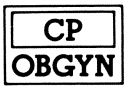
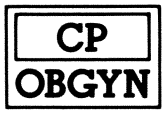


Clinical Perspectives in Obstetrics and Gynecology

Series Editor:

Herbert J. Buchsbaum, M.D.



Clinical Perspectives in Obstetrics and Gynecology

perspective *noun:* . . . the capacity to view subjects in their true relations or relative importance.

Each volume in Clinical Perspectives in Obstetrics and Gynecology will cover in depth a major clinical area in the health care of women. The objective is to present to the reader the pathophysiologic and biochemical basis of the condition under discussion, and to provide a scientific basis for clinical management. These volumes are not intended as "how to" books, but as a ready reference by authorities in the field.

Though the obstetrician and gynecologist may be the primary provider of health care for the female, this role is shared with family practitioners, pediatricians, medical and surgical specialists, and geriatricians. It is to all these physicians that the series is addressed.

Series Editor: Herbert J. Buchsbaum, M.D.

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Strategies in Gynecologic Surgery

Edited by
Herbert J. Buchsbaum
Leslie A. Walton

With 82 Illustrations

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To our families
Linda, Jonathan, and Julie
Jean, Sabrina, and Bradley

Preface

This volume is directed toward physicians who perform gynecologic surgery. The problems addressed are those encountered specifically in the performance of surgical procedures on the female genitalia. This book is not intended to be an atlas of surgery, but rather to supplement the many atlases available to the interested reader.

There is an increasing body of physiologic and clinical data that is directly related to gynecologic surgery. The editors have selected subjects for this volume that directly impact on the outcome of the operative procedure. Authors were chosen who have documented interest and expertise.

Many decisions made before the performance of the gynecologic operation can influence the outcome of the procedure, such as choice of abdominal incision, preparation of the operative field, and use of prophylactic anticoagulants and antibiotics. During the operation, additional choices that are open to the surgeon can affect the outcome: choice of suture material, technique of bladder drainage, and the use of drains. Attention to details of surgery, avoidance of genitourinary or gastrointestinal injury, and attention to potential problems in specific age groups can result in better outcome for the patient. The reader will also find chapters devoted to the legal implications of operative consent, the control of surgical bleeding, and intra- and postoperative monitoring.

It is our hope that the material in this book will help the gynecologic surgeon in his practice to improve the care of the female patient undergoing operative procedures on the genitalia.

We wish to thank our administrative assistants for their valuable contributions: Joyce Perry at the University of Pittsburgh, and Jackie Rogers and Dorcas Austin at the University of North Carolina. Their help was invaluable in coordinating the many aspects of manuscript preparation and editing. We would also like to acknowledge the efforts of Susan A. Kreml, Copy Editor, and the capable staff of Springer-Verlag.

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Preoperative Assessment 1

Leslie A. Walton

The Approach to the Patient

The gynecologist must utilize all available information in the delivery of surgical care to the patients. The advances in knowledge relevant to patient care, the increased availability of sophisticated technology, the heightened awareness of patients to diseases, and the continued refining of the physician–patient dialogue are among the factors that enhance the quality of care and contribute to the well-being of the patient.

The gynecologic surgeon is confronted by an array of challenging problems as he/she cares for the patients and aims for a successful outcome. The preoperative evaluation delves into the medical, surgical, social, and psychologic problems in an attempt to identify critical factors that might compromise the patient's hospital stay. This stay can be brief if the procedure is performed on an outpatient basis, or prolonged if an in-hospital stay is warranted. The patient involved can be young, healthy, and resilient, or older and burdened by diseases of aging. Whatever the patient's age, careful attention to all details is important as the patient approaches an operative procedure.

The increasing role of the federal government and third-party payers in health care costs has created a shift from in-hospital to outpatient care. In addition, there is an ongoing assessment of the need for many of the diagnostic studies that were routinely obtained in the past. Despite the increased emphasis on cost containment with curtailing of testing, the expectation of a good outcome from any surgical experience is still to be anticipated, and the delivery of medical care has been refined so that good outcomes are the rule rather than the exception. Thus, the preoperative evaluation of the patient is important in the total delivery of care of the surgical patient.

Finally, the most critical factor of the preoperative assessment is the patient herself. Operations on the female genital tract impinge heavily on the patient's psychosexual function. Whether the surgical procedures are for irregular bleeding, pelvic infection, or relaxation of pelvic structures, thoughts about sterility, cosmesis, and sexual function swirl in the patient's mind. These thoughts need to be addressed. It is important, for example, that we pay attention to the older patient's sexuality. Cognizance of these concerns and other problems play an important role in the preoperative period.

History and Physical Examination

Many significant details of the patient's history may be familiar to the physician because of prior outpatient care and treatment. These details should be reviewed and verified during a comprehensive preoperative history and physical examination. All facets of the chief complaint should be reviewed and attention directed to other organ systems. Since gyne-

cologists are now caring for an older population, the patient's history must emphasize a careful organ system review to look for cardiac, renal, or pulmonary disease. For example, a history of prolonged uterine bleeding alerts the surgeon to the deleterious effects of anemia on cardiac function and cardiac reserve, tissue healing, and response to stress. Endocrine abnormalities such as diabetes mellitus and gastrointestinal diseases such as diverticular disease are common among older women. A drug history is important in a search for allergic reactions. In addition, knowledge of drug dosage will alert the physician to look for side effects.

A history of multiple operations or repeat pelvic infections would alert the surgeon to the possibility of bowel adhesions and allow him to prepare the bowel preoperatively. Finally, bone and joint disease such as osteoporosis and arthritis may become important in positioning of the patient, planning for cushioning of joints if stirrups are used, and planning of postoperative activity of the patient. The existence of previously performed diagnostic studies is also important. These studies might alert the physician as to the need for further diagnostic studies or may assist him in assessing the severity of preexisting medical conditions.

Thus, a thorough assessment of the patient's health and disease status will enable the physician to provide total and comprehensive care. The process of obtaining a pertinent history will enhance the patient's confidence in her physician, and she may be comforted as she approaches the operating room setting.

Careful physical examination is mandatory in search of new findings that might indicate worsening of the patient's disease condition. It can never be assumed that previous physical findings have remained unchanged. An ovarian cyst diagnosed in the office could have ruptured and not be present at the in-hospital examination. Similarly, the presence of a 6-cm cystic mass in a patient now complaining of abdominal pain could indicate that the cyst has undergone torsion. In addition, a new and unrelated condition could have developed in the intervening days. As an example, the older patient with the diagnosis of bilateral adnexal masses could have developed a third abdominal-pelvic mass and have diverticular abscesses rather than tuboovarian abscesses. Careful attention to symptoms of the lower gastrointestinal tract would be helpful in the latter example.

The physical examination should be performed with keen emphasis on factors that will impinge on the patients's response to the surgical procedure. Weight and blood pressure are important. Complete examination of the head and neck might identify conditions that can complicate the operative process. A goiter or tracheal deviation could make endotracheal intubation difficult. Large, pendulous breasts would aggravate ventilation by mask and would compromise full diaphragmatic expansion postoperatively. Cardiac irregularities or murmurs require evaluation and, occasionally, the administration of prophylactic antibiotics.

The physical examination should look for deformities that might complicate the proper positioning of the patient. Careful evaluation of the extremities is necessary: for example, venous thromboses, may modify the intraoperative and postoperative care of the patient.

Abdominal and pelvic examinations are critical in the patient's evaluation. The working diagnosis must be confirmed. A detailed description of the pelvic findings should be carefully recorded. An examination under anesthesia should precede the surgical procedure for educational and diagnostic reasons.

A thorough and complete examination enables the physician to assess whether surgery is the appropriate method of treatment. In addition, a thorough evaluation sustains the patient's confidence and prepares her psychologically for surgery.

Preoperative Troubleshooting in the Gynecologic Patient

The dynamic approach to the patient about to undergo surgery requires that certain baseline questions be answered. Does the patient require further diagnostic tests? Does the present illness mimic a nonsurgical disease? Is this the appropriate time for a surgical intervention or would temporization and medical care relieve her suffering or improve her response to surgery? Are there therapeutic measures that can be instituted that would enhance the response to surgery and the recuperation

period? Is this a patient whose surgery could be delayed to obtain autologous rather than homologous blood should intraoperative transfusion become necessary? Is the patient about to be subjected to too many risks because of the operative procedure? Is the patient's psychologic support system geared toward a positive outcome to the operative procedure and the postoperative period?

The Assignment of Surgical Risk

The assignment of surgical risk is an important preoperative consideration. The classification outlined below has been in use for some time to assess the risk of mortality from surgical procedures and is known as the American Society of Anesthesiology (ASA) Physical Status Classification[1] (Table 1-1.). All preoperative patients should be assigned to one of the five categories.

It is obvious that anesthesia and surgical morbidity worsen as the physical status traverses from status I to status V.

Table 1-1. ASA Physical Status Scale[a]

Physical status	Patient category
I	A normal healthy patient.
II	A patient with mild-to-moderate systemic disease, e.g., anemia, morbid obesity.
III	A patient with severe systemic disease that limits activity not to the point of incapacitation, e.g., healed myocardial infarction, diabetes mellitus.
IV	A patient with incapacitating systemic disease that is life threatening, e.g., renal insufficiency.
V	A moribund patient who is not expected to survive, e.g., a patient with massive pulmonary embolus.

[a] From Owens et al. (1).

The Cardiovascular System

The absence of cardiac symptoms, confirmed by studies, is one indicator for a satisfactory recovery from the surgical procedure. However, normal physiologic and metabolic responses occur as a result of an operative approach, and if these responses are exaggerated, intraoperative and postoperative cardiac problems can develop.

In the perioperative period, it has been clearly shown that patients have an increased sympathetic activity as evidenced by an increased urinary output of catecholamines. In addition, increased levels of adrenocorticotrophic hormone (ACTH) and its accompanying stimulation of glucocorticoids and aldosterone result in elevation of these hormones. Antidiuretic hormone (ADH) levels also increase. Some patients will exhibit mild elevation of blood pressure, hyperglycemia, and fluid retention as a result.[2] While these alterations may not create major variations from normal baseline values, they can lead to systemic changes if aggravated.

All anesthetic agents depress myocardial function,[3] including halothane and nitrous oxide, anesthetics that are frequently used. Sodium pentothal, used for induction of anesthesia, also depresses myocardial contractility. As a result there is a reduction in venous return, and the available intravascular volume diminishes. The decreased myocardial contractility can lead to a significant diminution in cardiac output with resultant hypotension if not compensated by reflex tachycardia. The young, healthy patient with no cardiac disease can easily compensate for these changes with reflex tachycardia, while the older patient may experience a decrease in coronary perfusion and suffer from myocardial ischemia and other sequelae.

A number of older patients have some degree of myocardial fibrosis. Positive-pressure anesthesia results in an elevation of the intrathoracic pressure and a reduction in venous return and cardiac output. Since significant degrees of hypoxemia are often seen following surgery, this fall in oxygen tension and a decreased cardiac output might compound any preexisting cardiac disease and create organ system compromise in the postoperative period. Whenever a preoperative patient exhibits signs of hemodynamic instability, consideration should be given to the use of Swan–Ganz catheter monitoring.

Cardiac Risks. In 1977, Goldman and associates[4] published a large study in which they ex-

Table 1-2. Cardiac Risk Factors

Risk factor	Points (no.)
1. Age greater than 70 years	5
2. Myocardial infarction in previous 6 months	10
3. S3 gallop or jugular venous distension	11
4. Important aortic stenosis	3
5. Rhythm other than sinus or premature atrial contraction	7
6. More than five premature ventricular contractions per minute documented any time prior to surgery	7
7. Poor general medical condition, e.g., elevated BUN, bedridden patient	3
8. Intrathoracic, intraperitoneal, or aortic operation	3
9. Emergency operation	4

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amined cardiac risk factors involving 1001 patients over 40 years of age who were subjected to major, noncardiac operations. These authors evaluated the role of nine independent correlates and the role of these correlates in predicting life-threatening complications. These correlates were assigned a point value according to their relative importance (Table 1-2).

As one computes the patient's totals, four risk categories were defined (Table 1-3). Serious cardiac morbidity is directly proportional to class.

According to the Goldman classification, therefore, any patient with a risk index of 26 or more points should undergo surgery only if it is a life-saving procedure. Patients with index scores of 13–25 (class III) probably have sufficient cardiac risks to warrant routine preoperative medical consultation.[4]

Table 1-3. Class and Morbidity

Class	Points (no.)	Percentage of cardiac deaths or life-threatening complications
I	0–5	0.9
II	6–12	7.0
III	13–25	14.0
IV	>26	78.0

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Other categories of patients with cardiac disease need to be considered. Patients with stable angina pectoris who have not had a previous myocardial infarction do not appear to be at significantly increased risk when an elective surgical procedure is performed. Patients with unstable angina of less than 3 months duration constitute an absolute contraindication to noncardiac surgery except in emergency situations. Patients with documented coronary artery disease[5] constitute a special group and should be considered for cardiac bypass operation prior to major, noncardiac surgery. In addition, patients with fixed cardiac output as a result of diseases such as tight aortic or mitral stenosis are at increased risk. They require cardiac consultation prior to surgery.

Patients with myocardial infarction pose a special problem. The Mayo Clinic studies[6] showed that 6.6% of patients with a previous myocardial infarction and only 0.13% of patients without a previous infarction suffered a second infarction in the postoperative period. Advances in anesthesia did not change this morbidity rate when this study was repeated 6 years later.[7] The re-infarction rate was higher for upper abdominal and thoracic procedures. The risks of re-infarction increase dramatically if surgery is performed within the first 6 months after an acute myocardial infarction. Thus, elective surgery should always be postponed for at least 6 months following either a transmural or nontransmural myocardial infarction.[4,6]

Patients with "compensated" cardiac function following treatment for congestive heart failure are not at increased risk in noncardiac surgery. Patients with uncompensated congestive heart failure, especially patients with an S3 or jugular venous distension, are at risk for additional cardiac problems and more specifically pulmonary edema.[8] If a patient has more than five premature ventricular contractions per minute at any time prior to the surgical procedure, she is at increased cardiac risk. Intraoperative therapy with lidocaine is indicated, but it is not known whether administration of this drug decreases cardiac complications. The patient with atrial fibrilla-

tion does not seem to be at increased risk if cardiac rate is adequately controlled before surgery.[8] Conduction system disease such as third-degree A-V block, type I or II A-V block, and sinoatrial block are among the cardiac irregularities that require a temporary pacemaker prior to elective surgery.[9,10] Patients with unstable angina also require a temporary pacemaker prior to surgery.

Prophylaxis against Endocarditis. Transient bacteremia is a common phenomenon. While a higher incidence occurs in nongynecologic conditions, transient bacteremia occurs after urethral catheterization, sigmoidoscopy, and pelvic infections. Prophylactic antibiotic therapy is indicated in patients very susceptible to bacteremia. The cardiac lesions[11,12] that merit prophylactic antibiotics are listed in Table 1-4.

The Role of Hypertension. During intubation, the systolic blood pressure can be elevated by 20–45 mm Hg. This elevation occurs in 6% of normotensive patients and in 17% of patients with a history of hypertension.[8] Provided that there are no sequelae from this elevation, no further complications should arise.

Mild-to-moderate hypertension does not increase the morbidity from elective surgery.[13] Patients with elevated blood pressure prior to surgery have a high incidence of intraoperative *decrease* in blood pressure, but there is no increased incidence of cardiac complications. Patients with stable diastolic pressures up to 110 mm Hg can undergo surgery but require close intraoperative and recovery room monitoring. If hypertension is uncontrolled, however, elective surgery is contraindicated.

Antihypertensives and Other Cardiovascular Medications Prior to Surgery. The standard dictum has been to discontinue antihypertensive medications before surgery so as to decrease the risk of intraoperative hypotension.

Table 1-4. Cardiac Conditions Requiring Prophylactic Antibiotics

Prosthetic valves of all types
Acquired valvular lesions
Congenital cardiac lesions
Mitral valve prolapse

Patients with mild hypertension can discontinue their medication prior to elective surgery. The enforced bed rest of hospitalization contributes to normal pressure readings. However, most hypertensive patients will require the continuation of their medication. Some authors[13] recommend drug dose reduction. Other authors[14] recommend that antihypertensive drugs should be continued in full dosage until the night preceding surgery and be restarted in the immediate postoperative period. The anesthesiologist should be aware of the patient's medication.

Comments about a few drugs are in order. Clonidine hydrochloride should not be discontinued, because the abrupt cessation of the drug leads to severe rebound hypertension. Clonidine may cause hypotension because of the additive effect of anesthesia. Once the anesthesiologist is aware of this complication, careful intraoperative monitoring can be instituted. Propanolol should be continued pre- and postoperatively. Digitalis does not significantly increase the cardiac risks and can be continued, although bradyarrhythmias are more frequent in patients who are digitalized after hospital admission. This finding probably signifies an unstable digitalis level.[9] Nitroglycerine compounds should be continued during and after surgery.

The Respiratory System

The young gynecologic patient may have asymptomatic pulmonary disease that will become manifest with routine preoperative evaluation. The older patient can be more symptomatic, and a detailed and orderly review of pulmonary function is warranted.

The vital capacity can be decreased by as much as 45% for several days postoperatively in patients undergoing surgical procedures on the thorax and upper abdomen.[15] This decrease is less in pelvic procedures. In addition, residual volume and functional residual capacity are among the lung volume decreased by general anesthesia.

Hypoxemia occurs after surgery. The mean Pa_{O_2} drops from 88 preoperatively to 63 immediately after surgery.[15,16] This fall is caused primarily by small airway collapse intraoperatively and postoperatively. Shunting and V–Q mismatch is a sequelae.

A history of asthma, exposure to environmental pollutants, or illnesses such as sarcoidosis may predispose the patient to postoperative problems such as atelectasis and bronchitis. The incidence of these latter two conditions is about 10%–20% in the normal healthy postoperative patient. It is decreased to the 10% range in patients undergoing lower abdominal surgery.[17] Chronic smoking predisposes the patient to these complications and further sequelae such as pneumonia.

An awareness of medications being ingested for pulmonary disease is important. One of the common classes of asthmatic medication, the adrenergic drugs, cause vasoconstriction, cardiac stimulation, and bronchodilatation. Another class of drugs, the glucocorticoids, have no direct vascular effects, but affect the stress response, tissue healing, and fluid and electrolyte balance.

Many factors place the patient at risk for pulmonary complications:

a. Smoking, especially chronic
b. Obesity, especially morbid obesity
c. Known pulmonary disease
d. Age of the patient (over 70 years)

In addition, patients with an inability to complete a graded exercise test and patients with heavy sputum production (greater than 30 ml/day) are at risk for postoperative complications.

Simple assessment tests for respiratory function include arterial blood gases and pulmonary function testing (PFT). Preoperative PFT is indicated in the patients at risk for pulmonary complications. Preoperative arterial Pa_{O_2} less than 50 mm Hg[15] and/or Pa_{CO_2} greater than 15 mm Hg are significant contraindications to pelvic surgery.

The patient's pulmonary status can be optimized by such measures as cessation of smoking, weight reduction, and the use of incentive spirometry. Bronchodilators and chest physiotherapy for a few days preoperatively have been shown to increase the Pa_{O_2}. In one study,[16] patients given preoperative and postoperative intensive pulmonary therapy had a 22% incidence of pulmonary complications, while those not receiving treatment had a 60% incidence of pulmonary complications.

The patient with pulmonary diseases needs special attention. Acute asthmatic bronchitis affects approximately 25 million Americans.[18] Many patients might be relatively asymptomatic or might present with symptoms of dyspnea, chest tightness, and/or a chronic cough. When symptomatic, tachypnea and wheezing are the classic symptoms. If asthma or equivalent disease is suspected or diagnosed, blood gas determination and pulmonary function studies are indicated. A useful study is the measurement of the forced expiratory volume (FEV). If this value is less than 75% of predicted value, surgery can proceed with the use of general anesthesia. If the patient has moderate or severe symptoms, FEV changes more than 75% of predicted value,[19] and abnormal P_{O_2} and P_{CO_2} values, intensive evaluation and treatment prior to surgery is recommended.

It is important that bronchial irritants such as smoking be removed, that the patient is well hydrated to allow expectoration of secretions, and that premedication avoid drugs such as codeine, morphine, and cholinergic agonists. Quiescent asthma can flare up during general anesthesia and require the administration of intravenous theophylline.

The patient with chronic obstructive pulmonary disease deserves special attention. An established preoperative routine should be utilized to reduce the incidence of postoperative complications. The presence of purulent sputum indicates the need for a course of antibiotics in addition to the therapy outlined above.

The Hematologic System

Hemorrhage is a major risk for the patient undergoing surgery. A hematocrit in the 30% range or a hemoglobin level of 10 g/dl is required prior to elective gynecologic surgery. The requirement can be ignored when dealing with patients with sickle-cell disease, chronic renal disease, and other such conditions. A 30% hematocrit value does not allow a great margin of safety. In the young anemic healthy patient, it is advisable that pelvic surgery be postponed, if feasible, while the patient is offered iron, vitamins, and folic acid to facilitate the production of red blood cells. If the nature of her disease contradicts postponement of the operative procedure, transfusion with packed red cells is preferable to whole blood in order to reduce the problems associated with volume

expansion. Washing of the red cells reduces the possibility of transfusion reactions. One unit of red cells has an average hematocrit in the range of 70%–80% and will raise an adult's hematocrit by about 3 volumes percent.

On the other end of the spectrum, patients with hematocrit values as high as 57%, as in polycythemia vera, are at increased risk for hemorrhagic and thrombotic complications.[20] These patients have a surgical mortality rate approaching 36%. Elective surgery should be deferred until therapy reduces the hematocrit to the 42%–47% range and the platelet count to less than 500,000/mm^3.

Compromise of platelet function can be diagnosed preoperatively. Aspirin increases the bleeding complications because it inhibits platelet function with resultant prolongation of the bleeding time. This inhibition can occur for up to 10 days after aspirin ingestion. This bleeding problem is worsened when low-dose heparin is given concomitantly. Carbenicillin and ticarcillin inhibit platelet function and create a prolongation of the bleeding time. The effect can last for several days after the drugs are discontinued. Malnutrition and bowel sterilization reduce vitamin K levels and create clotting deficiencies. Indomethacin and phenylbutazone also inhibit platelet release but only for a short period of time. Antihistamines, dopamine, doptamine, and the nonsteroidal anti-inflammatory drugs also inhibit platelet function.

Functional platelet disorders are rare. Von Willebrand's disease, Bernard Soulier's syndrome, and thrombasthenia create abnormal bleeding time. These patients have a history of easy bruisability and a history of bleeding problems during prior surgical procedures. The platelet count is usually normal. Prothrombin time and partial thromboplastin time (PTT) will be normal in these conditions. Uremia creates bleeding by depression of platelet function.

While decreased platelet counts exposes the patient to increased risk of bleeding, only counts below 15,000–20,000/mm^3 may require prophylactic platelet transfusion. However, prolongation of bleeding should alert the physician to study clotting factors. Usually, patients with a platelet count above 100,000 are not at increased risk for bleeding unless the operation results in excessive blood loss and a consumptive coagulapathy. Although preoperative coagulation profiles are routinely ordered, there is little data to substantiate the need for these tests in the absence of a positive history, or drug ingestion.

The Urinary Tract System

The routine gynecologic patient presents with few renal problems. If symptoms are present, pressure or obstruction needs to be considered. Routine urinalysis is indicated.

During anesthesia, renal blood flow and glomerular filtration is reduced, resulting in decreased urine output. Age, renal disease, nephrotoxic medications, and peritonitis jeopardize renal status. A rough measure of renal function is obtained by the determination of creatinine clearance using the Cockroft–Gault formula.[21] The formula is as follows:

$$\text{Cr} + (140 - \text{age}) \times \text{kg}/72 \times \text{creat (mg/dl)}$$

It is age dependent and applicable to adult males. The creatinine clearance for women is 85% that of the male value. The creatinine clearance is valuable when measurements are made with stable renal function and a stable serum creatinine. (See Chapter 10.)

One of the basic radiologic studies utilized by the gynecologist is intravenous pyelography (IVP). This study will usually alert the physician as to the existence of anatomic distortions or anomalies of the tract or intrinsic renal disease. Knowledge of the BUN and creatinine is important before an IVP is ordered. Older patients, diabetics, patients who are dehydrated, or patients with intrinsic renal disease can develop renal insufficiency after dye studies such as intravenous pyelography.[22] Intravenous pyelogram can safely be performed if the serum creatinine is 2 mg/dl or less. When intravenous pyelogram is contraindicated, noninvasive renal ultrasound is a useful substitute.

The presence of chronic anemia could be the result of chronic renal disease. Patients with uremia have platelet dysfunction, which could result in a prolonged bleeding time during surgery. In addition, the presence of a chronic low hematocrit would predispose the patient to poor healing and cardiac dysfunction. Accordingly, it is desirable to have the

hematocrit corrected to an acceptable level before general anesthesia is administered.

The toxic effects of drugs on the bladder and kidney must be kept in mind. Gentamicin is known for its renal toxicity. This drug can create elevation of the BUN and creatinine after at least 2–3 days of administration. Adequate drug history will alert the physician to seek for any renal deficiency.

Patients with mild renal failure (serum creatinine less than 3 mg/dl) generally tolerate surgery very well.

Endocrine Considerations

Diabetes mellitus is encountered in the older gynecologic population. In addition to the difficulties of hypo-hyperglycemia and ketoacidosis, suboptimal control of the diabetic patient will result in persistent glycosuria. If patients have an accompanying osmotic diuresis, changes in electrolytes would occur. In addition, diabetic patients are at increased risk for postoperative infections, usually with a gram-negative organism. Accordingly, tighter control of diabetes is necessary to prevent electrolyte disturbances. In addition, better control enhances wound healing and lessen the chance of postoperative infection.

The diabetic patient might require hospitalization prior to surgery so that a battery of tests can be performed. The surgical procedure should be performed early in the morning. When insulin administration is necessary, a suggested protocol is as follows:

Minor surgery—withhold insulin until after the procedure

Major surgery—administer ⅓ of total daily regular insulin dose preoperatively and ⅓ postoperatively

The outpouring of catecholamines and corticosteroids secondary to surgical stress will accentuate the hyperglycemia. In addition, two commonly used anesthetics, halothane and ether, both cause hyperglycemia. Blood sugar determinations can be obtained intraoperatively to assist in the management of the patient. Urine glucose measurements are useful only if renal glucose threshold coincides with plasma glucose.

Adrenal insufficiency is rare in the gynecologic patient. However, relative adrenal insufficiency secondary to exogenous steroid administration can occur; inhaled steroids or a 7-day course of oral steroids can depress adrenal function for several days.[23] These patients are unable to mount an adrenocortical outpouring under the stress of surgery. Replacement therapy for adrenal insufficiency is necessary. One replacement protocol uses hydrocortisone sodium succinate as follows:

50 mg I.M. ½ hr preoperatively
15 mg/hr I.V. intraoperatively
10 mg/hr I.V. postoperatively
Postoperative days 1–2, 50–100 mg I.V./8 hr
Postoperative days 3–4, 50 mg IM q. 12 hr
Postoperative days 5–7, hydrocortisone 30 mg orally at 0700 hr and 20 mg orally at 1400 hr

In addition, occasionally patients are seen who are on steroid therapy for collagen or other disease conditions. Steroids are known to interfere with wound healing and suppress the immune response. Prednisone at a dose of 7.5 mg/day for at least 5 days can lead to adrenal suppression. These patients also require steroid supplementation at the time of surgery.

The duration of replacement therapy will depend on the extent of adrenal suppression and the extent of the surgical procedure. A brief surgical procedure with minimal adrenal challenge will allow the termination of steroids immediately after surgery.

Adrenal excess, that is, pheochromocytoma is always considered but rarely if ever seen. If symptoms of this disease are found, appropriate diagnostic studies are indicated and elective surgery is absolutely contraindicated.

The Gastrointestinal Tract

Gynecologic conditions may upset the functioning of the gastrointestinal tract as a result of direct pressure or involvement of the adjacent bowel or as a result of a generalized process in which the gastrointestinal tract is one of a multi-organ-system involvement. While the incidence of direct gastrointestinal involvement by benign pelvic diseases is low, the incidence of gastrointestinal symptoms is high.

In the preoperative evaluation, a history of gastrointestinal symptoms is important. Vomiting should alert the physician to assess serum

electrolyte levels. In addition, fluid replacement needs to be considered if vomiting and dehydration have created a contracted intravascular volume.

Changes in intestinal motility can create abdominal distension, and interfere with diaphragmatic excursion. Preoperative bowel decompression via long tube is sometimes necessary. In addition, emptying the colon via enemas enhances pelvic exposure, lessens the chance of bowel injury and subsequent contamination, and hastens bowel recovery after pelvic operations.

In the absence of intestinal involvement by pelvic disease, bowel symptoms are important if they point to primary diseases of the gastrointestinal tract. In addition, it is important that a dietary history be obtained from the patient as one measure of nutritional status.

General Considerations

The presurgical anesthesia visit is important to establish the doctor–patient relationship. The patient is able to meet one of the major members of her surgical team, and can acquire an understanding of the process that will enable her to tolerate the surgical procedure. A review of her suitability for anesthesia is another rung in the support system.

Body surface preparation of the gynecologic patient is important. Established routines are followed for the abdominal and vaginal preparation (see Chapter 4). Select patients may benefit from prophylactic antibiotics at the time of abdominal and vaginal hysterectomy (see Chapter 8). The use of support stockings properly applied before surgery is important. Numerous approaches are used to reduce or prevent thromboembolic phenomenon (see Chapter 11). These are some of the many considerations to be reviewed in the preoperative phase.

Fluid and Electrolye Considerations. Vomiting and diarrhea in the gynecologic patient can result in deficiency in serum electrolyte content. Of major importance are the serum levels of potassium and sodium. Correction of hypokalemia is mandatory prior to surgery. Hypokalemia can interfere with cardiac and renal function, aggravate effects of anesthesia, and create acid–base imbalance.

Gynecologic patients also experience relative clinical dehydration, especially patients under care for sepsis and patients whose overall oral fluid intake is restricted. Occasionally, the restriction of fluid and the bowel preparation necessary for diagnostic tests such as intravenous pyelography (IVP) and barium enema (BE) contribute to relative intravascular volume depletion. This volume must be replaced prior to surgery.

Nutritional Considerations. A satisfactory nutritional condition fortifies the patient's immune status, helps resist opportunistic infection, and enhances wound repair. Bistrian et al.[24] reported that at least half of the patients on both public and private wards are in some state of protein-caloric deprivation. The obvious nutritionally deficient patient will present with weight loss, muscle atrophy, poor skin turgor, and an inappropriate complexion. However, most patients on the gynecologic service would not exhibit such obvious signs of malnutrition.

The assessment of nutritional status can be made briefly by physical measurements and blood studies. Height and weight are not reliable indicators of nutritional deficiences. The amount of body fat can be measured by using the Lange caliper and measuring the nondominant midarm triceps skin-fold thickness[25] and then comparing this result with published standards. Muscle mass[25] can be measured by the use of the nondominant midarm circumference. This value is subtracted from the triceps skin-fold thickness and the result multiplied by 0.314. This figure is then checked for deviation from standard sources. Values greater than 90% of predicted values are within the normal range.

Finally, clinical studies can be used to screen for undernutrition. A serum albumin concentration less than 3.4 g/dl indicates some degree of malnutrition, if obvious liver and renal diseases are excluded. Similarly, reduced serum transferrin value indicates that malnutrition is present. Transferrin levels between 170 and 180 mg/dl have been associated with major operative complications.[26] A total lymphocyte count of less than 1,500/mm^3 indicates protein

depletion.[27] Most acute nutritional depletion can be corrected by an intensive 5-day nutritional support program.

Psychologic Considerations. A number of authors have carefully documented the fact that women have psychologic concerns after genital tract procedures such as tubal ligation or hysterectomy. While the exact incidence is variable, depending on the patient's presurgical psychologic state, many women experience varying degrees of depression after hysterectomy.[28] Women are concerned about the loss of the uterus and its effect on their femininity and sexual responsiveness. Some manifestations of the depressive state can be ascertained preoperatively by appropriate psychologic testing. Preoperatively, women have expressed other concerns about the effects of hysterectomy. When some or all of these concerns are expressed, assurances and supportive explanations can reduce or prevent the patient from encountering psychologic problems postoperatively.

In addition to psychologic counseling, other aspects of preoperative preparation have been shown to be beneficial. Wilson[29] prepared a group of 70 patients undergoing elective surgery (37 patients had abdominal hysterectomy) with education about the upcoming experience and muscle relaxation training. He found that these patients had a reduced hospital stay, required less pain medication, and experienced a return to general well-being that was faster and smoother.

The Role of Routine Tests

The current emphasis on assessing cost and yield of routine diagnostic studies has resulted in an ongoing evaluation of the routine tests ordered on the preoperative patient. A detailed history and physical examination will in large measure determine the need for tests beyond the routine baseline hemoglobin, hematocrit, and white blood cell count. The usefulness of routine urinalysis has not been substantiated, and this study can be replaced by multitest dipstick urine analysis at considerable savings. Only blood urea nitrogen (BUN) is indicated in the absence of a history of urinary tract problems. In two series of patients studied,[30,31] chest x-rays were of no benefit in patients under 30 years of age undergoing elective surgical procedures if a careful history and physical examination did not reveal any suspicion of cardiac or pulmonary disease.

Blood glucose determination is indicated in the older population because of the prevalence of diabetes mellitus. Prothrombin time (PT) activated partial thromboplastin time (aPTT) and thrombin clotting time (TCT) are not indicated[32] unless a careful history, physical examination, and disease diagnosis warrant suspicion of a bleeding disorder.

The contribution of some routine diagnostic studies needs to be reassessed. The intravenous pyelogram (IVP) is one of the standard studies utilized when extensive pelvic dissection is indicated. Indications for ordering this test have become selective. IVP should give way to ultrasound studies in the diabetic patient. Barium enemas should be ordered on specific indications for symptoms referable to the gastrointestinal tract or the patient at high risk for colonic cancer. Electrocardiograms can be deferred in the patient under 35 years of age[33] with a benign cardiac history and negative physical examination. Careful review of physical activity and exercise tolerance is an integral segment of the history-taking.

In summary, patient age, disease diagnosis and prevalence of associated other disease coupled with a careful and detailed history and physical examination will determine the need for specific preoperative testing.

References

1. Owens WD, Felts JA, Spitznagel EL Jr. ASA physical status classification: a study of consistency of ratings. Anesthesiology. 1978; 49:239–243.
2. Glasser SP, Spoto E Jr, Solomon DA, et al. When cardiac patients become surgical patients. Hosp Pract. March 1979; 165–173.
3. Philbin DM, Bland JHL. The cardiovascular effects of anesthetics: an introduction. Int Anesthesiol Clin. 1979; 17:1–11.
4. Goldman L, Caldera DL, Nussbaum SR, et al. Multifactorial index of cardiac risk in non-cardiac surgical procedures. New Engl J Med. 1977; 297:845–850.
5. Mahar LJ, Steen PA, Tinker JH, et al. Perioperative myocardial infarction in patients with coronary artery disease with and without aorto-coro-

nary artery bypass grafts. J Thorac Cardiovasc Surg. 1978; 76:533–537.
6. Tarhan S, Moffitt EA, Taylor WF, et al. Myocardial infarction after general anesthesia. JAMA. 1972; 220:1451–1454.
7. Steen PA, Tinker JH, Tarhan S. Myocardial reinfarction after anesthesia and surgery. JAMA. 1978; 239:2566–2570.
8. Goldman L, Caldera DL, Southwick FS, et al. Cardiac risk factors and complications in noncardiac surgery. Medicine. 1978; 57:357–370.
9. Pastore JO, Yurchak PM, Janis KM, et al. The risk of heart block in surgical patients with right bundle branch block and left axis deviation. Circulation. 1978; 57:677–680.
10. Venkataraman K, Madias JE, Hood WB. Indications for prophylactic pacemakers in patients with right bundle branch block and left anterior hemiblock. Chest. 1975; 68:501–506.
11. Kaplan EL, Anthony BF, Bisno A, et al. Prevention of bacterial endocarditis. Circulation. 1977; 56:139A–143A.
12. Sipes JN, Thompson RL, Hook EW. Prophylaxis of infective endocarditis: a re-evaluation. Ann Rev Med. 1977; 28:371–391.
13. Breslin DJ, Swinton NW Jr. Elective surgery in hypertensive patients preoperative consideration. Surg Clin North Am. 1970; 50:585–592.
14. Edwards WT. Preanesthetic management of the hypertensive patient. New Engl J Med. 1978; 301:158.
15. Tisi GM. Preoperative evaluation of pulmonary function. Am Rev Resp Dis. 1979; 119:293–310.
16. Cain HD, Stevens PM, Adaniya R. Preoperative pulmonary function and complications after cardio-vascular surgery. Chest. 1979; 76:130–135.
17. Pierce AK, Robertson J. Pulmonary complications of general surgery. Ann Rev Med. 1977; 28:211–221.
18. White WH. Social and economic aspects. NIAID task force report: asthma and other allergic disease, p. 7. 1979; NIH Pub No 79:387.
19. Gracey DR, Divertie MB, Didier EP. Preoperative pulmonary preparation of patients with chronic obstructive pulmonary disease: a prospective study. Chest. 1979; 76:2, 123–129.
20. Wasserman LR, Gilbert HS. Surgical bleeding in polycythemia vera. Ann NY Acad Sci. 1964; 115:122–130.
21. Martinez-Maldonade M. Handbook of renal therapeutics. London: Plenum Medical Book Co, 1983: p. 211–212
22. Byrd L, Sherman RL. Radiocontrast-induced acute renal failure: a clinical and pathophysiologic review. Medicine. 1979; 58:270–279.
23. Wyatt R, Waschek MS, Weinberger M, et al. Effects of inhaled beclomethasone diproprionate and alternate day prednisone on pituitary adrenal function in children with chronic asthma. New Engl J Med. 1978; 299:1387–1392.
24. Bistrian BR, Blackburn GL, Hallowell E, et al. Protein status of general surgical patients. JAMA. 1974; 230:858–860.
25. Wright RR. Comments on nutritional assessment. JAMA. 1980; 244:559–560.
26. Buzby GP, Mullen JL, Matthews E, et al. Prognostic index in gastro-intestinal surgery. Am J Surg. 1980; 139:160–167.
27. Bistrian BR, Blackburn GL, Scrimshaw NS, et al. Cellular immunity in semi-starved states in hospitalized adults. Am J Clin Nutr. 1975; 28:1148–1155.
28. Polivy J. Psychological reactions to hysterectomy: a critical review. Am J Obstet Gynecol. 1974; 118:417–426.
29. Wilson JF. Behavioral preparation for surgery: benefit or harm. J Behav Med. 1981; 4:79–102.
30. Loder RE. Routine preoperative chest radiography. Anaesthesia. 1978; 33:972–974.
31. Sagel SS, Evans RG, Forrest JV, et al. Efficacy of routine screening and lateral chest radiographs in a hospital-based population. N Engl J Med. 1974; 291:1001–1004.
32. Eisenberg JM, Clarke JR, Sussman SA. Prothrombin and partial thromboplastin times as preoperative screening tests. Arch Surg. 1982; 117:48–51.
33. Ferrer MI. The value of obligatory preoperative electrocardiograms. J Am Med Wom Assoc. 1978; 33:459–469.

Informed Consent to Gynecologic Surgery 2

Benjamin Gilbert and Ben A. Rich

Background of the Doctrine of Informed Consent

Every individual has a well established right to be free from nonconsensual touching. Violation of that right constitutes the common law tort of battery.[1] The application of this principle of tort law to the practice of medicine is certainly not of recent origin. In a 1914 decision, the respected jurist Benjamin Cardozo wrote the following often-quoted language:

> Every human being of adult years and sound mind has a right to determine what shall be done with his own body, and a surgeon who performs an operation without his patient's consent commits an assault for which he is liable in damages.[2]

The evolution of the doctrine of informed consent has been from the intentional tort of battery to the unintentional tort of negligence or medical malpractice.

A procedure performed without the patient's consent constitutes a battery or nonconsensual touching. The physician may be held liable for damages even though the procedure can be shown to have been a medically appropriate one which a reasonable person would have consented to under the circumstances.

In a recent case,[3] the Louisiana Supreme Court held a surgeon liable for performing a total hysterectomy and bilateral salpingo-oophorectomy without the patient's consent. The procedure was performed during a laparotomy to which the patient had given an informed consent in order to treat endometriosis. The written explanation provided by the physician to the patient read as follows:

> Dx (1) Pelvic inflammatory disease marked (2) endometriosis Rec (1) Laparotomy—Lysis of adhesions, Fulguration of endometrioma.[4]

Without mentioning any other procedure to the patient, the physician made the above entry on the hospital admission form but added the language: . . . probable salpingo-oophorectomy . . .[5]

The physician in this case argued unsuccessfully that the patient's prior consent was not necessary when the evidence indicated that the procedure was medically appropriate. The Court held that in the absence of an emergency, the standard of care for physicians requires that the physician obtain the patient's prior informed consent even when, as in this case, to do so would require a second operative procedure.

In another recent case, the New Jersey Supreme Court held that a battery had taken place when a physician other than the one named on the consent form performed the surgical procedure.[6] The patient consented to the removal of kidney stones by one of the physicians in a group practice. Although the patient was not advised of this, it was the practice of the group to deal with all patients as though they were patients of the group, rather

than of individual member physicians. Thus another surgeon from the group actually operated on this patient, and the surgeon whom he expected to perform the procedure was not even present.

The Court, in holding that such conduct constituted medical malpractice, cited extensively from statements of the American Medical Association and the American College of Surgeons that it is unethical to in any way mislead a patient as to the identity of the physician who is undertaking to provide treatment.[7]

The general rule that can be drawn from such cases is that no material divergence from the treatment that has been consented to, whether it is in the nature or extent of the procedure or the physician who will perform it, can be safely undertaken in the absence of an emergency without the prior understanding and approval of the patient. Also, the physician must adhere to any limitations or conditions that the patient has placed upon his treatment.

For example, a patient had consented to a surgical procedure on the express condition that another named physician be present in the operating room. Immediately prior to being placed under general anesthesia, the patient asked and was told that the physician whose presence she had insisted upon was not available. The patient was nevertheless anesthetized, and the procedure performed. The Court held that the patient's consent, being so conditioned, had been effectively revoked, and the surgery following the revocation was a battery.[8]

Conceptual Framework of Informed Consent

Informed consent, for the purpose of analysis, may be separated into two separate responsibilities on the part of the physician: the duty to inform the patient about his condition and the treatment thereof, and the duty to obtain the patient's consent to the proposed treatment.[9] The first aspect is the more problematical and hence accounts for the bulk of informed consent cases. Seven elements of the informational component of consent are generally recognized. These elements are

1. Patient's diagnosis
2. Nature and purpose of the procedure
3. Risks and consequences of the procedure
4. Probability of success of the procedure
5. Feasible alternatives to the procedure with their risks and consequences
6. Prognosis if nothing is done
7. Names of physician or physicians performing procedure[10]

The logical starting point for the discussion of any proposed medical procedure is the patient's present diagnosis. If the proposed procedure is diagnostic in nature, then the physician's disclosure may not, in fact, constitute a diagnosis. However, the physician should provide the patient with the medical basis upon which he is recommending a particular diagnostic procedure. For example, amniocentesis would be recommended to a 37-year-old woman who is early in her first trimester of pregnancy, because the risk of delivering a child with a genetic abnormality is statistically greater than the risk of harm to the mother or the fetus inherent in that diagnostic procedure.

The second element of disclosure, the nature and purpose of the procedure, is in most instances the least troublesome for the physician. Perhaps this is because patients and physicians alike tend to regard the question, "What are you going to do to me and why?" as the heart of informed consent. The manner in which this information is imparted depends primarily on the individual patient. Some patients expect and are able to understand a detailed and sophisticated description of the procedure. Other patients would find such a discussion confusing and excessive.[11] Consequently, the physician must be perceptive and sensitive to the patient's desire for such information and her ability to comprehend. Technical terms should be used sparingly and explained fully to avoid confusion or misunderstanding on the part of the patient.

For example, a patient suffering from bursitis executed a consent form that authorized a "hip replacement on the right side." However, the patient believed the procedure was the same as a "total hip replacement," which was the term used in his discussion with the surgeon. The Court held that the two procedures

were sufficiently dissimilar that the patient had not, in fact, consented to the placement of the prosthesis, and hence the performance of that procedure constituted a battery.[12]

Another instance in which poor communication led to a finding of battery based upon lack of consent involved a patient who presented to an ophthalmologist with a suspected sty. The patient had presented to the associate of the defendant with a similar problem in the past, which had been treated by puncturing the infected area and removing the infected matter. On this occasion, the physician diagnosed the condition as infected meibomian glands on the lower left eyelid and recommended that the marginal chalozion be opened and drained. The patient indicated his approval, and under local anesthesia the physician did this, but also removed some of the meibomian glands and a freckle.

When complications developed, the patient filed suit alleging that the physician had gone beyond the procedure for which he had obtained consent. The physician contended that the patient was awake and alert during the procedure and was impliedly consenting to everything that was done. The Court held that the portion of the procedure beyond that which was initially described to the patient was not consented to, impliedly or otherwise.[13]

The majority of the claims and lawsuits alleging lack of informed consent are based on the third element, that is, a failure to adequately disclose the risks and consequences of the procedure. Although it is generally agreed that a physician does not have a duty to disclose each and every risk of a procedure however slight, courts and legislatures utilize a variety of tests in order to determine those risks which a physician must disclose in order to avoid liability. On one end of the continuum is the professional standard of disclosure. This standard, which was first described in detail in the case of *Nathanson v. Kline,*[14] requires that the patient be told what a reasonable medical practitioner would tell a patient in that situation, at that time and place. The professional standard for disclosure is the rule in the majority of jurisdictions at this time.[15]

At the other end of the continuum, the case of *Canterbury v. Spence*[16] sets forth the material risk standard; under this, the physician must consider the likelihood that the risk will occur and the severity of the harm if it does, in order to determine which risks to disclose in a particular case. Using this standard, courts have held that whenever a procedure involves a risk of death or serious bodily harm, that the physician must disclose that risk to the patient.[17]

The *Canterbury* Court indicates that in order for a physician to accurately assess the materiality of a risk, he must put himself in the position of the patient. Thus, a risk becomes material, and, therefore, one that must be disclosed to a patient before consent is obtained, "when a reasonable person, in what the physician knows or should know to be the patient's position, would be likely to attach significance to the risk or cluster of risks in deciding whether or not to forego the proposed therapy."[18]

The *Canterbury* material risk standard remains very much of a minority view among the states. Therefore, in most jurisdictions, if a physician is disclosing that which his colleagues generally disclose in such situations, he will not be liable in an informed consent case, at least insofar as the substance of the disclosure is concerned.

The fourth element (probability of success) is a potentially serious pitfall for the unwary physician, in that it involves the physician walking a very narrow line between unwarranted optimism and pessimism. Physicians are acutely aware of the potential for frightening a patient away from a procedure by detailing the risks of the procedure or by suggesting a low probability of success. However, much of the litigation based on this element of informed consent involves situations in which physicians either unreasonably minimized the risk of failure, or even went so far as to give the patient the impression that the positive results were guaranteed. In a recent Colorado case,[19] the Court held that the plaintiff had stated a claim of negligent misrepresentation when he alleged that the physician had stated that he had performed ankle replacement surgery before, when, in fact, he had not.

When discussing the likelihood of success of a procedure, the physician should make no representations to the patient that will not withstand the test of strict medical and legal scrutiny. A less-than-satisfactory result, after inflated predictions of success by the physician,

will be more likely to cause the patient to bring suit than otherwise.[20] Based on a review of the case law, it is beyond reasonable argument that a physician is far more likely to be sued because he persuaded a patient to undergo a procedure by inflating the possibility of success than because he dissuaded a patient from undergoing a procedure by realistically discussing the possibility of failure.

Since the physician in most instances is advocating the procedure that he is explaining, a reasonable way of acknowledging the success potential without appearing to offer any guarantees is to shift to a discussion of the risks and consequences of the alternatives.[21] The risks and consequences of any reasonable alternative procedures constitute the fifth element of informed consent. If there are no reasonable alternatives, an accurately stated potential for failure will not be seen as threatening to the patient. Even when there are alternatives, presumably their risks or failure rate is greater than that of the procedure being advocated. However, once again, the physician should not make any representations about these alternative procedures that cannot be supported by the medical literature. For example, if a physician advocates one procedure over another because he has been trained to do one and not the other rather than because one is generally regarded as safer or more often successful than the other, he should disclose this to the patient. In the final analysis, a physician will have discharged his duty with regard to this element of consent when the patient has information sufficient to weigh the merits and demerits of each alternative independently.

The sixth element of consent is the disclosure to the patient by the physician of what the prognosis is reasonably expected to be if neither the procedure nor any of its alternatives is performed. Such information must be deemed relevant, because the patient has a right to forego all treatment modalities. However, in order for the selection of that alternative to be truly informed, the patient must know what consequences may reasonably be expected to flow from that decision. A California case[22] vividly demonstrated the developing judicial attitude toward this aspect of consent. Dr. Thomas, a family physician, urged his patient on several occasions between 1964 and 1969 to undergo a pap smear. She repeatedly declined. In 1969, when referred to a gynecologist, she was diagnosed as suffering from inoperable cervical cancer, from which she subsequently died. Plaintiff's experts testified that a timely pap smear would have revealed the tumor in time to save the patient's life. The court held that the defendant physician had an affirmative duty, on the patient's refusal to undergo the diagnostic procedure, to warn her of the dangers of undetected cervical cancer.[23]

The final element of the informational component of consent is the identity of the physician who will perform the procedure to which consent is being given. As indicated later in this chapter, the physician obtaining the consent should be the one who will perform the procedure. However, this is not always true, particularly in academic medical centers where residents are often assigned the task of obtaining the patient's consent to a procedure that may be performed by a more senior resident or an attending physician.

Whatever the circumstances may be in the particular case, the patient must understand and assent to them in order for the consent to be valid. In the teaching hospital context, the patient should also not be mislead as to the status of the person performing the procedure. The attending on the case may well believe that a senior resident has the requisite knowledge and experience to perform the procedure. If so, then the patient should understand that it is a senior resident rather than an attending who will actually be performing the procedure. A patient who learns after the procedure, particularly one that has not gone well, that a resident rather than an attending was responsible will be more likely to react negatively in the form of a malpractice suit.

Assuring Voluntariness

We shift now from the informational component to the assent component. In order for the patient's authorization of the procedure to be valid, the consent must be voluntary. The physician–patient relationship, at least historically, has been viewed as fiduciary in nature.[24] Such a relationship has been defined as existing where . . .

> there is special confidence reposed in one who in equity and good conscience is bound

> to act in good faith and with due regard to the interests of the one reposing the confidence.[25]

Because of the significant disparity in knowledge between the physician and patient, the physician must be extremely cautious that he does not overstate the merits of the proposed treatment or the demerits of the alternatives, or state opinion as fact to such an extent that the voluntariness of the patient's consent to the recommended procedure can be legitimately questioned.[26] Nor should the patient be given the impression that continued medical care, or the quality thereof, will in any way be compromised if the patient elects not to follow the recommendation of the physician. A very sick patient is in a vulnerable position, and great care must be taken to insure that his decision is not tainted by fear or insecurity.

Competence to Give an Informed Consent

When considering the validity of a consent, the patient's mental status is an important factor. A patient who is competent to give an informed consent is one whose mental faculties are not limited or impaired to such an extent that her ability to understand the relevant information and express her wishes has been materially compromised. Although a detailed discussion of substituted consent for patients who are legally or factually incompetent is beyond the scope of this chapter, it is elementary that a physician must not attempt to explain a complicated procedure to a patient whose ability to comprehend is restricted to any appreciable degree by medication, drowsiness, disorientation, pain, or other physiologic reason.[27] A consent that is obtained after a patient has been administered preanesthetic medication, although not uncommon, is potentially defective, if it can later be shown that the patient's ability to understand was materially compromised.

Verification of Informed Consent

With the exception of experimental procedures,[28] the laws governing informed consent do not require that consent be given in writing, only that informed consent be obtained. Thus, the purpose of consent forms is simply to document for the record that the patient has, in fact, consented to the procedure. Although such prestigious institutions as the Mayo Clinic have eschewed the use of written consent forms, the vast majority of hospitals and physicians have developed the practice of having the patient execute a written consent form before most invasive procedures.[29] Most of the forms currently in use bear a striking similarity to one another, containing boilerplate language such as the following:

> I request that the named physicians perform a _____ (Name of Procedure). I further request the performance of such additional procedures as the physician may consider necessary and advisable as a result of conditions arising in the course of the primary procedure. The nature and purpose of the procedure, the possible alternative methods of treatment, the risks involved, and the possibility of complications have been fully explained to me. No guarantee or assurance has been given by anyone as to the results that may be obtained.

Four complaints have characteristically been lodged against the typical consent form: (1) It contains too much technical legal jargon; (2) it contains too much technical medical jargon; (3) it is too long to be easily read and understood; and (4) it is too general.[30] The boilerplate passage above most clearly exemplifies the fourth complaint, which is also a crucial problem in defending the lack of informed consent aspects of a medical malpractice case. Since the majority of consent cases are informed consent cases, the issue for the trier of fact (judge or jury) is whether the information communicated by the physician was sufficient to meet the applicable standard of disclosure. Most often, the patient will contend that a particular risk or alternative treatment was not mentioned by the physician, while the physician contends that it was. Obviously, the typical consent form with boilerplate language provides no corroboration whatsoever that the physician made the particular declaration at issue. It verifies that some risks and alternatives were discussed, but not which ones.

In order for a consent form to adequately protect a physician, it must accurately memorialize not only that something about risks and alternatives was told to the patient, but what

those risks and alternatives were.[31] When the essential elements of the disclosure that the physician made in obtaining the patient's informed consent appear on the consent form bearing the patient's signature, the physician is in the optimal position to defend a potential claim alleging lack of informed consent. First, if there has been no departure from the relevant standard of care in the treatment of the patient, an attorney will be extremely reluctant to bring an action against a physician solely on the basis of lack of informed consent when the medical record contains a consent form signed by the patient that lists among the items disclosed the very risk that in fact resulted and the alternatives to the procedure. If such a claim is nevertheless instituted, the defense attorney may well be able to obtain a summary judgment in favor of the physician unless the plaintiff can somehow show that the patient's ability to understand was compromised or that there was some misrepresentation of fact made to induce the consent. Finally, even if the case goes to the jury, the consent form will severely test the credibility of the patient who tries to contend that she either did not know or understand what she was signing.[32]

Despite the tremendous difference that this type of consent form can make in a physician's ability to successfully prevent or defend against a claim of lack of informed consent, strong resistance to the use of a detailed form is the characteristic response of physicians. One of the arguments against a consent form that requires detailed information is that it will interfere with the physician–patient relationship at best, and at worst that it will create an adversarial relationship between the physician and the patient. However, a strong argument can be made to the contrary; that skillfully used, the complete disclosure to the patient in an attentive and caring demeanor will, in fact, enhance the quality of the relationship.[33] Since most patients sign some type of consent form anyway, the only difference to the patient is that she will find on the form the information that she has already been given orally, rather than legal boilerplate or medical jargon that more than likely will be unintelligible to her.

A second argument raised by physicians is that the completion of such a form for each patient would consume too much time. If we assume that the time necessary to advise the patient about the procedure and its risks and alternatives and to answer the patient's questions must be taken whether or not a consent form is used, and if we further assume that most hospitals require that some type of consent form be executed by each patient before an invasive procedure is performed, then the only additional increment of time required for the optimum protection afforded by a detailed consent form is that which it takes for the physician to write on the form what he is telling or has just told the patient. It strains credulity to imagine that we are speaking of more than an additional 3–5 minutes per patient, a brief period when compared to the amount of the physician's time that would be consumed in defending even one medical malpractice claim, which may well be avoided or nipped in the bud by the use of a detailed consent form.

Furthermore, many procedures that are performed on a regular basis can be the subject of procedure-specific forms with all of the relevant information already printed on the form, thereby reducing significantly the number of procedures for which detailed information must be written out by the physician.

The third objection to a detailed form commonly raised by physicians is that if some risk is left out on the form, the detailed consent form will make it more difficult, rather than less difficult, for the physician to defend a lawsuit. The response to this argument is that the same care that is exercised in the rendering of "hands-on" medical treatment must also be taken in the preparation of consent forms. This care goes beyond the actual listing of risks on the form and also encompasses the determination of what risks are generally disclosed in that area.

When Informed Consent is Required

Having analyzed the types of disclosures that should be made to patients and the nature of the consent itself, we must address another difficult issue: that is, in what instance is it necessary or appropriate to obtain consent. Clearly, consent must be obtained for all elective surgical and invasive diagnostic procedures. Consent should also be obtained for any diagnostic or treatment procedure that is accompanied by a high degree of risk or a low likelihood of

success, as well as one that may be considered experimental.

A recent decision by the North Carolina Court of Appeals deals with an embolization procedure, which the plaintiff contended was experimental. The Court held that when a health care provider offers an experimental procedure or treatment to a patient, there is a duty to inform the patient of the experimental nature of the proposed procedure and any uncertainty regarding the risks associated with the procedure.[34]

A physician should be familiar with the standard of care in his community with regard to the procedures for which consent is commonly obtained. As noted elsewhere in this chapter, although adherence to the community standard will not guarantee freedom from liability, a physician who does not obtain an informed consent for a procedure when most others in the community do is at an increased risk.

When Informed Consent Is Not Required

There are three generally recognized situations in which an informed consent by the patient is not a prerequisite to medical treatment of an invasive nature. The first is when the conscious and competent adult patient declines the offer of information by the physician and affirms his willingness to undergo the procedure based only on the physician's recommendation. The physician is well advised to have the patient execute a waiver-of-informed-consent form based on the same rationale as the execution of a consent form; that is, to document the nature of the interaction. The physician should also note in the patient's chart that the information was offered to the patient prior to the granting of consent and refused.

The second situation in which informed consent is not required is in a genuine medical emergency.[35] Such an emergency is usually defined as one in which immediate medical treatment is required to preserve life or prevent a serious impairment to health, and neither the patient nor someone authorized to grant consent on his behalf is available. Such consent is said to be implied by law.[36]

A different situation entirely is presented when a patient is on a gradual downhill course and has steadfastly refused treatment intended to reverse that decline. A physician may not, at the point at which the patient's condition has become critical, declare that an emergency now exists and proceed to treat the patient over the previously stated objection. Hence, the emergency doctrine only applies when the patient's position with regard to treatment is unknown. Once the physician knows that the patient rejects treatment for some condition, he must honor that declaration unless and until it is clearly revoked. Similarly, a patient's next-of-kin cannot override the patient's position on consent.

The third and most perilous circumstance is the one in which the physician seeks to withhold the information necessary for an informed consent on the ground that to inform the patient would further compromise his medical condition. Although formally recognized by courts[37] and referred to as the therapeutic privilege to withhold information, the circumstances under which it has been approved are strictly limited. The following language from the *Canterbury* opinion states the rationale for this limitation:

> The privilege does not accept the paternalistic notion that the physician may remain silent simply because divulgence might prompt the patient to forego therapy the physician feels the patient really needs. That attitude presumes instability or perversity for even the normal patient and runs counter to the foundation principle that the patient should and ordinarily can make the choice for himself. Nor does the privilege contemplate operation save where the patient's reaction to risk information, as reasonably foreseen by the physician, is menacing. (Footnotes omitted.)[38]

No physician should walk out onto the limb of therapeutic privilege alone, but only with a second medical opinion and a well documented medical record that supports the basis upon which he has invoked that privilege. Whenever possible, persons who know the patient well should be on record in support of the exercise of the privilege.[39]

Placing the Duty of Disclosure

The ultimate responsibility for obtaining the patient's informed consent falls upon the physician who will actually perform or supervise

the performance of the procedure being consented to.[40] In a situation in which a patient is admitted to a hospital for a diagnostic workup and possible surgery, the responsibility for obtaining consent to the various procedures cannot be shifted to the admitting physician. The radiologist, anesthesiologist, and operating surgeon must each undertake to explain to the patient their role in her treatment and obtain her consent to it. Although they may, in fact, delegate that task to a resident or other physician, the legal responsibility remains theirs throughout.

Until recently, the consensus opinion was that hospitals were not liable for procedures performed on their premises by independent physicians.[41] However, a recent Illinois decision held that it is a factual issue for the jury whether a hospital's duty to a patient includes requiring and ascertaining that physicians with staff privileges in fact obtain an informed consent before undertaking to treat a patient.[42] On the basis of this case, at least one commentator has suggested that a hospital may no longer be able to avoid liability by simply verifying that a properly executed consent form is in the medical record prior to an invasive procedure. Rather, it may now be necessary, under the reasoning in *Magana,* for the hospital to determine that the consent that was executed was truly informed.[43] Unless the hospital is in a jurisdiction such as Ohio, which has a statute that provides that a hospital cannot be held liable for a physician's failure to obtain an informed consent from his patient prior to a procedure unless the physician is a hospital employee,[44] it is advisable to monitor very carefully the manner in which consents are obtained for procedures conducted on hospital patients.

Case Discussions Pertaining to Informed Consent to Gynecologic Surgery

Court cases applying these general principles to the field of gynecologic surgery have focused primarily on three elements of informed consent: (1) whether the patient is legally capable of consenting to treatment; (2) whether risks and alternatives have been adequately disclosed; and (3) whether the physician may extend surgery and perform a different procedure than previously consented to by the patient. Discussing these three elements of informed consent by reference to a single area of surgery—sterilization procedures—will allow for a more comprehensive analysis of the factors considered in determining potential liability. The following case discussions are intended to illustrate some of the more frequently litigated aspects of consent for gynecologic surgery. The interplay of legal principles and factual variations in these cases, although specific to a single area of gynecologic surgery, should be instructive in developing an understanding of general principles that may be applied to other factual settings.

Capacity to Consent

The determination of a person's legal capacity to consent to medical or surgical treatment strikes at the heart of the law of consent. As previously quoted, "Every human being of adult years and sound mind has a right to determine what shall be done with his own body."[45] Questions of capacity to consent to sterilization procedures most often arise in relation to whether a woman's marital status, age, or mental capacity should influence her general right of self-determination. Two cases illustrate that even though the decision to end a woman's reproductive capacity through surgery may have a significant impact on her husband and the marital relationship, it is a decision that may be made by a woman herself.

An Oklahoma case[46] addressed the question of whether a husband can recover from a physician for damage to a marital relationship resulting from sterilization of the wife, consented to by her. In that case, there was no allegation that the operation was unsuccessful or that the physician was negligent. The husband alleged, instead, that the physician had interfered with the marital rights of the husband to "reproduce another child," and sought damages for this injury to him and to the marital relationship.

The court noted that there was no allegation in the legal proceedings that the wife was of diminished capacity or otherwise incapable of consent. The court approved the general rule that a married woman in full possession of her

faculties has the power, without the consent of her husband, to submit to a surgical operation on herself, including a sterilization procedure. The court specifically found that "the right of a person who is capable of competent consent to control his own body is paramount."[47] Furthermore, in relation to the husband's claimed damage to his "right to reproduce another child," the court refused to acknowledge a right in a husband to have a fertile wife and refused to allow recovery for damage to such a right.

A New Jersey case[48] based a woman's right to consent to a sterilization procedure, without spousal consent, on Constitutional guarantees of liberty and the right to privacy. The woman in the case bore three children during the period she cohabited with her husband. She began living apart from her husband and became pregnant by another man. The woman decided unilaterally that she no longer desired to utilize her capacity to procreate and wished to effectuate this end by means of sterilization. She planned to have her obstetrician-gynecologist perform the sterilization within 1 day after the delivery of her expected child. However, her physician would not perform such an operation unless she obtained the consent of her estranged husband, which she was unable to secure.

The court looked to *Roe* v. *Wade*[49] and related decisions for guidance in determining that a married woman has a Constitutional right to be sterilized without spousal consent. Those decisions sanctioned the ideology that a woman has a right in most circumstances to control her reproductive functions, and struck down restrictions on that right based on nonmedical criteria. The New Jersey court recognized "the sensible, logical and well-reasoned desirability of consultations between husband and wife regarding decisions in such matters. However, this is not to say that the spouse does or should have a power to veto."[50] The court held that the woman enjoyed a Constitutional right to obtain a sterilization operation without the consent of her husband.

This Constitutional right, which is enjoyed by a competent married woman, is also possessed by minors and incompetent women. There is no consistent legal guidance, however, in determining who shall decide for those who, either because of age or mental disability, are not permitted to or cannot give effective consent for themselves. Historically, parents and guardians have been afforded broad discretion over their children and disabled wards in the area of medical care and treatment, recognizing the parents' natural inclinations to act in the best interest of their children and wards. Due to the potential conflict of interest, and the nature of the rights at stake, consent that is given by a parent or guardian can be subject to either administrative or judicial review. To ensure that sterilization of minors or mentally disabled persons is not imposed solely for the convenience of parents, guardians, or institutional supervisors, such decisions must be closely reviewed. In some states,[51] statutes permit parents, guardians, or institutional supervisors to exercise "substituted consent" to authorize sterilizations. A number of other state statutes[52] require judicial review and require a court to make the ultimate determination of whether sterilization is warranted.

The remaining states that have not statutorily addressed sterilization of minors or mentally disabled persons are split among those that recognize the judiciary's authority to order sterilization in the absence of legislation[53] and those that do not recognize the judiciary's authority.[54] Recent cases in New Jersey[55] and Washington[56] establish guidelines, in the absence of legislation, for weighing the individual's rights of privacy and procreation against those competing interests favoring sterilization. These guidelines incorporate elements of procedural due process and involve an evaluation of risks, benefits, and alternatives to the requested sterilization.

Physicians faced with good-faith requests from parents or guardians of minors or mentally disabled persons should seek guidance on the status of the law in that jurisdiction. Physicians would be well advised to avoid performing sterilizations on minors or mentally disabled persons without statutory or judicial approval.

Disclosure of Risks and Alternatives

By far the most frequently litigated issues related to sterilization procedures have involved allegations of inadequate disclosure of the risks and alternatives of a surgical procedure. Spe-

cifically, there is a sizable body of case law involving the occurrence of fistulae following hysterectomy. Different legal standards are applied in determining whether a physician can be held liable for the occurrence of a fistula following hysterectomy.

One line of cases evaluated the adequacy of risk disclosure in terms of the materiality of such information to the patient's decision. The courts that utilize a materiality test usually find that the failure to disclose the risk of fistula formation is not a material deficiency. For example, in a Pennsylvania case[57] a woman sued her physician on the theory that a hysterectomy was performed without her informed consent. Following five miscarriages and the cesarean delivery of her fourth child, the patient underwent a tubal ligation for the purpose of sterilization. Some 7 months later she consulted another physician complaining of recurrent vaginal bleeding since the delivery of her fourth child. Following an evaluation of the patient, the physician recommended and performed a hysterectomy, removing her uterus, left fallopian tube, and left ovary. The patient remained in the hospital for over 2 weeks, during which time she developed abdominal distension, jaundice, fever, anemia attributable to hemorrhage into her abdominal cavity, and a vesicovaginal fistula. The patient consulted another physician, and the fistula was successfully repaired by surgery 6 months later.

The patient brought suit against the physician who performed the hysterectomy, alleging that his failure to inform her of the risk of vesicovaginal fistula rendered her consent ineffective. The jury determined that even though the physician had not informed the patient of the risk, a reasonable patient, aware of such a risk, would have proceeded with the operation nonetheless. On appeal, this verdict was upheld applying the "reasonable man" standard. According to this standard, the extent of risk disclosure will be evaluated against what a "reasonable man" in the situation which the physician knew or should have known to be that of the patient would deem significant in making a decision to undergo the recommended treatment. The court stated that this standard gives maximum effect to the patient's right to be arbiter of the medical treatment she will undergo without requiring the physician either to be a mind reader into the patient's most subjective thoughts or to disclose every risk. The physician is bound to disclose only those risks which a reasonable person would consider material to the decision whether or not to undergo treatment.

In another case[58] applying the "reasonable man" standard to the failure to disclose the risk of fistula formation, a Louisiana court denied recovery for a patient because the physician's failure to disclose a risk was not determined to be causally connected to the patient's injury. The patient in this case first consulted her physician complaining of abdominal pain and giving a history of chronic pelvic discomfort. The physician made an initial diagnosis of pelvic inflammatory disease, recommended a hysterectomy, and began antibiotic therapy. Over the next 5 years, including 15 office visits, the physician continued to recommend hysterectomy. The patient eventually became severely ill at work and was admitted to the hospital for a hysterectomy. On admission, the patient signed two separate consent forms, neither of which gave any details of the surgery or related risks.

The operation was completed without incident after finding severe pelvic inflammatory disease. On the tenth postoperative day the patient was not able to control her urine flow, and this problem persisted after her discharge from the hospital. Her physician became concerned that she might have developed a vesicovaginal fistula, and he made an appointment for her to see a urologist. The urologist confirmed the diagnosis and successfully performed surgery to correct the problem.

At trial, the patient argued that the physician failed to secure her informed consent to the operation in that he failed to inform her of the loss of function of an organ as required by Louisiana statute. The uncontroverted testimony at trial was that although the patient knew of the more common risks of hysterectomy surgery from previous discussions, the physician did not warn the patient of the possibility of a vesicovaginal fistula forming, or that such a condition would require correction by subsequent surgery, or that there might be a waiting period after initial surgery before operating again.

The court found that the physician had not obtained the patient's consent in accordance with Louisiana law, since there was an undisclosed risk of loss of function of an organ. However, the court noted that in order for there to be a finding of liability, not only must the patient show that the undisclosed risk actually occurred, but also that if the risk had been disclosed, the treatment and unwanted consequences would have been avoided. In short, the patient must prove that the failure to inform caused the damaging consequences. If the patient would have undertaken the treatment in any event, in spite of the deficient "informed consent" there would be no recovery. The court adopted an objective test for determining causation—whether a reasonable person in the patient's position would have consented to the operation if full disclosure had been made. Expert testimony at trial indicated that the rate of occurrence of this risk is less than 1%. The court held that such a low rate of that complication occurring, where the complication could be subsequently corrected, should not be a determinate factor to a reasonable person in the patient's position. Because the patient failed to prove causation, the physician was relieved of liability for his nondisclosure.

The alternate standard for evaluating the adequacy of risk disclosure is the professional standard, that is, what a reasonable physician would disclose under the same or similar circumstances. How the physician may best discharge this obligation to the patient involves primarily a question of medical judgment. While the legal analysis is very different from the reasonable man standard, the failure to disclose the risk of fistula formation after hysterectomy has generally not led to liability for physicians. This perspective is illustrated in a Colorado case[59] in which a woman sought a hysterectomy to correct long-standing and otherwise uncorrected difficulty with cramps and increased menstrual periods.

The patient signed a consent form which stated that she had been fully advised of the advantages of a hysterectomy, the complications of the operation, and alternate modes of treatment. The undisputed testimony at trial was that the physician did not inform the patient of the danger of fistula development or kidney loss, and did not inform her of other methods of treatment by which the hysterectomy could have been avoided. After the hysterectomy was performed, the patient developed a ureterovaginal fistula accompanied by several other complications requiring additional surgery and eventual loss of a kidney.

The patient contended that if her consent was obtained without her being informed of the risks inherent in the operation, the doctor should have been liable for the unfortunate result even if the manner in which he performed the hysterectomy was not negligent. The Colorado court, applying the professional standard test, first stated that it is not mandatory for a physician to make full and complete disclosure in all cases and under all circumstances. Once evidence has been presented indicating that the patient was not informed when she gave her consent due to a failure of disclosure, the physician must present evidence establishing that his failure to disclose conformed with community medical standards of care. Since the extent of disclosure is a medical judgment, a showing that nondisclosure complies with the community standard is a valid defense. The appeals court ordered a new trial to determine, by expert testimony, whether the failure to disclose the risk of a ureterovaginal fistula complied with what other physicians in the community disclosed under similar circumstances.

Although no precise formula can be stated as a guide for the necessity of disclosing any particular risk, the incidence of the risk and its severity when it occurs are important factors. Regardless of whether a court is applying the reasonable person or the reasonable physician, if the risk is statistically high or extremely severe the patient should be informed prior to surgery. If the statistical risk is minimal and transitory, the physician will probably not be required to disclose the risk.

The same theories applicable to risk disclosure are employed in determining liability for failure to inform the patient of medically feasible alternatives to a procedure. Reasonable person and reasonable physician standards apply to this informational aspect of consent as well. Allegations of undisclosed alternatives most often arise following a medical procedure which, even in the absence of negligent technique, is seen as unnecessary, unsuccessful, or

too drastic. The patient must assert that an undisclosed alternative, if known, would have been selected, and, that the undesired condition resulting from the actual procedure would not have occurred. For example, in a Kentucky case[60] a woman expressed her desire not to have any more children when pregnant for the eighth time. Her physician advised against a hysterectomy immediately after the delivery of a child because of the enlargement of the uterus and blood vessels and the danger of hemorrhage and infection. He advised that she have a tubal ligation instead. Although a subsequent hysterosalpingogram revealed that both tubes were blocked following the ligation, she became pregnant for the ninth time.

The woman alleged that she was not informed that the tubal ligation was not absolutely foolproof in preventing pregnancy, and argued that the physician knew of her eight children and that the physician should have suggested a hysterectomy as better suited for sterilization. The physician, on the other hand, testified that he had talked about the possibility of failure with a tubal ligation. The consent form that the woman signed included the following statement: "The risks involved and the possibility of complications have been explained to me. Even though good results are expected I acknowledge that no guarantee or assurance has been given to me as to the results that may be obtained." The trial court found the physician not liable for the woman's ninth pregnancy and related costs. On appeal, the court affirmed this finding, stating that it was inconceivable that a woman who knew of her propensity to become pregnant would not have discussed with her doctor the chances of success or failure of the tubal ligation procedure. The court stated that the physician performed an operation with a very low failure rate, which he thought was best for the patient's health following delivery of her eighth child and which he thought would be effective. In essence, the trial court did not believe that the woman was unaware of the alternatives to tubal ligation.

Another case[61] involving a failed tubal ligation reached a different result, possibly due to the different factual setting. A woman who experienced eclampsia with her first pregnancy sought to be sterilized at the time of her expected third cesarean delivery. The woman stated at trial that she informed her physician that she specifically did not want more children and could not afford any more. She also claimed that her physician warned her that a future pregnancy would imperil her health. Although the physician denied that he so warned the woman, he did sign a consultant's report prior to the operation stating that "future pregnancies would endanger her life. Sterilization is recommended for the following (sic) reason." A consent form, which stated that the operation was not effective in all cases, was given little evidentiary weight because the patient signed without reading it 10 min before being taken to the delivery room for the cesarean delivery and tubal ligation.

The trial court decided, without submitting the case to the jury, that the physician was not liable for the ensuing pregnancy subsequent to the tubal ligation. The appeals court reversed that decision and returned the matter to the trial court for a new trial, because the following evidence, in particular, should have been submitted to the jury for a determination of whether the patient was adequately informed of the alternatives to tubal ligation. First, there was evidence that the physician never directly disclosed to the patient that the operation might not be 100 percent effective regardless of what tubal ligation method he employed, nor did he disclose that other surgical methods would have been significantly more effective. Second, evidence was also produced to the effect that the patient elected to undergo sterilization because she was concerned about the possibility of damage to her physical well-being and the financial burden of raising another child. Third, it was undisputed at trial that the physician chose not to inform the patient about the increased risk of failure inherent in sterilizations performed at the time of cesarean delivery. She was therefore denied the opportunity of deciding whether to undergo sterilization at delivery or at a later time when the risk of failure would have been of material significance to a woman desirous of permanently preventing childbirth in the most effective manner. Finally, there was evidence permitting the jury to find that the physician did not discuss the possibility of vasectomy with either the patient or her husband, even

though, as the physician himself acknowledged, it was customary for physicians to discuss this subject when consulted by patients about sterilization.

Applying the reasonable person test to this evidence, the appeals court held that a jury could have found that a reasonable person in the patient's condition would have attached considerable significance to the projected risk of failure for the tubal ligation, and therefore should have been informed of the risks of fertility associated with the alternate methods or procedures for sterilization.

Exceeding the Scope of Consent

A third group of cases have addressed the question of whether a physician may extend surgery and perform an additional or different procedure than previously consented to by the patient. A general statement of the relevant law is that any material divergence from the treatment that has been consented to constitutes a battery upon the patient. A physician is justified in extending an operation and remedying a condition without the express consent of the patient when confronted with an emergency or an unanticipated condition and when immediate action is necessary for the preservation of the life or health of the patient and it is impractical to obtain consent of the operation. Two recent cases illustrate the application of this point.

The first case[62] involved a pregnant woman whose physician advised that her baby be delivered by cesarean section because of the baby's size and position. The woman agreed to the cesarean section and also requested that a tubal ligation be performed during the delivery for the purpose of rendering her completely sterile. When the patient was admitted to the hospital, she signed a consent form authorizing the medical partnership to perform the cesarean section and tubal ligation and also authorizing "such additional operations or procedures as are considered therapeutically necessary on the basis of findings during the course of said operation." The patient, a registered nurse familiar with these forms, testified in her deposition that she read and understood this language.

The next day, the physicians delivered a healthy, normal child through a vertical incision of the uterus. The placenta was removed after the delivery. The patient began bleeding from the uterine incision. To minimize blood loss the doctors quickly sutured the incision. Because of the patient's older age, 36 years, the large size of the child, and the presence of numerous fibroid tumors throughout the uterus, however, the patient's uterus did not contract well and she was losing more than a normal amount of blood. At this point in the operation her condition was reasonably stable, and the doctors began full abdominal-pelvic exploration and discovered a grapefruit-sized tumor arising from her right ovary, adherent to the uterus. Without laboratory study they could not determine whether the growth was benign or malignant. The doctors, without attempting to contact her husband, removed the tumor and sent it to the laboratory for histologic study, where subsequent tests revealed that the growth was benign.

Unfortunately for the patient, at the time of the removal of the tumor, blood vessels on the uterine surface were severed, resulting in more blood loss. The combined blood loss from removal of the tumor, from the cesarean incision, and from the placental site was too large. In their attempts to control the bleeding, the physicians utilized two methods; suturing and packing the surgical sites, and intramuscular Pitocin. Both methods were unsuccessful and her condition was deteriorating to the point where the doctors feared that this continuous bleeding might endanger her life. After a quick consultation the physicians removed her uterus in order to halt the hemorrhage. The husband learned of his wife's condition from one of the physicians prior to the removal of the uterus; however, he was told that a hysterectomy was necessary and he was not asked for his consent.

The net result was that the patient was treated with total hysterectomy and removal of her right ovary and fallopian tube instead of her requested tubal ligation. Having very strong feelings against the removal of her organs, as opposed merely to being rendered sterile, the woman, joined by her husband, brought a lawsuit against the physicians seeking damages for negligence, assault, and battery.

The trial court found that there was no issue

of negligence in the performance of the surgery and thus treated the claim as one of battery with the defense of consent. Finding no factual dispute, the trial court found the consent valid. The patient appealed and the court framed the issue on appeal as whether the physicians' actions were therapeutically necessary in the absence of an informed consent.

The Georgia standard for determining whether a procedure was "therapeutically necessary" is whether the doctor exercised that degree of care, skill, and diligence that any other surgeon in the community would be required to employ in reaching a decision under the same or similar circumstances, the reasonable physician standard. Examining the evidence presented at trial, the appeals court stated that it was undisputed that the patient was bleeding heavily and the doctors were unsuccessful in their initial efforts to halt it by using sutures and oxytocin. Once these measures failed to halt the bleeding, a hysterectomy was the next indicated step. The patient's own expert physician witness testified that the physicians attending the patient complied with the standard of care in determining that the hysterectomy was therapeutically necessary. The appeals court found that although the physicians removed the patient's uterus, right ovary, and fallopian tube without her consent, such action was therapeutically necessary, and therefore no liability would be imposed against the physician.

A Maryland court[63] analyzing a factual situation involving an unexpected hysterectomy during ovarian cyst surgery upheld a jury's finding of physician liability based on lack of informed consent because there was no emergency need for the procedure, because the material risks and complications of the initial surgery were not explained, and because a reasonable person would not have consented to the initial surgery had she known of the complications. The facts of the case, which are largely determinative of the result, are as follows. A 25-year-old unmarried woman had an abortion performed by dilation and curettage. Ten days later she complained of abdominal pains and cramps, which her physician believed were caused by a cyst on her left ovary. During surgery to remove the cyst, the physician observed that her left fallopian tube had ruptured as a result of a tubal pregnancy and her left ovary had a large cyst. He removed both the left tube and the left ovary. In his opinion, the condition of the right tube and the right ovary was such that the patient would never be able to conceive a child, and he decided it would be good surgical practice to remove them at that time. He thereupon removed the right tube, the right ovary, and the uterus. The woman filed suit against the physician claiming damages for injuries resulting from negligence in performing the operation and for failure to obtain her informed consent to the removal of those reproductive organs. The jury returned a verdict against the physician.

At trial, the woman testified that she never authorized the physician to remove her reproductive organs. She also testified that she signed some forms before the operation, but they "didn't have anything on it . . . I was in so much pain I would have probably signed anything." The physician testified that he discussed the necessity of the surgery and the possible need to remove all her reproductive organs. Expert witnessess for both sides testified about the pathologic condition of the right fallopian tube, right ovary, and uterus, and whether the physician acted in accordance with accepted medical standards in removing those organs. Experts for the patient testified that the right fallopian tube, right ovary, and uterus were normal on the basis of their review of the pathology results. Had these organs not been removed, the patient would have been able to bear more children. Furthermore, they testified that the loss of estrogen and associated use of Premarin exposed the patient to other risks. One of the surgeon's experts was the hospital pathologist who examined the tissues after the hysterectomy. In his original report, he found that the patient's right tube and right ovary were normal. After suit was filed, the pathologist was asked by the surgeon to review the pathology findings. The pathologist testified that his original report was in error, his subsequent findings indicated evidence of chronic inflammation of the right tube and right ovary.

The jury was instructed to return a verdict for the patient if there was no emergency need to remove the patient's right ovary, right tube, and uterus; if the physician did not inform the

patient of the material risks and complications associated with such surgery; and if a reasonable person would not have permitted removal of their reproductive organs had she known of those consequences. The jury was also instructed that if the patient indicated or gave consent to the physician to exercise his discretion as to what was necessary to remove, then the jury must find in favor of the physician with regard to the question of consent. The jury's verdict of liability against the physician, which was upheld on appeal, resulted from findings that there was no emergency need to remove the patient's right ovary, right tube, and uterus. In the absence of an emergency, removing those organs exceeded the scope of the patient's consent for surgery related to the tubal pregnancy in her left tube.

References

1. Prosser WL. Law of Torts, 4th ed. St. Paul. West Pub Co, 1971: p 34.
2. Schloendorff v. Society of New York Hospitals, 211 N.Y. 125, 129–30, 105 N.E. 92, 93 (1914).
3. Pizzalotto v. Wilson, 437 So. 2d 859 (La. 1983).
4. Id. at 862.
5. Id.
6. Perna v. Pirozzi, 92 N.J. 446, 457 A. 2d 431 (1983).
7. Judicial Council of the American Medical Ass'n., Op. 8.12 (1982); American College of Surgeons, "Statements on Principles." §IA. (June 1981).
8. Pugsley v. Privette, 220 Va. 892, 263 S.E. 2d 69 (1980).
9. Hiller MD. Medical Ethics and Law. Cambridge, Ballinger Pub. Co., 1981: p 198.
10. Rosoff A. Informed Consent. Rockville, Md, Aspen Systems Corp, 1981: p 318.
11. Presidential Commission. Making Health Care Decisions, Volume 1, pages 81–83 (1982).
12. Cathemer v. Hunter, 27 Ariz.App. 780, 558 P.2d 975 (1976).
13. Shulman v. Lerner, 2 Mich.App. 705, 141 N.W. 2d 348 (1966).
14. 186 Kan. 393, 350 P.2d 1093, rehearing denied, 187 Kan. 186, 354 P.2d 670 (1960).
15. Miller, L.J. Informed consent. JAMA. 1980; 244:2100–2103.
16. 464 F. 2d 772 (D.C. Cir. 1972), cert. denied, 409 U.S. 1064 (1972).
17. Cobbs v. Grant, 104 Cal.Rptr. 505, 502 P.2d 1 (Cal. 1972).
18. Canterbury, supra at 787.
19. Bloskas v. Murray, 646 P. 2d 907 (Colo. 1982).
20. Hold v. Nelson, 11 Wash.App. 230, 523 P.2d 211 (1974).
21. D. Louisell and H. Williams, Medical Malpractice, Vol. 1, §5.04 (1984).
22. Truman v. Thomas, 27 Cal. 3d 285, 611 P.2d 902, 165 Cal. Rptr. 308 (1980).
23. 165 Cal. Rptr. 308 at 311 (1980). See also: Keogan v. Holy Family Hospital, 95 Wash. 2d 306, 622 P.2d 1236 (1980).
24. Neagle v. McMullen, 334 Ill. 168, 165 N.W. 605 (Ill. 1929).
25. Black HD. Black's Law Dictionary, 5th ed. St. Paul, West Pub. Co., 1979: p. 564.
26. Preston T, The Clay Pedestal: A Re-examination of the Doctor-Patient Relationship. Seattle, Madrona Pub., Inc., 1981: p. 74–75.
27. For a discussion of competency to consent, see generally: Rosoff, supra note 9 at 233–243.
28. 21 C.F.R. §50.25(a)(1) (1984).
29. Moore RM Jr. Consent Forms—how, or whether, they should be used. Mayo Clin Proc 1978; 53:393–396.
30. Id. at 394.
31. Annas G, Glantz L, Katz B. The Rights of Doctors, Nurses and Allied Health Personnel. Cambridge, Ballinger Pub. Co., 1981: p. 75. Kaufer D, Steinberf E, Toney S. Revising Medical Consent Forms: An Empirical Model and Test. Law, Medicine, and Health Care. 1983; 11: p. 155–162.
32. The following statutes provide that a consent form that meets certain requirements, and which is signed by the patient, creates a presumption that the patient's informed consent was obtained: Fla. Stat. Ann. §768.46(a), Idaho Code §39-4305, Iowa Code Ann. §147.137, LA. Rev. Stat. §41A.110, Ohio Rev. Code §2713.54, N.C. Gen. Stat. §90=21.13(b), Tex. Rev. Civ. Stat. Art. §4590, Utah Code Ann. §78-14-5(e).
33. Southwick AF. The Law of Hospital and Health Care Administration. Ann Arbor, Health Admin. Press, 1978: p. 209.
34. Estrada v. Jacques, et al., 70 N.C.App. 627, 321 S.E. 2d 240, 254 (1984).
35. Canterbury, supra at 788–789.
36. Southwick, supra note 30.
37. Canterbury, supra at 789.
38. Id.
39. Rosoff, supra note 8 at 55.
40. Nelson v. Patrick, 58 N.C.App. 546, 293 S.E.2d 829 (1982).
41. Barfield v. South Highland Infirmary, 191 Ala. 553, 68 So. 30 (1915); Schloendorff, supra, note 2.
42. Magana v. Elie, 108 Ill. App.3d 1028, 439 N.E.2d 1319 (1982).

43. Greenlaw J, Should Hospitals Be Responsible for Informed Consent. Law, Medicine and Health Care. 1983; 11:173–176.
44. Ohio Rev. Code Ann. §2317.54 (1979 Supp.).
45. Schloendorf, supra note 2.
46. Murray v. Vandevander 522 P.2d 302 (Okla.App. 1974).
47. Id. at 304.
48. Ponter v. Ponter, 135 N.J.Super. 50, 342 A.2d 574 (1975).
49. 410 U.S. 113 (1973).
50. Ponter, supra note 48, at 577.
51. See, e.g., Idaho Code §39-3903 (1977); Utah Code Ann. §64-10-1 (1983) Supp.).
52. See, e.g., Conn. Gen. Stat. Ann. §§45-78q et seq. (1981); N.C. Gen. Stat. §35-36 (1984).
53. See infra, notes 55 and 56.
54. See, e.g., In the Matter of D.D., 64 A.D.2d 898, 408 N.Y.S.2d 104 (1978); In re MKR, 515 S.W.2d 407 (Mo. 1974).
55. In re Grady, 85 N.J. 235, 426 A.2d 467 (1981).
56. In re Hayes, 93 Wash.2d 228, 698 P.2d 635 (1980).
57. Bowers v. Garfield, 382 F. Supp. 503 (E.D.Pa. 1974).
58. LaCaze v. Collier, 434 So.2d 1039 (La. 1983).
59. Stauffer v. Karabin, 30 Colo.App. 357 492 P.2d 862 (1977).
60. Bennett v. Graves, 557 S.W.2d 893 (Ky.App. 1977).
61. Sard v. Hardy, 281 Md. 432, 379 A.2d 1014 (Md.App. 1977).
62. Davidson v. Shirley, 616 F.2d 224 (5th Cir. 1980).
63. Thimatariga v. Chambers, 46 Md.App. 260, 416 A.2d 1326 (1980).

Surgical Incisions and Their Anatomic Basis 3

Wayne A. Christopherson*

In selecting the abdominal incision for an operative procedure, the gynecologic surgeon must be cognizant of the requirements that will be placed on that incision. The incision must provide ready access and adequate exposure for the anticipated procedure as well as allow for extension, if necessary, for the performance of an unanticipated procedure. Ideally, the surgical incision should minimize the sacrifice of nerves, particularly those supplying the abdominal musculature. Choice of an abdominal incision must be based on sound, anatomic principles to ensure a satisfactory, secure closure.

Other factors, such as the patient's condition or the need for quick entrance to the abdominal cavity, may influence the choice of incision. The patient's body habitus, or previous surgical or medical history, may also make one type of incision preferable over another.

Anatomy of the Abdominal Wall

A working knowledge of the anatomy of the anterior abdominal wall is necessary before performing any abdominal incision. The abdominal wall is composed of skin, subcutaneous tissue, adipose tissue, muscle, lymphatics, fascia, blood vessels, and nerves.

Muscles and Fascia of the Anterior Abdominal Wall

The *external oblique* muscle (Figure 3-1A) forms the anteriolateral boundary of the abdominal wall. It originates from the lower eight ribs and courses obliquely downward and medially. The lower, posterior portion of the muscle inserts directly on the external lip of the iliac crest. The remainder of the muscle forms a broad aponeurosis, the medial portion of which passes over the rectus abdominis muscle contributing to the anterior rectus sheath. The inferior portion of the aponeurosis of the external oblique is attached to the anterior superior iliac spine and the pubic crest. Between these two bony prominences, the aponeurosis is turned back upon itself to form the J-shaped inguinal ligament. The external abdominal ring is located superior and medial to the pubic tubercle, where the aponeurosis of the external oblique is evaginated by the round ligament of the uterus. Fibers of the aponeurosis continue onto the round ligament as the external spermatic fascia.

The *internal oblique* muscle (Figure 3-1B) arises from the thoracolumbar fascia, intermediate line of the iliac crest, and inguinal ligament. The fibers arising from the thoracolumbar fascia course directly cephalad, inserting into the lower three ribs. The midportion of the internal oblique, which arises from the intermediate line, courses cephalad and medially forming an aponeurosis that subsequently splits into two lamellae which ensheath the rectus muscle. The lamellae then rejoin, and with the aponeuroses of the external oblique and transversus abdominis form the linea alba. The lower portion of the internal oblique, which

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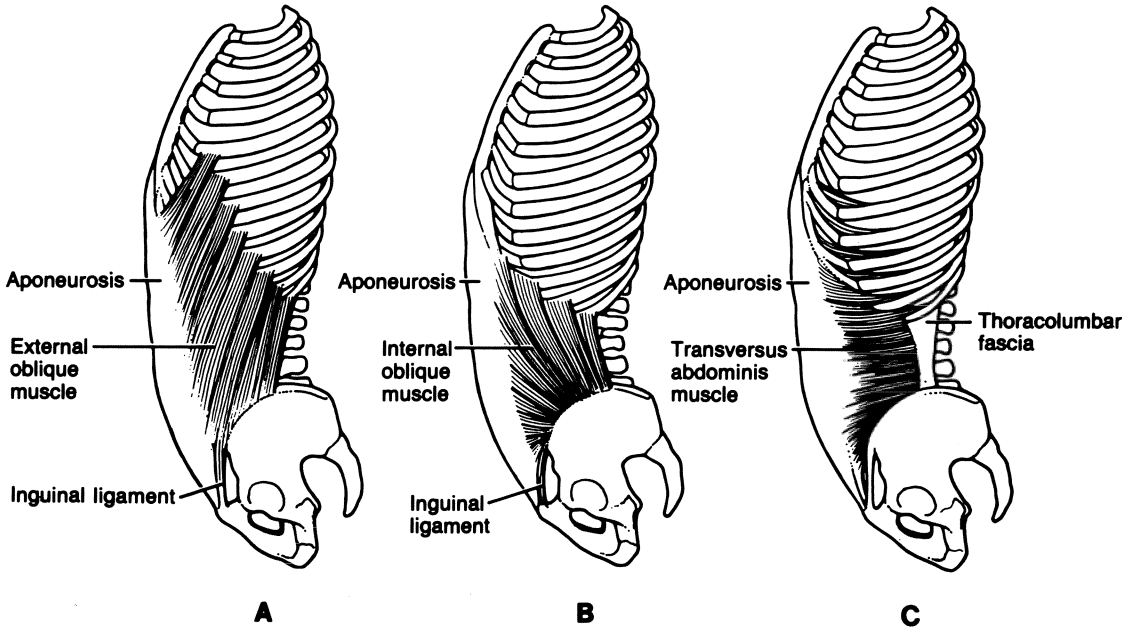


Figure 3-1. The muscular anteriolateral abdominal wall is composed of the external oblique muscle and aponeurosis **(A)**, internal oblique muscle and aponeurosis **(B)**, and the transversus abdominis muscle and aponeurosis **(C)**.

arises from the inguinal ligament, courses medially and inferomedially, over the round ligament. The aponeurosis of this portion of the muscle then continues medially, passing anterior to the rectus abdominis muscle. Medial to the rectus muscle, the aponeurosis of the internal oblique fuses with the aponeuroses of the external oblique and transversus abdominis to form the linea alba.

