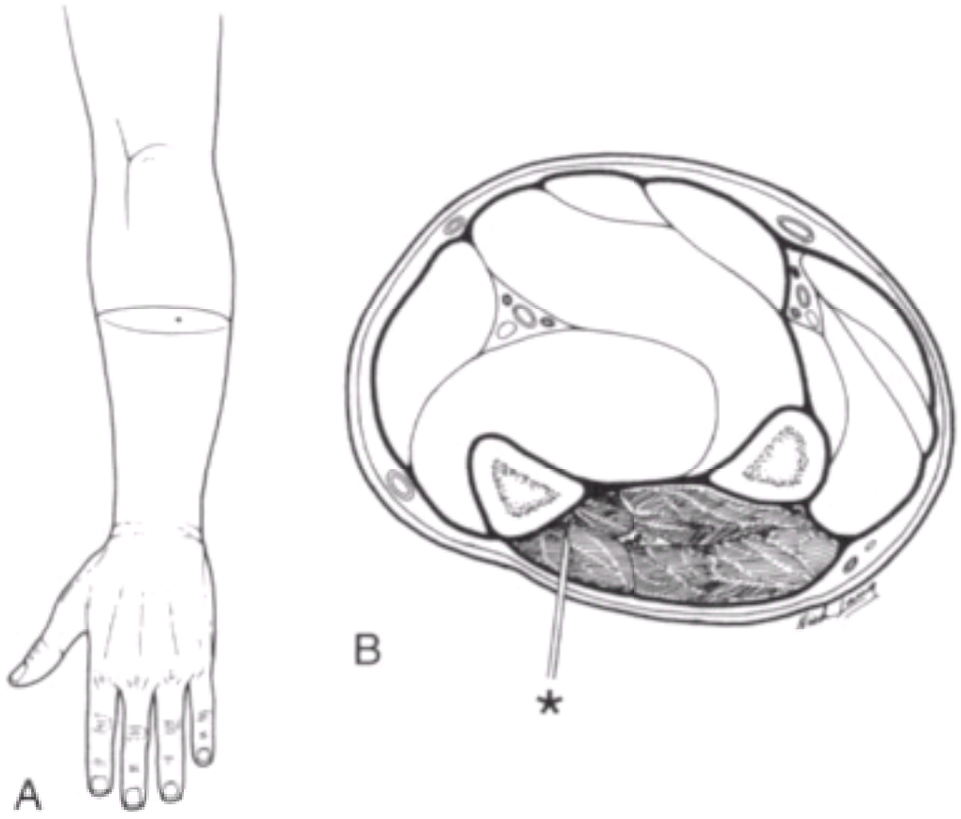


Figure 55-16 Mobile wad compartment syndrome of the forearm. Suggested needle entry point indicated by the small circle on lateral (radial) aspect of proximal fore arm *(A)*. The needle should be inserted (*) to a depth of 1 to 1.5 cm *(B)*. *(Modified with permission from Matsen FA (ed): Compartmental Syndromes. New York, Grune & Stratton, 1980, p 93.)*

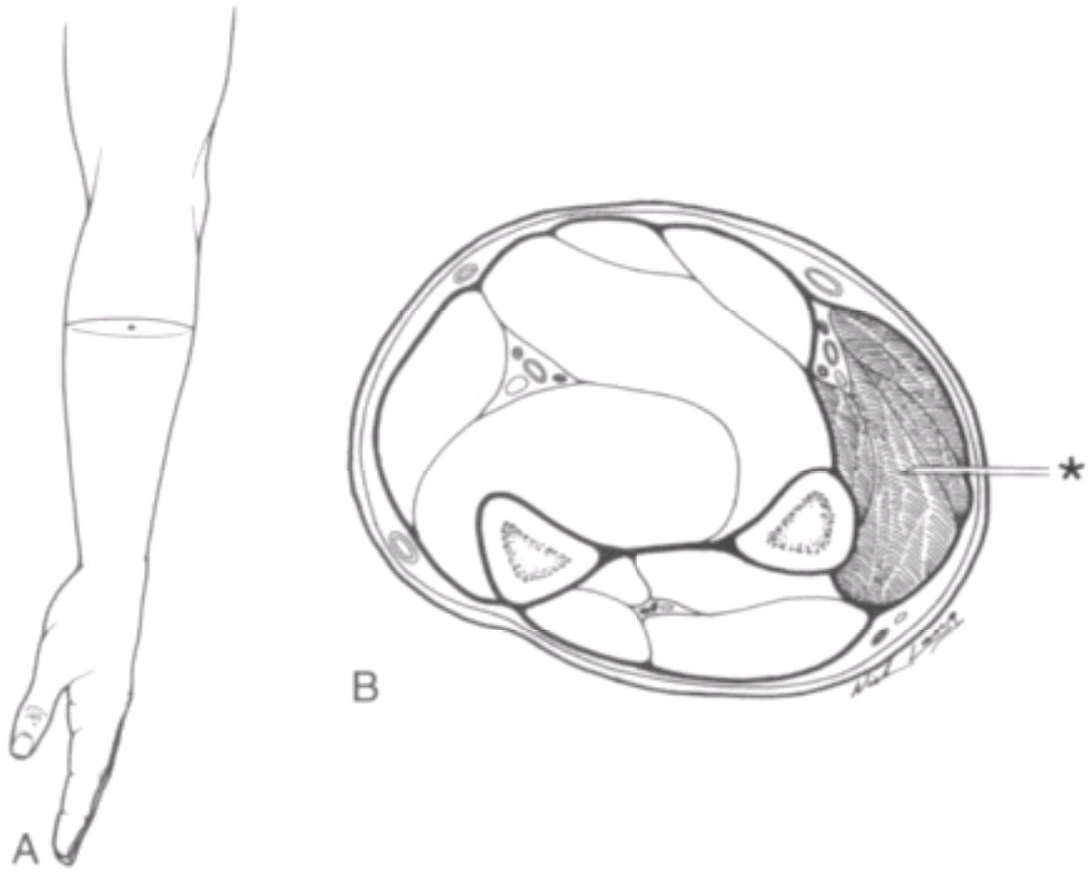


Figure 55-17 Gluteal compartment syndrome. Suggested entry points are indicated by the small circles *(A)*. The needle should be inserted to a depth of 4 to 8 cm depending on which compartment is being measured. Needle tips (*) shown entering muscle compartments *(B)*. *(Modified with permission from Owen CA, Moody PR, Mubarak SJ, et al: Gluteal compartment syndromes. Clin Orthop 132:57, 1978.)*

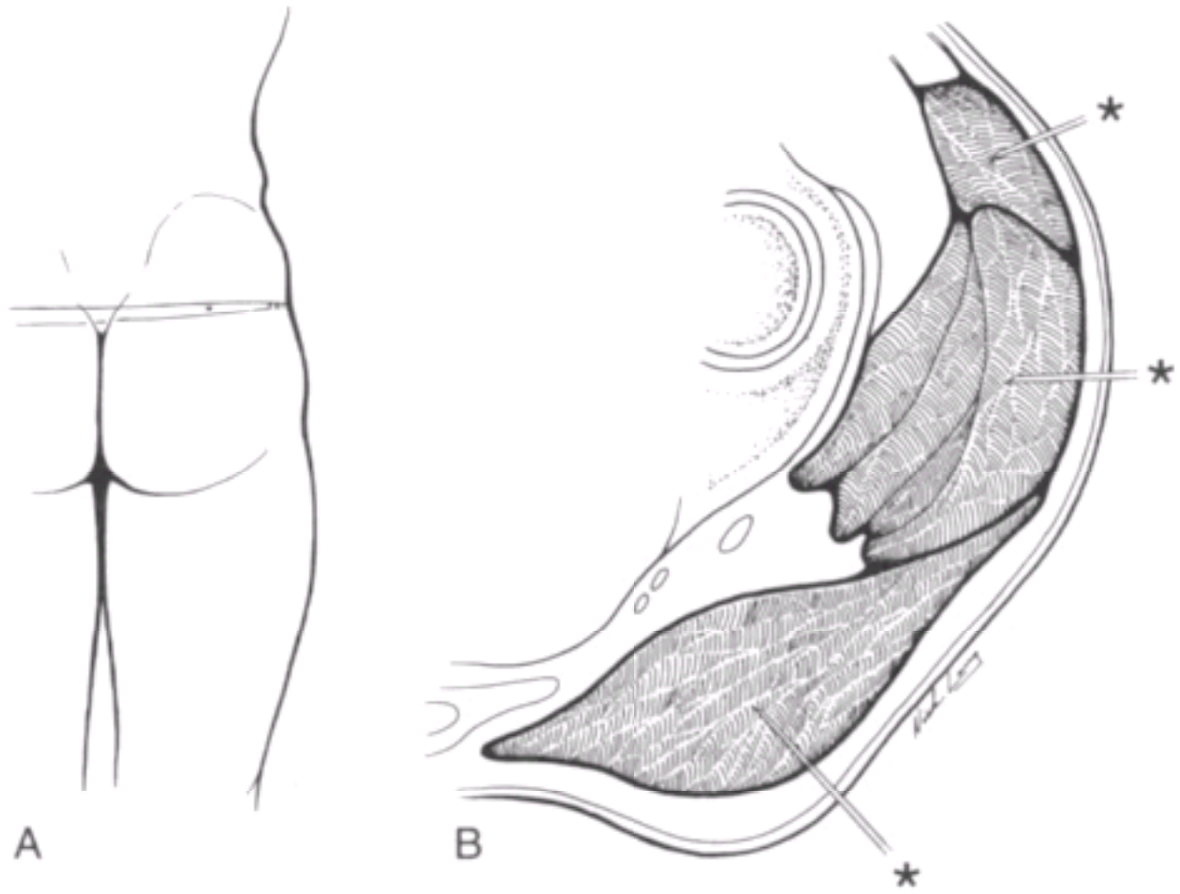


Figure 55-18 The compartments of the foot. *(From Mubarak SJ, Hargens AR: Compartment Syndromes and Volkmann's Contracture. Philadelphia, WB Saunders, 1981.)*

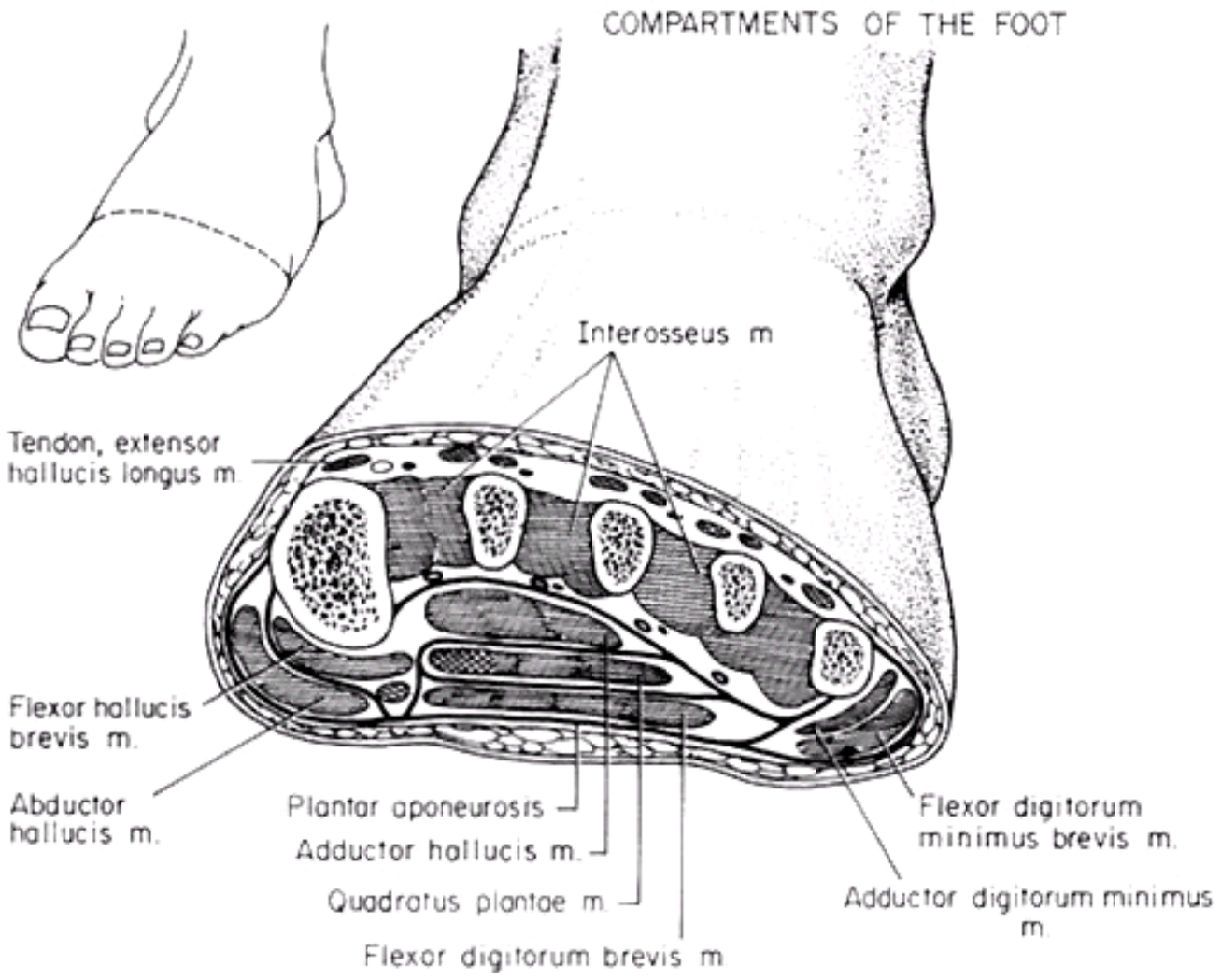


Figure 55-19 Compartment syndromes of the foot. Suggested needle pathways (*) to measure intracompartmental pressures: *A,* Medial. *B,* Lateral. *C,* Interosseous compartments. The central compartment is surrounded by these compartments. *(Modified from Myerson M: Acute compartment syndromes of the foot. Bull Hosp Jt Dis 47:251, 1987.)*

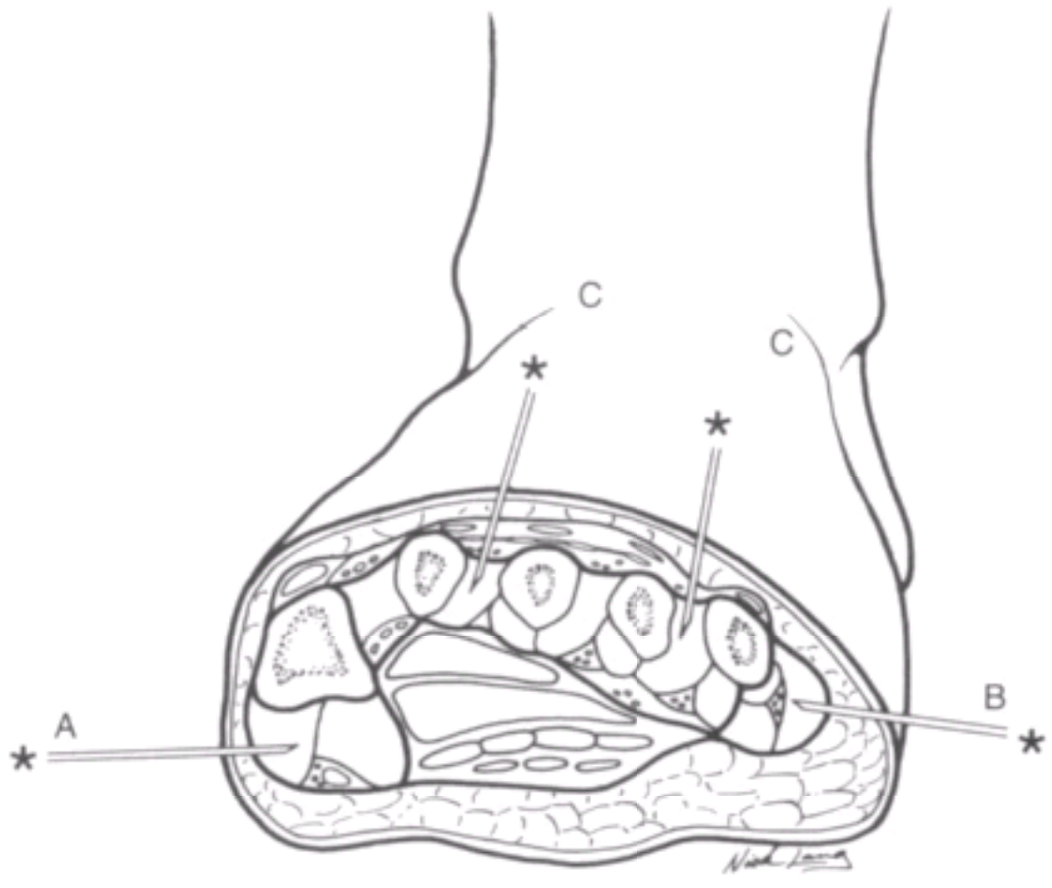


Figure 56-1 Technique for obtaining anesthesia before performing a dorsal slit.

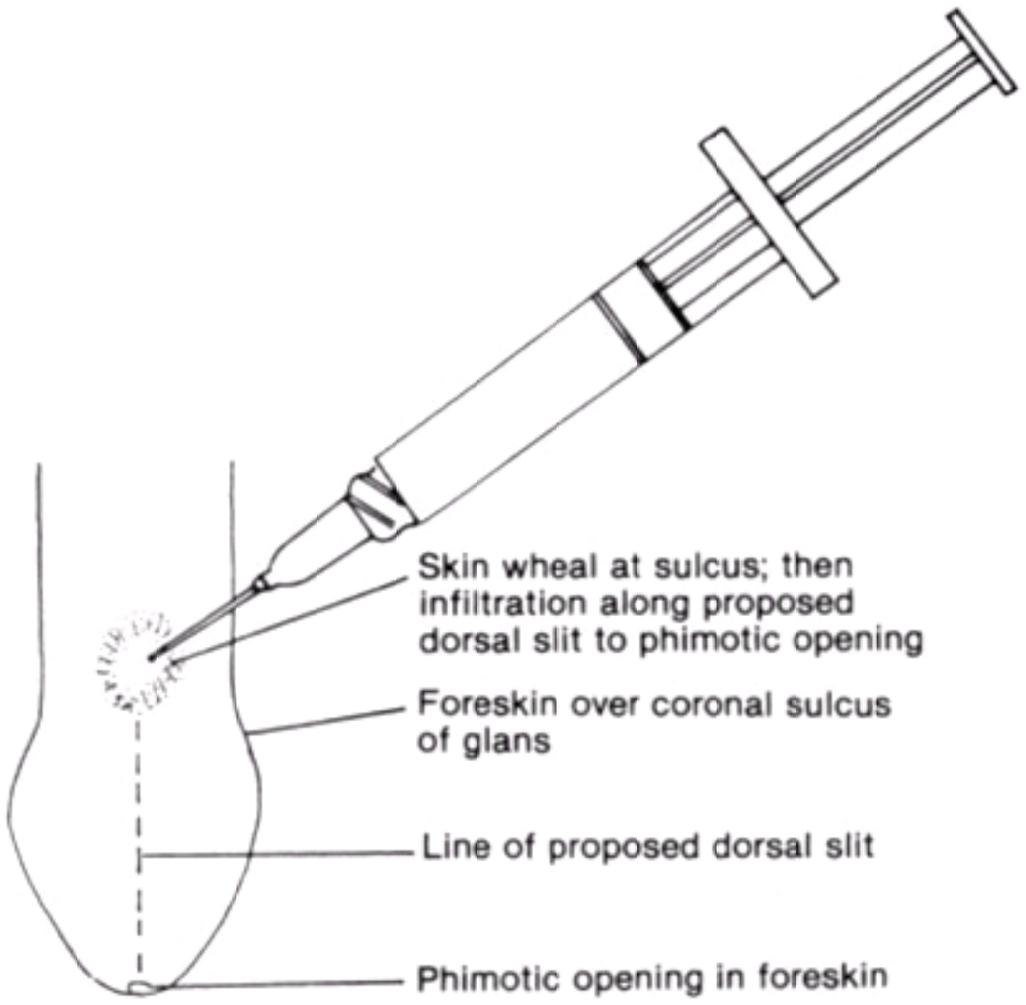


Figure 56-2 *A*, Dorsal nerve block at the base of the penis will provide anesthesia of only the dorsum of the penis. *B,* Circumferential subcutaneous lidocaine infiltration for a ("ring") field block at the base of the penis can provide anesthesia to the entire distal penis. *(A from Soliman MG, Tremblay NA: Nerve block of the penis for postoperative pain relief in children. Anesth Analg 57:495, 1978. Reproduced by permission.)*

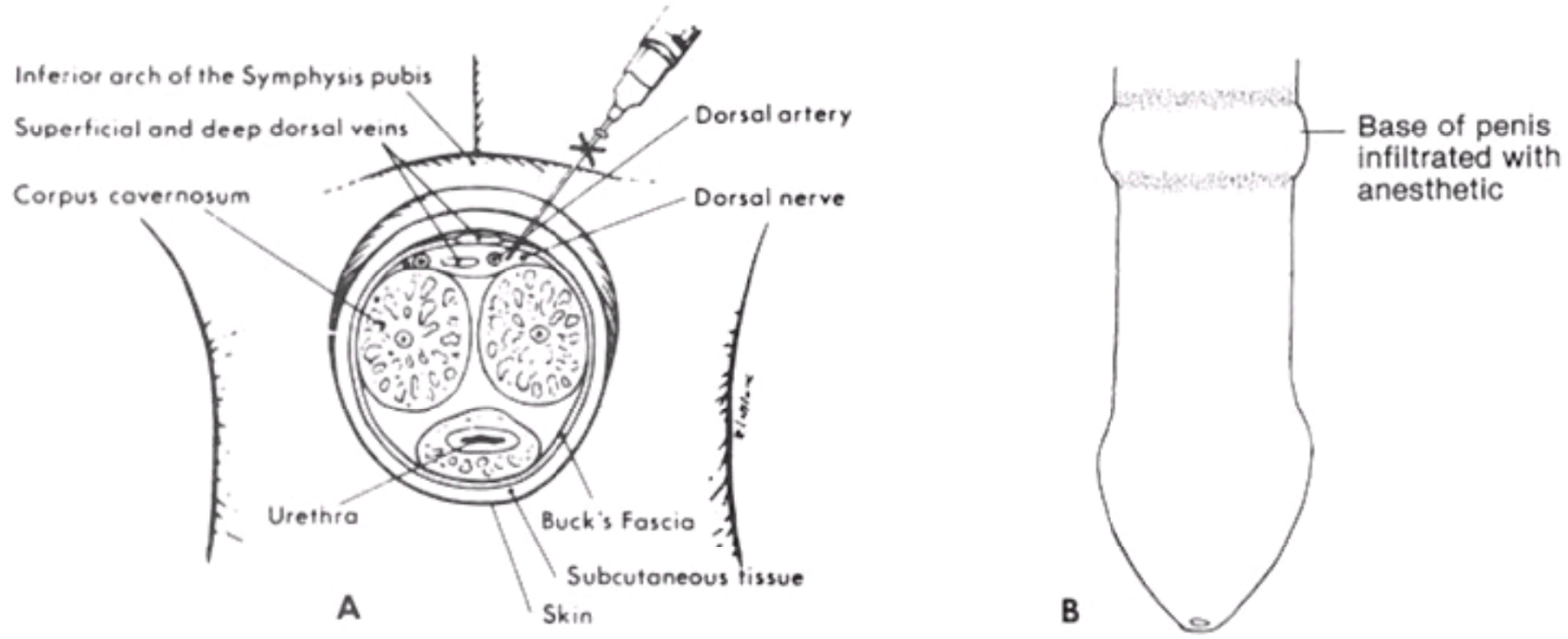


Figure 56-3 Placement of hemostat for treatment of phimosis. The "tenting up" of the foreskin in this manner proves that the tip of the hemostat is *not* in the urethra or under the glans. Once properly placed between the glans and overlying foreskin, the instrument is closed to crush the area of the foreskin to be cut.

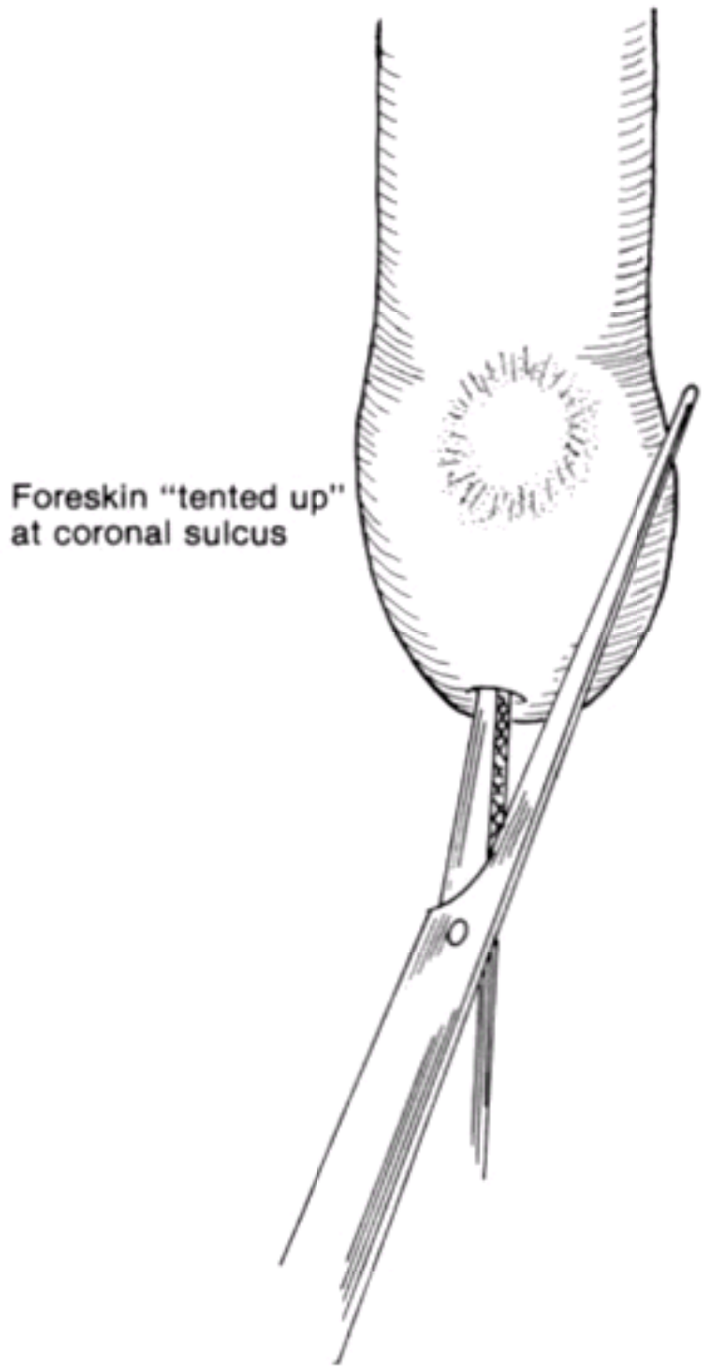


Figure 56-4 Treatment of phimosis. *A,* Dorsal slit in phimotic foreskin. Exposed glans is shaded. A single (dorsal) lengthwise incision has been made through crushed tissue (a^1, outer layer of foreskin; *a,* inner layer of foreskin). *B*, Cut edges of foreskin drawn back around glans penis. First, a^1 is sutured to *a*, then the remainder of the cut edges are sewn together for hemostasis. *C,* Final "beagle-ear" deformity of ventral transposed foreskin after the dorsal slit procedure has been completed. *D*, Postoperative appearance of dorsal slit.

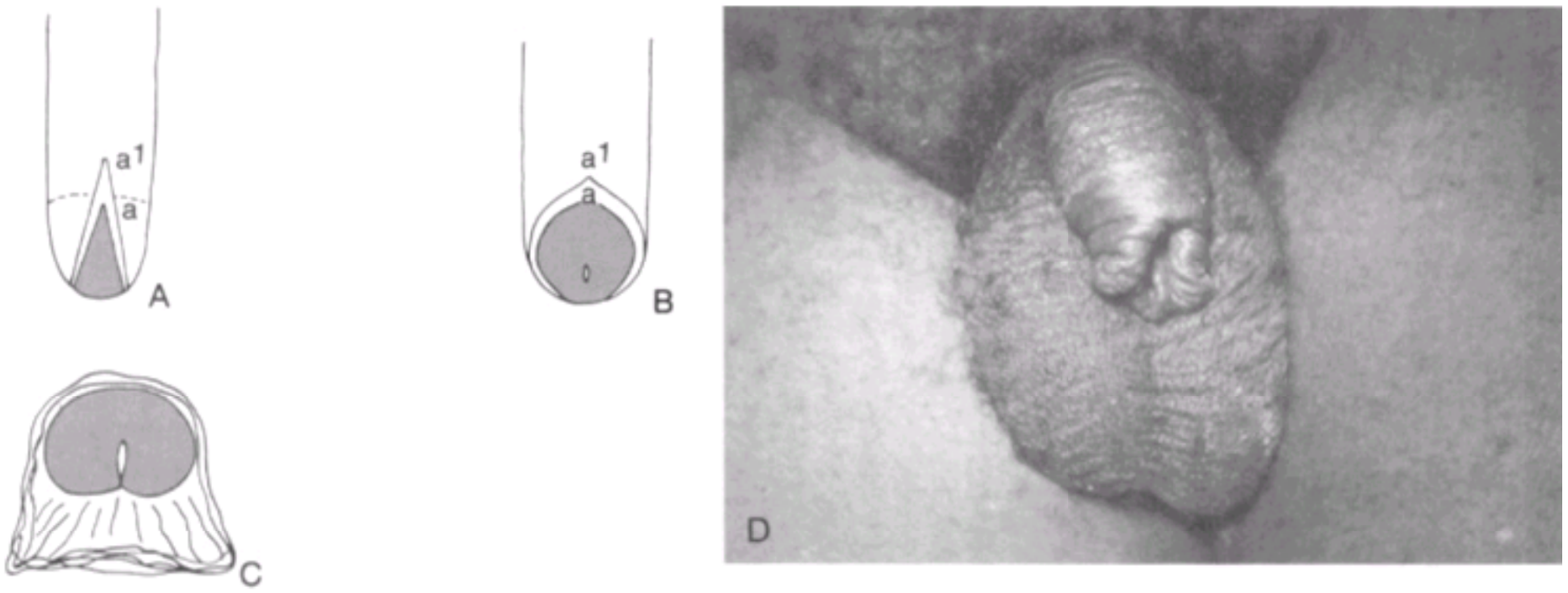
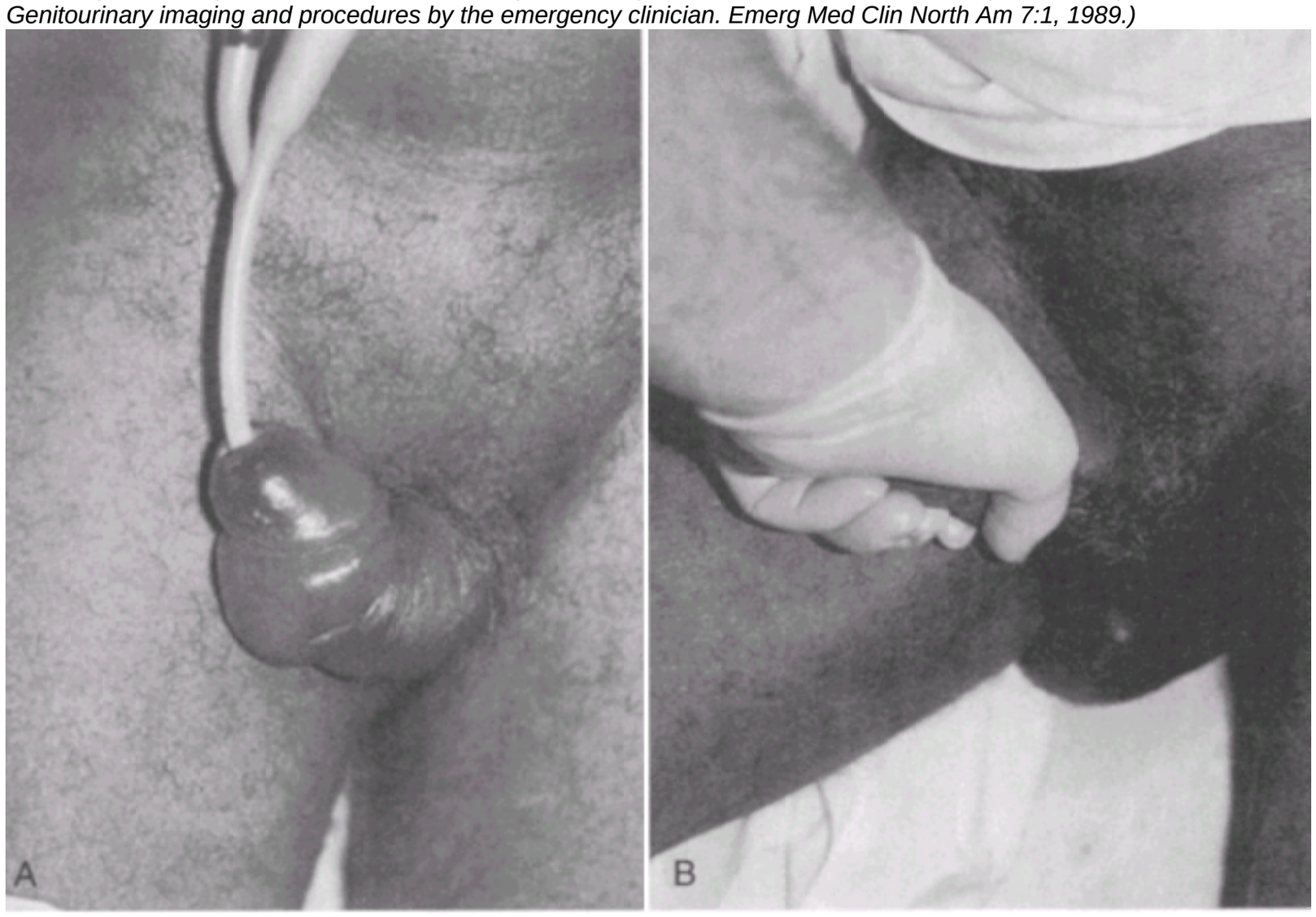


Figure 56-5 *A,* Paraphimosis, pictured here, may be mistaken for penile trauma, angioedema, or infection. The cause of paraphimosis in this case was failure to replace the foreskin following a catheter change in a demented nursing home patient. Prior to reduction, the catheter is usually removed. When the edema is minimal, the catheter may be left in place during reduction. *B,* Manual compression of the foreskin, or wrapping the penis with an elastic bandage for a few minutes, may reduce edema before a reduction of the paraphimosis is attempted. *C,* Technique for reduction of paraphimosis. Gentle, steady pressure is placed on the glans with the tips of the thumbs while gentle traction is applied to the foreskin. In this line drawing, the catheter is left in place during reduction. *D,* In a manner reminiscent of removing a rubber glove, the thumb forces the glans through the foreskin that is encircled by the entire palm to achieve final reduction *(E)*. *(C from Neuwirth H, et al: Genitourinary imaging and procedures by the emergency clinician. Emerg Med Clin North Am 7:1, 1989.)*



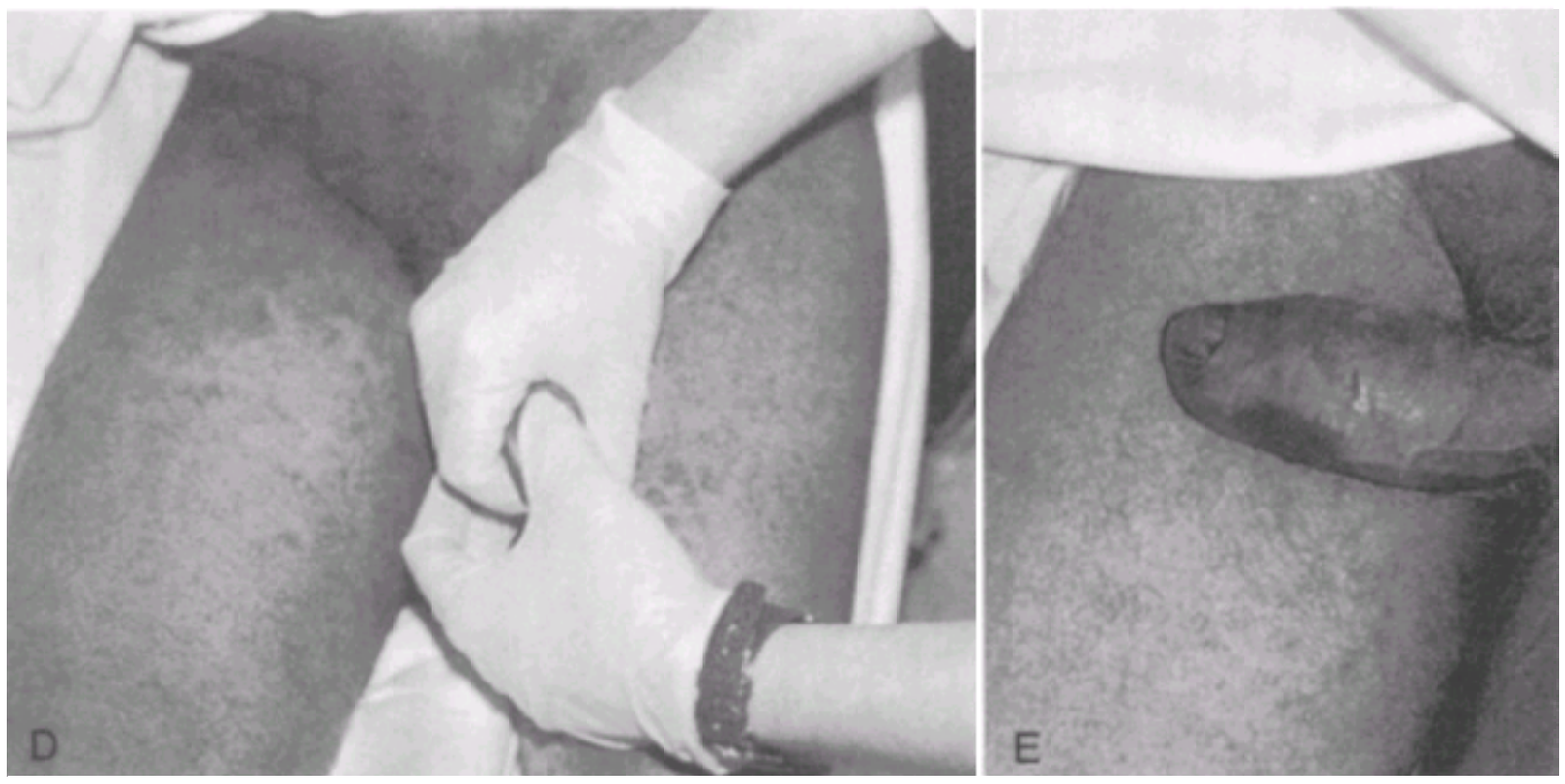


Figure 56-6 *A,* Application of Babcock clamps to reduce paraphimosis. *B,* Foreskin reduced. *(From Skoglund RW, Chapman WH: Reduction of paraphimosis. J Urol 104:137, 1970. Reproduced by permission. © Williams & Wilkins, 1970.)*

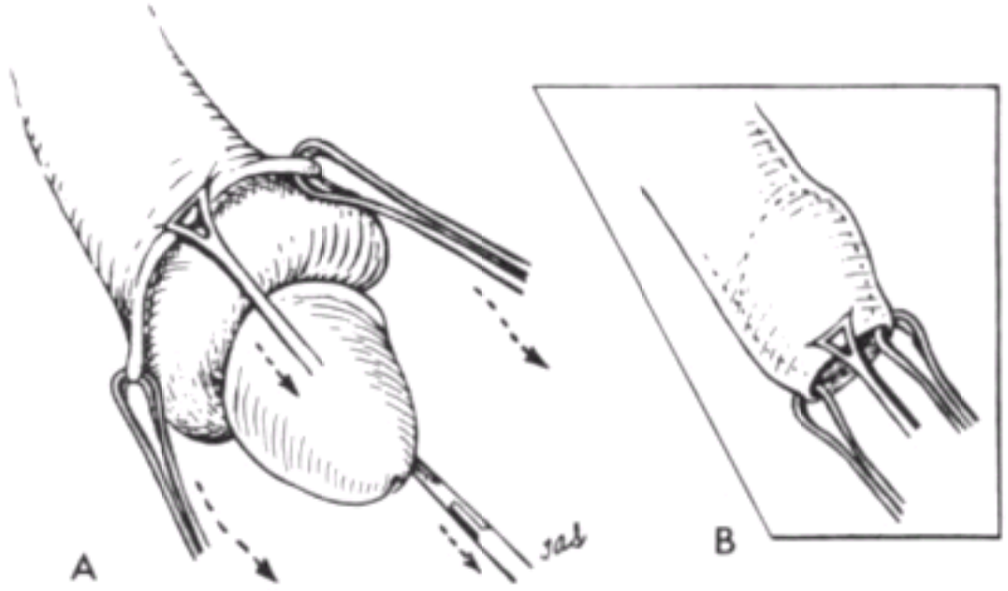


Figure 56-7 *A,* Anesthetizing the penis for surgical treatment of paraphimosis. Line of infiltration of local anesthesia used before performing dorsal slit. *B,* Incision for paraphimosis. Diamond-shaped defect resulting from incision of foreskin. The two apices of the dorsal slit *(a* and *b)* are approximated after the foreskin is reduced.

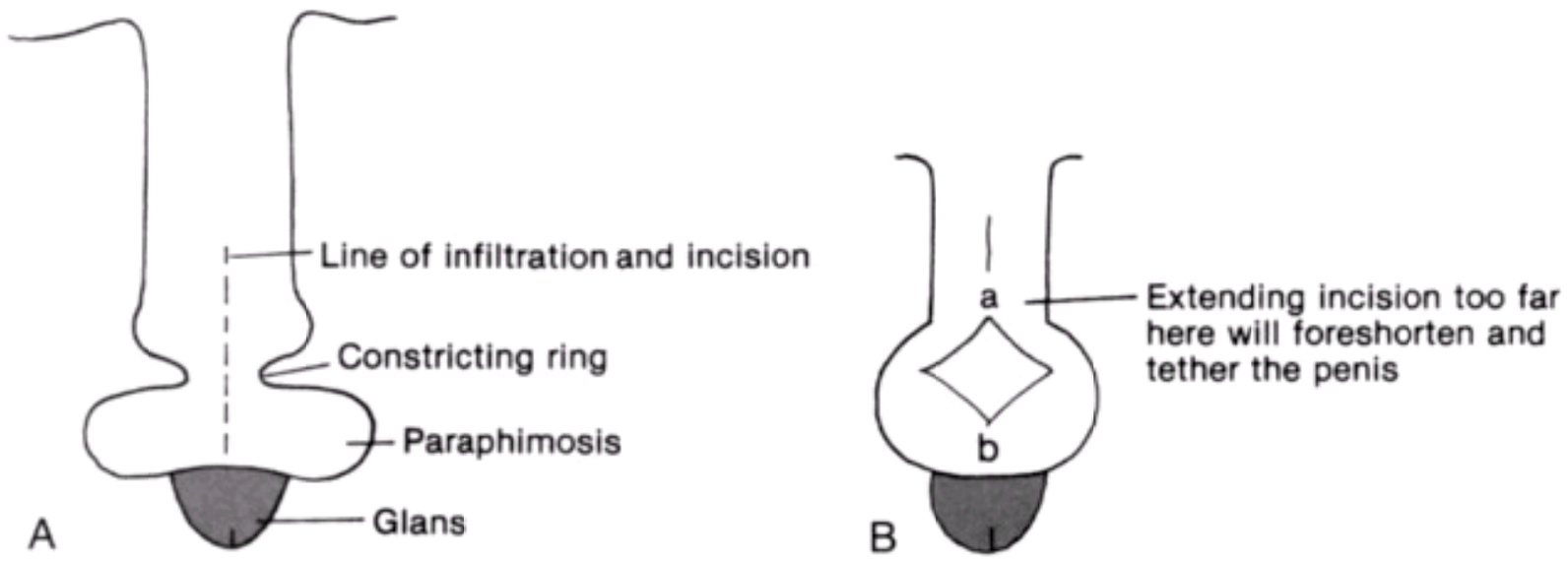
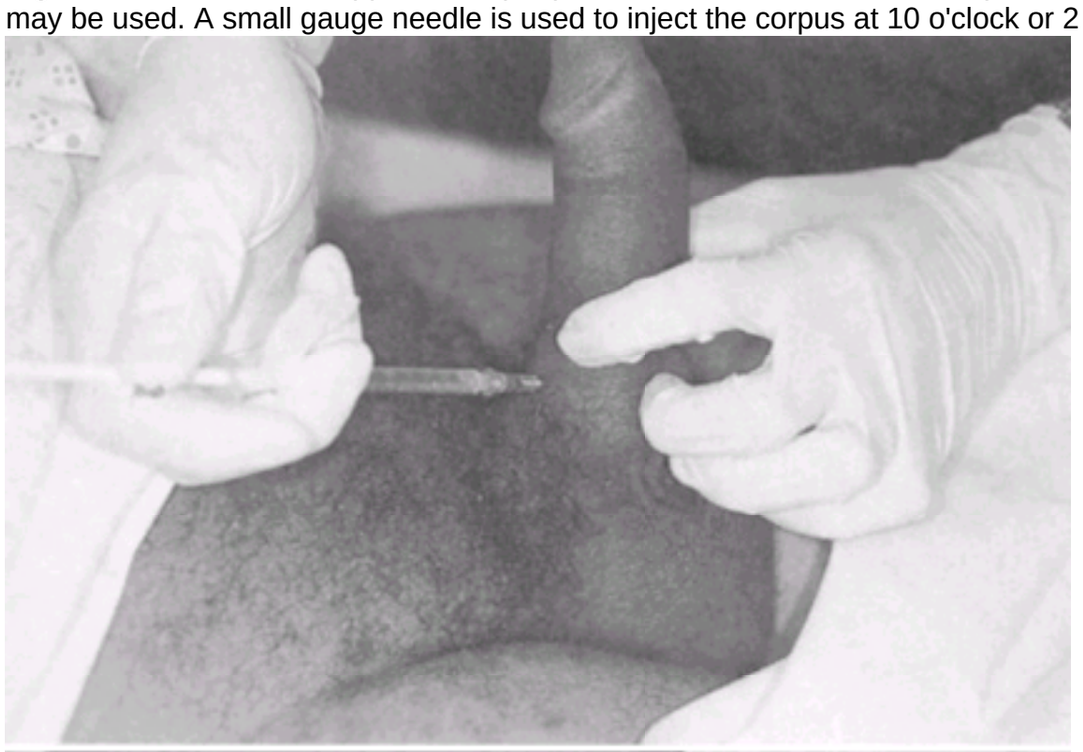


Figure 56-8 As an initial approach to priapism, and as an alternative to aspiration/irrigation, the intracorporeal injection of vasoactive drugs such as phenylephrine may be used. A small gauge needle is used to inject the corpus at 10 o'clock or 2 o'clock, at the base of the penis. Repeat injection may be required.



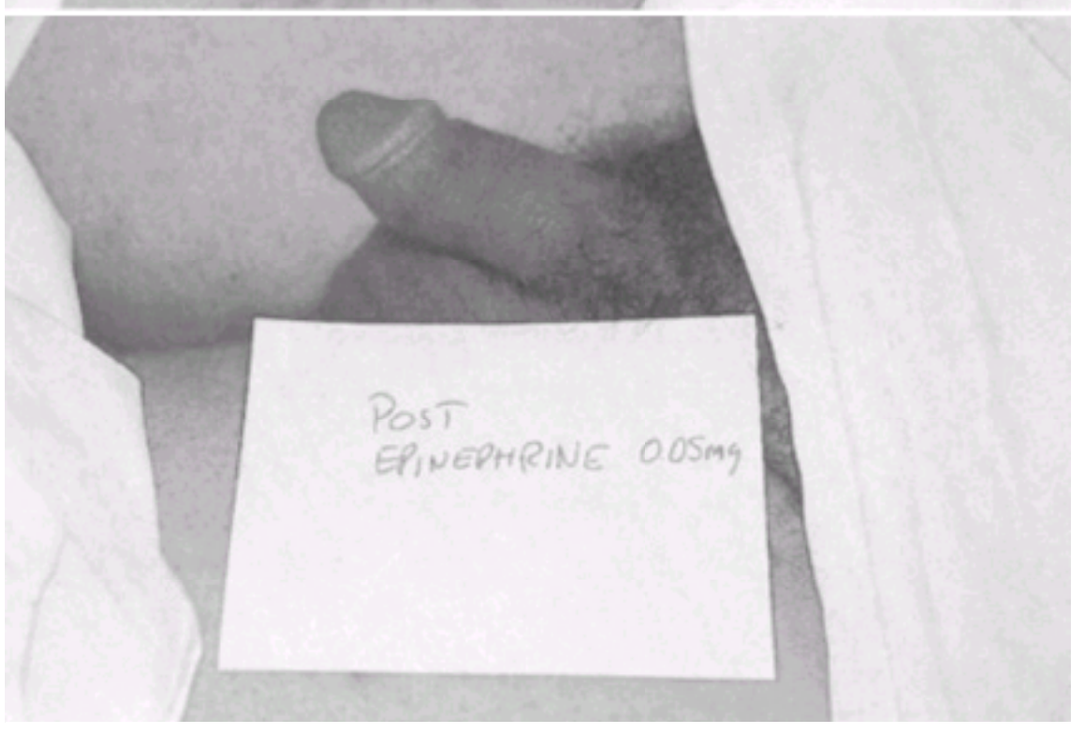


Figure 56-9 *A,* This patient experienced 18 hours of priapism after penile self-injection of papaverine as therapy for impotence. *B,* A penile dorsal nerve block is performed. *C,* A 19-ga butterfly needle is inserted into the corpus via the penile shaft at either the 2 o'clock or 10 o'clock position, and aspiration is performed. Slow steady suction will be most successful, while excessive suction may halt the aspiration. Do not puncture the corpus via the glans (see text). *D,* After detumescence with aspiration or with irrigation and injection of a vasoactive medication (see text), the penis is wrapped with an elastic bandage to discourage re-engorgement and to compress the puncture site.

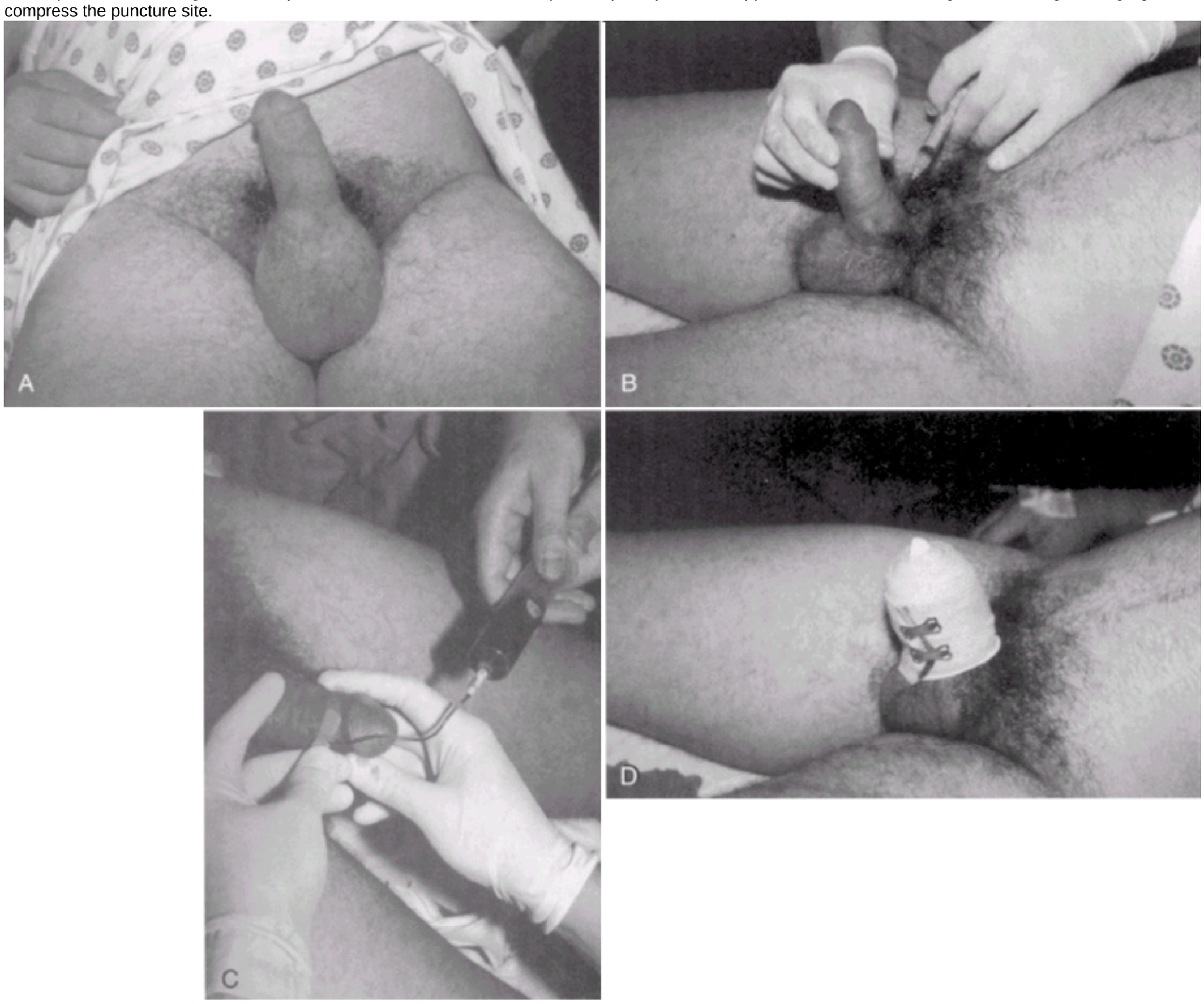


Figure 56-10 Proper position of the Doppler probe in the examination of the acute scrotum. Note caudal orientation of the probe (drawing is a lateral projection).

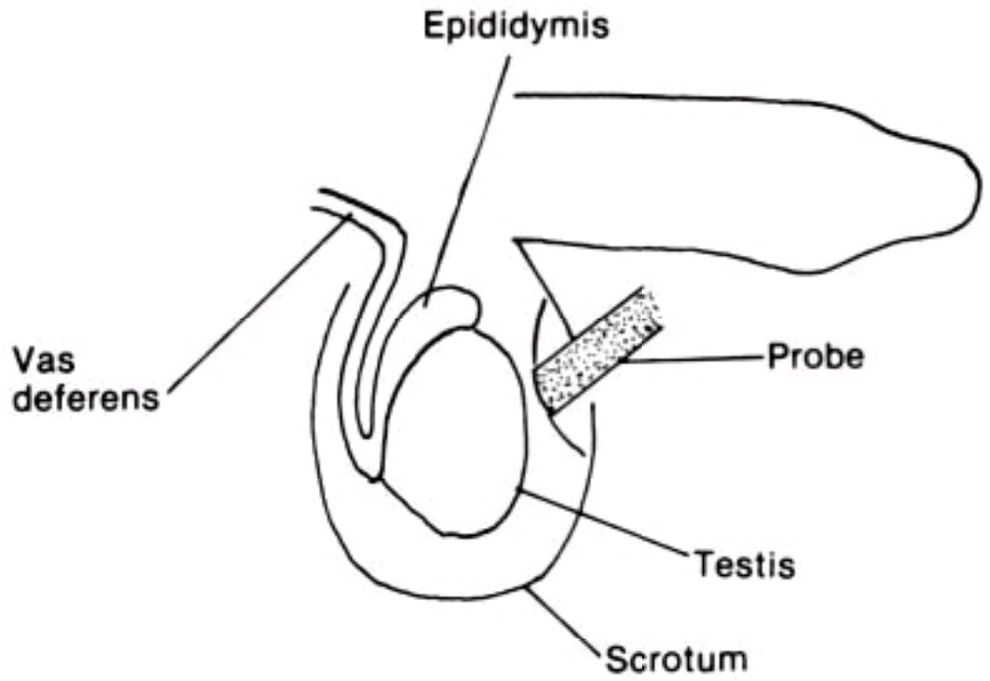
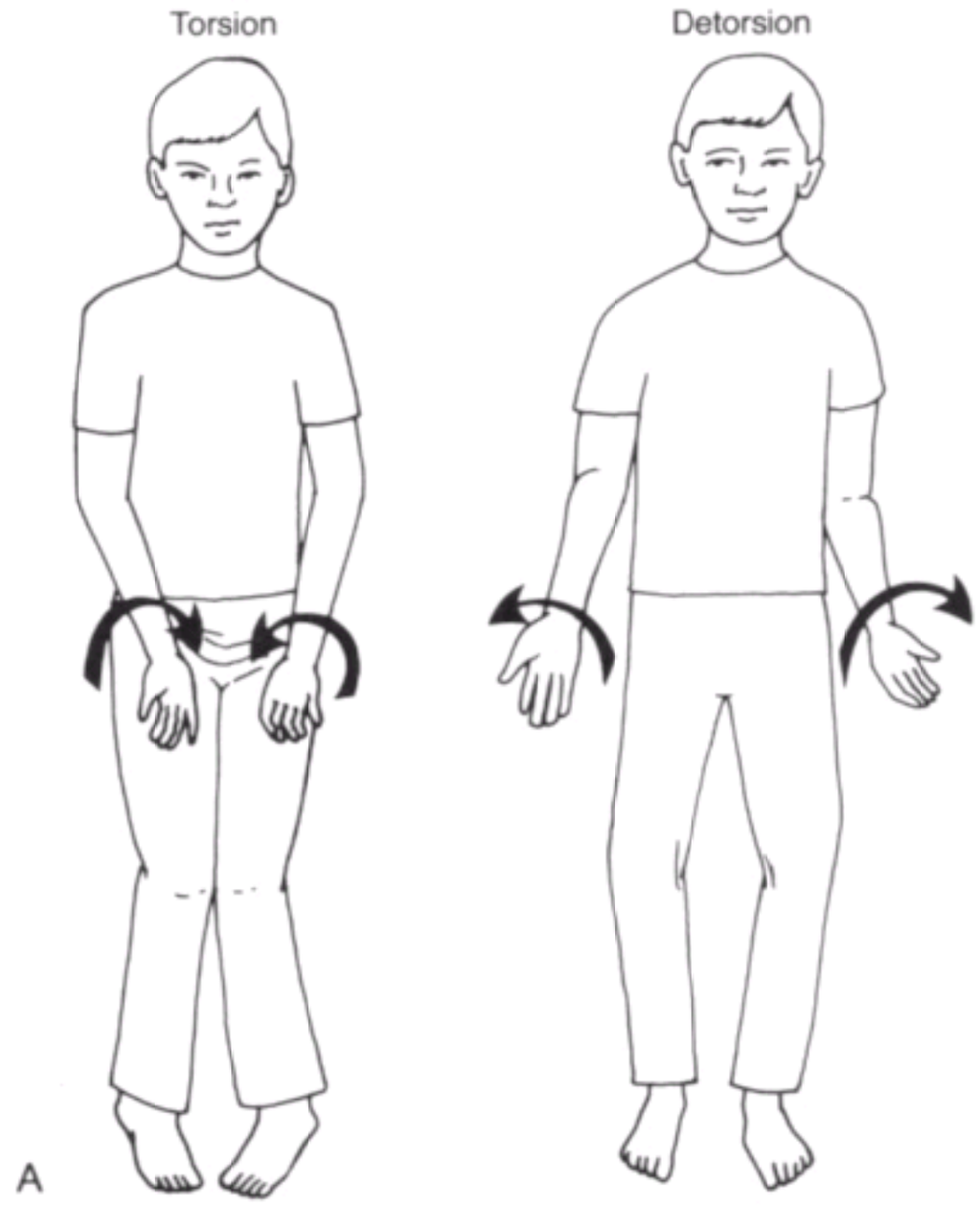


Figure 56-11 *A,* Testicular torsion usually occurs in a medial direction. Detorsion should be attempted initially by rotating the testis outward toward the thigh. This is most successful if attempted within the first few hours of torsion, before the onset of significant scrotal swelling. Intravenous narcotics (such as fentanyl) or a cord block can be administered before attempting detorsion. *B,* Detorsion of the testicle may require testicular rotation through two planes. To release the cremasteric muscle, the testis is rotated in a caudal to cranial direction simultaneously with medial to lateral rotation. The right testis is shown. *(B from Freeman S, et al: Urologic procedures. Emerg Med Clin North Am 4:543, 1986.)*



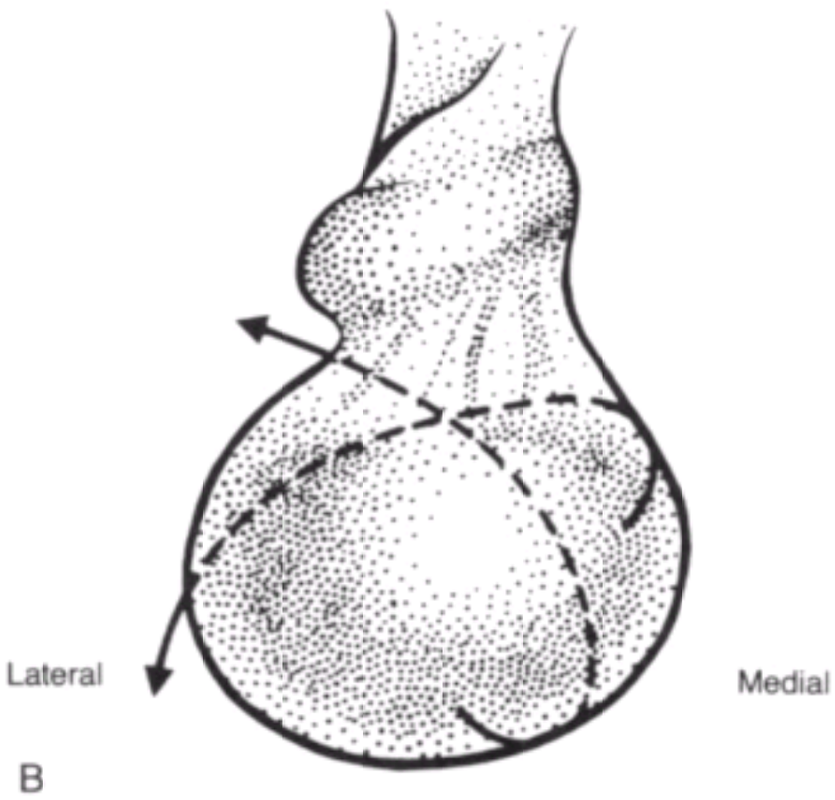


Figure 56-12 *A,* Anatomy of the female external genitalia. *B,* Uncomplicated catheterization in the female. *(A from Flocks RH, Culp DA: Surgical Urology: A Handbook of Operative Surgery, 4th ed. Chicago, Year Book Medical Publishers, 1975, p 357. B from Brunner LS, Suddarth DS: Lippincott Manual of Nursing Practice. Philadelphia, JB Lippincott, 1974, p 465. Reproduced by permission.)*

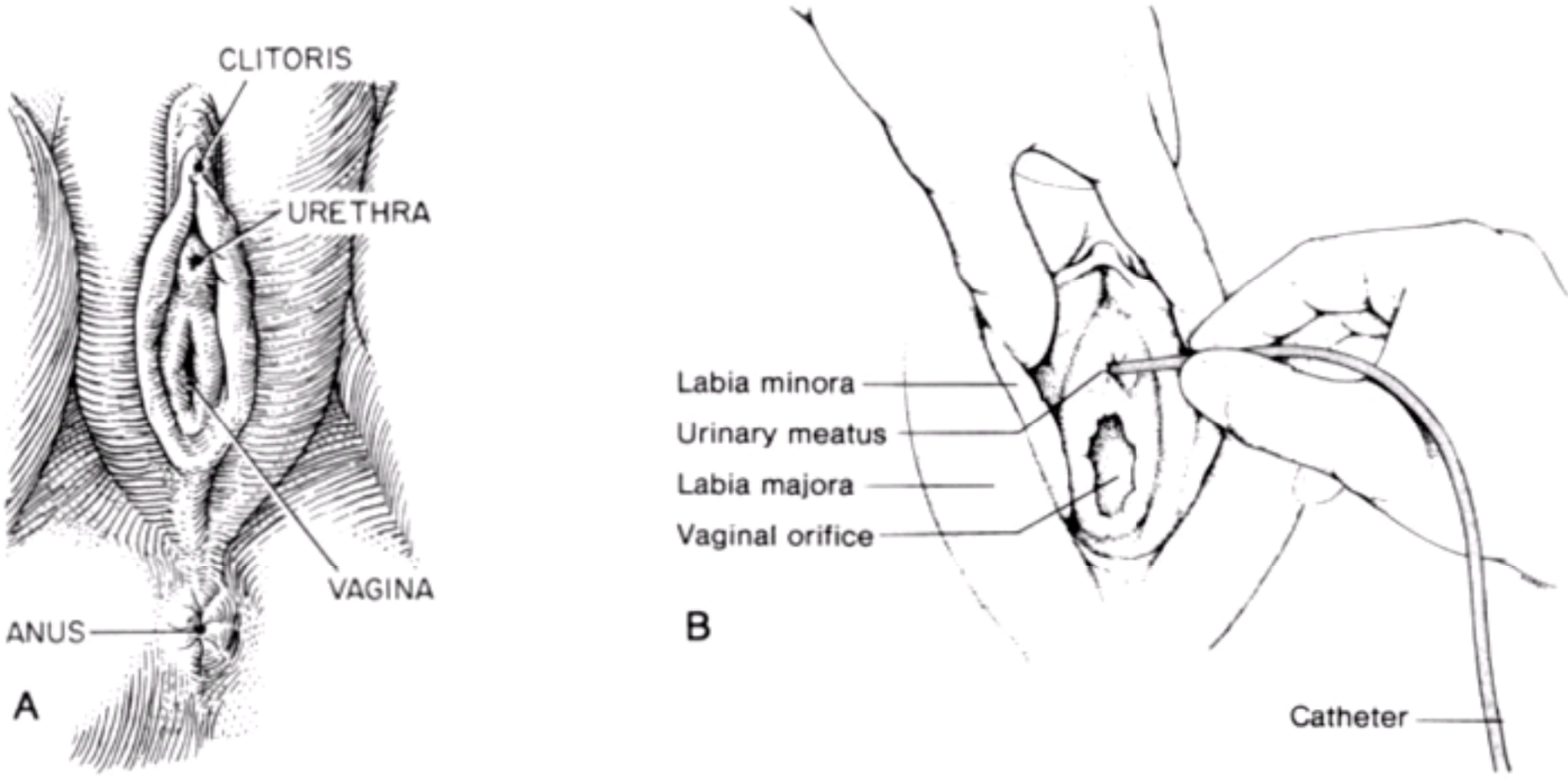


Figure 56-13 Female urethra. *A,* Sagittal view showing normal urethral course into the bladder. *B* and *C,* Cystourethrocele with loss of anterior support (frontal and sagittal views). *D* and *E,* Intravaginal re-creation of normal anatomic relationships with nondominant index and long fingers (frontal and sagittal views).

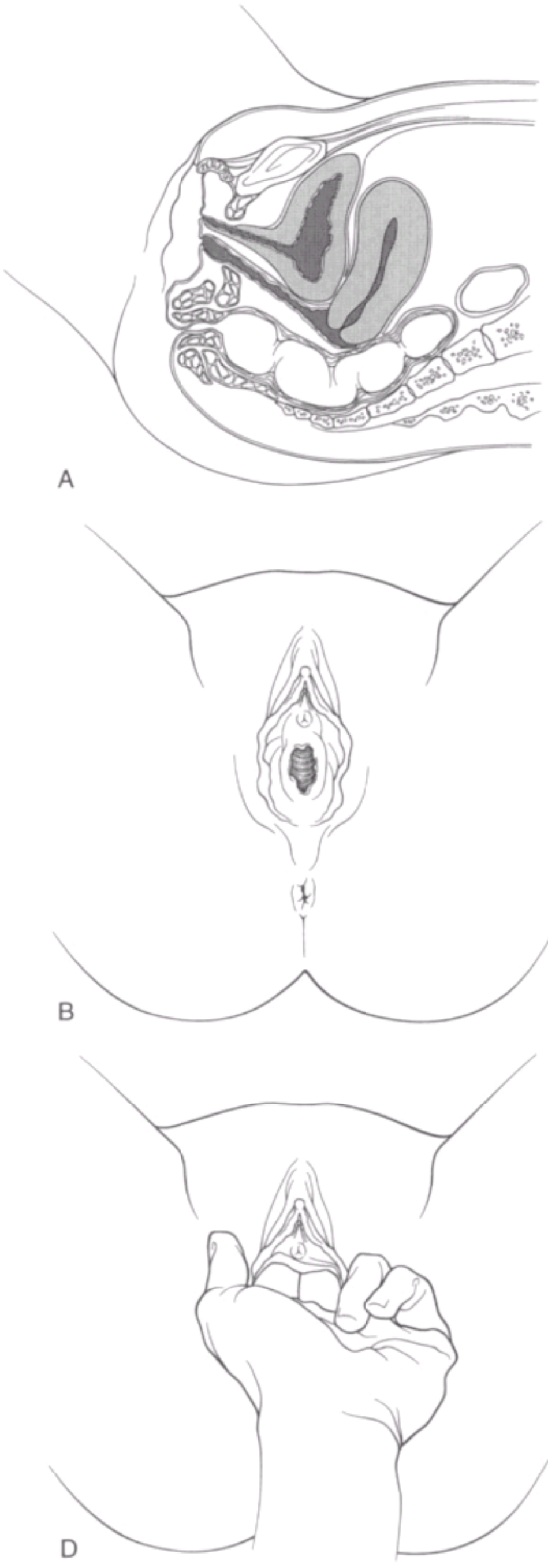


Figure 56-14 Anatomy of the male urethra. *(From Flocks RH, Culp DA: Surgical Urology: A Handbook of Operative Surgery, 4th ed. Chicago, Year Book Medical Publishers, 1975, p 359. Reproduced by permission.)*

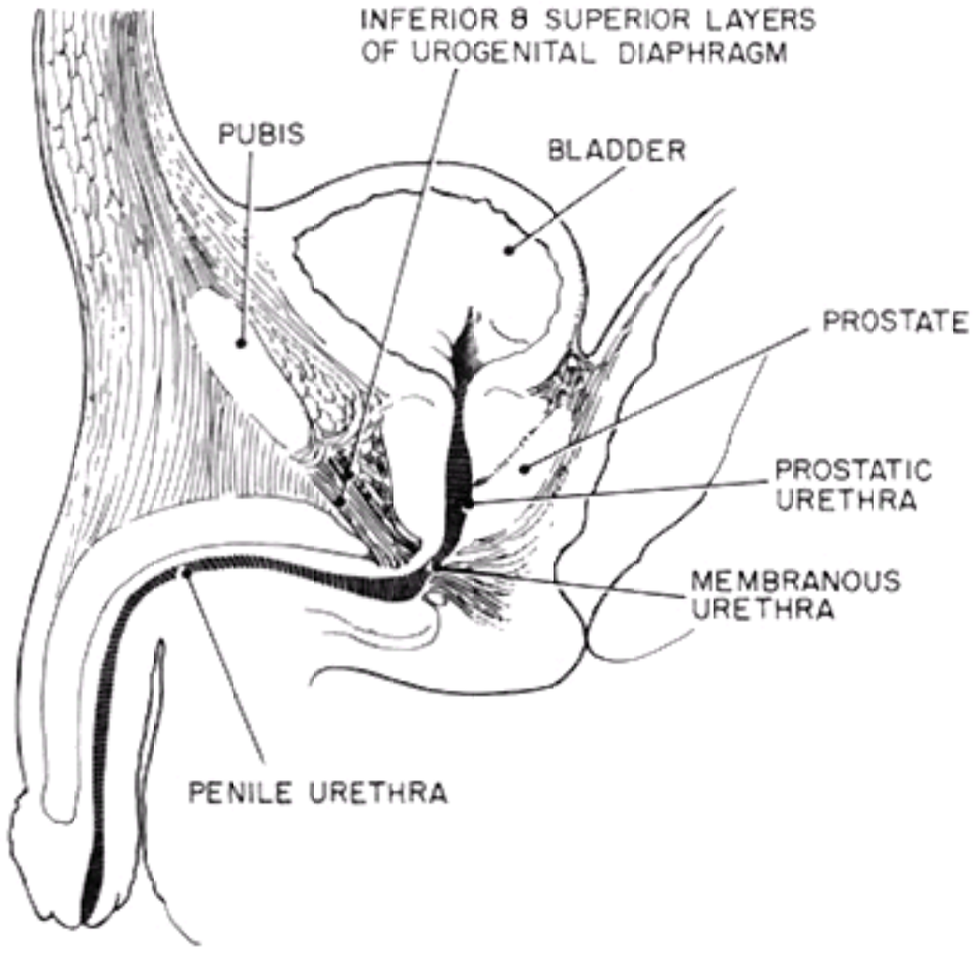


Figure 56-15 Foley catheter placement. Foley catheter is inserted fully (i.e., to the balloon port) before air or water is injected to inflate the balloon.

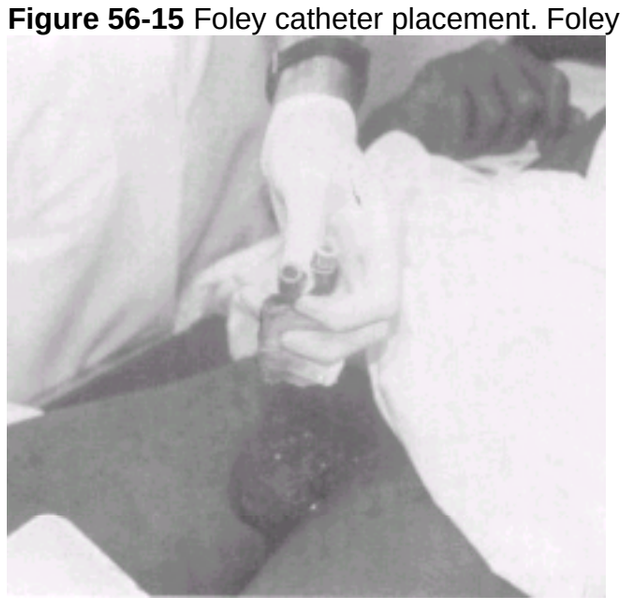


Figure 56-16 Foley catheter placement. *A,* Holding penis taut and upright prevents urethral folding, lessens external sphincter spasm, and promotes unobstructed catheterization. *B,* Proper uncomplicated male catheterization with upward traction on the penis. *(From Brunner LS, Suddarth DS: Lippincott Manual of Nursing Practice. Philadelphia, JB Lippincott, 1974, p 465. Reproduced by permission.)*

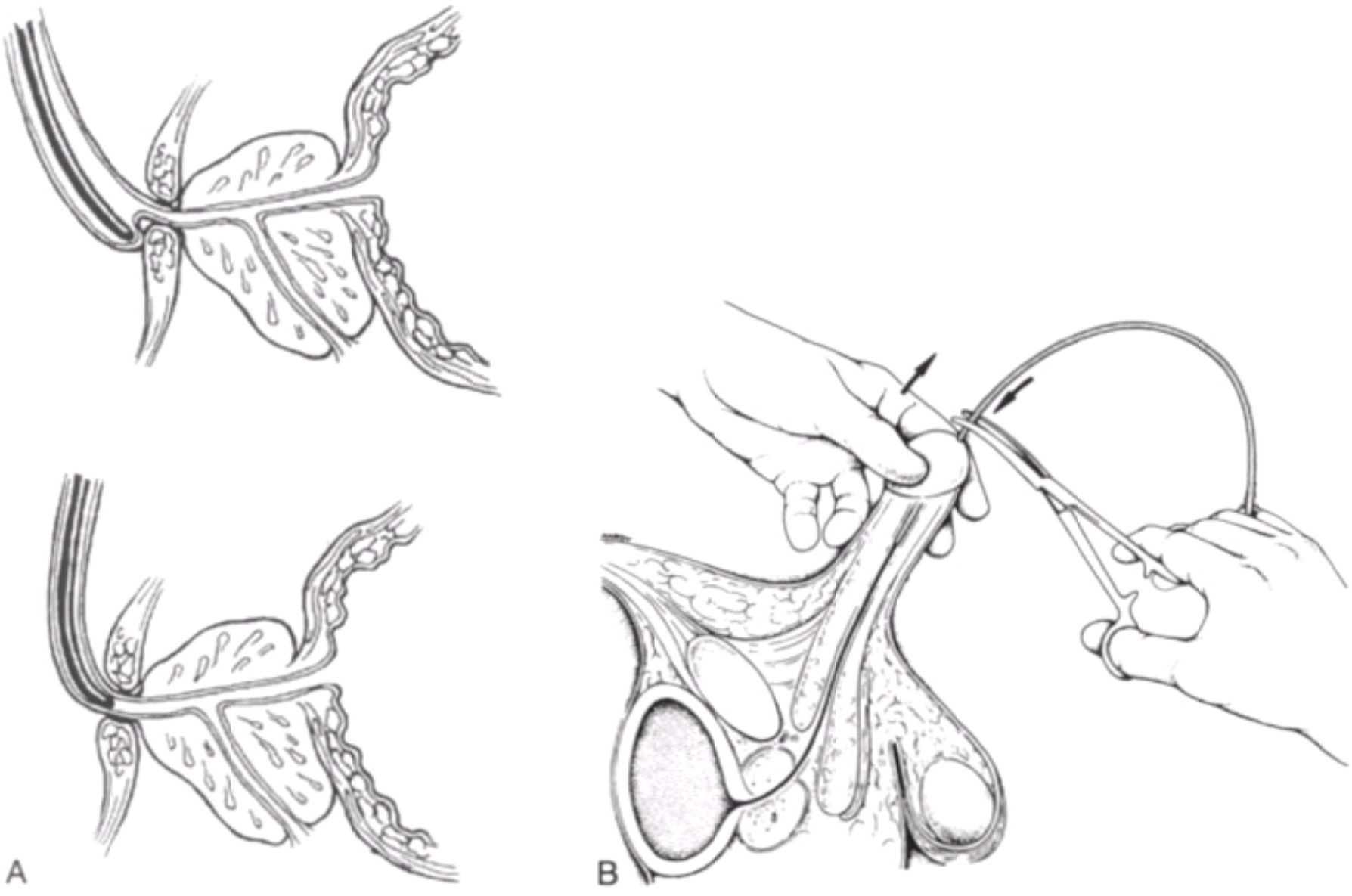


Figure 56-17 Foley catheter placement. It is important to fully retract the foreskin *(A),* especially in the uncircumcised male, so that a folded 4 × 4 gauze sponge can be wrapped around the coronal sulcus *(B)* and prevent foreskin reduction during the procedure. Once the procedure is terminated, the foreskin must be reduced to its normal anatomic position.

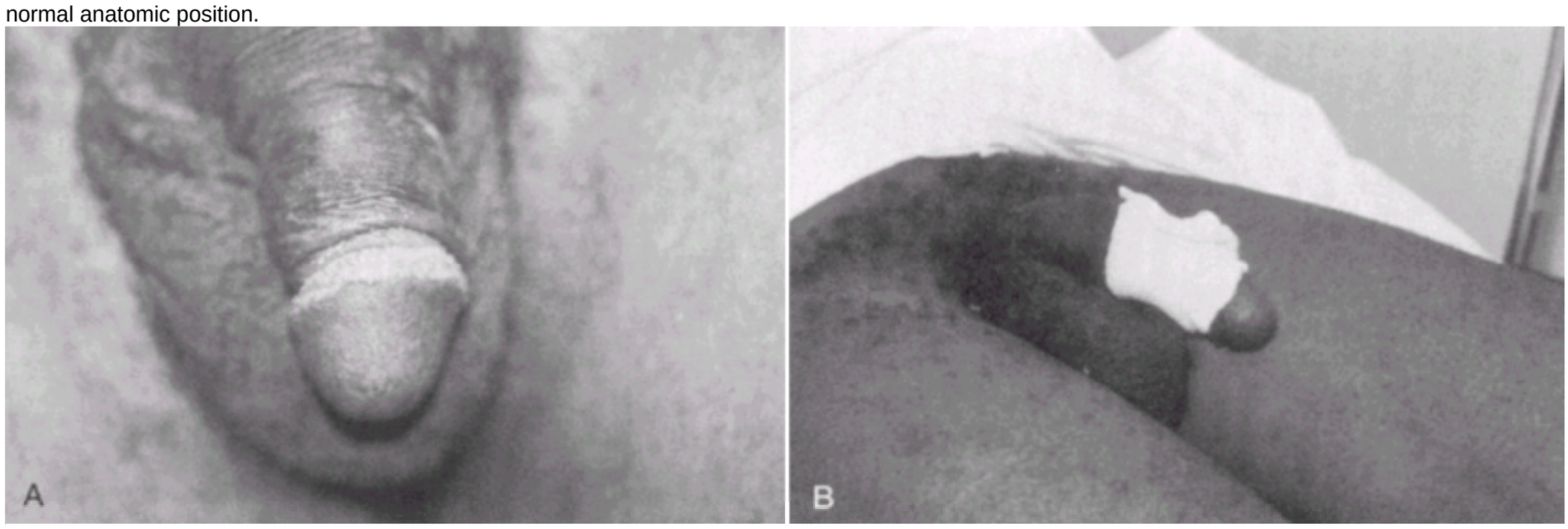


Figure 56-18 Penile stabilization. To optimize manual dexterity during urethral instrumentation, the prepared penis should be grasped between the nondominant long and ring fingers. This leaves the index finger and thumb available to stabilize the catheter or glans.

Figure 56-19 Paraphimosis. Tremendous foreskin edema from paraphimosis can make this diagnosis difficult unless the examiner searches diligently for the constricting band.

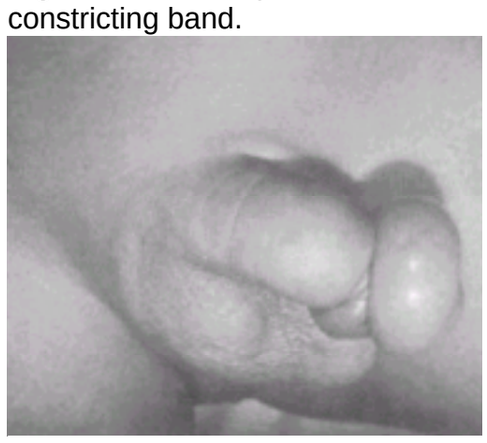


Figure 56-20 Paraphimosis. The appearance of this penis *(A)* could easily be mistaken as being normal. Proximal foreskin retraction reveals a normal coronal sulcus *(B)*. Distal foreskin manipulation *(C)* reveals a phimotic band causing paraphimosis.

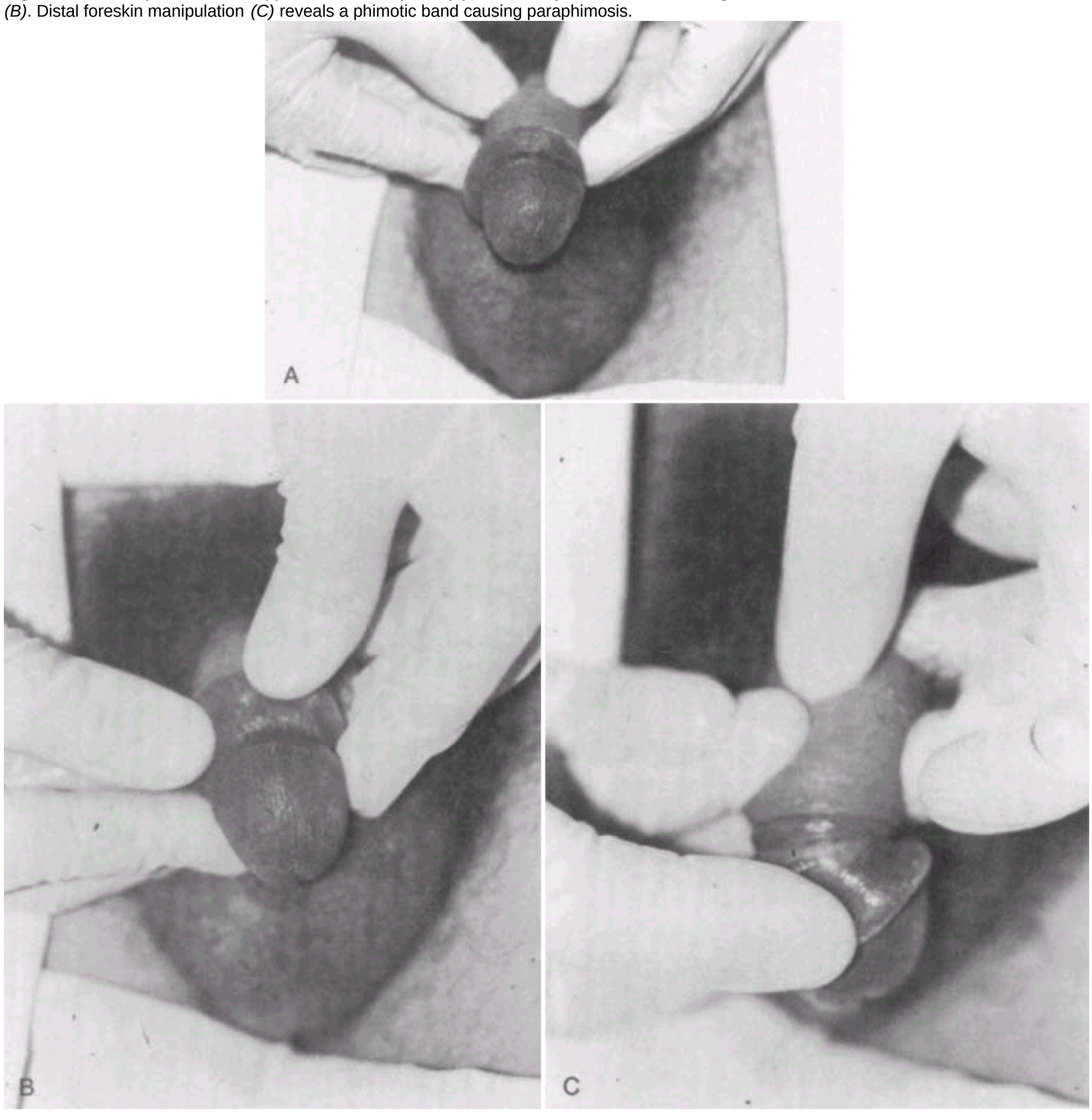
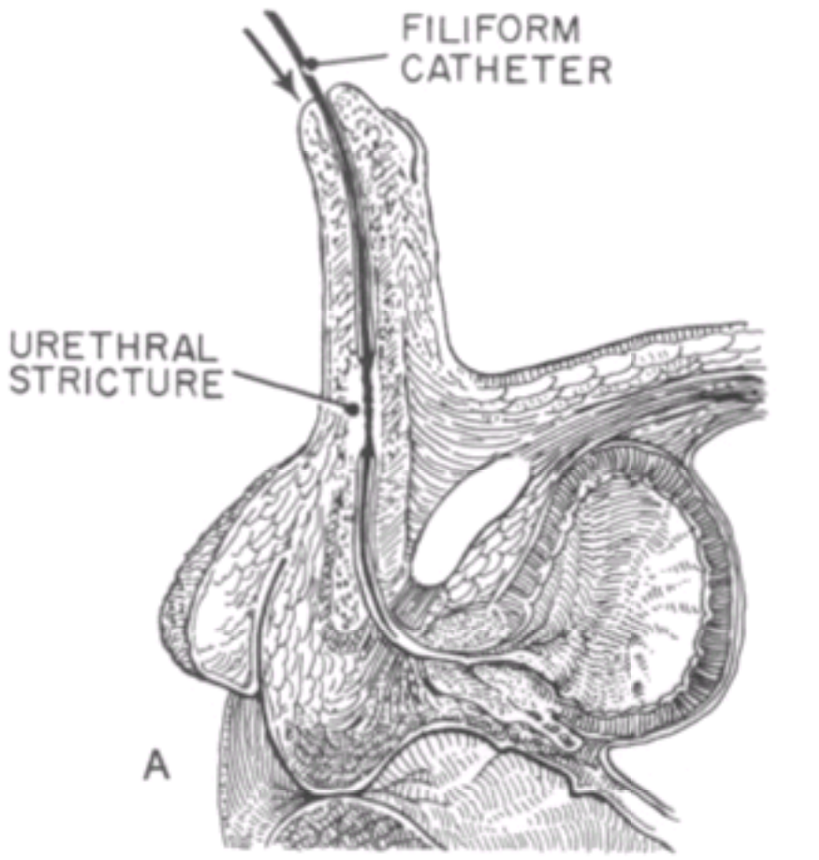
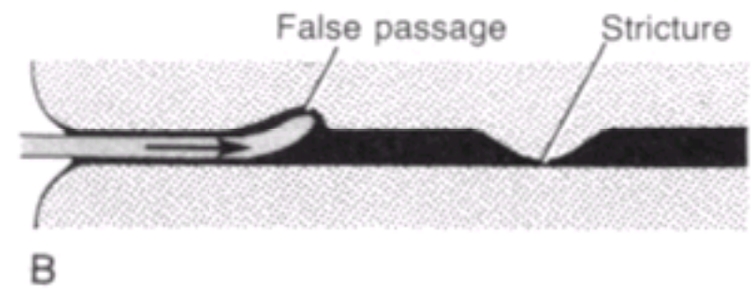
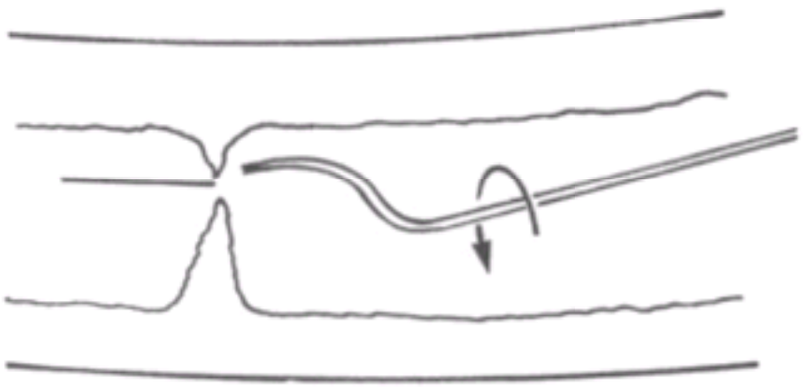
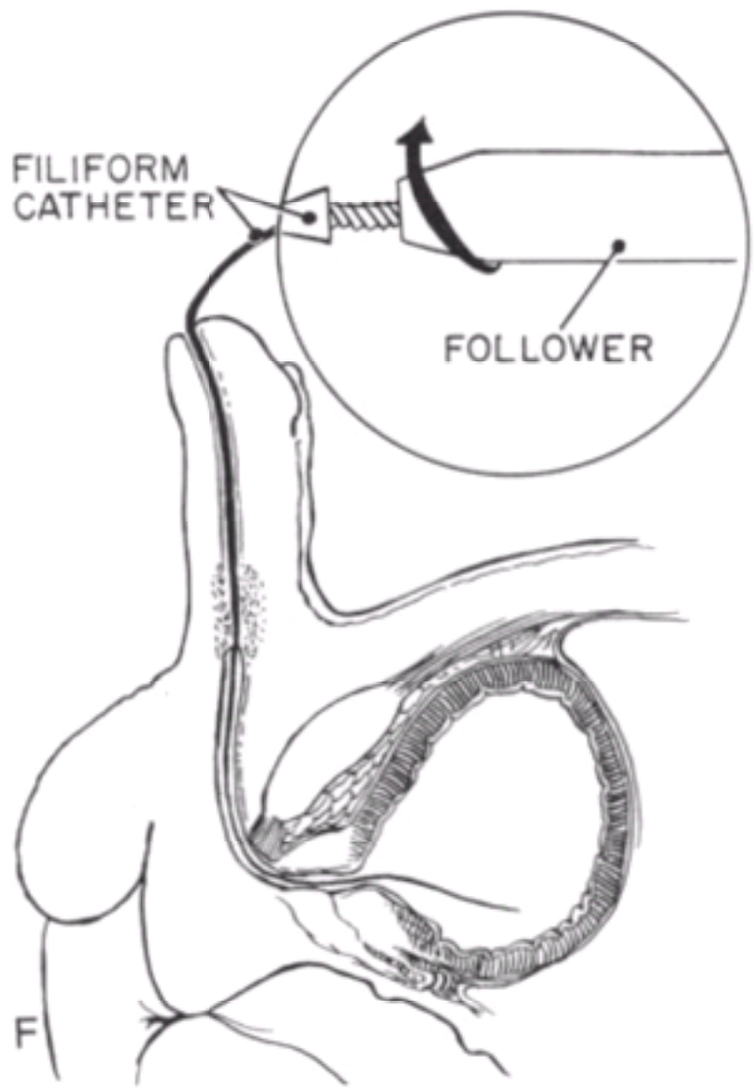


Figure 56-21 *A,* Passage of the filiform past the urethral stricture. *B,* Sequential passage of filiforms, with entry of the first catheter into the false passage. *C,* Advancement of the second filiform to the stricture site. *D,* Passage of the third filiform past the stricture. Following passage of the filiform into the bladder, redundant catheters are removed. *E,* Pigtail filiform passing through the stricture. *F,* Attachment of a follower to the filiform. *G,* Dilation of the stricture with a follower. *(E from Blandy J: Operative Urology. Oxford, Blackwell Scientific Publications, 1978, p 204. B, C, and D from Hill GJ: Outpatient Surgery. Philadelphia, WB Saunders, 1973. Reproduced by permission.)*









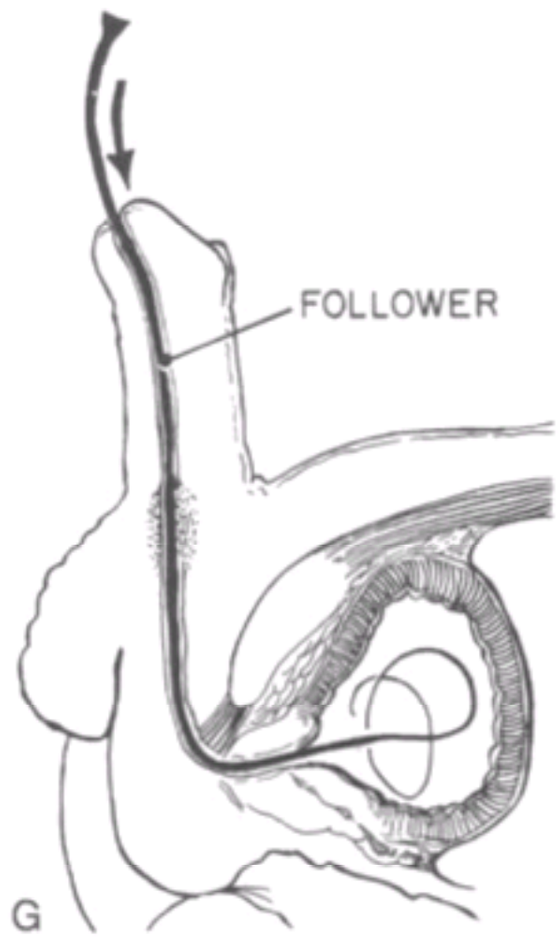
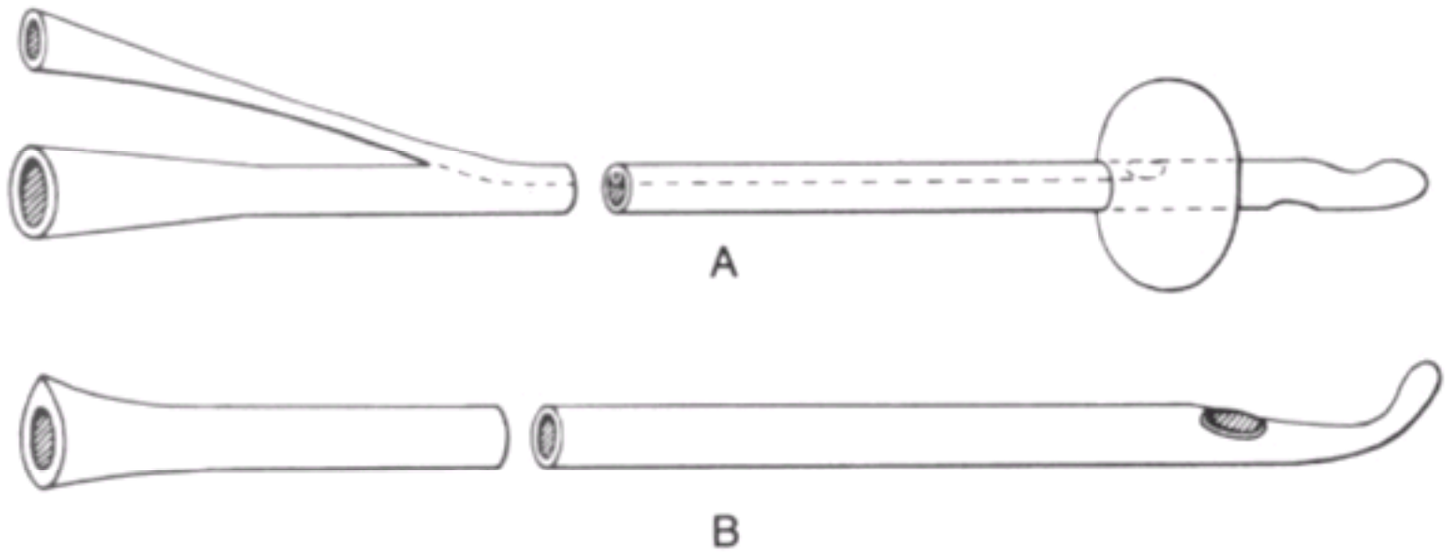
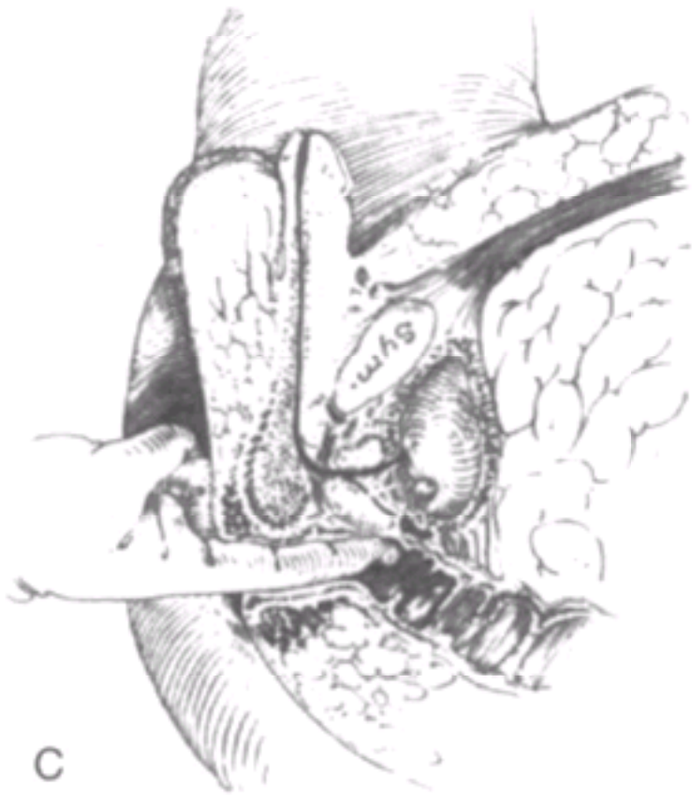


Figure 56-22 *A,* Self-retaining Foley-type catheter. *B,* Coudé catheter (also available in a non-self-retaining model). *C,* Superior pressure on the prostate may facilitate catheter passage. *D*, Catheter guide or stylet should be used only by a urologist.





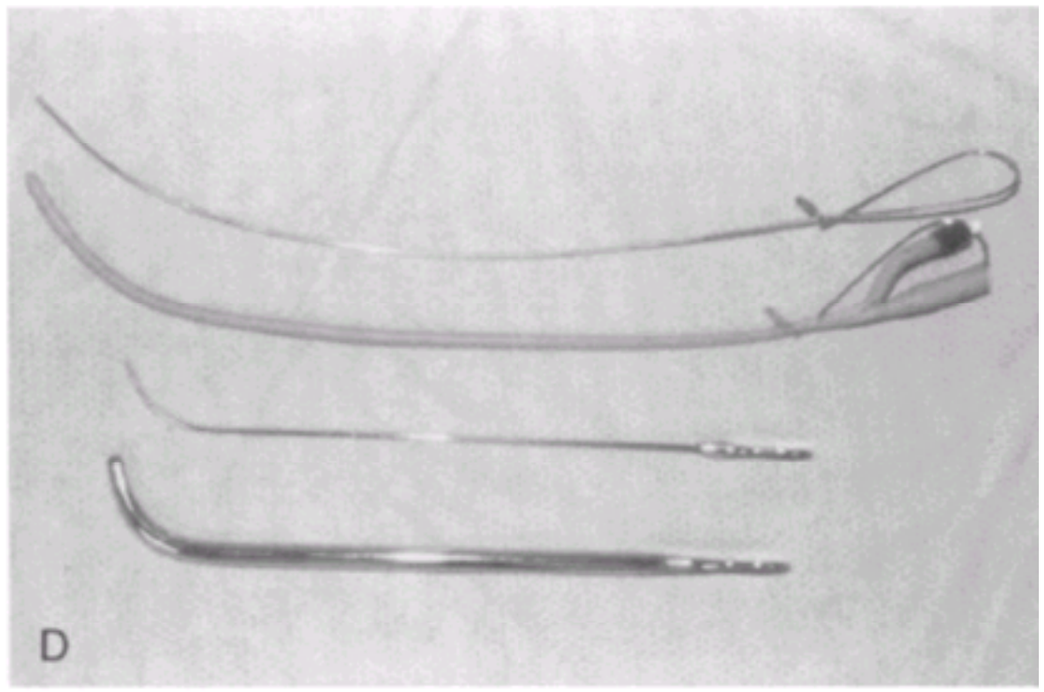


Figure 56-23 A flap-like defect in the inflating channel of a balloon catheter that is being raised by a wire stylet passed down the inflating channel to deflate the balloon. *(From Eichenberg HA, Amin M, Clark J: Nondeflating Foley catheters. Int Urol Nephrol 8:171, 1976. Reproduced by permission.)*

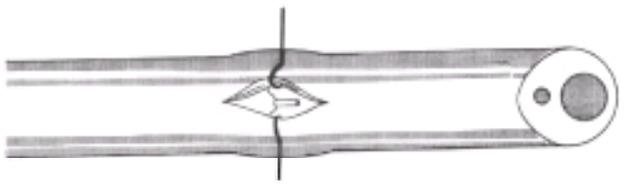
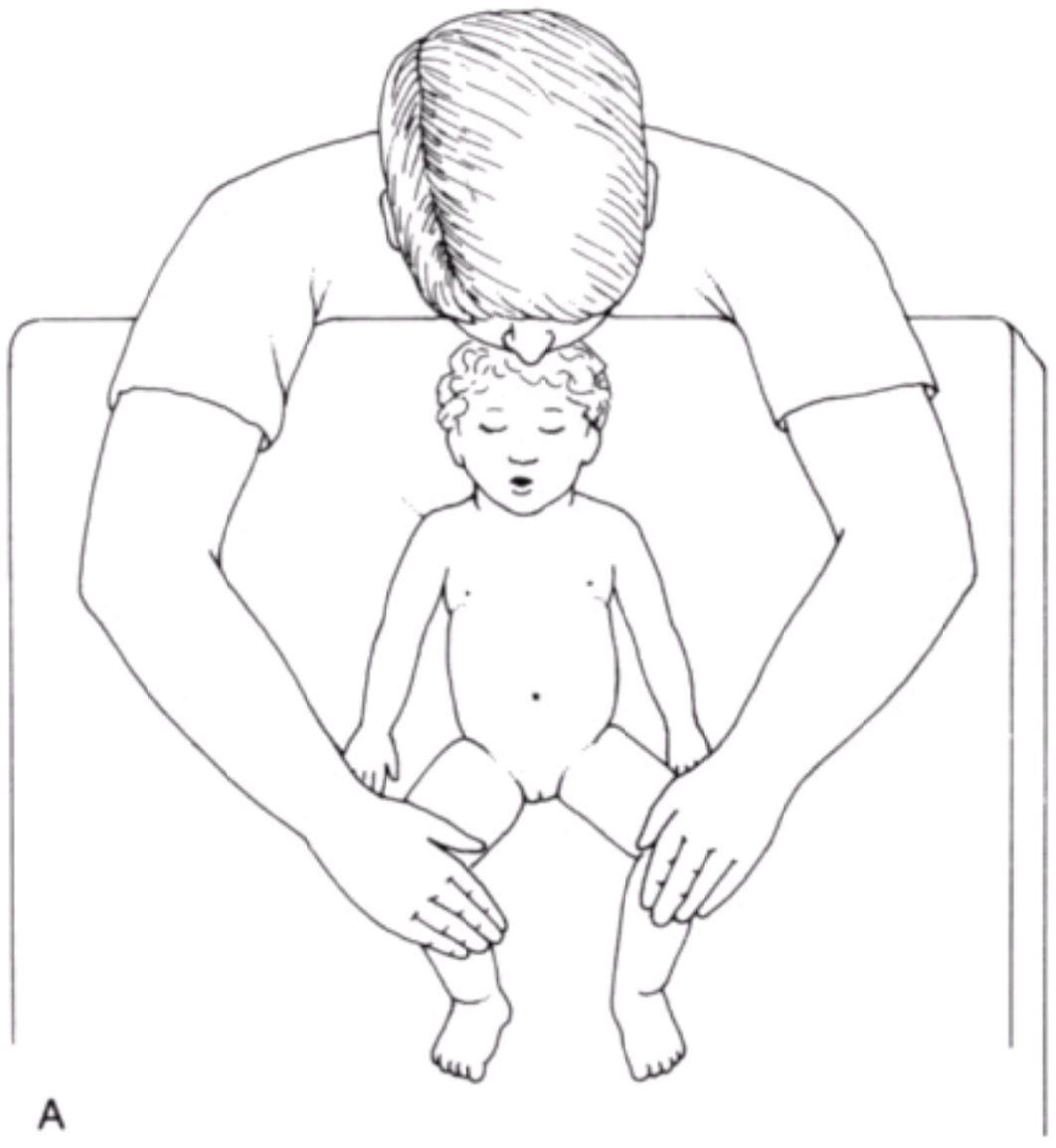


Figure 56-24 *A,* For a suprapubic bladder tap, the infant is restrained and placed in a frog-legged position. *B,* A 22-ga needle punctures the abdominal wall in the midline approximately 1 to 2 cm cephalad to the superior border of the pubic bone. The syringe is perpendicular to the plane of the abdominal wall (usually 10°–20° from the true vertical). The bladder is an abdominal organ in infants, and placing the needle too close to the pubic bone or angling toward the feet may cause the needle to miss the bladder. Localizing the bladder with bedside ultrasound facilitates this procedure.



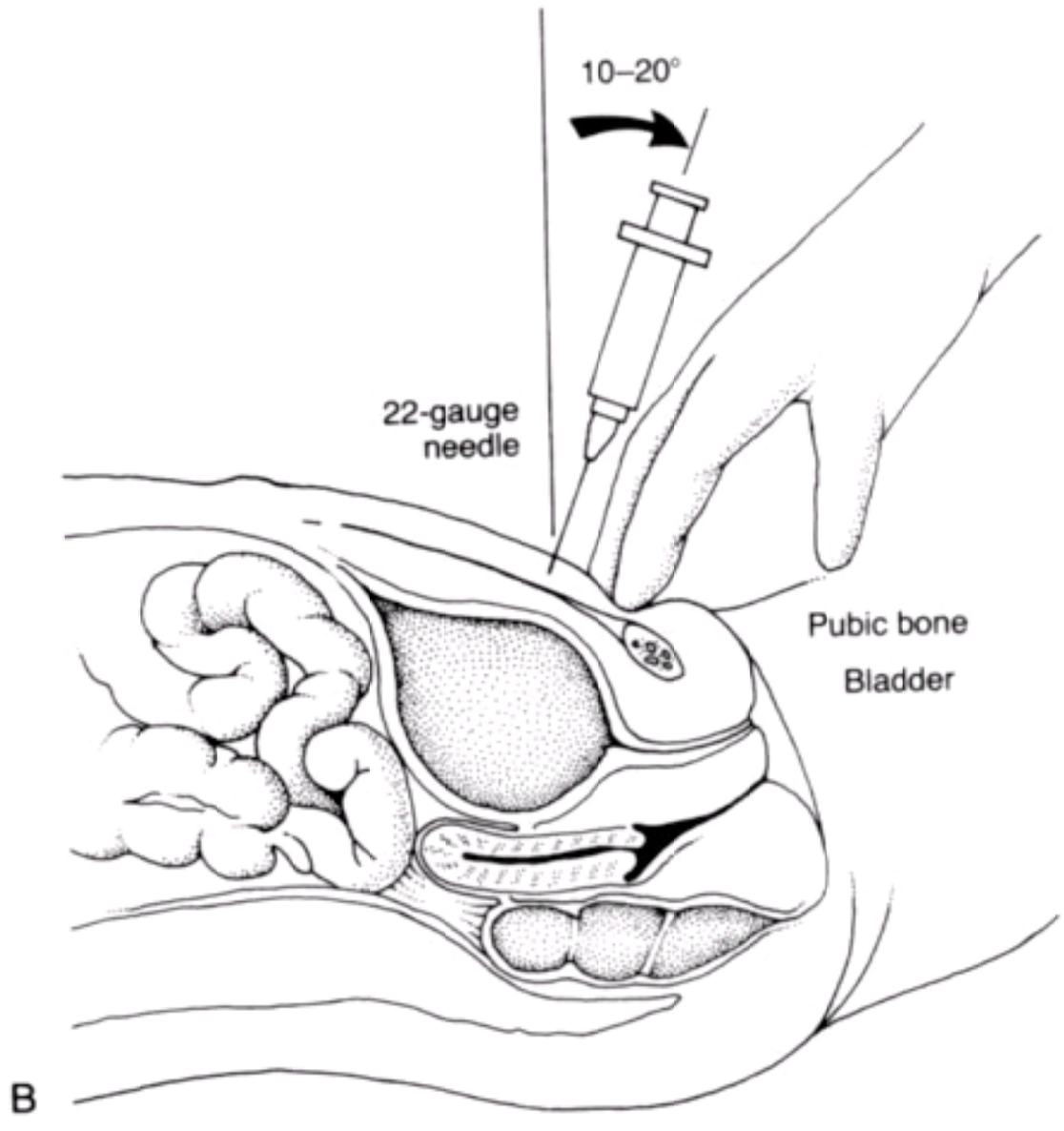


Figure 56-25 The peritoneum is pushed cephalad by the filled bladder during suprapubic aspiration in an adult. The needle is directed slightly caudad.