The *transversus abdominis* (Figure 3-1C) arises from the lower six costal cartilages, thoracolumbar fascia, internal lip of the iliac crest, and the inguinal ligament. The muscle fibers course transversally across the abdominal wall, and become aponeurotic at the lateral border of the rectus muscle. From the xiphoid process to a point approximately midway between the umbilicus and the pubis, the aponeurosis passes behind the rectus muscle. However, below that point the aponeurosis passes in front of the rectus abdominis. The crescentic border that is formed by the lower limit of the aponeurosis lying behind the rectus abdominis is the arcuate line. Medial to the rectus, the aponeurosis of the transversus abdominis combines with the aponeuroses of the internal oblique and external oblique to form the linea alba.

The *transversalis fascia* is the fascia on the inner surface of the transversus abdominis and should not be confused with the aponeurosis of the transversus abdominis. The transversalis fascia is part of the inner investing fascia of the abdominal wall, and is continuous with the fascia iliaca, diaphragmatic fascia, parietal pelvic fascia, and thoracolumbar fascia, as well as the contralateral transversalis fascia behind the rectus sheath.

The *rectus abdominis* (Figure 3-2) arises from the crest of the pubis, and is inserted into the costal cartilages of the fifth, sixth, and seventh ribs and the xiphoid process. The muscle is enclosed in an aponeurotic sheath that is adherent to three tendinous bands that transverse the muscle, the linea transversae. The uppermost linea transversae is at the level of the xiphoid process, and the lowermost at the level of umbilicus, with the third being approximately midway between these. Additional linea transversae are often present.

The *rectus sheath* (Figure 3-2) has previously been described in conjunction with the aponeuroses of the external oblique, internal oblique, and transversus abdominis muscles. In summary, the rectus sheath can be divided

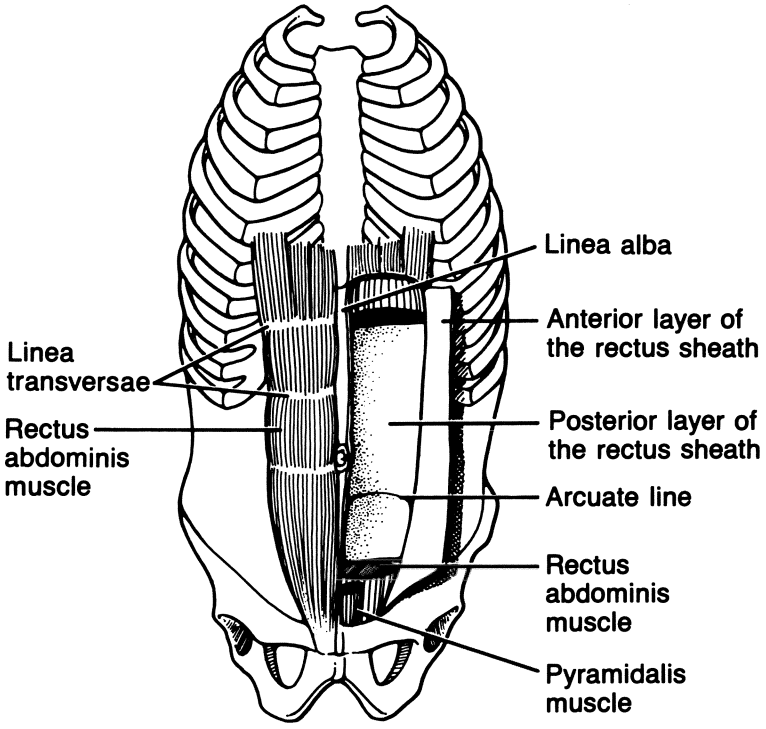


Figure 3-2. The rectus abdominis muscle is invested with an aponeurotic sheath. The rectus sheath is composed of the aponeuroses of the external oblique, internal oblique, and transversus abdominis muscles.

into a superior and an inferior portion. In the superior portion, which extends from the xiphoid process to a point midway between the umbilicus and the pubis, the rectus muscle is enveloped in a sheath formed by the splitting of the aponeurosis of the internal oblique. The aponeurosis of the external oblique contributes to the anterior wall of the sheath, while the aponeurosis of the transversus abdominis contributes to the posterior wall. In the inferior portion of the rectus sheath, the aponeuroses of both obliques and the transversus abdominis pass in front of the rectus muscle forming the anterior wall of the rectus sheath. Subsequently, only the transversalis fascia, extraperitoneal fat, and peritoneum lie deep to the rectus muscle. The free, crescentric border where the posterior wall of the rectus sheath ends is the *arcuate line,* which is located approximately midway between the umbilicus and the pubis. The anterior sheath of the rectus may contain the functionless pyramidalis muscle. When present, the pyramidalis originates from the body of the pubis and inserts into the linea alba.

The muscles of the abdominal wall are involved in numerous bodily functions. They are primarily responsible for the control of intraabdominal pressure, and consequently perform such varied functions as supporting the viscera, aiding venous return and lymphatic drainage, assisting respiration, and controlling all expulsive efforts such as urination, defecation, and parturition. The abdominal muscles also initiate flexion of the trunk on the hips and provide for rotation of the trunk at the waist. On contraction, the abdominal musculature forms a rigid wall that helps protect the abdominal viscera from trauma.

Blood Supply of the Anterior Abdominal Wall

There are four principal arteries supplying the abdominal wall: the superior epigastric artery and the musculophrenic branches of the internal (anterior) thoracic artery from above, and the deep circumflex iliac artery and the inferior epigastric artery from below (Figure 3-3).

The *superior epigastric* artery enters the sheath of the rectus from behind the seventh costal cartilage. It then descends behind the rectus muscle, supplying the muscle as well as other overlying tissues. The superior and inferior epigastric arteries anastomose freely, pro-

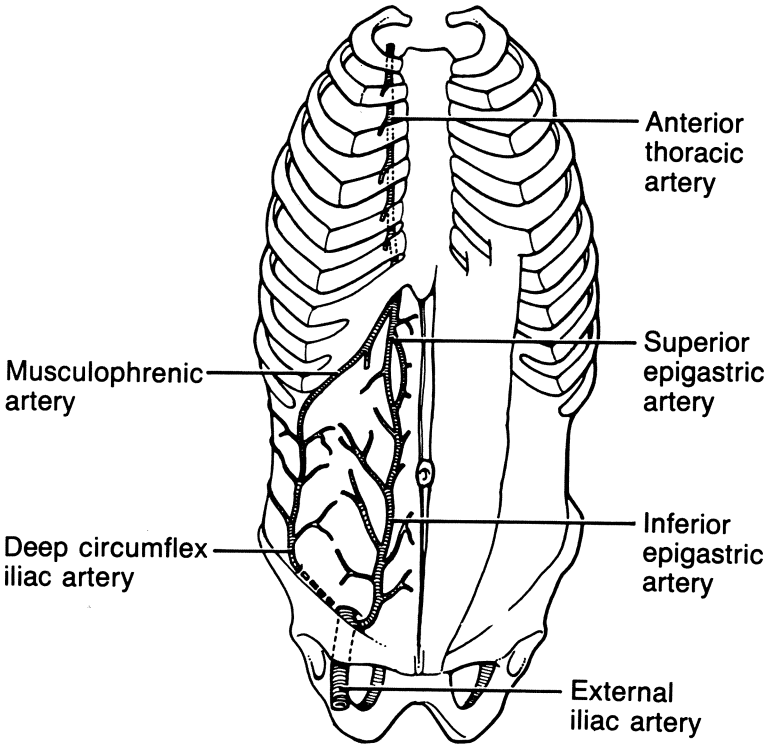


Figure 3-3. The blood supply of the anterior and lateral abdominal wall is derived from the superior epigastric and musculophrenic arteries superiorly and the inferior epigastric and deep circumflex iliac arteries inferiorly.

viding collateral circulation between the subclavian and external iliac arteries.

The *musculophrenic* artery lies behind the costal cartilages, and supplies the intercostal spaces, diaphragm, and upper abdominal wall.

The *deep circumflex iliac* artery, a branch of the external iliac artery, initially courses laterally and ventrally behind the inguinal ligament. The artery then turns medially and, following the arc of the iliac crest, pierces the transversus abdominis. The deep circumflex iliac artery ramifies between the transversus abdominis and the internal oblique muscles supplying the lower, lateral abdominal wall.

The *inferior epigastric* artery arises from the external iliac artery near the middle of the inguinal ligament. It then courses along the lateral border of the inguinal triangle, pierces the transversalis fascia, and enters the rectus sheath. The artery then ascends between the rectus muscle and the posterior wall of the rectus sheath supplying the overlying muscle, connective tissue, and skin. The inferior and superior epigastric arteries freely anastomose within the substance of the rectus muscle, providing collateral circulation between the subclavian and external iliac arteries.

Innervation of the Anterior Abdominal Wall

The abdominal wall is innervated by the *thoracoabdominal* (lower six intercostal) and the *iliohypogastric* and *ilioinguinal* nerves (Figure 3-4). The thoracoabdominal nerves descend obliquely behind the costal cartilages, gaining access to the interval between the transversus abdominis and the internal oblique, supplying these muscles as well as the external oblique and the overlying tissues. They continue medially, entering the sheath of the rectus, where they supply the rectus muscle as well as the overlying skin. The seventh intercostal nerve provides motor and sensory innervation to the region of the xiphoid process; the tenth intercostal nerve supplies the area of the umbilicus, and the twelfth intercostal nerve supplies the area just above the pubic symphysis. The iliohypogastric and the ilioinguinal nerves are derived chiefly from the first lumbar nerve and are cutaneous in distribution. The ilioinguinal nerve enters the inguinal canal, accompanying the round ligament to the labia majora. The iliohypogastric nerve supplies the skin overlying the mons pubis.

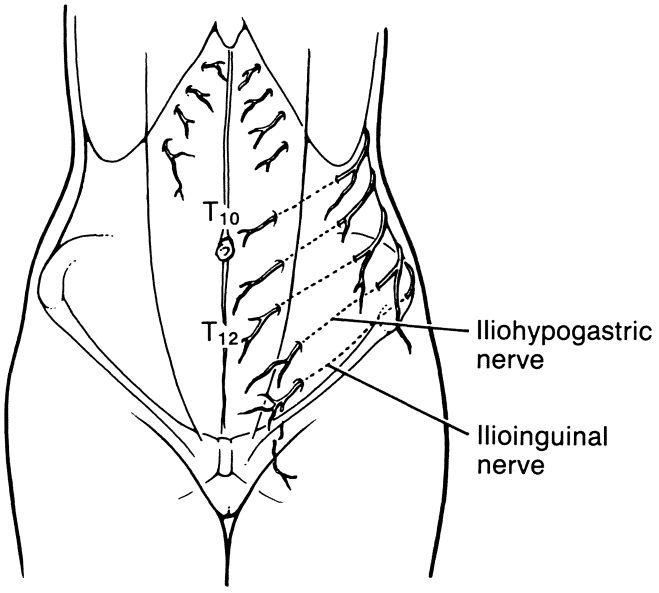


Figure 3-4. The anterior abdominal wall is innervated by the thoracoabdominal nerves (lower six intercostal nerves), iliohypogastric nerve, and ilioinguinal nerve.

Surgical Incisions

There are three major types of abdominal incisions through which pelvic surgery can be performed: (1) vertical or longitudinal incisions, (2) transverse incisions, and (3) muscle-splitting (gridiron) incisions.

Vertical Incisions

Several types of longitudinal incisions have been described (Figure 3-5). Each type of incision has advantages as well as limitations. The most commonly used vertical incisions are the midline incision and the paramedian incision. The transrectus (rectus-splitting) incision and the pararectus incisions, such as Battle's incision, are seldom indicated and rarely used.

The *midline incision* is the simplest, least hemorrhagic abdominal incision. It provides adequate exposure of the pelvic organs, and may easily be extended either through or around the umbilicus to provide access to the upper abdomen. The resulting scar is symmetric but does tend to broaden with time. The incidence of dehiscence and incisional hernia is relatively high with midline incisions for two reasons. First, healing is slowed by the relatively limited blood supply of the linea alba. Second, there is constant lateral tension on the suture line produced by the internal and external oblique and transversus abdominis muscles. The tension on the suture line is increased by coughing, retching, or straining postoperatively.

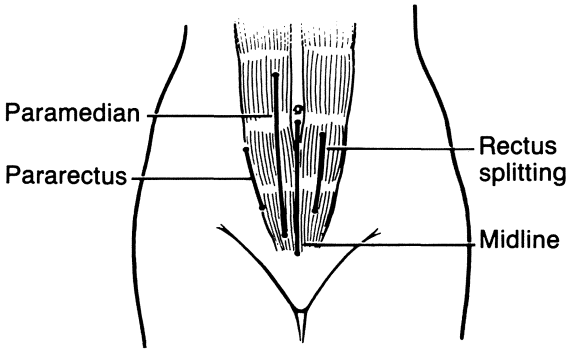


Figure 3-5. Vertical abdominal incisions. The location of the skin incision for midline, paramedian, pararectus, and rectus-splitting incisions is illustrated. The paramedian, pararectus, and rectus-splitting incisions can be located on either side of the abdomen and as far cephalad or caudad as is necessary for the operative procedure.

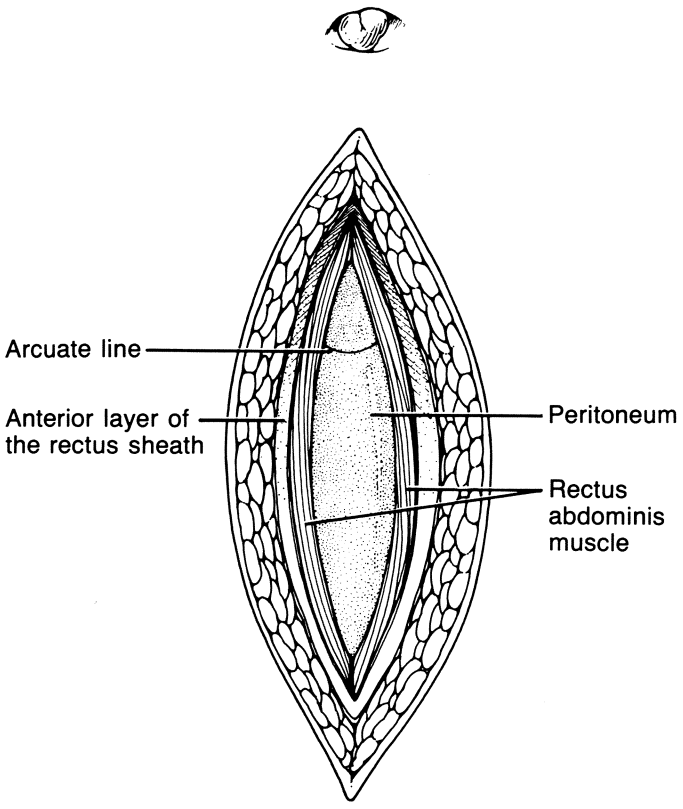


Figure 3-6. Midline incision. The skin, subcutaneous tissue, and anterior rectus sheath have been incised in the midline and the rectus muscles separated. The underlying posterior rectus sheath (above the arcuate line), transversalis fascia, and peritoneum are then incised.

The technique for performing a midline incision is uncomplicated (Figure 3-6). The skin is incised in the midline between the symphysis pubis and the umbilicus. The lower end of the incision should override the symphysis pubis for a short distance; otherwise, when the skin margins are retracted laterally the lower extent of the incision will be well above the symphysis pubis and will limit pelvic exposure. The underlying subcutaneous tissues are then divided and the linea alba identified. A short incision is made in the linea alba with the scalpel and extended for the desired distance with either scalpel or scissors. The underlying peritoneum can then be "tented" using small hemostats or fine pickups and incised. The initial peritoneal incision should be near the cephalad end of the incision to avoid injury to the bladder and inadvertent entry into pathologic pelvic processes.

Frequently, the fascial incision will be placed to one side of the linea alba, and the anterior wall of the rectus sheath will be opened exposing the underlying muscle. The medial flap of the rectus sheath is then reflected from the underlying muscle until the space between the two recti is identified. The rectus muscles are then separated by either sharp or blunt dissection for the full length of the incision and the abdominal cavity is entered as described above.

The *paramedian incision* has many of the advantages of the midline incision. It provides adequate exposure for all pelvic surgery and can easily be extended if necessary. Like the midline incision, it minimizes injury to thoracoabdominal nerves, limits trauma to the rectus muscle, and can be closed in an anatomic and secure fashion. The paramedian incision is located in a relatively vascular area and therefore heals well, making it less likely than the midline incision to give rise to an incisional hernia. Because the paramedian incision is more hemorrhagic than the midline incision, it requires more time to perform. Muscular branches of the inferior epigastric artery are often encountered in the lower portion of the

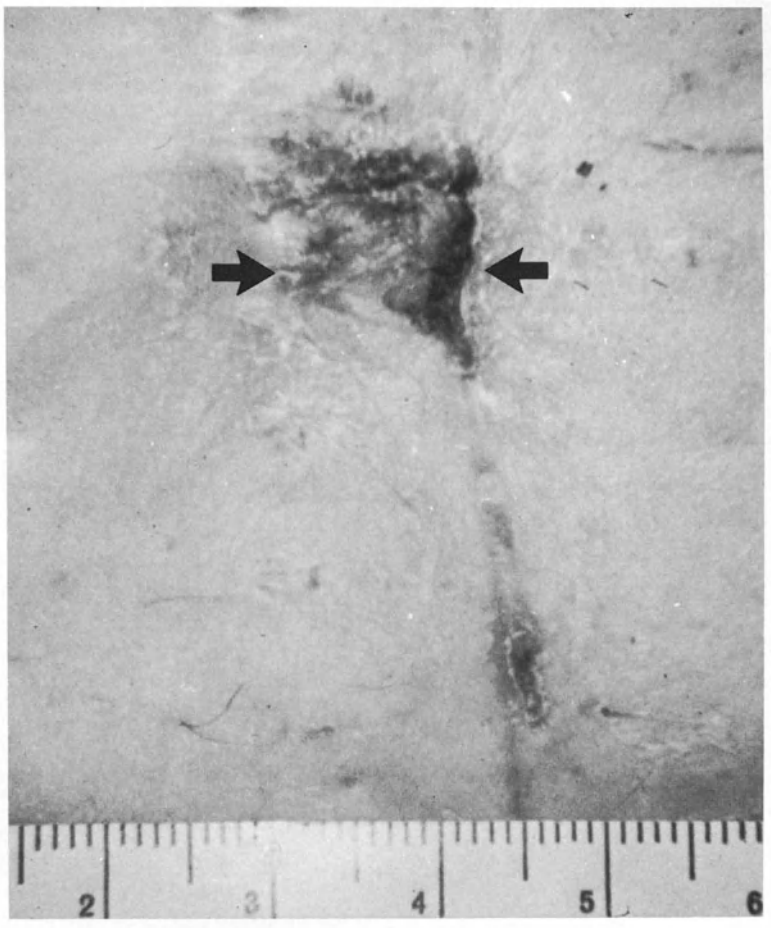


Figure 3-7. An area of aseptic necrosis between midline and paramedian incisions resulted when this patient had midline and right paramedian incisions (*arrows*) that compromised the blood supply from both lateral directions.

incision resulting in troublesome bleeding. The scar is asymmetric and often widens with time producing a poorer cosmetic result. A patient who has had previous surgery through a paramedian incision should not undergo a subsequent midline or contralateral paramedian incision, as this may interfere with the blood supply from both lateral directions and result in faulty healing (Figure 3-7).

To perform a paramedian incisions, a vertical incision is made through the skin and subcutaneous tissue approximately 1 in. lateral to the midline. Bleeding points are secured and the anterior leaf of the rectus sheath is opened through the extent of the incision (Figure 3-8A). The medial flap of the rectus sheath is then reflected from the muscle and the rectus muscle is gently retracted laterally, separating it from the linea alba. Care must be taken in retracting the rectus muscle laterally as the inferior epigastric vessels lie behind the rectus muscle and, if damaged, may cause considerable bleeding. The transversalis fascia and peritoneum underlying the rectus muscle are then incised (Figure 3-8B).

At the conclusion of the procedure, the incision can be closed in an anatomic and secure fashion that is unlikely to result in dehiscence or formation of a ventral hernia. The paramedian incision, like the midline incision, is under constant lateral tension from the abdominal musculature with the amount of tension being dependent on the length of the incision. Subsequently, long incisions extending into the upper abdomen have increased lateral tension on the wound, resulting in increased pain that is accentuated with inspiration. This can interfere with respiration and lead to an increased incidence of pulmonary complications.

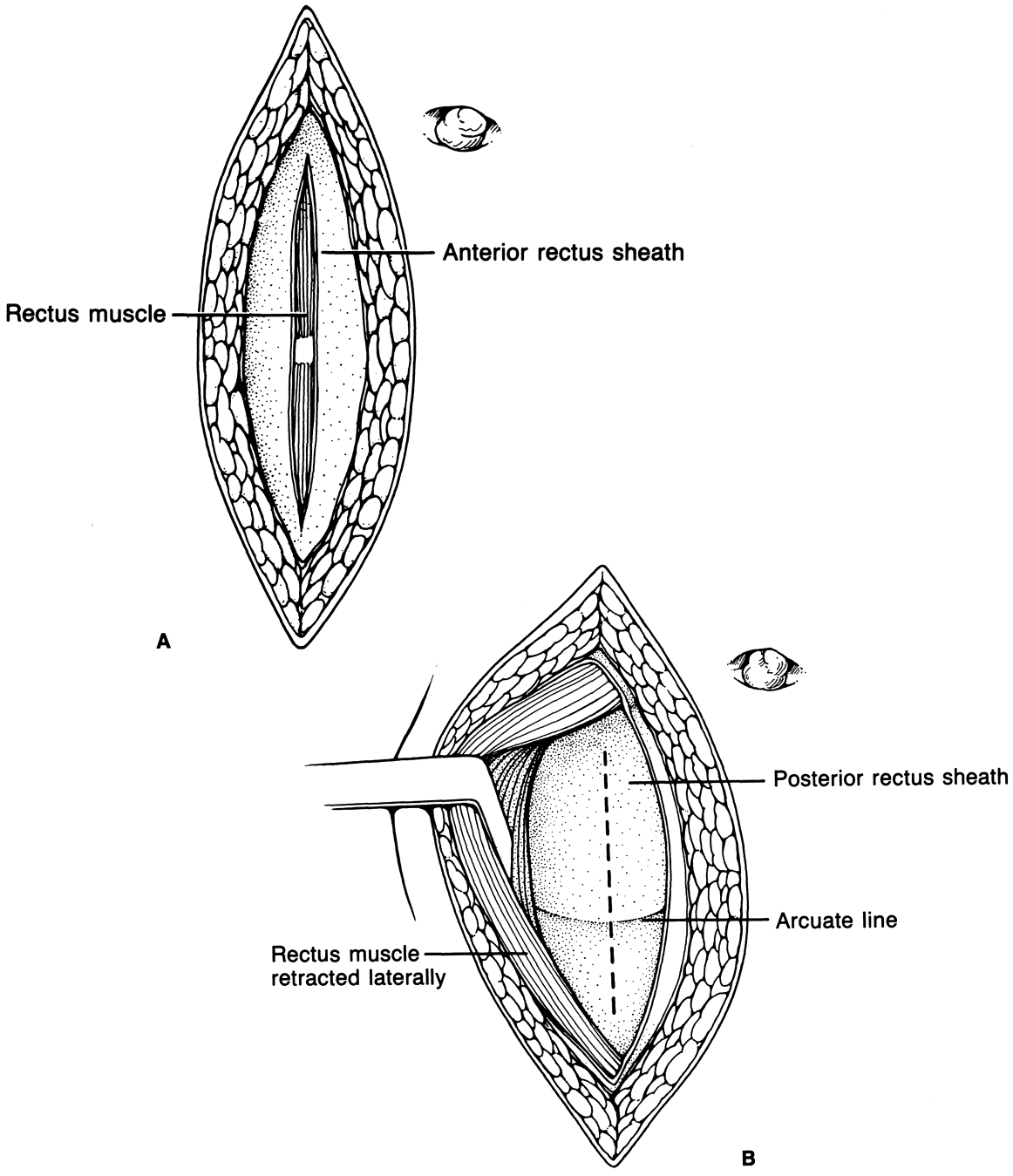


Figure 3-8. Paramedian incision. **A,** The skin, subcutaneous tissue, and anterior rectus sheath have been incised exposing the underlying rectus muscle. **B,** The rectus muscle is retracted laterally exposing the underlying posterior rectus sheath (above the arcuate line), transversalis fascia, and peritoneum, which are then incised.

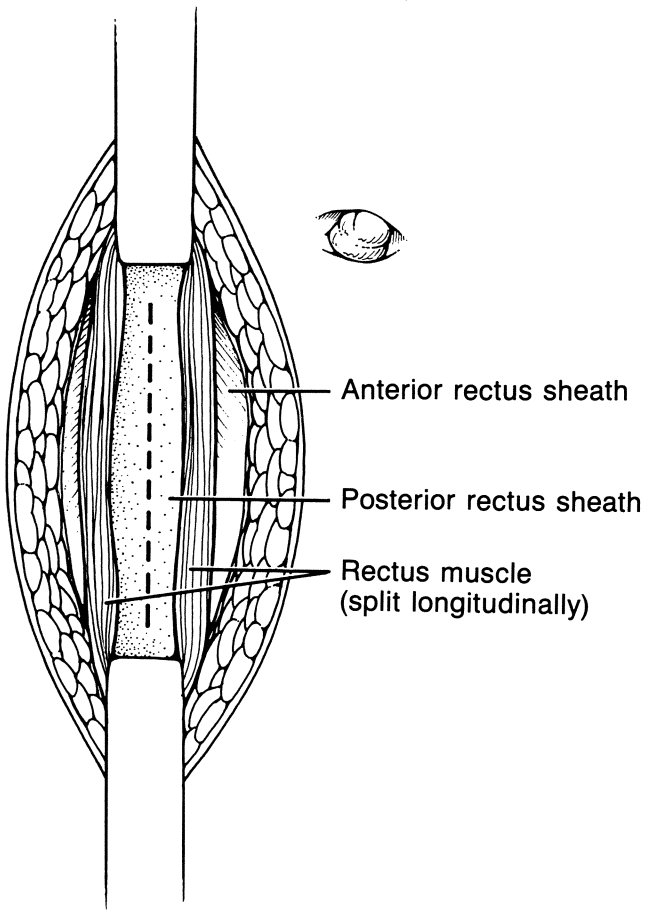


Figure 3-9. Transrectus or rectus-splitting incision. The anterior rectus sheath has been incised and the rectus muscle split longitudinally. This exposes the underlying posterior rectus sheath, transversalis fascia, and peritoneum, which are incised.

The *transrectus* or *rectus-splitting* incision can be performed more rapidly than the paramedian incision and will provide better lateral exposure, on the side of the incision, than the paramedian or midline incision. However, it must be of limited length. If more than two thoracoabdominal nerves are sacrificed, there will be some postoperative relaxation of the abdominal wall because the rectus muscle lying medial to the incision is deprived of nerve supply and subsequently atrophies. For this reason, a transrectus incision cannot be extended as can a paramedian or midline incision. However, a rectus-splitting incision may be converted to a paramedian incision if extension is found to be necessary.

The technique for the transrectus incision is similar to that for the paramedian incision. A vertical incision is made approximately ½–1 in. lateral to the midline, and the rectus sheath is identified. The anterior leaf of the rectus sheath is then incised longitudinally and the underlying rectus muscle divided for the length of the incision (Figure 3-9). The muscle fibers of the rectus can usually be easily separated by blunt dissection, but care must be taken to identify the underlying epigastric vessels, which may require ligation. The transversalis fascia and peritoneum underlying the rectus muscle are then incised as described above. The length of this incision is limited in that if more than two thoracoabdominal nerves are severed, the portion of the rectus muscle lying medial to the incision will be denervated and subsequently atrophy. Consequently, the denervated portion of the abdominal wall may be flaccid postoperatively.

The *pararectus* incision suffers from many of the same drawbacks as the rectus-splitting incision. The thoracoabdominal nerves are encountered at the lateral edge of the rectus muscle and should be preserved if possible.

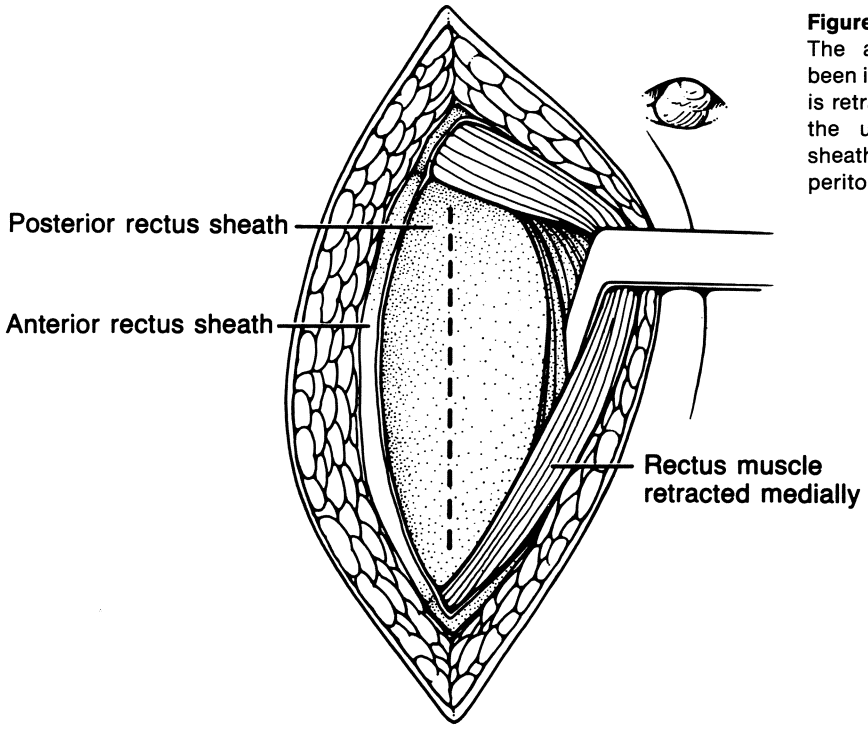


Figure 3-10. Pararectus incision. The anterior rectus sheath has been incised and the rectus muscle is retracted medially. This exposes the underlying posterior rectus sheath, transversalis fascia, and peritoneum, which are incised.

Consequently, the length of the incision is quite limited. This incision is not commonly used, but does provide better lateral exposure, on the side of the incision, than any of the incisions previously mentioned. A pararectus incision placed in the right-lower quadrant is often referred to as Battle's incision, and can be used for appendectomy.

The technique for pararectus incisions is similar to that for rectus-splitting incisions. A vertical incision is made approximately 1 in. lateral to the midline and carried through skin, subcutaneous tissue, and the anterior leaf of the rectus sheath. The lateral margin of the incised rectus sheath is then dissected from the underlying muscle, and the rectus muscle retracted medially (Figure 3-10). The transversalis fascia and peritoneum underlying the rectus muscle are then incised. Care must be taken to identify, and if necessary ligate, the inferior epigastric vessels, which are vulnerable to damage with this incision. The thoracoabdominal nerves, which may be identified where they enter the rectus muscle laterally, should also be preserved if possible. If these nerves are sectioned the rectus muscle medial to the incision is partially, or totally, denervated and may become flaccid postoperatively.

Transverse Incisions

As a group, transverse incisions offer several advantages over vertical incisions. Transverse incisions produce superior cosmetic results because they more closely follow Langer's skin lines, and the tension produced by the external oblique, internal oblique, and transversus abdominis muscles is in line with, or parallel to, the skin incision. These two factors combine to minimize the width of the scar. If the incision can be placed low on the abdominal wall, below the pubic hair line, the resultant scar is cosmetically pleasing and often difficult to detect.

The incidence of postoperative dehiscence or hernia formation is very low with transverse incisions. When the abdominal wall is incised transversely, the pull of the lateral abdominal musculature (internal oblique, external oblique, and transversus abdominis) is parallel to the incision, producing much less tension on the resultant suture line than occurs with vertical incisions. Transverse incisions of the lower abdomen interfere minimally with postopera-

tive respiration, and the incidence of pulmonary complications is subsequently low.

The principal disadvantage of transverse incisions is the inability to adequately explore the entire abdominal cavity, and the inability to extend the incision should upper abdominal exposure become necessary. The most commonly used tranverse incisions are the Pfannenstiel, Mackenrodt, Maylard, and Cherney incisions (Figure 3-11).

The *Pfannenstiel* incision is very popular in gynecologic surgery as it gives an excellent cosmetic result. The incision can be placed low on the abdominal wall, and the resultant scar does not tend to stretch. This incision closes in a secure, anatomic fashion, and postoperative dehiscence or hernia formation is almost unknown.

The principal disadvantage of the Pfannenstiel incision is the limited exposure provided by the incision, as noted above. Similarly, this incision is not suited for the removal of large pelvic or abdominal masses. The incision tends to be more hemorrhagic than a midline or paramedian incision and significantly slower to perform. For these reasons, a Pfannenstiel incision is contraindicated when speed in entering the abdomen is essential, when a large pelvic mass is present, or when upper abdominal exposure may be needed.

Although exposure is not as good as with a midline or paramedian incision, it is adequate for most obstetric and gynecologic procedures including lower segment cesarian section and relatively uncomplicated hysterectomy. If better exposure is required after a Pfannenstiel incision has been made, it can be converted to a Maylard incision; this will provide excellent exposure to the pelvis but will not allow adequate examination of the upper abdomen. If thorough examination of the upper abdomen is required, a supraumbilical midline or paramedian incision must be performed.

The technique for performing a Pfannenstiel incision was initially described in 1900.[1] A transverse incision, which is slightly convex downward, is placed two fingerbreadths above the symphysis pubis and extended for a total length of 10–15 cm. The incision is carried through skin and subcutaneous tissue down to the rectus sheath. The anterior leaves of the rectus sheaths are incised transversely on either side of the linea alba and the linea alba cut joining the two lateral incisions. This creates a superior, or cephalic, flap and an inferior, or caudad, flap. Each flap consists of skin, subcutaneous tissue, and anterior rectus sheath, and both flaps are elevated from the underlying rectus muscles by a combination of sharp and blunt dissection (Figure 3-12A). All bleeding points are carefully secured, as bleeding in this area can result in a subfascial hematoma postoperatively. The rectus muscles are then separated in the midline and the transversalis fascia and peritoneum incised longitudinally (Figure 3-12B). Care must be taken at the lower pole of the incision to avoid injury to the bladder.

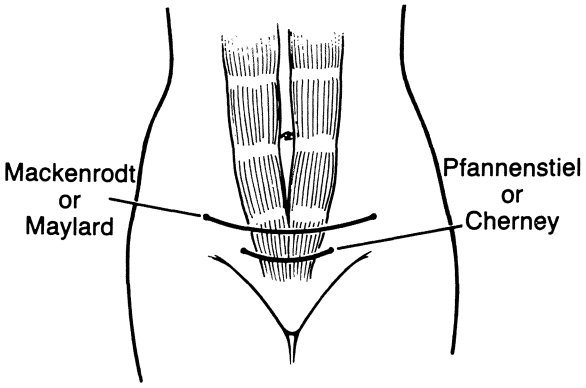


Figure 3-11. The location of the transverse skin incisions for Pfannenstiel, Mackenrodt or Maylard, and Cherney incisions. The position of the incision can be adjusted cephalad or caudad to suit the needs of the operation.

The *Kustner* incision[2] differs from the Pfannenstiel incision in that the subcutaneous tissue is dissected from the anterior rectus sheaths and the linea alba incised vertically as it is in a midline incision (Figure 3-13). This incision is often referred to as the "modified Pfannenstiel" incision and provides very poor exposure.

The *Mackenrodt* and *Maylard* incisions are similar. Mackenrodt[3] first described a low abdominal, transverse, muscle-splitting incision in 1901. This incision was specifically designed to optimize pelvic exposure for extensive pelvic procedures such as radical hysterectomy. In 1907 Maylard[4] described a similar incision, restricting location only to "below the umbilicus."

This type of incision has many characteristics in common with the Pfannenstiel incision.

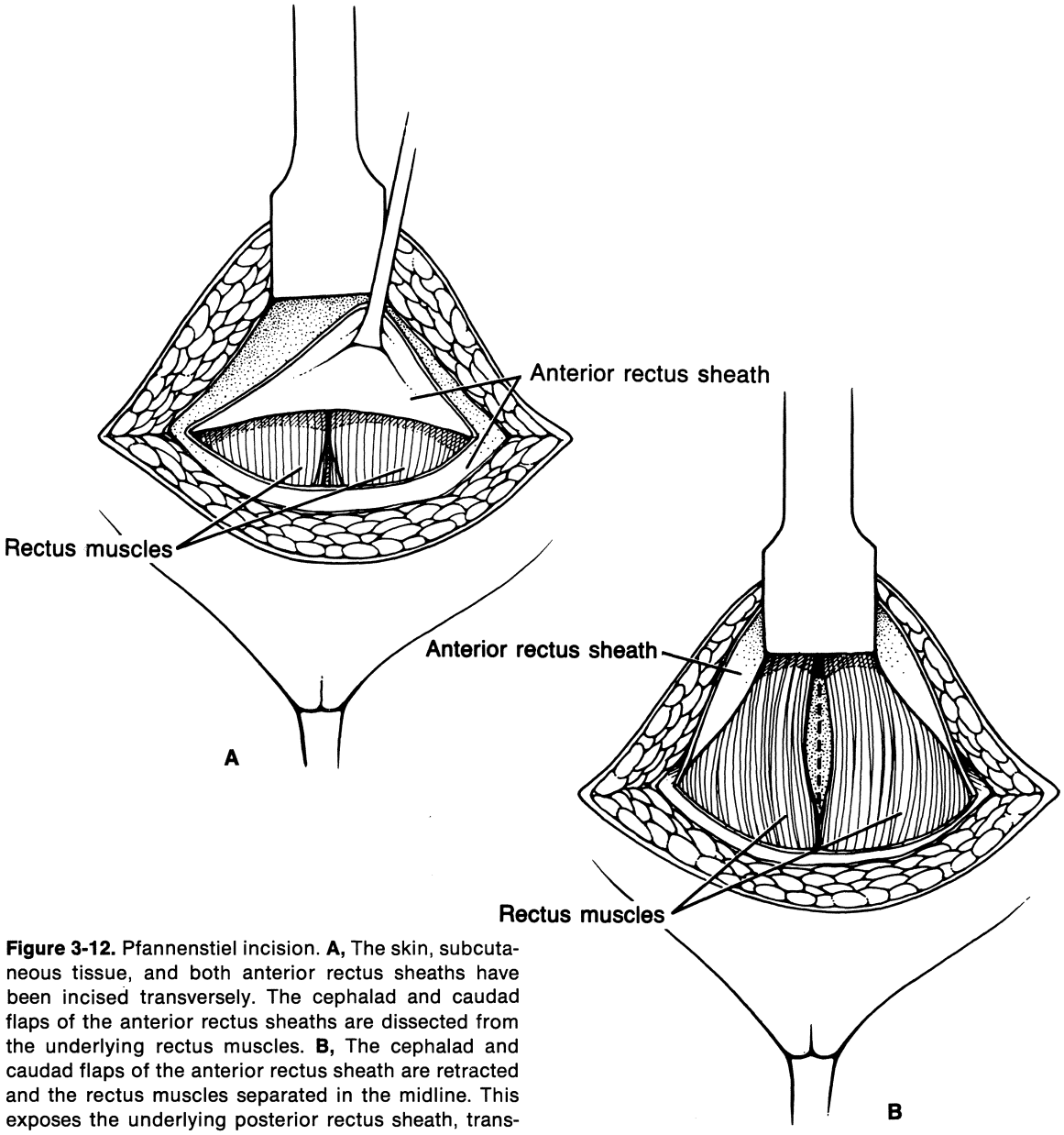


Figure 3-12. Pfannenstiel incision. **A,** The skin, subcutaneous tissue, and both anterior rectus sheaths have been incised transversely. The cephalad and caudad flaps of the anterior rectus sheaths are dissected from the underlying rectus muscles. **B,** The cephalad and caudad flaps of the anterior rectus sheath are retracted and the rectus muscles separated in the midline. This exposes the underlying posterior rectus sheath, transversalis fascia, and peritoneum, which are incised.

It results in a cosmetic scar that does not tend to widen with time, and the innervation of the abdominal musculature remains basically undisturbed. Tension from the transversus abdominis and oblique muscles is parallel to the line of incision and does not put tension on the suture line. Consequently, the incidence of postoperative dehiscence or hernia formation is low. The principal advantage of this incision over the Pfannenstiel incision is the excellent pelvic exposure that is provided. Unfortunately, upper abdominal exposure is compromised when the incision is placed low on the abdominal wall. The Mackenrodt/Maylard incision is significantly slower to perform than is the midline incision and tends to be more hemorrhagic. The rectus muscles must be divided, and both superficial and deep inferior epigastric vessels frequently require ligation.

This incision is desirable when speed in entering the abdomen is not a primary concern, a large pelvic/abdominal mass is not present,

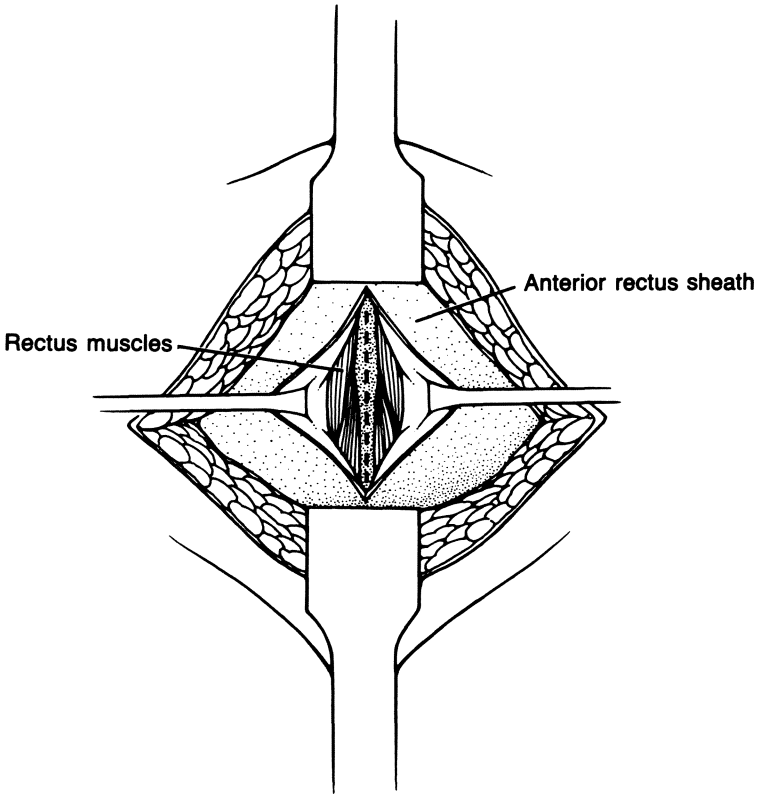


Figure 3-13. Kustner incision. The skin and subcutaneous tissue are incised transversly, as with a Pfannenstiel incision. The subcutaneous tissue is then separated from the underlying anterior rectus sheaths and the fascia and peritoneum incised in the midline as with a midline incision. The incision is often referred to as a "modified Pfannenstiel" incision.

Figure 3-14. Mackenrodt or Maylard incision. The anterior rectus sheaths and rectus muscles are divided transversely and the inferior epigastric vessels ligated. The transversalis fascia and peritoneum are then incised.

surgical procedures in the upper abdomen are not anticipated, and optimal exposure to the pelvic cavity is desired.

The technique for Mackenrodt/Maylard incisions is initially similar to that for Pfannenstiel incisions. A suprapubic transverse skin incision is made, the length of which is determined by the anticipated procedure. This incision is carried through the subcutaneous tissue to the anterior rectus sheath and aponeurosis of the external oblique. The superficial inferior epigastric vessels are often encountered and require ligation. The anterior rectus sheath and linea alba are incised transversely and the rectus muscles divided transversely (Figure 3-14). This can be accomplished with cutting cautery, to diminish blood loss. Care must be taken to identify and ligate the deep inferior epigastric vessels before they are cut. The tansversalis fascia and peritoneum then are incised and the incision extended laterally through the aponeuroses of the oblique

and transversus abdominis muscles for the desired distance.

This incision can be closed in an anatomic and secure fashion by first reapproximating the peritoneum and transversalis fascia with absorbable suture and then reapproximating the aponeuroses and anterior rectus sheath with absorbable or permanent suture. The rectus muscles need not be reapproximated with suture, as the area of the incision will form another "linea transversae" and no dysfunction will result. However, the rectus muscles are sutured to the anterior leaf of the rectus sheath to prevent their retraction and to minimize the "dead space" left within the rectus sheath. The cut edge of the rectus muscle must be carefully examined for hemostasis.

The *Cherney* incision[5] was described in 1941. Like the previously described transverse incisions, it results in a cosmetic scar and tends to close in a secure, anatomic fashion. The pelvic exposure utilizing a Cherney incision is better than that obtained with a Pfannenstiel incision, but not as good as that obtained with a Mackenrodt or Maylard incision. The Cherney incision provides very limited access to the upper abdomen, and is slower to perform and more hemorrhagic than a midline incision.

This incision can be utilized when speed in opening the abdomen is not essential, large pelvic/abdominal masses are not present, and surgical exposure of the upper abdomen is not required. The Cherney incision can be used for most routine gynecologic procedures, and provides excellent access to the space of Retzius for urinary stress incontinence procedures.

The technique for Cherney incisions is similar to that for the Maylard incision. A transverse incision, slightly convex downward, is made just about the level of the pubis and carried through skin and subcutaneous tissue to the rectus sheath and aponeurosis of the external oblique. The length of the incision is dependent on the requirements of the procedure being performed. The anterior wall of the rectus sheath and linea alba are opened transversely, and the caudad flap of the anterior rectus sheath is dissected from the underlying rectus muscle. This requires that the fibrous septum, which extends from the deep aspect of the anterior rectus sheath between the medial margins of the rectus muscles, be incised. When the lower flap of the rectus sheath has been dissected from the underlying rectus muscles as far as the pubic bone, the rectus muscles are transected at their insertion (Figure 3-15). This portion of the muscle is normally very fibrous, and bleeding is negligible. The rectus muscles are then reflected upward and the peritoneal cavity entered by incising the transversalis fascia and peritoneum.

An incision extending from one inferior epigastric artery to the other is sufficient for most types of pelvic surgery. However, if greater exposure is necessary, the inferior epigastric vessels can be ligated and the incision extended laterally into the aponeuroses of the oblique and tansversus abdominis muscles. If only access to the space of Retzius is required and no intraperitoneal surgery is planned, the incision can be performed as above with the exception that the transversalis fascia and peritoneum are not incised.

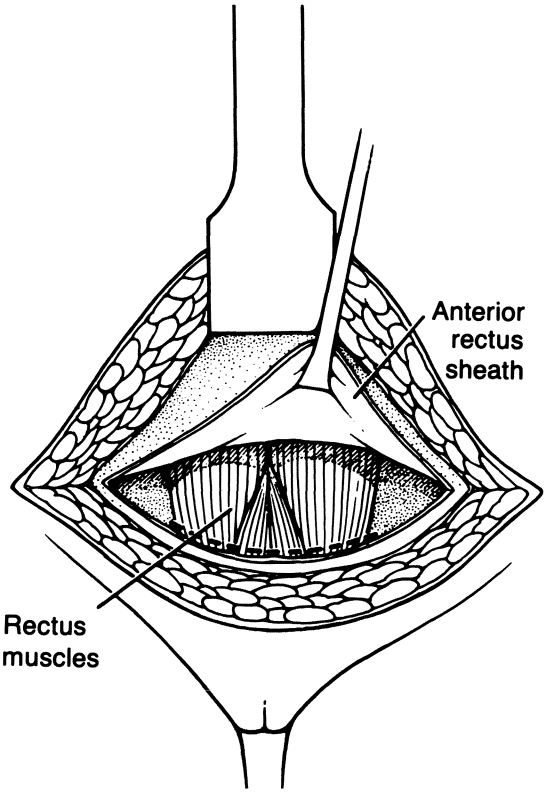


Figure 3-15. Cherney incision. The anterior rectus sheaths are incised transversely and separated from the underlying rectus muscles and the rectus muscles transected at their insertion to the pubic bone. This allows the rectus muscles to be reflected cephalad and the transversalis fascia and peritoneum to be incised transversely.

This incision can be closed in an anatomic, secure fashion by reapproximating the transversalis fascia and peritoneum with absorbable suture, tacking the ends of the rectus muscles to the undersurface of the rectus sheath with absorbable suture, and reapproximating the anterior rectus sheath with interrupted sutures of absorbable or nonabsorbable material.

The *gridiron* incision is seldom indicated in gynecologic surgery. Its principal use is for uncomplicated appendectomy, although this incision may be utilized for draining large pelvic/abdominal abscesses that do not point into the cul-de-sac and are not responsive to antibiotic therapy. In this situation, the position of the incision must be modified to suit its purpose.

For its usual application, the gridiron incision is made obliquely downward and medially over McBurney's point (Figure 3-16A). The incision is carried through the skin and subcutaneous tissue to the aponeurosis of the external oblique. The fibers of this aponeurosis course parallel with the incision and are separated in the line of the incision (Figure 3-16B). This exposes the underlying aponeuroses of the internal oblique and transversus abdominis, whose fibers run medially; these are also separated in the direction in which they run (Figure 3-16C). This exposes the transversalis fascia and peritoneum, which are incised.

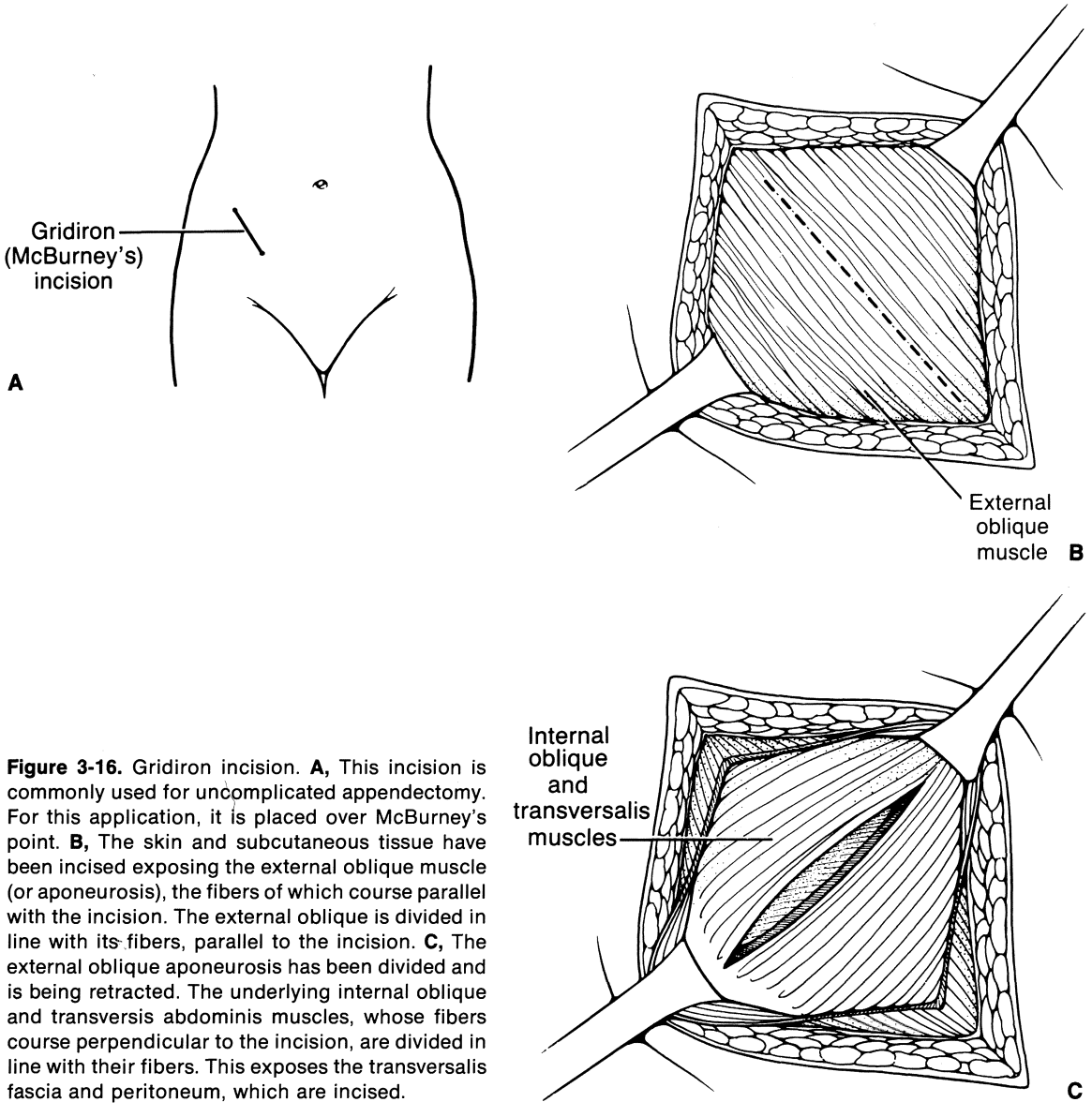


Figure 3-16. Gridiron incision. **A,** This incision is commonly used for uncomplicated appendectomy. For this application, it is placed over McBurney's point. **B,** The skin and subcutaneous tissue have been incised exposing the external oblique muscle (or aponeurosis), the fibers of which course parallel with the incision. The external oblique is divided in line with its fibers, parallel to the incision. **C,** The external oblique aponeurosis has been divided and is being retracted. The underlying internal oblique and transversis abdominis muscles, whose fibers course perpendicular to the incision, are divided in line with their fibers. This exposes the transversalis fascia and peritoneum, which are incised.

In summary, a number of incisions are available to the gynecologic surgeon. It is the responsibility of the surgeon to choose the incision that is best suited to the planned operative procedure, anticipated pathology, and needs of the individual patient.

References

1. Pfannenstiel J. Ueber die Vortheile des suprasymphysaren Fascienquerschnitts für die gynäkologischen Koliotomien zugleich ein Beitrag zu der Indikationsstellung der Operationswege. Samml Klin Vortr (Leipzig). 1900; 268:1735.
2. Kustner O. Methodik der gynaekologischen Laparotomie. Verh Dtsch Ges Gynäkol (9 Kongr). 1901; ix:580.
3. Mackenrodt A. Die Radikaloperation des Gebärmutterscheidenkrebses mit Ausräumung des Beckens. Verh Dtsch Gynokol 1901; ix:139.
4. Maylard AE. Direction of abdominal incisions. Br Med J. 1907; 2:895–901.
5. Cherney LS. A modified transverse incision for low abdominal operations. Surg Gynecol Obstet. 1940; 72:92–95.

Mechanical and Chemical Preparation of the Abdomen and Vagina

4

Leslie A. Walton and Vicki V. Baker

Introduction

Disruption of the tissue integrity of the abdominal or vaginal walls exposes the patient to colonization and possible infection by bacterial agents from her own flora or the surrounding environment. It is currently estimated that over 300,000 postoperative wound infections occur annually in the United States.[1] When infection occurs, wound healing is impaired. This complication not only results in a less favorable cosmetic result but increases the risks of wound disruption and generalized sepsis. The occurrence of a wound infection prolongs the duration of hospitalization by a factor of two with an attendant increase in medical care costs.[2] These findings are among the reasons why renewed emphasis is being placed upon preparation of the abdomen and vagina in an effort to decrease postoperative morbidity. This chapter will address the rationale and methodology of preparation of the abdomen and vagina for gynecologic procedures.

The Patient and Her Environment

Certain factors may predispose the patient to postoperative infections. Frequently cited factors include the following:

a. Advanced age of the patient
b. Malnutrition
c. Regional infection
d. Obesity
e. Concomitant illnesses, e.g., diabetes mellitus, hypertension, or neoplasia
f. Duration of the surgical procedure: The infection rate of a clean wound doubles every hour of surgery after the first hour
g. Duration of preoperative hospitalization: The patient is colonized by hospital pathogens when hospitalization is prolonged
h. Recent history of antibiotic ingestion: alteration of endogenous flora may contribute to antibiotic resistance
i. Compromised immunologic host

Among these general factors, medical diseases may play an adverse role. In a prospective study of the occurrence of infection in 23,649 surgical wounds during a 5-year period, the overall infection rate was 1.8% in "clean wounds".[3] However, the infection rate was 3.5% in obese patients, 10.7% in diabetic patients, and 16.6% in malnourished patients.[3] While diabetes mellitus and nutritional status are important, these factors become less dominant as predisposing factors when the other confounding variables such as age and the length of surgery are taken into account. It is important to identify and correct, when feasible, any of these associated conditions that might contribute to wound infection.

The surgical patient is subject to potential bacterial contamination from many sources.

a. Endogenous flora
b. The operating surgeon or surgical team

c. Airborne contaminants
d. Unsterile instruments

Of these factors, endogenous bacterial contamination is *the* most significant factor causing wound infections.[4]

Common resident skin flora include micrococci, corynebacteria, *Propionobacterium acnes* and occasionally *Staphylococcus aureus*. Quantitative and qualitative variations occur among different anatomic sites and among individuals.[5] The vagina and cervix are normally colonized by a variety of potentially pathogenic aerobic and anaerobic organisms, including gram-positive cocci, *Bacteroides* and *Proteus* spp., aerobic streptococci,[6–8] *Escherichia coli* and *Klebsiella*.

In the absence of unusually virulent organisms, the wound of a healthy patient with intact host defenses can withstand a bacterial innoculum of 10^5 organisms per milligram of tissue.[9] On the basis of the degree of potential endogenous bacterial contamination, surgical incisions may be classified as clean, clean-contaminated, contaminated, or dirty[10] (Table 4-1). The influence of the size of the bacterial innoculum is evidenced by the increasing incidence of infection in the spectrum from clean to dirty surgical cases.[3,10] The adequacy of host defenses alone is exemplified by a wound infection rate of only 7% in patients who underwent nonsterile open cardiac massage.[11]

The problem of disinfection of body surfaces in preparation for surgical procedures has been of interest to clinicians for many years. The dramatic reduction in the death rate from puerperal sepsis by hand disinfection as demonstrated by Semmelweiss in 1847 remains a landmark in infection control. Poor techniques acquired during training may be incorporated into practice in succeeding years. However, the scientific rationale for present techniques is not clearly and unequivocally documented. Reassessment of these time-honored methods is germane, since there has been little improvement in the incidence of postoperative wound infection during the past 20–30 years in spite of various efforts at infection control.[12]

Table 4-1. Classification of Operative Wounds in Relation to Potential Contamination[a]

Clean	No infection encountered, no break in aseptic technique and no hollow muscular organ entered. Cholecystectomy, incidental appendectomy, and hysterectomy are classified as clean wounds if no acute inflammation or pus is present. The incidence of infection is 1.5%–3.3%.
Clean-contaminated	A hollow muscular organ is entered, with minimal spillage of contents. The incidence of infection is 7.7%–10.8%.
Contaminated	Hollow muscular organ entered with gross spillage of contents or acute inflammation without pus formation is encountered. The incidence of infection is 15.2%–16.3%.
Dirty	Pus encountered or a perforated viscus found. The incidence of infection is 28.6%–40%.

[a] From Pollock and Evans.[4]

Preparation of the Abdominal Wall

A shower the night before surgery using an antiseptic type of soap to clean the abdomen, skin crevices, umbilicus, and genital area has been shown to decrease the incidence of wound infection.[3]

Traditionally, body hair at the operative site is removed prior to surgery. Advocates of this practice contend that hair is a source of bacterial contamination. However, the contribution of body hair to wound infection is debatable, based on studies demonstrating that deep portions of the hair follicles do not harbor organisms,[13] that the density of hair growth is not related to the magnitude of the surface flora,[13] and that hair removed from clean skin covered with sterile films is sterile.[7]

All methods of hair removal are associated with some degree of epidermal trauma Sufficient hair should be removed so that hair is not incorporated into wound closure. Methods of hair removal include the use of a depilatory, shaving with a razor, and hair removal with a clipper. Although a depilatory avoids any surface trauma, its application elicits a dermal lymphocytic reaction.[14] Shaving results in mac-

roscopic and microscopic surface lacerations and abrasions of the skin. When electron microscopy studies are performed after shaving, considerable skin damage is found, especially in areas with crevices and folds.[14] The traumatized areas can serve as a nidus for infection. The interval between hair removal and the performance of the surgical procedure influences the risk of infection. With time, endogenous saprophytic bacteria multiply in the dermis and therefore increase the size of the potential bacterial inoculum presented to the surgical incision.

Three modifications of the time-honored process of shaving the abdomen are now in vogue—immediate preoperative shaving, hair clipping, and the use of a depilatory. Seropian and Reynolds[15] reviewed the incidence of wound infection in 406 patients undergoing clean surgical procedures. The infection rate was 7.1% in patients who were shaved 24 hr before surgery as compared to 3.1% for patients who were shaved on the morning of surgery. Hair removal on the morning of surgery is preferred to shaving the night before surgery.

When hair removal is necessary, the use of an electric clipper rather than shaving is preferable. A group of 200 patients scheduled for elective inguinal herniorrhaphy were prospectively studied.[16] One group of patients was subjected to electrical hair clipping; the other group was prepared by razor shaving. Both procedures were performed immediately prior to surgery. The wound infection rate in the shaved group was 2% versus 1% for the clipped group. The difference in infection was not statistically significant, although hair clipping resulted in less skin trauma.[17] No data are presently available on the incidence of wound infection if the hair is clipped the night before surgery as compared to the morning of surgery. However, hair removal using an electric clipper immediately before surgery would seem preferable.

The third method of hair removal involves the use of a depilatory. Depilatories contain a calcium thioglycollate–calcium hydroxide–strontium hydroxide complex as the active agent. The depilatory can be applied the night before surgery without an increase in the incidence of infection and may be preferable for body areas that are difficult to prepare with electric clippers or a razor, for example, axilla and vulva.[18,19] After application, allergic reactions are unusual.[19,20] Although it can be applied by the patient and is an effective method of removing body hair, it is expensive. Compared to other methods of hair removal, use of a depilatory results in a 0.6% infection rate as compared to 5.6% if the hair is removed by shaving immediately before surgery.[15] Interestingly enough, when the hair is not removed, the wound infection rate is only 0.6%,[15] which is equivalent to the incidence associated with the use of depilatory.[18] Thus the use of a depilatory and/or immediate preoperative hair clipping constitute the best methods of hair removal.

Chemical Agents for Disinfection

The critical goal of the abdominal wall or vaginal mucosal preparation is a reduction of bacterial density at the prepared site. The preparation solution must be bacteriostatic or bacteriocidal, and this activity should be maintained for prolonged periods. In addition, there must be no adverse effect on wound healing. Several solutions and detergents, alone or in combination, have been studied as surface disinfectants.

Hexachlorophene (pHisoHex, pHisoDerm), a chlorinated bisphenol, is a bacteriostatic detergent that is active against many gram-positive organisms (Table 4-2). It exhibits very little activity against gram-negative bacteria. If applied repeatedly over 3–4 days, a protective residue forms that lasts for several days. However, the use of alcohol, even in perfume or aftershave lotion, can disrupt this protective residue.[21] Systemic absorption and accompanying potential toxicity limit the use of this agent. A questionable increase in the incidence of birth defects have been reported in association with the prolonged use of this substance.[22–25] Its routine use should be very carefully considered.

Benzalkonium chloride, a quaternary ammonium compound, is an excellent skin disinfectant. However, it is readily inactivated by absorbent materials or woven fabrics, both of which are commonly used materials. In addition, it is readily contaminated by *Pseudomonas* with epidemics of infection and septicemia re-

ported.[26–28] It does not currently enjoy wide usage.

Chlorhexidene gluconate (Hibiclens) is one of the newer antiseptic agents and is the only one currently approved by the F.D.A. (Table 4-2).[29] Chlorhexidene as Hibitane has enjoyed extensive use abroad for more than 20 years. Hibiclens is the trade name for a 4% chlorhexidene gluconate with 4% isopropanol in a sudsing skin cleanser. It is active against gram-negative and gram-positive organisms and, after repeated applications, provides cumulative continuing antibacterial action. Systemic absorption following mucocutaneous application does not occur.[30]

Iodine in some form has been used as the standard antiseptic solution for many years. It may be applied to the skin as a 2% solution in 70% alcohol. Although an excellent disinfectant, it is frequently irritating to the skin.

Polyvinylpyrrolidone-iodine (povidone-iodine) is a combination of iodine and a high molecular weight water-soluble polymer (Table 4-2). The slow release of iodine by the povidone–iodine complex creates bacteriocidal activity for a wide variety of organisms. Even in patients sensitive to iodine, toxicity rarely occurs because very little of the complex undergoes cutaneous absorption from intact skin.[31] In addition, skin, wound, and mucous membrane irritation is rarely reported.[31] Unlike chlorhexidene and hexachlorophene, povidone-iodine does not persist after repeated applications because it is water soluble.[32]

Several comparative studies of povidone-iodine, chlorhexidene, and hexachlorophene have been published. The efficacy and drawbacks of each of these agents must be considered. Povidone-iodine results in a greater decrease in bacterial skin counts in the first hour as compared to hexachlorophene, but this advantage is lost after 1 hr of glove time.[33] Another disadvantage of povidone-iodine is its inactivation by palmar sweat.[34] Chlorhexidene is superior to povidone-iodine and hexachlorophene in reducing bacterial skin counts and in maintaining greater residual activity.[35] Differences in study design, the use of prophylactic antibiotics, the statistical analyses (univariate versus multivariate), and the criteria for the diagnosis of wound infection make it difficult to compare the various studies using these disinfectants.

Operative Wound Infection

There is no apparent clinical difference between a 99% and a 70% reduction in bacterial skin counts. Using strict criteria for the diagnosis of wound infection, Berry et al.[36] could not demonstrate a clinically significant difference between povidone-iodine and chlorhexidene in a large series of prospectively randomized surgical procedures. Analysis of the subsets of patients who underwent "clean operations" demonstrated a 1.2% infection rate in the povidone-iodine group as compared to 3.1% in the chlorhexidene group. Cruse and Foord[3] were also unable to demonstrate a difference

Table 4-2. Contemporary Skin Disinfectants

Agent	Major range of activity	Potential toxicity	Absorption	Potential for contamination
Hexachlorophene	Gram-negative	Vacuolar encephalopathy ?? Increased birth defects	3% of topically applied product absorbed	Gram-negative *Candida*
Chlorhexidene	Gram-positive and Gram-negative fungi	Rare dermatologic reaction	None	None
Povidone-iodine	Gram-positive and gram-negative yeast	Allergic reaction; 1:500	None	None

in the incidence of wound infections when povidone-iodine, hexachlorophene, or chlorhexidene was used.

Preoperative Preparation

Because of a desire to reduce anesthesia and operating room time without increasing the risk of abdominal contamination, the duration of the hand and abdominal scrub in the surgical preparation was evaluated. Dineen[37] reported equivalent bacterial skin counts after a 5- or 10-min scrub using either povidone-iodine or hexachlorophene. In addition, the clinical report of Cruse and Foord[38] failed to demonstrate an increase in clean wound infections when a 3- to 5-min scrub was utilized for the first operation of the day and a 2- to 3-min scrub was performed before succeeding operations on that same day. Other available studies demonstrated equivalent or greater reductions in bacterial skin counts using a 90-sec jet wash or hydroscrub as compared to the more conventional scrub techniques presently used.[39–41] In summary, shorter scrub periods are as effective as longer scrub periods and are cost containing.

Operative Hand Preparation

The traditional use of a scrub brush is also subject to question. Apart from an increased incidence of dermatitis after repeated scrubs, there is a theoretical risk of uncovering and exposing the bacterial flora of the deeper skin layers. Galle et al.[42] were unable to demonstrate a difference in bacterial skin counts after 1 hr of glove time when a two-brush, 10-min povidone-iodine scrub was compared to a 5-min, one brush povidone-iodine scrub versus a 3-min, plain soap and hexochlorophene foam prep.[42] These refinements in hand scrub technique can further reduce operating room usage and costs without compromising antisepsis of the abdomen.

Considering the available information, there is no clearly superior antiseptic solution. The surgeon's preparation of choice is based on several considerations, including antibacterial efficacy, duration of continued activity, ease of application, and cost. The following are our recommendations for transabdominal pelvic surgery:

1. Use an effective solution
2. Hand scrub for 5 min
3. Apply the antiseptic solution for a minimum of 3–5 min
4. Extend the abdominal preparation from the pubic symphysis to the xiphoid process, and from the body–table interface on one side to the similar contralateral interface
5. Dry the surgical site with a sterile towel
6. The patient should not lie on wet covers saturated by antiseptic solution. This increases the likelihood of skin irritation or allergic reactions and also decreases body temperature.
7. Defer the use of plastic draping except when indicated to isolate specific problem areas. Waterproof drapes are impermeable adhesive-coated plastic sheets used to immobilize and prevent skin bacteria from entering the wound. Several studies failed to demonstrate reduction in postoperative wound infection when the use of these drapes was compared to the use of antiseptic solutions alone.[3,43,44]

Preparation of the Vagina

Operative procedures involving the vagina are fraught with the potential for infection because the vagina harbors a large number of aerobic and anaerobic bacteria.[45] It has been estimated that 29% of patients harbor pathogenic vaginal bacteria preoperatively.[46] Postoperative febrile morbidity is additionally influenced by the menstrual cycle. Because of increased cervical colonization by gram-negative bacilli, the likelihood of infection is greater during the follicular phase of the menstrual cycle.[47]

Several methods have been employed to disinfect the vagina preoperatively. These methods include the use of antiseptic douches, normal saline douches, and the administration of prophylactic antibiotics. Haeri et al.[48] compared douching and the use of suppositories as methods of vaginal preparation in 155 patients. One group of patients used two povidone-iodine douches 24 hr before surgery. Another group of patients utilized povidone-iodine gel and povidone-iodine douches 24 hr before surgery. A third group used 0.05%

chlorhexidene douches twice in 24 hr before surgery. The control group had no vaginal preparation. There was a significant decrease in postoperative febrile morbidity in the povidone-iodine group. There was a reduction in the postoperative vaginal bacterial colony count, although the reduction was not statistically significant. The postoperative morbidity in the chlorhexidene group was not reduced below that of the matched control group.[48] This is the only study to date that has evaluated chlorhexidene as a vaginal antiseptic. However, the concentration employed was much weaker than that which is currently available.

The apparent efficacy of povidone-iodine as a vaginal preparation is subject to question on the basis of a study by Blackmore et al.[49] in which there was no demonstrable difference in postoperative febrile morbidity whether povidone-iodine or no preparation was used. The increase in bacterial colonization of the vaginal apex on the fifth postoperative day was similar in both groups. The rapid bacterial regrowth following the vaginal use of povidone-iodine may explain the discrepancy between these studies.[4,50] It seems that this regrowth begins about 4 hr after the iodine application.

Chlorhexidene has been used for antiseptic studies in predelivery and prepregnancy termination patients.[51] When the vagina in the latter group of patients was prepared immediately before surgery with chlorhexidene, almost all bacteria in the vagina were killed 5 min after application of the antiseptic solution.

When using prophylactic antibiotics, the question arises as to the additional need for a vaginal antiseptic. Prior to using antibiotics, the infection rate following vaginal hysterectomy was 35%–55%.[52,53] This was reduced to 4%–13% after the administration of perioperative antibiotics.[54,55] In a study by Amstey and Jones,[56] all patients undergoing vaginal hysterectomy received prophylactic antibiotics and either a normal saline or a povidone-iodine douche. The postoperative febrile morbidity was the same regardless of the douching solution. Because povidone-iodine and possibly chorhexidene seem to offer no antiseptic advantage, and because there is a potential risk of toxicity secondary to transmucosal absorption of iodine,[57] the use of a normal saline douche in conjunction with prophylactic antibiotics is recommended for vaginal surgical procedures.

In summary, then, the efficacious methods of vaginal preparation include the immediate preoperative vaginal use of chlorhexidene and povidone-iodine, the administration of prophylactic antibiotics, and the use of a normal saline douche solution.

References

1. Altemeier WA. Surgical infections: incisional wounds. In: Bennett JV, Brachman PS, eds. Hospital infections. Boston: Little, Brown, 1979: p. 287–306.
2. Green JW, Wengel PP. Postoperative wound infection: a controlled study of the increased duration of hospital stay and direct cost of hospitalization. Ann Surg. 1977; 185:264–268.
3. Cruse PJE, Foord R. A 5-year prospective study of 23,649 surgical wounds. Arch Surg. 1973; 107:206–210.
4. Pollock AV, Evans M. The prophylaxis of surgical wound sepsis with cephaloridene—experiences in 2491 general surgical operations and reporting a controlled clinical trial against framycetin. J Antimicrob Chemother. 1975; 1(Suppl.):71–83.
5. Marples MJ. Life in the human skin. Sci Amer. 1969; 220:108–115.
6. Ohm MJ, Galask RP: Bacterial flora of cervix from 100 post-hysterectomy patients. Am J Obstet Gynecol. 1975; 122:683–687.
7. Wyatt B, Goldacre MJ, Loudon N, et al. Prevalence of bacteria in the vagina of normal young women. Br J Obstet Gynecol. 1981; 88:588–595.
8. Neary MP, Allen J, Okubedejo OA, et al. Preoperative vaginal bacteria and postoperative infections in gynecological patients. Lancet. 1973; 2:1291–1294.
9. Robson MC, Krizek TJ, Heggers JP. Biology of surgical infection. In: Current Problems in Surgery. Chicago, Yearbook Medical, 1973: p. 1–62.
10. Howard JM, Barker WF, Culbertson WR, et al. Postoperative wound infections: the influence of ultraviolet irradiation of the operating room and various other factors. Am Surg. 1964; 160 (Suppl. 2):1–192.
11. Altemeier WA, Todd J. Studies on the incidence of infection following open chest massage for cardiac arrest. Ann Surg. 1963; 158:596–607.
12. Center for Disease Control. National Nosocomial Infections Study report. Washington, D.C.:

Government Printing Office, 1976; 7. [DHEW Publication no. (CDC) 78-8257].
13. Percora DV, Landis RE, Martin E. Location of cutaneous microorganisms. Surgery. 1968; 64:1114–1118.
14. Hamilton HW, Hamilton KR, Lone FJ. Preoperative hair removal. Can J Surg. 1977; 20:269–275.
15. Seropian R, Reynolds BM. Wound Infection after preoperative depilatory versus razor preparation. Am J Surg. 1971; 121:251–254.
16. Balthazar ER, Colt JD, Nichols RL. Preoperative hair removal: a random prospective study of shaving versus clipping. South Med J. 1982; 75:799–801.
17. Court-Brown CM. Preoperative skin depilation and its effects on postoperative wound infections. J R Coll Surg Edinb. 1981; 26:238–241.
18. de Koos PT, McComas B. Shaving versus skin depilatory cream for preoperative skin preparation. A prospective study of wound infection rates. Am J Surg. 1983; 145:377–378.
19. Vestal PW. Preoperative preparation of skin with depilatory cream and detergent. Am J Surg. 1952; 83:398–402.
20. Prigot A, Garnes AL, Nwagbo LL. Evaluation of a chemical depilatory for preoperative preparation of five hundred fifteen surgical patients. Am J Surg. 1962; 104:900–906.
21. White JJ, Wallace CK, Burnett LS. Drug letter, skin disinfection. Johns Hopkins Med J. 1970; 126:169.
22. Butcher HR, Ballinger WF, Gravens DL, et al. Hexachlorophene concentrations in the blood of operating room personnel. Arch Surg. 1973; 107:73.
23. Feldmann RJ, Maibach HI. Absorption of some organic compounds through the skin in man. J Invest Dermatol. 1970; 54:399–404.
24. Check W. New study shows hexachlorophene is teratogenic in humans. JAMA. 1978; 240:513–514.
25. Janerich DT. Environmental causes of birth defects; the hexachlorophene issue. JAMA. 1979; 241:830–831.
26. Frank MJ, Schaffner W. Contaminated aqueous benzalkonium chloride: an unnecessary hospital infection hazard. JAMA. 1976; 236:2418–2419.
27. Kaslow RA, Mackel DC, Mallison GF. Nosocomial pseudobacteremia. Positive blood cultures due to contaminated benzalkonium antiseptic. JAMA. 1976; 236:2407–2409.
28. Plotkin SA, Austrian R. Bacteremia caused by *Pseudomonas* species following the use of materials stored in solutions of a cationic surface-active agent. Am J Med Sci. 1958; 235:621–627.
29. Food and Drug Administration. OTC topical antimicrobial products and drug and cosmetic products. Fed Reg. 1974; 39:33102.
30. Case DE, McAinsh J, Rushton A, et al. Chlorhexidene: attempts to detect percutaneous absorption in man. In: Chemotherapy. Williams JD, Geddes AM, eds. (Vol. III). New York: Plenum Publishing, 1979: p. 367–374.
31. Kothuis BJL. The effect of povidone-iodine on postoperative wound infection in abdominal surgery. Neth J Surg. 1981; 33:186–189.
32. Kaul AF, Jewett JF. Agents and techniques for disinfection of the skin. Surg Gynecol Obstet. 1981; 152:677–685.
33. Knudsin RB, Walter CW. The surgical scrub: practical consideration. Arch Surg. 1973; 107:75–77.
34. Smylie HG, Logie JR, Smith G. From Physohex to Hibiscrub. Br Med J. 1973; 4:586–589.
35. Peterson AF. Comparative evaluation of surgical scrub preparations. Surg Gyn Obstet. 1978; 146:63–65.
36. Berry AR, Watt B, Goldacre MJ, et al. A comparison of the use of povidone-iodine and chlorhexidene in the prophylaxis of postoperative wound infection. J Hosp Infect. 1982; 2:55–63.
37. Dineen P. An evaluation of the duration of the surgical scrub. Surg Gynecol Obstet. 1969; 129:1181–1184.
38. Cruse PJ, Foord R. The epidemiology of wound infection. A 10-year prospective study of 62,939 wound. Surg Clin N Am. 1980; 60:27–40.
39. Decker LA, Gross A, Miller FC, et al. A rapid method for the presurgical cleansing of hands. Obstet Gynecol. 1978; 51:115–117.
40. Cutright DE, Bhaskar SN, Gross A, et al. A new method for presurgical hand cleansing. Oral Surg. 1972; 33:162–167.
41. Gross A, Selting WJ, Cutright DE, et al. Evaluation of two antiseptic agents in surgical preparation of hands by a new method. Am J Surg. 1973; 126:49–52.
42. Galle PC, Homesley HD, Rhyne AL. Reassessment of the surgical scrub. Surg Gynecol Obstet. 1978; 147:215–218.
43. Maxwell JG, Ford CR, Peterson DE, et al. Abdominal wound infections and plastic drape protectors. Am J Surg. 1969; 118:844–848.
44. Jackson DW, Pollock AV, Tindal DS: The value of a plastic adhesive drape in the prevention of wound infection. Br J Surg. 1971; 58:340–342.
45. George JW, Ansbacher R, Otterson WN, Rabey F. Prospective bacteriologic study of women undergoing hysterectomy. Obstet Gynecol. 1975; 45:60–63.
46. Bonnar J, Bruce LC, Low RA. Noxytiolin (Gynaflex) in the preoperative preparation of

the vagina. J Obstet Gynecol Br Commonw. 1969; 76:850–852.
47. Thadepalli H, Savage EW, Salem FA, et al. Cyclic changes in cervical microflora and their effect on infections following hysterectomy. Gynecol Obstet Invest. 1982; 14:176–183.
48. Haeri AD, Klopper LL, Forder AA, et al. Effect of different pre-operative vaginal preparations on morbidity of patients undergoing abdominal hysterectomy, S Afr Med J. 1976; 50:1984–1986.
49. Blackmore MA, Turner GM, Adams MR, et al. The effect of pre-operative povidone-iodine vaginal pessaries on vault infections after hysterectomy. Br J Obstet Gynecol. 1981; 88:308–313.
50. Monif GRG, Thompson JL, Stephens HD, et al. Quantitative and qualitative effects of povidone-iodine liquid and gel on the aerobic and anaerobic flora of the female genital tract. Am J Obstet Gynecol. 1980; 137:432–438.
51. Vorherr H, Ulrich JA, Messer RH, et al. Antimicrobial effect of chlorhexidene on bacteria of groin, perineum, and vagina. J Reprod Med. 1980; 24:153–157.
52. Johnson CG. Vaginal hysterectomy and vaginectomy in personal retrospect. Am J Obstet Gynecol. 1969; 105:14–19.
53. White SC, Wartel LJ, Wade ME. Comparison of abdominal and vaginal hysterectomies. A review of 600 operations. Obstet Gynecol. 1971; 37:530–537.
54. Allen JL, Rampone JF, Wheeless CR: Use of prophylactic antibiotic in elective major gynecologic operations. Obstet Gynecol. 1972; 39:218–224.
55. Harralson JB, van Nagell JR Jr, Roddick JW Jr, et al. The effect of prophylactic antibiotics on pelvic infection following vaginal hysterectomy. Am J Obstet Gynecol. 1974; 120:1046–1049.
56. Amstey MS, Jones AP. Preparation of the vagina for surgery—a comparison of povidone-iodine and saline solution. JAMA. 1981; 245:839–841.
57. Vorherr H, Vorherr UF, Mehta P, et al. Vaginal absorption of povidone-iodine. JAMA. 1980; 244:2628–2629.

Mechanisms of Wound Healing, Suture Material, and Wound Closure

5

Luis Sanz and Samuel Smith

Introduction

The selection of the proper incision, suture material, and closure technique is very important to assist the patient's own repair mechanism and restore normal anatomic relationships after surgery. Attention to these details also prevents such complications as dehiscence and infection, assuring a good cosmetic result. A good understanding of the physiologic process of wound healing is needed to properly select the suture material and closure technique and consequently markedly decrease morbidity and mortality. We discuss the pathophysiology of wound healing, properties of suture materials, and techniques of wound closure.

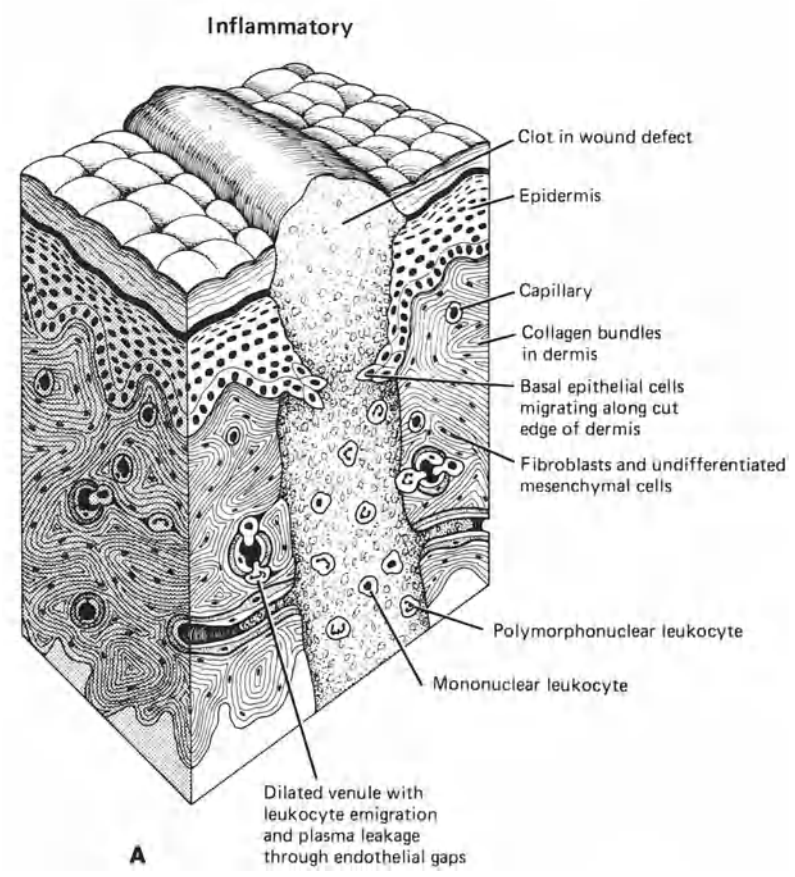
Pathophysiology

Four phases have been described in the healing process: inflammatory, migratory, proliferative or fibroplastic, and maturative.[4] Occurring concomitantly with the migratory phase are the processes of epithelialization and neovascularization[1,2] (Figure 5-1). Each of these phases is described.

Inflammatory Phase

Inflammation is the initial physiologic response to tissue trauma. When tissue is surgically incised, injury occurs on the cellular, tissue, and organ levels. Cells are damaged, vessels are injured, and numerous vascular and tissue factors interact in the area of injury. After a transient vasoconstriction, injured vessels thrombose and patent local small vessels dilate.[1,2,3] Platelets interact with damaged endothelium and exposed collagen to plug the vessel. The complement system is activated and initiates an inflammatory cascade involving the local coagulation, fibrinolytic, and kallikrein systems. Local deposition and lysis of fibrin occurs, and numerous inflammatory mediators are released, notably bradykinin, free radicals, hydrogen peroxide, and histamine. In addition, the complement system directly stimulates chemotactic response by granulocytes and promotes mast cell degranulation and histamine release. The result of these processes is an inflammatory response characterized by cellular autolysis, local vasodilatation, abnormal capillary permeability, and white blood cell migration into the area of injury. Proteins and plasma extravasate from injured vessels, while platelets stick to the injured endothelial lining and white blood cells migrate between endothelial cells. The injured tissue quickly fills with granulocytes, macrophages, erythrocytes, soluble plasma proteins, and fibrin. The magnitude of the inflammatory response generally reflects the magnitude of tissue injury.[1,2,3]

Granulocytes and monocytes are the predominant white blood cells involved in the inflammatory response. They are actively motile, migrate into the injured tissue, and actively phagocytize cellular debris and bacteria. Initially the polymorphonuclear cells predominate in the wound. However, as migration of



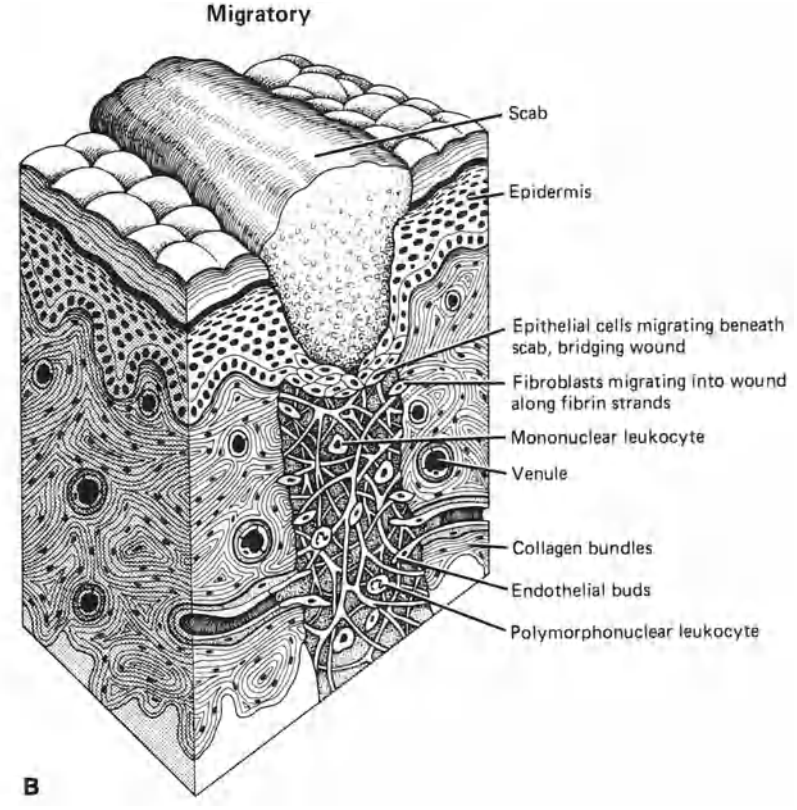
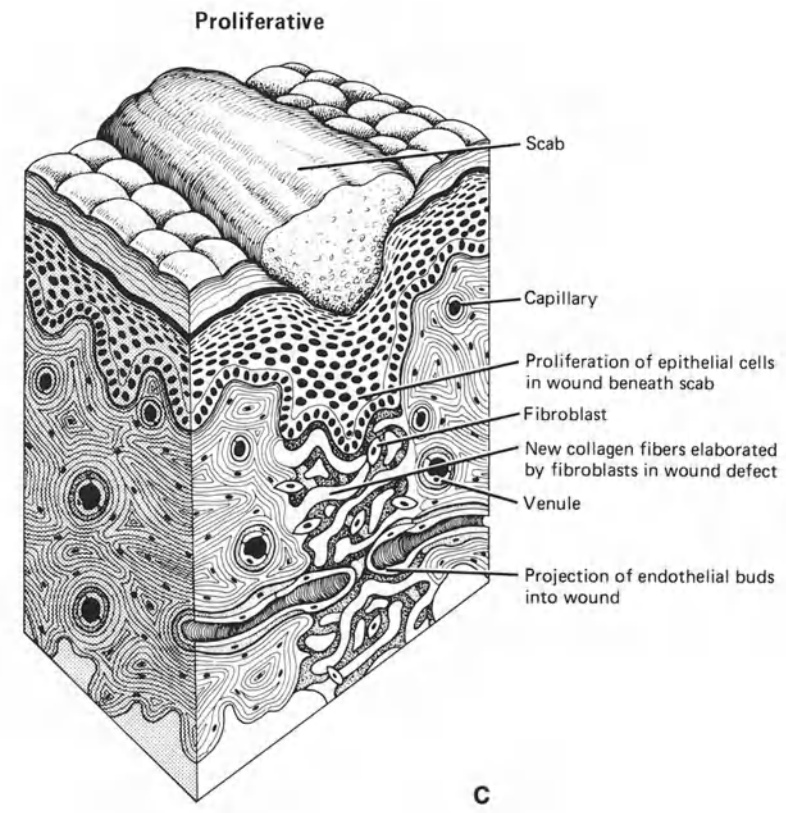
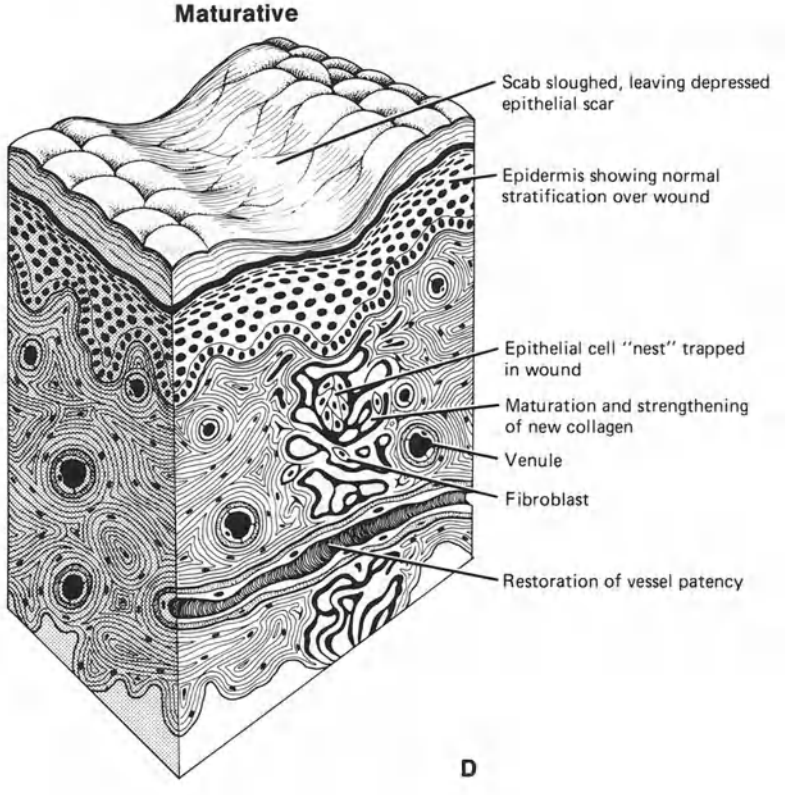


Figure 5-1. Four phases in the healing process. **A,** Inflammatory. **B,** Migratory. **C,** Proliferative. **D,** Maturation. Illustrations by Michael Reinhold. Figures A and D adapted from original paintings by John C. Craig, M.D., from Clinical Symposia, copyright © CIBA Pharmaceutical Co., Division of CIBA-GEIGY CORP., Summit, New Jersey.


Proliferative
Scab
Capillary
Proliferation of epithelial cells in wound beneath scab
Fibroblast
New collagen fibers elaborated by fibroblasts in wound defect
Venule
Projection of endothelial buds into wound
C


Maturative
Scab sloughed, leaving depressed epithelial scar
Epidermis showing normal stratification over wound
Epithelial cell "nest" trapped in wound
Maturation and strengthening of new collagen
Venule
Fibroblast
Restoration of vessel patency
D

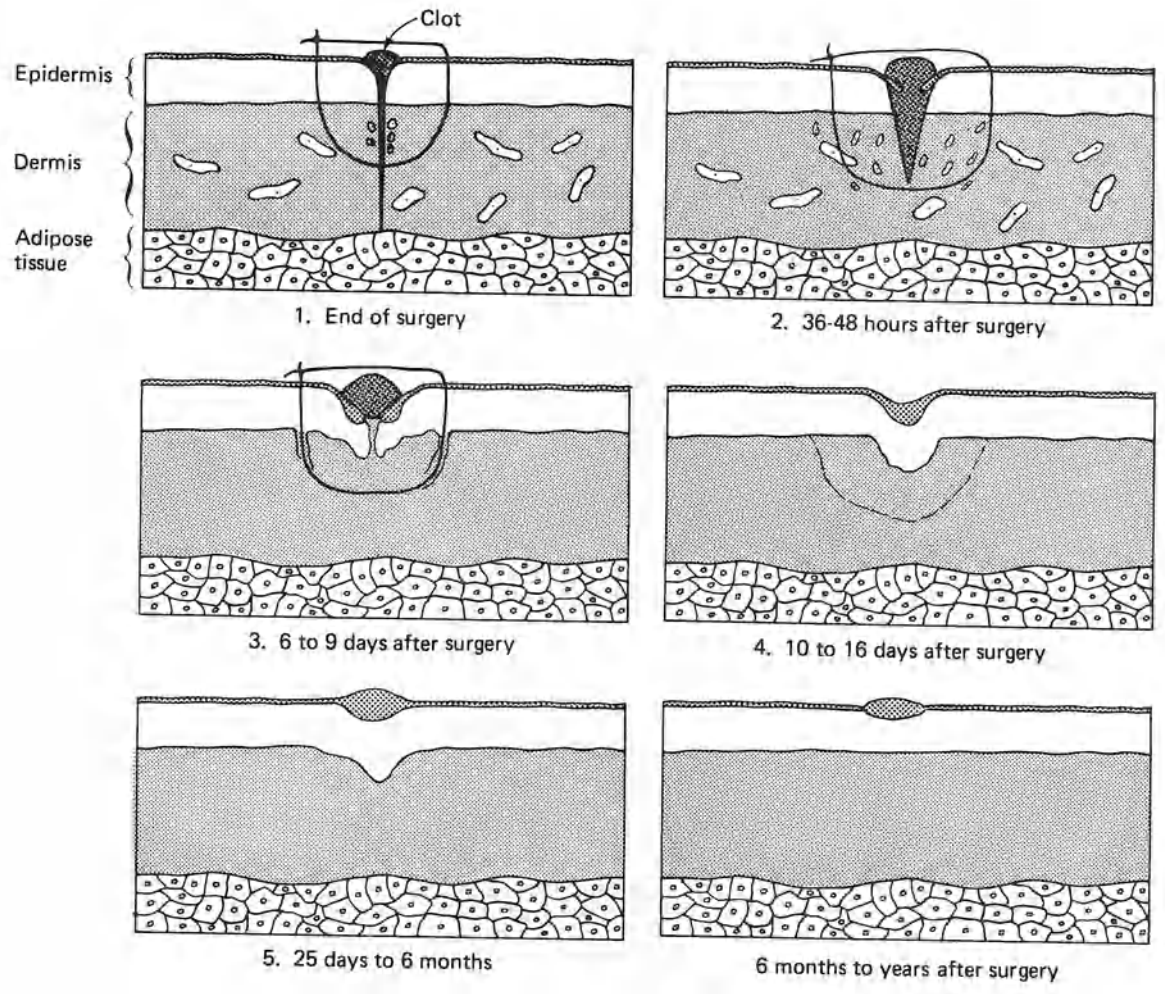


Figure 5-2. Epithelialization.

white blood cells into the wound subsides, the proportion of longer lived monocytes increases as the short-lived granulocytes die.[1,2,3]

Epithelialization

Important events occur at the wound edges simultaneously with the deeper inflammatory response. The epidermis immediately adjacent to the surgical wound edge begins thickening within 24–48 hr of injury[2] (Figure 5-2). Basal cells at the wound edge flatten and migrate toward the defect, eventually covering the defect. Some of these cells begin to reassume their cuboidal shape, and after the layer of epithelial cells is complete, mitotic division produces epithelial thickening. The healing epithelium, however, is thinner, less pigmented, and has less developed rete pegs than normal skin epithelium (Figure 5-2).

Squamous cells use a collagenase to cleave tissue ahead of them, facilitating their advance through the wound.[1,2] Epithelial cells advance in a fashion so as to undermine the eschar associated with the tissue defect. Epithelialization proceeds most efficiently in wounds where there is minimal eschar development.[1,2]

Skin sutures create a microwound into which epithelial cells migrate. Sometimes a plug of keratinized epithelium will be trapped in the suture tract, and small cysts may occur at the side of the mature wound. An epithelialized suture may leave an unsightly scar; using skin tapes instead of sutures or skin staples helps reduce this occurrence, as does early suture or

staple removal and replacement with skin tapes.[1,2]

Almost invariably large open wounds are covered by a blood clot. The migrating epithelial cells move under the clot, maintaining direct contact with the original wound bed. Proteolytic enzymes dissolve the base of the clot and facilitate epithelial cell migration. As epithelialization progresses, the undermined scar separates from the healing wound.[1] In large open wounds, epithelialization proceeds over a large bed of granulation tissue. Numerous projections of epithelial cells invade the newly formed connective tissue, creating pseudo rete pegs. These rete pegs do not persist; rather, they regress resulting in a relatively straight dermal-epithelial border. By 30 to 40 days, the surface of the reepithelialized wound is covered by scar epithelium that is characteristically thinner and more fragile than normal skin epithelium. Microscopically the scar epithelium lacks the rete pegs and is easily differentiated from normal epidermis.[1] The final scar is much narrower than the original defect, due to a process called contraction. Wound contraction is discussed shortly.

Neovascularization

Neovascularization is an important aspect of wound repair. The effective tissue circulation has been disrupted by surgical injury. Moreover, the added number of metabolizing inflammatory cells decreases the adequacy of the remaining circulation by increasing local oxygen and nutrient demand.[1,2] The local energy deficit is marked, emphasizing the necessary restoration of an effective microcirculation. New vessels must replace and supplement the old injured system. Neovascularization of wounds facilitates a continued nutrient supply to the healing tissue.

It is recognized that new vessels always arise from existing vessels and that new vessels begin as capillary buds.[1] Angiogenesis occurs in three ways. First is the formation of an entirely new vascular system in an open wound healing by secondary intention. Second, wound vessels may join with an unused circulation; this is typified by skin grafting in which the host bed provides circulation to the graft. Third, vessels may join across wounds that are healing by first intention.[1,32]

New vascular networks regenerate in secondary healing wounds.[1,2,3] Initially, endothelial cells sprout toward the wound edge from functioning vessels near the wound. Presumably, biochemical signals from platelets and macrophages initiate the sprouting process. Functional capillary loops form as capillary buds from various vessels join together, and circulation is reestablished. However, the basement membrane of these new vessels is incomplete, and the vessels are fragile and leaky. Capillary endothelial cells secrete a collagenase, which facilitates their movement through the collagenous gel that is produced by fibroblasts. Vessels formed in this fashion either participate in the formation of larger vessels or stop functioning and disappear.[1,2,3]

In primary repair, circulation can be reestablished and bridge the wound within 2–3 days. The close approximation of wound edges facilitates this. Larger thrombosed vessels may be recanalized by fibrinolytic enzymes; those enzymes also promote passage of red blood cells through the fibrin mesh. Endothelial cells apparently follow the course of these red blood cells.[1,2,3] Experimental work involving neovascularization following skin grafting demonstrates that the old vessels of the skin graft initially fill with red blood cells and later reestablish a functional vascular network.[1]

Migratory Phase

The inflammatory reaction ordinarily peaks and begins to subside after 2–3 days. At this time there is a change in character of the cells within the wound. Macrophages and fibroblasts replace granulocytes.[1,2] The most striking manifestation of this time period is the appearance of lush sprouts of very actively growing, tiny, fragile blood vessels and connective tissue cells. Granulation tissue is the aggregate of this fibroblastic and angioblastic proliferation; these processes result in bridging the gap where tissue is disrupted.[3] Fibrin that has been formed in the initial inflammatory exudate is invaded by granulation tissue.[2,3] Concurrently, or shortly thereafter, epithelial cells rapidly proliferate and overgrow the wound area. If healing proceeds normally, epithelialization is complete within a week; granulation tissue is receding and evidence of new collagen synthesis is visible.[1,2,3]

Macrophages are primarily responsible for digesting devitalized and necrotic tissue, removing foreign body particles, and attracting fibroblasts into the wound. Experimental work utilizing antiserum to macrophages has demonstrated that macrophages are necessary for proper wound healing.[1] Without macrophages, injured cells and tissues are not properly debrided and wound strength is poor. Macrophages appear to release substances involved in macrophage migration, fibroblast proliferation, and capillary budding. Similar studies have shown that neutrophils and lymphocytes are not necessary for wound healing.[1,2]

Undifferentiated mesenchymal cells begin to differentiate into migratory fibroblasts shortly after injury is incurred. Fibroblasts rapidly migrate into the space left after granulocytes and macrophages clear necrotic tissue, clot, and debris. Early in the inflammatory phase fibrin is deposited and acts as a hemostatic barrier and as a framework for the elements of repair.[1,2]

Migrating fibroblasts use the fibrin strands as a scaffold. It is uncertain, however, whether the fibrin strands provide active orientation or contact guidance. In fact, the large amount of fibrin may inhibit fibroblast and epithelial cell migration by obstructing migration mechanically. Fibroblasts do not contain fibrinolytic enzymes; these enzymes and plasminogen activator, instrumental in removal of fibrin, are contained in the capillary buds.[1,2]

Extensive hematomas, necrotic tissue, and bacteria block fibroblast migration. In general, however, debris is removed by the third to fifth day, by which time a strong fibroblastic and angioplastic response has occurred.

Fibroblast proliferation occurs simultaneously with migration. Fibroblasts initially synthesize and secrete components of ground substance, protein polysaccharides, and various glycoproteins.[1,2] Approximately 4 days after injury, *collagen synthesis of fibroplasia* begins.

Fibroplasia (Proliferative Phase)

During the stage of fibroplasia the character of cells in the wound remains constant.[8] Collagen is rapidly synthesized and deposited. Epithelium has already migrated across the granulating wound. These epithelial cells are known to secrete collagenase, which helps modify the collagen content of the wound. Fibroblasts that communicate with the scar epithelium also secrete collagenase.[1,2]

Fibroplasia lasts approximately 2–4 weeks. At the end of this phase much of the neovascular network has regressed. The number of fibroblasts, the glycoprotein and mucopolysaccharide content of the wound, and the rate of collagen synthesis all decrease. This stage is succeeded by the maturative phase of wound healing.

Collagen. Collagen is responsible for wound tensile strength. Wounds gain tensile strength in a particular fashion (Figure 5-3). The first 4–5 days after injury is called the lag phase, during which there is no appreciable gain in tensile strength. Subsequently, tensile strength rapidly increases, with maximal slope of the curve seen approximately 15 days postoperative (Figure 5-3). The period of fibroplasia is characterized by rapid gain in wound strength.[1,2]

Skin and fascia continue to gain strength after the period of fibroplasia. The rate of gain diminishes rapidly but gain in wound strength can occur 1 year postoperative. Importantly, however, even after 1 year skin wounds are 15%–20% weaker than surrounding normal tissue.[2,8] Increasing tensile strength during the period of fibroplasia parallels the rise in collagen content of the wound. Gain in tensile strength during the maturative phase is largely due to cross-linking of existing collagen fibers.[1,2]

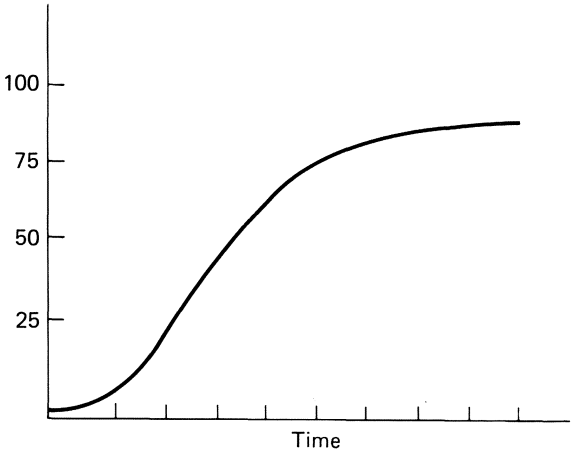


Figure 5-3. Change in wound tensile strength over time.

Elastin is the second major fibrous component of connective tissue. It is not replaced during wound healing, and it has no role in wound repair.[2]

Collagen Chemistry. Collagen is actually a group of glycoproteins with two common properties.[1,2] First, they are composed of three separate linear peptide chains, each chain approximately 1,000 amino acids long, termed alpha chains. Glycine residues occupy every third position in the alpha chains. One-third of the amino acids are either proline or hydroxyproline. Quantification of hydroxyproline correlates well with the collagen content of a tissue because the amino acid is virtually unique to collagen. Hydroxylysine is another amino acid virtually unique to collagen[1,2] (Figure 5-4).

Second, each alpha chain is twisted into a right-handed helix. Three alpha chains are aligned in parallel and twisted into a left-handed superhelix. This compound helical tertiary structure is unique to collagen; it accounts for collagen's rigidity despite its long length and narrow width (3,000 Å by 15 Å, respectively).

Four types of collagen have been identified by submolecular structural analysis (Table 5-1). Type I collagen, the largest component of skin- and tendon-derived collagen, consists of three alpha (α) chains, two of which are identical.[1,2,8] By convention the identical chains are called α_1 and the dissimilar chain α_2. Furthermore, two alpha chains may be covalently linked into a unit called the beta (β) chain, or three alpha chains may be covalently linked together into a gamma (γ) chain.[1,2]

Type II collagen is only found in cartilage. It has three identical alpha chains that are structurally similar to the α_1 chains of Type I collagen. Type III collagen contains three identical alpha chains that are similar to the alpha chains found in type I and II collagen. Type III collagen is found in small amounts in some adult tissue but mainly in embryonic connective tissue. Some type III collagen is laid down in skin wounds but is replaced by type I collagen as the wound matures. Type IV collagen is limited to basement membranes.[1,2]

Within the fibroblast, alpha chain synthesis proceeds similarly to other protein synthesis with the addition of a unique hydroxylation reaction (Figure 5-4). No transfer RNA exists to incorporate hydroxyproline or hydroxylysine into collagen. Instead, proline and lysine residues are incorporated into the alpha chains and are subsequently hydroxylated.[1,2] Ascorbic acid is an important cofactor for this hydroxyl-

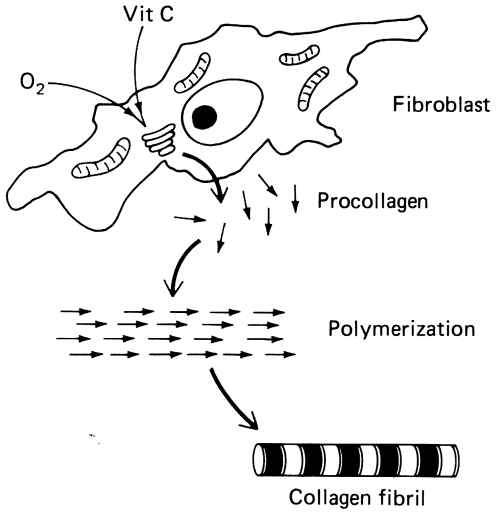


Figure 5-4. Collagen structure.

Table 5-1. Structurally and Genetically Distinct Collagens[a]

Type	Tissue distribution	Characteristics
I	Bone, tendon, skin, dentin, ligament, fascia, arteries, and uterus	Most prevalent form of collagen in the mature vertebrate organism; composed of two chain types
II	Hyaline cartilages	Relatively high content of hydroxylysine and glycosylated hydroxylysine
III	Skin, arteries, and uterus	High content of hydroxyproline; contains interchain disulfide bonds
IV	Basement membranes	High content of hydroxylysine and glycosylated hydroxylysine; contains interchain disulfide bonds and may contain large globular regions

[a] Modified from Hunt T, Dunphy JE[1].

ation step; it is required by the enzymes prolyl and lysyl hydroxylase. Without ascorbic acid, collagen is underhydroxylated, and characteristic collagen fibers fail to form. Scurvy, which results from ascorbic acid deficiency, is characterized by poor wound healing and capillary fragility.[1,2]

The polypeptide backbone of collagen can be constructed with energy derived from anaerobic glycolysis. However, adequate oxygenation is necessary for hydroxylation of proline and lysine residues. Without oxygen, collagen production and wound healing are compromised.[1,2]

The collagen molecule resulting from the alpha chain superhelix is intracellularly transported to the Golgi apparatus for glycosylation. Galactose residues are added to form the procollagen molecule, which is secreted by the fibroblast. The terminal peptides of procollagen molecules, called the registration peptides, facilitate the parallel assembly of alpha chains and the superhelical configuration. They are cleaved before the collagen molecules aggregate into fibers, forming the molecule tropocollagen.[1,2]

Tropocollagen molecules aggregate together to form fibrils. Each tropocollagen unit is displaced from adjacent tropocollagen molecules by one-fourth of its length. This quarter-staggered arrangement results in the characteristic banding patterns seen on electron microscopy. Later, cross-linking occurs between lysine residues of adjacent tropocollagen molecules, and significantly stronger collagen fibrils result.[1,2]

Maturation Phase

New collagen can be observed in healing wounds on the second postoperative day.[1,2,8] This early collagen exists essentially in a gel state,[1] and is very disorganized and weak compared to collagen that develops later in the healing process[1,2] (Figures 5-1 and 5-2). Rapid collagen synthesis occurs in the first months after injury. A balance exists between collagen synthesis and lysis that is important in scar remodeling and maturation. Early in wound repair, collagen lysis occurs in association with removal of noncollagenous protein and debris. Macrophages are largely responsible for this early lysis and removal. As repair continues, collagen lysis continues.

As the wound matures collagen synthesis and deposition changes. The vascular system has had time to regenerate, and tissue oxygenation is significantly improved.[1,2] The wound is also subjected to physical stresses and strains that serve to increase wound tensile strength by encouraging fibroblasts and collagen to line up along lines of tension. Wounds are stronger if slight stress is applied to the wound during the maturation phase than if the wound is protected from these forces.[1,2]

Several factors contribute to scar maturation. Intramolecular and intermolecular cross-linkages increase the tensile strength of collagen and stabilize the scar tissue by increasing the insolubility of collagen. Although collagen fibers become increasingly oriented along lines of tension during maturation, collagen fibers in scar tissue are more disorganized and clearly different in arrangement than the collagen fibers in nearby healthy tissue. Cutaneous scars are always weaker than surrounding normal skin, and are also more rigid due to the deficiency of elastin in scar tissue.[1,2]

Scar remodeling is associated with continued deposition of new collagen fibers and lysis of older fibers. Newly formed collagen attaches to older collagen and promotes continued wound approximation. Fibers are also packed more closely as the scar matures, facilitating intermolecular cross-linkage, insolubility, and resistance to enzymatic degradation. Interweaving and cross-linkage of older and new collagen fibers increase the wound's strength.[1,2] There are still points of weakness in the scar: The incision line is initially the point of maximal weakness, but after 2 weeks of healing, the junction between normal tissue collagen and scar collagen becomes the point of maximal weakness.[1,2]

As maturation proceeds, water and mucopolysaccharides are lost from the wound. Collagen fibers are compacted together, cross-linkage between molecules increases, and the wound scar assumes a dense white appearance.[1,2]

Wound Contraction

Wound contraction is the movement of full-thickness skin towards the center of the skin defect. It is an active process characterized by wound shrinkage and the drawing in of normal skin. Contraction is the process by which

large open wounds become smaller during second-intention healing; it is different from contracture, the loss of joint motion associated with scarring.[1,2] The force for contraction comes from special contractile cells of the wound, called myofibroblasts, that have attributes of both the fibroblast and the smooth muscle cell. Wound contraction, however, is not dependent on collagen.[1,2]

Suture Material

Historically, many materials have been used to approximate wounds. Early medical practitioners recognized that bleeding must be controlled and wounds secured for proper healing to occur.[34] Needles were used to pass suture material through traumatic wounds.[34] These materials included linen strips, animal sinews, flax, hemp, and grasses.[10,30] There was little change in suture material until the Renaissance,[10] and natural products sufficed as suture material until the 1940s. These included silk, cotton, linen-cotton, and catgut. During the 1940s, nylon and polyester were developed and became the first synthetic suture materials. Later polyethylene, polypropylene, polyglycolic acid, and polyglactin were developed, expanding the surgeon's choice of suture material.[2,10,34] For many years, however, silk and catgut were the mainstay suture materials.

Properties of Suture Materials

A surgeon must be familiar with the various available suture materials. The perfect suture material should have excellent handling qualities. It should be flexible, pliable, and easy to tie with good knot security. It should have lasting tensile strength, be nonallergenic, have good resistance to infection, and have predictable reabsorption after wound healing is complete.[34]

The described properties of suture material are knot security, tensile strength, tissue reaction, and wound security.[9,10] Surgeons must be familiar with these terms. Knot security describes a suture material's ability to maintain knot strength without slippage.[10] Square knots must be used when comparing the knot security of various materials.[10] Knot security is inversely proportional to memory of a suture material and directly proportional to its workability.[10] Suture materials with high memory are less pliable, tend to maintain their original shape, and frequently handle with difficulty.[10] They are more likely to untie or slip when placed under tension.

Tensile strength, used to describe suture material or the healing tissue, is the strength per unit area.[10] Tensile strength is a constant derived by dividing the force necessary to break a strand of material by the cross-sectional area of the material.[10] Breaking strength, in contrast, involves measurement of strength per unit width, and differs from tensile strength.[10] Skin and fascia are the tissues with greatest tensile strength.[10]

Tissue reaction is an important consideration in selecting a suture material. Two to seven days after suture placement, there is a marked and maximal tissue reaction around the suture that occurs to some degree with all suture materials studied.[9,10] The inflammation is characterized by an early infiltration of granulocytes, lymphocytes, and monocytes. By the fourth day monocytes, macrophages, and fibroblasts predominate.[2,9,10] Inflammation is always present for 7 days after suture placement regardless of suture material.[9,10,20] It is the subsequent chronic inflammatory reaction, however, that is dependent on the particular suture material chosen. In general, monofilament sutures incite less tissue reaction than multifilament sutures, and uncoated suture materials cause less tissue reaction than coated materials. Synthetic suture materials generally cause much less tissue inflammation than do natural suture materials.[2,9,10]

With respect to infection, the chemical nature of the suture seems to be more important than the physical configuration. The reabsorption of catgut in infected tissue is very fast. It has been demonstrated that the interstices present in multifilament sutures provide sites for bacterial infection to occur[9,20] (Figure 5-5).

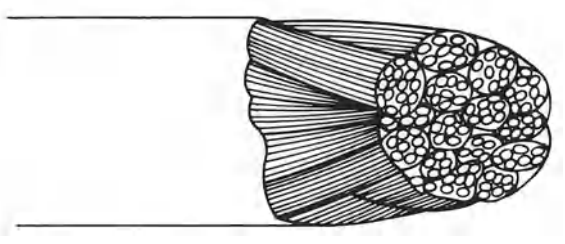


Figure 5-5. Braided suture.

Table 5-2. Classification of Suture Material

	Generic name	Raw material	Trade names
I. *Absorbable*			
Natural Collagen	Plain catgut	submucosa of sheep intestine	—
	Chromic catgut	+buffered chromicizing	—
Synthetics	Polyglycolic acid	homopolymer of glycolide	Dexon, Dexon-S
		+poloxamer 188 coating	Dexon-Plus
	Polyglactin-910	copolymer lactic and glycolic acid	Vicryl
		+calcium stearate coating	Coated-Vicryl
	Polydioxanone	monofilament	PDS
II. *Nonabsorbable*			
Natural fiber	Surgical cotton	twisted natural cotton	—
	Surgical silk	braided protein natural spun by silk worm	—
Synthetic	Nylon	polyamide polymer	—
		monofilament	Dermalon, Ethilon
		multifilament	Neurolon
		multifilament-silicone treated	Surgilon
	Polypropylene	polymer of polypropylene	—
		monofilament	Surgilene, Prolene
	Polybutester		NovaFil
	Polyethylene	thermoplastic synthetic resin	Dermalene
	Polyester	polyethylene terephthalate-multifilament	
		braided-plain	Dacron, Mersilene
		braided-silicone treated	Ti-Cron
		braided-polybutilate coated	Ethibond
		braided-PFTE (Teflon) coated	Polydek, Ethiflex
		braided-heavy PFTE (Teflon) impregnated	Tevdek
Metal	Stainless-steel wire	ferrous alloy	—
		twisted multistrand	Flexon
		monofilament strand	—
	Silver wire	silver wire	—

When sutures must be placed in contaminated or infected tissues, monofilament synthetic nonabsorbable sutures are recommended.

Wound security describes the strength imparted to the wound by the suture material.[10] It reflects the process of wound healing as well as the original tensile strength and absorbability of the suture material.[10] It also reflects the loss of tensile strength over time experienced with absorbable suture materials.[9,10]

Classification of Suture Material

The United States Pharmacopeia (USP) is the official compendium providing description of suture materials.[9] The USP also determines the standards and guidelines for the manufacture, packaging, labeling, and sterilization of suture materials.[9]

The USP classifies suture materials into two groups, the absorbable and the nonabsorbable.[9,10] In general, absorbable suture loses tensile strength in less than 60 days, and nonabsorbable suture maintains tensile strength for greater than 60 days.[10] These may be further subdivided into natural and synthetic and into monofilament and multifilament materials.[2,9,10,34] Table 5-2 describes the current USP classification.

Absorbable Sutures. *Catgut.* Catgut, historically the most popular suture material, is derived from the submucosa of sheep intestines.[2,10] The jejunum and ileum of these animals are sliced into longitudinal ribbons and undesired tissue layers are removed by mechanical and chemical processes. The resultant ribbons are chemically treated with a dilute formaldehyde solution, which blocks hydroxyl and amino groups on collagen (the major component of catgut suture), to increase strength and promote resistance to enzymatic lysis. Two or more ribbons are twisted together to develop the desired caliber suture, and the twisted strands are dried under tension. Once dry, the suture is polished, cut into designated lengths, attached to needles, and packaged and sterilized by Cobalt-60 irradiation.[2,10]

Catgut maintains significant tensile strength for approximately 4–5 days.[10,26] Wound security is essentially absent in 14 days.[10,26] Catgut also elicits the greatest tissue reaction of all absorbable sutures[9,10] (Table 5-3). Therefore, there is no reason to place catgut suture in abdominal wounds for purposes other than to secure hemostasis. Suturing with catgut mimics introducing a foreign body into the wound. It provides no additional wound security after skin sutures are removed.[10]

Chromic catgut is almost as old as catgut. It is manufactured by treating plain catgut with basic chromium salts,[2,10] which results in a suture with increased tensile strength and increased resistance to tissue absorption[7,9,10] (Table 5-3). Catgut is digested by acid proteases produced by inflammatory cells. Chromic catgut has greater initial tensile strength and slower tissue absorption and some greater wound security as compared to plain catgut. Wound security is maintained for approximately 14 days with chromic catgut but diminishes rapidly thereafter. Chromic gut is more useful than plain gut in approximating tissues, but where wound security is important it should be replaced by more modern suture material. Chromic gut produces a high degree of tissue reaction but less than that produced by plain catgut.[2,9,10,26,34]

Catgut suture can be used on visceral sorosal surfaces, which heal rapidly, and where rapid absorption is advantageous like vaginal mucosa. Catgut should not be used in skin closure, because tissue inflammation may facilitate bacterial superinfection and cosmetic results are frequently unsatisfactory.[10]

Synthetic Absorbable Sutures: Polyglycolic Acid and Polyglactin. Although catgut served surgeons well for many years, its several shortcomings stimulated a deliberate search for a synthetic substance that could be absorbed in human tissues and made into a usable suture material.[23] Desired properties included slower absorption and less tissue reaction than catgut with acceptable handling, knotting, and tensile strength characteristics. It had to withstand sterilization without changing its physical and biologic properties.[23]

Polyglycolic acid suture, marketed today as Dexon, and polyglactin suture, marketed as Vicryl, were released for general surgical use in 1970 and 1975, respectively.[23] Their commercial release was preceded by a decade of investigation and research.[23]

Table 5-3. The Absorbable Sutures[a]

Type	Knot security	Tensile strength	Wound security
Gut	+	++	5–7 days (50%)
Chromic	++	++	10–14 days (50%)
Dexon[b]	++++	++++	25 days (50%)
Vicryl[c]	+++	++++	30 days (50%)

[a] Modified from Swanson NA, Tromovitch TA.[10]
[b] Polyglycolic acid.
[c] Polyglycan 910.

Polyglycolic acid (Dexon) is a high-molecular-weight, linear copolymer of glycolic acid. It is synthesized as thin filaments, which are then braided into multifilament sutures of varying caliber.[2,34] Polyglactin (Vicryl) is a synthetic copolymer of lactic and glycolic acids. The formed polymer is melted and stretched into fibers that are braided into sutures.[2,23,34] Polyglactin sutures frequently exceed the minimum USP size requirements and are larger than polyglycolic acid sutures on a size-for-size comparison basis.[30]

Polyglycolic acid and polyglactin sutures have virtually identical biologic characteristics. Any differences between these suture materials reported in the primary literature are attributable to differences in investigational methodology, studies of varying tissues, and other variables unrelated to suture material.[9]

Polyglycolic acid and polyglactin do not depend on cellular proteolytic activity for absorption.[23,34] These polymers undergo slow hydrolysis; hydrolysis proceeds more rapidly in alkaline media than in acid or neutral media.[2] Little tissue inflammation accompanies their absorption; the principal cellular reaction to these sutures is some invasion of the interstices by macrophages. The absorption of these sutures is very constant and reliable.[23,34]

Polyglycolic acid and polyglactin undergo identical absorption in animal tissues.[23] There is essentially no absorption in the first 10 days; absorption becomes apparent between 15 and 30 days and is essentially complete in 60–90 days.[23] The absorption rate of catgut is not uniform, and its rate of dissolution is increased in infected tissues.

The initial and early tensile strength of synthetic absorbable sutures is greater than that of catgut.[23,26] Although the initial strength of an absorbable suture is related to its diameter, the rate of loss of tensile strength is also related to local tissue conditions and absorption rate.[2,19]

The synthetic absorbable sutures have an extremely high tensile strength, and retain approximately 55% of the original tensile strength at 14 days and 20% at 20 days.[9,23] Chromic catgut suture retains 34 percent of its original tensile strength after 14 days.[23] However, because polyglycolic acid and polyglactin have such a high initial tensile strength, wound security is maintained for several weeks longer than with catgut suture. Moreover, the synthetic absorbable sutures have greater knot security than catgut. Knot security of catgut is excellent in the dry state but the knots become insecure when exposed to tissue fluid.[21,26] Polyglycolic acid and polyglactin are not influenced by exposure to tissue fluid, and maintain good knot security in vivo.[2,9,10,21,23] A secure knot is one that will hold without slippage until the suture breaks; knot security very closely correlates with a suture material's coefficient of friction.[9] Other factors affecting knot security include the length of cut ends, the type of knot, and surgical technique.

Polyglycolic acid and polyglactin do not handle as well as catgut. The synthetic absorbable sutures have a tendency to slip when the surgeon attempts to tie a square knot.[9,10] However, this problem is much less common if a surgical knot is used. In addition, coated polyglycolic acid and coated polyglactin sutures are commercially available (Dexon-Plus, Coated Vicyrl) that have excellent handling qualities.[9,34] Dexon-S, which is also commercially available, is manufactured with finer filaments and provides improved handling because of its tighter and smoother braid.[34] All of these sutures slip through tissue without braided strands getting caught in the wound margins; this allows them to run through tissue similarly to catgut. The coated sutures have less memory and tie more easily than uncoated polyglycolic acid or polyglactin, thus improving handling properties. Knot security, however, is inferior to polyglycolic and polyglactin with conventional tying technique, and the coated sutures and Dexon-S have an increased tendency towards knot slippage if knots are not accurately thrown. The manufacturer recommends that several additional knots be thrown and that tails be longer than with uncoated synthetic absorbable sutures.[9,34]

Polyglycolic and polyglactin are superior to catgut with respect to tensile strength, tissue reactivity, wound strength, and knot security, and slightly inferior with respect to handling properties.[2,9,10,19,26,34] These newer sutures are preferable in a wound requiring fascial and subcutaneous tensile strength during wound healing, since these sutures maintain tensile

strength and wound security for 4–6 weeks, the period of fibroplasia and early wound maturation. Moreover, because of superior tensile strength, polyglycolic acid and polyglactin sutures of smaller caliber than previously possible with catgut may be used.

Nonabsorbable Sutures. The principal nonabsorbable sutures are silk, cotton, nylon, polyester, polypropylene, and steel. Nylon and steel are manufactured in monfilament and multifilament forms, polypropylene is provided only as a monofilament suture, and the other sutures are available in multifilament form[2,9] (Tables 5-3, 5-4, 5-5, 5-6, and 5-7).

Silk. Silk has been an important suture material for years. It is a suture that handles well and possesses good knot security. Its low memory makes these properties apparent. Silk, however, induces the greatest amount of tissue reaction of any nonabsorbable suture.[2,9,34] Surgical silk is a natural fiber that is classified as a nonabsorbable suture despite its gradual loss of tensile strength in tissue.[2,34] Silk is much stronger than cotton, but is not as strong as comparable calibers of synthetic nonabsorbable sutures. It loses approximately 50% of its tensile strength in 1 year, and there is no residual tensile strength after 2 years.[10] Silk is a slowly absorbable suture, generally disappearing in vivo after 2 years.[9,10]

Silk suture is now used less frequently. Its lack of memory and its good handling accounted for its popularity over the years. However, because of the great amount of associated tissue reaction, the use of silk is discouraged, especially when infection or contamination is present.[2,9] Newer synthetic nonabsorbable suture materials are making silk obsolete.

Nylon. Monofilament nylon (Ethilon, Dermalon) and multifilament, braided nylon (Neurolon) are essentially nonabsorbable sutures made from synthetic polymer fibers. They undergo absorption by slow hydrolysis over an extended period of time.[9,34] Nylon loses approximately 20% of its initial strength after 1 year without further diminution of tensile strength. Nylon sutures have lower tissue reactivity than silk suture and handle easily.[9,34] Its initial tensile strength is very similar to that of silk; that of braided nylon slightly less than that of monofilament nylon. Monofilament nylon, however, possesses the greatest tendency toward knot slippage. Proper surgical technique and additional knots are required because of the high memory of monofilament nylon.[9,34]

There is no clear advantage in using multifilament nylon over monofilament nylon suture. Monofilament nylon clearly has less risk of wound infection than does braided multifilament nylon. Monofilament nylon can also be used more safely than multifilament nylon in contaminated wounds. Monofilament nylon is an excellent suture for skin and fascial closure.[9,10]

Polyester. Dacron polyester is a braided synthetic suture. Its workability, ease of tying, and knot security are similar to that of silk, and it causes less tissue reaction than silk suture.[9,10] Sutures made of polyester are the strongest nonabsorbable synthetic sutures available, superior in tensile strength to nylon, silk, and polypropylene, and second only to stainless-steel sutures.[9] Uncoated polyester suture (Mersilene, Dacron) has excellent knot security. Lightly coated polytetrafluoroethylene (Teflon, PFTE) polyester suture (Polydek, Ethiflex) rank second in knot security and possess better handling properties than uncoated polyester.[9,34] The polytetrafluoroethylene-impregnated polyester sutures (Tevdek) and silicone-coated polyester suture (Ti-cron) have excellent handling characteristics, but demonstrate increased tissue reactivity and decreased knot security relative to uncoated polyester, lightly coated PFTE polyester, and silk sutures. Polybutilate-coated polyester suture (Ethibond) possesses similar handling characteristics, but has less tissue reaction, when compared to the uncoated or lightly coated polyester suture materials.[9,10]

Polypropylene. Polypropylene (Prolene, Surgilene) is a recently developed monofilament suture. It is extremely inert in tissue, having the lowest tissue reactivity of any nonabsorbable suture. It does not undergo degradation in vivo as does silk. Polypropylene is a smooth monofilament suture; the smoothness of the strand facilitates easy passage of the suture through tissue. It has excellent tensile

Table 5-4. Nonabsorbable Suture Materials[a]

Generic name	Construction and handling characteristics	Frequent uses
Surgical silk	Braided and siliconized *Dry* silk stronger than wet Do not moisten before use Has excellent "hand"; ties in secure knots	Should *not* be used when suture permanence for life required or in urinary tract. Contraindicated in known infected area. Very seldomly used today.
Polyester suture	Braided and siliconized High tensile strength retained indefinitely in tissues Smooth, supple in handling	Particularly useful in implanting heart valves and vascular prostheses; hand surgery, orthopedic and ophthalmic surgery
	Braided, uncoated polyester fiber; somewhat rougher and stiffer than coated High tensile strength; low tissue reactivity; good knot security	Cardiovascular, general, ophthalmic and orthopedic procedures
Nylon suture	Monofilament Strong, relatively inert and nonirritating in tissues	Skin closure and plastic surgery. May be buried in subcutaneous layer or used as pullout suture. Useful in ophthalmic, microsurgery, tendon repair, and as retention suture
	Braided and siliconized Handles like silk but is less reactive and much stronger Excellent knot security	May be used in place of silk with excellent results. Useful for general closure
Linear polyethylene	Monofilament with minimal elasticity Excellent tensile strength; soft and pliable for smooth passage through tissue	General closure, skin suture, plastic surgery, tendon repair, and vascular anastomoses
Polypropylene	Monofilament Smooth passage through tissues; minimal tissue reaction. Maintains tensile strength in vivo	General closure, vascular anastomosis, skin sutures; whereever nonabsorbables are indicated (except eye). Excellent pull-out suture; fascia
Stainless-steel wire	Twisted multistrand Exceptional strength; flexible noncorrosive, inert Almost no tissue reaction Nonmagnetic and electropassive in tissue fluids Cut only with wire scissors	Useful in known infected areas and when minimal tissue reaction desirable. Often used to repair disruption or eviscerated wound, or as retention sutures
	Monofilament strand Take care to avoid kinking during handling; avoid pricking gloves	Frequently used in orthopedic, plastic, thoracic and restorative procedures
Silver wire suture	Monofilament Very strong, more pliable than stainless steel. Has an antibacterial quality	For closure of dehiscence and for piercing ears

[a] Modified from Davis, Geck.[34]

strength, which is maintained in vivo. It has the best resistance to bacterial infection of any nonabsorbable suture. Polypropylene has slightly more memory than monofilament nylon; it elongates under tension but recovers its original dimension when tension subsides. Knot security with polypropylene is good, especially if surgical square knots and extra throws are employed.[9,10,34] Polypropylene is less likely to untie than other synthetic monofilament sutures because it is a softer, more plastic material than other synthetic nonabsorbable materials; if the knots are firmly set, a flattening occurs where strands cross one another, and this creates a locking effect.[9,10,34]

Table 5-5. Relative Tissue Reaction of Nonabsorbable Sutures[a]

Least reaction	
↑	Monofilament polypropylene
↑	Monofilament nylon
↑	Stainless steel wire
↑	Uncoated braided polyester
↑	Polybutilate coated polyester
↑	Teflon/silicone coated polyester
Most reaction	Natural fiber materials

[a] Modified from Yu GV, Caugliere R.[9]

Table 5-6. Relative Tissue Strength of Nonabsorbable Sutures[a]

Greatest strength	
↑	Stainless steel wire
↑	Coated braided polyester
↑	Uncoated braided polyester
↑	Monofilament nylon
↑	Braided nylon
↑	Monofilament polypropylene
↑	Monofilament polyethylene
	Natural fiber

[a] Modified from Yu GV, Caugliere R.[9]

Table 5-7. Relative Coefficient of Friction of Nonabsorable Sutures[a]

Greatest	
↑	Stainless steel
↑	Uncoated braided polyester
↑	Uncoated braided nylon
↑	Coated braided polyester
↑	Coated braided nylon
	Monofilament synthetics

[a] Modified from Yu GV, Caugliere R.[9]

Stainless Steel. Stainless-steel wire is made from a ferrous alloy of selected metallic ingredients. It is not as inert as some of the pure synthetic polymers; it causes a mild-to-moderate tissue reaction similar to monofilament nylon suture and is usually well tolerated in tissue. Stainless-steel wire provides the most tensile strength and greatest knot security of the available nonabsorbable sutures. However, it has poor handling properties and this may contribute to kinking, fatigue, fracture, or deformation at points of stress.

Twisted multistranded steel wire suture (Flexon) is more flexible and easier to handle and tie. The ends of wire suture must be handled with care to avoid puncture of gloves or tissue. Special instruments are necessary to cut wire sutures. Stainless-steel wire is useful for secondary repair of wound disruption or evisceration, as retention sutures, and in tissue sites of known infection.

Cotton. Surgical cotton is largely an obsolete suture material. It is weaker than silk, and handles similarly. Wet cotton is 10% stronger than dry cotton: Cotton sutures should be moistened prior to use to increase tensile strength and also to reduce its clinging to surgical gloves. Like silk, cotton produces a great amount of tissue reaction.[34]

Wound Closure

General Principles

The tensile strength of a sutured wound changes in a predictable way over time (Figure 5-3). The first 3–5 postoperative days is the lag phase, characterized by low tissue tensile strength. The lag phase is followed by a period of fibroplasia, collagen deposition, and increasing tensile strength. Wound strength during the lag phase and early fibroplasia is almost wholly dependent on suture tensile strength.[1,2]

It takes many months for abdominal wall fascia to heal. Durability and strength of the suture is particularly important since fascia regain only 25% of original strength after 20 days. When healing is delayed or incomplete, as in infected wounds, suture support is required for more extended periods.[6,12,31] The tensile strength of the fascial layer is greater than any other layer of the abdominal wall.

However, sutures must hold tissues together until sufficient healing has occurred (Figure 5-3). Selecting a rapidly absorbed suture for fascial closure increases the risk of wound dehiscence.

Early studies indicated that vertical incisions heal more slowly than transverse incisions. This was generally regarded as fact until recent studies demonstrated that vertical and transverse incisions have similar rates of dehiscence in randomized trials.[12,36,38] Earlier studies were nonrandomized and many have been biased, because vertical incisions can be performed faster than tranverse incisions and may have been used in sicker patients. Experimental studies have not supported the opinion that transverse incisions are under less tension than vertical incisions. It is probable that healing is more closely related to choice of suture and surgical technique than to choice of incisions.[12,37]

Catgut suture, first plain and later chromic, was the suture of choice for fascial closure for years. As stated earlier, catgut has less dependable tensile strength, wound security, and knot security than other sutures. One study demonstrated a 20%–30% loss of tensile strength within 2 hr when wet;[23] another indicated more wound dehiscences with catgut (14%) than with either catgut plus retention sutures (5%) or with wire (1%).[12,39] Clearly, catgut is suboptimal for fascial closure.

Suture selection should be based on the rate of healing of incised tissues. Generally, skin and fascia heal more slowly than visceral organs and peritoneal surfaces, and sutures should be selected that will maintain tissue approximation until adequate tissue tensile strength is regained.[12] The newer synthetic absorbable sutures, polyglycolic acid and polyglactin, retain their tensile strength and wound security better than chromic catgut. In the unused state, they are stronger than nonabsorbable sutures. After 14 days in vivo, these sutures have lost significant tensile strength but are still as strong as silk suture.[9,10,34] Polyglycolic acid loses much of its wound security after 30 days, but it is unusual for suture support to be necessary longer than that.[12]

Monofilament sutures such as nylon or polypropylene are also good choices for fascial closure. Monofilament nylon loses only 16% of its tensile strength after 70 days and only 20% after 200 days.[12] Two recent randomized studies demonstrated that polyglycolic acid, polyglactin, and polypropylene have similar rates of wound dehiscence.[40,41] Another study showed that wounds closed with silk dehisced more often than wounds closed with polyglycolic acid.[12,42]

There is little recent information on suture selection for abdominal wall closure in the presence of infection, but monofilament nonabsorbable sutures appear to be best. All sutures cause some degree of inflammatory reaction in tissues, in part because of the trauma of insertion and in part secondary to the physiochemical properties of the suture material.[6] Monofilament nylon and polypropylene remain relatively inert in infected wounds. In contrast, multifilament nylon is surrounded by a vigorous inflammatory reaction in vivo. Silk also manifests a vigorous inflammatory reaction and abscess formation in infected tissue. Polyglycolic acid, despite minimal tissue reaction in noninfected tissue, induces much more tissue reaction in infected tissue with small abscesses noted close to the suture.[6,31]

The increased reaction seen with multifilament nylon and polyglycolic acid is attributed to the braided nature of these materials and the persistence of bacteria in the interstices of the braid. Bacteria that penetrate multifilament threads promote and prolong inflammation because they are protected from the body's defense mechanisms. Granulocytes and macrophages are too large to work their way between tightly packed multifilament threads. Electron microscopy studies support this concept; bacteria have been demonstrated in the interstices of all the multifilament materials, particularly nonabsorbable silk and nylon. The implication is that these sutures provide a continuing nidus for infection. With monofilament suture there is no such nidus for infection unless the suture moves about and creates a space between it and the surrounding fibrous capsule that then fills with bacteria. Use of monofilament suture is also associated with a lower incidence of sinus formation.[6,7,31]

Infection seems to slow the absorption of polyglycolic acid suture.[6,31] Loss of tensile strength, however, does not parallel absorbability, and recent studies have demonstrated

that tensile strength is lost after 30 days.[6,31] If absorbable braided suture persists in the infected wound after its strength is gone, it continues to act as a foreign body without contributing to wound security.

Monofilament nylon and polypropylene are the best suture for closure of an infected abdominal wall fascia. Strength is sufficient to maintain approximation of fascial edges, even if healing is delayed by infection. These sutures are quickly walled off by a fibrous capsule. They are relatively free from infection, inert, and nonirritating to tissue.

Surgical Technique

Proper wound closure technique provides adequate tensile strength until the wound is healed. It approximates tissue to facilitate healing, secure infected wounds, utilizes well tolerated suture material, and is time efficient. Abdominal wound disruption (dehiscence) is a serious postoperative complication. Evisceration is associated with an average mortality rate of 20% (range, 11–35%).[17] The incidence of wound disruption has been relatively constant over time despite increased knowledge of wound healing, preoperative and postoperative care, surgical technique, and suture materials. The general surgery literature continues to report 0.5–5.0% rates of fascial dehiscence.[17,43,44] There is much less literature dealing with wound disruption following gynecologic surgery; the reported incidence is 0.3–0.7%.[17,45,46]

The slightly lower rates of wound dehiscence following gynecologic surgery have been attributed to the infraumbilical and low transverse incisions. Among the reasons for these reduced rates are elasticity of the abdominal wall associated with pregnancy, less physical activity in female patients, the younger mean age of gynecologic patients, lower infection rates, and fewer bowel procedures.

Clearly, many high-risk factors may be identified preoperatively and postoperatively. Table 5-8 classifies the etiologic factors in wound disruption. The technique of abdominal incision, especially fascial, may contribute to wound disruption. In one series, six of eight dehiscences were associated with the use of electrocautery to open the fascia.[14] In these six cases, reexploration revealed fascial necrosis without purulence at the sites where suture material had pulled through tissue. Most commonly, wound dehiscence occurs between the 5th and 12th postoperative day, with postoperative day 8 being the mode. Wound dehiscence is also dependent on the technique of wound closure.

There are several mechanical and biologic factors that contribute to dehiscence. First, the suture may be absorbed, resulting in too rapid a loss of tensile strength. Sixty percent or more of dehiscences were associated with the use of chromic catgut.[17] Second, the suture may break. Modern suture, however, seldom breaks if it is of appropriate tensile strength and chosen and tied properly, without fraying. Third, knots may untie or slip. A secure surgical knot is essential to wound security. Moreover, it is the surgical knot that usually fails when a tied suture is subjected to increasing tensile stress. The surgeon should obviously know the relative knot security of various suture materials[4,17] (Table 5-7).

The most common clinically demonstrated mechanical reason for wound disruption is suture cutting through tissue with the suture and knot remaining intact. This has been observed in 88% of disrupted wounds.[47] The size of the tissue bite and the diameter of the suture material are inversely related to the risk of disruption (Figure 5-6). Large bites with thick suture are less likely to cut through tissue than small bites with thin suture material.[48] Moreover, maximal wound strength is achieved when fascial edges are loosely approximated with big bites[5] (Figure 5-7). Tying sutures loosely decreases tissue ischemia and necrosis and subsequently weakening of the suture line. Several studies have demonstrated that mass closure incorporating muscle is mechanically sound, because it allows for the 30% expansion of the wound that occurs with postoperative abdominal distension.[12] Midline incisions closed with a mass-closure technique, such as Smead-Jones, are thrice more resistant to disruption than paramedian incisions closed in layers, twice more resistant than midline incisions closed in the linea alba, and 1.7 times more resistant to disruption than a transverse supraumbilical incision.[49]

Midline incisions provide better operative exposure and are frequently preferred. Most

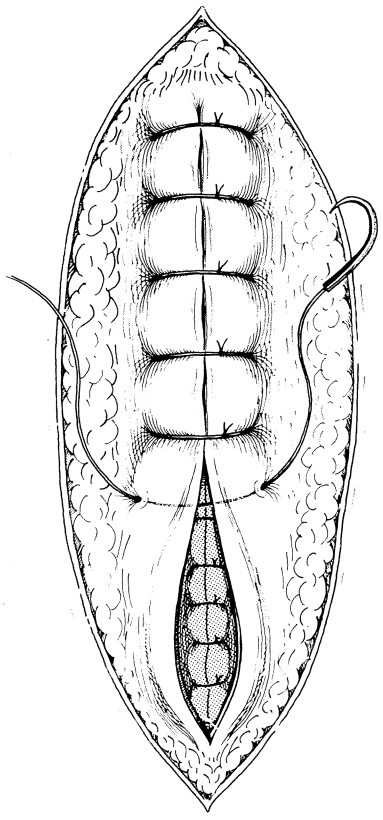


Figure 5-6. Single-layer closure. End result of fascial closure with simple interrupted technique. Sutures are loose, with big bites.

surgeons believe that interrupted sutures provide greater security than continuous suture closure. However, the fact is not well supported in the literature.[12] Closure technique, such as adequacy and spacing of tissue bites, is perhaps more important than the method of closure. This is confirmed by several series.[12,24] Nevertheless, we do not recommend continuous fascial closure with catgut on a vertical incision.

Patients at high risk for wound dehiscence must be recognized preoperatively (Table 5-8). In closing the abdominal wall, we recommend using a layer-by-layer technique in low-risk patients and a mass-closure technique, such as the Smead–Jones techniques or external retention sutures, in partients at high risk for dehiscence. There is little support for the use of external retention sutures. Retention sutures left in situ for 10–15 days postoperatively will leave a significant scar. If used, it is important to choose a suture material for fascial closure that maintains tensile strength longer than the retention sutures remain in tissue. We do not recommend the use of external retention sutures, because they are less cosmetic, more painful, and more likely to cause infection along the skin sinus tract than is the internal mass closure technique.

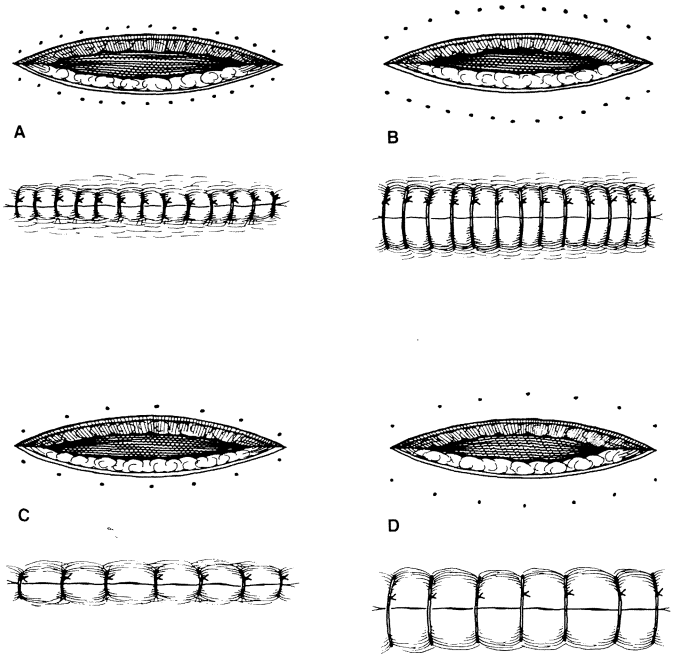


Figure 5-7. Primer of suturing technique. In their animal study, Sanders and colleagues evaluated the tensile strength and microscopic appearance of the fascia, and found that closure is stronger when sutures are placed 1 cm from the fascial edge and tied loosely.[7] Thus, techniques shown in (**A**), (**B**), and (**C**) are wrong; (**D**) is correct.

Table 5-8. High-Risk Factors for Wound Dehiscence

Preoperative
Age
Chemotherapy
Chronic cough
Chronic illness
Malignant disease
Nutrition
Obesity
Previous abdominal surgery
Prior radiotherapy
Pulmonary disease
Steroid therapy
Intraoperative
Improper selection of incision, suture material, or closure technique
Postoperative
Ileus
Intestinal obstruction
Vomiting
Wound infection

Regardless of technique, close attention to hemeostasis and proper handling tissues will minimize trauma and help prevent dehiscence.

Layer-by-Layer Closure. In layer-by-layer closure, the parietal peritoneum, fascia, subcutaneous tissue, and skin are closed separately. Before closing the peritoneum, it is important to achieve operative site hemostasis and to wash the pelvic cavity with large volumes of warm saline to remove debris. This approach reduces the chance for operative site infection and adhesion formation.

The parietal peritoneum should be closed with a small-diameter absorbable suture such as 3/0 polyglycolic acid or polyglactin, since little tensile strength is required to approximate this layer, which will heal within 24–48 hr. A large randomized trial demonstrated no difference in dehiscence rates between patients with peritoneum closed and in patients with peritoneum left open during layer-by-layer closure. A minimal amount of suture material should be left within the peritoneal cavity, and very tight suturing that may produce ischemia should be avoided. A continuous suture incorporating the parietal peritoneum, and also the transversalis fascia when prominent, is sufficient. Interlocking continuous sutures may slow the healing process by decreasing vascularization of the peritoneal surface. Peritoneal ischemia may deprive tissue of its ability to lyse fibrin and interface with the process of wound healing.

It is most important to keep fascia properly approximated while it develops sufficient tensile strength through fibroplasia. Proper closure of this layer requires a suture that will produce the least inflammatory reaction, preserve tensile strength, and minimize the chances of infections. The best way to close fascia in low-risk patients is with simple, loosely-tied interrupted stitches using size 0 or 1 synthetic absorbable suture. A nonabsorbable monofilament suture such as Prolene size 0 is also appropriate. The distance to the edge of the incision and between sutures should be about 1 cm (Figure 5-7). Decreasing the space between sutures temporarily increases wound strength, but this initial benefit is followed by weakening of the fascial layer, which may result in incisional hernia. Sutures should be tied securely so as to approximate but not strangulate tissues. In transverse incisions, the fascia can be closed with continuous, synthetic absorbable sutures.

The main objective of suturing subcutaneous fat is to control capillary bleeding and obliterate the space that would form by pulling skin edges together over the top of a subcutaneous defect. Blood, liquefied fat, serum, and debris are excellent media for bacterial growth. Several loosely tied sutures in subcutaneous fat will obliterate dead space beneath the skin. Small-caliber absorbable sutures such as 3-0 polyglycolic acid are most appropriate, since sutures inciting significant tissue reaction should be avoided.

Desired cosmetic appearance should influence the method used for skin closure. Continuous subcuticular suture achieves the most cosmetic result. An absorbable synthetic suture size 5-0 on a small cutting needle is recommended for subcuticular closure (Figure 5-8). However, a continuous pullout subcuticular suture with 4/0 prolene is recommended for patients who tend to form keloids. These sutures may be removed after 7–10 days, thus avoiding further inflammatory reaction associated with suture absorption. Steri-strips are applied to the skin to reduce tension on subcuti-

cular sutures. A steri-strip may be placed on the patient's skin a few days prior to surgery to detect hypersensitivity to the tape. Staples are also acceptable for skin closure; these are generally removed after 5 days and replaced with steri-strips.

Interrupted skin sutures may be utilized, with monofilament nylon or polypropylene preferred. Every surgeon must be fully familiar with the various techniques of suture placement (simple, vertical mattress, horizontal mattress) and know how to produce everted skin edges as opposed to inverted skin edges during closure. Most standard textbooks of general surgery and plastic surgery describe these suture techniques in detail.

It is important to realize that early postoperative edema increases the tension on skin edge. This frequently results in cutting of the skin even though sutures were snug at the time of skin closure. This excess tension contributes to permanent skin suture marks, especially if the sutures remain longer than 7 days. Lateral pull on the skin may occur secondary to excessive removal of tissue or because the wound is at right angles to muscle pull. This lateral pull can be reduced by subcutaneous sutures placed so as to reduce tension on skin edges.

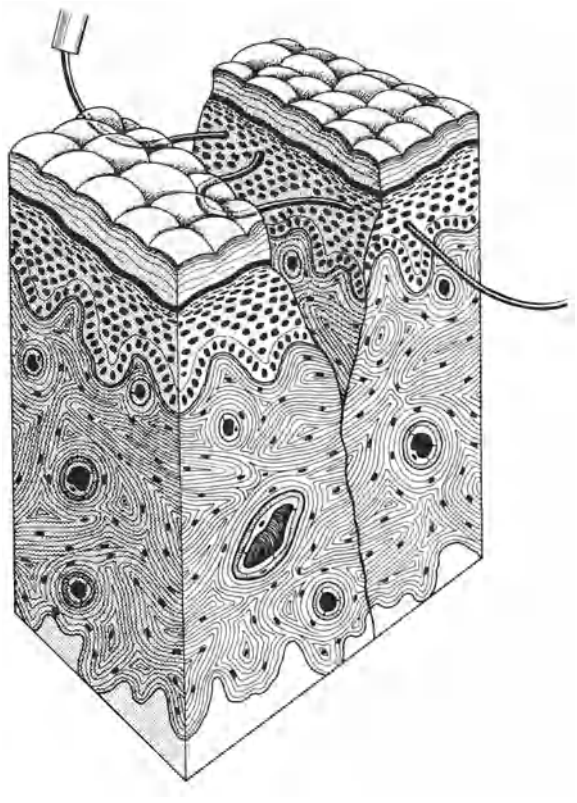


Figure 5-8. Subcuticular skin closure. For best appearance, use a continuous subcuticular 4-0 or 5-0 suture, such as Dexon or Vicryl. The alternative is a continuous pullout subcuticular closure, with a nonabsorbable monofilament suture, such as Prolene. The latter must be removed in 7–10 days.

Mass-Closure Technique. The Smead–Jones technique of mass closure is the best and most comfortable wound closure for patients at high risk for dehiscence. Clinical experience verified the usefulness of this closure technique in reducing the wound dehiscence rate in high-risk patients.[14,16,17,29] Wound dehiscence rates may be as low as 0.1% using Smead–Jones closure.

The Smead–Jones technique applies the principle of mass closure to prevent suture material from tearing through weakened tissue. The Smead–Jones closure has been called the far–near closure technique[29] (Figure 5-9). The closure incorporates the anterior fascia, rectus muscle, posterior fascia, and peritoneum as the internal retention portion and anterior fascia as the second portion of the closure.[29] Care must be taken not to incorporate excessive peritoneum or to leave too much permanent suture material within the peritoneal cavity, which might result in adhesion formation or entrapment of bowel if the suture loosens. A variation of the Smead–Jones technique is to place several interrupted fascial sutures to reinforce the mass closure. This step helps to reduce incisional hernia by allowing better approximation of fascial edges.

Monofilament permanent suture is the best for mass closure. Excellent results have also been reported using polyglycolic acid suture.[29] The outermost sutures should be placed at least 2.5 cm from the fascial edge. The distance between sutures should be 1–2 cm. The sutures must be tied so that tissue is loosely approximated; tying these sutures too tightly results in devascularization of large amounts of muscle and fascia which will eventually weaken the incision.

Delayed Primary Closure. Wound healing occurs by first, second, or third intention (Figure 5-10). A surgical incision that is reapproximated heals by first intention. Second intention

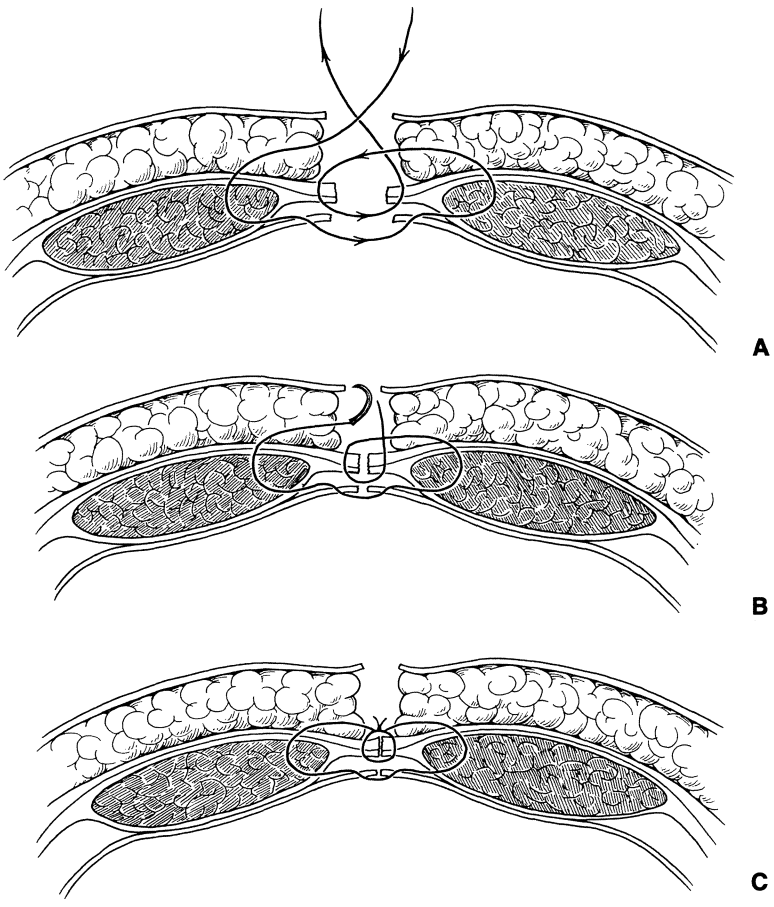


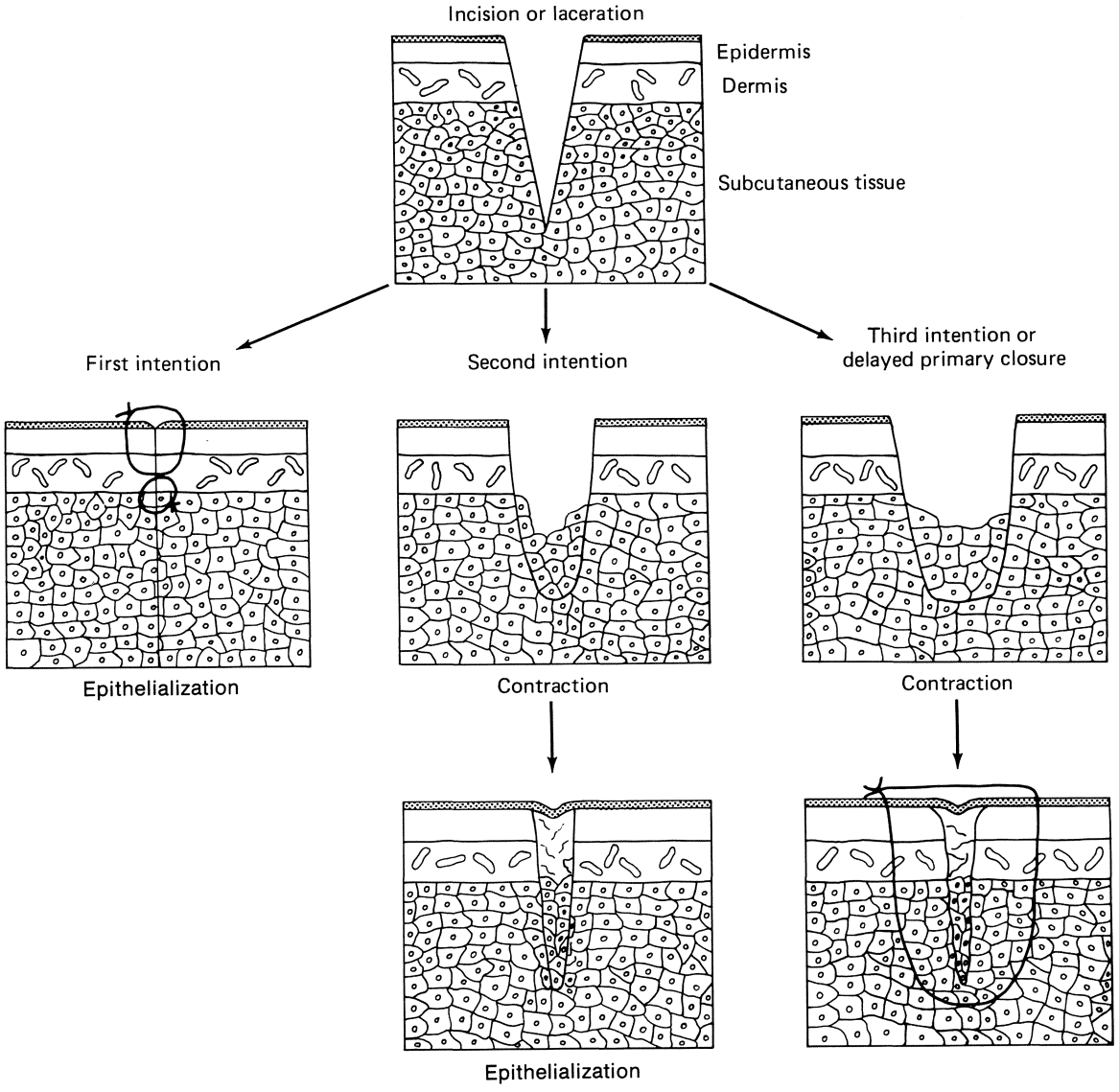
Figure 5-9. Modified Smead–Jones closure. In this approach, only 1 cm of the peritoneum is enclosed by the suture, to prevent entrapment of the bowel by the large intraabdominal loop. (**A**), Suture going through the anterior and posterior fascia should be at least 2.5 cm from the edge of the fascia. (**B**), Minimize the amount of suture material inside the abdominal cavity. (**C**), Approximate the edge loosely to prevent ischemia and necrosis.

healing refers to an open wound healing through the processes of epithelialization and granulation tissue formation. During second intention healing, the open defect is filled with new tissue prior to complete epithelialization. Third intention healing is alternately referred to as delayed primary closure. The wound is initially left open to prevent infection and it is subsequently closed to allow healing to complete by first intention. If the probability of wound infection is high, delayed primary closure is recommended to prevent wound abscess[11] (Table 5-9). Military surgeons have long recognized the value of delayed primary closure in managing contaminated wounds. Several recent studies emphasize the value of delayed primary closure in reducing the incidence of wound infections following gynecologic oncology surgery, surgery for tuboovarian abscess, surgery in the morbidly obese patient, contaminated bowel surgery, and suppurative appendicitis. Delayed primary closure is associated with a reduction in

Table 5-9. Indications for Delayed Primary Wound Closure[a]

Patient
Obesity
Malnutrition
Chronic or debilitating disease
Diabetes
Immunosuppression
Disease process
Malignancy
Infection
Pelvic inflammatory disease
Intraperitoneal abscess
Diverticulitis
Prolonged rupture of membranes
Clinical endometritis or amnionitis
Surgical procedure
Opened bowel
Break in aseptic technique
"Above and below" procedure, e.g., radical vulvectomy, urethrovesical sling procedure
Excessively moist wound
Any incision when infection is endemic in the hospital

[a] Modified from Brown SE, Allen WH, Robins RN.[11]

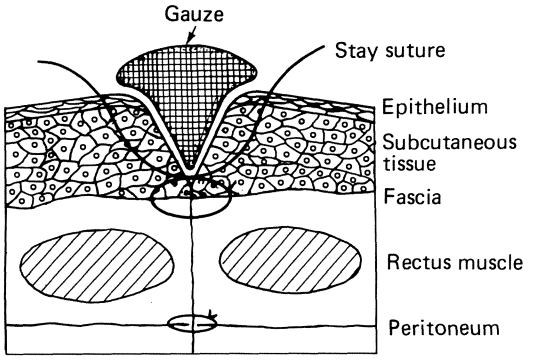


the wound infection rate from 34.1% to 2.3% in patients with peforating appendicitis.[11]

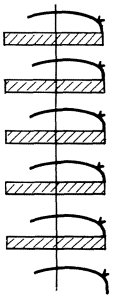
Delayed closure involves closing the peritoneum and fascia (Figure 5-11). The wound is irrigated with sterile saline, and vertical interrupted mattress sutures of monofilament nylon or polypropylene are placed at 2-cm intervals and left untied, thus leaving skin and subcutaneous tissue open. Sterile gauze soaked with dilute povidone-iodine solution is placed in the wound to cover subcutaneous tissue down to the fascia. A large dry dressing then covers the incision. The wound is inspected and cleansed daily with peroxide, and if there is no evidence of infection the sutures are tied at the patient's bedside on the fifth postoperative day. This is done under regional anesthesia and/or intravenous sedation.

Several basic physiologic mechanisms of wound healing support the use of delayed primary closure. Epithelialization produces a watertight seal within 24 hr in primarily closed incisions.[1,2,11] Exudation into a wound is maximal during the first 24 hr and decreases rapidly over the subsequent 48 hr. Therefore, any contaminated wound closed primarily results in entrapment of bacteria in the subcutaneous tissues, where exudate, blood clot, and debris

◀ **Figure 5-10.** Schematic diagram of healing by first, second, and third intention.



A. The wound is kept open above the fascia



B. Closure after 5 to 7 days if the wound is clean

Figure 5-11. Technique of delayed primary closure. The wound is left open above the fascia and irrigated twice daily. A Betadine-soaked gauze is left inside the wound and covered with a dressing. On day five or seven the wound is closed with the permanent monofilament stay sutures.

facilitate bacterial multiplication. Bacteria cultured from such infected wounds are generally the same as those cultured at the time of operation, while infections developing after delayed primary closure are usually due to bacteria acquired nosocomially.[11]

Moreover, collagen is not synthesized in significant amounts until the fifth postoperative day. Therefore, there is little benefit, with respect to wound tensile strength, in closing contaminated wounds before the fifth postoperative day.[1,2,11]

In most cases, risk factors can be evaluated in the preoperative assessment. Delayed primary wound closure is a simple, safe, and effective method of reducing the incidence of wound infection in patients at risk. It is well tolerated and economical. Hospital stay is not extended, and wound strength and appearance are not affected.[11]

Bibliography

1. Hunt T, Dunphy JE. Fundamentals of wound management. New York: Appleton-Century-Crofts, 1979.
2. Peacock EE. Wound repair, 3rd edition. Philadelphia: WB Saunders, 1984.
3. Lavia MF, Hill, Jr RB. Principles of Pathobiology, 2nd edition. New York: Oxford University, 1975.
4. Sanz L. Choosing the right wound closure technique. Contemp Obstet Gynecol. 1983, 21:142–154.
5. Sanders R, DiClementi D, Ireland K. Principles of abdominal wound closure II. *Arch Surg* 1977; 112:1184–1191.
6. Bucknall TE. Abdominal wound closure: choice of suture. J R Soc Med. 1981; 74:580–585.
7. Edlitch RF, Panek PH, Rodeheaver, GT et al. Physical and chemical configuration of sutures in the development of surgical infection. Ann Surg. 1973; 177:679–688.
8. Madden JW, Arem AJ. Wound healing: biologic and clinical features. In: Sabiston Textbook of Surgery.
9. Yu GV, Cauqliere R. Suture materials—properties and uses. J Am Podiatry Assoc. 1981; 73:57–63.
10. Swanson NA, Tromovitch TA. Suture materials; 1980's properties, uses, and abuses. Int J Dermatol. 1982; 21:373–378.
11. Brown SE, Allen WH, Robins RN. The use of delayed primary wound closure in preventing wound infections. Am J Obstet Gynecol. 1977; 127:713–717.
12. Kenady DE. Management of abdominal wounds. Surg Clin North Am. 1984; 64:803–807.
13. Stillman RM, Bella FJ, Seligman SJ. Skin wound closure—the effect of various wound closure methods on susceptibility to infection. Arch Surg. 1980; 115:674–675.
14. Greenburg AG, Saik RP, Peskin GW. Wound dehiscence: pathophysiology and prevention. Arch Surg. 1979; 114:143–146.
15. Trimbus JB. Security of various knots commonly used in surgical practice. Obstet Gynecol. 1984; 64:274–280.

16. Helmkamp BF. Abdominal wound dehiscence. Am J Obstet Gynecol. 1977; 128:803–807.
17. Wallace D, Hernandez W, Schlaerth J, et al. Prevention of abdominal wound disruption utilizing the Smead–Jones closure technique. Obstet Gynecol. 1980; 56:226–230.
18. Murray DH, Blaisdell FW. Use of synthetic absorbable sutures for abdominal and chest wound closures. Arch Surg. 1978; 113:477–480.
19. Howes EL. Strength studies of polyglycolic acid versus catgut sutures of the same size. Surg Gynecol Obstet. 1973; 137:15–20.
20. Gomel V, McComb P, Boer-Meisel M. Histologic reactions to polyglactin-910, polyethylene, and nylon microsuture. J Reprod Med. 1980; 25:56–62.
21. Herrmann JB. Changes in tensile strength and knot security of surgical sutures in vivo. Arch Surg. 1973; 106:707–710.
22. Rahmann MS, Way S. Polyglycolic and surgical sutures in gynecologic surgery. J Obstet Gynecol Br Commonw. 1972; 79:849–851.
23. Laufman H. Synthetic absorbable sutures. Surg Gynecol Obstet. 1977; 145:597–608.
24. Malt RA. Abdominal incisions, sutures and sacrilege. N Engl J Med. 1977; 297:722–724.
25. Jones TE, Newell ET, Brubaker RE. The use of alloy steel wire in the closure of abdominal wounds. Surg Gynecol Obstet. 1941; 72:1056–1059.
26. Haxton HA, Clegg JF, Lord MG. A comparison of catgut and polyglycolic acid sutures in human abdominal wounds. J Abdom Surg. 1974; 16:239–244.
27. Viljanto J. Disinfection of surgical wounds without inhibition of normal wound healing. Arch Surg. 1980; 115:253–256.
28. Sindelar WF, Mason R. Irrigation of subcutaneous tissue with povidone-iodine solution for prevention of surgical wound infections. Surg Gynecol Obstet. 1979; 148:227–231.
29. Baggish MS, Lee WK. Abdominal wound disruption. Obstet Gynecol. 1975; 46:530–534.
30. Aston SJ. The choice of suture material for skin closure. J Dermatol Surg. 1976; 2:57–61.
31. Bucknall TE, Ellis LT. The choice of a suture to close abdominal incisions. Eur Surg Res. 1983; 15:59–66.
32. Hunt TK. Wound healing. In: Current surgical diagnosis and treatment. Lange Medical Publications, 1977.
33. Laufman H. Is catgut obsolete. Surg Obstet Gynecol. 1977; 145:587–588.
34. Stroumtsos O. Perspectives on sutures. Monograph by Davis and Geck, 1978.
35. Stone HH, Hoefling SJ, Strom PR. Abdominal incisions: transverse vs vertical placement and continuous vs interrupted closure. South Med J. 1983; 76:1106–1108.
36. Wasiljew BK, Winchester DP. Experience with continuous absorbable suture in the closure of abdominal incisions. Surg Gynecol Obstet. 1982; 4:378–380.
37. Greenall MJ, Evans M, Pollock AV. Midline or transverse laparotomy? A random controlled clinical trial. Part I: Influence on healing. Br J Surg. 1980; 67:188–190.
38. Goligher JC, Irvin TT, Johnston D. A controlled clinical trial of three methods of closure of laparotomy wounds. Br J Surg. 1975; 62:823–829.
39. Corman ML, Veidenheimer MC. Controlled clinical trial of three suture materials for abdominal wall closure after bowel operations. Am J Surg. 1981; 141:510–513.
40. Irvin TT, Koffman CG, Duthie HL. Layer closure of laparotomy wounds with absorbable and nonabsorbable suture materials. Br J Surg. 1976; 63:793–796.
41. Kronborg O. Polyglycolic acid versus silk for fascial closure of abdominal incisions. Acta Chir Scand. 1976; 142:9–12.
42. Keill RH, Keitzer WF, Nichols SK. Abdominal wound dehiscence. Arch Surg. 1973; 106:573–577.
43. Mendoza CB, Postlethwait RW. Incidence of wound disruption following operation. Arch Surg. 1970; 101:396–399.
44. Pratt JH. Wound healing—evisceration. Clin Obstet Gynecol. 1973; 16:126–129.
45. Tweedie FJ, Long RC. Abdominal wound disruption. Surg Gynecol Obstet. 1954; 99:41–45.
46. Alexander HC, Prudden JF. The causes of abdominal wound disruption. Surg Gynecol Obstet. 1966; 122:1223–1227.
47. Dudley HAF. Layered and mass closure of the abdominal wall. Br J Surg. 1970; 57:664–668.
48. Tera W, Aberg C. Tissue strength of structures involved in musculo-aponecrotic layer sutures in laparotomy incisions. Acta Chir Scand. 1976; 142:349–355.
49. Ellis H, Heddle R. Does the peritoneum need to be closed at laparotomy? Br J Surg. 1977; 64:733–736.
50. Karipincni RC, Wilk PJ, Danese CA. The role of the peritoneum in the healing of abdominal incisions. Surg Gynecol Obstet. 1976; 142:729–730.
51. Haxton HA. The absorption of catgut in human abdominal wounds. Br J Surg. 1963; 50:534–537.

Avoiding Urinary Tract Injuries 6

Herbert J. Buchsbaum

The close anatomic and functional relationships of the female genital and urinary tracts have their origins in embryologic development. From the clinical perspective, pathologic processes in the genital tract can cause morphologic and physiologic alterations in the urinary tract; for example, large uterine myomas displace, and endometriosis and cervical carcinoma obstruct, the ureters.

Preoperative Evaluation

The avoidance of urinary tract injuries begins with appropriate preoperative evaluation. While many gynecologic conditions impact on the urinary tract, we do not feel that every patient scheduled for pelvic surgery needs an intravenous pyelogram (IVP). The risks are grave and the costs, on a national basis, prohibitive. In view of the rising medical costs, one must consider that approximately 800,000 hysterectomies (and nearly as many procedures on the adnexa) are performed annually in this country. If every patient were to have a pre- and postoperative excretory urogram, the costs would be, based on an average cost of $150.00 per study, $240,000,000 annually. The risks are appreciable: Bird and Sherman[1] reported an incidence of acute renal failure of 0.15% with intravenous pyelography; patients with certain diseases are at increased risk. It has been estimated that 1 in 14,000 patients has a serious reaction at the time of IVP, and 1 in 40,000 studies results in a death. If routine IVPs were performed, based on the above figures, 125 lives might be unnecessarily lost.

Certain patients do require preoperative urologic investigation. Patients with genital anomalies and conditions that commonly affect the ureters or bladder, either by invasion, obstruction, or displacement, should be evaluated. In most situations an IVP suffices; some patients may require cystograms, cystoscopic examinations, or urodynamic studies. Where appropriate, retrograde pyelograms or double-bulb urethrograms may be indicated.

Patients who would benefit from preoperative evaluation are those with gynecologic processes that are likely to involve the urinary tract. Documentation of the distortion and/or displacement of the urinary tract structures helps the gynecologic surgeon in avoiding injury to these structures, but will also identify measurable changes by which appropriateness of therapy can be assessed.

Genital Anomalies. Any patient who presents with primary amenorrhea or with a genital anomaly needs at least an intravenous pyelogram. Approximately one-half the patients with Müllerian agenesis (Mayer–Rokitansky–Kuster–Hauser syndrome) have some urinary anomaly.[2] About one-third have either an absent or ectopic kidney (Figure 6-1). Lesser defects include rotation or ptosis of the kidney and double collecting systems. It would be sad indeed if a solitary pelvic kidney was inadvertently excised or was traumatized during a McIndoe vaginoplasty. Granat and colleagues[3]

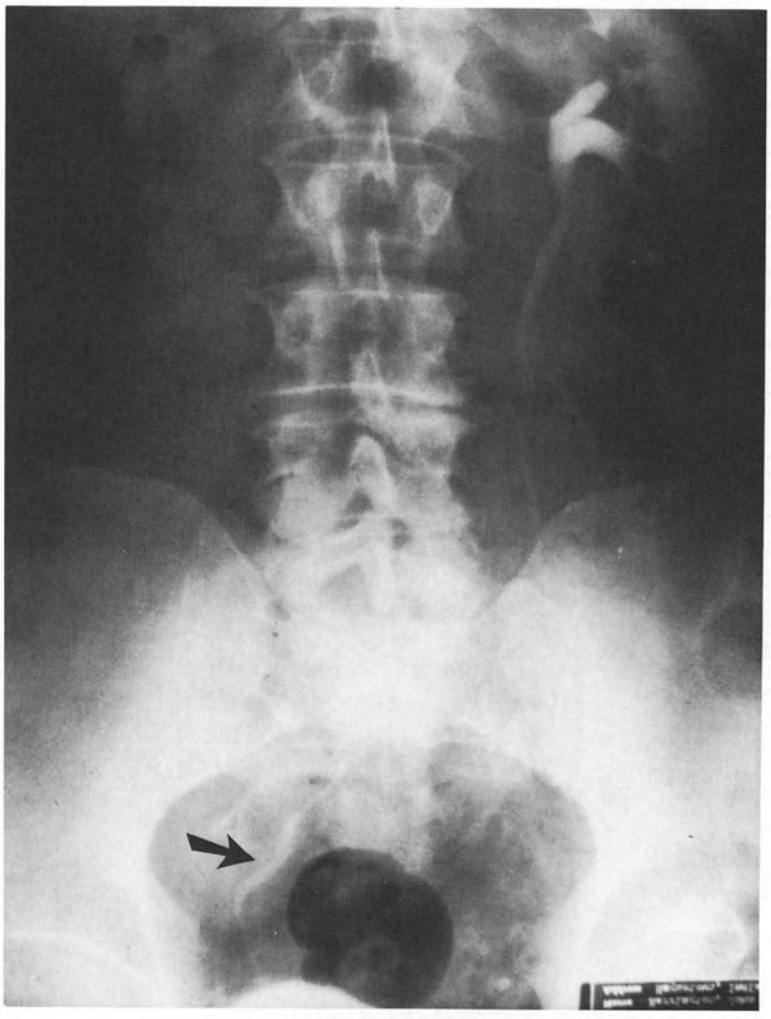


Figure 6-1. Intravenous pyelogram in patient with Mullerian agenesis. Note normal left kidney and collecting system. The right kidney is ectopic, located in the pelvis, with its ureter marked by *arrow*.

reported considerable retroperitoneal bleed from a pelvic kidney injured at culdocentesis.

Patients with uterine anomalies should undergo an excretory urogram. The highest incidence of renal anomalies is found among women with an obstructed double uterus or rudimentary uterine horn. Rock and Jones[4] reported that 100% of patients with this condition have renal agenesis on the side of the deformity. Patients with a uterus didelphys, a bicornate or septate uterus, have fewer and less significant urinary anomalies such as double collecting system, malrotation, and ptosis.

Leiomyomata. Leiomyomata uteri are the most common tumors of the female pelvis, present in 25% of women of reproductive age. The likelihood of ureteral obstruction and/or displacement secondary to uterine myomata is related to the size and location of the tumor. When the tumors are small, they are not likely to cause ureteral obstruction or displacement. When the uterus is enlarged and extends above the pelvic brim, ureteral obstruction is present in 20%–30% of women. The right ureter is involved three to four times more commonly than the left as a result of the cushioning effect of the sigmoid colon and its mesentery. The presence of pelvic inflammatory disease or endometriosis significantly heightens the risk of ureteral obstruction in patients with leiomyomata uteri.

Intraligamentous myomas are unique in their ability to obstruct a ureter. Nearly one-

Figure 6-2. Patient with total procidentia. **A,** Intravenous pyelogram showing medial displacement of the ureters (*arrows*) when uterus is replaced. **B,** Ureteral catheters demonstrating extrapelvic location of the bladder trigone. From Buchsbaum HJ and Schmidt JD. Gynecologic and obstetric urology, 2nd ed. Philadelphia, WB Saunders, 1982, chapter 10. Used with permission.

half of the patients with intraligamentous myomas will have either obstruction or deviation or both. Depending on location, the ureter can be deviated either medially or laterally, superiorly or inferiorly.

Pelvic, Inflammatory Disease. As with any adnexal tumor, the likelihood of pelvic inflammatory disease compromising the ureters is directly related to the size of the inflammatory mass. A pyosalpinx is not likely to cause ureteral obstruction; partial obstruction with a tuboovarian abscess is common.

Endometriosis. The most common site of endometriosis in the urinary tract is the bladder. Ureteral obstruction, resulting from either intrinsic (involving the ureteral wall) or extrinsic pressure, generally occurs in the distal one-third of the uteter. Any patient with significant endometriosis should have a pyelogram. The findings on the study may be an indication for surgery. Only 10% of patients with ureteral blockage secondary to endometriosis have cyclic flank or abdominal pain, but approximately 25% of kidneys are lost.[5]

Ovarian and Paraovarian Tumors. The likelihood of an adnexal lesion obstructing a ureter is related to its size and mobility. Small benign or malignant ovarian tumors are unlikely to obstruct the ureter. When a lesion reaches the pelvic brim, the likelihood of ureteral compromise is as high as 40%. Ovarian tumors, benign or malignant, are generally softer and more plastic than uterine myomas, and can accommodate to the shape of the bony pelvis. For this reason they present a greater risk to the ureter, and ultimately to renal function, than do leiomyomata.

Corpus and Cervical Malignancies. Cervical carcinoma, more than any other gynecologic condition, is likely to affect the ureters. Nearly one-third of patients presenting with invasive cervical carcinoma have ureteral obstruction on pyelogram. A higher proportion of patients with stage III disease will have ureteral obstruction. Obstruction by central tumor is near the ureterovesical junction, by enlarged pelvic lymph nodes at the lateral pelvic wall, and, less commonly, by metastases in the paraaortic lymph nodes.

Uterine Prolapse. Uterine descensus and prolapse are now the most common indications for vaginal hysterectomy. Although total procidentia is rarely seen these days, we continue to see patients with marked prolapse. When coming out of the pelvis, the uterus brings the bladder with it, resulting in medial displacement of the ureters and possibly in ureteral obstruction (Figure 6-2). Obstruction of the ureters by uterine prolapse was recognized as early as 1847 by Virchow. The degree and severity of changes in the upper tract are related to the age of the patient and the degree and duration of prolapse. The ureteral dilatation is usually bilateral, and can be severe. The site of obstruction is the most distal (intramural) portion of the ureter.

Preoperatively placed ureteral catheters have a very limited place in gynecologic surgery. Their placement requires an additional operative procedure, traumatizes the urothelium, and carries the risk of perforation. In severely indurated tissue, as in pelvic inflammatory disease, endometriosis, and cancer, the catheter cannot be palpated and the surgeon proceeds with a false sense of security. The best protection for the ureter is a surgeon familiar with its course through the pelvis.

Abdominal Surgery

The bladder should be emptied by catheterization just prior to pelvic surgery. For all but the shortest procedures, an indwelling catheter assures decompression of the bladder throughout the operative procedure, thus assuring better exposure and reducing the likelihood of bladder injury.

Laparoscopic Procedures

There are several points during a laparoscopic procedure at which the urinary structures are at risk. The reported incidence of bladder injury varies from 2% to 0.00012%.[6,7] Bladder injury can result from perforation with the Veerhis needle or from the placement of the trochar. A less frequent but far more serious injury is thermal injury to the bladder or ureter. Thermal burns result in extensive vascular injury, requiring broad resection before repair.

Laparotomy, and Abdominal Hysterectomy

Bladder. The most likely time of bladder injury is in the very beginning of the procedure when the peritoneal cavity is entered. Whether a vertical or transverse skin incision is made, the peritoneum should be incised vertically and the cavity entered as cephalad as possible. We do not open the peritoneum in "layers" as is frequently suggested. Too often, the surgeon forgets that the urinary bladder is an *extraperitoneal* structure and is more likely to be injured by this "layering." Furthermore, the peritoneum is never closed in layers, thus weakening the suture line. Approaching the bladder, the areolar tissue is thickened and vessels are more evident. To get a maximum incision, the surgeon can transilluminate this area, a technique that clearly identifies the border of the bladder. Graber and colleagues[8] reported that bladder injury occurred in 1.9% of 819 abdominal hysterectomies.

The second point in the performance of abdominal hysterectomy at which the bladder is at risk is during its mobilization off the lower anterior aspect of the corpus and cervix. Mobilization of the bladder assures not only its safety but also that of the ureters. By mobilizing the bladder the ureters are brought inferiorly and away from the vaginal angles. To accomplish this, the vesicouterine fold of the visceral peritoneum is best entered laterally after the round ligaments have been divided and ligated. The peritoneum over the cervix is elevated with tissue forceps, and a curved incision directed to that point. Too often the bladder incision is made too high on the corpus of the uterus. At this point the peritoneum is closely applied to the uterus with the areolar tissue absent. Incising the peritoneum at this site causes bleeding, which obscures the field and exposes the bladder to possible injury. When the peritoneum can be tented by elevating it with tissue forceps, one can be assured that the incision will be atraumatic. The edge of the inferior bladder flap can then be grasped with forceps and the bladder dissected off the cervix and vagina. The areolar tissue can be sharply incised or developed with a gauze-wrapped finger or a dental roll dissector.

In patients with previous cesarean section the tissue planes are obliterated. In such cases the dissection is best performed with the scissors, with the tip directed against the cervix. After the dissection is started, the portion of the bladder over the cervix and over the vaginal angles is generally easily mobilized. If the dissection is difficult and the surgeon suspects the bladder has been entered, it should be distended with methylene blue to identify any full-thickness defects. Should one be found, it is best closed with two layers of 3/0 chromic catgut. The first layer should be a continuous suture and the second layer interrupted sutures. Continuous bladder drainage with an indwelling catheter should be maintained for 10–12 days. Bladder injuries identified at surgery, and repaired, rarely result in fistulas.

Ureteral Injury. Injury to the ureters carries more dire consequences than bladder trauma. The incidence of ureteral injury in gynecologic surgery has been approximately 2%. The side of ureteral injury is approximately equal. There are five sites at which the ureter is likely to be injured in the performance of total abdominal hysterectomy and/or adnexectomy:

1. Infundibulopelvic ligament. The ureter enters the pelvis just medial to the bifurcation of the common iliac artery. At this site, adnexal pathology is likely to obstruct and/or displace the ureter. In pelvic inflammatory disease and endometriosis, the anatomy is generally distorted and the tissue indurated, making it difficult to palpate the ureter. If the infundibulopelvic ligament is to be clamped, it is best to open the peritoneum over the lateral wall between the round and infundibulopelvic ligaments. Once the peritoneum is opened and the space developed by finger dissection, the ureter is found on the medial leaf of the peritoneum. The external iliac artery is easily identified lying at the medial edge of the psoas muscle. If the artery is followed retrograde to its origin at the bifurcation of the common iliac artery, the ureter will be just medial. The ureter can then be followed retrograde into or behind the adnexal mass. With the ureter under direct vision, the infundibulopelvic ligament can be safely clamped, divided and ligated. In a rare situation where the entire pelvic peritoneum may be involved with a disease process, the peritoneum can be opened at

the pelvic brim to identify the ureter. This is done in a vertical fashion parallel to the ureter and allows the surgeon to follow the ureter in an antegrade fashion. Higgins[9] reported that 5 of 59 ureteral injuried (8.5%) in gynecologic surgery occurred during salpingo-oophorectomy.

2. Uterine artery. The ureter lies approximately 2–2.5 cm from the lateral margin of the cervix, where the uterine artery crosses. If, during dissection, bleeding occurs in the area of the uterine artery, the surgeon is likely to clamp laterally. This places the ureter at great risk, since the ureter lies approximately 2–3 cm lateral to the cervix and under the uterine artery (Figure 6-3). The most appropriate management would be to use direct pressure with a sponge stick by an assistant to control bleeding while the operator opens the peritoneum over the lateral pelvic wall to identify the ureter. The hypogastric artery can then be ligated or the bleeders isolated and clamped. One must exercise caution in the use of hemostatic preparations such as microfibrillar collagen (Avitene), oxydized cellulose (Surgicel), and absorbable gelatin sponge (Gelfoam). Several earlier reports have cited retroperitoneal fibrosis with ureteral obstruction when these materials were used. Wahlstrom and colleagues[10] did not find an adverse inflammatory or fibrotic reaction when these agents were used in the recommended fashion.

3. Cardinal ligaments. The ureters are at risk in the performance of extrafascial total abdominal hysterectomy when clamps are placed at the cardinal ligaments. If an extrafascial

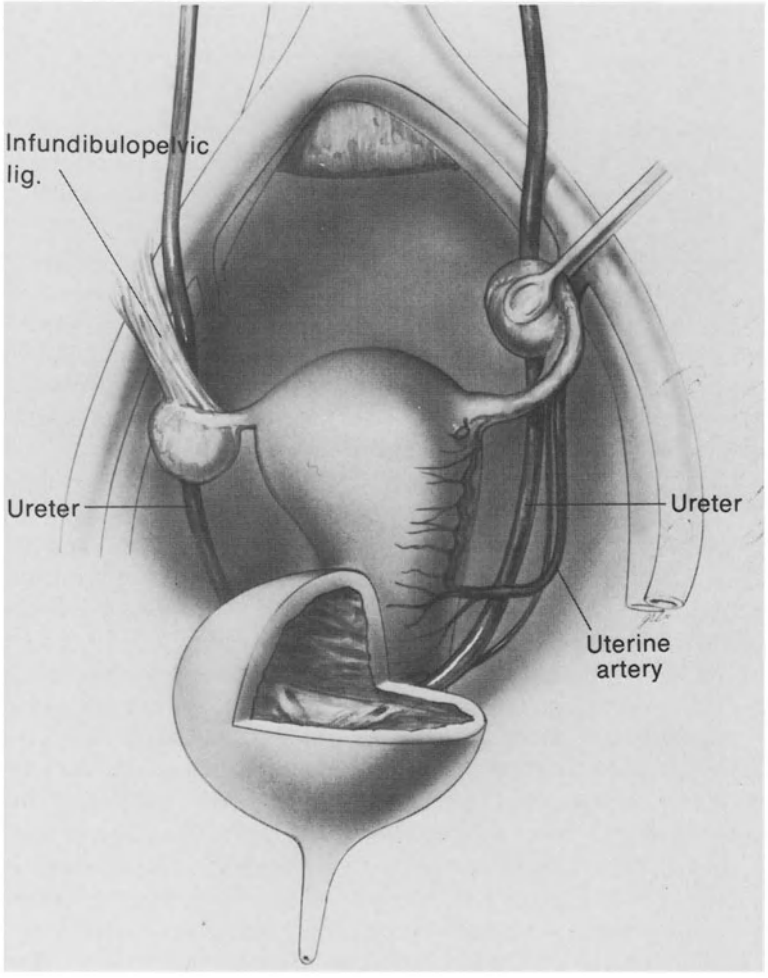


Figure 6-3. Pelvic anatomy showing relationship of the ureter to the uterine artery. From Buchsbaum HJ and Schmidt JD. Gynecologic and obstetric urology, 2nd ed. Philadelphia, WB Saunders, 1982, chapter 10. Used with permission.

technique is used, the tips of the clamp must be directed medially toward the cervix with the clamp in the axis of the uterus.