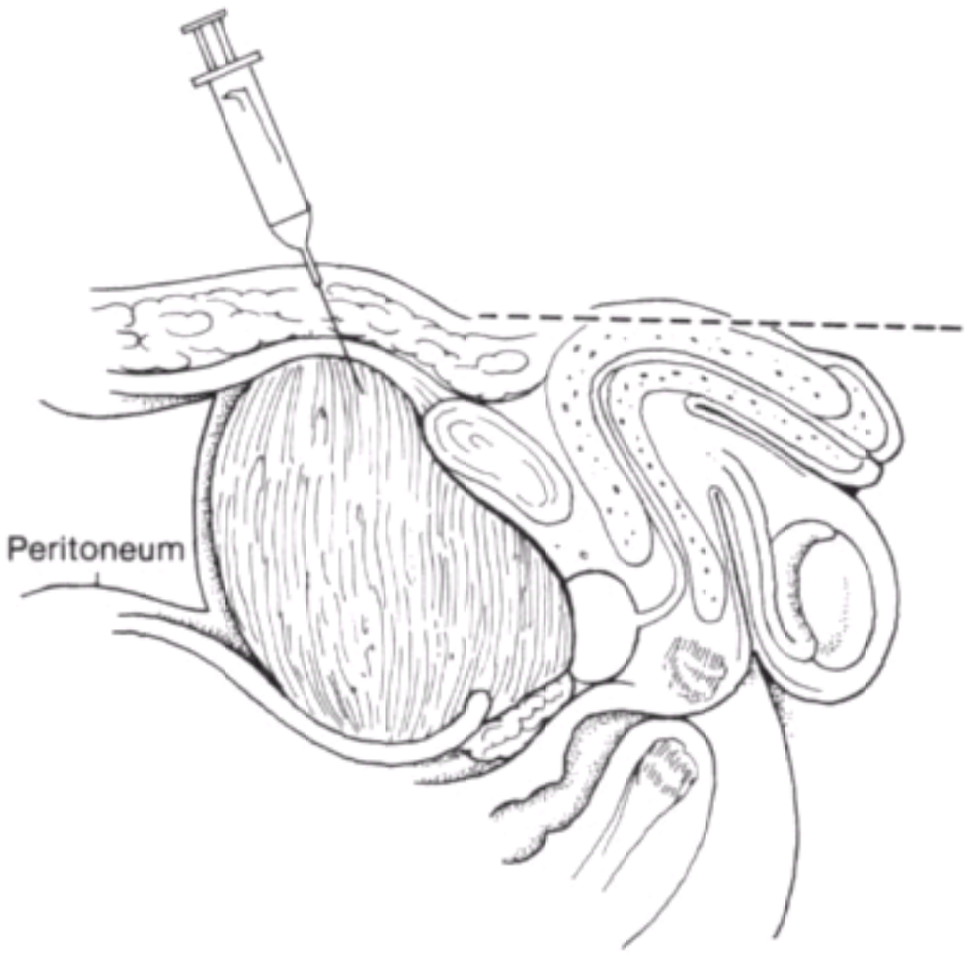


Figure 56-26 Suprapubic cystostomy with the Cook peel-away sheath introducer. *A,* Bladder is entered with a syringe and needle. Location is confirmed by the aspiration of urine. *B,* Syringe is removed and the guide wire is passed through the needle into the bladder. *C,* The needle is removed, then the dilator and peel-away sheath are passed over the wire into the bladder. A small stab wound in the anterior abdominal fascia may be required to accommodate the dilator and sheath. *D,* The dilator and wire are removed, leaving only the sheath inside the bladder. *E,* The preselected Foley balloon catheter is passed through the sheath into the bladder. Urine is aspirated to confirm location. *F,* The balloon is inflated with a minimum of 10 mL of air, saline, or water. A 5-mL balloon will accommodate 10 mL easily and make accidental catheter distraction less likely. *G,* The sheath is removed from the bladder, anterior abdominal wall, and cutaneous entry site, and is then literally peeled away from the indwelling catheter. *H,* The catheter is withdrawn until a snug fit is ensured at the cystostomy site.

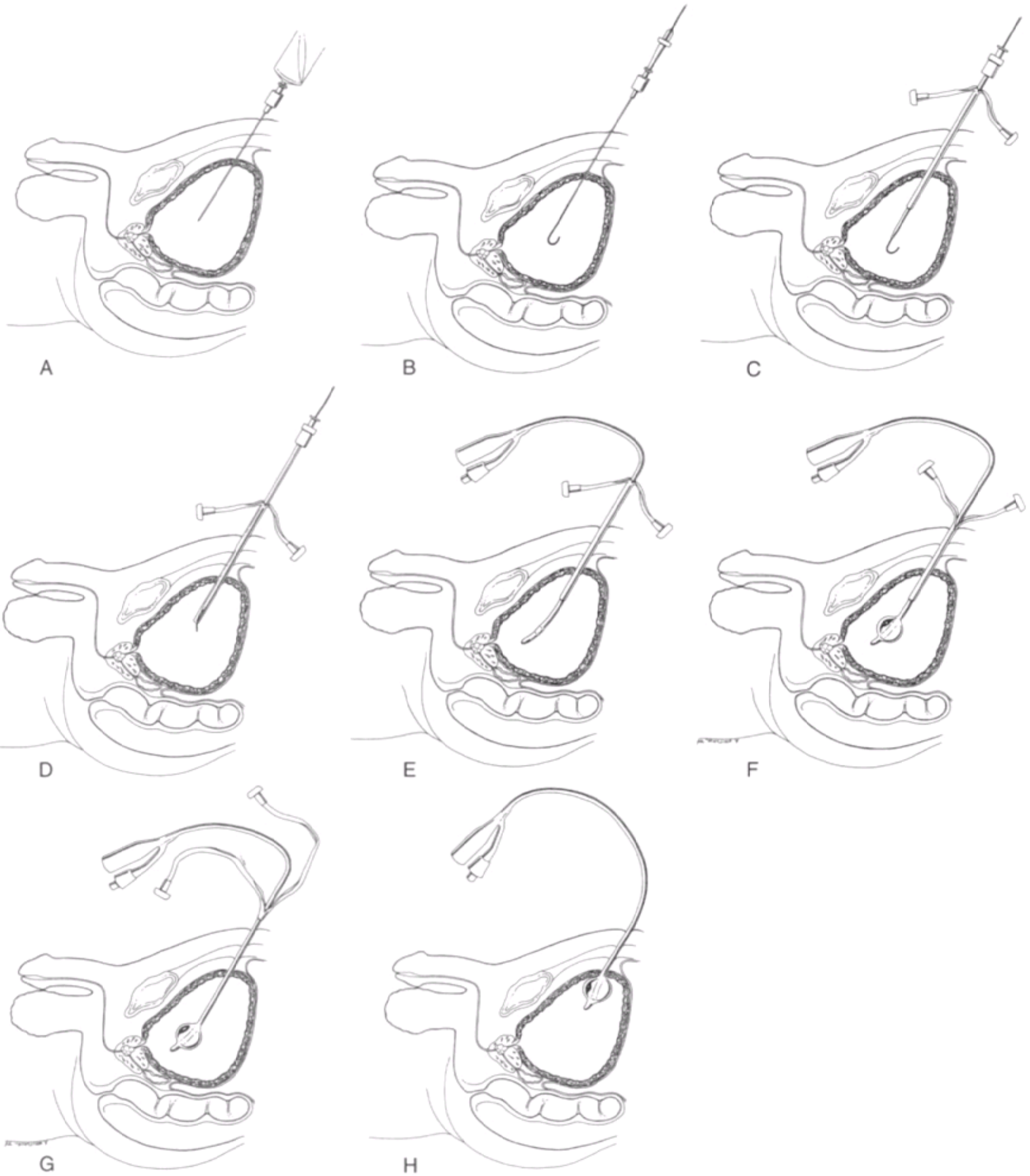


Figure 56-27 A common posterior urethral injury is a disruption of the membranous urethra. In this case, a distended bladder and attached prostate gland are sheared from the fixed membranous urethra. Note the development of a perivesical hematoma and the presence of a "high-riding" prostate gland.

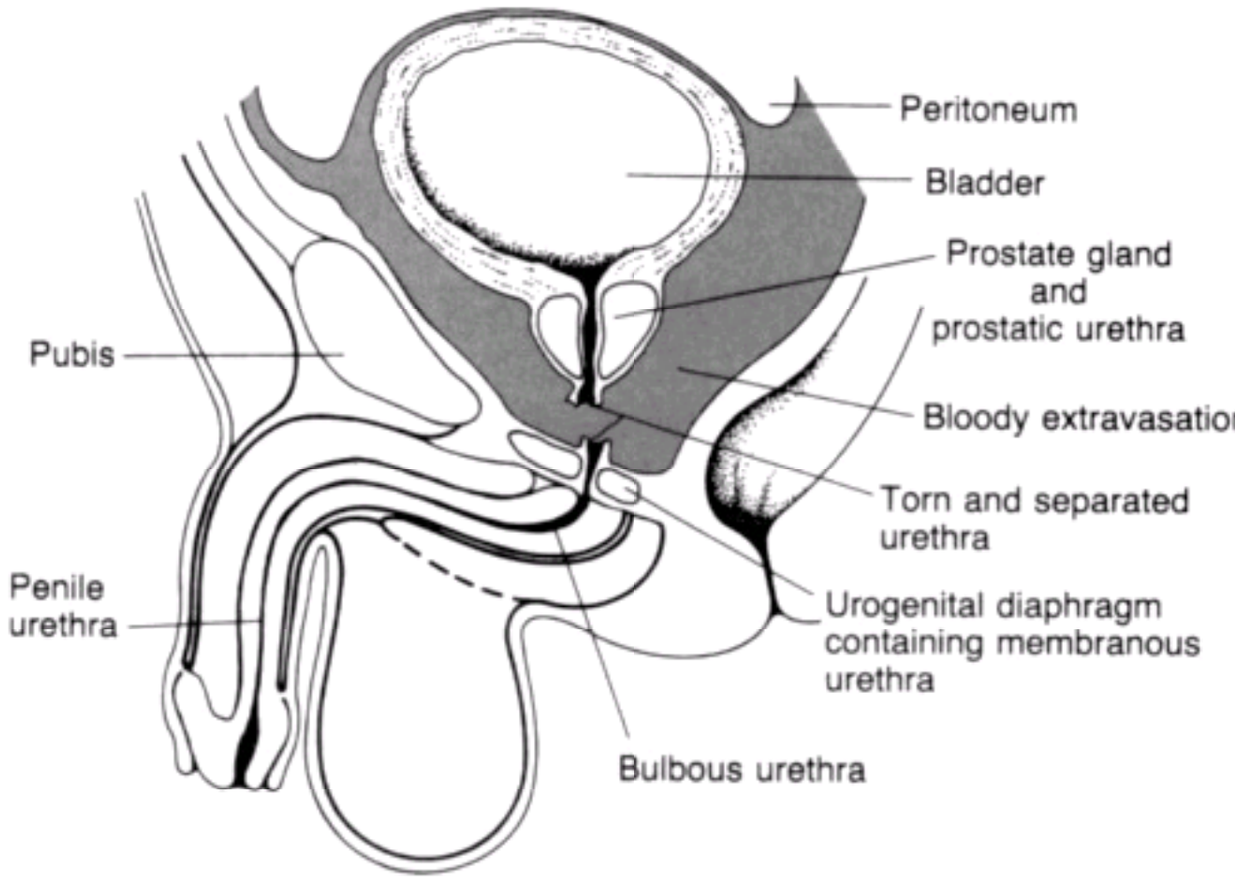
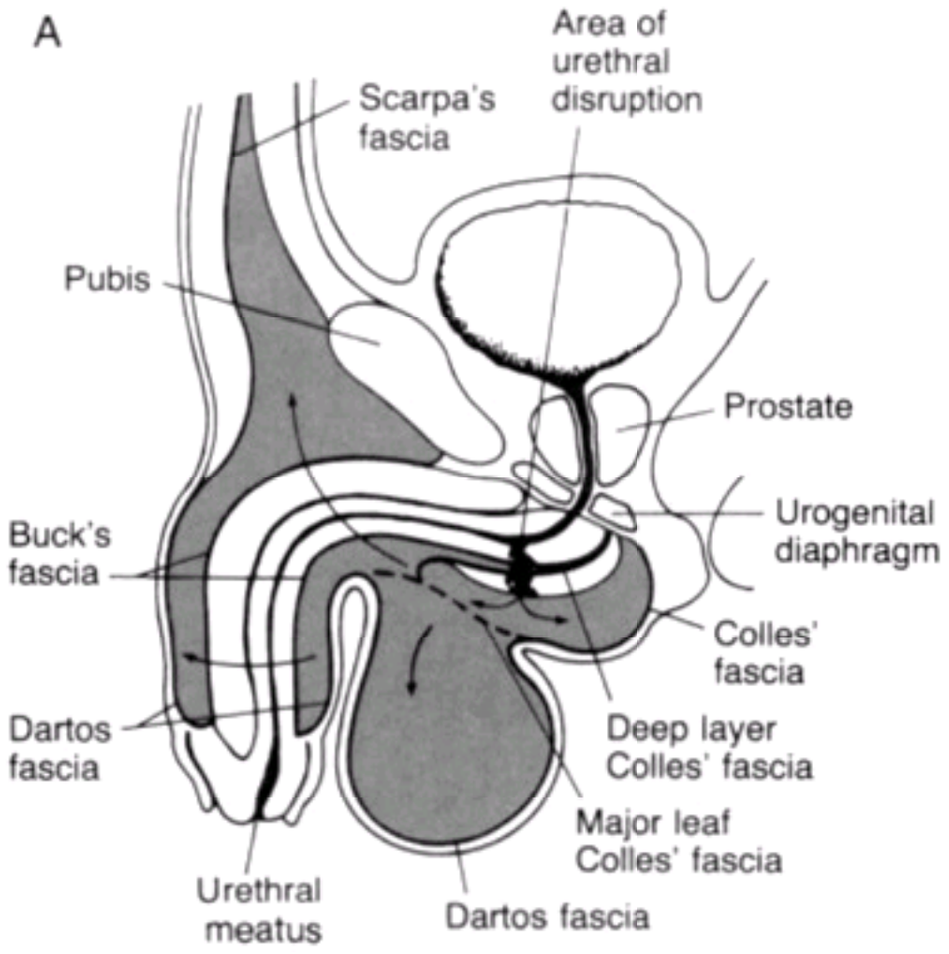


Figure 56-28 *A,* Disruption of the anterior urethra (bulbous urethra) occurs with straddle-type injuries in the male. Extravasation of urine and blood may occur in the perineum or scrotum, or along the anterior abdominal wall. Note that in this diagram, Buck fascia has been penetrated. *B,* Anterior urethral injury in which Buck fascia remains intact. In this situation, extravasation is confirmed and results in a swollen and ecchymotic penis. Such an injury usually results from instrumentation of the anterior urethra.



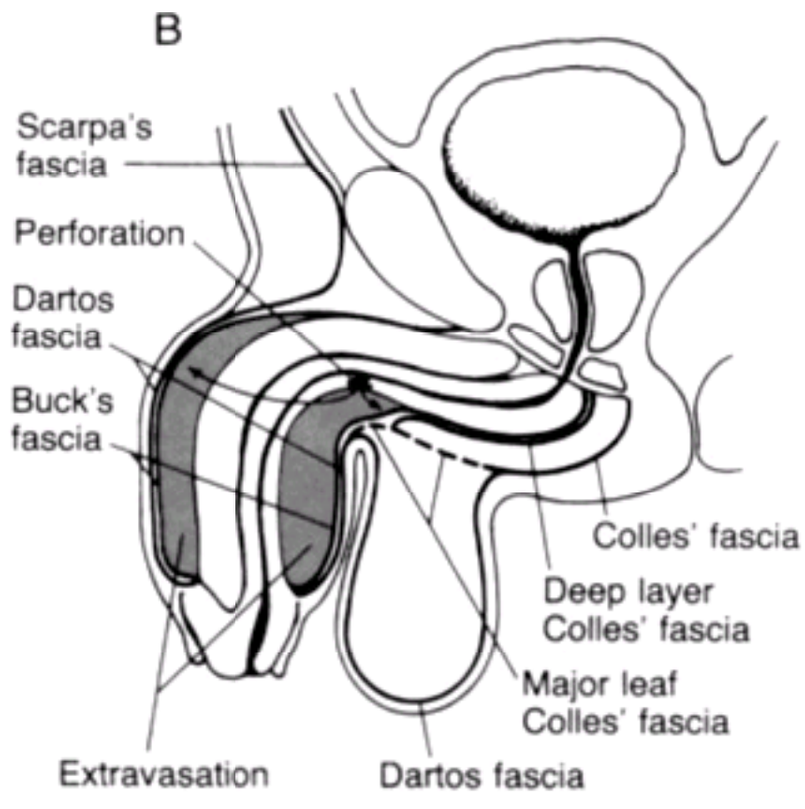
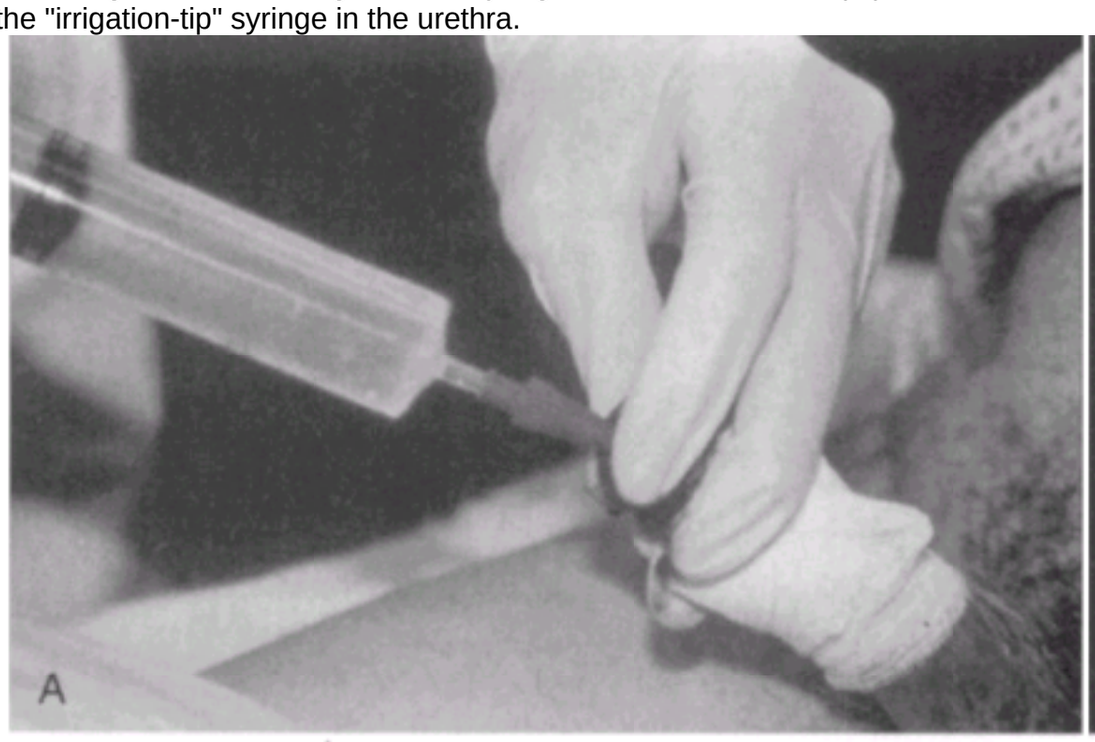
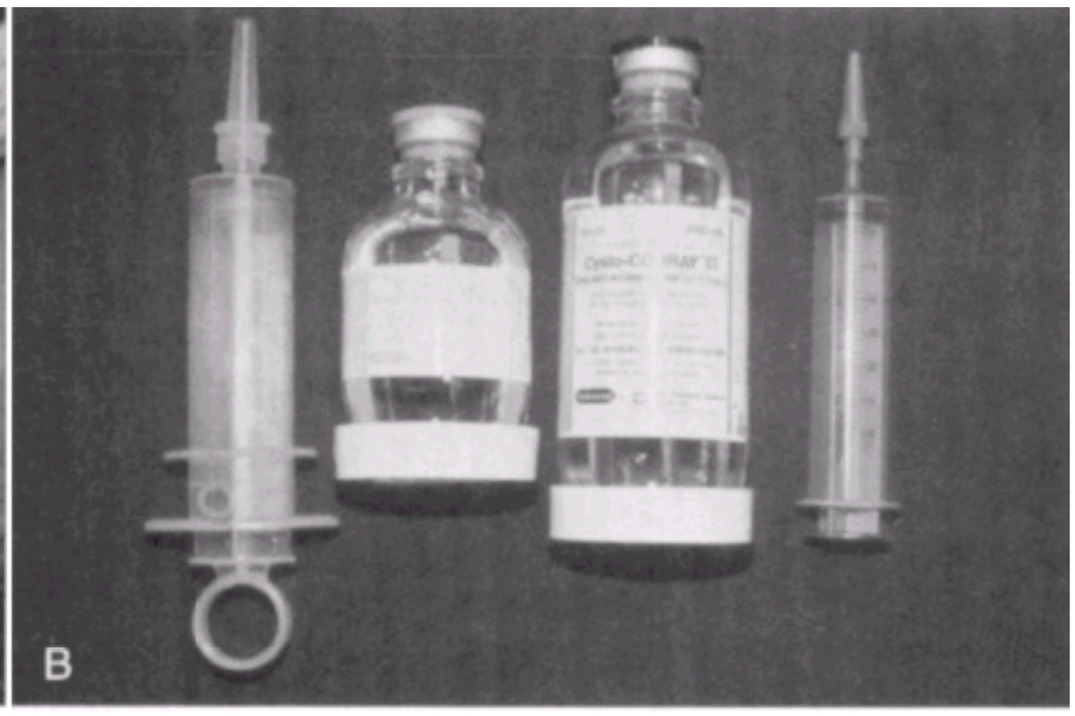


Figure 56-29 Retrograde urethrogram. The foreskin is fully retracted and an unwrapped 4 × 4 gauze sponge is folded in half longitudinally and wrapped around the penis proximal to the coronal sulcus, to prevent foreskin reduction. *A,* The penis is held between the long and ring fingers of the nondominant hand. The thumb and index finger ensure a snug fit of the syringe in the urethra. *B,* Equipment needed for retrograde urethrography and cystography. *C,* Alternative technique for securing the "irrigation-tip" syringe in the urethra.





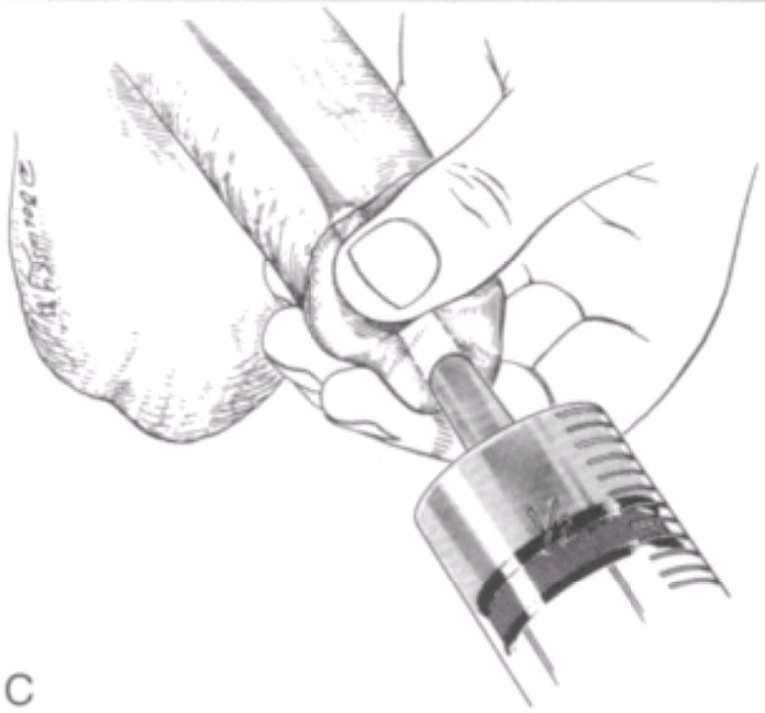


Figure 56-30 Normal retrograde urethrogram. The patient is supine on the examination table. The penis is stretched perpendicularly across the patient's right thigh to allow urethral unfolding and complete urethral visualization.

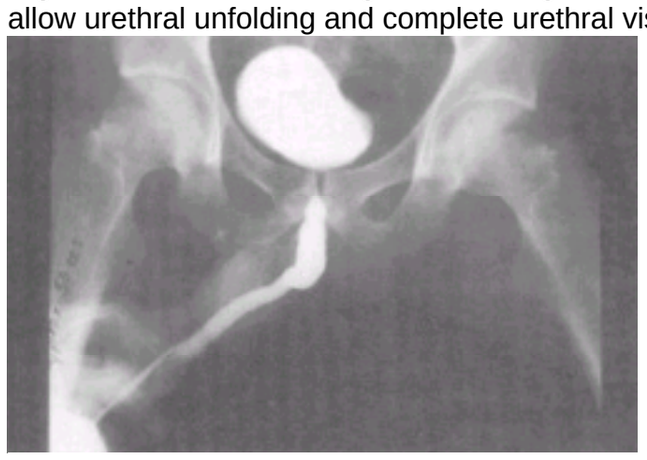


Figure 56-31 Venous intravasation (*arrows*) during a forceful retrograde urethrogram. This may mimic urethral extravasation, but it clears immediately, as opposed to actual extravasation, which remains indefinitely. The presence of intravasation is benign. *(From Richter MW, Lytton B, Myerson D, Grnja V: Radiology of genitourinary trauma. Radiol Clin North Am 11(3):626, 1973.)*

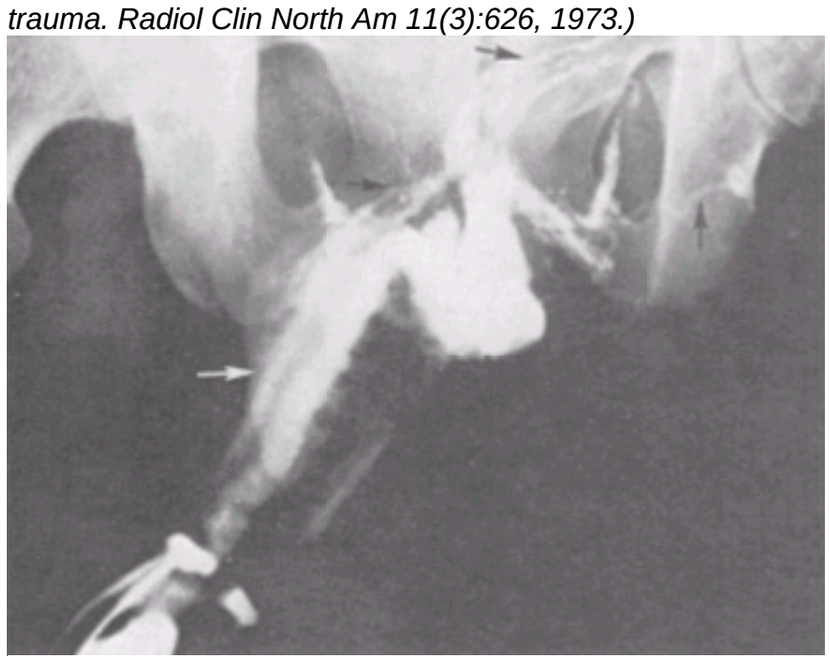


Figure 56-32 Retrograde urethrogram using a Foley catheter (8 Fr). Slowly inflate the balloon with 2 mL of air, tap water, or sterile saline to create a snug fit; then slowly inject 60 mL of a 10% solution of contrast material through the catheter lumen (see text).

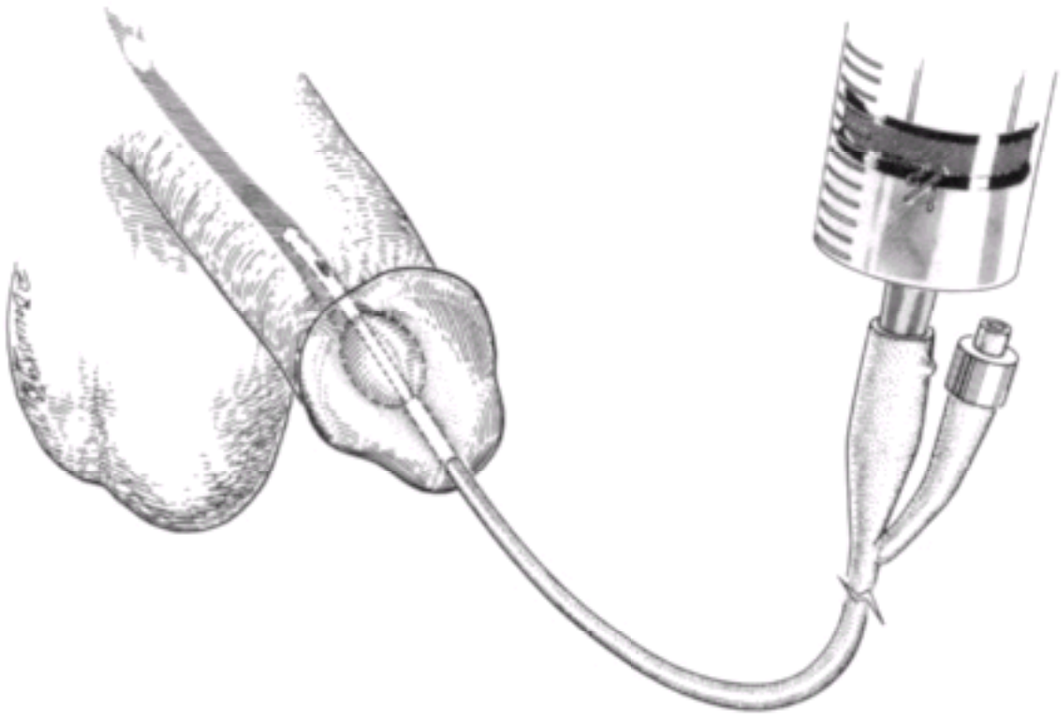


Figure 56-33 *A,* Retrograde urethrogram. Urethrogram in case of supramembranous urethral rupture. Contrast extravasation is typical of that seen with this type of injury. *B,* A rupture at the proximal bulbous urethra into the scrotum (*arrows*). *C,* Residual contrast material within perineum and scrotum. *(A from Morehouse DD, MacKinnon KJ: Posterior urethral injury: Etiology, diagnosis, initial management. Urol Clin North Am 4:74, 1977. C from Richter MW, Lytton B, Myerson D, Grnja V: Radiology of genitourinary trauma. Radiol Clin North Am 11:627, 1973.)*

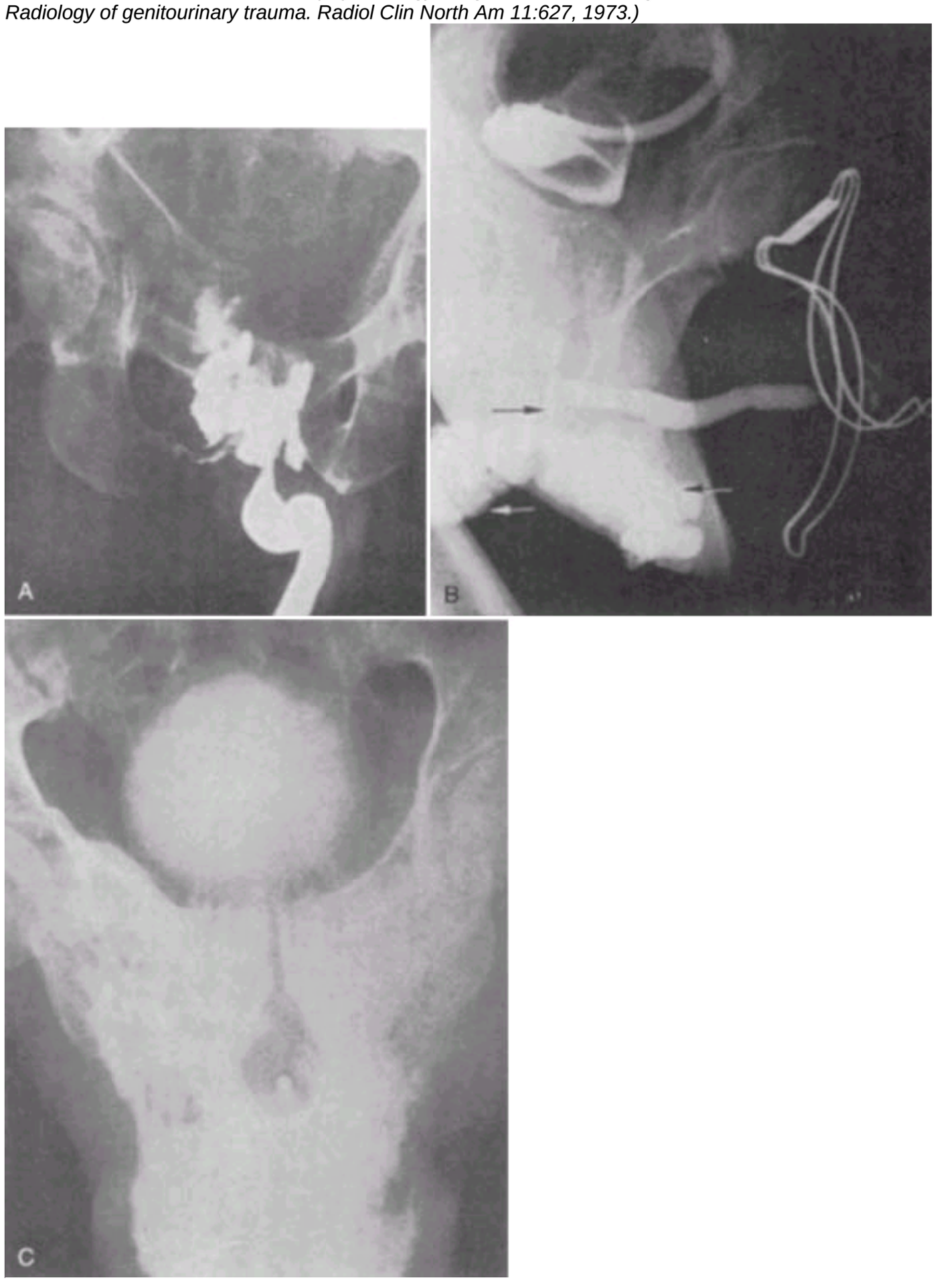


Figure 56-34 Evaluation of a urethral injury with a Foley catheter in place. A lubricated pediatric feeding tube has been advanced into the urethra beside the indwelling Foley catheter.

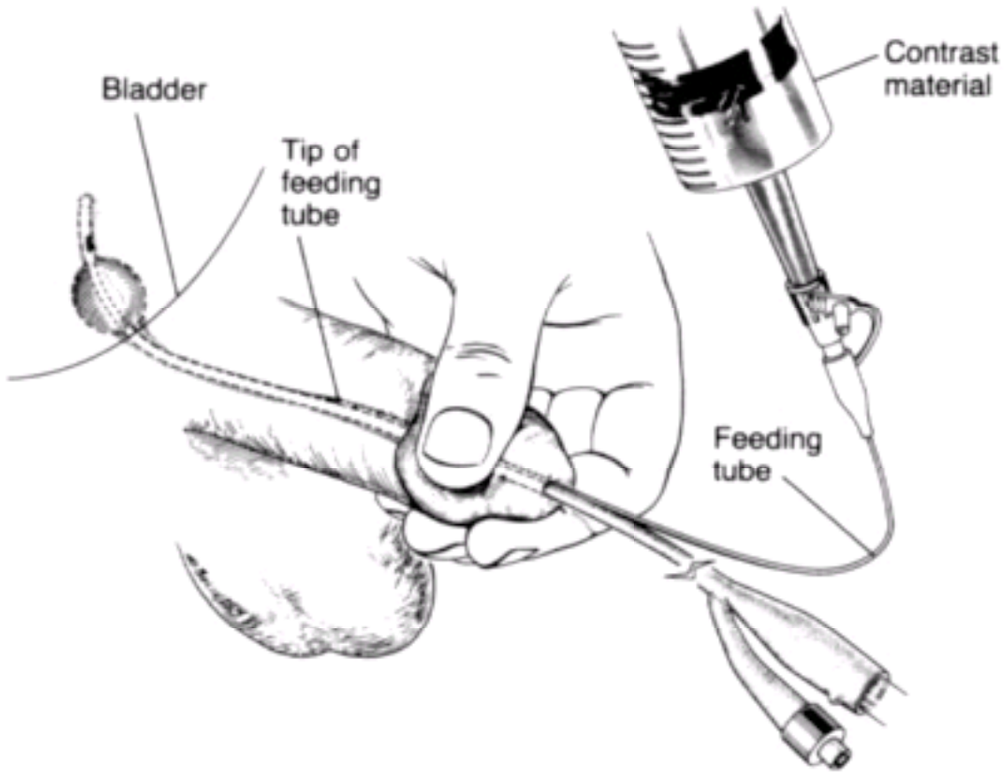


Figure 56-35 Retrograde cystogram. *A,* In patients with pelvic fracture, retrograde cystography should be done with the patient supine throughout the examination. Here, gross extravasation is evident, but its superior extent is not well defined. *B,* A lateral film may help define the extent of any extravasation. This film shows no intraperitoneal extension, so the extravasation must be totally extraperitoneal.

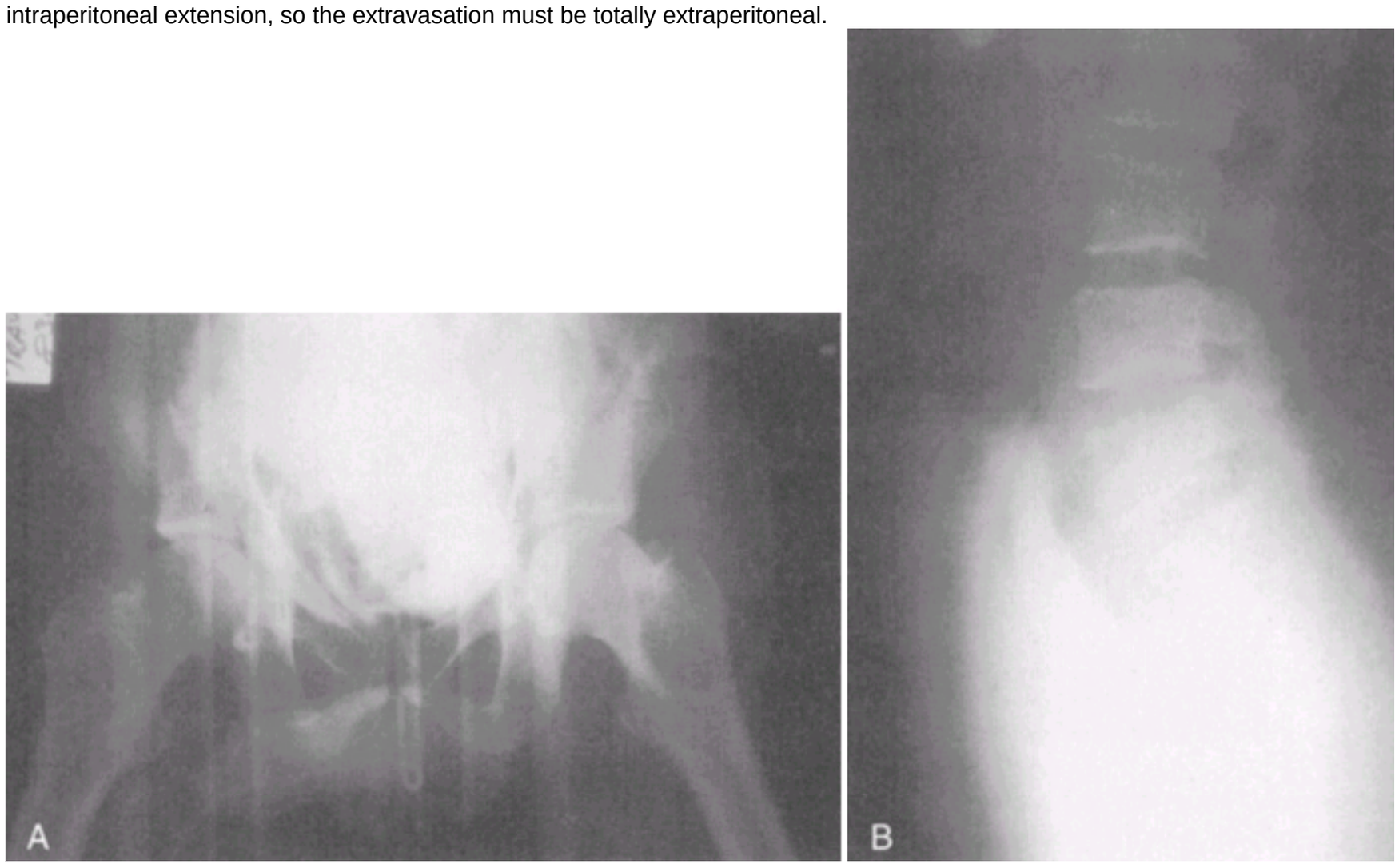


Figure 56-36 Retrograde cystogram. *A,* Anteroposterior (AP) filled-bladder film. *B,* AP postevacuation film of same patient showing extensive extravasation not seen on the AP filled-bladder film. Balloon of catheter is identified by *arrows*. *C,* "KUB" (kidneys, ureters, and bladder) showing bullet in the area of pelvis. *D,* AP filled-bladder film of same patient showing bladder displacement to right, presumably from a pelvic hematoma. No extravasation is visible with the bladder full of contrast. *E,* AP postevacuation film of same patient, showing subtle contrast extravasation in area of bullet that could easily be missed without a high-quality preliminary KUB and post-evacuation film.

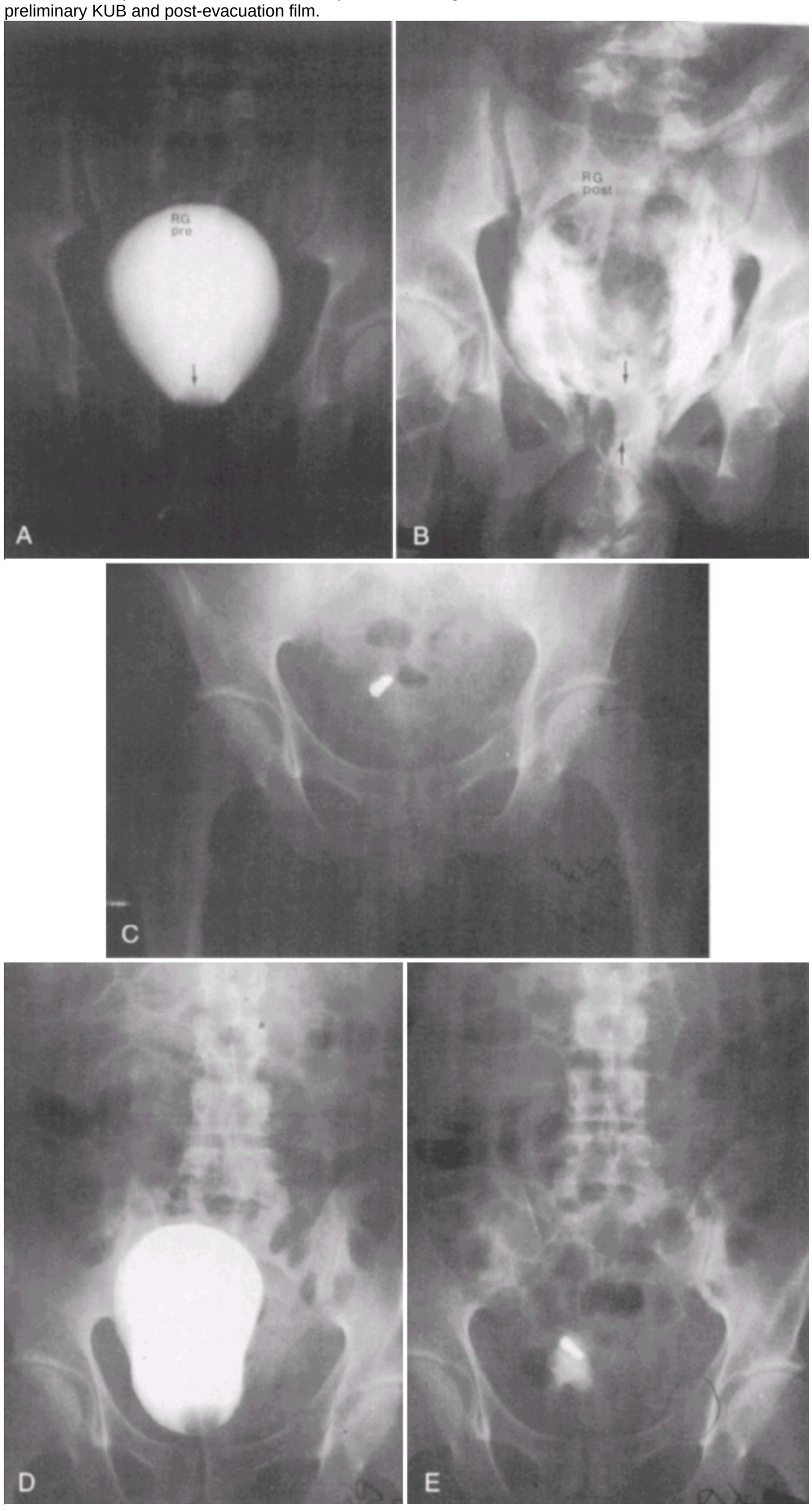


Figure 56-37 Examples of extraperitoneal bladder rupture. *A,* Note the amorphous extravasation of contrast material within the perivesical space (*arrows*) in a patient with a right pelvic fracture (*arrowheads*). *B,* A second patient with a pelvic fracture (*arrowhead*) and perivesical hematoma shows the teardrop shape of a deformed bladder and extraperitoneal extravasation (*arrows*). *(From Richter MW, Lytton B, Myerson D, Grnja V: Radiology of genitourinary trauma. Radiol Clin North Am 11:623, 1973.)*

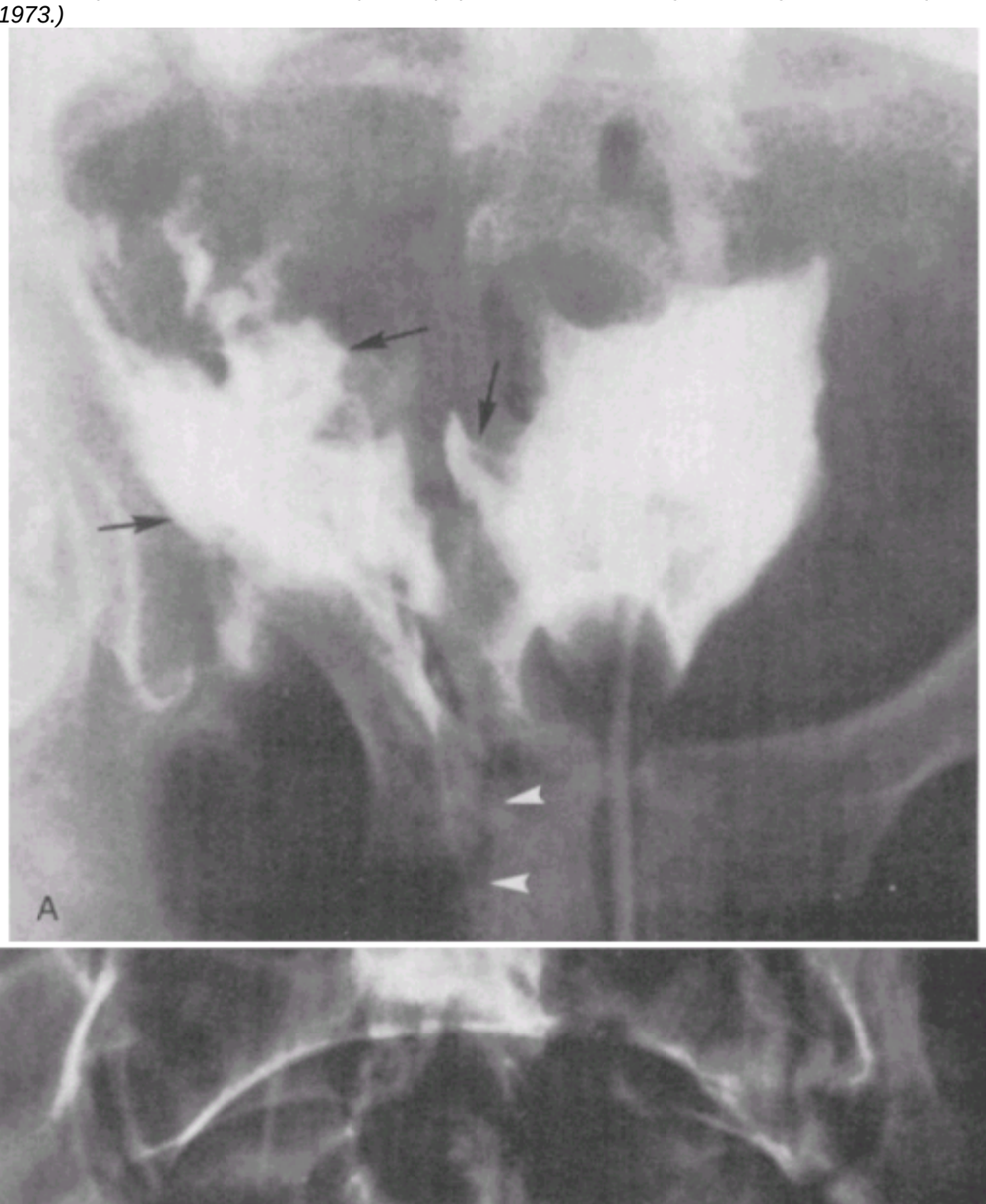


Figure 56-38 Intraperitoneal bladder rupture. *A,* A 22-year-old pedestrian hit by an automobile. Note extravasation of contrast material beginning at the dome and tracking up the left paracolic gutter (*arrows*). *B,* This 57-year-old man had fulguration of a bladder tumor at the bladder dome and sustained perforation. A cystogram dramatically demonstrates the extravasation of contrast material that outlines the bowel loops (*arrows*) and the paracolic gutters. *(Courtesy of Morton A, Bosniak MD, New York.) (From Richter MW, Lytton B, Myerson D, Grnja V: Radiology of genitourinary trauma. Radiol Clin North Am 11:623, 1973.)*

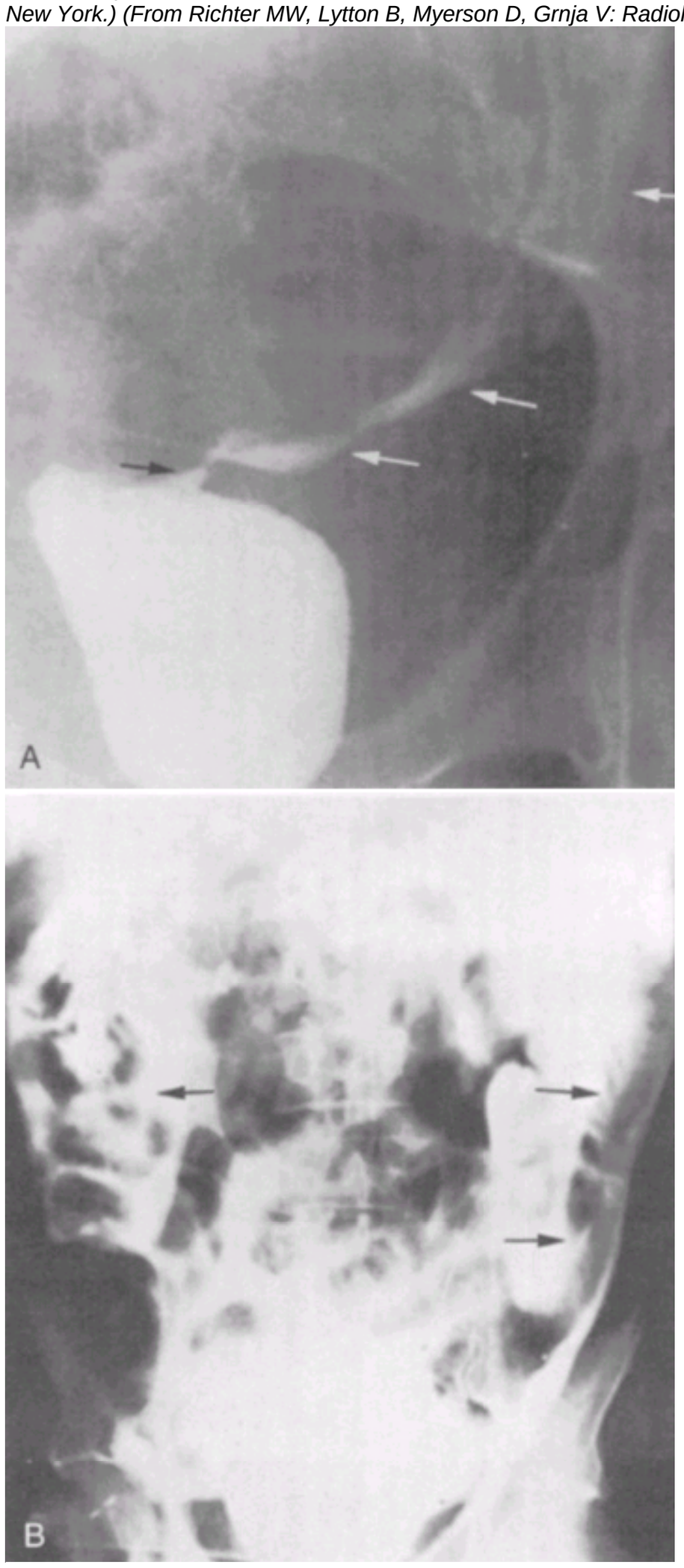


Figure 56-39 Retrograde cystogram and abdominal computed tomography (CT) scan. These two procedures can be done concomitantly. The bladder is filled in the standard retrograde fashion and the catheter is clamped. Intravenous and oral contrast can then be administered and CT scanning performed. This film demonstrates contrast ascites, which is consistent with intraperitoneal bladder rupture and extravasation.

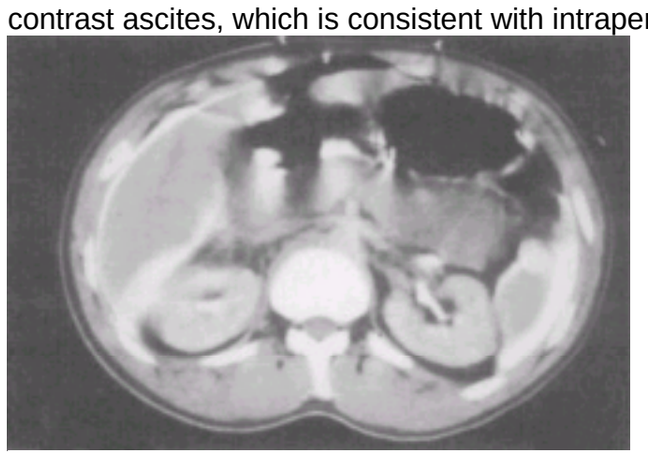


Figure 57-6 Station of the fetal head. As a reference point, the level of the ischial spines is zero station. *(From Benson RC [ed]: Current Obstetric and Gynecologic Diagnosis and Treatment, 3rd ed. Los Altos, Calif., Lange Medical Publications, 1980.)*

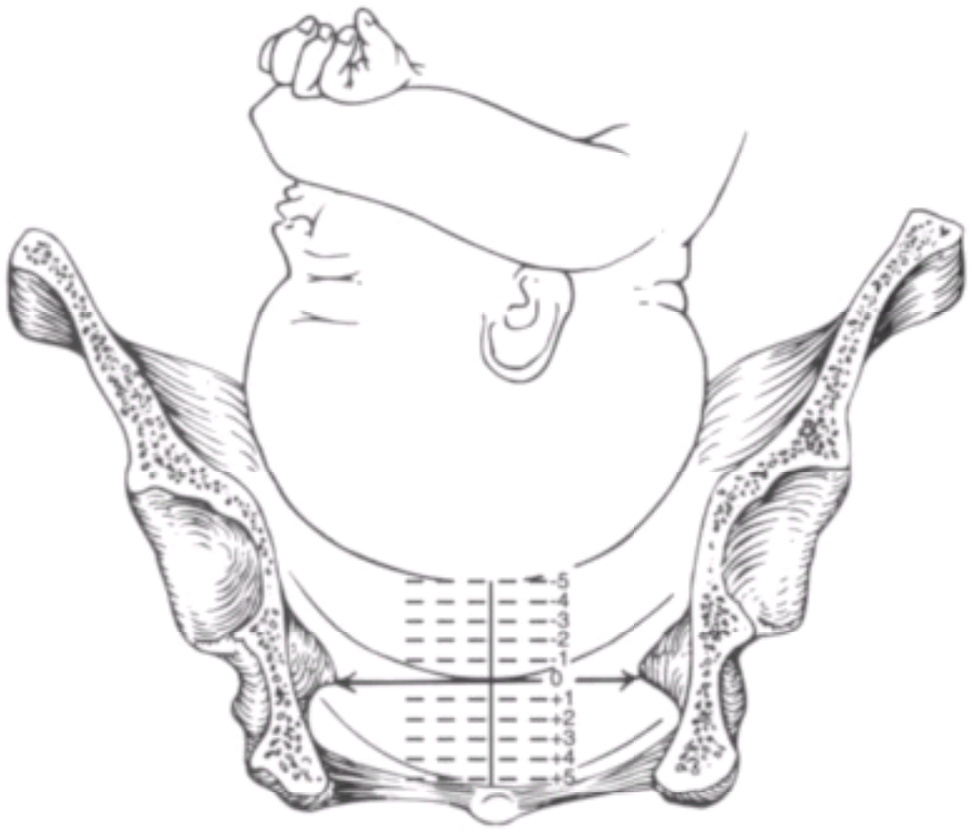


Figure 57-8 Mechanism of labor for breech presentation. *A,* Before labor. *B,* Engagement of the buttocks, internal rotation. *C,* Lateral flexion of the trunk, delivery of the buttocks. *D,* External rotation of the buttocks, engagement of the shoulders. *E,* Internal rotation of the shoulders, delivery of the posterior shoulder. *F,* Lateral flexion of the trunk, delivery of the anterior shoulder. *(Adapted from Benson RC [ed]: Current Obstetric and Gynecologic Diagnosis and Treatment, 3rd ed. Los Altos, Calif., Lange Medical Publications, 1980.)*

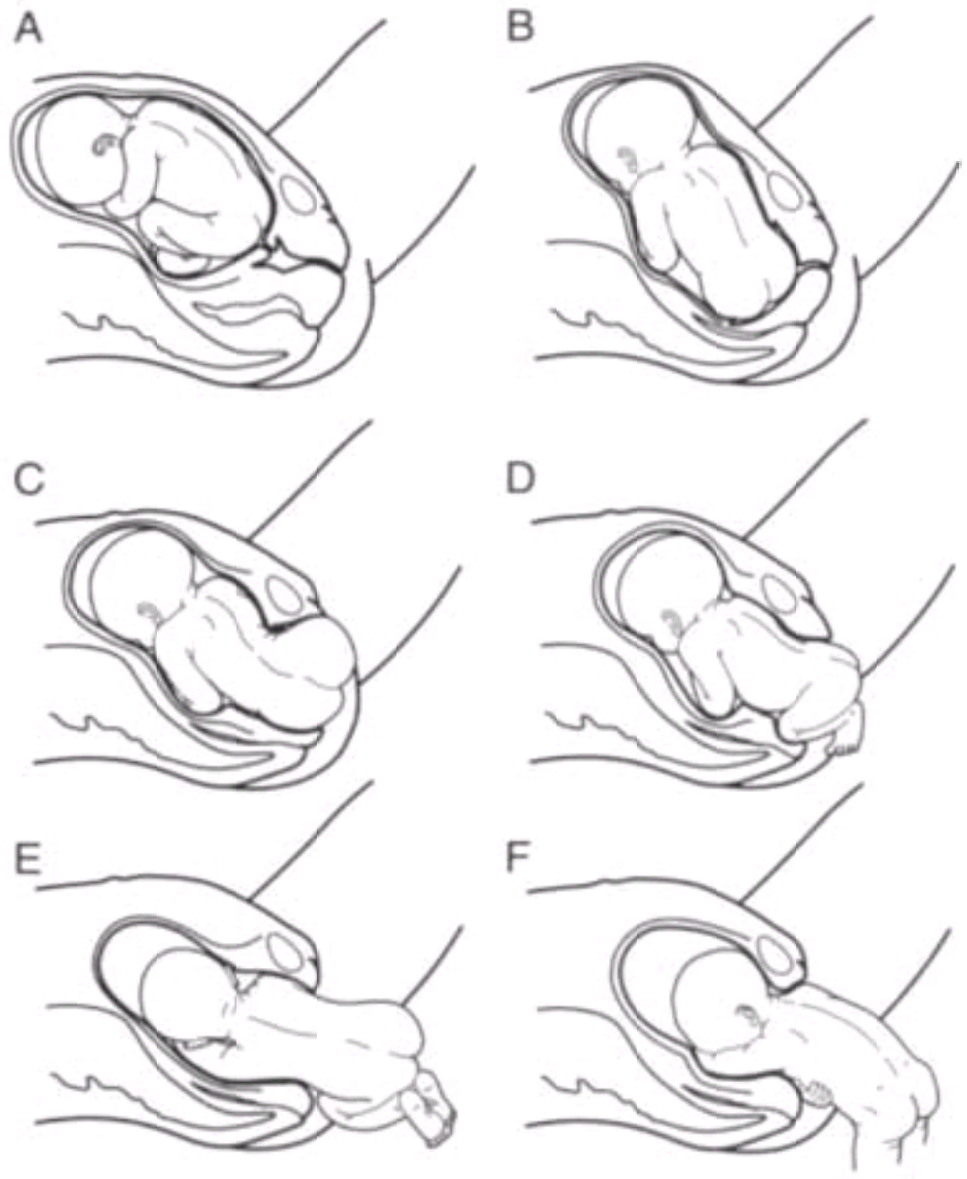


Figure 57-9 Modified Ritgen maneuver. *(From Pritchard JA, MacDonald PC: Williams Obstetrics, 16th ed. New York, Appleton-Century-Crofts, 1980.)*

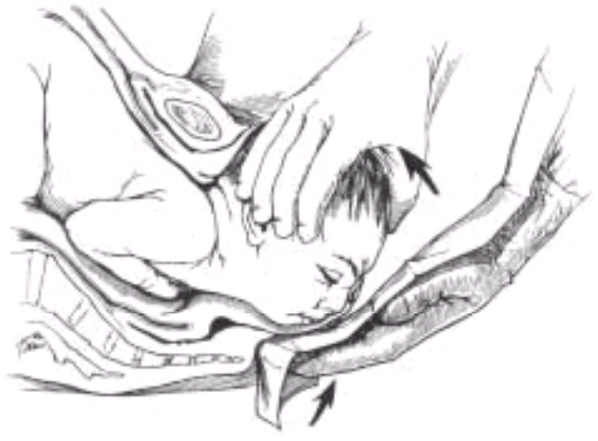


Figure 57-10 Checking for the cord around the infant's neck.

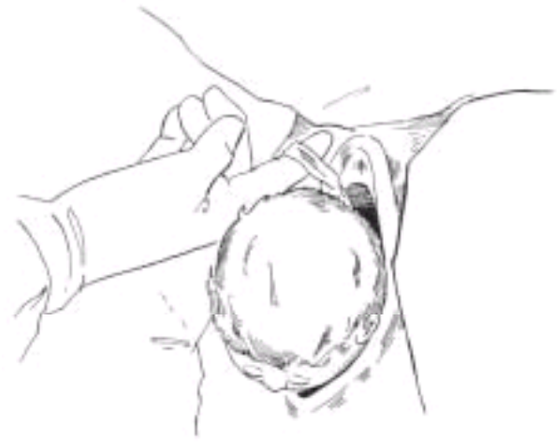


Figure 57-11 Delivery of the placenta. Gentle pressure is exerted through the abdominal wall to lift the uterine fundus upward while keeping the umbilical cord slightly taught. This maneuver is repeated until the placenta reaches the introitus.

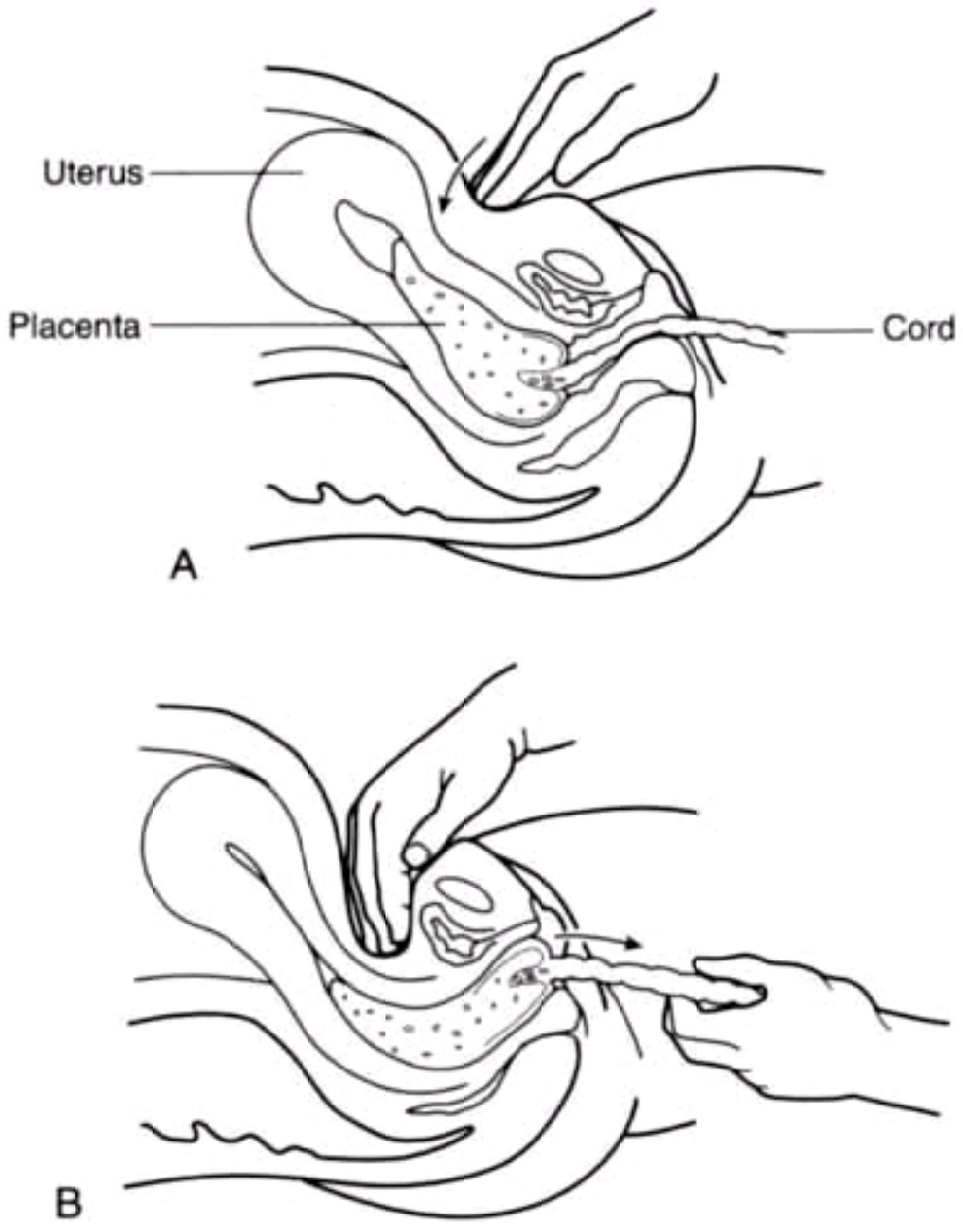


Figure 57-12 McRoberts maneuver for shoulder dystocia. The maternal hips are completely flexed, allowing the knees to rest on the abdomen. This causes cephalad rotation of the maternal pelvis and flattening of the lumbar lordosis resulting in an increase in the useful size of the pelvic outlet.

Figure 57-13 Rubin or reverse Wood's screw maneuver for shoulder dystocia. *A,* Rotation of the posterior shoulder. *B,* Delivery of the rotated shoulder.

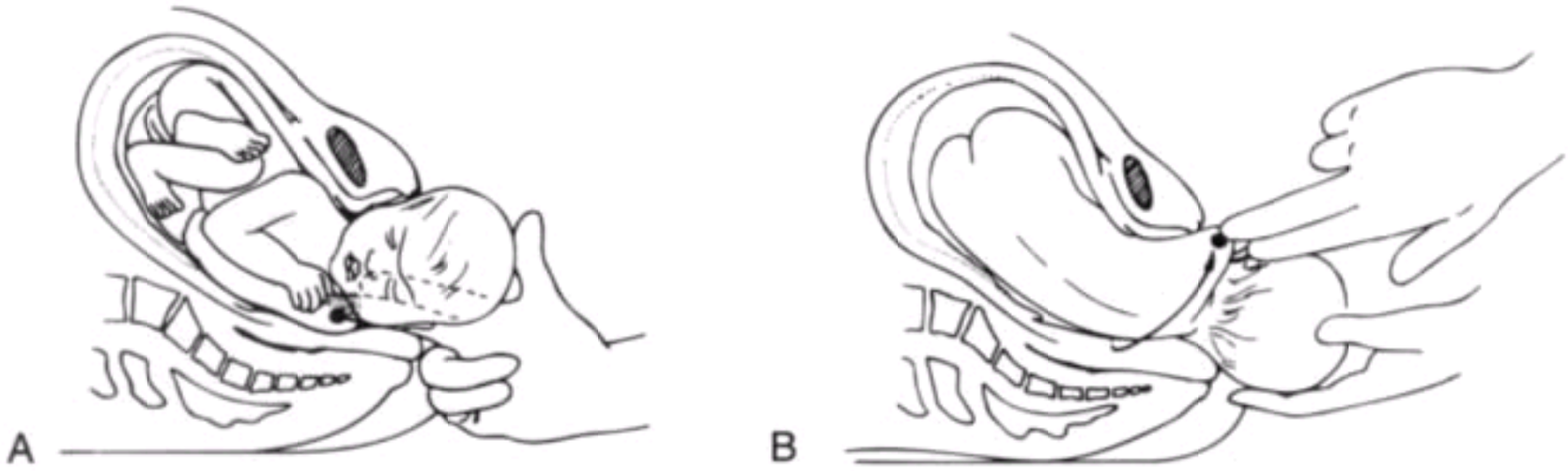


Figure 57-14 Delivery of the posterior arm for shoulder dystocia. *A,* Flexion of the posterior arm. *B,* delivery of the posterior arm to permit delivery of the anterior shoulder.

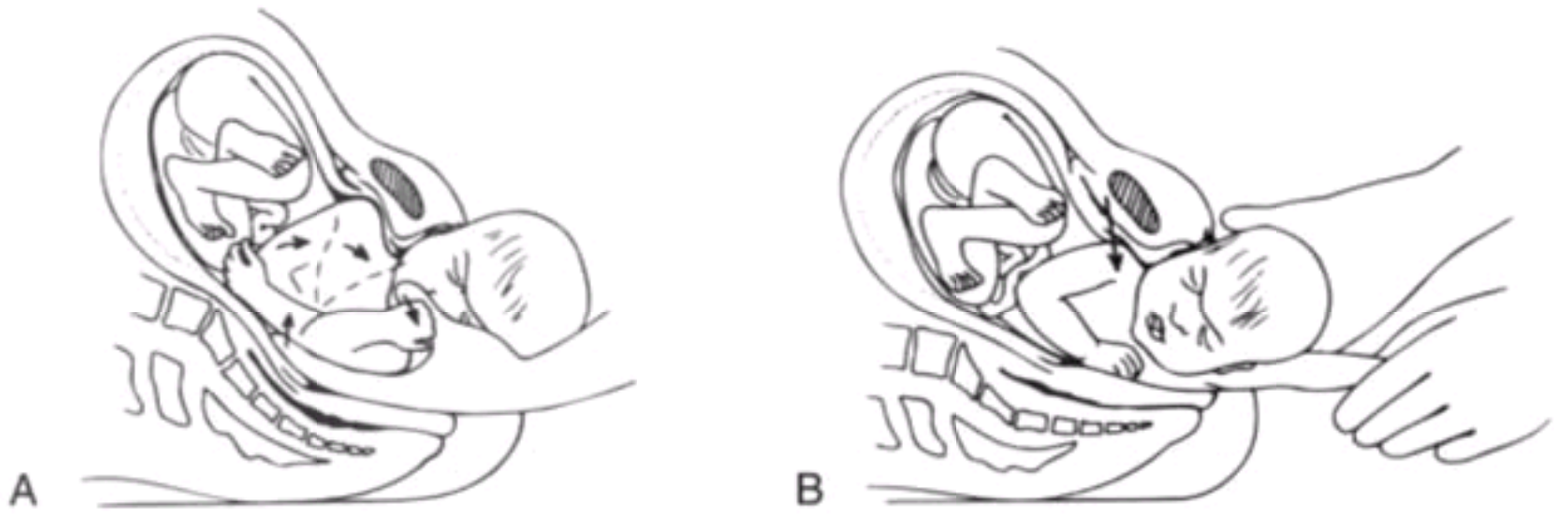


Figure 57-15 Assisted breech delivery. Once the breech has delivered to the level of the umbilicus, delivery of the legs may be aided by placing the fingers parallel to the medial thigh and sweeping the legs laterally away from the midline.

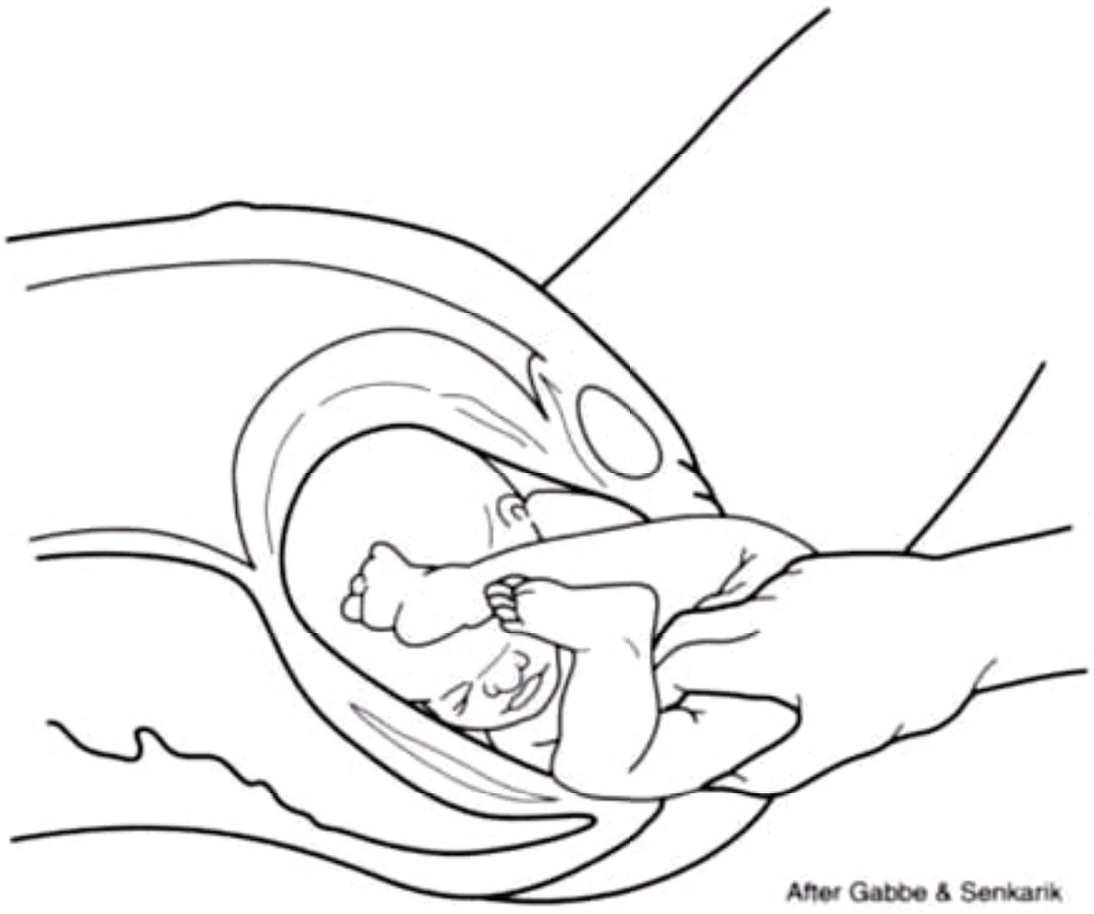


Figure 57-16 Assisted breech delivery. The clinician's hands are placed over the infant's sacrum to deliver the body. *(From Taylor ES: Obstetrics and Fetal Medicine, 2nd ed. Baltimore, Williams & Wilkins, 1977.)*

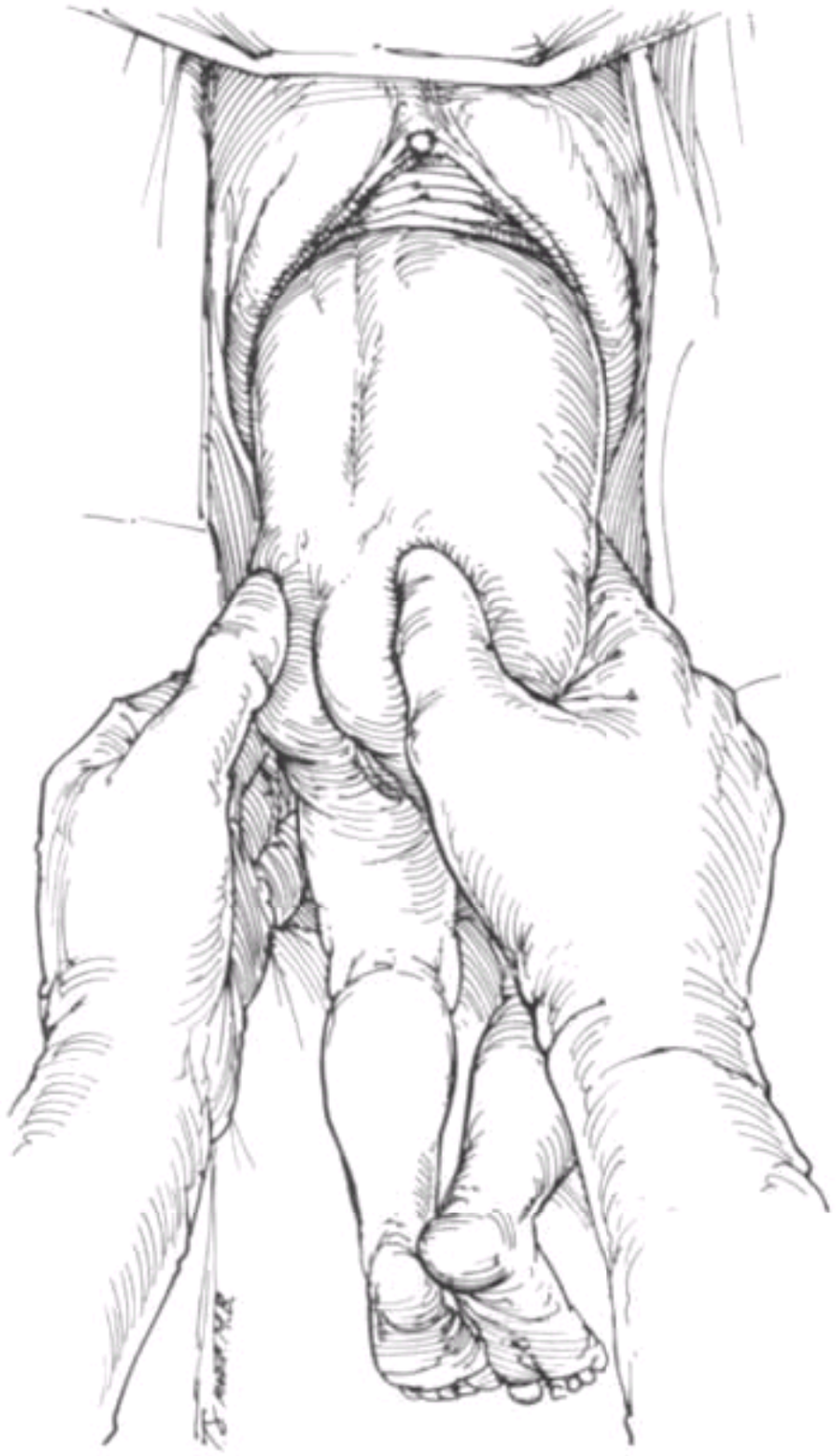


Figure 57-17 Assisted breech delivery. Rotation occurs as the scapulae emerge. *(From Taylor ES: Obstetrics and Fetal Medicine, 2nd ed. Baltimore, Williams & Wilkins, 1977.)*

Figure 57-18 Assisted breech delivery. *A,* Delivery of the posterior shoulder by upward traction on the fetal body. *B,* Delivery of the anterior shoulder beneath the symphysis by downward traction. *(From Taylor ES: Obstetrics and Fetal Medicine, 2nd ed. Baltimore, Williams & Wilkins, 1977.)*

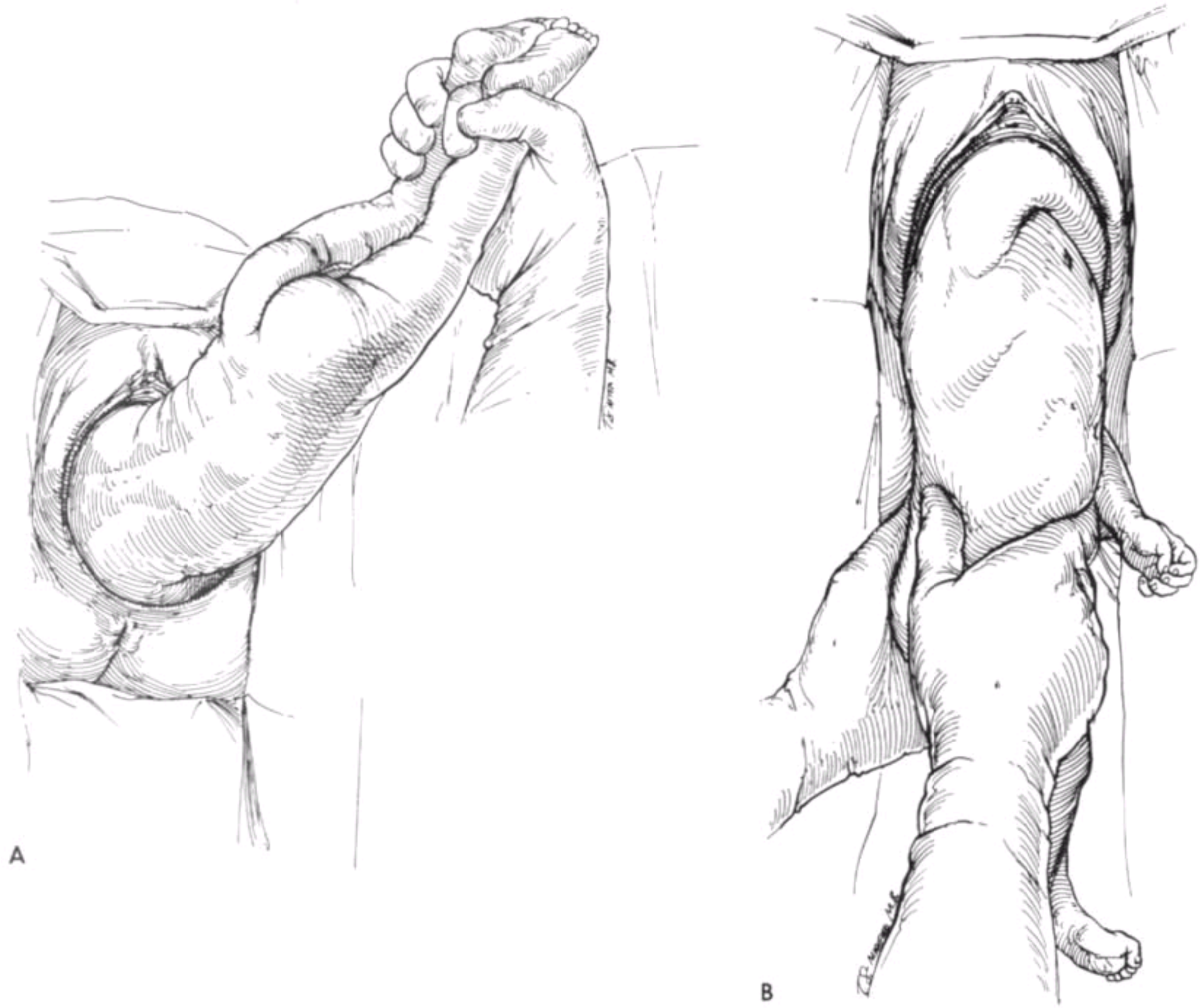


Figure 57-19 Mauriceau maneuver: delivery of the aftercoming head. While suprapubic pressure is applied by an assistant, the head is gently flexed by pressure on the maxilla. *(From Taylor ES: Obstetrics and Fetal Medicine, 2nd ed. Baltimore, Williams & Wilkins, 1977.)*

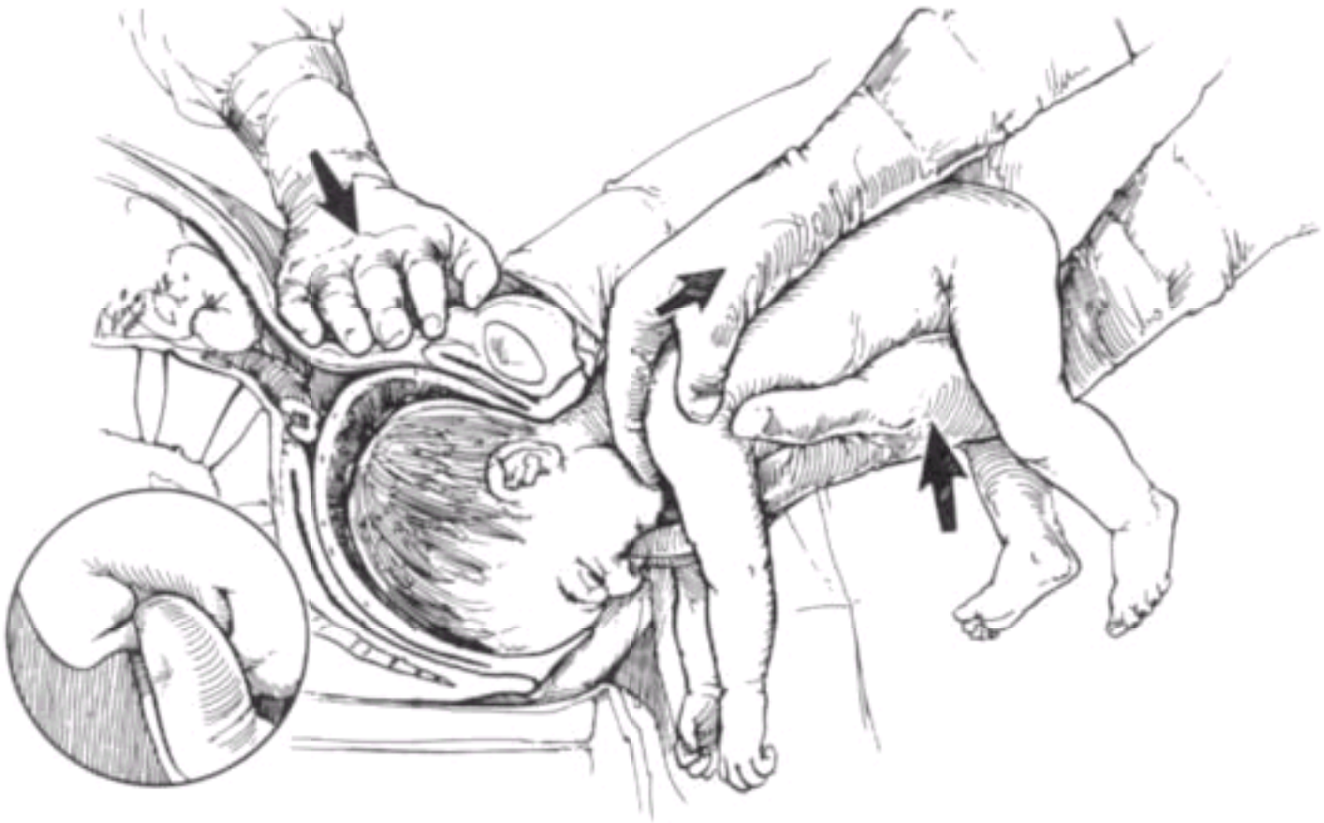


Figure 57-20 Breech extraction. Traction of the feet and ankles. Note that the index finger is placed between the ankles. *(From Pritchard JA, MacDonald PC: Williams Obstetrics, 16th ed. New York, Appleton-Century-Crofts, 1980.)*

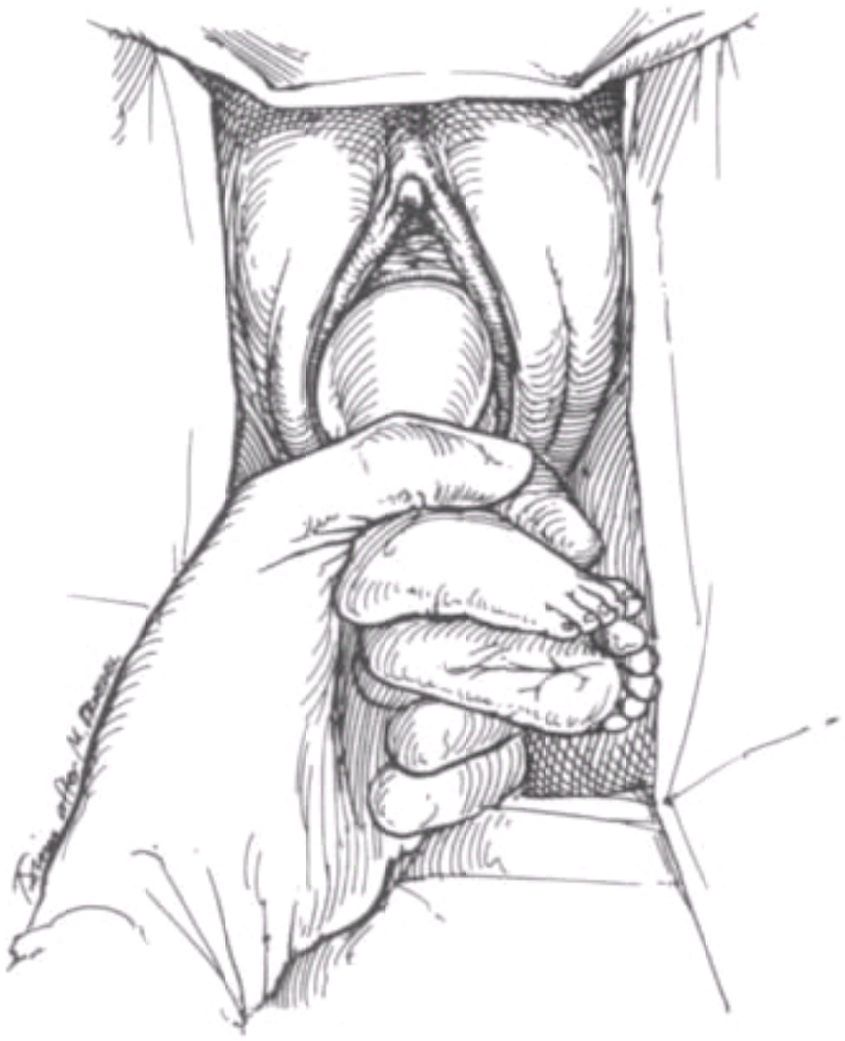


Figure 57-21 Breech extraction. Traction of the legs and thighs. *(From Taylor ES: Obstetrics and Fetal Medicine, 2nd ed. Baltimore, Williams & Wilkins, 1977.)*

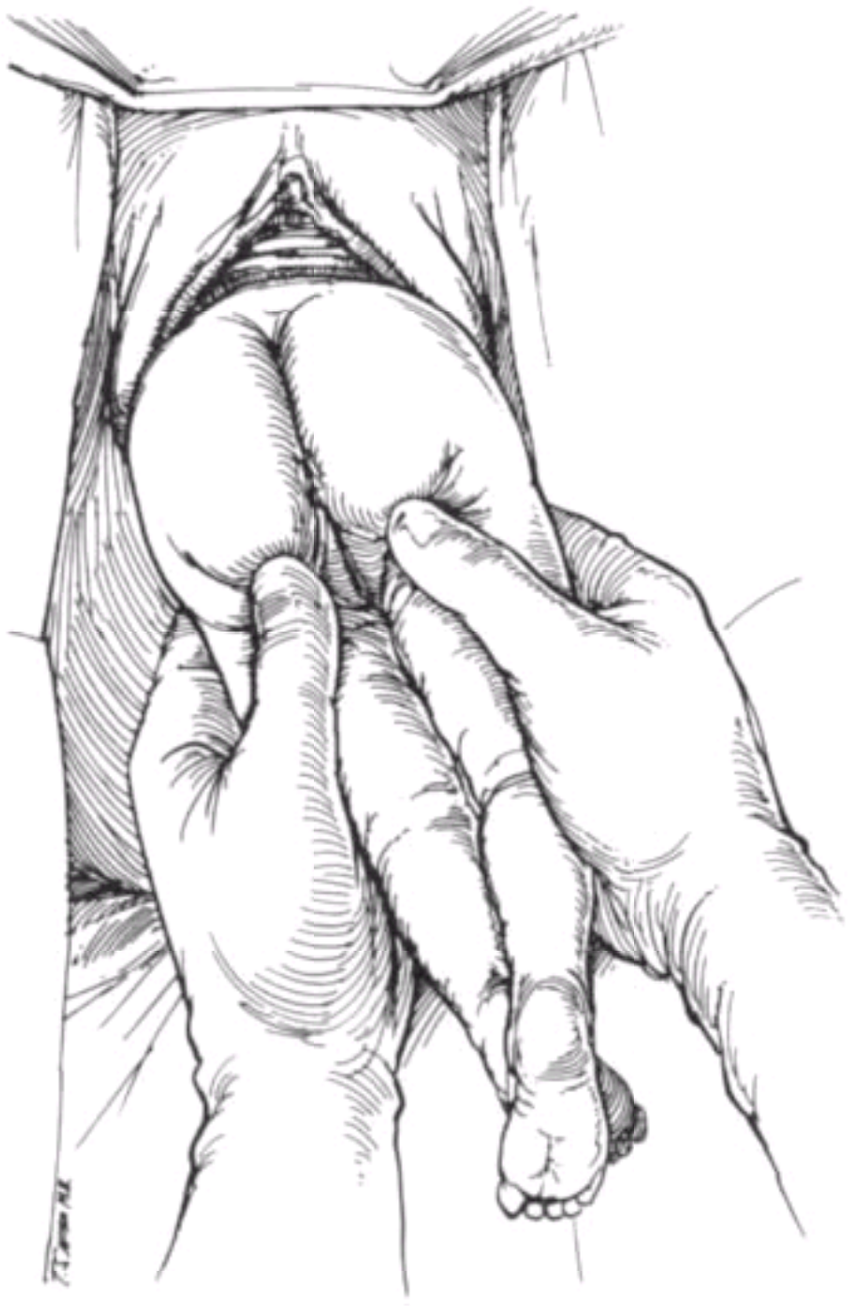


Figure 57-22 Breech extraction. Extraction of a frank breech by moderate traction exerted with a finger in each groin. *(From Pritchard JA, MacDonald PC: Williams Obstetrics, 16th ed. New York, Appleton-Century-Crofts, 1980.)*

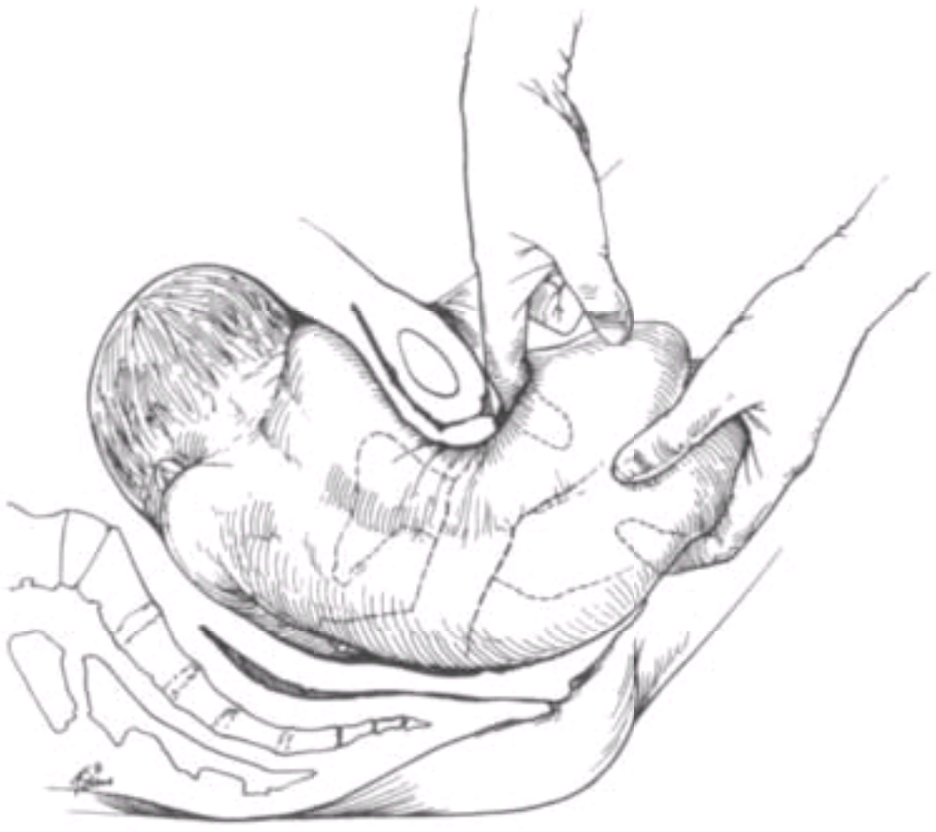


Figure 57-23 Types of episiotomy. The median episiotomy is the easiest to perform and repair.

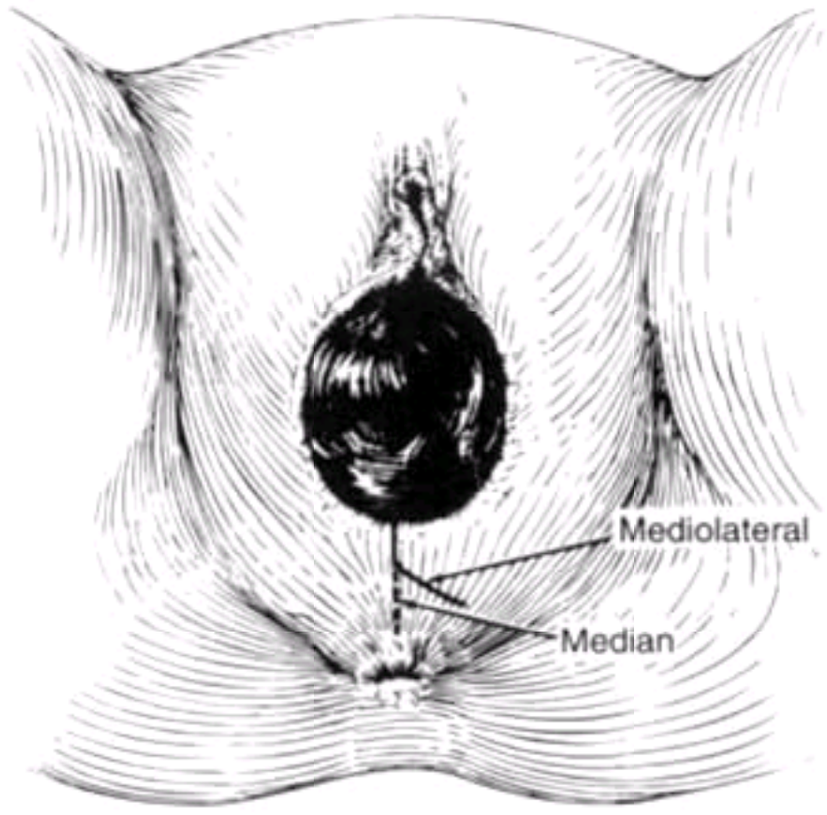


Figure 57-24 Midline (median) episiotomy.

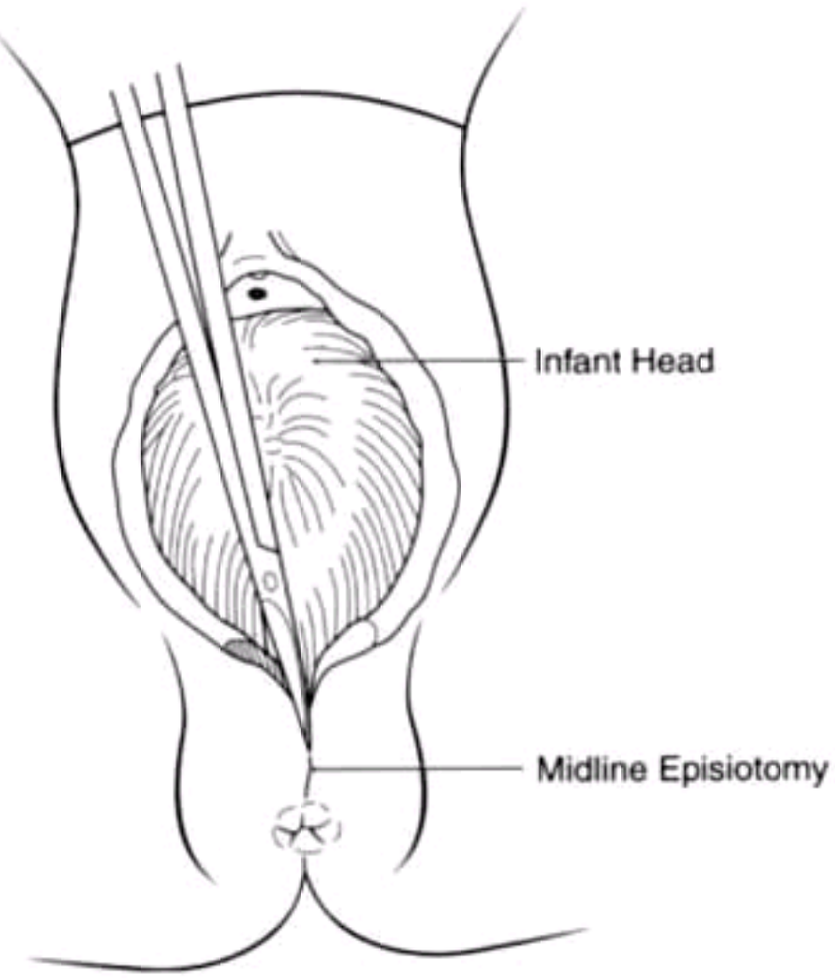


Figure 57-25 Closure of median episiotomy. *A,* Closure of mucosa and hymenal ring with continuous suture. *B,* Approximation of perineal musculature with interrupted sutures. *C,* Continuous suture to unite superficial fascia. *D,* Completion of repair by carrying continuous suture upward as a subcuticular stitch. *E,* Alternatively, closure of the superficial fascia and skin (*C* and *D*) may be accomplished by a series of loosely tied interrupted sutures. *(From Pritchard JA, MacDonald PC: Williams Obstetrics, 16th ed. New York, Appleton-Century-Crofts, 1980.)*

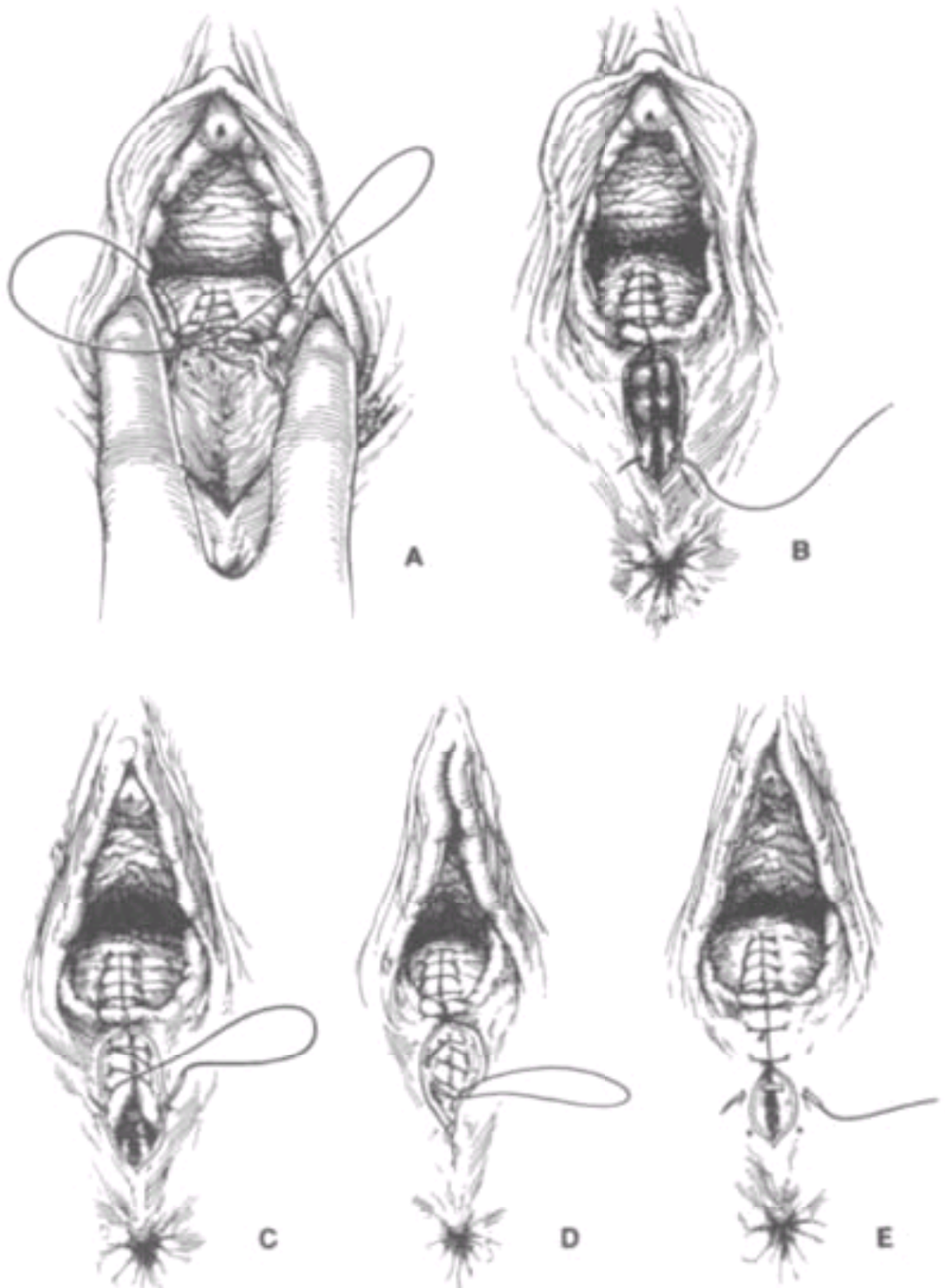


Figure 57-26 Uterine massage. One hand is used to massage the posterior aspect of the uterus through the abdominal wall. The other hand, inserted in the vagina, compresses the anterior uterus. *(From Pritchard JA, MacDonald PC: Williams Obstetrics, 16th ed. New York, Appleton-Century-Crofts, 1980.)*

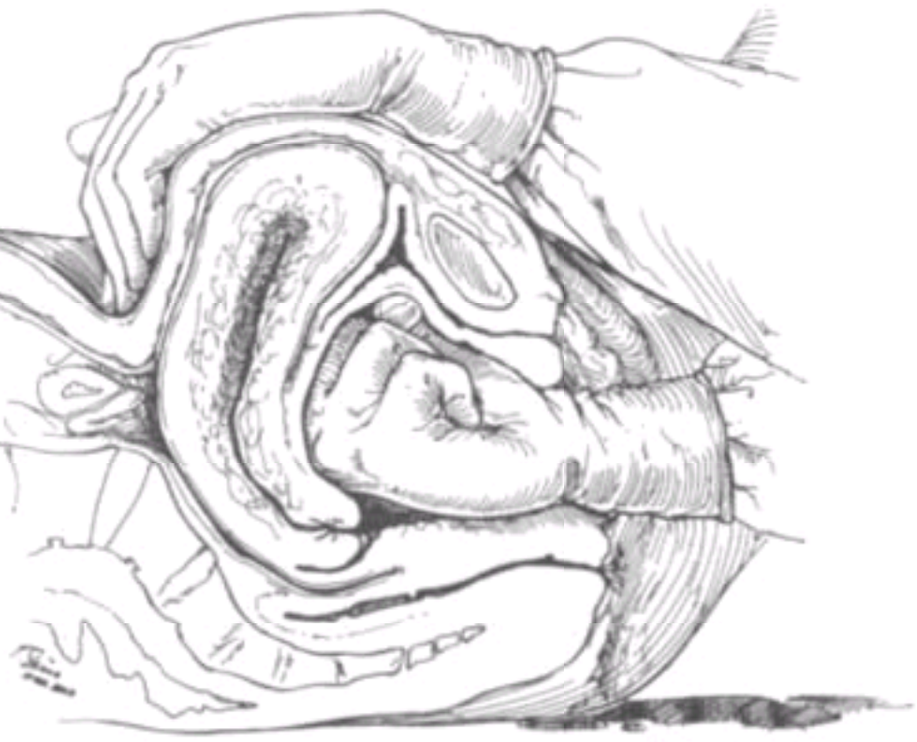


Figure 57-27 Replacement of the inverted uterus.

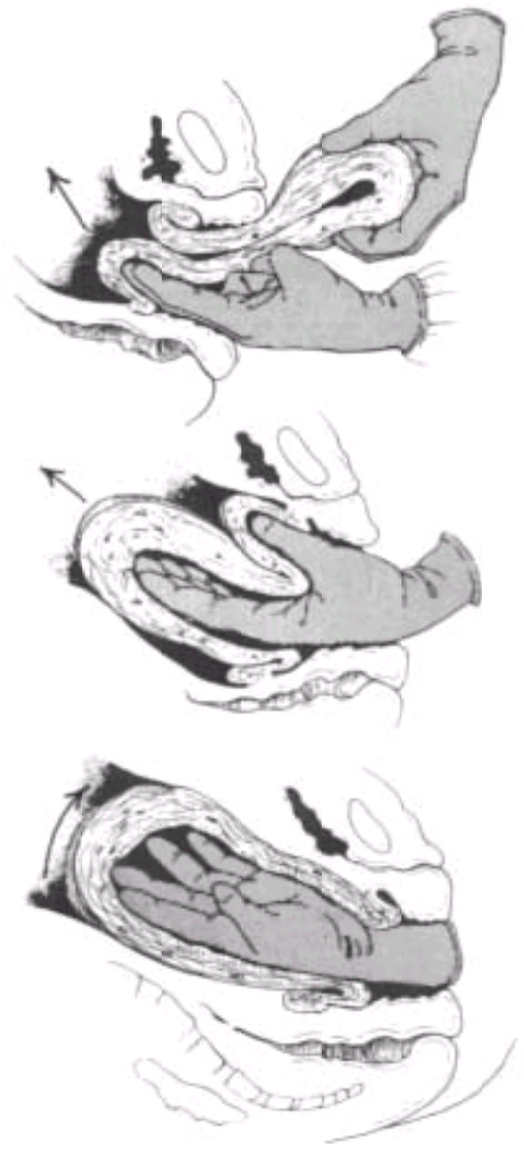


Figure 57-28 Cesarean delivery. A vertical incision is made through the abdominal wall from the level of the uterine fundus to the symphysis pubis.