4. Angle of the vagina. Adequate mobilization of the bladder can avoid ureteral injury at this point in the operation. When the bladder has been adequately mobilized inferiorly, the ureters are moved further from the vaginal angles.

5. Reperitonealization. After the uterus has been removed and the vagina closed or a running lock suture placed, reperitonealization of the pelvic peritoneum places the ureters at risk. When large amounts of peritoneum have been excised, as is the case in inflammatory disease or endometriosis, the surgeon should examine the medial leaf of the peritoneum for the location of the ureter. Carelessly placed sutures can either incorporate the ureter or kink it.

Mechanisms of Injury. At any of the sites listed above, the ureter can be injured by one or more of mechanisms listed in Table 6-1. If in the performance of gynecologic surgery the surgeon suspects ureteral injury, every effort should be made to clarify the issue. Unfortunately, only one-third of ureteral injuries are recognized at surgery. Identification and correction of the injury spares the patient another operation at a time when inflammation, fibrosis, and adhesions will be present. Furthermore, it insures that renal function will not be lost.

If ligation is suspected, the ureter should be isolated by careful dissection, both proximal and distal to the site of suspected injury. Indigo carmine (5–6 ml) is injected into the lumen of the ureter proximal to the suspected site of ligation. If the urine in the collecting system turns blue, it is safe to assume that the ureter is not completely occluded. With transection or excision of a ureter, the extent of injury and the site can be identified by administering 8 ml of indigo carmine systemically and searching for extravasation of the colored urine. Crush injuries of the ureter require individualization. Generally, if there is discoloration or disruption of Waldeyer's sheath, resection is most appropriate. If the injury is less severe, a stent may be all that is required.

Table 6-1. Mechanisms of Ureteral Injury

Ligation
Transection
Crush
Puncture
Kinking
Anoxia, ischemia

Puncture wounds require no correction. If the ureter is incorporated into a pedicle by a ligature, careful examination of the ureter is essential. If no crush injury is evident, nothing need be done beyond removing the suture. Large pedicles ligated with heavy suture material kink, narrow, or partially obstruct the ureter. This can be avoided by using fine absorbable suture material and keeping the pedicles small in the vicinity of the ureter. Injury resulting from anoxia and ischemia occurs most often when there has been extensive dissection, compromising the ureter's internal blood supply.[11] The ureter has an extensive intrinsic blood supply, which will withstand ligation of its major external sources. When the ureter is circumferentially dissected and traumatized with instruments, this intrinsic blood supply is compromised, leading to anoxia, ischemia, and necrosis.

Site of Injury. Surgical correction of a ureteral injury is directed by site of the injury. Injuries of the distal ureter 3–4 cm from the ureterovisical junction are best managed by ureteroneocystotomy performed in an antireflux technique. There is generally enough mobility to accomplish anastomosis without tension. When the length of the ureter is inadequate to reach the bladder without tension, the bladder can be mobilized and brought toward the ureter. The bladder is fixed to the medial aspect of the psoas muscle—a "psoas hitch"—and the ureter anastomosed in an antirefluxing technique. If the bladder cannot be mobilized, and the ureteral injury is in the distal third of the ureter, a flap or pedicle can be developed utilizing full-thickness bladder wall. The ureter is then anastomosed to the tube.

With injuries in the middle third of the ureter and an adequate distal ureter, an end-to-end anastomosis can be attempted. The ends of the ureter are spatulated to increase the size

of the anastomosis, and a ureteral stent is brought out through the bladder and attached to a Foley catheter. When the injury is in the middle or upper third of the ureter or large segments have been excised or injured, a transureteroureterostomy is the most appropriate method of repair. The procedure can be done from right to left or from left to right. The injured ureter is mobilized retroperitoneally and brought across the midline and anastomosed to the intact ureter. If this is not feasible because of inadequate length of ureter or absence of the contralateral collecting system, a segment of small bowel can be interposed as a ureteral replacement and anastomosed to the bladder.

Vaginal Surgery

Considering the proximity of the bladder, ureters, and uterus and the restricted operative field with limited exposure, it is surprising how rarely bladder and ureteral injuries occur in the performance of vaginal hysterectomy. The incidence of bladder injury in the performance of vaginal hysterectomy varies from 0.2% to slightly over 1%.[12–14] If the bladder injury is recognized and repaired, there are usually no sequelae. The site of injury is most commonly on the posterior aspect, above the trigone. A two-layered repair utilizing 3/0 chromic catgut suture with prolonged bladder drainage is adequate. Copenhaver[12] reported 11 cystotomies recognized and repaired in the performance of 1,000 vaginal hysterectomies. Of the two vesicovaginal fistulas that developed, only 1 occurred in the 11 cases in which the bladder defect was recognized and repaired.

When vaginal hysterectomy is performed for uterine prolapse, the surgeon must remember that the bladder prolapses with the uterus. The bladder is no longer a pelvic structure, and great care must be taken in developing the planes in hysterectomy. As the bladder descends, the ureters are brought more medial and may be dilated (Figure 6-1).

Ureteral injuries are far less likely in vaginal than in abdominal hysterectomy. Nevertheless, there is a report of bilateral ureteral obstruction following anterior colporrhaphy.[15] If ureteral injury is suspected, a retrograde ureteral catheter should be passed with the patient still anesthetized. If this is unsuccessful, it is best to perform an extraperitoneal exploration of the distal ureter and bladder.

In the event that ureteral obstruction is identified, cutaneous ureterostomy or nephrostomy should be performed. This spares renal function and allows a delayed repair.

Summary

A knowledge of the course of the pelvic ureter is the most critical factor in avoiding injuries to it in the performance of gynecologic surgery. Patients with genital anomalies or those with disease likely to compress or displace bladder or ureter should have at least a preoperative intravenous pyelogram.

References

1. Byrd L, Sherman RL. Radiocontrast-induced acute renal failure. Medicine. 1974; 58:270–279.
2. Griffin JE, Edwards C, Madden JD, et al. Congenital absence of the vagina. Ann Intern Med. 1976; 85:224–230.
3. Granat M, Gordon T, Issaq E, et al. Accidental puncture of pelvic kidney: A rare complication of culdocentesis. Am J Obstet Gynecol. 1980; 138:233–235.
4. Rock JA, Jones HW Jr. The double uterus associated with an obstructed hemivagina and ipsilateral renal agenesis. Am J Obstet Gynecol. 1982; 138:339–342.
5. Moore JG, Hibbard LT, Growdon WA, et al. Urinary tract endometriosis: Enigmas in diagnosis and treatment. Am J Obstet Gynecol. 1979; 134:162–170.
6. Phillips JM, Keith D, Keith L. Gynecological laporoscopy 1973: The state of the art. In: Phillips JM, Keith L, eds. Gynecological laparoscopy: principles and techniques. New York: Stratton International Medical Book Corp, 1974.
7. Pattinson RC, Louw NS, Engelbrecht B, et al. Complications in 8509 laparoscopic Falopering sterilizations performed under local anaesthesia. S Afr Med J. 1983; 64:975–976.
8. Graber EA, O'Rourke JJ, McElrath T. Iatrogenic bladder injury during hysterectomy. Obstet Gynecol. 1964; 23:267–273.

9. Higgins CC. Ureteral injuries during surgery. JAMA. 1967; 199:82–84.
10. Wahlstrom E, Yadegar J, Amodeo P, et al. Comparative tissue reactivity to topical hemostatic agents in the periureteral area. Arch Surg. 1983; 118:1375–1377.
11. Buchsbaum HJ. The urinary tract and radical hysterectomy. In: Buchsbaum HJ, Schmidt JD, eds. Gynecologic and obstetric urology, 2nd edition. Philadelphia: WB Saunders, 1982: chapter 10.
12. Copenhaver EH. Vaginal hysterectomy. An analysis of indications and complications among 1,000 operations. Am J Obstet Gynecol. 1962; 84:123–128.
13. Pratt JH. Common complications of vaginal hysterectomy: Thoughts regarding their prevention and management. Clin Obstet Gynecol. 1976; 19:645–659.
14. Porges RF. Vaginal hysterectomy at Bellevue Hospital. Obstet Gynecol. 1970; 35:300–313.
15. Norman RW, Mack FG, Awad SA, et al. Acute renal failure secondary to bilateral ureteric obstruction: review of 50 cases. Can Med Assoc J. 1982; 127:601–604.

The Gastrointestinal Tract 7

Herbert J. Buchsbaum and Julius Mazer

Because of the proximity of the internal female genital structures to the gastrointestinal tract, diseases in one system can involve the other. Primary pathology in one system can present with signs, symptoms, and physical findings mimicking disease in the other. The gynecologic surgeon can better plan the operative procedure and thereby provide better care for the patient if this information is available preoperatively.

There are at least three reasons for obtaining preoperative diagnostic studies of the gastrointestinal tract: (1) pathologic processes involving the female genital structures can involve the gastrointestinal tract, e.g., endometriosis, pelvic inflammatory disease, and ovarian carcinoma; (2) benign and malignant processes of the gastrointestinal tract can present with signs, symptoms, and physical findings mimicking gynecologic disease, e.g., ruptured colon diverticulum or appendiceal abscess; (3) lesions which, like the Krukenberg tumor, primarily involve both the gastrointestinal and female genital tracts.

Diagnostic Studies

Selection of studies is based on medical history and findings on physical examination. Evaluation of the gastrointestinal tract should begin with routine testing for occult blood in the rectum in asymptomatic patients over 45 years of age. The study is cheap, easily performed, and an effective screening technique. Patients who give a history compatible with primary bowel disease should have a complete evaluation prior to pelvic surgery. The radiographic and endoscopic studies available to evaluate the gastrointestinal tract are shown in Table 7-1. A barium enema should be performed in all asymptomatic patients over the age of 50 who are being evaluated for gynecologic cancer.

For a gastrointestinal study to be of diagnostic value, the patient must be adequately prepared. For sigmoidoscopic examination, we prefer cleansing with a cathartic and enemas. For colonoscopic examination, whole gut irrigation should be employed (see below). Prior to the performance of a barium enema, the bowel should be cleansed utilizing two laxatives and 2 liters of water. We prefer to use 1 liter in the morning and 1 liter in the afternoon. A saline laxative (magnesium citrate) that draws fluid into the intestine is used, as well as a second laxative (bisacodyl) that aids in propelling fluid and stool. A cleansing enema should be administered the morning of the study.

Sigmoidoscopic Examination. This is best performed with the patient somewhat sedated and

Table 7-1. Diagnostic Studies of the Gastrointestinal Tract

Sigmoidoscopic examination
Colonoscopic examination
Flexible gastroscopic examination
Upper gastrointestinal contrast study
Small-bowel contrast study
Barium enema
Ultrasound
Computed tomography (CT)

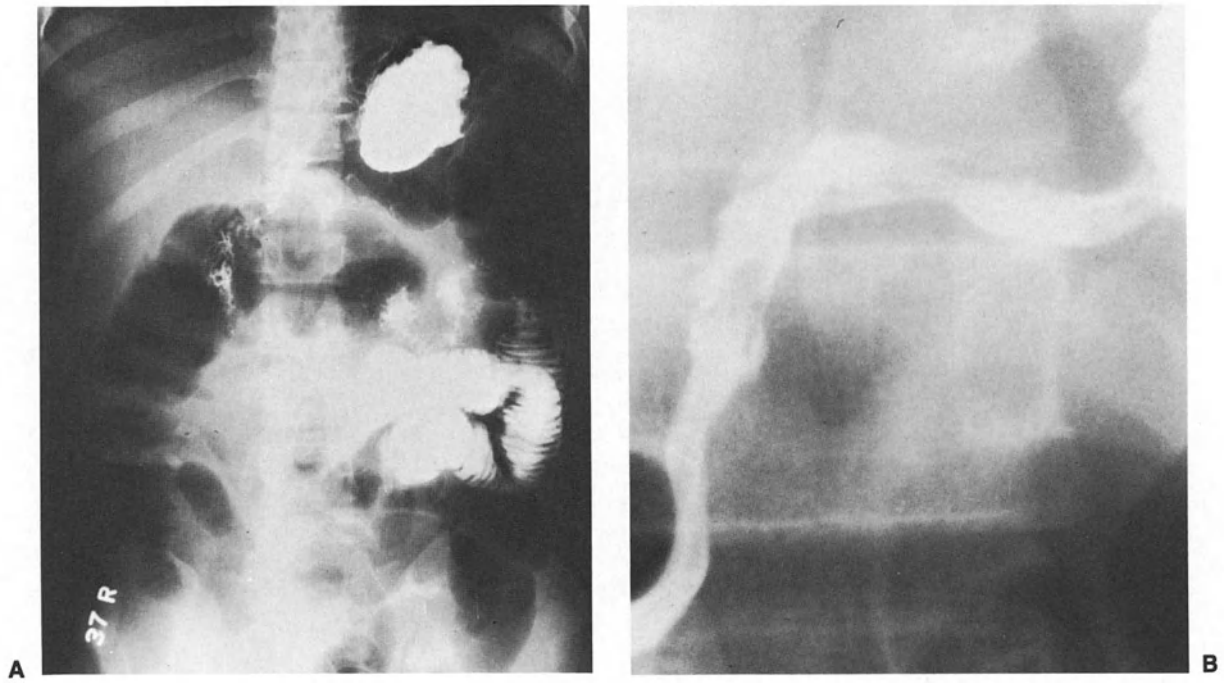


Figure 7-1. Studies in patient with Krukenberg tumor. **A,** Upper gastrointestinal study showing barium in gastric fundus and small bowel. **B,** Detailed view of stomach. Narrowed lumen of the body and antrum of stomach is compatible with tumor in wall. Biopsy of stomach and ovary revealed Krukenberg tumor.

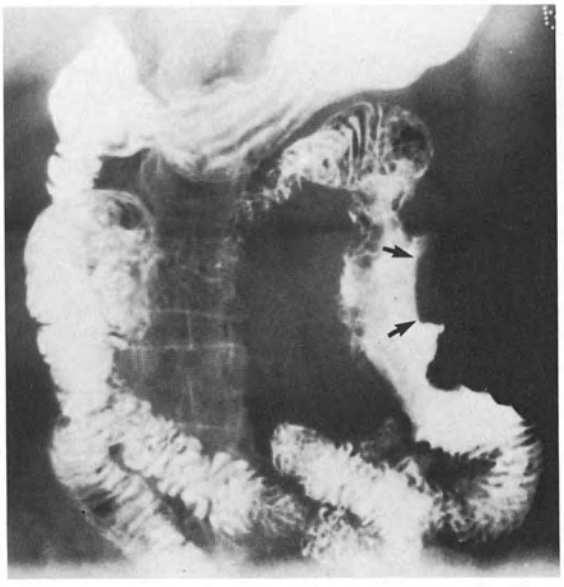


Figure 7-2. Upper gastrointestinal series with small-bowel follow-through in metastatic carcinoma. Mucosal destructive change (*arrows*) of proximal jejunum is shown just below the ligament of Treitz, resulting from carcinoma metastatic to the small bowel.

in the knee-chest position. The fiberoptic sigmoidoscope provides good light; suction and biopsy equipment should be available when the examination is performed.

Colonoscopic Examination. Colonoscopic examination with a flexible scope is not performed by gynecologists and requires specialized training. With the incidence of bowel carcinoma shifting from the left to the right colon, this study is appropriate in patients with radiographic evidence of an intraluminal lesion.

Upper Gastrointestinal Tract Barium Study. This has limited value in the evaluation of patients scheduled for gynecologic surgery. It may be useful in patients with Krukenberg tumors (Figure 7-1).

Small-Bowel Barium Study. This study is appropriate in selected patients, generally those with gynecologic malignancies (Fig. 7-2). In ovarian carcinoma, up to 25% of patients may develop intestinal obstruction because of the disease. Early in the disease process, serosa and small-bowel mesentery may be involved. Small-bowel studies may also be of value in evaluating pa-

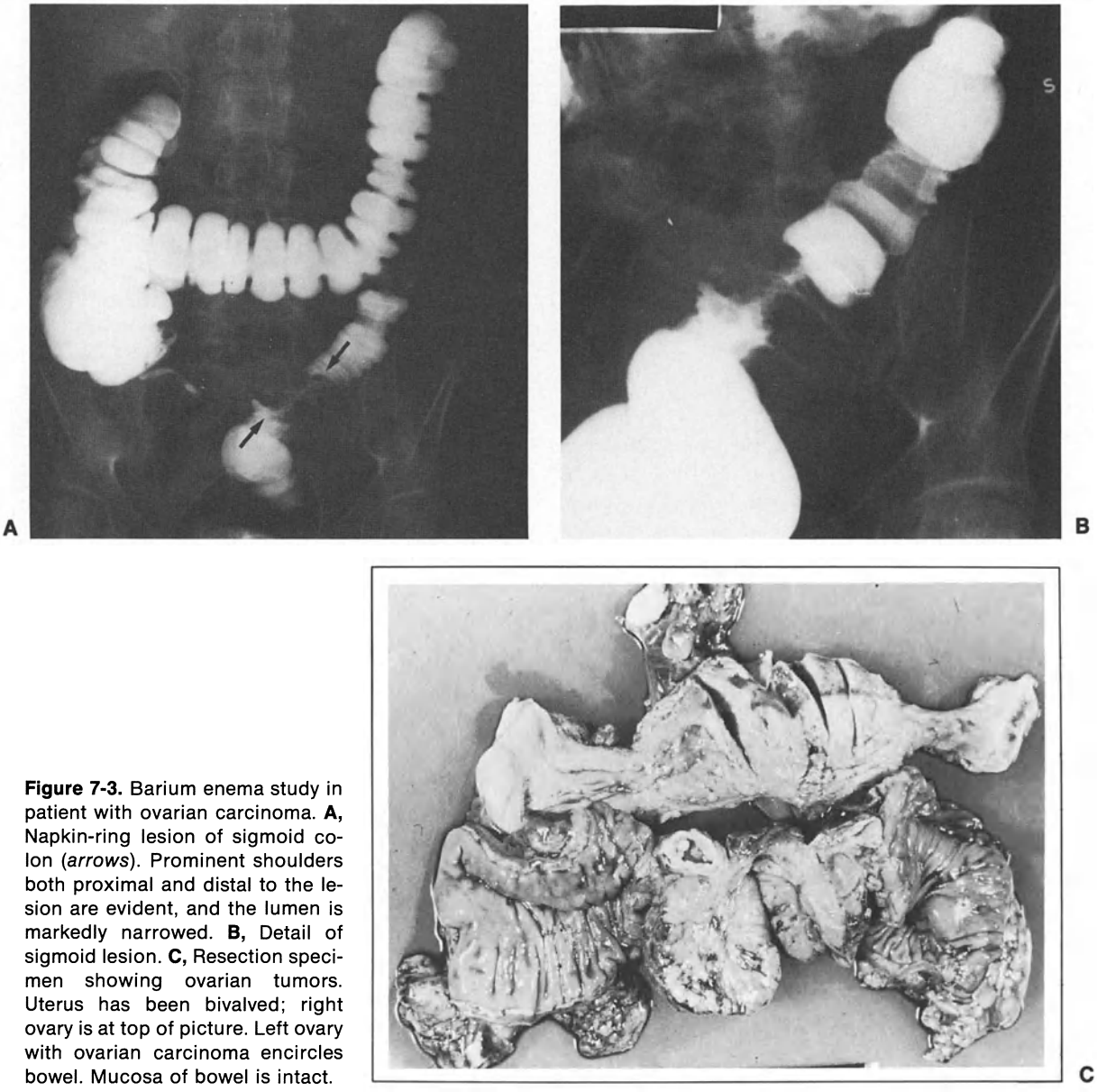


Figure 7-3. Barium enema study in patient with ovarian carcinoma. **A,** Napkin-ring lesion of sigmoid colon (*arrows*). Prominent shoulders both proximal and distal to the lesion are evident, and the lumen is markedly narrowed. **B,** Detail of sigmoid lesion. **C,** Resection specimen showing ovarian tumors. Uterus has been bivalved; right ovary is at top of picture. Left ovary with ovarian carcinoma encircles bowel. Mucosa of bowel is intact.

tients with cervical carcinoma. Farmer and Hawk[1] found 5 of 14 metastatic tumors of the small bowel to be from a primary carcinoma of the cervix. Buchsbaum[2] reported small-bowel involvement in 8 of 150 patients (5%) with cervical carcinoma evaluated by pretreatment celiotomy. The patients with small-bowel metastases represented one-half of patients with visceral metastases.

Barium Enema (Double-Contrast Study). High-density barium is instilled to the midtransverse colon. The barium is then evacuated by gravity drainage and air instilled. The barium is visible as a thin lining on the bowel mucosa. This study is far more sensitive than single-contrast studies, and is likely to pick up small polyps, early inflammatory changes, and malignancies (Figure 7-3). Where obstruction of the colon is suspected, a standard single-contrast study should be obtained. Severini and colleagues[3] evaluated double-contrast barium studies in patients with ovarian carcinoma. Serosal involvement was noted in the studies of 9 of 21 (42.8%) new patients, 8 of whom had advanced disease.

Ultrasonography. Ultrasonography is of only limited value for evaluation of gastrointestinal involvement by gynecologic disease processes. The thickness of the bowel wall, as determined by ultrasonic examination, may suggest inflammatory disease or primary and metastatic malignancy.[4]

Computed Tomography. These studies (CT) are of only limited value in preoperative evaluation of the gastrointestinal tract.

Bowel Preparation

If intestinal surgery is anticipated, based on clinical history, physical examination, or radiographic studies, mechanical and antibiotic bowel preparation should be instituted preoperatively. There is no longer any doubt about the value of mechanical bowel preparation with diet, enemas, and cathartics in reducing fecal bulk and bowel flora, combined with antibiotics in reducing the incidence of postoperative infectious complications in patients undergoing bowel resection.

In 1975, Crapp and associates[5] reported on bowel preparation utilizing whole gut irrigation. They used 9–12 liters of isotonic saline administered by mouth to mechanically clean the gut. The fluid was administered at a rate of 3–4 liters/hr until the effluent contained no feces. In their study of 81 patients, the mean volume of irrigation fluid was 9.5 liters and the patients experienced a mean weight gain of 1.9 kg. Davis and co-workers[6] later described a balanced electrolyte solution for this purpose (polyethlene glycol–electrolyte), which is now commercially available as Golytely and Colyte. These solutions reduce the likelihood of fluid retention, as well as potassium loss, making this type of preparation better tolerated by the elderly with cardiac or renal disease.

Other investigators have tried elemental diets utilizing Vivonex for 5 days combined with enemas to reduce fecal bulk, without any clear-cut advantage over traditional preparation.[7] Irvin and Goligher[8] documented the local benefit of appropriate bowel preparation: The incidence of anastomotic disruption was 24% in poorly prepared bowel, and only 7% in the well prepared colon.

Mechanical preparation of the bowel should be accompanied by antibiotic preparation. Controlled clinical studies have documented the value of antibiotic bowel preparation in reducing postoperative sepsis, peritonitis, and wound infection.[9–11] Orally administered and poorly absorbed antibiotics effectively reduce the bowel flora. Appropriately selected antibiotics are effective and carry a reduced likelihood of systemic drug toxicity, are unlikely to be used in other settings, are cheap, and carry a reduced risk of overgrowth of resistant bacteria because of short-term use. The drugs used in the bowel prep must cover both aerobic and anaerobic organisms. The use of mechanical and oral antibiotics has reduced the wound infection rate from 40% to 10%.

The standard bowel preparation used on the Gynecologic Oncology service at Magee–Womens Hospital is shown in Table 7-2. The regimen utilizes both mechanical preparation and the oral administration of antibiotics. Sustacal is given for nutritional support, and intravenous fluids are administered for hydration and electrolyte replacement. We utilize the whole gut irrigation in patients where bowel surgery is a great likelihood, for example, exenteration with transverse colon conduit.

Most recently, parenteral antibiotics have been tested either as substitutes for orally administered agents or with them. These studies were recently reviewed by Wilson and Sokol.[12] It appears that the addition of cephalosporins to mechanical preparation and oral antibiotics further reduces infectious complications following bowel surgery.

The Bowel in Surgery

Abdominal Incision. Avoidance of injury to the bowel during gynecologic surgery begins with choice of the abdominal incision (Chapter 2). In patients who have had prior surgery, it is wise to select an incision removed from the original one or to extend the earlier incision into the upper abdomen to avoid loops of small bowel that may be adherent to the anterior parietal peritoneum. Ratcliff and colleagues[13] found that in 15% of patients with small-bowel obstruction following gynecologic surgery, the site of obstruction was an adhesion to the anterior abdominal wall, suggesting that a far

Table 7-2. Preoperative Bowel Preparation

Pre-op day 3
1. Clear liquid diet (2000 ml)
2. Sustacal (12 oz 4 times daily)
3. Enemas at bedtime
4. Ducolax 5-mg tabs h.s.

Pre-op day 2
1. Clear liquid diet
2. Sustacal (12 oz × 14)
3. Enemas until clear h.s.
4. Magnesium citrate 150 ml b.i.d.

Pre-op day 1
1. Clear liquid diet, n.p.o. after 1600 hr
2. IV infusion started at 1600 hr; 2000 ml until surgery
3. Enemas until clear at 1100 hr
4. Magnesium citrate 150 ml b.i.d.
5. Neomycin 1 g and erythromycin base 1 g orally at 1300, 1400, 1800, and 2000 hr

greater number of patients have less severe adhesions at this site.

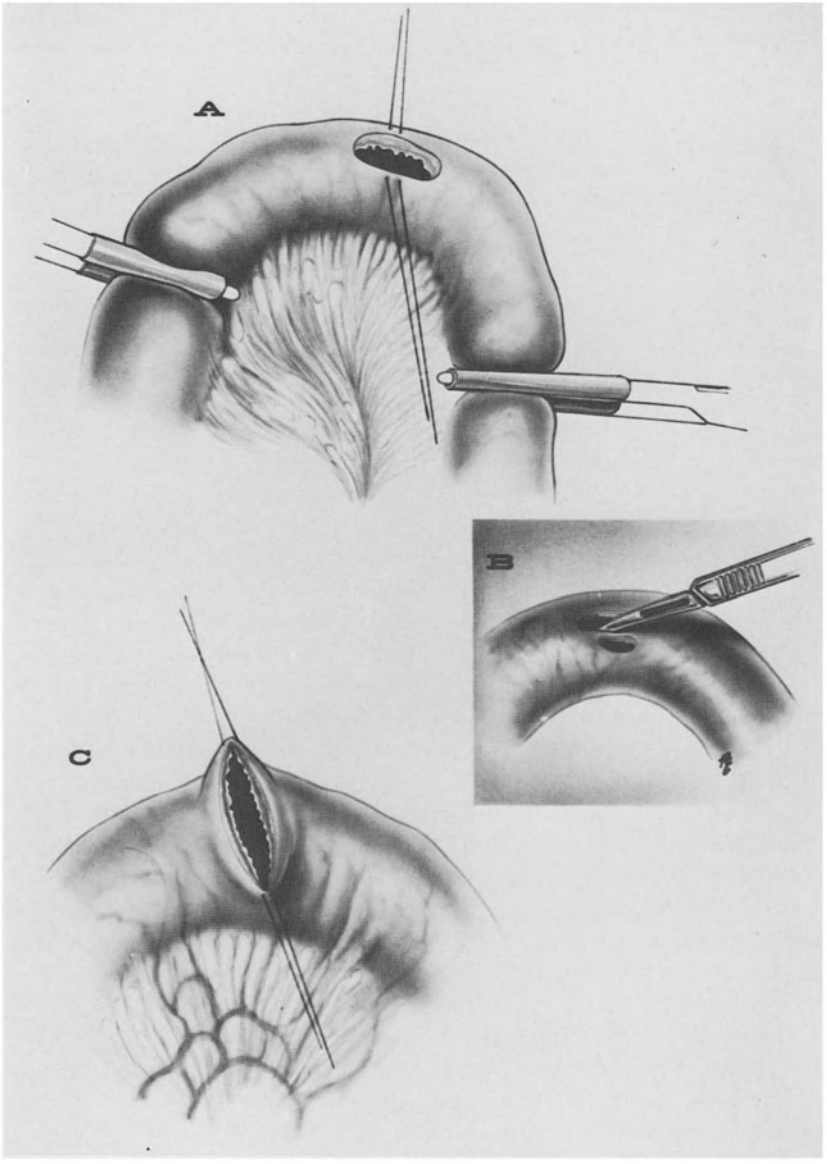
Lysis of Adhesions. When the peritoneal cavity is entered above the old incision, the bowel is protected and the adhesions are easily lysed. Bowel can become adherent to the parietal peritoneum, to pelvic viscera, or to other loops of bowel. Filmy adhesions can be broken by blunt dissection with the thumb and forefinger; fibrous bands and adherent loops of bowel are best separated by sharp dissection. Adhesions in patients who have received radiation therapy may be thick and fixed, obliterating tissue planes. The bowel may be anoxic and easily traumatized, and the dissection must be carried out with sharp instruments. In separating loops of small bowel, the surgeon must continually identify bowel mesentery and layers of the bowel wall. A scalpel may be required to establish a plane of dissection.

Should the bowel be distended, decompression can be accomplished by passing a large-bore needle through a pursestring suture and attaching the needle to low suction. The pursestring suture is tied as the needle is withdrawn. If extensive adhesions are found, it is wise to have the anesthesiologist pass a nasogastric tube into the stomach; the position of the tube is checked by the surgeon.

If extensive adhesions are encountered and the dissection results in denudation of considerable small-bowel serosa, use of a Baker tube should be considered. The Baker tube is a long, double-lumen intestinal tube (~3 m) with an inflatable bulb at the tip. The tube is passed into the intestinal tract via a gastrostomy or jejunostomy incision. The stomach or small bowel is fixed to the anterior parietal peritoneum. The balloon is then partially inflated and mechanically advanced through the small intestine until the tip is in the cecum. The balloon is fully inflated, fixing the catheter at this position. The relatively rigid tube avoids sharp angulation of the bowel by new adhesions. The tube is left in place for 8–10 days; the balloon is deflated before the tube is removed.

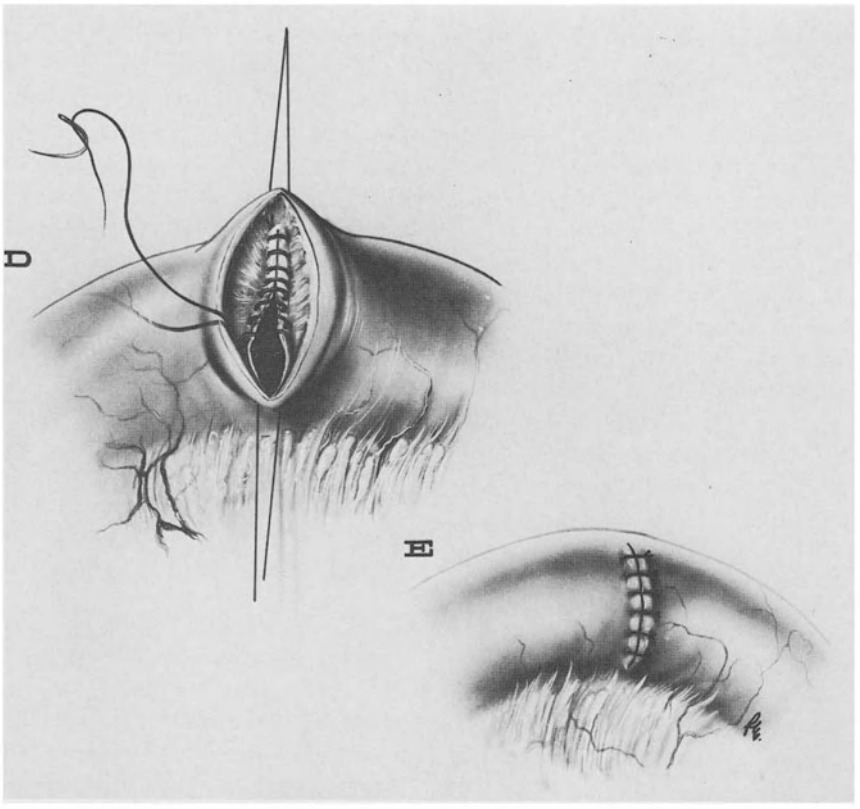
Examination of Bowel. On entering the peritoneal cavity, or after adhesions have been lysed, the surgeon should visually and palpably examine the entire small and large bowel and their mesenteries. A convenient way to start this systemic examination is for the surgeon (standing on the patient's left side) to run his left hand, palm up, cephalad from the pelvis until he encounters the cecum. The cecum is secondarily fixed and presents an easy landmark. The appendix is next: the serosa is examined for hyperemia, and the appendix palpated to rule out a fecalith or tumor. The ileocecal junction should next be identified, and the small bowel examined. The examination proceeds proximally, including the mesentery, to the ligament of Treitz. The small-bowel mesentery should be palpably examined to detect any enlarged lymph nodes. The examination may also disclose a Meckel's diverticulum (see below) approximately 2 ft from the ileocecal junction.

Examination of the colon is next carried out in an antegrade fashion beginning at the cecum. The ascending colon is mobile and lends itself to careful visual examination; the colon should also be carefully palpated to identify solid intraluminal tumors. The hepatic flexure is fixed and immobile; the transverse colon, suspended by its mesentery, can often be brought out through a vertical lower abdominal incision. The splenic flexure is also fixed; the descending colon and sigmoid are more mobile. The descending colon and sigmoid must be palpated for intraluminal tumors, as well as visually examined for the presence of diverticuli.



Packing the Bowel. Prior to beginning pelvic surgery, the bowel must be packed out of the operative field. The Trendelenburg position, head down with the hips extended, allows the small bowel to recede into the upper abdomen by gravity and brings the pelvis into better view. In using laparotomy pads, the surgeon should attempt to pack the bowel in as atraumatic a fashion as possible. Laparotomy pads abrade the surface of the bowel, and this effect may prolong postoperative ileus. Three laparotomy pads are all that are required to provide adequate exposure for pelvic surgery with the patient in the Trendelenberg position. One folded pad is placed in each gutter and the third is used to displace the bowel. If the patient has a large omentum it can be interposed as a buffer between the bowel and the last laparotomy pad, thereby sparing the small bowel trauma.

Small-Bowel Injury. If the small bowel is injured during lysis of adhesions or during performance of gynecologic surgery, the defect should be repaired immediately. Injuries to the serosa need not be repaired; those involving serosa and muscularis should be repaired with 4/0 synthetic absorbable suture material



◀ **Figure 7-4.** Repair of small-bowel injury. **A,** Segment of injured bowel isolated between two noncrushing clamps. Sutures of 4/0 silk have been placed at the lateral borders of the defect. **B,** Two defects close to one another converted to a single defect for repair. **C,** Tension on stay sutures converts longitudinal wound to a transverse one. **D,** Beginning repair. First layer: Connell suture of 3/0 absorbable synthetic suture is used to close mucosa. **E,** Completed repair. Second (seromuscular) layer closed with Lembert sutures of 4/0 silk or 4/0 absorbable synthetic material. From Buchsbaum HJ, Lifshitz S. Bowel complications in gynecologic surgery. In: Wynn RM, ed. Obstetrics and gynecology annual, 1976. New York, Appleton-Century-Crofts, 1976, pp. 222-223. Used with permission.

(polyglycolic acid or polyglactone sutures) on an intestinal needle. Small, full-thickness defects of the bowel wall (up to 2–3 mm in diameter) can be repaired with a pursestring suture. Larger defects should not be closed in this fashion, since the repair may compromise the lumen of the bowel. When larger defects are evident, noncrushing clamps are placed proximal and distal to the injury to immobilize the loop of bowel and prevent further spillage of bowel content (Figure 7-4A). Repair should always be carried out perpendicular to the axis of the bowel. Two small defects close to one another are best converted to a single wound and closed (Figure 7-4B). Stay sutures are placed at the angles to convert a longitudinal into a transverse wound (Figure 7-4C). A single layer closure generally suffices in the small bowel. In heavily irradiated or indurated tissue, a two-layered closure is appropriate, utilizing a running Connell or Cushing suture of 4/0 synthetic absorbable suture material for the mucosa and interrupted Lembert type sutures of similar material for the seromuscular layer (Figures 7-4D and 7-4E).

The bowel mesentery must be carefully examined. Injuries perpendicular to the axis of the bowel (parallel to the mesenteric vessels)

generally offer no problem; those parallel to the axis of the bowel must be carefully examined to insure that the blood supply to the intestine has not been compromised. When repair of the mesentery is required, small shallow bites of tissue should be taken on both sides of the defect. This requires a second row of sutures on the opposite side of the mesentery.

When speed is essential in repairing a small-bowel injury, a stapler may be used. The same principles apply as in suture repair: the staple line must be at right angles to the axis of the bowel. The edges of the laceration are elevated by stay sutures and the incision closed with a TA-30 stapler utilizing 3.5 staples (United States Surgical Corporation). The excess tissue is excised.

Large-Bowel Injury. Injuries to the colon in gynecologic surgery occur most commonly to the rectosigmoid in operations for pelvic inflammatory disease, endometriosis, and ovarian cancer. Less commonly, a bowel lesion can involve the genital structures; for example, ruptured appendiceal abscess, ruptured diverticulum.

As with small bowel-lacerations, small defects can be closed with a reinforced pursestring suture of 3/0 or 4/0 synthetic absorbable material. The pursestring suture is first placed and tied; a Lembert suture is then placed to reinforce the first suture. Larger lacerations require a two-layered closure, with the suture line perpendicular to the axis of the bowel. Recently, Entner and Heimberger[14] reported excellent results in both elective and emergent colon surgery with a single-row anastomosis in patients who had antibiotic prophylaxis. A proximal diverting colostomy is rarely indicated after surgical repair of colonic injury, even when resection and anastomosis are performed. A colostomy is done only when there is concern about the integrity of the suture line or in the face of extensive inflammation.

Appendectomy. The diagnosis of acute appendicitis continues to be an enigma to the clinician. The signs, symptoms, and physical findings may be confused with those of pelvic inflammatory disease in the young patient. The gynecologist may have to perform an appendectomy for acute appendicitis in a patient explored for suspected gynecologic pathology. In this situation, there is no debate as to what to do. Greater controversy has surrounded the performance of incidental appendectomy at the time of celiotomy for gynecologic pathology. Mattingly and Thompson[15] give the cogent reasons supporting incidental appendectomy:

1. To reduce mortality and morbidity from appendicitis, including infertility following perforated appendix
2. To eliminate undiagnosed incidental pathology in the appendix
3. To eliminate the appendix from diagnostic consideration when the patient has abdominal or pelvic complaints in the immediate postoperative period and in future years

The main arguments raised against incidental appendectomy at the time of a gynecologic surgical procedure are that (1) it is inappropriate to remove an apparently normal structure; (2) it prolongs the operative procedure; (3) it increases the postoperative morbidity, and (4) it potentially increases the surgeon's liability. These points are discussed below.

1. The "normal"-appearing appendices removed incidentally at the termination of gynecologic procedures are found to contain considerable pathology when examined. The incidence of gross and microscopic pathology in the appendix removed incidentally ranges from 10% to over 25%. Taniguchi and Kilkenny[16] reported that over 10% of appendices revealed catarrhal appendicitis and 0.9% showed subacute or early appendicitis, while Waters[17] found abnormalities in 22% of the appendices removed at the time of gynecologic surgery. A strikingly higher figure was reported by Melcher[18], who found that only 12 of 45 (27%) of the appendices were *normal* following incidental removal at the time of abdominal hysterectomy. In addition to inflammation, the appendix can be the seat of carcinoid tumor, and less frequently, a Krukenberg tumor.

2. Removal may prolong the operation for 10–15 min. This represents less than 15% of the total operating time, an insignificant prolongation.

3. In reported series of gynecologic procedures, the performance of incidental appendectomy did not add to the postoperative morbidity or prolong hospitalization.[16–19] In fact, Waters[17] reported a higher incidence of postoperative temperature elevation among patients who did *not* have appendectomy, compared to those who did.

4. A discussion of the medical liability is beyond the scope of this chapter. In this litigious society the physician can be found as equally liable for excising as for not excising the appendix.

In summary, we feel that appendectomy should be performed in conjunction with pelvic surgery, elective or emergent, when in the judgment of the surgeon the patient's condition is stable and the appendix is accessible, and the surgeon is trained and familiar with the procedure. Whether appendectomy should be performed at the time of tubal surgery for infertility is unresolved.

There are nearly as many ways of removing an appendix as there are surgeons. Time of training and geography are factors contributing to a gynecologic surgeon's technique in performing appendectomy. The mesoappendix is serially clamped and tied with absorbable synthetic sutures of 3/0. We prefer to excise the appendix at a crush site between a proximal tie of 2/0 chromic catgut and a distal straight hemostat. The suture on the stump is held with a curved hemostat, and a pursestring suture of 4/0 silk placed around the base of the appendix. The appendiceal stump is inverted and the pursestring suture tied.

Meckel's Diverticulum. Meckel's diverticulum is the most common gastrointestinal anomaly, with a reported incidence of about 2%. Although it was first described by Fabricius Hildanus in 1598, it was Johann Friedrich Meckel, a German anatomist, who described its embryologic development and clinical significance in 1808. It is a true diverticulum of the ileum, containing all layers of the gut wall, and arises on the antimesenteric side.

The stalk between the yolk sac and the embryologic midgut is the vitelline or omphalomesenteric duct. The yolk sac stops growing when the fetus is about 3.5 mm in size, and the duct starts regressing. By 5 weeks gestation the duct atrophies and undergoes resorption, freeing the gut from the abdominal wall. Failure of complete obliteration and absorption can result in a variety of anomalies. A fibrous band may connect the small bowel to the umbilicus if there has been obliteration without reabsorption. Persistence of the middle of the duct results in a vitelline cyst connected by a cord to both umbilicus and bowel. By far the most common anomaly, accounting for approximately 85%–95% of vitelline duct persistence, is Meckel's diverticulum, a true diverticulum.

A popular "rule of twos" has evolved regarding Meckel's diverticulum: It occurs in 2% of the population, is twice as common in men as in women, is located 2 ft from the ileocecal junction, is 2 in. in length, and contains two heterotropic types of tissue; there are two major types of complications. While this rule is easy to remember, it represents an oversimplification.

The reported incidence varies, depending on the population studied. The incidence in collected autopsy series is 1.2%–2.5%, while in surgical series it is 2.0%–4.5%.[20] Collins[21] reported the presence of a Meckel's diverticulum in 2% of 50,000 patients undergoing appendectomy. While the anomaly is twice as common in males as in females, in symptomatic cases the ratio favors males by 5% to 6.1%. In adults, the ratio is closer to 1 : 1.

The site of Meckel's diverticulum varies from 2 to 160 cm, most often 40–50 cm, from the ileocecal junction. The shape, length, and diameter vary considerably (Figure 7-5). The extremes of reported length are from 1 to 26 cm. Five, not two, different types of heterotropic mucosa may be found in the diverticulum: gastric, pancreatic, duodenal, colonic, and bile duct. Gastric mucosa is by far the most common; 85% have fundal (hydrochloric acid producing) glands.

Eighty percent of all individuals with Meckel's diverticulum are asymptomatic; in the symptomatic group, 50% of patients have symptoms before the age of 2 and 80% by 16 years of age. The most common clinical manifestation of Meckel's diverticulum is ulceration. Small-bowel obstruction is second most common, and can affect individuals at any age. Inflammation resulting in an appendicitis-like picture is most common in infants and chil-

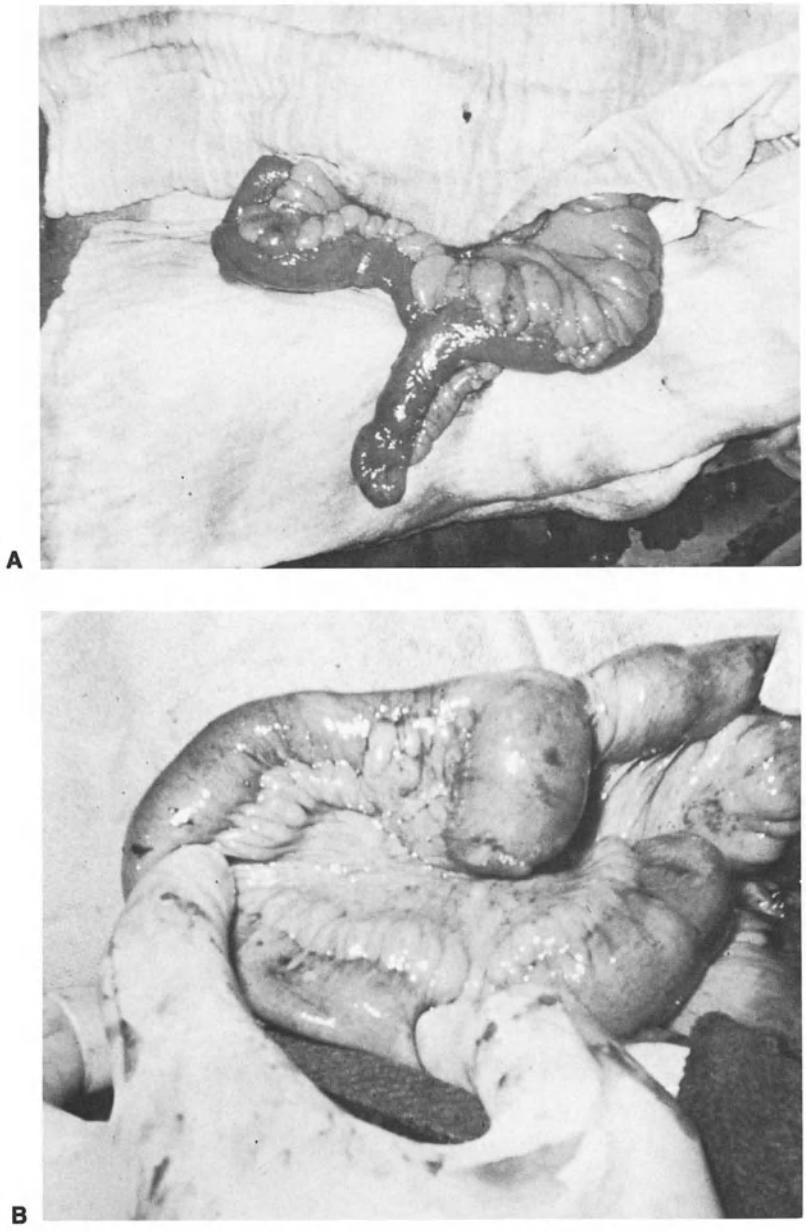


Figure 7-5. Meckel's diverticulum showing difference in size and shape. **A,** Diverticulum approximately 5 cm in length and 1.5 cm in diameter. **B,** Broad-based Meckel's diverticulum.

dren, while neoplasms (leiomyomata, fibroma, carcinoid, leiomyosarcoma, and adenocarcinoma) are rare (1.5%) and occur more commonly in adults.

Various techniques have been applied to the nonoperative detection, including contrast studies of the upper and lower gastrointestinal tracts (Figure 7-6), technetium and pertechnate scanning, antegrade small-bowel infusion, and arteriography. In adults, any single study is of limited value in establishing the diagnosis.[22]

The gynecologist who explores the upper abdomen and routinely examines the small bowel is likely to encounter this most common of gastrointestinal anomalies in the performance of gynecologic surgery.[23] He is then faced with the question of whether to excise the asymptomatic Meckel's diverticulum. It is our feeling that a Meckel's diverticulum found

incidentally at gynecologic surgery should be removed. In our experience, approximately 50% of the diverticuli excised will have ectopic mucosa and approximately 15% will have gastric mucosa. Neoplasms are more likely to arise in diverticuli in later life, and this pouch can be the leading edge of an intussusception or can result in a volvulus when there is a persistent band.

In the absence of an acute abdomen, overwhelming infection, or a prolonged operative procedure, diverticulectomy should be performed. It can be done either by suture technique (Figure 7-7) or with surgical staples (Figure 7-8). There is usually a dominant vessel, the remnant of the right vitelline artery, which must be isolated and ligated. In both techniques the suture line must be perpendicular to the axis of the bowel. Prior to excision, the surgeon should carefully examine and palpate the diverticulum to determine if there is a tumor present. The presence of a malignant tumor requires a more extensive procedure: removal of the distal ileum and ascending colon with ileotransverse colon anastomosis to remove the primary lymphatics.

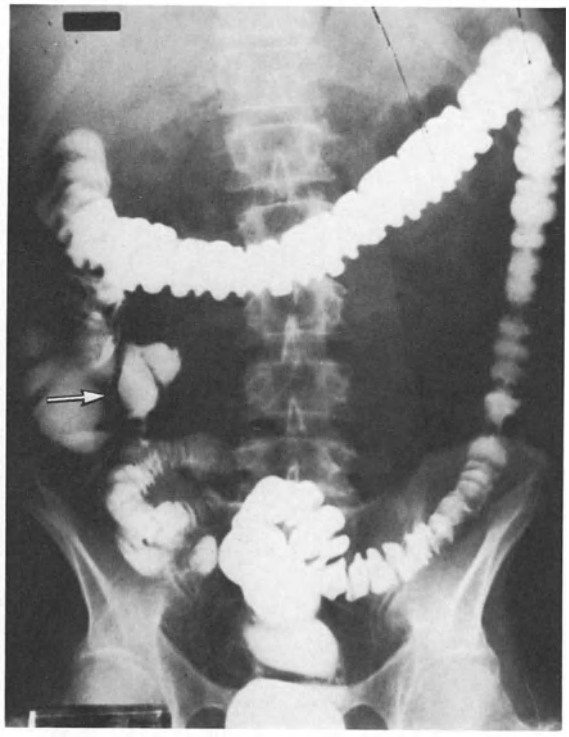
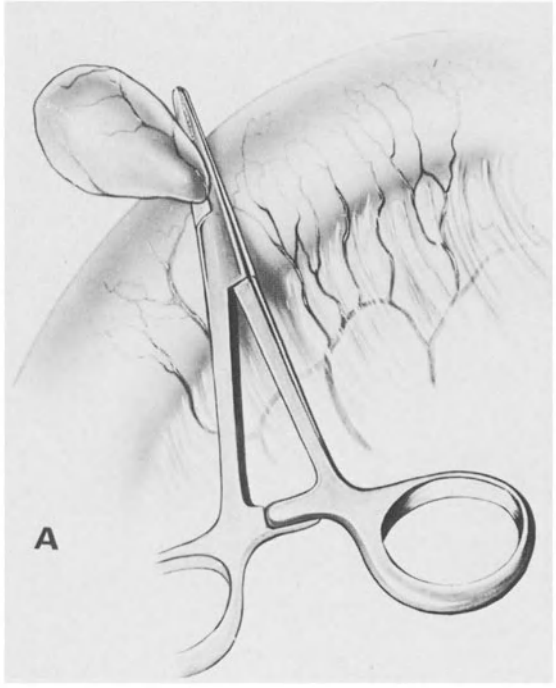


Figure 7-6. Barium enema in patient with Meckel's diverticulum. Retrograde filling of ileum reveals diverticulum (*arrow*).



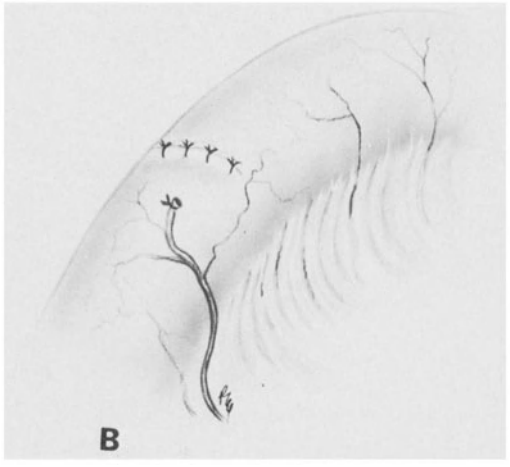
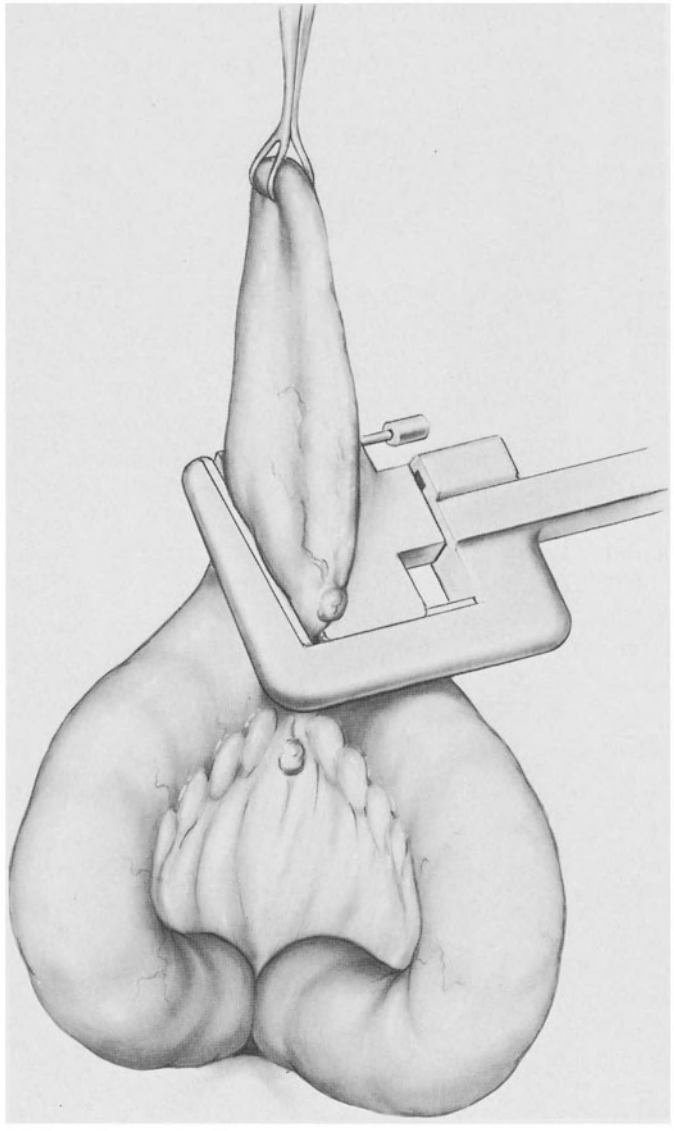


Figure 7-7. Excision of Meckel's diverticulum. Arterial supply to diverticulum has been isolated and ligated. **A,** Clamp placed across the diverticulum at right angles to the axis of the small bowel. **B,** Incision has been closed in one layer utilizing 3/0 absorbable synthetic suture. Reprinted with permission from The American College of Obstetricians and Gynecologists. (Obstetrics and Gynecology, 45, 1975, 311–314.)



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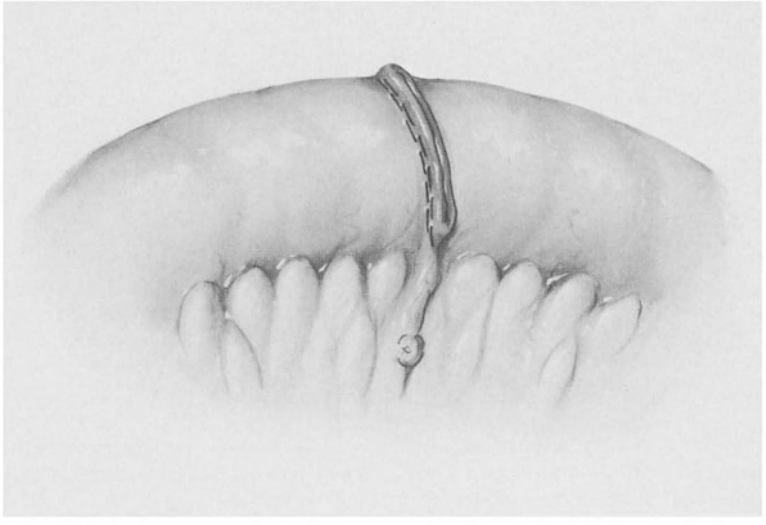

B

Figure 7-8. Diverticulectomy performed with staples. **A,** Meckel's diverticulum held with Babcock clamp. TA-30 stapler applied across base at right angles to axis of the ileum. **B,** Completed diverticulectomy shows staples perpendicular to axis of ileum and ligature on artery. Reprinted with the permission of U.S. Surgical Corporation.

Postoperative Problems

Paralytic (Adynamic) Ileus. Some degree of ileus follows most abdominal/pelvic operations. The severity and duration of the condition depend on the length and type of surgery, the amount of trauma the bowel has sustained, and the chemical or bacterial contamination of the peritoneal cavity. The pathophysiology of ileus is not completely understood. There is a lack of motor activity within the intestinal musculature, but electrical activity is present. The dysfunction appears to result from continuous activity of the intrinsic inhibitory neurons.[24] The patient may complain of bloating, abdominal discomfort, or pain 24–48 hr after surgery. She has no interest in food, and has not passed flatus. On physical examination the abdomen is found to be distended, with tympani to percussion and absent or markedly hypoactive bowel sounds. The patient may have a tachycardia, but vital signs are generally normal. There may be some shortness of breath or tachypnea resulting from elevation of the diaphragms.

The treatment of postoperative adynamic ileus involves withholding of food and administration of intravenous fluids. Rarely does this condition require placement of a nasogastric tube and mechanical decompression. Serum electrolytes should be monitored as a guide to fluid and electrolyte replacement. If there is no improvement, a nasogastric tube should be placed and attached to suction, and abdominal x-rays—supine (flat), upright (erect), and lateral decubitus films of the whole abdomen—should be obtained. The lateral decubitus films give better views and greater detail of the colon, particularly if there are full-length air–fluid levels. On the supine film there is distension of both small and large bowel, with a considerable amount of gas in the stomach. The erect and lateral decubitus film shows air–fluid levels throughout the small and large intestine. The air–fluid levels tend to be at about the same level (Figure 7-9; Table 7-3).

Attempts to stimulate the propulsive activity of the intestine with drugs are ineffective and may even be hazardous. The condition is self-limiting and generally resolves by the fourth

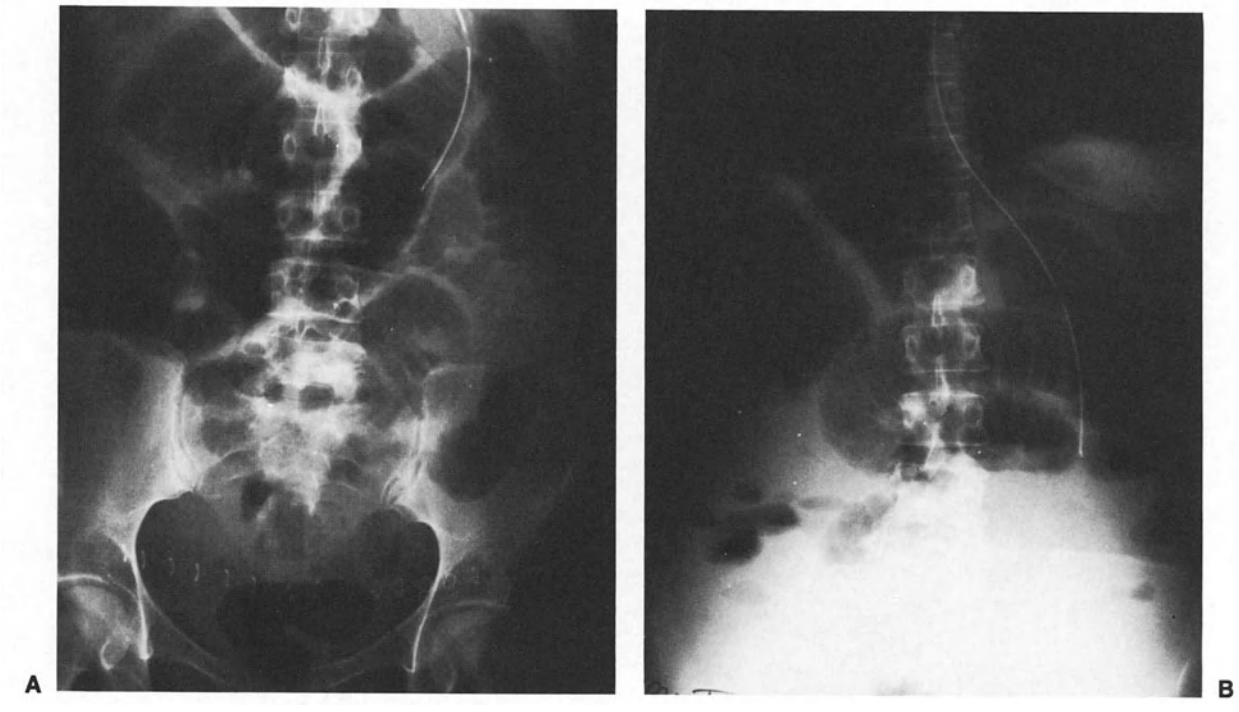

Figure 7-9. Fourth postoperative day. **A,** Supine film. Nasogastric tube is seen in stomach. Small and large bowel are distended in relative proportion to one another. **B,** Upright film. Air–fluid levels are evident at the same level in dilated loops of small bowel.

Table 7-3. Differential Radiographic Findings in Ileus and Mechanical Obstruction

Adynamic Ileus	*Mechanical Obstruction*
1. Small and large bowel are distended in proportion to each other	1. In small-bowel obstruction there is dilated small bowel proximal to site of obstruction. In colonic obstruction the colon is distended; small-bowel distension is present with incompetent ileocecal valve
2. Air–fluid levels in small bowel are infrequent; when present, they are at the same levels	2. Air–fluid levels are common and at different levels in the bowel
3. Quantitative difference in small-bowel distension	3. Greater small-bowel distension than with ileus
4. Small-bowel distension in central part of abdomen with colon in periphery	4. Small-bowel distension present in central part of abdomen; no peripheral large-bowel distension

postoperative day. Bowel sounds return and the patient's abdominal distension regresses as gas moves through the intestine and the patient passes flatus.

A rare form of paralytic ileus is segmental reflex ileus, which involves an isolated segment and can result from localized intra- or extraperitoneal infection: cuff abscess, subdiaphragmatic abscess, pancreatitis, or even basal pneumonia. The diagnosis can only be established by abdominal radiography, which reveals persistent distention of an isolated loop of small bowel. Correction of the underlying condition results in resolution of the ileus.

Mechanical Obstruction. Although mechanical small-bowel obstruction becomes clinically evident at a time remote from the surgery, it may manifest itself as early as 5–7 days postoperatively. The most common cause of small-bowel obstruction is adhesions. Ratcliff and colleagues[13] found that 83% of female patients with small-bowel obstruction had prior abdominal surgery. Of the patients with prior surgery, 33 of 49 (77.4%) had an obstetric or gynecologic procedure. Other causes of mechanical bowel obstruction are hernia, intussusception, volvulus, and tumor.

With postoperative mechanical small-bowel obstruction, the patient appears more toxic and the clinical picture is more acute than with ileus. The patient is lethargic, has colicky abdominal pains, and usually vomits. The colicky pains occur in rushes and result from active bowel peristalsis proximal to the site of obstruction, attempting to pass bowel contents past the site of obstruction. Occasionally the examiner may see the hyperactive peristaltic waves through the abdominal wall and hear the rushes (borborygmi) without a stethoscope. On auscultation of the abdomen, loud, high-pitched, metallic bowel sounds are heard just before the patient experiences the colicky pain. The time of onset of vomiting is related to the site of obstruction: early in proximal, and later in distal small-bowel obstruction. The patient may have bowel movements early in the course as she empties the small bowel and colon distal to the site of obstruction.

The findings of severe pain, tachycardia, temperature elevation, and leukocytosis are suggestive of infarction. If partial or complete small-bowel obstruction is suspected, radiographic studies should be ordered and repeated as frequently as necessary. The plain films generally reveal distension of the proximal bowel, with little or no gas evident in the colon. On upright or lateral decubitus films, multiple air–fluid levels may be seen at different levels in a given loop of bowel (Figure

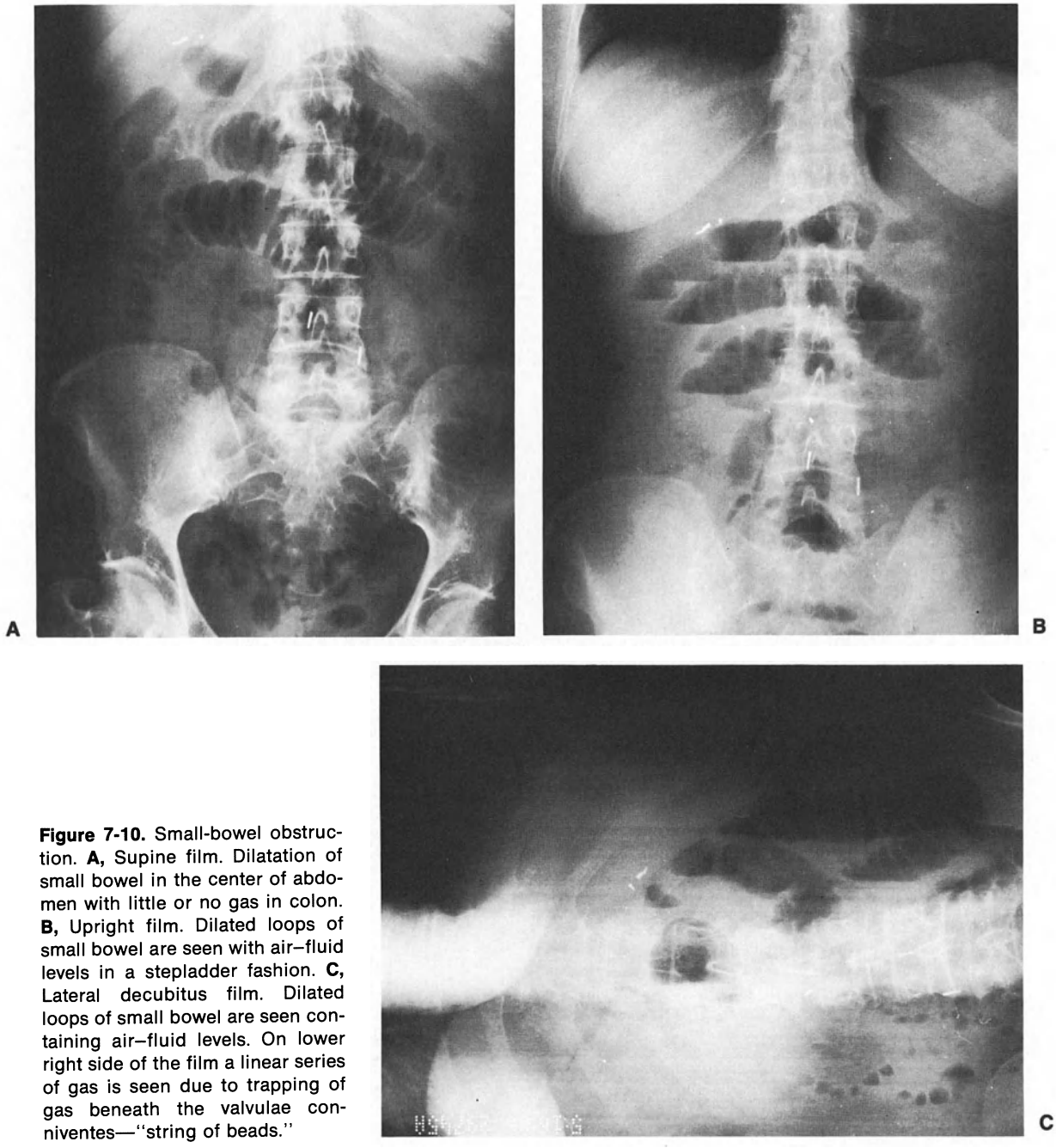

Figure 7-10. Small-bowel obstruction. **A,** Supine film. Dilatation of small bowel in the center of abdomen with little or no gas in colon. **B,** Upright film. Dilated loops of small bowel are seen with air–fluid levels in a stepladder fashion. **C,** Lateral decubitus film. Dilated loops of small bowel are seen containing air–fluid levels. On lower right side of the film a linear series of gas is seen due to trapping of gas beneath the valvulae conniventes—"string of beads."

7-10). If the history and physical findings suggest colon obstruction, a retrograde contrast study should be obtained.

Rarely, one may find multiple sites of mechanical obstruction in small and/or large bowel. We cared for a patient recently with recurrent cervical carcinoma who had obstruction at two sites in the small bowel as well as the transverse colon.[25] In postoperative partial or complete small-bowel obstruction, passage of a long intestinal tube (Miller–Abbott or Cantor) may obviate the need for surgery. If there is no suggestion of strangulation of small bowel, the patient can be managed conservatively so long as her condition does not worsen.

Wolfson and colleagues[26] studied 112 patients who had 127 episodes of small-bowel obstruction resulting from postoperative adhe-

sions and were managed conservatively with long-tube intubation. Critical to the outcome was success in passing the tube into the small bowel. When the tube passed the pylorus, only 11 of 20 (55%) patients with complete, and only 7 of 52 (13%) with partial small-bowel obstruction, required surgery. Overall, 62% of the patients in their series were spared an operation. Patients managed conservatively were hospitalized for about half as long as patients who underwent surgery. Although the authors developed a numerical system of "risk factors" indicative of strangulation, it has only limited application.

To facilitate passage of a long tube, the tip of the balloon should be positioned in the pylorus under fluoroscopic control[27] and the patient maintained on her right side. If the tube fails to pass into the duodenum with these methods, Kim[28] described a technique utilizing flexible endoscopic guidance. Once the tube has advanced (Figure 7-11), barium and water can be administered in an antegrade fashion to identify the site and degree of obstruction.

Since these long tubes have openings only at the distal end, once the tube has passed into the small bowel some patients require intermittent gastric suction to remove the 2000–3000 ml of saliva and gastric secretions that are produced every 24 hr. In mechanical obstruction, large volumes of fluid are pooled in the lumen of the intestine, and the patient requires careful fluid and electrolyte monitoring and replacement. Intermittent suction of the long tube allows the physician to better estimate fluid and electrolyte needs (Table 7-4). Furthermore, as a result of decompression of the bowel, the pathophysiologic conditions are interrupted: collection of fluid in the bowel lumen stimulates greater secretion. Although absorption from the bowel continues, the flow into the lumen exceeds that going out. If the distension is allowed to continue, blood flow to that segment of bowel is compromised. Since the vessels enter the intestinal wall tangentially, they become narrowed with increasing distension. Eventually the capillary flow is compromised, resulting in gangrene.

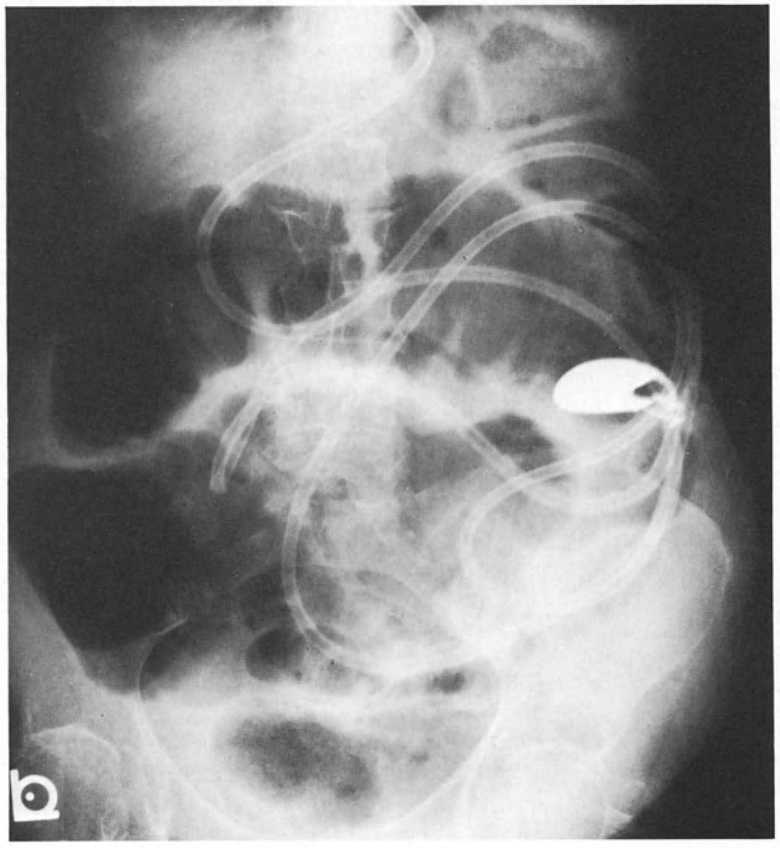

Figure 7-11. Supine film in patient with partial small-bowel obstruction managed by placement of long intestinal tube. Cantor tube tip is seen in ileum.

Table 7-4. Average Daily Volume and Electrolyte Concentrations of Gastrointestinal Secretions

	Volume (ml/day)	Electrolyte Concentrations (meq/liter)		
		Na^+	K^+	Cl^-
Saliva	1000–1500	10–40	10–20	6–30
Gastric juice	2000–2500	60–120	10–20	10–30
Hepatic bile	600–800	130–155	2–12	80–100
Pancreatic juice	700–1000	150–155	5–10	30–50
Duodenal secretions	300–800	90–140	2–10	70–120
Jejunal and ileal secretions	2000–3000	125–140	5–10	100–130
Colonic mucosal secretions	200–500	140–148	5–10	60–90
Total	8000–10,000			

References

1. Farmer RG, Hawk WA. Metastatic tumors of the small bowel. Gastroenterology. 1964; 47:496–504.
2. Buchsbaum HJ. Extrapelvic lymph node involvement in cervical cancer. Am J Obstet Gynecol. 1979; 133:814–824.
3. Severini A, Petrillo R, Kenda R, et al. The value of double contrast enema in the assessment of ovarian carcinoma's diffusion. Gynecol Oncol. 1981; 11:17–22.
4. Fleischer AC, Muhletaler CA, James AE Jr. Sonographic assessment of the bowel wall. AJR. 1981; 136:887–891.
5. Crapp AR, Tillotson P, Powis SJA, et al. Preparation of the bowel by whole gut irrigation. Lancet. 1975; 2:1239–1240.
6. Davis GR, Santa Ana CA, Morawski SG, et al. Development of a lavage solution associated with minimal water and electrolyte absorption or secretion. Gastroenterology. 1980; 78:991.
7. Cooney DR, Wassner JD, Grosfeld JL, et al. Are elemental diets useful in bowel preparation? Arch Surg. 1974; 109:206–210.
8. Irvin TT, Goligher JE. Aetiology of disruption of intestinal anastomosis. Br J Surg. 1973; 60:461–465.
9. Clarke JS, Condon RE, Bartlett JG, et al. Preoperative oral antibiotics reduce septic complications of colon operations: Results of prospective, randomized, double-blind clinical study. Ann Surg. 1977; 186:251–258.
10. Nichols RE. Use of prophylactic antibiotics in surgical practice. Am J Med. 1981; 70:686–692.
11. Condon RE, Bartlett JG, Greenlee H, et al. Efficacy of oral and systemic antibiotic prophylaxis in colorectal operations. Arch Surg. 1983; 118:496–501.
12. Wilson SE, Sokol T. Antimicrobials in elective colon surgery. Infect Surg. 1985; 8:609–618.
13. Ratcliff JB, Kapernick P, Brooks GG, et al. Small bowel obstruction and previous gynecologic surgery. South Med J. 1983; 76:1349–1350.
14. Entner M, Heimberger H. One row anastomosis in colonic operations with antibiotic prophylaxis. Surg Gynecol Obstet. 1985; 161:136–138.
15. Mattingly RF, Thompson JD. TeLinde's operative gynecology. Philadelphia: JB Lippincott, 1985: chapter 20, p. 481.
16. Taniguchi T, Kilkenny GS. Prophylactic appendectomy in gynecological surgery. Am J Obstet Gynecol. 1950; 60:1359–1362.
17. Waters EG. Elective appendectomy with abdominal and pelvic surgery. Obstet Gynecol. 1977; 50:511–517.
18. Melcher DH. Appendectomy with abdominal hysterectomy. Lancet. 1971; 1:810–812.
19. Loeffler F, Stearn R. Abdominal hysterectomy with appendectomy. Acta Obstet Gynecol Scand. 1967; 46:435–443.
20. Gray SW, Skandalakis JE. Embryology for surgeons. Philadelphia: WB Saunders, 1972: chapter 5.
21. Collins DC. A study of 50,000 specimens of the human vermiform appendix. Surg Gynecol Obstet. 1955; 101:437–441.
22. Schwartz MJ, Lewis JH. Meckel's diverticulum: Pitfalls in scintigraphic detection in the adult. Am J Gastroenterol. 1984; 79:611–618.
23. Buchsbaum HJ. Meckel's diverticulum. Obstet Gynecol. 1975; 45:311–314.
24. Wood JD. Physiology of the enteric nervous system. In: Johnson RL, ed. Physiology of the gastrointestinal tract, Vol. 1. New York: Raven Press, 1981: chapter 1, p. 1–37.
25. Christopherson WA, Voet R, Buchsbaum HJ.

Recurrent cervical cancer presenting as small bowel obstruction. Gynecol Oncol. 1985; 22:109–114.
26. Wolfson PJ, Bauer JJ, Gelernt IM, et al. Use of the long tube in the management of patients with small-intestinal obstruction due to adhesions. Arch Surg. 1985; 120:1001–1006.
27. Shipps FC, Saylor CB. Fluoroscopic placement of the intestinal tubes. Radiology. 1979; 132:226–227.
28. Kim IG. Endoscopic guided intubation of long nasointestinal decompression tube. Surg Gynecol Obstet. 1985; 161:283–284.

Prophylactic Antibiotics for Abdominal and Vaginal Hysterectomy

8

David L. Hemsell

Adoption of the Diagnosis-Related Groups (DRG) concept has altered many aspects of health care, and may be the impetus necessary to alter the overutilization of antimicrobials for the prevention of major infection after hysterectomy. There is no disagreement about the necessity to prevent infection in the pelvis or in the abdominal wound following hysterectomy. Hospital stay may be prolonged for up to 2 weeks if an infection develops. Because of the pathogens involved, abscesses may form and a second operative procedure may be required. Gynecologic surgeons are interested in a zero percent incidence of infection. Even *with* antimicrobial prophylaxis, this standard is usually not attainable. Major infection after hysterectomy with prophylaxis is between 5% and 10%. If the incidence of major infection *without* prophylaxis is within this range, prophylaxis is not indicated, unless the gynecologist can identify a high-risk group of patients.

According to DiPiro et al.,[1] "clean contaminated" surgical procedures (see Table 4-1, Chapter 4), including hysterectomy, account for approximately 16% of all operations and are associated with a wound infection rate of about 10%. Hysterectomy, however, was the major surgical procedure most frequently performed on women of reproductive age during the last decade.[2] In agreement with data reported by DiPiro et al. are other studies with infection rates as low as 0.4%[3] or 1.6%.[4] In contrast, Ledger and Child[5] reported that 54% of 3,564 women after vaginal hysterectomy and 45% of 8,462 women after abdominal hysterectomy were treated for infection. In prospective, randomized controlled studies of abdominal hysterectomy without prophylaxis, wound and pelvic infection ranged from 4%[6] to 50%;[7,8] after vaginal hysterectomy, it ranged from 4%[9] to 64%.[10]

Major infection obviously prolongs hospital stay and increases health care costs. Swartz[11] and Shapiro and co-workers[12] reported that temperature elevations alone (febrile morbidity) after hysterectomy significantly increased hospital costs. Falk and Bunkin[4] reported that 21.2% of 500 women in their study developed febrile morbidity, while only 8 women (1.6%) required treatment. These data were similar to those reported by Gray.[3] The incidence of febrile morbidity in Gray's report was 20.8% of 2,421 women, but only 9 women (0.4%) required treatment. The wide range in the incidence of infection underscores the necessity of identifying the source of temperature elevation and the incidence of infection in each hospital.

Risk Factors

Although it is commonly believed that all premenopausal women undergoing vaginal hysterectomy require prophylaxis, available data from controlled studies indicate that antimicrobial administration did not uniformly lower the infection rate.[6,9,13–15] Many reports confirm the lack of efficacy of prophylactic antimicrobials in preventing major infection after abdominal hysterectomy.[6,9,16–24] If all women do not need prophylaxis, is it possible to identify a

group of women who are at increased risk for the development of major postoperative infection?

In the mid-1960s, Taylor and Hansen[25] and Pratt and Galloway[26] reported increased morbidity, including febrile morbidity and major infection, after vaginal hysterectomy in younger women. Shapiro and co-workers[27] confirmed these findings in their report in 1982. They reported that infections occurred more frequently in younger women after both vaginal and abdominal hysterectomy. Others have reported that menopausal status had no impact on the incidence of infection after vaginal hysterectomy,[22,28–33] that is, the younger patient is not at increased risk after vaginal hysterectomy[9,19,30,34,35] or abdominal hysterectomy.[9] Osborne and colleagues[36] provided a bacteriologic explanation for these data when they reported no significant differences in the number or type of bacterial species isolated from the cervix or vagina of premenopausal and postmenopausal women.

Thadepalli and colleagues[37] reported that the major infection rate was higher after hysterectomy if the operation was performed during the proliferative phase of the menstrual cycle. Others have reported a higher incidence of morbidity if the hysterectomy was performed during the secretory phase.[29,38] Several authors have shown that the phase of the menstrual cycle was not a risk factor for infection after either abdominal or vaginal hysterectomy.[19,28]

Gallup[39] reported that modification of celiotomy techniques in obese women significantly decreased wound morbidity from 42.2% to 3.1%. Factors that were associated with an increased abdominal wound infection rate were older patient, preoperative shaving, use of Penrose-type or passive drains, and duration of the operation. In contradiction, Grossman et al.[19] and Shapiro and colleagues[27] did not identify obesity as a risk factor. Obesity was shown to increase the risk for infection after abdominal hysterectomy,[40] but not after vaginal hysterectomy[41] when studied by a single investigator. Shapiro and co-workers[27] found that increased operative time was associated with an increased infection rate after vaginal hysterectomy. By contrast, Roberts and Homesley[9] found that increased operative time did not increase febrile morbidity in women undergoing vaginal or abdominal hysterectomy.

Pratt and Galloway[26] stated that there is an increased infection rate when additional procedures are performed at the time of hysterectomy, especially in women over 60 years of age. An increased infection rate with added procedures has not been supported by other reports.[16,19,26,28–32,35,36,42] The incidence of postoperative infection is not higher when a resident surgeon rather than an attending surgeon performs the hysterectomy.[27,28,43,44] When the same physicians provided surgical care to both private and indigent patients, the latter were at increased risk for major infection following hysterectomy.[19,27,45] Increased blood loss was not a factor associated with increased infection rates after hysterectomy.[9,27–29] Prolonged catheter usage after hysterectomy may increase the risk for urinary tract infection. Short-term usage is not associated with bladder infection in the absence of a previous infection.

Case Studies

Over 1,900 women undergoing vaginal and abdominal hysterectomy in our hospital were prospectively evaluated for efficacy and safety of prophylactic antibiotic regimens. Temperature elevation alone (febrile morbidity), menopausal status, age, phase of the menstrual cycle, obesity, prolonged operative time, additional surgical procedures, increased blood loss, and catheter usage have not emerged as factors increasing the risk for the development of major postoperative infection.[46–52] Younger age was found to be a risk factor in one of our studies, and that risk was seen in women who were given placebo at the time of abdominal hysterectomy.[48] In a recent study of 225 women undergoing either vaginal or abdominal hysterectomy, it was the older woman who was at greater risk for the development of major pelvic and/or wound infection.[51] In one study of 193 women undergoing vaginal hysterectomy, the incidence of pelvic infection requiring antibiotic therapy was increased with additional surgical procedures, but febrile morbidity alone was not increased.[47]

Poor surgical technique can contribute to the development of infection after hysterectomy. "Mass ligation of tissue, collection of blood and serum in the pelvic structures or anterior abdominal wall, and loud and boisterous conversation at the operating table" were observed as factors increasing the morbidity rates.[53] "Morbidity caused by pelvic infection was found to be more closely correlated with errors in operative technique than with the age or endometrial histology of the patient."[38] The above data demonstrate that theoretical considerations are not always confirmed by clinical trials, and that the administration of prophylactic antibiotics needs to be individualized. Gentle handling of tissues, careful attention to hemostasis, avoidance of dead spaces, and avoiding large pedicles are important surgical techniques in preventing infection. The use of prophylactic antibiotics cannot compensate for poor surgical techniques.

The use of prophylactic antibiotics at hysterectomy may be difficult to defend. As indicated earlier, there is much less controversy about prophylaxis for vaginal hysterectomy than for abdominal hysterectomy. While prophylaxis seems to be more effective in preventing major infection after vaginal hysterectomy, the abdominal approach increases the risk for major postoperative infection; this was noted when both surgical procedures required antimicrobial prophylaxis and both populations were studied by the same investigator.[6,16,17,19,22,24,33,42,43,51,54] The reasons for this finding may relate to the fact that there are two incisions rather than one, that more serious conditions necessitate the abdominal approach, and that the patients are usually older. In addition, contamination with lower reproductive tract flora occurs later in abdominal than in vaginal hysterectomy, and therefore tissues are exposed to bacteria for a shorter period of time. In addition, irrigation of the pelvis before closure and of the abdominal incision should reduce the bacterial inoculum.

Theoretically, these factors should be protective for abdominal hysterectomy patients when compared to those undergoing vaginal hysterectomy. These theoretical considerations are not substantiated by clinical trials, however. The severity of the patient's illness is a major factor. Debilitating diseases, immunosuppression, and malnutrition all predispose a woman to infection following hysterectomy. Host-related factors may well be the most important variable in the development of major infection following hysterectomy.

Historical Perspective

The concept of prophylaxis is certainly not new. Turner[55] reported in 1950 that 100,000 units of penicillin G in a cocoa-butter suppository inserted intravaginally 12–14 hr before vaginal hysterectomy reduced the postoperative febrile morbidity rate from 37.5% to 7%. The reported incidence of febrile morbidity at other institutions during the same period ranged from 26% to 42%. In a comprehensive report of 1,000 women undergoing both abdominal and vaginal hysterectomy, Cron and colleagues[53] reported that a Zephiran-saturated pack inserted into the vagina the night preceding surgery did not significantly alter febrile morbidity after either procedure. A penicillin suppository identical to that used by Turner inserted intravaginally the night before surgery was associated with a reduction in febrile morbidity from 40% to 21% in 1,000 women. Turner[55] stated that "A considerable number of operators prescribe injectable penicillin for their patients the day of operation and for at least three additional days. . . . It appears that injectable penicillin administered postoperatively does not appreciably alter the incidence of febrile reactions in any of the groups. . . . The use of therapeutic penicillin was reduced 50% following the introduction of a penicillin suppository into the vagina. A considerable financial benefit may thereby accrue to the patient."[55] Additional surgical procedures did not significantly alter the incidence of major postoperative infection in Turner's report, written 35 years ago. It is refreshing to know that three decades ago there were investigators interested in cost containment.

Regimens

Few articles concerning prophylaxis appeared until the late 1960s. These evaluated the efficacy of 4- to 6-day drug administration in

women undergoing vaginal hysterectomy.[13,28,29,42,43] Antibiotics were administered parenterally for the first 1 to 2 days, followed by oral antibiotics to complete the course of "prophylaxis." Later studies indicated that the prophylactic administration of antibiotic for only 12 hr effectively prevented infection.[45,56] Among the antibiotics evaluated were ampicillin,[29,31] penicillin,[19] tetracycline,[29] chloramphenicol,[28] cephalothin,[13,43] cefazolin,[17,19,33,35,54] cephradine,[10,22] and cephaloridine.[45,56] More recent studies have included second-generation cephalosporins (cefoxitin[21,46,57] and cefamandole[20]) and third-generation cephalosporins such as cefotaxime,[58,59] moxalactam,[47] cefoperazone,[50] and ceftriaxone.[51,60] Metronidazole[61] and trimethoprim-sulfamethoxazole[14] were also evaluated. Clinical trials have, in general, revealed comparable efficacy among the drugs studied.

There are several factors to be addressed when a patient is being considered for antimicrobial prophylaxis. Among these factors are duration, route, and timing of administration. Burke[62] demonstrated in an experimental animal model that the most effective period of antibiotic administration ranged from immediately before making the incision to about 1 hr after the procedure. Ideally, the antibiotic agent should be in sufficient concentrations in the tissues when the contamination occurs. Data for cesarean sections have confirmed Burke's animal model data by showing that initial prophylaxis administration after clamping the infant's umbilical cord does not increase the maternal risk for infection when compared to that observed with administration prior to incision. Preoperative administration is easily achieved with hysterectomy.

Pharmacokinetics

French and co-authors[63] reported that 1 g of either cefoxitin or cefamandole administered rapidly intravenously before hysterectomy resulted in peak pelvic tissue concentration 20 min later. Two hours after injection, pelvic tissue concentrations of both antimicrobials were very low. We found significant concentrations of cefoxitin ($\geq 8.7\ \mu g/g$) in pelvic tissues for as long as 199 min after a 2-g intramuscular dose.[50] If the surgeon uses a single dose of antimicrobial for prophylaxis, intravenous administration in the operating room is preferred for vaginal hysterectomy and routine abdominal hysterectomy. A single dose of prophylactic antibiotic given prior to induction of anesthesia is to be preferred. For prolonged vaginal or abdominal hysterectomy, the intramuscular route of administration appears more appropriate. If the half-life of the antimicrobial exceeds 2 hr, the route of administration probably does not make a difference.

Do pharmacokinetic data such as serum half-life and protein binding play a role in preventing infection? It is more likely that serum concentration of an antibiotic is not the important factor, but rather the amount of antibiotic present in potentially contaminated tissue sites.[64] Serum concentration contributes to this, but protein binding is important because the greater the protein binding, the lower the fraction of free antibiotic available to diffuse into tissue. Factors other than protein binding affect half-life in serum, as evidenced by the fact that penicillin G is 55% bound and ampicillin is 70% bound and their respective half-lifes in serum are 4 and 5 hr. Cephalothin is 70% protein bound, yet the half-life in serum is about 0.5 hr.[61] Does a higher concentration of antibiotic in tissues offer better protection against infection? Theoretically it should. We have not been able to correlate concentration of antibiotic in pelvic tissues or serum with the ability of an antibiotic to prevent infection.[50,51] An agent with a prolonged half-life should require fewer administrations, and perhaps at lower doses. Clinical trials are necessary to confirm or refute this premise.

Bacterial Spectrum

Antibiotic selection, whether for therapy or prophylaxis, is empiric. Selection is based on the knowledge of the potential pathogens causing infection and the training and experience of the surgeon. Are the newer antimicrobials—because of the increased in vitro activity, the expanded spectrum of bactericidal activity, and the enhanced pharmacodynamics—more effective or more efficient than older agents? Without doubt, the newer cephalosporins and penicillins are very effective as therapeutic agents. There are several comparative clinical trials reporting that a newer agent alone was as

effective as was combination therapy in the treatment of women with a variety of pelvic infections.[65–68] With such therapeutic efficacy, the newer agents should be significantly more effective at preventing infection than older agents. This assumption has not been supported by clinical trials. The newer and older agents appear to be of equal efficacy.[47,50,51,58–60] The possibility exists that the newer agents might be as effective as older agents, even when given at significantly lower doses. Data to confirm or refute this concept are not available. If this finding is confirmed with equivalent doses, it would be medically and economically imperative that we utilize older agents for prophylaxis, saving the newer agents for therapy. This practice would allow fulfillment of several of the prophylaxis guidelines proposed by Ledger et al.[69] He also recommended that *prophylaxis* should be terminated in the recovery room.[70]

Cefotaxime was the first antimicrobial approved by the Food and Drug Administration for preoperative single-dose hysterectomy prophylaxis. Approval was granted in 1984. Yet many older studies have shown that a single preoperative dose of antimicrobial was effective at preventing major infection after both vaginal and abdominal hysterectomy.[10,14,34,35,49,51,52,60,61,71] In many instances the single dose was compared to multiple doses of the same or other antibiotics. The benefits to the patient and hospital from single-dose prophylaxis are numerous and include, but are not limited to, diminished toxic or allergic/adverse reaction, minimal alteration in host flora, decreased likelihood for the development of selective resistance in hospital and patient bacterial flora, decreased likelihood for the development of superinfection, decreased pharmacy and nursing service space and time obligation, and reduced expense for both the hospital pharmacy and the patient

Risks of Prophylaxis

The risks of true prophylaxis are relatively small. Anaphylaxis is the most immediate and potentially life-threatening adverse reaction; its occurrence is extremely rare. Accurate documentation of a history of allergy should identify problems in this category. There may be cross allergenicity between cephalosporins and penicillin. Fatal reactions have been reported following the administration of a cephalosporin (cephalothin) to a patient with a penicillin allergy.[72] Approximately 10%–15% of women undergoing hysterectomy in our hospital admit to an allergy to penicillin. Skin testing is not routinely performed. If the reaction to penicillin does not indicate an anaphylactic or immediate hypersensitivity reaction, we administer a cephalosporin for prophylaxis. In the 1,900 women whom we have prospectively evaluated, we observed a 0.2% incidence of skin rash and pruritus without a rash. These women denied antimicrobial allergy prior to antibiotic administration. Theoretically, each dose in excess of the preoperative dose increases the potential for reaction by that order of magnitude.