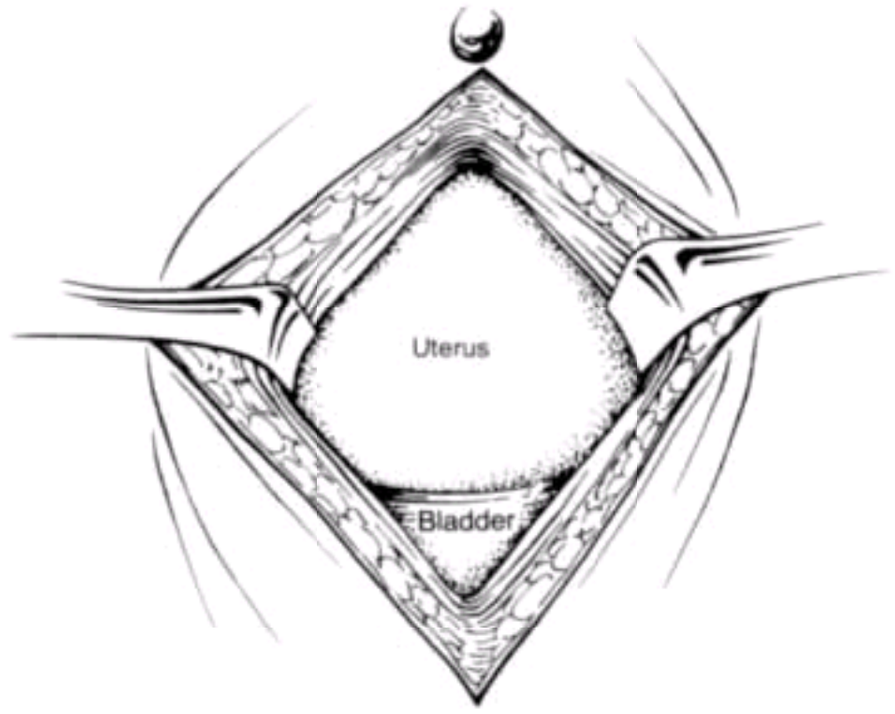


Figure 57-29 Cesarean delivery. If available, retractors are used to expose the anterior surface of the uterus, and the bladder is retracted inferiorly.

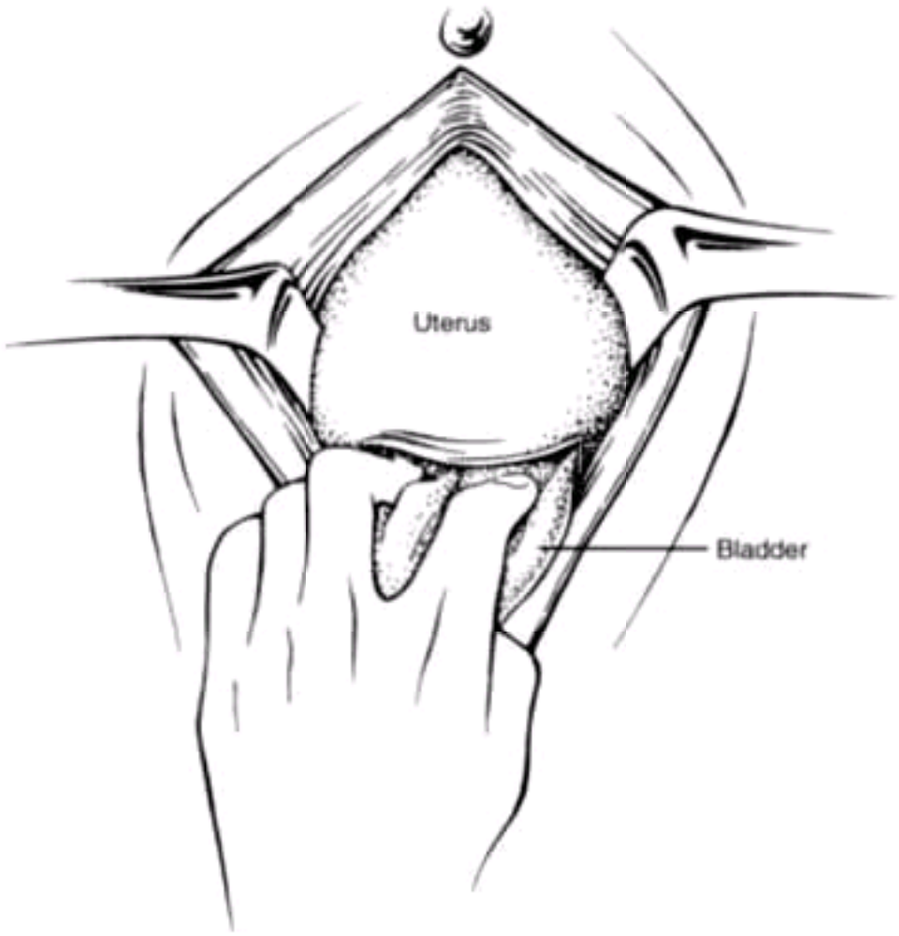


Figure 57-30 Cesarean delivery. *A,* Small vertical incision is made with a scalpel through the lower uterine segment. *B,* Bandage scissors are used to extend the incision vertically to the fundus.

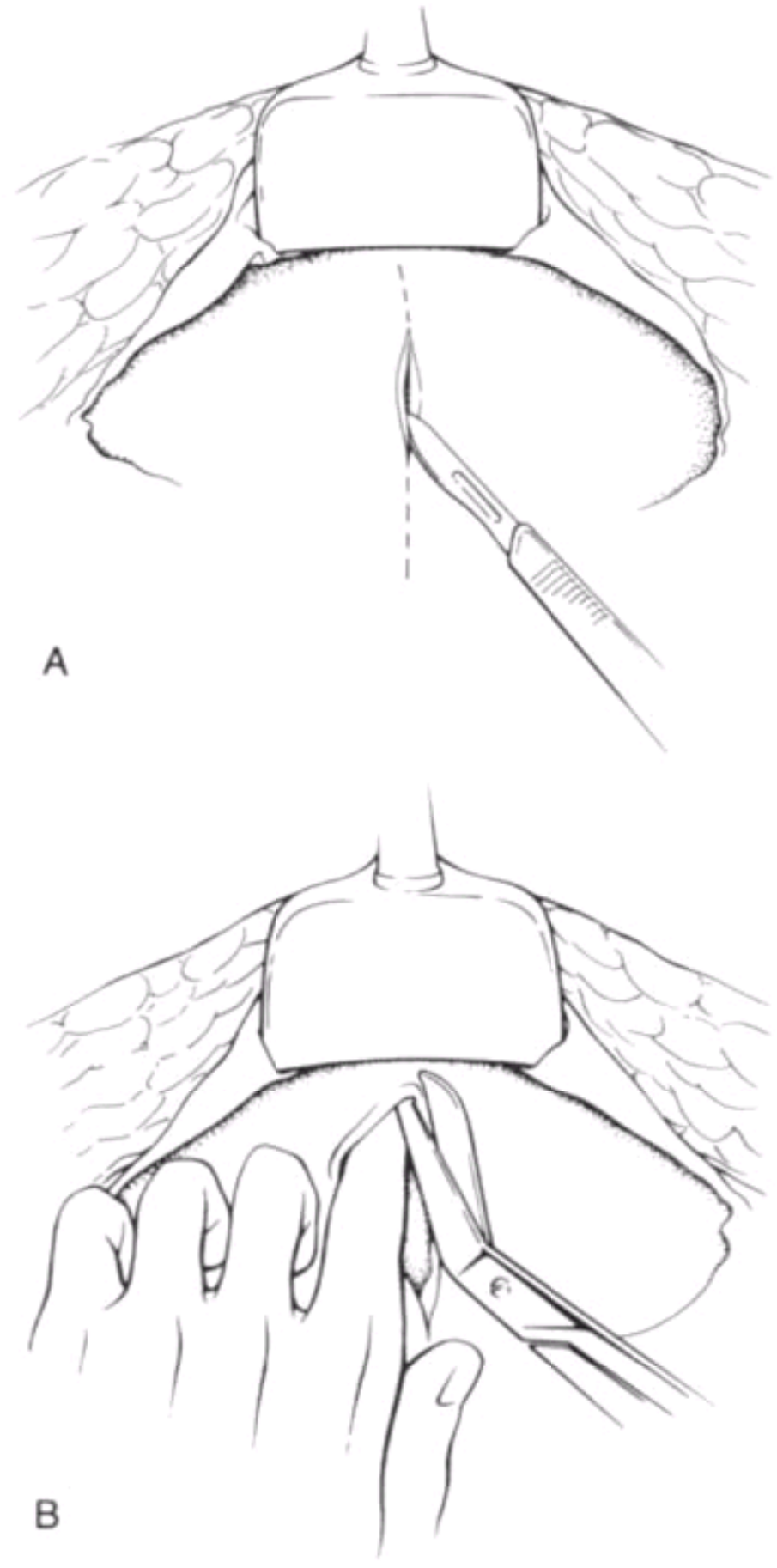


Figure 57-31 Cesarean delivery. The infant is delivered, the nose and mouth suctioned, and the cord clamped and cut.

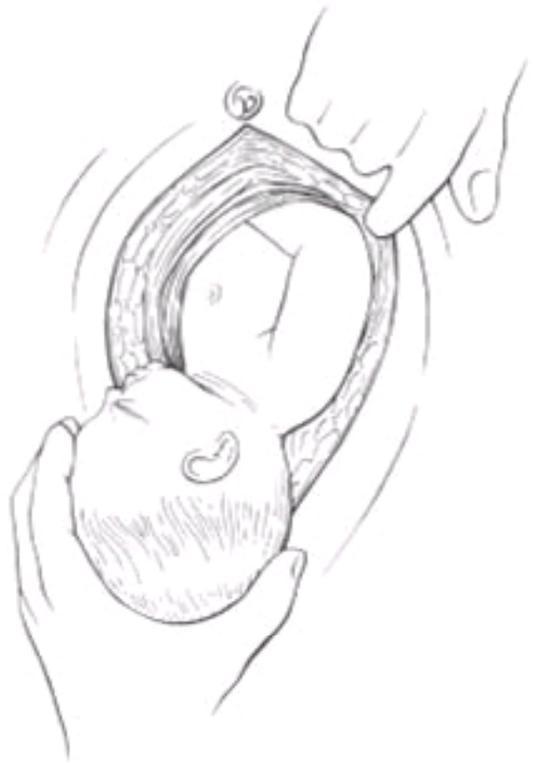


Figure 58-1 This decidual cast, a perfect outline of the uterine cavity, was initially thought to be products of conception when found in the vaginal vault of a pregnant woman treated for abdominal pain and vaginal bleeding. The initial diagnosis was a spontaneous abortion, but this cast is virtually diagnostic of an ectopic pregnancy. The woman later developed hypotension and was found to have a ruptured tubal pregnancy.

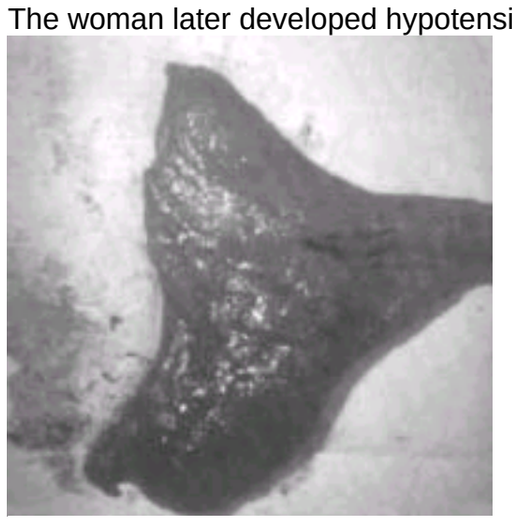
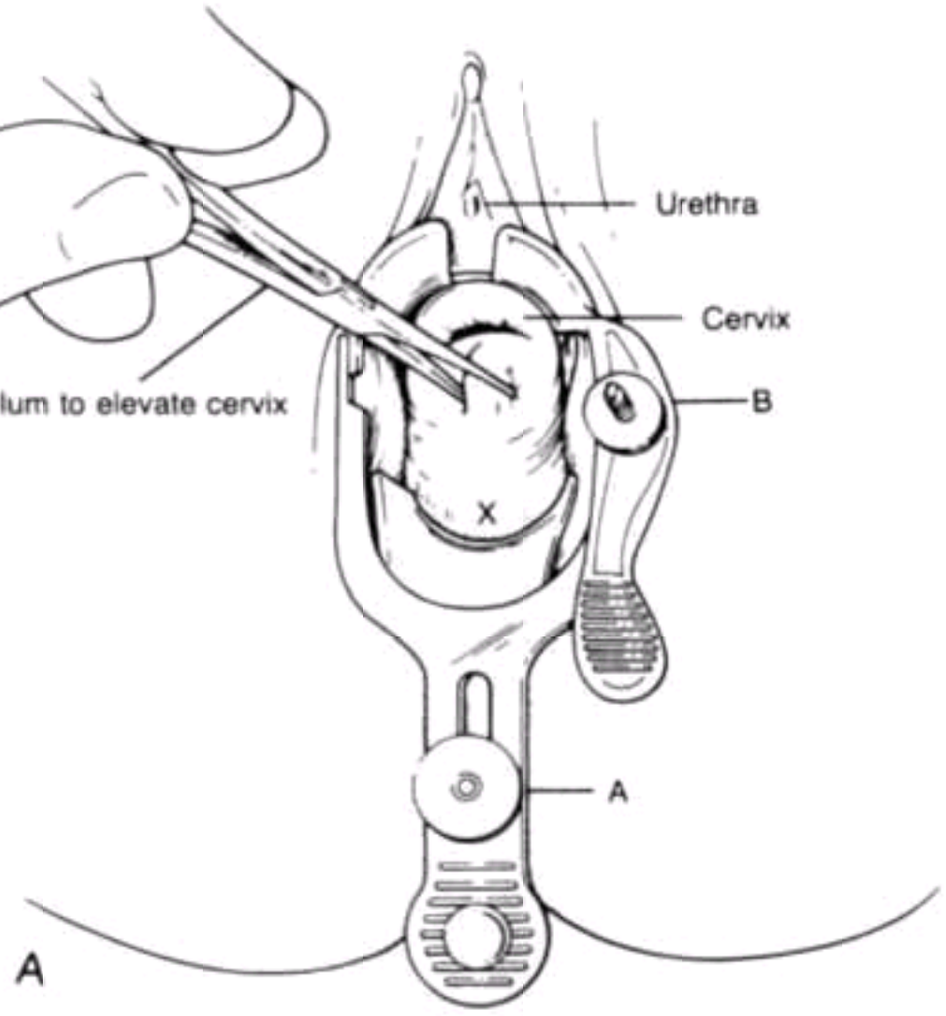


Figure 58-2 *A,* Preparation for culdocentesis. Note that one opens the speculum widely by using both the height *(A)* and the angle *(B)* adjustments. The cervix is grasped on the posterior lip with a toothed tenaculum. X marks the site for puncture of the vaginal wall. *B,* The use of a butterfly needle for culdocentesis. The needle is inserted 1 cm posterior to the point at which the vaginal wall joins the cervix. *(A, From Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. B, From Webb MJ: Culdocentesis. JACEP 7:452, 1978.)*



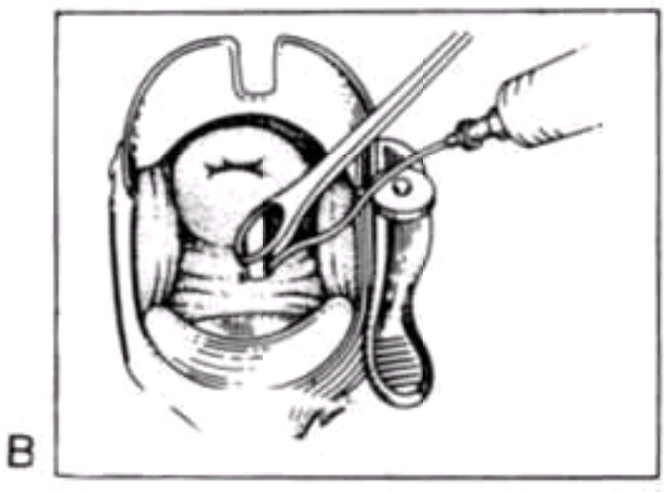
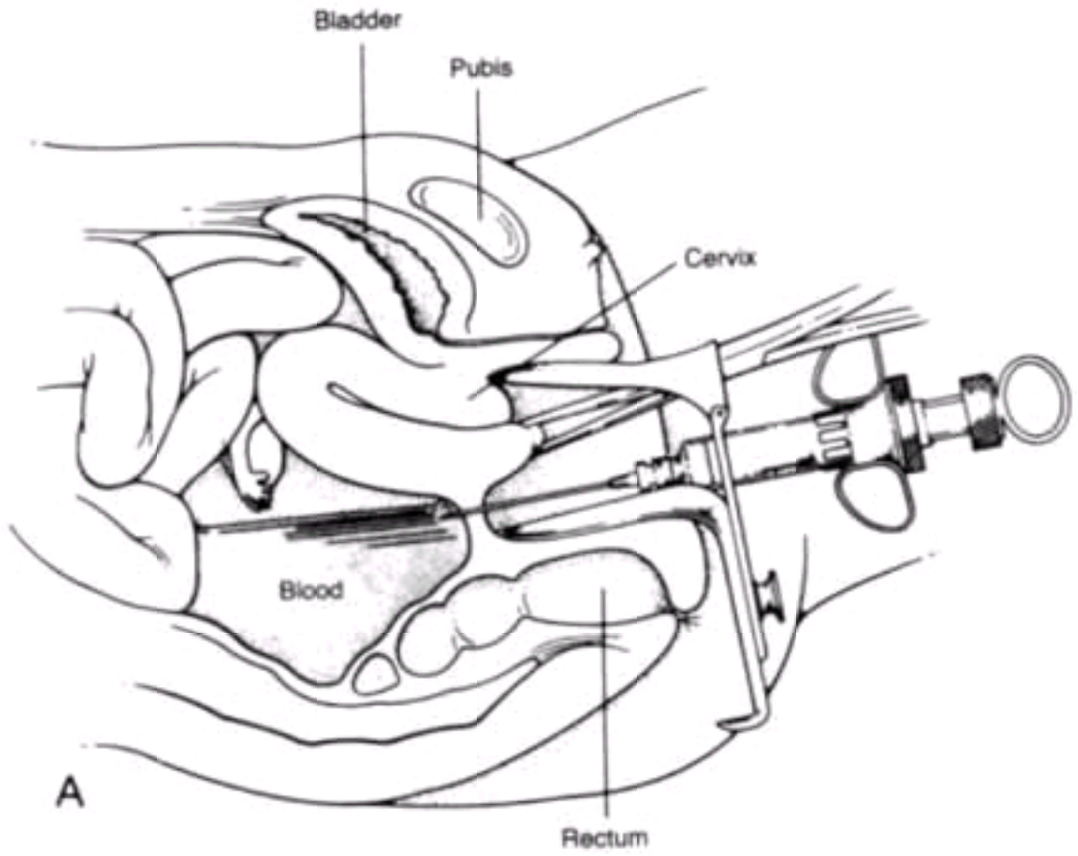


Figure 58-3 The needle is advanced parallel to the lower blade of the speculum. Aspiration is continued throughout the gradual withdrawal of the needle. *A,* The use of a spinal needle. *B,* the use of a butterfly needle and ringed forceps. *(A, from Vander Salm TJ, Cutler BS, Wheeler HB: Atlas of Bedside Procedures. Boston, Little, Brown, 1979. B, from Webb MJ: Culdocentesis. JACEP 7:452, 1978.)*



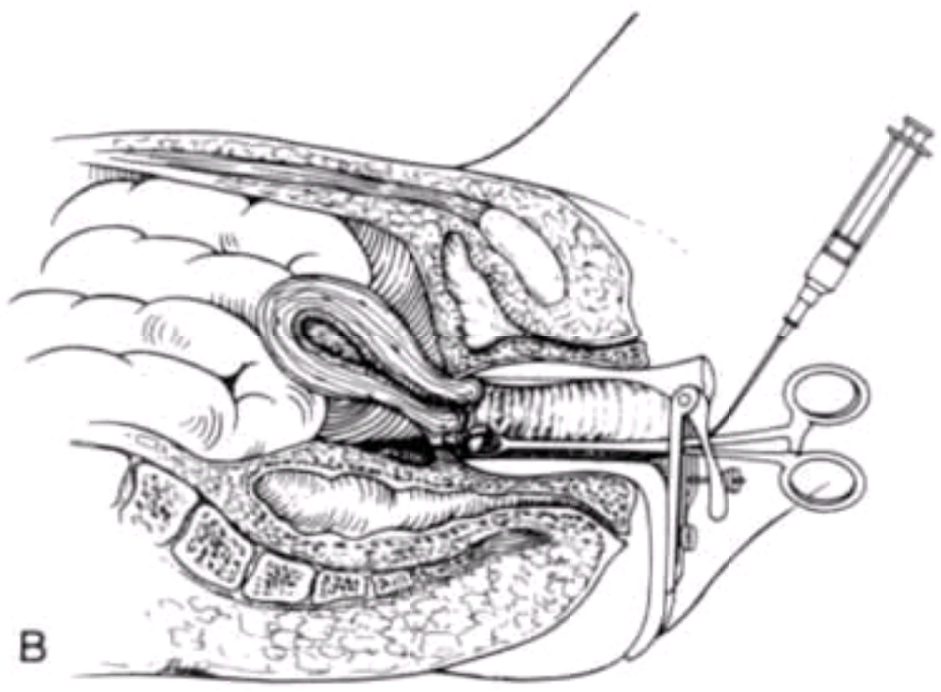


Figure 58-4 Culdocentesis may be performed with a 19-ga butterfly needle held with ring forceps. The ringed forceps grasp the wings of the butterfly needle to direct its placement. An assistant aspirates for fluid through the proximal end of the tubing. *(From Webb MJ: Culdocentesis. JACEP 7:451, 1978.)*

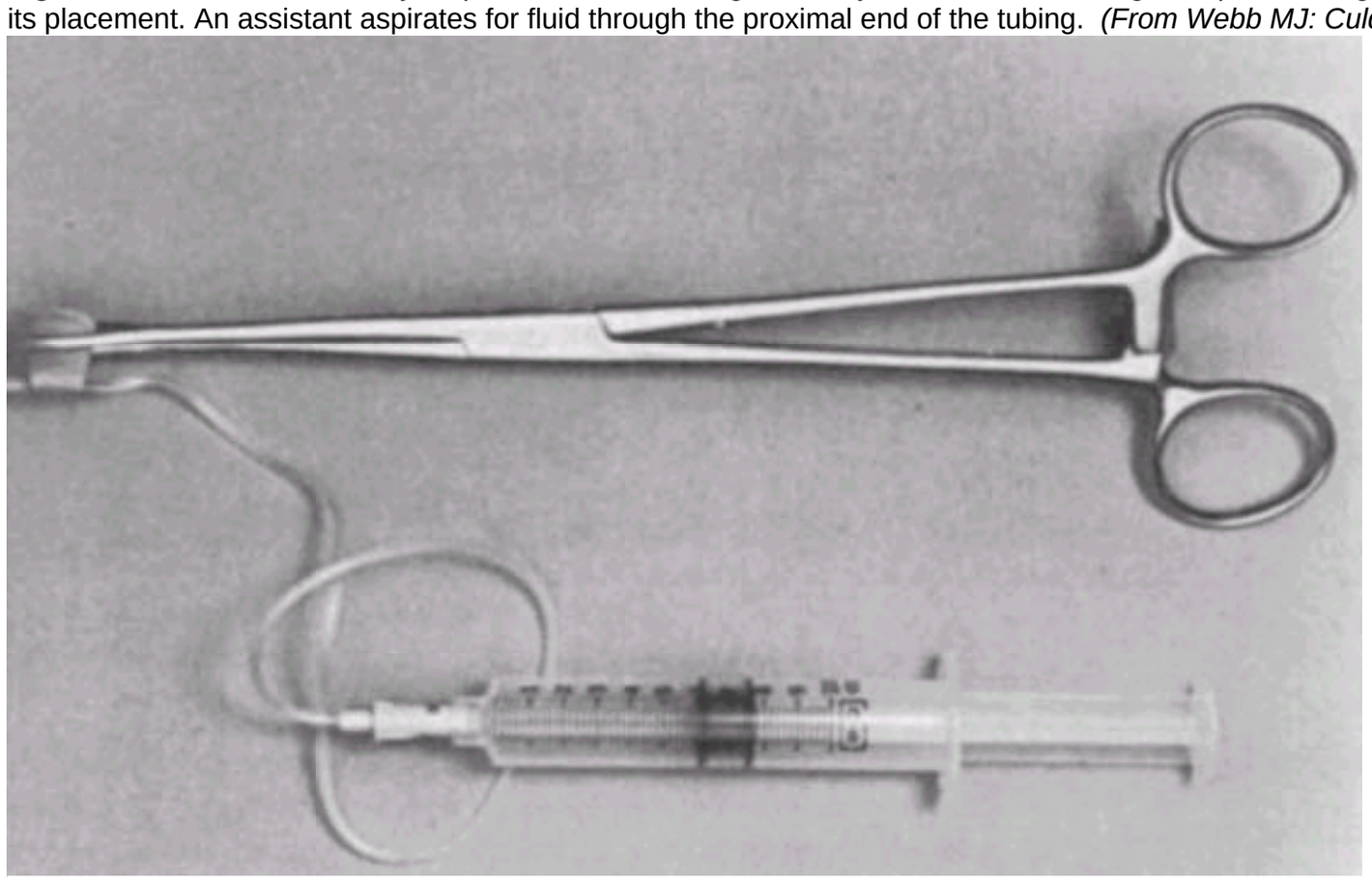


Figure 59-1 Sexual assault examination kit used in Los Angeles County.

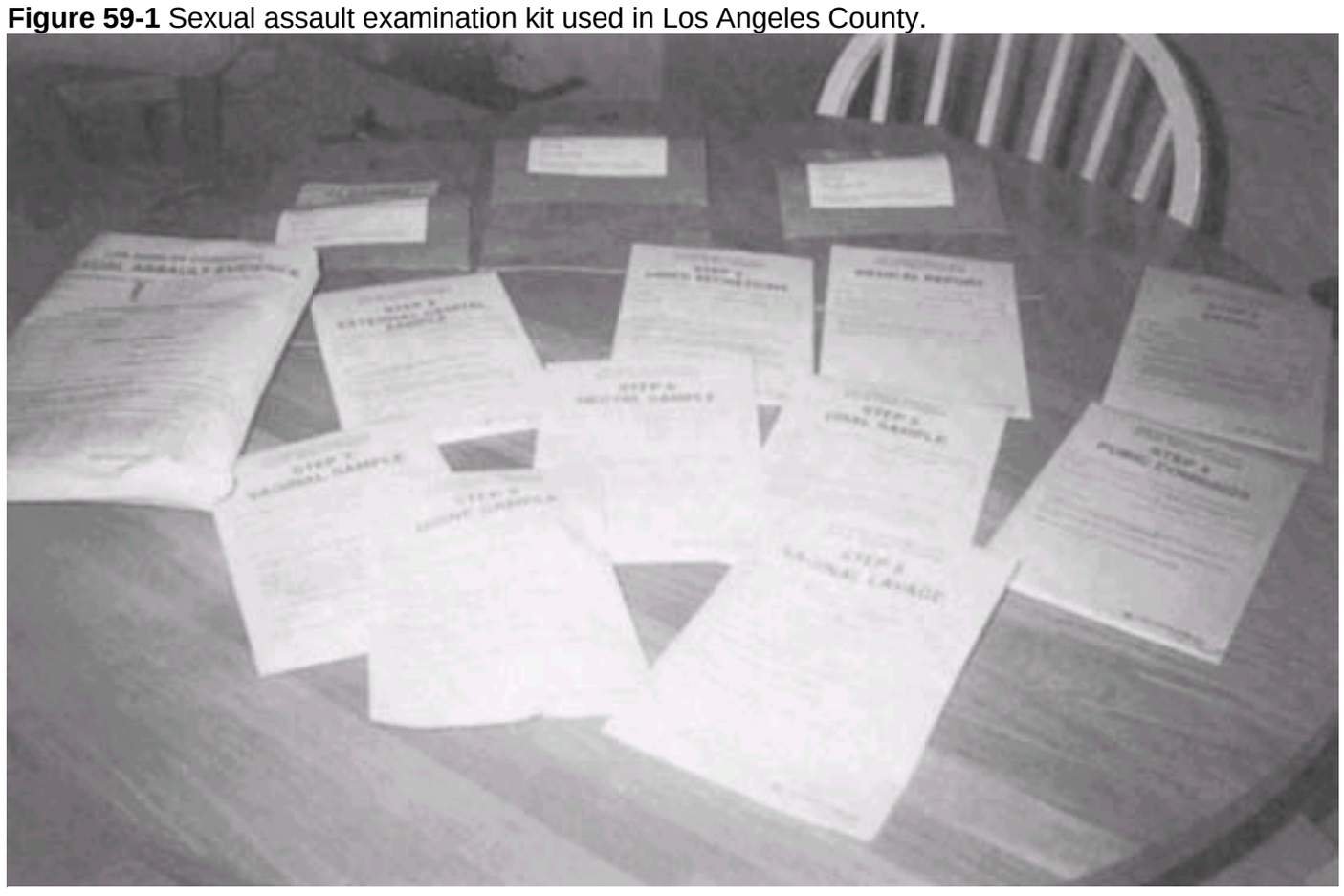


Figure 59-2 Alternative light source.

Figure 59-3 Female anatomy. *(From Girardin BW, Faugno DK, Seneski PC, et al: Color Atlas of Sexual Assault. St. Louis, Mosby-Year Book, 1997. Originally from Seidel HM, et al: Mosby's Guide to Physical Examination, 3rd ed. St. Louis, Mosby, 1995.)*

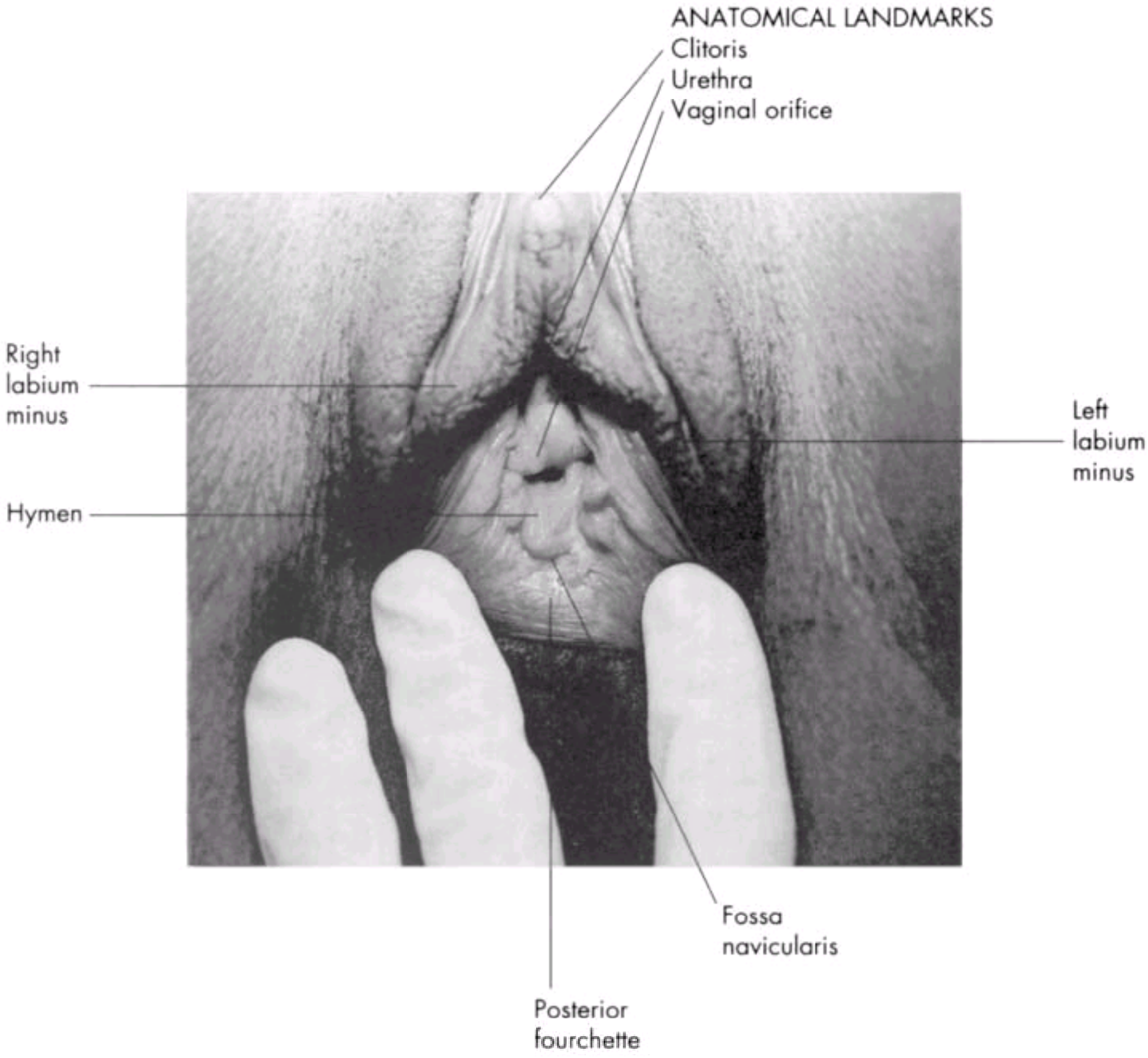


Figure 59-4 Male anatomy. *(From Girardin, et al: Color Atlas of Sexual Assault. St. Louis, Mosby-Year Book, 1997.)*

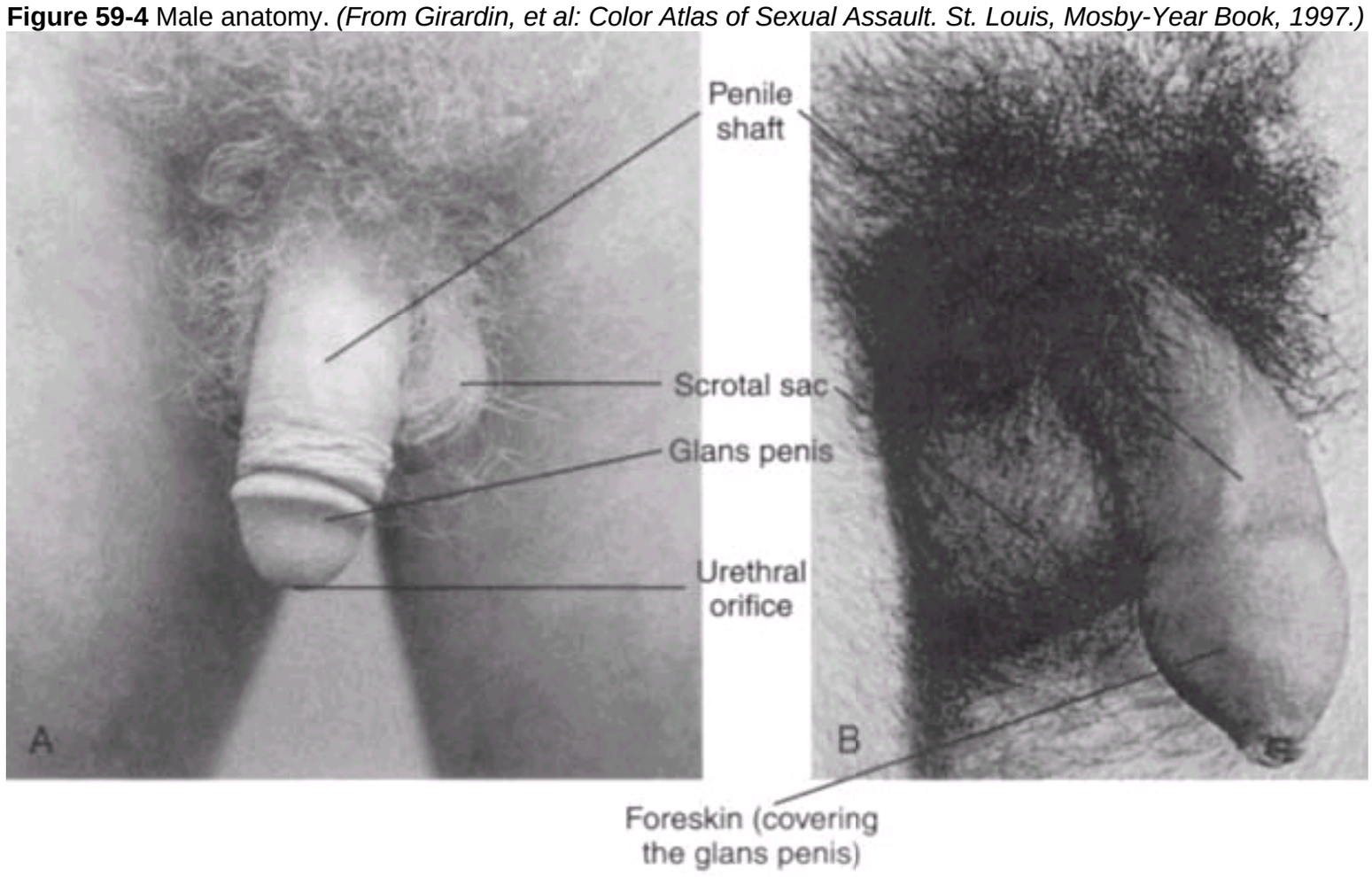


Figure 59-5 Video colposcope with real time visualization on monitor shown. Image is eye of teddy bear at 15× magnification.

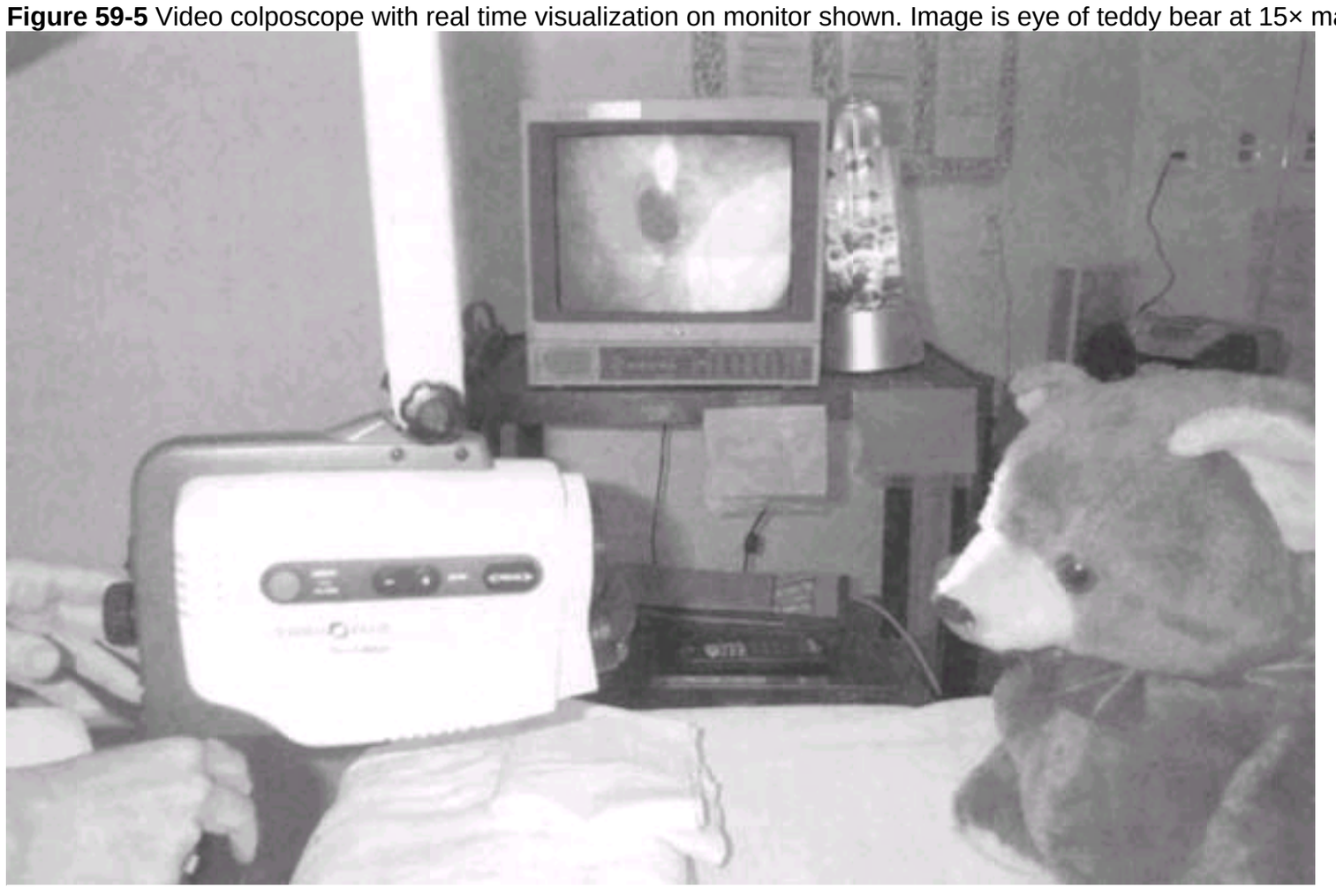


Figure 59-6 Hymenal injury at the 6-o'clock position in adolescent female.

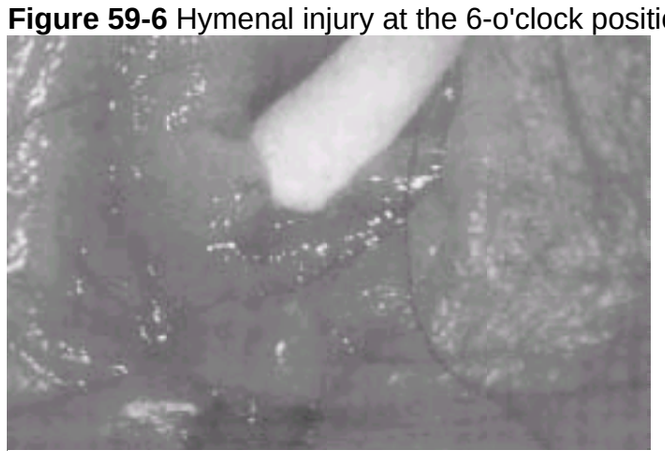


Figure 59-7 Device used to hold swabs and slides to speed drying.

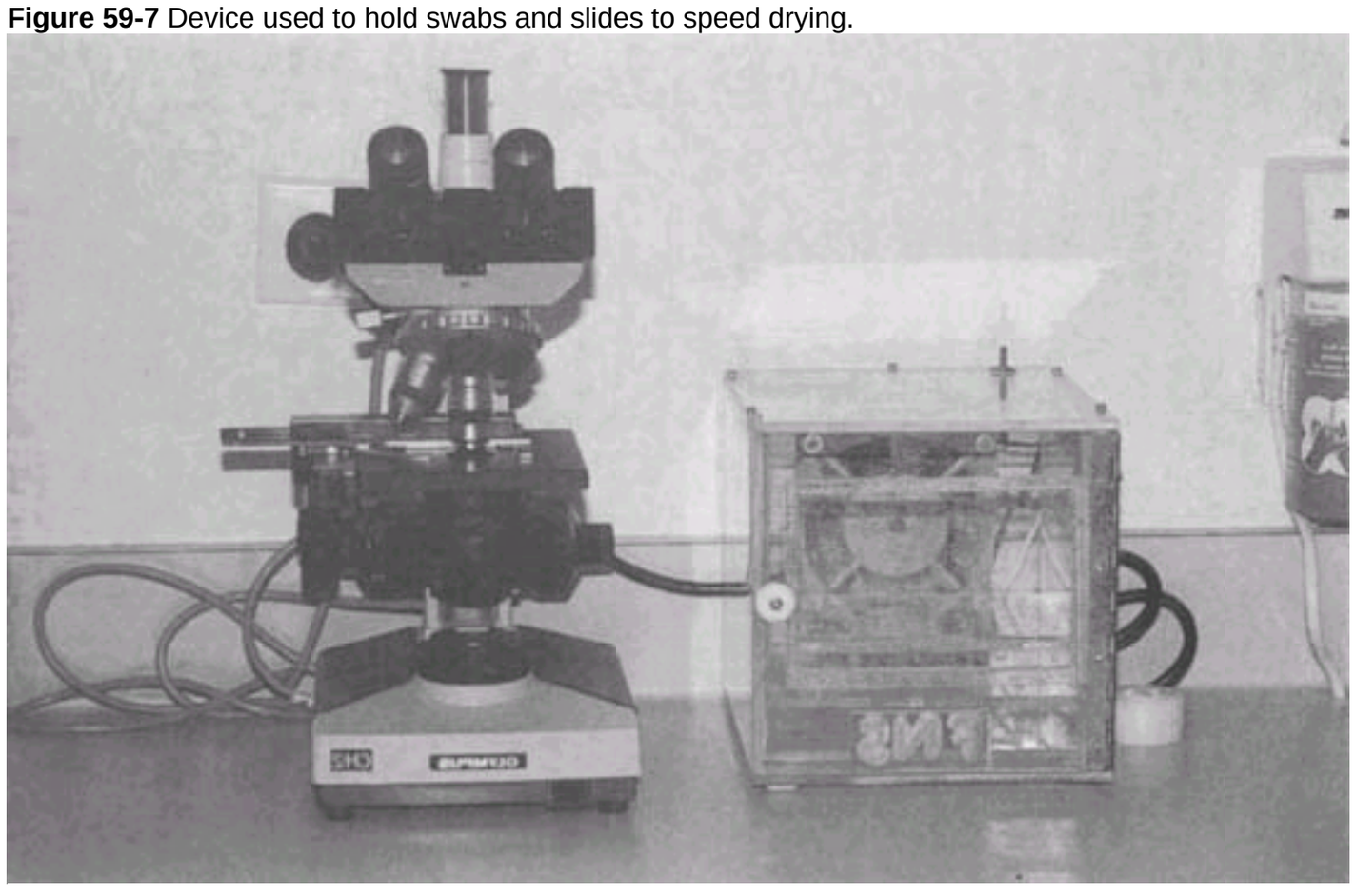


Figure 59-8 Posterior fourchette injuries. *A,* Viewed through the colposcope without the use of toluidine blue dye. *B,* Same posterior fourchette injuries outlined with toluidine blue dye.

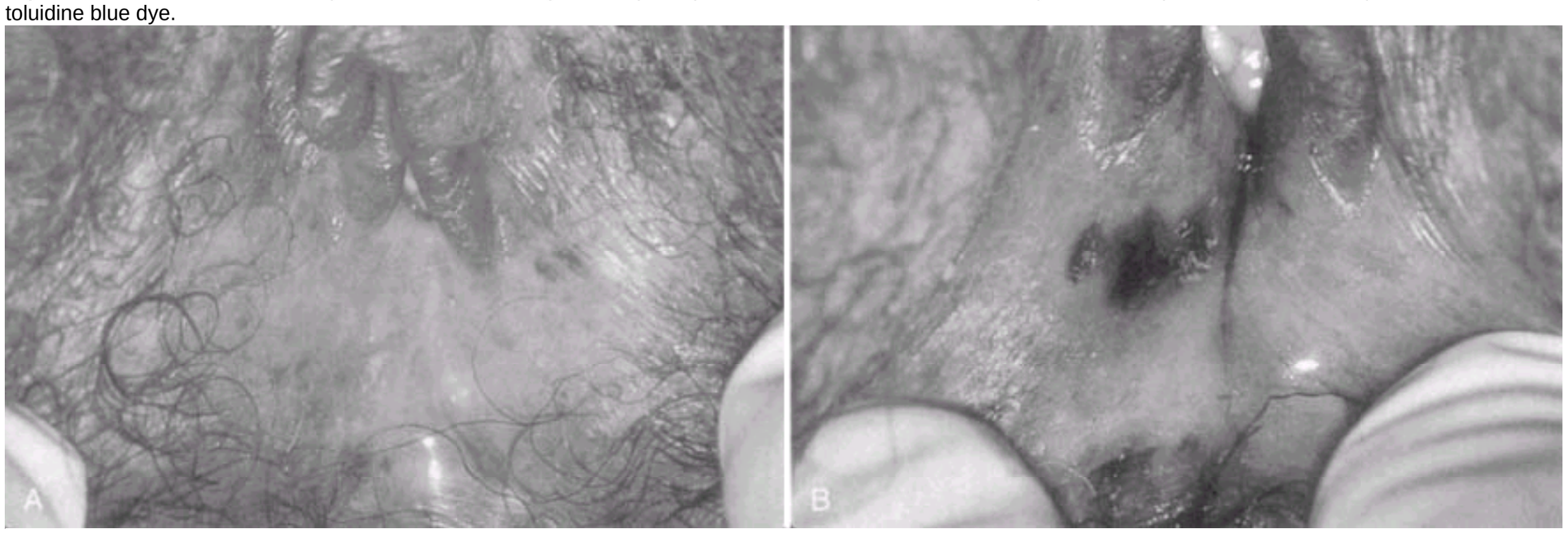


Figure 59-9 Toluidine blue application procedure. *(From McCauley J, Guzinski G, Welch R, et al: Toluidine blue in the corroboration of rape in the adult victim. Am J Emerg Med 5:106, 1987.)*

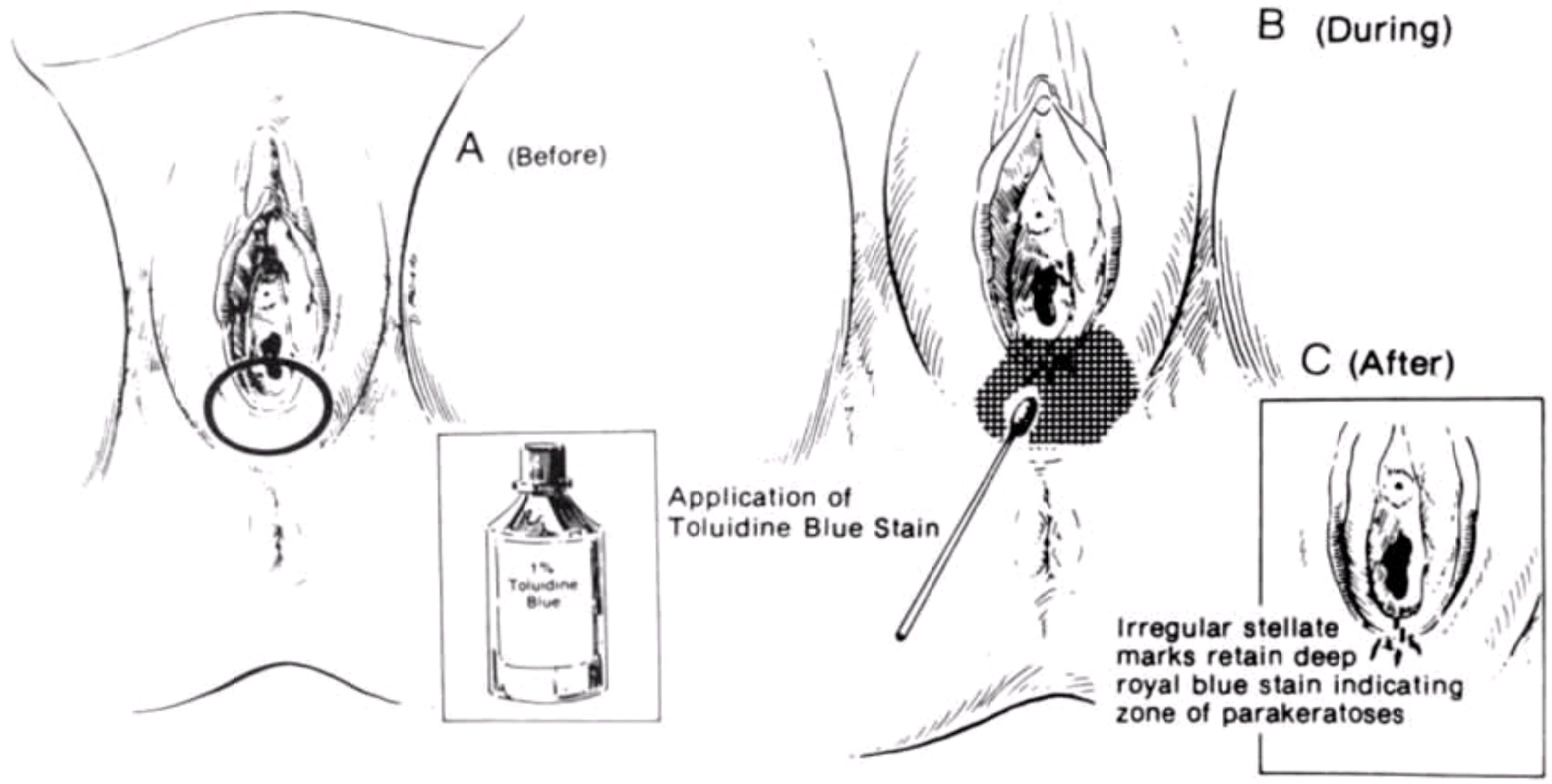


Figure 59-10 Anal injury is best seen with separation of perianal tissues. *A,* Without toluidine blue dye. *B,* Visualized after toluidine blue dye.

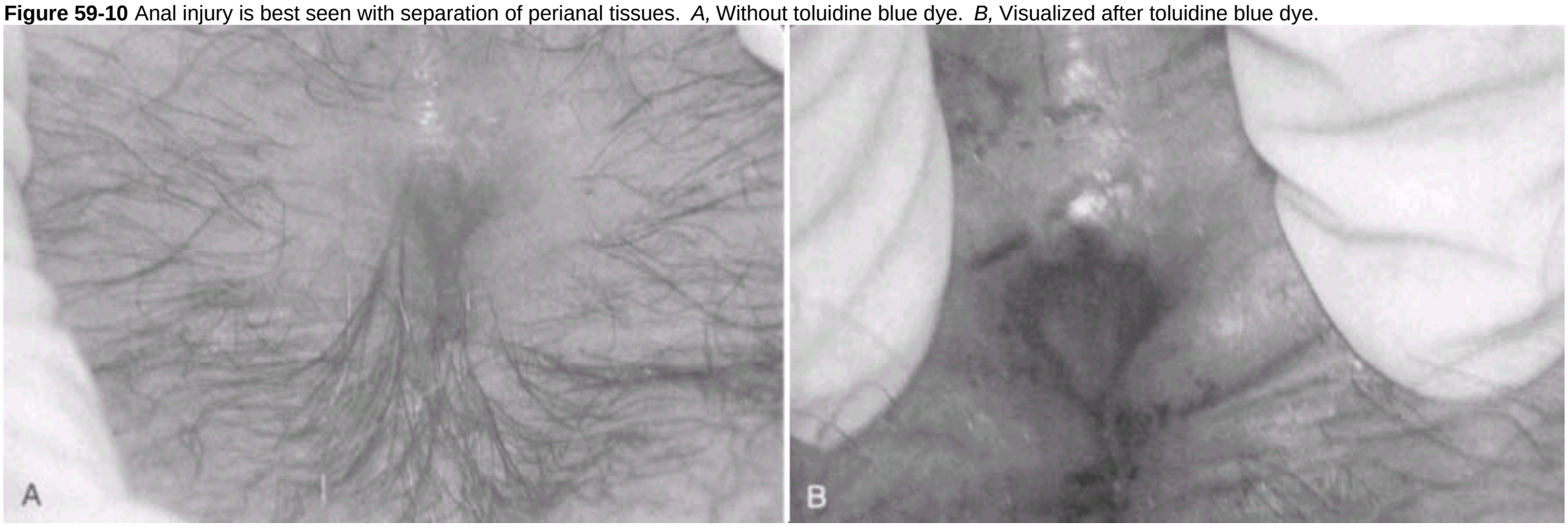


Figure 59-11 Rectal injury visualized with anoscopy.

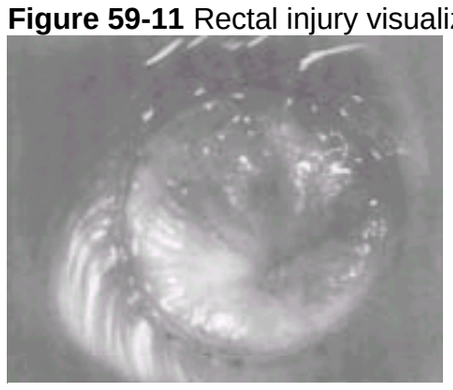


Figure 59-12 Recommended "frog leg" position to examine children.

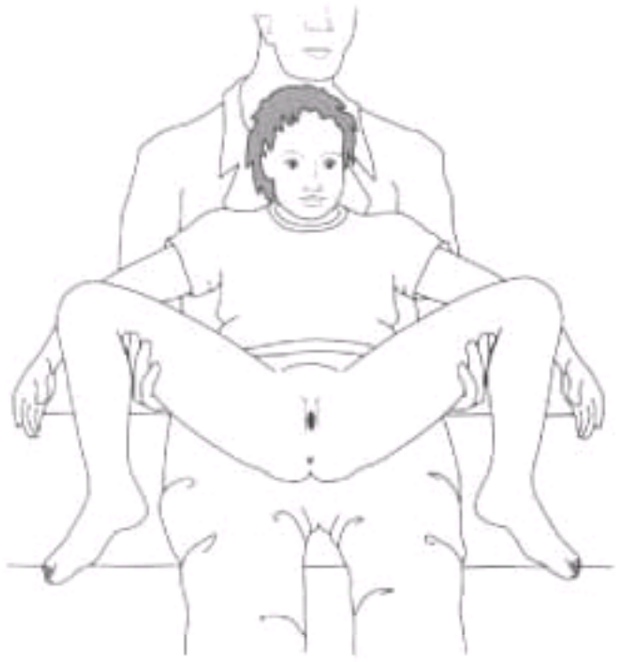


Figure 60-1 Schematic presentation of the various adverse effects associated with radiation and their relative incidence at different stages of gestation. *(Adapted from Mettler FA Jr, Upton A: Medical Effects of Ionizing Radiation, 2nd ed, Chapter 8, WB Saunders, 1995.)*

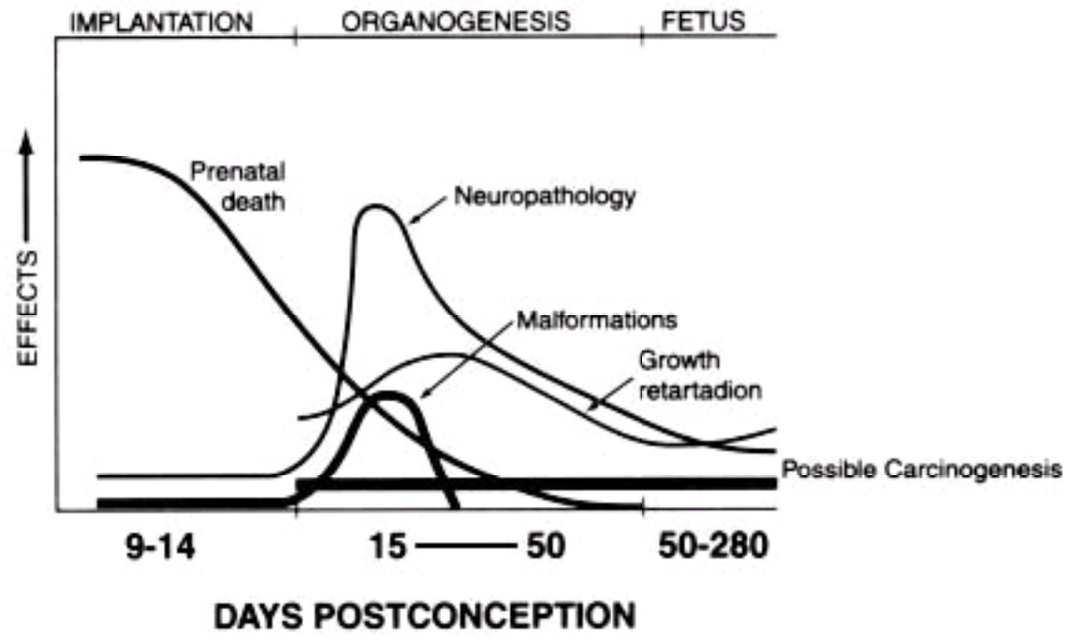


Figure 60-2 *Graph A*: Comparison of common radiographic studies with the accepted 5 rad cumulative fetal exposure limit. *Graph B:* Low fetal exposure studies equated to equivalent dose from background terrestrial radiation. *Graph C*: Fetal exposure level compared to the Nuclear Regulatory Commission's accepted fetal dose during pregnancy.

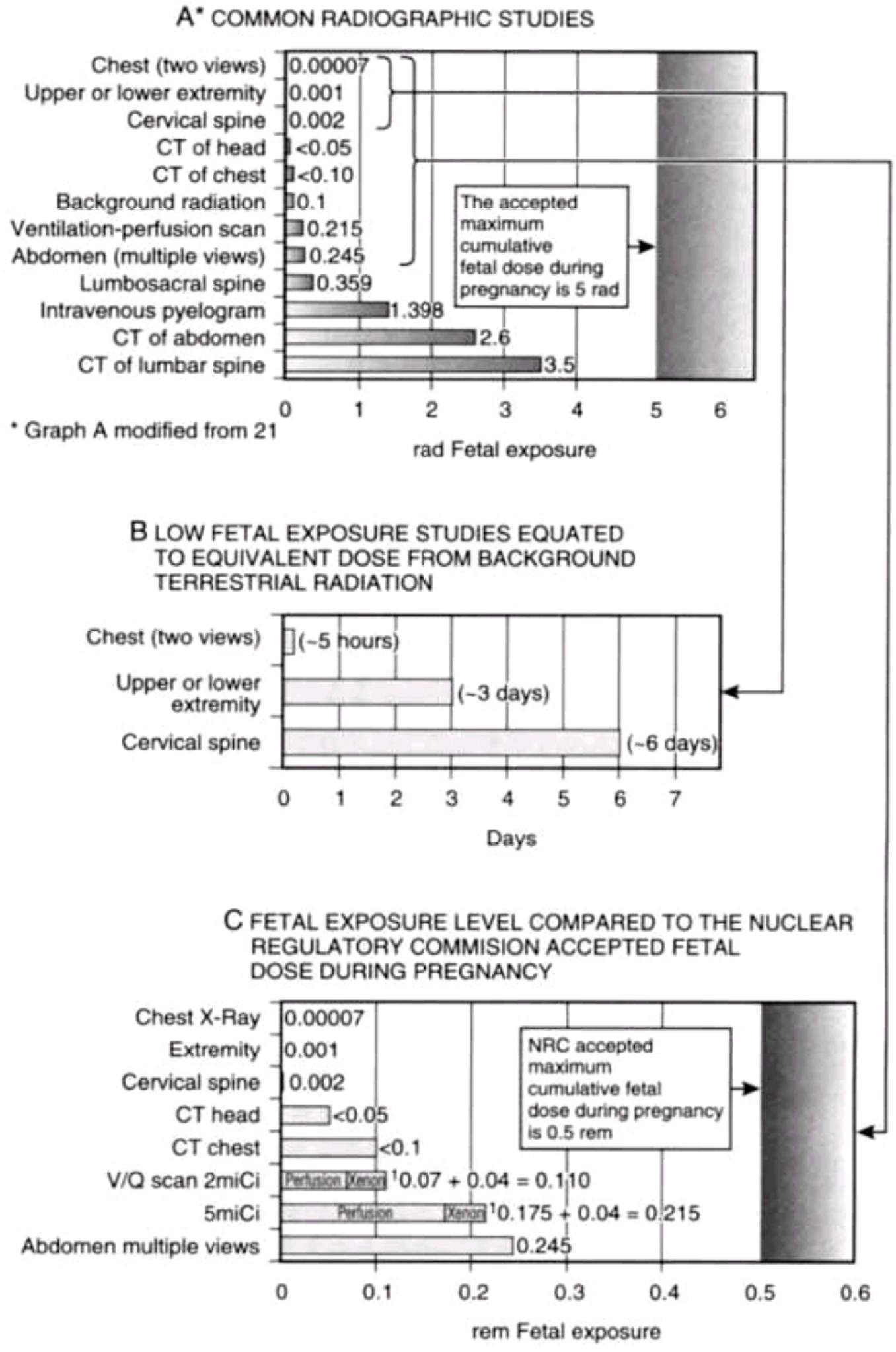


Figure 61-1 Intracranial contents and their volumes for healthy adults.

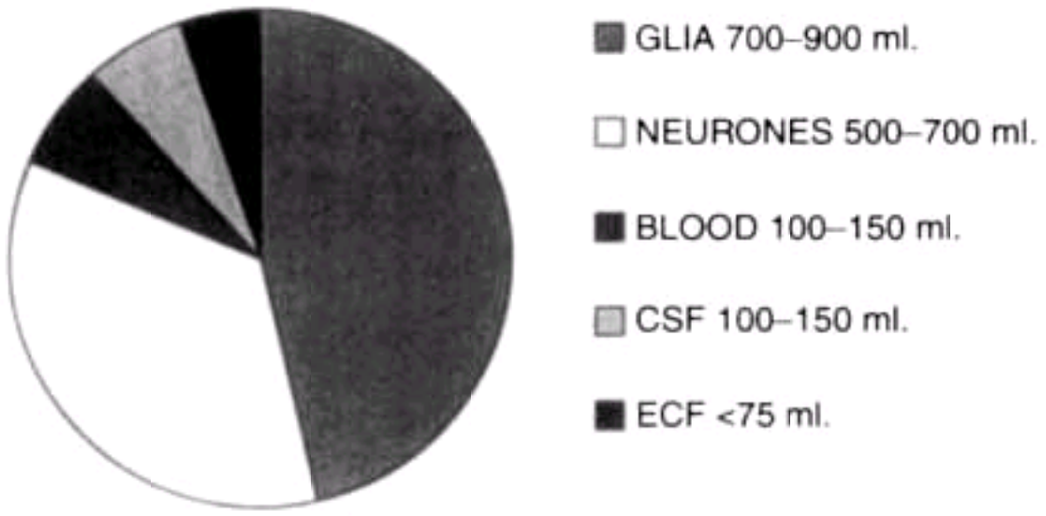


Figure 61-2 Intracranial volume-pressure relationship, demonstrating limits of compensatory mechanisms.

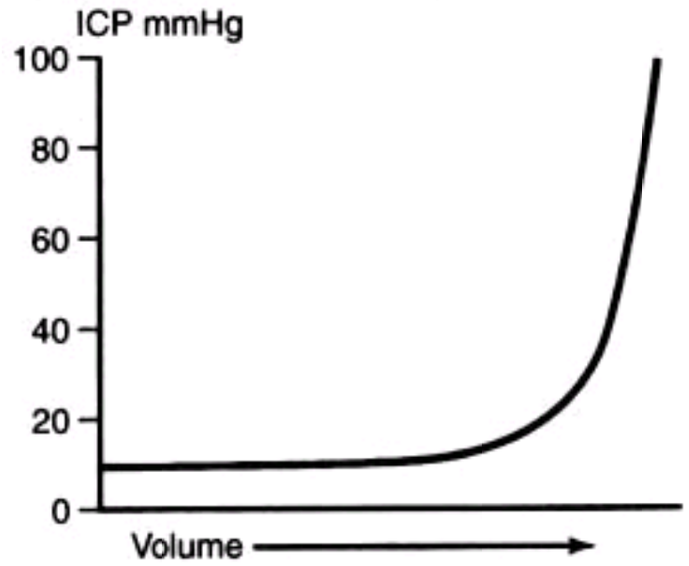


Figure 61-3 Intracranial shifts from supratentorial lesions. *A,* Relationships of the various supratentorial compartments as seen in a coronal section. *B,* Herniation of the cingulate gyrus under the falx (*1*); herniation of the temporal lobe into the tentorial notch (*2*); compression of the opposite cerebral peduncle against the unyielding tentorium, producing Kernohan's notch (*3*); and downward displacement of the brainstem through the tentorial notch (*4*). *(From Plum F, Posner JB: The Diagnosis of Stupor and Coma, 2nd ed. Philadelphia, FA Davis, 1972. Reproduced by permission.)*

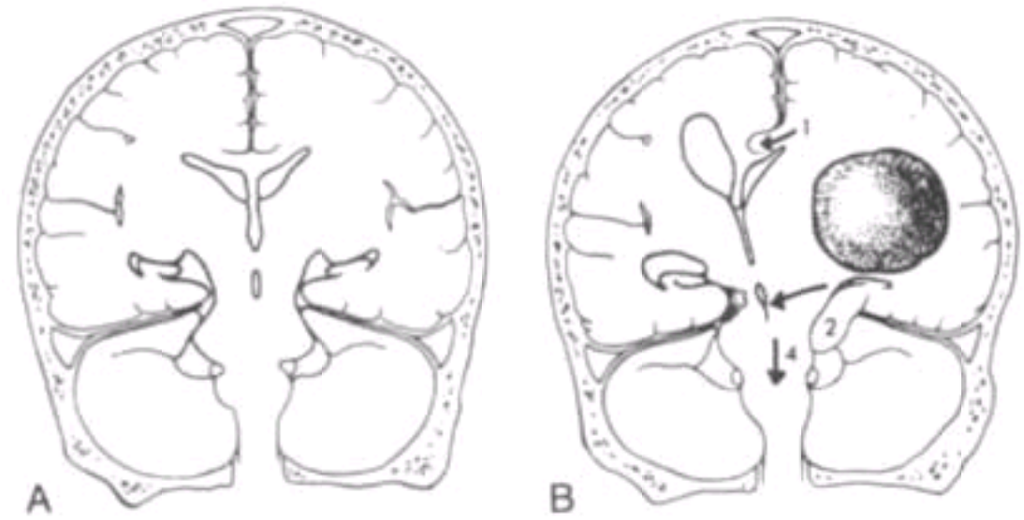


Figure 61-4 Cerebrospinal fluid production and flow *(From Rengachary and Wilkins: Principles of Neurosurgery. Philadelphia, Mosby, 1994).*

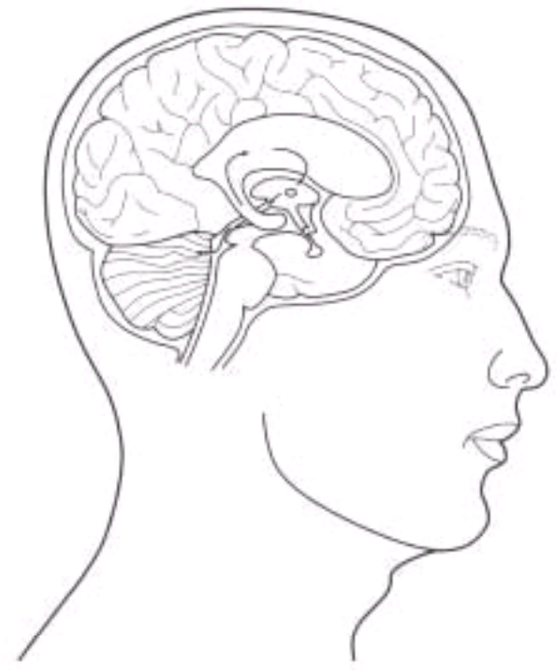
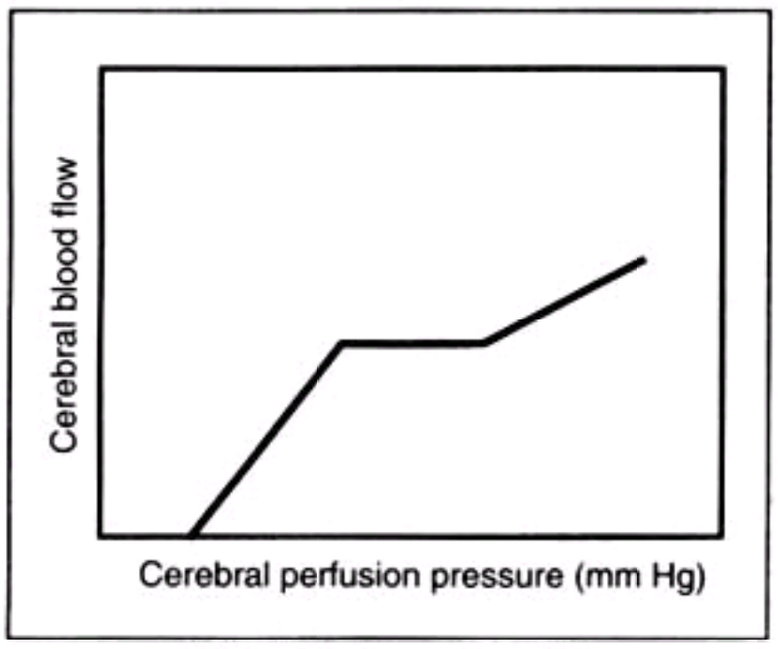


Figure 61-5 Cerebral autoregulation.



CPP= Blood pressure–ICP

Figure 61-7 Shunt components *(From Rengachary and Wilkins: Principles of Neurosurgery. Philadelphia, Mosby, 1994).*

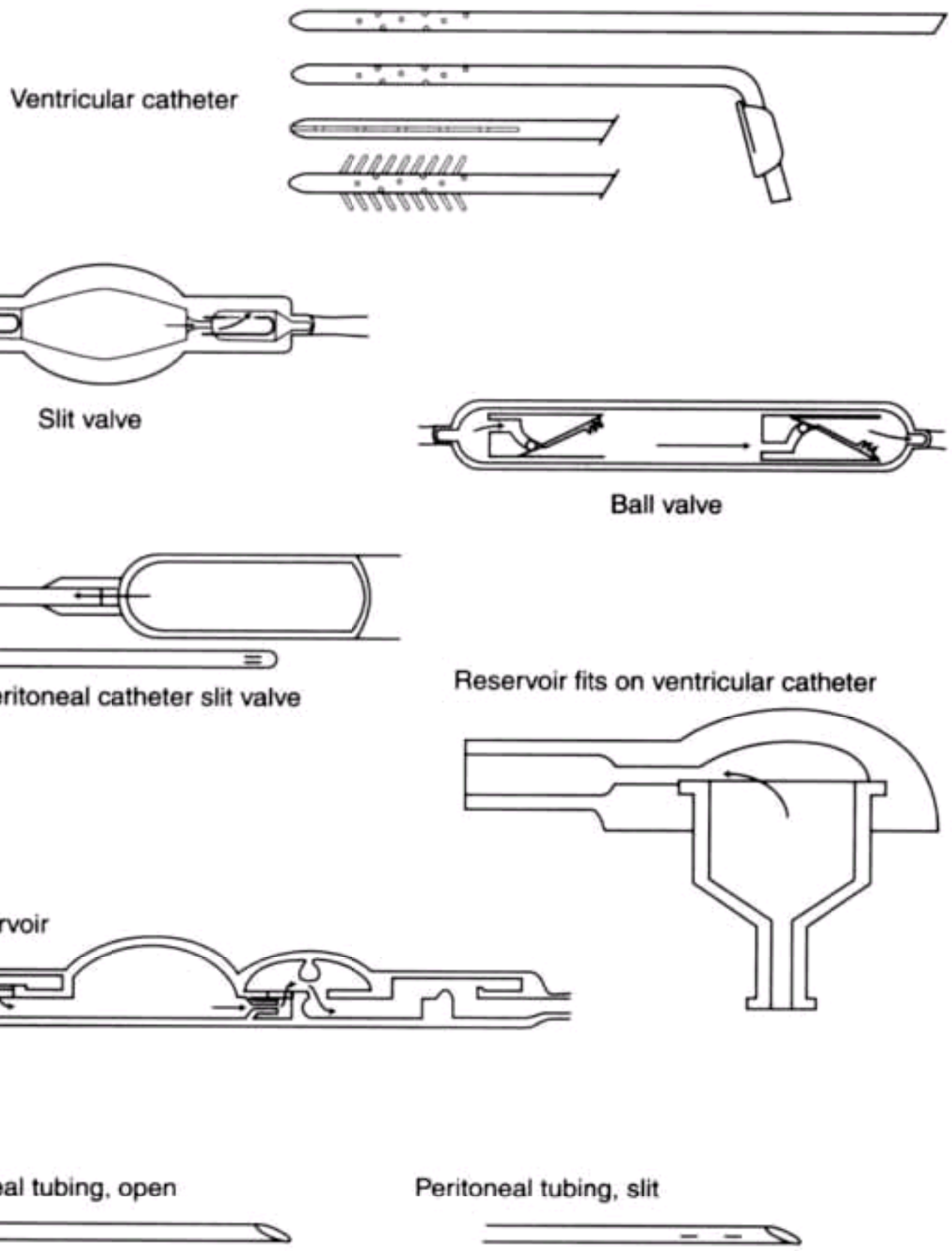


Figure 61-8 Cross section of a Pudenz flushing valve (American Heyer-Schulte, Santa Barbara, CA) illustrating the diaphragm valve. The proximal inlet tube and silicone base are placed in the burr hole so that only the reservoir (silicon dome) protrudes above the skull *(Courtesy of PS Medical, Goleta, CA).*

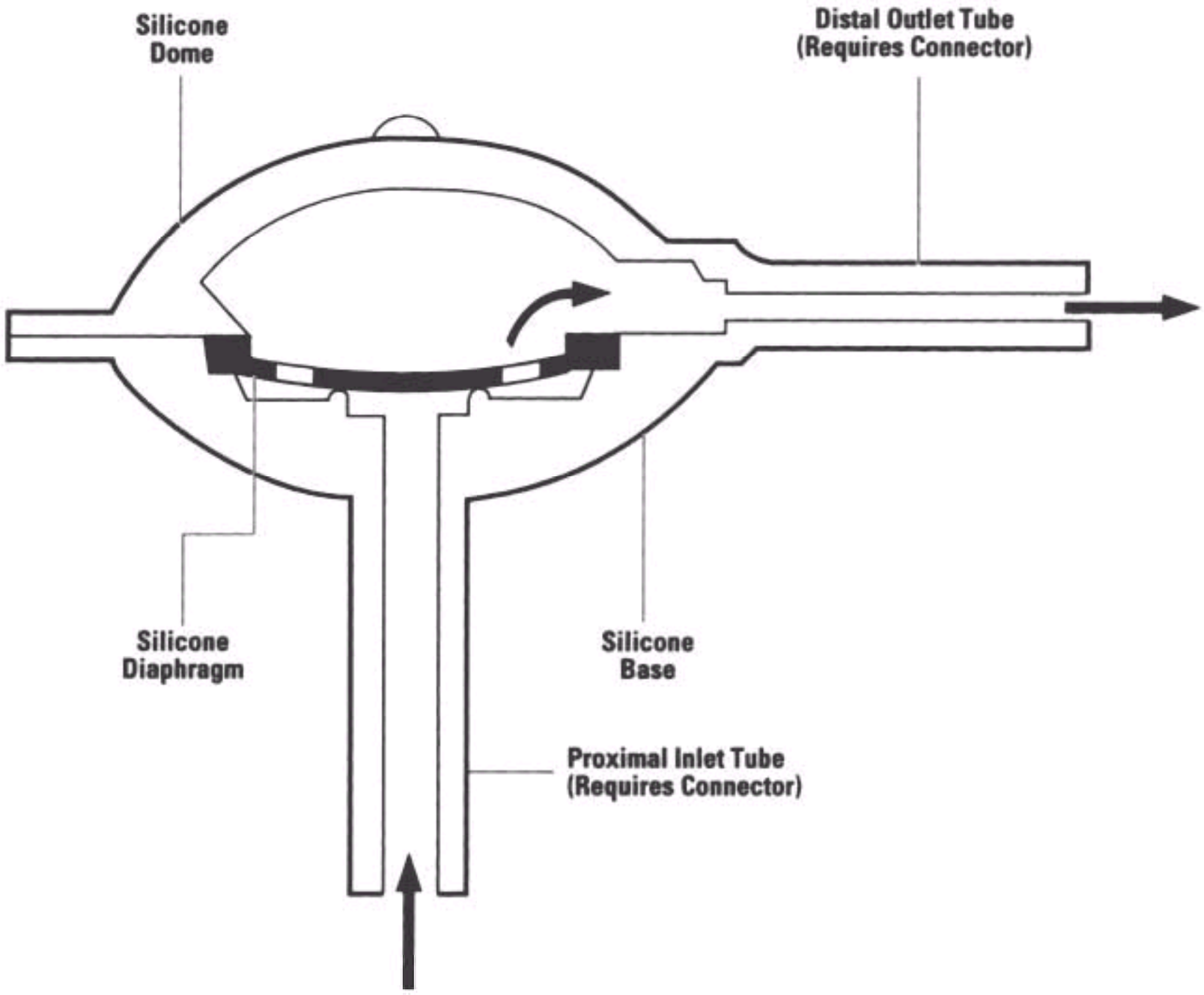


Figure 61-9 Ventriculoperitoneal shunt and alternative occipital placement *(From Rengachary and Wilkins: Principles of Neurosurgery. Philadelphia, Mosby, 1994, Fig. 7-6).*

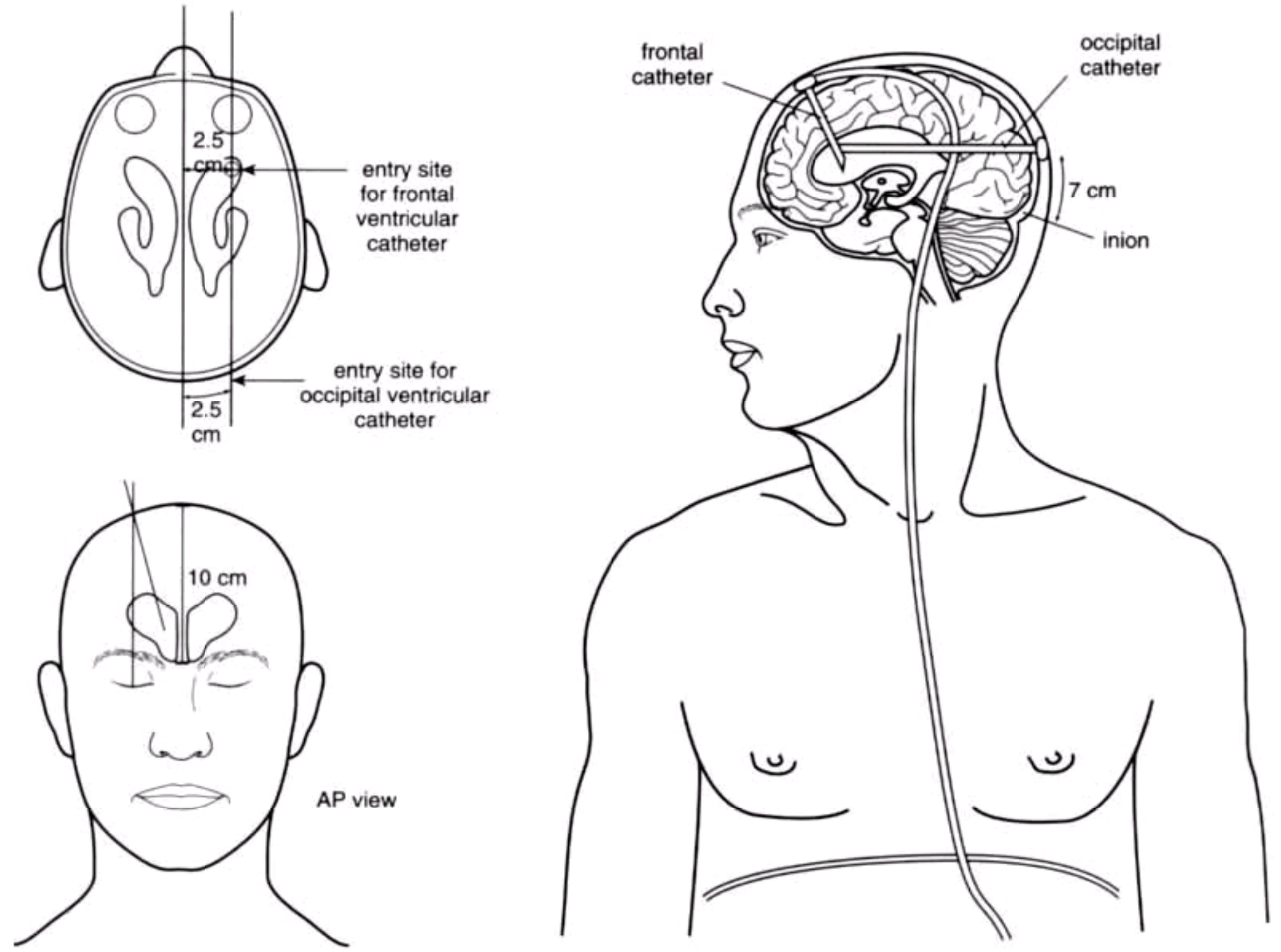


Figure 61-10 A standard shunt series includes an anteroposterior (AP) skull, lateral skull, and chest radiograph and kidney, ureters, bladder (KUB) series. This intact shunt is a Rickham reservoir over the burr hole, with a cylindrical Holter valve a few centimeters distal. This AP skull radiograph reveals the proximal intracranial portion of the shunt and a Rickham reservoir over the burr hole.

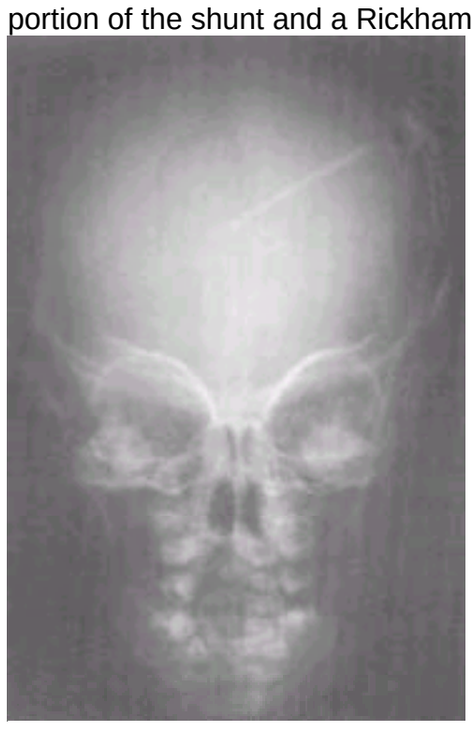


Figure 61-11 Lateral skull radiograph of patient in Figure 61-10 . This view better reveals a cylindrical Holter valve situated a few centimeters distal to the Rickham reservoir.

Figure 61-12 Anteroposterior chest and abdominal radiograph of patient in Figure 61-10 . This radiograph reveals the distal portion of the shunt. Note the length of the catheter to allow for patient growth.

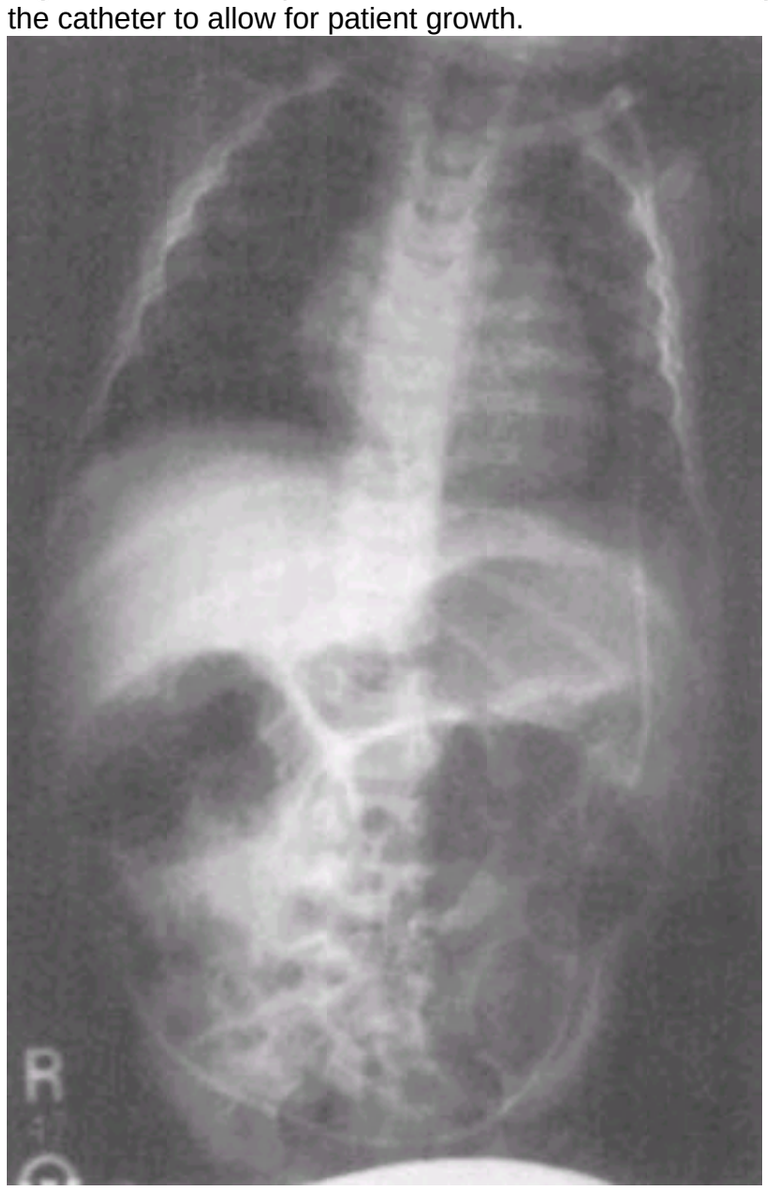


Figure 61-13 Lateral chest and radiograph of the patient in Figure 61-10 .

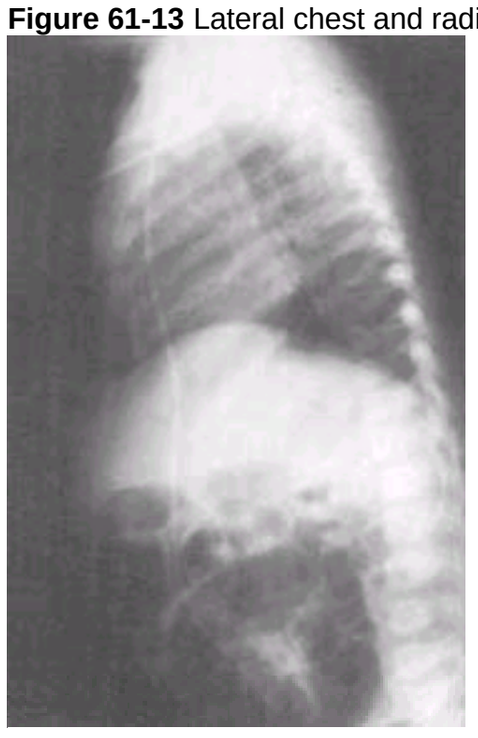


Figure 61-14 Connector discontinuity of a ventricular peritoneal shunt, at the level of the clavicle, is illustrated in this chest radiograph from a shunt series.

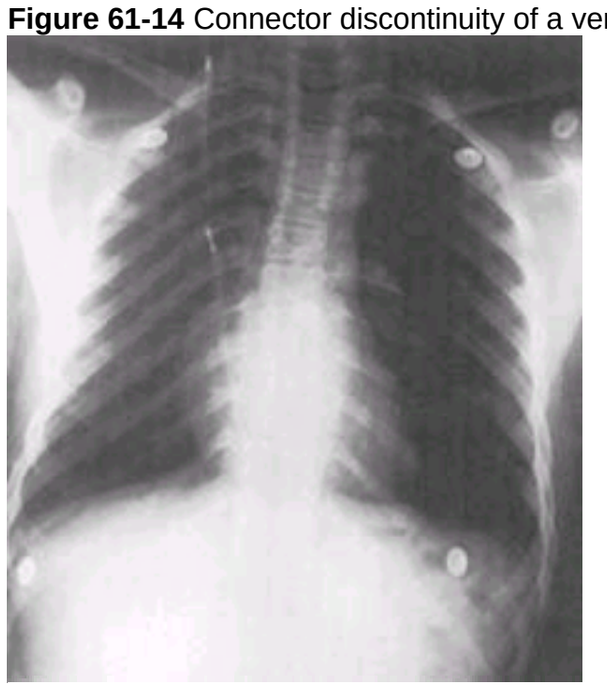


Figure 61-15 Normal lateral skull radiograph demonstrating an intact ventricular peritoneal shunt in a young patient with a Holter valve. This shunt later became disconnected (see Fig. 61-17 and Fig. 61-18).

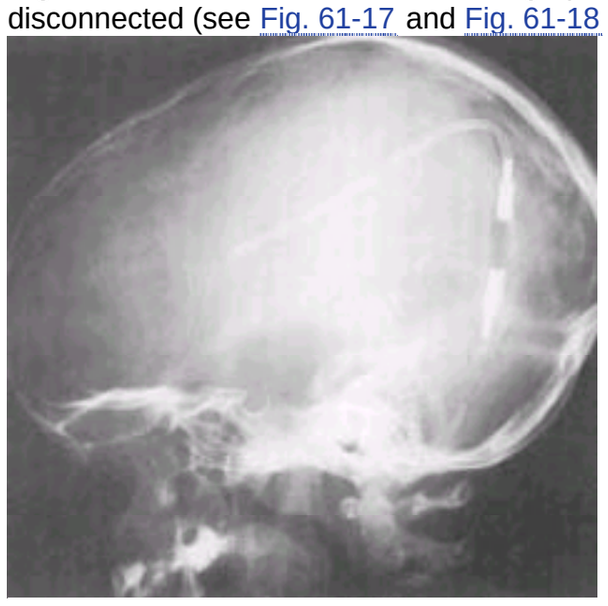


Figure 61-16 Normal anteroposterior chest and abdominal radiograph demonstrating an intact ventricular peritoneal shunt in the patient in Figure 61-15 .

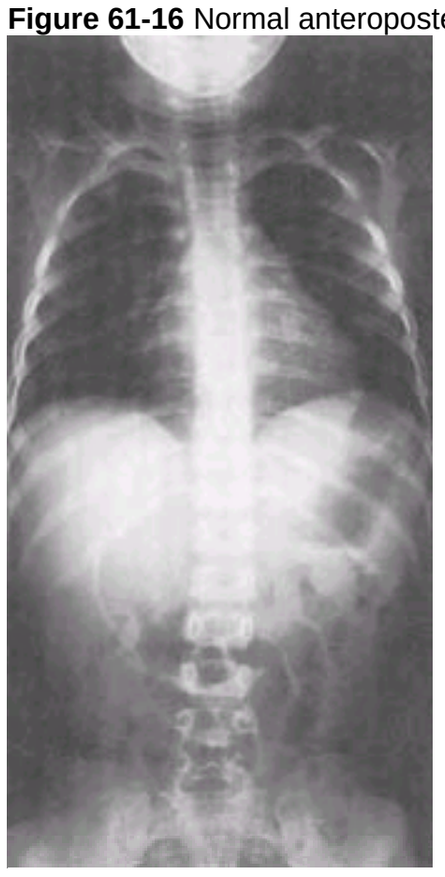


Figure 61-17 Repeat films from the patient in Figure 61-15 and Figure 61-16 illustrating shunt disconnection at the level of the valve and subsequent migration of the distal catheter into the pelvis. This lateral skull radiograph demonstrates the disconnection from the valve.

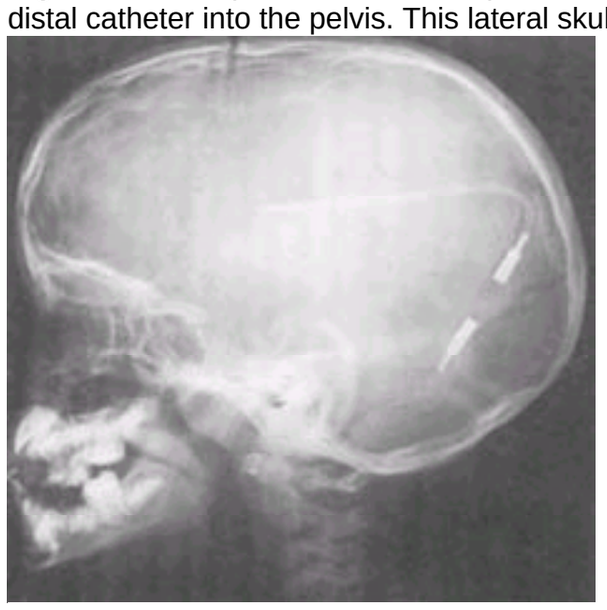


Figure 61-18 Abdominal radiograph of patient in Figure 61-17 demonstrating migration of the catheter into the pelvis.

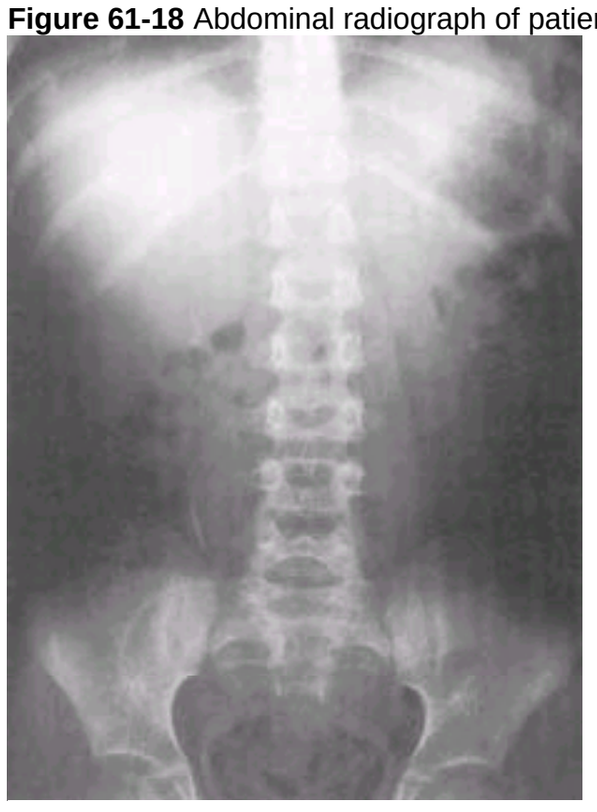


Figure 61-19 Cranial computed tomography scan of the patient with shunt disconnection shown in Figure 61-17 . This CT scan at presentation reveals ventriculomegaly. After revision, the ventricles returned to normal size (see Fig. 61-20).

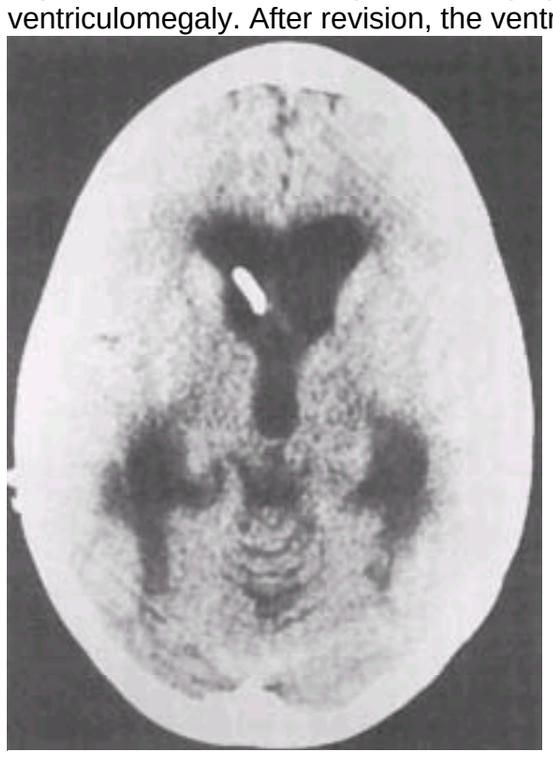


Figure 61-20 Cranial computed tomography scan of patient in Figure 61-19 after shunt revision. The ventricles have returned to normal size.

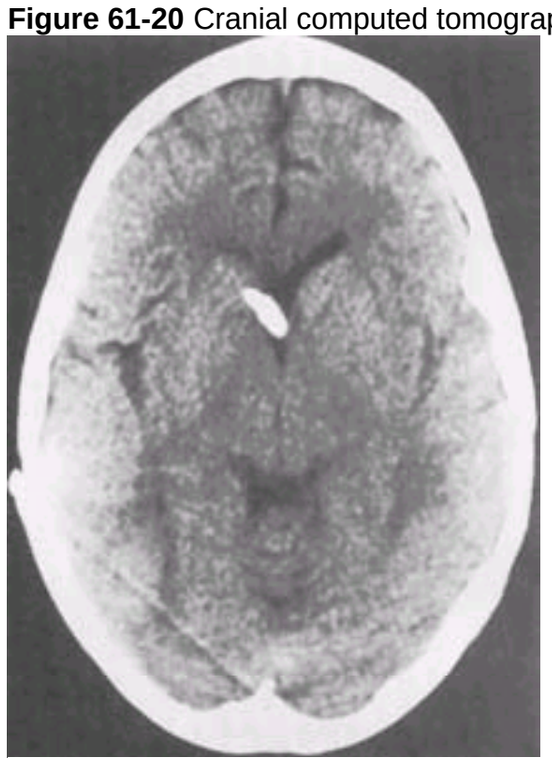


Figure 61-21 A 25-ga butterfly needle puncture of a reservoir. To avoid damage to the reservoir, the angle should be approximately 20° to 30°. Note that the dome reservoir is under the skin. Prior to passage of the butterfly needle, the skin is anesthetized, sterilized with povidone-iodine, and nicked with a number 11 scalpal or larger needle. Fluid is not aspirated but is allowed to drain passively.

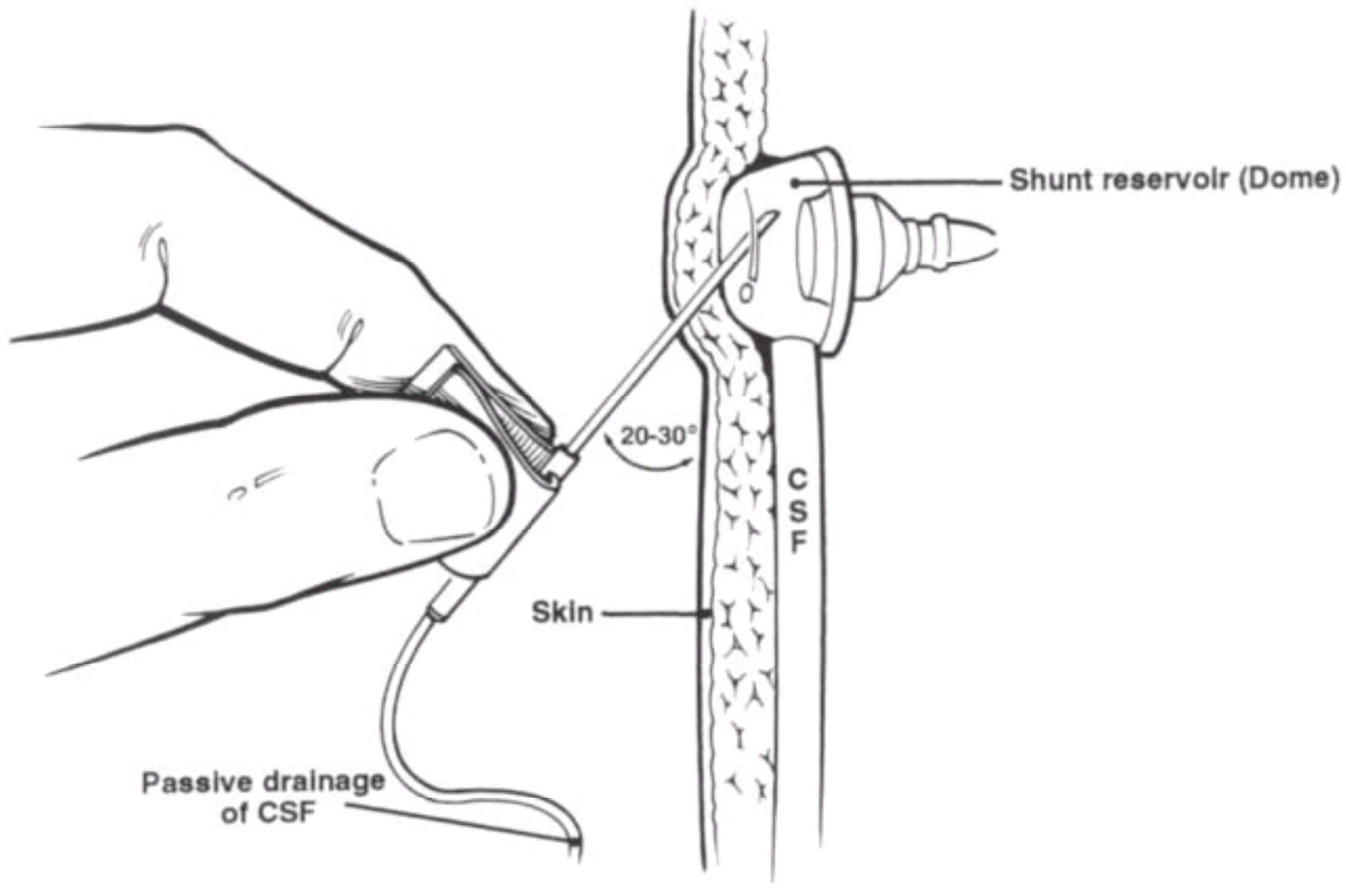


Figure 62-1 Standard lumbar puncture tray. Separate equipment is available for infants, children, and adults. *(Image courtesy of the American Pharmaseal Company, Glendale, CA.)*

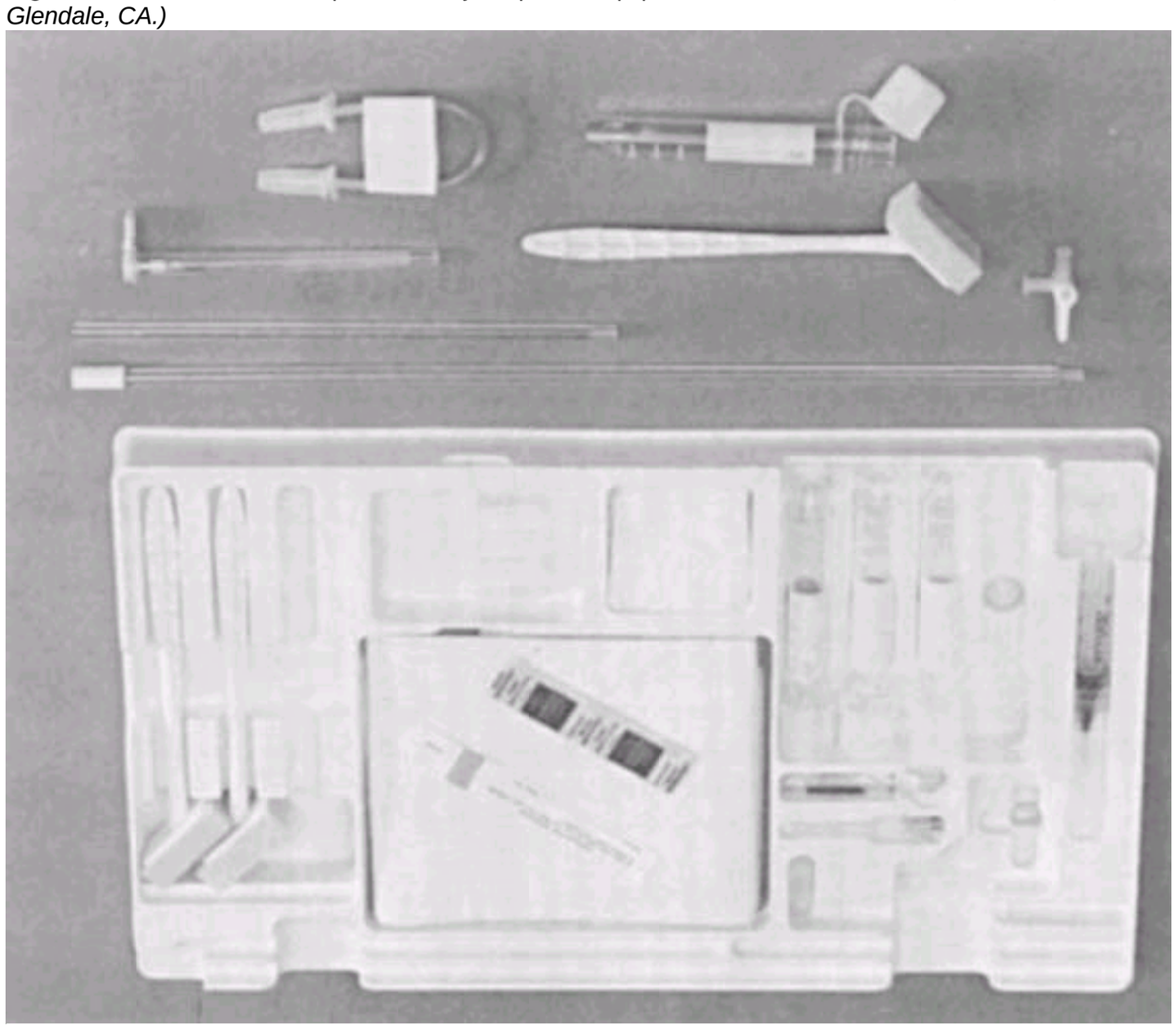


Figure 62-4 The spinal needle is usually advanced ½ to ¾ of its length before the spinal canal is reached. In this obese patient the needle was advanced all the way to the hub of the needle before spinal fluid was returned.