Opponents of the concept of prophylaxis declare that the use of antimicrobial in the absence of infection results in the masking of clinical signs and symptoms of infection during the initial hospitalization, thereby delaying diagnosis and prolonging hospital stay. This delay in diagnosis may extend into the period after hospitalization, possibly resulting in the need for hospital readmission and therapy. We have monitored patients closely after discharge, and have observed that few women given antimicrobial prophylaxis at hysterectomy develop a late infection after discharge from the hospital. The number of patents with late infection is significantly less now that we give prophylaxis than it was before the institution of antimicrobial prophylaxis. Since the inception of prospective evaluations, the most significant infections after hospital discharge were those observed in women receiving placebo. These infections can usually be treated with oral antimicrobials on an outpatient basis. It is imperative that we include these infections in statistical analyses when infection rates and success with various prophylactic regimens are being evaluated.

Death from pseudomembranous enterocolitis has been reported following *prolonged* prophylactic use of regimens of ampicillin and cephalothin, oral cephalosporins,[73] and cephaloridine[74] at hysterectomy. Pseudomembranous enterocolitis has also been reported

following cephalothin and cephradine "prophylaxis."[75] *Clostridium difficile,* the anaerobic bacteria responsible for this condition, is a gram-positive spore-forming bacillus. It is a component of normal intestinal flora in only about 3% of healthy human beings. Alteration of the normal enteric flora by antibiotics with resultant overgrowth of this bacteria and release of its toxin is the proposed pathophysiology of this disease.

Alteration of host flora and hospital bacterial resistance patterns may result from the prophylactic use of antimicrobials, especially if overutilized. Drug dosage appears to be an important factor. We found that a single preoperative dose of cefoxitin caused significantly less alteration of lower reproductive tract flora, manifested by the development of resistance, than did three doses over 12 hr in women undergoing both vaginal[49] and abdominal[52] hysterectomy. Long-term follow-up of hospital bacterial flora and antibiotic sensitivity patterns are important and necessary for adequate patient care. Increased usage of multiple antibiotics has resulted in their being less effective because of the development of in vitro bacterial resistance. Many strains of *Staphylococcus aureus* are now resistant to penicillin G, and more strains are demonstrating resistance to methicillin. In earlier studies *Bacteroides fragilis* was sensitive to tetracycline. Now, very few *Bacteroides* species are sensitive to tetracycline, and the numbers of strains resistant to cefoxitin may be increasing. Clindamycin-resistant strains have also been identified.[76] Without doubt, the longer an antibiotic is given, the more likely it is that resistant organisms will develop among patient flora and hospital bacteria.

Disulfiram- or Antabuse-like reactions occur when individuals ingest alcohol within several days of treatment with cephalosporins containing a methyltetrazolethiol side chain. These antibiotics are cefamandole, cefoperazone, and moxalactam. The reaction, believed to be caused by accumulation of acetaldehyde due to inhibition of acetaldehyde dehydrogenase, can be prevented by avoiding alcohol after injection of these antibiotics. This reaction should be an insignificant problem with the prophylactic application of these antibiotics, since women are usually in the hospital for at least 3 days after hysterectomy, and alcohol is not a frequently prescribed medication.

In summary, it has been shown that data relating to infection after hysterectomy are diverse, that generalizations may be misleading, and that risk factors are unpredictable and inconsistent. Surgeons in each hospital must determine the incidence and importance of postoperative temperature elevations, the incidence of major infections requiring antimicrobial therapy following vaginal and abdominal hysterectomy, the incidence of antibiotic administration for prophylaxis and also for "therapy" following surgery, the efficacy of various antibiotics and regimens in preventing infection, the required duration of prophylaxis, and the impact of the antibiotics on hospital flora. Prophylactic antibiotic usage is significant, accounting for one-third of antibiotic usage in many hospitals.[77] It is our current impression that antibiotics are being overutilized at the time of hysterectomy for both prophylactic and therapeutic endeavors. Appropriate selection and application of antimicrobial regimens for both prevention of and treatment of major infection after hysterectomy is mandatory to protect the patient from the complications of overusage.

References

1. DiPiro JT, Record KE, Schanzenbach KS, et al. Antimicrobial prophylaxis in surgery: Part 1. Am J Hosp Pharm. 1981; 38:320–334.
2. Dicker RC, Greenspan JR, Strauss LT, et al. Complications of abdominal and vaginal hysterectomy among women of reproductive age in the United States. The collaborative review of sterilization. Am J Obstet Gynecol. 1982; 144:841–848.
3. Gray LA. Open cuff method of abdominal hysterectomy. Obstet Gynecol. 1975; 46:42–46.
4. Falk HC, Bunkin IA. A study of 500 vaginal hysterectomies. Am J Obstet Gynecol. 1946; 52:623–630.
5. Ledger WJ, Child MA. The hospital care of patients undergoing hysterectomy: an analysis of 12,026 patients from the Professional Activity Study. Am J Obstet Gynecol. 1973; 117:423–433.
6. Mayer W, Gordon M, Rothbard MJ. Prophylactic antibiotics. Use in hysterectomy. NY State J Med. 1976; 76:2144–2147.
7. Appelbaum PC, Moodley J, Chatterton SA, et al.

Metronidazole in the prophylaxis and treatment of anaerobic infection. S Afr Med J. 1978; 54:703–706.
8. Appelbaum PC, Moodley J, Chatterton SA, et al. Tinidazole in the prophylaxis and treatment of anaerobic infection. Chemotherapy. 1980; 26:145–151.
9. Roberts JM, Homesley HD. Low-dose carbenicillin prophylaxis for vaginal and abdominal hysterectomy. Obstet Gynecol. 1978; 52:83–87.
10. Mendelson J, Portnoy J, DeSaint Victor JR, et al. Effect of single and multidose cephradine prophylaxis on infectious morbidity of vaginal hysterectomy. Obstet Gynecol. 1979; 53:31–35.
11. Swartz WH. Prophylaxis of minor febrile and major infectious morbidity following hysterectomy. Obstet Gynecol. 1979; 54:284–288.
12. Shapiro M, Schoenbaum SC, Tager IB, et al. Benefit-cost analysis of antimicrobial prophylaxis in abdominal and vaginal hysterectomy. JAMA. 1983; 249:1290–1294.
13. Bivens MD, Neufeld J, McCarty WD. The prophylactic use of Keflex and Keflin in vaginal hysterectomy. Am J Obstet Gynecol. 1975; 122:169–175.
14. Mathews DD, Agarwal V, Gordon AM, et al. A double-blind trial of single-dose chemoprophylaxis with co-trimoxazole during vaginal hysterectomy and repair. Br J Obstet Gynecol. 1979; 86:737–740.
15. Vincelette J, Finkelstein F, Aoki FY, et al. Double-blind trial of perioperative intravenous metronidazole prophylaxis for abdominal and vaginal hysterectomy. Surgery. 1983; 93:185–189.
16. Ohm MJ, Galask RP. The effect of antibiotic prophylaxis on patients undergoing total abdominal hysterectomy. I. Effect on morbidity. Am J Obstet Gynecol. 1976; 125:442–447.
17. Swartz WH, Tanaree P. T-tube suction drainage and/or prophylactic antibiotics: a randomized study of 451 hysterectomies. Obstet Gynecol. 1976; 47:665–670.
18. Mathews DD, Ross H, Cooper J. A double-blind trial of single-dose chemoprophylaxis with cotrimoxazole during total abdominal hysterectomy. Br J Obstet Gynecol. 1977; 84:894–897.
19. Grossman JH III, Greco TP, Minkin MJ, et al. Prophylactic antibiotics in gynecologic surgery. Obstet Gynecol. 1979; 53:537–544.
20. Creasman WT, Hill GB, Weed JC Jr, et al. A trial of prophylactic cefamandole in extended gynecologic surgery. Obstet Gynecol. 1982; 59:309–314.
21. Duff P. Antibiotic prophylaxis for abdominal hysterectomy. Obstet Gynecol. 1982; 60:25–29.
22. Stage AH, Glover DD, Vaughan JE. Low-dose cephradine prophylaxis in obstetric and gynecologic surgery. J Reprod Med. 1982; 27:113–119.
23. Popkin DR, Martinez LA, Carswell GA. Metronidazole in the prophylaxis of anaerobic infections in gynecologic surgery. Surgery. 1983; 93:180–184.
24. Savage EW, Thadepalli H, Rambhatla K, et al. Minocycline prophylaxis in elective hysterectomy. J Reprod Med. 1984; 29:81–87.
25. Taylor ES, Hansen RR. Morbidity following vaginal hysterectomy and colpoplasty. Obstet Gynecol. 1961; 17:346–348.
26. Pratt JH, Galloway JR. Vaginal hysterectomy in patients less than 36 or more than 60 years of age. Am J Obstet Gynecol. 1965; 93:812–821.
27. Shapiro M, Munoz A, Tager IB, et al. Risk factors for infection at the operative site after abdominal or vaginal hysterectomy. N Engl J Med. 1982; 307:1661–1666.
28. Goosenberg J, Emich JP Jr, Schwarz RH. Prophylactic antibiotics in vaginal hysterectomy. Am J Obstet Gynecol. 1969; 105:503–506.
29. Bolling DR Jr, Plunkett GD. Prophylactic antibiotics for vaginal hysterectomies. Obstet Gynecol. 1973; 41:689–692.
30. Scott JR. Pelvic infections following vaginal hysterectomy. Hosp Pract. 1973; 8:101–110.
31. Jackson C, Amstey MS. Prophylactic ampicillin therapy for vaginal hysterectomy. Surg Gynecol Obstet. 1975; 141:755–757.
32. Boyd ME, Garceau R. The value of prophylactic antibiotics after vaginal hysterectomy. Am J Obstet Gynecol. 1976; 125:581–585.
33. Holman JF, McGowan JE, Thompson JD. Perioperative antibiotics in major elective gynecologic surgery. South Med J. 1978; 71:417–420.
34. Lett WJ, Ansbacher R, Davison BL, et al. Prophylactic antibiotics for women undergoing vaginal hysterectomy. J Reprod Med. 1977; 19:51–54.
35. Jacobson JA, Hebertson R, Kasworm E. Comparison of ceforanide and cephalothin prophylaxis for vaginal hysterectomies. Antimicrob Agents Chemother. 1982; 22:643–647.
36. Osborne NG, Wright RC, Grubin L. Genital bacteriology: a comparative study of premenopausal women with postmenopausal women. Am J Obstet Gynecol. 1979; 135:195–198.
37. Thadepalli H, Savage EW Jr, Salem FA, et al. Cyclic changes in cervical microflora and their effect on infections following hysterectomy. Gynecol Obstet Invest. 1982; 14:176–183.
38. Sprague AD, van Nagell JR Jr. The relationship of age and endometrial histology to blood loss and morbidity following vaginal hysterectomy. Am J Obstet Gynecol. 1974; 118:805–808.
39. Gallup DG. Modification of celiotomy tech-

niques to decrease morbidity in obese gynecologic patients. Am J Obstet Gynecol. 1984; 150:171–178.
40. Pitkin RM. Abdominal hysterectomy in obese women. Surg Gynecol Obstet. 1976; 142:532–536.
41. Pitkin RM. Vaginal hysterectomy in obese women. Obstet Gynecol. 1977; 49:567–569.
42. Ohm MJ, Galask RP. The effect of antibiotic prophylaxis on patients undergoing vaginal operations. I. The effect on morbidity. Am J Obstet Gynecol. 1975; 123:590–596.
43. Allen JL, Rampone JF, Wheeless CR. Use of a prophylactic antibiotic in elective major gynologic operations. Obstet Gynecol. 1972; 39:218–224.
44. Poulsen HK, Borel J, Olsen H. Prophylactic metronidazole or suction drainage in abdominal hysterectomy. Obstet Gynecol. 1984; 63:291–294.
45. Breeden JT, Mayo JE. Low dose prophylactic antibiotics in vaginal hysterectomy. Obstet Gynecol. 1974; 43:379–385.
46. Hemsell DL, Cunningham FG, Kappus S, et al. Cefoxitin for prophylaxis in premenopausal women undergoing vaginal hysterectomy. Obstet Gynecol. 1980; 56:629–634.
47. Hemsell D, Hemsell P, Nobles B, et al. Moxalactam versus cefazolin prophylaxis for vaginal hysterectomy. Am J Obstet Gynecol. 1983; 147:379–385.
48. Hemsell DL, Reisch J, Nobles B, et al. Prevention of major infection after elective abdominal hysterectomy: Individual determination required. Am J Obstet Gynecol. 1983; 147:520–528.
49. Hemsell DL, Heard ML, Nobles BJ, et al. Single-dose cefoxitin prophylaxis for premenopausal women undergoing vaginal hysterectomy. Obstet Gynecol. 1984; 63:285–290.
50. Hemsell DL, Johnson ER, Bawdon RE, et al. Cefoperazone and cefoxitin prophylaxis for abdominal hysterectomy. Obstet Gynecol. 1984; 63:467–472.
51. Hemsell DL, Johnson ER, Bawdon RE, et al. Ceftriaxone and cefazolin prophylaxis for hysterectomy. Surg Gynecol Obstet. 1985; 161:197–203.
52. Hemsell DL, Hemsell PG, Heard ML, et al. Preoperative cefoxitin prophylaxis for elective abdominal hysterectomy. Am J Obstet Gynecol. 1985; 153:225–226.
53. Cron RS, Stauffer J, Paegel H Jr. Morbidity studies in one thousand consecutive hysterectomies. Am J Obstet Gynecol. 1952; 63:344–350.
54. Polk BF, Tager IB, Shapiro M, et al. Randomised clinical trial of perioperative cefazolin in preventing infection after hysterectomy. Lancet. 1980; 1:437–441.
55. Turner SJ. The effect of penicillin vaginal suppositories on morbidity in vaginal hysterectomy and on the vaginal flora. Am J Obstet Gynecol. 1950; 60:806–812.
56. Ledger WJ, Sweet RL, Headington JT. Prophlactic cephaloridine in the prevention of postoperative pelvic infections in premenopausal women undergoing vaginal hysterectomy. Am J Obstet Gynecol. 1973; 115:766–774.
57. Mickal A, Curole D, Lewis C. Cefoxitin sodium: double-blind vaginal hysterectomy prophylaxis in premenopausal patients. Obstet Gynecol. 1980; 56:222–225.
58. Wideman GL, Matthijssen C. Comparative efficacy of cefotaxime and cefazolin as prophylaxis against infections following elective hysterectomy. Clin Ther. 1982; 5 (Suppl. A):67–73.
59. Roy S, Wilkin J. Comparison of cerfotaxime with cefazolin for prophylaxis of vaginal or abdominal hysterectomy. Clin Ther. 1982; 5 (Suppl. A):74–82.
60. Hemsell DL, Menon MO, Friedman AJ. Ceftriaxone or cefazolin for prophylaxis for the prevention of infection following vaginal hysterectomy. Am J Surg. 1984; 148 (4A):22–26.
61. Hamod KA, Spence MR, Rosenshein NB, et al. Single-dose and multidose prophylaxis in vaginal hysterectomy: a comparison of sodium cephalothin and metronidazole. Am J Obstet Gynecol. 1980; 136:976–979.
62. Burke JF. The effective period of preventive antibiotic action in experimental incisions and dermal lesions. Surgery. 1961; 50:161–168.
63. French MA, Quintiliani R, Nightingale CH, et al. Serum and tissue concentrations of cefoxitin and cefamandole in women undergoing hysterectomy. Obstet Gynecol. 1983; 61:42–47.
64. Neu HC. Clinical pharmacokinetics in preventive antimicrobial therapy. South Med J. 1977; 70 (Suppl. 1):14–24.
65. Cunningham FG, Hemsell DL, DePalma RT, et al. Moxalactam for obstetric and gynecologic infections. In Vitro and dose-finding studies. Am J Obstet Gynecol. 1981; 139:915–921.
66. Gibbs RS, Blanco JD, Duff P, et al. A double-blind randomized comparison of moxalactam versus clindamycin-gentamicin in treatment of endomyometritis after cesarean section delivery. Am J Obstet Gynecol. 1983; 146:769–772.
67. Hemsell DL, Cunningham FG, DePalma RT, et al. Cefotaxime sodium therapy for endomyometritis following cesarean section: dose-finding and comparative studies. Obstet Gynecol. 1983; 62:489–497.

68. Gilstrap LC III, Maier RC, Gibbs RS, et al. Piperacillin versus clindamycin plus gentamicin for pelvic infections. Obstet Gynecol. 1984; 64:762–766.
69. Ledger WJ, Gee C, Lewis WP. Guidelines for antibiotic prophylaxis in gynecology. Am J Obstet Gynecol. 1975; 121:1038–1045.
70. Ledger WJ. Prevention, diagnosis, and treatment of postoperative infections. Obstet Gynecol. 1980; 55 (Suppl. 5):203S–206S.
71. Lander JL, Steigrad SJ. Single dose preoperative prophylactic antibiotic in vaginal hysterectomy. Aust NZ J Obstet Gynaecol. 1978; 18:73–75.
72. Spruill FG, Minette LJ, Sturner WQ. Two surgical deaths associated with cephalothin. JAMA. 1974; 229:440–441.
73. Ledger WJ, Puttler OL. Death from pseudomembranous enterocolitis. Obstet Gynecol. 1975; 45:609–613.
74. Tan J, Bayne LH, McLeod PJ. Pseudomembranous colitis. A fatal case following prophylactic cephaloridine therapy. JAMA. 1979; 242:749–750.
75. Hutcheon DF, Milligan FD, Yardley JH, et al. Cephalosporin-associated pseudomembranous colitis. Am J Dig Dis. 1978; 23:321–326.
76. Cuchural GJ Jr, Tally FP, Jacobus NV, et al. Antimicrobial susceptibilities of 1,292 isolates of the *Bacteroides fragilis* group in the United States: comparison of 1981 with 1982. Antimicrob Agents Chemother. 1984; 26:145–148.
77. Guglielmo BJ, Hohn DC, Koo PJ, et al. Antibiotic prophylaxis in surgical procedures. A critical analysis of the literature. Arch Surg. 1983; 118:943–955.

Control of Surgical Bleeding 9

David Gal

Celsus first described the use of ligatures in 100 A.D. Galen, however, influenced the medical profession to use cautery for many centuries. Ambroise Paré, the French military surgeon, reintroduced the principles of ligature in 1552. Since then, great strides have been made in surgical techniques and suture material usage, especially in the second half of this century. Finally, improved understanding of the hemostatic process has contributed to the control of surgical bleeding.

Hemostasis is a physiologic phenomenon requiring the interfacing of several factors. The achievement of hemostasis depends on the nature of insult to the blood vessel, the type of vessel injured, the anatomic site of injury, platelet reaction, the coagulation mechanism, and fibrinolytic activity (Figure 9-1). The support of the surrounding tissues is of primary importance in the process of hemostasis. Thus, in a young healthy individual, the rupture of a small submucosal venule in the nose may result in extensive bleeding while rupture of the same caliber venule in the calf or arm may result in minimal bleeding. Centuries ago, Hunter demonstrated that small arteries contract after complete transection with minimal bleeding; however, if longitudinally incised, the same size vessel will bleed extensively.

The sequence of normal hemostasis (Figure 9-2) is not completely understood; however, the general sequence of events is as follows: Vascular disruption is immediately followed by vasoconstriction, platelet adhesion, and formation of a platelet plug. Concomitantly, the coagulation cascade is activated and fibrin strands form and reinforce the platelet plug. A recovery phase follows. A delayed event during the recovery phase is the activation of the fibrinolytic system. Plasmin is formed and it degrades fibrin strands and dissolves the plug to allow recanalization.

Preoperative Evaluation of Coagulopathy

The preoperative evaluation should be as extensive and cost effective as time and local economics allow. The patient's history is of primary importance. Bleeding tendencies (e.g., prolonged bleeding after dental care or minor injuries) as well as family history of coagulation abnormalities should be sought. In gynecology, a coagulation problem may present as excessive menstrual flow rather than intermenstrual bleeding. A curettage in such a patient, who may have an unrecognized thrombocytopenia, may lead to massive hemorrhage. In the family history, attention should be directed to sex-linked and recessive modes of genetic transmission. Finally, the patient should list all drugs being ingested, some of which may have an effect on her coagulation profile.

The physical examination should include a search for evidence of a bleeding disorder such as ecchymoses, petechiae, or purpura. Senile purpura are most common on the forearms

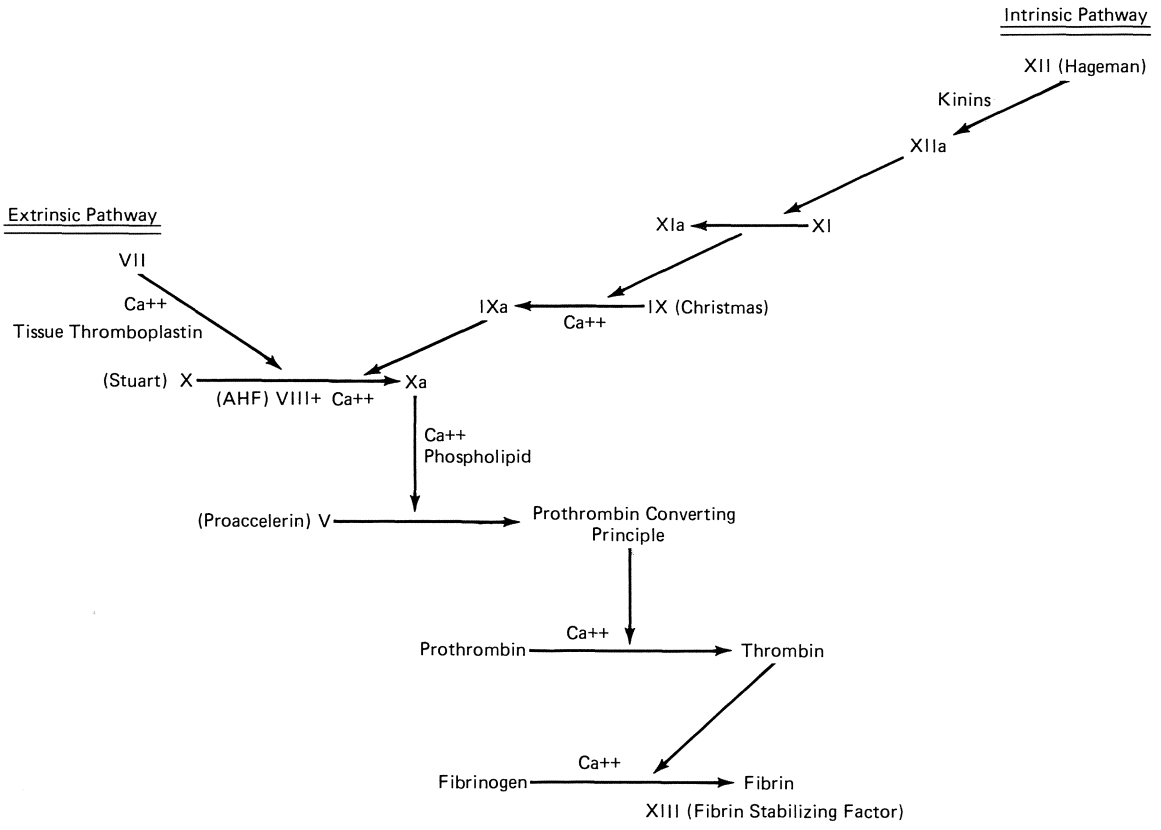

Figure 9-1. The coagulation system. Lower-case "a" following factor number indicates activated coagulation factor.

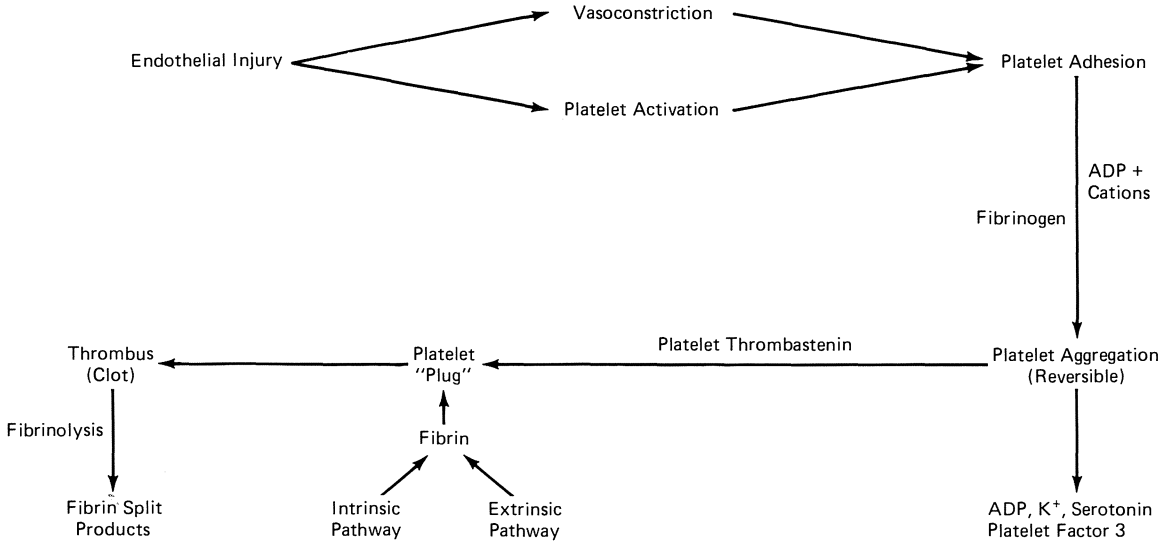

Figure 9-2. Mechanisms of hemostasis.

Table 9-1. Diagnosis of Inherited Disorders of Coagulation

Deficient factor	Bleeding time	Prothrombin time	Partial thromboplastin time	Thrombin time	Fibrinogen time	Platelet adhesiveness
I (fibrinogen)	P[a] or N[b]	P	P	D[c]	D	N
II (thrombin)	N	P	P	N	N	N
V	N	P	P	N	N	N
VII	N	P	N	N	N	N
VIII	N	N	P	N	N	N
Von Willebrand's	P	N	P	N	N	D
IX	N	N	P	N	N	N
X	N	P	P	N	N	N
XI	N	N	P	N	N	N
XII	N	N	P	N	N	N
XIII	N	N	N	N	N	N

[a] Prolonged.
[b] Normal.
[c] Decreased.

and hands, while Henoch–Schonlein purpura generally are distributed over the buttocks, elbows, and ankles; spider angiomas signify the presence of liver disease and possible reduction of prothrombin. Hepatosplenomegaly and lymphadenopathy should raise the possibility of a reticuloendothelial disorder.

The preoperative laboratory evaluation should include:

1. Peripheral blood smear for platelet and erythrocyte morphology and platelet count
2. Partial thromboplastin time (PTT) or activated PTT (aPTT)
3. One-stage prothrombin time (PT)

When the PTT is used in conjunction with PT for a bleeding disorder, the abnormality can be subdivided into first-stage (hemophilia group) and second-stage problems (prothrombin complex). A dilute whole blood or euglobulin plasma clot-lysis test should be performed in patients with metastatic disease in order to screen for fibrinolysis problems.

If patients have a hemostatic dysfunction or personal or family history of a bleeding disorder, a more elaborate hemostatic profile is required. This profile should include a bleeding time, thrombin time, fibrinogen level, platelet adhesiveness, and additional clotting factor studies (Table 9-1).

Intra-and Postoperative Bleeding

Bleeding during surgery that is unrelated to an acute vascular injury is usually due to one or more of the following causes:

1. Ineffective local hemostasis
2. Complication of blood transfusion
3. A previously present but undetected coagulation defect
4. Sepsis
5. Fibrinolysis

A generalized hemostatic defect will cause bleeding from multiple sites, for example, incision, drain wounds, venous puncture, etc.

Although the gynecologist may suspect that surgical bleeding is related to local problems, laboratory investigation must confirm it. All the basic coagulation studies (platelet count, PTT, PT, and thrombin time) should be performed. The results of these studies should confirm the clinical impression.

Intraoperatively a patient may develop a transfusion reaction (chills, diaphoresis), which is sometimes difficult to diagnose because the patient is anesthetized. The only evidence may be a progressive failure of hemostasis and unanticipated oozing. The pathogenesis of this phenomenon, although poorly understood, may be related to platelet consumption. Aden-

osine disphosphate (ADP), released from hemolyzed erythrocytes, causes platelet aggregation, which together with the release of a procoagulant platelet factor (phospholipid) may bring about intravascular defibrination. When the hemostatic defect manifests itself during surgery, the patient will bleed from an intravenous site and demonstrate hematuria or hematemesis.

Patients with sepsis may develop continuous bleeding during surgery. This is the result of endotoxin-induced thrombocytopenia and labile factor V. In addition, the occurrence of hemolysis as a result of sepsis can lead to defibrination in these patients. Thrombin time, PT, and PTT are mandatory in their evaluation. Euglobulin clot lysis and dilute whole blood clot lysis are sensitive indices of fibrinolysis.

Management of Bleeding due to Hemostatic Defect

Acute blood loss should be replaced by equal volumes of whole blood. However, it is difficult to evaluate the volume deficit. Values for "normal blood volume" vary according to patient's age, size, and disease status. Measurement of hemoglobin and hematocrit may be misleading in the face of acute blood loss. These studies may be normal in the immediate postoperative period in spite of severely contracted blood volume, and the actual values will become evident over a period of 72 hr[1] (Table 9-2). A healthy patient can lose 1 liter of blood in a short period without a fall in blood pressure so long as she remains supine. Significant hypotension develops after blood loss of about 2 liters (40% of total volume). The surgical blood loss is the sum of suctioned blood and blood on sponges and laparotomy pads. The weight of the sponges and laparatomy pads should be multiplied by 1.4 to obtain true blood loss.[2] Losses up to 1000 ml can be safely treated with lactated Ringer's and colloid solutions.

Table 9-2. Hematocrit Stabilization after Loss of 1000 ML of Blood[a]

Time from bleeding (hr)	Hematocrit decrease (%)
1	3
24	5
48	6
72	8

[a] Modified from Ebert and Colleagues. Data from a healthy adult female. (Archives of Internal Medicine, 1941, Volume 68, 578–594. Copyright 1941, American Medical Association.)

Red blood cells should be transfused to improve oxygen-carrying capacity. The Committee on Blood of the American Medical Association declared that "It is poor practice to order 2 units of blood to escape criticism for using a single unit." Only the appropriate volume should be given. "Massive transfusion" is defined as transfusion greater than 2500 or 5000 ml transfused over a period of 24 hr.

The problems associated with massive transfusion are dilution thrombocytopenia, impaired platelet function, and deficiencies of Factors V, VIII, and XI. Hypothermia can occur after transfusions if a blood warmer is not used. Hypocalcemia due to citrate binding and hyperkalemia without clinical effects have also been described. Finally, if a patient bleeds after massive transfusion, thrombocytopenia should be considered and treated by the administration of fresh platelet concentrate.

The cause of defibrination and clotting factor deficit is often multifactorial; thus, it is best managed by fresh-frozen plasma, which usually carries less transfusion-associated risk than the concentrated components (Table 9-3).

Vascular Anatomy

With the exception of the ovarian vessels, which originate from the abdominal aorta just below the renal arteries and course in the infundibulopelvic ligament to anastomose with the uterine plexus, the pelvic blood supply begins at the bifurcation of the aorta.

The middle sacral artery, the embryologic continuation of the aorta, is small because of the development of the common iliac arteries. It supplies muscles and bones of the posterior pelvic wall. The common iliac arteries are divisions of the abdominal aorta. Except for minor collaterals, the iliac arteries have no major branches until they divide to form the external and internal iliac (hypogastric) arteries. The ovarian vessels and ureters traverse the com-

Table 9-3. Blood Components Therapy

Component	Content	Indications	Risks	Special precautions	Comments
Whole blood	All components	Hypoxia and hypovolemia Massive acute blood loss	Hepatitis, volume overload	ABO compatibility. Essential labile coagulation factors deteriorate 24 hr after collection	Consider component therapy
Red blood cells	Red cells White cells	Symptomatic anemia Blood replacement	Allosensitization Hepatitis	ABO compatibility. Essential coagulation deficiency	Hematocrit will increase 3–5% per 200 ml (1 unit)
RBC, leucocytes poor	Red cells	Anemia and febrile reaction from leukocyte antibodies	Same as above	Same as above	Same as above
Fresh-frozen plasma	All clotting factors, no cells	Hypovolemia and hypoalbuminemia, DIC and deficiency of clotting factors of immunoglobulins	Hepatitis	ABO compatibility	When treating hypovolemia, give 1 unit (200 ml) for every 4 units of red cells. Fibrinogen increases 10 mg/dl per unit
Cryoprecipitate	I, V, VIII, XIII	Hemophilia A, Von Willebrand's, DIC, and Factor XIII deficiency	Hepatitis	Rapid infusion and frequent repeat doses may be necessary	Fibrinogen increases 10 mg/dl per unit (40 ml). Contraindicated if coagulation defect undefined.
Platelet concentrate	Platelets	Thrombocytopenia (acquired and hereditary). Platelet function anomaly	Rh isoimmunization	Do not use microaggregate filter	Increases platelet count 1500/ml per unit (50 ml). Consider component Rx.
Factor concentrate	VIII, IX	Hemophilia A, IX deficiency	Hepatitis	None	IU equals factor activity in 1 ml pooled plasma

mon iliac arteries at the brim of the pelvis, just medial to the bifurcation. The external iliac artery lies on the medial border of the psoas major muscle. It has no major branches until it approaches the groin. At the groin, the deep circumflex iliac artery branches laterally, while the inferior epigastric artery courses anteromedially as the iliac artery becomes the femoral artery. The inferior epigastric artery may anastomose with the obturator artery and the epigastric artery cephalad to communicate with the mammary plexus.

The hypogastric artery is the medial terminal branch of the common iliac artery. The posterior division gives rise to three parietal branches, the iliolumbar, lateral sacral, and the superior gluteal arteries. The anterior division gives rise to parietal branches (obturator, inferior gluteal, and internal pudendal) and visceral branches (superior vesical, middle hemorrhoidal, uterine, and vaginal) arteries.

The superior vesical artery supplies the distal ureter and bladder. The middle hemorrhoidal has a variable origin. It anastomoses with the superior hemorrhoidal branch of the inferior mesenteric artery and the inferior hemorrhoidal artery (a division of the internal pudendal artery). The vaginal artery may rise from the hypogastric artery, the uterine artery or the superior vesical artery. It anastomoses with the descending branches of the uterine artery. The inferior gluteal artery, a parietal branch of the hypogastric artery, supplies the gluteus maximus muscle and the muscles of the back of the thigh.

There are two major pelvic collateral systems, a vertical and a horizontal (Table 9-4). The vertical system functions to a greater extent than the horizontal, especially after bilateral hypogastric artery ligation.

Arterial Injury

Two types of arterial trauma that may occur during pelvic surgery are blunt compression and penetrating lacerations or puncture. The former injury is usually a result of incorrect use of retractors and traction maneuvers. The injury seldom results in vascular or distal tissue damage, unless the traumatic action was prolonged and produced thrombosis and distal ischemia. Occasionally, the vascular injury is underestimated and the arterial contusion may appear innocent, without bleeding or disruption of the arterial wall. However, intimal damage may occur, which causes gradual thrombus formation or intramural hematoma. In addition, the vessel intima can be lifted and a false channel formed. This can result in arterial occlusion and loss of distal pulses.

Arterial puncture wounds can be managed by direct compression. When small arteries are severed, the muscular layer causes them to contract. As a result, thrombus formation occurs in the divided ends. In such a case, no specific hemostatic measures are required. An

Table 9-4. Major Pelvic Anastomoses[a]

Vertical	Horizontal
1. Ovarian (aorta–uterine (hypogastric)	1. Left vesical (hypogastric)–right vesical
2. Superior hemorrhoidal (inferior mesenteric)–middle hemorrhoidal (hypogastric)	2. Left pubic (obturator from hypogastric)–right pubic
3. Middle hemorrhoidal–inferior hemorrhoidal (internal pudendal from hypogastric)	
4. Obturator (hypogastric)–inferior epigastric (external iliac)	
5. Inferior gluteal (hypogastric)–circumflex and perforating branches of deep femoral artery	
6. Superior gluteal (hypogastric–lateral sacral (hypogastric)	
7. Lumbar–iliolumbar (hypogastric)	

[a] Arterial origin in parentheses.

exception to this rule is the arteriosclerotic vessel that has lost its contractility. After arterial injury, the distal pulse will generally disappear. However, peripheral pulses can sometimes be palpable, so long as the vessels are not completely transected or thrombosed, since the pressure wave may be transmitted through the soft thrombus or a large collateral branch.

When a vessel is lacerated rather than transected, blood loss may be increased because the contractile factors tend to separate the vessel edges even more. If the vessel is not exposed, a hematoma will form. This hematoma may or may not be contained by the adjacent tissues, depending on the size of the laceration. If venous injury accompanies the arterial damage, an arteriovenous fistula or a false aneurysm may form at the site of the laceration.

Of special interest to gynecologists in recent years is bleeding due to laparoscopic procedures. Such bleeding may be fatal if not recognized and corrected. If the bleeding is intraperitoneal, it will be visualized through the laparoscope or the exit of blood through the verres needle. Retroperitoneal bleeding may go unnoticed for a long time, and often will be discovered only after a change in vital signs.

Management of Vascular Injuries

An acute arterial injury should be repaired promptly. Bleeding from the site of injury should be controlled by direct pressure. The least traumatic methods involve the use of the surgeon's fingers, a sponge, or an instrument. The next step should be resotration of blood volume and requisition of appropriate vascular instruments and suture material. An artificial graft might be necessary. The defect can be repaired if necessary with an artificial graft or a venous autograft.

After direct pressure has been applied, the proximal and distal ends of the injured artery should be inspected and clamped 2–3 cm from the site of injury with atraumatic vascular clamps.[3] Temporary intraluminal occlusion with a Fogarty catheter or even a Foley catheter is sometimes helpful.[3] When a major vessel is to be repaired (e.g., aorta, common iliac artery), the patient must be anticoagulated by the administration of 5,000–10,000 units of heparin administered intravenously. At this point, a decision is made regarding the use of a patch or a graft. Monofilament synthetic suture is utilized (5/0 or 6/0; 4/0 for the aorta). Many authors recommend that a concomittant venous injury should be repaired first, in order to improve peripheral circulation and outflow.[4]

The basic aim of arterial reconstruction is restoration of blood flow. Techniques utilized include the following: longitudinal repair, longitudinal repair with patch angioplasty, primary end-to-end anastomosis, and replacement with graft.

Debridement of the edges with removal of injured adventitia and intima should precede the repair. Sutures should be placed to prevent inversion of the edges. However, lacerations involving more than 30% of the vessel circumference usually require a patch to prevent constriction of the lumen. Synthetic materials are not used in a contaminated field, and autogenous vein graft should be used. These grafts must be tailored to restore the normal arterial lumen. Tension on the suture line should be avoided.

A loss of 2 cm or more of the arterial wall requires placement of an intervening graft. After transection, the physiologic retraction of both ends may magnify the gap. Proximal and distal dissection of the divided arterial segments will provide extra length to decrease tension on the suture line. As a rule the graft should be slightly larger than the vessel that it is replacing. Before completing the anastomosis, the vascular clamps, first the distal and then the proximal, are momentarily released to expel residual air and thrombi.

If the vessel was occluded for more than a few minutes, it is advisable to assure the absence of distal thrombi by gentle insertion of a Fogarty catheter[5] as far as possible; after inflation of the balloon, the catheter is withdrawn. This thrombectomy procedure requires expertise to avoid distal perforation and severe spasm or even rupture of the vessel due to

inappropriate balloon inflation. After completion of the anastomosis, gentle pressure over the suture line will control oozing of blood from the suture puncture. On releasing the clamps, a strong pulsation should be felt distal to the anastomosis. A significant pressure gradient across the suture line may indicate mechanical narrowing, or thrombus formation and revision may be necessary. Occasionally, an intraoperative arteriogram may be required to clarify the reason for the diminished distal pulse.

It is preferable to cover the anastomotic site with soft tissue such as fat or peritoneum. Heparin neutralization is not mandatory if the anastomatic site is dry. If the procedure lasts a few hours, heparin degradation would have taken place.

Arterial Injury

Inferior Mesenteric. The inferior mesenteric vessels deserve special consideration at the time of paraaortic lymph node dissection and laparoscopic procedures. Up to 1% of patients undergoing paraaortic dissection may have some degree of left colon ischemia, secondary to trauma to this vessel.[6] If the integrity of the inferior mesenteric artery is interrupted distal to the origin of the left colic artery, its first branch, the viability of the descending and rectosigmoid colon may be compromised. The anastomoses from the superior to the inferior mesenteric systems are established between the middle and left colic arteries via the marginal artery of Drummond. Ligation of the inferior mesenteric artery proximal to the takeoff of the left colic artery will preserve these anastomoses. If there is inadequate blood supply to the lower colon, or if this vessel seems unusually large (usually due to superior mesenteric occlusion), reimplantation of the injured vessel into the aorta should be undertaken.

Common and External Iliac Arteries. The iliac arteries, unlike other arteries, do not retract and constrict their lumen on transsection, possibly because the adjacent tissues do not provide adequate compression of the divided arterial ends. An acute occlusion of either one of these vessels, by thrombosis or ligature, is followed by gangrenous change of the involved extremity in up to 50% of cases.[7]

Hypogastric Artery. The hypogastric artery may be ligated in cases of intractable pelvic bleeding. The clinical condition of the patient determines the surgical approach and exact site of ligature. In the hemodynamically stable patient, an extraperitoneal approach is advised. However, this technique is time consuming because two incisions are required. In the hemodynamically unstable patient, the transabdominal approach is advised. An alternative approach in situations such as uterine hemorrhage involves ligating the uterine artery at its origin from the hypogastric artery.

The hypogastric artery is ligated close to its origin from the common iliac artery. This ligation must be performed proximal to the posterior parietal branches (i.e., iliolumbar, lateral sacral, and superior gluteal) to maintain these collaterals (Table 9-4).

The surgeon must avoid injuring the hypogastric and external iliac veins, which lie just posterior and lateral to the hypogastric artery. The ureters should be visualized throughout the procedure. Pulsation in the external iliac artery, if present before the ligation, should be present after the ligation. There is continued blood flow in the hypogastric artery distal to the site of ligation.[8] The collateral system functions immediately after ligation (Table 9-4). However, the arterial pulse pressure is significantly decreased. This is usually associated with reduction in mean blood pressure and rate of blood flow in the collateral system. As a result, the trip-hammer effect of arterial pulsation disappears and the net pressure is equal to that in the vein. Clots at the site of the arterial injury will remain localized without being dislodged by arterial pulsation.

Femoral Artery. This artery is in danger of injury during groin dissection, at the time of radical vulvar surgery. Since this artery is the main blood supply to the lower extremity, an injury to this artery should always be repaired.

Venous Injury

Isolated major venous trauma, particularly to the inferior vena cava and common iliac veins, may occur during radical surgery and major gynecologic procedures. Perforations of the vena cava and iliac veins during laparoscopy have been reported.[9,10] Iliac vein stenosis can result from radiation therapy. This stenosis may be followed by lymphatic blockage and leg edema. Veins of small diameter can be ligated with minimal or no disability. Major veins should be preserved. If a major vein needs to be ligated, early supportive measures, such as elevation of the extremity and anticoagulation, should be instituted to prevent extension of venous thrombosis. However, even in these cases minimal or no immediate disability will occur. The ligation of major veins impairs venous return although the end result may not be apparent until months or years later.[11] Although the thin, fragile vein wall with low intraluminal pressure make the repair more difficult than repair of arteries, ligation should be discouraged.

To avoid vein constriction, an autogenous saphenous vein can be used. The long-term results of direct repair are much more satisfactory than the use of prosthetic grafts, which are followed by a high incidence of thrombosis. Thrombophlebitis and pulmonary embolism were considered common complications following venous repair; however, recent reports refute this belief.[12] When tumor encases a vein, requiring resection and a prosthetic graft, the performance of an arteriovenous fistula has been advocated.[13,14]

The postoperative care of the patient with venous repair should include prophylaxis for local thrombosis by the use of anticoagulation and avoidance of extremity edema by elevation.

Iliac Veins. These are the main conduits for venous return from the lower half of the body. Any interruption of the venous system between the femoral vein and inferior vena cava can create chronic venous insufficiency, particularly when the patient is standing. End-to-end anastomoses can sometimes be performed after mobilization of the divided segments. Because of the greater incidence of thrombosis, thrombi should be removed from both ends before completing the repair.

Vena Cava. If ligation is indicated because of extensive injury to the vessel, the site of ligation should be immediately below the major tributary, the renal, or the lumbar veins to prevent thrombosis in the residual pouch and subsequent emboli. The ligation of the vena cava at the renal or suprarenal segment may lead to death in 80%–90% of patients.[15,16] Bleeding from the vena cava is best controlled by direct pressure. Atraumatic clamps are applied distal and proximal to the injury. Sometimes application of a partial occlusion clamp may be of benefit. An alternative to clamp application is direct compression of the vena cava with two folded sponges held by ring forceps. After hemostasis and adequate exposure, the laceration can be repaired with 5/0 or 6/0 nonabsorbable monofilament continuous suture.

Thermal Hemostasis

Bovie and Cushing, during the first decade of this century, resurrected Galen's concept of cautery for hemostasis. Cautery is effective because heat denatures protein and coagulates tissue with resultant vascular occlusion. The electrocautery amplitude setting should be high enough to produce prompt coagulation. If the amplitude is too high, an arc is formed between the cautery and the tissue and may damage tissue and interfere with monitoring devices. A negative place applied to the patient serves to prevent severe skin burns. Explosive anesthetic agents must not be used in the presence of electrocautery.

Chemical Hemostasis

There are two mechanisms of chemical hemostasis—vasoconstrictors and coagulants. Epinephrine, a vasoconstrictor, has a temporary effect.

Cushing was the first to employ skeletal muscle for local hemostasis during brain surgery.[17] Shortly thereafter, hemostatic fibers were developed. Locally functioning material should

be easy to handle, should be rapidly absorbed, nonirritating, and create hemostasis independent of the clotting system. The most widely used are oxidized cellulose (Oxycel), oxidized regenerated cellulose (Surgicel), micronized collagen (Avitene), and gelatin foam (Gelfoam). The latter, derived from denatured animal resin gelatin, is widely used. Prior to the placement of Gelfoam, the sponge should be moistened in saline or thrombin solution, and all the air should be removed from the interstices. Its main hemostatic activity is related to the contact with blood, the large surface area of the sponge, and the pressure exerted by the weight of the sponge.

Invasive Radiologic Embolization for Hemostasis

Gelfoam can also be used for arterial embolization when surgical intervention is questionable. The technique utilizes radiographic image visualization and selective arterial embolization.[18–23] Gelfoam pads are cut into particles small enough to obstruct the intraarterial lumen but too large to reach the arteriolar level, thus minimizing the risk of acute infarction and tissue necrosis.[24] Gelfoam is completely reabsorbed in 2 days–2 weeks, minimizing the likelihood of any long-term compromise of the viability and function of the treated organs. Several complications have been described with this technique, such as vesicovaginal fistula,[23] thrombosis of the femoral artery,[22] and renal toxicity from excessive volumes of contrast medium.[25] However, these can be avoided by careful attention to angiographic technique.

References

1. Ebert RV, Stead EA, Gibson JG. Response of normal subjects to acute blood loss. With special reference to the mechanism of restoration of blood volume. Arch Int Med. 1941; 68:578–594.
2. Schwartz SI. Hemostasis, surgical bleeding, and transfusion. In: Schwartz SI, Shires GT, Spencer FC, et al, eds. Principles of surgery. New York: McGraw-Hill, 1984: p. 106.
3. Smiley K, Perry MO. Balloon catheter tamponande of major vascular wounds. Am J Surg. 1971; 121:326–341.
4. Hewitt RL, Drapanas T. Vascular injuries. In: Haimovici H, ed. Vascular surgery: principles and techniques. New York: McGraw-Hill, 1976: p. 560–562.
5. Hardy JD, Raju S, Neely WA, et al. Aortic and other arterial injuries. Ann Surg. 1975; 181:640–648.
6. Belinson JL, Goldbert MI, Averette HE. Paraaortic lymphadenectomy in gynecologic cancer. Gynecol Oncol. 1979; 7:188–198.
7. Rich NM, Spencer FC eds. Iliac artery injury. In: Vascular trauma. Philadelphia: WB Saunders, 1978: p. 475–493.
8. Burchell RC, Mengert WF. Internal iliac artery ligation: a series of 200 patients. J Int Fed Obstet Gynecol. 1969; 7:85–94.
9. McDonald PT, Rich NM, Collins GJ, et al. Vascular trauma secondary to diagnostic and therapeutic procedures: Laparoscopy. Am J Surg. 1978; 135:651–667.
10. Katz M, Beck P, Tancer ML. Major vessel injury during laparoscopy: anatomy of two cases. Am J Obstet Gynecol. 1979; 135:544–552.
11. Rich NM, Spencer FC. Venous injuries. In: Vascular trauma. Philadelphia: WB Saunders, 1978: p. 156.
12. Rich NM, Hughes CW, Baugh JH. Management of venous injuries. Ann Surg. 1970; 171:724–730.
13. Johnson V, Eiseman B. Evaluation of arteriovenous shunt to maintain patency of venous autograft. Am J Surg. 1969: 118:915–924.
14. Hobson RW, Wright CB. Peripheral side to side arteriovenous fistula: hemodynamics and application to venous reconstruction. Am J Surg. 1973; 126:411–419.
15. Cassebaum WH, Bukanz SL, Baum V, et al. Ligation of the inferior vena cava above the renal vein of a sole kidney with recovery. Am J Surg. 1967; 113:667–669.
16. Ramnath R, Walden EC, Caguin F. Ligation of the suprarenal vena cava and right nephrectomy with complete recovery. Am J Surg. 1966; 112:88–92.
17. Cushing H. The control of bleeding in operations for brain tumors. Ann Surg. 1911; 54:1–21.
18. Haseltine FP, Glickman MG, Marchesi S, et al. Uterine embolization in a patient with postabortal hemorrhage. Obstet Gynecol. 1984; 63:78S–80S.
19. Heaston DK, Meaneu DE, Brown BJ, et al. Transcatheter arterial embolization for control of persistent massive puerperal hemorrhage after bilateral surgical hypogastric ligation. Am J Roentgenol Radium Ther Nucl Med. 1979; 133:152–171.

20. Pais SO, Glickman MG, Schwartz P, et al. Embolization of pelvic arteries for control of postpartum hemorrhage. Obstet Gynecol. 1980; 55:754–763.
21. Glickman MG. Pelvic artery embolization. In: Berkowitz RL, ed. Critical care of the obstetrical patient. New York: Churchill-Livingston, 1983.
22. Jander HP, Russinovich NAE. Transcatheter gelfoam embolization in abdominal, retroperitoneal and pelvic hemorrhage. Radiology. 1980; 136:337–344.
23. Lang EK. Transcatheter embolization of pelvic vessels for control of intractable hemorrhage. Radiology. 1981; 140:331–339.
24. Braf ZF, Coontz WN Jr. Gangrene of the bladder. Complication of hypogastric artery embolization. Urology. 1977; 9:670–671.
25. Matalon TSA, Athanasoulis CA, Margolis MN, et al. Hemorrhage with pelvic fractures: efficacy of transcatheter embolization. Am J Roentgenol Radium Ther Nucl Med. 1979; 133:859–868.

Monitoring High-Risk and Critically Ill Patients 10

Daniel J. Polacek

Within the past several years, numerous advances in technology have enabled physicians to monitor high-risk and critically ill patients more closely. Invasive and noninvasive monitoring techniques are employed with three goals in mind. The first objective is to recognize, as early as possible, any acute changes in the patient's clinical condition. Second, intensive monitoring allows us to measure the effects of many therapeutic interventions and facilitates precise titration of therapy. Third, monitoring may prevent or assist in ameliorating complications that are related to the patient's underlying pathology or to iatrogenic diagnostic or therapeutic interventions.

Many patients may be adequately followed by using the traditional noninvasive techniques, such as frequent vital signs, hourly intake and output measurement, and daily weights. If invasive monitoring techniques are utilized, the physician must be aware of their capabilities, limitations, associated complications, and the indications for their use.

This chapter reviews commonly used noninvasive and invasive methods of monitoring high-risk and critically ill patients in the preoperative, intraoperative, and postoperative periods. Special emphasis is placed on monitoring of cardiac and pulmonary function; however, routine screening techniques for monitoring critically ill adults in an intensive care unit and in the operating room are also examined. The capabilities and limitations of common monitoring techniques, the indications for their use, and associated complications are reviewed.

Bedside Evaluation

The relatively recent proliferation of automated invasive and noninvasive monitoring devices has led to a tendency to deemphasize the bedside clinical exam; however, there is no substitute for a well trained and skilled observer at the bedside. For example, mental status, which is a very sensitive indicator of overall patient function, can only be assessed by a trained clinician. A change in mental status may be the first sign of hypoxemia, hypercapnia, acidosis, or impending septic shock. Observation of the patient's ventilatory rate and pattern may lead more quickly to the diagnosis and treatment of many pathologic processes than any laboratory examination. For example, the diagnosis of ketoacidosis can be made at the bedside by observing the characteristic hyperpnea and fruity breath. Impending ventilatory failure in chronic obstructive pulmonary disease is often preceded by paradoxical thoracoabdominal breathing, and the development of Cheyne–Stokes respiration is often the first sign of life-threatening central nervous system disease. Tachypnea with nasal flaring, tachycardia, and anxiety are clinical indications for elective endotracheal intubation despite "normal" arterial blood gases in a patient with pulmonary disease. In all of these

situations, clinical observation and judgment are of paramount importance.

Data Acquisition

One of the most important aspects of patient monitoring is the collection and organization of clinical and laboratory data so that trends are readily apparent. The easiest way to accomplish this is to develop a multipurpose clinical flow sheet. The commonly monitored physiologic variables including vital signs and cardiac, pulmonary, and renal parameters must be available at the patient's bedside. The flow sheet should document not only total fluid balance, but also the type and amount of each intake or output, daily weights, and laboratory results. It is helpful to leave additional room on the flow sheet to document intermittent drug administration and pertinent nursing observations.

An example of the flow sheet used in the Adult Intensive Care Unit at Magee–Womens Hospital is shown in Figure 10-1. Vital signs may be recorded as frequently as every 15 min. Space is available to record central venous, pulmonary capillary wedge, and mean arterial blood pressures. Several different intakes and outputs can be followed on an hourly basis and as a consequence daily trends are more easily followed. Inclusion of laboratory data on the flow sheet allows temporal correlation between changes in clinical status and lab values.

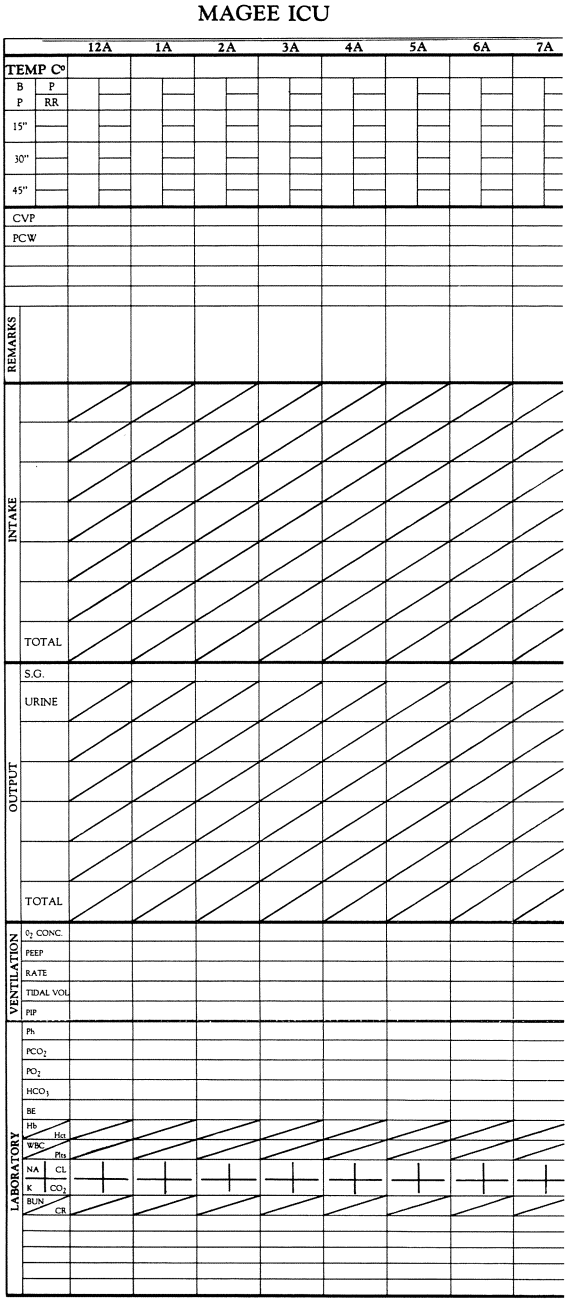

Figure 10-1. Multipurpose clinical flow sheet.

Monitoring Renal Function

Measurement of urine output coupled with daily weights is appropriate for all patients and sufficient monitoring for many patients. A urine output of 0.5 ml/kg/hr should be maintained in all patients. Decreases in urine output often lead to the insertion of the most commonly used invasive monitoring device, namely, the Foley catheter. Monitoring of urine output alone may be inadequate as the sole index of renal function, since nonoliguric renal failure is not an uncommon occurrence.[1] Serum blood urea nitrogen (BUN) and creatinine concentrations, urine specific gravity, urine osmolality, and urine electrolytes are also helpful in determining overall renal function.

Serum creatinine and BUN are largely dependent on glomerular filtration rate (GFR). Creatinine is produced and released from muscle at a relatively constant rate. Its excretion is dependent almost entirely on GFR with minimal tubular secretion. When a 50% reduction in GFR is present, the serum creatinine doubles under steady-state conditions.[2] Thus, a serum creatinine of 2 mg/dl indicates approximately 50% of normal GFR, a serum cre-

atinine of 4 mg/dl indicates about 25% of normal GFR, and a creatinine of 8 mg/dl about 12% of normal renal function. Serum creatinine alone as a measure of GFR is unreliable since the patient may not be in a steady state when the determination is made. Creatinine clearance is a much more sensitive index of GFR than the serum creatinine and should be directly measured when necessary. Aging itself is associated with decreases in GFR and muscle mass despite "normal" serum creatinine. Age-related decreases in GFR can be estimated by the following equation for females:[3]

$$\text{Creatinine clearance} = \left[\frac{(140 - \text{age})(\text{weight in kg})}{72 \times \text{serum creatinine}}\right] \times 0.85$$

The BUN is less reliable than serum creatinine as a measure of GFR, since urea clearance varies with the rate of urine flow as well as with changes in the GFR. Tubular reabsorption of urea can vary from as little as 40% of the filtered load, when urine output is high, to as much as 60% of the filtered load in oliguric states. Urea levels in the blood are also dependent on protein intake and catabolism, intravascular volume, liver disease, and gastrointestinal blood loss. A BUN/creatinine ratio of greater than 20 : 1 may indicate intravascular volume depletion; however, gastrointestinal blood loss, hypercatabolism, and drug-related changes in the BUN must be excluded before this diagnosis can be entertained.

Urine specific gravity has been used for many years as a monitor of renal concentration and consequently an indirect estimate of intravascular volume. Specific gravity is a function of both the total number of particles in solution and the relative size and density of these particles. Temperature changes will affect specific gravity measured by a hydrometer. In addition, protein, glucose, and radiopaque contrast material all disproportionately increase specific gravity. The measurement itself is also subject to large interobserver variations.[4] For these reasons, specific gravity is a very crude test of renal concentrating ability and should be replaced, when possible, with measures of urine osmolality.

Osmolality is a measure of the number of particles in solution and is not affected by their size or density. Consequently, osmolality is a more accurate measure of urinary concentration of solids and renal tubular function when compared to specific gravity measurements. In renal failure, the urine/plasma (U/P) osmolality (U/P_{osm}) ratio does not significantly differ from unity. In contrast, patients with prerenal azotemia generate $U/P_{osm} > 1.15$.[5]

Several other measures of renal tubular function, in addition to specific gravity and urine osmolality determinations, have been developed. These include urine sodium, urine/plasma creatinine ratios, and measurements of fractional excretion of filtered sodium (FE_{Na}).[6]

$$(FE_{Na}) = \frac{\text{urine Na/serum Na}}{\text{urine creatinine/serum creatinine}} \times 100$$

These measures of concentrating capacity may be abnormal in patients with renal dysfunction prior to any significant elevations in serum BUN or creatinine and they are helpful in differentiating renal hypoperfusion ("prerenal" azotemia) from intrinsic renal disease in the setting of acute oliguria. As can be seen in Table 10-1, prerenal azotemia is characterized by $U_{osm} > 500$, urine Na <20, U/P creatinine >40, and FE_{Na} <1%. Prerenal azotemia with low fractional excretion of sodium occurs not only with systemic hypovolemia, but also in states characterized by decreased effective circulating blood volume such as congestive heart failure and the hepatorenal syndrome.

Low urinary sodium and FE_{Na} <1% have also been reported with oliguric renal failure induced following radiopaque contrast media, in early obstructive uropathy, in patients who are septic, and in myoglobinuric renal failure.[7,8,9,10] Acute glomerulonephritis may

Table 10-1. Summary of Urinary Indices in Oliguric Renal Failure[a]

	Prerenal	Oliguric acute renal failure
U_{osm}, Mosm/kg H_2O	>500	<300
Urine sodium, meq/liter	<20	>40
Urine/plasma creatinine	>40	<10
Fractional excretion of filtered sodium	<1	>1

[a] From data in references 6 and 67.

present with urinary diagnostic indices similar to those of prerenal azotemia; however, in this instance, the urinalysis can distinguish between these two entities since glomerulonephritis is characterized by red cells and red cell casts in the urine while prerenal azotemia will have a "benign" sediment. In a similar manner, ultrasonography is a very sensitive test to exclude urinary tract obstruction.[11]

Urinary diagnostic indices are less useful in the setting of nonoliguric acute renal failure. In general, the fractional excretion of sodium is greater than 3%; however, fractional excretion of sodium less than 1% has been reported in patients with acute nonoliguric renal failure following burns and in patients with severe liver dysfunction.[12,13] Many patients with nonoliguric renal failure have FE_{Na} >1% and <3%.

Radiographic Monitoring

The value of routine admission and preoperative chest radiographs has been questioned in two recent publications.[14,15] In contrast, daily chest x-rays are indicated for critically ill patients with unstable cardiac pathology, pulmonary disease, or indwelling pulmonary artery catheters or endotracheal tubes. About 20% of these patients will have a previously unsuspected radiographic abnormality. This information causes a change in therapy for more than 90% of these patients.[16] Additional chest x-rays should be obtained following invasive procedures or any major changes in cardiopulmonary status such as fever, worsening hypoxemia, or significant changes in peak airway pressures in mechanically ventilated patients.

Pulmonary interstitial emphysema, an early sign of barotrauma, can be recognized radiographically prior to any clinical signs. This is important since more than half of these patients may develop life-threatening pneumomediastinum or pneumothorax.[17] An early change in ventilatory management in patients with pulmonary interstitial emphysema alone may prevent the progression to pneumothorax. Chest x-rays of patients on mechanical ventilation and high levels of positive and expiratory pressure (PEEP) may be misleading, since radiographic signs of improvement in the absence of change in the underlying pulmonary pathology can occur by virtue of the fact that PEEP leads to an increase in lung volumes. In this instance, radiographic improvement does not necessarily correlate with clinical improvement.

Noninvasive Blood Pressure Monitoring

One of the oldest and most frequently monitored clinical signs is the arterial blood pressure. The Reverend Stephen Hales reported the first measure of blood pressure in a horse in 1731. The modern day sphygmomanometer was introduced in the late 1890s, and in 1905 Korotkoff described the indirect measure of blood pressure by auscultation, which is the technique in clinical use today. Korotkoff described five sounds produced by the flow of blood as the blood pressure cuff is gradually deflated. Phase I describes the first appearance of tapping sounds, which corresponds to systolic blood pressure. During phase II, the tapping sounds change to soft murmurs, followed by louder murmurs during phase III. Phase IV denotes muffling of these murmurs, and phase V marks the absence of sound. Diastolic blood pressure corresponds most closely to phase V sounds.[18] Mean arterial pressure (MAP) is approximated by the equation:

$$MAP = \frac{\text{systolic pressure} + 2\ (\text{diastolic pressure})}{3}$$

Which of these three pressures should be monitored most closely in high-risk and critically ill patients is open to debate and depends, in part, on the clinical situation. For example, during neurosurgical procedures the risk of bleeding correlates most closely with the level of systolic pressure. Myocardial oxygen consumption also correlates most closely with systolic blood pressure in association with heart rate.[19] This is called the rate-pressure product. The importance of diastolic blood pressure rests mainly on the fact that myocardial perfusion occurs during diastole. Overall tissue perfusion seems to correlate best with MAP and for this reason, MAP is the most frequently monitored pressure, especially in low flow states and when vasoactive medications are used.

The auscultatory measurement of blood pressure is not accurate in shock states characterized by high systemic vascular resistance

(SVR). In one study, auscultatory pressure underestimated directly measured systolic blood pressure by 64 mm Hg in hypotensive patients with high SVR. A similar discrepancy between direct and auscultatory blood pressures was not observed in hypotensive patients with low or normal vascular resistance.[20]

Oscillometric methods of blood pressure measurement may be used for the noninvasive measurement of systolic, diastolic, and mean arterial pressures when Korotkoff sounds are diminished or inaudible. Automated machines using this technique are commercially available (Dinamap), and can be programmed to obtain readings as frequently as 1 min apart. Oscillometric measures of arterial pressure are more accurate than simultaneous auscultatory measurements.[21] When pooled data from many patients is obtained comparing oscillometric MAP to directly measured MAP, the mean error is as low as 0.23 mm Hg ± 4.21 mm Hg.[22] Single measurements on an individual patient have a mean error ±2 SD as high as 14 mm Hg or 17%.[23] Although oscillometric pressure measurements are useful in many clinical situations, the rather large error for individual determinations and the fact that as long as 45 sec may be required to obtain a pressure limits the value of this monitoring device for patients who are hemodynamically unstable and for those who require vasoactive drugs for blood pressure control.

Table 10-2. Direct arterial pressure monitoring

Indications:
Cardiovascular instability
Shock
Hypertensive crisis (preeclampsia)
Vasoactive drug infusion
Pulmonary disease
ARDS
Frequent arterial blood gas sampling
Intraoperative
Moderate or severe coronary artery disease
Moderate or severe pulmonary disease
Obesity
Old age
Pelvic exenteration
Complications:
Common
Thrombosis
Infection
Uncommon
Thromboembolism
Air embolism
Aneurysm/pseudoaneurysm
Arteriovenous fistula
Compressive neuropathy
Hemorrhage
Distal ischemia

Invasive Arterial Pressure Monitoring

Direct intraarterial catheterization remains the "gold standard" of blood pressure measurement. Arterial catheters are most frequently required for patients with unstable cardiovascular or pulmonary problems, especially when potent intravenous vasoactive drug therapy is required (Table 10-2). Examples include hypertensive crisis treated with nitroprusside, unstable angina treated with intravenous nitroglycerine, and shock. As noted above, auscultatory blood pressures are inaccurate in the presence of hypotension and elevated systemic vascular resistance. Fluid and vasopressor requirements will change rapidly, and continuous blood pressure measurement is vital for prompt and accurate titration of therapy.

In general, arterial monitoring is a prerequisite whenever infusions of epinephrine, norepinephrine, neosynephrine, or high-dose dopamine are used. The treatment of adult respiratory distress syndrome (ARDS) with mechanical ventilation and positive and expiratory pressure (PEEP) is also facilitated by intraarterial monitoring because of the frequent need for arterial blood gases and because changes in ventilatory management will require concomitant changes in fluid and or vasopressor therapy.

Intraarterial monitoring in the operating suite is indicated for procedures in which major fluid shifts are anticipated, such as pelvic exenterations. Patients with a prior history of cardiac disease often benefit from invasive blood pressure monitoring, since it has been shown that the incidence of perioperative myocardial reinfarction may be reduced by limiting intraoperative blood pressure fluctuations.[24] Obese patients have lower room air P_{O_2} than age-matched nonobese patients, and they are more likely to develop intraoperative hypoxemia, especially when placed in the Trendelenburg position.[25,26] In addition, it is more difficult to obtain accurate auscultatory blood

pressures in obese patients. For these reasons, intraarterial blood pressure monitoring should be considered for this patient population. Arterial lines should also be inserted when patients require frequent intraoperative vasopressor therapy, in the presence of intraoperative shock, and in those patients with significant preoperative pulmonary insufficiency. Since resting arterial oxygenation declines with age, elderly patients may also benefit from arterial catheterization and frequent intraoperative blood gas measurements.[27]

Arterial catheterization is indicated when more than three arterial blood gas measurements are required within 24 hr. Maintaining an arterial line is more comfortable for patients than frequent arterial punctures, there is probably less overall trauma to the vessel, and the measurements should be more accurate since hyperventilation during arterial puncture is eliminated.

Thromboemboli, cerebral embolization following irrigation, aneurysm, and pseudoaneurysm formation, arteriovenous fistula, compressive neuropathy, hemorrhage, and digital ischemia have all been reported as unusual complications of arterial catheterization.[28] The most common complications of arterial catheterization are thrombosis and infection.

The incidence of thrombosis of the radial artery following arterial catheterization is as high as 30%.[29,30] The incidence of thrombosis is proportional to both the duration of cannulation and the proportion of the vessel lumen occupied by the catheter. Cannulations lasting 3 days or less lead to thrombosis in 11% of cases, whereas cannulations lasting 4–10 days result in thrombosis in 29% of cases.[30] Eighteen-gauge cannulas cause radial artery thrombosis four times as frequently as 20-gauge catheters.[31] In almost all cases, the vessel recannulates with no major ischemic complications despite the high incidence of temporary thrombosis.[29,32] Patients who are hypotensive, those receiving vasoactive agents, and prolonged catheterization increase the risk of thrombosis.[33] Multiple punctures of the artery and surgical cutdown also increase the risk of thrombosis.[34,35]

Catheter-associated septicemia occurs in 0%–4% of patients and local infections at the catheter site in about 20%.[36,37] The risk of local or systemic infection becomes significant with catheterizations in excess of 4 days and, in general, rises with increasing duration of catheterization. Surgical cutdown also increases the risk of infection.[36] Rates of local and systemic infection are similar for radial and femoral arterial catheters.[37]

Any peripheral artery may be cannulated; however, the radial and femoral arteries are preferred by many clinicians. Prior to radial artery cannulation, the adequacy of collateral circulation from the ulnar artery should be established by means of an Allen's test.[38] Both radial and ulnar arteries are digitally occluded, and the hand is drained of blood by repetitive opening and closing of the hand. The ulnar artery is then decompressed, and the time for reperfusion should not exceed 5–7 sec. The radial artery should be checked in a similar manner in patients with prior catheterization at this site, since a proximal radial artery occlusion may escape detection if collateral flow from only the ulnar circulation is checked.[39]

Once collateral circulation is established, the wrist should be stabilized and hyperextended on an arm board. Local xylocaine (without epinephrine) anesthesia may be used. Under sterile conditions, a 20-gauge catheter over a stylet is then advanced at a 30° angle directly into the arterial lumen. As soon as blood return is established, the plastic catheter is advanced and the stylet removed. An alternative method involves penetration through both anterior and posterior walls of the vessel. The stylet is removed, and the plastic cannula is slowly withdrawn until blood flows freely. The catheter is then advanced proximally in the arterial lumen.

In recent years, femoral artery catheterization has become as popular as radial artery catheterization, and the femoral artery is the preferred site at our institution. The advantages of femoral catheterization include ease of insertion and increased durability when compared to radial artery catheters. The overall complication rates and the incidence of local and systemic infection are similar for radial and femoral catheters.[34,37,40] The early fear that femoral arterial catheterization would produce significant thromboembolic complications has not been justified.

Femoral catheters are inserted utilizing the Seldinger technique. The groin is shaved, and under sterile conditions an 18-gauge stylet is advanced at an angle of 45° into the lumen of the femoral artery just distal to the inguinal ligament. When a luxuriant blood return is obtained, a guide wire is advanced proximally in the arterial lumen and the stylet is removed. An 18-gauge, 12-cm catheter is then threaded over the guide wire, the wire is removed, and the hub of the catheter is attached to saline-filled pressure tubing. The catheter is secured with two sutures, and the adequacy of peripheral pulses and the arterial wave form are then documented.

Other potential sites for arterial catheterization include the axillary, dorsalis pedis, ulnar, and brachial arteries. Brachial artery catheterization should rarely if ever be attempted, because of the poor collateral circulation to the upper arm. The axillary artery has a relatively large lumen and a good collateral circulation; however, its proximity to the cerebral circulation increases the risk of air or particulate embolization to the brain. Surgical cutdowns at any site should be avoided, if possible, because of the increased incidence of infection.[34,36]

Patency of the lines is maintained with a continuous flush system delivering 3 ml/hr of heparinized solution with a concentration of 2 units/ml. The Center for Disease Control recommends changing flush solutions every 24 hr, replacing tubing, continuous flush devices, and transducer domes every 48 hr, and replacing arterial cannulas every 4 days.[41] The last of these recommendations has recently been challenged;[37] however, it remains a desirable practice if alternate sites for arterial catheterization are available. Any catheter should be removed at the first sign of digital ischemia or local infection and in the presence of systemic signs of infection with no clinically obvious source.

Cardiac Function

Systemic blood pressure is determinated by the product of cardiac output and systemic vascular resistance (BP = CO × SVR). Cardiac output in turn is dependent on ventricular preload, ventricular contractility, and afterload. Although cardiac output from the right ventricle must equal that of the left ventricle over time, the two chambers function with different preloads; they eject blood against significantly different afterloads, and even contractility may vary between the two ventricles. Consequently, ventricular independence as well as ventricular interdependence must be considered in the overall assessment of cardiovascular function.

Starling's law of the heart (Figure 10-2) describes the relationship between ventricular end-diastolic volume, or preload, and force of ventricular contraction. As end-diastolic volume increases, the force of contraction and/or extent of myocardial fiber shortening increases, leading to increases in stroke volume. It is important to emphasize that preload is a measure of ventricular end-diastolic volume. Although changes in ventricular end-diastolic pressures often reflect changes in ventricular end-diastolic volumes, and are followed clinically by central venous and pulmonary capillary wedge pressures, ventricular end-diastolic volumes rather than end-diastolic pressures are synonymous with preload. Increased contractility will shift the Starling curve up and to the left, whereas decreased contractility will shift the curve down and to the right.

Compliance is a reflection of the relationship between changes in volume and associated changes in pressure. Figure 10-3 illustrates a family of ventricular compliance curves.

Figure 10-2. Starling's law of the heart. If left-ventricular end-diastolic volume (*LVED Vol*) or preload increases, left-ventricular stroke work (*LVSW*) and stroke volume (*SV*) will increase. For any given LVED vol, the absolute SV will depend on ventricular contractility. Ventricles with increased, normal, and decreased contractility are illustrated in this figure. *NL*, normal contractility.

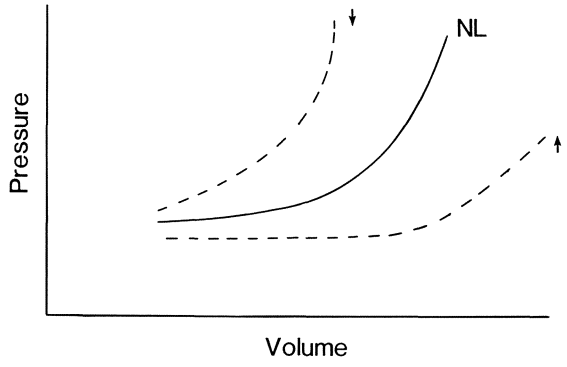

Figure 10-3. Ventricular compliance curves. Compliance measures the change in pressure for any given change in volume. Ventricles with decreased, normal, and increased compliance are illustrated. Decreases in ventricular compliance will shift the pressure–volume curve up and to the left, whereas increases in compliance will shift the curve down and to the right (*arrows*). For any given ventricular pressure, more compliant ventricles will accommodate larger volumes of fluid. *NL,* normal compliance.

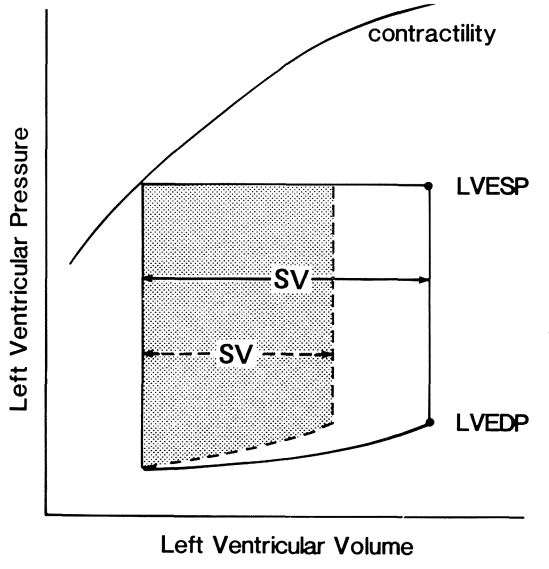

Figure 10-4. Effect of ventricular compliance with afterload and contractility remaining constant. During diastole, the ventricle fills until it reaches a given left-ventricular end-diastolic pressure (*LVEDP*). Ventricular contraction begins and is isovolumetric until the onset of ejection, when left-ventricular end systolic pressure (*LVESP*) is reached. The stroke volume is ejected, then isovolumetric relaxation completes the cardiac cycle. The shaded ventricular pressure–volume loop represents a less compliant ventricle when compared to the open loop. For any given LVEDP, the less compliant ventricle will have a decreased LVEDVol (i.e., preload) and consequently eject a smaller stroke volume. *SV,* stroke volume.

Myocardial ischemia, shock, positive end expiratory pressure (PEEP), pericardial effusion, and inotropic drug therapy are associated with decreased ventricular compliance. Some cardiomyopathies and vasodilating drugs such as nitroprusside and nitroglycerine are associated with increases in ventricular compliance.

Figure 10-4 illustrates the effects of changes in ventricular compliance on stroke volume. In this example, ventricular end-diastolic pressure, contractility, and afterload are constant; however, the shaded ventricle is less compliant. Although ventricular contraction is initiated at the same left ventricular end-diastolic pressure (LVEDP), the more compliant ventricle will accommodate an increased volume (i.e., increased preload) and consequently eject a larger stroke volume. Figure 10-5 shows that increasing afterload will lead to a decrease in stroke volume when contractility and preload remain constant.

In clinical practice, cardiac contractility cannot be measured. Afterload can be approxi-

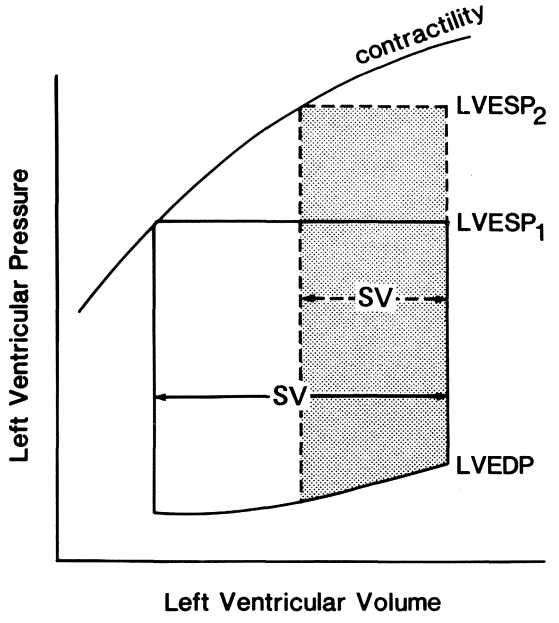

Figure 10-5. Effect of increasing afterload on stroke volume when preload and contractility are constant. When afterload is increased, a higher left-ventricular end-systolic pressure ($LVESP_2$) must be generated prior to opening of the aortic valve and ejection of the stroke volume. The shaded ventricular pressure–volume loop illustrates how increases in afterload will lead to decreases in *SV* for any given preload and contractility. *LVEDP,* left-ventricular end-diastolic pressure.

mated by measuring the systemic vascular resistance index (SVRI) by the following equation:

$$SVRI = \frac{(MAP - CVP) \times 80}{CI}$$

(CI = cardiac index)

Preload is synonomous with end-diastolic volume. End-diastolic volume is dependent on both ventricular compliance (Figure 10-3) and the sum of pressures acting to distend the ventricle, or the transmural pressure (P_{TM}). Transmural pressure of the left ventricle, for example, is equal to pressure within the vascular system, or left ventricular end-diastolic pressure (LVEDP) minus juxtacardiac pressure or pleural pressure (P_{PL}) (Figure 10-6). Consequently, P_{TM} = LVEDP − P_{PL}. Pulmonary capillary wedge pressures provide the best clinical estimate of LVEDP. P_{PL} can only be estimated in clinical situations, since physicians do not routinely insert pressure catheters into the pleural cavity. This interaction between ventricular compliance, pleural pressure, and intravascular pressure is of great clinical importance since absolute values for PCWP are of value only if the effect of ventricular compliance and pleural pressure, neither one of which is easily measured, are considered.

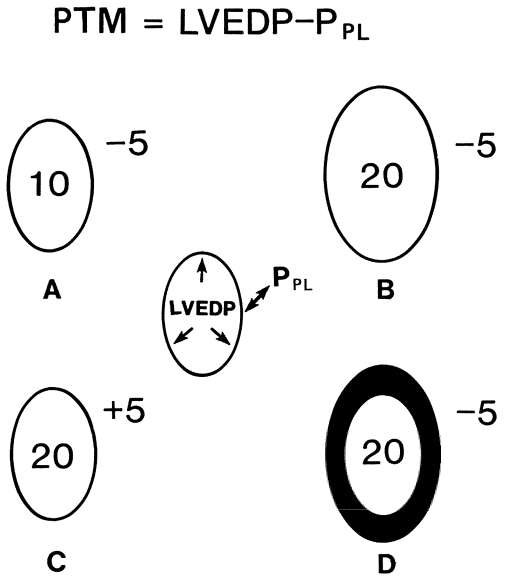

Figure 10-6. Transmural pressure and ventricular preload. The ovals represent a left ventricle. Ventricular preload is dependent on transmural pressure and ventricular compliance. **A,** A ventricle with normal compliance, *LVEDP*, and P_{PL}. *PTM* = 10 − (−5) = 15. Preload is normal. **B,** Increased LVEDP with normal compliance and P_{PL}. PTM = 20 − (−5) = 25. Preload is elevated with ventricular distension. Congestive heart failure is an example. **C,** Elevated LVEDP and P_{PL} with normal compliance. PTM = 20 − 5 = 15. Preload is normal. Mechanical ventilation with positive end-expiratory pressure is an example. **D,** Elevated LVEDP and normal P_{PL} with a poorly compliant ventricle. PTM = 20 − (−5) = 25. Elevated transmural pressures are necessary to maintain a normal LVEDVol or preload when the ventricle is less compliant. Hypertensive cardiomyopathy is an example.

Three different interpretations for an elevated PCWP are illustrated in Figure 10-6. Therapeutic interventions should never be based on single PCWP readings unless they are very high or very low. Monitoring of trends following diuresis or volume loading is necessary, since we are unable to measure all the variables affecting ventricular function. PCWP measurements should also be accompanied by intermittent measurement of cardiac output and mixed venous oxygen saturation.

Preload may be directly measured by two-dimensional echocardiography or through the use of nuclear ventriculograms. These two methods of measuring preload are not routinely available in the clinical setting. Consequently, we can only estimate right- and left-ventricular preload through the use of central venous and pulmonary capillary wedge pressure measurements, respectively.

Central Venous Pressure Monitoring

Central venous pressure monitoring became widely used in the 1960s. Central venous pressures (CVP) accurately measure right-ventricular filling pressures, and consequently provide an indirect measure of both right-ventricular preload and right-ventricular function. In patients with no underlying cardiac or pulmonary pathology, the CVP correlates reasonably well with pulmonary capillary wedge pressures (PCWP).[42] Central venous pressure measurements are valuable guides to fluid therapy early in the treatment of hypovolemic or septic shock and during surgical procedures that are associated with large fluid flux (Table 10-3). Central venous lines are also essential for the administration of total parenteral nutrition. They may be used to gain venous access when peripheral veins are not available, and for the

Table 10-3. Central venous pressure monitoring

Indications:
- Measurement of central venous pressure
- Rapid fluid administration
- Parenteral nutrition
- Venous access

Complications:
- Common
  - Pneumothorax
  - Arterial puncture
  - Bleeding
  - Hematoma formantion
  - Local/systemic infection
- Uncommon
  - Air embolus
  - Hydrothorax
  - Hydromediastinum
  - Thoracic duct laceration
  - Hemothorax
  - Hemomediastinum
  - Aortic puncture
  - Catheter embolus
  - Catheter knotting
  - Horners Syndrome
  - Tracheal perforation
  - Thrombophlebitis
  - Thrombosis
  - Arteriovenous fistula

administration of irritating medications such as concentrated potassium chloride solutions or for chemotherapy.

Central venous lines may be connected to water manometers to obtain pressure measurements; however, when possible, it is preferable to obtain readings from a pressure transducer. Water manometer measurements are relatively inaccurate even if the system fluctuates well with respiration, and blood is easily aspirated since the system can respond to only one or two pressure variations or 1–2 Hz because of the physical limitations of fluid movement in narrow tubing. If the pressure varies considerably during a cardiac or respiratory cycle or if pressure changes are rapid, the water column falls to the highest level. Consequently, large *a* waves, significant tricuspid regurgitation, large variations in intrathoracic pressure during mechanical respiration, and tachycardia (>120 BPM) all tend to result in erroneously high pressure measurements when a water manometer system is utilized (Figure 10-7).

Pressure transducers convert hydraulic pressure changes into electrical signals that can be displayed on an oscilloscope. Central venous pressure is characterized by three positive deflections called *a, c,* and *v* waves (Figure 10-7). The *a* wave is produced by right atrial contraction and begins after the first heart sound. It follows the P wave of the ECG. The second deflection, or *c* wave, is produced by bulging of the tricuspid valve into the right atrium during the onset of ventricular contraction occurring after the QRS complex of the ECG. The *x* descent results from additional atrial relaxation and downward displacement of the ventricle and tricuspid valve during ventricular systole. The *v* wave represents right atrial filling against a closed tricuspid valve. The *y* descent corresponds to opening of the tricuspid valve with emptying into the right ventricle. The central venous pressure represents the mean

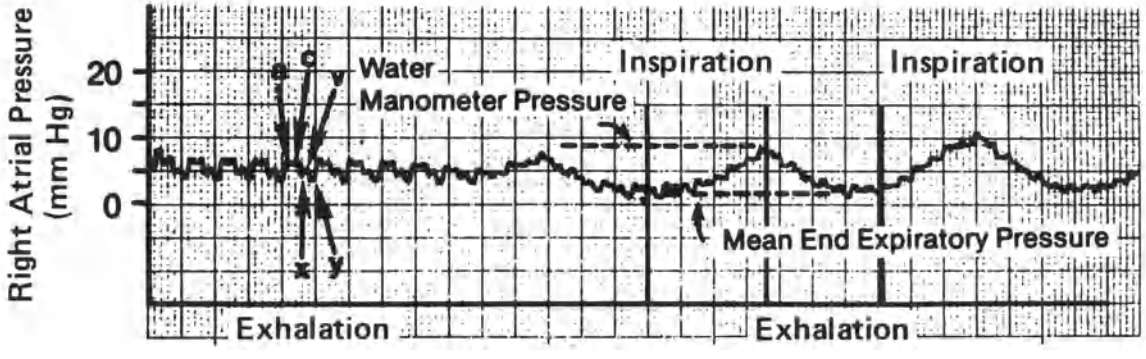

Figure 10-7. Typical CVP waveform of patient receiving mechanical ventilation. The *a, c,* and *v* waves and *x* and *y* descents are illustrated. The lowest pressure measured by a water manometer is often the highest pressure measured by the transducer during mechanical inspiration. The most accurate central venous pressure is the mean end expiratory pressure. (From Otto CW. Central venous pressure monitoring. In: Blitt CD, ed. Monitoring in anesthesia and critical care medicine. New York: Churchill-Livingstone, 1985: p. 127, Figure 7-5.)

value of these pressure waves obtained at end expiration.

The incidence of complications associated with the insertion of central venous catheters appears to be inversely proportional to the skill of the physician. Complication rates of 0.4%–9.9% have been reported.[43] Pneumothorax, bleeding, hematoma formation, and inadvertent arterial puncture are the most common complications during catheter insertion (Table 10-3). Air embolus, hydrothorax, hydromediastinum, thoracic duct laceration, hemothorax, hemomediastinum, aortic puncture, catheter embolus, catheter knotting, Horner's syndrome, bilateral vocal cord paralysis, and tracheal perforation are uncommon, but reported, complications of central vein catheterization.[44] Late complications of central venous lines include thrombophlebitis, thrombosis, cellulitis of the insertion site, catheter-related sepsis, and arteriovenous fistula formation.[44]

Internal and external jugular, subclavian, antecubital, and femoral veins may all be used to gain central venous access. The advantage of the antecubital and external jugular approach is that they are both peripheral veins and the morbidity of insertion is that of any peripheral vein insertion. One of these sites should be considered for patients with a coagulopathy, since tamponade of bleeding is feasible. Disadvantages of the antecubital approach include a high incidence of thrombophlebitis, thrombosis, and failure to reach the central circulation.[45] Difficulty in passing a J wire through the valves of the external jugular vein and into the central circulation occurs in about 25% of external jugular vein catheterization attempts.[46] Femoral veins provide easy access; however, because of difficulty in threading the catheter into the right atrium in the absence of fluroscopy, femoral venous catheters are infrequently used for pressure monitoring.

The internal jugular and subclavian veins are the most common sites for central venous catheter insertion. The various techniques of insertion are beyond the scope of this chapter; however, several good reviews are available.[45,47] Internal jugular catheterization is associated with a lower incidence of pneumothorax when compared to a subclavian approach and hematomas are visible and compressible. In contrast, subclavian catheters are more comfortable for most patients, and it is easier to maintain a sterile occlusive dressing. Subclavian catheterization of patients with a coagulopathy should be avoided since tamponade of bleeding is ineffective.

The initial enthusiasm for monitoring central venous pressures was predicated on the belief that knowledge of right-ventricular filling pressures and right-ventricular function would reflect similar changes in left-ventricular filling pressures and function. As noted above, this is true for many patients with no significant cardiopulmonary dysfunction following trauma or major abdominal surgery. Does the same correlation hold true for other critically ill patients?

Forrester simultaneously measured filling pressures of the right (CVP) and left (PCWP) ventricles following acute myocardial infarction.[48] He found a poor correlation between absolute values of CVP and PCWP. When filling pressures were correlated with the presence or absence of radiologic pulmonary edema, a PCWP of 18 provided an excellent separation between these two groups, whereas no absolute value for CVP was able to distinguish between those patients with or without pulmonary edema. Moreover, changes in PCWP following volume loading or diuresis were accompanied by either no change in CVP (12%) or directionally opposite changes in CVP (16%). The relative magnitude of changes for the remaining patients was highly variable. A similar lack of correlation is found in patients with cirrhosis, chronic obstructive airway disease, depressed left-ventricular ejection fractions, regional left-ventricular wall motion abnormalities, sepsis, or multiple trauma with associated cardiac and pulmonary disease.[42,49,50] Conditions in which CVP is less accurate than PCWP are frequently present in many critically ill patients.

Swan–Ganz Catheterizations

Over 2 million Swan–Ganz catheters have been inserted since 1970 when Swan, Ganz, and Forrester introduced this valuable tool to clinical medicine. The standard 7-French Swan–Ganz catheter contains four lumens; one for balloon inflation, a second for the thermistor wires, and two lumens for infusion or pressure monitoring. The distal lumen ends at

the tip of the catheter, and may be utilized to monitor pulmonary artery pressures and to obtain mixed venous blood samples. The proximal lumen is located 30 cm from the catheter tip and may be used for drug infusions or to monitor central venous pressures. Recently, an oximetric Swan–Ganz catheter has been developed containing all of the features noted above in addition to fiberoptic filaments that provide continuous monitoring of mixed venous oxygen saturation. Both of these catheters may be used to obtain thermodilution cardiac outputs.

The data provided by Swan–Ganz catheterization has markedly improved our ability to monitor cardiovascular function, since changes in clinical signs and even changes in central venous pressure measurements occur much later or even fail to detect subtle but significant changes in left-ventricular function. Diagnostic speed and accuracy are improved by monitoring pulmonary capillary wedge pressures, cardiac output and mixed venous oxygen saturation.

The principle of pulmonary capillary wedge pressure measurement is illustrated in Figure 10-8. Occlusion of the Swan–Ganz catheter balloon sets up a static column of blood between the catheter tip and the remainder of the pulmonary venous system. Constriction of the vessel distal to the alveolus, associated for example with pulmonary hypertension, will not cause any significant variation between PCWP and LVEDP. However, if a constriction is located more distally in the path of the flowing column of blood, as in mitral stenosis or pulmonary venous occlusion, then PCWP will exceed LVEDP. Although structural pulmonary venous constriction is rare, functional pulmonary venous constriction may occur in septic patients with adult respiratory distress syndrome.[51]

Thermodilution cardiac output determinations are comparable in accuracy to dye-dilution methods.[52] Since cardiac output is exquisitely sensitive to changes in intrathoracic pressure and associated changes in venous return, measures of cardiac output may vary by ±20% depending on where in the ventilatory cycle the measurement was obtained.[53] Consequently, thermodilution cardiac outputs should be done in triplicate at different phases of the ventilatory cycle and the results averaged.