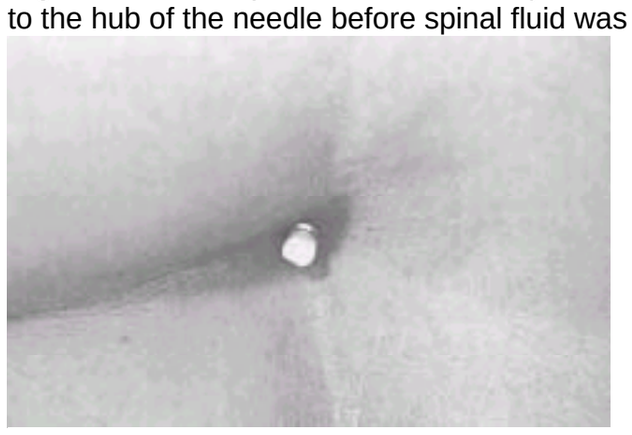


Figure 63-1 Schematic drawing of the brainstem, showing the major elements of the vestibulo-ocular reflex (VOR) pathway. The *solid line* indicates the direct projection between the vestibular nuclei and the third and sixth nuclei. The *stippled area* represents the indirect projections between the nuclei. *(Modified from Barr ML, Kiernan JA: The Human Nervous System—An Anatomical Viewpoint, 5th ed. Philadelphia, JB Lippincott, 1988, p 330.)*

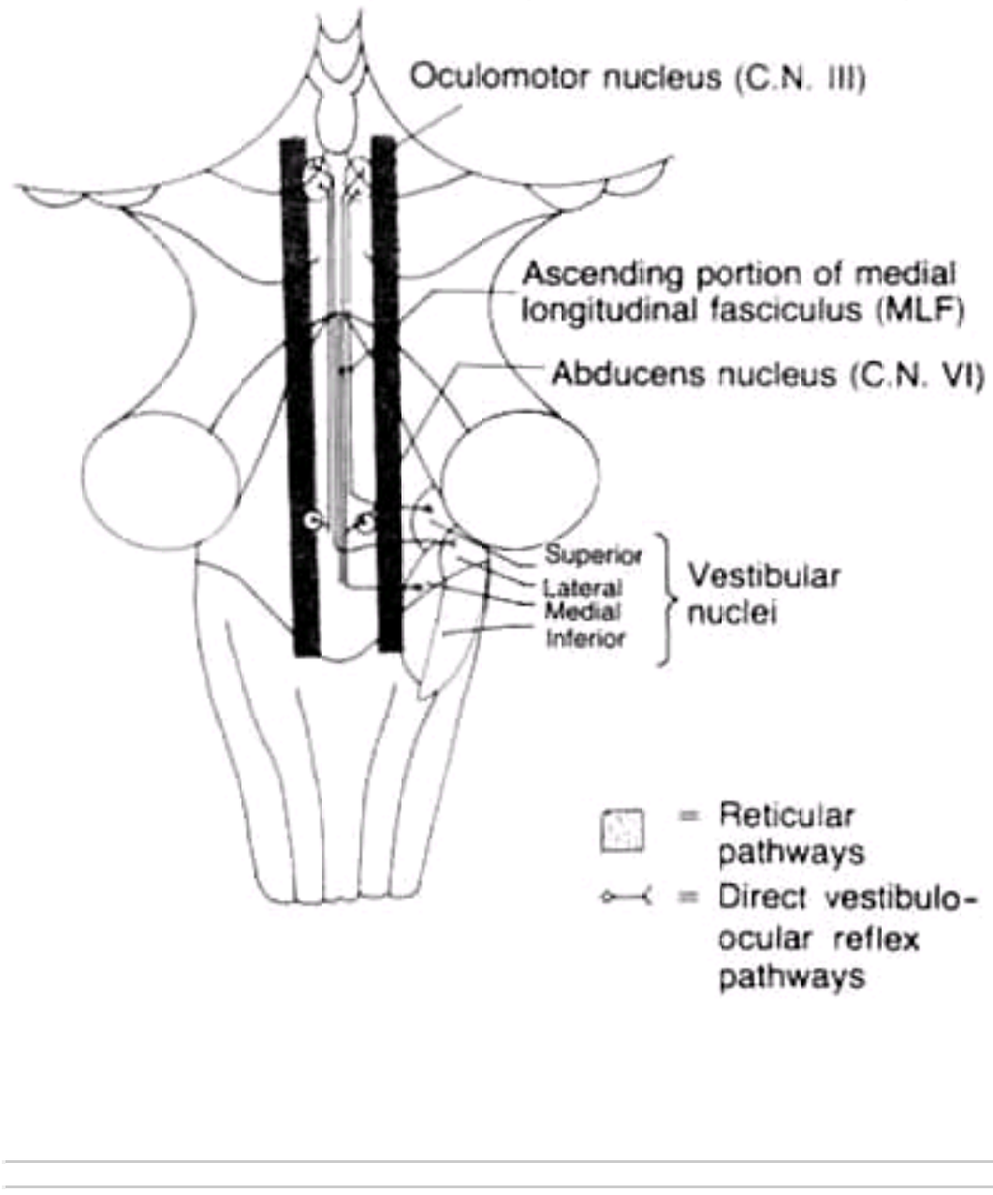


Figure 63-7 Neurotransmitter action at a neuromuscular junction. Acetylcholine *(Ach)* is formed and stored in the nerve terminal. It is released by nerve terminal depolarization in the presence of calcium and binds with receptors on the postsynaptic membrane. After producing an ionic conductance charge, it is hydrolyzed by acetylcholinesterase. *(From Daube JR, Reagen TJ, Sandok BA, Westmoreland BF: Medical Neurosciences: An Approach to Anatomy, Pathology and Physiology, and Physiology by Systems and Levels. Boston, Little, Brown, 1986, p 278.)*

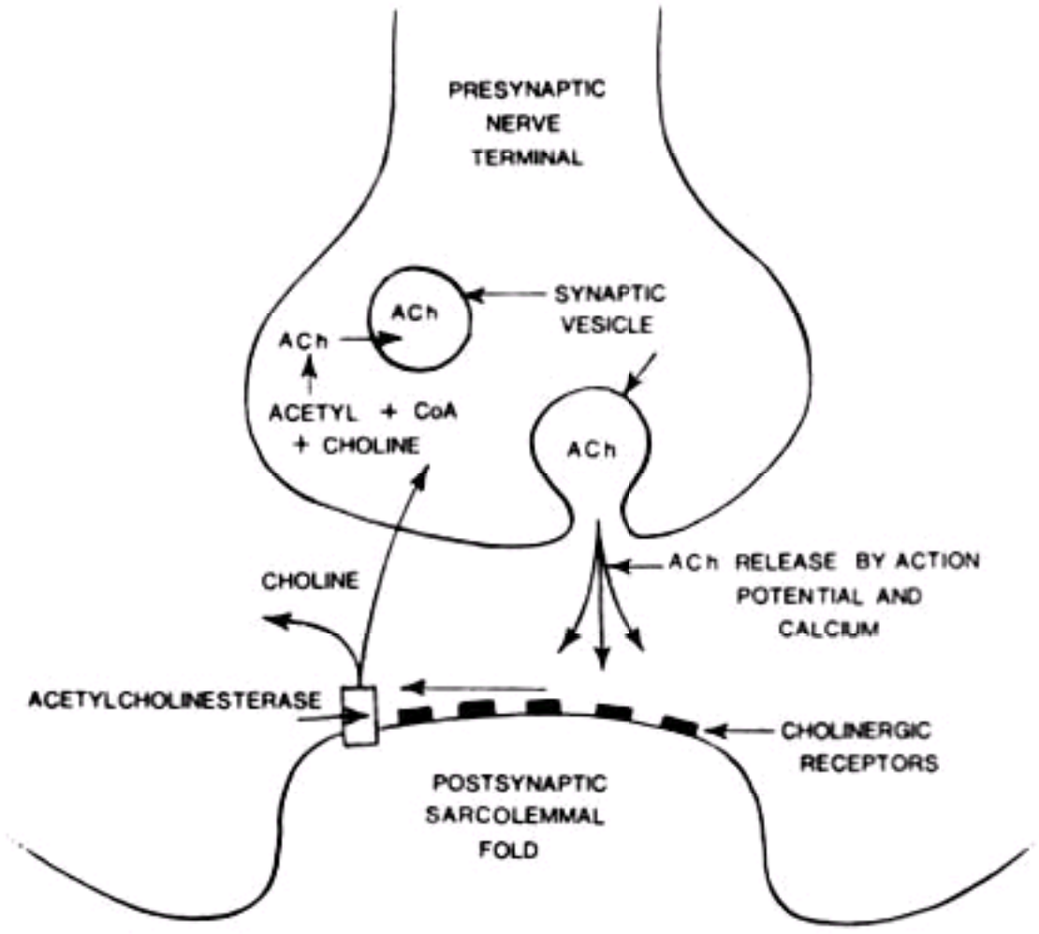


Figure 63-8 *A,* Before ice pack placement. *B,* After ice pack placement, improvement is noted in ptosis of the right eye. *(From Sethi KD, Rivner MH, Swift TR: Ice pack test for myasthenia gravis. Neurology 37:1383, 1987.)*

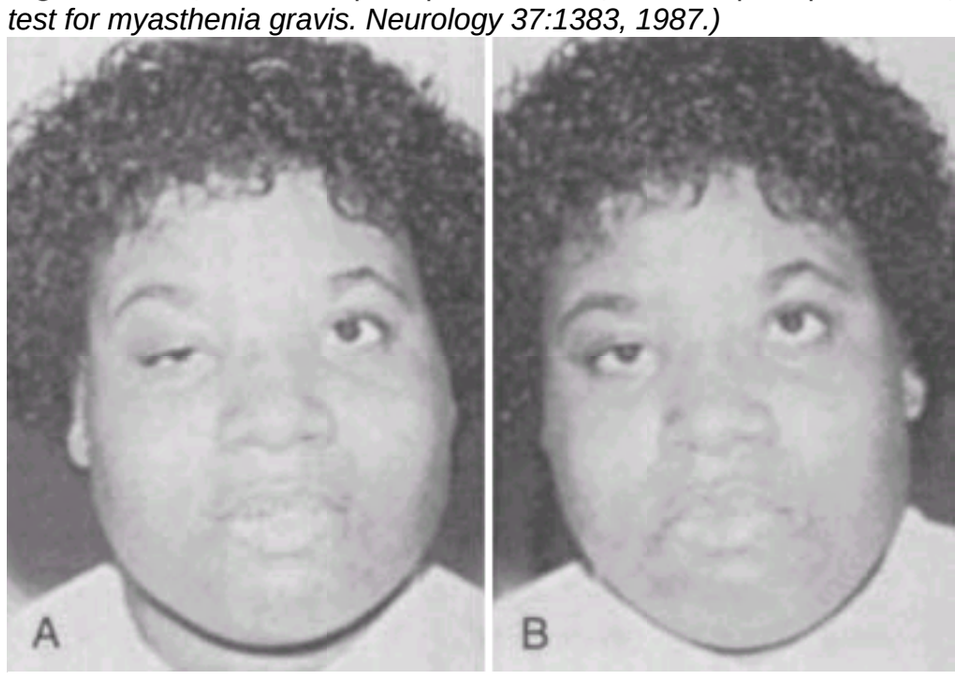


Figure 64-1 The "routine" progression of visual acuity assessment is reversed in the emergent presentation. Assessing an intact visual pathway begins with quickly discerning if the patient has light perception (LP), can see hand motion (HM), and count fingers at 3 feet (CF). Subsequent progression to assess vision at 10, then 20, feet from a standard eye chart ensues.

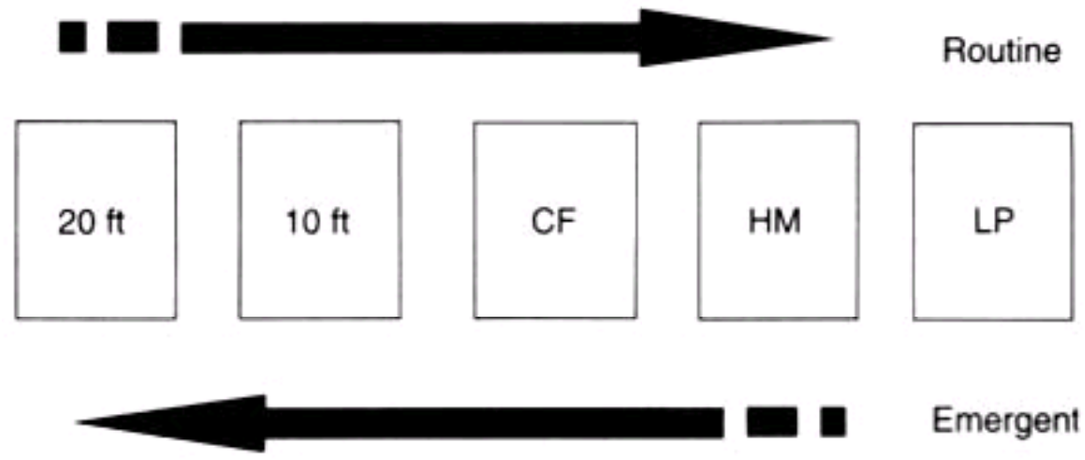


Figure 64-2 A commercial pinhole device reveals refractive error caused by corneal aberration (excess tearing or nearsightedness). *A*, First measure visual acuity without the device. *B*, Then measure with the pinhole cover lowered. Acuity with and without the pinhole device should be documented. *C*, If the patient cannot stand or a formal eye chart is not available, ask the patient to read this "distance equivalent" chart by holding this figure 14 inches away from the patient.

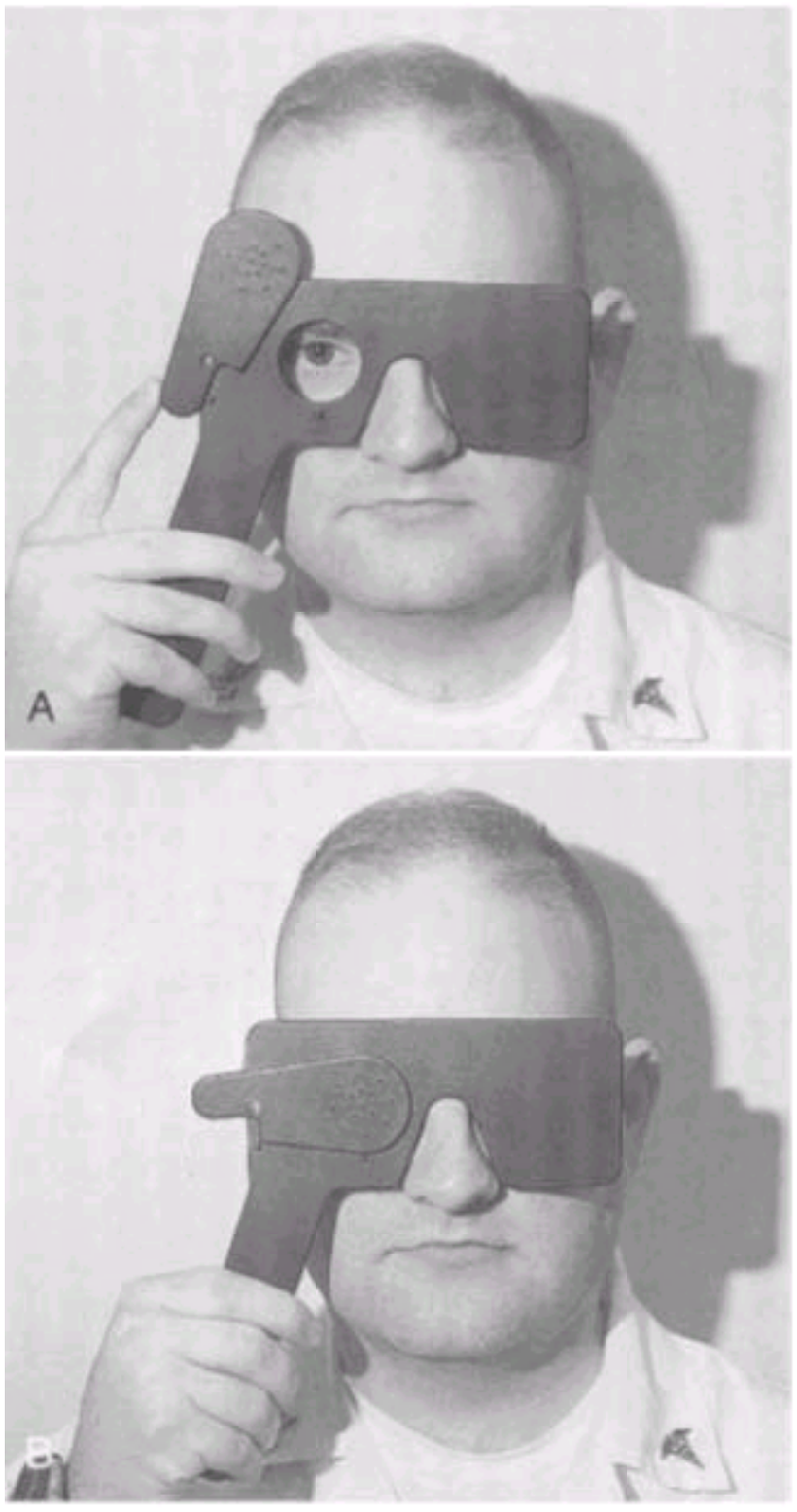


Figure 64-3 Optokinetic Nystagmus (OKN) testing will determine if there is an intact visual pathway. OKN is induced by the passage of a regularly sequenced pattern in front of the eye such as this commercially available drum. The patient is directed to look at the drum that is held in front of the patient and rotated slowly. Alternatively, a tape measure can be drawn across the line of sight while asking the patient to look directly at it as it passes.

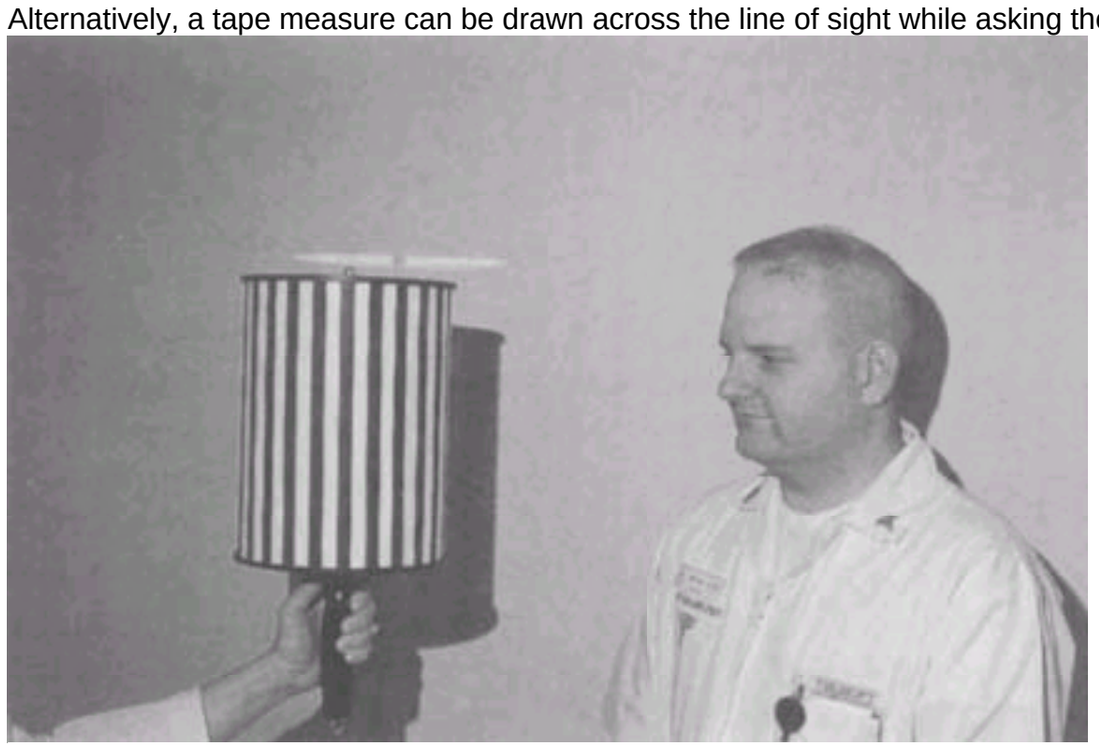
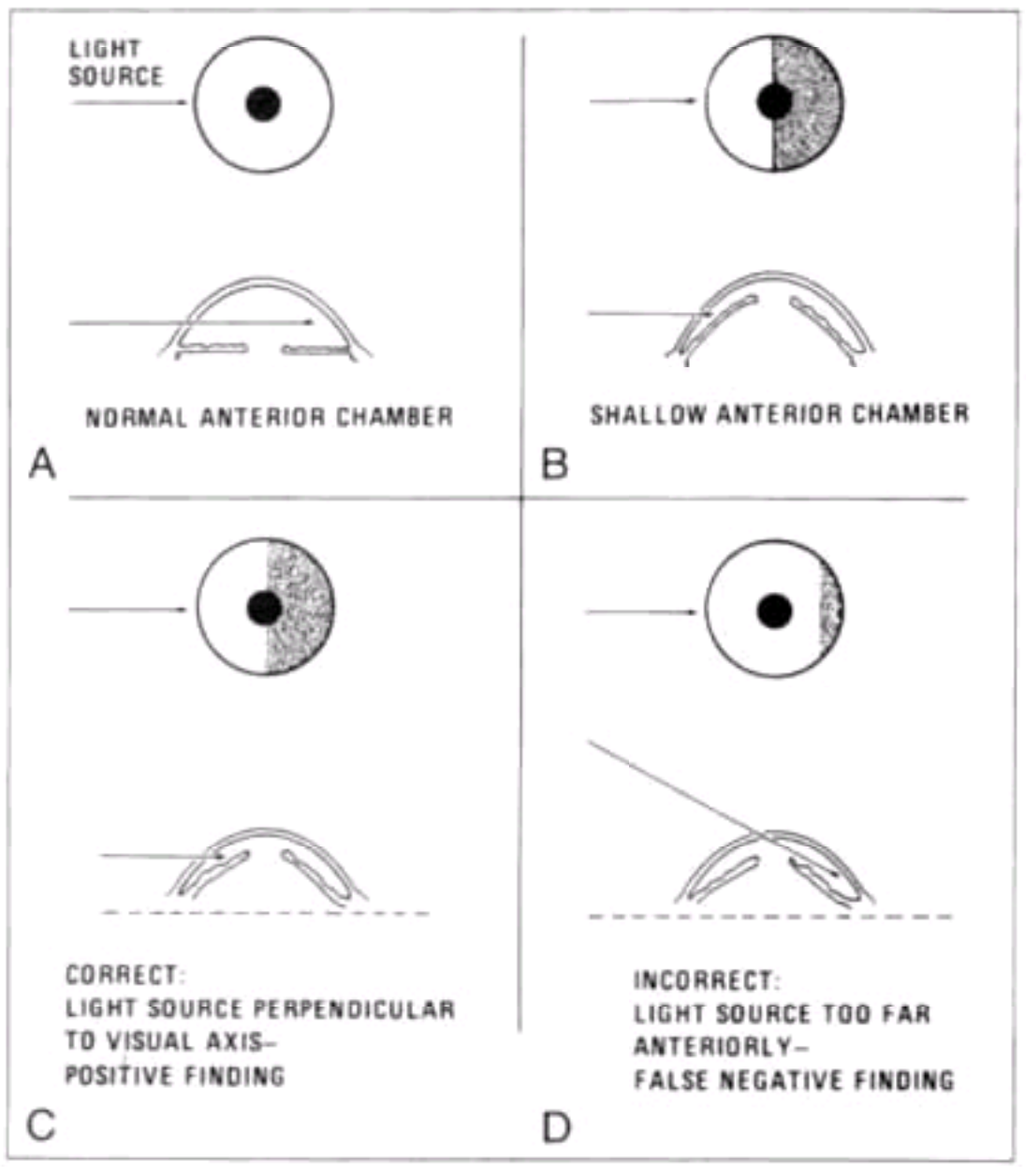


Figure 64-4 Anterior chamber depth and transillumination test. *A,* Normal anterior chamber with negative transillumination test. Note that the entire iris is illuminated. *B,* Shallow anterior chamber with positive transillumination test. Note the shadow on the outer half of the iris. *C,* Shallow anterior chamber with correctly placed light source yielding a true-positive test result in the presence of a shallow anterior chamber. *D,* Shallow anterior chamber with incorrectly placed light source giving a false-negative test result. *E,* Clinical use of the penlight examination to assess the depth of the anterior chamber. The examiner sits face-to-face with the patient to ensure that the light source is perfectly perpendicular to the line of vision. *F*, Note the shadow cast on the nasal portion of the iris, indicating a very narrow anterior chamber in this patient with acute angle closure glaucoma. *(A–D from Bresler MJ, Hoffman RS: Prevention of iatrogenic acute narrow-angle glaucoma. Ann Emerg Med 10:535, 1981. Reproduced by permission.)*



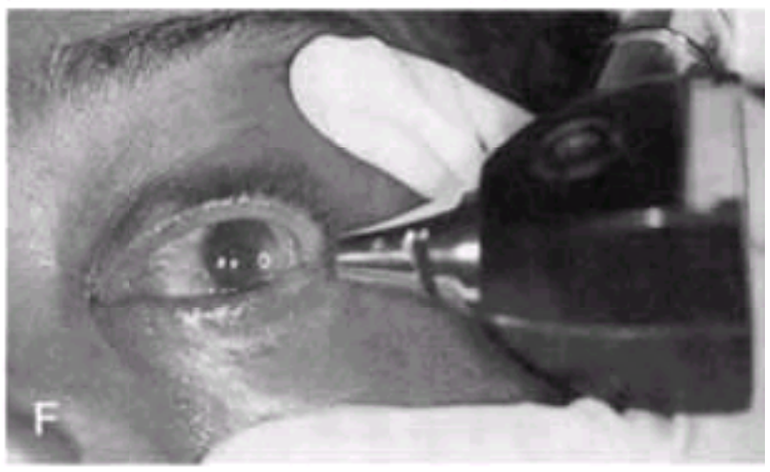


Figure 64-5 *A*, After neosynephrine drops were instilled in the nose to facilitate tube passage, this comatose patient was nasotracheally intubated for his drug overdose. *B*, On a subsequent examination a unilateral fixed and dilated pupil was noted. The pupil dilation was from neosynephrine nose drops that were snorted from the nose into the eye during intubation, simulating cerebral herniation. Other unusual causes of a fixed and dilated pupil are endotracheal epinephrine expelled from the lungs and splashed in the eye during cardiopulmonary resuscitation and inadvertant contamination of the eye following application of a scopolamine patch behind the ear.

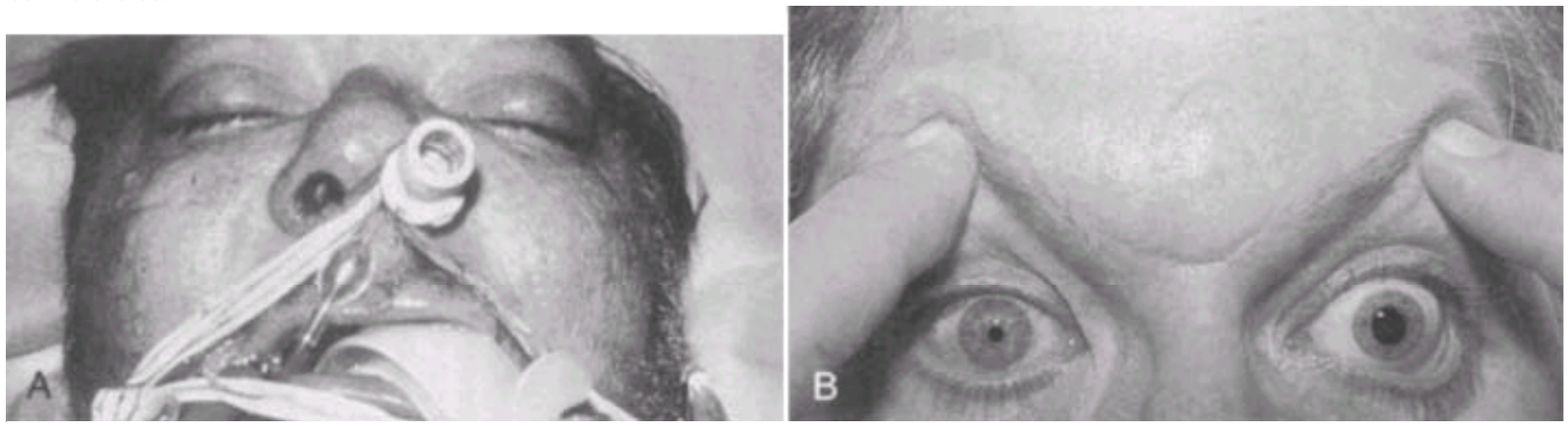


Figure 64-6 *A*, Administration of eyedrops. The patient should lie in a supine position or with the head tilted back. The patient's gaze should be directed upward. The lower lid is pulled downward and a single drop of medicine is instilled in the lower conjunctival fornix. The patient should be instructed to close the eyelids for 1 minute to increase the contact of the medicine with the globe and to decrease the medication outflow down the tear duct and over the lid margin. *B*, If one is administering large amounts of eye drops that have systemic effects, such as beta blocker drops, the operator's index finger is placed under the inferior eyelid along the nasal borders of the eye, firmly compressing the nasolacrimal duct against the globe for a few minutes, thereby preventing migration of the drops into the nose and reducing systemic absorption.

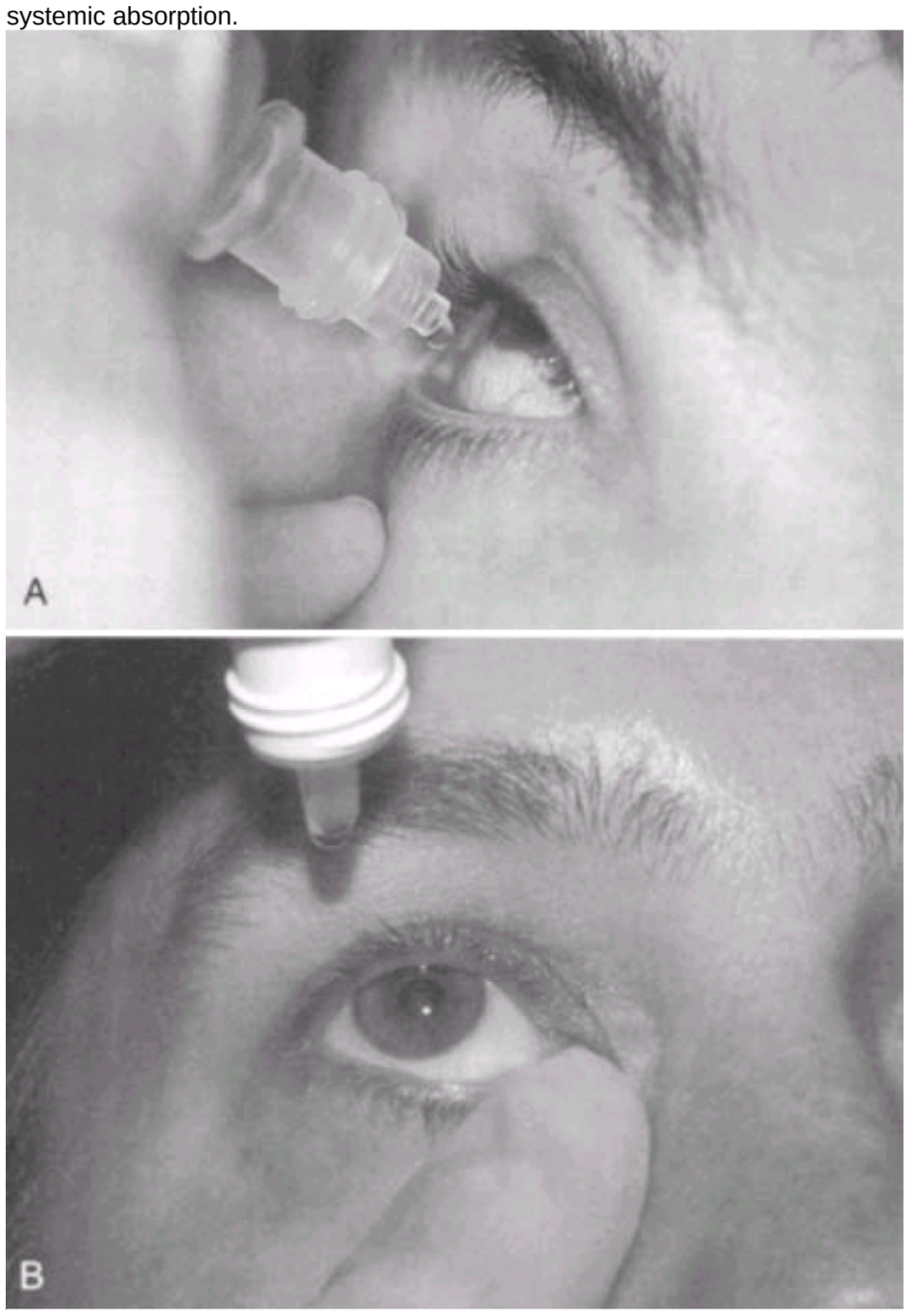
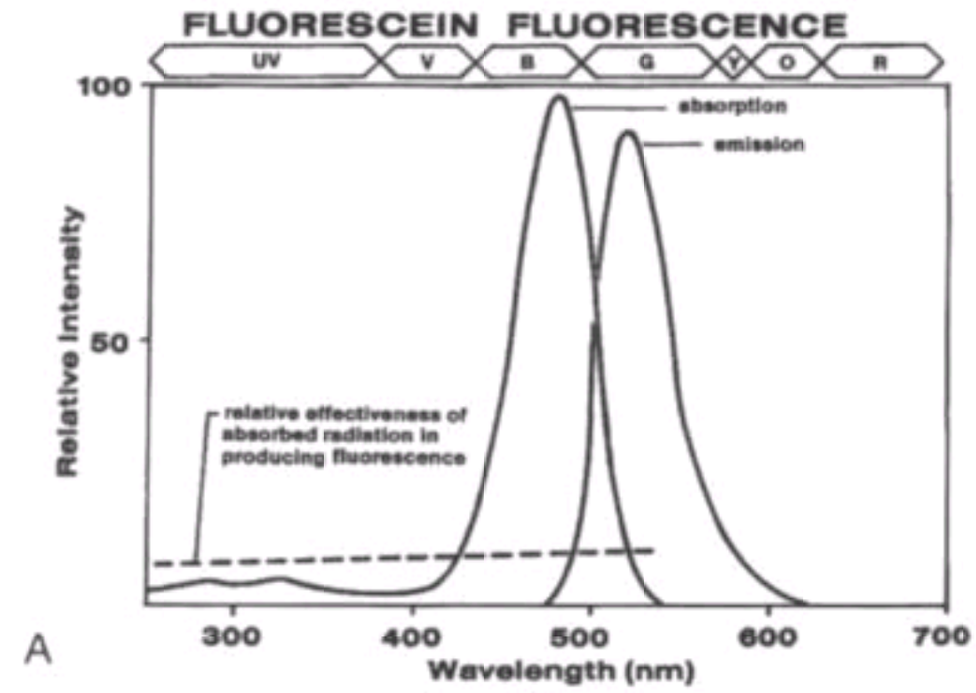


Figure 64-7 *A*, Sodium fluorescein absorbs light in the blue wavelengths and emits the energy in the longer green wavelengths. *B*, This large corneal abrasion is readily seen without the slit lamp when fluorescein is instilled into the eye. Smaller abrasions, or corneal injuries produced by keratitis or welder's arc flash injuries, require slit lamp evaluation to identify minor corneal defects. Even minor traumatic abrasions will escape detection with only a blue light examination by the naked eye.



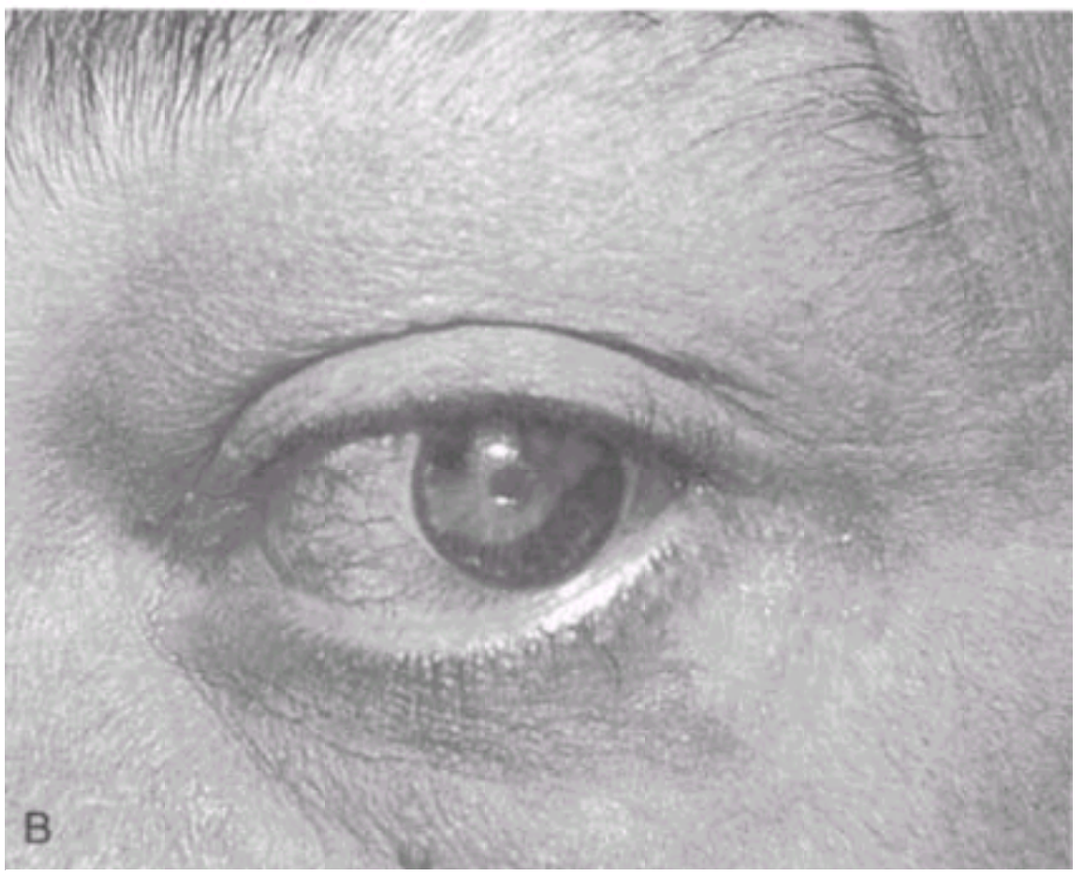


Figure 64-8 The fluorescein strip has been moistened with 1 drop of saline or tap water. The lower lid is depressed and a wetted strip is then placed gently onto the inside of the patient's lower lid so that only the smallest amount is instilled. Excess fluorescein may obscure subtle findings and thus should be avoided. Fluorescein will permanently stain contact lenses not removed (as seen in this model).

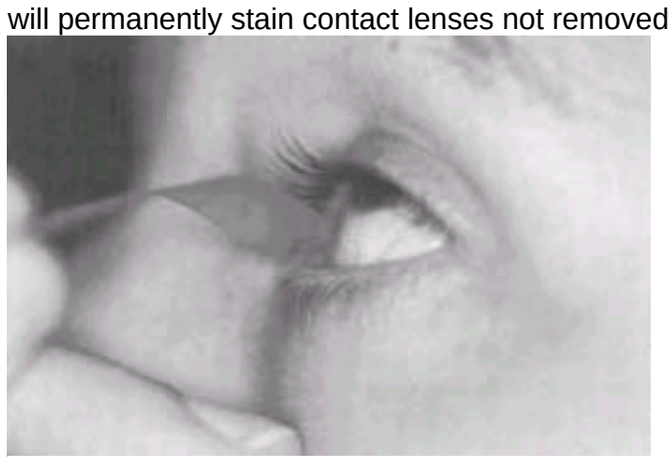


Figure 64-10 Patterns of acute corneal injury.

1. Traumatic abrasion: usually with linear features and sharp borders when seen early (< 24 hours). Occurs more in the central cornea.
2. Abrasion from foreign body (FB): vertical abrasions on the upper cornea seen when an FB is embedded in the upper lid. Also shown is a rust ring with a metallic FB.
3. Exposure pattern: seen with prolonged exposure to ultraviolet (e.g., welding flash, sunlamp exposure); produces bandlike keratitis over lower half of the cornea. Squinting in the setting of the bright light protected the upper corneal surface.
4. Herpes simplex keratitis: classic dendritic pattern.
5. Adenovirus keratitis: Diffuse minute corneal staining seen in epidemic keratoconjunctivitis about 7 days after symptoms.
6. Contact lens overuse: Central punctate staining.

(From Knopp, K, Trott A: Ophthalmologic procedures in the emergency department—Part III: Slit lamp use and foreign bodies. Acad Emerg Med 2:227, 1995. Reproduced by permission.)

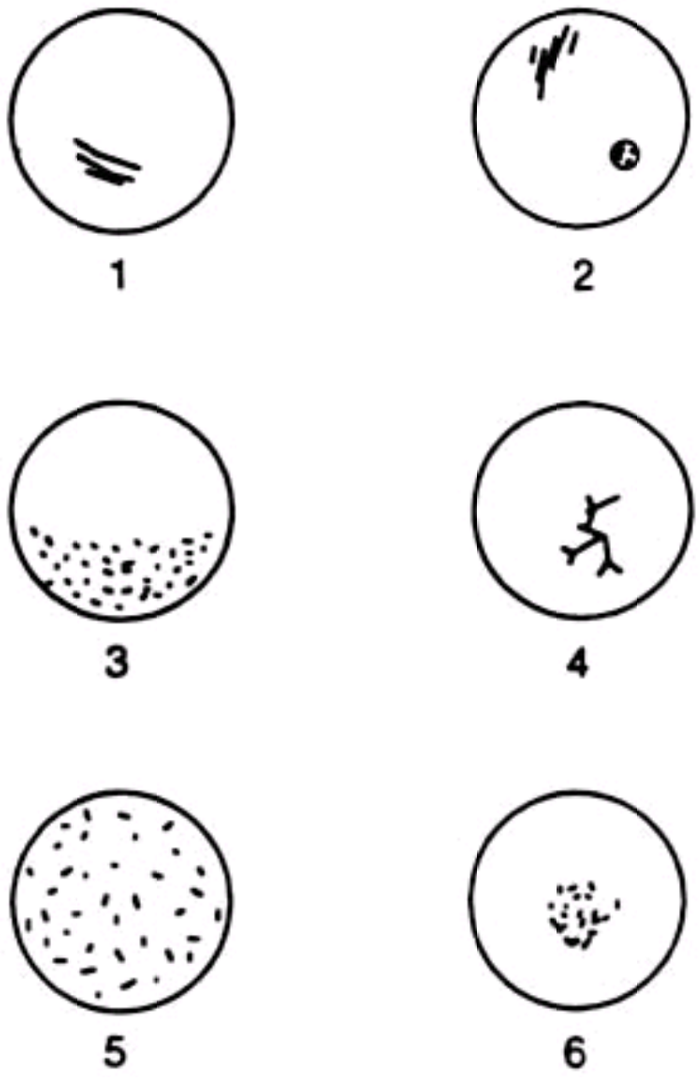


Figure 64-11 Double eversion of upper lid using a lid retractor. From Fogle JA, Spyker DA: Management of chemical and drug injury to the eye. *(In Haddad LM, Winchester JF (eds): Clinical Management of Poisoning and Drug Overdose, 2nd ed. Philadelphia, WB Saunders, 1990 Reproduced by permission.)*

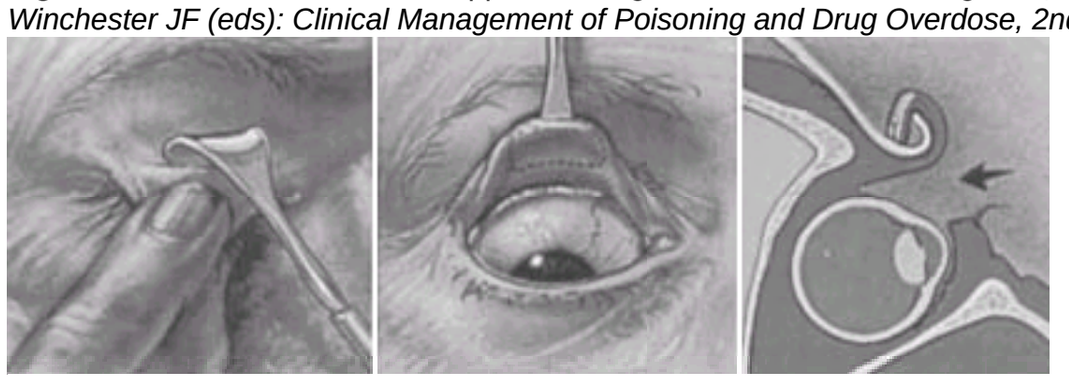


Figure 64-12 Devices for separating eyelids. A Desmarres retractor and a retractor improvised from a paper clip allow active manipulation of lids. Freestanding specula may require a seventh nerve block to reduce blepharospasm. *(From Fogle JA, Spyker DA: Management of chemical and drug injury to the eye. In Haddad LM, Winchester JF (eds): Clinical Management of Poisoning and Drug Overdose, 2nd ed. Philadelphia, WB Saunders, 1990. Reproduced by permission.)*

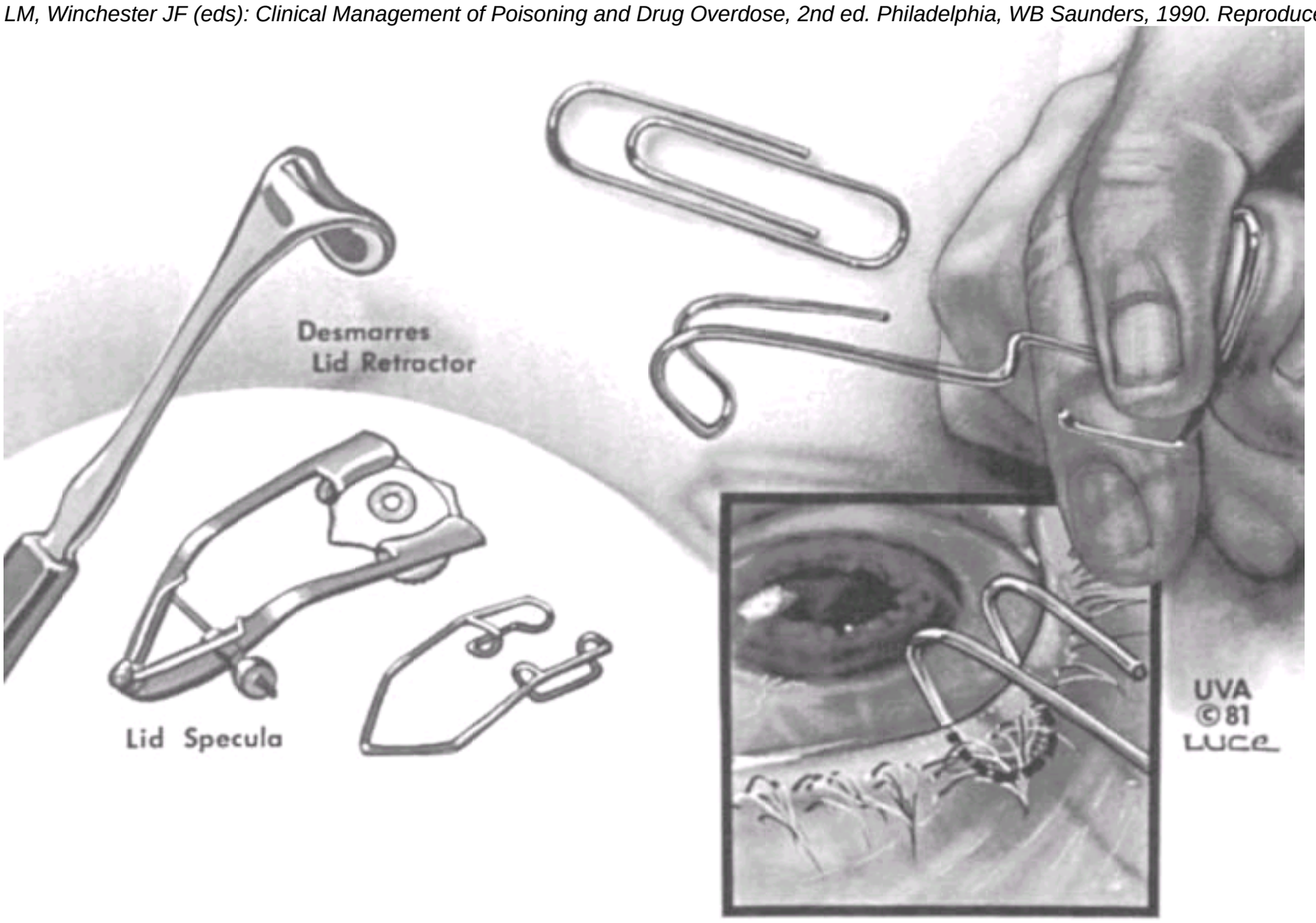


Figure 64-13 Irrigation technique using a Desmarres retractor for lid separation Irrigation is performed with saline or lactated Ringer's solution. For alkaline injuries the pH of the eye should be checked with pH paper to assess the adequacy of irrigation (normal pH = 7.4). Since a rebound may occur with alkaline products, the pH should be rechecked 20 to 30 minutes *after irrigation* to assure complete removal. *(From Fogle JA, Spyker DA: Management of chemical and drug injury to the eye. In Haddad LM, Winchester JF: Clinical Management of Poisoning and Drug Overdose, 2nd ed. Philadelphia, WB Saunders, 1990. Reproduced by permission.)*

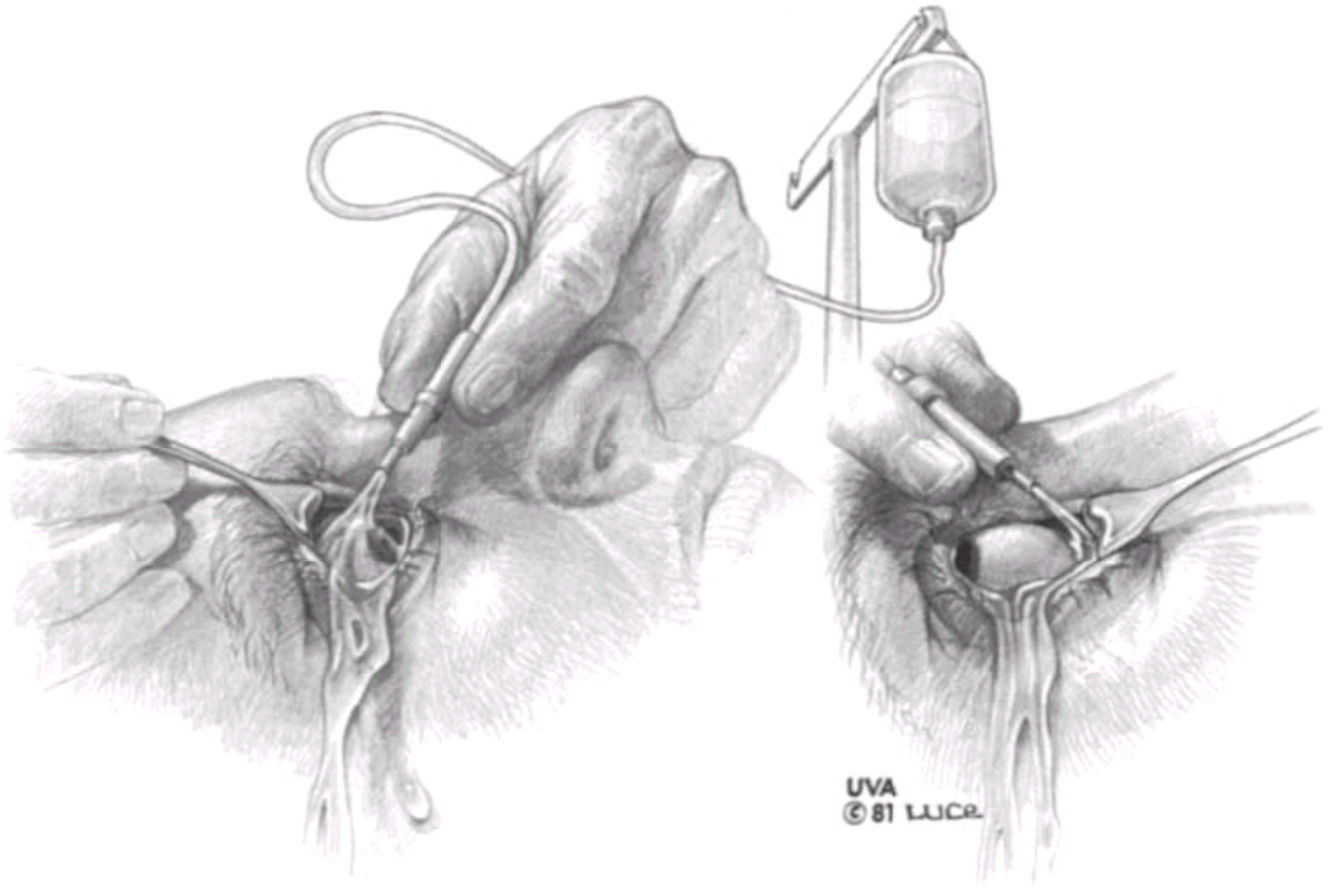


Figure 64-14 Injection points for facial and orbital anesthesia and akinesia. *A,* Van Lint technique of orbicularis infiltration. *B,* Retrobulbar injection site. *C,* O'Brien facial nerve block. *C',* Alternative facial nerve block by tympanomastoid fissure injection. *D,* Infraorbital sensory block. *E,* Supraorbital sensory block. Injection of orbicularis *(A)* or facial nerve *(C* or *C')* permits examination and treatment of the eye in the setting of severe blepharospasm. Anesthesia is placed within several mm of the nerves. *(From Deutsch TA, Feller DB: Paton and Goldberg's Management of Ocular Injuries, 2nd ed. Philadelphia, WB Saunders, 1985, p 17.)*

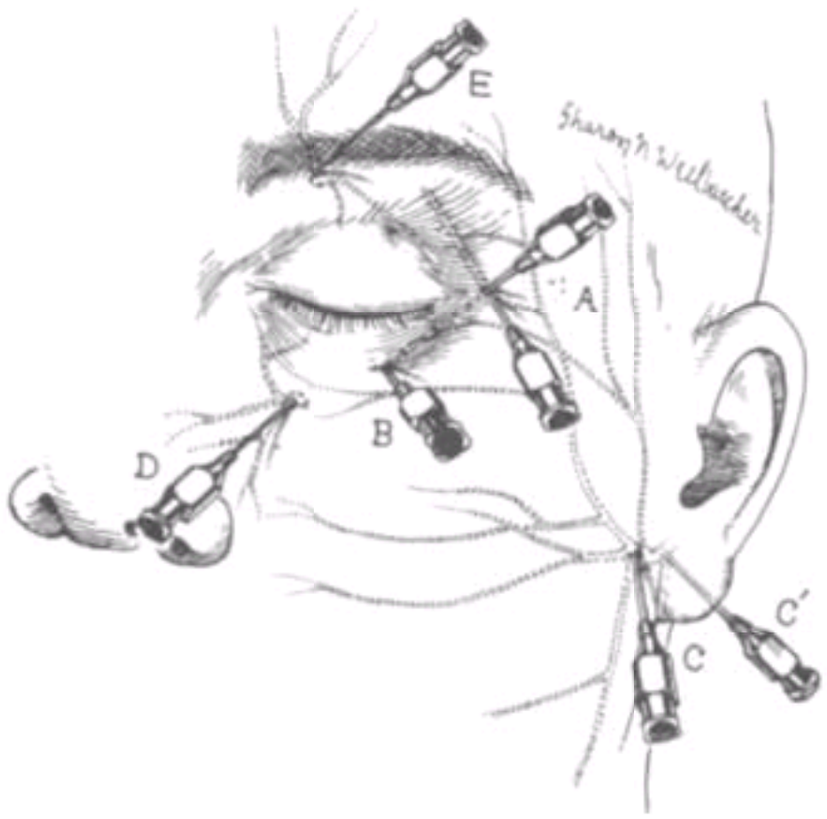


Figure 64-15 *A,* The Morgan therapeutic lens attached to intravenous tubing. *B,* Placement of the device into the anesthetized eye for irrigation. *(Courtesy of MorTan, Inc, Missoula, MT.)*

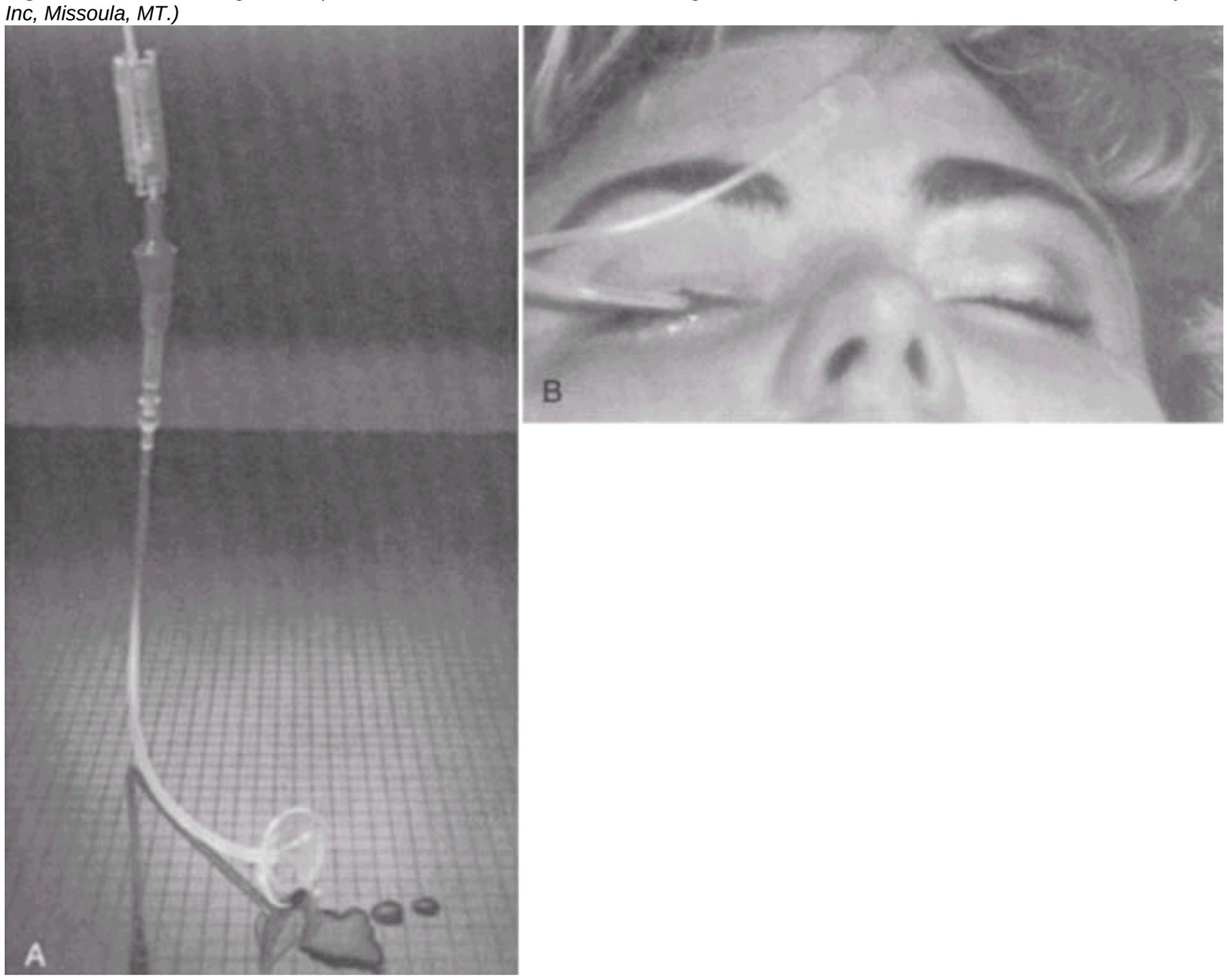


Figure 64-16 Continuous irrigation device. A central venous catheter (after perforation by the clinician) is looped in the fornices of the anesthetized eye, beginning from the temporal border of the eyelids. The eye is lightly patched after catheter placement.

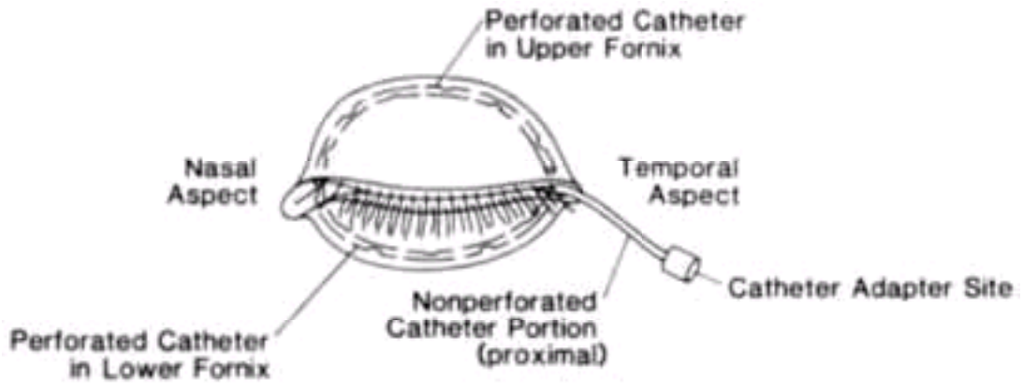


Figure 64-17 The Eye Irrigator is designed to slide up under the upper lid as shown. Irrigation is accomplished by connecting the device to an intravenous bag containing the desired irrigation fluid. It is used primarily at the injury site to supplement flushing at the eyewash station. *(Courtesy of the Eye Irrigator, American Health and Safety, Inc.)*

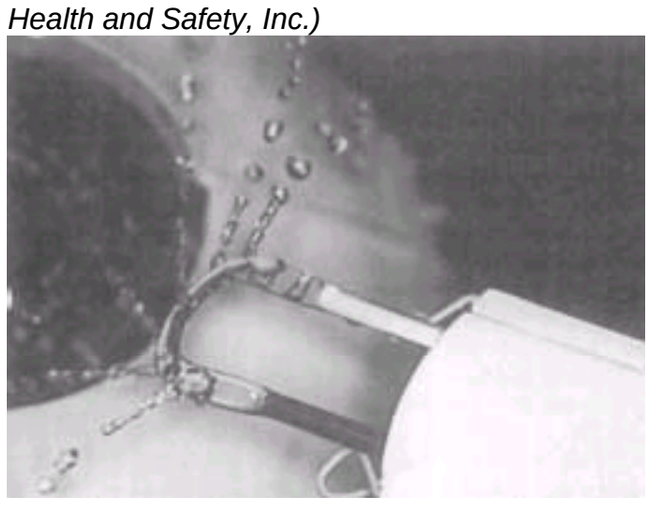


Figure 64-18 *A*, Corneal laceration with prolapse of the iris. The extruded iris is dark, mimicking a corneal foreign body. Often the only clue is an abnormal pupil and the extruded iris may not be appreciated as intraocular tissue. The pupil is irregular (often pear- or teardrop-shaped), pointing toward the laceration (photo courtesy of Lawrence B. Stack, MD). *B*, A pear-shaped pupil without protrusion of the lens is a more subtle, yet characteristic, indication of a perforated globe. *C*, Another indication of a penetrating globe injury is periorbital fat protruding from an upper eyelid laceration. This patient was stabbed by a knife. *D*, This patient has an obviously cloudy lens soon after trauma. A projectile entered the temporal portion of the globe and produced a seemingly minor scleral hemorrhage. Patients with penetrating injuries to the globe should be treated with systemic antibiotics (such as cefazolin/gentamycin combination), tetanus toxoid if indicated, and antiemetics to control vomiting (which raises intraocular pressure).

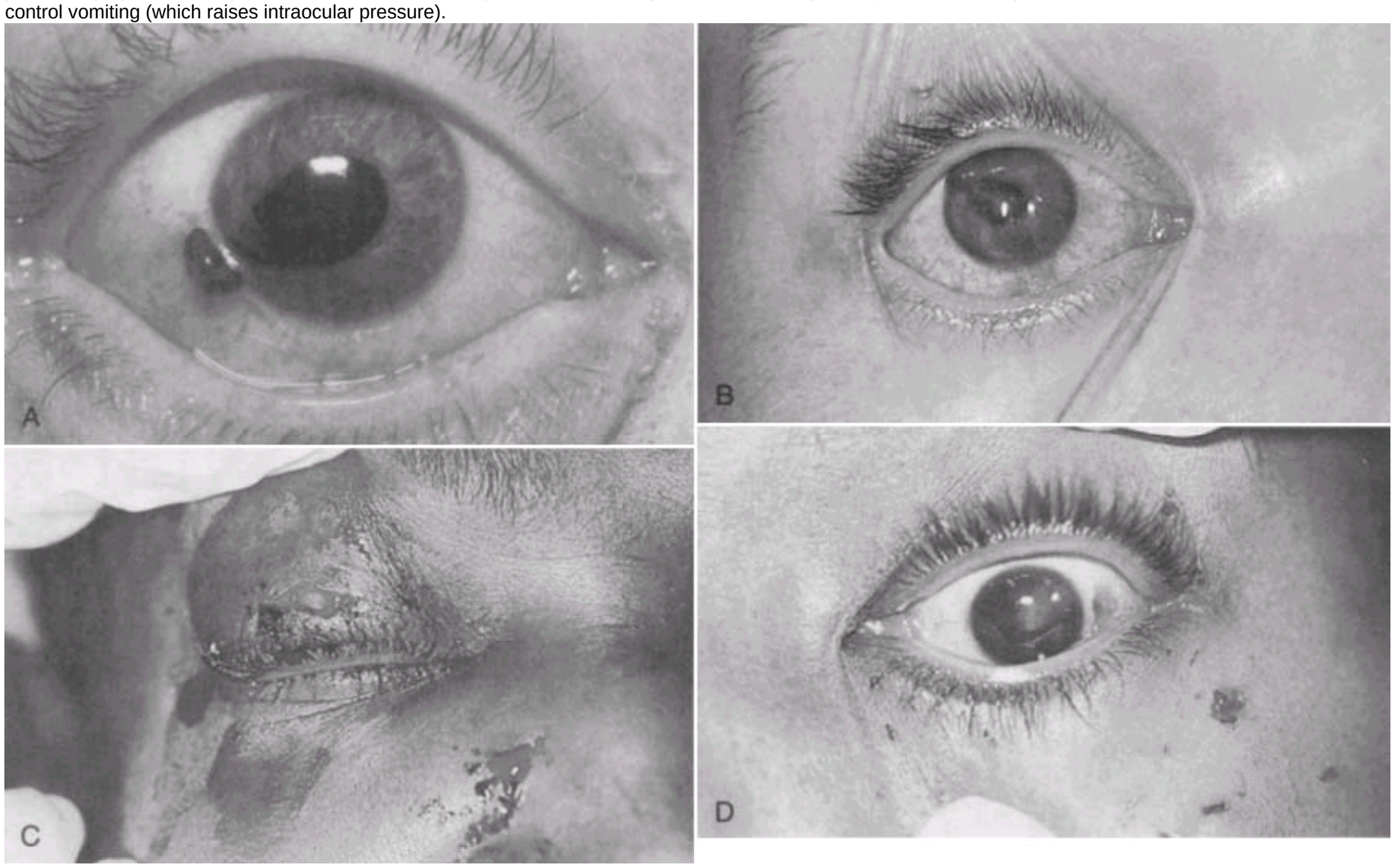


Figure 64-19 When a penetrating globe injury is suspected and a metal shield is not available in the emergency department or prehospital setting, a makeshift shield can be fashioned with available materials. A paper cup was used to fashion this shield.

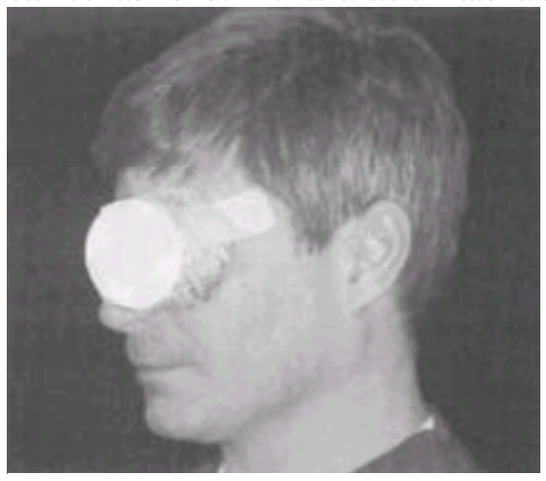


Figure 64-20 Single upper lid eversion. *A,* The patient is instructed to look downward, and the end of a cotton-tipped applicator is placed above the tarsal plate while the lashes and the lid margin are pulled down, out and up. *B,* One everts the lid and holds it by pressing the lashes against the superior orbital rim. If the foreign body is seen on the lid, it can usually be removed by touching it with a moistened cotton-tipped applicator. *(From Pavan-Langston D: Manual of Ocular Diagnosis and Therapy, 3rd ed. Boston, Little, Brown & Co, 1991. Reproduced by permission.) C*, This patient presented with a swollen and tender upper eyelid thought to be secondary to a stye. *D*, With lid eversion a small pustule was found under the upper eyelid. *E*, With a 27-gauge needle the pustule was incised and a drop of pus was expressed, and she made a rapid and uneventful recovery.

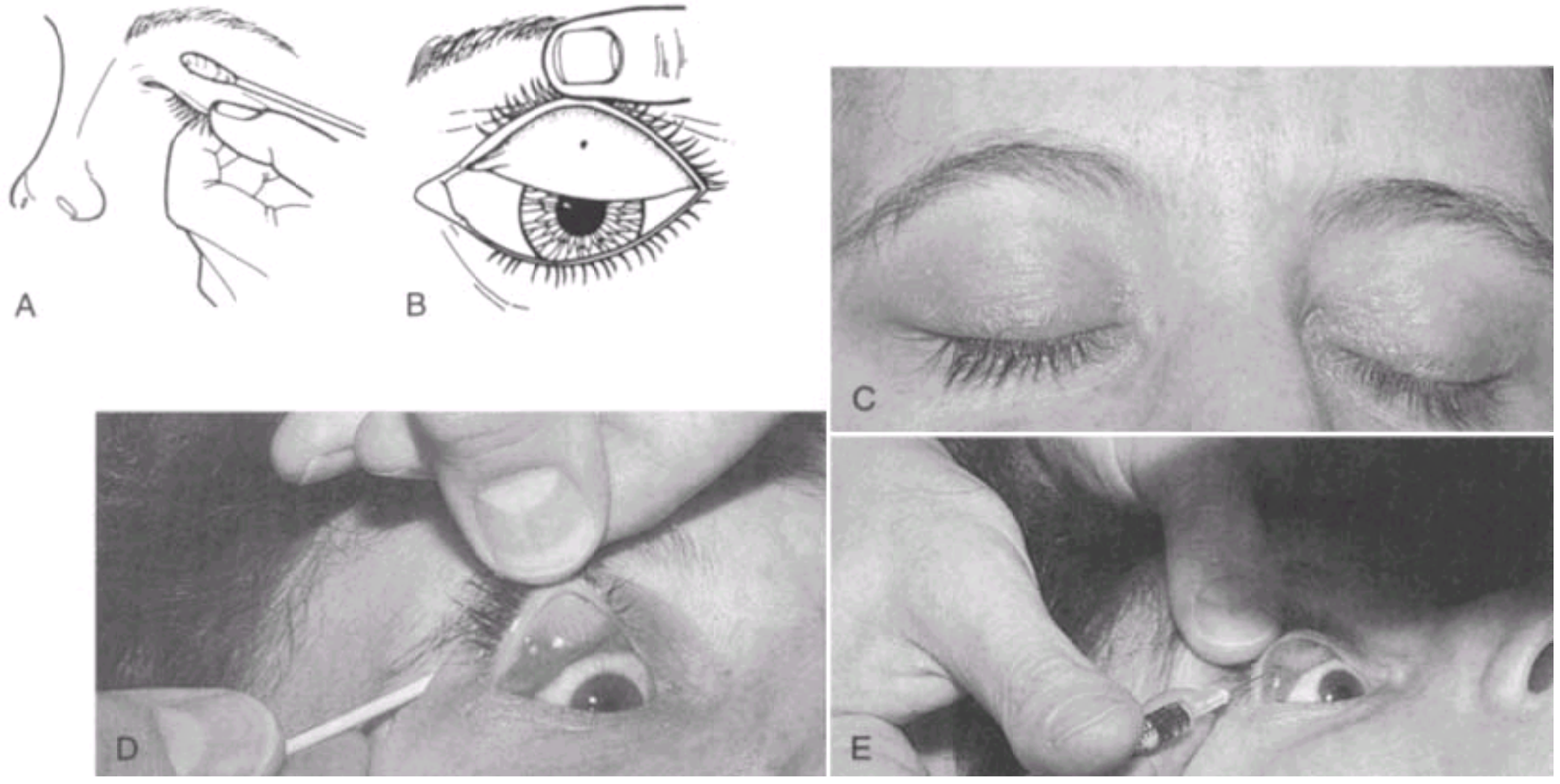


Figure 64-21 An "invisible" corneal abrasion casts an obvious shadow on the iris. The relative positions of *(a)* the corneal abrasion and *(b)* the iris shadow depend on the direction of the incident light (as in *A* and *B*). *(From Paton D, Goldberg MF: Management of Ocular Injuries. Philadelphia, WB Saunders, 1976. Reproduced by permission.)*

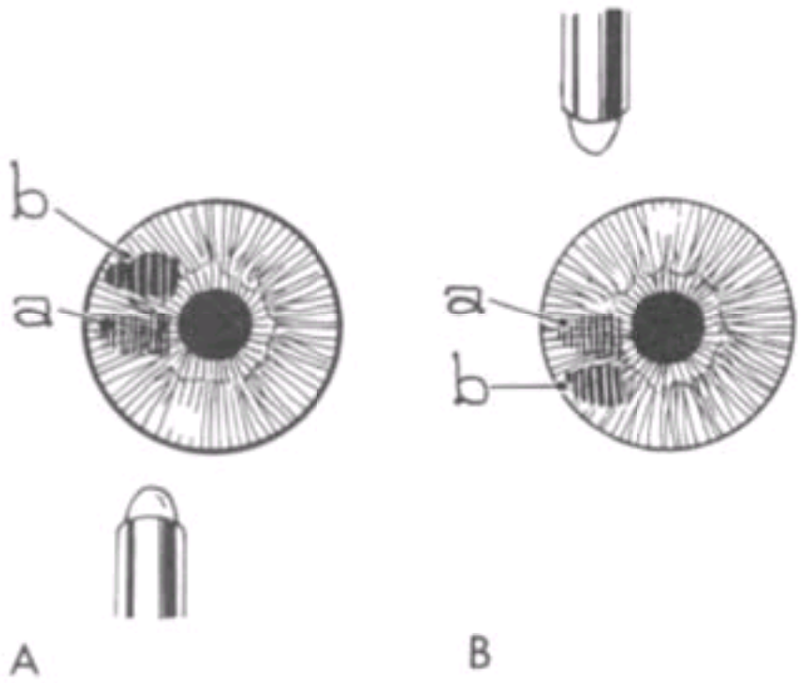


Figure 64-22 This embedded corneal foreign body is readily seen under slit lamp examination. A removal device (needle, spud, or burr drill) should be used for careful removal.

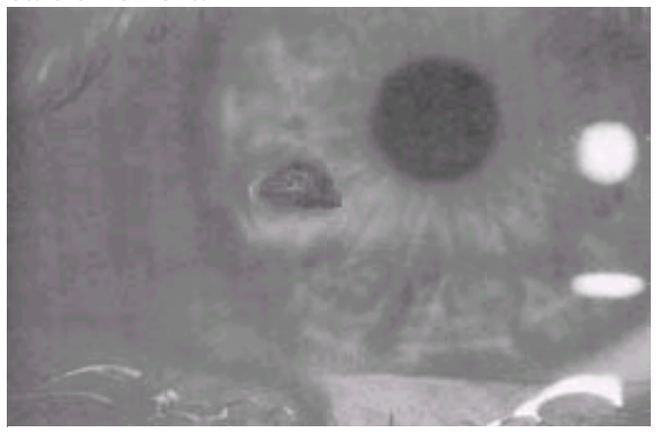
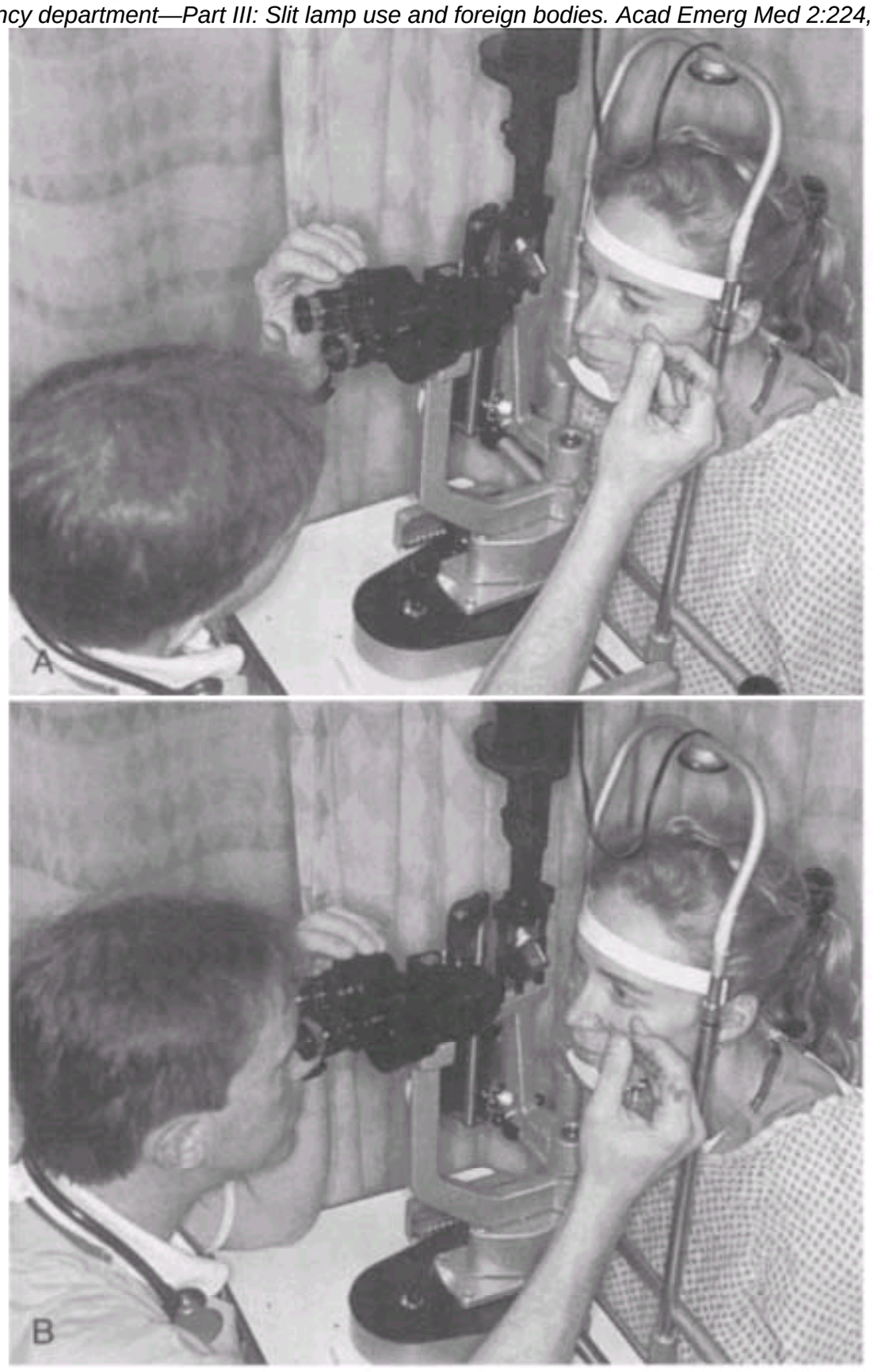


Figure 64-23 It is preferable to remove the corneal foreign body (FB) under the slit lamp. A topical anesthetic is applied and the operator uses a small syringe with a short 25- to 27-ga needle (such as a tuberculin syringe). Be certain that the needle is firmly attached to the syringe. *A*, *Under direct vision*, the syringe is brought close to the eye and the operator rests the hand on the patient's cheek. Be sure that the patient's forehead maintains continual contact with the cross bar on the slit lamp. *B,* While looking through the slit lamp, the needle is brought to the cornea and the FB is removed. *C,* The side of the instrument (drill bit or beveled edge of the needle) should be held tangential to the cornea. Arrows indicate the direction of device in order to remove the FB. *(From Knoop K, Trott A: Ophthalmologic procedures in the emergency department—Part III: Slit lamp use and foreign bodies. Acad Emerg Med 2:224, 1995. Reproduced by permission.)*



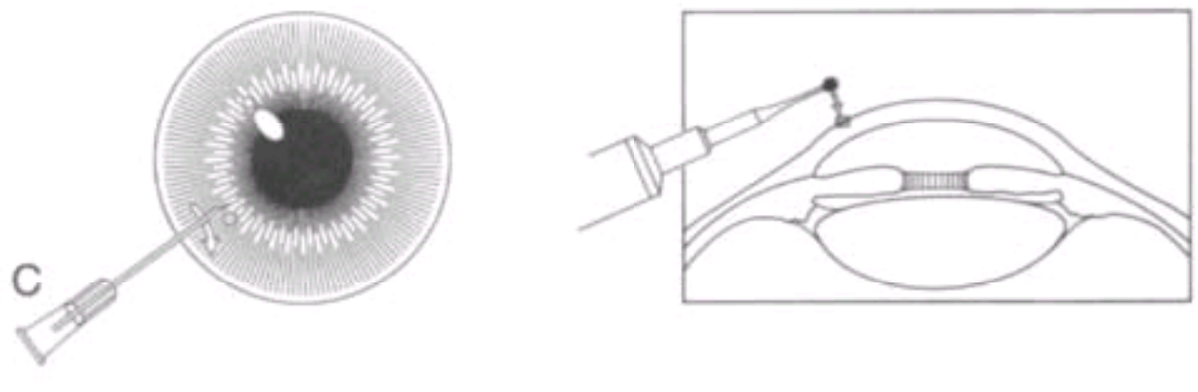


Figure 64-24 A typical rust ring is seen at 8 o'clock on the cornea. A burr drill can be used for attempted removal, which if unsuccessful, can be reattempted in 24 hours. *(From Knoop KJ, Stack LB, Storrow AB (eds). Atlas of Emergency Medicine, 2nd ed. New York, McGraw-Hill, 2002. Reproduced by permission.)*

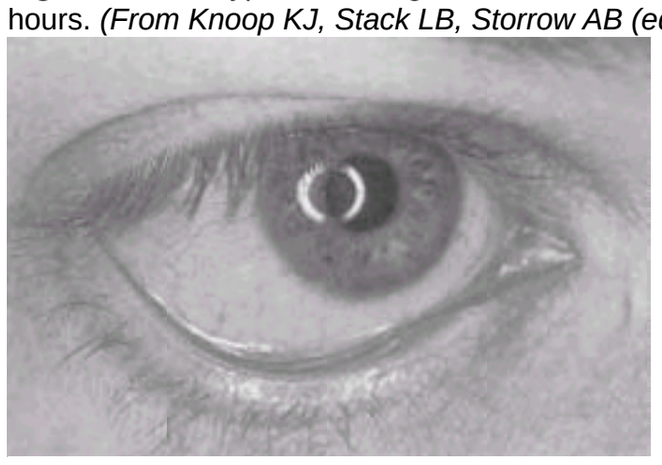


Figure 64-25 Application of eye patch. *A,* Vertically folded first patch in orbital recess. *B,* Horizontally oriented second patch with forehead to cheek taping. The contralateral eye should be kept closed throughout patching.

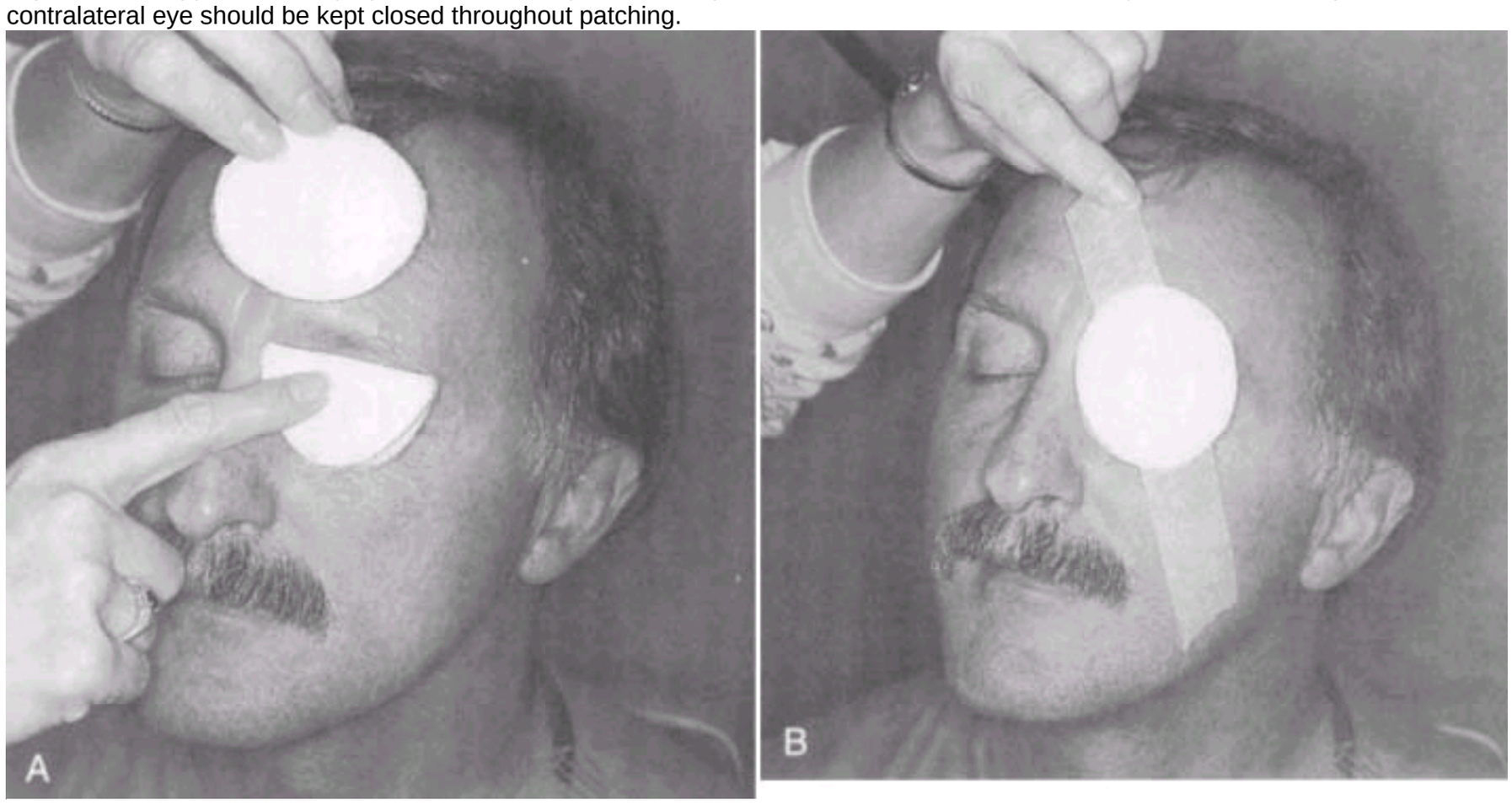


Figure 64-26 Final appearance of pressure eye patch.

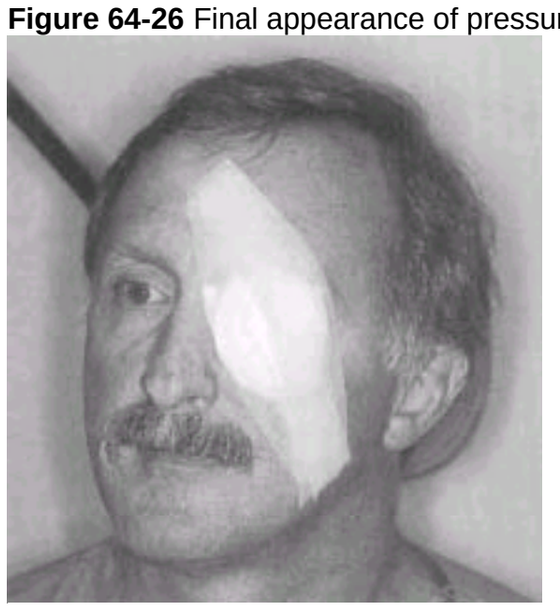


Figure 64-27 Use of the adjustable elastic strap pressure patch, Presspatch II. The clinician should ensure that the elastic straps for the patch are adjusted to avoid excessive globe pressure. *(Courtesy of Precision Therapeutics, Inc, Las Vegas, NV.)*

Figure 64-28 Donaldson eye patch (Keeler Instruments, Inc, Broomall, PA). *Inset* shows the means by which the patch can be released for eye inspection or medication administration.

Figure 64-29 Manual technique for removing a hard contact lens. *A,* Separation of lids. *B,* Entrapment of lens edges with lids. *C,* Expulsion of lens by forcing of lower lid under inferior edge of lens. *(From Grant HD, Murray RH, Bergeron JF: Brady Emergency Care, 5th ed. Englewood Cliffs, NJ, Prentice Hall, 1990, p 338. Reproduced by permission.)*

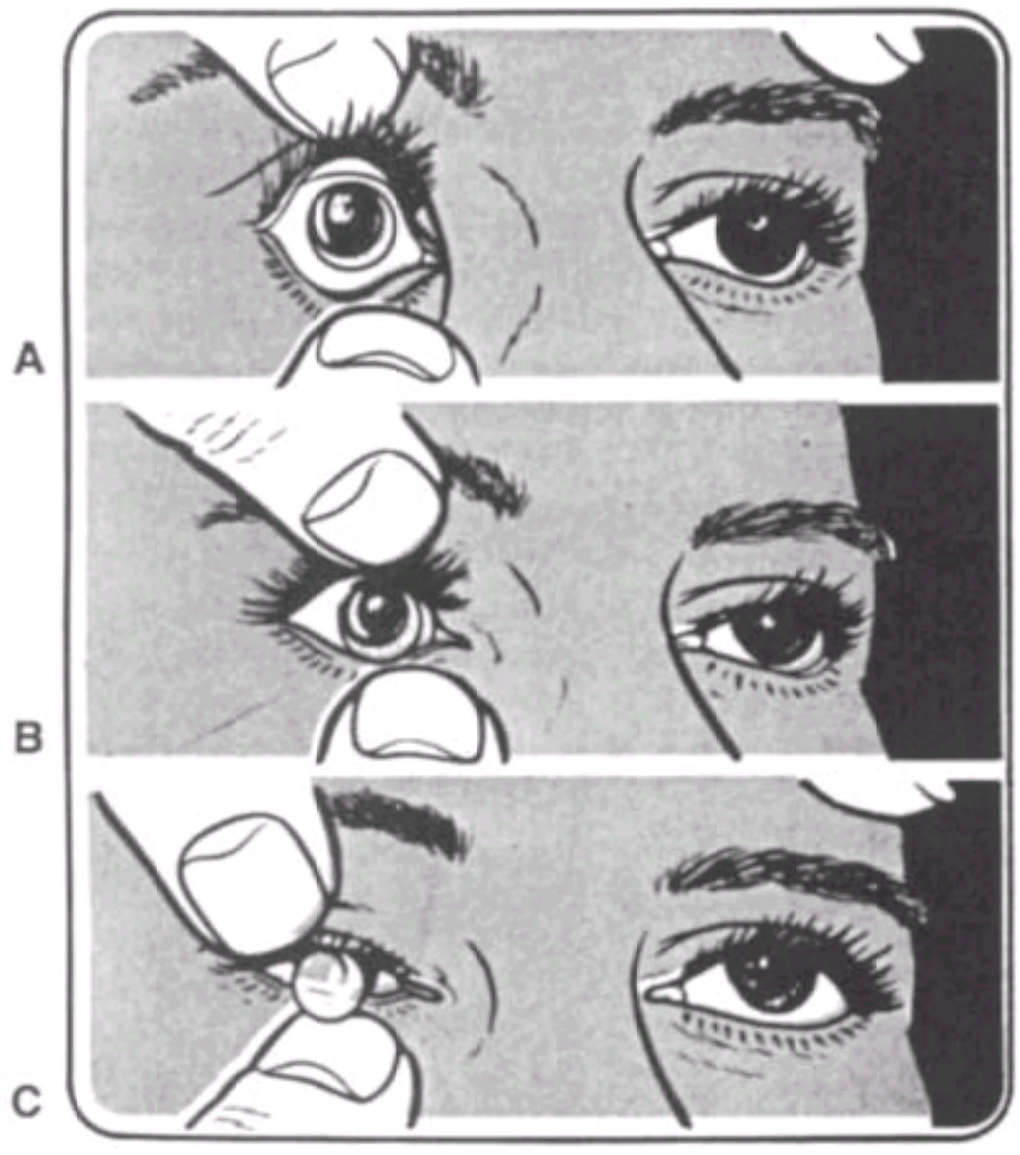


Figure 64-30 Use of a moistened suction cup to remove a hard contact lens. *(From Grant HD, Murray RH, Bergeron JF: Brady Emergency Care, 5th ed. Englewood Cliffs, NJ, Prentice Hall, 1990, p 338. Reproduced by permission.)*

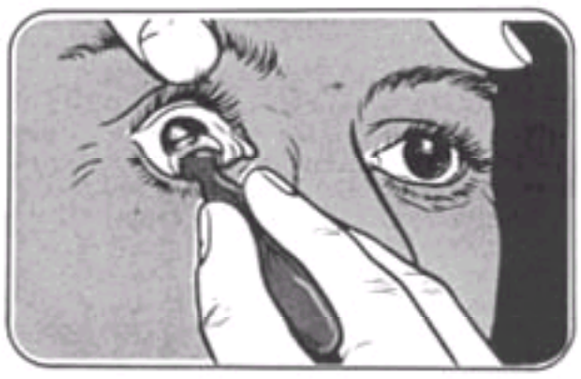


Figure 64-31 Removal of a hard scleral lens. *A,* Separation of lids. *B,* Forcing of lower lid beneath edge of scleral lens by temporal traction on lower lid. *C,* Lifting of lens off eye. *(From Grant HD, Murray RH, Bergeron JF: Brady Emergency Care, 5th ed. Englewood Cliffs, NJ, Prentice Hall, 1990, p 338. Reproduced by permission.)*

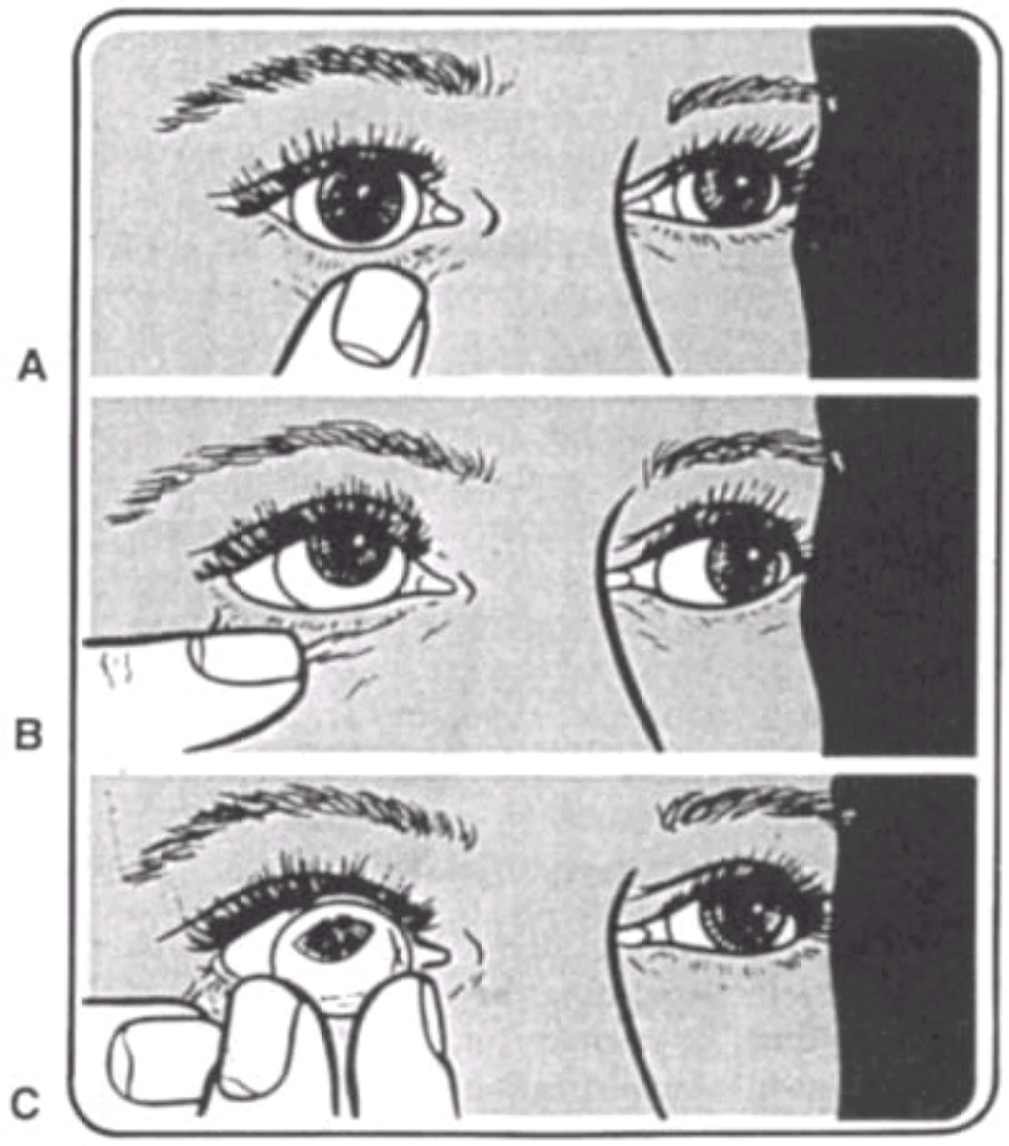


Figure 64-32 Removal of a soft contact lens. *A,* Separation of lids and movement of contact onto sclera using index finger. *B,* Pinching of lens between thumb and index finger. *(From Grant HD, Murray RH, Bergeron JF: Brady Emergency Care, 5th ed. Englewood Cliffs, NJ, Prentice Hall, 1990, p 338. Reproduced by permission.)*

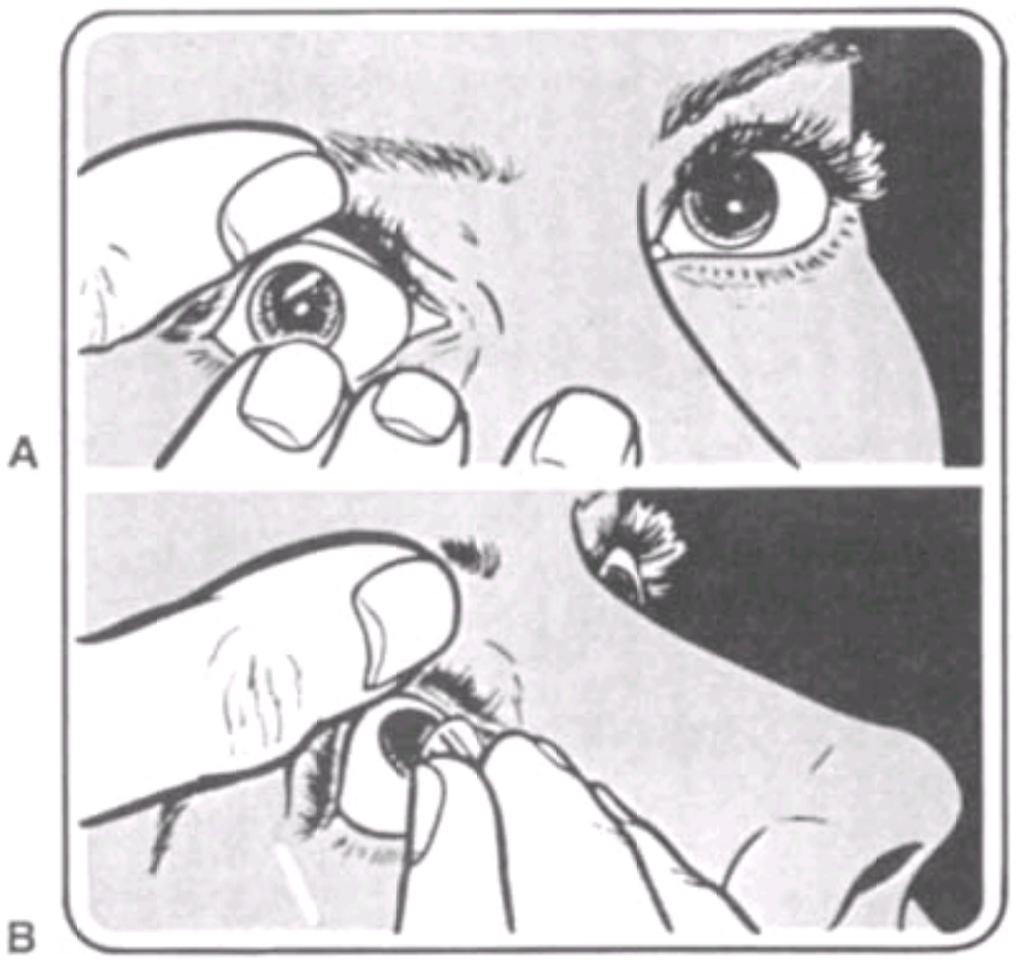


Figure 64-33 Hard contact lens embedded in conjunctival tissue of upper lid. *(From Mandell RB: Contact Lens Practice, 3rd ed. Springfield, IL, Charles C Thomas, 1981. Reproduced by permission.)*

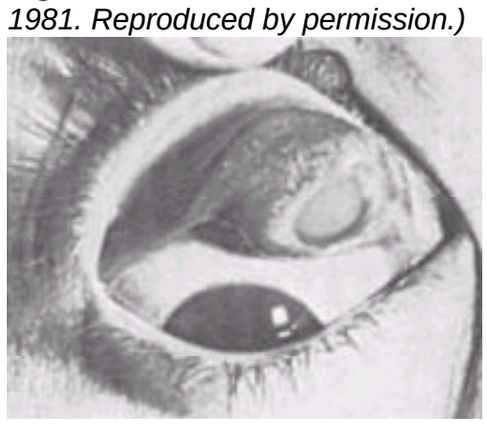


Figure 64-34 Principle of tonometry. At equilibrium: $P_t = F/A$. *(From Draeger J, Jessen K: Tonometry and tonography. In Bellows JG (ed): Glaucoma: Contemporary International Concepts. New York, Masson Publishing USA, 1979. Reproduced by permission.)*

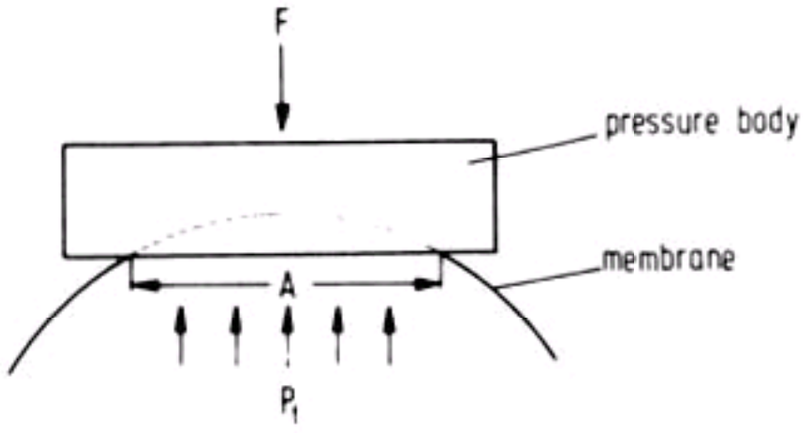


Figure 64-35 Principle of impression tonometry. In reality, P_t is increased slightly by the weight of the instrument. *(From Draeger J, Jessen K: Tonometry and tonography. In Bellows JG (ed): Glaucoma: Contemporary International Concepts. New York, Masson Publishing USA, 1979. Reproduced by permission.)*

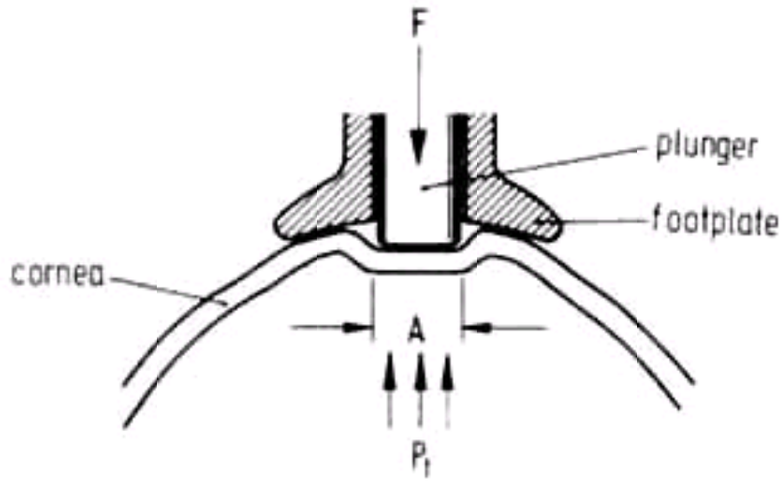


Figure 64-36 Principle of applanation tonometry. The effect of surface tension counters the pressure rise produced by application of the instrument. *(From Draeger J, Jessen K: Tonometry and tonography. In Bellows JG (ed): Glaucoma: Contemporary International Concepts. New York, Masson Publishing USA, 1979. Reproduced by permission.)*

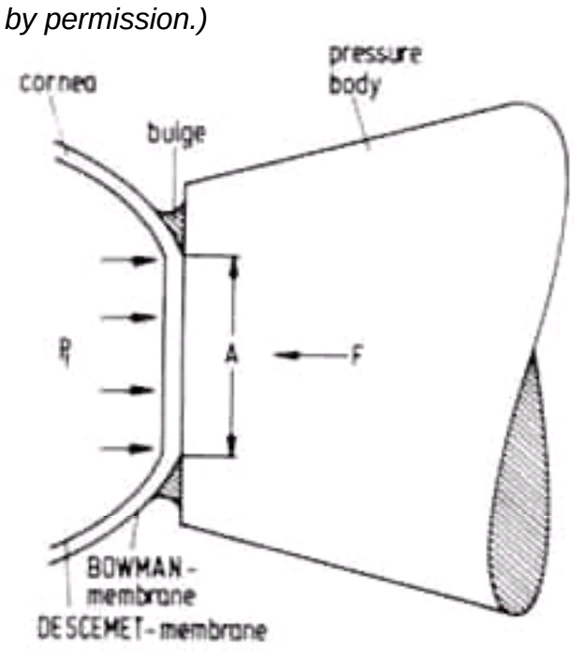


Figure 64-37 The relatively unskilled examiner can detect very high intraocular pressure of acute angle closure glaucoma with tactile tonometry. The examiner rests both hands upon the patient's forehead and alternately applies just enough digital pressure on the globe to indent it slightly with one index finger while feeling the compliance of the globe with the other.

Figure 64-38 One technique of lid separation and Schiotz tonometer placement. Lid separation pressure is applied to the bony orbital rims. An assistant may separate the lids while the operator concentrates on proper placement of the tonometer. The tonometer is held vertically during use, and the clinician's hand is established against the patient's facial bones. After instillation of anesthetic drops, the patient will not experience any pain from this procedure. It is important to have a relaxed patient, since squinting and blepharospasm may interfere with the reading. *(From Keeney AH: Ocular Examination, 2nd ed. St Louis, CV Mosby, 1976. Reproduced by permission.)*

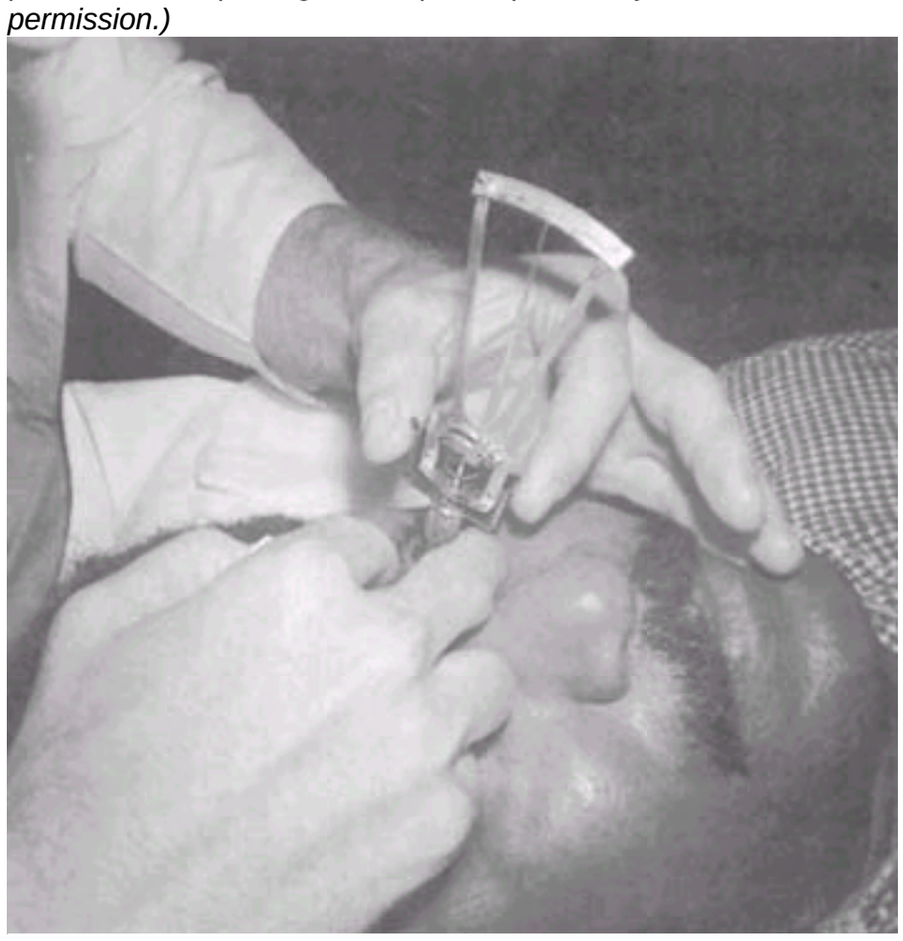


Figure 64-39 Bedside tonometry is easily accomplished with the Tono-Pen XL. The battery-powered device averages four consecutive readings and reports statistical reliability.

Figure 64-40 After topical anesthesia, the Tono-Pen XL is touched lightly and briefly to the cornea with a tapping motion, then withdrawn. Disposable tip covers are used to minimize any cross contamination.

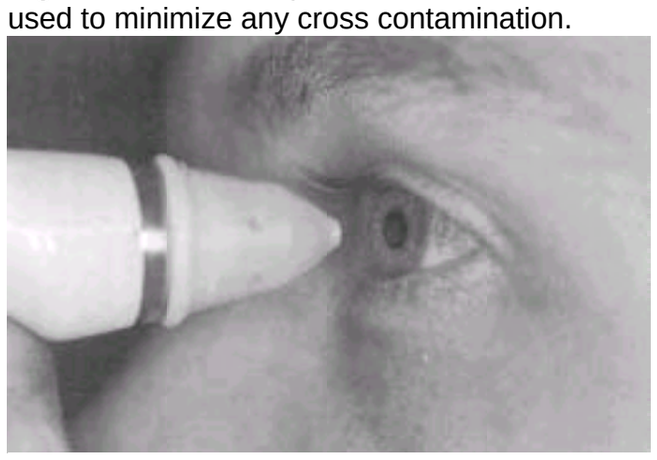


Figure 64-41 Goldmann applanation tonometer with the biprism aligned with the patient's right cornea.

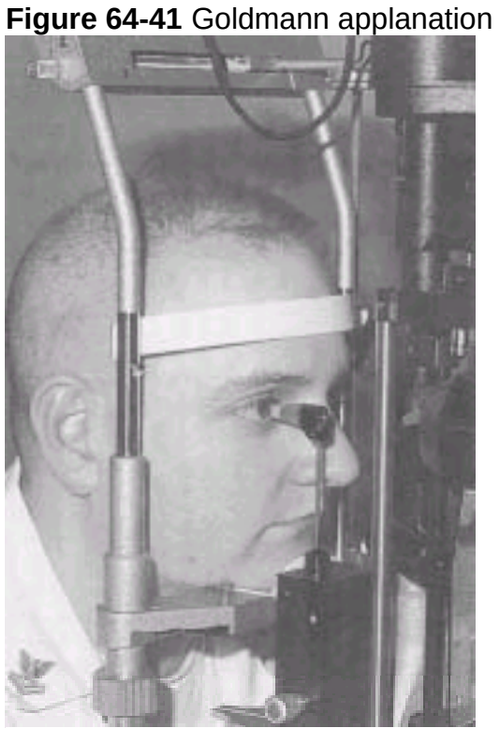


Figure 64-42 Schematic representation of semicircles seen through the contact applanation prism of the Goldmann tonometer. *A,* Semicircles are too wide, suggesting excessive moistening of the prism or cornea. The prism must be withdrawn and dried. *B,* Semicircles are too narrow, suggesting that the lacrimal fluid has dried out, as during a prolonged measurement. The prism must be withdrawn so that the patient may blink a few times. The measurement is then repeated. *C,* Semicircles are of appropriate width, and their inner borders just touch. Cardiac pulsations transmitted through the globe cause rhythmic or pulsating movement of the semicircles over each other through a small amplitude. *D,* Semicircles are slightly separated, indicating applied pressure below that of the eye. The measuring drum must be turned to increase applanation pressure until the end point is reached. *(From Keeney AH: Ocular Examination, 2nd ed. St Louis, CV Mosby, 1976. Reproduced by permission.)*

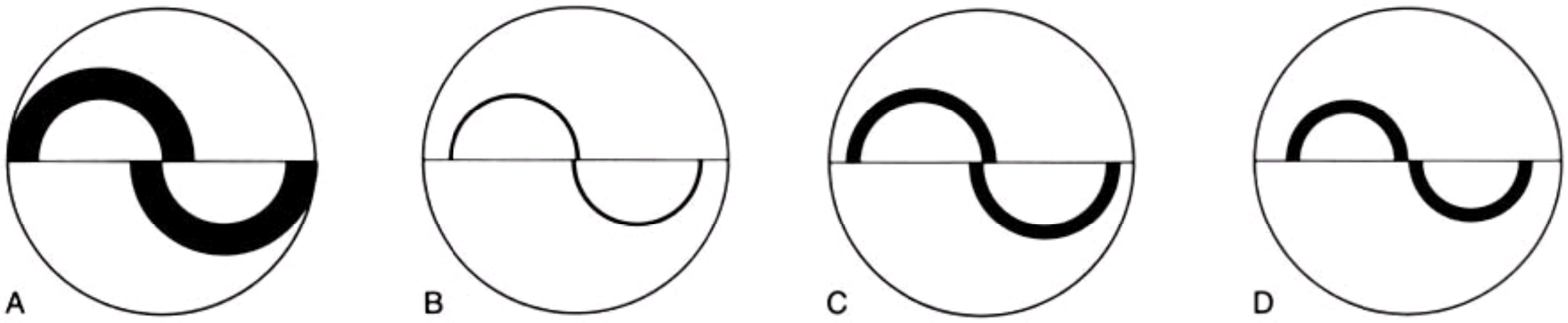


Figure 64-43 Appearance of the semicircles in applanation tonometry. Rotate the pressure knob to align the innermost concave margins of the two semicircles. IOP = intraocular pressure. (1) Pressure on the tonometer head is too high—rotate the knob to decrease reading. (2) Pressure on the tonometer head is too low—rotate the knob to increase reading. (3) Pressure on the tonometer head is equal to IOP—the dial reading equals IOP. *(From Knoop K, Trott A: Ophthalmologic procedures in the emergency department—Part III: Slit lamp use and foreign bodies. Acad Emerg Med 2:224, 1995. Reproduced by permission.)*

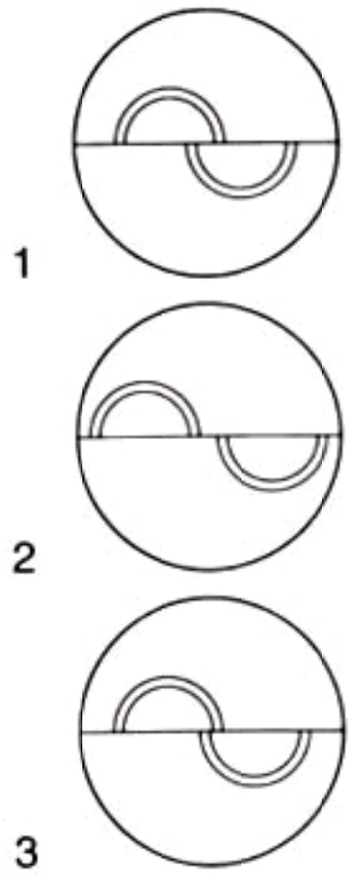
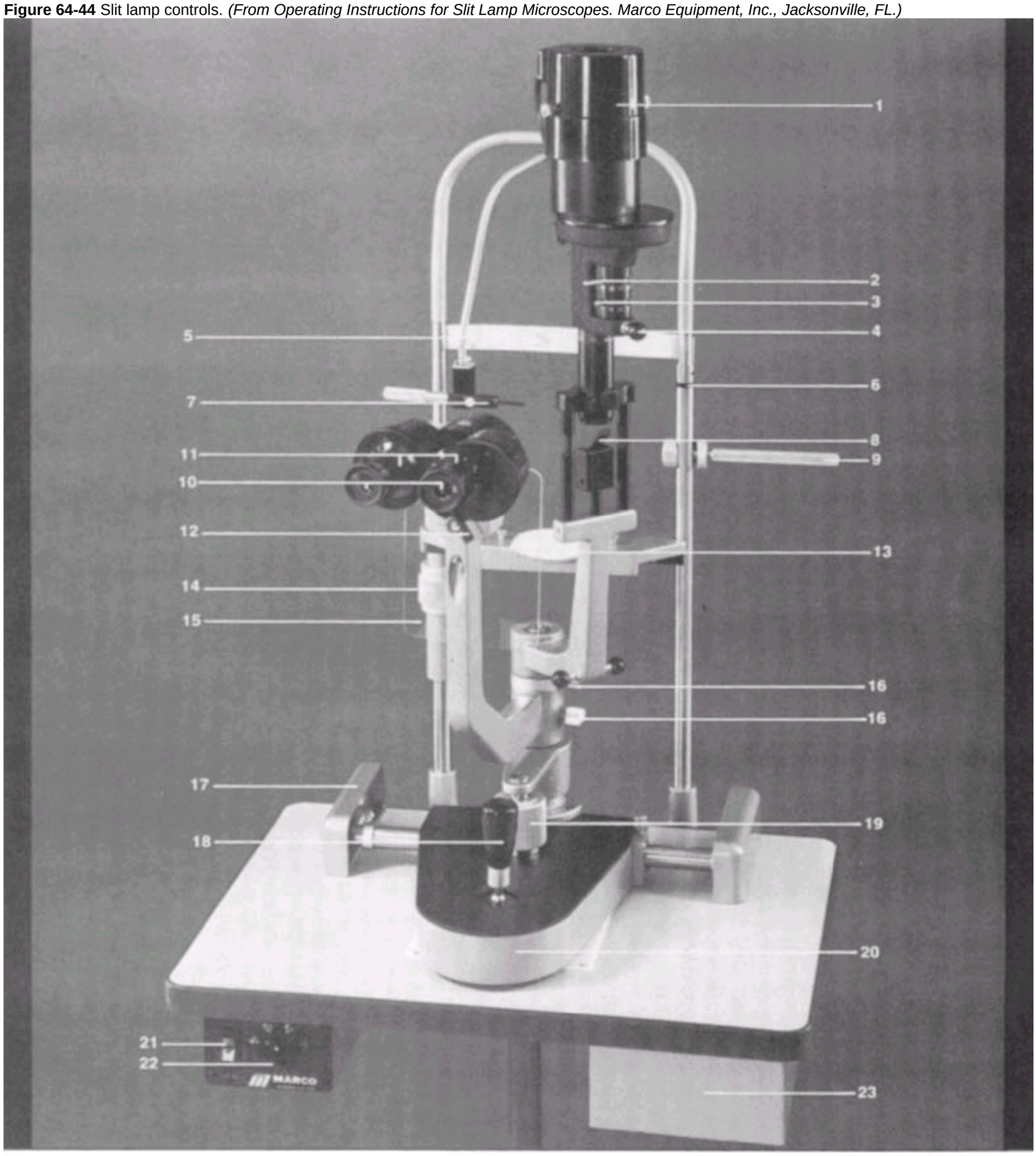


Figure 64-44 Slit lamp controls. *(From Operating Instructions for Slit Lamp Microscopes. Marco Equipment, Inc., Jacksonville, FL.)*



MARCO I PRIMARY CARE SLIT LAMP

NOMENCLATURE

1. Cover for Lamp Bulb
2. Slit Width Controls (Red-Free Filter)
3. Slit Height Control (Cobalt Blue Filter)
4. Control of the Rotation of Slit
5. Headrest
6. Eye Level Marker
7. Fixation Lighthead
8. Mirror
9. Examiner's Handrest
10. Eyepieces
11. Knurled Rings for Refractive Error Adjustment
12. High-Low Magnification Lever
13. Patient's Chinrest
14. Headrest Elevation Control
15. Breath Shield
16. Fixing Screws for Arm
17. Rail Covers
18. Joystick
19. Elevation Control
20. Slit Lamp Base
21. On-Off Switch
22. Intensity Control
23. Accessory Storage Drawer

Figure 64-45 Slit lamp photograph of a normal right eye under low power. The curved slit of light on the left is reflected off the cornea while the slit on the right is reflected off the iris. The depth of the anterior chamber can easily be appreciated under this low magnification setup. *(Courtesy of D. Price.)*

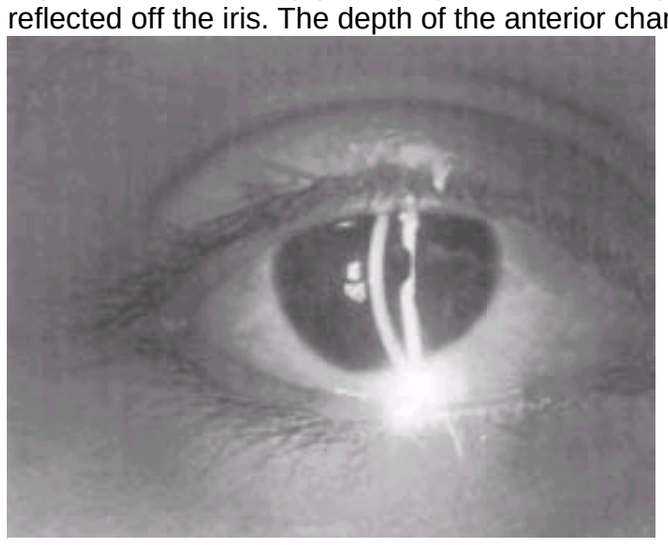


Figure 64-46 Appearance of the left eye during anterior chamber examination under low power: *a,* corneal epithelium; *b,* corneal stroma; *c,* corneal endothelium; *d,* anterior chamber (potential location of cells or flare); *e,* iris; *f,* lens reflection. The slit of light shines in the temporal to nasal direction at 45° to the anterior surface of the cornea. The depth of the cornea and anterior chamber examination are best done under high power in a dark room.

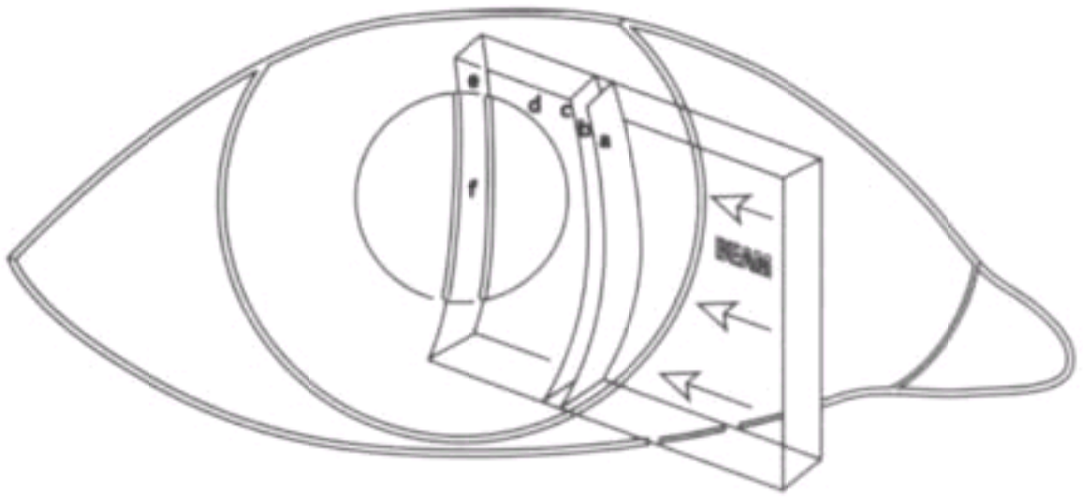


Figure 64-47 Digital globe massage is performed by applying firm steady pressure on the globe with the examiner's thumb for approximately 5 seconds, followed by abruptly releasing the pressure for 5 to 10 seconds. The process is repeated for up to 20 minutes or until improvement of vision is observed.

Figure 64-48 Anterior chamber paracentesis. After topical and subconjunctival anesthesia (see text), a 30-ga needle is directed obliquely from the 4:30 or 7:30 o'clock position toward the 6 o'clock position to avoid the lens. An assistant stabilizes the globe with forceps, grasping the conjunctiva (see text). *Top*, Anteroposterior projection. *Bottom*, Tangential projection. *(From Knoop K, Trott A: Ophthalmologic procedures in the emergency department: I. Immediate sight-saving procedures. Acad Emerg Med 1:408, 1994.)*

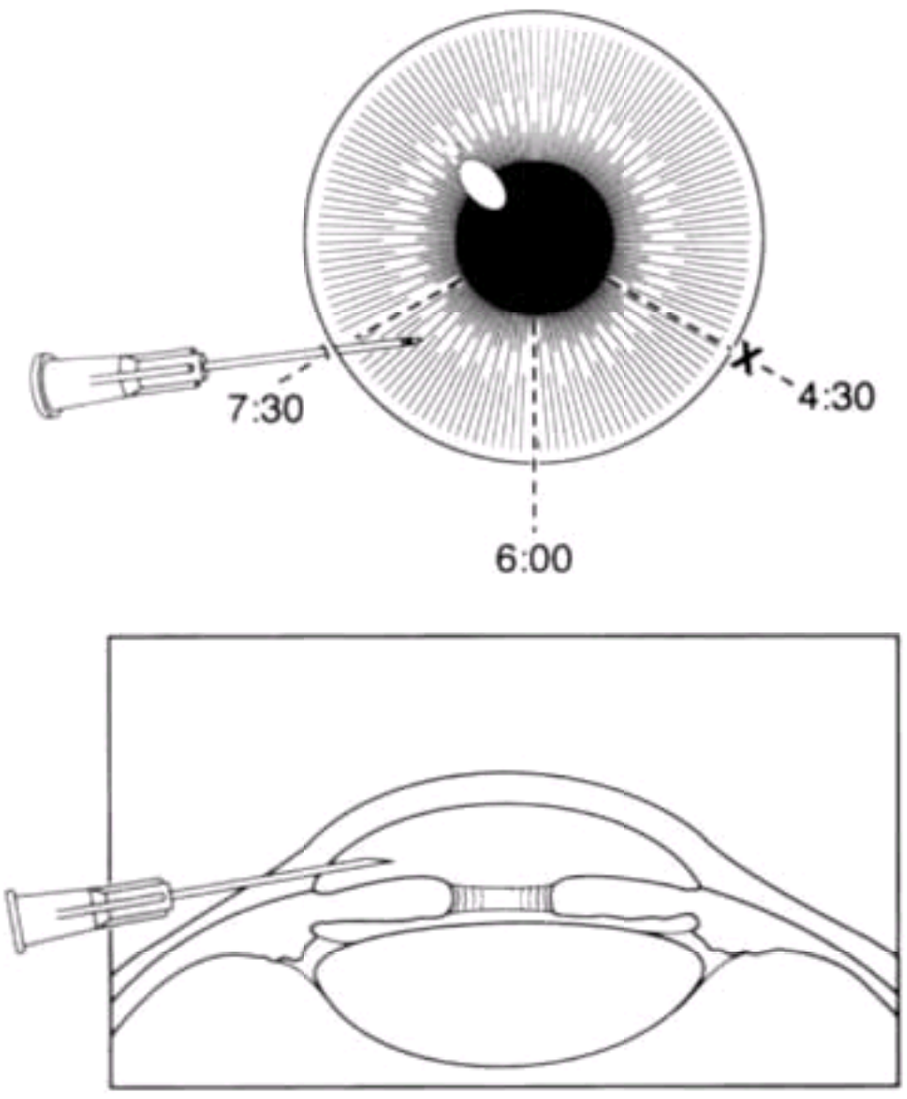
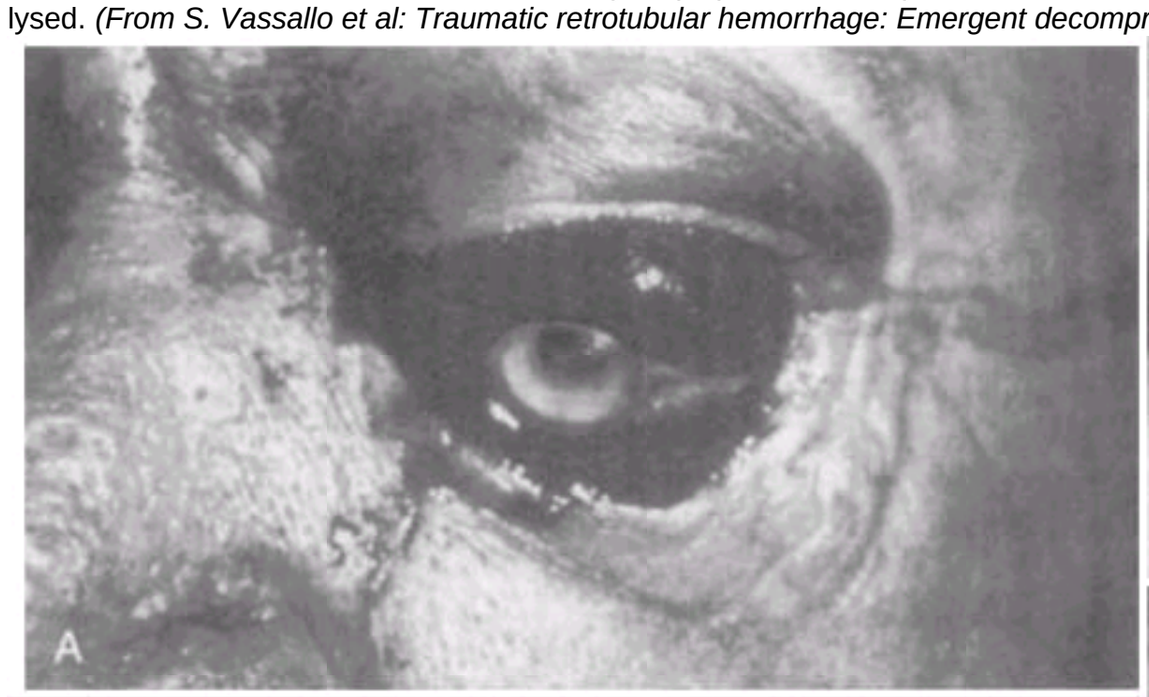
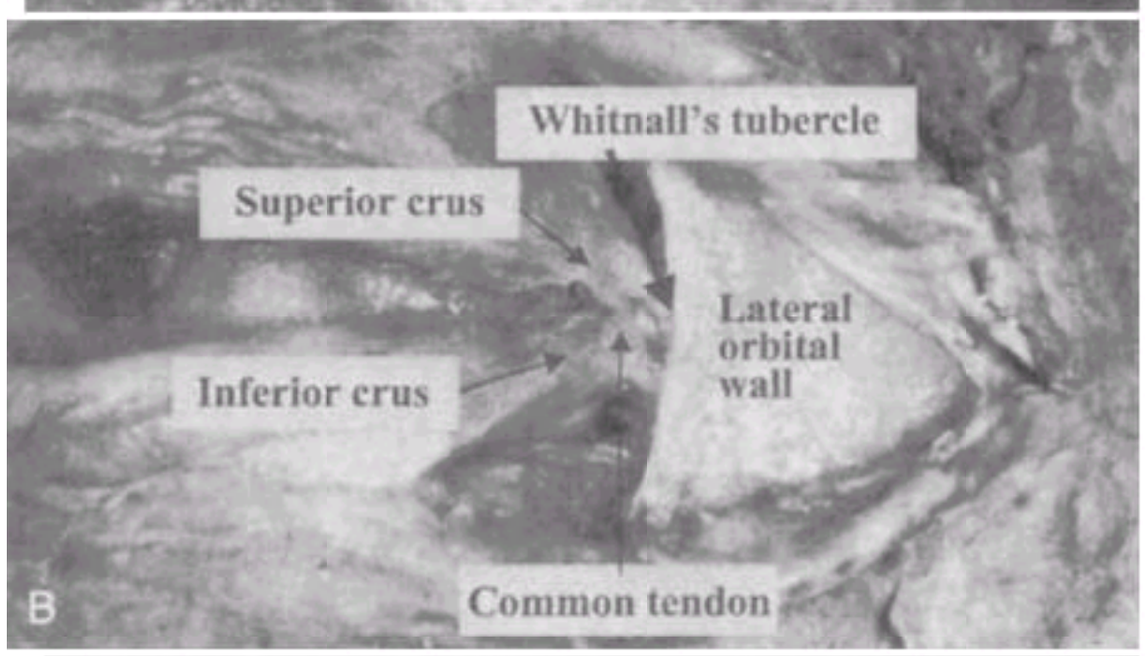
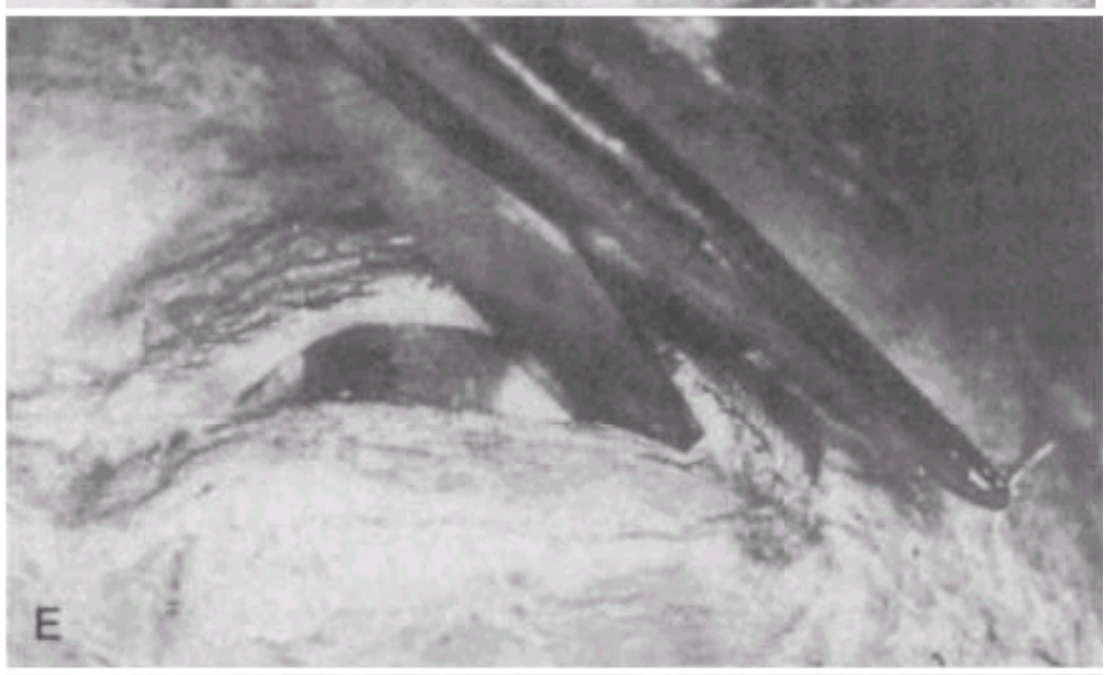


Figure 64-49 *A,* Severe proptosis secondary to acute traumatic retrobulbar hemorrhage. *B,* Anatomy of orbital structures demonstrating the inferior and superior crura of the lateral canthal tendon beneath the lateral canthus. The crura join and as a common tendon are attached to the inner aspect of the lateral orbital wall, forming Whitnall's tubercle. The lateral canthus, formed by the upper and lower eyelid, has been removed. *C, D,* A clamp crushes the lateral canthus to reduce bleeding when it is incised. The canthotomy allows the inferior crus to be exposed and cut to decompress the eyeball. *E,* A 1 centimeter horizontal incision is made in the lateral canthus, through the tissue that was compressed. *F,* The lower lid is pulled down and away from the lateral orbital rim, separating the skin and conjunctiva. If bleeding hinders identification of the inferior crus, it may be palpated. *G,* Only the inferior crus need be lysed initially. If intraocular pressure is not reduced, the superior crus is lysed. *(From S. Vassallo et al: Traumatic retrotubular hemorrhage: Emergent decompression by lateral canthotomy and cantholysis. J Emerg Med 22:21, 2002.)*







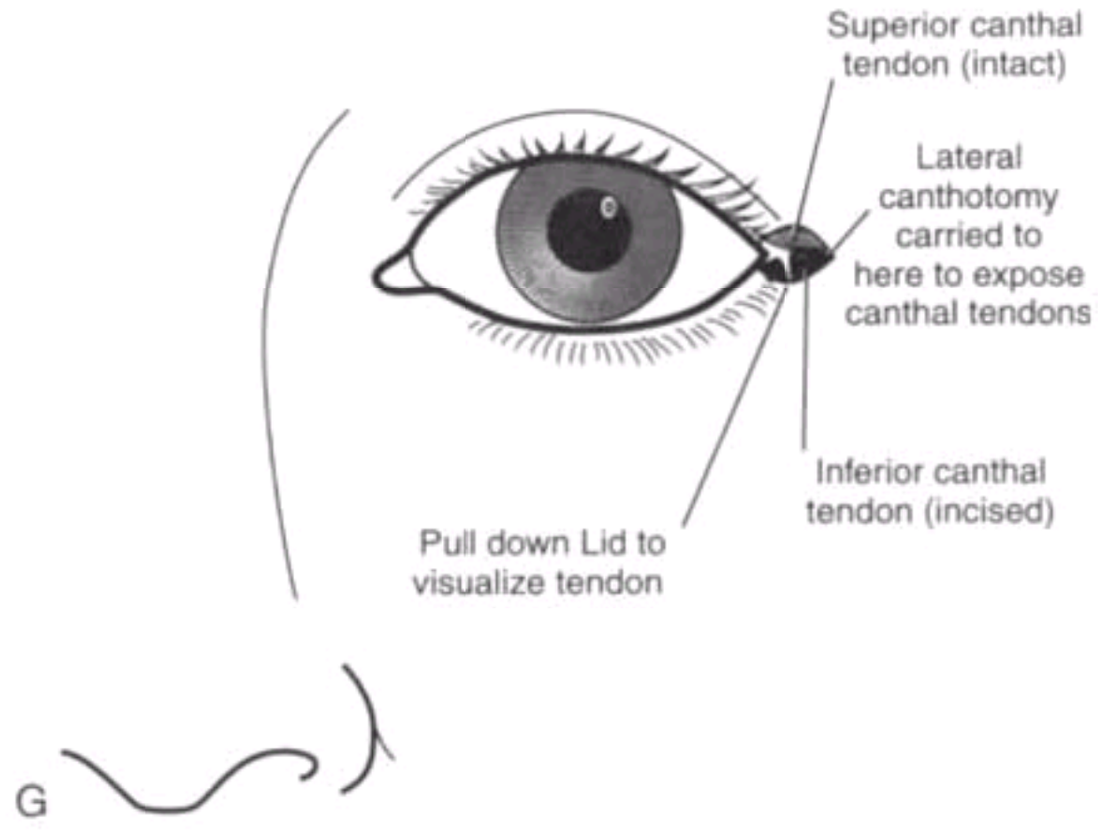


Figure 64-50 Appearance of bilateral luxated globe; superior (*top)* and lateral (*bottom*) projections. *(From Love JN, Bertram-Love JE: Luxation of the globe. Am J Emerg Med 11:61, 1993; original photographs courtesy of WR Green, MD, Wilmer Eye Institute, Baltimore.)*

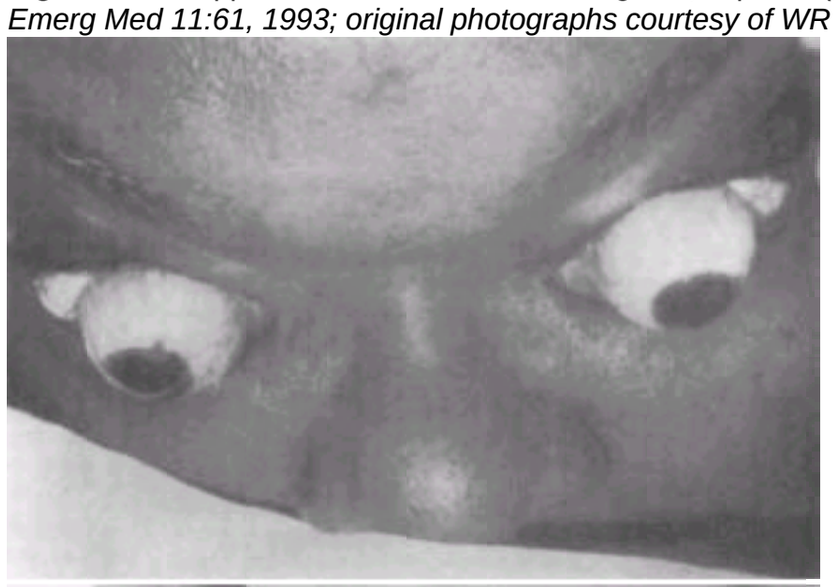


Figure 65-1 Anatomy of the oropharynx. Sagittal section of the neck. Also depicted is the use of the nasopharyngeal scope through the nasopharynx and the oropharynx.

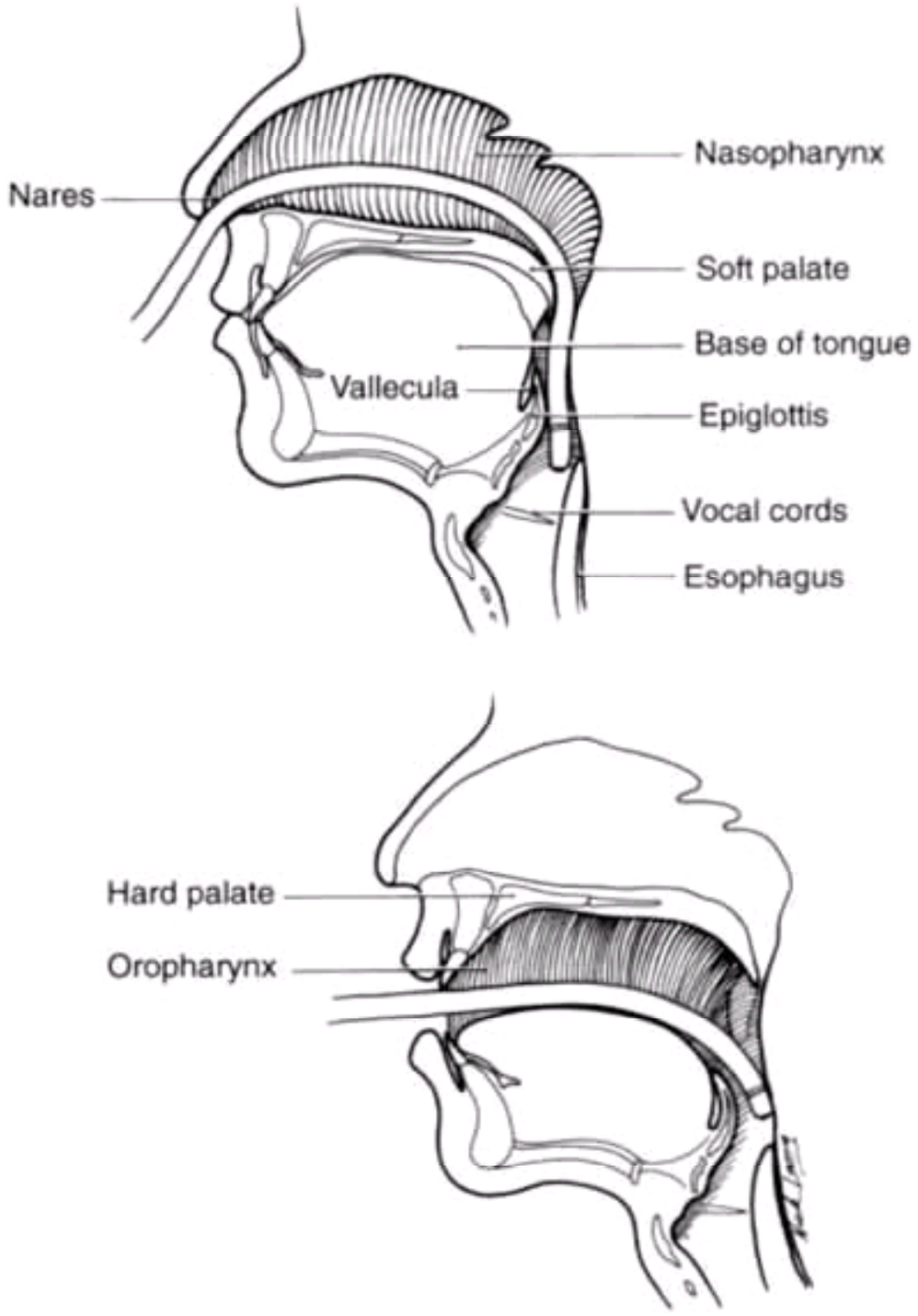


Figure 65-2 Indirect mirror evaluation of oropharynx. Grasp the patient's tongue between the thumb and first finger, using a gauze pad to provide traction. Elevate the upper lip with the middle finger. Advance the warmed laryngeal mirror into the posterior oropharynx, taking care not to stimulate the posterior tongue or pharynx. Remember that the structures in the mirror will be reversed. Always use universal precautions.

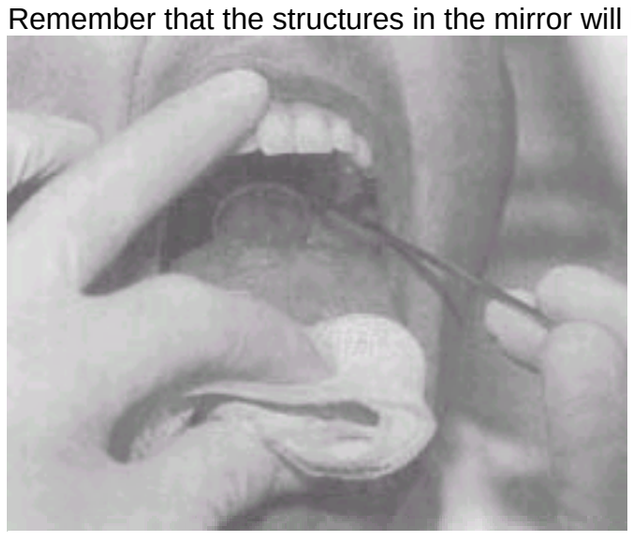


Figure 65-3 View of the larynx from above. The true and false vocal cords are sketched, with the arytenoid eminences behind them on each side. The epiglottis, piriform fossae, and valleculae are also labeled.

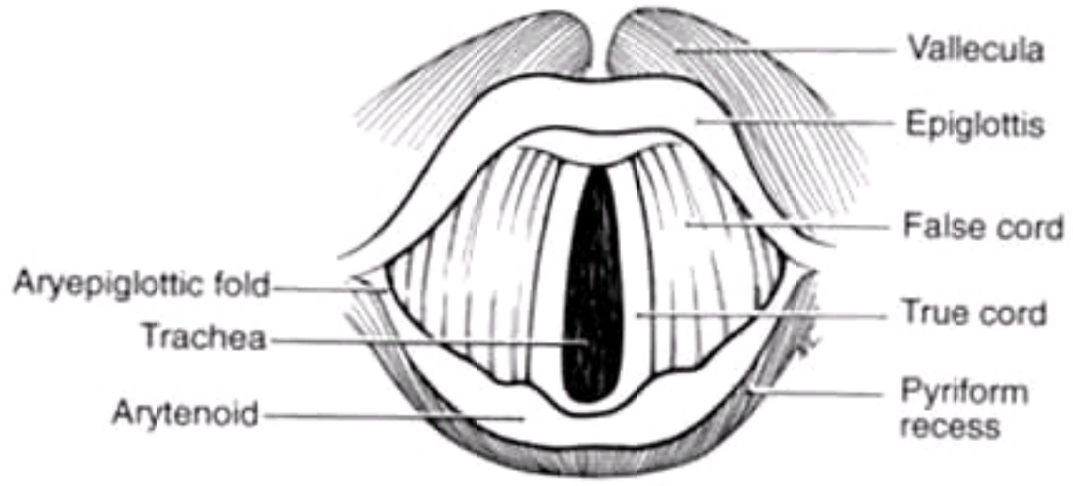


Figure 65-4 Fiberoptic scope with light source.

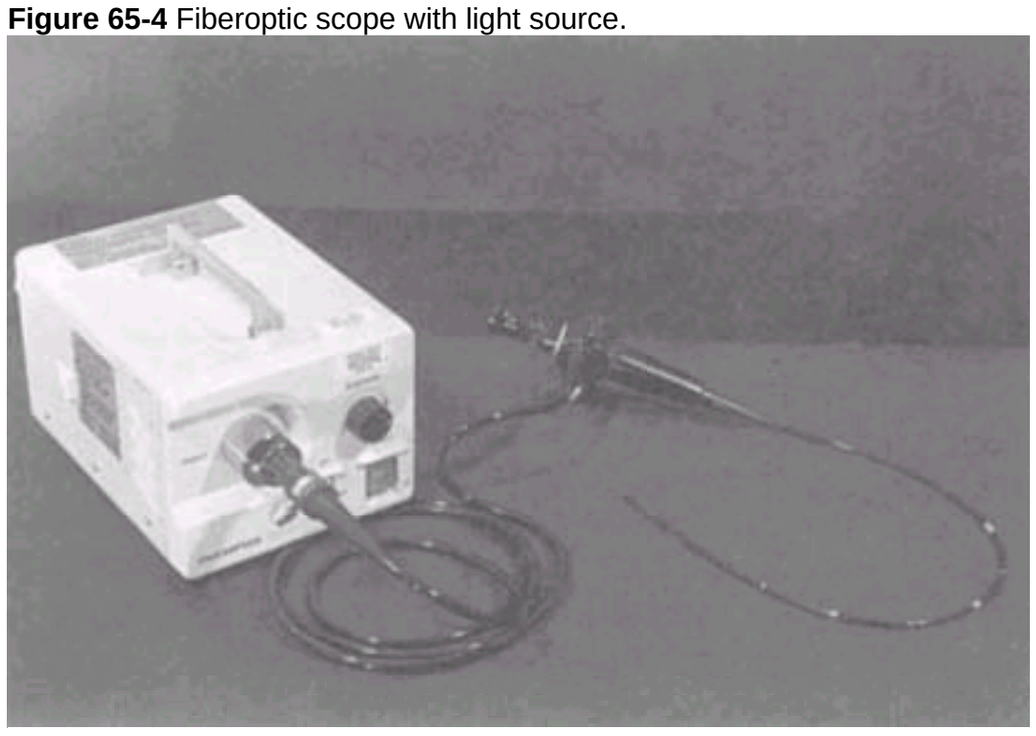


Figure 65-5 Fiberoptic nasopharyngoscope in use. Prepare the patient's throat and nares with topical anesthetic. Topical vasoconstrictors may also be used in the nares. The use of a nasal trumpet is optional. Advance the scope slowly into the naris with the hand stabilized on the patient's nose; guide the scope using the thumb and index finger. Visualize the passage of the scope through the naris into the posterior nasopharynx. Always use universal precautions.

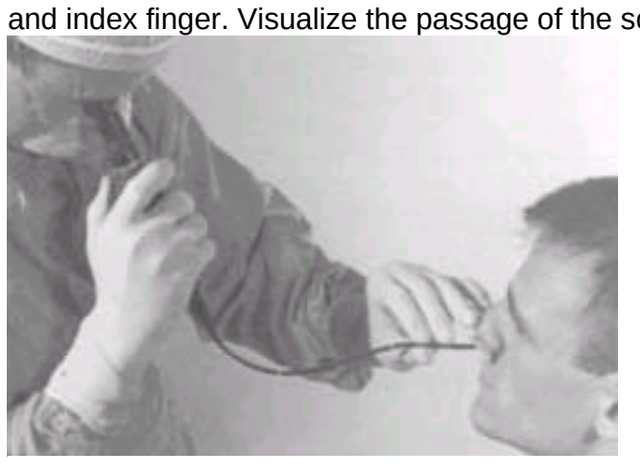


Figure 65-6 Anatomy of a peritonsillar abscess. The palatine tonsil and peritonsillar space are identified on the patient's right. A peritonsillar abscess is shown on the patient's left. Note that the abscess can extend medially, displacing the uvula. The carotid artery and jugular vein are posterior and lateral to the abscess. Avoid lateral angulation of the aspirating needle and use a needle guard to prevent injury.

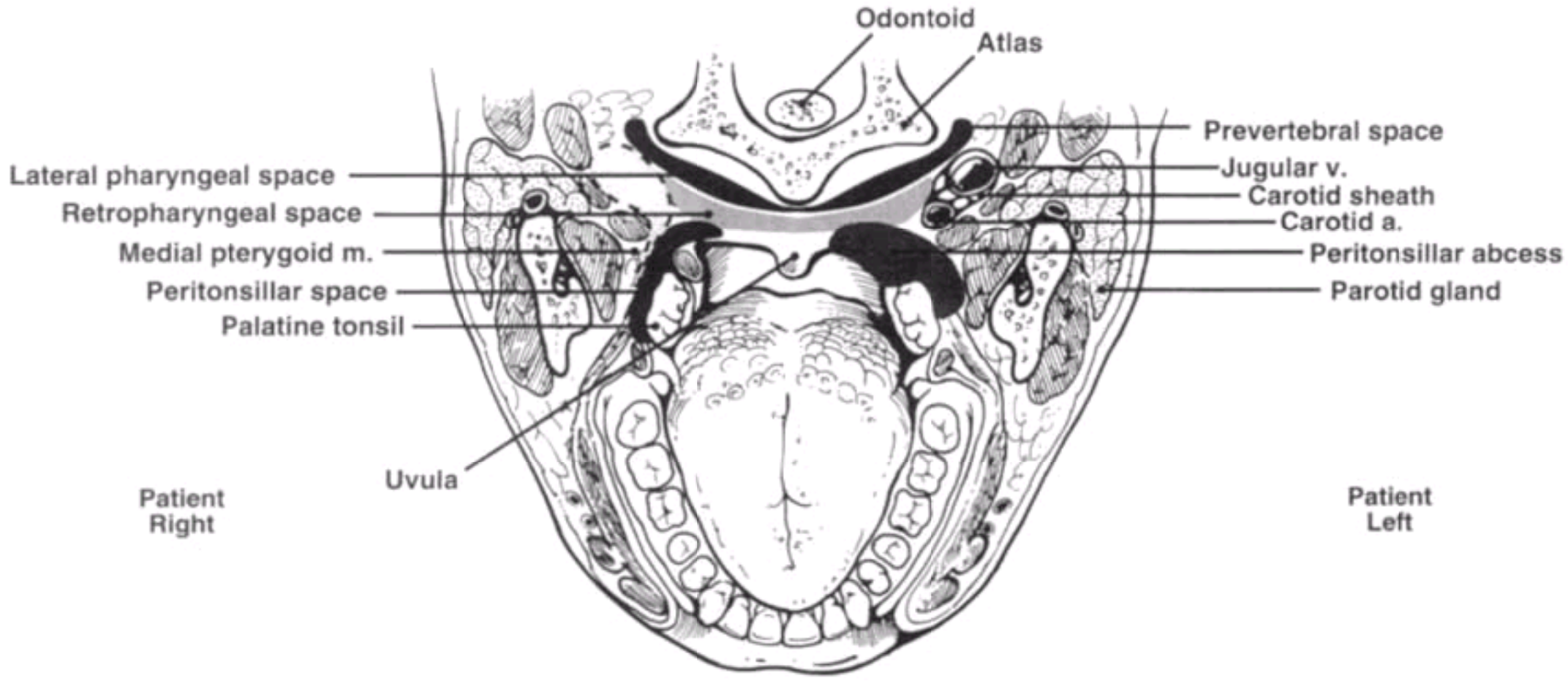


Figure 65-7 Clinical symptoms and visual inspection may not be sufficient to differentiate peritonsillar abscess from cellulitis. The clinician's gloved index finger is used to palpate the peritonsillar area to search for fluctuance and localized swelling.

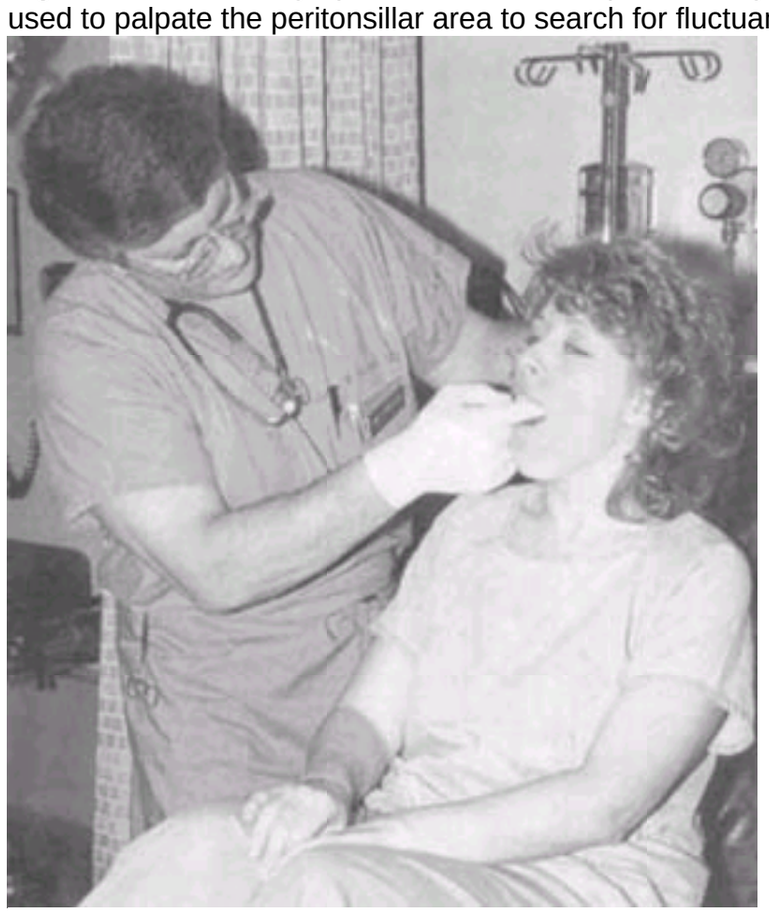


Figure 65-8 Needle aspiration of a peritonsillar abscess. The patient sits upright with the head supported by the back of the stretcher (or a dental chair head rest). A head lamp provides light and an assistant retracts the cheek laterally to maximize visibility.

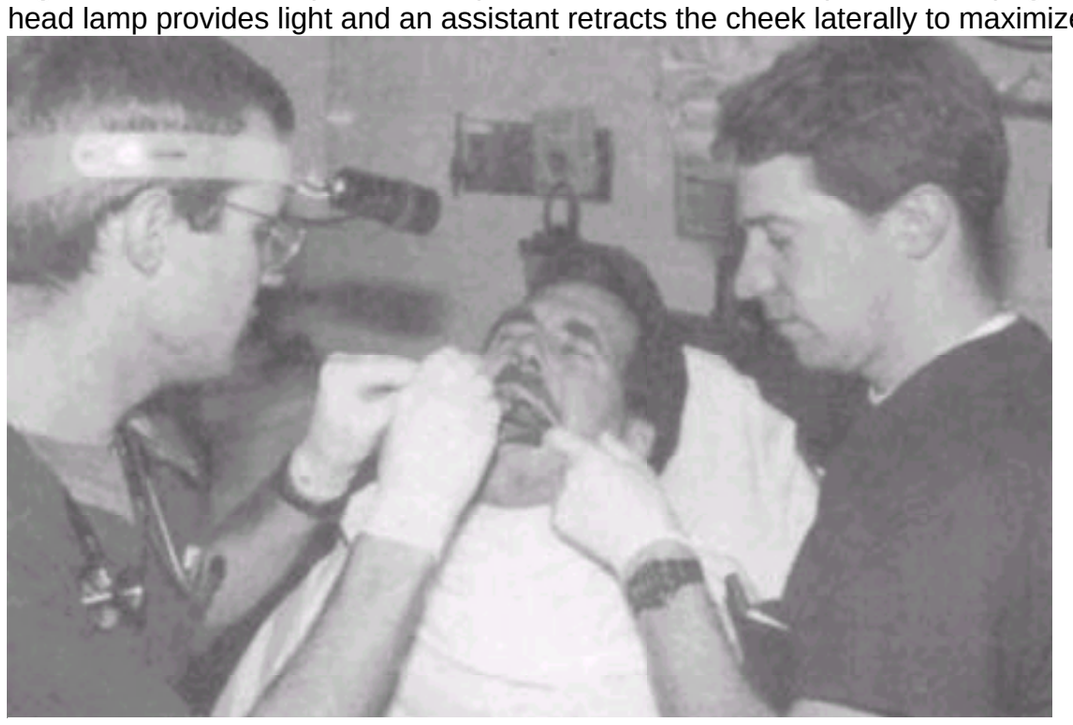


Figure 65-9 *A*, As a safeguard to prevent deep penetration of a needle used to drain a peritonsillar abscess, select a long 18- to 20-gauge needle. Remove the plastic needle guard and cut off the distal 1 cm. *B*, Replace the guard on the needle and tape it to the hub.

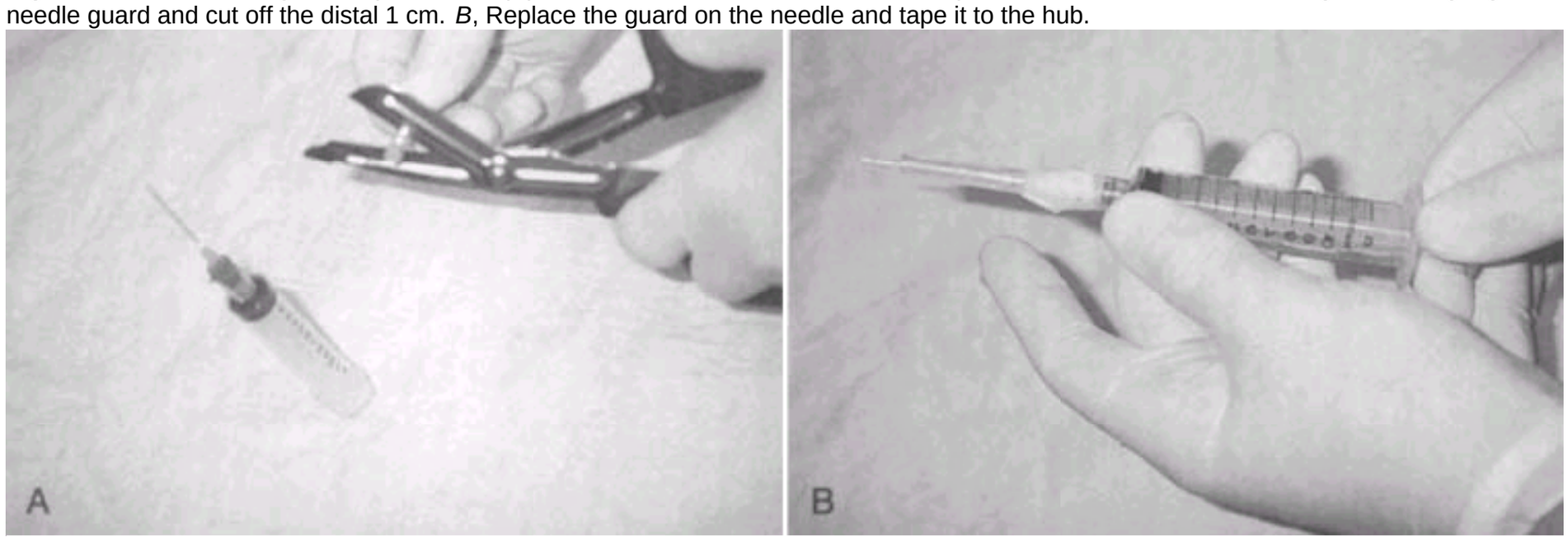


Figure 65-10 *A*, Needle aspiration of a peritonsillar abscess. Anesthetize the posterior pharynx with topical lidocaine spray. Blanch the mucosa with lidocaine/epinephrine with a 27-gauge (ga) needle on a long syringe (to allow visualization of the site) in the area to be aspirated. Advance an 18- or 20-ga needle with needle guard into the area of greatest fluctuance, usually the superior pole. Aspirate as you advance the needle. Advance the needle in the sagittal plane. Do *not* direct the needle laterally toward the carotid artery or jugular vein. *B,* The superior pole is aspirated first, but the middle and inferior poles should be aspirated if pus is not obtained initially. Note that the tonsil itself is not aspirated. The *peritonsillar space* contains the abscess.

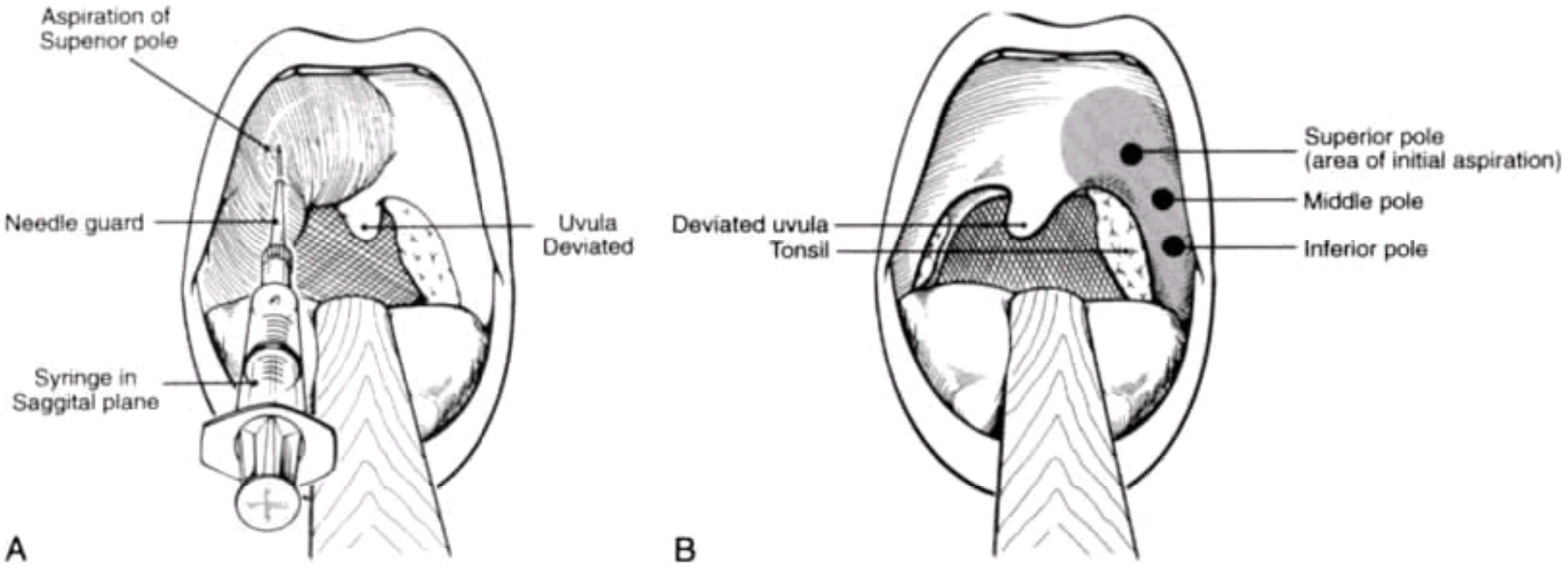


Figure 65-11 Needle aspiration usually yields 2 to 6 mL of thick pus. Greater volumes are unusual. Removing only a small amount will produce a marked reduction in symptoms.

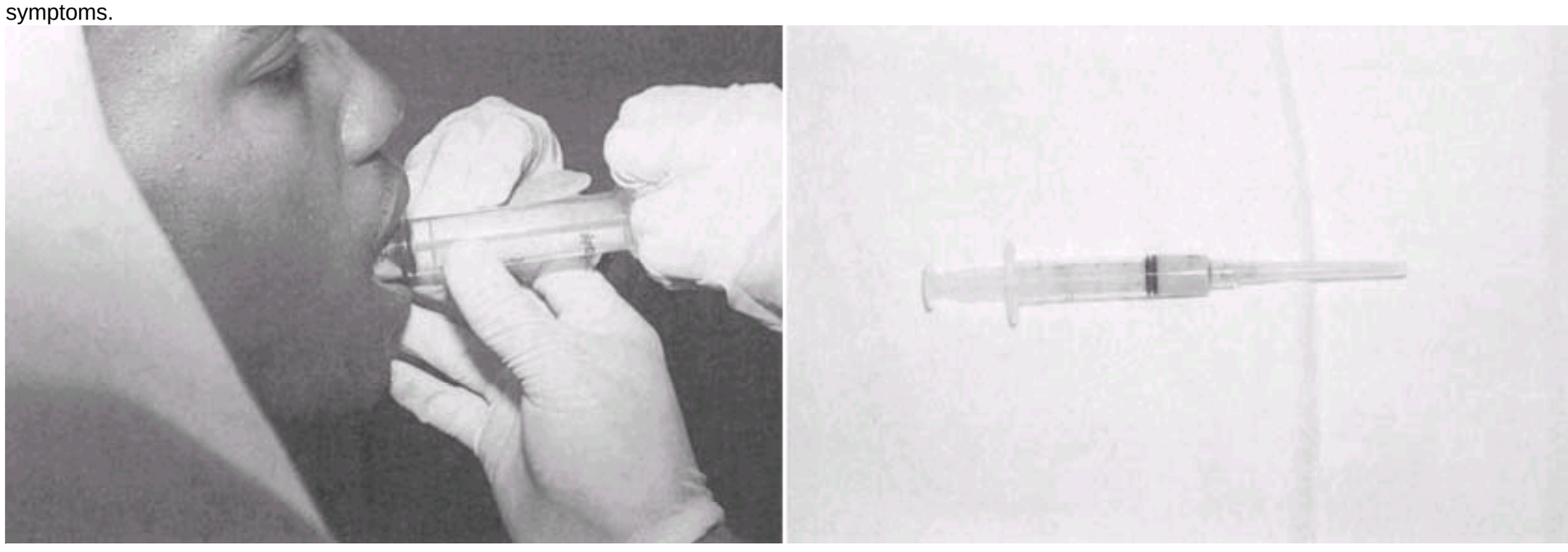


Figure 65-12 Aspiration is often the only procedure required to successfully treat a peritonsillar abscess, but it has a 10% failure rate. In some instances the clinician will opt for incision and drainage of a peritonsillar abscess. This procedure may used initially, or after aspiration if copious pus is aspirated, or if pus continues to drain or reaccumulates. *A,* Normal-appearing oropharynx. *B,* Peritonsillar abscess on the right side of the throat. *C,* Incision of the abscess at the area of greatest fluctuance. Notice that the scalpel is taped to prevent deep penetration. *D,* Loculations are removed by gentle probing with hemostats.

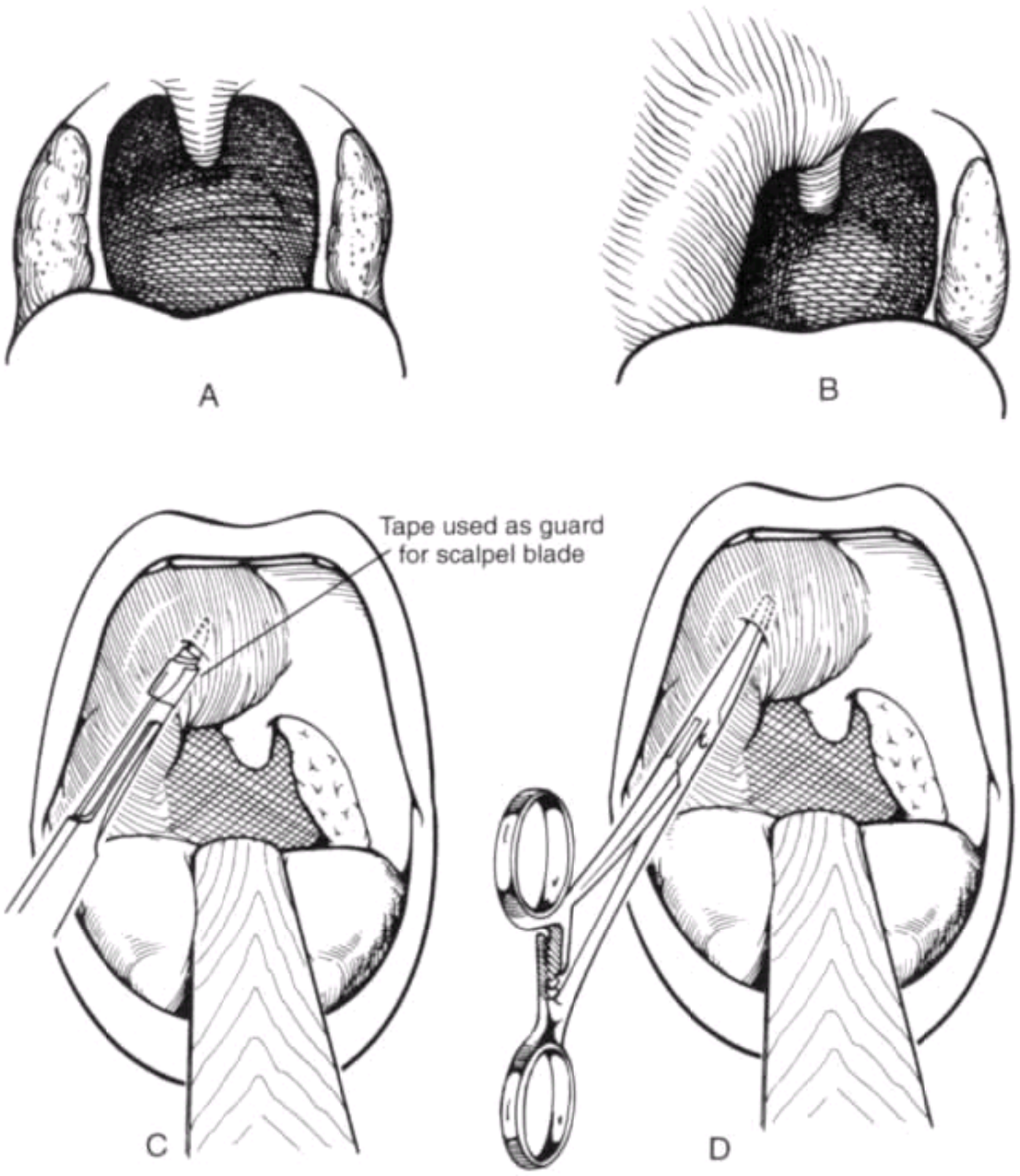


Figure 65-13 *A* and *B,* External anatomy of the ear and innervation of the auricle.

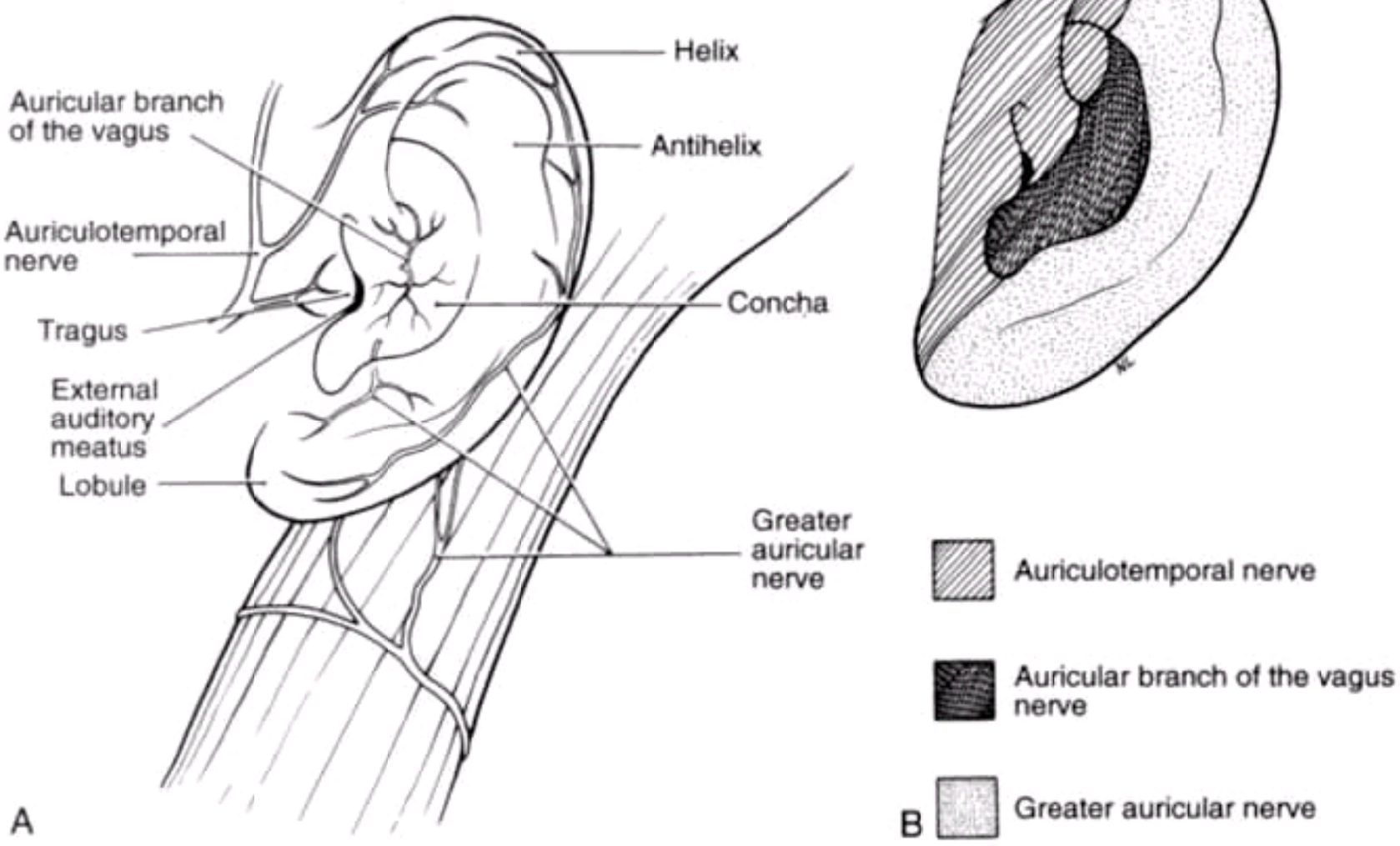


Figure 65-14 Field blocks of the auricle. *A,* One method uses approximately 3 to 4 mL of anesthetic, both in the posterior sulcus and at a point just anterior to the tragus. *B,* Alternative field block technique that deposits 2 to 3 mL of anesthetic for each needle pass. See text for more details.

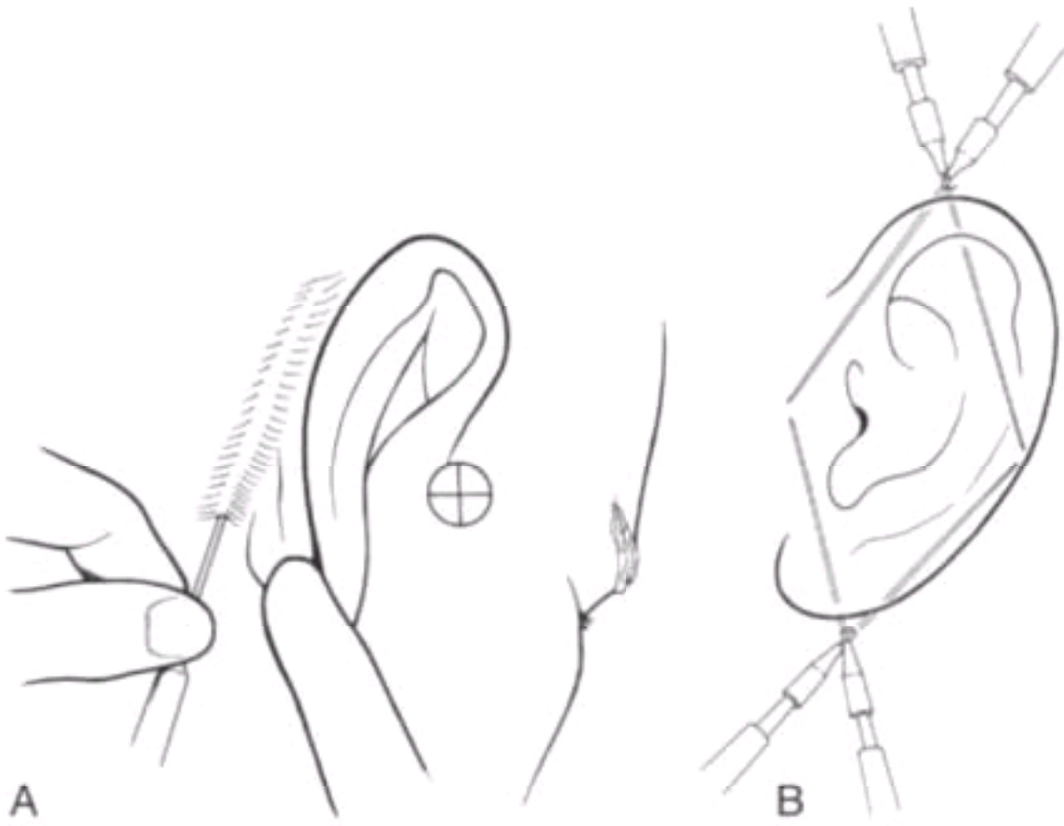


Figure 65-15 Four-quadrant field block anesthesia of the external auditory canal. Local anesthetic is injected subcutaneously in the four quadrants of the lateral portion of the ear canal. The largest speculum that will fit is used to guide the injections. The speculum is withdrawn slightly, tilted toward each of the four quadrants, and the needle is inserted subcutaneously *(x)*. A very small amount of anesthetic (0.25 to 0.50 mL) is injected to produce a slight bulge in the soft tissue. A total of 1.5 to 2.0 mL of anesthetic is usually sufficient to anesthetize the ear canal and permit painless removal of a foreign body.

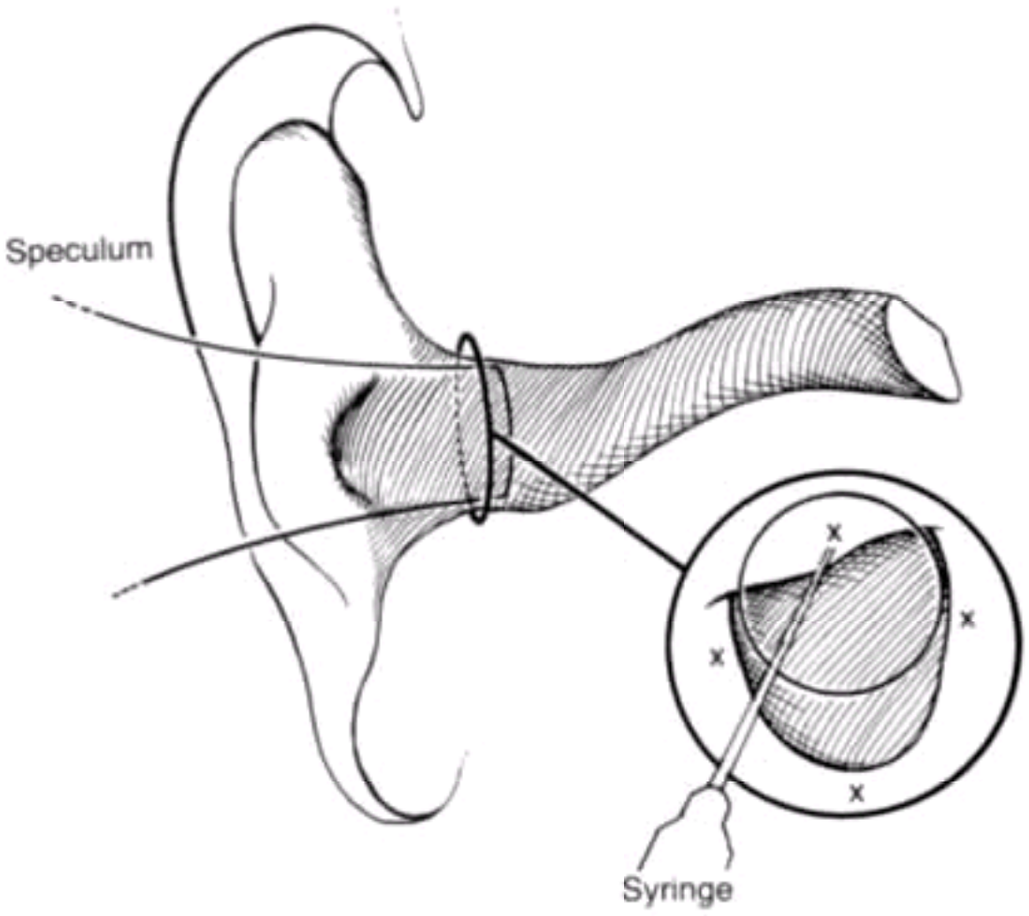


Figure 65-16 Diagram of injection sites for an alternative technique to anesthetize the ear canal and central concha. Each site should be injected with approximately 0.5 mL of 1% lidocaine. Do not inject if external signs of infection are present.

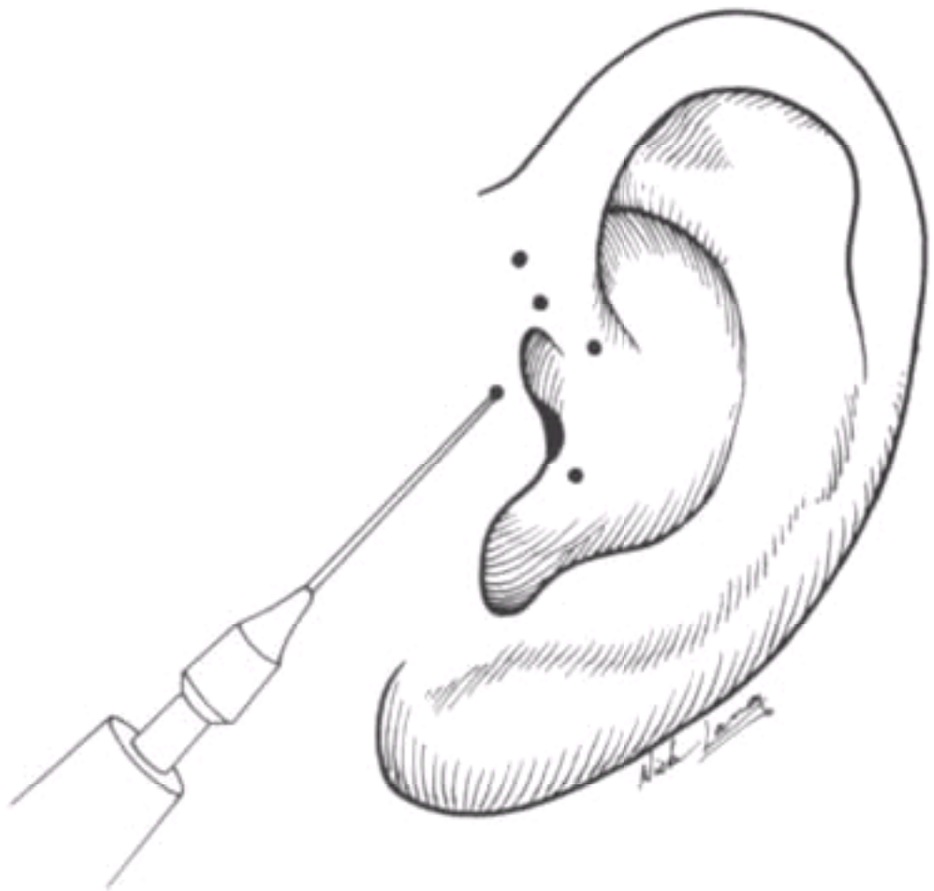


Figure 65-17 Examination of ear canal. The pinna is retracted in a superior and posterior direction to straighten out the ear canal. The scope is held in the other hand and stabilized against the patient's head. This prevents inadvertent injury if the patient moves unexpectedly.

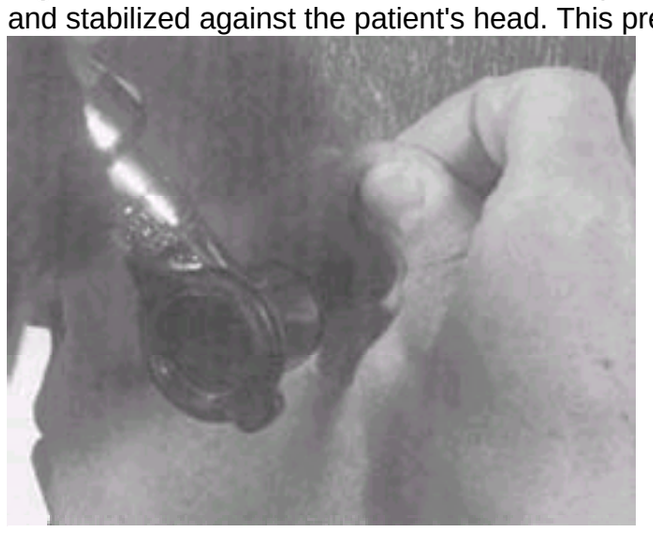


Figure 65-18 Irrigation is a simple, painless, and usually successful way to remove cerumen. After a wax-softening agent has been instilled for 15 to 20 minutes, an assistant applies traction on the ear to straighten the canal, and the plastic tubing of a 19-ga butterfly device (needle and wings removed) is inserted 1 cm into the canal. A basin is held by the patient, and warm water is introduced with a 20-mL syringe. A number of irrigations may be required, and the procedure may be supplemented with careful removal of large cerumen pieces with a curette.

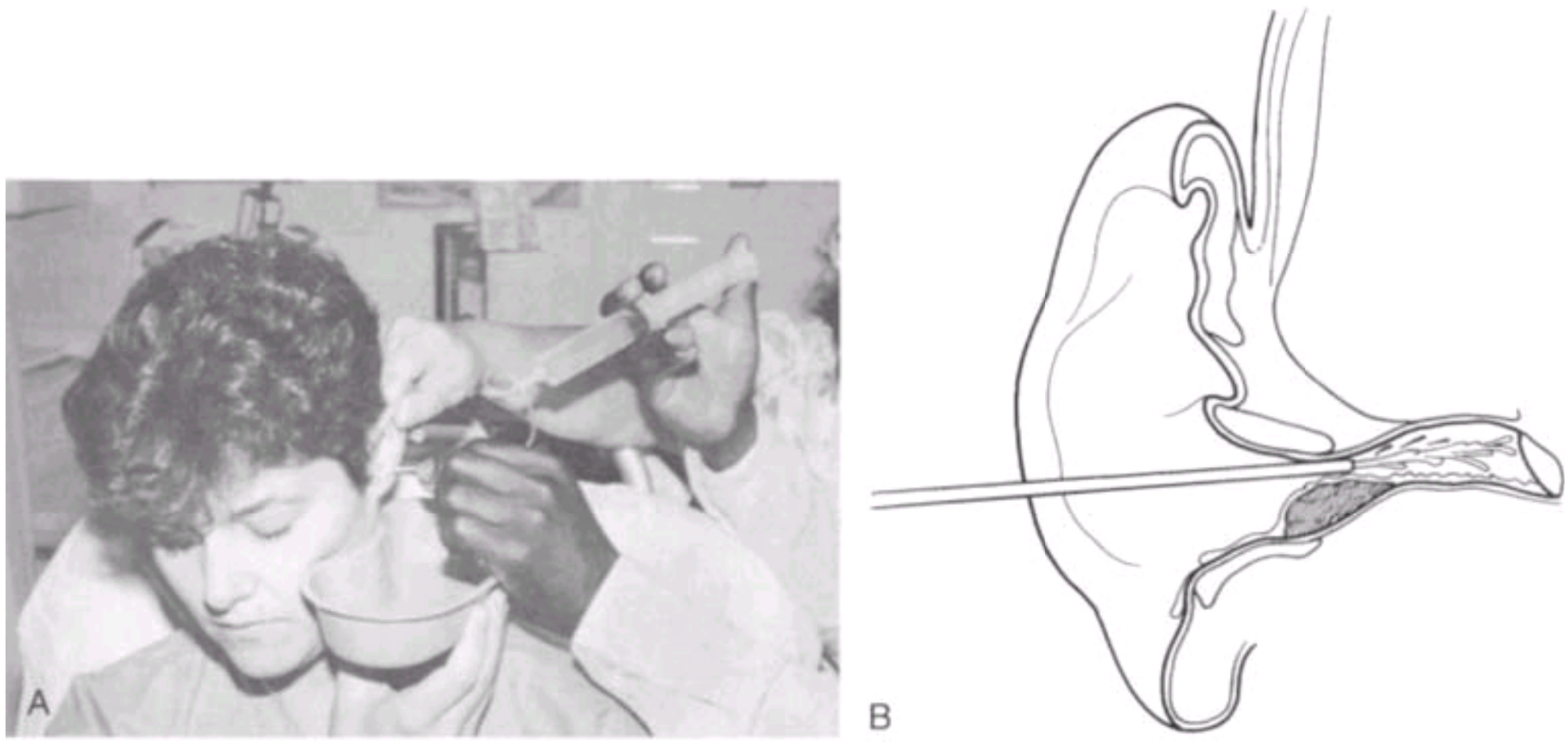


Figure 65-19 The most readily available device for ear irrigation is an 18-gauge flexible catheter attached to a 60-mL syringe. Since multiple irrigation may be required, small syringes are counterproductive.

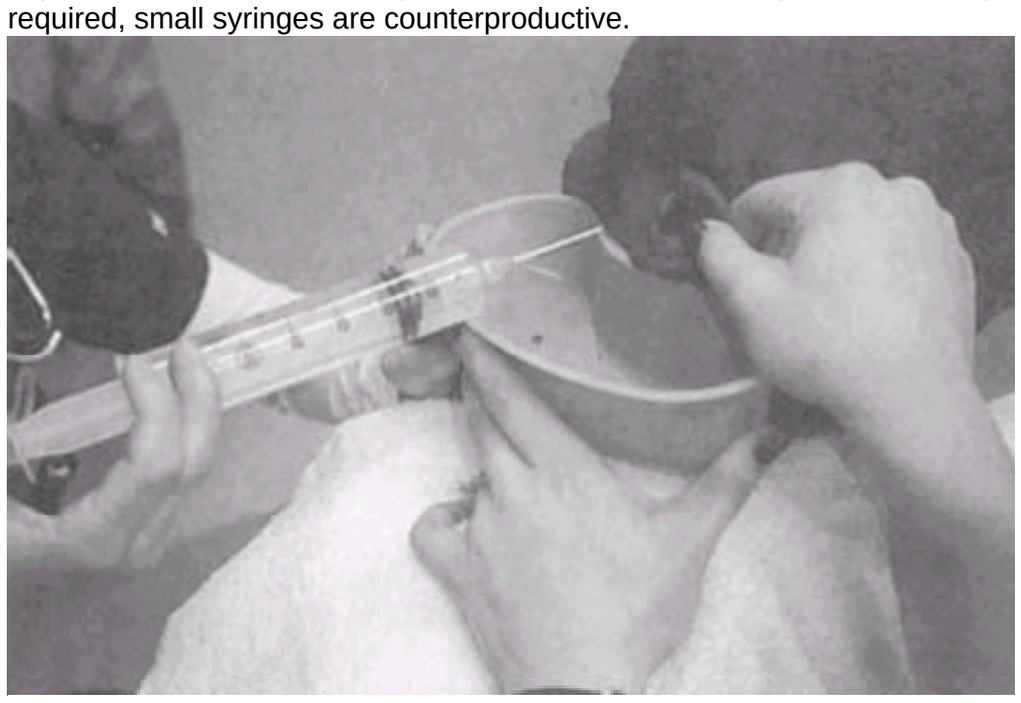


Figure 65-20 This DeVilbiss irrigator uses compressed air to eject the irrigating solution, which should be near body temperature to avoid caloric stimulation of the inner ear. The more conventional metal ear syringe can be equally effective and does not require a source of compressed air to function.

Figure 65-21 Technique for direct visualization and mechanical removal. Use of alligator forceps through a diagnostic otoscope. Note that the magnification device has been slid laterally and that no ear speculum has been attached. *Inset,* Use of ear curettage through operating otoscope. *(From Fritz S, Kelen GD, Sivertson KT: Foreign bodies of the external auditory canal. Emerg Med Clin North Am 5:184, 1987.)*

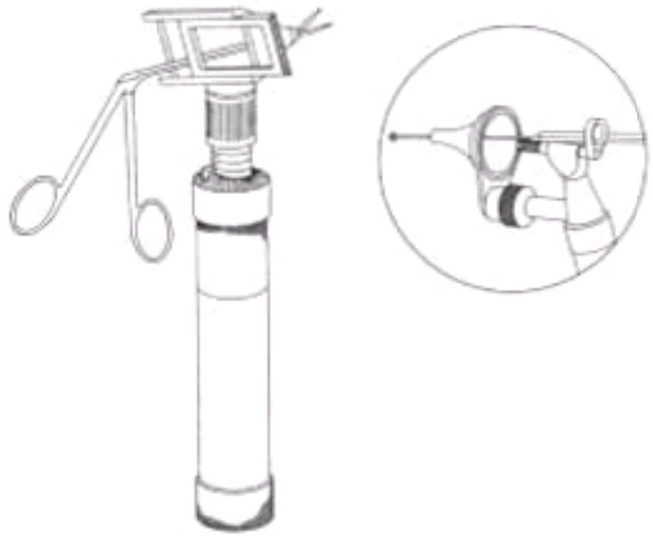


Figure 65-22 Instruments used for foreign body extraction. From left to right: alligator forceps, bayonet forceps, right-angle hook, wire loop, soft-tipped suction, Frazier suction.

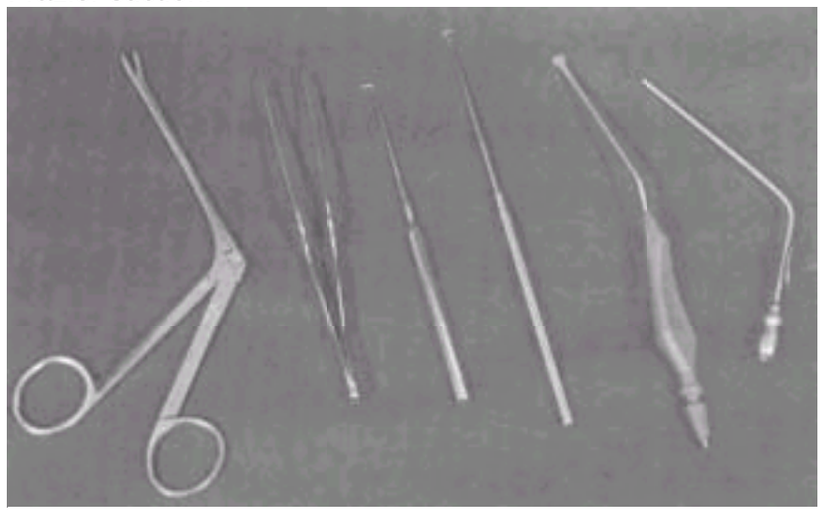


Figure 65-23 Removal of impacted cerumen. Pass the tip of the wire loop beyond the wax and gently tease the wax off the ear canal wall. Extract the wax slowly from the canal. Under direct visualization, avoid contact with the skin of the ear canal to prevent pain and excoriation.

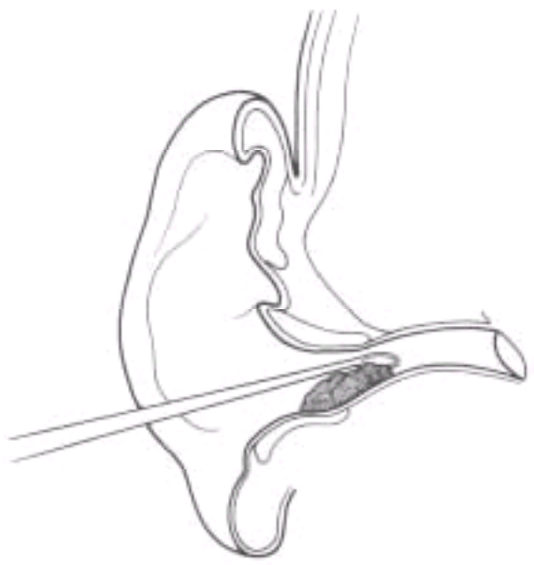


Figure 65-24 Debridement of external otitis. A cotton-tipped applicator is inserted into the canal and debris is gently removed from the canal. Irrigation of the ear may also be helpful if the tympanic membrane is not perforated. *Inset,* the edema has almost closed the canal and will not allow medication to be instilled into the inner canal. An ear wick will prove useful in this situation.

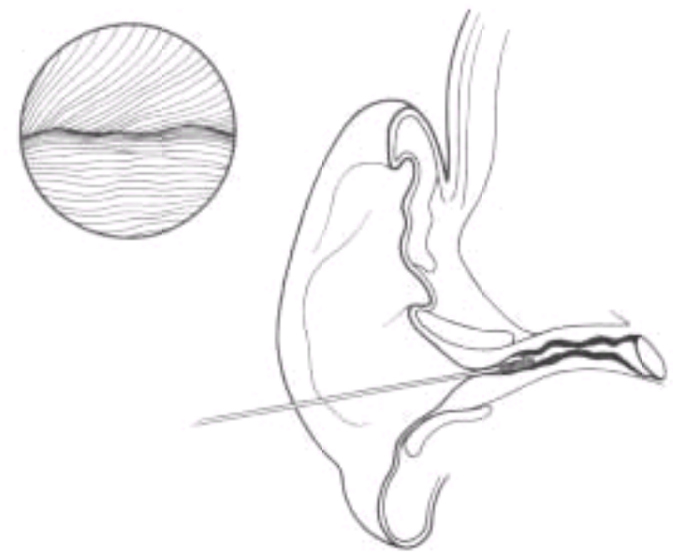


Figure 65-25 The Hognose device for foreign body removal. *A*, The Hognose attached to the otoscope and to wall suction. *B*, Occlusion of the open insufflation port to engage suction and remove the foreign body (attached).

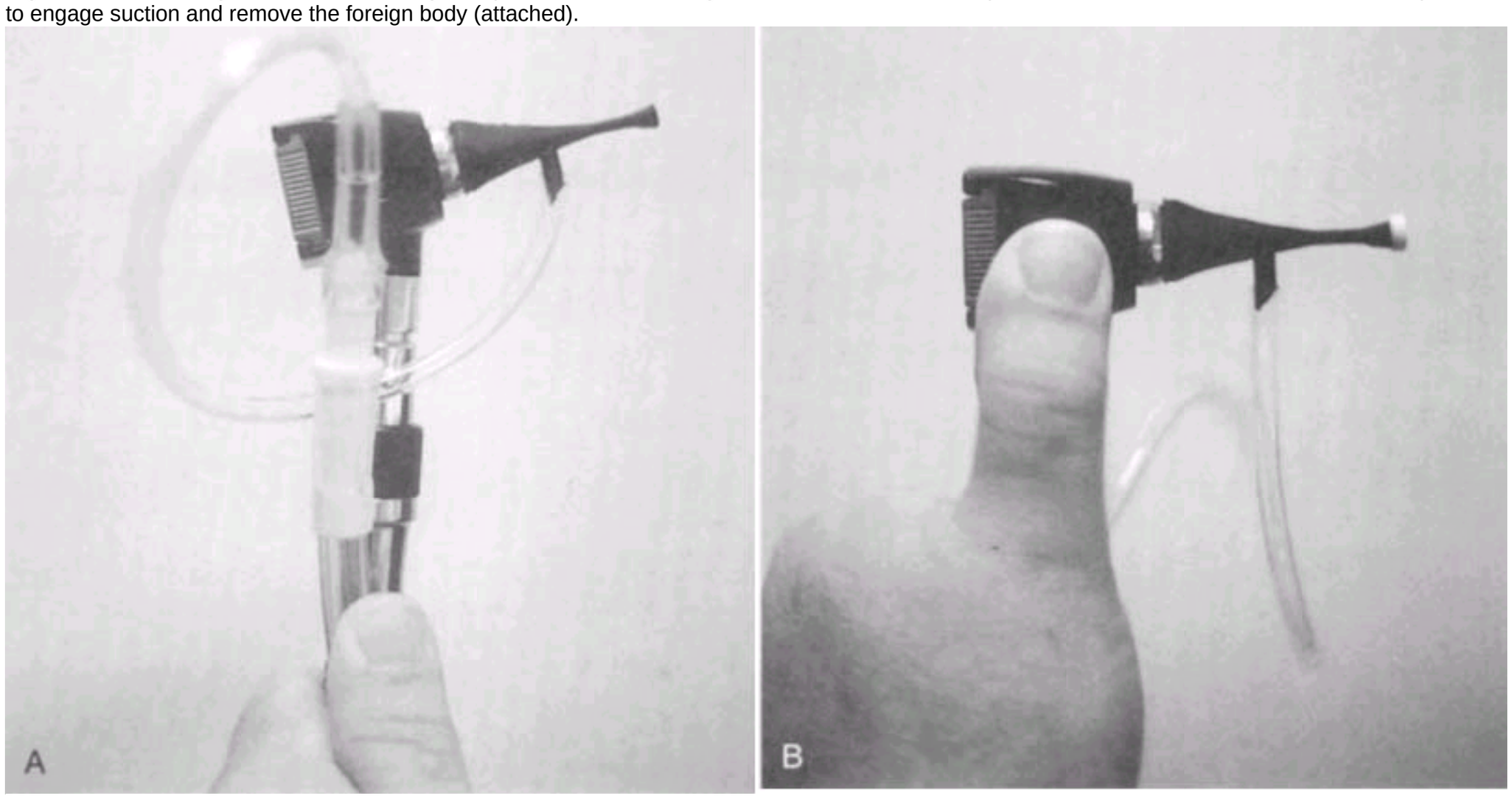


Figure 65-26 Application of various methods of foreign body removal from the ear canal. *A*, Right-angle hook. *B*, Irrigation. *C*, Alligator forceps. *D*, Soft-tipped suction.

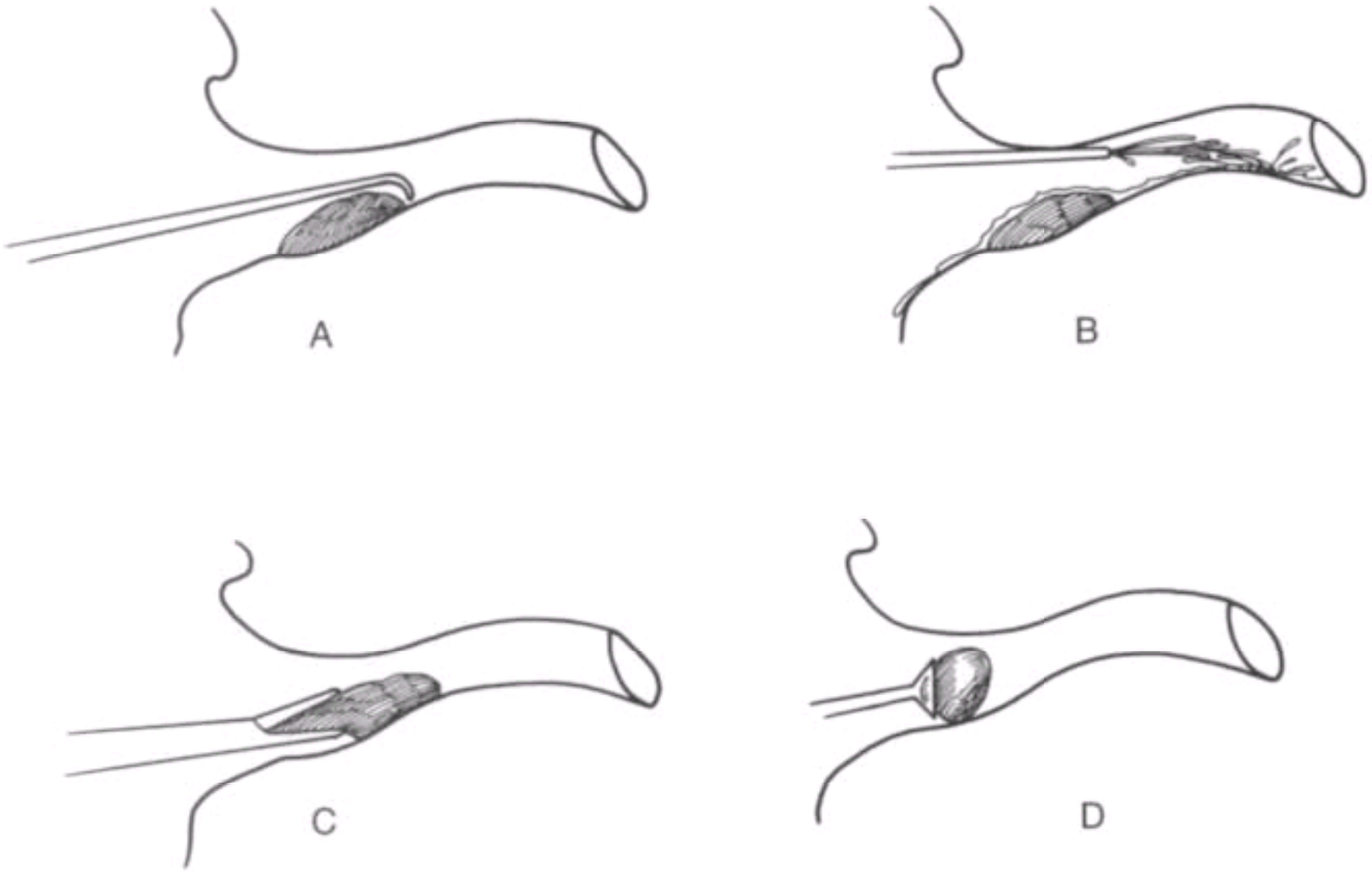


Figure 65-27 *A,* Subperichondrial hematoma within the concha of the ear. *B,* Needle aspiration of an auricular hematoma. A topical antiseptic is used to clean the ear, but local anesthesia is seldom required. While stabilizing the pinna with the thumb and fingers, the most fluctuant part of the hematoma is punctured with a 20-ga needle. The thumb "milks" the hematoma into the syringe until the entire hematoma has been evacuated. The thumb maintains continued pressure on the ear for 3 minutes after the needle has been withdrawn. A pressure dressing is then applied, and the ear is checked for reaccumulation of blood in 24 hours. Reaspiration may be required, and persistent accumulations require incision and drainage. *(B redrawn with permission from Fleisher GR, Ludwig S, Henretig FM, et al: Textbook of Pediatric Emergency Medicine. Baltimore, Williams & Wilkins, 1983.)*

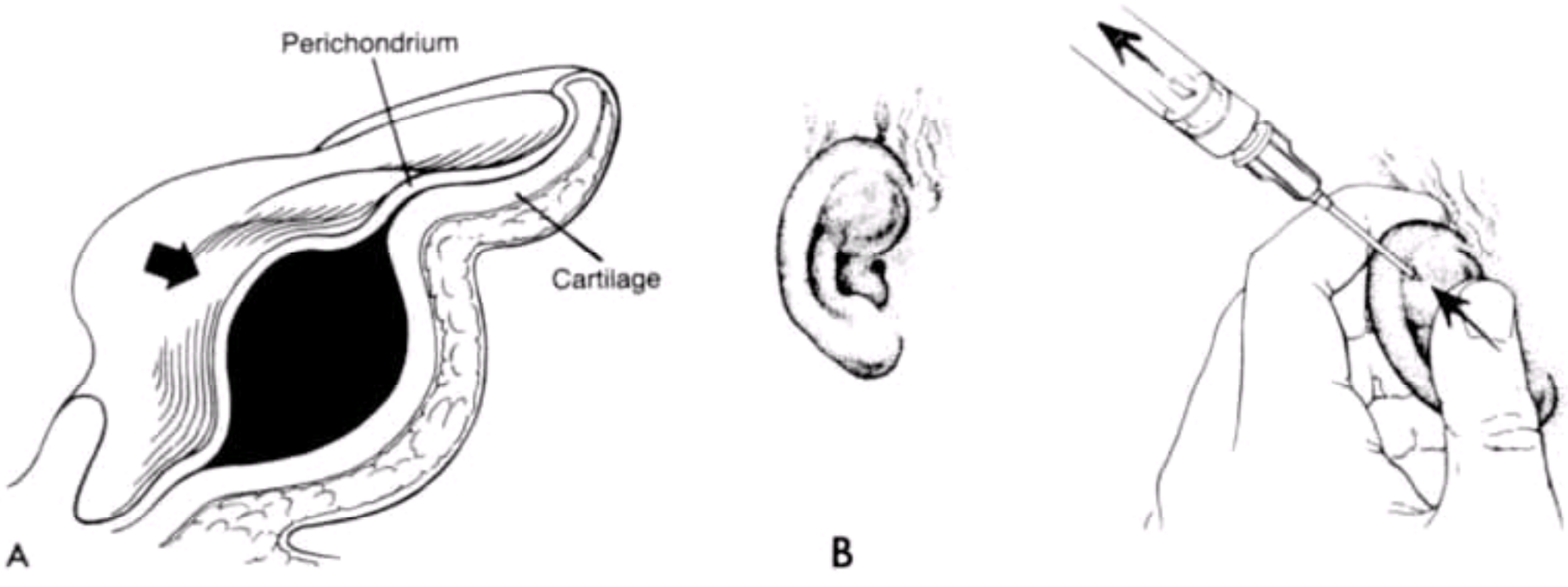


Figure 65-28 Auricular hematoma. *A*, Hematoma separating the perichondrium from the cartilage. *B*, Incision *(arrows)* made along the skin curvature at the posterior edge of the hematoma. Hematoma is evacuated and area irrigated. *C*, Two anterior dental rolls are secured with sutures to a posterior dental roll to maintain normal anatomy of the pinna. *D*, Side view illustrates position of sutures and dental rolls in relation to incision site. Note that the perichondrium is apposed to the cartilage. *(From Clemons JE, Seveneid LR: Otohematoma. In Cummings CW (ed): Otolaryngology—Head and Neck Surgery, 2nd ed. St Louis, Mosby-Year Book, 1993, p 2866.)*

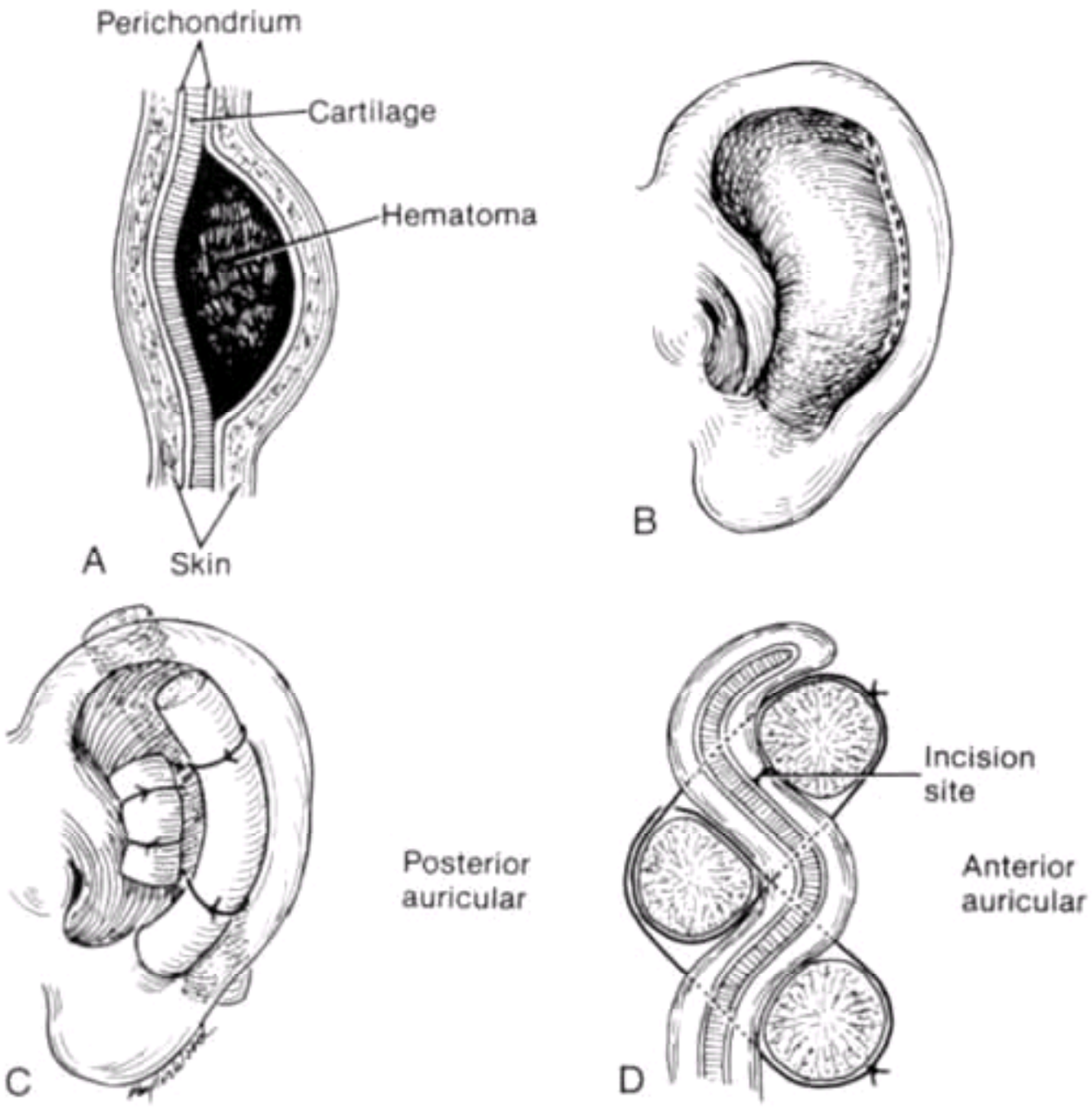
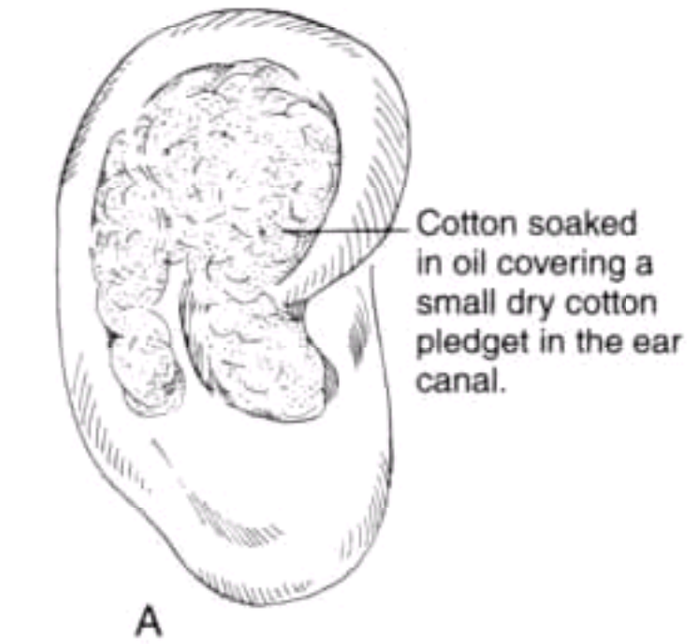


Figure 65-29 Compression dressing of the ear. Following successful aspiration of an auricular hematoma, a compression dressing is used to prevent reaccumulation of the hematoma or fluid. *A,* Dry cotton is first placed into the ear canal. A conforming material is then carefully molded into all the convolutions of the auricle. One may use Vaseline gauze or cotton soaked in mineral oil or saline. *B,* When the convolutions are fully packed, a posterior gauze pack is placed behind the ear. A V-shaped section has been cut from the gauze to allow it to fit easily behind the ear. *C,* Multiple layers of fluffed gauze are placed over the packed ear, and the entire dressing is held in place with Kling gauze or an elastic gauze roll. *D,* The ear is thus compressed between two layers of gauze, and the packing ensures even distribution of pressure to all parts of the auricle.