Mixed venous oxygen saturation ($P_{\bar{V}_{O_2}}$) is another important variable to monitor in the critically ill patient. Although there is a rather large interorgan variation in oxygen utilization, mixed venous oxygen saturation provides a gross measure of the adequacy of total body oxygen delivery. Blood for intermittent sampling of mixed venous oxygen saturation may be obtained by aspirating slowly from the distal

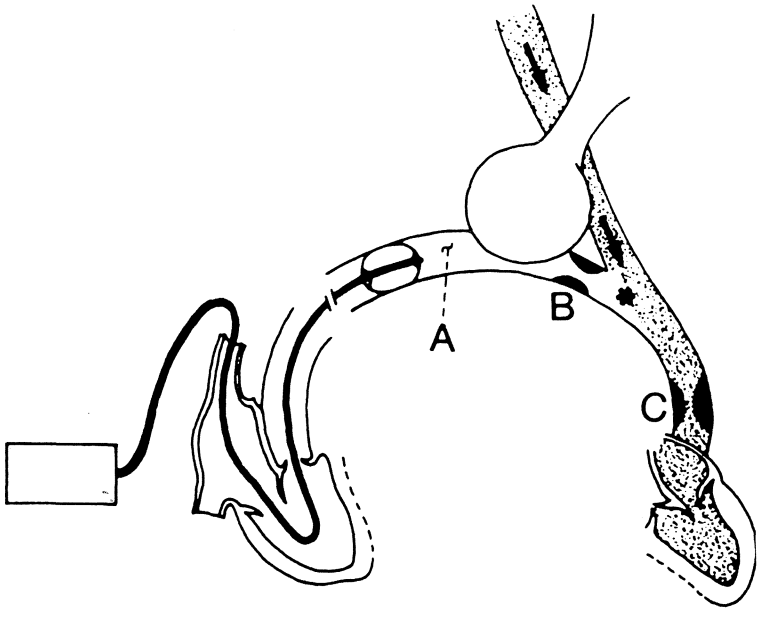

Figure 10-8. Principle of the pulmonary artery wedge pressure measurement. During balloon occlusion of the pulmonary artery, blood flow ceases between the catheter tip (*A*), and the junction point for veins serving occluded and nonoccluded units (*asterisk*). Narrowing of the static column (*B*) will not influence PCWP; however, similar narrowing of the flowing column at point *C* caused, for example, by pulmonary venous constriction, will cause PCWP to overestimate LVEDP. Mitral stenosis would lead to a similar elevation of the PCWP. (From O'Quin R, Marini JJ. Pulmonary artery occlusion pressure: Clinical physiology, measurement and interpretation. Am Rev Respir Dis. 1983; 128:320, Figure 1. Reprinted with permission.)

port of standard Swan Ganz catheters. Continuous $P\bar{v}_{O_2}$ saturation monitoring is available through the use of oximetric Swan–Ganz catheters. Continuous $P\bar{v}_{O_2}$ saturation monitoring offers significant advantages over intermittent cardiac output and $P\bar{v}_{O_2}$ determinations.

Dr. H.J.C. Swan[54] suggested that pulmonary artery catheterization is indicated for "any situation in which a physician would consider placing a central venous pressure line for the purpose of cardiovascular monitoring" (Table 10-4). This statement is based on the fact that although central venous pressures adequately assess right-ventricular function, in the presence of cardiac or pulmonary disease the CVP often fails to provide accurate information of instantaneous left-ventricular function. In shock states, a CVP may be adequate for initial fluid resuscitation; however, Swan–Ganz catheter insertion is indicated if the patient does not respond quickly to initial fluid replacement. This is especially true for cases of septic shock, which are associated with a high incidence of noncardiogenic pulmonary edema.

Swan–Ganz catheterization is indicated for myocardial infarction complicated by hemodynamic instability, so that vasoactive or inotropic agents can be titrated to the desired endpoint.[55] Right-heart catheterization is also necessary to differentiate acute mitral regurgitation from acute rupture of the interventricular septum.

Patients requiring vasoactive drugs for blood pressure support often require pulmonary artery catheterization to monitor therapy. Therapy of malignant hypertension utilizing nitroprusside may be instituted with intraarterial blood pressure monitoring alone; however, if pulmonary edema or cardiac dysfunction are associated problems, Swan–Ganz monitoring is necessary to adequately monitor vasodilator therapy. This principle would apply to the preeclamptic patient as well. Meaningful titration of intravenous nitroglycerine in patients with unstable angina also requires Swan–Ganz pressure monitoring.

Swan–Ganz catheterization is almost invariably necessary to monitor therapy of patients with adult respiratory distress syndrome (ARDS). This syndrome, which has numerous causes, is characterized by noncardiogenic pulmonary edema. Ventilatory support with high positive end expiratory pressure (PEEP) is the cornerstone of therapy; however, PEEP depresses cardiac output. When high levels of PEEP are used, volume loading and inotropic support are often required to maintain cardiac output without excessive elevation in the PCWP. For all of these reasons, Swan–Ganz catheterization is helpful to guide changes in ventilatory management, fluid therapy, and vasoactive drug support.

Thus far we have discussed nonelective indications for Swan–Ganz catheterization. Many high-risk surgical patients, especially those with physical status scale IV or V, benefit from elective Swan–Ganz catheterization. Although criteria defining these patients are not well established, patients with preexistent cardiac or pulmonary disease who are scheduled for extensive surgical procedures or procedures in which large volume requirements are anticipated may benefit from hemodynamic monitoring, for example, pelvic exenteration.

Complications associated with pulmonary artery catheterization occur while obtaining central venous access, during insertion of the catheter through the heart, and with long-term maintenance of the catheter. The first and last types of complications are essentially identical in type and incidence to those of central venous lines such as pneumothorax, thrombosis, hemorrhage, or catheter-related sepsis. Complications inherent to the Swan–Ganz catheter include arrhythmias, conduction disturbances, catheter knotting, balloon rupture, pulmonary

Table 10-4. Indications for Pulmonary Artery Catheterization[a]

Differentiation of cardiogenic and noncardiogenic pulmonary edema
Guidance of fluid and vasopressor therapy in septic shock
Titration of intravenous vasoactive medication
Optimization of PEEP and fluid therapy in ARDS
Myocardial infarction complicated by hypotension, acute rupture of the interventricular septum, or acute mitral regurgitation
Diagnosis of cardiac tamponade
Malignant hypertension (preeclampsia) with pulmonary edema
High-risk surgical patients

[a] From *Journal of the American Medical Association.* 1975; 233:865–867. Copyright 1975, American Medical Association.

infarction, and pulmonary artery rupture. Most of these complications can be avoided by meticulous attention to technique.

Change in cardiac rhythm is the most common complication of pulmonary artery catheterization.[56,57] Ventricular premature contractions and ventricular tachycardia are the most common arrhythmias. These arrhythmias are usually precipitated during passage of the catheter through the right ventricle and respond, in almost all cases, to lidocaine and removal of the catheter. Myocardial ischemia, hypoxemia, and acidosis increase the risk of ventricular ectopy.[58] Atrial arrhythmias including fibrillation and flutter have been reported but are uncommon.[59] Transient right bundle branch block occurs in about 5% of right-heart catheterizations.[58]

Pulmonary infarction initially was one of the major complications of pulmonary artery catheterization; however, in recent years it has become a relatively infrequent problem due to more meticulous catheter care and the recent introduction of heparin-bonded catheters. Pulmonary artery rupture occurs with a frequency of 0.08%, and is almost universally lethal.[57] Patients with pulmonary hypertension, the elderly, and females are at increased risk for this complication.[60] Placement of the pulmonary catheter in the proximal pulmonary artery and slow inflation of the balloon until a wedge pressure is obtained should prevent this lethal complication.

Respiratory Monitoring

Although methods for monitoring the cardiovascular system have changed dramatically during the last two decades, routine monitoring of pulmonary function has changed little during this time. Clinical examination and arterial blood gas determinations remain the most important monitoring modalities, supplemented by relatively simple tests of ventilatory volumes and mechanics. Transcutaneous measurement of arterial oxygen and carbon dioxide tensions, mass spectrometry, and pneumotachometers are available, but are not in routine use for pulmonary monitoring because of numerous technical problems, high cost, and lack of reliability.

Bedside observation of respiratory rate, patterns of ventilation and the use of the accessory muscles of ventilation is the most important respiratory monitoring technique. Although respiratory rate can be monitored electronically by utilizing special electrocardiographic electrodes that measure changes in chest impedance, detection of abnormal ventilatory patterns and other signs of respiratory distress depend on close bedside observation.

Tachypnea is often the first clinical manifestation of pulmonary pathology. Both hypoxia and hypercarbia will initiate an increase in respiratory rate.[61] A decrease in pulmonary compliance, caused by pulmonary edema or pneumonia for example, will increase the work of breathing. The patient's most efficient compensatory response is to increase respiratory rate coupled with a decrease in tidal volume.[62]

Use of the accessory muscles of respiration and nasal flaring are sensitive signs of respiratory distress, which may serve as indications for endotracheal intubation despite normal blood gases. Abnormal breathing patterns such as Cheyne–Stokes respirations or paradoxical thoracoabdominal breathing are other signs of significant cardiac or pulmonary pathology that can be detected only by close bedside observation.

Clinical evaluation of skin color is an important although grossly insensitive estimate of the adequacy of oxygenation. Almost 40 years ago, Jules Comroe showed that cyanosis could not be detected clinically until oxygen saturation fell to 75%–80%.[63] In addition, there were marked variations in the ability of the clinician to note cyanosis in different patients and even in the same patients at different times. For this reason, arterial blood gas monitoring is an essential diagnostic tool not only for monitoring of acid–base balance but also for documenting adequate oxygenation and ventilation. The frequency of blood gas measurement depends on the clinical situation. Certainly, blood gases should be monitored following every ventilator adjustment or if there is any significant change in vital signs or mental status.

Tidal volume, forced vital capacity, minute ventilation (tidal volume × respiratory rate) and maximum inspiratory force are easily measured at the bedside for both intubated and mechanically ventilated patients. Serial measurements of these variables help to quantitate improvement or deterioration in pulmo-

nary function and are valuable for predicting successful weaning from mechanical ventilation. Clinical estimates of alveolar ventilation, on the other hand, are grossly inaccurate.[64]

Normal tidal volume averages 7 ml/kg; 2 ml/kg represents dead-space ventilation and the remaining 5 ml/kg represents alveolar ventilation. The forced vital capacity should exceed 10 ml/kg prior to extubation so that tidal volumes after extubation do not exceed 60% of forced vital capacity. Minute ventilation (MV) of less than 10 liters/min and the ability to double MV on command also correlate with successful extubation.[65]

Maximal negative inspiratory force (NIF) is a crude estimation of patient strength. Normally, NIF exceeds -80 cm/H_2O. A NIF more negative than -25 cm/H_2O usually predicts successful weaning from mechanical ventilation.[65]

When a patient is intubated and mechanically ventilated, the pressure manometer of the

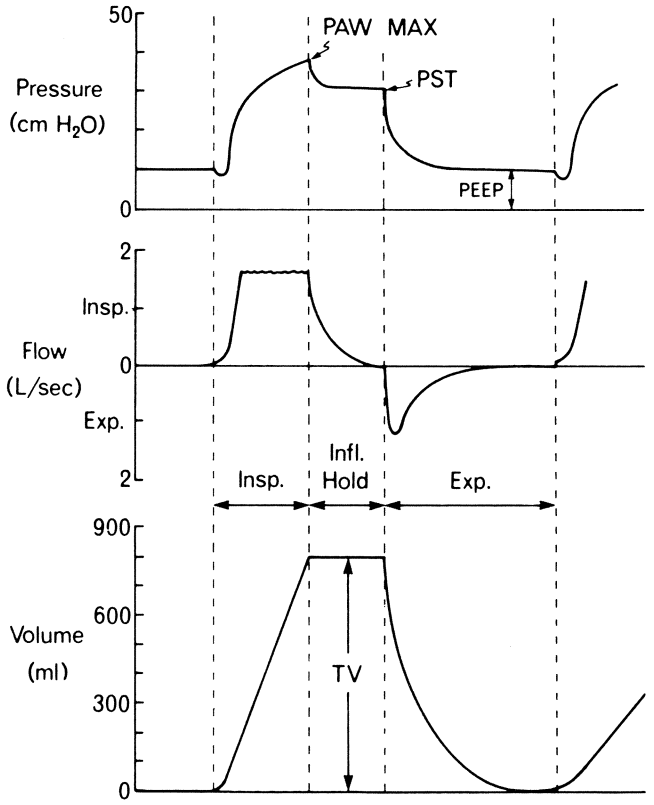

Figure 10-9. Pressure, flow, and volume changes with mechanical ventilation. Airway pressures may be obtained from the pressure gauge on the ventilator. Flow is measured by a spirometer. The mechanical breath is initiated by a decrease in airway pressure generated by the patient. The ventilator delivers a preset tidal volume with airway pressure rising to peak levels at end-inspiration (*PAW*). During the inflationary hold, flow falls to zero; however, the lungs remain distended and a static airway pressure (*PST*) is obtained prior to exhalation. Effective dynamic compliance (C_{dyn}) = TV ÷ (PAW − PEEP). Static lung compliance (C_{st}) = TV ÷ (PST − PEEP). *TV* = tidal volume. *PEEP* = positive end expiratory pressure. (From Fallat RJ. Respiratory Monitoring. In: Bone RC, ed. Critical care. A comprehensive approach. Park Ridge, IL: American College of Chest Physicians. 1984;194, Fig. 4.)

volume ventilator provides useful information about pulmonary mechanics.[66] Peak inspiratory pressures during a mechanical breath can be obtained, and the effective dynamic compliance (C_{dyn}) of the lungs and chest wall calculated by the following equation:

C_{dyn} = tidal volume (TV) ÷
[Peak Airway Pressure (PAW) −
Positive end expiratory pressure (PEEP)].

With an inspiratory hold, or if the exhalation port is occluded following inspiration, the peak airway pressures will decrease to a plateau pressure (PST), as noted in Figure 10-9. The static lung compliance can then be calculated by the following equation: C_{st} = TV ÷ (PST − PEEP). Changes in airway resistance, caused by bronchospasm or mucous plugs, will increase PAW without changing plateau pressures. Changes in overall lung compliance from pneumonia, pulmonary edema, pneumothorax, or an occluded or misplaced endotracheal tube will all increase peak inspiratory pressures and PST prior to any sign of clinical deterioration. Consequently, close monitoring of changes in static and dynamic lung compliance is a valuable monitoring device in intubated patients.

Concluding Remarks

This chapter provides an overview of basic monitoring modalities for critically ill and high-risk patients and some general guidelines as to when these diagnostic strategies should be employed. In the future, less invasive methods of monitoring will become available. Whether monitoring is invasive or not, intimate knowledge of the capabilities and deficiencies of our monitoring techniques is crucial so that informed therapeutic decisions can be made. The monitoring techniques now at our disposal, and those of the future, must complement but never displace careful bedside clinical examination.

References

1. Anderson RJ, Linas SL, Berns AS, et al. Nonoliguric acute renal failure. New Engl J Med. 1977; 296:1134–1138.
2. Kassirer JP. Clinical evaluation of kidney function–glomerular function. New Engl J Med. 1971; 285:385–389.
3. Cockroft DW, Gault MH. Prediction of creatinine clearance from serum creatinine. Nephron. 1976; 16:31–41.
4. Galambos JT, Herndon EG, Reynolds GH. Specific gravity determination. Fact or fancy? New Engl J Med. 1964; 270:506–508.
5. Myers BD, Hilberman M, Spencer RJ, et al. Glomerular and tubular function in non-oliguric acute renal failure. Am J Med. 1982; 72:642–649.
6. Miller TR, Anderson RJ, Linas SL, et al. Urinary diagnostic indices in acute renal failure. A prospective study. Ann Intern Med. 1978; 89:47–50.
7. Steiner RW. Low fractional excretion of sodium in myoglobinuric renal failure. Arch Intern Med. 1982; 142:1216–1217.
8. Fang LST, Sirota RA, Ebert TH, et al. Low fractional excretion of sodium with contrast media—induced acute renal failure. Arch Intern Med. 1980; 140:531–533.
9. Hoffman LM, Suki WN. Obstructive uropathy mimicking volume depletion. JAMA. 1976; 236:2096–2097.
10. Vaz AJ. Low fractional excretion of urine sodium in acute renal failure due to sepsis. Arch Intern Med. 1983; 143:738–739.
11. Ellenbogen PH, Scheible FW, Talner LB, et al. Sensitivity of grey-scale ultrasound in detecting urinary tract obstruction. AJR. 1978; 130:731–733.
12. Planas M, Wachtel T, Frank H, et al. Characterization of acute renal failure in the burned patient. Arch Intern Med. 1982; 142:2087–2091.
13. Diamond JR, Yoburn DC. Nonoliguric acute renal failure associated with low fractional excretion of sodium. Ann Intern Med. 1982; 96:597–600.
14. Rucker L, Frye EB, Staten MA. Usefulness of screening chest roentgenograms in preoperative patients. JAMA. 1983; 250:3209–3211.
15. Hubbell FA, Greenfield S, Tyler JL, et al. The impact of routine admission chest x-ray films on patient care. New Engl J Med. 1985; 312:209–213.
16. Strain DS, Kinasewitz GT, Vereen LE, et al. Value of routine daily chest x-rays in the medical intensive care unit. Crit Care Med. 1985; 13:534–536.
17. Johnson TH, Altman AR. Pulmonary interstitial gas: first sign of barotrauma due to PEEP therapy. Crit Care Med. 1979; 7:532–535.
18. Braunwald E. The physical examination. In: Braunwald E, ed. Heart disease. A textbook of cardiovascular medicine (Vol. 2). Philadelphia: WB Saunders, 1984: p. 14–40.
19. Gobel FL, Nordstrom LA, Nelsen RR, et al. The

rate-pressure product as an index of myocardial oxygen consumption during exercise in patients with angina pectoris. Circulation. 1978; 57:549–556.
20. Cohn JN. Blood pressure measurement in shock. JAMA. 1967; 199:118–122.
21. Van Bergen FH, Weatherhead DS, Treloar AE, et al. Comparison of indirect and direct methods of measuring arterial blood pressure. Circulation. 1954; 10:481–490.
22. Ramsey M. Noninvasive automatic determination of mean arterial pressure. Med Biol Eng Comput. 1979; 17:11–18.
23. Yelderman M, Ream AK. Indirect measurement of mean blood pressure in the anesthetized patient. Anesthesiology. 1979; 50:253–256.
24. Rao TLK, Jacobs KH, El-Etr AA. Reinfarction following anesthesia in patients with myocardial infarction. Anesthesiology. 1983; 59:499–505.
25. Vaughan RW, Englehardt RC, Wise L. Postoperative hypoxemia in obese patients. Ann Surg. 1974; 180:877–882.
26. Vaughan RW, Wise L. Intraoperative arterial oxygenation in obese patients. Ann Surg. 1976; 184:35–42.
27. Sorbini CA, Grassi V, Solinas E, et al. Arterial oxygen tension in relation to age in healthy subjects. Respiration. 1968; 25:3–13.
28. Gauer PK, Downs JB. Complications of arterial catheterization. Respir Care. 1982; 27:435–444.
29. Slogoff S, Keats AS, Arlund C. On the safety of radial artery cannulation. Anesthesiology. 1983; 59:42–47.
30. Bedford RF. Long-term radial artery cannulation: effects on subsequent vessel function. Crit Care Med. 1978; 6:64–66.
31. Bedford RF. Radial artery function following percutaneous cannulation with 18- and 20-gauge catheters. Anesthesiology. 1977; 47:37–39.
32. Bedford RF, Wollman H. Complications of percutaneous radial-artery cannulation. Anesthesiology. 1973; 38:228–236.
33. Gardner RM, Schwartz R, Wong HC, et al. Percutaneous indwelling radial-artery catheters for monitoring cardiovascular function. New Engl J Med. 1974; 290:1227–1231.
34. Russell JA, Joel M, Hudson RJ, et al. Prospective evaluation of radial and femoral artery catheterization sites in critically ill adults. Crit Care Med. 1983; 11:936–939.
35. Davis FM, Stewart JM. Radial artery cannulation. A prospective study in patients undergoing cardiothoracic surgery. Br J Anesth. 1980; 52:41–46.
36. Band JD, Maki DG. Infections caused by arterial catheters used for hemodynamic monitoring. Am J Med. 1979; 67:735–741.
37. Thomas F, Burke JP, Parker J, et al. The risk of infection related to radial vs femoral sites for arterial catheterization. Crit Care Med. 1983; 11:807–812.
38. Allen EV. Thromboangitis obliterans: methods of diagnosis of chronic occlusive arterial lesions distal to the wrist, with illustrative cases. Am J Med Sci. 1929; 178:237–244.
39. Meyer RM, Katele GV. The case for a complete Allen's test. Anesth Analg. 1983; 62:947–948.
40. Soderstrom CA, Wasserman DH, Dunham CM, et al. Superiority of the femoral artery for monitoring. Am J Surg. 1982; 144:309–312.
41. Centers for Disease Control: Guidelines for prevention of infections related to intravascular pressure-monitoring systems. Infect Control. 1982; 3:68.
42. Toussaint GPM, Burgess JH, Hampson LG. Central venous pressure and pulmonary wedge pressure in critical surgical illness. Arch Surg. 1974; 109:265–269.
43. Borja AR. Current status of infraclavicular subclavian vein catheterization. Ann Thorac Surg. 1972; 13:615–624.
44. McGoon MD, Benedetto PW, Greene BM. Complications of percutaneous central venous catheterization. A report of two cases and review of the literature. Johns Hopkins Med J. 1979; 145:1–6.
45. Otto CW. Central venous pressure monitoring. In: Blitt CD, ed. Monitoring in anesthesia and critical care medicine. New York: Churchill-Livingstone. 1985:121–165.
46. Belani KG, Buckley JJ, Gordon JR, et al. Percutaneous cervical central venous line placement: A comparison of the internal and external jugular vein routes. Anesth Analg. 1980; 59:40–44.
47. Civetta JM. Pulmonary artery catheterization. In: Sprung CL, ed. The pulmonary artery catheter. Methodology and clinical applications. Baltimore: University Park, 1983: p. 21–71.
48. Forrester JS, Diamond G, McHugh TJ, et al. Filling pressures in the right and left sides of the heart in acute myocardial infarction. New Engl J Med. 1971; 285:190–193.
49. Civetta JM, Gabel JC. Flow-directed pulmonary artery catheterization in surgical patients: indications and modifications of technic. Ann Surg. 1973; 176:753–756.
50. Mangano DT. Monitoring pulmonary artery pressures in coronary-artery disease. Anesthesiology. 1980; 53:364–370.
51. Lefcoe MS, Sibbald WJ, Holliday RL. Wedged balloon catheter angiography in the critical care unit. Crit Care Med. 1979; 10:449–453.

52. Fischer AP, Benis AM, Jurado RA, et al. Analysis of errors in measurement of cardiac output by simultaneous dye and thermal dilution in cardiothoracic surgical patients. Cardiovasc Res. 1978; 12:190–199.
53. Synder JV, Powner DJ. The effects of mechanical ventilation in the measurement of cardiac output by thermodilution. Crit Care Med. 1982; 10:677–682.
54. Swan HJC. Balloon flotation catheters: their use in hemodynamic monitoring in clinical practice. JAMA. 1975; 233:865–867.
55. Shaver JA. Hemodynamic monitoring in the critically ill patient. New Engl J Med. 1983; 308:277–279.
56. Boyd KD, Thomas SJ, Gold J, et al. A prospective study of complications of pulmonary artery catheterizations in 500 consecutive patients. Chest. 1983; 84:245–249.
57. Rao TLK, Gorski DW, Laughlin S, et al. Safety of pulmonary artery catheterization. Anesthesiology 1982; 57:A116.
58. Sprung CL, Pozen RG, Rozanski JJ, et al. Advanced ventricular arrhythmias during bedside pulmonary artery catheterization. Am J Med. 1982; 72:203–208.
59. Elliott CG, Zimmerman GA, Clemmer TP. Complications of pulmonary artery catheterization in the care of critically ill patients. Chest. 1979; 76:647–652.
60. Sprung CL. Complications of pulmonary artery catheterization. In: Sprung CL, ed. The pulmonary artery catheter. Methodology and clinical applications. Baltimore: University Park, 1983: p. 73–101.
61. Kronenberg RS, Drage CW. Attenuation of the ventilatory and heart rate response to hypoxia and hypercapnia with aging in normal men. J Clin Invest. 1973; 52:1812–1819.
62. Douglas ME, Downs JB. Applied physiology and respiratory care. In: Shoemaker WC, Thompson WL, eds. Critical care state of the art (Vol 3). Fullerton, CA: Society of Critical Care Medicine, 1982; IIIE1–35.
63. Comroe JH, Botelho S. The unreliability of cyanosis in the recognition of arterial anoxemia. Am J Med Sci. 1947; 214:1–6.
64. Mithoefer JC, Bossman OG, Thibeault OW, et al. The clinical estimation of alveolar ventilation. Am Rev Respir Dis. 1968; 98:868–871.
65. Sahn SA, Lakshminarayan S. Bedside criteria for discontinuation of mechanical ventilation. Chest. 1973; 63:1002–1005.
66. Bone RC. Monitoring ventilatory mechanics in acute respiratory failure. Respir Care. 1983; 28:597–604.
67. Espinel CH, Gregory AW. Differential diagnosis of acute renal failure. Clin Nephrol. 1980; 13:73–77.

11 Postoperative Venous Thromboembolic Disease: Natural History, Risk Factors, and Prophylaxis

Daniel L. Clarke-Pearson

Introduction

Deep venous thrombosis and pulmonary embolism, while largely preventable, are significant complications occurring in postoperative patients. It is estimated that between 150,000 and 200,000 patients die annually in the United States secondary to pulmonary embolism. The magnitude of this problem is relevant to the gynecologist, because 40% of all deaths following gynecologic surgery are directly attributed to pulmonary emboli.[1] Pulmonary embolism is also the second leading cause of death in women who undergo a legally induced abortion.[2] In a higher risk group of patients with uterine or cervical carcinoma, pulmonary embolism is the leading cause of postoperative death.[3,4] The morbidity from nonfatal pulmonary embolism and deep venous thrombosis also consumes important health care dollars through intensive in-hospital treatment as well as the long-term economic loss and suffering of patients who are unable to sustain gainful employment secondary to chronic postthrombotic venous stasis changes.[5]

The prevention of fatal pulmonary embolism is directly tied to the prevention of deep venous thrombosis in the lower extremities and pelvis. It is estimated that more than 90% of pulmonary emboli arise from the venous system distal to the inferior vena cava. With the advent of accurate and sensitive noninvasive techniques, primarily ^{125}iodine fibrinogen counting, a number of prophylactic methods have been evaluated for their effectiveness in preventing lower extremity deep venous thrombosis. Over the past decade, a wide range of prophylactic methods have been developed for postoperative thromboembolism prophylaxis. These methods, however, are not equally effective, and many carry risks of complications and expense that must be considered before selecting an appropriate mode for an individual patient. In order to intelligently select a prophylactic modality, the risks of thromboembolism for an individual patient must be considered.

Risk Factors

The causal factors of venous thrombosis were first proposed by Virchow in 1858.[6] The three factors are hypercoagulable state, venous stasis, and vessel intima injury. These are the key events or risk factors that must be reduced or eliminated in order to prevent deep venous thrombosis and pulmonary emboli. Translation of these three general etiologies to clinically significant risk factors has been further elucidated by the use of sensitive, relatively noninvasive tests to detect deep venous thrombosis.

Of tests available today, ^{125}iodine fibrinogen counting of the leg has been used most widely to detect occult thrombi and thereby screen large patient populations for deep venous thrombosis. ^{125}Iodine fibrinogen leg counting is based on the incorporation of fibrinogen, labeled with ^{125}iodine, into an acutely developing venous thrombus. The detection of a thrombus that has incorporated ^{125}iodine-labeled fibrinogen is accomplished by counting

over the venous distribution of the leg with a scintillation counter. The diagnosis is made at the patient's bedside using a portable counter, and is amenable to daily counting for prospective surveillance. ^{125}Iodine fibrinogen counting is extremely sensitive in the detection of small thrombi forming in the calf veins, but has diminished accuracy above the midthigh due to increased background activity from adjacent vessels and the bladder. In addition, this test may be falsely positive in the region of a fresh surgical wound or hematoma that may also have incorporated ^{125}iodine-labeled fibrinogen. For purposes of postoperative surveillance, ^{125}iodine fibrinogen counting has become the diagnostic method of choice because of its sensitivity and ease of application to most surgical patients.[7] It is important to emphasize that calf vein thrombosis is considered by most investigators to represent the primary source of significant deep venous thrombosis and subsequent pulmonary emboli. When the thrombosis propagates from the calf to the proximal popliteal or femoral veins, a very high incidence of pulmonary emboli is found. The logic to the detection of calf vein thrombosis by ^{125}iodine fibrinogen counting when a prophylactic method is evaluated is based on the assumption that if small thrombi in the calf may be prevented by a specific prophylactic method, a subsequent reduction in the incidence of fatal pulmonary emboli will follow.

In assessing venous thrombosis risk factors in general surgical patients Nicholaides and Irwing,[8] Gallus,[9] and Kakkar et al.[10] found clinical risk factors that were significantly associated with the development of ^{125}iodine fibrinogen-detected leg thrombi. For the general surgical patient, age and the extent of the surgical procedure were considered to be the two most significant factors associated with postoperative deep venous thrombosis. Other modifying risk factors include a past history of deep venous thrombosis or pulmonary embolism, varicose veins, concurrent malignancy, obesity, use of synthetic estrogens (oral contraceptives), inactivity, female sex, black patients, congestive heart failure, trauma, and duration of the major surgical procedure. Siegel et al.[11] assessed the risk of developing pulmonary emboli in 6,527 hospitalized patients. With logistic regression analysis techniques, they found congestive heart failure, black race, inactivity, and female sex were significantly associated with clinically significant pulmonary embolism.

When the patient undergoing gynecologic surgery is specifically considered, two prospective studies have evaluated risk factors associated with the postoperative occurrence of ^{125}iodine fibrinogen-detected deep venous thrombosis.[12,13] Clayton et al.[12] studied the risk factors of 124 patients undergoing vaginal and abdominal surgery for gynecologic disease. Most of these patients had benign gynecologic conditions. Utilizing logistic regression analysis, the five factors identified to be associated with postoperative deep venous thrombosis included age, varicose veins, percentage overweight, euglobulin lysis time, and serum fibrin-related antigen. A prognostic index score, which was created on the basis of on these five variables, was applied in a subsequent study to select high-risk patients who might most benefit from intensive perioperative thromboembolism prophylaxis.[14] The results of that study confirmed the usefulness of these criteria for patient selection in the application of prophylactic methods.

The risk factors associated with venous thromboembolic complications have also been assessed in the gynecology service at Duke University Medical Center in 411 patients undergoing major abdominal and pelvic surgery.[13] Of these patients, 84% had gynecologic malignancies or complications secondary to their treatment for malignancy. Preoperative risk factors identified in this study include age, nonwhite patients, increasing stage of malignancy, past history of deep venous thrombosis, lower extremity edema or venous stasis changes, varicose veins, weight, and a past history of radiation therapy. When considered individually, these preoperative factors were all significantly associated with postoperative deep venous thrombosis. Intraoperative factors associated with postoperative deep venous thrombosis included increased anesthesia time, increased blood loss, and transfusion requirements in the operating room. When these factors are considered in a stepwise logistic regression model, the type of surgical procedure, age, edema, nonwhite patients, varicose veins, prior radiation therapy, a past history of deep venous thrombosis, and duration of surgery

were found to be the most important variables associated with postoperative thromboembolic complications. The recognition of these factors, which are associated with postoperative venous thromboembolism, should allow the clinican to stratify patients into low-risk, medium-risk, and high-risk groups and thereby apply appropriate prophylactic methods while at the same time not exposing a low-risk group of patients to the potential complications of some prophylactic treatment regimens.

Natural History of Postoperative Venous Thromboembolism

Prior to considering venous thromboembolism prophylaxis, an understanding of the natural history of postoperative deep venous thrombosis and pulmonary embolism is necessary since the time sequence and site of deep venous thrombosis is relevant to the most effective use of prophylactic methods. The advent of noninvasive methods to detect deep venous thrombosis, especially ^{125}iodine fibrinogen counting, has allowed a clear understanding of the natural history of postoperative deep venous thrombosis. Reports in general surgery patients have shown that most deep venous thrombosis arise in the calf veins.[10,15] These thrombi might lyse spontaneously, remain in the calf, or propagate to the proximal deep venous system of the leg. It is these more proximal thrombi that are the major source of pulmonary emboli and cause long-term sequelae and morbidity of the post thrombotic stasis syndrome.

The natural history of thromboembolic complications following gynecologic surgery have some similarities and some differences when compared with the general surgery patient. Three-hundred-eighty-two gynecologic surgery patients were studied in a prospective fashion using ^{125}iodine fibrinogen leg counting throughout their postoperative hospitalization.[16] This group of patients had undergone major abdominal and pelvic surgery and included a high proportion of patients with gynecologic malignancies. Seventeen percent of patients developed postoperative venous thromboembolic complications; 85% of the thrombi were located in the veins of the calf, and most were of little clinical significance. Nearly one-third of these calf thrombi lysed spontaneously, 4% propagated to the proximal leg veins, and 4% became symptomatic pulmonary emboli. The remaining 65% of thrombi in the calf did not propagate beyond the calf throughout postoperative surveillance, and long-term follow-up showed there were no clinically significant complications of these thrombi. These findings are similar to those reported from general surgery patients and further emphasize that calf vein thrombosis, while a frequent event, is of minimal clinical significance. The 4% incidence of pulmonary emboli originating from calf veins is similar to reports by Doouss[17] and Moser and DeMoine[18] and conflict with the report of Kakkar et al.[10] who found an incidence of 10% of pulmonary emboli associated with calf-vein thrombosis.

The 4% of gynecologic patients who developed deep venous thrombosis proximal to the calf are a much more significant group of patients in that these thrombi represent potentially life-threatening pulmonary emboli. In our experience, these proximal thrombi often arose in conjunction with calf-vein thrombosis, but in approximately half the patients no calf-vein thrombosis was found. It is also important to emphasize that 40% of gynecology patients who developed symptomatic pulmonary emboli postoperatively had no evidence of deep venous thrombosis on noninvasive ^{125}iodine fibrinogen leg counting. This finding suggests that thrombi and subsequent pulmonary emboli arising from pelvic veins is a much more frequent event as compared with the general surgery patient. Unfortunately, a satisfactory test for the detection of pelvic vein thrombosis has not been established, although preliminary work with ^{111}indium-labeled platelets, which are incorporated into acutely forming thrombi and emboli, may be a reasonably noninvasive technique to image pelvic veins.[19,20]

The time of occurrence of venous thromboembolic events is important to recognize so that effective prophylaxis may be applied during the interval the patient is at greatest risk to develop deep venous thrombosis or pulmonary emboli. Of all venous thromboembolic complications, 50% occur within the first 24 hr

postoperatively and 75% are detected by the third postoperative day.[16] It seems then that the surgical procedure itself including venous stasis in the legs following induction of general anesthesia as well as the release of tissue thromboplastins secondary to surgical trauma lead to the immediate formation of occult postoperative thrombi. The early postoperative occurrence of most thromboembolic events therefore requires the application of prophylaxis in the operating room and throughout the perioperative period to achieve maximal reduction in thromboembolic complications. The later occurrence of thromboembolic complications, however, must not be forgotten as 15% of venous thromboembolic complications following gynecologic surgery were diagnosed after the seventh postoperative day.[16] The patient who remains at risk for thromboembolic complications because of immobility, prolonged postoperative recovery, or possibly a hypercoagulable state secondary to advanced malignancy should continue to receive prophylaxis beyond the conventional perioperative recommendations.

Prevention of Postoperative Venous Thromboemboli

Awareness of the risk factors associated with postoperative venous thromboembolic complications as well as the natural history of these complications allow us to develop strategies for the prevention of postoperative thromboembolic complications. Preoperative evaluation must include a careful history and physical examination in order to detect those variables known to be associated with subsequent thromboembolic complications. Patients using oral contraceptives should discontinue these medications at least 6 weeks before elective major abdominal and pelvic surgery. Patients who have diminished capabilities to ambulate should have aggressive physical therapy to increase their activity prior to undergoing elective surgery. Identification of high-risk factors should allow for perioperative use of prophylactic methods to be discussed subsequently. In addition, and occasional patient who is considered to be at low-risk preoperatively may develop unforeseen high-risk factors in the operating room that should lead to the use of appropriate prophylactic measures postoperatively.

Over the past decade a number of prophylactic methods have undergone clinical trials showing significant reduction in the incidence of deep venous thrombosis, and a few studies have been completed which have demonstrated a reduction in fatal pulmonary emboli. Of the methods reported, each has its advantages and disadvantages that will be discussed shortly. The ideal prophylactic method would be effective, free of significant side effects, well accepted by the patient and nursing staff, and widely applicable to most patient groups. In addition, in these days of cost consciousness, the ideal method would be inexpensive.

All methods of venous thrombosis prophylaxis attack one or more aspect of Virhow's triad and are primarily directed at reducing hypercoaguability and/or venous stasis. Specific prophylactic techniques may be divided into pharmacologic methods and those relying on mechanical reduction of venous stasis. In the former group, low-dose heparin and intravenous dextran have received the broadest evaluation in general surgery and gynecology. External pneumatic calf compression, a newer technique, has received less evaluation in general surgery and has had limited evaluation in gynecology. The general discussion of these methods is primarily based on experiences in general surgical populations, which in many ways may be extrapolated to gynecologic surgery patients. Where applicable, studies specifically addressing prophylaxis in gynecology will be cited and discussed.

Pharmacologic Methods

Low-Dose Heparin. The use of small doses of subcutaneously administered heparin for the prevention of deep venous thrombosis and pulmonary embolism is the most widely studied of all prophylactic methods. Over 25 controlled trials have demonstrated that heparin given subcutaneously 2 hr preoperatively and every 8–12 hr postoperatively is effective in reducing the incidence of ^{125}iodine fibrinogen scan-detected thrombi.[21] The value of low-dose heparin in preventing fatal pulmonary

emboli was assumed from the knowledge that most pulmonary emboli arose in the legs as thrombi. This was established by the randomized, controlled, multicenter international trial reported in 1975, which demonstrated a reduction in fatal postoperative pulmonary emboli in general surgery patients receiving low-dose heparin every 8 hr postoperatively.[22]

The primary mode of action of low-dose heparin is thought to be the inhibition of activated Factor X (Xa) via the action of antithrombin III. To be effective, the low-dose heparin regimen requires that circulating levels of heparin be present prior to the initiation of surgery and continue throughout the immediate postoperative period while the patient is at highest risk to develop deep venous thrombosis. The recommended routine is to give 5,000 units of heparin subcutaneously 2 hr preoperatively and then every 8–12 hr postoperatively for approximately 7 days or until the patient is fully ambulatory. Both the 8- and 12-hr dosing schedules appear to be effective in reducing deep venous thrombosis. However, the 8-hr regimen is associated with increased bleeding complications; thus, more recent studies have evaluated only the 12-hr regimen.[23]

Trials of low-dose heparin in gynecologic surgery patients are limited and a clear consensus as to the value of low-dose heparin in all groups of patients has not been established, due to differences in patient selection and length of follow-up. There are three controlled randomized trials of low-dose heparin used in gynecology in the English medical literature to date and only one North American study.[24,26] All studies used the same regimen of low-dose heparin administration: 5,000 units subcutaneously 2 hr preoperatively and every 12 hr for 7 days postoperatively. ^{125}Iodine fibrinogen leg counting for the detection of thrombosis was used for final diagnosis in all studies. The trials reported by Ballard et al.[24] and Taberner et al.[25] were conducted in patients with benign gynecologic conditions [98%]. All patients were over 40 years of age and follow-up was discontinued at the time of discharge from hospital. The American study by Clarke-Pearson and associates[26] evaluated a larger group of patients on a gynecologic oncology unit. Only 16% had benign gynecologic conditions, and follow-up included the first 6 weeks postoperatively.

The results of the trial by Taberner et al.[25] of 97 patients showed a 23% incidence of deep venous thrombosis in the control group, as compared with a 6% incidence of deep venous thrombosis in the low-dose heparin-treated patients. This difference was statistically significant ($p < .05$). Unfortunately, although this was a randomized trial, the control group contained a larger number of patients with malignancy. When the cancer patients were excluded from the trial analysis, there remained no significant value to the use of low-dose heparin in patients with benign conditions. In the second study, also from England, Ballard and associates[24] evaluated a group of 110 patients who also had a predominance of benign gynecologic diseases. The nontreated control group had a 29% incidence of deep venous thrombosis as compared with a 3.6% incidence in the low-dose heparin-treated group ($p < .001$). In this trial, none of the patients developed deep venous thrombosis proximal to the calf and none developed a pulmonary embolus.

In contrast is the study conducted by Clarke-Pearson et al.[26] at Duke University Medical Center. In this randomized trial of 185 patients undergoing major abdominal and pelvic surgery on the gynecologic oncology service, there was no difference in the incidence of thromboembolic complications between the control group [12.4%] and the low-dose heparin-treated group [14.8%]. When analysis of thromboembolic complications was confined to the first 7 postoperative days, the incidence of deep venous thrombosis was 12.4% for the control group compared with 6.8% in the low-dose heparin-treated group. This difference was not statistically significant [$p = 0.2$], but it does suggest that low-dose heparin may be effective while the patient is receiving heparin prophylaxis but that the beneficial effect is lost when the drug is discontinued. This may also explain the beneficial results of low-dose heparin noted by Taberner[25] and Ballard et al[24] in that venous thromboembolic complications were recorded only until hospital discharge. Patients with malignancy or decreased ambulatory capacity due to age or extent of surgery

may remain at risk longer and may require a longer duration of low-dose heparin prophylaxis in order to truly benefit from this regimen. In summary, with regard to gynecologic surgery, only the trial reported by Ballard et al.[24] has found a beneficial effect of low-dose heparin in patients with benign gynecologic conditions. Taberner et al., in benign gynecology patients, and Clarke-Pearson et al.,[26] in gynecologic oncology patients, did not find low-dose heparin to be of benefit. Other groups of surgical subpopulations have also found low-dose heparin to be of no benefit, especially in total hip replacement,[27] open prostatectomy,[28] cystectomy,[28] and inguinal lymphadenectomy patients.[29]

Attempts to improve the prophylactic results of low-dose heparin by utilizing adjusted doses based on minimal prolongation of the activated partial thromboplastin time or the addition of other drugs used in combination with heparin have also been reported.[30,31] Of particular note are studies in which dihydroergotamine has been administered concurrently with low-dose heparin.[32–34] Dihydroergotamine, a vasoconstrictor, appears to have a selective effect on capacitance vessels (veins) with minimal effect on resistance vessels (arteries), thereby reducing venous stasis. A multicenter trial in the United States of 744 general surgery patients found that 0.5 mg of dihydroergotamine mesylate given with 5,000 units of heparin subcutaneously 2 hr preoperatively and every 12 hr postoperatively for 5–7 days significantly reduced the incidence of deep venous thrombosis detected by ^{125}iodine fibrinogen leg counting.[34] Curiously, low-dose heparin when given alone did not significantly reduce the occurrence of deep venous thrombosis. Adolf et al.[32] have reported beneficial results of dihydroergotamine plus low-dose heparin in gynecologic surgery patients in a study reported in the German literature.

Although the most widely studied prophylactic method, low-dose heparin has not received widespread use from general surgeons or gynecologists. The primary reasons for resistance to use are either a lack of evidence that low-dose heparin is effective or the feeling that bleeding complications outstrip the anticipated benefits in the prevention of pulmonary emboli and deep venous thrombosis.[35,36] Although low-dose heparin is considered to have no effect on measurable coagulation parameters, most large series have noted an increase in the bleeding complication rate, especially a higher incidence of wound hematoma.[22,23,37] Up to 10%–15% of otherwise healthy patients have their activated partial thromboplastin time (APTT) prolonged greater than 1.5 times control value 2–4 hr after 5,000 units of heparin is given subcutaneously.[38] These transiently anticoagulated patients have also been noted in one carefully monitored trial of low-dose heparin in gynecology.[37] It was these patients in which the major bleeding complications were encounted postoperatively. Dockerty et al.[39] also found that estimated blood loss increased from 246 to 401 ml in low-dose heparin-treated patients undergoing abdominal hysterectomy. In patients undergoing inguinal or pelvic lymphadenectomy, retrospective studies have suggested that low-dose heparin contributed to an increased occurrence of lymphocysts.[40,41] These clinical reports were confirmed in a prospective study that demonstrated a two-fold increase in retroperitoneal lymph drainage in patients treated with low-dose heparin.[37] Finally, although relatively rare, thrombocytopenia is associated with low-dose heparin use and has been found in 6% of patients after gynecologic surgery.[37] Although many authors feel that no monitoring of coagulation parameters is necessary for effective and safe low-dose heparin use, periodic postoperative assessment of activated thromboplastin time and platelet count seems prudent to maximize the identification of the 22% of patients who either had prolonged APTT or thrombocytopenia and who are most prone to develop major clinical hemorrhagic complications.

Errors of administration are also frequently encountered with low-dose heparin use. Properly timed administration may be a concern on some busy surgical units. Further, the proper dose may be in error if heparin from multidose vials is used. Gallus et al.,[23] in a study of 820 general surgery patients, found that in nearly one of five patients the heparin was either stopped because of complications (8.6%) or it was not given as prescribed (11.2%). This problem may be partially avoided by using heparin supplied in unit dose vials or prefilled

syringes. Dihydroergotamine should be avoided in patients with peripherial vascular disease, angina, coronary insufficiency, severe hypertension, severely traumatized patients, or patients with debilitating conditions such as sepsis, because they appear to be most prone to vasospastic reactions. Further, dihydroergotamine should be discontinued in the presence of severe sepsis, shock, myocardial infarction, angina, and hypotension.[34]

Dextran. Dextran of molecular weight 70,000 or 40,000 has been found to be effective in the prevention of venous thromboembolism in general surgery and gynecologic surgery patients in many trials reported to date.[42–48] Both Dextran preparations appear to be equally effective in preventing postoperative deep venous thrombosis. The antithrombotic properties of dextran are uncertain, but may be related to an indirect decrease in platelet adhesiveness, a decrease in blood viscosity, and indirectly to an increased venous flow rate. Controlled clinical trials of patients undergoing general surgical procedures have shown that dextran is effective in preventing deep venous thrombosis and fatal pulmonary emboli to a magnitude similar to that achieved with low-dose heparin therapy. Several studies have been performed evaluating the effectiveness of dextran therapy in patients undergoing gynecologic surgical procedures. Bonnar et al.[44] demonstrated that Dextran 70 reduced the incidence of ^{125}iodine fibrinogen-detected deep venous thrombosis in patients undergoing surgery for benign gynecologic conditions as well as for malignancy. In studies comparing low-dose heparin with intravenous dextran, results are more variable. McCarthy et al.[45] and Hohl et al.[46] showed no significant difference between a group of patients treated with low-dose heparin and those treated with Dextran 70. On the other hand, Borow and Goldson[47] found a significant decrease in the incidence of deep venous thrombosis in patients treated with intravenous dextran as compared to a group treated with low-dose heparin.

Dextran administration regimens vary considerably from one study to another. In general, 500–1000 ml of Dextran 70 are infused during surgery, and subsequent 500 ml infusions are given immediately postoperatively and a third unit on the first postoperative day. Some regimens have continued to give Dextran every other day postoperatively. The best regimen for Dextran administration in moderate- and high-risk groups of patients has not been clearly identified. Complications associated with Dextran therapy include the potential for fluid overload in patients with limited cardiovascular and renal reserve, and (rarely) an anaphylactic reaction has been noted. Bleeding complications associated with Dextran therapy are considered to be significantly less frequent than those associated with the use of low-dose heparin.[48]

Antiplatelet Agents. Platelet aggregation is generally considered to be an important event in arterial thrombosis but less important in the formation of deep venous thrombi. Fibrin formation and deposition is usually the initiating event in venous thrombosis, and the presence of platelet aggregates at the site of origin of venous thrombi demonstrates a less significant role of platelets in the initiation of venous thrombosis. The potential prophylactic benefit of antiplatelet drugs has been evaluated alone and in combination clinical trials.[49] Results of trials evaluating acetylsalicylic acid (aspirin), dipyridamole (persantine), hydroxychloraquine (plaquenil), and sulfinpyrazone have been conflicting relative to the reported efficacy in preventing deep venous thrombosis. Aspirin has received the widest evaluation and appears to be effective in male patients undergoing orthopedic surgical procedures.[50] At the present time, however, there are few data to support the widespread use of antiplatelet drugs for the prevention of postoperative venous thromboembolic complications in either general surgical patients or in patients undergoing surgery for gynecologic diseases.

Warfarin. The use of sodium warfarin to prevent a postoperative thromboembolic complication relies on theraputic anticoagulation to achieve effective results. Controlled trials utilizing accurate diagnostic methods to document venous thromboembolic events demonstrated that warfarin therapy is extremely effective in reducing the frequency of venous thromboembolism as well as fatal pulmonary emboli.[51,52] To achieve these results, however,

warfarin must prolong the prothrombin time to 1.5–2 times the control value. As might be expected, intraoperative and postoperative bleeding complications are greater with this method of prophylaxis as compared with those of low-dose heparin or intravenous dextran therapy.

A single trial comparing warfarin therapy with low-dose heparin in gynecologic surgery showed both to be beneficial when compared to a control group.[25] However, both the warfarin and the low-dose heparin treatment groups had approximately the same incidence of deep venous thrombosis. Taberner et al.[25] concluded that in moderate-risk patients, such as those undergoing surgery for benign gynecologic conditions, low-dose heparin appears to be a more convenient mode of therapy, as it does not require 5 days of preoperative adjustment of prothrombin time or continued laboratory monitoring of the prothombin time. Warfarin, while an effective method for prevention of postoperative deep venous thrombosis and pulmonary embolism, should be reserved for the very-high-risk patient. When the risks of postoperative pulmonary embolus exceed the higher bleeding complication rate, such as in patients undergoing a total hip replacement, this may be the regimen of choice. Some patients with gynecologic malignancies or those patients who have demonstrated repeated bouts of deep venous thrombosis may also be suitable candidates for this more intensive therapy.

Mechanical Methods

Stasis in the veins of the legs has been clearly demonstrated on the operating table, and continues postoperatively for varying lengths of time. Many authors feel that the combination of stasis occurring in the capacitance veins of the calf during surgery, plus the hypercoagulable state induced by surgery, are the prime factors contributing to the development of acute postoperative deep venous thrombosis. Prospective studies of the natural history of postoperative venous thrombosis have shown that the calf veins are the predominant site of thrombi and that most thrombi develop within 24 hr of surgery. Reduction of venous stasis in the perioperative period by various methods has been less extensively investigated than pharmacologic methods such as low-dose heparin. However, a growing body of literature supports the important role that these mechanical prophylactic methods may play in the prevention of postoperative deep vein thrombosis.

Although probably of only modest benefit, reduction of stasis by short preoperative hospital stays and early postoperative ambulation should be encouraged for all patients. Elevation of the foot of the bed 20°, thus raising the calf above heart level, allows gravity to drain the calf veins and should further reduce stasis. More active forms of mechanical prophylaxis include electrical calf-muscle stimulation, intraoperative foot-pedaling devices, postoperative physical therapy, elastic stockings, and external pneumatic leg compression. The two most widely studied methods are elastic stockings and external pneumatic compression.

Elastic Stockings. In a survey of general surgeons in the United States, elastic stockings used alone were second only to low-dose heparin as the prophylactic method of choice in high-risk and moderate-high-risk surgical patients.[35] The simplicity of elastic stockings and the absence of significant side effects are probably the two most important reasons that they are included in the routine postoperative orders of many surgeons. The literature supporting this widespread practice, however, is limited and controversial. Early studies of static uniform compression stockings demonstrated no benefit from this style of stocking.[53,54] Further evaluation of venous flow dynamics demonstrated better venous emptying and increased venous flow from stockings that had a gradient of pressure higher at the ankle and diminishing in the thigh.[55,56] Controlled studies of gradient elastic stockings are limited but do suggest modest benefit when carefully fitted.[57–59] Insuring a proper fit of the stockings is a major stumbling block, and may make the stockings hazardous to some patients who develop a tourniquet effect at the knee or midthigh by poorly fitted stockings. Variations in human anatomy do not allow perfect fit of all patients to stocking sizes manufactured. As an example of this problem, a retrospective study of 281 patients undergoing radical hysterectomy or total abdominal hysterectomy found a

four-fold increase in the incidence of postoperative deep venous thrombosis and pulmonary embolism in patients weighing over 90 kg who wore elastic stockings perioperatively.[3] It was suggested that a tourniquet effect of these stockings may have lead to increased stasis in this group of patients already at high risk due to obesity, age, malignancy, and major surgery.

External Pneumatic Compression. The largest body of literature dealing with the reduction of postoperative venous stasis deals with intermittent external compression of the leg by pneumatically inflated sleeves placed around the calf and/or leg during intraoperative and postoperative periods.[28,47,60–69] Various pneumatic compression devices and leg sleeve designs are available, but the current literature has not demonstrated superiority of one system over another. The single-chambered calf compression device has been studied the most extensively, and appears to significantly reduce the incidence of deep venous thrombosis on a par with that of low-dose heparin. In addition to increasing venous flow and pulsatile emptying of the calf veins, external pneumatic compression also appears to augment endogenous fibrinolysis.[70,71] Activation of the fibrinolytic system should allow for lysis of very early thrombi before they become clinically significant.

The duration of postoperative external pneumatic compression has been different in various trials. Our understanding that the onset of most deep venous thrombosis occurs intraoperatively and in the first 48 hr postoperatively suggests that this time interval should be a minimum length for external pneumatic compression. Several investigators have found external pneumatic compression to be effective when used only in the operating room or in the operating room and for the first 24 hr postoperatively.[60,65,66] Salzman and associates[65] reported that external pneumatic compression was as beneficial when used only intraoperatively as when applied for a longer period of time in patients after urologic surgical procedures. On the other hand, Turpie et al.[63,64] found continuing benefit in the use of external pneumatic compression for up to 14 days postoperatively.

External pneumatic compression used in patients undergoing major surgery for gynecologic malignancy has been found to reduce the incidence of postoperative venous thromboembolic complications by nearly threefold.[67] Calf compression was applied intraoperatively and for the first 5 postoperative days. In a subsequent trial of similar patients designed to evaluate whether external pneumatic compression might achieve similar benefits when used only intraoperatively and for the first 24 hr postoperatively, there is no reduction of deep venous thrombosis compared with the control group.[68] It appears that patients with gynecologic malignancies remain at risk because of stasis and hypercoagulable states for a longer period of time than the general surgical or urology patients, and if compression is to be effective, it must be used for at least 5 days postoperatively.

Two studies have directly compared external pneumatic compression and low-dose heparin in gynecology.[47,69] Jobson et al.[69] reported external pneumatic compression to be superior to low-dose heparin in a randomized trial of 330 patients undergoing major gynecologic surgical procedures. In this uncontrolled randomized trial, the clinical end point was the diagnosis of symptomatic pulmonary emboli. Of 164 patients in the low-dose heparin group, 7 developed postoperative pulmonary emboli, including two that were fatal, but none of the 139 patients with external pneumatic calf compression prophylaxis developed a pulmonary embolis ($p = .006$). This large trial would have been more significant had a prospective method been used to screen all patients for postoperative deep venous thrombosis or pulmonary emboli. Without such prospective screening, one might argue that bias in the clinical detection of pulmonary emboli may have occurred. Borow and Goldson,[47] in a smaller group of gynecologic surgery patients, also found the incidence of ^{125}iodine fibrinogen counting detected thrombi to be lower in an external pneumatic compression treatment group (7.6%) as compared with a low-dose heparin-treated group (9.1%) and in the untreated control group (16.6%). There was no statistically significant difference in the incidence of deep venous thrombosis between the external pneumatic calf compression and low-dose heparin-treated groups. External pneumatic compression has also been found to be

beneficial in other groups of patients who have not benefited from low-dose heparin prophylaxis, including patients after suprapubic prostectomy,[28] cystectomy,[28] and total hip operations.[62]

External pneumatic leg compression has no significant side effects or risks, although patient tolerance has been cited as a drawback to the use of this equipment. However, we have had only 2 patients of nearly 200 treated with external pneumatic compression request removal because of discomfort. The equipment is easily managed by the nursing staff, and while initial capital outlay for external pneumatic compressors may seem large, Salzman and Davis[72] calculated that the cost per patient of this prophylactic method is slightly less than that of low-dose heparin given for 7 days postoperatively.

Inferior Vena Cava Interruption. Interruption of the vena cava, while not preventing deep venous thrombosis, is effective in preventing fatal pulmonary emboli. Although most often indicated for patients with recurrent pulmonary emboli resistant to conventional anticoagulant therapy, a retrospective analysis of high-risk patients with hip fractures found that placement of an inferior vena cava filter lowered the clinical incidence of pulmonary embolism and the incidence of fatal pulmonary embolism.[73] A vena cava filter or umbrella appears more attractive when compared to cava ligation, plication, or clipping, because of relative ease of application and the lower incidence of late sequelae. This invasive prophylactic technique should be reserved only for very-high-risk patients.

Surveillance

Under selected circumstances, it may be deemed that prophylaxis is inappropriate, even though a patient is considered to be at high risk to develop a venous thromboembolic complication. This may be due to evidence that suggests that any prophylaxis is ineffective under these specific circumstances, that the risk of bleeding cannot be tolerated, or that a specific prophylactic method is not available in a particular institution. Although deep venous thrombosis might not be prevented, recognition (diagnosis) and treatment of occult thrombi may prevent more ominous sequelae of pulmonary embolism. Early detection of symptomatic deep venous thrombosis in the leg is most appropriately performed by ^{125}iodine fibrinogen leg counting initiated immediately after surgery and continued throughout the postoperative period while the patient remains at risk. Surveillance with ^{125}iodine fibrinogen counting and the prevention of fatal pulmonary embolism is supported by nine studies in which 1,373 patients were screened with ^{125}iodine fibrinogen counting. Of these patients, none suffered a fatal pulmonary embolus.[72]

Other noninvasive diagnostic techniques such as impedance plethysmography, phleborheography, or Doppler scanning are relatively insensitive as screening methods and do not usually detect small thrombi in the calf or nonocculsive thrombi in the femoral-popliteal segments. Finally, in extremely high-risk settings, such as in total hip replacement patients, the incidence of deep venous thrombosis is so frequent that some investigators routinely perform venography prior to hospital discharge to be certain the patient does not have an occult deep venous thrombosis.

Surveillance of the patient at risk for pelvic venous thrombosis is a more difficult problem. From prospective studies of gynecologic patients, it appears that approximately 40% of pulmonary emboli must arise from the veins in the pelvis.[16] These vessels are not accessible to ^{125}iodine fibrinogen counting or any other widely available noninvasive diagnostic method. Preliminary studies imaging ^{111}indium-labeled platelets injected intravenously suggests that occult pelvic vein thrombi may be diagnosed by this method.[19,20] Figure 11-1 demonstrates the diagnosis of occult pelvic vein thrombosis by ^{111}indium-labeled platelet imaging. The full potential of ^{111}indium-labeled platelet imaging as a diagnostic method for the detection of pelvic vein thrombosis remains to be investigated as to its sensitivity and specificity as well as the true incidence and significance of pelvic vein thrombosis.

Strategies to Prevent Postoperative Thromboembolic Complications

The wide variety of prophylactic methods reviewed in this chapter must be used in a logical fashion in order to have maximal impact on

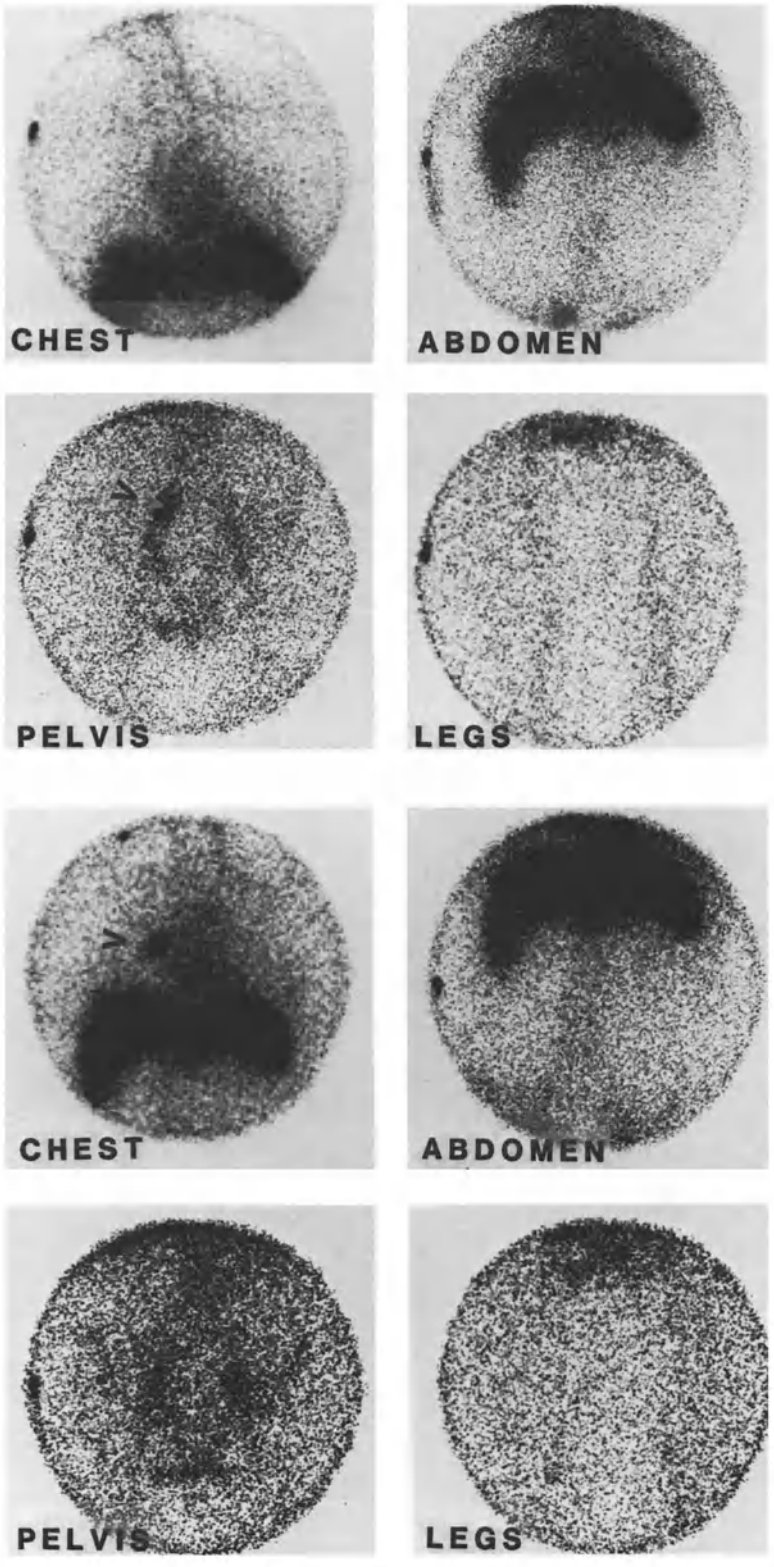

Figure 11-1. A, ^{111}Indium-labeled platelet images of the chest, abdomen, pelvis, and legs of a patient 24 hr after radical hysterectomy and pelvic lymphadenectomy. The area of increased uptake in the right side of the pelvis is a venous thrombosis. **B,** Images at 48 hr postoperatively show clearing of ^{111}indium activity in the pelvis but a focal area of indium in the right chest. A pulmonary embolus was documented by pulmonary arteriography. (Reprinted by permission of C.V. Mosby Co. from Am J Obstet Gynecol. 1984; 149:796–798.)

the prevention of postoperative deep venous thrombosis and pulmonary embolism. The application of a single method to all gynecologic patients would certainly not be cost effective, and would place some patients at excessive risks while others would be inadequately treated. Selection of prophylaxis must be based on reasonable assessment of the risk of venous thromboembolic complications in a particular patient. Those risks, discussed previously, may be divided into low, moderate, or high risks as described by Salzman and Davies,[72] Nicholaides and Irving,[8] and Gallus.[9] Taking into consideration the risk factors identified by Clayton et al.[12] and Clarke-Pearson et al.,[13] a modified set of risk factors may be proposed for gynecologic surgery patients. Table 11-1 outlines the levels of risks suggested for use in selecting prophylactic intervention. Careful review of the patient's history and physical findings relative to previous venous disease and other known risk factors is very important prior to placing a patient in the *low-risk* category. If no risk factors are identified, there is no advantage to subjecting the patient to the expense or potential discomfort or complications of any prophylactic method. Patients undergoing major surgery who are over 40 years of age make up the majority of *moderate-risk* patients in gynecologic surgery. Deep venous thrombosis and pulmonary embolism are real threats to this group of patients, and effective prophylaxis should routinely be employed. Considering the studies previously cited, which have been performed in gynecology, options of prophylactic methods include low-dose heparin, dextran, or external pneumatic compression. Of these methods, external pneumatic compression appears to be slightly more effective, has less significant side effects, and has a cost approximately equal to that of low-dose heparin and approximately one-half that of dextran regimens. If external pneumatic compression is not available, then low-dose heparin appears to be as effective as dextran with less cost and equal side effects.

Table 11-1. Risk Levels for Postoperative Deep Venous Thrombosis or Pulmonary Embolism

Low risk
Minor surgery[a]
Age <40 years and major surgery[a]
Moderate risk
Minor surgery + other risk factors
Age <40 + major surgery + other risk factors
Age ≥40 <60 + major surgery
High risk
Age ≥40 + major surgery + other risk factors
Age ≥60 + major surgery
Extremely high risk (any age)
Pelvic exenteration
Radical vulvectomy + inguinal lymphadenectomy
Major surgery + history of deep venous thrombosis or pulmonary embolus
Major surgery + severe postthrombotic stasis changes in the lower leg
Other risk factors increasing the risk of postoperative thromboembolism
Malignancy
Postthrombotic stasis changes in the leg
Ankle edema
Varicose veins
Prior pelvic radiation therapy
Black race
Obesity
Not ambulatory preoperatively
Surgery lasting longer than 300 min
Estimated blood loss >600 ml

[a] No other risk factors

Patients at *high risk* to develop postoperative venous thromboemboli should receive maximum prophylactic effort. These are usually patients who are elderly and have cancer, and other complicating risk factors. While studies of prophylaxis in this group of gynecologic patients are limited, it appears that low-dose heparin is inferior to external pneumatic compression. In this group of patients, every effort should be made to have external pneumatic compression available for perioperative and postoperative use. Continued use of external pneumatic compression for a minimum of 5 days is highly recommended. An additional strategy in management might also include surveillance with ^{125}iodine fibrinogen leg counting postoperatively. While this increases the cost of patient care, the early detection and treatment of occult deep venous thrombosis should lead to a near complete elimination of fatal pulmonary emboli.[72]

The small group of patients at *extremely high risk* includes patients undergoing pelvic exenteration or radical vulvectomy and patients with a confirmed past history of deep venous thrombosis and/or pulmonary embolism.

While external pneumatic compression, dextran, and low-dose heparin have some impact on these patients, additional measures should also be considered. The successful use of warfarin in the extremely high risk group of total hip surgery patients should be strongly considered. One approach to prophylaxis might include perioperative use of external pneumatic compression combined with ^{125}iodine fibrinogen surveillance, switching to warfarin on the third postoperative day when the risks of bleeding are reduced. Other approaches including vena cava ligation or placement of a cava filter may offer effective prophylaxis of fatal pulmonary emboli while minimizing hemorrhagic risks.

Even with the best-laid management plans preoperatively, occasionally unexpected circumstances occur in the operating room that move the patient into a higher risk category. Prolonged operating time and increased intraoperative blood loss are two factors associated with postoperative thromboemboli that are independent of other risk factors identified preoperatively. At the point where the risk increases, especially in a previous low-risk patient, prophylaxis may still be employed in the form of external pneumatic compression and/or ^{125}iodine fibrinogen counting surveillance may be instituted immediately postoperatively.

The duration of prophylaxis also warrants an additional comment. Most studies have found that the occurrence of thromboemboli is in the first few days postoperatively, and therefore intensive prophylaxis has been used preoperatively and for 5–7 days postoperatively. It appears, however, that some high-risk patients continue to be at risk for a longer period of time. Cancer patients and others with prolonged postoperative recovery, immobility, and other factors such as hypercoagulability would probably benefit from longer prophylaxis and/or surveillance. Unfortunately, the duration of prophylaxis has not been fully studied in this type of patient.

In preparing a strategy for the prevention of postoperative venous thromboembolic complications, the gynecologist must evaluate the individual patient's potential risks and select from a number of prophylactic methods currently available. Recognition of risk factors and individualized management schemes should lead to the most effective prophylaxis while minimizing side effects and costs of therapy. Many ongoing trials evaluating new and old prophylactic methods should further identify the best prophylaxis for gynecologic surgery patients.

References

1. Jeffcoate TNA, Tindall VR. Venous thrombosis and embolism in obstetrics and gynecology. Aust N Z J Obstet Gynaecol. 1965; 5:119–130.
2. Kimball AM, Hallum AV, Cates W. Deaths caused by pulmonary thromboembolism after legally induced abortion. Am J Obstet Gynecol. 1978; 132:169–174.
3. Clarke-Pearson DL, Jelovsek FR, Creasman WT. Thromboembolism complicating surgery for cervical and uterine malignancy: Incidence, risk factors, and prophylaxis. Obstet Gynecol. 1983; 61:87–94.
4. Creasman WT, Weed JC Jr. Radical hysterectomy. In: Schaefer G, Graber EA, eds. Complications in obstetrics and gynecologic surgery. Hagerstown, MD: Harper and Row, 1981: p. 389–398.
5. O'Donnell TF Jr, Browse NL, Burnaud KG, et al. The socio-economic effects of an iliofemoral venous thrombosis. J Surg Res. 1977; 22:483–488.
6. Virchow R. Die cellularpathologie in ihrer begrundung auf physiologiche und pathologiche gewebslehre. Berlin: A. Hirshwald, 1858.
7. Kakkar VV. Fibrinogen uptake test for the detection of deep venous thrombosis—A review of current practice. Semin Nucl Med. 1977; 7:229–244.
8. Nicolaides AN, Irving D. Clinical factors and the risk of deep venous thrombosis. In: Nicolaides AN, ed. Thromboembolism: etiologies, advances in prevention and management. Lancaster: Medical and Technical Publishers, 1975: p. 193–204.
9. Gallus AS. Venous thromboembolism: Incidence and clinical risk factors. In: Madden JL, Hume M, eds. Venous thromboembolism. New York: Appleton-Century-Crofts, 1976: p. 1–32.
10. Kakkar VV, Flanc C, Howe C, et al. Natural history of postoperative deep vein thrombosis. Lancet. 1969; 2:230–233.
11. Siegel B, Justin JR, Gibson RJ, et al. Risk assessment of pulmonary embolism by multivariate analysis. Arch Surg. 1979; 114:188–192.
12. Clayton JK, Anderson JA, McNicol GP. Preoperative prediction of postoperative deep vein thrombosis. Br Med J. 1976; 2:910–916.

13. Clarke-Pearson DL, DeLong E, Synan IS, et al. A prospective study of risk factors associated with postoperative venous thromboembolism in gynecology. Obstet Gynecol. 1986 (in press).
14. Crandon AJ, Peel KR, Anderson JA, et al. Prophylaxis of postoperative deep vein thrombosis: Selective use of low-dose heparin in high-risk patients. Br Med J. 1980; 2:345–347.
15. Nicolaides AN, O'Connell JD. Origin and distribution of thrombi in patients presenting with clinical venous thrombosis. In: Nicolaides AN, ed. Thromboembolism: etiology, advances in prevention and management. Baltimore: University Park, 1975: p. 167–180.
16. Clarke-Pearson DL, Synan IS, Coleman RE, et al. The natural history of postoperative venous thromboembolism in gynecologic oncology: a prospective study of 382 patients. Am J Obstet Gynecol. 1984; 148:1051–1054.
17. Doouss TW. The clinical significance of venous thrombosis of the calf. Br J Surg. 1976; 63:377–378.
18. Moser KM , DeMoine JR. Is embolic risk conditioned by location of deep venous thrombosis? Ann Intern Med. 1981; 94:439–444.
19. Clarke-Pearson DL, Coleman RE, Petry N, et al. Postoperative pelvic vein thrombosis and pulmonary embolism detected by Indium-111-labeled platelet imaging: a case report. Am J Obstet Gynecol. 1984; 149:796–798.
20. Clarke-Pearson DL, Coleman RE, Siegel R, et al. ^{111}Indium-labeled platelet imaging for the detection of deep venous thrombosis and pulmonary embolism in patients without symptoms after surgery. Surgery. 1985; 98:98–103.
21. American Heart Association Special Report: Prevention of venous thromboembolism in surgical patients by low-dose heparin. Circulation. 1977; 55:423A–426A.
22. Kakkar VV. Prevention of fatal postoperative pulmonary embolism by low dose heparin. An international multicenter trial. Lancet. 1975; 2:145–151.
23. Gallus AS, Hirsh J, O'Brien E, et al. Prevention of venous thrombosis with small, subcutaneous doses of heparin. JAMA. 1976; 235:1980–1982.
24. Ballard M, Bradley-Watson PJ, Johnstone ED, et al: Low doses of subcutaneous heparin in the prevention of deep venous thrombosis after gynecologic surgery. J Obstet Gynaecol Br Commonw, 1973; 80:469–472.
25. Taberner DA, Poller, L, Burnstein RW, et al. Oral anticoagulants controlled by British comparative thromboplastin versus low dose heparin prophylaxis of deep venous thrombosis. Br Med J. 1978; 1:272–274.
26. Clarke-Pearson DL, Coleman RE, Synan IS, et al. Venous thromboembolism prophylaxis in gynecologic oncology: A prospective controlled trial of low-dose heparin. Am J Obstet Gynecol. 1983; 145:606–613.
27. Barber HM, Galaski CBS, Feil EJ, et al. A comparative study of dextran-70, warfarin, and low-dose heparin for the prophylaxis of thromboembolism following total hip replacement. Postgrad Med J. 1977; 53:130–133.
28. Coe NP, Collins REC, Klein LA, et al. Prevention of deep vein thrombosis in urologic patients: a controlled, randomized trial of low dose heparin and external pneumatic compression boots. Surgery. 1978; 83:230–234.
29. Arbeit JM, Lowery SF, Line BR, et al. Deep venous thromboembolism in patients undergoing inguinal lymph node dissection for melanoma. Ann Surg. 1981; 194:648–655.
30. Leyvraz PF, Richard J, Bachmann F, et al: Adjusted versus fixed-dose subcutaneous heparin in the prevention of deep vein thrombosis after total hip replacement. N Engl J Med. 1983; 309:954–958.
31. Negus D, Cox SJ, Friedgood A, et al. Ultra low dose intravenous heparin in the prevention of postoperative deep vein thrombosis. Lancet. 1980; 1:891–894.
32. Adolf J, Butterman A, Weidenbath A, et al. Optimization of postoperative prophylaxis of thrombosis in gynecology. Geburtshilfe Fraunheilkd. 1978; 38:98–100.
33. Morris WT, Hardy AE. The effect of dihydroergotamine and heparin on the incidence of thromboembolic complications following total hip replacement: a randomized controlled clinical trial. Br J Surg. 1981; 68:301–304.
34. Sasahara AA. Dihydroergotamine heparin prophylaxis of postoperative deep vein thrombosis. JAMA. 1984; 251:2960–2966.,
35. Conti S, Daschbach M. Venous thromboembolism prophylaxis. A survey of its use in the United States. Arch Surg. 1982; 117:1036–1040.
36. Kotz HL, Geelhoed GW. Lethal Thromboembolism and its prevention in pelvic surgery: a review. Gynecol Oncol. 1981; 12:271–280.
37. Clarke-Pearson DL, DeLong, ER, Synan IS, et al. Complications of low-dose heparin prophylaxis in gynecologic oncology surgery. Obstet Gynecol. 1984; 64:689–694.
38. Gurewich V, Numm T, Thazhathekudyil T, et al. Hemostatic effect of uniform low-dose subcutaneous heparin in surgical patients. Arch Intern Med. 1978; 138:41–44.
39. Dockerty PW, Goodman JDS, Hill JG, et al. The effect of low-dose heparin on blood loss at ab-

dominal hysterectomy. Br J Obstet Gynaecol. 1983; 90:759–762.
40. Catalona WJ, Kadmon D, Crane DB. Effect of mini-dose heparin on lymphocele formation following extraperitoneal pelvic lymphadenectomy. J Urol. 1979; 123:890–895.
41. Piver MS, Malfetano JH, Lele SB, et al. Prophylactic anticoagulation as a possible cause of inguinal lymphocyst after radical vulvectomy and inguinal lymphadenectomy. Obstet Gynecol. 1983; 62:17–21.
42. Clagett GP, Collins GJ. Platelets, thromboembolism and other clinical utility of antiplatelet drugs. Surg Gynecol Obstet. 1978; 1476:257–272.
43. Gruber VF, Saldeen T, Brokop T, et al. Incidences of fatal postoperative pulmonary embolism after prophylaxis with dextran-70 and low dose heparin: an international multicenter trial. Br Med J. 1980; 1:83–86.
44. Bonnar J, Walsh JJ, Haddon M, et al. Coagulation system changes induced by pelvic surgery and the effect of dextran-70. Bibl Anat. 1973; 12:351–356.
45. McCarthy TG, McQueen J, Johnstone FD, et al. A comparison of low dose subcutaneous heparin and intravenous dextran-70 in the prophylaxis of deep venous thrombosis after gynecological surgery. J Obstet Gynecol Br Commonw. 1974; 81:486–491.
46. Hohl MK, Luscher KJP, Tichy J, et al. Prevention of postoperative thromboembolism by dextran-70 or low dose heparin. Obstet Gynecol. 1980; 55:497–500.
47. Borow M, Goldson H. Postoperative venous thrombosis: evaluation of five methods of treatment. Am J Surg. 1981; 141:245–251.
48. Gruber UF, Duckert F, Fridrich R, et al. Prevention of postoperative thromboembolism by Dextran-40, low doses of heparin, or xanthinol nicotinate. Lancet. 1977; 1:207–210.
49. Genton E, Gent M, Hirsh J, et al. Platelet-inhibiting doses in the prevention of clinical thrombotic disease. N Engl J Med. 1975; 293:1174–1178, 1236–1240, 1296–1300.
50. Harris WH, Salzman EW, Alhanasoulis C, et al. Aspirin prophylaxis of venous thromboembolism after total hip replacement. N Engl J Med. 1977; 297:1246–1249.
51. Clagett GP, Salzman EW. Prevention of venous thromboembolism. Prog Cardiovasc Dis. 1975; 17:345–366.
52. Belding HH. Use of anticoagulants in the prevention of venous thromboembolic disease in postoperative patients. Arch Surg. 1965; 90:566–573.
53. Browse NL, Jackson BT, Maye ME, et al. The value of mechanical methods of preventing postoperative calf vein thrombosis. Br J Surg. 1974; 61:319–323.
54. Rosengarten DS, Laird J, Jeyasingh K, et al. The failure of compression stocking (Turbigrip) to prevent deep venous thrombosis after operation. Br J Surg. 1970; 57:296–299.
55. Sigel B, Edelstein AL, Savitch L, et al. Type of compression for reducing venous stasis. Arch Surg. 1975; 110:171–175.
56. Lawrence D, Kakkar VV. Graduated, static, external compression of the lower limb: a physiological assessment. Br J Surg. 1980; 67:119–121.
57. Holford CP. Graded compression for preventing deep venous thrombosis. Br Med J. 1976; 2:969–970.
58. Lewis CE, Antoine J, Mueller C, et al. Elastic compression in the prevention of venous stasis; a critical re-evaluation. Am J Surg. 1976; 132:739–743.
59. Scurr JH, Ibrahim SZ, Faber RG, et al. The efficacy of graduated compression stocking in the prevention of deep vein thrombosis. Br J Surg. 1977; 64:371–373.
60. Roberts VC, Cotton LT. Prevention of postoperative deep vein thrombosis in patients with malignant disease. Br Med J. 1974; 1:358–360.
61. Hills NNH, Pfug JJ, Jeyasingh K, et al. Prevention of deep vein thrombosis by intermittent pneumatic compression of calf. Br Med J. 1972; 1:131–135.
62. Lee BY, Trainor FS, Kavner D, et al. Noninvasive prevention of thrombosis of deep veins of the thigh using intermittent pneumatic compression. Surg Gynecol Obstet. 1976; 142:705–714.
63. Turpie AGG, Gallus AS, Beattie WS, et al. Prevention of venous thrombosis in patients with intracranial disease by intermittent pneumatic compression of the calf. Neurology. 1977; 27:435–438.
64. Turpie AGG, Delmore T, Hirsh J, et al. Prevention of venous thrombosis by intermittent sequential calf compression in patients with intracranial disease. Thromb Res. 1979; 15:611–615.
65. Salzman EW, Ploet J, Bettlemann M, et al. Intraoperative external pneumatic calf compression to afford long-term prophylaxis against deep vein thrombosis in urological patients. Surgery. 1980; 87:239–242.
66. Nicolaides AN, Fernandes e Fernandes J, Pollock AV. Intermittent sequential pneumatic compression of the legs in the prevention of venous stasis and postoperative deep venous thrombosis. Surgery. 1980; 87:69–76.
67. Clarke-Pearson DL, Synan IS, Hinshaw W, et al.

Prevention of postoperative venous thromboembolism by external pneumatic calf compression in patients with gynecologic malignancy. Obstet Gynecol. 1984; 63:92–98.
68. Clarke-Pearson DL, Creasman WT, Coleman RE, et al. Perioperative external pneumatic calf compression as thromboembolism prophylaxis in gynecologic oncology: report of a randomized controlled trial. Gynecol Oncol. 1984; 18:226–232.
69. Jobson VW, Homesley HD, Welander CE. Comparison of heparin and intermittent calf compression for prevention of pulmonary embolism. Gynecol Oncol. 1983; 15:143.
70. Allenby F, Boardman L, Pflug JJ, et al. Effects of external pneumatic intermittent compression on fibrinolysis in man. Lancet. 1976; 2:1412–1414.
71. Knight MTN, Dawson R. Effect of intermittent compression of the arms on deep venous thrombosis in the legs. Lancet. 1976; 2:1265–1268.
72. Salzman FW, Davies GC. Prophylaxis of venous thromboembolism: analysis of cost effectiveness. Ann Surg. 1980; 191:207–218.
73. Fullen WD, Miller DH, et al. Prophylactic vena caval interruption in hip fractures. J Trauma. 1973; 13:403–410.

Reconstructive Techniques 12

Leslie A. Walton

Introduction

The increasing longevity of women, the renewed emphasis on physical well-being, the increasing attention to preservation of body image, and the expectation of a return to near-normal function are among the reasons why reconstructive approaches to the female genital tract are being increasingly emphasized and utilized in gynecologic surgery.

While extirpative procedures may require reconstructive surgery, sequelae of nonsurgical treatment may also compromise genital function and require some type of reconstruction. In addition, anatomic abnormalities of the genital tract may require surgical correction to augment function and enhance psychosocial well being.

This chapter will examine the reconstructive techniques related to the vulva, vagina, pelvic cavity, and abdominal wall (Table 12-1). The reconstructive needs can be simple or complex, and are clearly within the purview of the gynecologic surgeon. Emphasis will be placed on indications for reconstruction without detailed step-by-step diagramatic representation of the technical approaches; the interested reader can find detailed descriptions in surgical atlases. When special refinements in surgical technique exist, these refinements will be discussed. Some of these approaches utilize techniques that are common to disciplines such as plastic and reconstructive surgery. Advances in technologic equipment and surgical techniques may reduce the number of women requiring reconstruction after treatment for many conditions.

Procedures Involving the Vulva

The vulva and introitus may be scarred with resulting contracture after surgery, radiation therapy, and inflammatory diseases, especially chronic suppurative infections. In addition, perianal diseases with occasional fistula formation can contribute to vulvar and perineal disfigurement.

The choice of the surgical procedure is dictated by the extent of scarring, the degree of compromise of vulvovaginal function, the viability of the normal remaining tissue, and the status of any associated diseases. The operative

Table 12-1. Reconstruction

Vulva	
Relaxing incisions	Perineal or buttock flaps
Z-plasty	Bulbocavernosus fat pad
Grafting	Vulvovaginoplasty
Rhomboid flap	
Vagina	
Grafting with skin or human amnion	
Use of peritoneum to extend vaginal depth	
Creation of neovagina	
Vaginal vault suspension	
Pelvis	
Repositioning of ovary	
Omentum or mesocolon in pelvic reconstruction	
Pelvic peritoneum for a pelvic floor	
Abdominal wall	
Correction of incisional hernias, including use of prosthetic material	

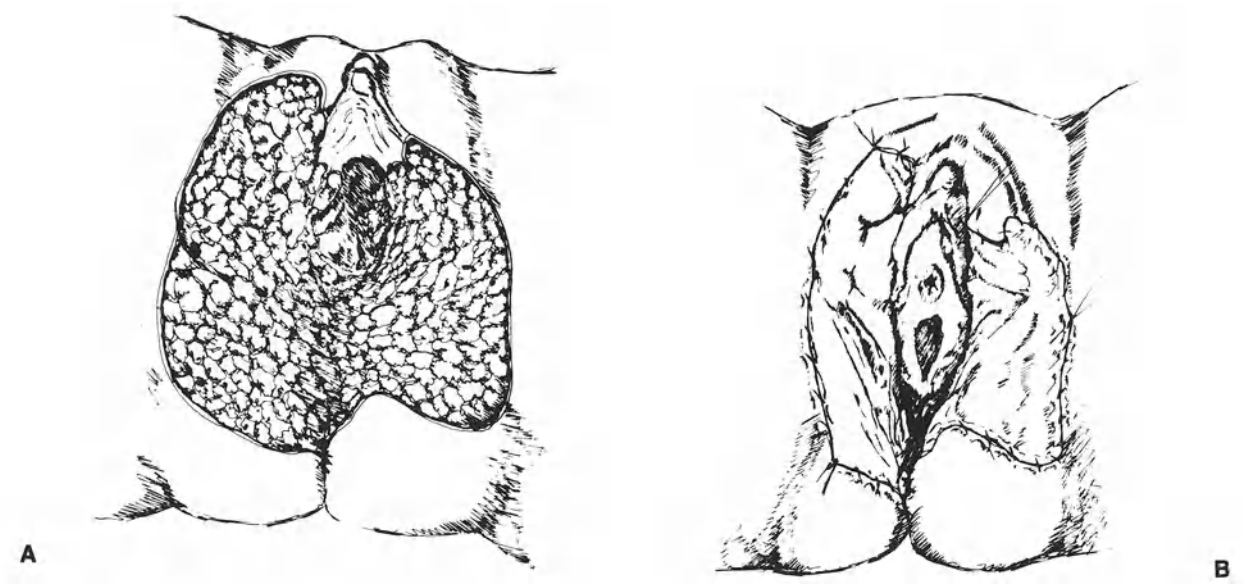

Figure 12-1. A, Vulva after "skinning vulvectomy." The raw surgical bed is ready for graft application. **B,** A split-thickness skin graft is applied. From Walton LA. Carcinoma in situ of the vulva. A self-instructional program. Chapel Hill, NC: Health Science Consortium, 1982.

site should be free of infectious processes; any existing disease should be quiescent.

Among the tenets that help to ensure a good cosmetic result when reconstructive surgery is performed are the following:

a. Tissue should be gently handled
b. The residual tissue should be healthy
c. Dead spaces should be obliterated
d. Hemostasis must be adequate
e. Fine suture material should be used
f. Suture lines should not be closed under tension.

Initial therapy for vulvovaginal introital stenosis usually involves careful counseling, the use of graduated dilators, and the vaginal application of estrogen cream. When an operative approach is selected, a simple surgical procedure for introital stenosis involves the use of relaxing incisions created on one axis with resuturing of the vulvovaginal tissue at a 90° axis. These incisions are made in a radial fashion around the introitus; the number of incisions is determined by the desired enlargement of the introitus. Occasionally, when the patient has a good perineal body, one incision can be made vertically into the perineal tissue and closed horizontally to enable the introitus to be widened.[1]

Second, a Z-plasty type of procedure may be indicated.[2] Careful anticipation of the desired result will dictate the length of the Z incisions that are created during surgery. A functional introitus can be created without utilizing an extensive surgical procedure. Postoperative recovery is usually uncomplicated.

Wide local excision is one of the standard approaches used in treatment of diseases such as vulvar intraepithelial neoplasia. When such surgical procedures on the labia majora, labia minora, or perineum create skin defects, split-thickness skin grafts can be used in closure rather than suturing of the surgical margins under tension. These skin grafts can be applied to segmental areas of the vulva and perineum. The skin graft can be obtained from a donor site on the posterior thigh or the posterior medial buttocks utilizing a dermatome. These areas are chosen to minimize the cosmetic effects of a residual scar. The graft varies in thickness from 0.015 to 0.017 in. The grafted surgical field usually heals within 14–21 days. Occasionally depigmentation with minimal scarring is seen at the donor site.

A large superficial denuded area is created on the vulva when a "skinning vulvectomy"—removal of vulvar skin without subcutaneous tissue—is performed for multifocal carcinoma

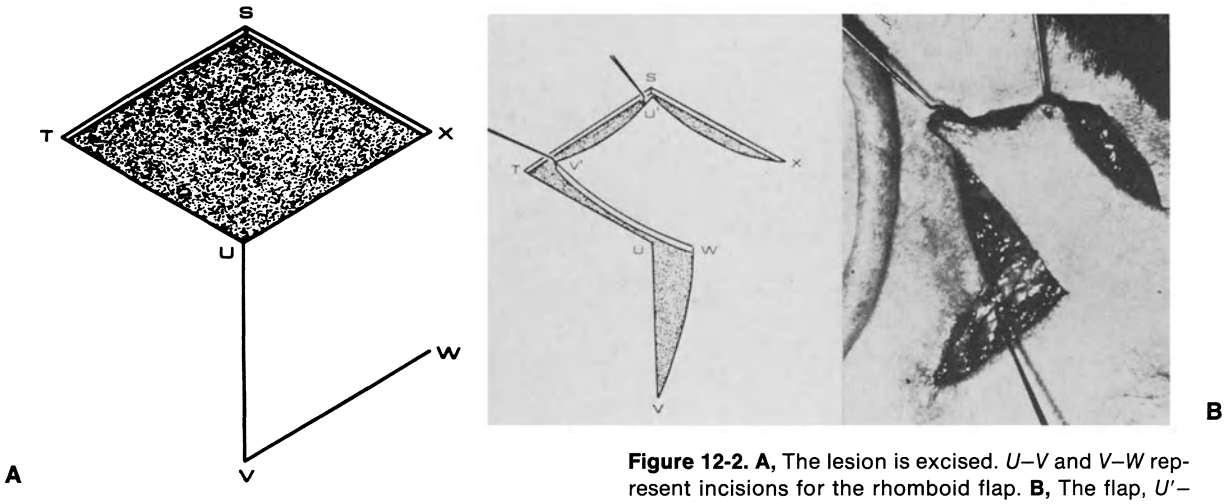

Figure 12-2. A, The lesion is excised. *U–V* and *V–W* represent incisions for the rhomboid flap. **B,** The flap, *U′–V′–WX,* is ready to cover the defect. The edges of *V′–U′* will be sutured to *S–T, V′–W* to *T–U,* and *X–U′* to *X–S.* From Lister GD, Gibson T. Closure of rhomboid skin defects: the flaps of Limbert and Duforementel. Br J Plast Surg. 1972; 25:300–314.

in situ of the vulva (Figure 12-1A). Usually, split-thickness skin grafts are taken from sites previously mentioned to cover the denuded area. A clean dry surgical bed is procured, and the graft is applied to cover the surgical field (Figure 12-1B). Graft take is usually in the 90% range in this category of patients. These patients can become ambulatory within 3–5 days. They experience a good return of sensation to the vulva and very minimal vulvar scarring, and can resume sexual function within 6 weeks.

Another complicated procedure to cover vulvar defects involves the use of a rhomboid flap.[3,4] A rhombus is a parallelogram with equal sides. A rhomboid flap (Figures 12-2A and -2B) is useful when lesions are large and difficult to include in an elliptical incision that will allow skin edge approximation. Preoperative marking to outline the placement of the flap is important. A satisfactory cosmetic result is usually obtained because skin excision for the long axis of the rhomboid is minimal. Attention to the principles of closure as outlined above apply when this procedure is utilized. Complications of infection and dyspareunia are rare.

Occasionally, large vulvar or perineal defects exist after tissue resection for a perineal sinus or after vulvectomy for hidradenitis. When the defects are closer to the perineum, full-thickness skin flaps from the perineum or the buttocks can be used.[5] These perineal or buttock flaps yield good cosmetic results. Sensory perception in the healed flap segment is equivalent to sensation in the preoperative period.