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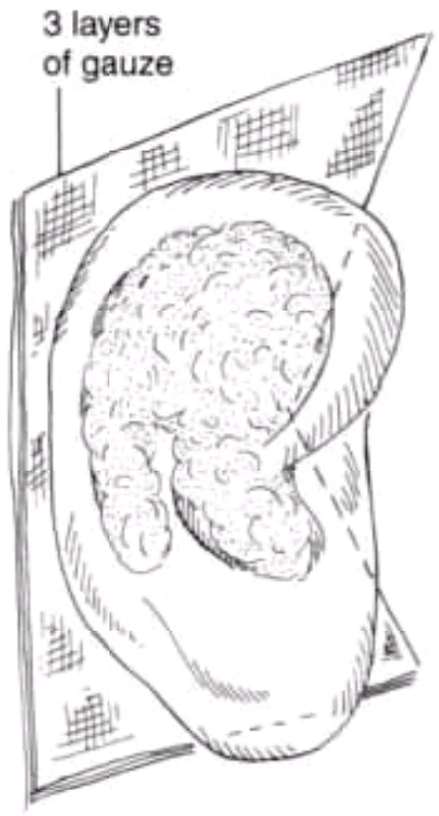

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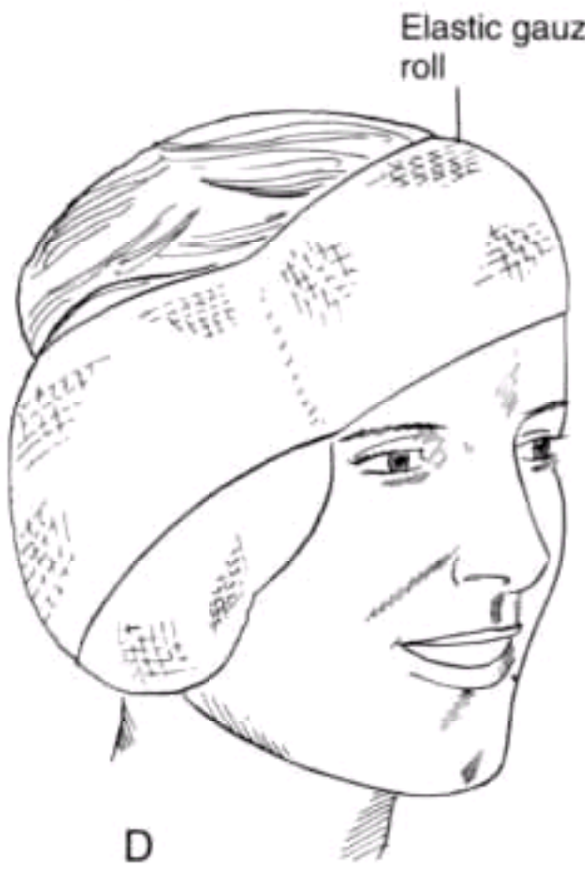


D

Figure 65-30 Vascular supply to the lateral wall. The most common site of posterior epistaxis is the sphenopalatine artery as it emerges posterior to the middle turbinate. *(From Maceri DR: Epistaxis and nasal trauma. In Cummings CW (ed): Otolaryngology—Head and Neck Surgery, 2nd ed. St Louis, Mosby-Year Book, 1993, p 728.)*

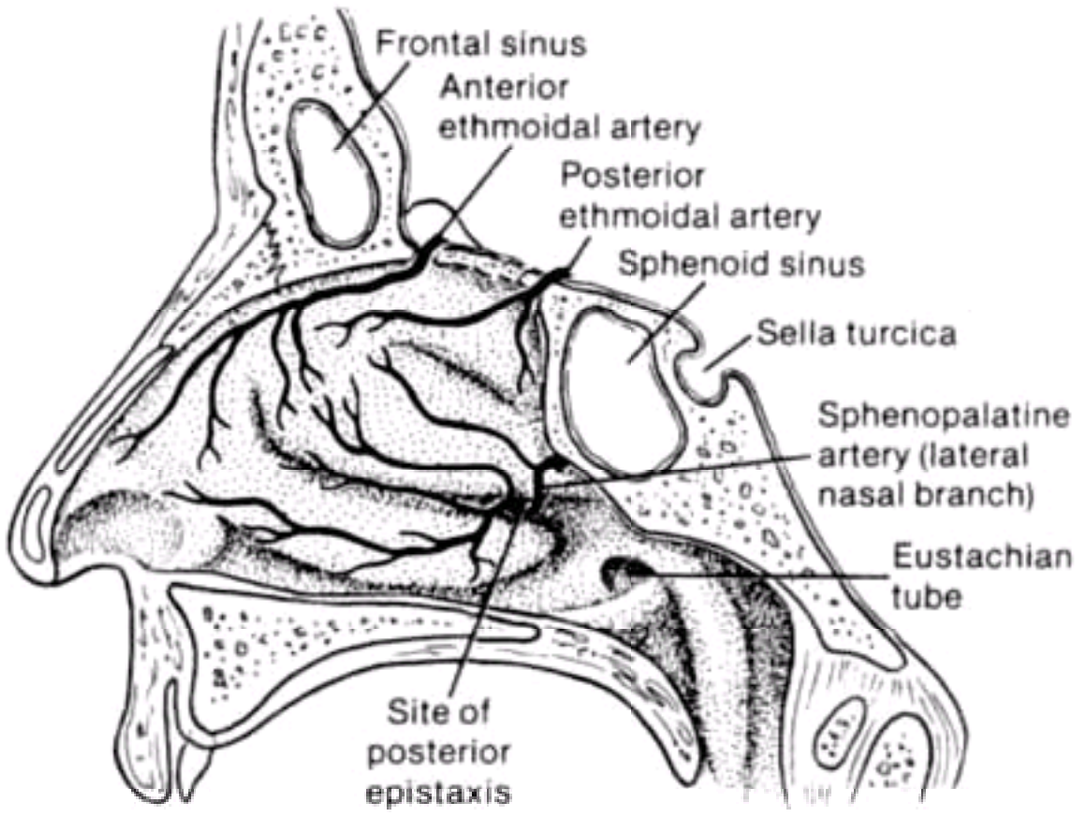


Figure 65-31 Vascular supply to the septum. The most common site of anterior epistaxis is within the area labeled Kiesselbach's plexus. *(From Maceri DR: Epistaxis and nasal trauma. In Cummings CW (ed): Otolaryngology—Head and Neck Surgery, 2nd ed. St Louis, Mosby-Year Book, 1993, p 728.)*

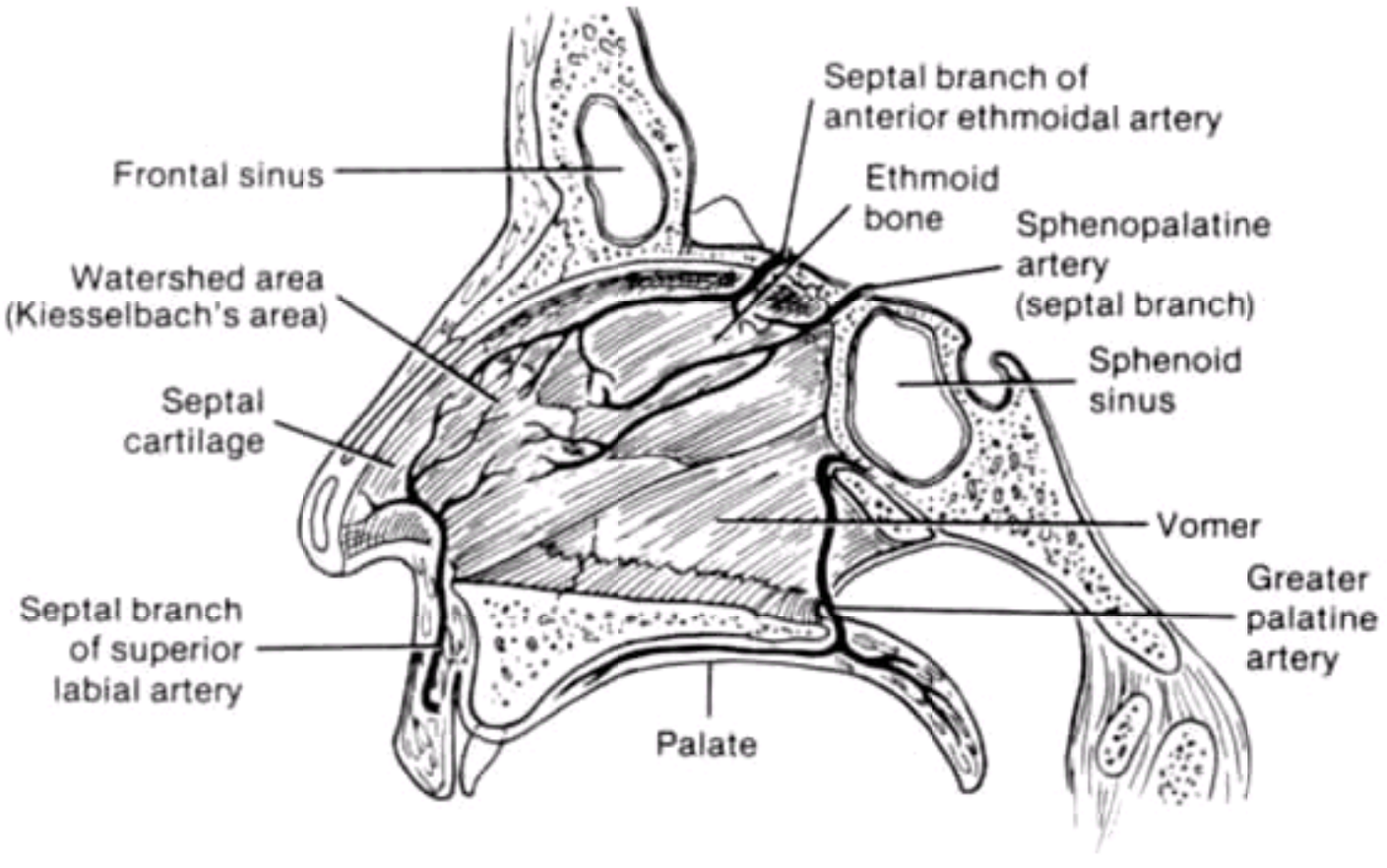


Figure 65-32 Placement of local anesthetic in the nose for anesthesia before reduction of nasal fracture by blockage of the anterior ethmoidal nerve superiorly and the sphenopalatine ganglion at the posterior end of the middle turbinate. *(From DeWeese DD, Saunders WH, Schuller DE, Schleuning AJ II (eds): Otolaryngology—Head and Neck Surgery, 7th ed. St Louis, CV Mosby, 1988.)*

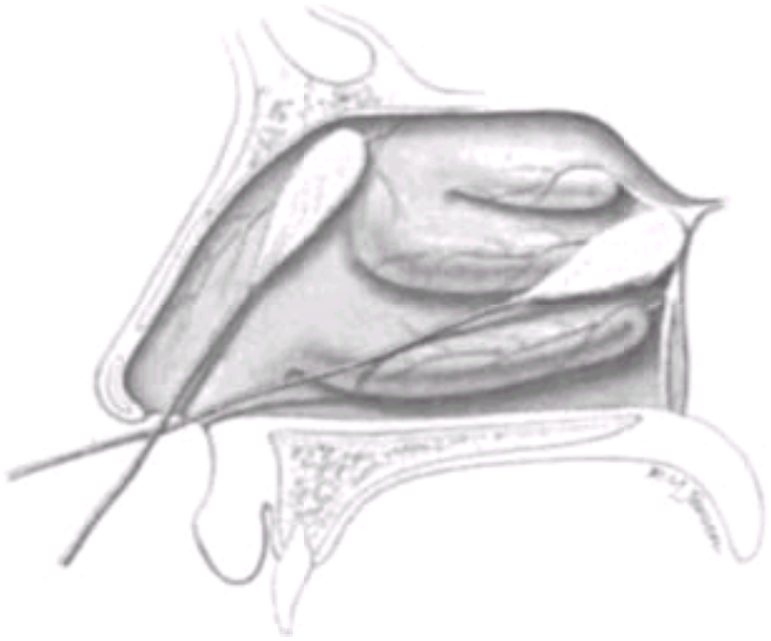


Figure 65-33 Topical anesthetic and vasoconstrictors are applied on individually made cotton pledgets. The size of the pledget may be changed according to the extent of the nasal cavity to be anesthetized and the size of the patient. *A,* An appropriately sized cotton pledget is grasped in a bayonet forceps. *B,* The cotton is then grasped with the opposite hand, and the forceps is rotated. *C,* The pledget is removed and is ready for insertion. *D,* To completely anesthetize the nasal cavity, three pledgets are necessary. The first is placed on the floor of the nose, the second in the middle meatus between the inferior and middle turbinates, and the third in the roof of the nasal cavity and the anterior nasal vestibule. *Note:* This pledget technique can be used to make a cotton wick for the treatment of otitis externa.

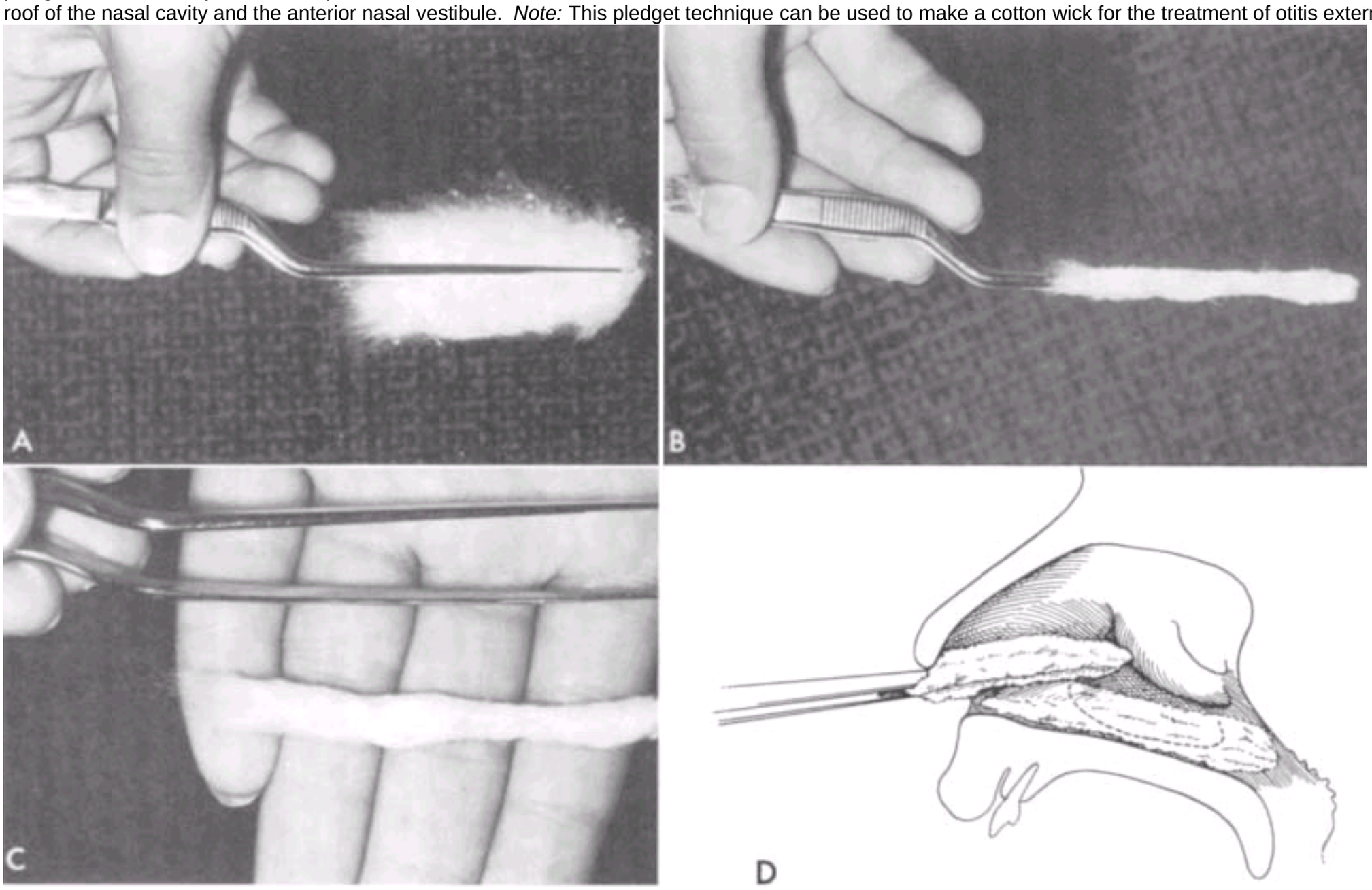


Figure 65-34 To examine a nose properly, a nasal speculum must be used. *A,* The clinician's index finger rests on the bridge of the nose, and the speculum is spread in an inferior-to-superior direction. *B,* It is *incorrect* to spread the speculum laterally or to use the instrument in an unsupported manner.

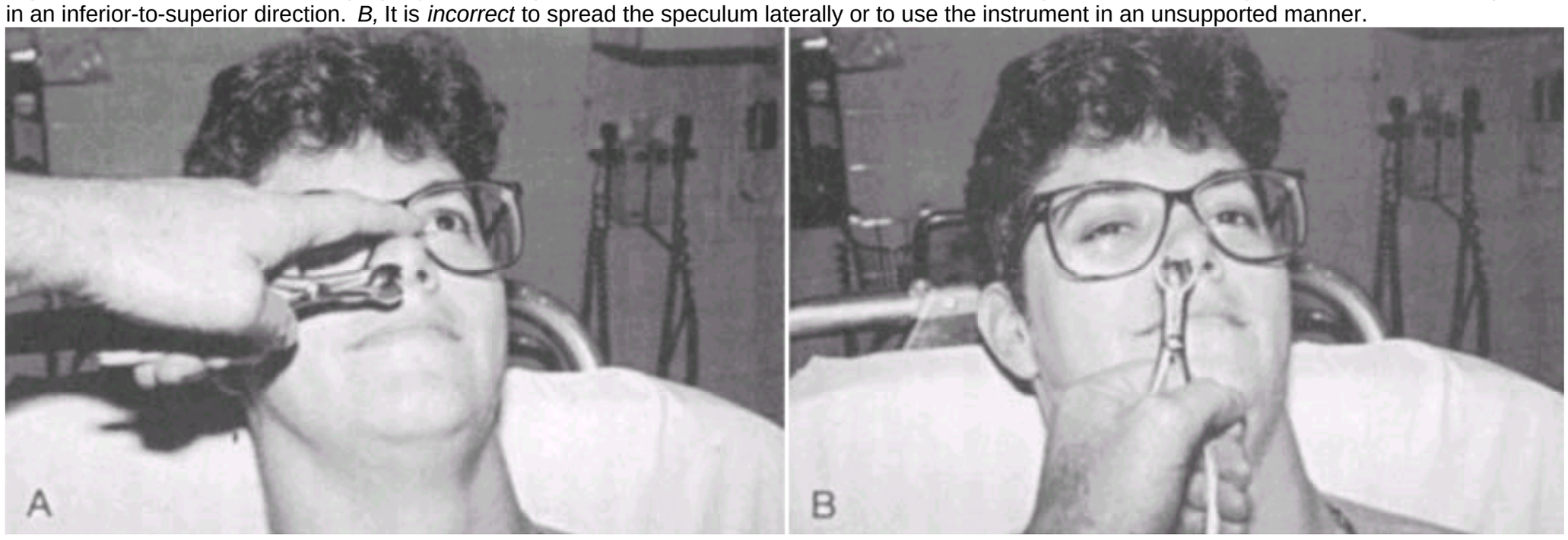
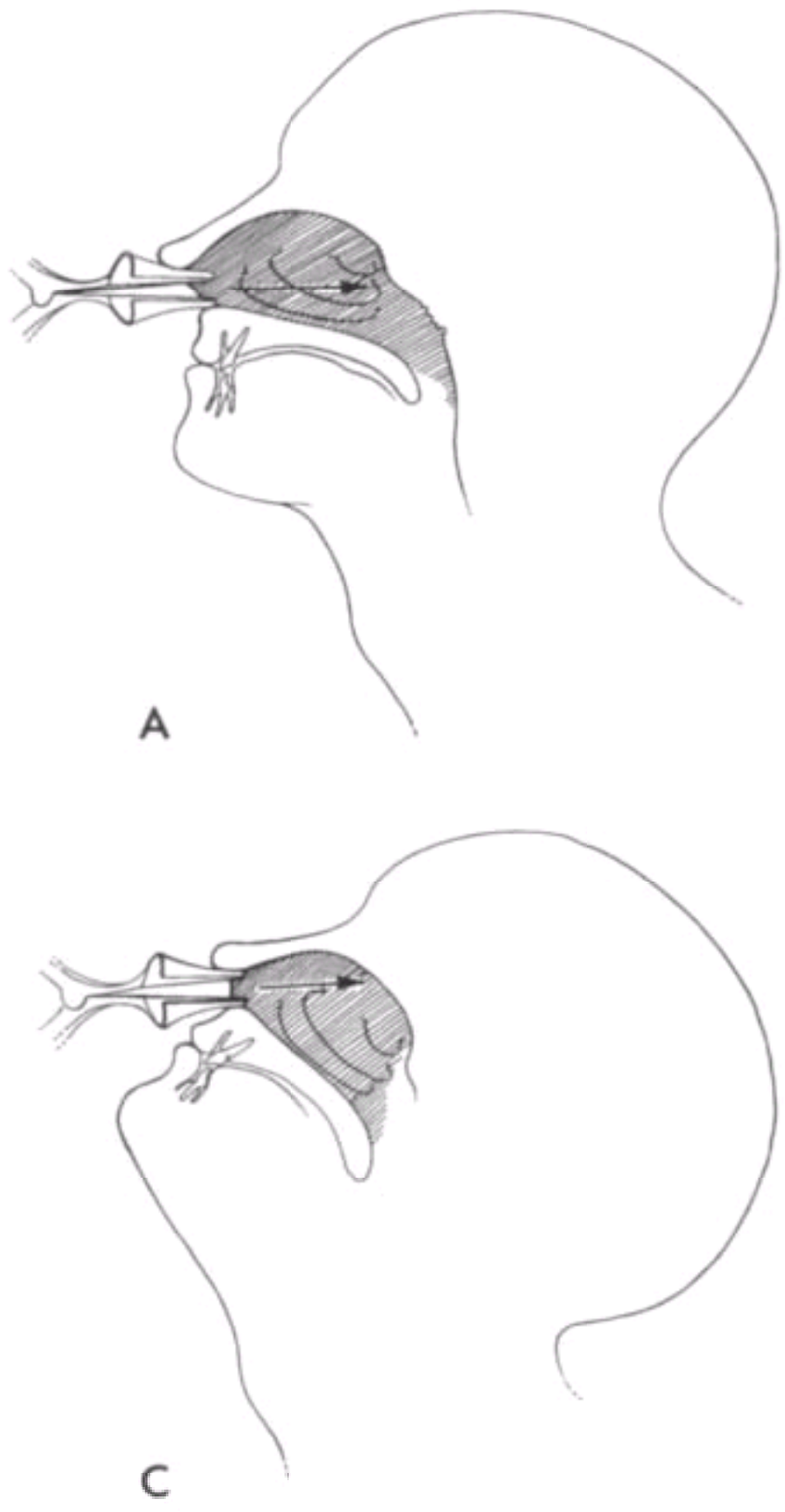


Figure 65-35 *A* and *B,* Correct position for examining and treating diseases of the nasal cavity. The patient is in the "sniffing" position, sitting upright or leaning slightly forward with the head only slightly extended. When the nasal tip is raised with the nasal speculum, the view is parallel to the floor of the nose and allows visualization of the entire nasal cavity. *C* and *D,* When told that their nose will be examined, most patients extend the neck and look toward the ceiling. In this position, only the most anterior portions of the nasal cavity are visible.



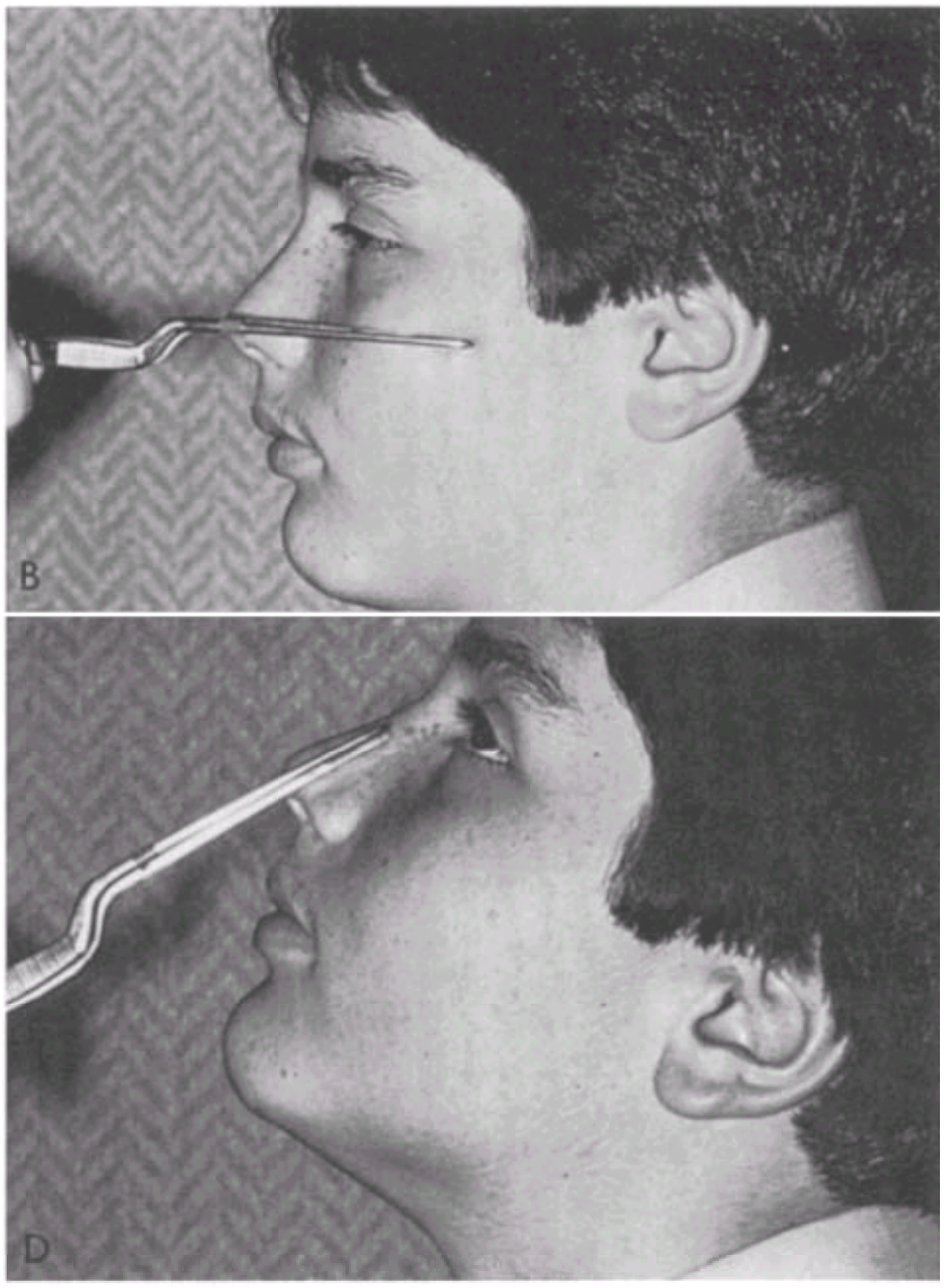


Figure 65-36 If an arterial bleeding site is found in the nasal septum, both hemostasis and anesthesia for cautery can be accomplished by injecting the mucosa at the base of the bleeder with a small amount of lidocaine with epinephrine via a tuberculin or insulin syringe. This procedure best follows an initial application of topical anesthesia.

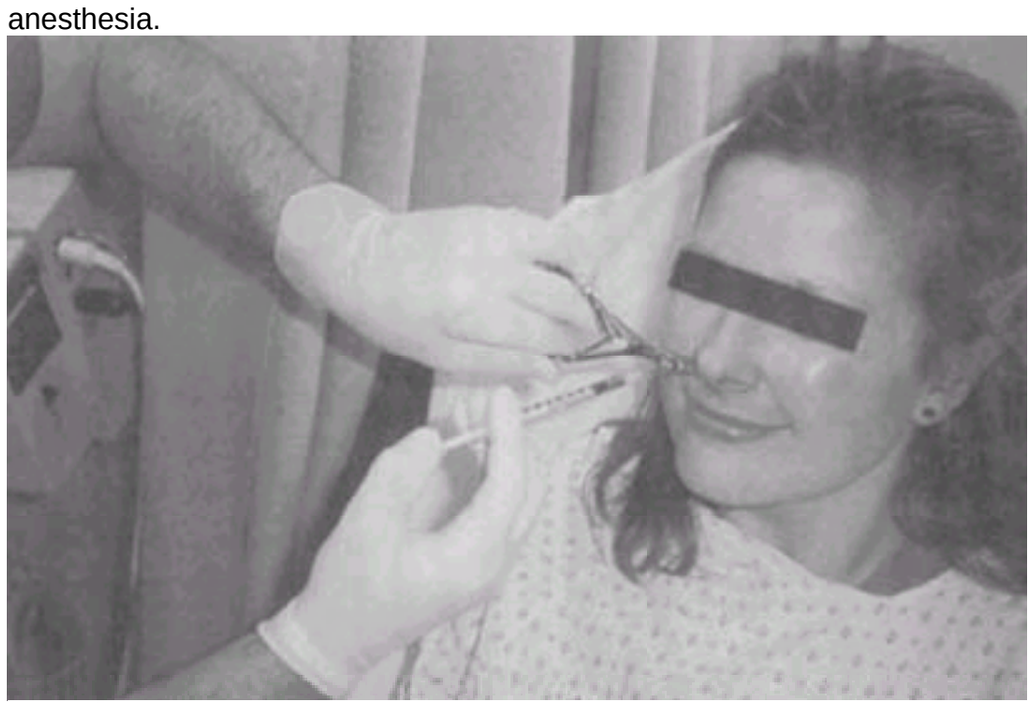


Figure 65-37 Equipment used for treatment of epistaxis. *Top:* Electrocautery device and silver nitrate sticks. Left to right, bottom row: Merocel nasal tampon, posterior packing ball, Merocel nasal tampon with catheter included (Doyle Pack), anterior packing gauze with bayonet forceps, Foley catheter, Epistat dual balloon catheter.

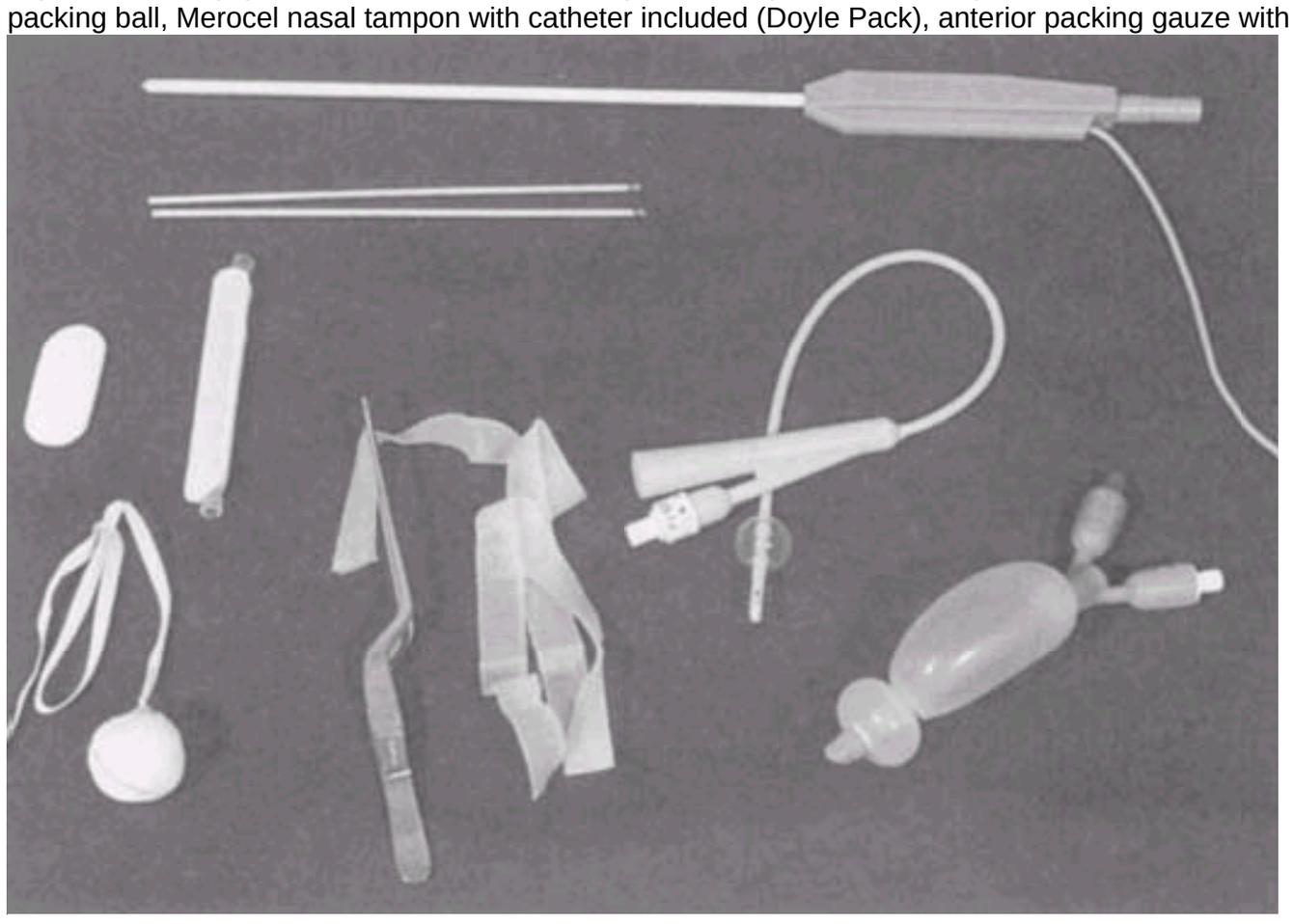


Figure 65-38 The key to placement of an anterior nasal pack that will control epistaxis adequately and stay in place is to lay the packing into the nasal cavity in an "accordion" manner, so that part of each layer of packing lies anteriorly, preventing the gauze from falling posteriorly into the nasopharynx. *A,* The first layer of 0.25-in. Vaseline gauze strip is grasped approximately 2 to 3 cm from its end. *B,* The first layer is then placed on the floor of the nose through the nasal speculum (not pictured here). The bayonet forceps and nasal speculum are then withdrawn. *C,* The nasal speculum is reintroduced on top of the first layer of packing, and a second layer is placed in an identical manner. After several layers have been placed, it is often useful to reintroduce the bayonet forceps to push the previously placed packing down onto the floor of the nose, making it tighter and more secure. *D,* A complete anterior nasal pack can tamponade a bleeding point anywhere in the anterior nasal cavity and will stay in place until removed by the clinician or patient.

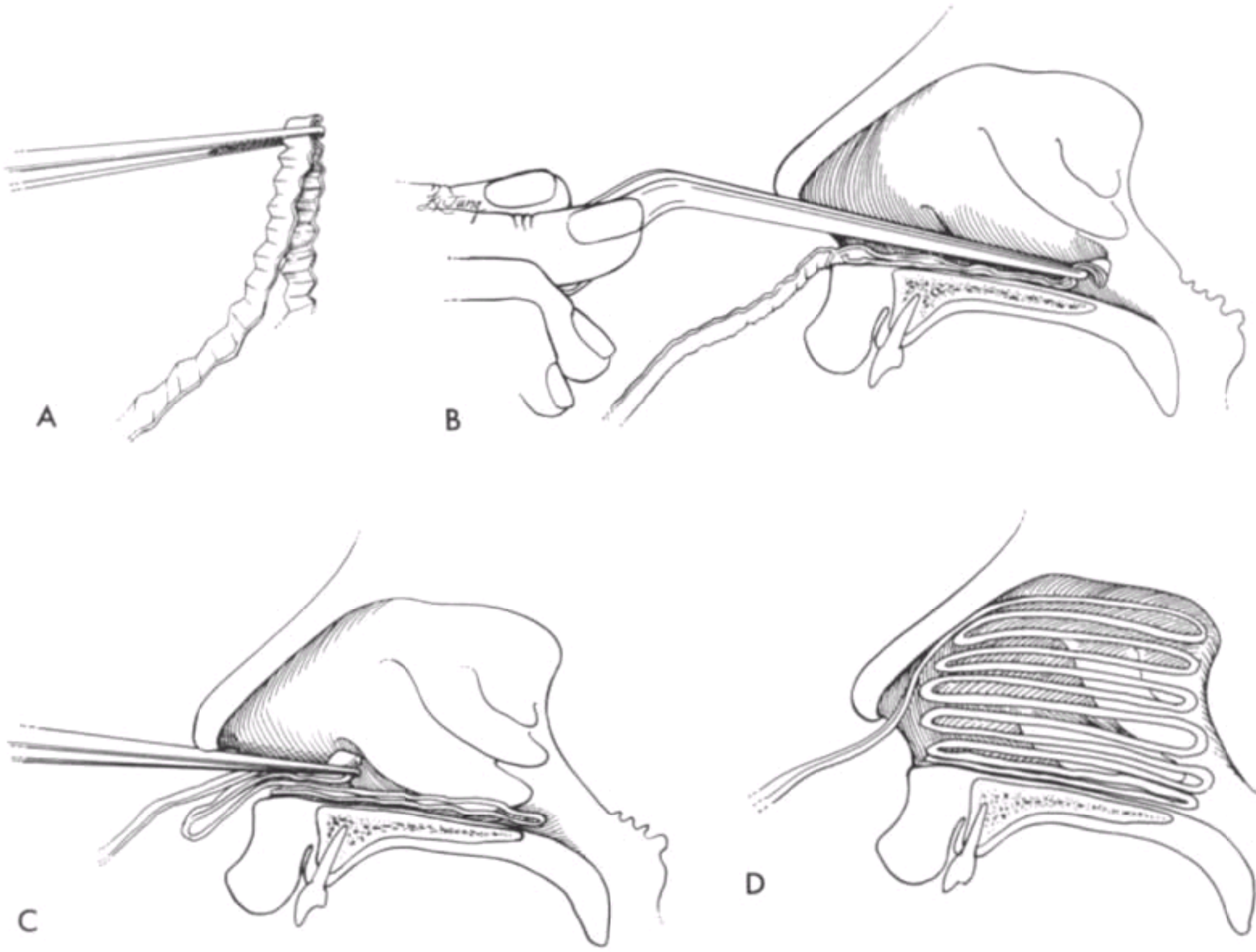
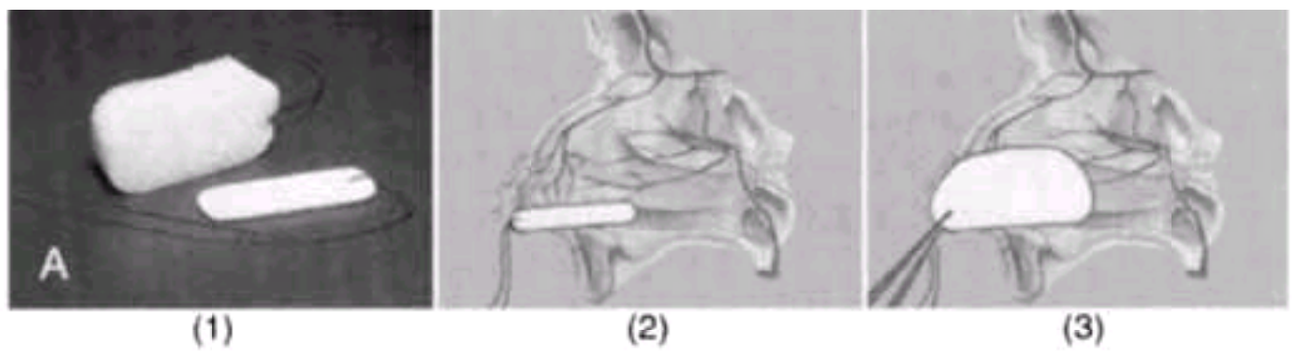


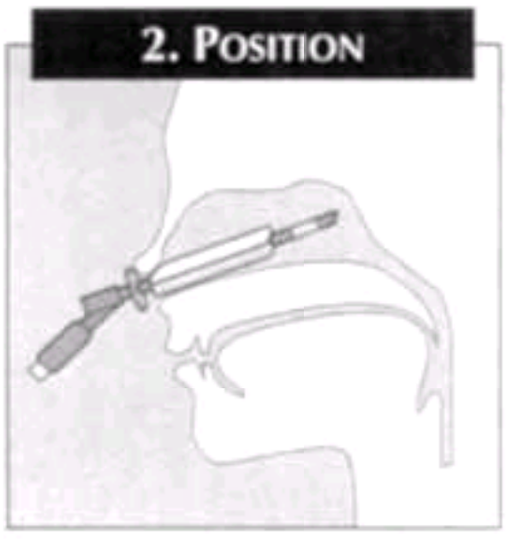
Figure 65-39 Nasal packing alternatives. *A,* Merocel nasal pack. (1) Merocel sponge compressed and expanded. To insert the sponge packing, lubricate the device with an antibiotic ointment and place it in the nasal cavity. (2) Some trimming of the product may be needed and prior use of topical anesthesia is advised. When the sponge is in place, hydrate it with saline. Expansion of the moist sponge will compress the bleeding site. (3) It may remain in place for up to 3 days. Rehydrate the sponge before removal. *B,* Epistat II nasal catheter. The catheter is usually left in place up to 3 days and can be used in outpatients. A hollow inner airway tube allows the patient to breathe.



1. LUBRICATION

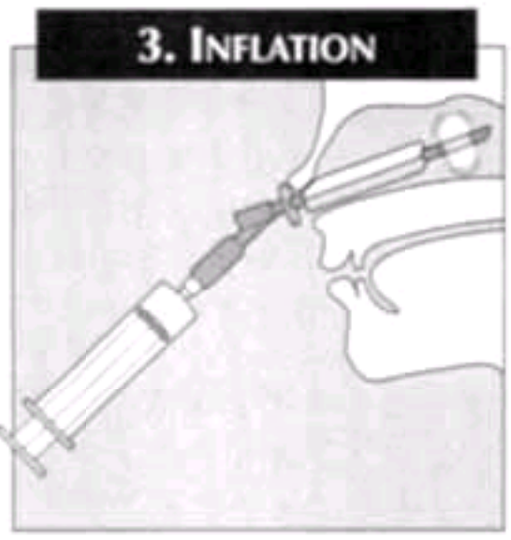
Clean blood clots from the nose. Use a topical anesthesia to numb the nose. Liberally apply antibiotic ointment or lubricant to the **EPISTAT II** Nasal Catheter.

2. POSITION



Position the EPISTAT II Catheter so the posterior balloon rests in the nasopharynx.

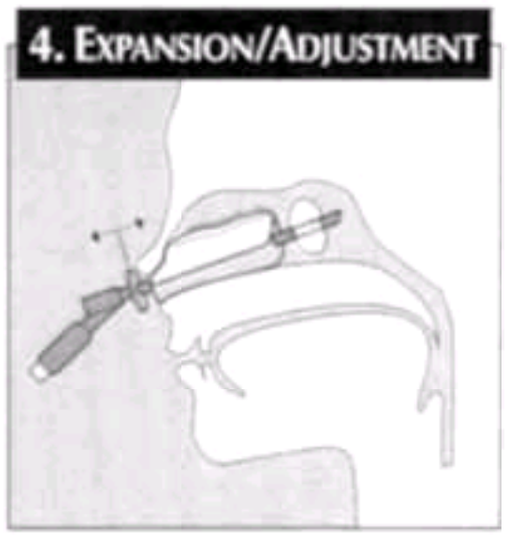
3. INFLATION



Using a syringe, inflate the balloon by injecting approximately 10cc sterile saline into the valve.

After inflation of the balloon, gently pull the catheter outward to position it in the nasopharynx.

4. EXPANSION/ADJUSTMENT



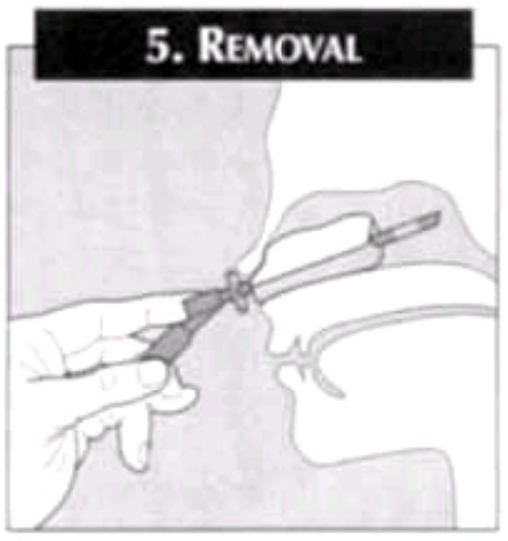
Hydrate the MEROCEL sponge with sterile saline until fully expanded.

Advance the retaining ring until it gently rests against the nose.

The 4" x 4" gauze pad can be placed between the nose and retaining ring for added comfort.

Check the catheter periodically. Release or add pressure as needed.

5. REMOVAL



Introduce a syringe firmly into the valve and withdraw the solution. Rehydrate the MEROCEL sponge with sterile saline until saturated (approximately 10-20cc).

Physicians recommend allowing the pack to rehydrate for 5-10 minutes before removal.

Slowly withdraw the catheter from the nose. The sponge can be left in place by removing only the catheter.

B

Figure 65-40 Examples of various packs for epistaxis in a cadaver model. *A*, The posterior Merocel sponge, not inflated, will stop most nosebleeds and is more comfortable than some balloon devices. *B*, Wetting the Rapid Rhino before placement activates the slippery covering for easier insertion. *C*, Anterior/posterior Rapid Rhino in place. *D*, Posterior pack with balloon inflated.

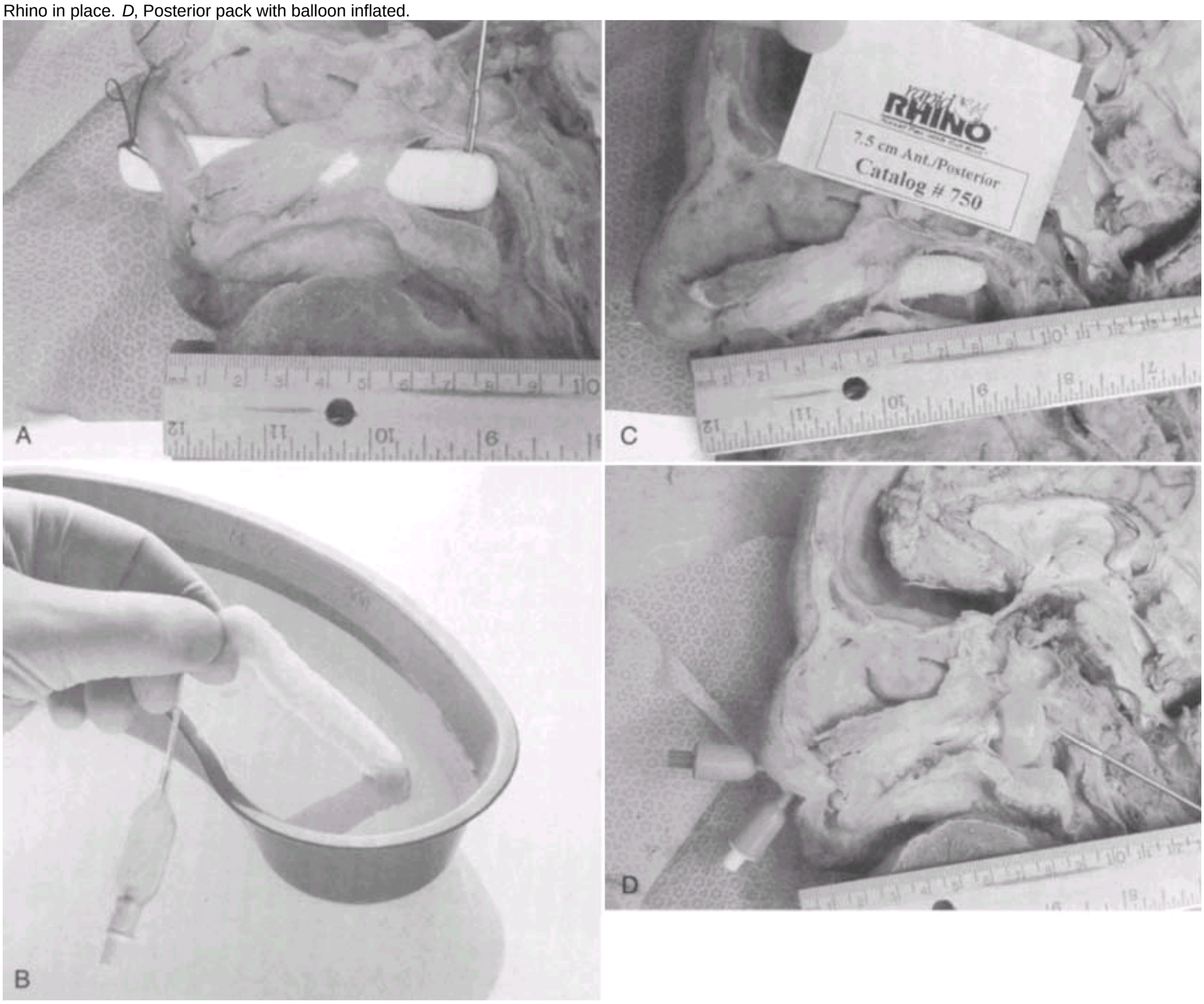
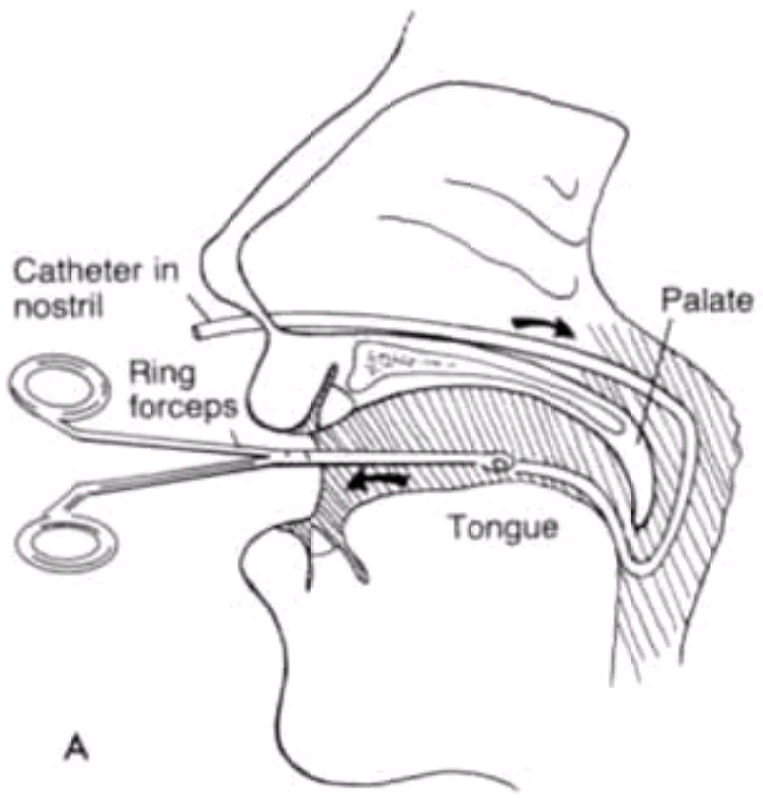
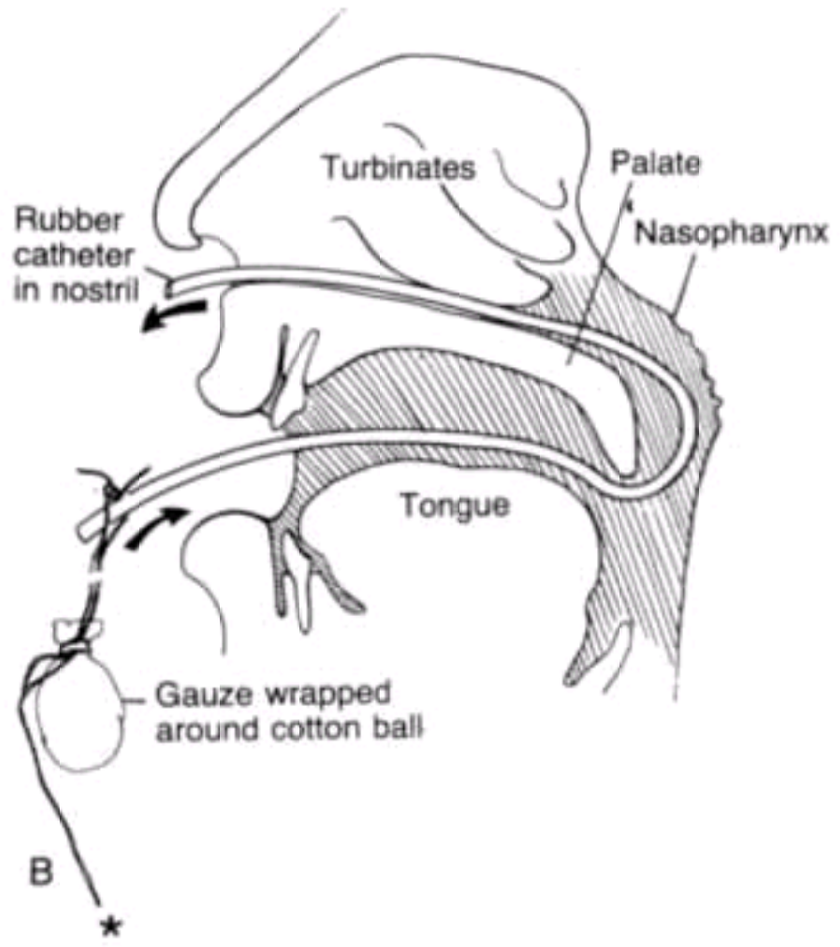
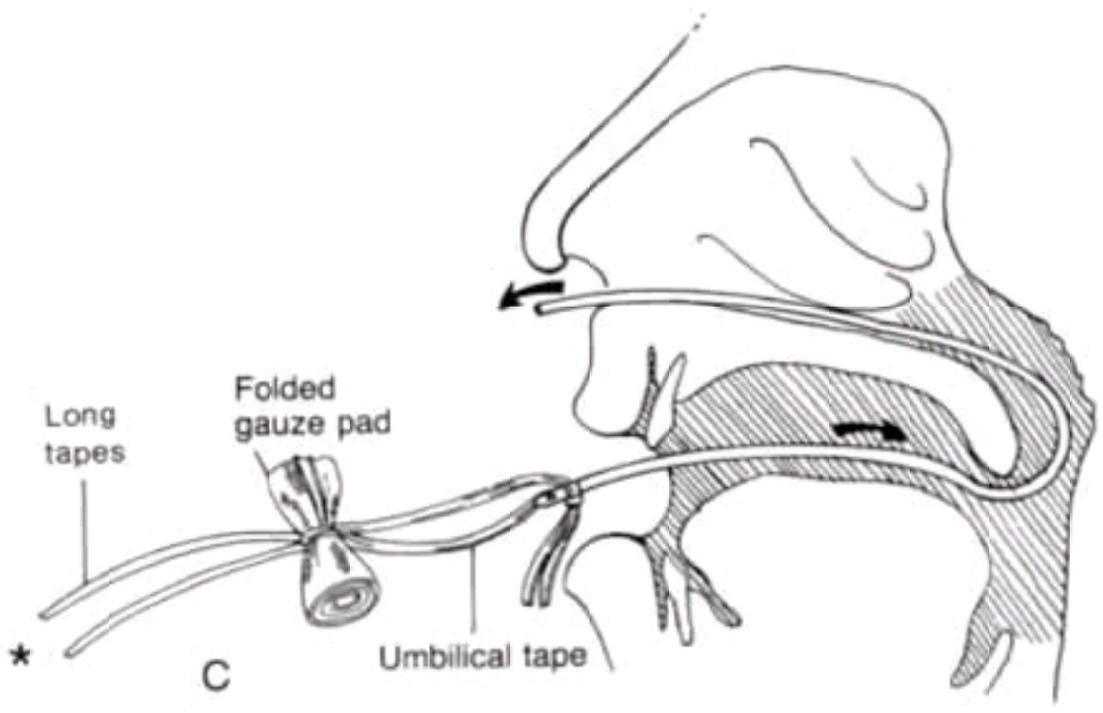


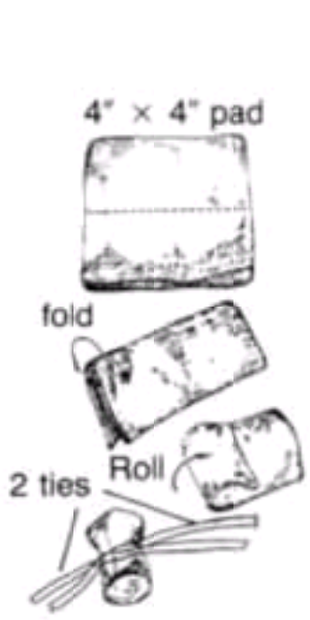
Figure 65-41 An expanding Merocel tampon is ideal for minor anterior bleeds. The dehydrated pack is trimmed to fit the nose and generously lubricated with antibiotic ointment. A swift single motion is used to insert the pack to its full length. The pack expands when hydrated with saline. The patient continues to keep the pack moist at home and it is again well hydrated just before removal.

Figure 65-42a Posterior nasal pack. *A,* Following topical anesthesia, a red rubber catheter is passed through the nose and carefully grasped in the oropharynx with ringed forceps and brought out through the mouth. *B (upper right),* A posterior nasal pack made by wrapping a cotton ball in a 4 × 4-in. gauze pad and tying 2 long silk sutures or umbilical tapes around the neck of the pack. One tie must be left long so that it can be taped to the cheek until needed for removal of the pack. *C (c*enter), Alternatively, a gauze pad can be folded and rolled into a cylinder and tied with two strings. Two of the long strings are used to tie the pack to the tip of the catheter and the other two will be used to remove the pack. *D,* As an option, a second catheter, which has been passed through the nonbleeding side and brought out the mouth, can be used to retract the palate forward to aid in the placement of the pack (not shown). The optional "retraction" catheter is removed after the pack is in the proper position.









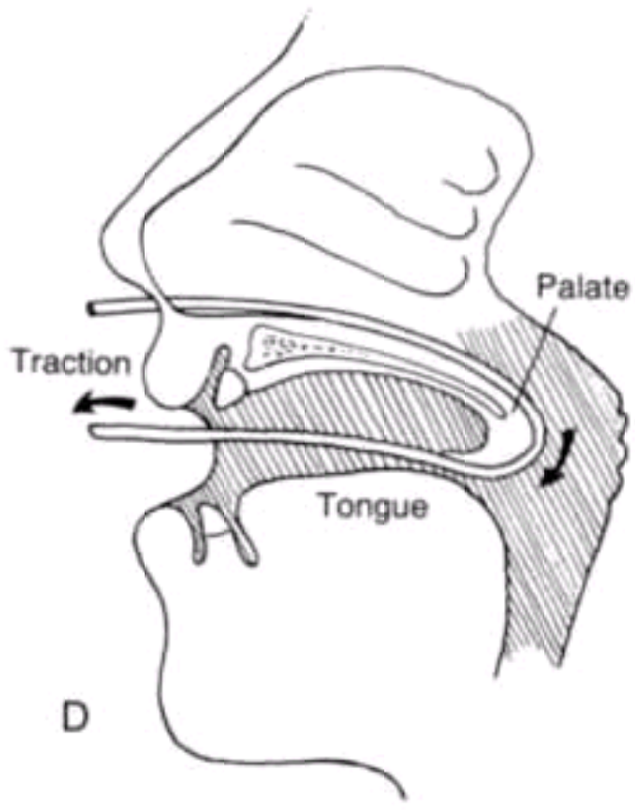


Figure 65-42b *E,* The pack is digitally guided into the nasopharynx. *F,* A gauze roll secures the pack to the nose and the rescue ties are taped to the cheek.

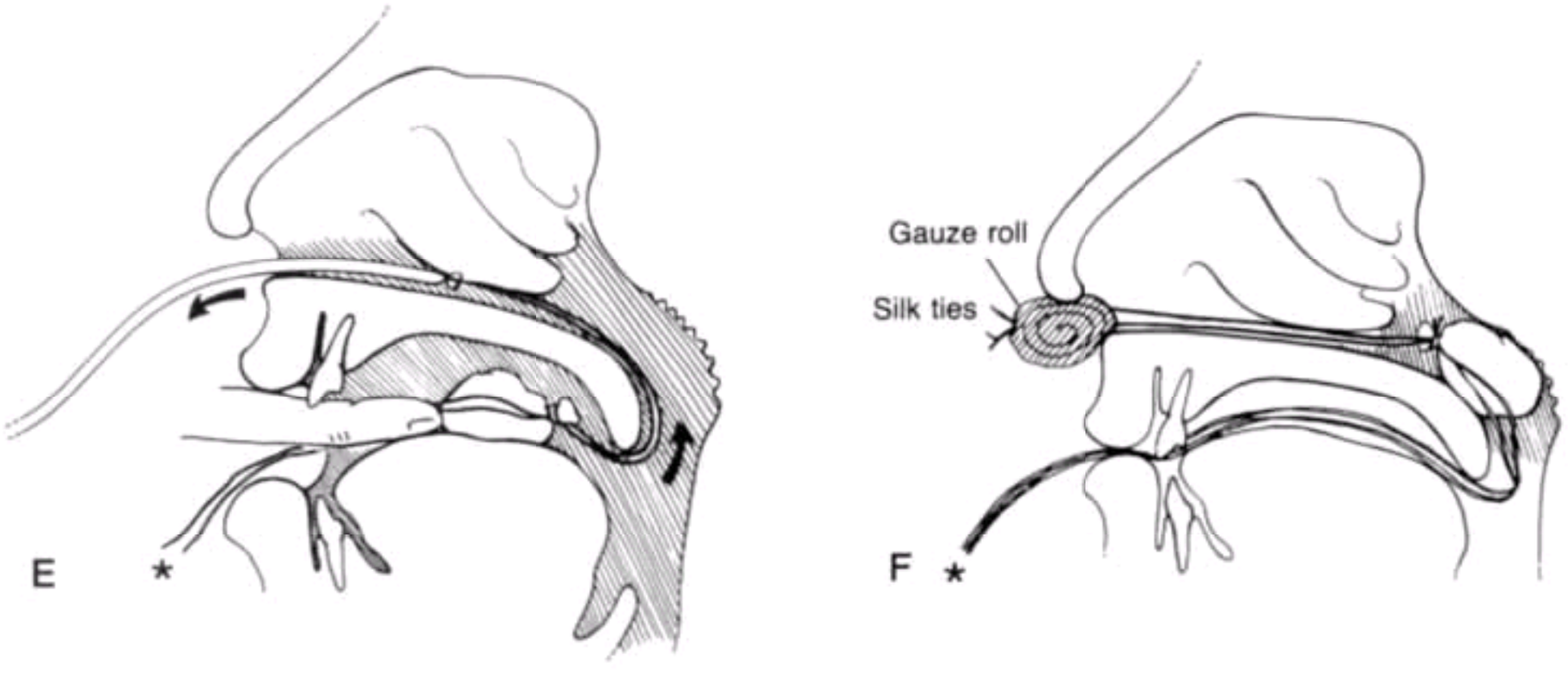


Figure 65-43 Foley catheter placed into the nasopharynx, inflated with water, and retracted into position. The distal tip of the catheter has been cut off. An anterior pack (not shown) is then placed around the catheter. The ala and columella are protected with gauze padding, and a plastic umbilical clamp or nasogastric clamp is applied to the catheter to maintain slight tension on the balloon.

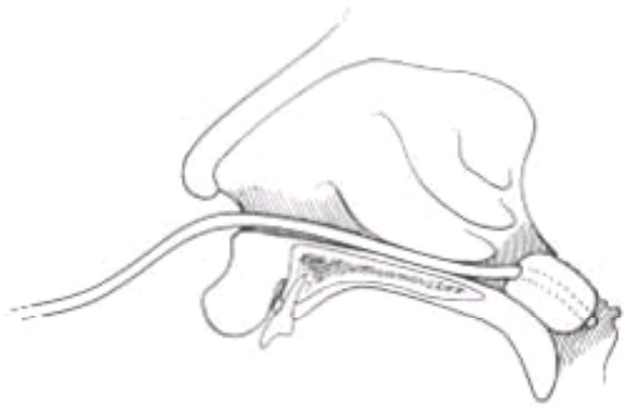
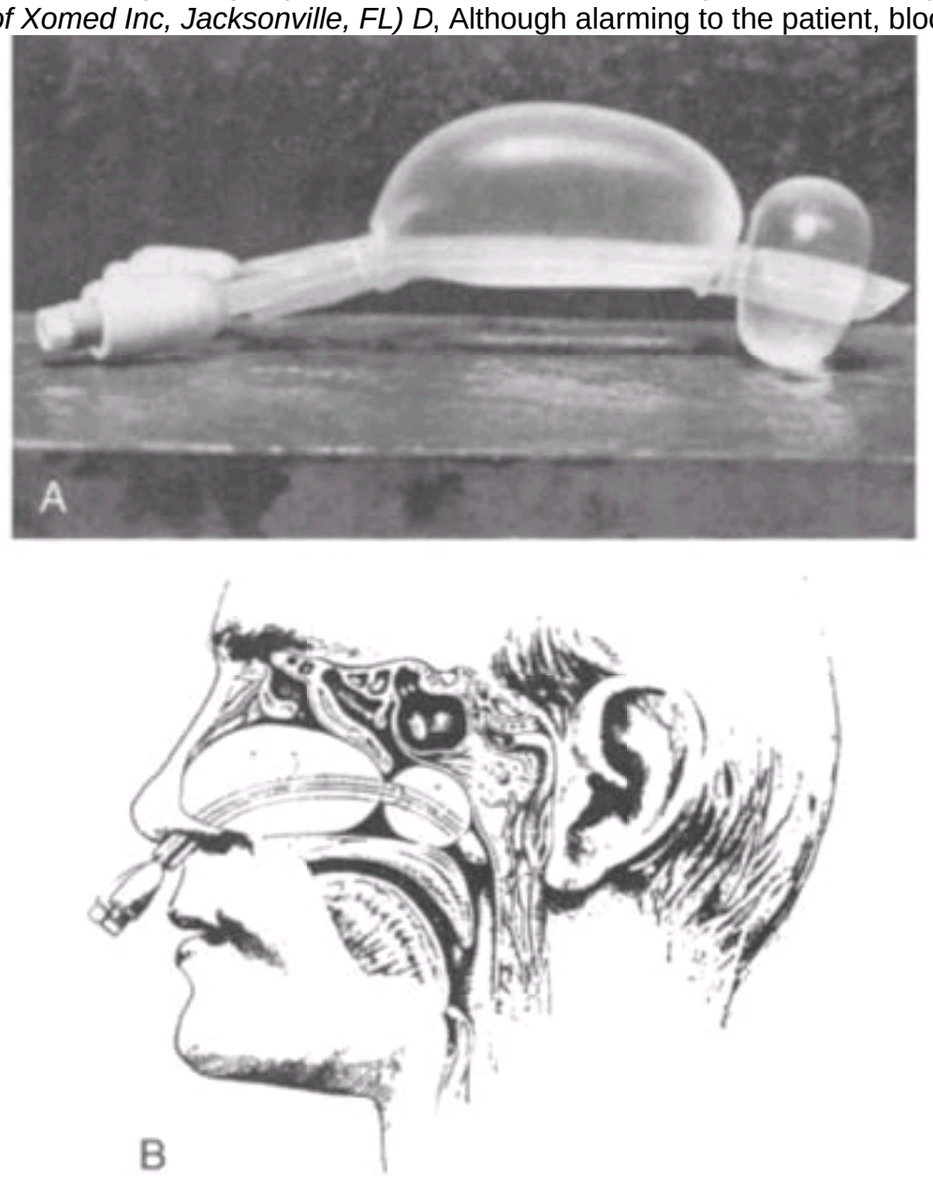


Figure 65-44 *A* and *B,* The balloon tamponade device serves as both an anterior and a posterior pack. It is easily inserted and is often successful for the temporary control of posterior epistaxis in the emergency department. *C,* Although often effective for hemorrhage control, balloon inflation within the nasal cavity can be quite painful and prolonged pressure on the nasal alae can produce tissue injury. Gauze should be placed between the external balloon and the skin of the nose. *(Courtesy of Xomed Inc, Jacksonville, FL) D*, Although alarming to the patient, blood in the eye from back bleeding via the nasolacrimal duct from a balloon pack is benign.



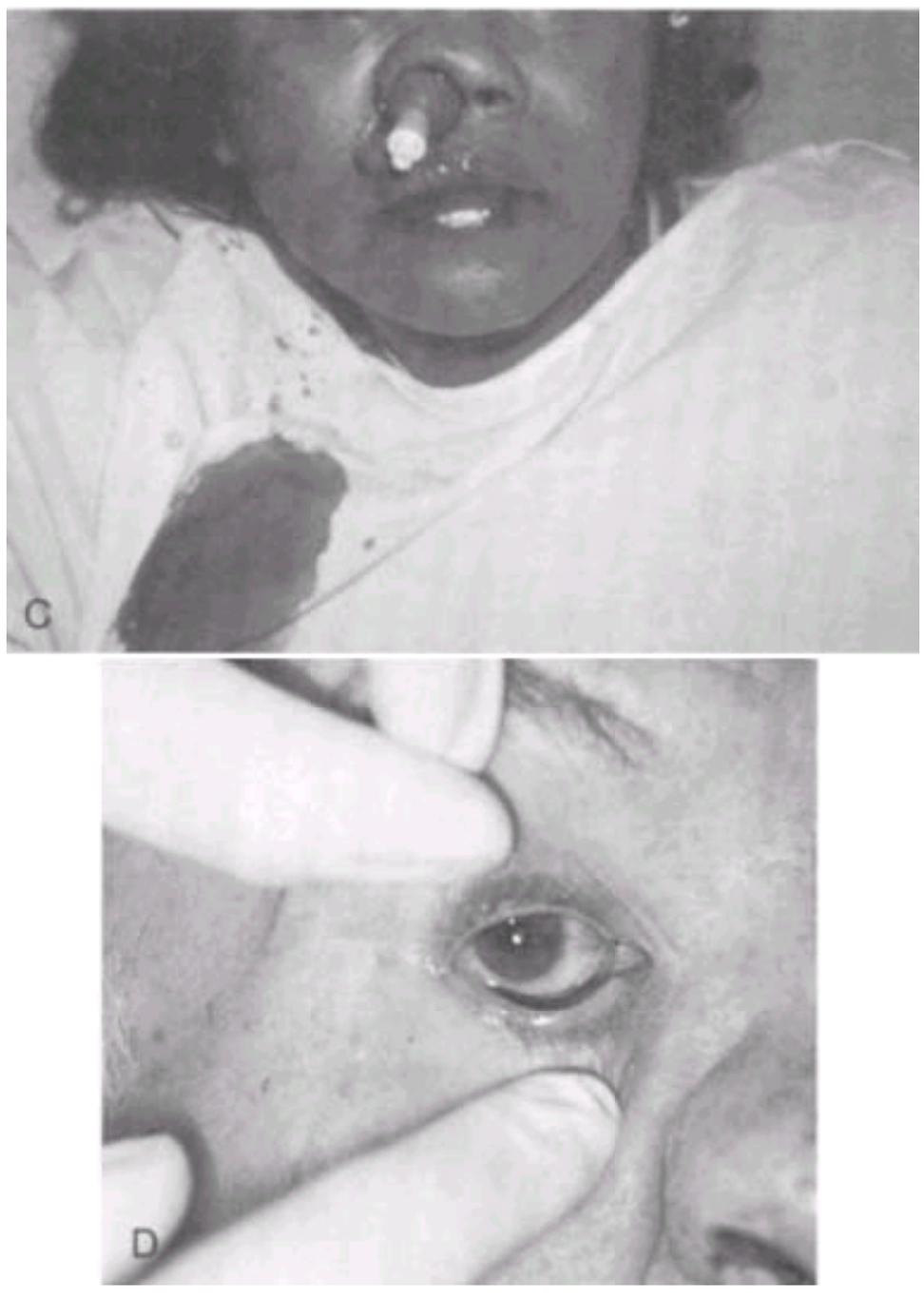


Figure 65-45 Xomed Rhinology Products. *A,* Xomed Epistat nasal catheter. *B,* Pope Flex-Pak nasal packing (shown expanded and compressed). *C,* Xomed Epistat II nasal catheter. *D,* Staxi-Stat pack without drawstring. *E,* Large Fast-Pak nasal pack with applicator. *F,* Weimert epistaxis packing. *G,* Small Fast-Pak nasal pack with applicator.

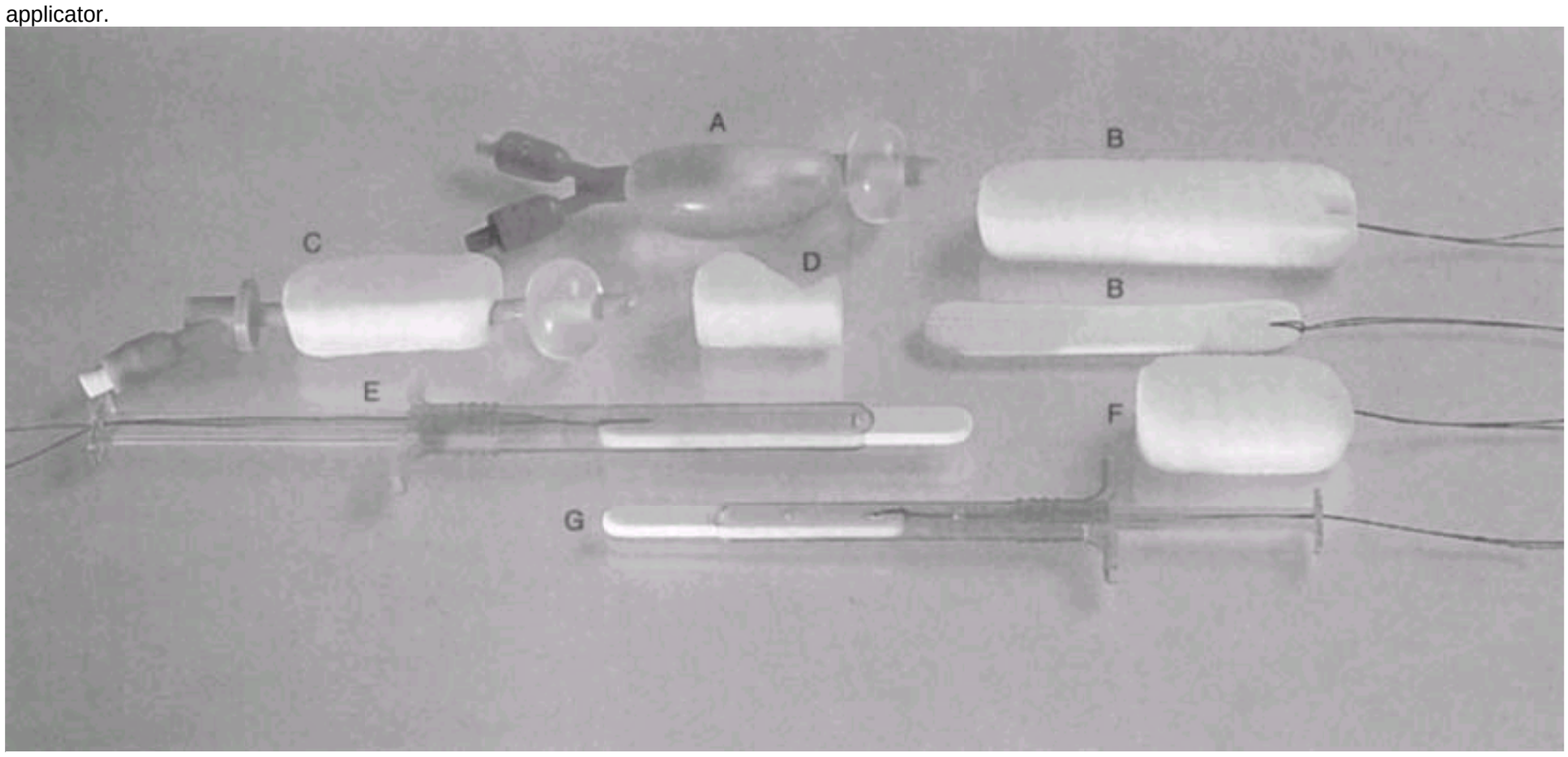


Figure 65-46 If visual inspection of the nose with a speculum does not rule out a septal hematoma, the clinician's gloved fingers, passed posteriorly along both sides of the septum, may reveal bulging or fluctuance.

Figure 65-47 *A*, A small left-sided septal hematoma. *B*, After applying appropriate topical anesthesia, supplemented with local infiltration, if necessary, a *horizontal* incision is made through the mucosa and the perichondrium covering the hematoma. *C,* A small cup forceps or scissors is used to remove enough mucosa to prevent premature closure of the wound and reaccumulation of hematoma. *D,* A sterile rubber band is then placed as a drain, and the naris is packed.

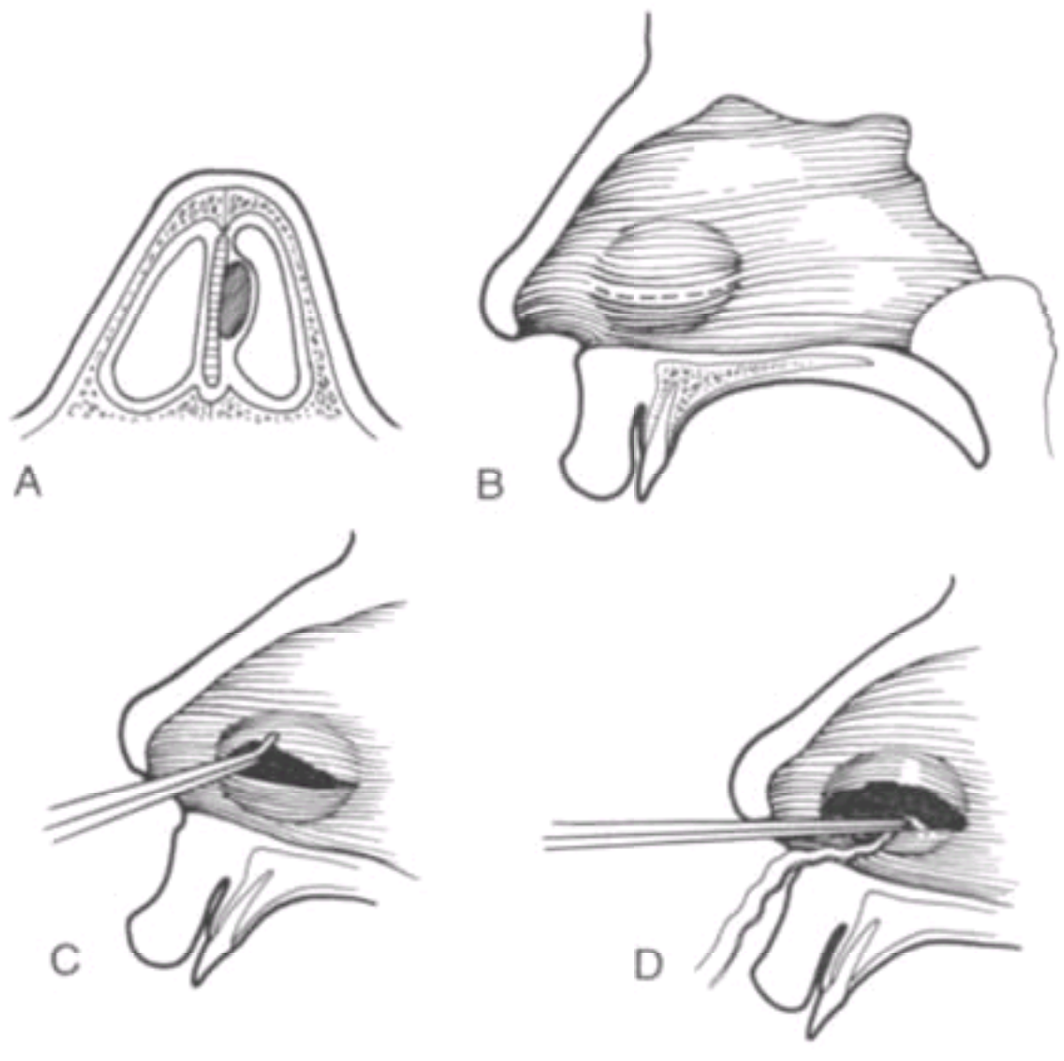


Figure 65-48 Reduction of a depressed and dislocated nasal bone fracture. This reduction is accomplished in two steps following anesthesia by first elevating the depressed nasal bone as illustrated and then manually displacing the pyramid to the midline. The handle of a scalpel may be used if an elevator is unavailable. *(From Adams GL, Boies LR Jr, Hilger PA (eds): Boies Fundamentals of Otolaryngology, 6th ed. Philadelphia, WB Saunders, 1989.)*

Figure 65-49 *A* and *B,* Fogarty balloon extraction of nasal foreign body. Insert the lubricated catheter tip above the foreign body, then gradually inflate the balloon. Slowly withdraw until resistance is met, then pull the object out of the naris.

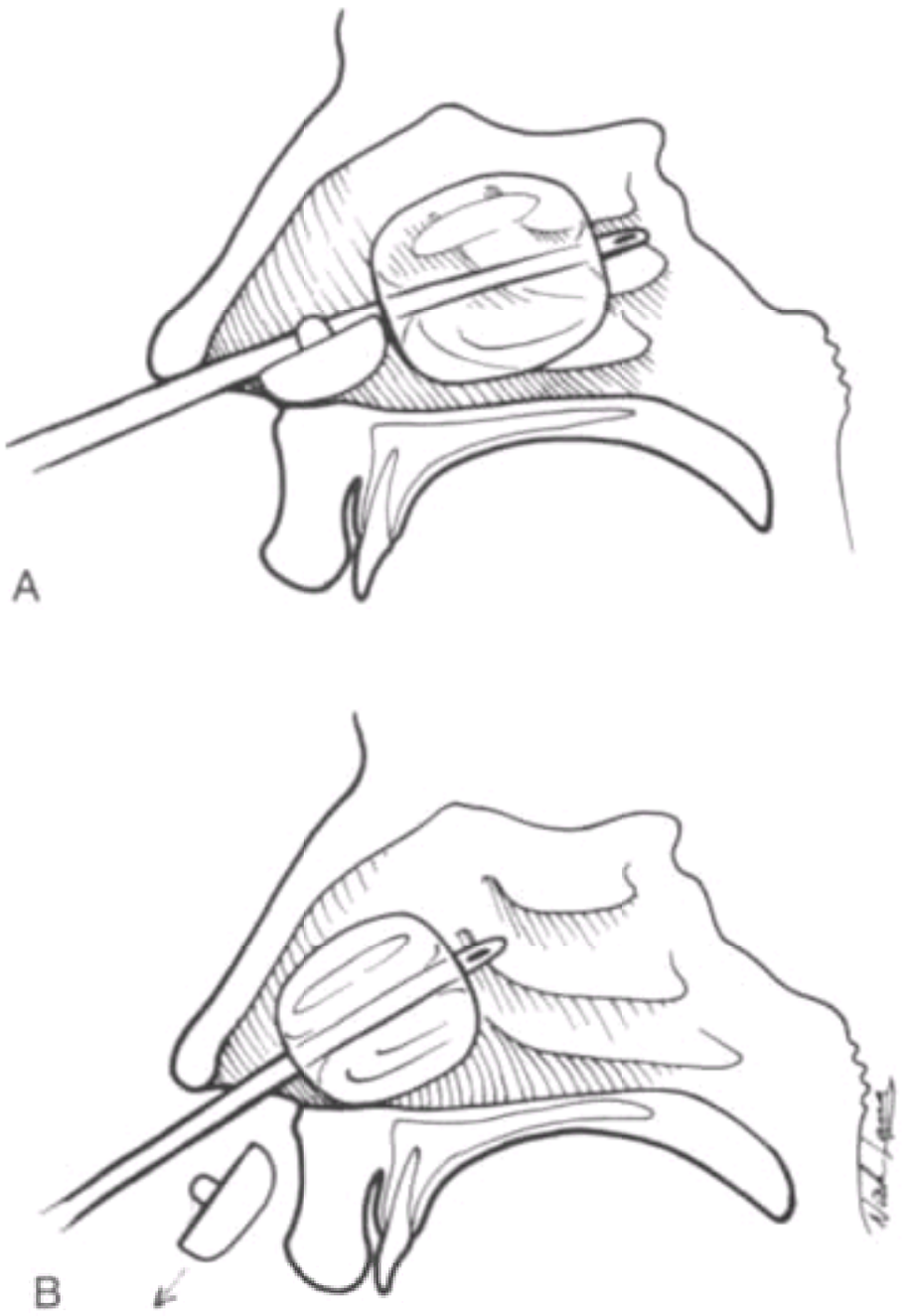


Figure 65-50 The Katz Extractor (InHealth Technologies, Carpinteria, CA) for oto-rhino foreign body removal shown with balloon inflated.

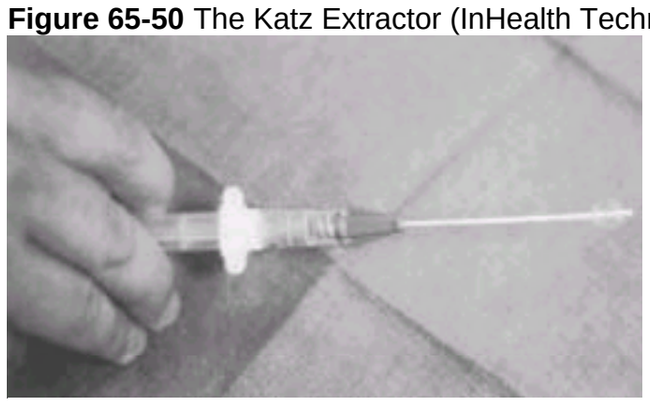


Figure 65-51 Bag-valve-mask technique to blow foreign body out of the naris. Ensure that the face mask forms a tight seal around the patient's mouth and that the unaffected nostril is completely occluded. Attempt to firmly compress the bag as the patient exhales (an assistant is helpful to hold the mask snugly and to occlude the other nostril). This technique works best with objects that completely occlude the nostril.

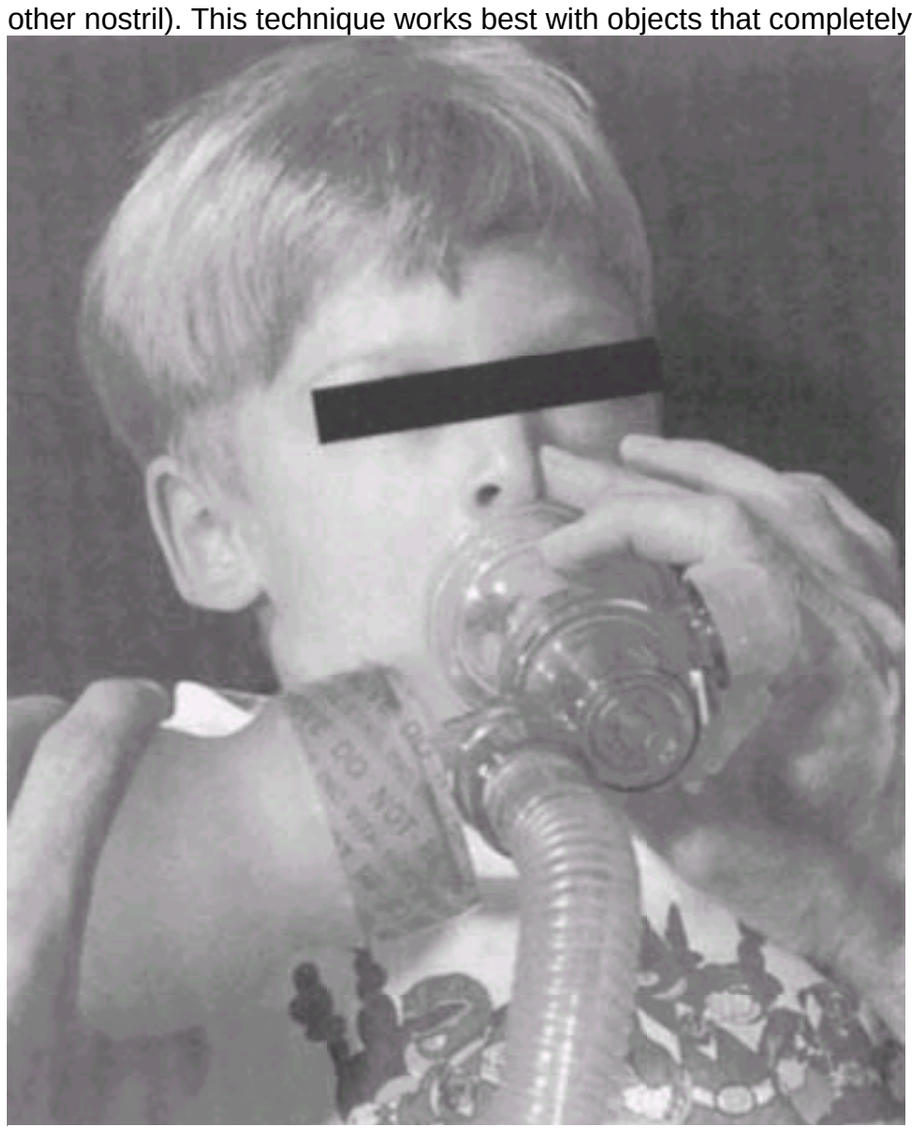


Figure 66-1 Muscles responsible for closing and excursive mandibular movements. Sagittal skull views illustrating the anatomical positions of the following muscles: *A,* Temporalis. *B,* Superficial masseter. *C,* Deep masseter. *D,* Medial pterygoid. *E,* Lateral pterygoid. *(From King R: Oral-Facial Emergencies-Diagnosis and Management, 1st ed. Portland, OR, JBK Publishing, 1994.)*

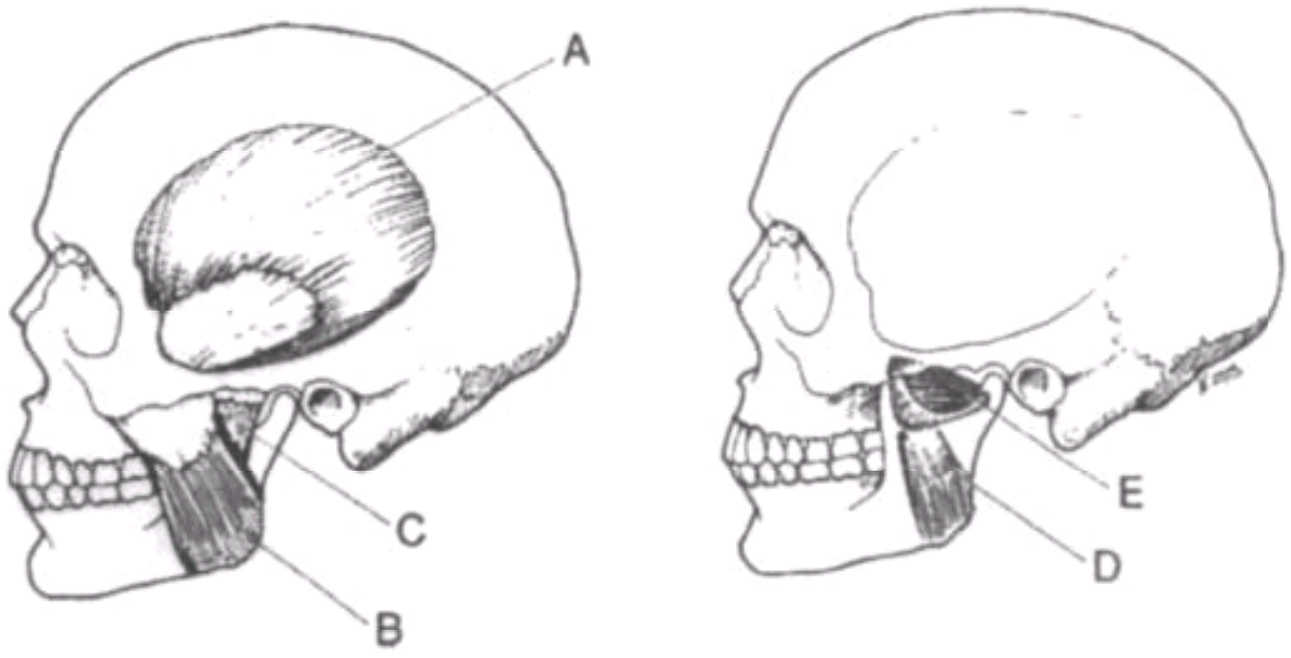


Figure 66-2 Muscles responsible for mandibular opening. Oblique skull views illustrating the anatomical positions of the following muscles: *A,* Mylohyoid. *B,* Anterior belly of the digastric. *C,* Posterior belly of the digastric. *D,* Geniohyoid. *(From King R: Oral-Facial Emergencies-Diagnosis and Management, 1st ed, Porland, Or: JBK Publishing, 1994.)*

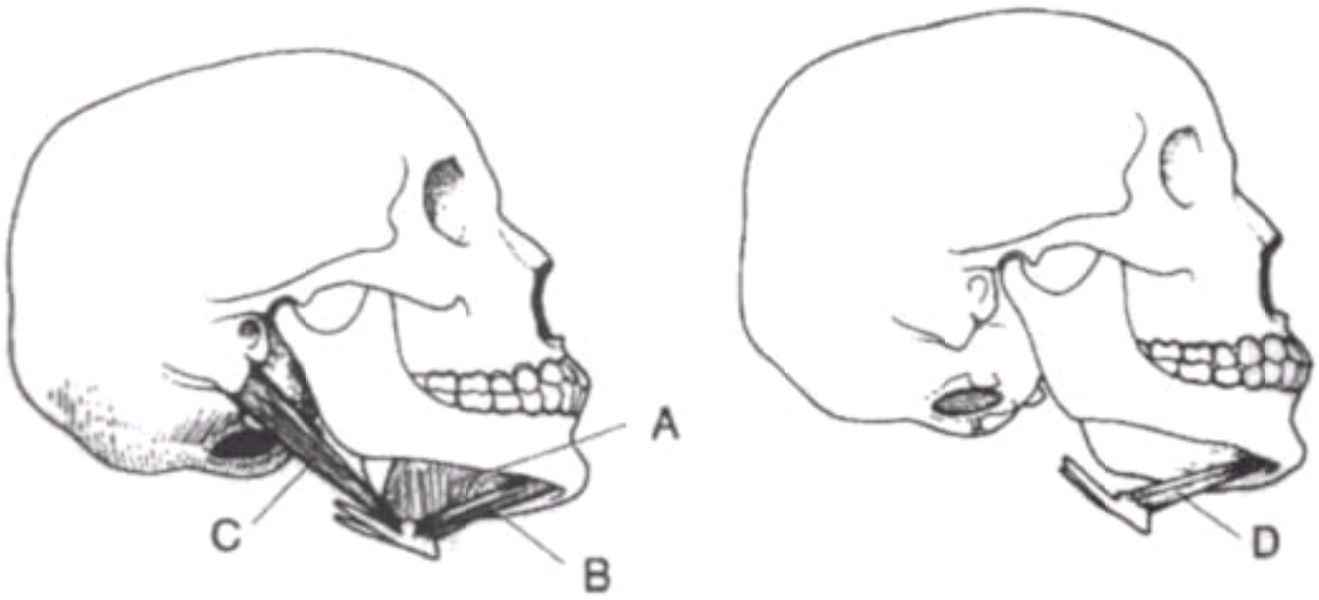


Figure 66-3 Axial view of the floor of the mandible. The arrows indicate the direction of pull of the lateral pterygoid, medial pterygoid, and mylohyoid muscles. *(From Eisele D and McQuone S: Emergencies of the Head and Neck, 1st ed. St. Louis, MO, Mosby, 2000.)*

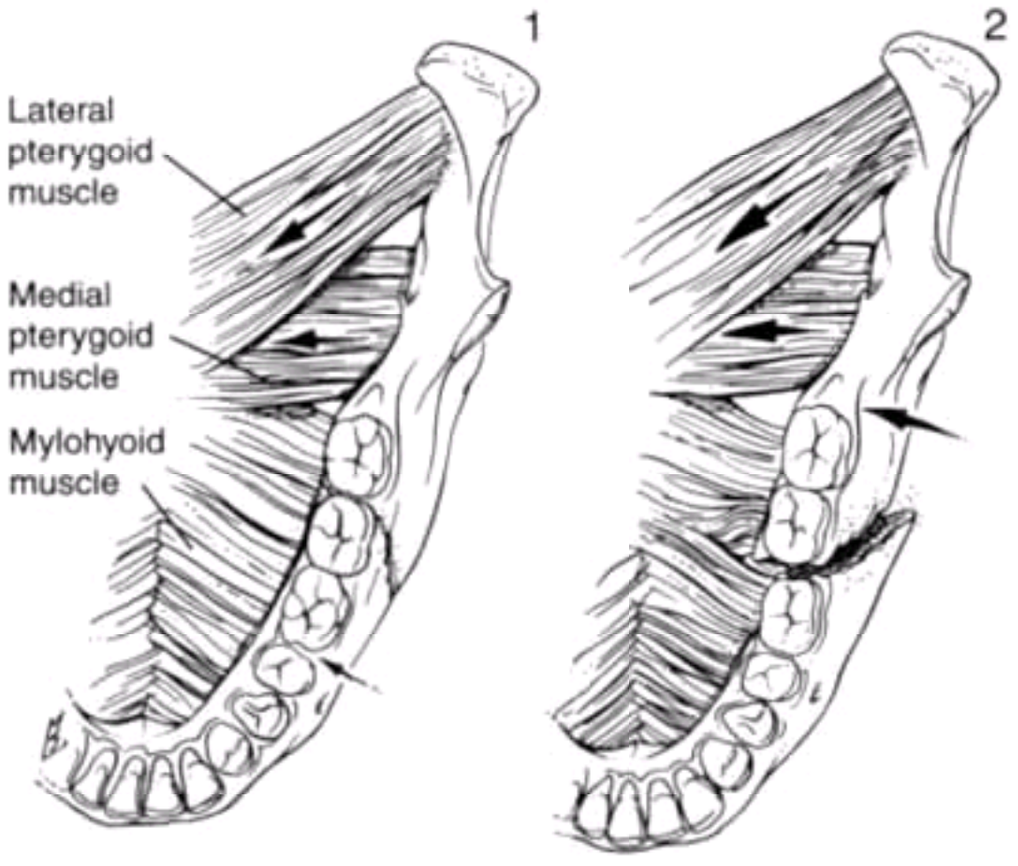


Figure 66-4 Anatomy of the mandible. *Top,* View from lateral (buccal) perspective. *Bottom,* View from medial (lingual) perspective. *(Redrawn from Grant JC: Grant's Atlas of Anatomy, 5th ed. Baltimore, Williams & Wilkins, 1962.)*

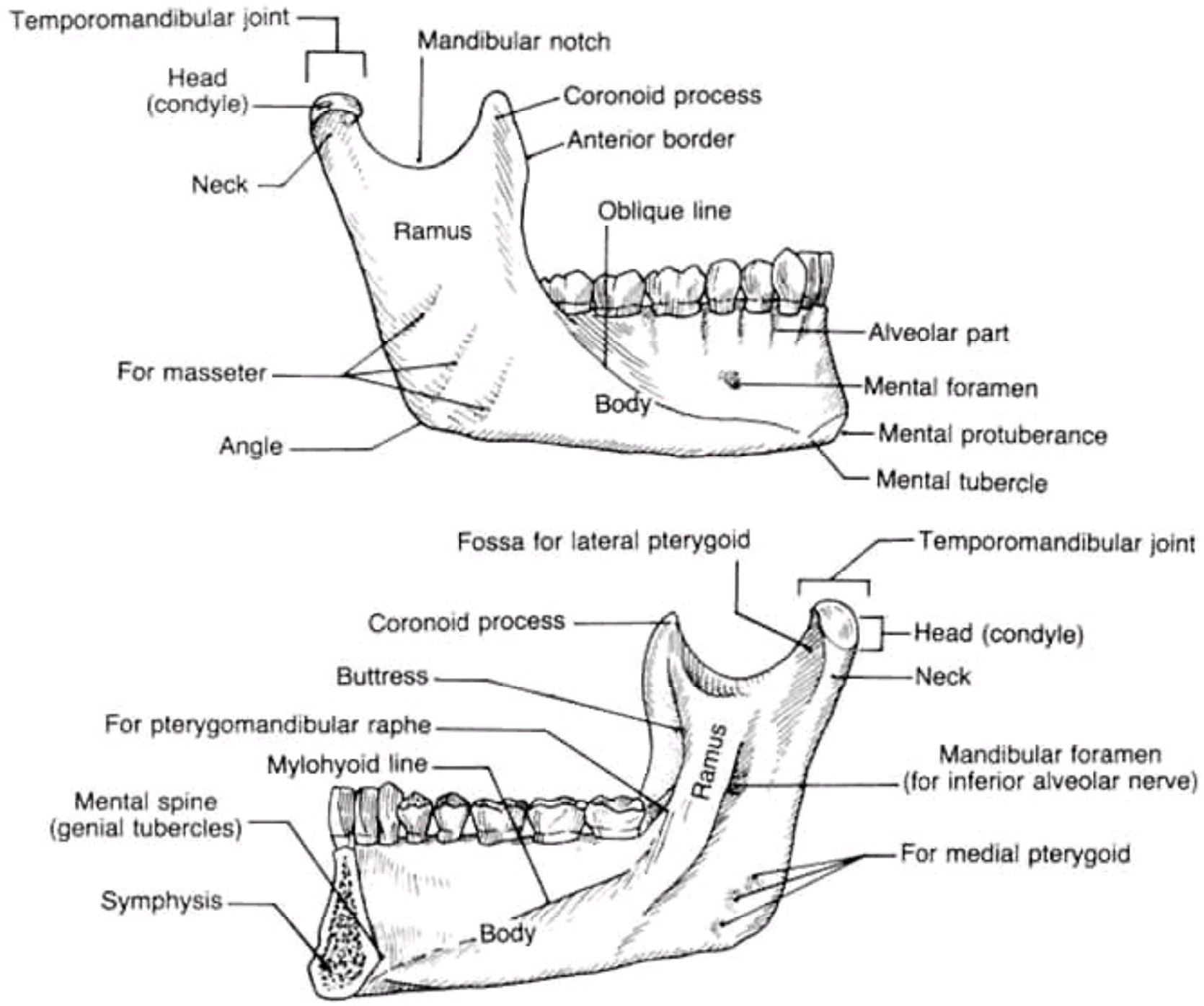
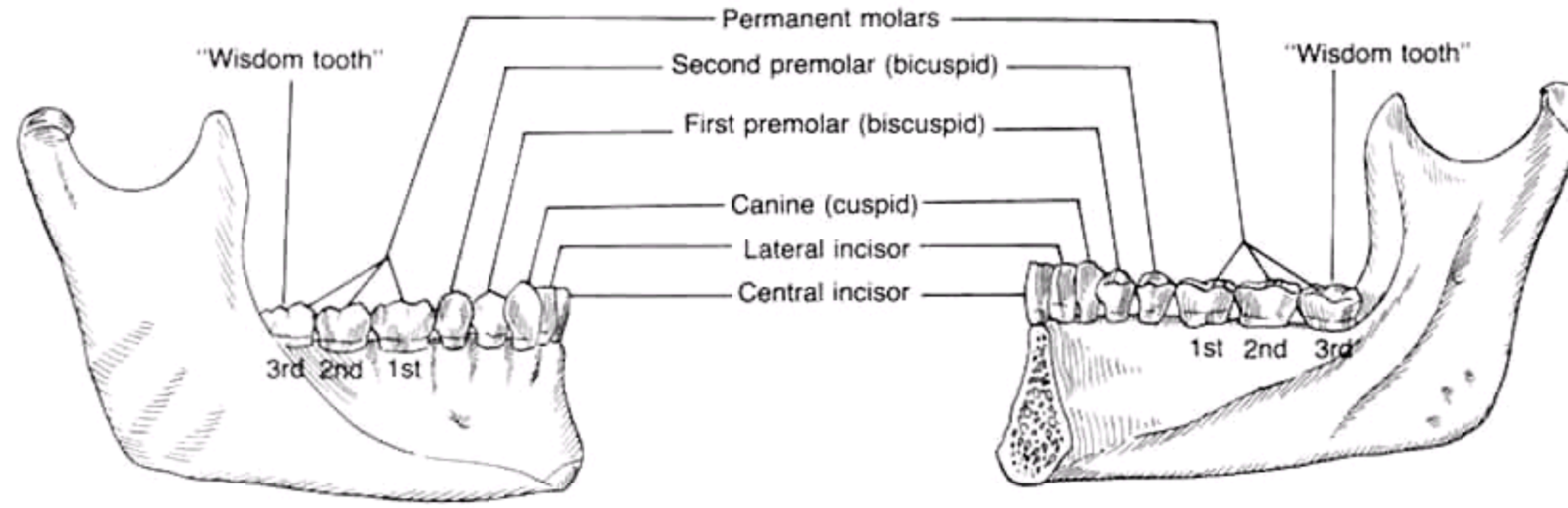


Figure 66-5 Classification of teeth. *Top left,* Lateral view. *Top right,* Medial view. *(Redrawn from Grant J, Basmajian J: Grant's Method of Anatomy, 7th ed. Baltimore, Williams & Wilkins, 1965.)*



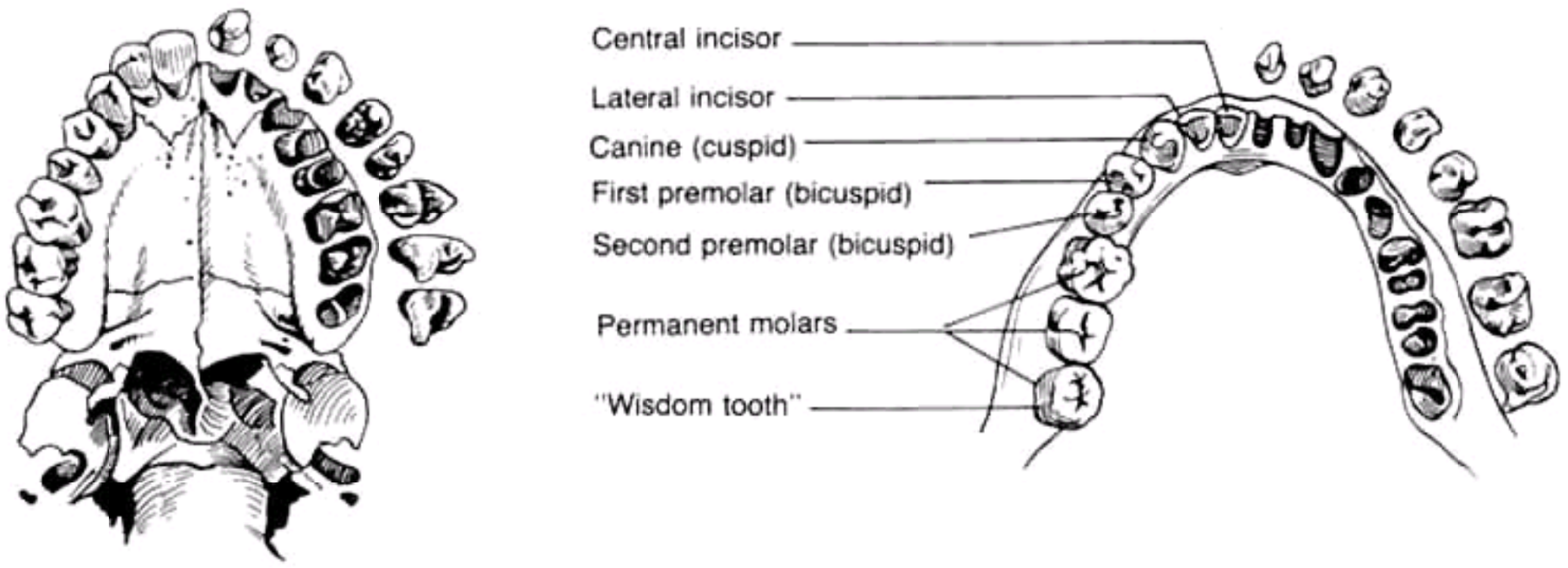


Figure 66-7 The dental anatomic unit.

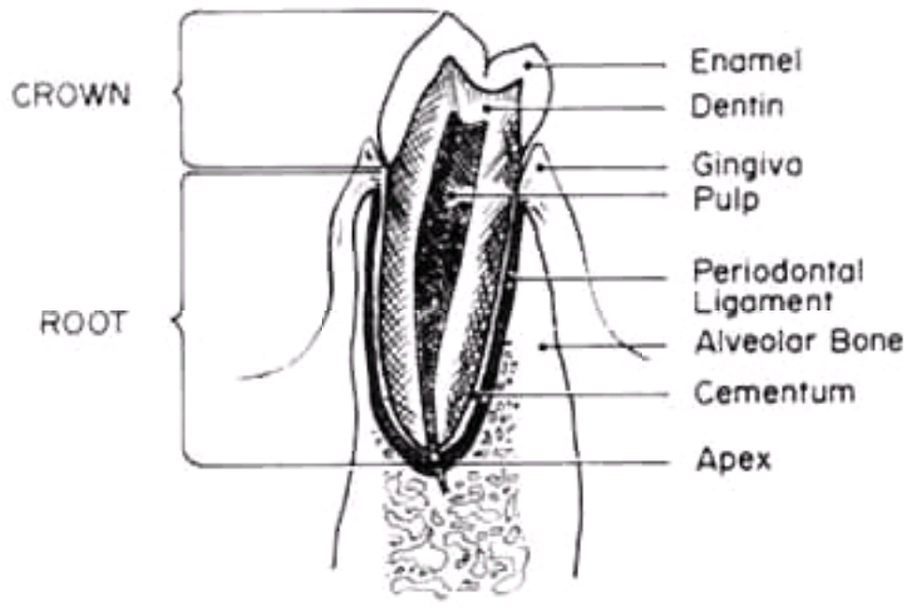


Figure 66-8 The Ellis classification for fractured alveolar teeth. The easiest method to classify fractured teeth is by description (e.g., fracture through the dentin of the first upper right molar).

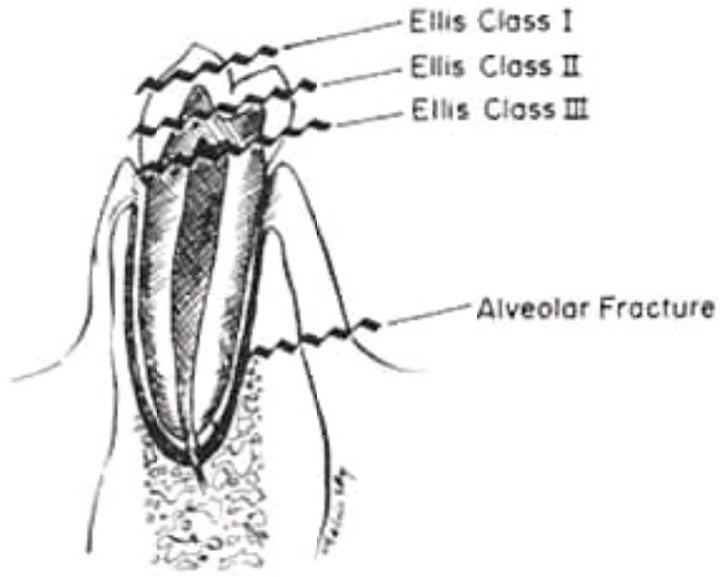
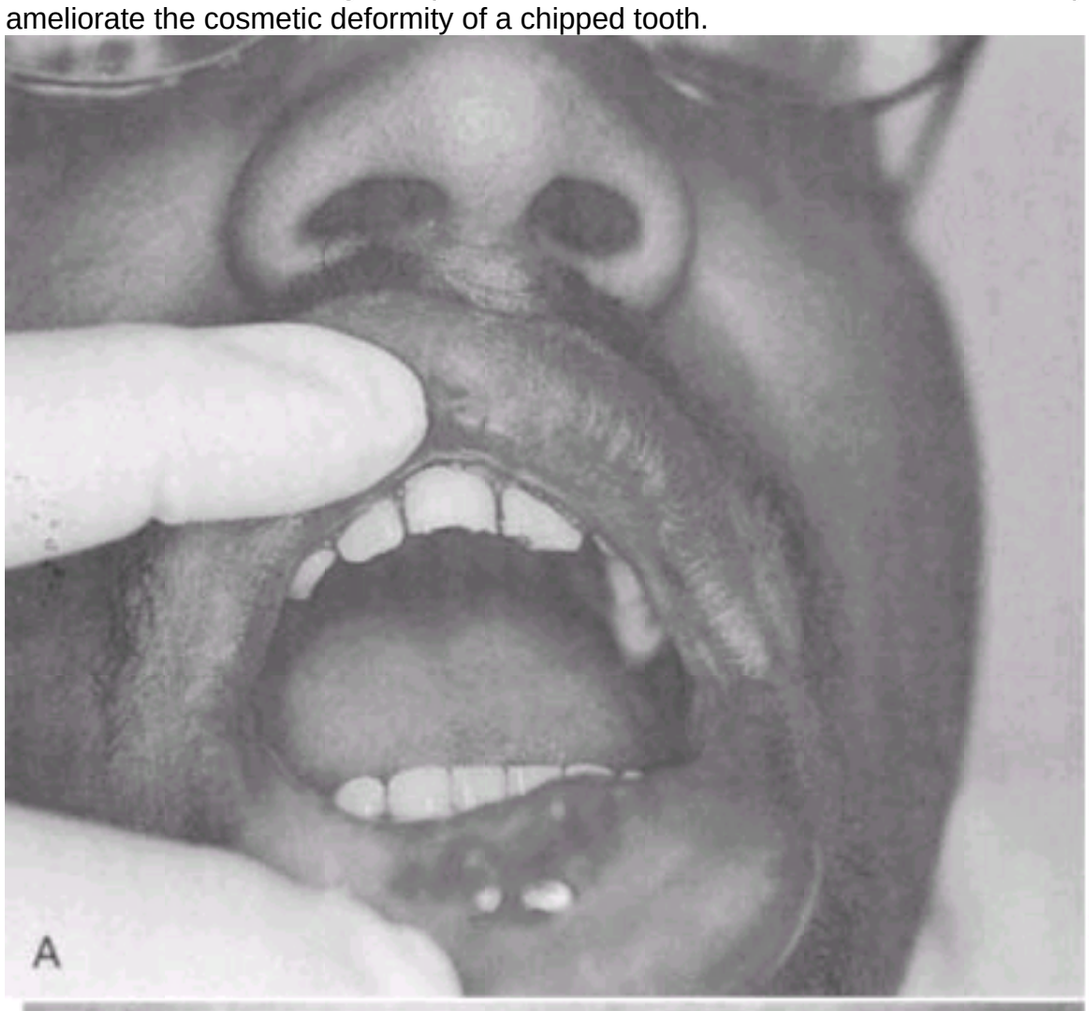
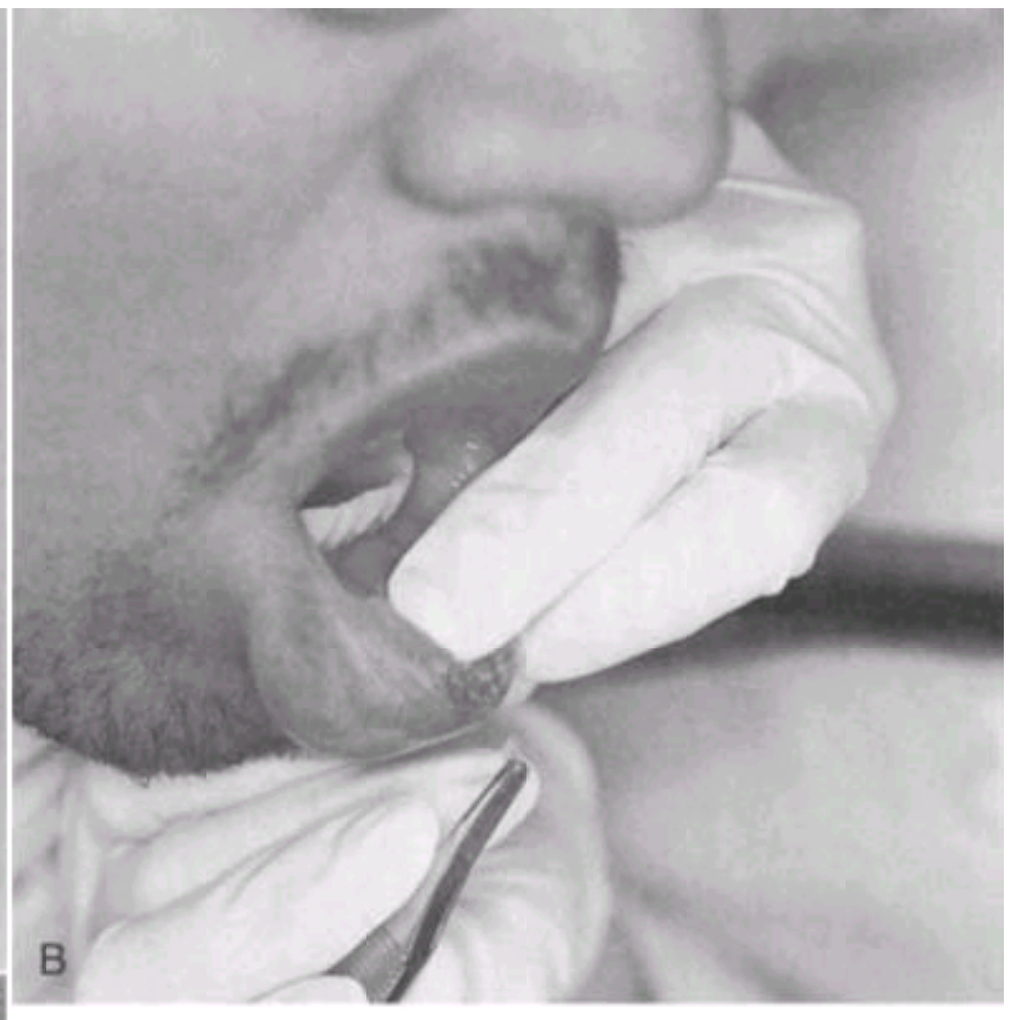


Figure 66-9 *A,* A common injury from a fist to the mouth results in a chipped tooth (here, fractured through the enamel only) and a laceration of the lip. One should always explore the mucosal laceration, looking for the missing piece of tooth. *B,* When this lip laceration was explored, a piece of tooth was found embedded within the laceration. If this foreign body is not removed, an infection is certain a few days later. *C,* Smoothing the enamel with a disc sander or an emory board will ameliorate the cosmetic deformity of a chipped tooth.