When small areas of scarring or ulceration are present in the vulvovaginal area, the bulbocavernosus muscle and its accompanying fat pad can be used to replace the tissue injured in this area. An additional benefit of this procedure is the introduction of a well vascularized pedicle with its blood supply from the perineal branch of the pudendal artery. As an example, the fat pad can be swung in to cover an area of ulceration in the lower vagina, the vulva, or the vulvovaginal junction. In addition, this labial fat pad has been useful in the management of difficult fistulas of the urinary or lower intestinal tract; the closure of urethrovaginal,[6] vesicovaginal (Figure 12-3), or rectovaginal fistulas[7] (Figures 12-4A and -4B). When a fistula is present, inflammation at the edges must be treated. At the time of the surgical repair, necrotic or fibrotic tissue is excised and the fat pad is brought to the fistulous site or the site of ulceration via a paravaginal tunnel. In addition to introducing a new blood

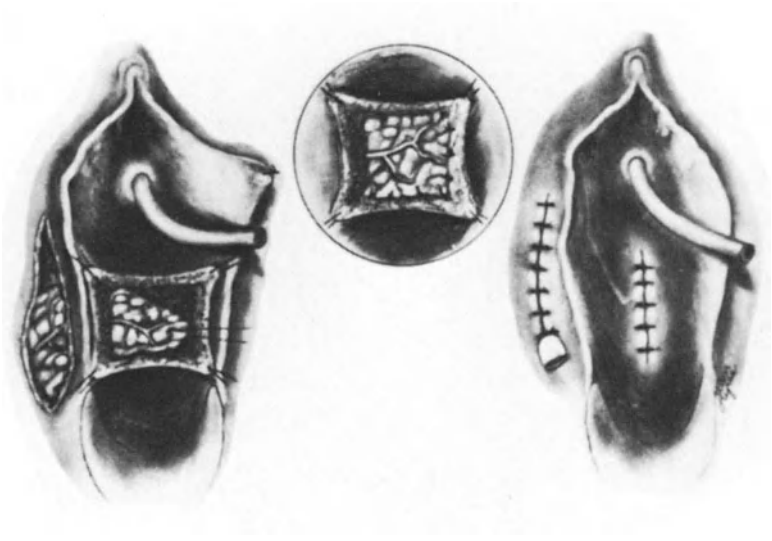

Figure 12-3. The labial fat pad is surgically created and brought through the paravaginal incision to the site of a vesicovaginal fistula (left). The pedicle with its blood supply is sutured in place after closure of the bladder defect (center). Approximation of the labial and vaginal mucosa (right). From Patil U, Waterhouse K, Laungani G. Management of eighteen difficult vesico-vaginal and urethro-vaginal fistulas with modified Ingelman–Sundberg and Martius operations. J Urol. 1980; 123:654. © 1980. The Williams and Wilkins Co., Baltimore.

supply, the fat pad provides healthy tissue to enhance repair and to fill the tissue gap that was present.

The Williams vulvovaginal plasty[8] offers another opportunity for vulvar-vaginal reconstruction. The original approach was used in patients with congenital absence of the vagina. If the vagina had previously been compromised by radiation scarring or foreshortened by surgery, modifications[9] of the original approach would allow the vaginal tube to be lengthened or a new canal to be created. In addition patients with androgen insensitivity (testicular feminization) who have a blindly ending vaginal pouch may benefit from this surgical approach. Creation of a neo-vagina offers better results.

Labial skin and subcutaneous tissue are used to create a vagina. After making a U-shaped incision into the vulva (Figure 12-5A), the incised skin edges are sutured so that a tubular pouch is created (Figure 12-5B). This surgical approach does not require a prolonged hospital stay. The neovagina is functional in about 6–8 weeks. This technique maintains the sensory function of the vagina and removes the need for continued dilation of the canal. On initial observation, the neo-vagina is at a right angle to the horizontal plane when the patient is lying down. However with time and after intercourse, the angulation disappears and a near normal vaginal axis results.

If large vulvar, perineal, or groin defects exist, more complicated types of flaps are used. One type of complicated flap in wide usage in gynecologic surgery is the myocutaneous flap. These flaps enable muscle, its blood supply, and overlying subcutaneous tissue and skin to be swung in to cover large defects after radical surgery, massive trauma, or radiation damage. Flaps currently used include the gracilis muscle flap[10,11] and the tensor fascia lata flap.[12]

Difficult vesicovaginal or rectovaginal fistulas, which might occur after radiation therapy, can be corrected with the use of the gracilis muscle flap.

Procedures Involving the Vagina

Reconstructive procedures involving the vagina vary in complexity from treatment of vaginal ulcers to creation of a neovagina. Shortening of the vagina can occur as a result of surgery, erosion, ulceration, radiation therapy, or vaginal adhesions. In addition, fistula formation can create the need for reconstruction. Congenital anomalies, such as partial vaginal agenesis and vaginal septa, may require surgery. Finally, the congenital absence of the vagina mandates the construction of a neovagina. The timing of the reconstructive procedure is a very important phase in the sexual rehabilitation function and psychosocial development of the young woman. Careful counseling, patient motivation, and continued vaginal dilation with the use of stents and molds are important in the management of vaginal reconstruction (see Chapter 13).

Occasionally only a segment of the lower vagina is present, as in mullerian aplasia or vagi-

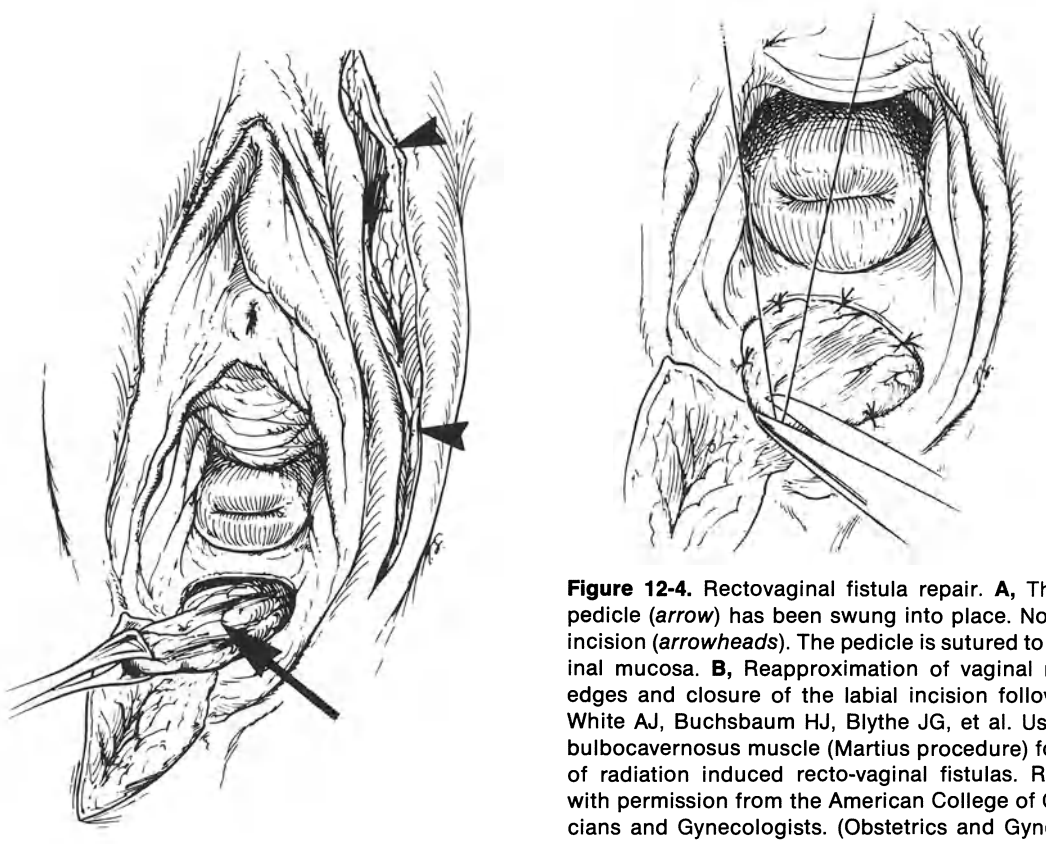

Figure 12-4. Rectovaginal fistula repair. **A,** The labial pedicle (*arrow*) has been swung into place. Note labial incision (*arrowheads*). The pedicle is sutured to the vaginal mucosa. **B,** Reapproximation of vaginal mucosal edges and closure of the labial incision follow. From White AJ, Buchsbaum HJ, Blythe JG, et al. Use of the bulbocavernosus muscle (Martius procedure) for repair of radiation induced recto-vaginal fistulas. Reprinted with permission from the American College of Obstetricians and Gynecologists. (Obstetrics and Gynecology, 60, 1981, pp. 115–116.)

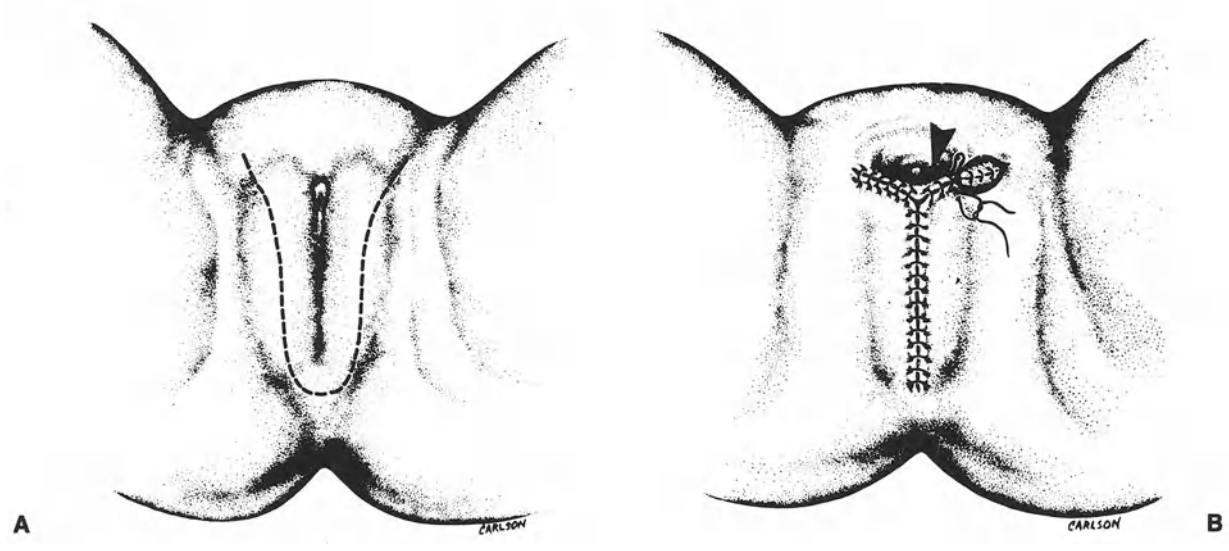

Figure 12-5. Vulvovaginoplasty. **A,** The vulvar incision is outlined. **B,** Appearance of the vulva after vulvoplasty. The vaginal pouch is behind the central line of sutures. *Arrowhead* indicates vaginal orifice. From Day TG, Stanhope R. Vulvovaginoplasty in gynecologic oncology. Reprinted with permission from the American College of Obstetricians and Gynecologists. (Obstetrics and Gynecology, 50, 1977, p. 362.)

nal atresia. Dilatation as advocated by Frank[13] or a modification as advocated in the saddle technique[14] can be used in the creation of a functional vagina. If lengthening of an abbreviated cavity is required, dissection is used to deepen the cavity, which can then be lined with a split-thickness skin graft or with human amnion.[15] If the latter is to be used, fetal amnion is harvested, prepared, and applied using the same techniques as in skin grafting. The amnion must be chosen from an Rh-negative patient. Adjacent epithelium usually grows and covers the amnion. Continued dilatation and the use of estrogen cream is necessary to maintain an adequate canal.

Creation of a neovagina requires training and expertise. A canal between the urethral meatus and anus is created by sharp and blunt dissection to about the level of the pelvic peritoneal reflection.[16] When an adequate cavity is created and absolute hemostasis is achieved, a vaginal mold covered with a split-thickness skin graft is inserted into the vaginal space. The mold is held in place for 7 days by suturing to the labia. Modifications of the original technique include infiltrating the space with neosynephrine and the topical application of thrombin. After the mold has been removed, patency of the canal is maintained by the use of soft stents. Appropriate counseling and dilatation are an important part of care. If intromission is not routinely practiced, the vaginal stents have to be used for long periods of time, occasionally up to 6 months, to keep the canal open. Application of topical estrogen cream is routinely practised.

When a partial vaginectomy accompanies radical hysterectomy for cervical cancer, the vaginal length will be compromised. Bladder and cul-de-sac peritoneum can be utilized to form a pouch at the apex of the existing vagina.[17] Essentially, a 2–3 cm of bladder flap peritoneum and an equivalent length of anterior rectal or cul-de-sac peritoneum (Figure 12-6A) are sutured in the middle (Figure 12-6B), superior to the plane of the vagina. This pouch can be lined with amnion or skin. The cavity must be kept open with a mold or vaginal stent. With time, vaginal epithelium from the preexisting vagina will extend onto this peritoneal lining.

Occasionally, the entire vagina is obliterated or is replaced by scar tissue such that the surgical process cannot utilize the previous vaginal canal. Under these circumstances, careful dissection into the perineum[18,19] immediately below the posterior vagina is an alternative. Using sharp and blunt dissection, a tunnel is created in the rectovaginal septum. Entry into the rectum must be avoided. When hemostasis is adequate and satisfactory canal depth is achieved, the canal is lined with a split-thickness skin graft. Human amnion can be used as alternative tissue to line this canal. It is impor-

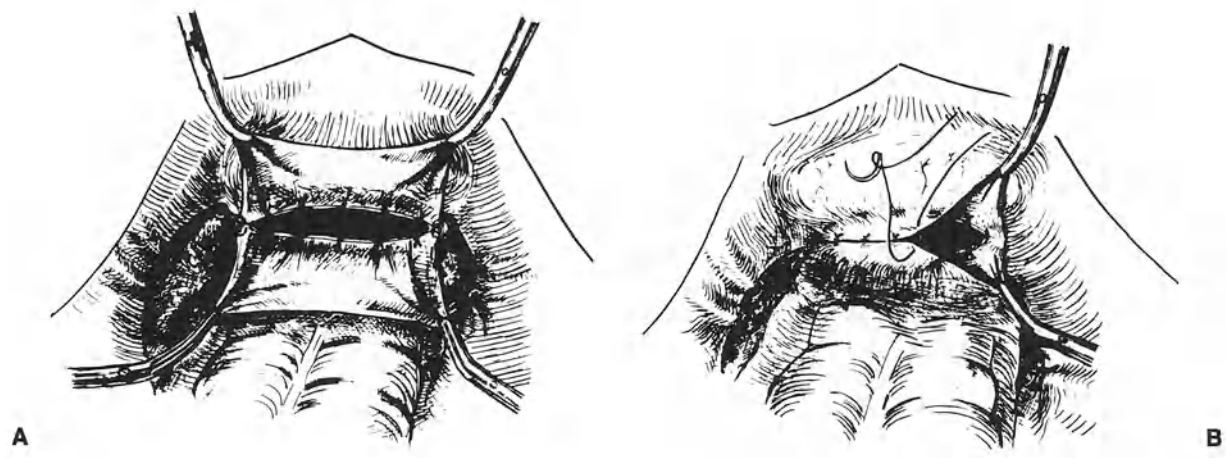

Figure 12-6. Use of the pelvic peritoneum. **A,** Unattached bladder and rectal peritoneal flaps are created. **B,** The anterior and posterior flap edges are reapproximated. From Saito M, Kumasaka T, Kato K, et al. Vaginal repair in the radical operation for cervical carcinoma. Acta Obstet Gynecol Scand. 976; 55:152–153.

tant that the patient use estrogen cream, wear a vaginal stent, and use vaginal dilators. The application of a topical anesthetic may facilitate this latter process.

Reconstruction of the vagina is sometimes indicated after radical or ultraradical surgery, such as a radical vulvectomy or pelvic exenteration. Usually musculocutaneous flaps such as the gracilis muscle are utilized. In addition, the bulbocavernosus muscle and skin have been utilized in vaginal reconstruction.[20] Significant segments of the posterior vagina must be preserved at the time of radical surgery when this latter technique is used.

Vaginal Vault Suspension

A small but increasing number of patients are seeking gynecologic care for vaginal vault prolapse. The patients are usually postmenopausal. In a review of four series with 55 patients with vault prolapse,[21–24] only 4 patients were younger than 49 years of age and 46 patients were 52 years or older. When the older healthy, active patient seeks surgical correction, a challenging problem exists.

When a woman is standing, the upper vagina normally points towards the hollow of the sacrum and is located almost horizontal to the ground. The posterior aspect of the upper vagina receives support from the endopelvic fascia and levator ani muscle fibers. Attenuation or atrophy of endopelvic structures, chronic pulmonary problems, and obesity are among the conditions contributing to vaginal vault prolapse or eversion.

Both the transvaginal and transabdominal approaches or a combination of these procedures have been used in the repair of vault prolapse. The additional presence of symptomatic prolapse of the bladder or rectum dictates an initial vaginal approach. The vault prolapse can then be corrected by suturing or fixing the vaginal apex as high as possible. The success of the approach will depend on restoring the vaginal apex as much as possible to its normal position.

When a vaginal approach is utilized, the vaginal (usually posterior) mucosa is incised to the apex of the vagina. An enterocele is usually present, and a diligent effort should be made to identify it and ligate the sac. The vaginal apex is then sutured to sacrospinous tissue, perirectal fascia, or uterosacral tissue. It is difficult to easily locate these supporting structures, especially the latter two, at the time of surgical correction. Any space between the vaginal apex and sacrum must be obliterated to thwart reformation of another enterocele, which could contribute to repeat apex prolapse. Redundant vaginal mucosa is excised, and midline reapproximation of the mucosa is accomplished as the final step in the procedure.

Current surgical preference in the management of vault prolapse favors sacral colpopexy through an abdominal incision.[25] The peritoneum over the hollow of the sacrum is incised. Sutures are placed into the periosteum, and the vaginal apex is attached to the sacrum. Various prosthetic materials commonly used for suspension include Teflon, Mersilene, and Proline. These are all synthetic materials and rejection and sinus formation are known complications. Autogenous grafts like the rectus fascia have been tried to circumvent these complications. Other surgeons have used cadaver dura mater;[24] in addition, completely absorbable Vicryl mesh has been used in a small series of patients.[26] Injury to the ureters and sigmoid colon can occur during the sacrospinous suspension. More commonly, however, bleeding from the presacral blood vessels occurs and poses an irritating problem.

The success rate of vaginal prolapse correction is difficult to determine because of confounding contributory factors.

Procedures Involving the Pelvis

Ovarian Transposition. The preservation of ovarian function has assumed greater import in the functioning of the female patient. Patients with lymphomas constitute the largest group requiring ovarian preservation. Transposition of the ovary is important in surgical patients and especially in the younger patient who is to undergo external radiation therapy involving the pelvic cavity or pelvic- and abdominal-node–bearing areas. Two surgical approaches have been utilized: (a) the ovary is displaced laterally into the iliac fossa and out of the field of radiation,[27] and (b) the ovary is displaced medially behind the uterine fun-

dus.[28] The latter approach is not used as often as the former, but has the potential for maintaining fertility. Long-term follow-up of these patients has demonstrated satisfactory ovarian function.

Use of the Omentum or Colon Mesentery. The use of healthy adipose vascular tissue to increase vascularity of another area or to serve as replacement tissue comes into focus when the omentum is used in reconstruction. Occasionally, a denuded or traumatized pelvic floor results from surgery, and the need exists to cover the floor to reduce the possibility of multiple loops of bowel becoming adherent to the pelvic floor. The omentum can be surgically lengthened after deciding whether the right or left gastroepiploic artery will serve as the blood supply for the omental flap. The lengthened omentum is then sutured to the floor of the cul-de-sac. On other occasions, the omentum is used as a sling[29] to prevent multiple loops of bowel from adhering to the raw pelvic floor.

Another approach to covering the pelvic floor involves the use of peritoneum from the bladder, rectosigmoid, and the lateral pelvic wall. After peritoneal flaps from these organs are preserved, the peritoneal edges are reapproximated and also attached to areas of the pelvic sidewall so as to reduce the raw surface area of the pelvic floor. Finally, synthetic material such as the Vicryl mesh[30] has been used to cover the raw pelvic floor.

Approach to Incisional (Ventral) Hernia Repair

The incidence of postoperative incisional hernia is 1%, rising to about 10% if a wound infection occurs and to about 30% if the patient has suffered wound dehiscence with reclosure. Infection and dehiscence are the leading causes of incisional herniation. The transverse abdominal incision has a significantly lower incidence of herniation than the midline incision (see Chapter 3).

Once the diagnosis of incisional herniation is established, a careful assessment must be made as to whether surgery or conservative therapy is indicated. The possibility for successful treatment of the hernia and the possible morbidity of the surgical procedure must be carefully evaluated. The risk of intestinal incarceration is one reason for surgical intervention. The risk is increased if the hernial defect is small, and is probably nonexistent if a full-length (large) incisional hernia is present. However, some patients with large hernias may demonstrate skin thinning and ulceration requiring hernia repair. Finally, a few patients with large hernias have respiratory problems due to their inability to cough, and require surgical correction to improve continuing respiratory ailments.

Conditions contributing to a high failure rate of repair are

a. Significant pulmonary disease: chronic coughing can lead to failure of the repair
b. Size: hernias extending the entire length of the incision are difficult to repair
c. Obesity: if repair is contemplated, a 30% weight loss is recommended before surgical correction is attempted.

Overweight is a contraindication to ventral hernia repair.

When repair is contemplated, all nutritional and metabolic deficits must be corrected. In addition, it is best to wait for at least 6 months and possibly 1 year after initial surgery, if feasible, before surgical correction is attempted. At that time all reparative processes are probably stabilized (see Chapter 5).

Adherence to certain principles will increase the changes for a successful repair:

a. Fibrotic tissue should be resected
b. The skin edges should be reflected away from the wound
c. The rectus fascia must be identified
d. The entire wound should be examined from the peritoneal side to rule out other hernias
e. Meticulous hemostasis must be obtained
f. Appropriate drains should be used to avoid hematoma and seroma formation
g. Tension on the suture line should be avoided.

During the hernia repair, careful dissection is required. After the hernial sac is identified and excised, the peritoneal cavity is inspected. Occasionally, easy reapproximation of the fascial edges is feasible. Some surgeons recommend using the fascia to reinforce the incision

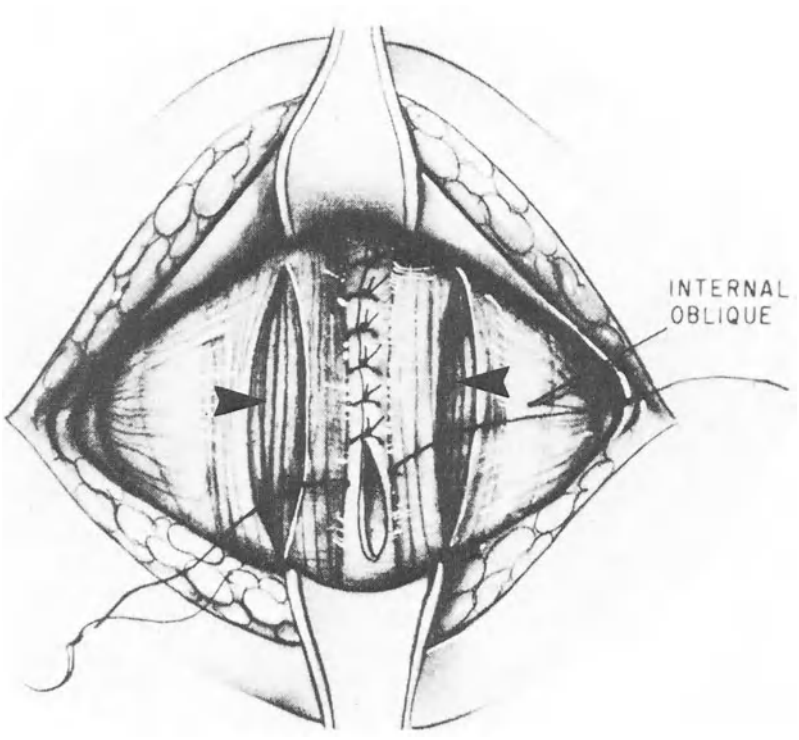

Figure 12-7. Relaxing incisions, indicated by *arrowheads,* in anterior rectus sheath. Sutures indicate reapproximated fascial edges. Reproduced with permission from Morton JH. Abdominal wall hernias. In: Schwartz SI, ed. Principles of surgery, 4th Edition. New York: McGraw-Hill, 1984:1473.

site. To accomplish this, a wide strip of rectus fascia is obtained by carefully dissecting it from the subcutaneous tissue and rectus muscle. The edge of one fascial margin, for example, the right edge, is sutured to the underside of the fascial margin of the left side, and the left fascial margin is then sutured to the surface of the right rectus fascia.[31] The superiority of this type of closure over careful edge to edge fascial reapproximation has not been established.

When fascial edges are being sutured together, a closure without tension can be accomplished by the use of longitudinal relaxing incisions[32] (Figure 12-7) in the anterior rectus sheath. These incisions are made on either side of the original hernia. The rectus fascial edges are then approximated without tension. These relaxing incisions do not cause herniation because of the underlying rectus muscle.

If the defect is large and the fascial edges cannot be approximated, a bridge technique is utilized. Available bridge material include Tantalum, gauze-braided polyester (Mersilene), or polyprophylene mesh (Marlex). Tantalum does not enjoy wide usage. These materials have been successful in hernia repair. They must be sewn to the fascial edges with strong sutures such as prolene. However, if the wound becomes infected, they must be removed.

References

1. Crossen HS, Crossen RJ. Diseases of Women, 9th Edition. St. Louis: C.V. Mosby, 1944; p. 346.
2. Wilkinson EJ. Introital stenosis and Z-plasty. Obstet Gynecol. 1971; 38:638–640.
3. Lister GD, Gibson T. Closure of rhomboid skin defects: The flaps of Limbert and Duforementel. Br J Plast Surg. 1972; 25:300–314.
4. Barnhill DR, Hoskins WJ, Metz P. Use of the rhomboid flap after partial vulvectomy. Obstet Gynecol. 1983; 62:444–447.
5. Hirshowitz B, Peretz BA. Bilateral superomedial thigh flaps for primary reconstruction of scrotum and vulva. Ann Plast Surg. 1982; 8:390–396.
6. Patil U, Waterhouse K, Laungani G. Management of eighteen difficult vesico-vaginal and urethrovaginal fistulas with modified Ingelman-Sundberg and Martius operations. J Urol. 1980; 123:653–656.
7. White AJ, Buchsbaum JH, Blythe JG, et al. Use of the bulbo-cavernosus muscle (Martius procedure) for repair of radiation-induced recto-

vaginal fistulas. Obstet Gynecol. 1982; 60:114–118.
8. Williams EA. Congenital absence of the vagina. A simple operation for its relief. J Obstet Gynecol Br Commw. 1964; 80:511–516.
9. Day TG, Stanhope R. Vulvovaginoplasty in gynecologic oncology. Obstet Gynecol. 1977; 50:361–364.
10. Becker DW Jr, Massey FM, McCraw JB. Musculocutaneous flaps in reconstructive pelvic surgery. Obstet Gynecol. 1979; 54:178–183.
11. Wheeless CR, McGibbon B, Dorsey JH, Maxwell GP. Gracilis myocutaneous flap in reconstruction of the vulva and female perineum. Obstet Gynecol. 1979; 54:97–102.
12. Chafe W, Fowler WC, Walton LA, Currie JL. Radical vulvectomy using the tensor fascia lata myocutaneous flap. Am J Obstet Gynecol. 1983; 145:207–213.
13. Frank RT. The formation of an artificial vagina without operation. Am J Obstet Gynecol. 1938; 35:1053–1055.
14. Ingram JM. The bicycle seat stool in the treatment of vaginal agenesis and stenosis: A preliminary report. Am J Obstet Gynecol. 1977; 129:543–547.
15. Tancer MC, Katz M, Veridiano NP. Vaginal epithelialization with human amnion. Obstet Gynecol. 1979; 54:345–349.
16. McIndo AH, Banister JB. An operation for the cure of congenital absence of the vagina. J Obstet Gynecol Br Emp. 1938; 45:490–494.
17. Saito M, Kumasaka T, Kato K, et al. Vaginal repair in the radical operation for cervical carcinoma. Acta Obstet Gynecol Scand. 1976; 55:151–154.
18. Simmons RJ, Millard DR. Reconstruction of a functioning vagina following radiation therapy for cancer of the cervix. Surg Gynecol Obstet. 1961; 112:761–762.
19. West JT, Ketcham AS, Smith RR. Vaginal reconstruction following pelvic exenteration or post irradiation necrosis. Surg Gynecol Obstet. 1964; 118:788–794.
20. Hatch KD. Construction of a neovagina after exenteration using the vulvobulbo cavernosus myocutaneous graft. Obstet Gynecol. 1984; 63:110–114.
21. Thornton Jr WN, Peters III WA. Repair of vaginal prolapse after hysterectomy. Am J Obstet Gynecol. 1983; 147:140–148.
22. Seigworth GR. Vaginal vault prolapse with eversion. Obstet Gynecol. 1979; 54:255–260.
23. Feldman GB, Birnbaum SJ. Sacral colpopexy for vaginal vault prolapse. Obstet Gynecol. 1979; 53:399–401.
24. Lansman HH. Posthysterectomy vault prolapse: sacral colpopexy with dura mater graft. Obstet Gynecol. 1983; 63:577–582.
25. Nichols D. Sacrospinous fixation for massive eversion of the vagina. Am J Obstet Gynecol. 1982; 142:901–904.
26. Buchsbaum HJ, Lifshitz S. Vicryl mesh for vaginal vault suspension. personal communication, 1985.
27. Belinson J, Doherty M, McDay JB. A new technique for ovarian transposition. Surg Gynecol Obstet. 1984; 159:157–160.
28. Ray GR, Trueblood HW, Enright LP, et al: Oophoropexy: a means of preserving ovarian function following pelvic megavoltage radiotherapy. Pathology. 1970; 96:175–200.
29. Buchsbaum HJ, White AJ. Omental sling for management of the pelvic floor following exenteration. Am J Obstet Gynecol. 1973; 117:407–412.
30. Buchsbaum HJ, Christopherson WA, Lifshitz S, et al. Pelvic floor reconstruction: use of Vicryl mesh. *Arch Surg.* 1985; 120:1389–1391.
31. Zollinger RM, Zollinger Jr RM. Atlas of surgical operations, 4th edition. New York: MacMillan, 1975: Plate CLXI.
32. Schwartz SI. Principles of surgery, 4th edition. New York: McGraw-Hill, 1984: p. 147B.

The Pediatric Patient 13

PonJola Coney

Introduction

The leading causes of death in children are injuries due to blunt and penetrating trauma, malignancies, and congenital malformations. Vaginal bleeding and abnormal development are among the challenging problems that channel the pediatric patient to the gynecologist. These problems, adolescent pregnancy and unannounced emergencies, often bring the pediatric patient to the gynecologist for the first time. When called on to care for the pediatric patient, the gynecologist is confronted with a tremendous challenge that requires recognition and appreciation of signs and symptoms that may represent major or minor disease processes. A diagnosis should be made with confidence and a management plan begun at once. Training programs in obstetrics and gynecology do not always provide experience in caring for pediatric patients. The specialties of pediatrics, pediatric surgery, and adolescent medicine essentially provide comprehensive health care for this population of patients with the obstetrician–gynecologist serving in an occasional consultant role. The intent of this chapter is to emphasize the gynecologic problems particular to infants, preadolescents, and adolescents.

Initial Evaluation

The aim in caring for the pediatric patient is not different than in adult practice. Exquisite bedside manner and gentleness must be utilized to solve the problem. Abnormalities of the reproductive system are laden with emotion, and the first encounter offers the physician the opportunity to maintain normal physiologic function and avoid complications. Careful attention must be paid to the psychologic profile of the patient.

Before attempting the examination, it is important to become familiar with the child and to establish rapport with the child's parents to alleviate apprehension. The older child should be treated as an adult. A complete history and physical examination including a pelvic examination are mandatory to make a diagnosis and to start the timetable for surgical intervention if indicated.

The exam in the prepubertal child can be performed without the routine "stirrup" formality. If the patient is not cooperative and the examination cannot be performed in the usual manner, anesthesia or a pediatric cocktail should be administered without hesitation. Normal variants in the prepubertal patient examination include a more anteriorly located introitus and a more prominent clitoris. The vagina appears erythematous, and the pH is alkaline rather than acid. In the absence of estrogen influence, the vaginal epithelium is uncornified. The hymen is redundant, and the cervix may appear as an ectropion. The uterus is small, 2.5–3.0 cm, with the cervix comprising two-thirds of the organ. Proper instruments should be utilized for inspection of the vagina. Usually, the rectal examination is more helpful because of the poor distensibility of the upper vagina. A diligent and gentle search should be

made to confirm normal internal genital anatomy. The uterus and ovaries should be identified.

Routine laboratory tests and procedures such as blood chemistry, hematology, cytology, biopsy, and x-rays should be utilized in the evaluation of the pediatric patient. The uterus can be identified on sonographic examination regardless of the child's age. The size, however, may vary from 2.5 cm to 7 cm after puberty. The ovary, which can be visualized with a high-frequency transducer (3.5 mHz), is 1–2 cm in size. Endocrine assays, FSH (follicle-stimulating hormone), LH (Luteinizing hormone), estradiol, testosterone, and HCG (human chorionic gonadotropin) are important in defining conditions of intersexuality and abnormal maturation. Karyotype determination is also necessary in many cases.

Management of Specific Problems

Major conditions encountered in childhood requiring surgical treatment by the gynecologist occur in the prepubertal and adolescent periods (see Table 13-1).[1,2]

Ambiguous Genitalia

The most common cause of female pseudohermaphroditism in the newborn is congenital adrenal hyperplasia (CAH).[3–5] Ninety percent of cases are due to deficiency of the enzyme 21-hydroxylase. Other enzyme deficiencies that primarily result in CAH and ambiguous genitalia include 11β-hydroxylase and 3β-hydroxysteroid dehydrogenase (3β-01).[4,5] CAH is an autosomally recessive inherited disorder and can be diagnosed prenatally.

The virilizing effect on the external genitalia of the female can range from simple clitoral enlargement to complete virilization with a phallus and fused labia much like the scrotum. The diagnosis of CAH is confirmed by elevated plasma 17-hydroxyprogesterone in a genetic (XX) female. Patients will present as salt-losing or non-salt-losing variety. Maternal ingestion of androgenic hormones and maternal androgen-producing tumor should be excluded.

Management of the ambiguous genitalia should begin only after treatment is instituted and the patient is medically stable. The genital ambiguity is not progressive once treatment is

Table 13-1. Most Common Gynecologic Disorders in Pediatric Patients[a]

Age	Disorder	Signs/symptoms	Diagnostic aid(s)	Management
Infant	Elevated maternal estrogens	Vaginal bleeding	Inspection	No intervention
	Trauma	Vaginal bleeding	Inspection	Intervene for specific repair
	Congenital adrenal hyperplasia, adrenal tumors, exposure to androgenic hormones	Ambiguous or anomalous genitalia (clitoromegaly, fused labia)	Plasma 17-Hydroxyprogesterone Urinary 17-Ketosteroids Karyotype	May require clitoral reduction, labial separation, vaginoplasty
	Foreign body	Vaginal discharge	Inspection	Removal
	Labial agglutination	Asymptomatic cryptomenorrhea	Inspection; vertical line in center	Surgically separate if medical therapy fails
	Ectopic ureter with vaginal terminus	Cystic mass protruding from vagina, vaginitis, hydroureter, hydronephrosis	IVP	Surgery indicated
	Prolapsed urethra/urethrocele	Dysuria, red mass at vestibule	Retrograde cystourethrogram Attempt to catheterize center of mass	Surgery indicated
	Vaginal ectopic anus	Dimple at anal site, constipation Intestinal obstruction	Inspection Lateral x-rays	Surgery indicated

Table 13-1. *Continued*

Age	Disorder	Signs/symptoms	Diagnostic aid(s)	Management
Puberty	Lichen sclerosus et atrophicus	White, papular lesion on vulva and perianal area	Biopsy	Medical therapy
	Vaginal adenosis	Erythematous or strawberry-red areas in the vagina	Cytology/biopsy	As indicated
	Vaginal clear-cell adenocarcinoma	Vaginal bleeding, discharge, pain	Biopsy	Radical surgery
	Sarcoma botryoides	Edematous, grapelike friable mass protruding from the vagina	Biopsy	Chemotherapy, surgery
	Condylomata acuminata	Vaginal spotting, asymptomatic	Podophyllin Trichloroacetic acid	May require surgical excision or laser ablation.
	Ovarian tumors and cysts	Abdominal pain, nausea, vomiting, vaginal bleeding, precocious puberty (isosexual, heterosexual), pelvic mass	Ultrasound, IVP	Surgery if indicated
	G-I disturbances: Acute appendicitis Inflammatory bowel disease Intussusception Meckel's Diverticulum	Abdominal pain, nausea, vomiting fever, intestinal obstruction	Abdominal films, CBC with differential, electrolytes	Surgery if indicated
	Androgen Insensitivity (TF)[b]	Blind vagina, inguinal–labial masses	Karyotype, FSH, LH, plasma testosterone	Gonadectomy
	Imperorate hymen	Cryptomenorrhea	Ultrasound	Surgical incision/ excision
	Vaginal septum	Dysmenorrhea, pelvic pain, abdominopelvic mass		
	Vaginal atresia	Rare cryptomenorrhea	Inspection, IVP, laparoscopy, karyotype	Vaginal construction
	XY Gonadal dysgenesis	Delayed puberty Ambiguous genitalia	Karyotype, FSH, LH, testosterone estradiol, X-ray	Gonadectomy
	DUB[c]	Menorrhagia, menometrorrhagia, pelvic/ back pain, fatigue	CBC, platelets, PT/ PTT, bleeding time, FSH/LH, testosterone, HSG, hysteroscopy, TSH/T_4	Surgery *only* if medical therapy fails

[a] Reprinted with permission from W.B. Saunders Co.[2] and Lippincott/Harper and Row[1].
[b] TF, Testicular feminization.
[c] DUB, Dysfunctional uterine bleeding.

begun. If any consideration is given to rearing as a male, the urologist should be consulted. In the usual case, femaleness can be preserved in every sense including reproduction. Clitoroplasty and surgical opening of the introitus and vestibule can be done any time after the patient is stable. Vaginoplasty and vaginal dilatation should be deferred until there is full cooperation from the patient to avoid secondary surgery. The parents should be fully informed of the good prognosis and of the inheritance pattern.

Vaginal Atresia (Rokitansky–Kuster–Hauser Syndrome)

Vaginal atresia results from failure of canalization of the vaginal plate. The most minor type is imperforate hymen. The incidence is 1 in 5000 females.[6,7,8] These patients most often present with primary amenorrhea. Ovarian development is normal as evidenced by normal, feminine secondary sex characteristics. Associated developmental abnormalities of the renal (50%) and skeletal systems (25%) are frequent. Thus, an IVP and study of the spine should be performed.[7] A karyotype should be done to differentiate the Androgen Insensitivity Syndrome (AIS). Less than 10% of patients will have a functional uterus. If it is present, cyclical abdominal pain and hematometra may be presenting complaints.

Creation of a new vagina should be deferred until there is full development of pelvic structures. The child must be counseled regarding her reproductive incapability. She must understand that the only goal is to create a vagina to permit sexual intercourse. Surgical correction can be considered if the patient is psychologically mature and motivated. Rudimentary Mullerian structures should be removed.

The desired technique for creating a neovagina is that of MacIndoe, although numerous techniques have been described[6–13] (Figures 13-1 through 13-5). Preoperative preparation

Figure 13-1. Vaginal atresia. The external genitalia appear normal.

Figure 13-2. A split-thickness skin graft (*STSG*) is obtained in one piece as thin as possible.

Figure 13-3. The stent is fully draped with the STSG.

should include emptying of the lower bowel, shaving of the perineum, pubic area, thighs, and buttocks, and prophylactic antibiotics. The patient is placed in the dorsolithotomy position, and a Foley catheter to gravity drainage is inserted into the bladder. Laparoscopy should be performed prior to the procedure to evaluate any Mullerian structures that may be present. A transverse incision is made between the urethra and rectum. A space is bluntly and sharply dissected proximally to the level of the peritoneal reflection, large enough to accomodate a 12 × 3 cm stent, being careful to avoid damage to the urethra and rectum. A split-thickness skin graft should be taken from the thigh or buttock and kept moist with saline until used. The donor site is covered with iodine-impregnated gauze and Teflon. Complications primarily include hematoma formation and infection; therefore, meticulous care should be given to hemostasis.

The stent, made of styrofoam covered with a condom, is covered with the skin graft and placed into the space. The edges of the graft are sutured to the labia and the mold is left in place for 1 week. The patient is kept at bedrest during this time on a low-residue diet. Antibiotics are continued until the mold is removed. A suprapubic catheter is preferable during this postoperative period. After 1 week, the stent is removed, the neovagina is cleansed by saline irrigation, and a temporary stent is placed. An adjustable inflatable vaginal stent (10 cm × 3 cm, Heyer Schulte Corp., Goleta, CA) is commonly used to continue daily intermittent dilation in lieu of the styrofoam–condom stent until healing is complete and the several months of the contractile phase are over.

The patient has to be entirely committed to daily mechanical dilation of her neovagina for at least 6 months once she leaves the hospital. Failure to do so will result in contracture. Sec-

Figure 13-4. Neovagina is successfully maintained by daily dilatation with stent.

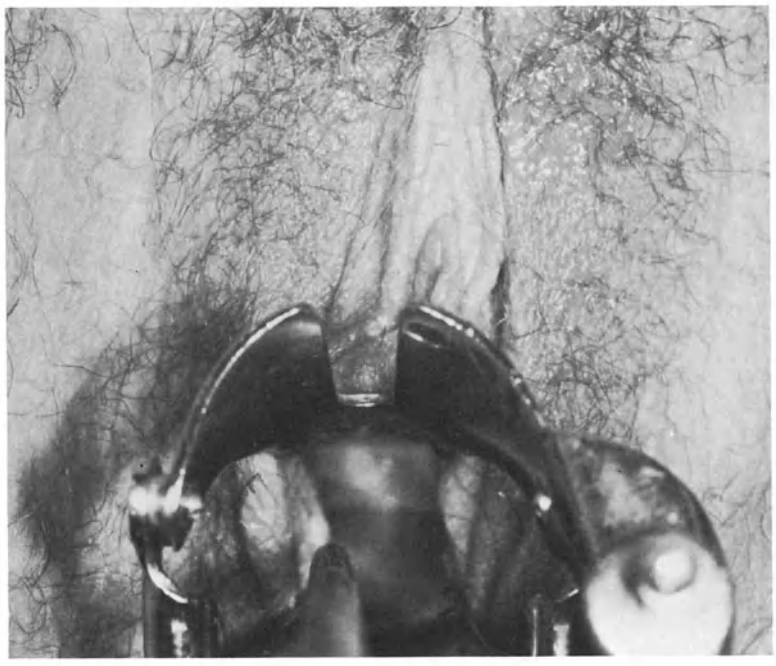

Figure 13-5. Appearance of vaginal mucosa several months later after complete epithelization.

ondary surgery may be required. Coitus can begin as early as 6 weeks following surgery, and she can expect normal sexual function.[13–15]

Imperforate Hymen

This anomaly is usually brought to medical attention with complication of acute onset of abdominal pain. The presence of a fluctuant mass is often noted on rectal examination and occasionally may extend to the umbilicus.[6] The hymen is intact and may be bulging. A cruciate incision is made into the hymen. If hematometra is present, antibiotics should be administered.

Vaginal Septum

Vaginal septa are usually found at the junction of the upper one-third and lower two-thirds of the vagina.[6] History may reveal cryptomenorrhea and dyspareunia. Treatment is excision and repair.

Disorders of Intersex

Swyer syndrome is XY gonadal dysgenesis characterized by an XY genotype in phenotypic females.[16,17] Patients with the syndrome are tall with normal Mullerian development and bilateral streak gonads. They most often present with failure to develop secondary sex characteristics and amenorrhea. The differential diagnosis is AIS.

AIS or testicular feminization is familial male pseudohermaphroditism in which androgen binding to the cytosol receptor is impaired.[18] The patient is always phenotypically female with male genotype (XY). The internal genitalia consists of a blind vaginal pouch with cryptorchid testes in the inguinal area or in the labia. Breast development is excellent. Pubic and axillary hair are sparse or absent. The gonadotropins, FSH and LH, are elevated. Testosterone levels are in the range of normal for males.

Mixed gonadal dysgenesis is characterized by an ovotestis on one side and a streak gonad on the contralateral side.[19] The sex chromosomal composition is predominantly 45 XO : 46 XY. These patients have ambiguous external genitalia, female internal genitalia, and may virilize at puberty. Amenorrhea and failure to develop feminine secondary sex characteristics are also the presenting complaints.

In any patient with abnormal sexual development or unusual sexual maturation, think of *chromosomes* and *hormones*. Germ-cell tumors occur at a higher frequency in patients with in-

tersex disorders.[20–22] Patients with gonadal dysgenesis and a Y chromosome should be recognized as early as possible and surgically explored; gonadectomy should be performed. The propensity for malignancy is earlier in the dyskinetic gonad than in the cryptorchid testes. Family members of patients with AIS should be screened, as this is an X-linked inherited disorder.

Eighty percent of gonadoblastomas and dysgerminomas arise in patients with dyskinetic gonads and cryptorchid testes.[22] The gonadoblastoma is a mixture of germ, Sertoli, Leydig, and granulsoa-type cells, and is a form of in situ cancer. Dysgerminomas can secrete testosterone and estradiol. These tumors are exceptionally radiosensitive, and the survival rate approaches 95%.[22] The prognosis in pure gonadoblastomas is good with bilateral gonadectomy.[22] Tumors of higher malignancy potential such as teratoma, embryonal carcinoma, choriocarcinoma, and endodermal sinus do occur in these patients.[22]

Intraabdominal and pelvic tumors occur less frequently in infants than in older children. The incidence is lower overall in the patient with normal development and ovarian function, but the frequency of certain histologic types is different than in the adult[23–25] (Table 13-2). The most common ovarian tumors in pediatric patients are from the teratoma-dermoid group (one-third), followed by epithelial tumors (cystadenomas).[23,24] Surgical management depends on the size, bilaterality, and the potential for malignant change. Low malignant-potential ovarian tumor (stage I) may occur in these patients and justify conservative surgery in the form of unilateral oophorectomy and associated other biopsies. The risk of developing malignancy in the remaining ovary is always uncertain.

The long-term consequences of radiation and combination chemotherapy on gonadal function are unknown. The prepubertal and adolescent ovary seems to be less susceptible to damage by radiation therapy than the older ovary, but premature ovarian failure does occur.[26,27] Normal ovarian function with normal childbearing does occur after therapy with irradiation and combination chemotherapy.[28] Several techniques are available to re-route the ovary out of the irradiated field.

Table 13-2. Ovarian Pathology in the Pediatric Group

Benign
Simple, follicular cysts
Cystic and hemorrhagic corpus luteal cysts
Endometrioma
Cystic teratoma
Polycystic ovarian disease (PCOD)
Cystadenoma
Mucinous cystadenoma
Sclerosing stromal tumor
Malignant
Germ-cell tumors
Dysgerminoma
Embryonal carcinoma
Teratoma
Endodermal Sinus
Choriocarcinoma
Sex Cord—Sex Mesenchyme Tumor
Granulosa Cell Tumor
Sertoli-Leydig Cell Tumor
Gynandroblastoma
Others
Burkitt's Lymphoma
Sarcomas

Tumors in the vagina occur infrequently, but as a rule of thumb are highly malignant. They are usually of the clear-cell adenocarcinoma or sarcoma botryoides variety. Surgery and chemotherapy are the usual modes of therapy available.

Dysfunctional Uterine Bleeding (DUB)

Dysfunctional uterine bleeding (DUB) is acyclic, excessive and/or prolonged bleeding from nonorganic causes. DUB is seen in ages 12–50; most often, however, it appears in the extreme ages of reproductive life, adolescence and perimenopause.

Before patients with abnormal uterine bleeding are treated, a number of potential causes should be included in the differential diagnosis (Table 13-3). The diagnosis of DUB remains one of exclusion.

The average blood loss during menses is 50 ml over 3- to 7-day period. DUB implies a deviation from the normal or average menstrual pattern, which may be established during adolescence and continued for the lifetime. The abnormal pattern may be daily spotting, menorrhagia, or menometrorrhagia (sometimes profuse and life-threatening), often ac-

Table 13-3. Dysfunctional Uterine Bleeding: Common Organic Etiologies

Cervical
Neoplasia
Cervicitis
Trauma
Uterine
Chronic PID
Polyps
Leiomyomata
Exogenous steroids/endocrine therapy
Neoplasia
Pregnancy (POC, decidua, Arias, stella)
H–P–O Dysfunction
PCOD (polycystic ovarian disease)
Drugs (antihypertensives)
Ovarian cysts, tumors
Miscellaneous
Bleeding dyscrasias
Hepatic—renal disease
Anticoagulant therapy
Thyroid disease

Table 13-4. Laboratory Studies for Dysfunctional Uterine Bleeding

CBC with differential
Platelet count
PT/PTT
Bleeding time
Urinalysis
FSH, LH
TSH, T_4
DHEAS, testosterone
HSG, hysteroscopy

companied by pelvic pain, backache, and chronic fatigue.

Studies of the pituitary gonadotropins and endocrine profiles (although limited) have been performed on patients with DUB, and these reveal a pattern compatible with a disturbance of ovulation in both the follicular and luteal phases of the cycle.[29–31] In one study of 5,575 cycles in 216 women over a 10-year period, 55% were anovulatory the first year past menarche. After 10 years, only 2.9% were anovulatory.[32]

The first step in managing DUB is to obtain a *complete* history with special attention to the menstrual history from menarche. Adolescent menstrual abnormalities are important, and may be the beginning of a poor prognosis for normal menstrual function in adulthood. A *complete physical examination* should always be performed. The external genitalia should be carefully inspected and the vagina examined for tears, lacerations, neoplasia, and vaginitis. The majority of adolescent patients with abnormal uterine bleeding have complications of pregnancy. Fifty percent of adolescents 15–19 years of age were sexually active in one report. Other infrequent causes of DUB include infectin, blood coagulopathies, hyperthyroidism, and polyps.

Thorough investigation for organic causes is imperative before any therapy is instituted (Table 13-4). Medical therapy (cyclic, endocrine) should be employed first in the adolescent with DUB. Any organic cause should be managed as indicated, and a diagnosis of DUB applied only when no treatable organic cause is found. Dilatation and curettage (D & C) should be the last alternative. Studies show that one-third of patients treated by D & C require more than one procedure and a large number will have hysterectomies at a young age. One must keep in mind, however, the possibility of associated minor unrelated pathology.

If bleeding continues, sharp curettage should be performed for therapeutic as well as diagnostic purposes. Seventy percent of patients will respond to curettage alone for 6 months or longer; 20% will require hormonal therapy, and 10% will fail on all therapy and require hysterectomy for recurrent persistent bleeding. For patients who desire pregnancy, ovulation induction is indicated once acute or prolonged bleeding episodes are controlled.

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References

1. Huffman JW. Gynecologic endocrine disorders in childhood and adolescence. Clin Obstet Gynecol. 1973; 16:51–81.

2. Carrington ER. Gynecologic problems in infants and prepubertal girls. Surg Clin N Am. 1954; 34:1615–1626.
3. Bongiovanni AM, Eberlein WR. Defective steroidal biogenesis in congenital adrenal hyperplasia. Pediatrics. 1958; 21:661–672.
4. Rosenfield RL, Miller WL. Congenital adrenal hyperplasia. In: Mahesh VB, Greenblatt RB, eds. Hirsutism and virilism. Boston: John Wright, 1983: p. 87–119.
5. Bongiovanni AM. Unusual steroid pattern in congenital adrenal hyperplasia: Deficiency of 3β-hydroxydehydrogenase. J Clin Endocrinol Metab. 1961; 21:860–862.
6. Capraro V. Surgical correction of genital anomalies. In: Sciarra JJ, McElin TW, eds. Gynecol and Obstetrics. Hagerstown: Harper and Row, 1981: Chapter 53, p. 1–12.
7. Farber M, Noumoff J, Freedman M, et al. Understanding and correcting genital anomalies. Contemp Obstet Gynecol. 1984; 24:113–129.
8. Evans TN, Poland ML, Boving RL. Vaginal malformations. Am J Obstet Gynecol. 1981; 141:910–920.
9. Fraser IS, Baird DT, Hubson BM, et al. Cyclical ovarian function in women with congenital absence of the uterus and vagina. J Clin Endocrinol Metab. 1973; 36:634–637.
10. Frank RT. Formation of an artificial vagina without operation. Am J Obstet Gynecol. 1938; 35:1053–1055.
11. MacIndoe A. The treatment of congenital absence and obliterative conditions of the vagina. Br J Plast Surg. 1950; 2:254–267.
12. Addison WA, Hammond CB. Successful management of vaginal agenesis—a team effort. Contemp Obstet Gynecol. 1981; 17:61–74.
13. Rock JA, Reeves LA, Retto H, et al. Success following vaginal creation for mullerian agenesis. Fertil Steril. 1983; 39:809–813.
14. Vasquez SB. Mullerian duct agenesis and other congenital anomalies. J Adolesc Health Care. 1982; 2:289–290.
15. David A, Carmil D, Bar-David E, et al. Congenital absence of the vagina. Clinical and psychologic aspects. Obstet Gynecol. 1975; 46:407–409.
16. Sarto GE. Cytogenetics of fifty patients with primary amenorrhea. Am J Obstet Gynecol. 1974; 119:14–23.
17. Sarto GE. Genetic disorders affecting genital development in 46 XY individuals (male pseudo hermaphroditism). Clin Obstet Gynecol. 1972; 15:183–202.
18. Morris JM, Mahesh VB. Further observations on the syndrome "testicular feminization." Am J Obstet Gynecol. 1963; 87:731.
19. Davidoff F, Federman DD. Mixed gonadal dysgenesis pediatrics 1973; 52:725–742.
20. Khalid AK, Bond AG, Ennis G, et al. Dysgerminoma—Gonadoblastoma and familial 46 XY pure gonadal dysgenesis: case report and review of the genetics and pathophysiology of gonadal dysgenesis and H-Y antigen. Aust NZ J Obstet Gynaecol. 1982; 22:175–179.
21. Schellhas HF, Trujillo JM, Rutledge FN, et al. Germ cell tumors associated with XY gonadal dysgenesis. Am J Obstet Gynecol. 1971; 109:1197–1203.
22. Scully RE. Gonadoblastoma. A review of 74 cases. Cancer. 1970; 25:1340–1355.
23. Heald FP. Ovarian tumors during adolescence. In: Heald FP, ed. Adolescent gynecology. Baltimore: Williams and Wilkins, 1966; p. 69–76.
24. Reis R, Koop C. Ovarian tumors in infants and children. J Pediatr. 1962; 60:96–102.
25. Morris JM. Functional tumors of the ovary. Clin Obstet Gynecol. 1961; 4:821–833.
26. Shalet SM. Effects of cancer chemotherapy on gonadal function of patients. Cancer Treat Rev. 1980; 7:141–152.
27. Stillman RJ, Schiff I, Schinfeld J. Reproductive and gonadal function in the female after therapy for childhood malignancy. Obstet Gynecol Surv. 1982; 37:385–393.
28. Horning SJ, Hoppe RT, Kaplan HS, et al. Female reproductive potential after treatment for Hodgkin's disease. N Engl J Med. 1981; 304:1377–1382.
29. Fraser IS, Michie EA, Wide L, et al. Pituitary gonadotropins and ovarian function in adolescent dysfunctional uterine bleeding. J Clin Endocrinol Metab. 1973; 37:407–414.
30. Southam AL, Richart RM. Prognosis for adolescents with menstrual abnormalities. Am J Obstet Gynecol. 1966; 94:637–645.
31. Southam AL. Metropathia hemorrhagica and nonpsychogenic amenorrhea. In: Heald FP, ed. Adolescent gynecology. Baltimore: Williams and Wilkins, 1966: p. 45–57.
32. Vollman RF. Patterns of menstrual performance in adolescent girls. Proc Second World Congr Fertil Steril, Naples, 1956, p. 27.

Surgery in the Aged 14

Daniel J. Polacek and Herbert J. Buchsbaum

The life expectancy of American women, which has increased dramatically during this century, is now 77 years. Women who are presently 65 years of age have an additional 18.4 years of life expectancy, and those who are 75 an additional 11.5 years. Individuals over 65 years of age, less than 4% of the population at the turn of the century, now account for over 11% of the total population. Americans over 85 years of age, of whom 69% are women, now constitute 1.1% of the population. By the year 2050 this age group is expected to constitute 5% of the population.[1] These demographic data indicate that a greater proportion of health care resources will be devoted to the care of elderly patients. It therefore becomes increasingly important to understand the unique medical and surgical needs and problems of this age group.

One problem in discussing the role of surgery in the health care of the elderly is the lack of definition for the term "elderly." In a review of 108 papers published in the medical literature since 1941 and dealing with operations in over 50,000 elderly patients, Linn and colleagues[2] found the lower limit for this category ranged from less than 60 to greater than 90 years of age.

Most of the elderly are active members of society. Nearly 55% of the individuals over 85 years of age are living independently, whereas only 23.2% reside in nursing homes. These patients present with a variety of medical complaints and findings ranging from annoying to life threatening. Urinary stress incontinence, which is irritating and socially disabling at any age, affects 5%–20% of the elderly living in the community and 40%–75% of those in nursing homes.[3] The incidence of more serious problems such as hypertension, diabetes, and cardiovascular disease increases dramatically with age. The elderly do have a disproportionate risk of developing cancer. Over 70% of the patients with vulvar, 40% with endometrial, 35% with ovarian, and 25% with cervical carcinoma are over the age of 65. The average age of patients treated for vulvar carcinoma is in the mid-seventies.[4]

Well into this century, operating on individuals over 50 years of age was considered risky. Katlic[5] cites a statement by Ochsner, who felt that ". . . an elective operation for inguinal hernia in a patient older than 50 years was not justified." In contrast, Katlic[5] recently reported six surgical procedures performed on centenarians. The relative proportion of elderly patients hospitalized for major operative procedures is greater than that of younger patients. Krauer[6] reported that although only 5% of the patients on a gynecologic unit were over 70 years of age, this group accounted for 8.9% of the major surgery. Since surgery may be "corrective, ablative or palliative in order to prolong life, restore function or alleviate pain," no procedure should be denied the elderly solely on the basis of age.[7]

While health care costs are covered for most elderly women, concern has been expressed about the increased cost of prolonged hospitalization for the elderly. A number of studies, both in this country and abroad, showed that the postoperative stay is *not* prolonged for the

elderly.[8–11] Since more extensive preoperative evaluation and therapy is often necessary for elderly patients, their preoperative hospitalization may be prolonged. In this era of disease-related groups (DRGs), every effort must be made not to deny quality care to the elderly who qualify for surgical management. Innovative measures must be devised to shorten the preoperative hospital phase so as not to compromise the preoperative evaluation.

Several structural and functional changes in each organ system are associated with the process of aging. These alterations in morphology and physiology may not be present in each elderly individual, but are characteristic of elderly groups when compared to younger control groups. These changes may not affect basal function; however, they often adversely affect the capacity of the organ system to respond to stress and reduce the margin of error. Consequently, it is important to understand changes in physiology that occur in the cardiovascular, pulmonary, renal, gastrointestinal, and immune systems with advancing age and the impact of these changes on surgical management.

Cardiovascular Physiology in the Elderly

Healthy elderly individuals exhibit normal cardiovascular function at rest; however, age-related changes in the cardiovascular system clearly lead to a diminished cardiovascular response to stress in the elderly (Table 14-1).[12] This is due to changes in cardiac output, vascular resistance, baroreceptor reactivity, and the response of the sympathetic nervous system.

Cardiac Function. Studies of resting cardiac output in the aged population have shown conflicting results. Brandfonbrener[13] measured resting cardiac output utilizing dye-dilution techniques and found a consistent decline in cardiac index of 0.79% per year beginning after the third decade of life. Cardiac index in this study changed from a mean of 3.72 liters/min/m^2 in the third decade of life to 2.36 liters/min/m^2 in the ninth decade. This occurred primarily as a result of a decrease in stroke index from 48.9 ml/beat/m^2 to 36.5 ml/beat/m^2. Another study by Strandell[14] showed a similar age-related decline in cardiac index in the supine position; however, the mean pulmonary artery pressures were lower in his aged population suggesting that a decreased venous return was responsible for his observed decrease in cardiac output. Furthermore, when cardiac index was measured at rest in the sitting position, there was no difference between the younger and elderly groups. A third study utilizing noninvasive techniques was unable to detect any age-related differences in stroke volume or cardiac output at rest in the supine position.[15] Both left-ventricular ejection time and isovolumetric relaxation are prolonged in elderly individuals. This may result from increased afterload rather than an intrinsic change in the myocardial inotropic state.[16,17]

Table 14-1. Age-Related Cardiovascular Changes[a]

Variable	Effect of age
Resting cardiac output	Heart rate unchanged
	(?) ↓ Stroke volume when supine
Exercise	↓ Maximal heart rate
	↓ Stroke volume
	↓ Ejection fraction
	Regional cardiac wall motion abnormalities
Peripheral vascular resistance	Increased
Sinus node function	Decreased maximal heart rate
Baroreceptor sensitivity	Decreased
Hypoxia	↓ Tachycardic response
Hypercarbia	Absent tachycardic response
Ventricular compliance	(?) Decreased

[a] From Gerstenblith G, Lakatta EG, Weisfeldt ML. Age changes in myocardial function and exercise response. *Progress in Cardiovascular Diseases* 1976; 19:1–21. By permission.

Although changes in resting cardiac function in elderly individuals are not impressive, there are clear differences in the cardiovascular response to stress in the elderly.

Advancing age is associated with exercise-induced changes in regional cardiac wall motion and ejection fraction. Port et al.[18] performed radionuclide angiography at rest and during exercise in healthy subjects 20–95 years of age measuring left-ventricular regional wall motion and ejection fraction. At rest, regional wall motion was normal in all age groups. During exercise, the frequency of wall motion abnormalities increased from 10% in the sixth decade to 44% by the eighth through tenth decades of life. At rest, there were no age-related differences in ejection fraction. In contrast, during exercise only 1 of 48 (0.2%) patients under the age of 60 had an ejection fraction less than 60%, compared to 13 of 29 (44%) subjects over the age of 60. Whereas only 4 of 48 (8%) subjects under the age of 60 had an increase in ejection fraction during exercise of less than the normal 5%, 24 of 29 (82%) elderly subjects had an ejection fraction increase of less than 5%, and 21 of these actually had a decrease in ejection fraction. These findings suggest that there may be an age-related decrease in cardiac contractility.

Sinus Node. During exercise, the maximal heart rate response is 18% lower in subjects over 50 years of age and 7% lower in subjects 35–49 years of age when compared to subjects less than 34 years of age.[19] Maximal cardiac output is depressed in the elderly group due to decreases in both stroke volume and heart rate. The decreased maximal heart rate response to stress may result from both structural changes in the sinoatrial node and decreased sensitivity to β-adrenergic stimuli. With advancing age the sinoatrial node exhibits an increase in fat, elastic tissue, and reticular fibers, all of which may adversely affect impulse generation.[20] The dose of isoproterenal required to increase the heart rate by 25 beats/min increases with age. This effect is probably due to decreased receptor sensitivity.[21]

Baroreceptor Function. The bradycardic response to acute elevations in arterial blood pressure, or baroreceptor reflex, is attenuated with age. Gribben et al.[22] measured the slowing of the pulse rate in response to blood pressure elevations following intravenous injection of phenylephrine. Plotting the pulse interval against blood pressure gives a linear relationship, the slope of which is a measure of baroreflex sensitivity. Gribben et al. found a progressive decrease in sensitivity with age. The baroreceptors, which lie within the arterial wall of the carotid sinus, respond to changes in vessel caliber with alterations in afferent nerve activity. The decreased elasticity and increased thickness of the major vessels, rather than age-related alterations in the receptors themselves, provide the most likely explanation for the depressed baroreceptor response in the aged population.

Hypoxia and Hypercarbia. Kronenberg and Drage[23] studied the heart rate response to hypoxia and hypercarbia in normal young men aged 23–30 and in elderly men aged 64–73 with no clinical evidence of cardiopulmonary disease. When alveolar oxygen was lowered from 100 to 40 mm Hg, the heart rate of the young men increased by 34% versus an increase of 12% in the elderly group. Similar but less pronounced changes in heart rate were evident when the alveolar carbon dioxide was raised to 55 mm Hg; the young men increased their heart rate by 15% while the elderly men decreased theirs by 1%. Although the exact mechanism of this response to hypoxia and hypercarbia is not known, alterations of chemoreceptor function, baroreceptor function, or response of the sympathetic nervous system function may all play a role. This attenuated response to hypoxia and hypercarbia has major clinical implications. First, it indicates that the elderly are less able to improve oxygen delivery to the tissues during periods of hypoxemia by increasing their heart rate and cardiac output. In addition, tachycardia, one of the early clinical signs of hypoxia, may be absent in elderly individuals.

Vascular Changes. Aging is associated with increased vessel wall thickness and reduced elasticity, and with increases in volume of the aorta.[24] The increased end-diastolic aortic volume increases impedance to ejection, because the stroke volume must be accelerated against larger inertial forces at the beginning of sys-

tole. The decreased elasticity leads to a loss of aortic recoil and thus diminishes the aortic contribution to forward flow. Similar morphologic changes develop in the walls of the peripheral vasculature, resulting in an increase in the systemic vascular resistance and the frequent finding of systolic hypertension in the elderly population. These changes in the aorta and systemic vasculature increase the work load of the left ventricle. Over time, one would expect a compensatory hypertrophy of the left ventricle, and echocardiographic data do show some age-related increases in left-ventricular posterior wall thickness.[25]

Although this mild cardiac hypertrophy associated with aging is not a universal finding, it suggests that the increased systemic vascular resistance in the elderly is one of the primary events leading to a decreased cardiac output from an increased afterload rather than a compensatory response. In addition, with cardiac hypertrophy we would expect to see a decrease in left-ventricular compliance. This may explain the frequent finding of an S_4 gallop (73%) in the elderly population, even in the absence of clinically evident cardiovascular disease.[26]

Surgical Implications. In summary, although resting cardiovascular function in healthy elderly individuals is not appreciably compromised, even healthy elderly women have a significant decrease in cardiovascular reserve. The cause of this diminished cardiovascular response to stress is multifactorial in origin. Maximal heart rate is decreased because of both structural changes in the sinoatrial node and decreased sensitivity to catecholamines. Stroke volume appears to be decreased in the supine position. Other experimental data suggest that decreases in contractility and ventricular compliance also occur as a result of the aging process.

A decrease in left-ventricular compliance will lead to increases in the pulmonary capillary wedge pressure for any given preload or left-ventricular end-diastolic volume. Consequently, higher absolute pulmonary capillary wedge pressure values in the elderly, when compared to younger patients, may be necessary to maintain preload and cardiac output. In addition, changes in the vascular system and in baroreceptor sensitivity contribute to the decreased cardiovascular response to stress. Although all these factors diminish cardiovascular reserve and the intraoperative margin of error, with meticulous management, age per se should not be a contraindication to surgery.

Pulmonary Physiology of the Aged

Aging of the pulmonary system is associated with changes in anatomy, static lung volumes, dynamic mechanical properties, gas exchange, and the control of ventilation (Table 14-2). These age-related changes lead to an overall decrease in baseline pulmonary function and in pulmonary reserve, and consequently increase the risk of hypoxia and hypercarbia in the elderly surgical patient.

Morphology. Several anatomic changes that occur in the lungs of elderly individuals appear to be attributable to aging per se. Although total lung capacity remains relatively constant throughout life, after 40 years of age the alveolar duct volume increases while alveolar volume decreases.[27] Total lung elastin content increases with age as a result of increased pleural, bronchial, and vascular elastin; however, the parenchymal connective tissue composition appears to remain the same.[28] Vascular changes in the aged include intimal thickening in pulmonary arteries less than 200 μm in diameter and in those greater than 3,000 μm in diameter. These changes are associated with medial hypertrophy of the pulmonary vessels.[29] Bronchial gland hypertrophy also appears to occur with aging independent of smoking history or environmental exposure.[30] The configuration of the chest changes with aging as a result of degenerative changes in the intervertebral discs. The normal thoracic kyphosis is accentuated leading to a progressive increase in the anterioposterior diameter of the chest and a decrease in height.[31]

Lung Mechanics. With senescence, the chest wall loses compliance while the abdominal wall becomes more compliant. Consequently, the diaphragmatic contribution to breathing in-

Table 14-2. Age-Related Pulmonary Changes

Variable	Effect of age
Anatomy	Intimal thickening and medial hypertrophy of the pulmonary vasculature
	Bronchial gland hypertrophy
	↑ AP diameter of the chest
Pulmonary mechanics	↓ Chest wall compliance
	↑ Lung compliance
	↓ Total lung–chest wall compliance
Lung volumes	↓ Forced vital capacity
	↓ Forced expiratory volume in 1 sec
	↑ Residual volume
	↑ Closing volume
Gas exchange	↓ Pa_{O_2}
	↓ Diffusing capacity
Control of ventilation	↓ Hypoxic and hypercapnic ventilatory drive

creases in importance. In young subjects, movement of the rib cage accounts for 40% of the volume change with each breath in the sitting position and diaphragmatic motion accounts for the remaining 60%. In elderly individuals, rib cage excursion accounts for only 30%.[32] Although the chest wall becomes stiffer with aging, the lung itself becomes more compliant. The elastic recoil of the lung, or the tendency of the lung to collapse, decreases with aging. This increase in the compliance of the lung is counterbalanced by a quantitively greater decrease in chest wall compliance, and as a consequence total work of breathing is slightly increased in the elderly.[33] The decrease in total lung–chest wall compliance may result in elevated peak inspiratory pressures during mechanical ventilation.

Lung Volumes. Although total lung capacity (TLC) remains constant throughout adult life, specific lung volumes show significant changes with aging (Figure 14-1). Vital capacity decreases by 24 ml/year in females after the age of 20, probably as a result of the loss of chest wall mobility with aging.[34,35] Associated with this decrease in vital capacity is an increase in residual volume (RV) with age. Residual volume, as a percentage of total lung capacity, remains relatively constant at about 20% until the mid-thirties, when the RV/TLC begins a steady rise to 40% at age 70.[36] Functional residual capacity, as a percent of total lung capacity, increases slightly with age while expiratory reserve volume shows a steady decrease.[37]

The loss of lung elastic recoil contributes to the age-related increase in closing capacity or closing volume. This value is a measurement of the volume at which airways in the dependent portions of the lung begin to collapse during resting tidal breathing. The closing volume exceeds functional residual capacity in patients above the age of 65 in the seated position, and in patients older than 44 years of age in the supine position, due to the positional decrease in functional residual capacity.[38] This is of major clinical importance since altered ventilation to perfusion ratios and subsequent hypoxia may occur, especially in the supine position.

Diminished elastic recoil of the lung also contributes to decreases in both the forced expiratory volume in 1 sec (FEV_1) and the maximal midexpiratory flow rate. In women above the age of 20, FEV_1 decreases by 25 ml/year and the maximal midexpiratory flow rate decreases by 30 ml/year.[34] Maximal voluntary ventilation (MVV) also declines with age (Figure 14-2).[37] This test is not only a gross measurement of intrinsic airway function but also reflects overall strength of the muscles of respiration, patient cooperation, and motivation. Declines in these indices of airway function, coupled with increases in the closing volume, lead to a significant reduction in pulmonary reserve and an increased risk of hypoxemia in elderly patients.

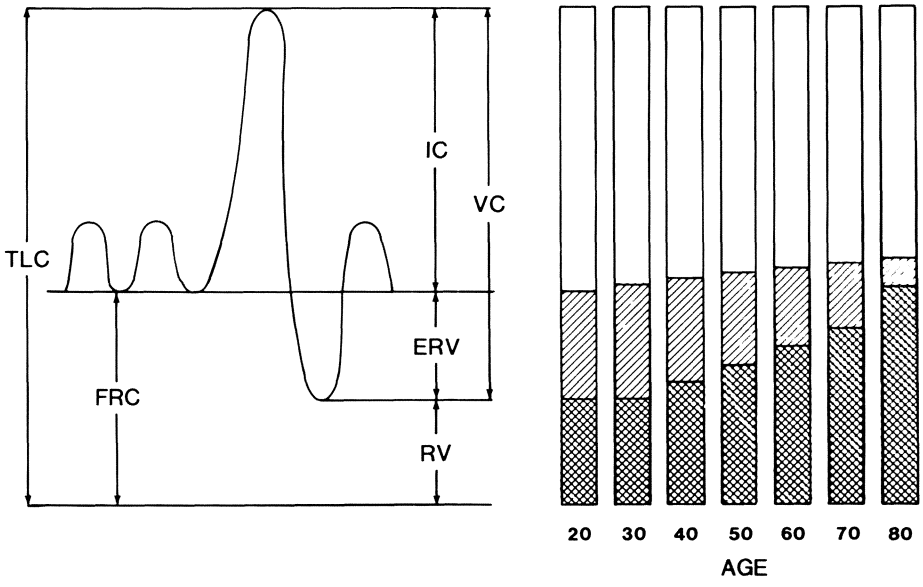


Figure 14-1. Changes in lung volumes with aging. Total lung capacity (*TLC*) remains constant. Vital capacity (*VC*), inspiratory capacity (*IC*), and expiratory reserve volume (*ERV*) decrease whereas functional residual capacity (*FRC*) and residual volume (*RV*) increase. (From data contained in References 34–37.)

Gas Exchange. The arterial P_{O_2} declines from a mean of 94 mm Hg in subjects less than 30 years old to a mean of 74 mm Hg in subjects over 60 years old when measured in the supine position.[39] In contrast, arterial pH and P_{CO_2} show no age-related changes (Figure14-2).[39] This age-related decrease in Pa_{O_2} is associated with decreases in both oxygen saturation and oxygen content. The relative hypoxemia of the elderly is probably due to not one but a constellation of age-related pulmonary changes. Carbon monoxide diffusing capacity decreases lin-

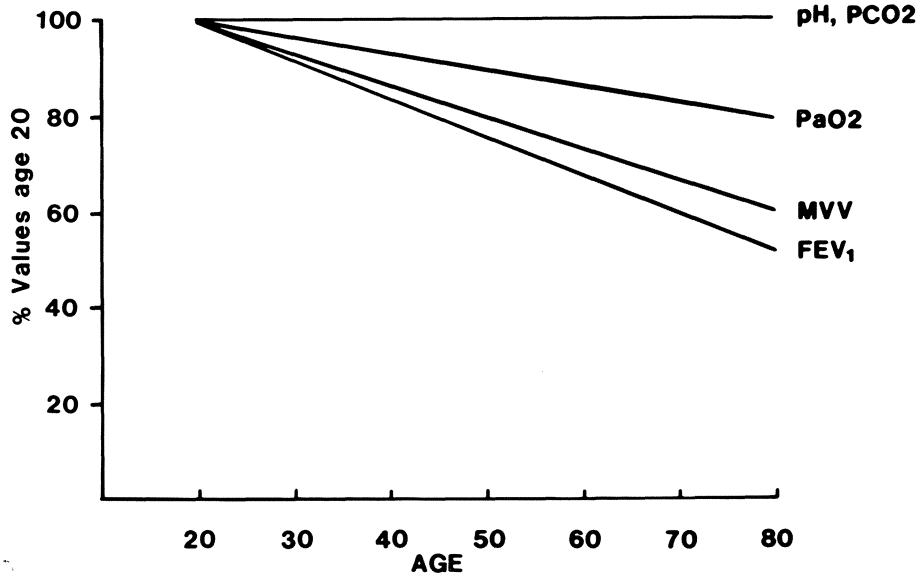


Figure 14-2. Age-related change in maximal voluntary ventilation (*MVV*), forced expiratory volume in 1 sec (FEV_1), and arterial pH, P_{CO_2}, and Pa_{O_2}. (From data in References 34, 37, 39.)

early with age by 0.06–0.18 ml/min/mm Hg per year for females.[40] Elderly people also have less uniform ventilation than younger adults during tidal breathing and relatively increased blood flow to the upper lung regions.[41] This imbalance in ventilation–perfusion relationships, which can cause hypoxemia, is probably caused by the loss of elastic recoil of the lungs and increases in closing volume that occur as a function of aging.

Control of Ventilation. Hypoxic and hypercapnic ventilatory drive diminishes with increasing age. When the alveolar oxygen is decreased to 40 mm Hg, young men increased their ventilation by 40 liters/min, while elderly males increase their ventilation by only 10 liters/min. When the alveolar carbon dioxide was elevated in both groups of patients, the young men increased their ventilation by 3.4 liters/min/mm Hg whereas the response in elderly males was 2.0 liters/min/mm Hg.[23] The change in pulmonary mechanics described above may explain part of this diminished ventilatory response to hypoxia and hypercarbia; however, age also appears to attenuate chemoreceptor function. This altered response is significant since hypoxia or hypercarbia in the elderly may not be associated with the usual clinical sign of tachypnea.

Surgical Implications. The decreased baseline Pa_{O_2} present in the elderly with its associated decrease in oxygen content increases the importance of maintaining preoperative and intraoperative hemoglobin levels of 10 gm/dl or greater. The elderly are more liable to develop intraoperative and postoperative hypoxemia, especially in the supine position, and yet they may not exhibit the usual clinical signs of hypoxemia such as tachycardia and tachypnea. For this reason, more frequent postoperative supplemental oxygen therapy and arterial blood gas monitoring is warranted. Incentive spirometry and early ambulation in the postoperative period are also vitally important. Bedridden elderly patients should have the head of the bed elevated at least 30°, if possible, to minimize the positional decrease in functional residual capacity.

Renal Physiology in the Elderly

The aging process leads to significant decreases in renal blood flow, glomerular filtration rate, and tubular function (Table 14-3). Despite these changes, elderly individuals are able to maintain relatively normal intravascular volumes and electrolyte concentrations in the basal state; however, the renal response to stress is blunted. Sodium excess or deprivation and excess acid, alkaline, or potassium loads are handled less quickly and not as completely in the elderly population compared to younger individuals.

Body Composition. Body composition undergoes significant changes with aging. There is a drop with aging in total body water (TBW) calculated as the percentage of body weight. In women 20–30 years of age, 51.2% of total body weight is water. In contrast, in females over the age of 60 the percentage of total body weight due to water decreases to 46.2%.[42] Although there are no age-related changes in blood volume, plasma volume, or extracellular fluid volumes, intracellular volume decreases with age. The decrease in TBW also reflects the loss of lean body mass with aging and a

Table 14-3. Age-Related Renal Changes

Variable	Effect of age
Anatomy	↓ Weight and volume of kidney ↓ Glomerular capillary surface area ↓ Tubular length
Glomerular filtration rate	Decreased
Tubular function	↓ Concentration and diluting capabilities Slower response to Na^+ flux Diminished capacity to excrete acid

relative increase in the proportion of fat, which contains comparatively less water per gram of fatty tissue.

Changes in TBW and total body fat content will affect the volume of distribution (V_d) of drugs that distribute into water spaces or fat, respectively. Diazepam, which is lipophilic, will have a larger V_d in the elderly, because of their increased total body fat content, than in the younger population. Although the loading dose must be increased to achieve a given blood level of diazepam because of the increased V_d, the dosage interval for this drug can be prolonged since its half-life increases in proportion to the increase in its V_d. Conversely, if the V_d decreases, the loading dose of the drug must be decreased and its dosing interval shortened.

Anatomic Changes. The total weight and volume of the kidneys decrease by 20–30% with age. This is due primarily to loss of cortical tissue with relative sparing of the medulla. Both the number and surface area of the glomeruli decrease with age. Subjects less than 36 years of age have 1.96×10^6 glomeruli in contrast to older subjects whose mean number of glomeruli dropped to 1.44×10^6. The overall glomerular capillary basement membrane surface area becomes smaller.[43] Proximal tubular volume shows a similar decline, which parallels the changes in glomerular size.[44]

Many of the anatomic changes in the kidney are thought to be secondary to changes in the renal vasculature. Intimal proliferation, medial hypertrophy, and hyalinization of the renal arterial vessels are more common in the elderly. Reduplication and focal thickening in the basement membrane develop in both the glomeruli and tubules.[44] Absolute renal blood flow and blood flow per gram of tissue significantly diminish after the age of 50 as a result of these morphologic changes. Vascular reactivity of the kidney also changes with age. The vasodilatory response to acetylcholine or sodium loading is decreased in the elderly; however, the vasoconstrictive response to angiotension remains intact.[45]

Glomerular Filtration. Perhaps the most important renal change with senescence is the decrease in glomerular filtration rate. There is a progressive linear decline in creatinine clearance from 140 ml/min/1.73 m^2 at age 30 to 97 ml/min/1.73 m^2 at age 80.[46] The minimal increase in serum creatinine levels from 0.81 to 0.84 during this time period reflects the concurrent decrease in muscle mass with aging. The decreased glomerular filtration rate in the elderly is of major clinical importance, as many commonly used drugs including digoxin, procainamide, the aminoglycoside antibiotics, and penicillin are primarily excreted through the kidney. A complete list of medications whose dose must be altered in patients with renal insufficiency is reviewed elsewhere.[47] Since the serum creatinine remains within the normal range as the creatinine clearance and lean body mass decrease in parallel, serum creatinine cannot be used as an accurate guide to renal function in the elderly woman. Ideally, creatinine clearance should be measured. The following formula[48] is helpful for calculating creatinine clearance (ml/min) from serum creatinine alone:

$$\left[\frac{(140 - \text{age})\ (\text{weight in kg})}{72\ (\text{serum creatinine in mg/100 ml})}\right] - 15\%$$

Tubular Function. Alterations in kidney tubule function with aging lead to deficits in concentration and dilution of urine, sodium conservation, and acid excretion. Following a 12-hr dehydration test, subjects 20–39 years of age changed their urine osmolality from 969 to 1109 mOsm/kg versus a change from 832 to 882 mOsm/kg in subjects older than 60 years.[49] In this study, urine flow decreased from 1.02 to 0.49 ml/min in the younger group whereas the elderly subjects had no change in total urine flow. This decrease in concentrating ability did not correlate with creatinine clearance. The impairment in urinary concentration may result from the age-related relative increase in medullary blood flow causing decreased medullary tonicity and a subsequent decline in the efficiency of the countercurrent system.

A defect in arginine vasopressin (antidiuretic hormone) release does not appear to contribute to the decreased ability to concentrate urine. In fact, elderly subjects have a heightened sensitivity to hyperosmolality. When the

serum osmolality was raised to 306 mOsm/kg in two groups of young (22–48 years) and old (52–66 years) subjects by infusing hypertonic saline, the elderly patients increased serum arginine vasopressin levels 4.5 times the baseline value, whereas the younger men elevated serum arginine vasopressin levels 2.5 times the baseline.[50] Age-related decreases in free water clearance and maximum dilution are closely related to falls in the glomerular filtration rate and the decrease in functioning glomeruli.[51]

The ability of the aged kidney to achieve sodium balance remains intact. However, the rapidity of response to sodium depletion is slowed. The half-time for the reduction in renal sodium excretion in subjects placed on a 10-meq sodium diet was 17.6 hr for those under 30 years of age, 23.4 hr in the 30–59 age group, and 30.9 hr in subjects over the age of 60.[52] Age-related decreases in renin and concomitant decreases in aldosterone may play a role in the diminished speed of sodium retention.[53] The relatively lower levels of aldosterone may also predispose the elderly to the development of hyperkalemia. This is especially true since the diminished glomerular filtration rate of the elderly will also limit potassium excretion. The duration of hyperkalemia associated with acidosis may be prolonged, since the capacity to excrete an acid load is diminished in the elderly due to a reduction in ammonium excretion.[54] This is another example of decreased renal tubular function in the elderly.

Surgical Implications. The age-related changes in glomerular filtration and tubular function predispose elderly patients to derangements in fluid and electrolyte balances. They cannot respond as quickly or as completely as younger patients to volume and electrolyte shifts that occur during surgery or as a result of physician-mediated fluid therapy. Drug therapy in this population is complicated by changes in total body water, fat, and lean body mass, as well as by the decreased renal excretion of a host of medications. In addition, the usual indices of renal function including serum creatinine, specific gravity, and urine output are less reliable aids in evaluating intravascular volume status and renal function.

Physiology of the Digestive System in the Aged

Gastrointestinal and hepatobiliary diseases are a significant source of morbidity and mortality in the elderly population; however, information concerning age-related changes in these systems is sparse. This is due, in part, to the immense functional reserve of the hepatic and gastrointestinal systems and to the difficulty in separating changes resulting from systemic disease from age-related changes. Nevertheless, several pathophysiologic changes occur more frequently in elderly women, and their consequences are more severe.

Esophagus. Studies of esophageal function in the elderly have not separated changes related to aging per se from changes occurring as a result of concurrent systemic disease.[55] An increased incidence of abnormal peristalsis and tertiary contractions and inadequate relaxation of the lower esophageal sphincter have been reported. In contrast, a manometric study of normal males over the age of 80 failed to detect any abnormalities of esophageal motility, although a marked decline in the amplitude of peristaltic waves was present.[56] This finding is compatible with a decrease in the mass of esophageal muscle, which parallels the overall loss of lean body mass with aging. The incidence of hiatus hernia increases with age from 14% in women under the age of 30 to 58% in women over 60 years of age.[57] Hiatus hernia increases the risk of aspiration during intubation.

Stomach. The incidence of atrophic gastritis, characterized by thinning of the gastric mucosa and muscular wall, loss of parietal cells, and goblet-cell metaplasia, increases with age. Atrophic gastritis is associated with delayed gastric emptying and decreased hydrochloric acid secretion. However, age per se does not appear to affect gastric motility.[58] Atrophic gastritis has also been associated with pernicious anemia and an increased incidence of dyspepsia, gastric ulceration, and gastric carcinoma. Absorption of vitamin B_{12} and iron may also be decreased.

Intestine. With aging, there is a reduction in villous height and an increase in the breadth of

villi in the small intestine. This results in a significant reduction in surface area.[59] Nevertheless, there is no conclusive evidence that these morphologic changes cause clinically significant malabsorption in healthy elderly women. Age-related changes in the colon appear to be limited to an increased prevalence of diverticuli and increased tortuousity of the blood supply. In women less than 60 years of age, diverticuli are present in 8% compared to an incidence of 39% in the older age groups.[60]

Liver. Studies in experimental animals have shown reduced liver microsomal drug-metabolizing enzymes with increasing age. Hepatic clearance of drugs is dependent on both liver blood flow and intrinsic enzymatic activity. Liver blood flow decreases with age. Conclusive evidence that age itself adversely affects liver enzymatic activity is not available in human studies.[55] Consequently, the metabolism of drugs with low hepatic clearance does not appear to be affected by age. For drugs with high hepatic clearance, such as lidocaine, liver blood flow is the limiting step in their metabolism, and the age-related decrease in blood flow will necessitate decreases in the dose requirement.[61]

Surgical Implications. The increased frequency of hiatal hernia and atrophic gastritis, with its associated decrease in gastric motility, increases the risk of aspiration in the elderly population. Although age-related change in hepatic drug metabolism is poorly defined, in most instances it is prudent to initiate therapy with lower doses of hepatically metabolized drugs in the elderly.

Immune Capacity

Linn and Jensen[62] compared the immune status of patients averaging 66 ± 6 years of age with patients aged 48 ± 10 years before and after inguinal hernia repair. Lymphocyte blastogenic responses to PHA and Con A were similar in both groups prior to surgery. Five days after surgery, the responses to PHA and Con A were depressed in the elderly patients but remained normal in the younger patients. These findings suggest that the T-cell response to stress is depressed in older patients.

Surgical Morbidity and Mortality

Factors that are thought to conribute to morbidity and mortality in surgery in the aged include concurrent medical diseases, age, type of surgery, elective versus emergent surgery, type of anesthesia, and operating time.

Medical Conditions. Elderly patients presenting for gynecologic surgery frequently have significant medical problems. Pierson et al.[9] performed a case-control study to identify the incidence of medical disease in women over 75 (mean, 79.9 years) undergoing surgery for gynecologic malignancy. The control group contained women under 55 (mean, 47.6 years) undergoing identical procedures for similar indications. The incidence of major medical problems was 76% in the elderly group and only 28% in the control group. The most common problems of the elderly were hypertension (52%), arteriosclerotic heart disease (38%), and diabetes (20%). In the control group, the most common problem was varicose veins. Other studies report similar results.[11,63]

Operative risk rises in proportion to the number and severity of medical problems.[64,65] The risk of surgery may not be justified if the patient's medical status is associated with a more dismal prognosis than the underlying surgical problem itself.

Age. Early studies in the surgical literature reporting higher-than-normal surgical risk in the elderly often did not separate morbidity secondary to coexistent medical problems from that due to age itself.[66] More recent studies fail to demonstrate an unequivocal association between age and surgical risk.[67,68] Panayiotis and co-workers[8] reported no increase in the postoperative complication rate among patients over 65 when compared to a control group aged 40–55 years. Pierson et al.[9] reported a postoperative complication rate of 1.12 per patient in a study group averaging 79.2 years of age and 1.04 per patient in a control group averaging 47.6 years among patients undergo-

ing operative procedures for gynecologic malignancy. The major procedures ranged from celiotomy with biopsy to radical vulvectomy with groin dissection and pelvic exenteration.

Type of Surgery. The risk of transabdominal surgery is greater than that of vaginal or vulvar surgery. Marshall and Fahey[69] reported a mortality of 16.4% in extraabdominal and 28.7% in intraabdominal general surgical procedures in patients over 80 years of age. The percentage of vaginal procedures in the elderly undergoing gynecologic surgery ranges from 54% to 85%. [8,10,63,70] The balance are abdominal procedures done for suspected or proven malignancy. Paldi and co-workers[70] reported that the incidence of benign and malignant tumors was equal in their series (15%). O'Leary and Symmonds,[71] reviewing the experience at the Mayo Clinic in radical surgery in 133 patients over 65 years of age, reported that radical vulvectomy was the most frequently performed procedure (43%), followed by radical hysterectomy (26%). While the indications for surgery usually dictate the route of surgery, the vaginal route should be used preferentially when there is a choice.

Elective versus Emergency Surgery. Mortality is greater in emergency than in elective procedures. Underlying pathology contributes to the three- to five-fold higher mortality associated with emergency surgery in the aged.[67,69,72] There is rarely an occasion for emergency gynecologic surgery in the elderly. In Tancer and Matseoane's series,[63] one finds a difference in postoperative complications in "indicated" and "elective" procedures of 33% versus 19%, respectively. Malignancies that increase the risk for certain postoperative complications, for example, pulmonary embolism, contributed to this difference. In truly emergent procedures, the patient's preoperative evaluation is compromised, and optimal facilities for intra- and postoperative monitoring may not be available.

Anesthesia. Many anesthesiologists prefer conduction anesthesia for elderly women. In Ellenbogen and co-workers' series[11] of women over the age of 65 undergoing vaginal hysterectomy, 85.8% had conduction anesthesia (77.8% spinal and 7.8% epidural). Similarly, Panayiotis and colleagues,[8] in a case-controlled study, found 67.4% of the elderly had conduction anesthesia for 25 abdominal and 135 vaginal procedures. In the control group of younger women with similar operations, 82.5% had general anesthesia and only 17.5% had conduction anesthesia. In the case-control study by Pearson et al.,[9] 36% of 25 women over 75 years of age undergoing major abdominal operations for gynecologic cancer had general anesthesia and 64% had conduction anesthesia. In the control group, 92% had general anesthesia and only 8% had conduction anesthesia. McKeithen,[10] reporting the experience of a St. Petersburg, Florida hospital in 185 gynecologic operations performed on women over 65 years of age, found that 98% of the patients had general anesthesia for 112 vaginal and 73 abdominal operations.

There is no inherent reason for choosing conduction anesthesia over general inhalation anesthesia in abdominal surgery in the aged. General anesthesia, both during induction and maintenance, may adversely affect cardiac function. However, conduction anesthesia may be associated with changes in systemic vascular resistance, which can have dire consequences in the elderly. Anesthetic management must be based on the patient's overall medical status rather than age itself.

Operating Time. Most investigators feel that length of operation is not related to morbidity or mortality in the aged.[69,72] There is therefore no reason to hurry through a procedure on the elderly. In fact, when operating time in women over 75 years of age is compared with that in women under 55 years of age, the operating time is slightly shorter for the elderly women.[9]

Preoperative Evaluation

The preoperative evaluation in the elderly should follow the protocol outlined in Chapter 1. The elderly may suffer from dementia and impaired hearing and vision. These deficits may limit communication and effective history-taking.[3] Nevertheless, the physician must

carefully screen patients for a history of cardiovascular, pulmonary, and renal disease and obtain a complete history of medication ingestion. All elderly patients should have an electrocardiogram, chest x-ray, complete blood count with red cell indices, serum electrolytes, blood urea nitrogen, and creatinine. Magnesium levels should be obtained if the patient is receiving diuretics. We feel that room air blood gasses should be determined preoperatively in all elderly patients. Patients with a history of pulmonary disease or abnormal blood gasses should have pulmonary function studies performed.

Great care must be taken in explaining the indications and risks of the planned operative procedure. Geriatric patients in general show significantly poorer comprehension of consent information and therefore have some impairment in their competency to give informed consent.[73] They also may have impaired memory and cognitive abilities. These individuals frequently need a longer period of time to recover full mental function following anesthesia and surgery.[74]

We routinely use prophylactic antibiotic and heparin therapy in the elderly. Cefoxitin is administered preoperatively, intraoperatively, and postoperatively for a total of three doses. Nephrotoxic drugs such as aminoglycosides and vancomycin may be used with caution so long as peak and trough drug levels are closely monitored. Five thousand units of subcutaneous hepain is administered 2 hr prior to surgery and then every 12 hr postoperatively until the patient is ambulatory.

The elderly need to be carefully monitored intra- and postoperatively as described in Chapter 10. If the above precautions are taken, surgery can be made safe for the elderly, and none need to be denied surgery for life-threatening conditions or procedures that improve the quality of life solely on the basis of age.