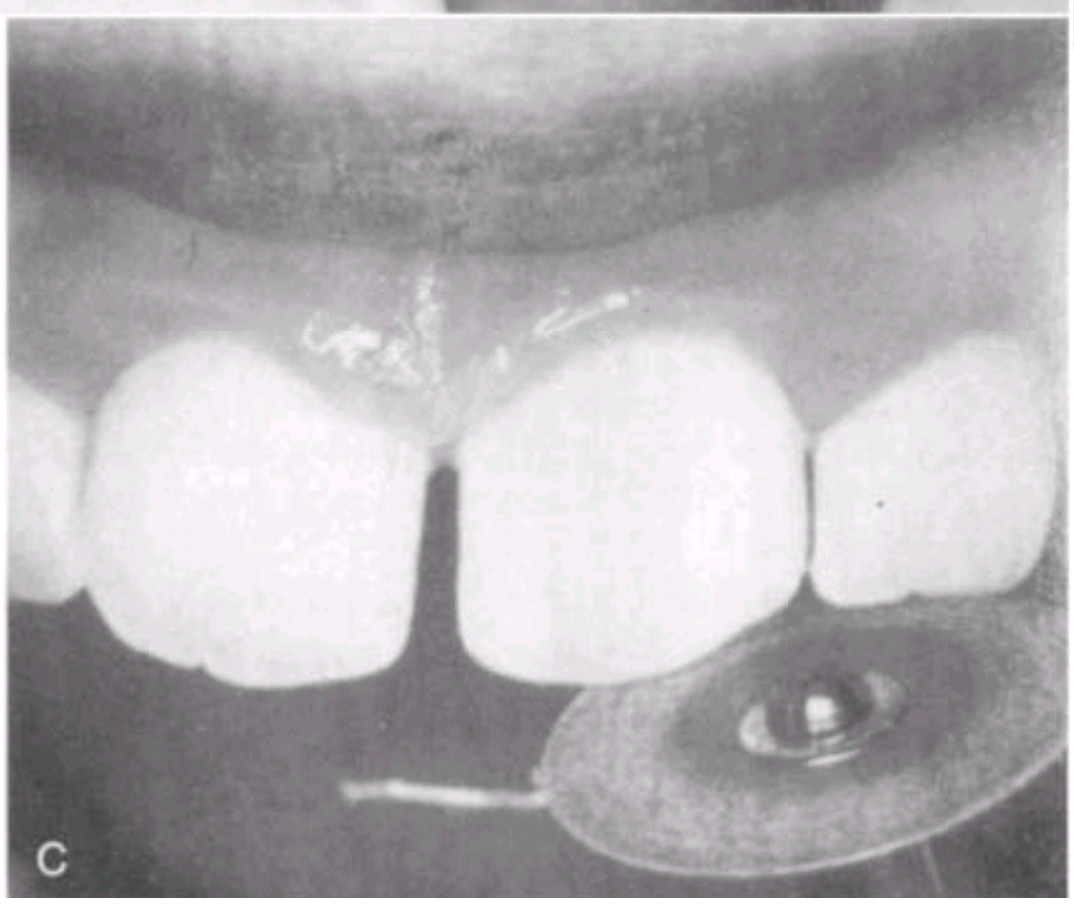


Figure 66-10 A fracture through the dentin.

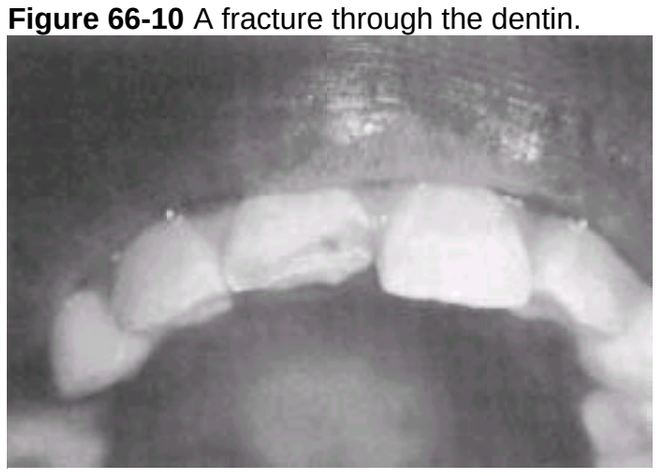


Figure 66-11 *A,* Calcium hydroxide paste is one acceptable material to cover dentin or pulp fractures. *B,* Mixing the calcium hydroxide paste with a spatula on a mixing pad. *C,* Application of the periodontal paste to the fractured surface of the tooth.

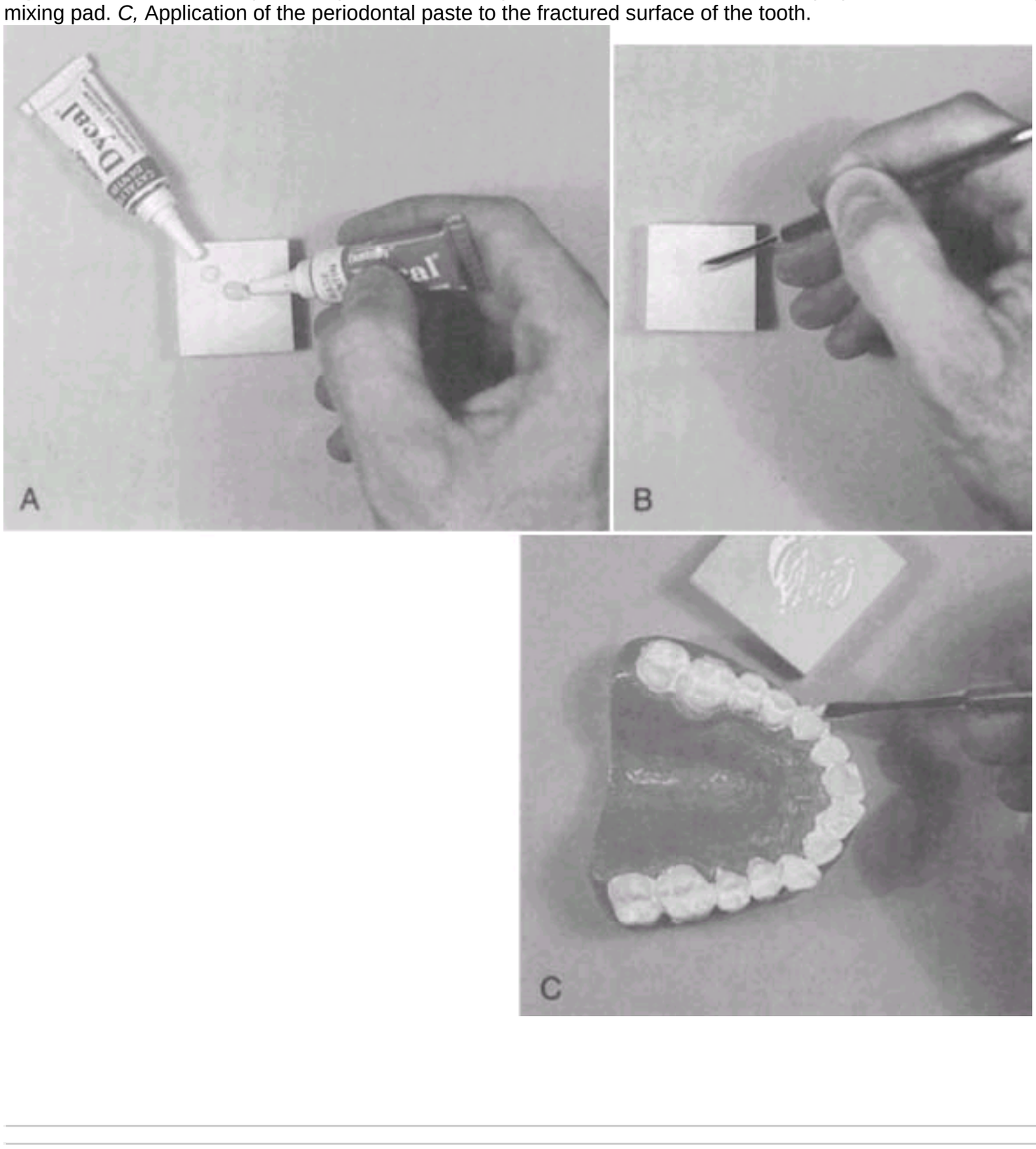


Figure 66-12 *A,* Covering the pulp fracture with calcium hydroxide paste. *B,* The calcium hydroxide paste hardens quickly in the moist environment of the mouth.

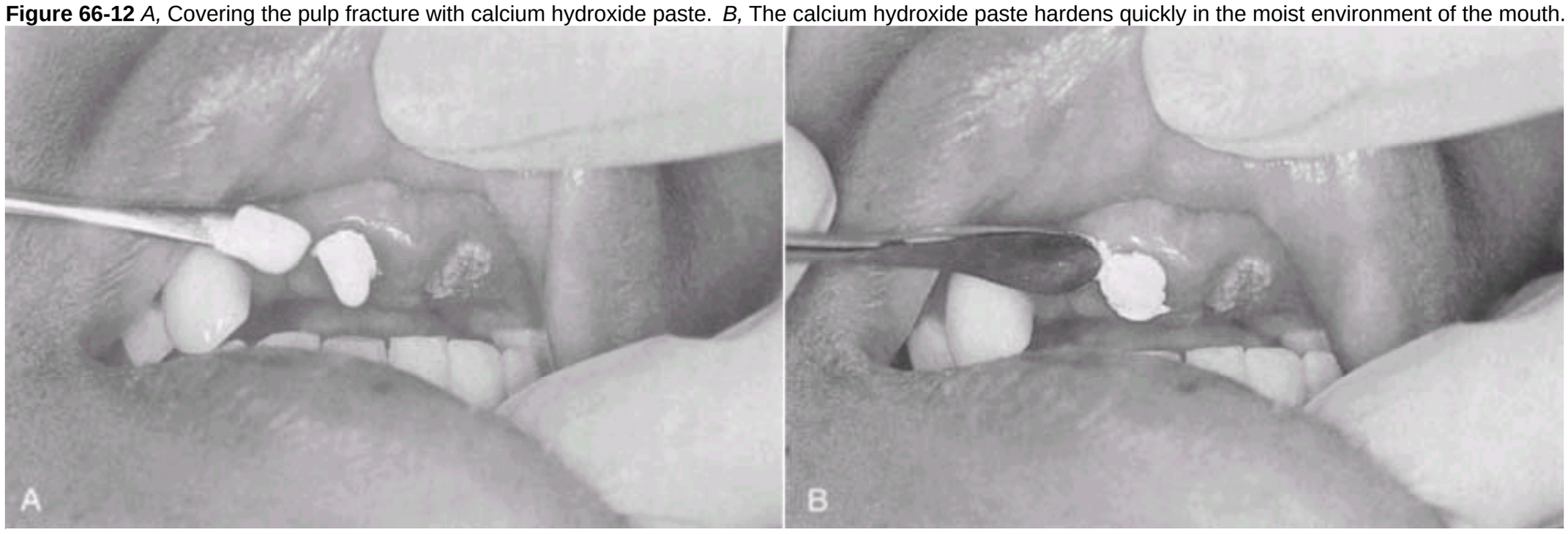


Figure 66-13 Extrusive luxation occurs when the tooth is forced partially out of the socket in an axial direction. *(From King R: Oral-Facial Emergencies-Diagnosis and Management, 1st ed. Portland, OR, JBK Publishing, 1994.)*

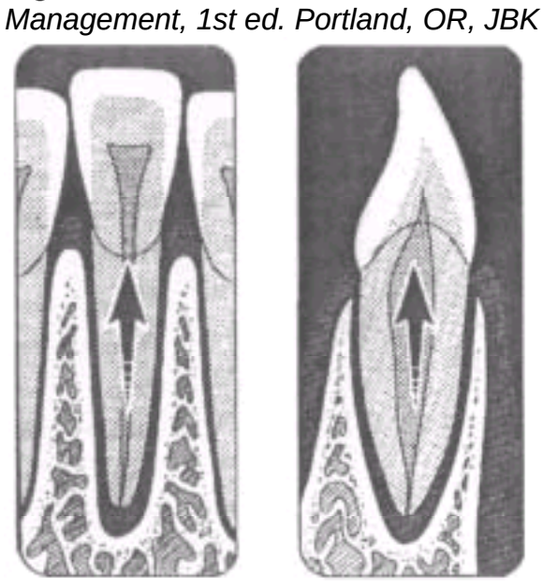


Figure 66-14 Intrusive luxation of a tooth compresses the periodontal ligament and vascular supply of the pulp. It may even crush the apical bone. *(From King R: Oral-Facial Emergencies-Diagnosis and Management, 1st ed. Portland, OR, JBK Publishing, 1994.)*

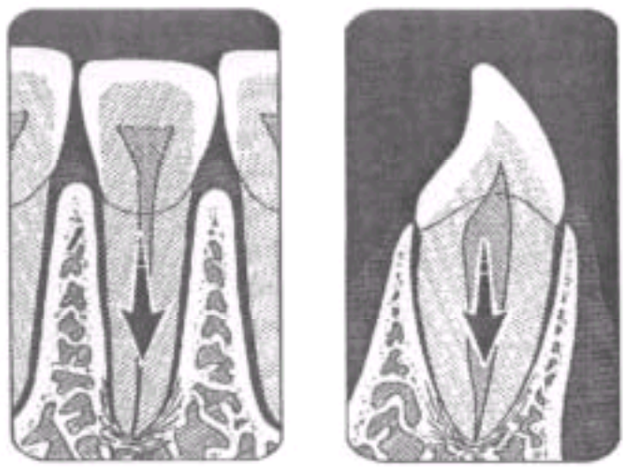


Figure 66-15 Intruded tooth secondary to trauma. On superficial examination it appears that the tooth was simply knocked out. A dental radiograph or CT scan is necessary to determine intrusion or avulsion. Intruded teeth create the potential for infection or cosmetic deformities. Intrusion of an upper tooth into the maxillary sinus can cause recurrent sinusitis. Teeth can also intrude into the nasal cavity and cause infection or bleeding, or they can be aspirated into the airway. The incisors are the most commonly intruded teeth. *(From Johnson R: The treatment of the traumatized incisor in the child patient. In University of Pennsylvania School of Dental Medicine: Continuing Dental Education, vol 2. Philadelphia, University of Pennsylvania, 1978.)*

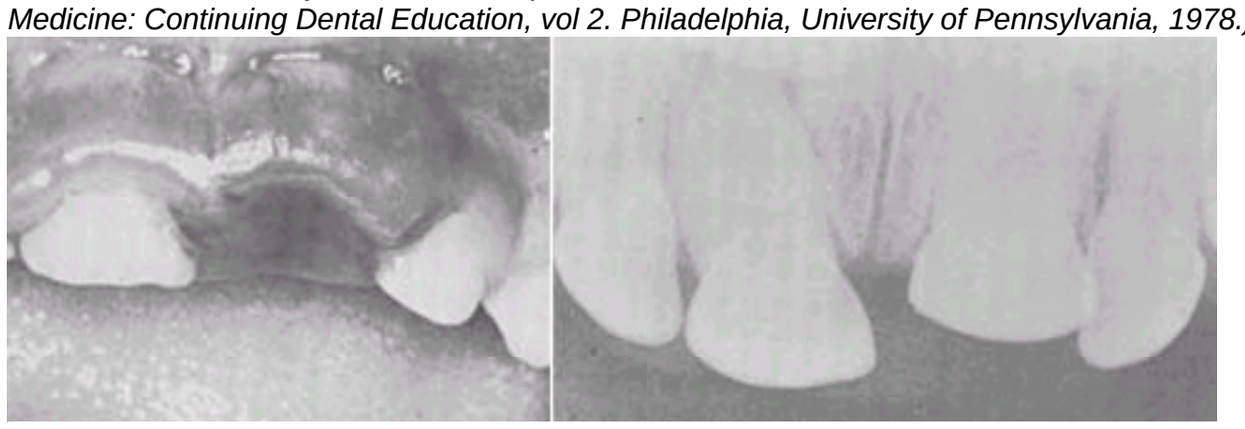


Figure 66-16 Lateral luxation occurs when the tooth is displaced in either a lingual, mesial, distal, or facial direction. Fractures of the alveolus frequently accompany lateral luxation injuries. *(From King R: Oral-Facial Emergencies-Diagnosis and Management, 1st ed. Portland, OR, JBK Publishing, 1994.)*

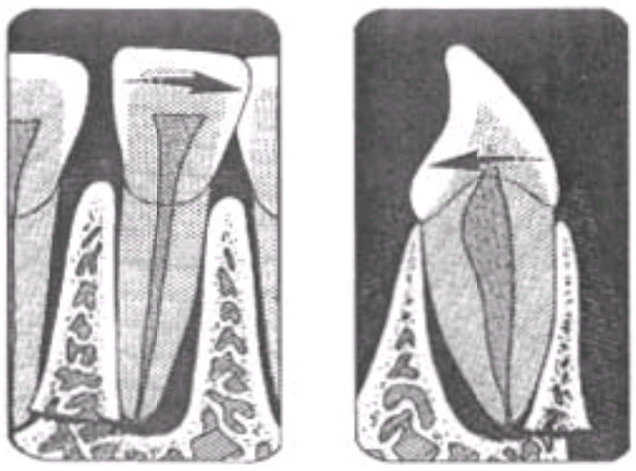


Figure 66-17 Application of Erich arch bars. *A through C,* A commercially available arch bar is shaped to fit the maxillary and mandibular arches. *D and E,* Stainless steel wire secures the bar to the necks of the teeth. *F,* The maxilla and the mandible are brought into occlusion and held in place with rubber bands. This technique is generally not used by the emergency clinician, but this equipment should be available for the oral or maxillofacial surgeon. *(From Converse JM: Reconstructive Plastic Surgery, 2nd ed, vol 2. Philadelphia, WB Saunders, 1977.)*

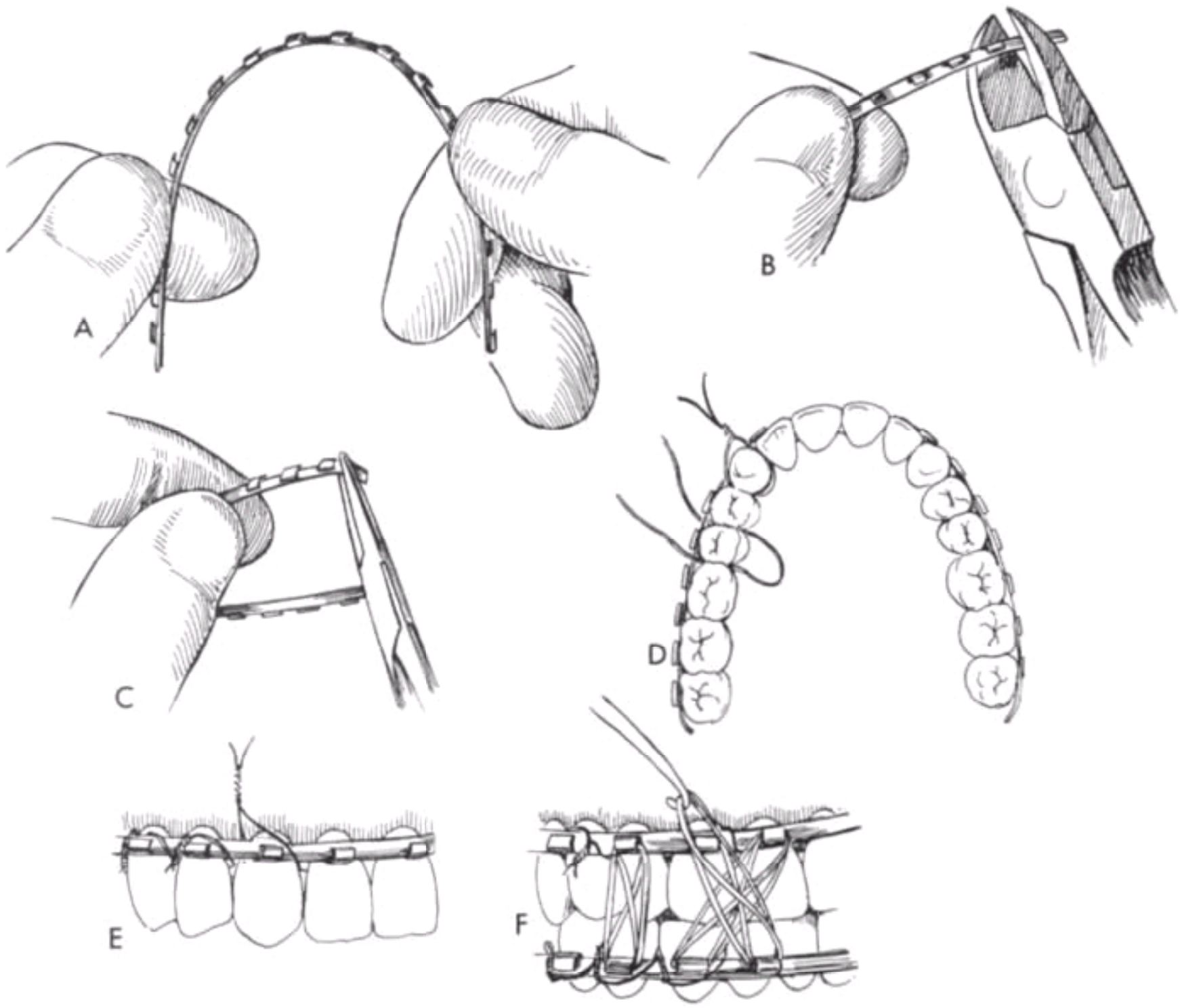


Figure 66-18 *A,* Squeeze out equal-sized ribbons of the periodontal paste. *B,* Mix the base and catalyst together with a tongue blade. *C,* Using moistened gloves, apply the paste to the dry enamel and gingiva.

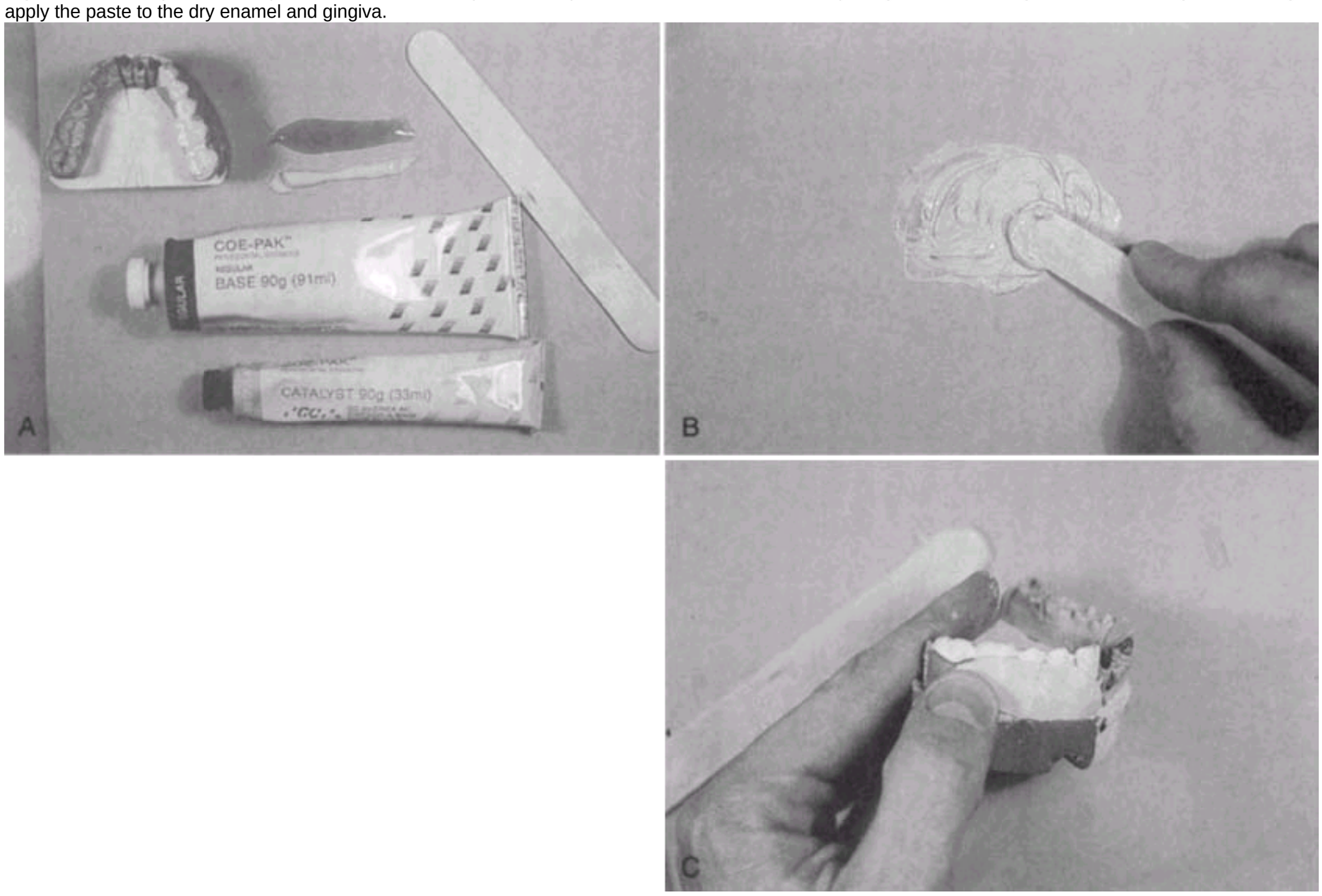


Figure 66-19 Application of periodontal paste. *A,* Equal-length ribbons of base and catalyst are squeezed onto the mixing pad. *B,* The base and catalyst are mixed together using a moistened spatula or tongue blade. *C and D,* Using moistened gloves, roll the mixture into a ribbon. *E through H,* Apply to the enamel and gingiva, extending the paste two teeth on each side of the loosened tooth. The paste adheres best when the surface of the enamel and gingiva are as dry as possible.

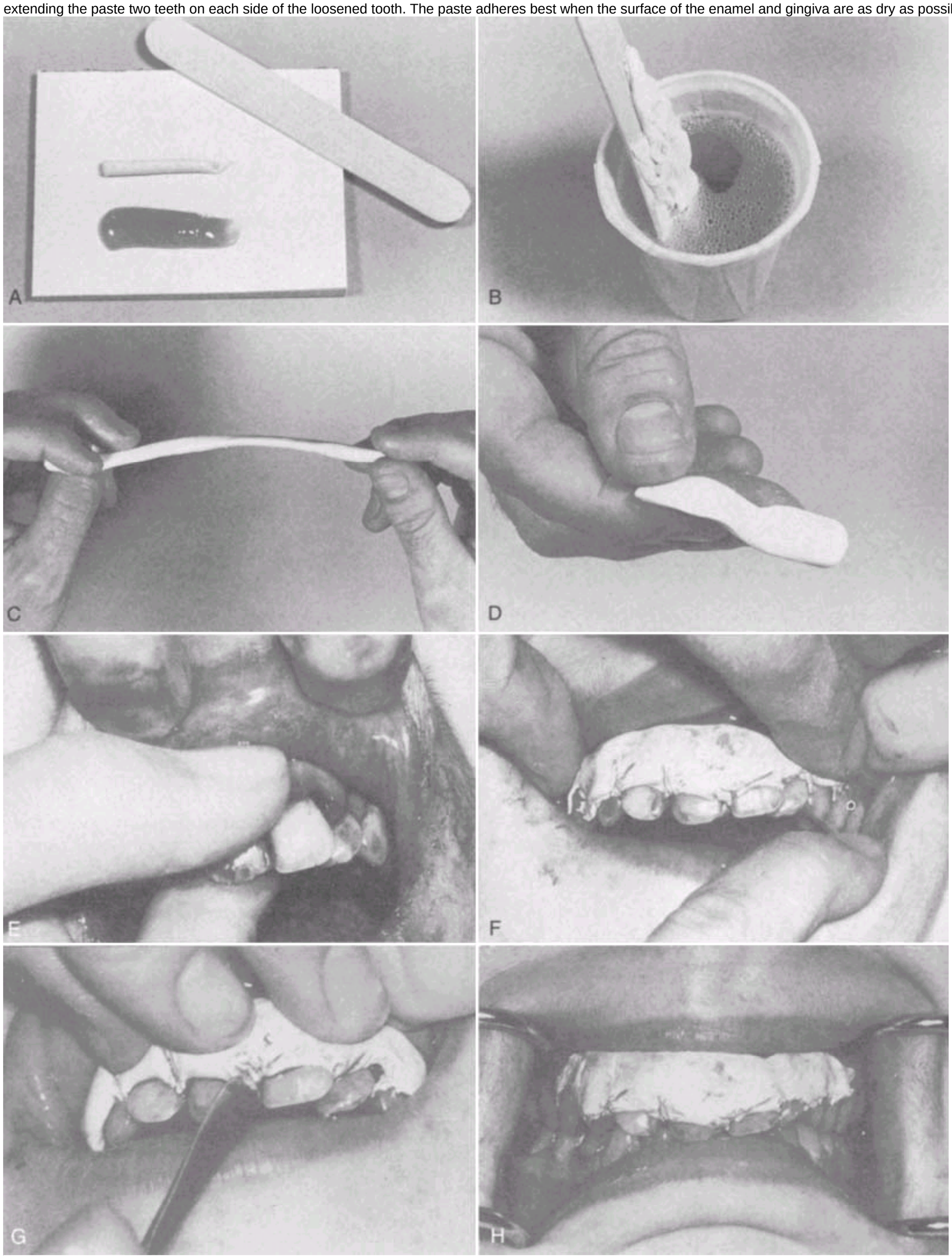


Figure 66-20 *A and B,* Equal amounts of the self-cure composite are placed on the mixing pad and mixed together. *C,* Subluxed teeth should be repositioned prior to application of the composite. *D,* The mixture is applied only to the dry enamel of the teeth. The composite should extend to one tooth on each side of the loosened tooth.

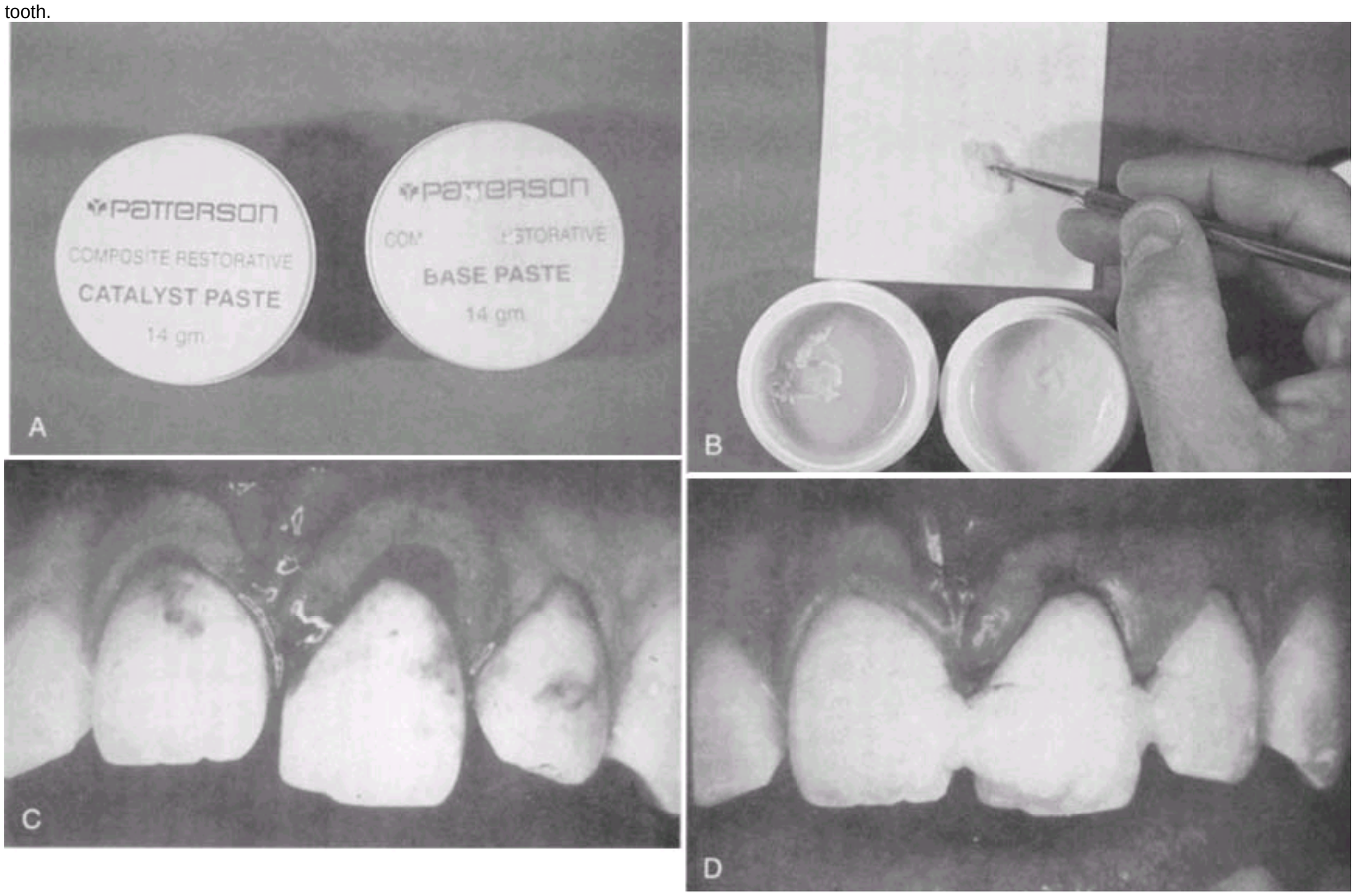


Figure 66-21 Using the "Save A Tooth" system, a tooth is placed into the container and closed. The preservative will increase the life span of traumatized periodontal ligament cells.

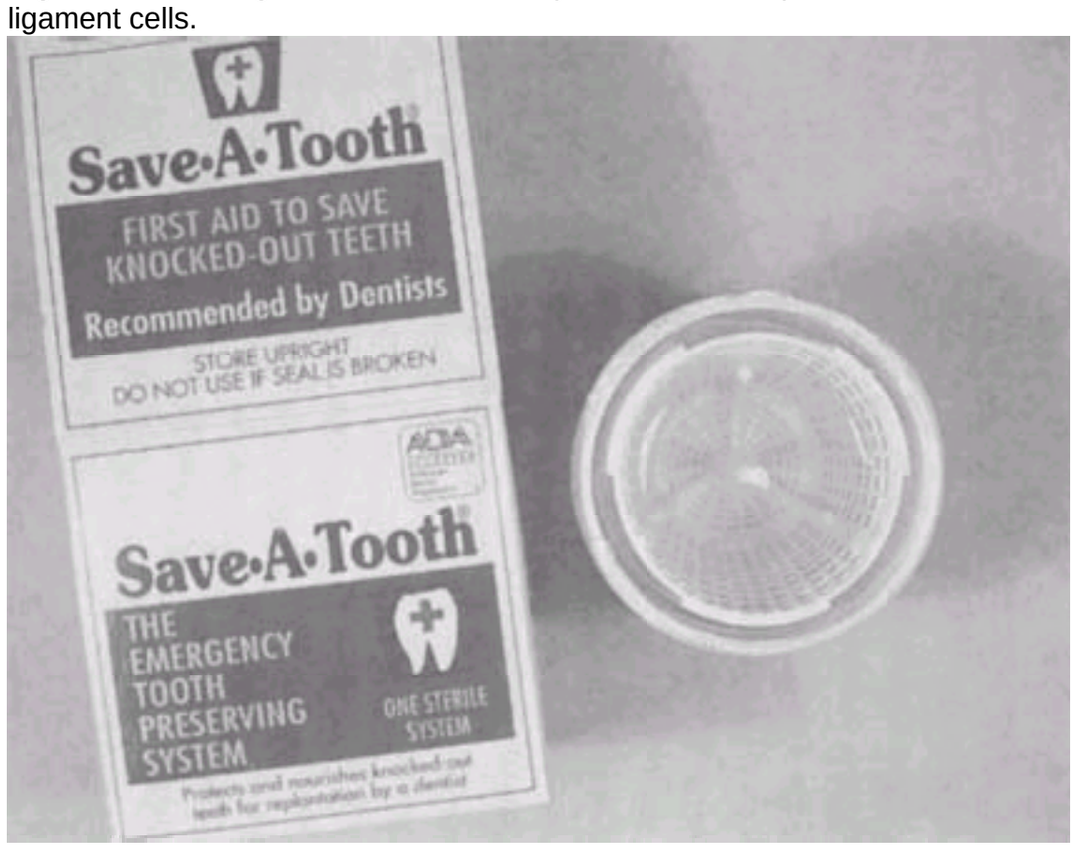


Figure 66-22 The Ora 5 Topical Bactericidal Agent can be applied to intraoral lacerations or injuries to help prevent infection.

Figure 66-23 *A,* Gingival lacerations sometimes leave little tissue for approximation. *B,* The teeth can be used as anchors for sutures and help approximate the lacerated tissue. *C,* Gingival lacerations usually heal rapidly. (See also Fig. 66-24 .)

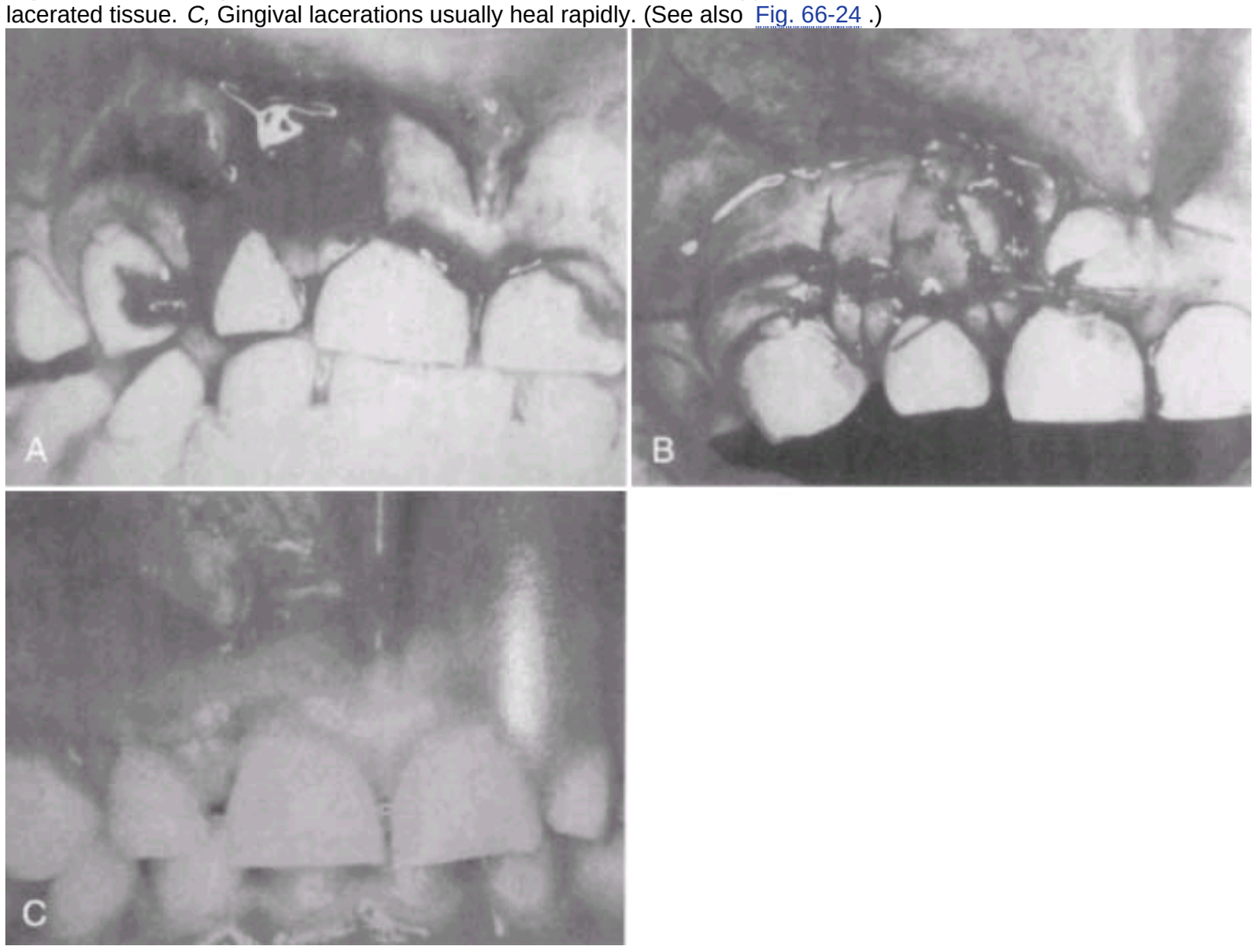
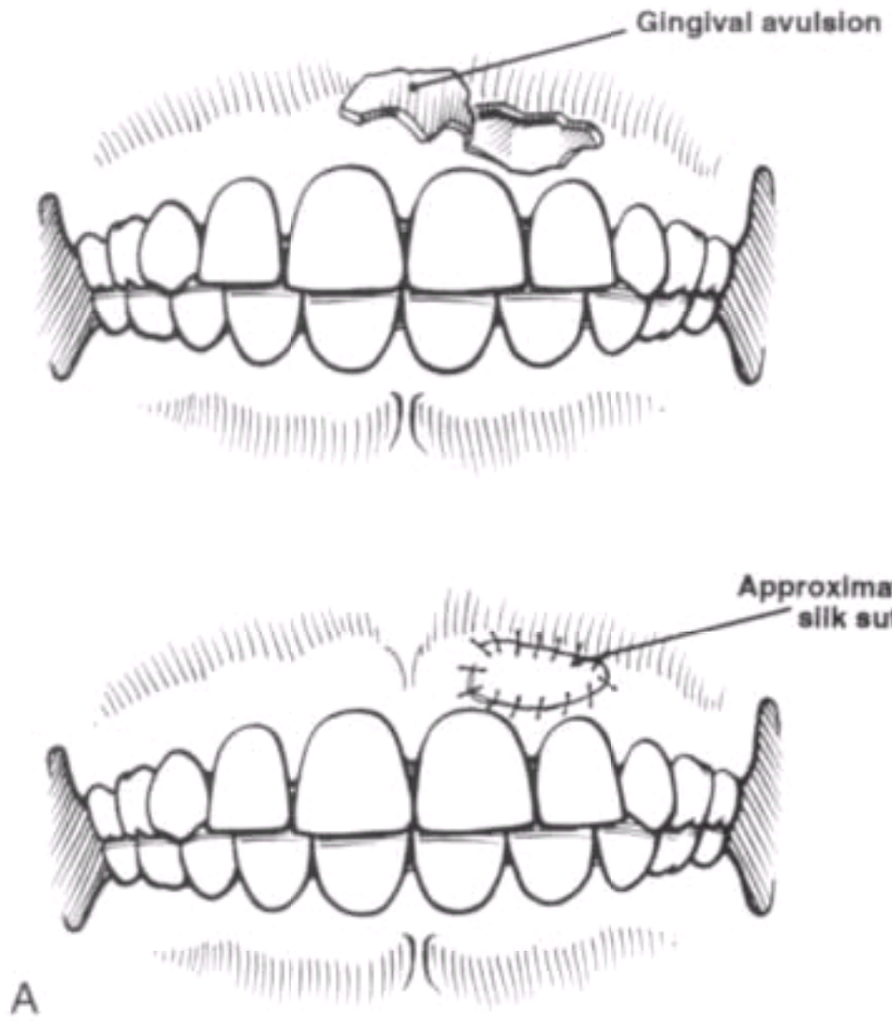
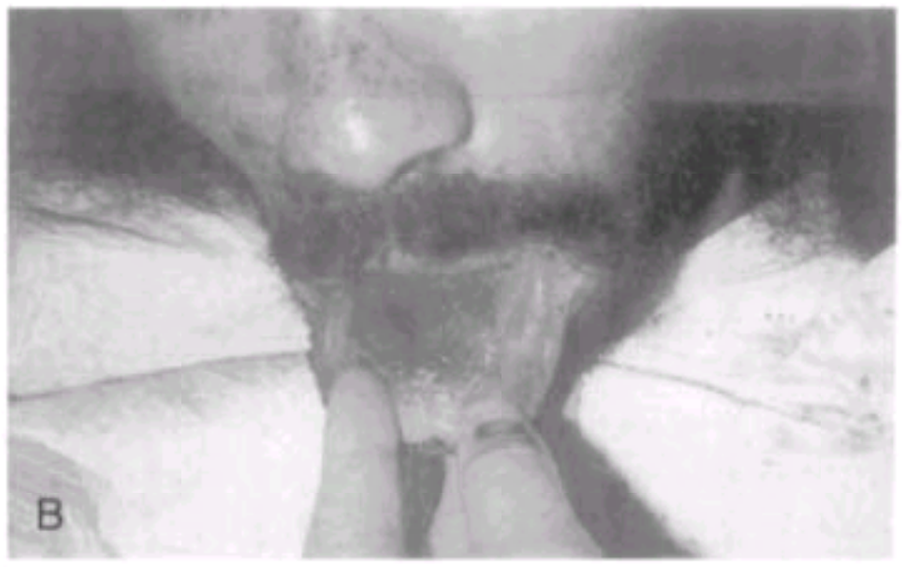


Figure 66-24 Repair of avulsed or lacerated gingiva. *A,* simple flap lap lacerations are approximated and closed with interrupted soft sutures, such as dexon or vicryl. *B,* Large gingival avulsions should be approximated to an anatomical position with interrupted sutures. *C1,* The exposed roots of the teeth should be covered. The thin and friable avulsed gingiva can not be sutured to the remaining gingiva or submucosal tissue. *C2,* The suture begins on the outer surface of the avulsed flap and is passed behind an anchoring tooth, like dental floss. The underside of the avulsed segment is then entered by the suture needle so the needle exits on the gingival surface. *C3,* Sutures pull the gingival to an anatomical position to cover the roots of the teeth and are tied on the outer surface. Sutures are removed in 5–7 days. See also Figure 66-23 .





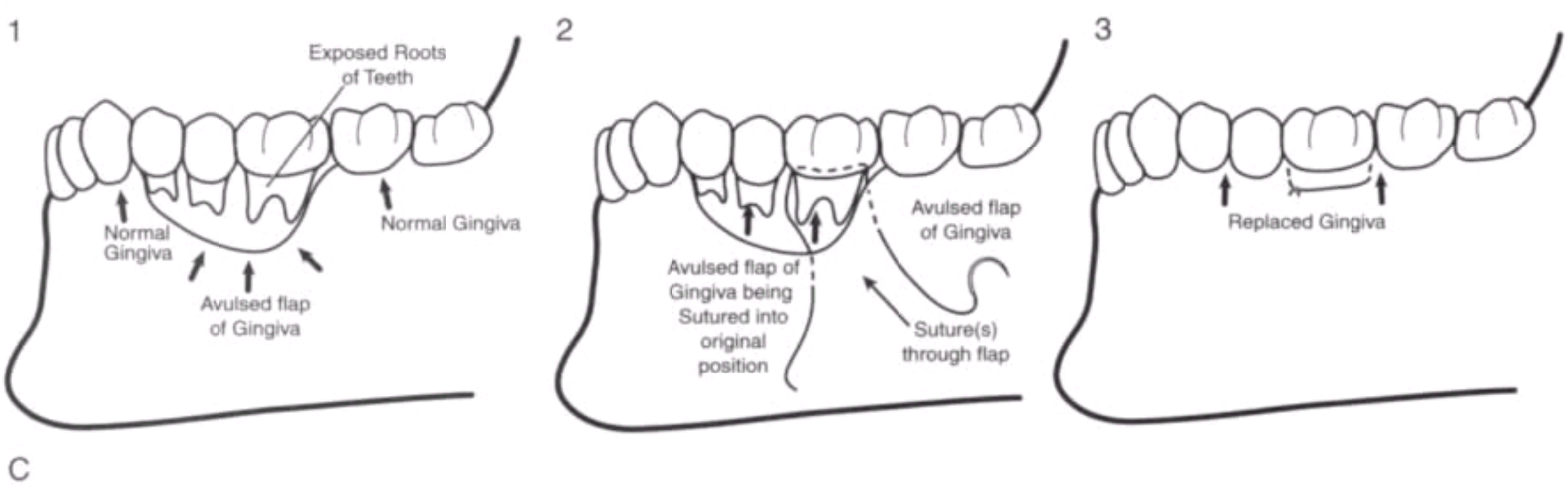


Figure 66-25 Dry Socket Paste. Apply alone or as a slurry mixed with Gelfoam into the socket to relieve alveolar osteitis pain.

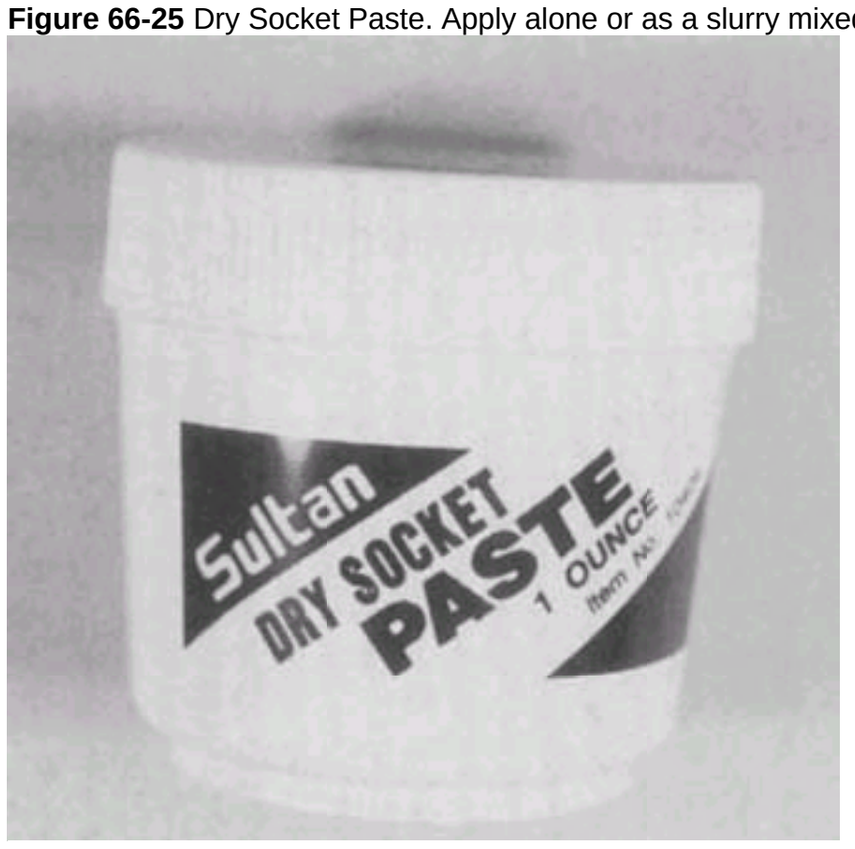


Figure 66-26 Equipment for incision and drainage of dental infection. From left to right on lower row. Cotton-tipped applicators, dental (aspirating) syringe with local anesthesia, topical anesthetic and 2 × 2 gauze, scalpel with No. 11 blade, hemostat, iris scissors, gauze (Nu-gauze) packing, and needle holder.

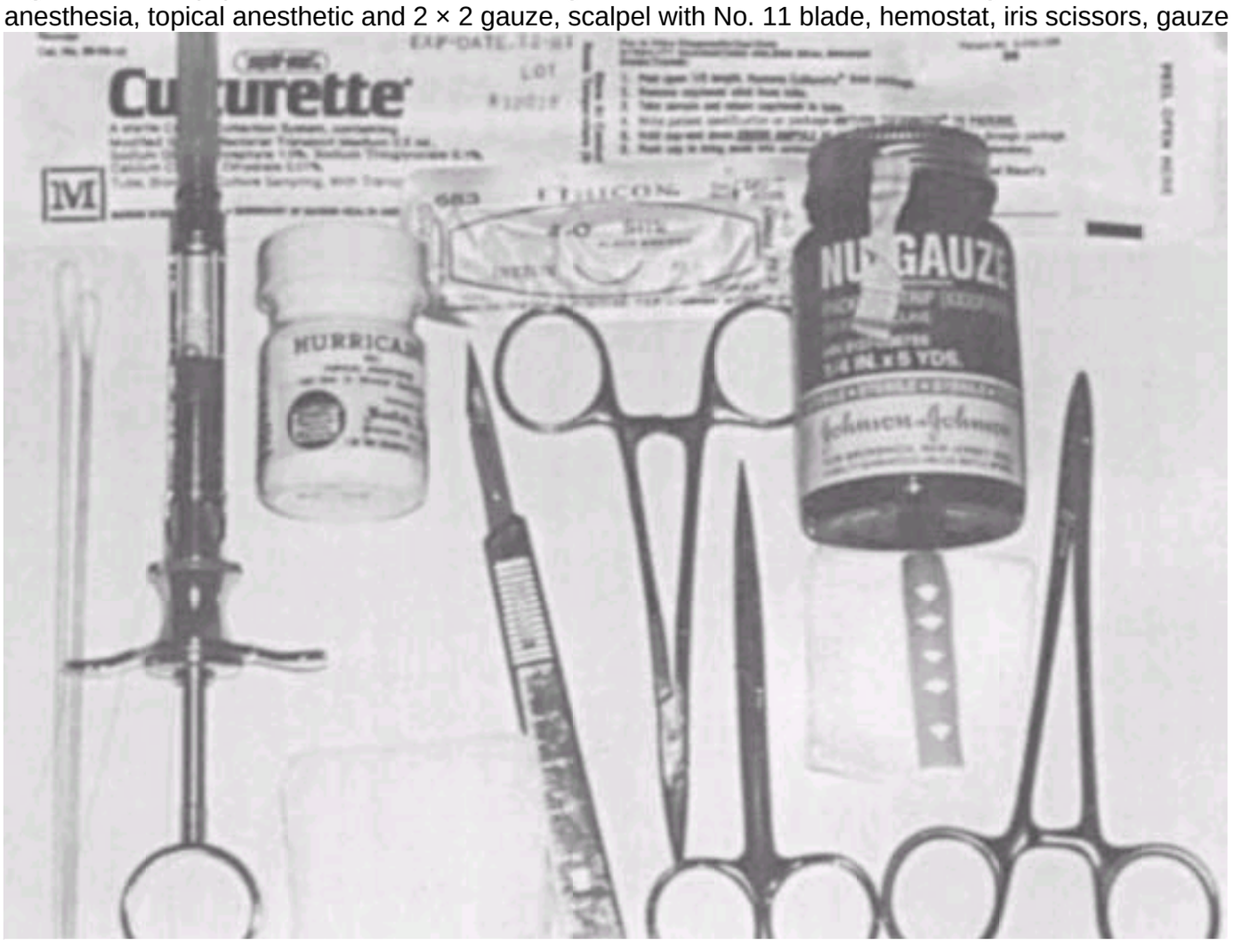


Figure 66-27 *A,* This patient presented with facial swelling up to the right eye. She had minor tooth pain but mostly complained of an ache in the face. X-rays revealed maxillary sinusitis with an air-fluid level. *B,* Intraoral examination revealed a pea-sized pointing abscess at the base of an upper tooth, the cause of sinusitis. *C,* Following local anesthesia, a blade incised the abscess, with drainage of copious pus. *D,* A hemostat was inserted into the abscess cavity to assure drainage, and a small piece of gauze was placed into the cavity. IV antibiotics (clindamycin) followed by oral antibiotics were given, the patient saw her dentist the next day, and plans to extract the tooth were made. She recovered fully.

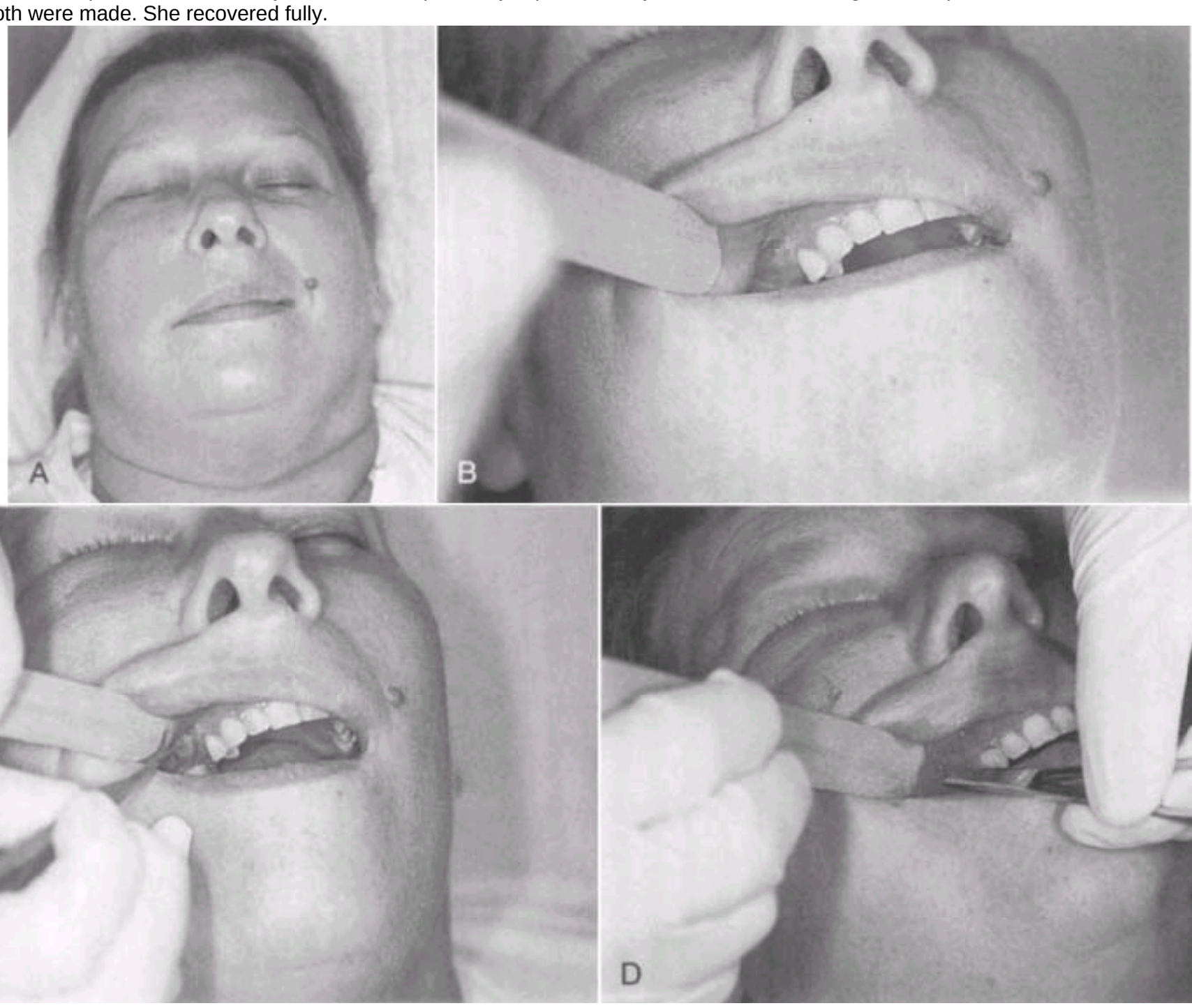


Figure 66-28 *A,* Most dental infections associated with facial swelling should be drained intraorally. Illustration of a facial abscess that points extraorally. *B through F,* Technique of cutaneous drainage in a second patient. *B,* Anesthetic infiltrated below abscessed area. *C,* Incision. *D,* Drainage with blunt dissection. *E,* Gauze packing. *F,* Gauze sutured in place. This step may be omitted if desired. *G and H,* Osteomyelitis of the jaw presenting as a dental problem. This patient had a toothache for weeks that had turned into an abscess pointing on the side of the face. She had been to several emergency departments and was given prescriptions for antibiotics. No history of trauma was forthcoming. *G,* An obvious pointing abscess over the mandible. *H,* A panorex x-ray revealed an old fractured mandible with osteomyelitis draining into the soft tissues.

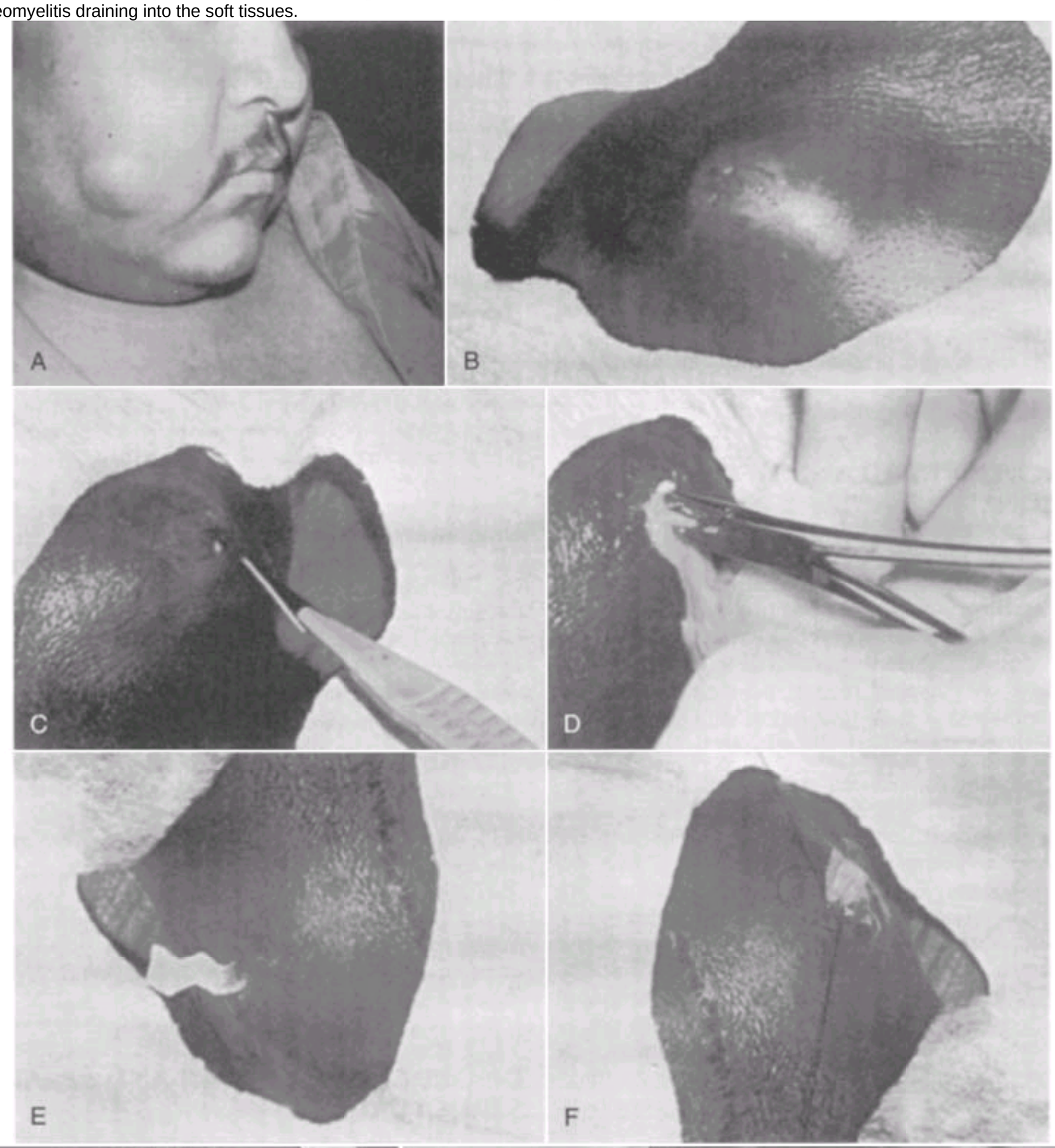


Figure 66-29 *A*, For persistent bleeding of a tongue, mucosa, or dental extraction site, topical thrombin can be sprayed onto gelfoam. *B*, A tongue laceration like this will heal well without sutures once bleeding is controlled. Topical thrombin may be an option if hemostasis is problematic.

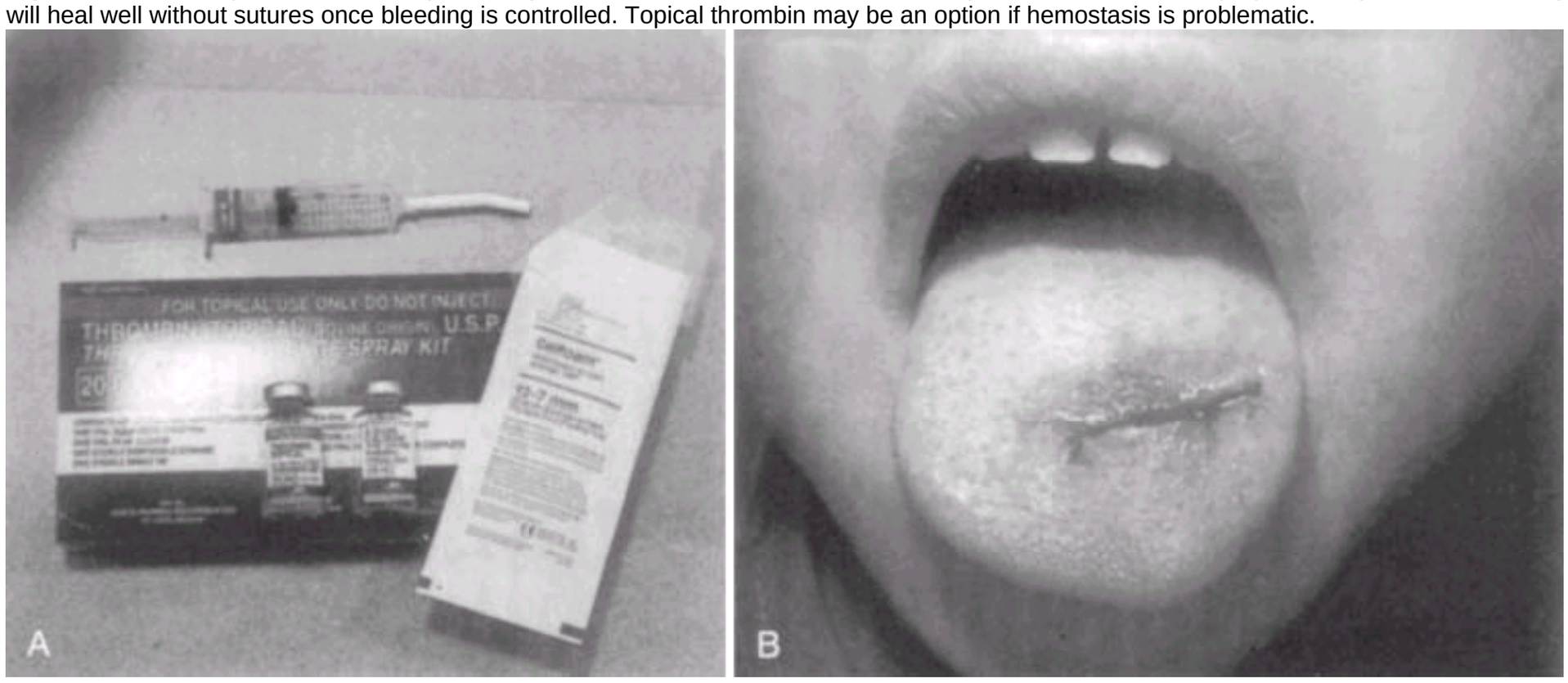
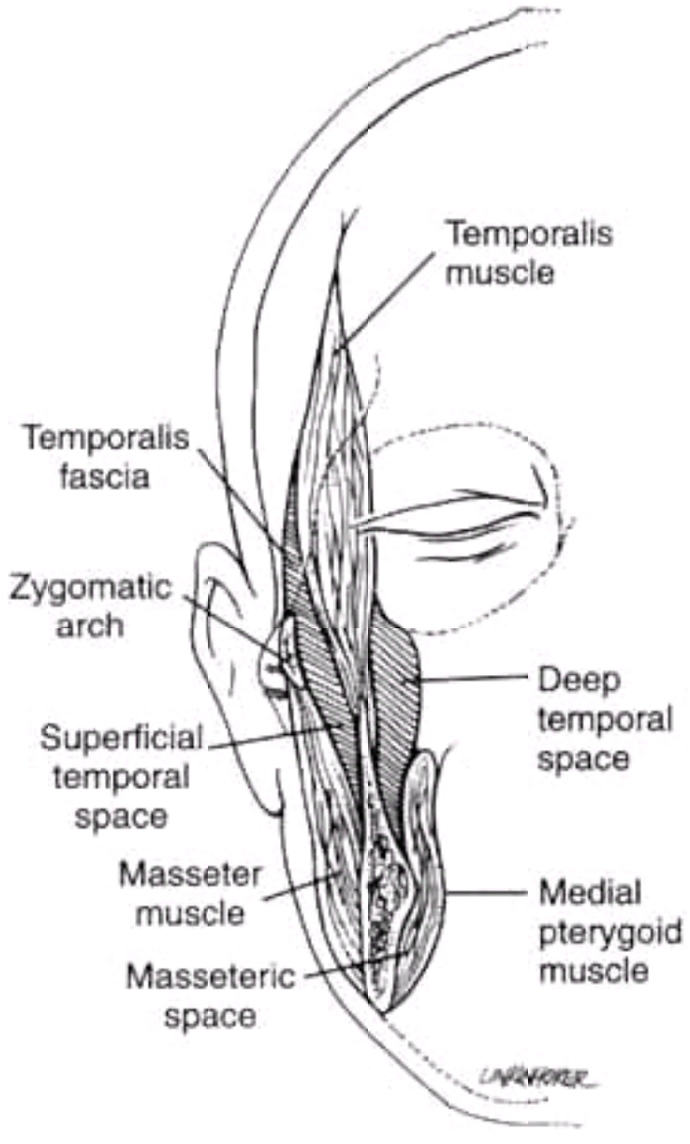
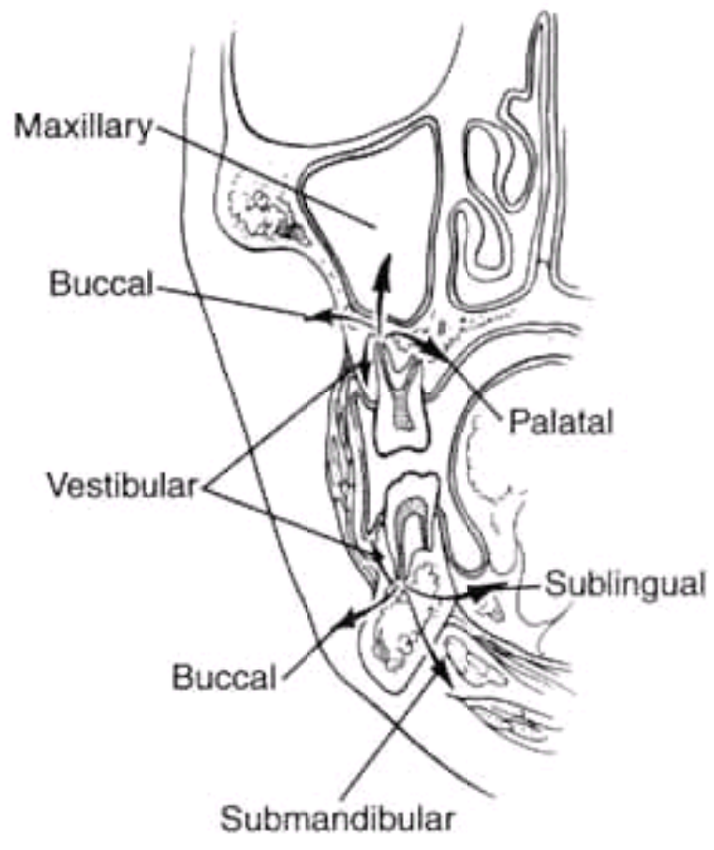


Figure 66-30 *A,* Location of temporal space abscesses. *B,* Route of infection into the buccal space, vestibular spaces, submandibular space, sublingual space, and palatal space. *(From Eisele D, McQuone S: Emergencies of the Head and Neck, 1st ed. St. Louis, MO, Mosby, 2000.)*



A



B

Figure 66-31 *A and B,* Masticator space infection with trismus. *C,* Combined fascial space infections involving the masticator, parapharyngeal, and temporal spaces.

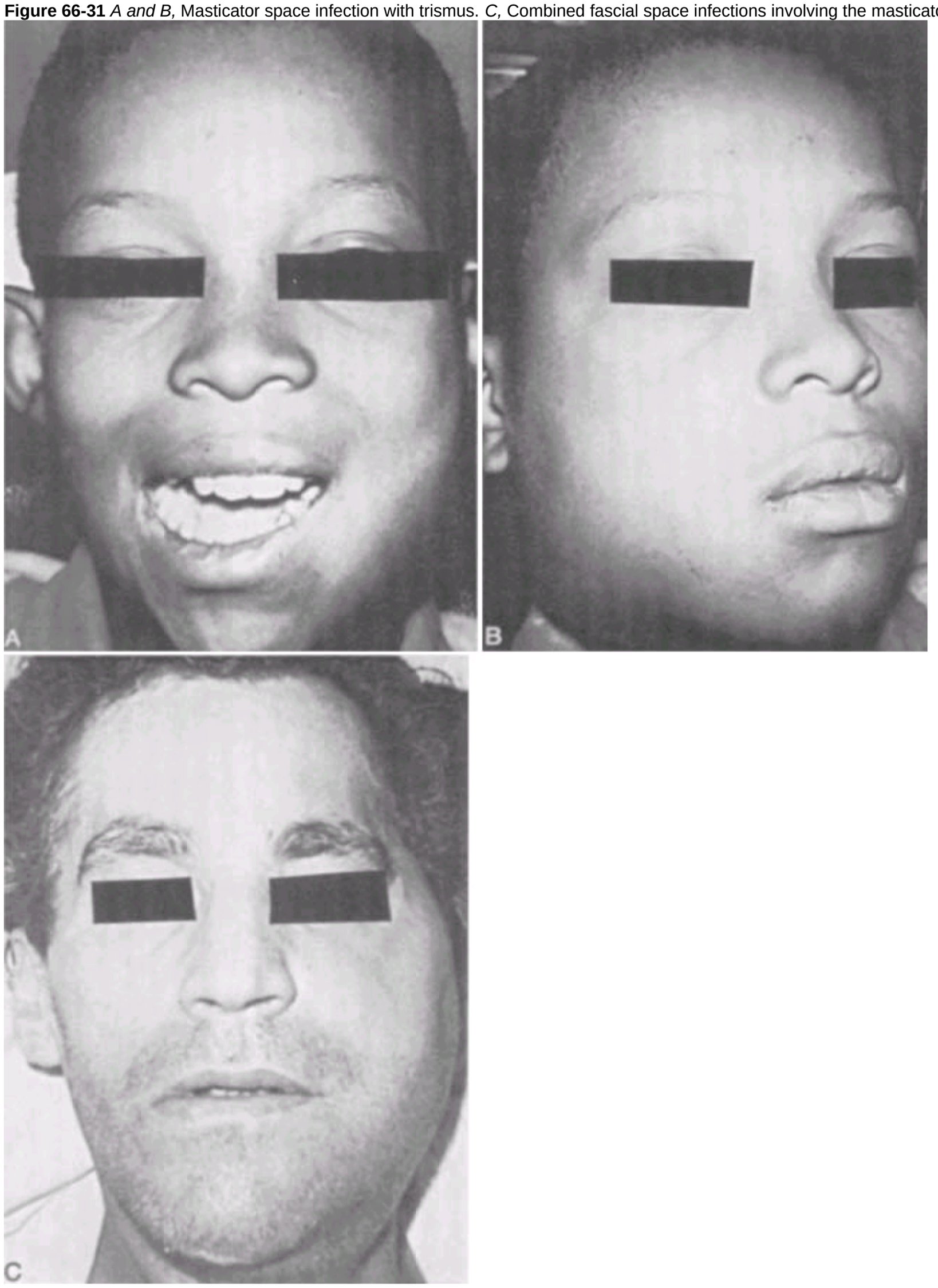


Figure 66-32 *A,* Ludwig angina may initially appear benign. *B,* In Ludwig angina, rapid progression may compromise the airway in a few hours.

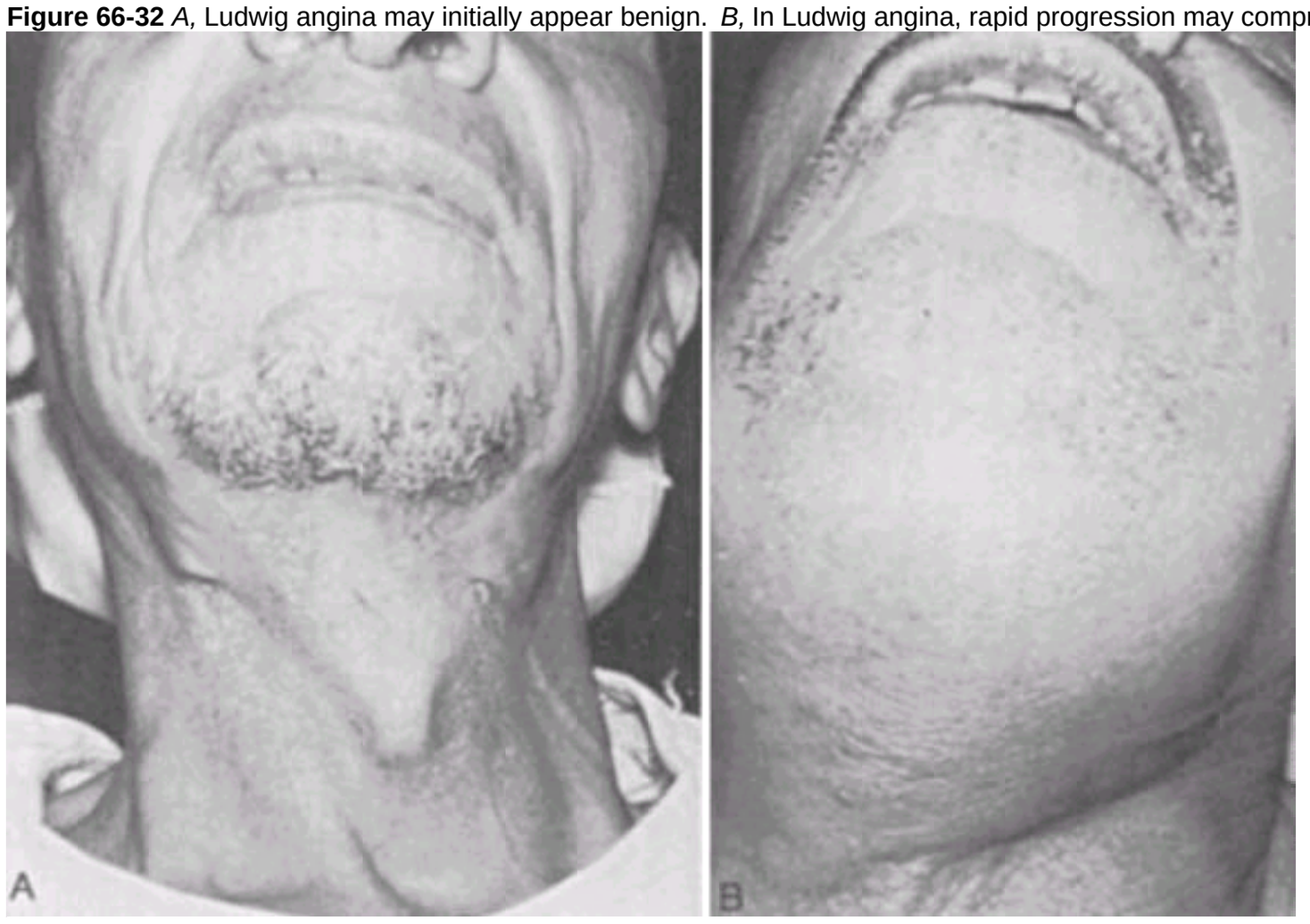


Figure 67-1 Electric thermometer (without probe). This is model 43TA with Fahrenheit and Celsius scales. Infrared tympanic membrane thermometers are acceptable alternatives, but the rectal probe has the advantage of providing a continuous readout. *(Image courtesy of Yellow Springs Instrument Company, Yellow Springs, OH.)*

Figure 67-2 Temperature conversion scale. To change Celsius (centigrade) to Fahrenheit, multiply the Celsius temperature by 9/5 and add 32. To change Fahrenheit to Celsius, subtract 32 from the Fahrenheit number and multiply by 5/9.

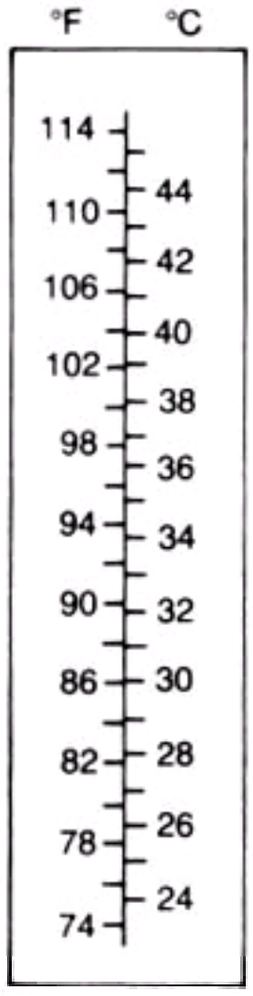


Figure 67-3 In severe hypothermia, the electrocardiogram (ECG) exhibits marked elevation of the J deflection, so-called Osborne waves. The height of the J wave is proportionate to the degree of hypothermia, and this finding is usually most marked in the midprecordial leads. The ECG is of a patient with sinus bradycardia, but approximately half of patients with a temperature below 32°C (89.6°F) develop slow atrial fibrillation, a rhythm that usually converts spontaneously with rewarming. *(From Marriott HJ: Practical Electrocardiology, 8th ed. Baltimore, Williams & Wilkins, 1988.)*

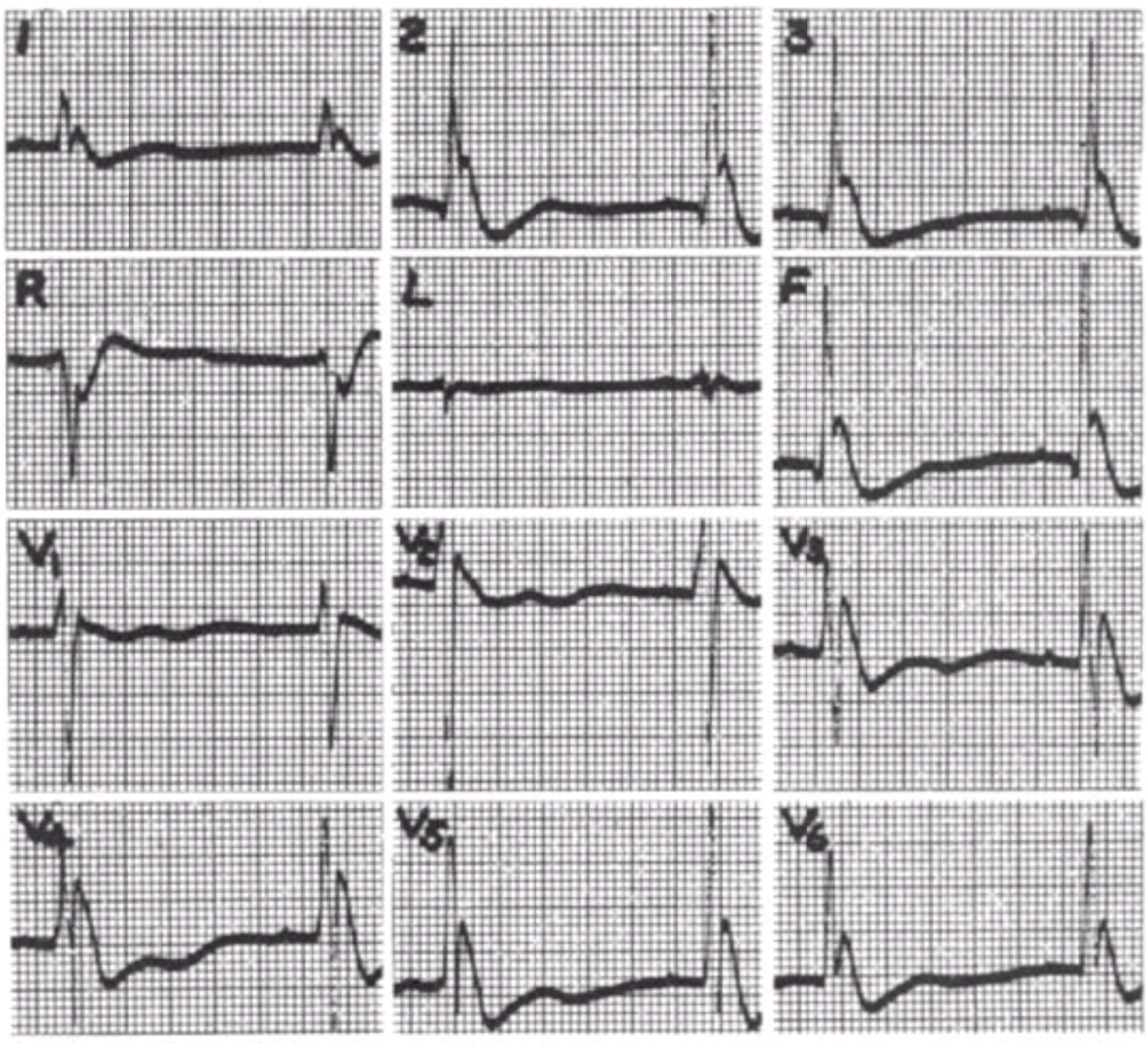


Figure 67-4 Passive external rewarming features. Further heat loss must be stopped through insulation and environmental manipulation. Damp clothing is removed. Dry, warmed blankets are applied. Warmed intravenous fluids are given to counteract the cold-induced diuresis. Internal heat generation is required for rewarming. Rewarming rates are relatively slow. Aggressive intervention with drugs and invasive monitoring may be more harmful than helpful in the otherwise stable hypothermic patient. Note the absence of tracheal intubation, central lines, and Foley catheter in the mild to moderately hypothermic patient.

Figure 67-5 Active external rewarming features. A heat source warms the skin. Traditionally, warm water immersion has been used (as shown). Rates of rewarming are very rapid in some series. There have been suggestions of increased mortality with this method when used for patients with moderate to severe hypothermia, but certain conditions may warrant its use. A major drawback is the inability to closely monitor the patient undergoing immersion. Use of warm air convection (e.g., use of Bair-Hugger device, Eden Prairie, MN), however, allows rewarming in the ED with full patient monitoring and less rapid vasodilation during rewarming.

Figure 67-6 Active core rewarming methods. Inhalation of warm, humidified air or oxygen causes gradual core rewarming and should be a *mainstay of rewarming therapy*. This method can be combined with other methods such as heated intravenous fluids. Peritoneal lavage is a more rapid method of core rewarming that requires placement of one or more intraperitoneal catheters. Other techniques are described in the text.

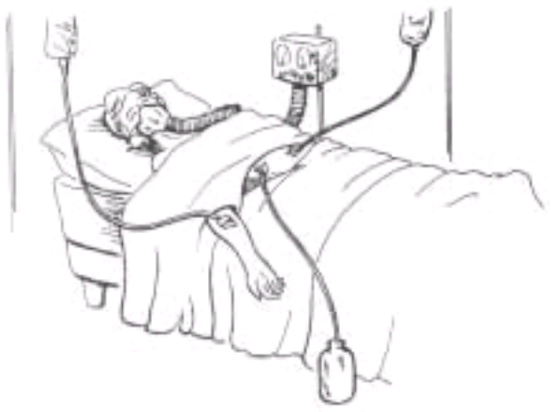


Figure 67-7 Active core rewarming cardiac bypass features. Bypass technology and a surgical team are needed. The rewarming rates are rapid. This method is useful in a patient with cardiac arrest; the technique is invasive, expensive, and technically difficult.

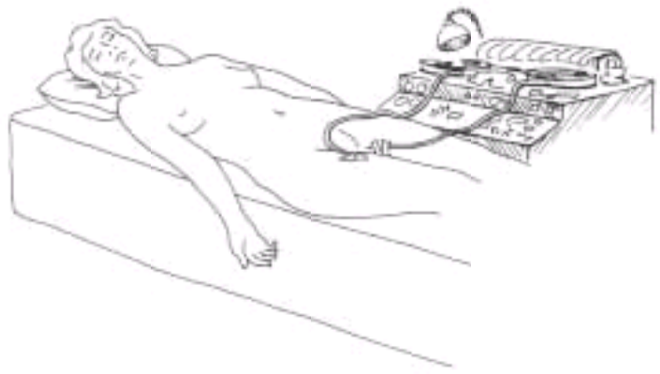


Figure 67-8 Active core rewarming open thoracic lavage features. The heart is warmed directly. This method requires surgical backup and is relatively invasive. The rewarming rates are rapid. An alternative that is more practical for the ED is pleural rewarming repeatedly using 200 to 300 mL warmed saline intermittently placed, then withdrawn through a chest tube (see text).

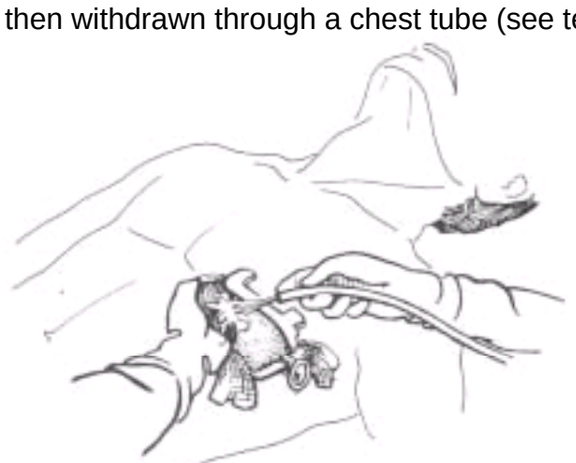


Figure 68-1 It is absolutely essential to rapidly lower the core temperature of a severely hyperthermic patient by instituting cooling techniques as soon as possible. Evaporative cooling (see text) is usually quite effective and technically easy. An alternative approach, albeit poorly studied, is to literally pack the patient in ice. In this case, plastic trash bags were used to hold the ice and to prevent water from dripping on the floor. A child's plastic wading pool is another option for this ice packing technique.

Figure 69-1 Longitudinal image of the gallbladder, demonstrating variability in tissue echogenicity. *A,* The anechoic appearance of fluid. Water, plasma, non-clotted blood, and urine will have the same appearance. *B,* The highly reflective appearance of a calcified stone in the gallbladder. Foreign bodies, needles, and bone will have a similar brightly echogenic appearance. *C,* The relatively hypoechoic appearance of tissue. Clotted blood, particulate material within fluid (lipid or purulent material) will appear the same, with echogenicity intermediate to bone and fluid.

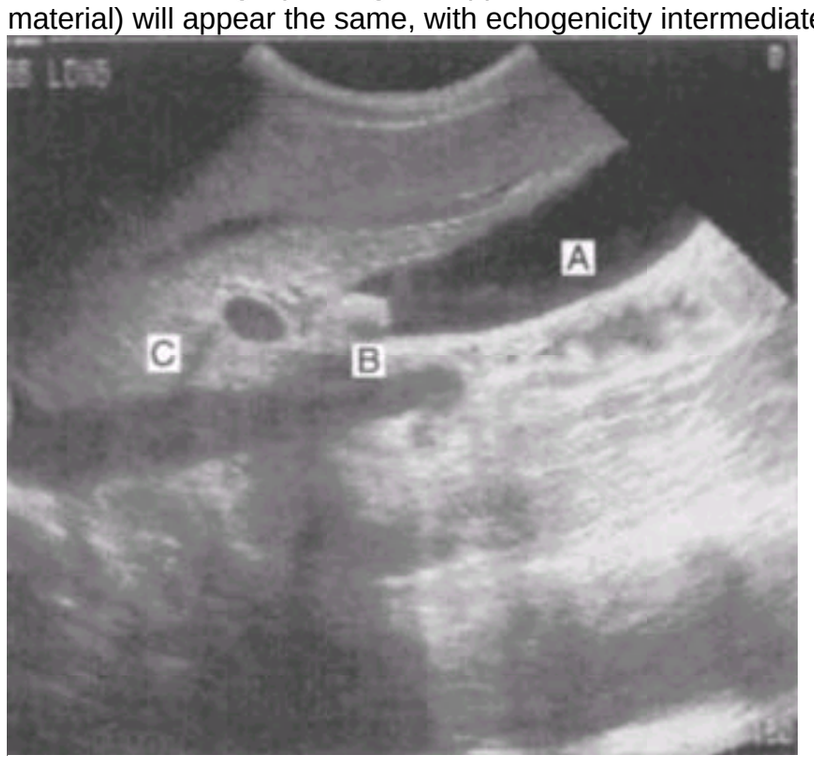


Figure 69-2 The curvilinear array probe is used for lower frequency (2.0 to 5.0 MHz) scanning of the abdomen and chest. It is best used for cardiac and abdominal imaging. The linear array probe is used for high frequency (6 to 10 MHz) scanning of superficial tissues, vessels, subcutaneous masses, and foreign bodies.

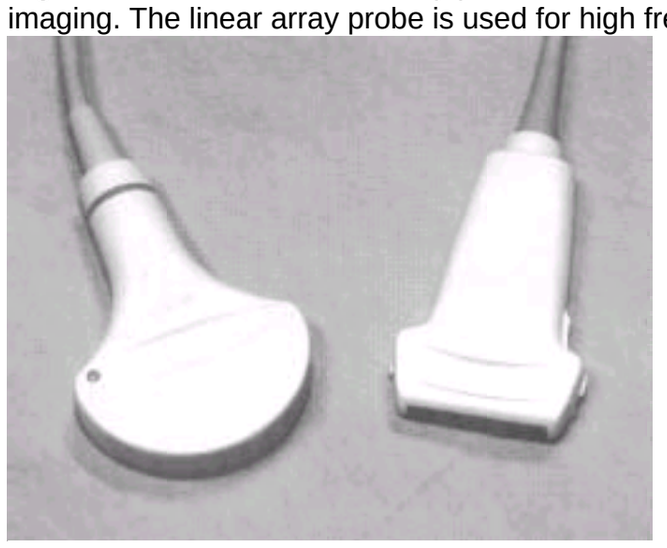


Figure 69-3 An ultrasound needle guide is ideally used to guide needle placement into deep or small structures. *(Courtesy of Dymax Corp, a subsidiary of Bard Access Systems.)*

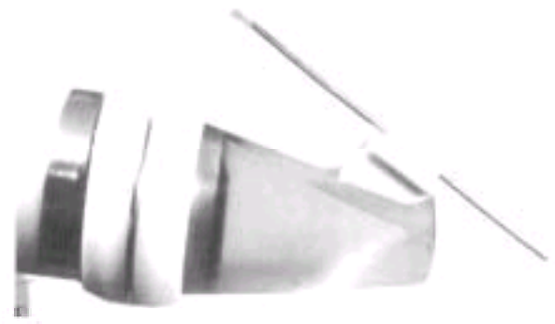


Figure 69-4 Imaging window for the internal jugular vein and carotid artery. The ultrasound probe should be covered with acoustic gel and then a sterile cover or glove. An additional layer of sterile gel should be placed over the cover. The probe is then placed parallel and superior to the clavicle over the groove made by the two heads of the sternocleidomastoid muscle. The probe should be pointed toward the patient's right. Care should be taken not to apply undue pressure on the probe to avoid compression of the easily collapsible internal jugular vein. Asking the patient to blow on his or her thumb or performing a Valsalva maneuver will increase the luminal diameter of the vein. *(Lydia F. Roberts, Photographer)*

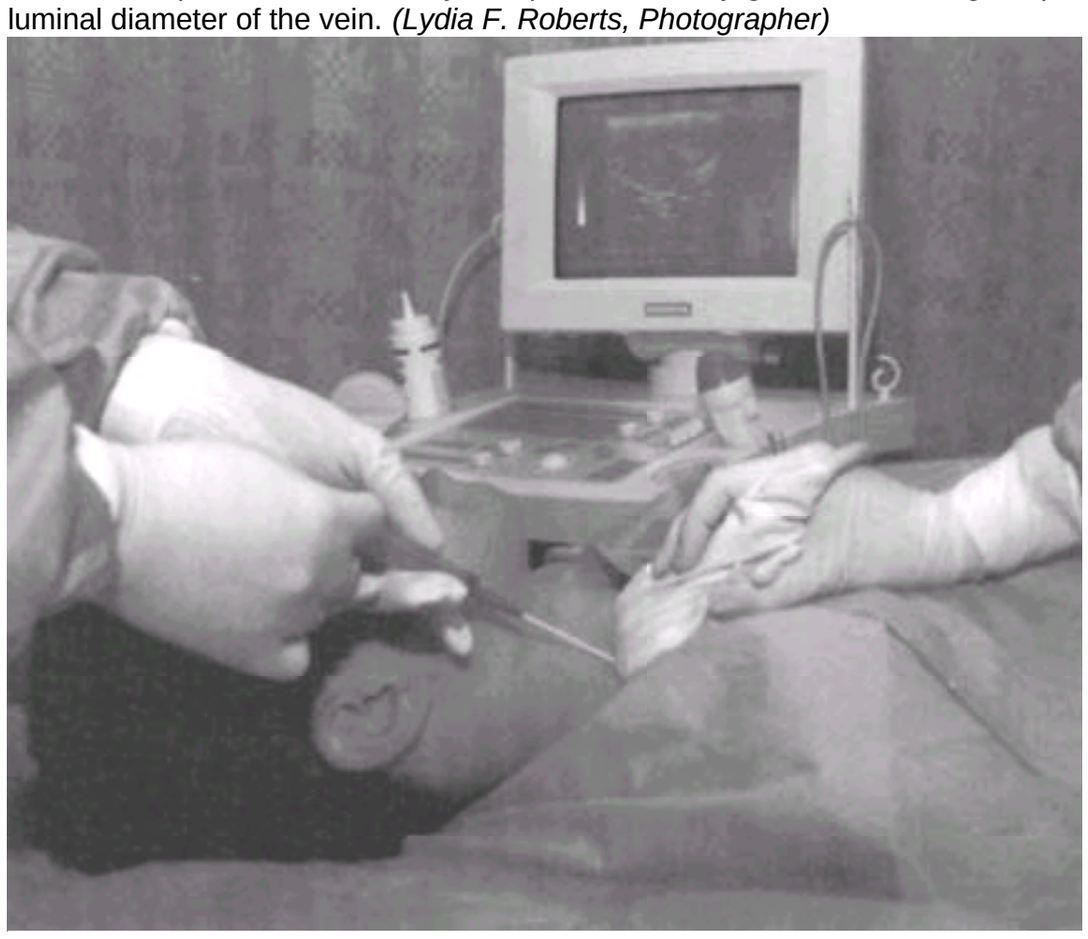
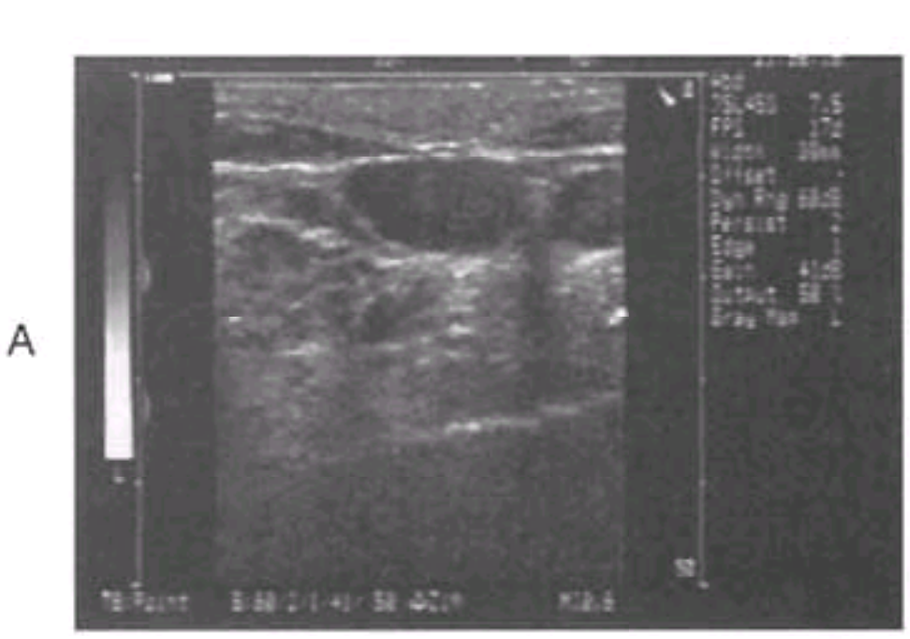
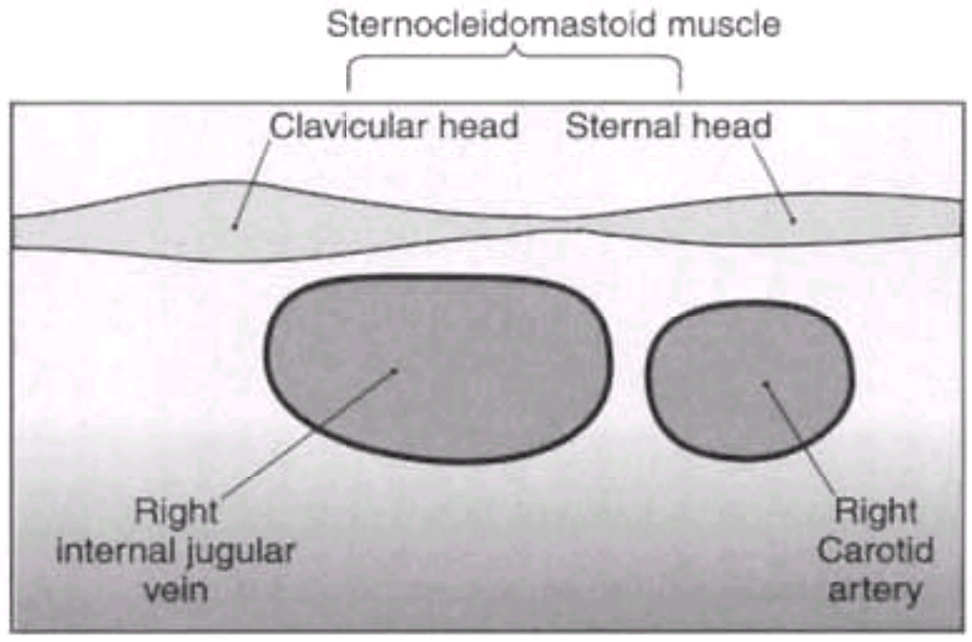


Figure 69-5 The internal jugular vein and carotid artery. *A,* Sonographic image; *B,* Schematic representation. The beam of the ultrasound should intersect the carotid artery and the internal jugular vein in a transverse or cross-sectional plane. The internal jugular vein is easily identified by its compressibility and response to Valsalva maneuvers (i.e., it increases in diameter).



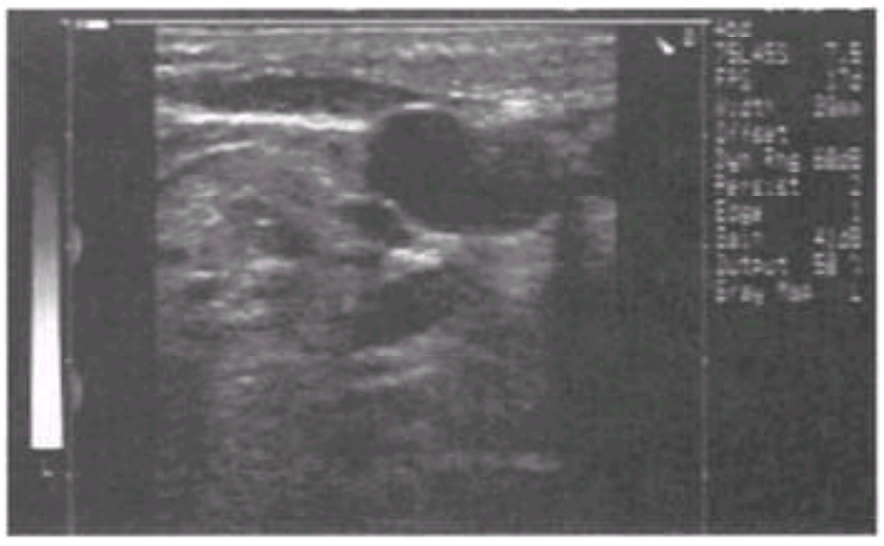
A

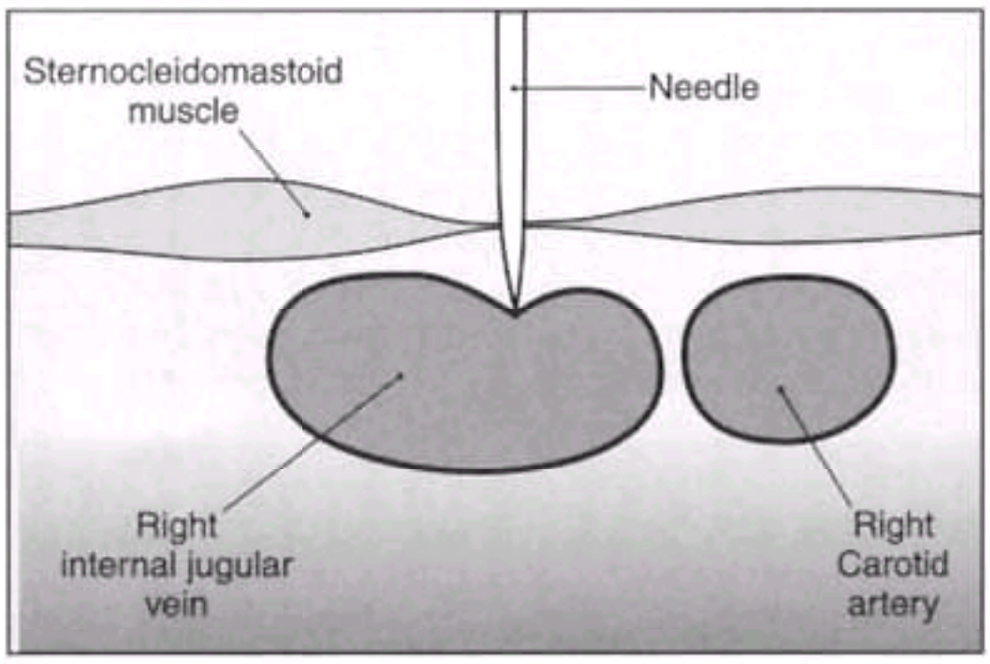


B

Figure 69-6 *A* and *B,* Once the vein is positioned directly under the probe, needle aspiration can be attempted. If the site of skin puncture is correct, the needle will indent the vein and the vein will appear to collapse as it is entered.

A

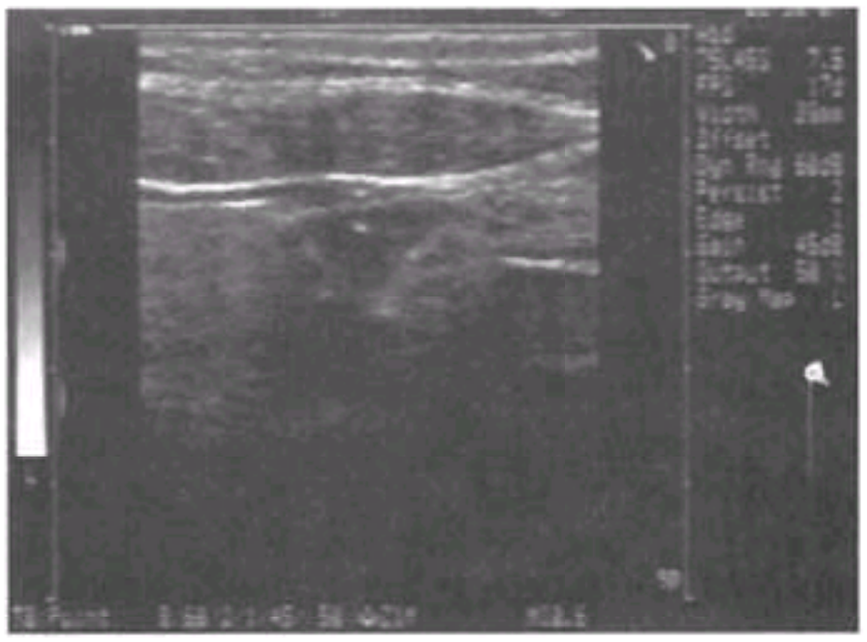


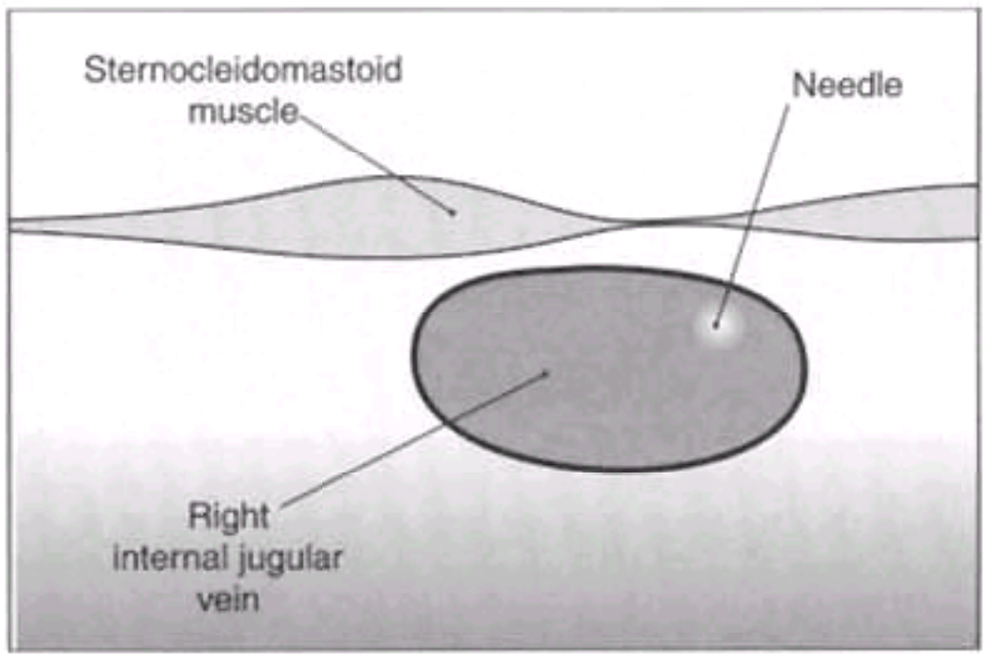


B

Figure 69-7 *A* and *B,* The needle may be visualized within the vessel lumen, and will appear as a brightly echogenic structure.

A





B

Figure 69-8 To obtain a subxiphoid view of the heart and pericardium, the probe should be placed beneath the xiphoid process with the probe marker pointing to the patient's right. The probe should be angled upward and slightly to the left. *(Lydia F. Roberts, Photographer)*

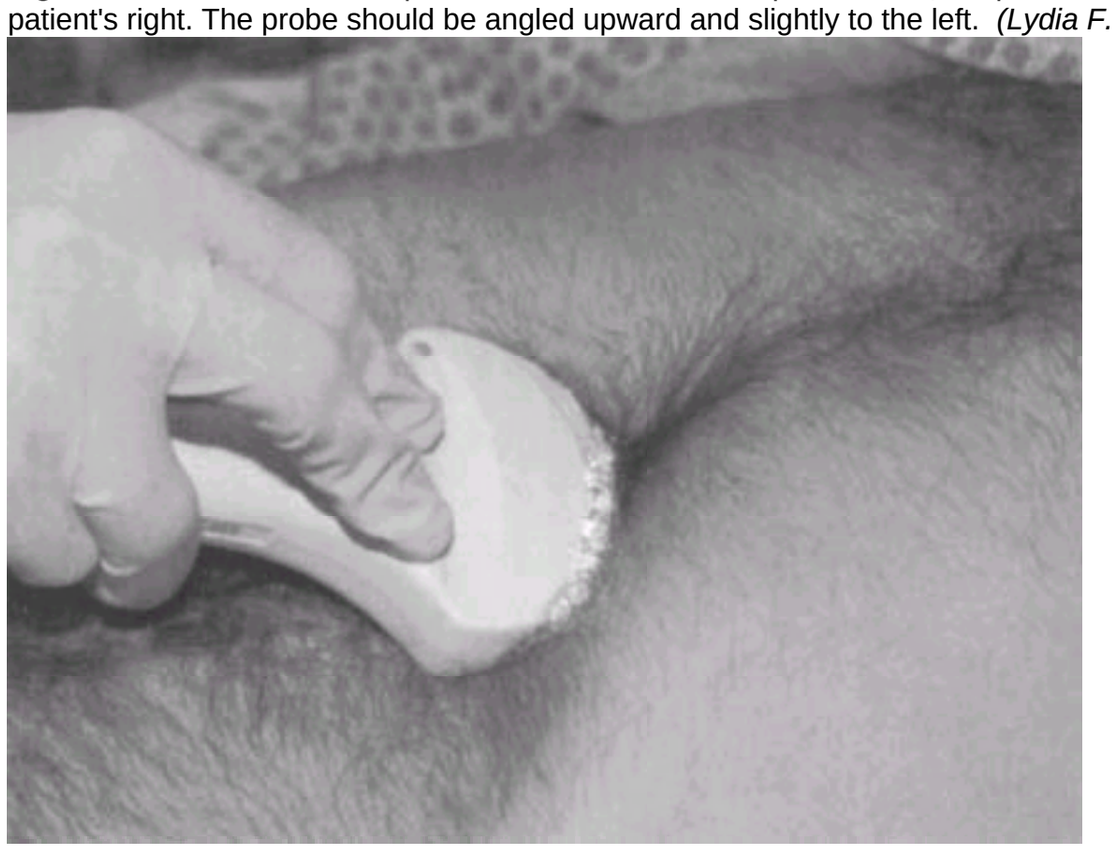


Figure 69-9 *A,* Sonographic appearance of a normal heart and pericardium obtained through the subxiphoid window. The right ventricle will be the most anterior cardiac structure, bordered superiorly by the pericardium and diaphragm. *B,* Schematic representation.

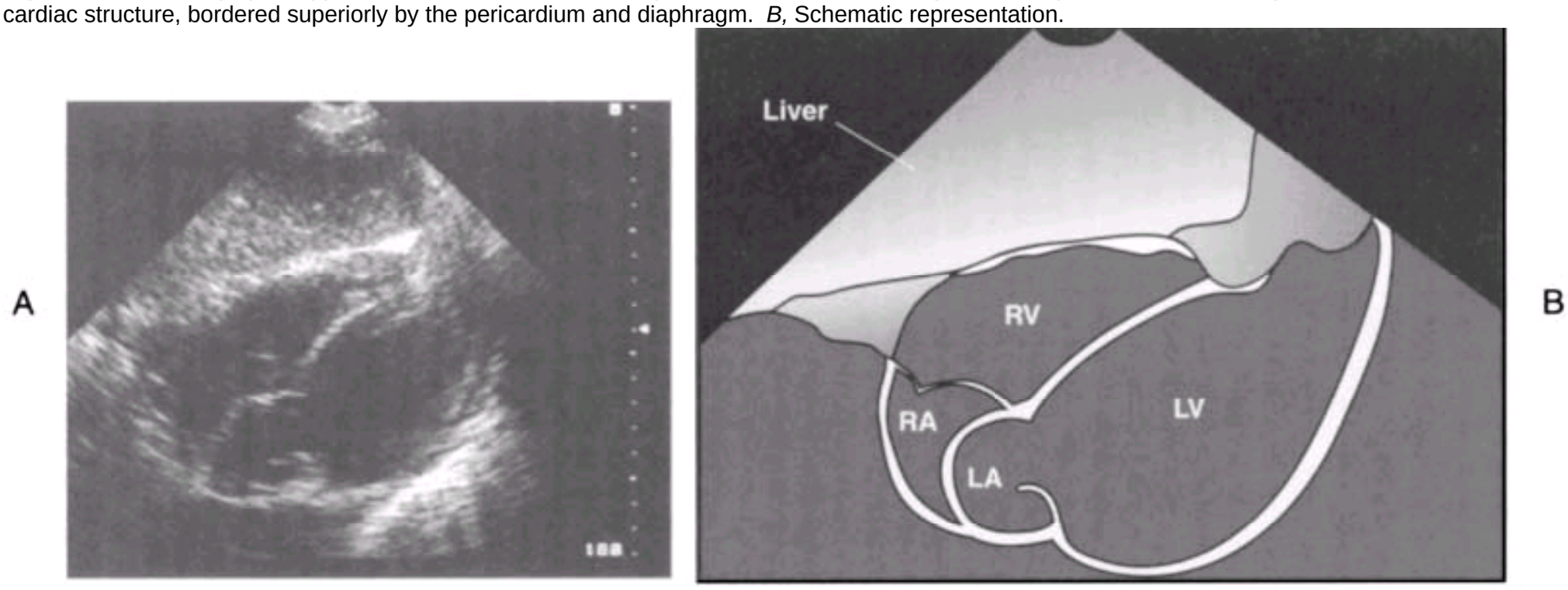


Figure 69-10 To obtain a parasternal view of the heart, the probe should be placed adjacent to the left sternal border in the left second or third intercostal space. The patient's head is at the lower edge of the photograph. *(Lydia F. Roberts, Photographer)*

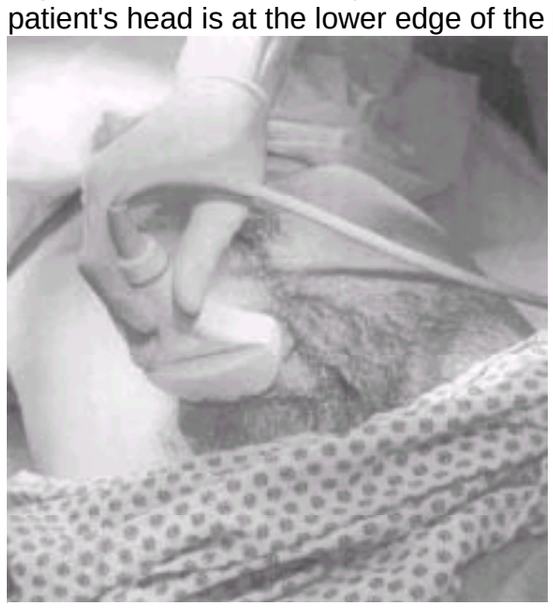
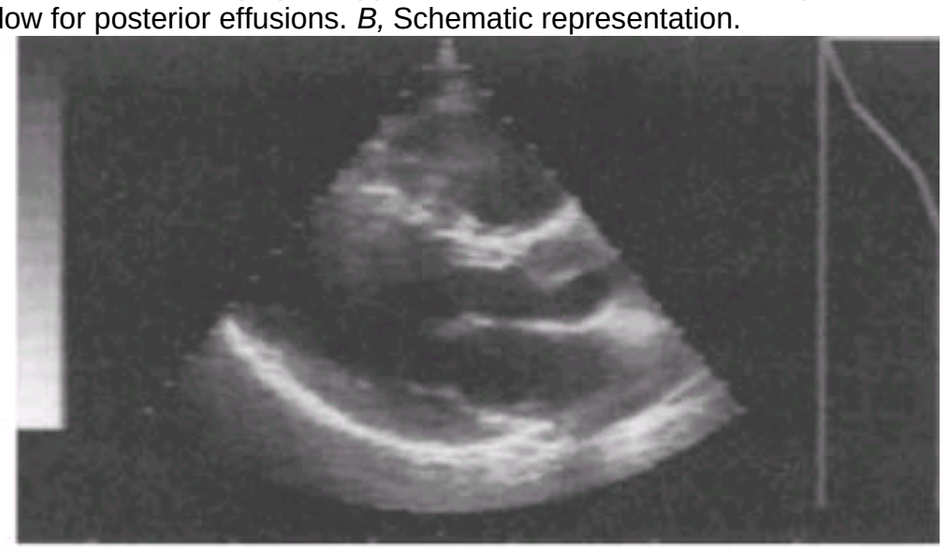


Figure 69-11 *A,* Sonographic appearance of a normal heart and pericardium obtained through the parasternal long axis window. This view is an excellent imaging window for posterior effusions. *B,* Schematic representation.

A



B

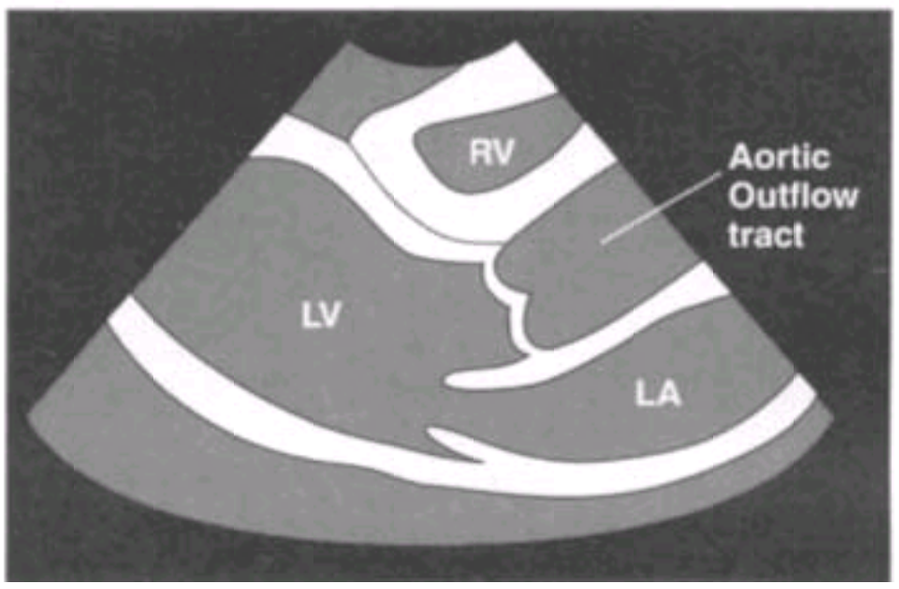
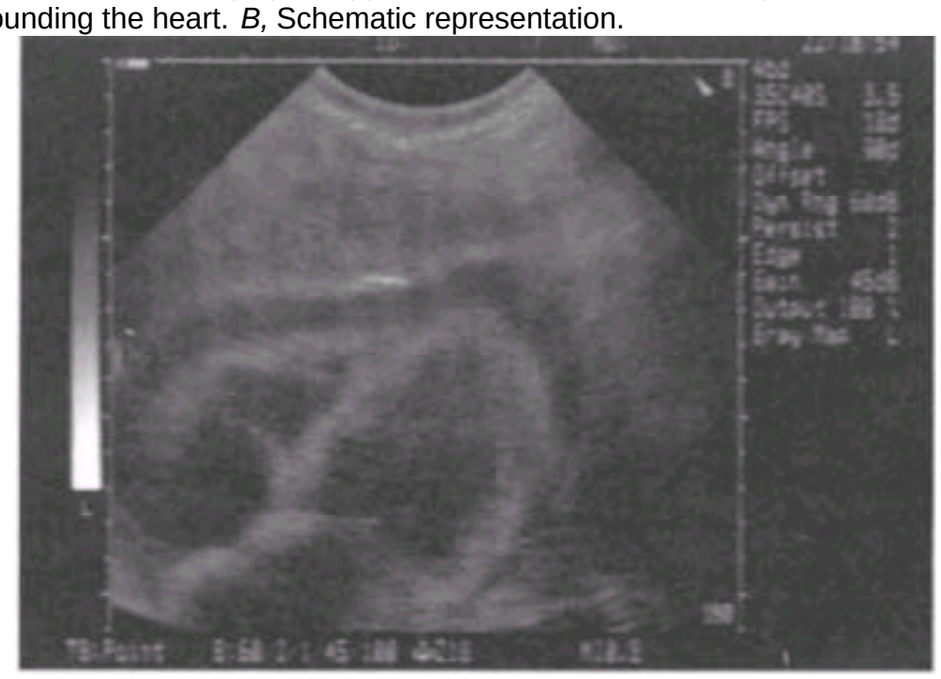


Figure 69-12 *A,* Sonographic appearance of a circumferential pericardial effusion obtained through the subxiphoid window. The effusion is seen as an anechoic stripe surrounding the heart. *B,* Schematic representation.

A



B

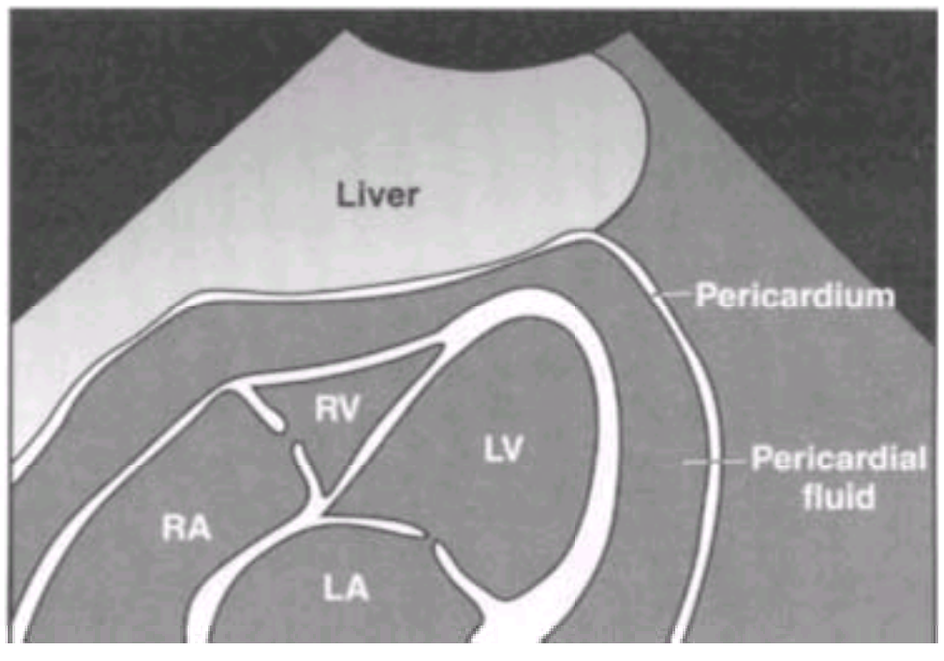
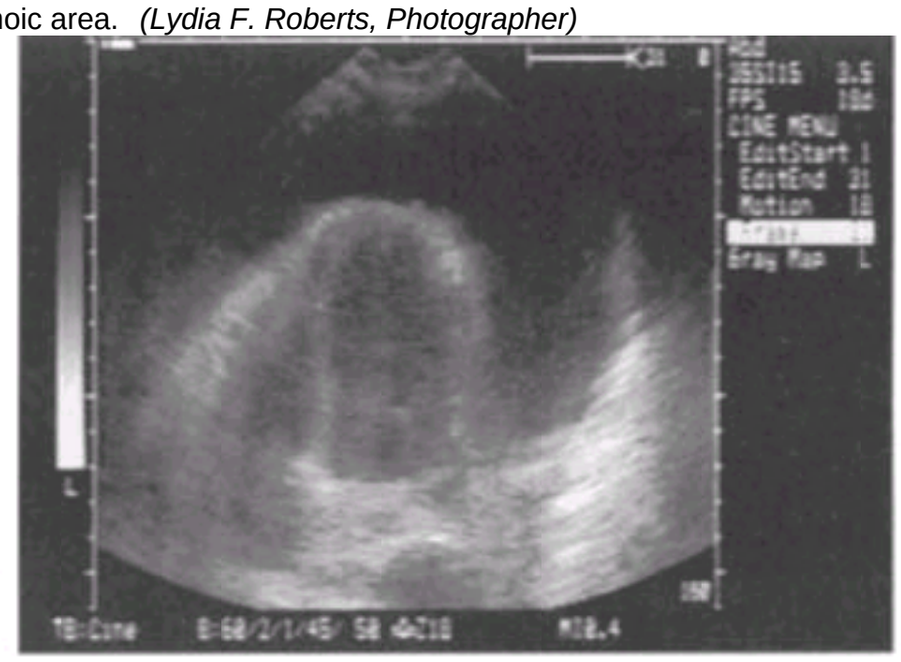
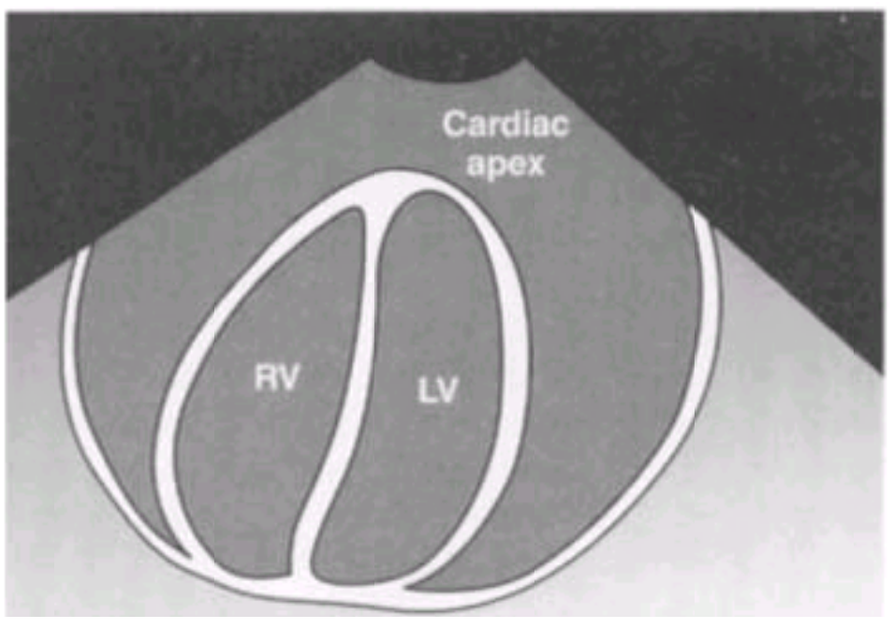


Figure 69-13 Photograph (*A*), sonographic image (*B*), and schematic representation (*C*) demonstrating the sonographic window that places the largest area of accumulated fluid nearest the probe (top of screen). There should be no vital structures between the probe and the pericardial space when the aspirating needle/catheter is placed over the superior border of the rib closest to the anechoic area. *(Lydia F. Roberts, Photographer)*

A



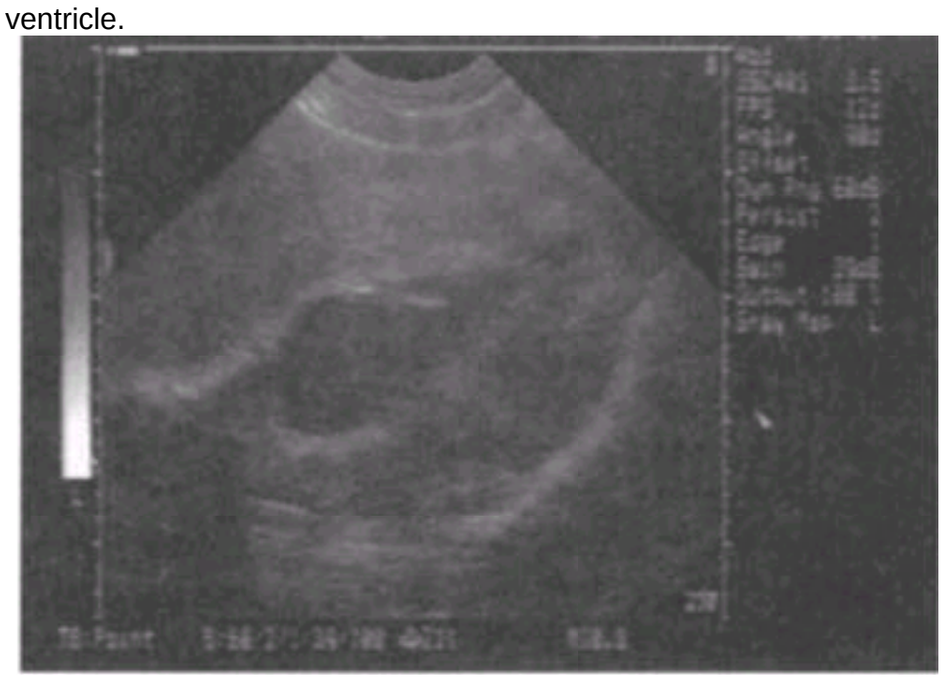
B



C

Figure 69-14 Pacing wire within the right ventricle. *A,* Songraphic image. *B,* Schematic representation. The right ventricle is seen through the subxiphoid window, which provides excellent views of the heart without interfering with pacemaker line placement. The pacemaker wire is seen as a brightly echogenic structure within the right ventricle.

A



B

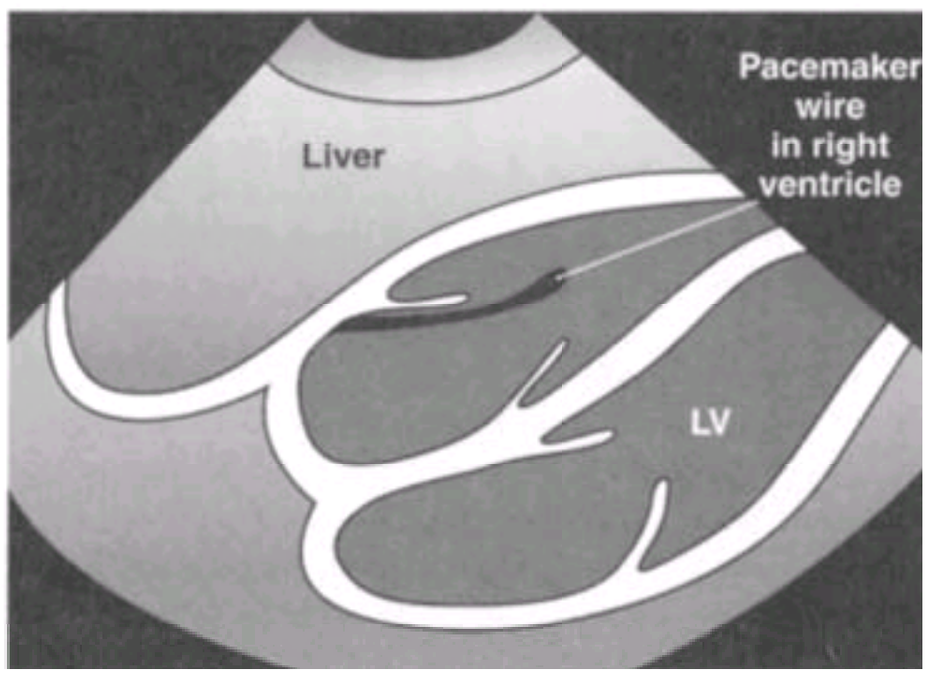
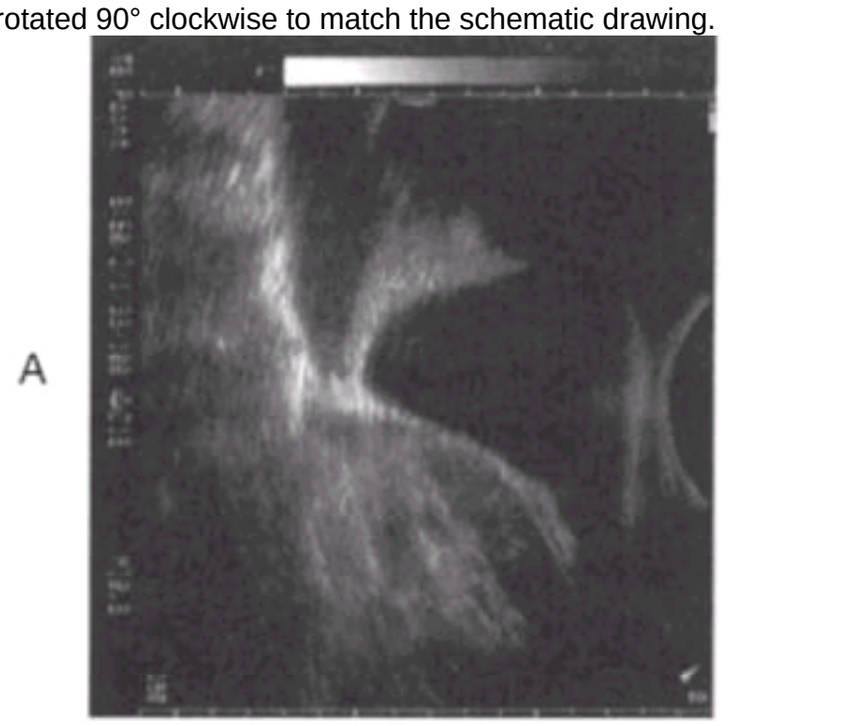


Figure 69-15 Sonographic appearance (*A*) and schematic representation (*B*) of a pleural effusion. Fluid will appear as an anechoic area over the brightly echogenic diaphragm. The collapsed lung can usually be seen as a wedge-shaped echogenic structure moving with respirations within the fluid. Note that the sonographic image has been rotated 90° clockwise to match the schematic drawing.



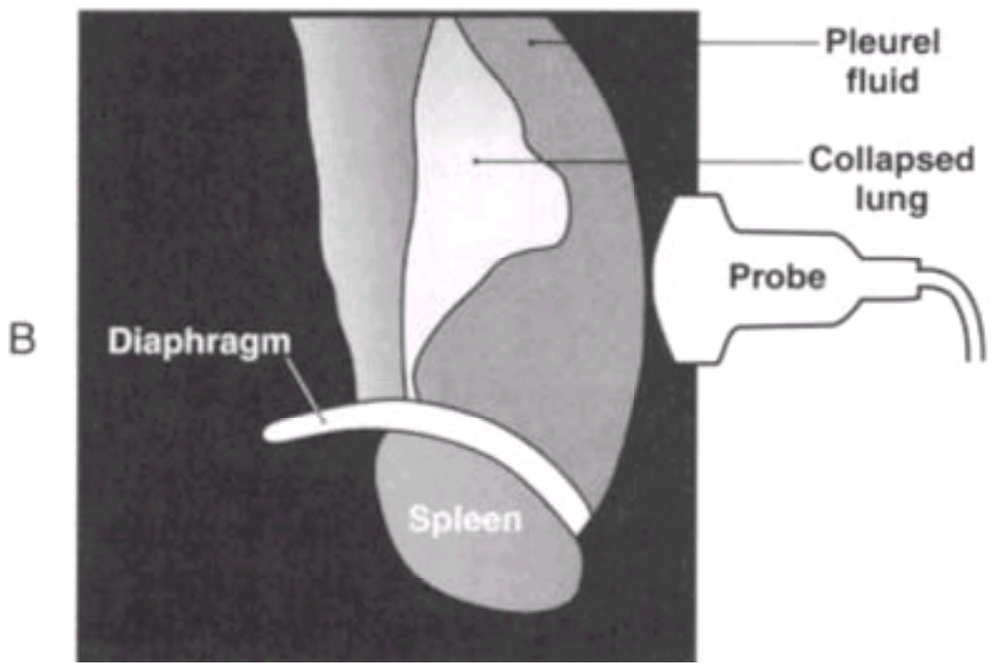


Figure 69-16 Probe positioning for identification of a pleural effusion. The probe should be placed longitudinally over the lateral or posterior chest wall on the side of the suspected effusion. The probe should be moved inferiorly to identify the kidney, liver or spleen, and diaphragm. *(Lydia F. Roberts, Photographer)*

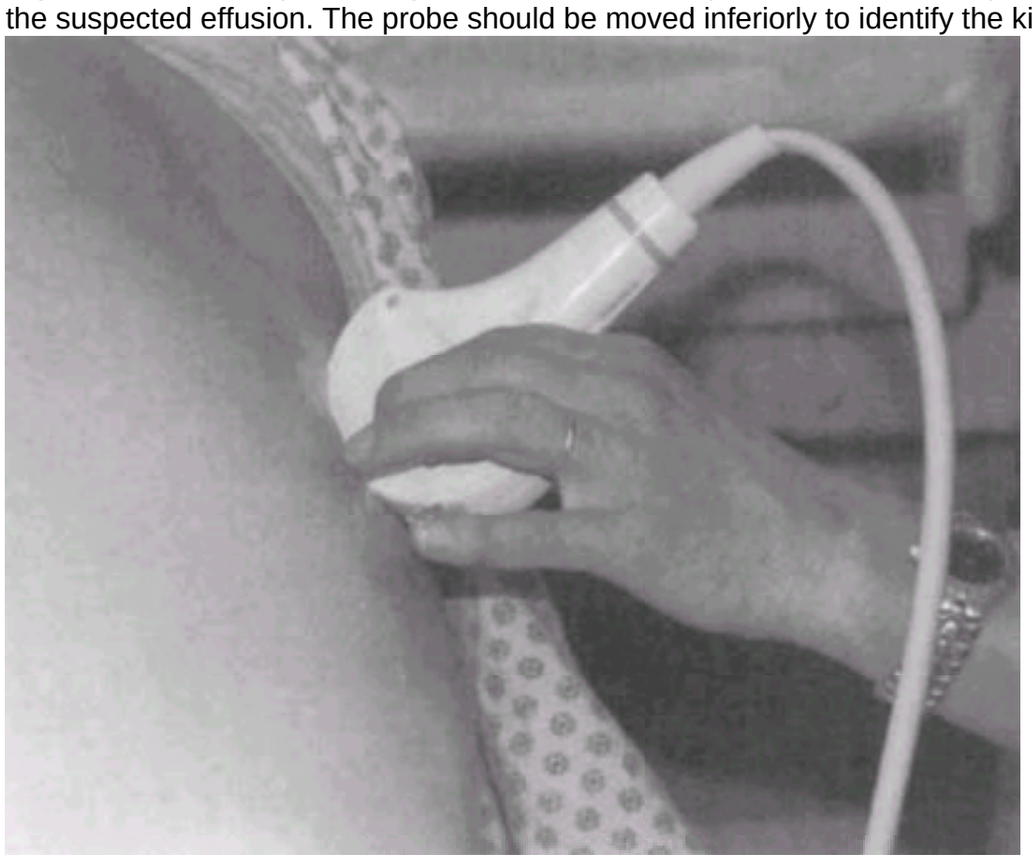


Figure 69-17 Sonographic appearance of peritoneal free fluid. Fluid will appear as a large anechoic area with loops of bowel floating freely. The optimal area for needle insertion is where there is a large pocket of fluid just under the skin surface, closest to the probe.

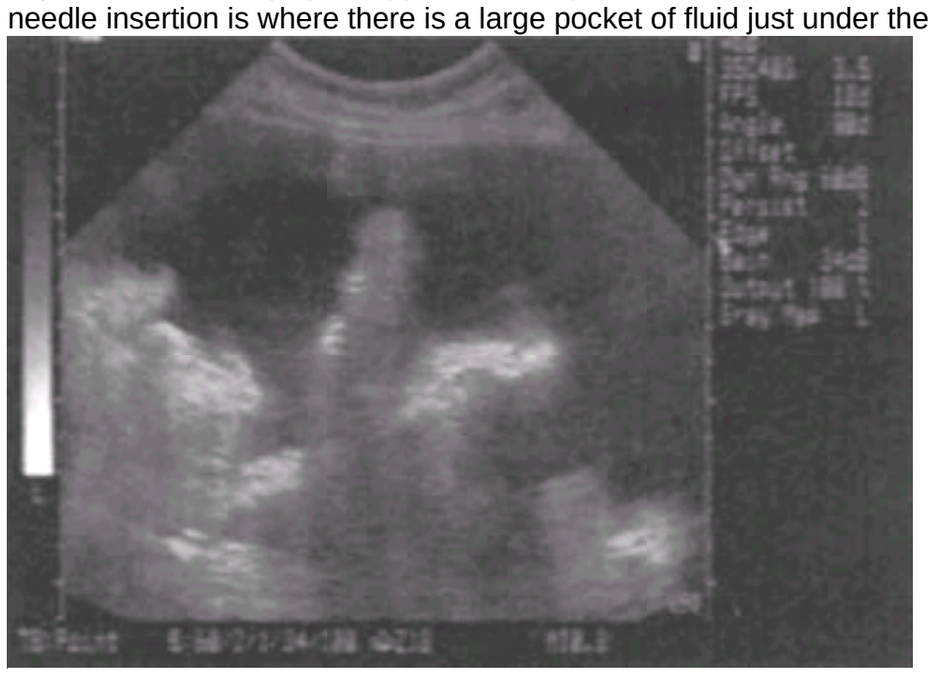
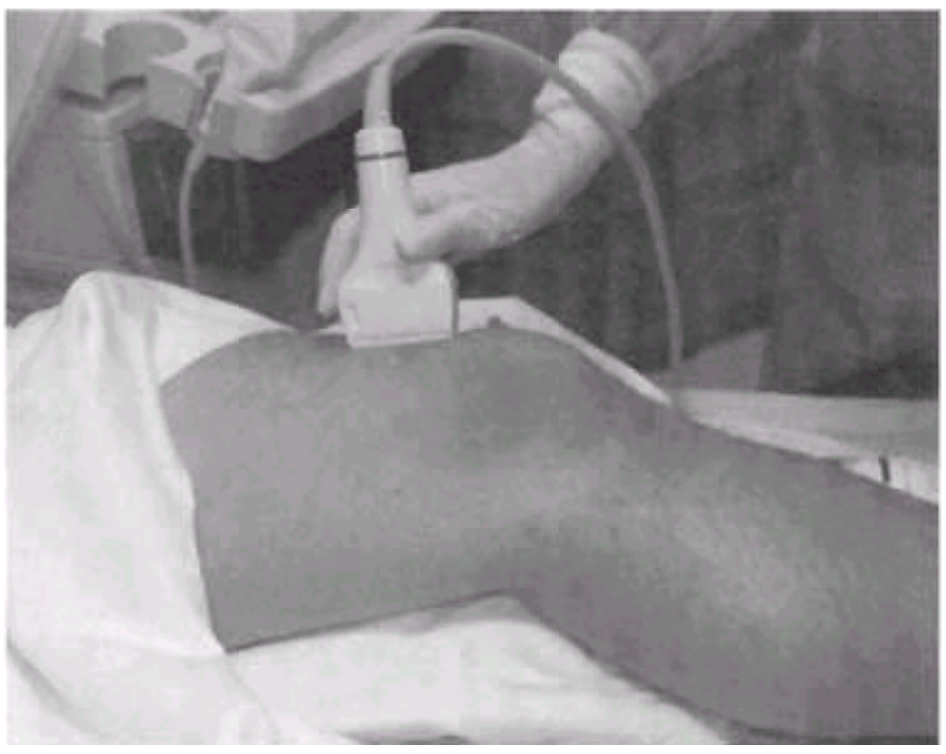
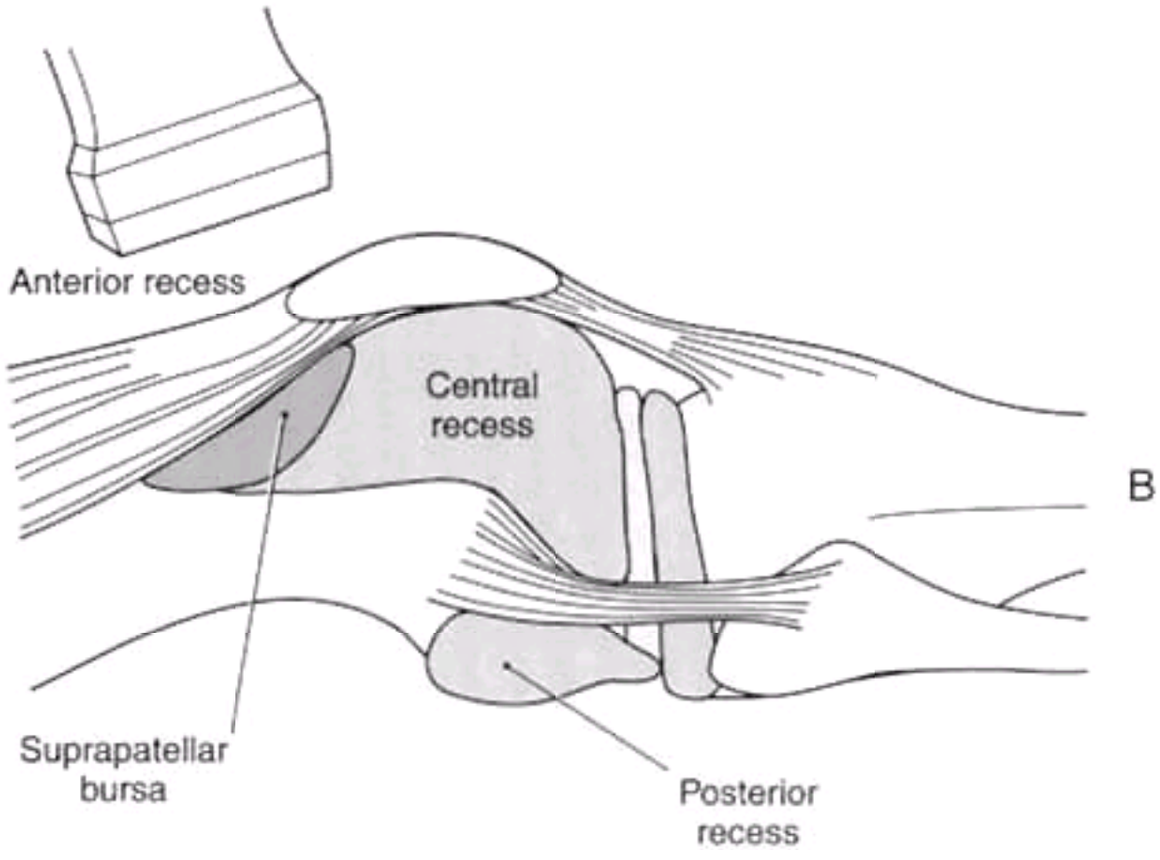


Figure 69-18 *A,* Probe positioning for sonographic imaging of the suprapatellar bursa. The probe should be placed in the longitudinal position superior to the patella. Following identification of fluid within the bursa, the probe can be rotated transversely to identify the medial and lateral extent of the fluid. *B,* Schematic representation. *(Lydia F. Roberts, Photographer)*

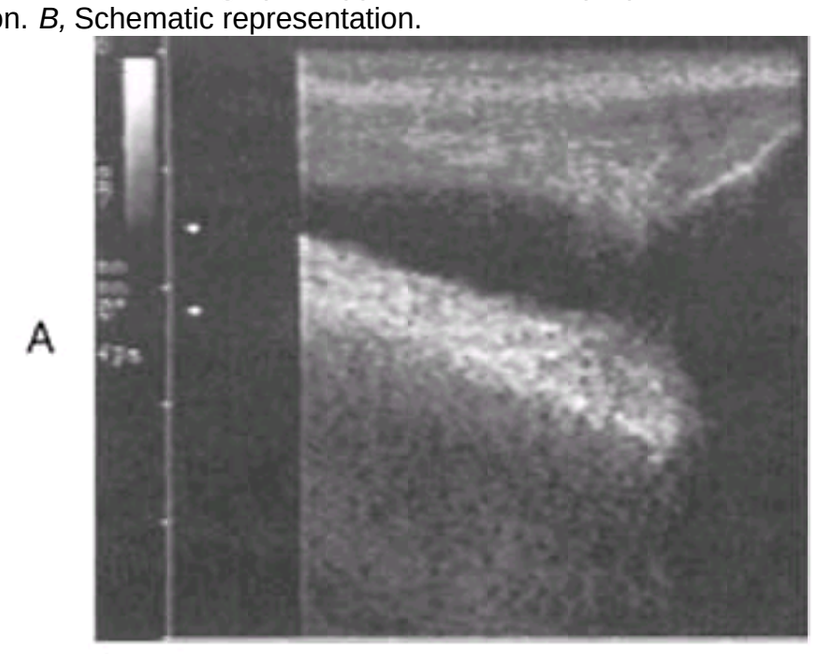


A



B

Figure 69-19 *A,* Sonographic appearance of a suprapatellar effusion. Fluid will appear as an anechoic area superior to the patella and beneath the quadriceps tendon. *B,* Schematic representation.



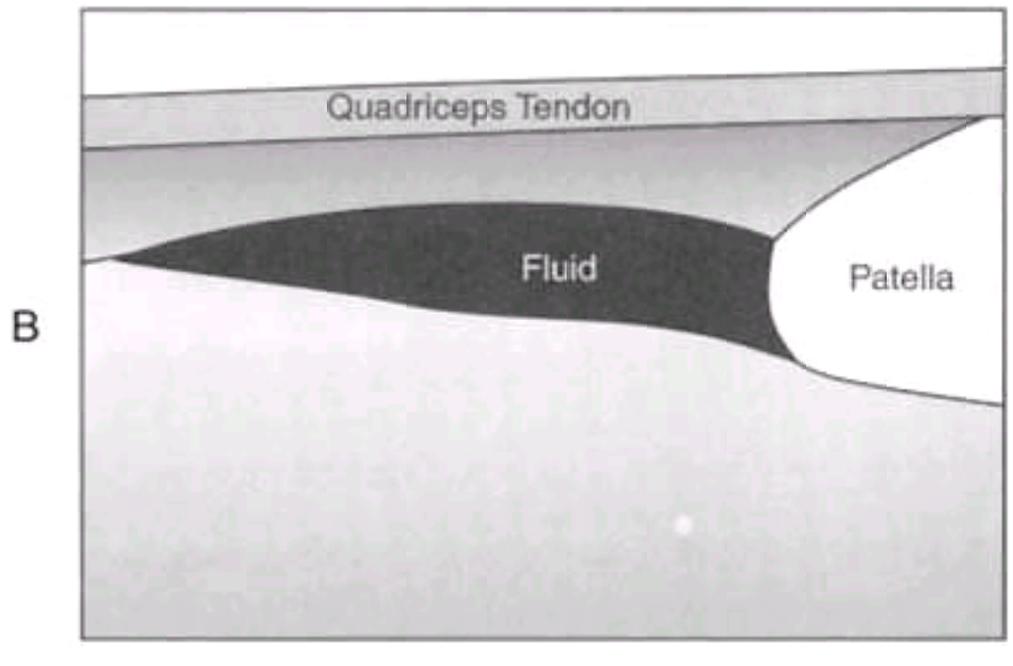


Figure 69-20 Probe positioning for the anterior view of the shoulder joint. The patient's arm should be placed palm up and the elbow flexed and supported. The arm should be rotated externally. The probe should be placed in the transverse position over the anterior aspect of the shoulder and the biceps groove visualized. *(Lydia F. Roberts, Photographer)*

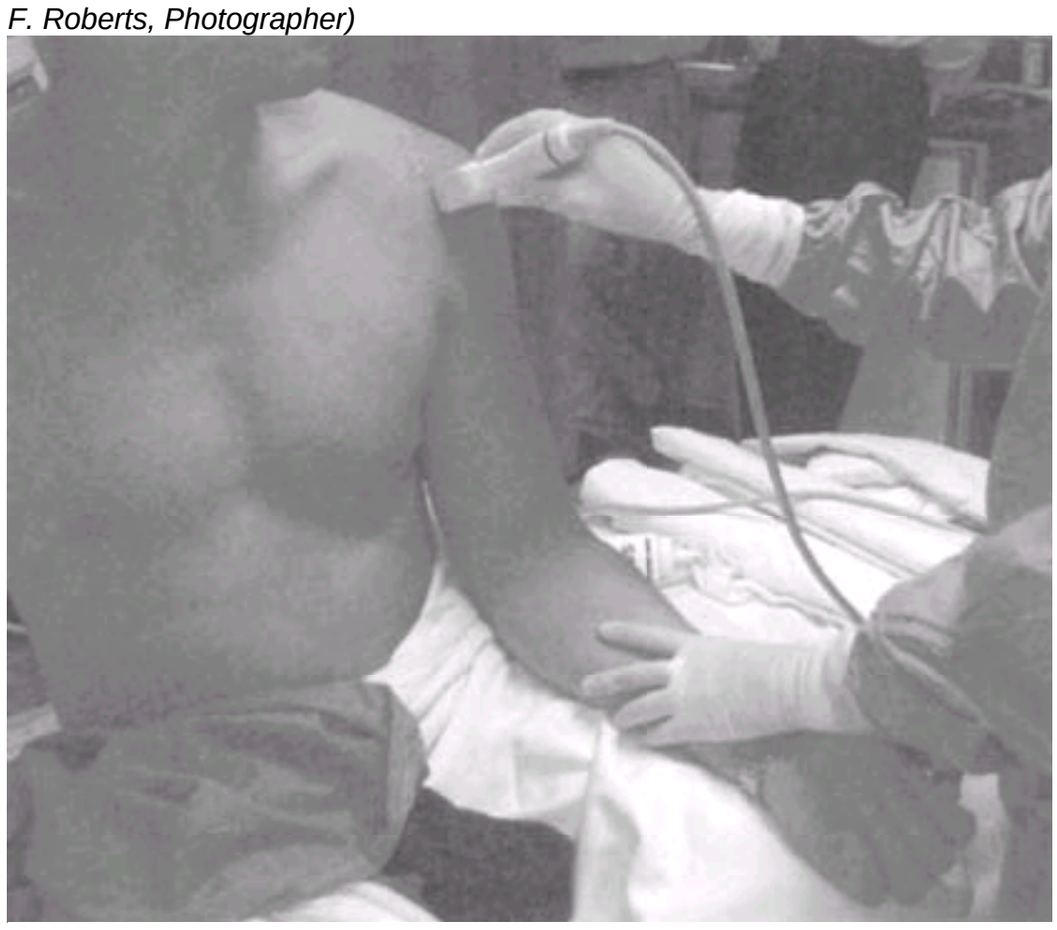
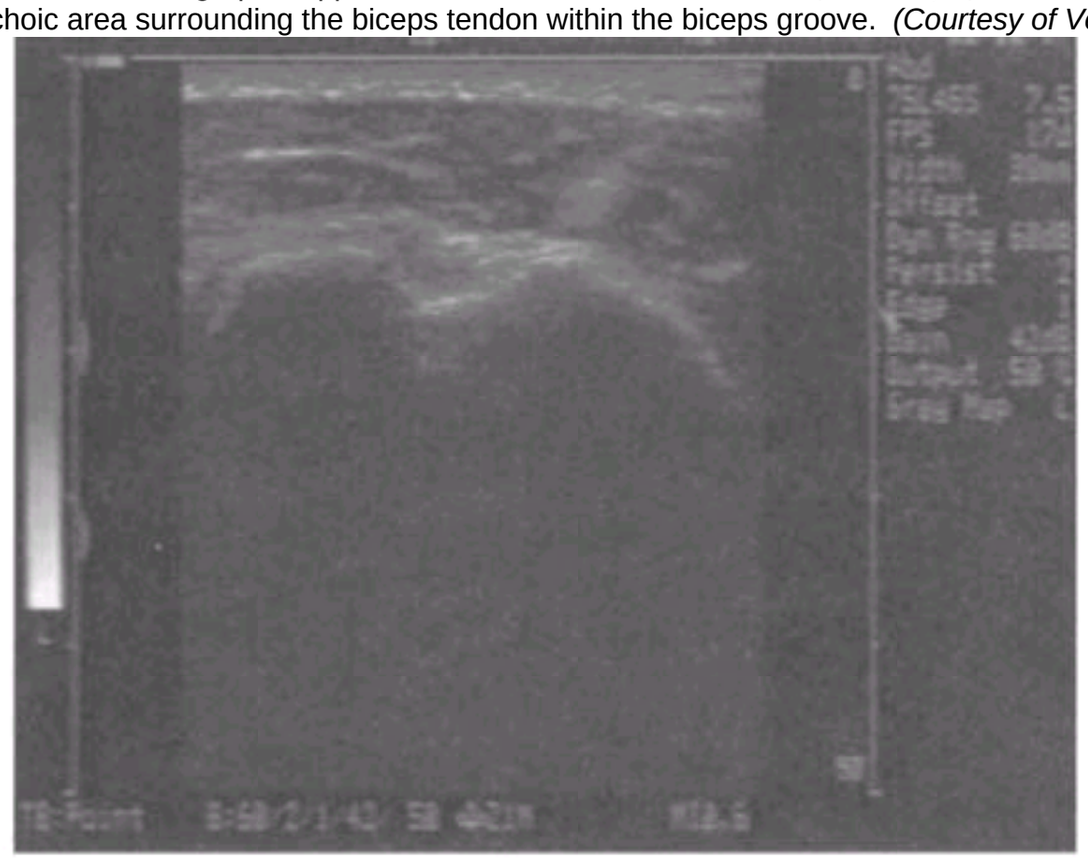
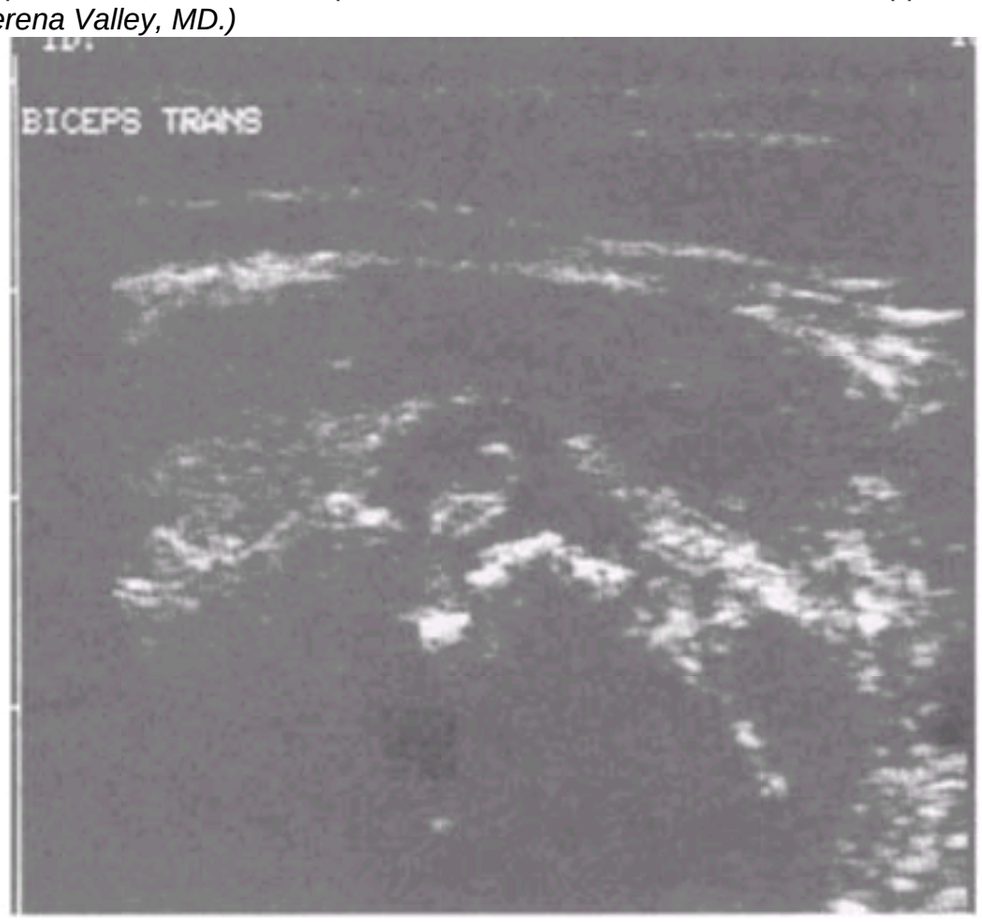


Figure 69-21 Sonographic appearance of the anterior shoulder. *A,* Normal; *B,* Effusion present; *C,* Schematic representation. Fluid within a shoulder will appear as an anechoic area surrounding the biceps tendon within the biceps groove. *(Courtesy of Verena Valley, MD.)*

A





B

C

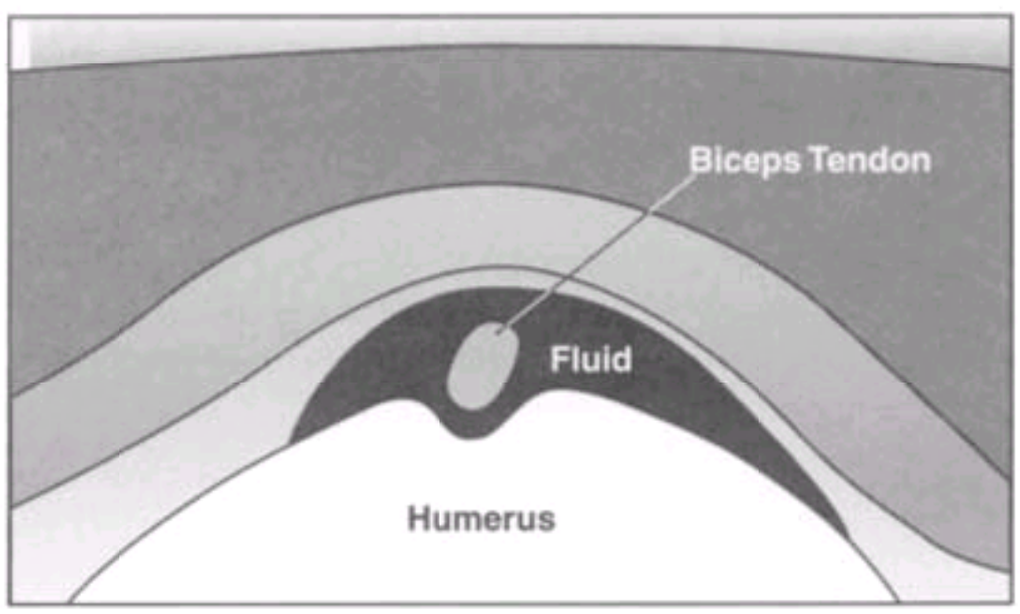


Figure 69-22 Probe positioning for the posterior view of the shoulder joint. The probe should be placed in the transverse position over the posterior aspect of the shoulder. The glenoid rim can be readily identified in this position. *(Lydia F. Roberts, Photographer)*

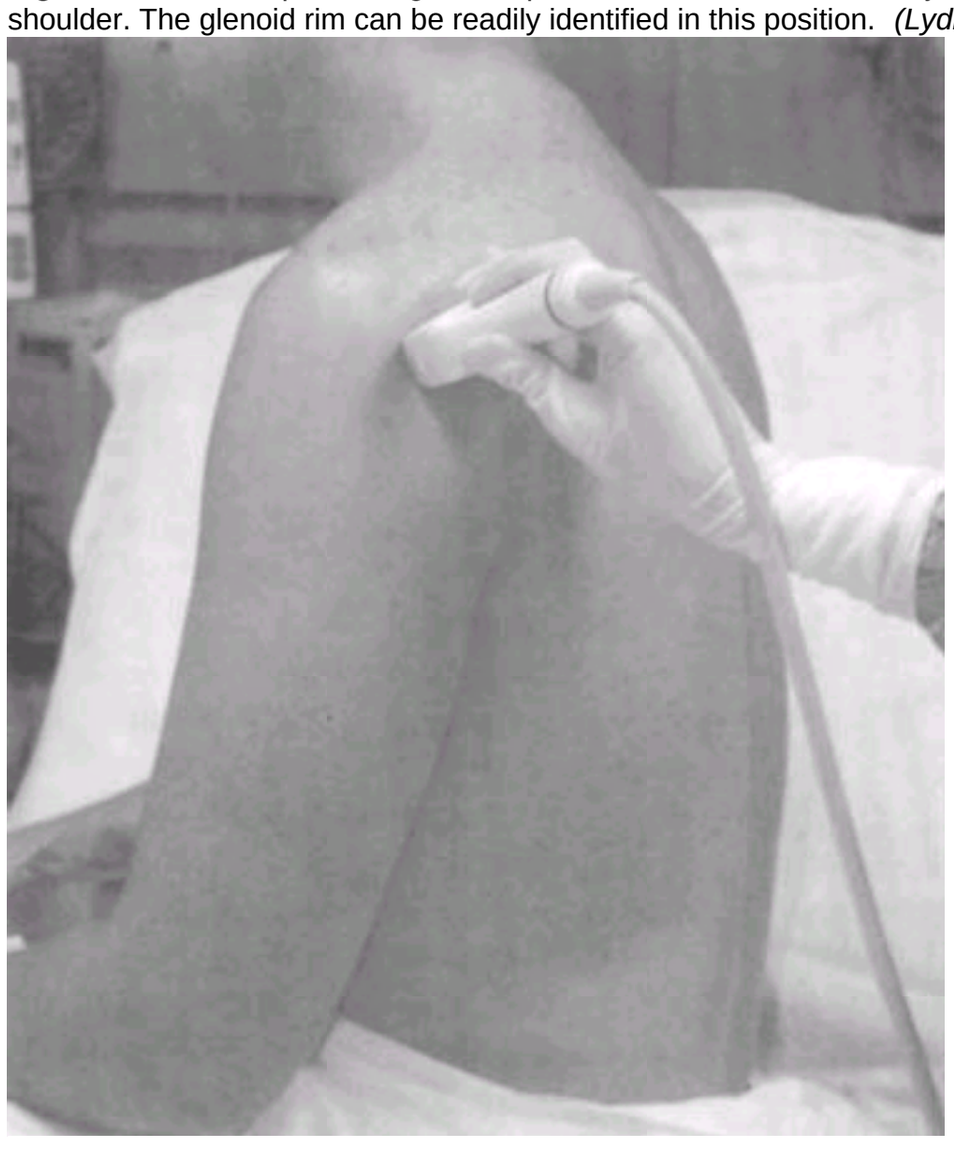
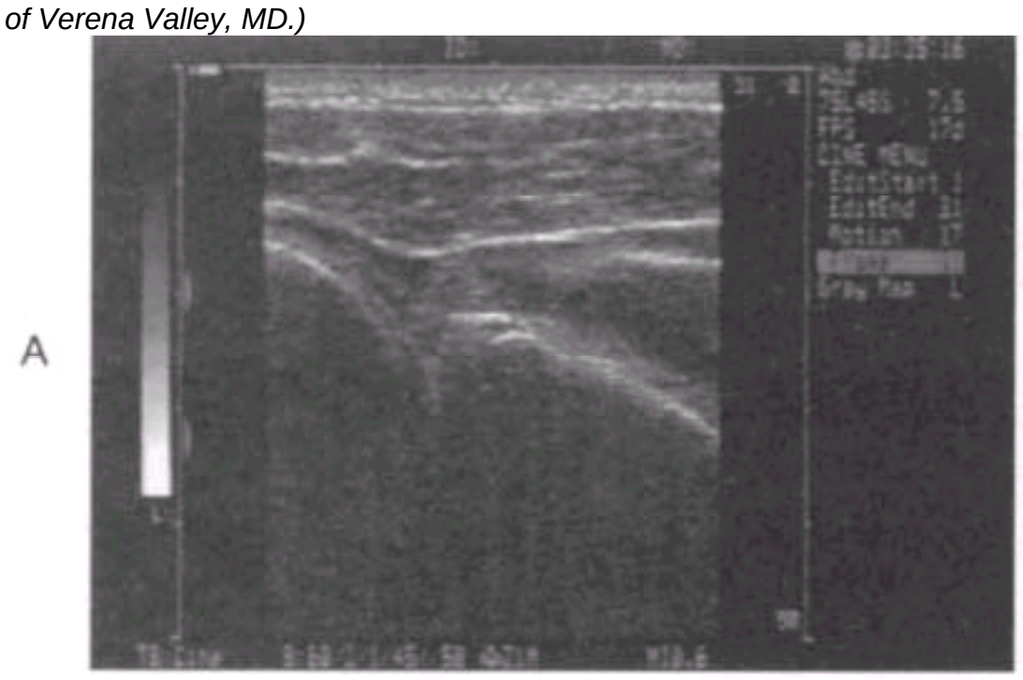
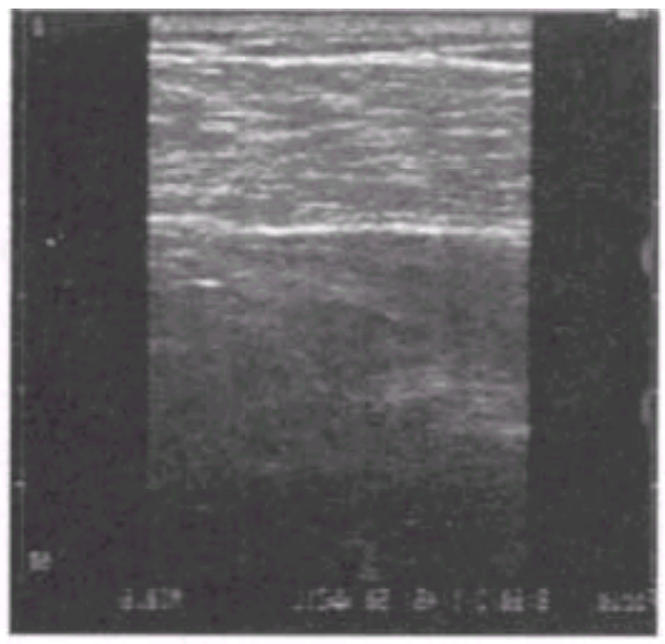


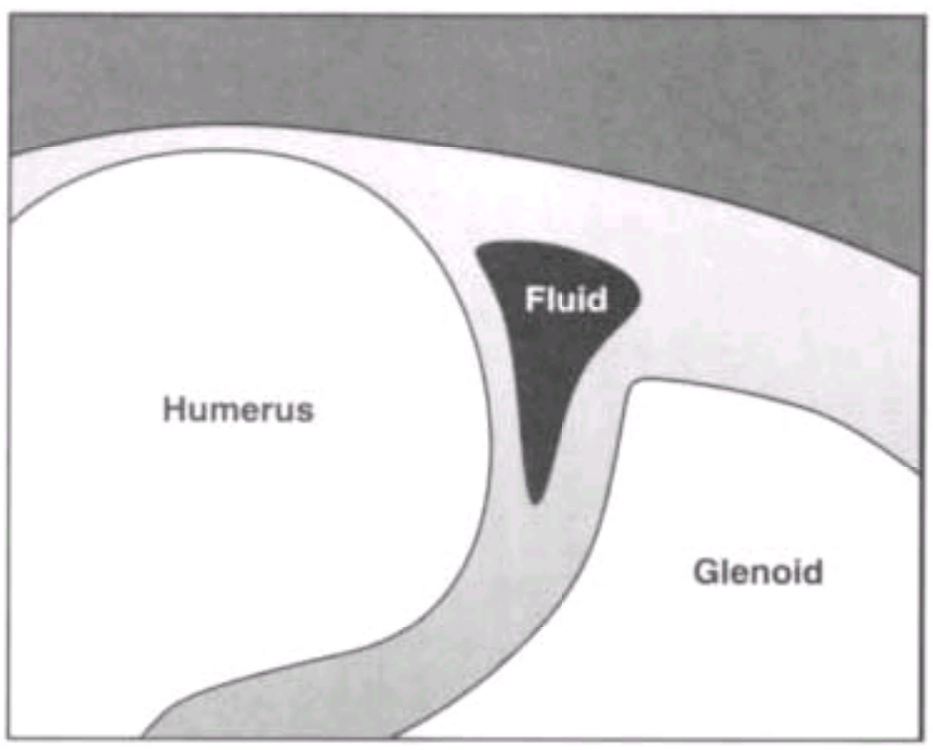
Figure 69-23 Sonographic appearance of the posterior shoulder. *A,* Normal; *B,* Effusion present; *C,* Schematic representation. Free fluid will appear as an anechoic collection medial to the glenoid rim. When fluid is identified in the anterior view, fluid can often be "milked" into the posterior space by moving the shoulder. *(Courtesy of Verena Valley, MD.)*



A



B



C

Figure 69-24 Probe positioning for assessment of ankle effusion. The foot should be positioned in plantar flexion, and the transducer oriented longitudinally over the anterior surface of the tibia. Moving the transducer toward the foot and seeing the curved dome of the talus will identify the joint space. *(Lydia F. Roberts, Photographer)*

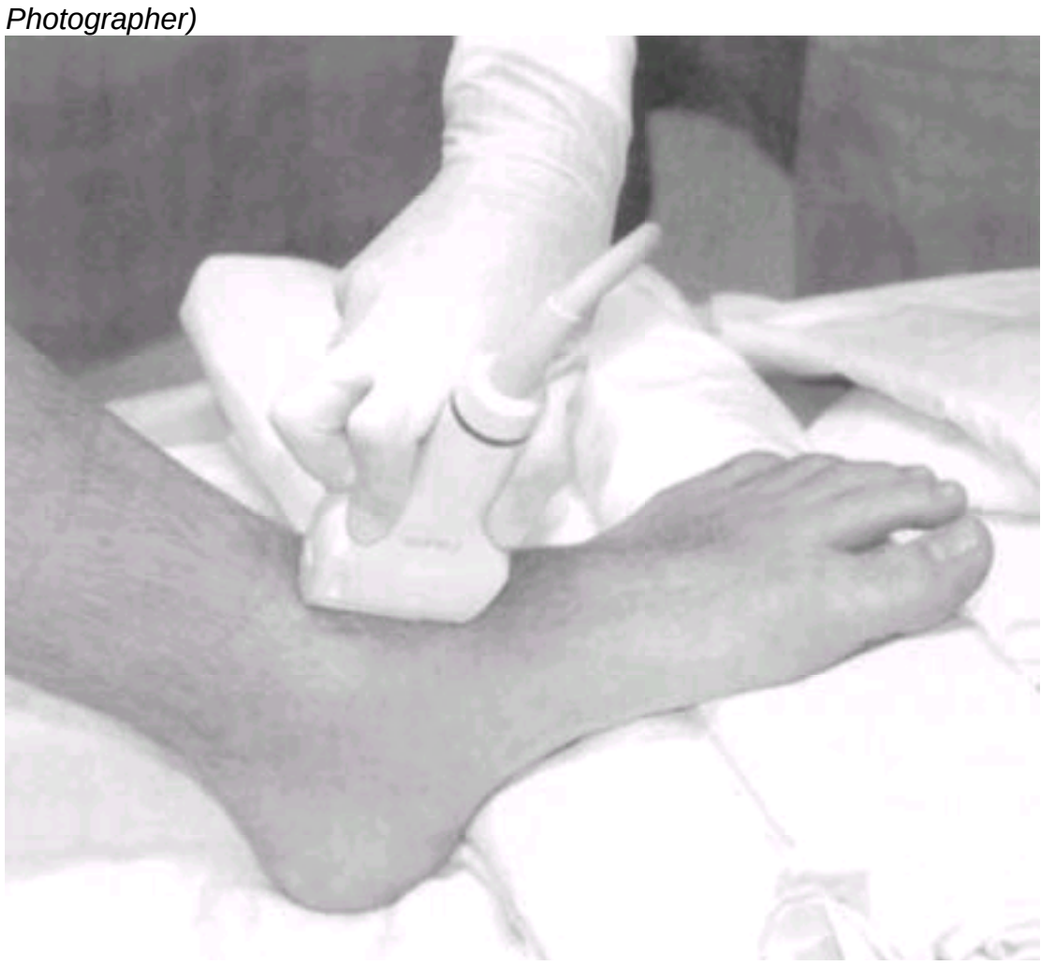
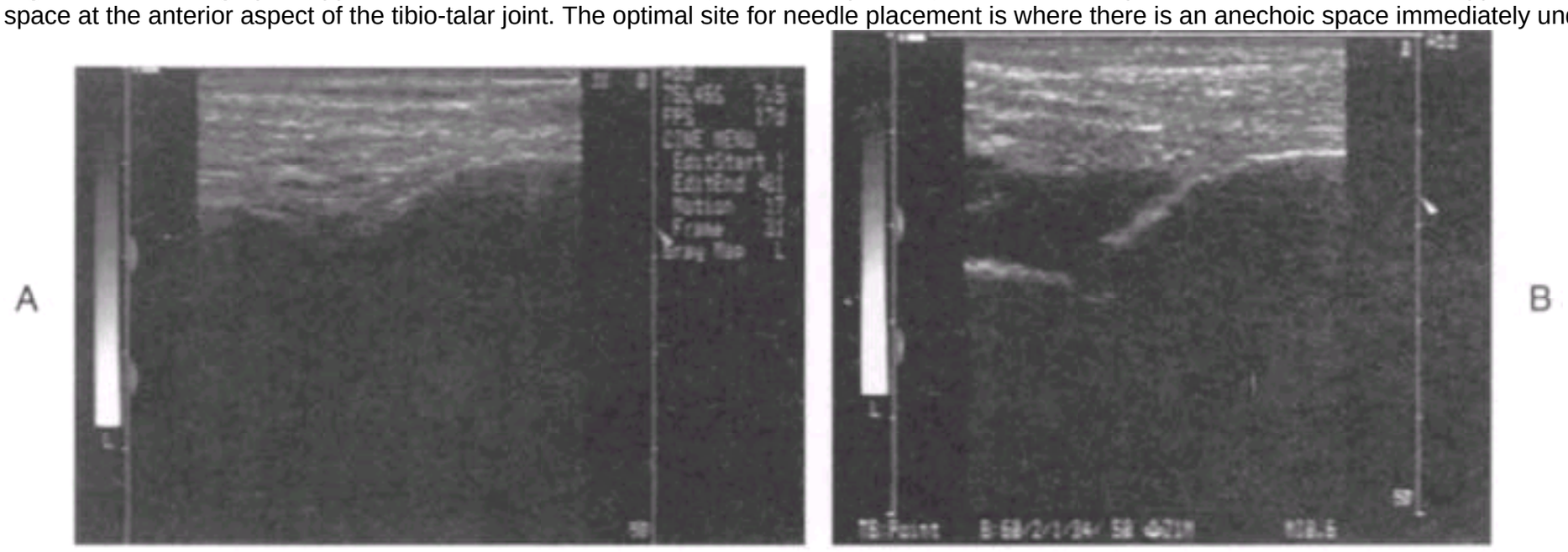
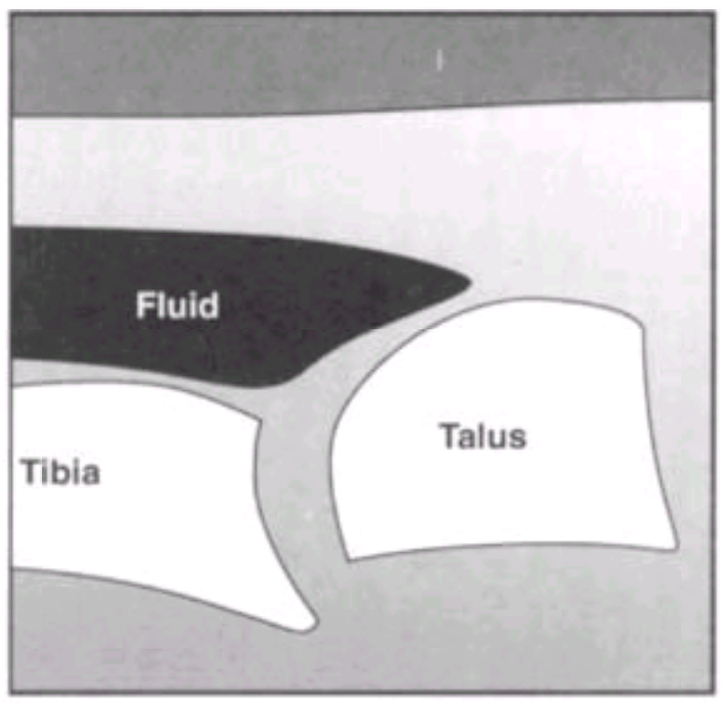


Figure 69-25 Sonographic appearance of the ankle. *A,* Normal; *B,* Effusion present; *C,* Schematic representation. An effusion will appear as an increased anechoic space at the anterior aspect of the tibio-talar joint. The optimal site for needle placement is where there is an anechoic space immediately under the probe.



A

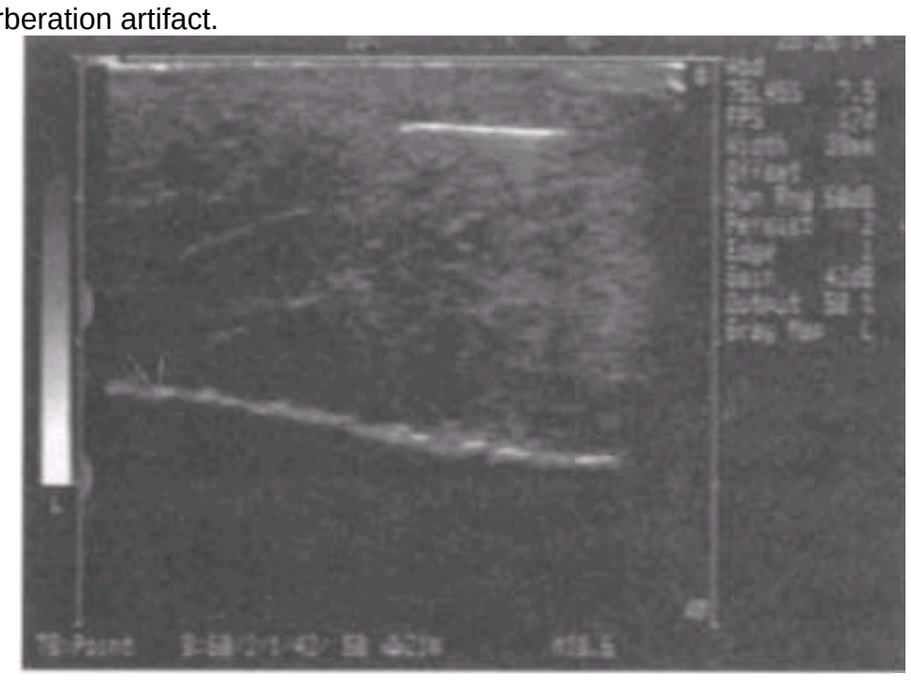
B



C

Figure 69-26 Sonographic appearance of a retained metallic foreign body (*A*) and a retained glass foreign body (*B*). Foreign bodies are usually hyperechoic, and will appear as brightly echogenic structures, distinct from surrounding bone or calcifications. Clues to the location of a foreign body will be acoustic shadowing and reverberation artifact.

A



B

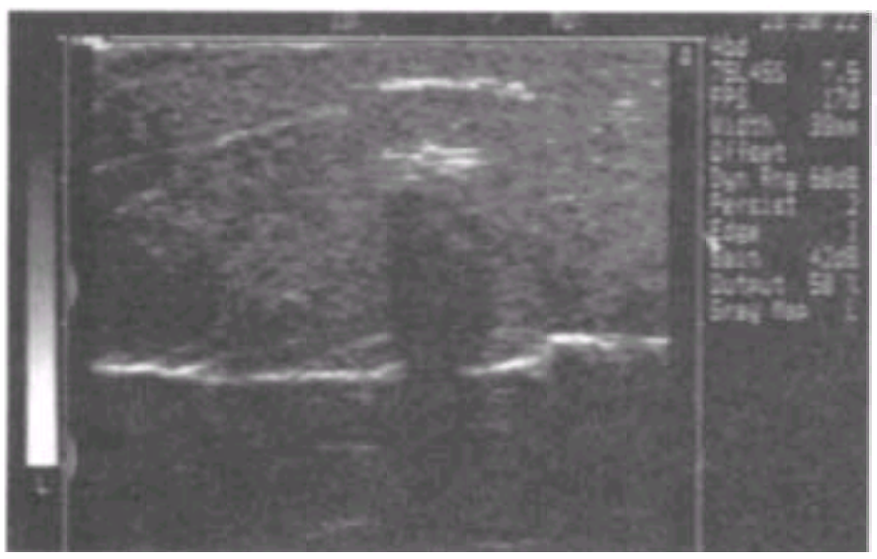


Figure 69-27 Sonographic appearance of the bladder in transverse view. The bladder will appear as an anechoic structure immediately beneath the skin surface, with a characteristic rhomboid appearance on transverse view.

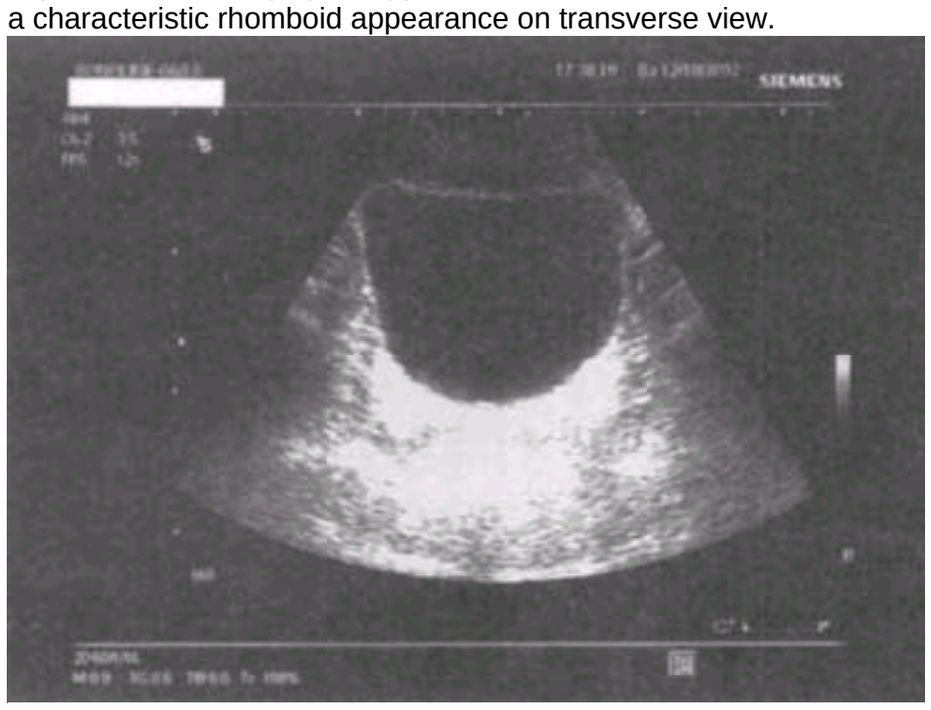


Figure 69-28 Sonographic appearance of a subcutaneous fluid collection. An abscess appears as a discrete anechoic area that may be surrounded by a brightly reflective wall. The fluid will be anechoic, but will often contain scattered echoes representing purulent material or necrotic debris. The fluid may also contain septations, which appear as echogenic walls traversing the fluid cavity.

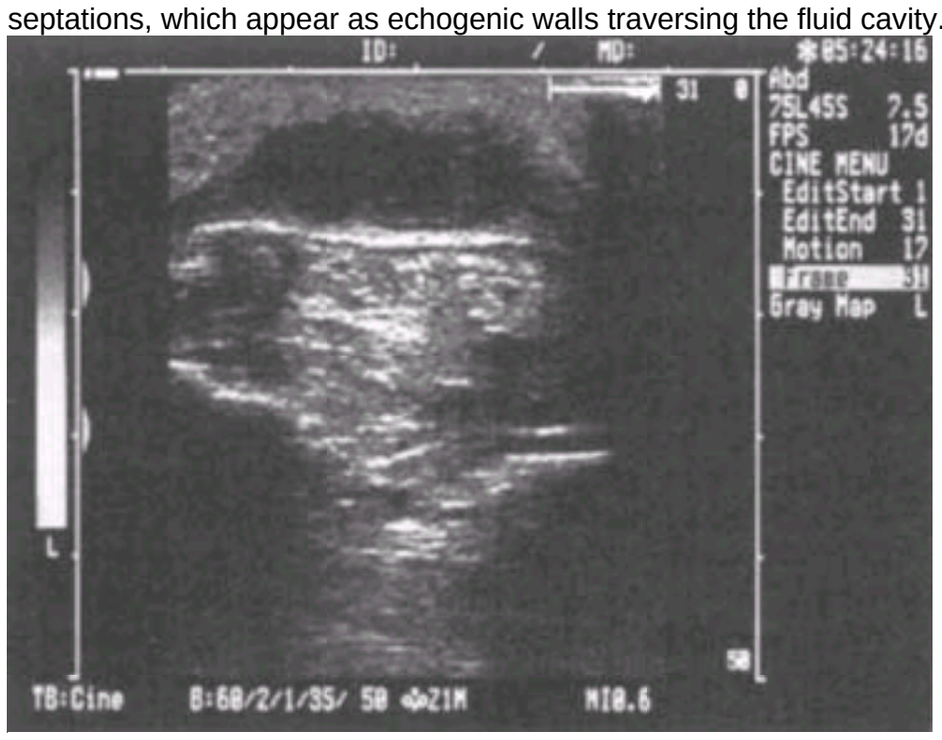


Figure 70-1 Algorithm for deciding method of obtaining urine specimen for evaluation of possible urinary tract infection. MSCC, midstream clean catch; SPA, suprapubic aspiration; UTI, urinary tract infection.

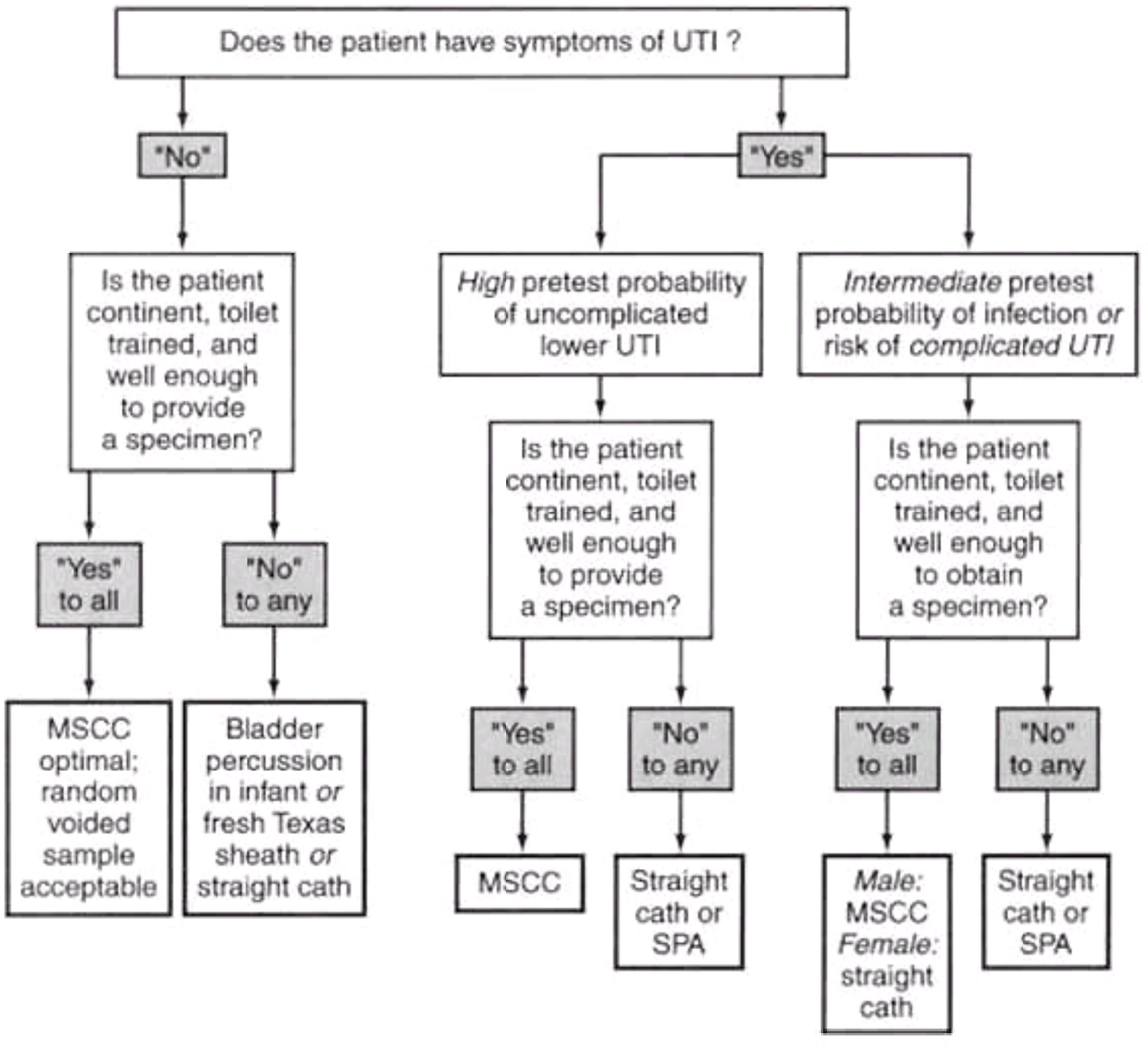


Figure 70-3 "Prism-shaped" calcium monohydrate crystals *(right)* resembling hippurate or urate crystals, and octagonal calcium dihydrate crystals *(left)*. *(Illustration by NJ Miller.)*

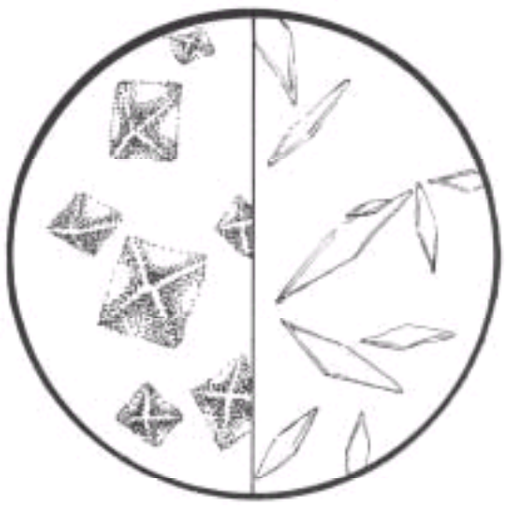


Figure 71-1 *A,* Recapping a needle by holding the cap in the hand is the most common way to sustain a needle puncture. *B,* It is best to discard the needle/syringe without recapping, but an alternative is to partially recap *without* holding the guard (needle cap), so that at least 80% of the needle is covered before completing the recapping with the second hand.

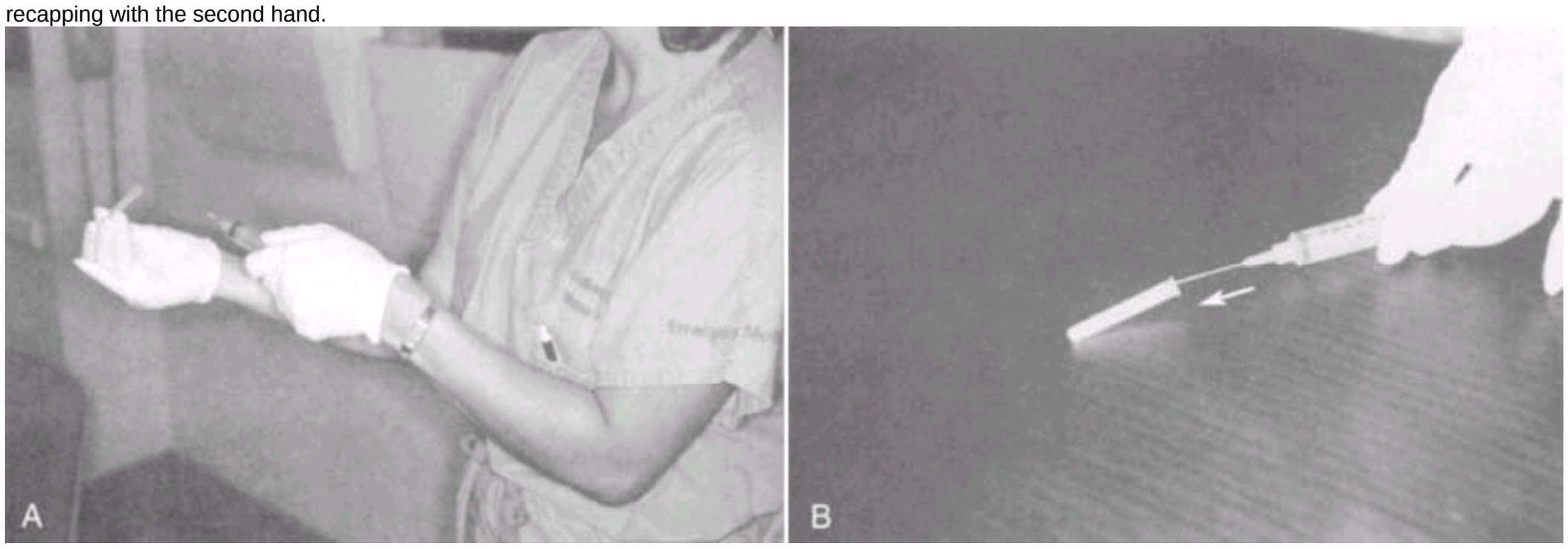


Figure 72-1 Procedural training at the bedside is an important part of learning a new skill. *A,* This ankle dislocation requires prompt relocation in the ED. *B,* The instructor first reviews the procedure with the trainee, including indications, contraindications, procedural steps, and complications. The trainee then performs the reduction while the instructor observes and provides guidance during the procedure. *C,* Following the procedure, the instructor examines the patient and assesses the adequacy of reduction.

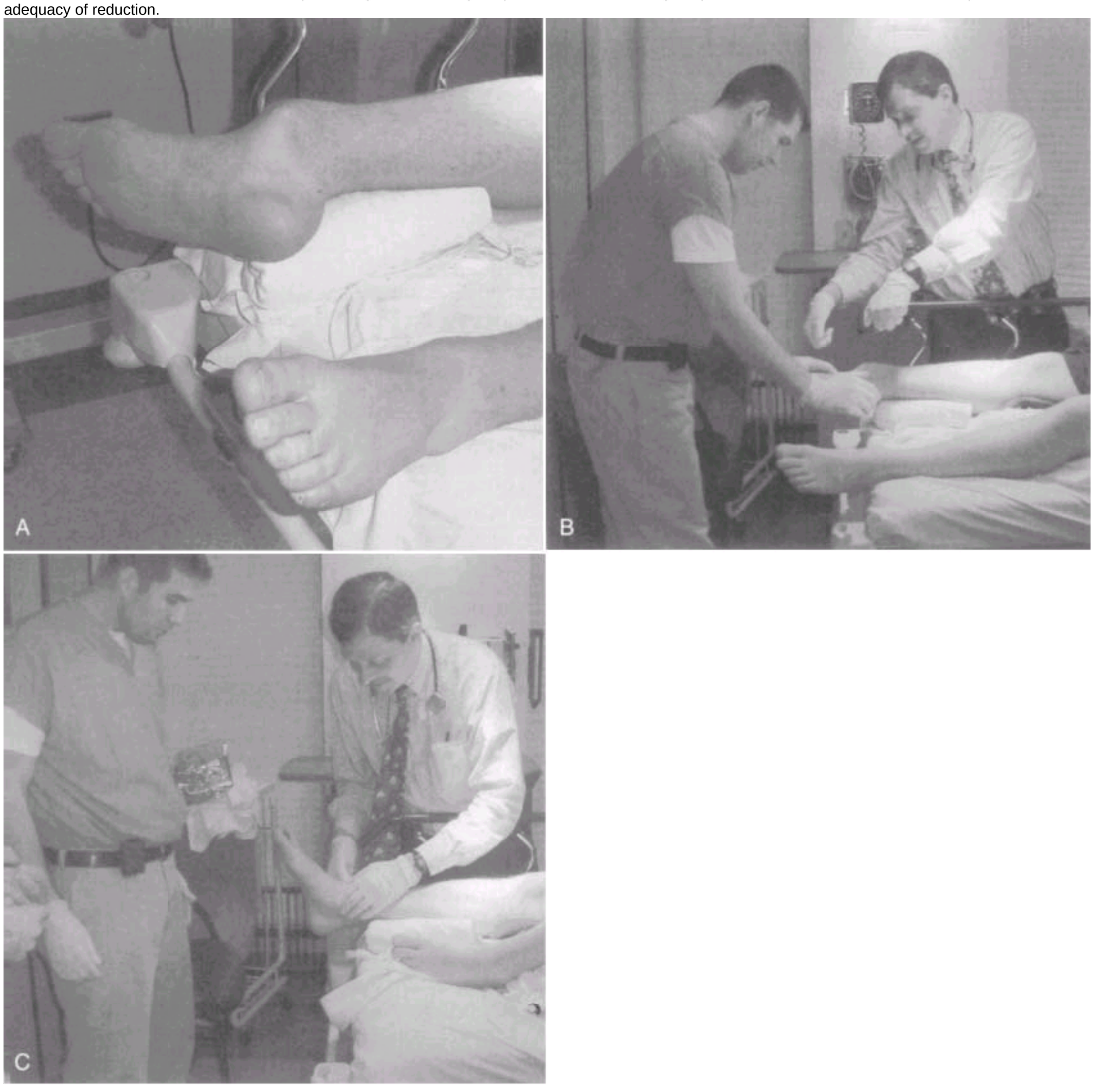


Figure 72-2 Volunteers are ideal models for teaching certain noninvasive procedures such as splinting and casting.

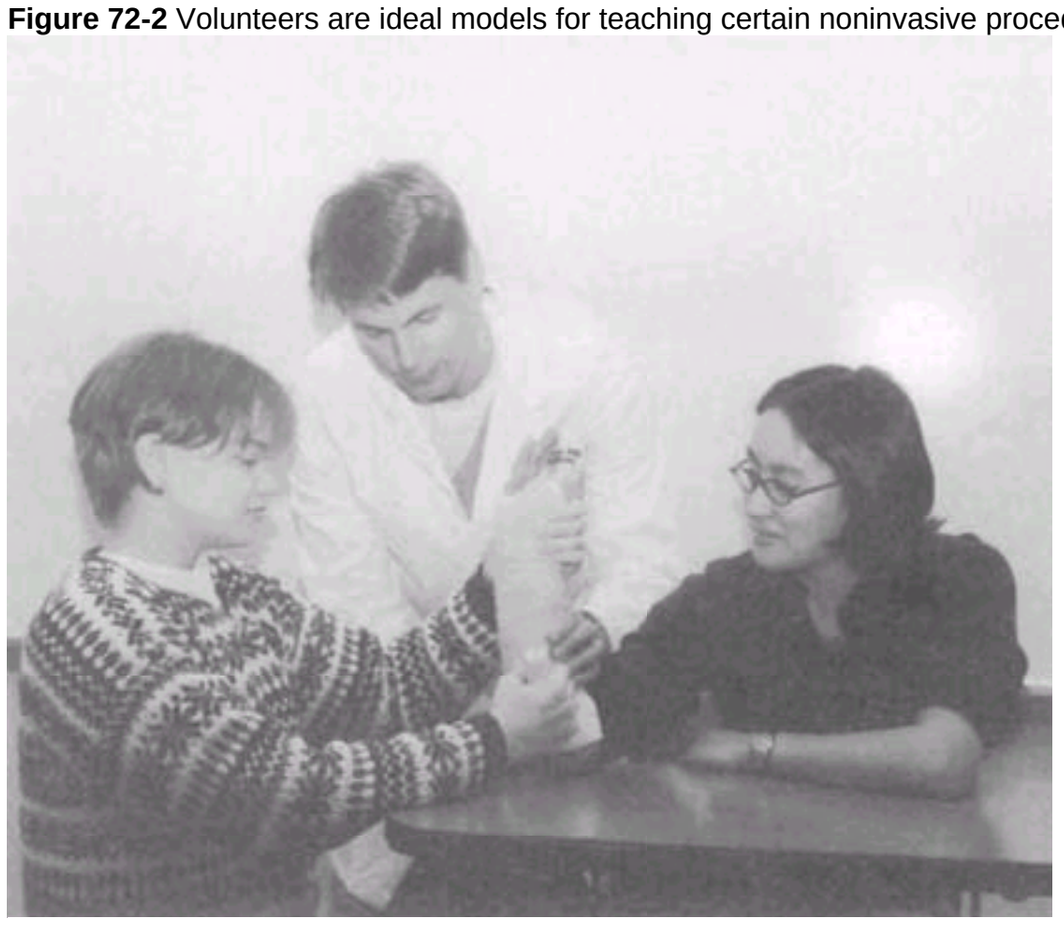


Figure 72-3 Mannequins are available to teach many procedures, including endotracheal intubation.

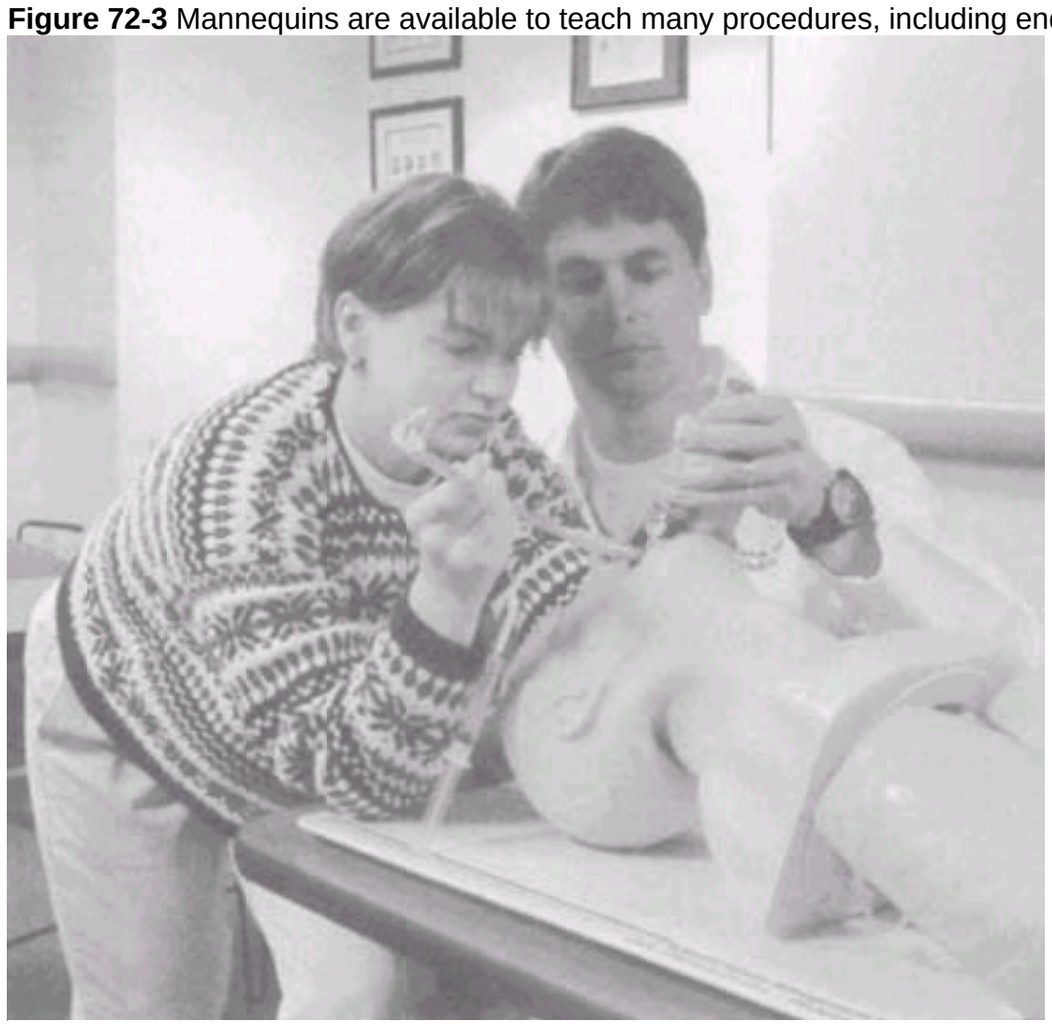


Figure 72-4 Human patient medical simulator. *A,* In the control room the educator is able to create and monitor clinical scenarios. *B,* The trainee performs electrical cardioversion on a human patient simulator and receives feedback from his actions by watching the monitor (successful conversion of ventricular tachycardia to sinus rhythm).

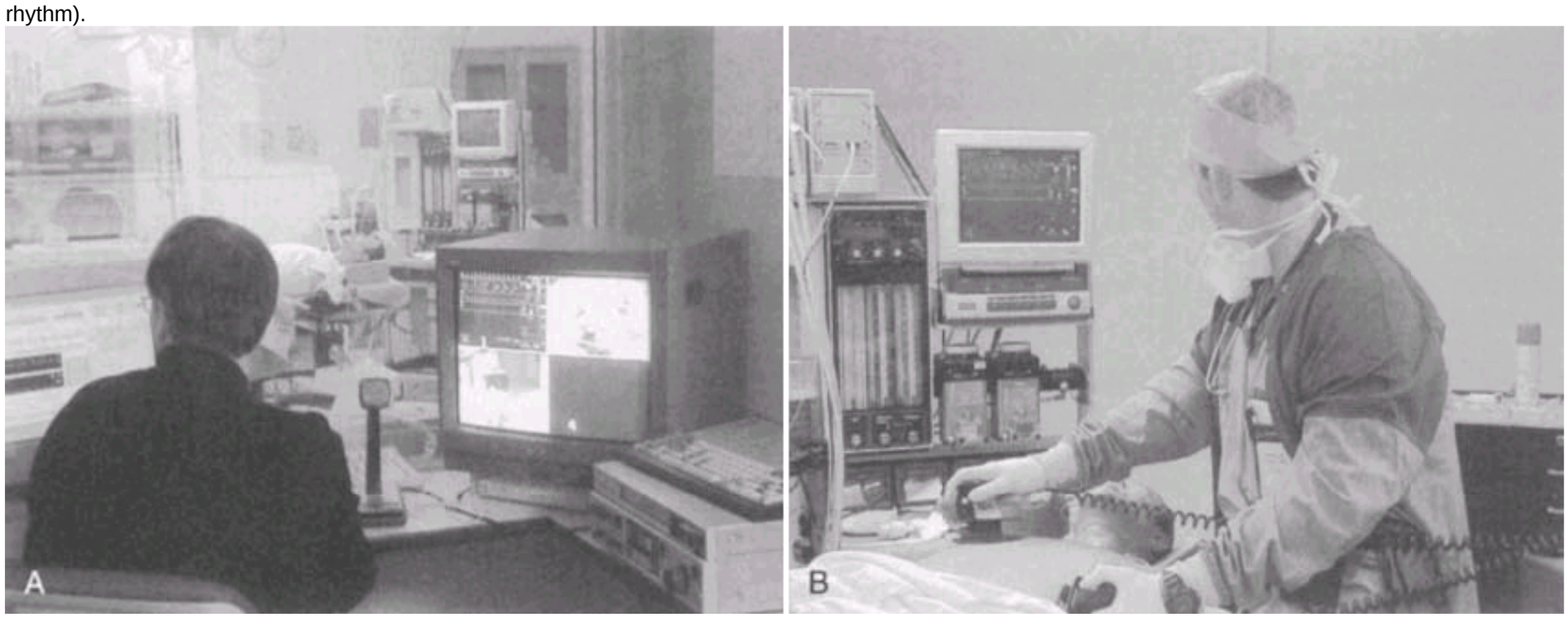


Figure 72-5 Isolated pig's feet are useful models to teach basic wound closure techniques.

Figure 72-6 In the live animal laboratory an instructor assists while the trainee performs a venous cutdown.

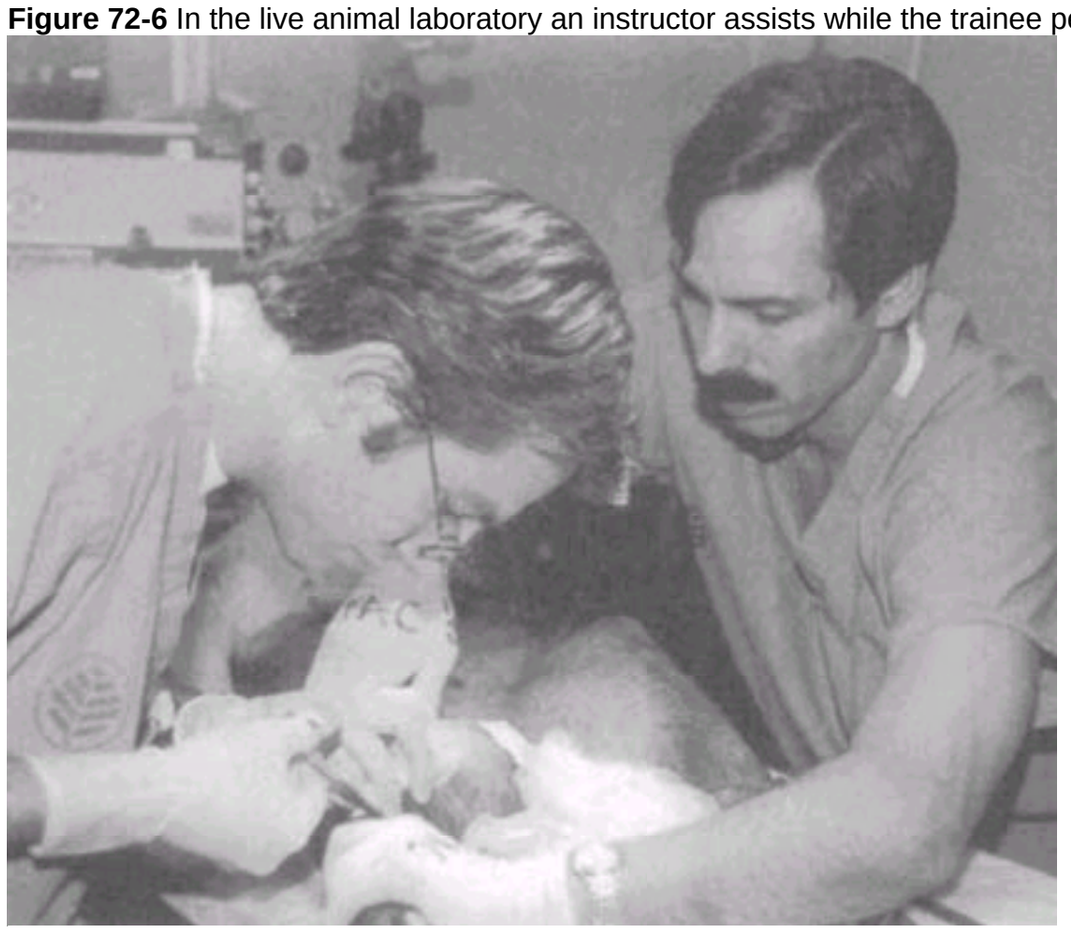
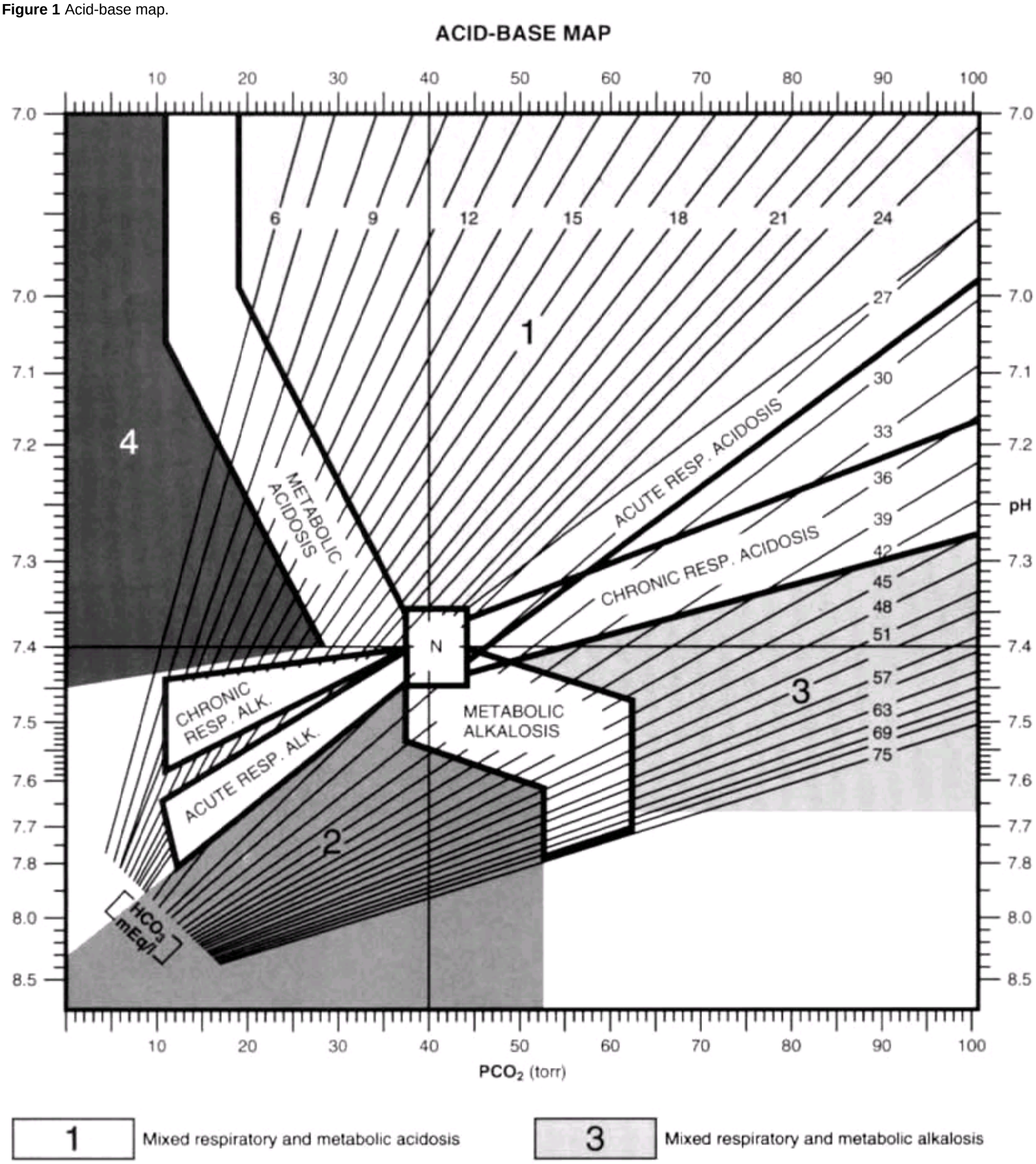


Figure 1 Acid-base map.



1 Mixed respiratory and metabolic acidosis	3 Mixed respiratory and metabolic alkalosis
2 Metabolic alkalosis and respiratory acidosis	4 Metabolic acidosis and respiratory alkalosis