References

1. 1980 Census of Population: General Population Characteristics, United States. U.S. Census Bureau, 1983.
2. Linn BS, Linn MW, Wallen N. Evaluation of results of surgical procedures in the elderly. Ann Surg. 1982; 195:90–96.
3. Rowe JW. Health care of the elderly. N Engl J Med. 1985; 312:827–835.
4. Lifshitz S, Savage JE, Yates SJ, Buchsbaum HJ. Primary epidermoid carcinoma of the vulva. Surg Gynecol Obstet. 1982; 155:59–61.
5. Katlic MR. Surgery in centenarians. JAMA. 1985; 253:3139–3141.
6. Krauer F. Surgery in the postmenopausal woman. In: Buchsbaum HJ, ed. The menopause. New York: Springer-Verlag, 1983: chapter 10.
7. Glenn F. Pre- and postoperative management of elderly surgical patients. J Am Geriat Soc. 1973; 21:385–393.
8. Panayiotis G, Ellenbogen A, Grunstein S. Major gynecologic surgical procedures in the aged. J Am Geriat Soc. 1978; 26:459–462.
9. Pierson RL, Figge PK, Buchsbaum HJ. Surgery for gynecologic malignancy in the aged. Obstet Gynecol. 1975; 46:523–527.
10. McKeithen WS. Major gynecologic surgery in the elderly female 65 years of age and older. Am J Obstet Gynecol. 1975; 123:59–63.
11. Ellenbogen A, Agranat A, Grunstein S. The role of vaginal hysterectomy in the aged woman. J Am Geriatr Soc. 1981; 29:426–428.
12. Gerstenblith G, Lakatta EG, Weisfeldt ML. Age changes in myocardial function and exercise response. Prog Cardiovascular Dis. 1976; 19:1–21.
13. Brandfonbrener M, Landowne M, Shock NW. Changes in cardiac output with age. Circulation. 1955; 12:557–566.
14. Strandel T. Circulatory studies on healthy old men. Acta Med Scand. 1964; 175 (Suppl. 414):1–44.
15. Proper R, Wall F. Left ventricular stroke volume measurements not affected by chronologic aging. Am Heart J. 1972; 83:843–845.
16. Willems JL, Roelandt J, DeGeest H, et al. The left ventricular ejection time in elderly subjects. Circulation. 1970; 42:37–42.
17. Harrison TR, Dixon K, Russell RO, et al. The relation of age to the duration of contraction, ejection, and relaxation of the normal human heart. Am Heart J. 1964; 67:189–199.
18. Port S, Cobb FR, Coleman RE. et al. Effect of age on the response of the left ventricular ejection fraction to exercise. New Engl J Med. 1980; 303:1133–1137.
19. Julius S, Amery A, Whitlock LS. et al. Influence of age on the hemodynamic response to exercise. Circulation. 1967; 36:222–230.
20. Lev M. Aging changes in the human sinoatrial node. J Gerontol. 1954; 9:1–19.
21. Vestal RE, Wood AJJ, Shand DG. Reduced B-

Adrenoceptor sensitivity in the elderly. Clin Pharmacol Ther. 1979; 26:181–186.
22. Gribbin B, Pickering TG, Sleight P, et al. Effect of age and high blood pressure on baroreflex sensitivity in man. Circ Res. 1971; 29:424–431.
23. Kronenberg RS, Drage CW. Attenuation of the ventilatory and heart rate responses to hypoxia and hypercarbia with aging in normal men. J Clin Invest. 1973; 52:1812–1819.
24. Lindenfeld J, Groves BM. Cardiovascular function and disease in the aged. In: Schrier RW, ed. Clinical internal medicine in the aged. Philadelphia: WB Saunders, 1982: 87–123.
25. Gerstenblith G, Frederiksen J, Yin FCP, et al. Echocardiographic assessment of a normal adult aging population. Circulation. 1977; 56:273–278.
26. Spodick DH, Quarry VM. Prevalence of the fourth heart sound by phonocardiography in the absence of cardiac disease. Am Heart J. 1974; 87:11–14.
27. Weibel ER. Morphometrics of the lung. In: Fenn WO, Raln H, eds. Handbook of physiology. Respiration. Sec. 3, Vol. 1. Washington, D.C.: American Physiologic Society, 1964: Chapter 7, 285–306.
28. Pierce JA, Ebert RV. Fibrous network of the lung and its change with age. Thorax. 1965; 20:469–476.
29. Semmens M. The pulmonary artery in the normal aged lung. Br J Dis Chest. 1970; 64:65–72.
30. Hernandez JA, Anderson AE, Holmes WL, et al. The bronchial glands in aging. J Am Geriatr Soc. 1965; 13:799–803.
31. Pierce JA, Ebert RV. The barrel deformity of the chest, the senile lung and obstructive pulmonary emphysema. Am J Med. 1958; 25:13–22.
32. Rizzato G, Marazzini L. Thoracoabdominal mechanics in elderly men. J Appl Physiol. 1970; 28:457–460.
33. Turner JM, Mead J, Wohl ME. Elasticity of human lungs in relation to age. J Appl Physiol. 1968; 25:664–671.
34. Morris JF, Koski A, Johnson LC. Spirometric standards for healthy nonsmoking adults. Am Rev Respir Dis. 1971; 103:57–67.
35. Cherbotarev DF, Korkushko OV, Ivanov LA. Mechanisms of hypoxemia in the elderly. J Gerontol. 1974; 29:393–400.
36. Brody AW, Johnson JR, Townley RG, et al. The residual volume. Predicted values as a function of age. Am Rev Respir Dis. 1974; 109:98–105.
37. Grimby G, Soderholm B. Spirometric studies in normal subjects III. Static lung volumes and maximum voluntary ventilation in adults with a note on physical fitness. Acta Med Scand. 1963; 173:199–206.
38. Leblanc P, Ruff F, Milic-Emili J. Effects of age and body position on airway closure in man. J Appl Physiol. 1970; 28:448–451.
39. Sorbini CA, Grassi V, Solinas E, et al. Arterial oxygen tension in relation to age in healthy subjects. Respiration. 1968; 25:3–13.
40. Muiesan G, Sorbini CA, Grassi V. Respiratory function in the aged. Bull Physiopathol Respir. 1971; 7:973–1009.
41. Holland J, Milic-Emili J, Macklem PT, et al. Regional distribution of pulmonary ventilation and perfusion in elderly subjects. J Clin Invest. 1968; 47:81–92.
42. Edelman IS, Shukla KK, Cohn SH, et al. Further observations in total body water. I. Normal values through the life span. Surg Gynecol Obstet. 1952; 95:1–12.
43. Dunnill MS, Halley W. Some observations on the quantitative anatomy of the kidney. J Pathol. 1973; 110:113–121.
44. Darmady EM, Offer J, Woodhouse MA. The parameters of the aging kidney. J Pathol. 1973; 109:195–207.
45. Hollenberg NK, Adams DF, Solomon HS, et al. Senescence and the renal vasculature in normal man. Circ Res. 1974; 34:309–316.
46. Rowe JW, Andres R, Tobin JD, et al. The effect of age on creatinine clearance in men: a cross-sectional and longitudinal study. J Gerontol. 1976; 31:155–163.
47. Bennett WM, Muther RS, Parker RA, et al. Drug therapy in renal failure: dosing guidelines for adults. Ann Intern Med. 1980; 93:62–89, 286–325.
48. Cockroft DW, Gault MH. Prediction of creatinine clearance from serum creatinine. Nephron. 1976; 16:31–41.
49. Rowe JW, Shock NW, DeFronzo RA. The influence of age on the renal response to water deprivation in man. Nephron. 1976; 17:270–278.
50. Helderman JH, Vestal RE, Rowe JW. The response of arginine vasopressin to intravenous ethanol and hypertonic saline in man: the impact of aging. J Gerontol. 1978; 33:39–47.
51. Lindeman RD, Lee TD, Yiengst MJ, et al. Influence of age, renal disease, hypertension, diuretics and calcium on the antidiuretic responses to suboptimal infusions of vasopressin. J Lab Clin Med. 1966; 68:206–223.
52. Epstein M, Hollenberg NK. Age as a determinant of renal sodium conservation in normal man. J Lab Clin Med. 1976; 87:411–417.
53. Weidmann P, DeMyttenaere-Bursztein S, Maxwell MH, et al. Effect of aging on plasma renin and aldosterone in normal man. Kidney Int. 1975; 8:325–333.

54. Agarwal BN, Cabebe FG. Renal acidification in elderly subjects. Nephron. 1980; 26:291–295.
55. Vierling JM, Reichen J. Physiology and diseases of the digestive system in the aged. In: Schrier RW, ed. Clinical internal medicine of the aged. Philadelphia: WB Saunders, 1982: p. 149–166.
56. Hollis JB, Casstell DO. Esophageal function in elderly men: A new look at "presbyesophagus." Ann Intern Med. 1974; 80:371–374.
57. Pride RB. Incidence and coincidence of hiatus hernia. Gut. 1966; 7:188–189.
58. Davies WT, Kirkpatrick JR, Owen GM, et a . Gastric emptying in atrophic gastritis and carc - noma of the stomach. Scand J Gastroentero.. 1971; 6:297–301.
59. Warren PM, Pepperman MA, Montgomery RD. Age changes in small intestinal mucosa. Lancet. 1978; 2:849–850.
60. Manousos ON, Truelove SC, Lumsden K. Prevalence of colonic diverticulosis in the general population of the Oxford area. Br Med J. 1967; 3:762–763.
61. Gerber JG. Drug usage in the elderly. In: Schrier RW, ed. Clinical internal medicine in the aged. Philadelphia: WB Saunders, 1982: p. 51–65.
62. Linn BS, Jensen J. Age and immune response to a surgical stress. Arch Surg. 1983; 118:405–409.
63. Tancer ML, Matseoane SL. Gynecologic surgery in patients over 65. Geriatrics. 1966; 21:189–196.
64. Hirsh RA. An approach to assessing perioperative risk. In: Goldman DR, Brown FH, Levy WK, et al., eds. Medical care of the surgical patient. Philadelphia: JB Lippincott, 1982: p. 31–39.
65. Goldman L. Cardiac risks and complications of noncardiac surgery. Ann Intern Med. 1983; 98:504–513.
66. Johnson JC. Surgery in the elderly. In: Goldman DR, Brown FH, Levy WK, et al., eds. Medical care of the surgical patient. Philadelphia: JB Lippincott, 1982: p. 578–590.
67. Blake R, Lynn J. Emergency abdominal surgery in the aged. Br J Surg. 1976; 63:956–960.
68. Robbins RE, Budden MK. Major abdominal surgery in patients over 70 years of age: Results during 1962 to 1966 compared with those during 1950 to 1959. Can J Surg. 1972; 15:73–78.
69. Marshall WH, Fahey PJ. Operative complications and mortality in patients over 80 years of age. Arch Surg. 1964; 88:896–904.
70. Paldi E, Peretz A, Pascal B. Gynecologic surgery in geriatric patients. Geriatrics. 1966; 21:131–136.
71. O'Leary JA, Symmonds RE. Radical pelvic operations in the geriatric patient. Obstet Gynecol. 1966; 28:745–753.
72. Young HD, Tanga MR, Wellington JL. Major abdominal surgery in the elderly: A review of 172 consecutive patients. Can J Surg. 1971; 14:324–327.
73. Stanley B, Guido J, Stanley M, et al. The elderly patient and informed consent. JAMA. 1984; 252:1302–1306.
74. Blundell E. A psychological study of the effects of surgery on eighty-six elderly patients. Br J Soc Clin Psychol. 1967; 6:297–303.

The Role of Surgical Drains 15

Hans-B. Krebs and B. Frederick Helmkamp

Historical Notes

The earliest recorded use of surgical drainage was by Hippocrates (400 B.C.), who used cannulas to treat empyema.[1] Claudius Galen (200 B.C.), whose teachings were held infallible for the next 1500 years, described tubes for the management of ascites.[2]

Metal tubes, and, subsequently, glass were used throughout the centuries until Chassaignac in 1859 introduced soft rubber tubes. In spite of this innovation, gauze drains remained popular for several decades. These drains, after remaining in the wound for 3–4 days, were painful to remove, and occasionally portions of omentum or loops of the small intestine would push alongside the drain onto the abdominal wall.[2] Kehrer, in 1882, solved this problem by placing gauze within a rubber sheath, thus creating the first "cigarette drain."[3] In the 1880s, Mikulicz[4] popularized a drain or tampon that he constructed by placing a fenestrated sheet of rubber in the area to be drained and filling it with strips of gauze. Penrose, in 1897, cut the end off a condom and filled it with gauze.

The last innovation in drainage in the 19th century was a forerunner of the present sump-type drain and was described by Kellogg in 1895.[3] Heaton, in 1898, was the first to apply water activated suction to a sump tube, which he constructed by placing a smaller perforated catheter inside a glass drain.

In 1905, Yates,[5] in one of the classic papers of the surgical literature, proposed the concept of both prophylactic and therapeutic drainage. He recommended the selection of soft, less irritating drains as well as their early removal to reduce complications. The late 1920s witnessed the final demise of gauze capillary drainage, although its use persisted in rubber sheaths as cigarette drains.[2] The use of suction drainage and sump drains increased; commercial sump drains were introduced by Chaffin in 1932.[2,6] Further improvements in drainage were made by Murphy[7] who applied intermittent suction with a syringe attached to a catheter. Three years later, Barron publicized continuous suction in closed drainage systems.[8] The units were cumbersome and required constant supervision to prevent retrograde infection from the suction bottles.[2] Finally, Redon and Jost developed a portable closed suction unit in 1954.

Indications for Drainage

Drains are used therapeutically to aid the egress of fluid from an abscess, hematoma, or a fecal fistula. The prophylactic use of drains to inhibit fluid accumulation (subcutaneous space; "wet" surgical field) is controversial. Proponents will argue that prophylactic use of drains will decrease the rate of postoperative infection. Opponents claim that such drains facilitate migration of bacteria into the wound and result in a higher wound infection rate.

Both Cruse and Foord[9] and Higson and Ketlewell[10] showed the lowest wound infection rate in those wounds that had *no* drains. Since patients who had drains and those who had no

drains were not randomized, it is possible that drains were used in a population at high risk for wound infection (e.g., obesity, diabetes, and advanced age), whereas patients at low risk had no drains. Therefore, it remains unclear from these studies if wound drains increase wound infection.

Theoretic considerations would support prophylactic wound drainage. Alexander et al.[11] found that after 24 hr the complement level in wound fluid falls so that opsonization of bacteria is impaired. Removal of stagnant wound fluid with closed suction drainage would allow fresh fluid with opsonins to enter the wound. Altemeyer[12] and McIlreth et al.[13] have shown that removal of accumulating tissue fluid and blood by suction tubes will expedite the healing process. Swartz and Tenaree[14] decreased febrile morbidity using a T tube for retroperitoneal drainage of the pelvis following abdominal and vaginal hysterectomy. They suggested drainage as an alternative to antibiotic administration.

Most authors[15–19] agree that there is no role for abdominal drainage in generalized peritonitis, as, for example, from a ruptured tuboovarian abscess. Yates[5] demonstrated 80 years ago that it is physically and physiologically impossible to drain the entire peritoneal cavity, since local drain encapsulation occurs within hours. By contrast, well defined cavities, such as the pelvis, lend themselves readily to drainage.

Types of Drains

Drains may be either passive or active (Table 15-1). Passive drains function primarily by overflow; they are assisted occasionally by gravity or capillary action. Active drains are connected to suction. The indications for each type of drain are discussed in the following sections.

Table 5-1. Types of Surgical Drains

Passive	
Penrose	
Foley	
Malecot	
Active	
Open:	Sump
Closed:	ReliaVac
	HemoVac
	Jackson–Pratt
	T-tube

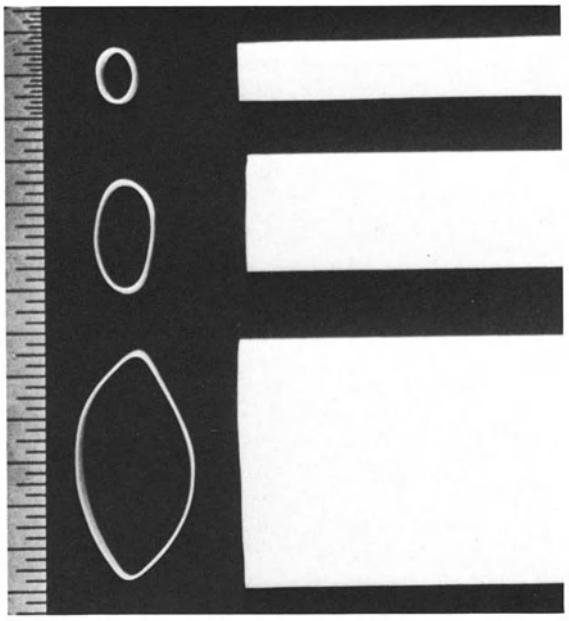


Figure 15-1. Penrose drains of various diameters (¼, ½, and 1 in.). Reproduced with permission from Helmkamp BF, Krebs, HB. Correct use of surgical drains. Contemp Obstet Gynecol. March 1984, p. 123–129.

Passive Drains

Penrose. The Penrose drain is still widely used.[19] It is available as a thin rubber tube in different diameters (Figure 15-1), and is therefore adaptable to different anatomic areas. A small Penrose drain may be used after scalene node biopsy or evacuation of a vulvar hematoma; larger Penrose drains are used for pelvic abscesses or for draining pelvic or subfascial hematomas. If much drainage is expected, an ostomy appliance may be placed over the drain(s) for accurate fluid measurement.[20] The small Penrose drain is ideal for gentle traction on the ureter during its dissection from the pelvic floor or from an adjacent retroperitoneal mass. In the authors' opinion, the use of Penrose drains in *noninfected* wounds should be quite limited for the following reasons:

1. Availability of modern closed suction drains

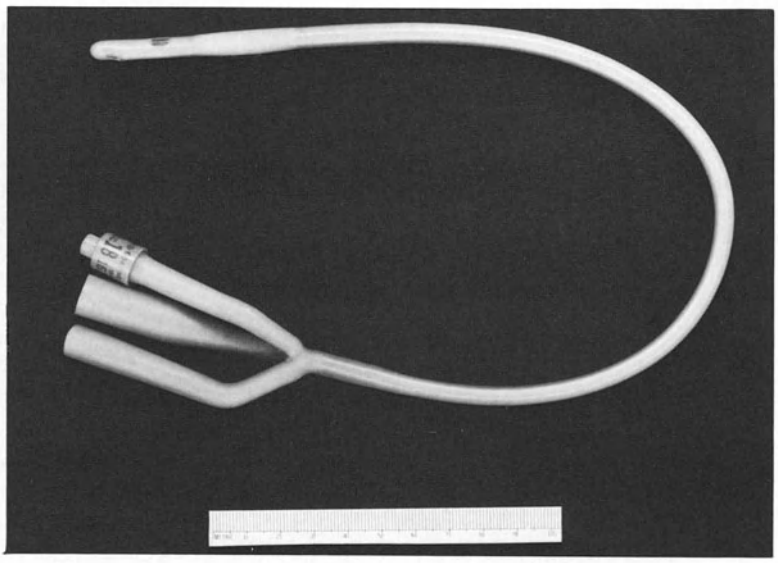


Figure 15-2. Three-way Foley catheter. This triple-lumen tube is suitable for continuous irrigation.

2. Penrose drains are two-way conduits; surface bacteria quickly colonize the drain tract and facilitate secondary infection[5]
3. Penrose drainage by gravity is difficult and inefficient.

A *cigarette drain* is formed by placing gauze inside a Penrose drain. Although capillary action may be enhanced by the gauze, plugging can occur. The authors have found no advantage to cigarette drains as compared to standard Penrose drains.

Foley. The Foley catheter is the standard for bladder drainage. This inflatable balloon catheter was devised in 1935 by Frederick E. B. Foley to control hemorrhage after transurethral prostatectomy.[21,22] In cases in which the bladder has been incised or damaged or in case of severe hemorrhagic cystitis, a three-way Foley catheter will allow continuous bladder irrigation minimizing blockage by clot formation (Figure 15-2). Foley catheters may also be used for long-term suprapubic bladder drainage, decompression of the stomach via gastrostomy, or as active drains. A three-way Foley provides excellent drainage of a pyometra. The uterine cavity may be irrigated continuously with a povidone-iodine solution until the endometrial cavity is cleared of infection.

When the catheter is used for this purpose, it should remain in place for at least 5 days to assure patency of the endocervical canal and allow subsequent spontaneous drainage. After the channel leading to the Foley bulb is selectively tied off, thereby preventing deflation and loss of the catheter, the catheter may be shortened to the level of the vaginal introitus. This modification of a Foley may also be used instead of the Word catheter. The Word catheter (Figure 15-3) is a short latex stem with an

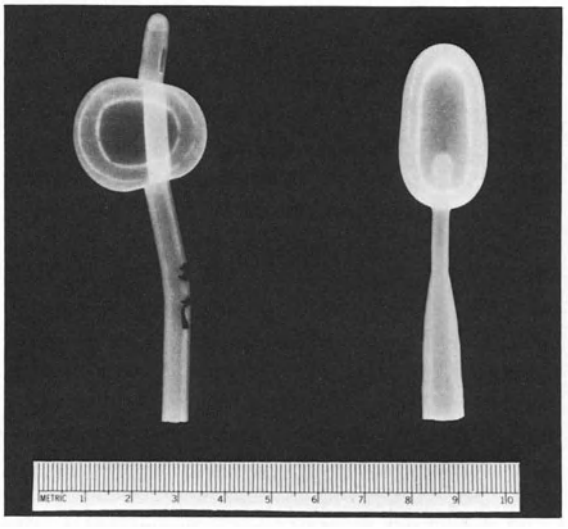


Figure 15-3. Shortened inflated two-way Foley catheter (left). Spontaneous decompression of the bulb is prevented by selective obstruction of the insufflation channel with one or two sutures. The Foley catheter may be used instead of the Word catheter (right) to maintain patency of cysts or abscess cavities (e.g., Bartholin cysts or abscess, pyometra).

inflatable bulb at the end designed specifically for treatment of Bartholin cysts and abscesses. If it is left in place for about 6 weeks, a fistulous tract will develop about the catheter and provide continued drainage of the gland.

Malecot. The Malecot (mushroom or wing-tipped) catheter (Figure 15-4) was introduced in France in 1892.[23] It was originally designed by Achille-Etienne Malecot for use as a self-retaining urethral catheter or as a pleural drainage tube. It is commonly used for posterior colpotomy drainage of a pelvic abscess.[24,25] It is also used to drain the presacral space following posterior or total pelvic exenteration.

Active Drains

Open Drains or Sump Drains. These are double-lumen drains consisting of a smaller tube placed within a larger tube (Salem sump tube) (Figure 15-5). The sump drain allows air to enter the drainage area through the smaller tube. This maintains patency of the larger tube and minimizes the amount of vacuum that is required.[15,26]

Sump drains may also consist of two tubes adjacent to one another. They may be improvised by using two rubber or plastic tubes tied side by side or by placing one inside the other (Figure 15-6). They are primarily used for drainage of the stomach, abscess cavities,[2,6,15] and enterocutaneous fistulas.[27] Sump drains are preferred to Penrose drains for the removal of purulent fluid and for preventing premature closure of an abscess cavity. They may also be used for cavity irrigation. Irrigating fluids can be made to enter the drainage site via the air vent tube during intermittent suction.

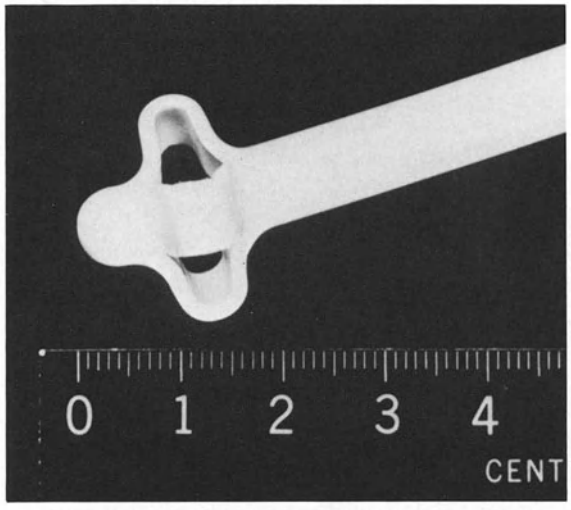


Figure 15-4. Malecot catheter (close-up view). Reproduced with permission from Helmkamp BF, Krebs, HB. Correct use of surgical drains. Contemp Obstet Gynecol. March 1984, p. 123–129.

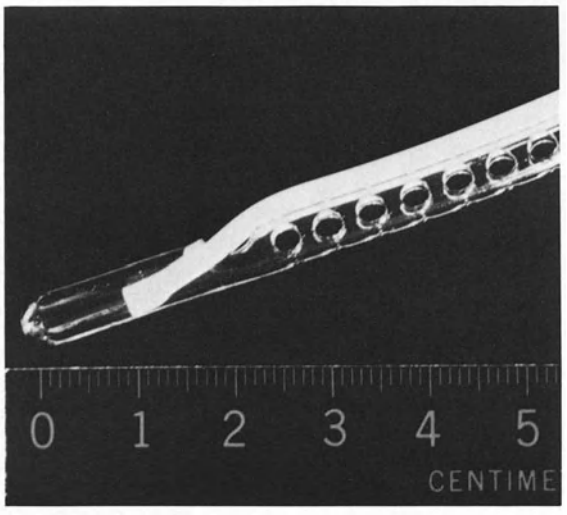


Figure 15-5. Salem sump tube (close-up view). The drain has multiple perforations and a second lumen for air to maintain patency. Reproduced with permission from Helmkamp BF, Krebs, HB. Correct use of surgical drains. Contemp Obstet Gynecol. March 1984, p. 123–129.

Drawbacks to vented double-lumen drains include possible entrance of airborne bacteria and rigidity of the tube.[28] The addition of a filter to the vented lumen has reduced the amount of particulate matter and bacteria entering the tube.[29] When not used for irrigation, sump drains like the Chaffin tube are easily converted to closed drains by clamping one of the two tubal lumina, as shown in Figure 15-7.

Modifications of the sump drains are the triple-lumen drain, which allows continuous irrigation through a third lumen, and the Penrose sump drain. This triple-lumen tube is made by placing a Penrose drain over the sump tube. It has been shown that the Penrose sump drainage system provides superior drainage when compared to either Penrose or sump drain alone.[26]

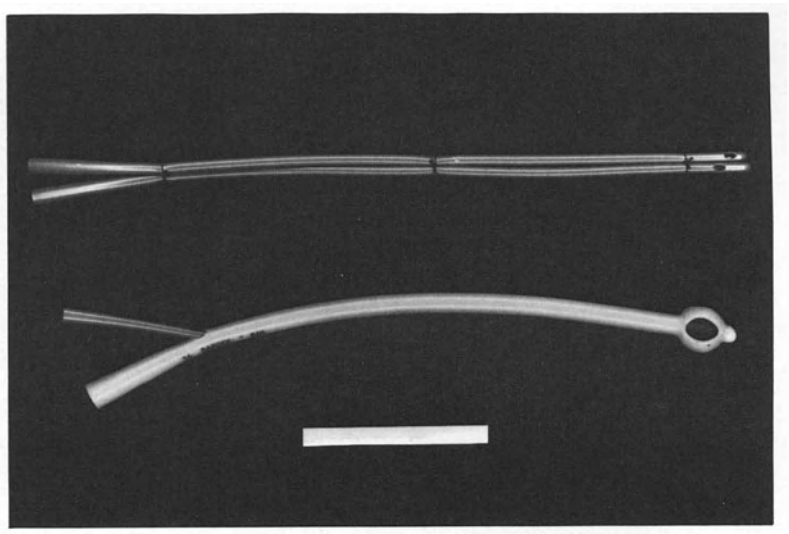


Figure 15-6. Improvised sump tubes. A red rubber tube is inserted through a small opening into the lumen of a large Malecot catheter (bottom). The sump tube on top consists of two red rubber tubes tied together.

Closed Drains. Closed-suction wound drainage is preferred to open or Penrose drainage.[2,9,28,30] All drains create a route for contamination of the drain site; however, this occurs much less frequently when closed suction drains are used. Examples of these drains are shown in Figure 15-8.

The drains may be used prophylactically in any wound area where an accumulation of blood, lymph, or other secretions may promote infections or impair wound healing. In the mid-1970s, Swartz and Tanaree[14,31] studied the role of closed-suction drainage following hysterectomy. When a T-tube drain (Figure 15-8B) was used following abdominal or vaginal hysterectomy, an average of 40 ml of serosanguinous fluid was collected from the retroperitoneal space in 48 hr. Those patients with T-tube drainage had a marked decrease in febrile morbidity when compared to a control group.[14] In a second study, these authors reported on T-tube drainage and cephalosporin prophylaxis in 451 women undergoing hysterectomy. Both the drainage and antibiotic groups had a decrease in postoperative pelvic infection and febrile morbidity when compared to the control group. The combination of T-tube drainage and prophylactic antibiot-

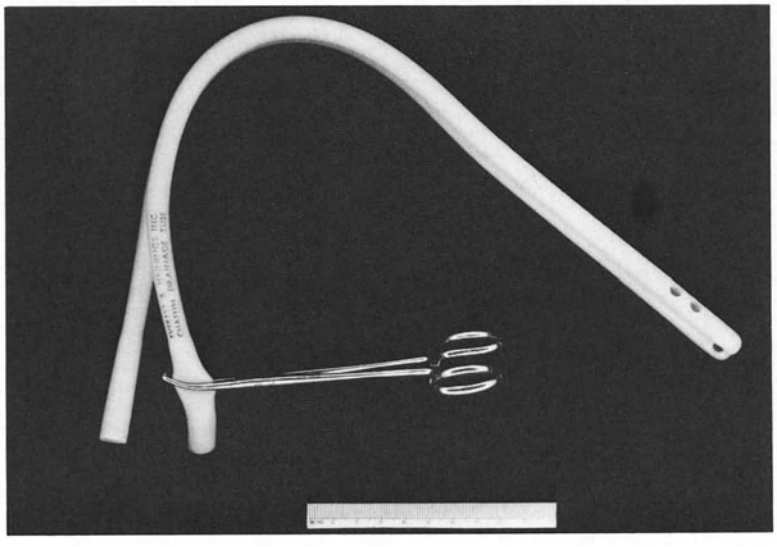


Figure 15-7. Chaffin tube. The drain consists of two adjacent tubes of equal lumen. The clamp (right) converts the tube into a closed suction drain.

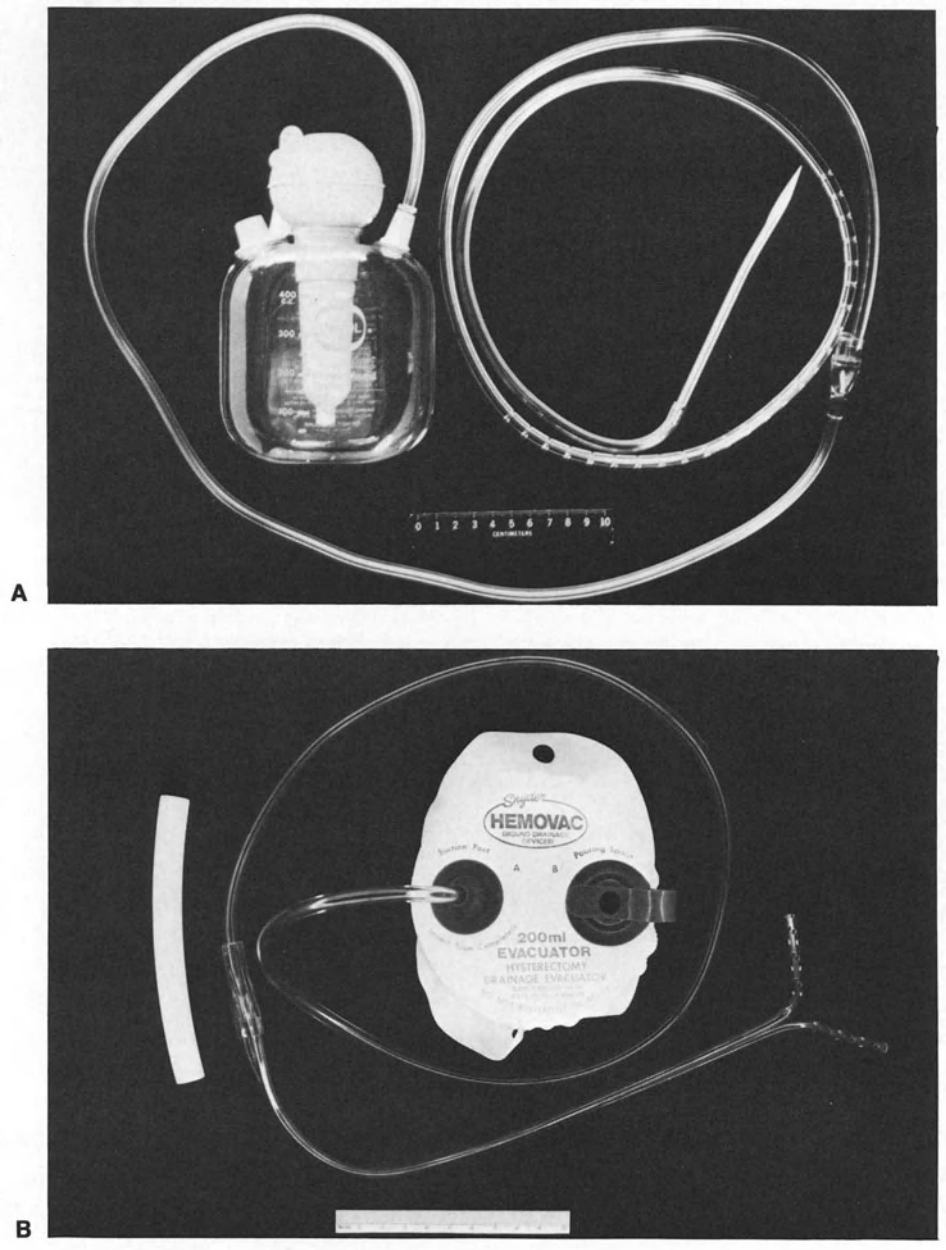
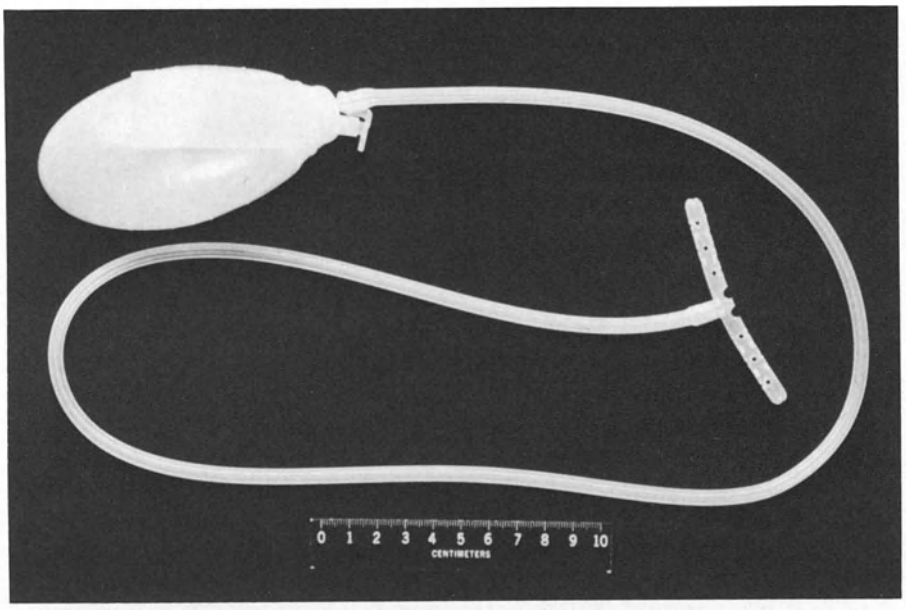


Figure 15-8. Examples of closed suction drains. **A,** ReliaVac. **B,** HemoVac system (200-ml evacuator with Y-tube and inserter sheath).

ics led to a further, but not statistically significant, decrease in pelvic infections. These results show that retroperitoneal T-tube drainage is an acceptable alternative to prophylactic antibiotics for hysterectomy.

T tubes for drainage of the vaginal cuff after vaginal or abdominal hysterectomy may conveniently be let out through the open or partially closed vagina (Figure 15-9). Alternatively, after abdominal hysterectomy, a drain may also be placed into the wound area and let out retroperitoneally through the lateral abdominal wall (Figure 15-10). This approach allows complete closure of the vagina, thereby



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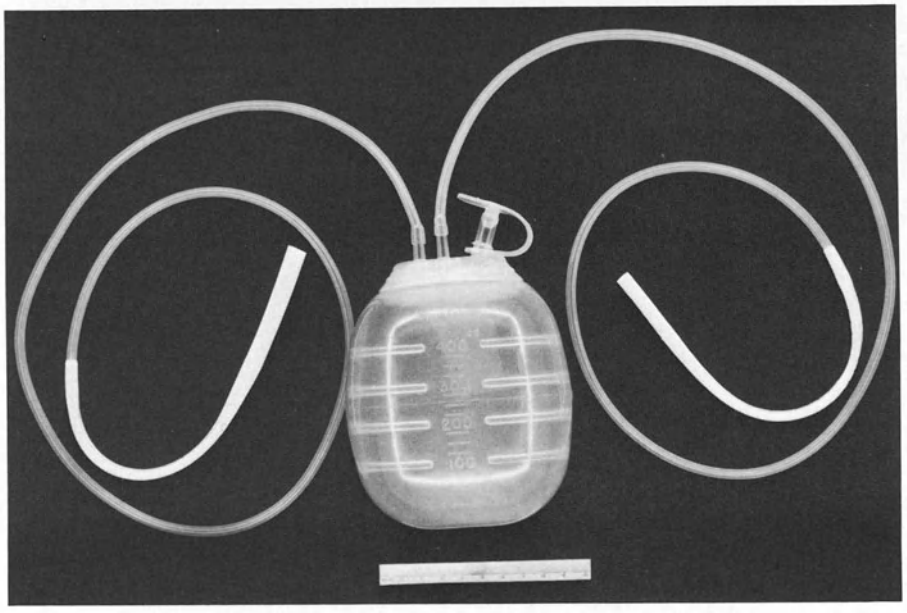

D

Figure 15-8 (*Cont.*). **C,** T tube with Jackson–Pratt small-volume suction reservoir (100 ml). **D,** Flat 7-mm (right) and 10-mm (left) Jackson–Pratt drains with large (400-ml) suction reservoir. **A** and **C** are reproduced with permission from Helmkamp BF, Krebs HB. Correct use of surgical drains. Contemp Obstet Gynecol. March 1984, p. 123–129.

preventing transvaginal bacterial contamination of the wound.

The introduction of retroperitoneal drainage by Symmonds and Pratt[32] following pelvic lymphadenectomy has greatly reduced the incidence of lymphocyst, hematoma, infection, and urinary fistulas. The drain is placed into the obturator fossa between the external iliac vessels and psoas muscle and above the obturator nerve (Figure 15-11). Using long Sarot or Rumel forceps or a Varco clamp, the drain is brought out in the lower quadrant and sewn to

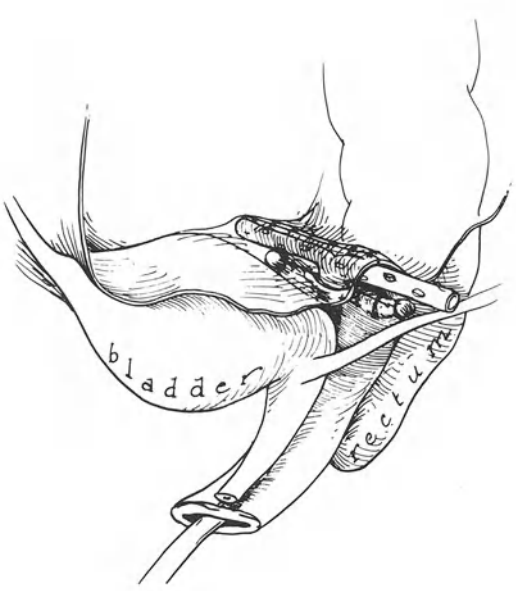

Figure 15-9. T tube. The tube is placed retroperitoneally during abdominal or vaginal hysterectomy. It exits transvaginally.

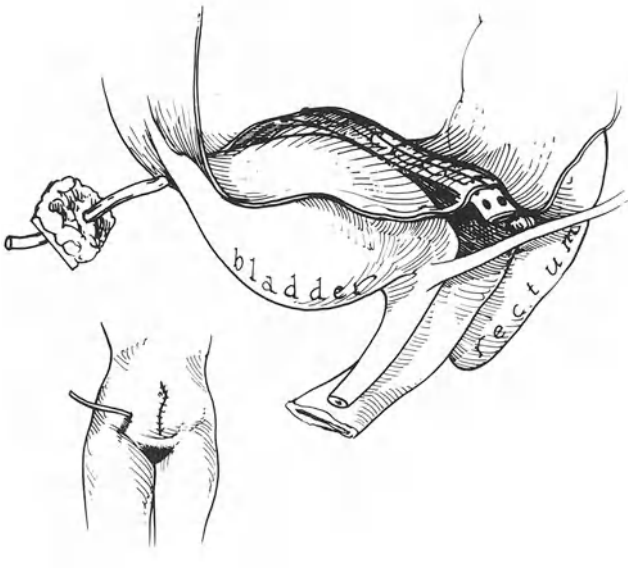

Figure 15-10. Flat Jackson–Pratt drain. The drain is placed retroperitoneally during abdominal hysterectomy. It exits through the anterior abdominal wall.

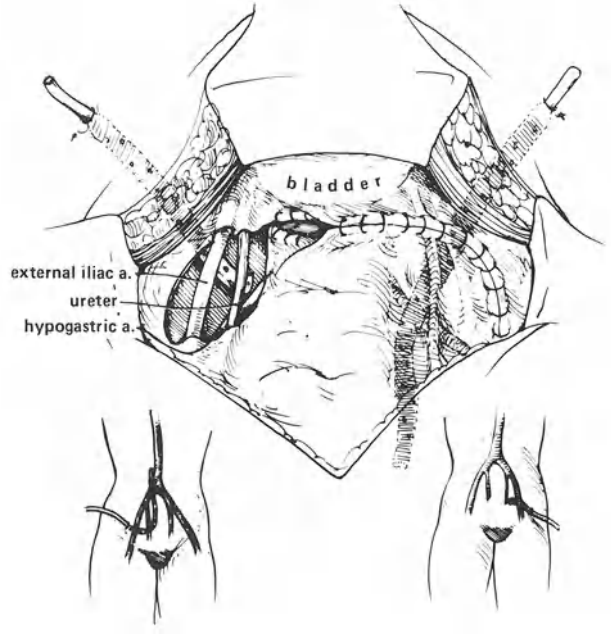

Figure 15-11. Upper: Flat Jackson–Pratt drains in radical hysterectomy. The drain on the left is located retroperitoneally lateral to the external iliac artery and the ureter. The drain on the right drains the right obturator fossa and extends upwards retroperitoneally to simultaneously drain the area of a paraaortic lymph node dissection. Lower right: Location of drain in obturator fossa. Lower left: Flat Jackson–Pratt drain extends cephalad into paraaortic region.

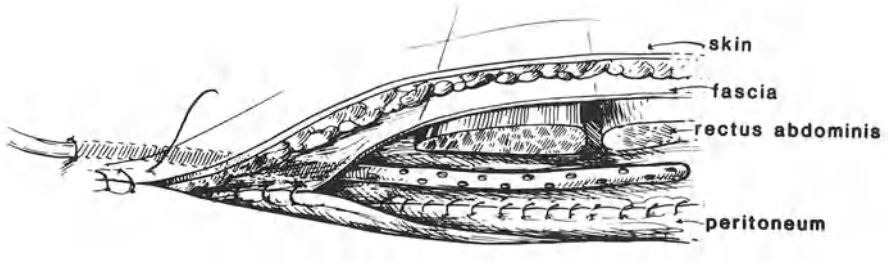

Figure 15-12. Closure of a transverse muscle-cutting (Maylard) incision. A round Jackson–Pratt drain is located in the subfascial space.

the skin with permanent suture. Total drainage may be copious and prolonged; it is not unusual to collect more than 1000 ml. Drains are removed when there is minimal drainage (30–50 ml) within a 24-hr period.

Drains are also used following selective paraaortic lymphadenectomy[33] (Figure 15-11) and inguinal lymphadenectomy.[34,35] Closed-suction drainage should also be considered following transverse muscle-splitting incisions (Figure 15-12).[36,37] The subfascial placement of the drain will help minimize the incidence of subfascial hematoma or seroma.

The authors prefer the Jackson–Pratt flat or round suction drains for closed-suction drainage.[36] These drains are soft, pliable, radiopaque, multiperforated, and have a ridged lumen to prevent collapse (Figure 15-8D).

Technical Aspects of Wound Drainage

For best results, the following principles with regard to the placement and management of drains should be observed.

1. Only soft drains should be used adjacent to blood vessels, nerves, bowel, ureter, or bladder
2. To avoid infection, wound disruption, and hernia, drains should never exit through the operative incision[2,9,10,15,19]
3. The stab wound or incision for the drain must be adequate. If it is too tight, drainage cannot take place; if it is too loose, bowel or omentum may herniate alongside the drain[3]
4. The peritoneum and fascia should be placed into normal position when the stab wound is being made. This will give the drain the most direct route of exit and will prevent kinking of the drain during closure of the abdominal wall[3]
5. Drains should be removed as soon as there is no significant drainage. Drains placed prophylactically can usually be removed within the first 2 postoperative days, if drainage is minimal (30-50 ml within a 24 hr period)
6. Passive drains should exit in dependent site for gravity drainage
7. Passive drains placed for therapeutic reasons are advanced slowly each day once significant drainage has ceased.[15,18]

The suction setting for active drains varies between open and closed drains. "High" suction is appropriate for gastric decompression using a sump drain attached to a Thermotic Pump (e.g., Gomco 120 mm Hg). A "low" suction (30–40 mm Hg) is preferred as continuous suction or intermittent suction from a central source (wall suction). A three-way Foley catheter for drainage or irrigation of a pyometra or abscess cavity may also be attached to low-level continuous or intermittent suction.

The optimal level of suction for closed drains has not been established. A recent report suggested that high-pressure systems creating negative pressures in excess of 100 cm H_2O water cause more damage and tissue necrosis than low-pressure systems.[38] However, the clinical relevance of these findings is presently not clear. A well controlled low level of suction may be obtained by attaching a drain to a Pleur-evac collection chamber. The filling of the suction chamber (e.g., 15–20 cm of water) will determine the amount of negative pressure.[33]

Complications of Drains

Complications related to drains may largely be prevented by selecting the proper drain, using it for sound indications, and adhering to basic surgical principles during placement and subsequent management. If a vessel is cut when the stab wound is made, the bleeding may be controlled by compression. A deeply placed U stitch securing the drain to the skin may also achieve hemostasis. Persistent hemorrhage requires surgical exploration of the stab wound and ligation of bleeding vessels.

Bowel that herniates alongside a drain may become obstructed. This complication has become exceedingly rare since the advent of closed drainage systems.

Infection of the drain tract is not uncommon.[5] Only occasionally will an abdominal wall abscess form after removal of the drain. Infection of the actual drain site below the drain tract calls for aggressive antibiotic treatment. It is usually best to remove the infected drain. Alternatively, wound irrigation with antibiotic solution through the drain should be considered.

A sudden decrease in the drainage suggests obstruction. It is usually due to small tissue fragments rather than blood clot.[37] Although it may be possible to restore patency of the drain by flushing it with a sterile solution of normal saline,[37] this procedure risks introducing bacteria into the wound. Drain obstruction can usually be avoided by clearing the drain site of all debris during surgery and by *early* use of suction.

Drains should be considered foreign bodies and must not be used as a substitute for hemostasis or meticulous surgical technique. This is particularly true when drains are employed for prophylactic reasons.

References

1. Robb H. The management of the drainage tube in abdominal surgery. Johns Hopkins Hosp Rep, Baltimore. 1891; 2:184–188.
2. Moss JP. Historical and current perspectives on surgical drainage. Surg Gynecol Obstet. 1981; 152:517–527.
3. Postlethwait RW. Principles of operative surgery; antisepsis, technique, sutures, and drains. In: Sabiston Jr DC, ed. Davis–Christopher textbook of surgery. The biological basis of modern surgical practice. Philadelphia: WB Saunders, 1981: p. 317–332.
4. Gibson CL. The rubber dam Mikulicz tampon. Ann Surg. 1921; 73:470–472.
5. Yates JL. An experimental study of the local effects of peritoneal drainage. Surg Gynecol Obstet. 1905; 1:473–492.
6. Chaffin RC. Drainage. Am J Surg. 1934; 24:100–104.
7. Murphey DR. Use of atmospheric pressure in obliterating axillary dead space following radical mastectomy. South Surg. 1947; 13:372–375.
8. Vallfors B, Hanssong HA, Larsson J, et al. Studies on optimal conditions in surgical suction systems. Acta Chir Scand [Suppl]. 1976; 474:1–36.
9. Cruse PJE, Foord R. A five year prospective study of 23,469 surgical wounds. Arch Surg. 1973; 107:206–210.
10. Higson RH, Kettlewell MGW. Parietal wound drainage in abdominal surgery. Br J Surg. 1978; 65:326–329.
11. Alexander JW, Korelitz J, Alexander NS, et al. Prevention of wound infections: a case for closed suction drainage to remove wound fluids deficient in opsonic proteins. Am J Surg. 1976; 132:59–63.
12. Altemeyer WA. Surgical infections; incisional wounds. In: Bennett JC, Brachman PS, eds. Hospital Infection. Boston: Little, Brown, 1979: p. 287.
13. McIlreth DC, Van Heerden JA, Edis AJ, et al. Closure of abdominal incision with subcutaneous catheters. Surgery. 1977; 80:411–416.
14. Swartz WH, Tanaree P. Suction drainage as an alternative to prophylactic antibiotics for hysterectomy. Obstet Gynecol. 1975; 45:305–310.
15. Duthie HL. Drainage of the abdomen. N Engl J Med. 1972; 287:1081–1083.
16. Greenall MJ, Evans M, Pollack AV, et al. Should you drain a perforated appendix? Br J Surg. 1978; 65:880–882.
17. Karlen JR. Personal communication, 1984
18. Barone RM. Care of drains and tubes. In: Condon RE, Nyhus LM, eds. Manual of surgical therapeutics. Boston: Little, Brown, 1975: p. 164–174.
19. Abramson DJ. Charles Bingham Penrose and the Penrose drain. Surg Gynecol Obstet. 1976; 143:285–286.
20. Firlit CF, Canning JR. Surgical wound drainage: a simple device for collection. J Urol. 1972; 108:327–329.
21. Zorgniotti AW, Foley FEB. Early development of balloon catheter. Urology. 1973; 1:75–80.
22. Foley FEB. A self-retaining bag catheter for use

as an indwelling catheter for constant drainage of the bladder. J Urol. 1937; 38:132–140.
23. Malecot A. Sonde se fixant d'elle-même à demeure das la vessie. Archivs de Tocologie et de Gynecologie. 1892; 19:321–323.
24. Rubenstein PR, Mishell DR Jr, Ledger WJ, et al. Colpotomy drainage of pelvic abscess. Obstet Gynecol. 1976; 48:142–146.
25. Benigno BB. Medical and surgical management of the pelvic abscess. Clin Obstet Gynecol. 1981; 24:1187–1197.
26. Hanna EA. Efficiency of peritoneal drainage. Surg Gynecol Obstet. 1970; 131:983–987.
27. MacFarlane M, Frawley JE. A technique for drainage of enterocutaneous fistulas. Surg Gynecol Obstet. 1975; 141:263–264.
28. Magee C, Rodeheaver GT, Golden GT, et al. Potentiation of wound infection by surgical drains. Am J Surg. 1976; 131:547–549.
29. Golden GT, Roberts TL, Rodeheaver GT, et al. A new filtered sump tube for wound drainage. Am J Surg. 1975; 129:716–717.
30. Nova PF, Vanecko RM, Bransfield JJ. Prophylactic abdominal drains. Arch Surg. 1972; 105:173–176.
31. Swartz WH, Tanaree P. T-tube suction drainage and/or prophylactic antibiotics. A randomized study of 451 hysterectomies. Obstet Gynecol. 1976; 47:665–670.
32. Symmonds RE, Pratt JH. Prevention of fistulas and lymphocysts in radical hysterectomy. Obstet Gynecol. 1961; 17:57–64.
33. Helmkamp BF, Krebs HB, Isikoff MB, et al. Para-aortic lymphocyst. Am J Obstet Gynecol. 1980; 138:395–398.
34. Disaia PJ, Creasman WT, Rich WM, et al. An alternative approach to early cancer of the vulva. Am J Obstet Gynecol. 1979; 133:825–832.
35. Hacker NF, Leuchter RS, Berek JS, et al. Radical vulvectomy and bilateral inguinal lymphadenectomy through separate groin incisions. Obstet Gynecol. 1981; 58:574–579.
36. Jackson FE, Fleming PM. Jackson–Pratt brain drain. Use in general surgical conditions requiring drainage. Int Surg. 1972; 57:658–659.
37. Zacharski LR, Colt J, Mayor MB, et al. Mechanism of obstruction of closed wound suction tubing. Arch Surg. 1979;114:614–615.
38. Cherry G, McClatchey K. Wound drainage: effects of negative pressure on healing. Infect Surg. 1983; 2:243–247.

Postoperative Bladder Drainage 16

Ellen Brock and J. Andrew Fantl

The need for postoperative bladder drainage in gynecologic surgery and the optimal method to accomplish it remain subjects of some controversy. Although the use of catheters for bladder drainage is many centuries old, modern techniques for continuous drainage began when Foley introduced the self-retaining balloon catheter in 1937.[1] A similar catheter, used for achieving hemostasis following prostatectomy, was described by him 10 years earlier. The use of the catheter had numerous clear advantages over previous methods. There was no need for repetitive catheterizations, and the latex material irritated the urothelium far less than other materials. In addition, there was significantly less urethral trauma since positional changes caused less catheter movement.

Suprapubic catheterization for continuous drainage was introduced with success to the gynecologic community by Hodgkinson and Hodari in 1966,[2] and has gained acceptance. It had, however, been described in 1953 by Madden and Goddard as a bladder drainage technique following vaginal repairs.[3] Numerous reports followed, proclaiming the superiority of suprapubic over transurethral bladder drainage. The most commonly cited advantages included: (1) decreased patient discomfort, (2) easier nursing management, (3) ability to clamp the catheter and allow a voiding trial, (4) lower incidence of significant bacteriuria, and (5) decreased time to normal voiding.[2,4,5]

There were, however, new complications associated with the insertion and use of the suprapubic catheter. These included (1) perforation of a viscus, (2) hematuria, (3) infection of the insertion site, (4) hematoma formation, (5) catheter breakage, and (6) occlusion. In attempts to avoid these complications, several different catheters have been developed, utilizing different materials and increasing lumen size.

Urinary tract infection has long been recognized as a risk of bladder catheterization. Kass[6] in 1956 found a 2% incidence of dysuria and bacilluria in normal healthy women after a single catheterization. In addition, he indicated that 95% of patients with indwelling transurethral catheters would develop significant bacilluria within 96 hr and that prophylactic antibiotics would not prevent such infections. The debate continues as to whether antibiotic coverage is of any value. Mattingly et al.,[7] Ingram,[8] Kass,[6] and others have testified against the need for antibiotics while Hofmeister et al.[5] and others advocated their use. Most importantly, Mattingly et al.[7] in 1972 demonstrated the absence of long-term sequelae in healthy women with catheter-induced urinary tract infection.

Reasons for Bladder Drainage Following Gynecologic Surgery

The reasons for postoperative bladder drainage vary according to the specific situation. A clinically oriented classification is presented in Table 16-1. It is important to recognize the indications for catheter drainage and select the

Table 16-1. Bladder Drainage Following Gynecologic Surgery

Complaint	Clinical situation
Spontaneous voiding is inconvenient and/or painful	Difficult or traumatic vaginal delivery Nonradical pelvic surgery Noncompressive surgery for genuine stress incontinence Genitourinary fistula
The tissue of the lower urinary tract has been disrupted	Bladder or urethral diverticulum Intraoperative cystotomy or injury to bladder or urethra
The bladder and/or urethra have been denervated or obstructed	Radical pelvic surgery Compressive surgery for genuine stress incontinence

best route. An across-the-board system applicable to all circumstances does not exist.

Inconvenient and/or Painful Spontaneous Voiding

Bladder drainage is rarely necessary after vaginal delivery. On occasion, however, after a difficult delivery, tissue trauma may produce edema and pain that cause inability to void. Pain is usually the main reason for difficulty in spontaneous bladder emptying. Striated muscle spasm inhibits relaxation, and the normal voiding sequence is interrupted. Transurethral bladder drainage may be necessary for a short period of time, although analgesic therapy and local physiotherapy in the form of sitz baths may be all that are needed. In this regard, requesting the patient to void while in the bath tub may result in successful voiding and avoidance of catheterization.

Immediately following pelvic surgery, incisional pain and postoperative sedation make spontaneous voiding difficult and inconvenient. Such inconveniences include patient discomfort as well as nursing care. Most surgeons use transurethral drainage for this short period (usually 24 hr). Richardson et al.,[9] however, found that only 6 of 151 patients required insertion of an indwelling catheter postoperatively when none had been placed at the time of surgery. In general, there is agreement that the infectious morbidity incurred by the short-term postoperative drainage is offset by the advantages in patient comfort and nursing care.

The indications for bladder drainage following nonradical pelvic surgery also apply usually to noncompressive or repositional operations for urinary incontinence. However, in cases where extensive bladder neck or periurethral dissection was performed, bladder drainage is recommended for 3–7 days until tissue edema has subsided. We have used either transurethral or suprapubic drainage in such conditions.

All patients are preoperatively instructed on bladder function and techniques of perineal relaxation. Specific efforts are made by the medical and nursing team to relieve anxiety and provide reassurance of successful postoperative micturition. After catheter discontinuation or clamping, urinary symptoms and micturition frequency and volumes are monitored for 24 hr. If the patient is asymptomatic and frequency and volumes are within normal limits, no attempts are made to obtain residual volumes in those cases where transurethral drainage as used. If knowledge of residual urine volume is desired, we recommend that it not be obtained following initial micturition, regardless of the route of drainage. It is not uncommon to observe irritative urinary frequency and small voided volumes immediately following catheter removal or clamping. This usually resolves after two or three spontaneous voidings.

"Significant" residual volume is still a matter of controversy. Absolute figures, such as 50 ml, seem less clinically sound than volume/residual ratios or percentage of voided volumes. In general, we consider residuals that represent 20%–25% of the voided volume clinically acceptable provided that at least 200 ml are voided. Recatheterization is, of course, necessary when symptoms of bladder fullness persist or total inability to void exists. In our opinion, clinical judgment rather than strict formulas or volumes should guide the clinician.

Timing of catheter removal or clamping varies according to surgeon's preference; reports vary from 3 to 15 days. There is no significant difference in time for return to normal voiding

when suprapubic and transurethral bladder drainage are compared.[5,7,10] It seems reasonable to expect the extent and involvement of the surgery rather than the route of bladder drainage to be the predictive factor in the time to restoration of voiding function.

There has been considerable attention devoted to the issue of bacterial infection and the route of bladder drainage. Wiser et al.,[11] in a randomized double-blind prospective study, reported a significant increase in the incidence of postoperative bacteriuria (at 4 days and at 6 weeks postoperatively) in patients using transurethral drainage over those with suprapubic drainage when no prophylactic antibiotics were used. In addition, they found that the dominant organisms in positive cultures from the suprapubic group were *Staphylococcus* and *Proteus;* in the group with transurethral drainage, the dominant organisms were *E. coli, Klebsiella, Enterococcus,* and *S. fecalis.* This finding differs somewhat from Ingram's report in which, in the suprapubic group, 94% of the positive cultures were *E. coli* and *Klebsiella.*[3,8,10] These organisms accounted for only 57% of the positive cultures in the group with transurethral drainage, the remainder being species of *Proteus, Pseudomonas,* and *Enterococcus.*

Mattingly et al.,[7] in a series in which no prophylactic antibiotics were used, and Hofmeister et al.,[5] in a series in which sulfa drug prophylaxis was employed, found that in the absence of urinary tract anomalies or altered host susceptibility, chronic urinary tract anomalies or altered host susceptibility, chronic urinary tract infection did not develop from catheter-induced bacteriuria.

When the suprapubic route is selected, the technique of insertion is of significant importance. When the trocar method is used, distension of the bladder, appropriate midline suprapubic location of the insertion site, and caudal direction of the trocar are essential for safe catheter insertion (Figure 16-1). An alternate method is the use of a male urethral sound to push the distended bladder to the anterior abdominal wall. A skin incision is made over the sound, and the catheter is drawn into the bladder after attachment to the sound (Figure 16-2). Wilson et al.[12] found that few complications are associated with this method of insertion.

Relative contraindications to the use of closed suprapubic cystotomy are inability to distend the bladder, recent cystotomy, gross hematuria, previous extensive abdominal surgery, bladder carcinoma, or extreme obesity. In general, catheter lumen diameter is an important consideration in using suprapubic catheters. Small-bore catheters are easily occluded, especially when macrosopic hematuria exists or prolonged drainage is expected.

Recently, Broberg[13] reported on the use of a #8 feeding tube for transurethral drainage after gynecologic surgery. The tube, which is prepared by cutting extra holes in the distal end, is inserted through the urethra and sutured in place. This minitube has the advantage of allowing spontaneous voiding around it, thereby eliminating the need to remove the catheter from the urethra to allow a voiding trial. In addition, the risk of trauma associated with suprapubic catheter placement is avoided. More experience and future clinical trials will determine applicability of this innovative method.

Disruption of the Tissues of the Lower Urinary Tract

Bladder drainage is indicated following procedures that involve, or result in, interruption of the integrity of the urinary tract. Such procedures include cystotomy, repair of urethral diverticulum, or repair of genitourinary fistula. Following cystotomy without other complications, bladder drainage is indicated for 5–7 days. This can be accomplished by the transurethral or suprapubic route with equal efficacy.

With regards to vesicovaginal fistula, Marshall[14] recommended using suprapubic drainage, while Keettel et al.[15] have reported good results using transurethral drainage. The technique of postoperative drainage is often dictated by the route of repair (abdominal versus vaginal) and by the size and location of the defect. If the fistula involves the trigone or the base of the bladder, suprapubic drainage is desirable to avoid pressure on the suture line from a transurethral catheter bulb. This would be particularly important in radiation-induced fistulae where healing is already impaired. In utilizing a vaginal approach to repair large

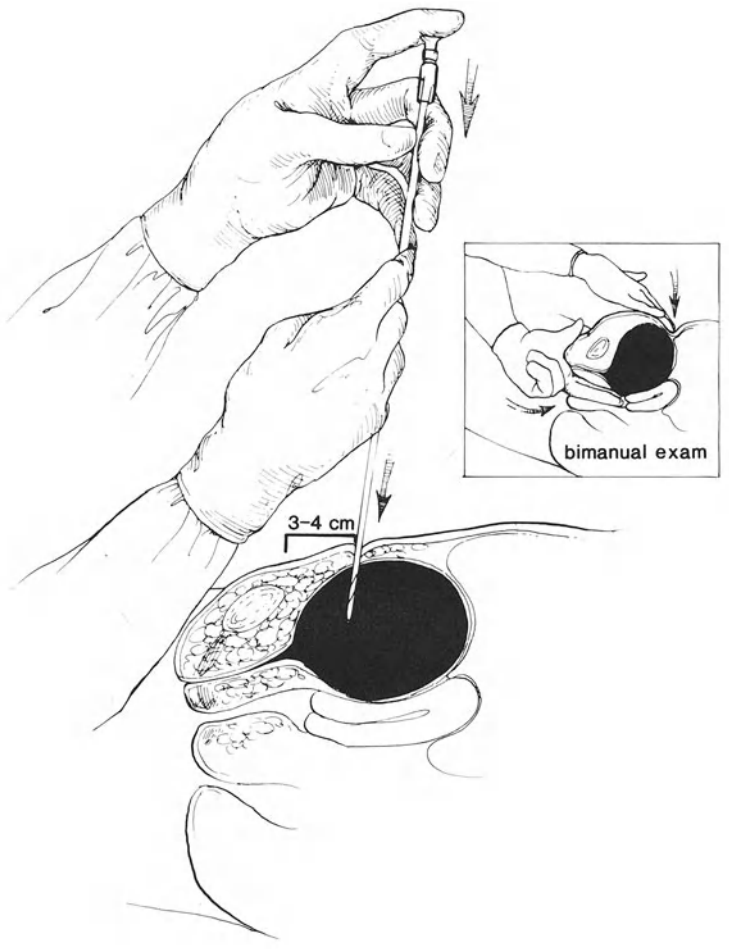

Figure 16-1. Technique of insertion of suprapubic catheter using trocar method. Note bladder distension, placement of trocar relative to pubic symphysis, and direction of insertion of trocar.

vesicovaginal fistulae, bladder overdistension needed for the insertion of a closed suprapubic cystotomy may jeopardize the integrity of the repair. An open cystotomy may be a consideration in such cases.

The length of time that postoperative bladder drainage is employed depends on the cause and size of the fistula. Recommendations range from 7 days in small simple postoperative fistulae to 2–6 weeks in large radiation-induced fistulae. It should be mentioned that attempts at achieving spontaneous healing of small postoperative vesicovaginal fistulae with catheter drainage alone should be encouraged. Symmonds[16] reported that 15%–20% of postoperative vesicovaginal fistulae will heal spontaneously with catheter drainage alone within 6 weeks.[16]

The importance of using large-caliber catheters for drainage after fistula repair is obvious, as macroscopic hematuria is always present. When the fistula involves the urethra, most authors recommend the suprapubic route of drainage for approximately 2 weeks. The same applies to drainage after urethral diverticulectomy. In treating a urethral diverticulum by partial diverticulectomy, we have used transurethral drainage for 2 weeks with very satisfactory results. When marsupialization is done for a distal diverticulum, drainage time is reduced to 3–4 days.

Denervated or Obstructed Bladder and/or Urethra

These conditions may occur after radical surgery for cervical carcinoma or deliberate obstructive surgery for genuine stress inconti-

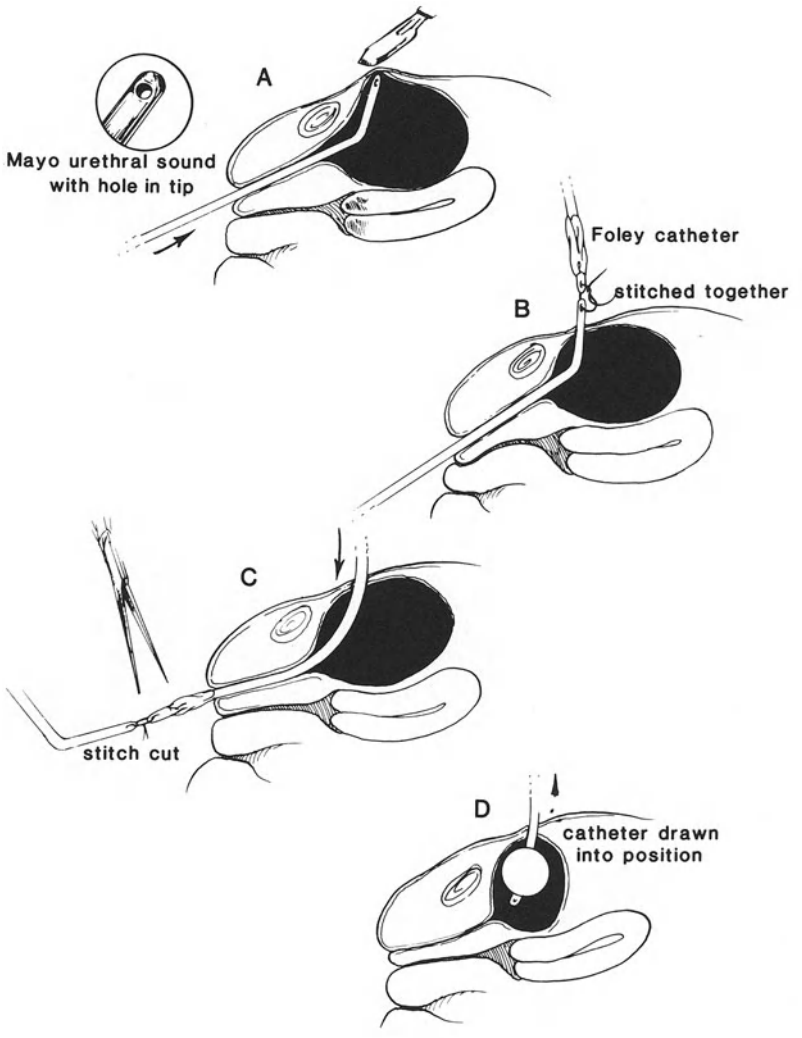

Figure 16-2. Technique of suprapubic catheter insertion using male urethral sound. The anterior abdominal wall is elevated with the sound, incised, and the catheter sutured to the sound tip. The sound and catheter tip are withdrawn through the urethra, the suture cut, and the Foley catheter positioned properly in the bladder.

nence (sling procedures). Under these circumstances in which prolonged bladder drainage might be anticipated, suprapubic, large-bore catheter drainage should be utilized. Open cystotomy is preferred, and a Foley- or Malecot-type catheter can be used. These patients should be counseled preoperatively on catheter care, as this counseling minimizes anxiety and has a significant impact in their clinical management. We do not encourage clamp–release–clamp techniques, as the literature offers weak evidence that such methodology accelerates or facilitates micturition. Passive distension and collapse will probably not accelerate reinervation or muscle hypertrophy, which is necessary to overcome denervation and/or obstruction. On the other hand, time intervals and/or *active* detrusor contractions seem to represent a more logical approach.

Summary

Bladder drainage after gynecologic surgery is needed for various reasons. The reasons should be identified, and appropriate techniques used to accomplish the desired therapeutic objectives. It makes clinical sense to tailor the route, catheter type, and length of drainage to each case. Strict protocols utilized without individualization should be discouraged. Preoperative patient counseling incorporating knowledge on bladder function as well as catheter care represents an important addition to appropriate management.

References

1. Foley FEB. A self-retaining bag catheter. J Urol. 1937; 38:140.
2. Hodgkinson CP, Hodari AA. Trocar suprapubic cystotomy for postoperative bladder drainage in the female. Am J Obstet Gynecol. 1966; 96:773.
3. Ingram J. Suprapubic cystotomy by trocar catheter: A preliminary report. Am J Obstet Gynecol. 1972; 113:1108.
4. McGruder CJ. Suprapubic bladder drainage following gynecologic operations. Am J Obstet Gynecol. 1972; 113:267.
5. Hofmeister FJ, Martens WE, Strebel RL. Foley catheter or suprapubic tube? Am J Obstet Gynecol. 1970; 107:767.
6. Kass C. Asymptomatic infections of the urinary tract. Trans Assoc Am Physicians. 1956; 56.
7. Mattingly RF, Moore DE, Clark DO. Bacteriologic study of suprapubic bladder drainage. Am J Obstet Gynecol. 1972; 114:732.
8. Ingram J. Postoperative management of the bladder. In: Sciarra J, ed. Gynecology and obstetrics. New York: Harper & Row, 1984.
9. Richardson AC, Lyon JB, Graham EE. Abdominal hysterectomy: relationship between morbidity and surgical technique. Am J Obstet Gynecol. 1973; 115:953.
10. Ingram J. Further experience with suprapubic drainage by trocar catheter. Am J Obstet Gynecol. 1975; 121:885.
11. Wiser WL, Morrison JC, Loveday GL, et al. Management of bladder drainage following vaginal plastic repairs. Obstet Gynecol. 1974; 44:65.
12. Wilson EA, Sprague AD, VanNagell JR Jr. Suprapubic cystotomy in gynecologic surgery: a comparison of two methods. Am J Obstet Gynecol. 1973; 115:991.
13. Broberg C. Catheter drainage after gynecologic surgery: a comparison of methods. Am J Obstet Gynecol. 1984; 149:18.
14. Marshall VS. Vesicovaginal fistulas on one urological service. J Urol. 1979; 121:25.
15. Keetel WC, Sehring FG, deProsse CA, et al. Surgical management of urethrovaginal and vesicovaginal fistulas. Am J Obstet Gynecol. 1978; 131:425.
16. Symmonds RE. Prevention and management of genitourinary fistula. J Contin Educ Obstet Gynecol. 1979; 21:13.

Major Posthysterectomy Infections: Diagnosis and Management

17

David L. Hemsell

Posthysterectomy Infection

Elective hysterectomy was the most frequently performed major surgical procedure on reproductive age women during the last decade.[1] Adherence to good surgical principles such as gentle handling of tissue, sharp dissection, careful attention to hemostasis, avoidance of large pedicles of devitalized tissue and large areas of dead space, use of less reactive suture, and close attention to aseptic techniques have reduced complications. In addition, Ledger[2] emphasized that the use of plastic adhesive drapes, excessive use of electrocautery for cutting, and the use of passive wound drains increase the risk for postoperative infection. Because of the potential pathogens involved in postoperative infections, abscess formation is possible. Postoperative pelvic or wound infection may prolong hospital stay from 5 to 14 days and necessitate a second operative procedure. These infections continue to pose a significant problem for gynecologic surgeons.

Pelvic infection occurs more frequently than infection of an abdominal incision. The higher pelvic infection rate may be due to collections of 10–200 ml of blood and serum above the vaginal surgical margin. These collections are ideal culture media for the normal flora of the lower reproductive tract.[3] Theoretically, reduction of this volume should lower the infection rate. Indeed, Swartz and Tanaree[3] reported that T-tube suction drainage (see Chapter 8) of this extraperitoneal space was as effective as prophylactic antibiotics in preventing postoperative pelvic infection. They showed that when suction drain was combined with a prophylactic antibiotic, a significant lowering of the infection rate did not occur.[4] This result has not been uniformly observed. Poulsen and co-workers[5] reported significant reduction in the incidence of febrile morbidity, infectious morbidity, and urinary tract infection after hysterectomy if a prophylactic antibiotic was administered, but not with suction drainage alone. Galle and colleagues[6] reported similar results in a series of women undergoing vaginal hysterectomy. The volume of fluid collected varied from zero to 720 ml in the latter study and was much greater than that reported by Swartz.

Gray[7] described an open cuff technique designed to reduce or prevent the extraperitoneal, supravaginal collection of blood and serum and thereby to decrease postoperative infections after abdominal hysterectomy. Application of this technique to women undergoing vaginal hysterectomy at our hospital resulted in an incidence of pelvic infection that was significantly lower than the infection rate observed when the cuff was closed and if placebo was given.[8] When prophylaxis was given, no difference in infection rate was observed if the vaginal cuff was left open or closed. Perhaps this observation occurred because the infection rate was so low, and the numbers of patients in each arm were small.

In interpreting prophylaxis or therapy data, the definition of "infection" is essential. Conventional teaching emphasizes temperature elevation as one of the hallmark signs of infection. In many reports, the term "febrile

morbidity" has been used interchangeably with "infection requiring antibiotic therapy." Gray,[7] Falk and Bunkin[9], and Ledger and Child[10] emphasize that the two entities should not be confused. They reported an incidence of febrile morbidity (temperature elevation only) that was significantly higher than the incidence of infection requiring antibiotic therapy.

To further confuse the issue, definitions of febrile morbidity are far from uniform. Listed below are several of the more commonly reported definitions of febrile morbidity:

1. Temperature of 38°C or greater[11]
2. Temperature of 38°C or greater on 2 different days[12]
3. Temperature of 38°C or greater on 2 of the first 10 postoperative days more than 24 hr after surgery[13,14]
4. Temperature of 38°C or greater on 2 days more than 24 hr after surgery[15,16]
5. Temperature of 38°C or greater on two or more occasions 6 or more hr apart more than 24 hr after surgery[17,18,19]
6. Temperature of 38°C or greater on two or more occasions 6 or more hours apart more than 24 hr after surgery on 2 consecutive days[5]
7. Temperature of 38°C or greater on at least two occasions more than 48 hr after surgery[20]
8. Temperature of 38°C or greater on at least two occasions 4 or more hours apart more than 48 hr after surgery[21]
9. Temperature of 38.3°C or greater on 2 occasions six hours apart more than 48 hr after surgery[22]
10. Temperature of 38.1°C or greater on two occasions 4 hr apart[23]
11. Temperature of 38.3°C or greater on at least two occasions 8 or more hr apart more than 48 hr after surgery[24]
12. Temperature of 38.3°C or greater on at least two occasions 8 or more hr apart more than 48 hr after surgery[25]

Most authors, as noted above, use a reading of 38°C or greater as an indication of infection requiring therapy.

The findings at vaginal examination may be of limited value in establishing the diagnosis of pelvic infection. Vaginal examination on the second or third day after hysterectomy is frequently associated with physical findings that are compatible with infection. Irrespective of temperature reading, there is usually purulent material in the vagina. The surgical margin is somewhat erythematous and indurated, and may be tender. A logical assumption may be that all vaginal cuffs are infected after hysterectomy whether the patient develops temperature elevations or not.

Treatment for cuff cellulitis is not mandatory in the majority of patients. The addition of temperature elevation to the above physical findings does not require the use of parenteral or oral antimicrobials or the prolongation of prophylactic antibiotics. Most gynecologic surgeons will not treat patients with antibiotics if they are not febrile. Frequently, they will treat patients with parenteral or oral antimicrobials if they develop temperature elevations postoperatively whether symptoms are present or not. The use of antibiotics in an asymptomatic patient constitutes antimicrobial overuse in most instances.

As a result of prospective evaluations, we learned that it was safe to withhold antimicrobial therapy for women who had temperature elevations after hysterectomy if they remained asymptomatic.[8] In these patients, abdominal examination remained normal, and a pelvic examination did not contribute useful information. Up to 40% of women undergoing hysterectomy may have temperature elevations to 38°C or above on two or more occasions more than 24 hr after surgery. A pelvic examination is not necessary after each temperature elevation since this examination may represent a source of infection. For this reason, we do not routinely perform vaginal examinations at temperature elevation after hysterectomy.

If a patient complains of increasing lower abdominal, pelvic, or back pain after hysterectomy, lower abdominal tenderness, usually asymmetric and away from the abdominal incision, was a common finding. On bimanual examination parametrial tenderness was frequently present. The temperature elevation associated with infection in these patients occurs at a mean of some 80 hr after surgery, about 30 hr later than temperature elevations not associated with subjective symptoms.

These findings of pelvic infection (pelvic cellulitis) usually occur on or after the third postoperative day and require parenteral antibiotic therapy.

Temperature elevation alone disappeared without therapy. Unless a hematoma was suspected, we found it unnecessary to perform a bimanual examination if the patient experienced temperature elevations in the absence of the above described symptoms. Antimicrobial therapy should never be initiated without pelvic examination.

Pelvic abscess is uncommon following elective hysterectomy. It is usually a complication of pelvic cellulitis. It occurs more frequently after vaginal than abdominal hysterectomy. During vaginal hysterectomy, contamination occurs early and continually while it occurs late in abdominal hysterectomy. A pelvic abscess is frequently not diagnosed until response to initial antimicobial therapy is less than anticipated. When clinical response to antimicrobial therapy for pelvic cellulitis is suboptimal after 48–72 hr, or if symptoms worsen on therapy, repeat pelvic examination is indicated. If an abscess is palpated, an anaerobic-specific antibiotic such as clindamycin, metronidazole, or chloramphenicol should be added to the antibiotic regimen. If there is adnexal infection at the time of hysterectomy, an abscess may develop at that location. Pelvic abscesses almost uniformly respond to antimicrobial therapy alone; reexploration is seldom required. If the abscess is accessible, drainage will facilitate a cure. In our experience, treatment for an abscess complicating pelvic cellulitis will extend hospital stay for about 3 days longer than the stay necessary for treatment of pelvic cellulitis without abscess formation. A single preoperative dose of antibiotic has abolished this complication of pelvic infection.

Ovarian abscess is an extremely rare infection. Symptoms are usually minimal and nonspecific and may not become evident until several weeks after the patient has been discharged from the hospital.[26] The patient may not seek care until rupture of the abscess. This requires emergent treatment for stabilization and surgery. The advent of antimicrobial prophylaxis has resulted in the disappearance of this potentially life-threatening catastrophe. It is the view of most gynecologic surgeons that excision of ovarian cysts at the time of hysterectomy does not predispose to ovarian abscess.

It is difficult to identify an early pelvic hematoma. Ultrasonography may locate it, but sonography is useful primarily to document increasing size. Significant increases in size can be judged by monitoring the hematocrit. If the hematocrit continues to fall despite transfusion, surgical intervention with ligation of the bleeding site must be performed. Administration of antibiotics when a hematoma is suspected or diagnosed has not altered the incidence of clinical infection in our patients, so that "prophylactic" antimicrobials are not routinely used. A hematoma in the pelvis may become infected, form an abscess, and require drainage. Suppurative infections confined to the fallopian tube and infections within the leaves of the broad ligament are not prone to intraabdominal rupture, and drainage of these infection sites would require an operative procedure, which is not usually necessary. The pathogens of all these infections derive from the normal flora of the lower reproductive tract.

"Anatomically, the vagina is a wonder. Physiologically, it gives us a view of one patient's endocrine health. Socially, it is a delight and joy to all—but bacteriologically it is a paradise. Dark, warm, moist, and rich in all sorts of culture media, it grows many organisms."[24] Cron and coworkers[27] reported that in studying 1,000 women, a Zephiran-saturated pack inserted into the vagina the night before vaginal or abdominal hysterectomy did not significantly alter febrile morbidity. Other types of preparation of the vagina performed immediately prior to surgery did reduce the bacterial count of the vaginal mucosa but did not reduce the bacterial count in the endocervix.[28] Indeed, saline is as effective as povidone-iodine in decreasing vaginal bacterial count.[29] Osborne and colleagues[30] showed that hot conization of the cervix immediately before vaginal hysterectomy was as effective as prophylactic antibiotics in preventing postoperative pelvic infection, thus confirming the importance of endocervical bacteria. Qualitatively, the bacterial flora of the vagina and cervix are similar to organisms found in the lower alimentary tract,

although significant quantitative differences do exist.[28,29,31–34]

Hysterectomy without prophylactic antibiotics is associated with alteration of lower reproductive tract flora.[8,35–37] Other authors showed that the addition of prophylaxis at hysterectomy is associated with alteration of the lower reproductive tract flora when preoperative and postoperative cultures are compared. Differences in types of bacteria recovered with and without prophylaxis are not significant. If a patient develops clinical infection in the pelvis following hysterectomy with prophylaxis, the bacteria recovered would be expected to demonstrate in vitro resistance to the prophylactic agent; this has not been found.

Identification of the specific bacteria present in the vagina or endocervix preoperatively or even at surgical margins intraoperatively does not allow prediction of the pathogens responsible for a major infection after vaginal or abdominal hysterectomy.[8,31,35,37–40] It is, however, from this lower reproductive tract polymicrobial milieu that pathogens of pelvic and abdominal wound infections originate.[41–44] It is frequently difficult to identify the true pathogens in pelvic infections because usually three to eight potential pathogens are recovered from transvaginal cultures. Also, infection can exist in the parametrial or adnexal structures, sites not easily cultured. Culturing the vaginal apex may not accurately identify pathogens at these remote locations. A transvaginal culture should be taken from the space cephalad to the vaginal suture line. Although septicemia is uncommon, a pathogen identified in the blood provides reliable and important information.

Therapy for these infections is empiric and is begun before culture data, if obtained, are available. Failure rates of initial empiric regimens were as high as 35% in many studies. Although the presence of anaerobes was recognized in the earliest studies, their importance did not become evident until the early 1970s. Combination therapy for these postoperative infections was common because of the polymicrobial origin. Penicillin and aminoglycoside or penicillin and tetracycline were the most frequently employed regimens. If initial therapy failed, an anerobic-specific agent was added.[45]

DiZerega and colleagues[46] demonstrated that the combination of penicillin and gentamicin was signifiantly less effective than the combination of clindamycin and gentamicin in the treatment of women with pelvic infections after cesarean section. The latter antimicrobials have become the combination of choice for many major pelvic infections. Recently, results of treatment of serious pelvic infections were comparable when either clindamycin, chloramphenicol, or ticarcillin was combined with gentamicin.[47] Similar results were observed when metronidazole or clindamycin was combined with tobramycin.[48] Penicillin or ampicillin are frequently added to combinations resulting in administration of three antibiotics, frequently at different dosing intervals. Success rates are not improved. Therapeutic regimens providing predictable anaerobic coverage are associated with the most favorable cure rates.

Questions have been raised about the role of *Streptococcus faecalis,* enterococcus, as a pathogen in pelvic infections. It is frequently recovered from pelvic infection sites. Women who developed pelvic infections treated with cephalosporins, which are generally ineffective against the enterococcus, were clinically cured even when enterococcus was recovered from the pretreatment culture. If a pelvic culture is repeated at discharge from the hospital, enterococcus is frequently recovered after successful cephalosporin therapy. This leads us to believe that enterococcus is not a pathogen in the majority of instances. Any regimen selected for therapy should be active against gram-negative and gram-positive aerobes and anaerobes.

The newer semisynthetic penicillins and cephalosporins have increased bactericidal activity against an expanded spectrum of aerobic and anaerobic bacteria, including *Bacteroides fragilis.* Many of these antibiotics as single agent therapy have been shown to be as effective as combination therapy, and in most instances is cheaper than combination therapy, for pelvic infections.[49–54] There are many obvious advantages and benefits to be realized from effective monotherapy. Indeed, many gynecologists have used ampicillin alone to treat these infections with excellent success rates. There is no need to alter regimens that are successful.

For posthysterectomy pelvic infection, intra-

venous therapy should be instituted immediately on establishing the diagnosis. In the past, women who developed pelvic infections after hysterectomy were treated for a finite period of time, such as 5–7 days. They were sent home on an oral antimicrobial to be taken for another 1–2 weeks to complete 14 days of therapy. Many gynecologists have found that intravenous therapy administered until the patient has been afebrile for 24–48 hr is sufficient; oral antimicrobials in the hospital or after discharge are not necessary and may be detrimental.[53]

Pelvic Thrombophlebitis

Another infection that has been almost entirely eradicated by antibiotic prophylaxis and early therapy for pelvic infection is septic pelvic thrombophlebitis.[55] It was seen more frequently after puerperal or postabortal sepsis than after hysterectomy. It is difficult to establish the diagnosis with certainty. Without surgical confirmation, it was primarily a diagnosis of exclusion unless septic pulmonary emboli or metastatic abscesses were diagnosed in other organs. With computerized axial tomography scanning, thrombosis in pelvic and/or ovarian veins can now be identified. The "classical" presentation of chills, spiking temperatures, and protracted clinical course described by Collins and colleagues[56–58] is rarely seen. The most common presentation now is presistent temperature elevation after more than 3 days of parenteral antimicrobial therapy for clinical pelvic infection. At that time, the patient is relatively asymptomatic, and no longer has tenderness at infection sites; the pulse rate is elevated above the normally expected rate.

This temperature pattern might exist with the presence of resistant pathogens or a superinfection, but with either of those conditions, the worsening symptoms and physical findings will indicate therapeutic failure. Often, antimicrobial therapy is altered. Without specific symptoms and clinical findings, a cause more plausible than resistant bacteria or superinfection might be pelvic phlebitis or thrombophlebitis. Blood culture should be obtained, and bimanual pelvic examination should be performed. Heparin therapy, introduced in 1964 by Schulman and Zatuchni,[59] and continued antimicrobial therapy will result in a normal temperature and pulse rate reading within 24 hr. A response is presumptive evidence of the presence of at least phlebitis. Ligation or clipping of the vena cava and/or ovarian veins is rarely indicated, and then usually only when a patient continues to experience embolization after anticoagulation has been achieved. Medical treatment with antibiotics and heparin appears to be the preferred treatment regimen for suspected pelvic thrombophlebitis.[60]

Drug Reactions

Drug fever, perhaps observed more frequently with the newer semisynthetic antimicrobials, may present as persistent fever without other signs of drug reaction. Signs and symptoms of clinical infection disappear after initiation of appropriate antimicrobial therapy. Sustained pyrexia without circadian fluctuation is suggestive of drug fever. This is a diagnosis of exlcusion in most instances. The onset of eosinophilia and a rash constitute strong supportive evidence for the diagnosis; the absence of both does not exclude the diagnosis. Withholding antimicrobial therapy, a justifiable event in such patients, results in a fairly prompt return to normal temperature in the asymptomatic patient. The only means of firmly establishing the diagnosis is a drug rechallenge, which is usually not indicated.

Gastrointestinal Complication

A problem seen occasionally in patients treated with broad-spectrum antibiotic regimens is diarrhea. Almost all antibiotics can cause diarrhea. The occurrence of diarrhea is primarily a nuisance in over 80% of cases, and responds to supportive care. Severe diarrhea may require alteration of antibiotics or stopping antimicrobial therapy if the infection being treated has resolved. If colitis develops with pseudomembrane and *Clostridium difficile* is identified, appropriate and prompt additional therapy is necessary. Although presentation of pseudomembraneous enterocolitis is variable, it is frequently severe, beginning 4–9 days after initiation of antimicrobial therapy. It has been reported after oral, intramuscular, intravenous, or topical uses of penicillins, cephalosporins, or clindamycin. The diarrhea is usually watery, profuse, and accompanied by significant abdominal cramps, high fever, marked

abdominal tenderness, and leukocytosis to as high as 35,000 cells/mm^3. In some cases, however, symptoms do not begin until days to weeks after antibiotic therapy has been completed. If untreated, electrolyte imbalances and dehydration may be severe and even fatal.

The diagnosis is quickly and accurately made by endoscopy and visualization of the nodular inflammatory and pseudomembraneous plaques. Biopsy is confirmatory, but not essential if the appearance of the bowel is characteristic. *C. difficile* cytotoxins are present in stools. Only a small percentage of people carry that potential pathogen, so Crohn's colitis, idiopathic ulcerative colitis, and colitis caused by pathogens such as *Salmonella, Shigella,* and *Campylobacter* must be ruled out. The treatment of choice for *Clostridium difficile* colitis is oral vancomycin, 500 mg four times daily.[61] Improvement begins soon after treatment in most cases; treatment should be continued for at least 5 days, and beyond the time when toxin is recovered from the stool. Before the efficacy of vancomycin was identified, cholestyramine, 4 g 3–4 times daily was somewhat effective, especially in mild cases. Medications to slow gastrointestinal motility should not be administered.

Gram-Negative Bacteria

The aminoglycosides are the most effective agents in the treatment of infection caused by gram-negative aerobic bacteria. They are ototoxic and/or nephrotoxic. These adverse toxic reactions are observed more frequently with prolonged treatment of the older patient, representing a higher proportion of patients now seen by the gynecologist. Prolonged therapy is not necessary for the treatment of most infections following hysterectomy. Serum measurements of aminoglycoside concentration and monitoring renal function can be used to avoid oto- and nephro-toxicity. Adjustment of doses to "therapeutic levels" is not usually a necessity, since most gram-negative pathogens of pelvic infections are exquisitely sensitive to these agents. If necessary, dose adjustment can be accomplished safely. In 249 gynecologic patients, Zaske[62] found a dose of 1.9–14.0 mg/kg/day of gentamicin was necessary to maintain a therapeutic level.

Wound Infections

The second most common infection following hysterectomy that prolongs hospital stay but infrequently requires antimicrobial therapy is wound infection. Infection in an abdominal incision is not usually a difficult diagnosis to make compared to pelvic infections for several reasons. The diagnosis of an abdominal wound infection is made by clinical examination. Diagnosis is usually made between the fourth and sixth postoperative day. The patient presents with low-grade temperature elevation. Induration and erythema are present in the wound edges, and when the skin edges are separated, pus is evacuated and should be cultured. Pathogens recovered are usually those recovered from the lower reproductive tract. Occasionally, pure *Staphylococcus aureus* is recovered, and treatment with penicillinase-resistant antimicrobials may be necessary. This is an uncommon etiology, and represents a true nosocomial, or hospital-acquired, infection.

The infected incision should be thoroughly evaluated to ensure that the fascia is intact and that lateral loculations of infected material do not exist. It is frequently not necessary to open the entire length of the incision to accomplish this evaluation. Debridement of necrotic material and abscess or hematoma wall are necessary; this does not usually involve extensive removal of tissue. Hydrogen peroxide, saline, wet-to-dry dressing changes three to four times daily with fine-mesh gauze carefully applied to incision edges stimulates granulation tissue growth. Parenteral or oral antimicrobials are usually unnecessary. Subcutaneous infection can be avoided in obese patients or patients with infectious processes, such as PID and ruptured diverticuli, by not primarily closing the incision above the fascia. The wound is closed by delayed primary closure on the third to fourth day.

Hematomas or seromas may be extensive and may require daily wound care. Even when these collections may not appear purulent, culture should be performed.

Summary

Good surgical technique as noted above plays a key role in the prevention of pelvic and wound infection. For women who are at high risk for

the development of infection, single-dose application of an antimicrobial prophylaxis is indicated. Caution must be used when evaluating temperature elevation following surgery. Most women with this finding will not require antimicrobial therapy. Although uncommon, complications are associated with antimicrobial therapy and may be fatal. Treatment of these infections should include coverage for the anaerobic organism. In many instances, single-agent therapy is as safe and effective and more cost effective than combination therapy.

References

1. Dicker RC, Greenspan JR, Strauss LT, et al. Complications of abdominal and vaginal hysterectomy among women of reproductive age in the United States. The Collaborative Review of Sterilization. Am J Obstet Gynecol. 1982; 144:841–848.
2. Ledger WJ. Prevention, diagnosis, and treatment of postoperative infections. Obstet Gynecol. 1980; 55 (Suppl. 5):203S–206S.
3. Swartz WH, Tanaree P. Suction drainage as an alternative to prophylactic antibiotics for hysterectomy. Obstet Gynecol. 1975; 45:305–310.
4. Swartz WH, Tanaree P. T-tube suction drainage and/or prophylactic antibiotics. A randomized study of 451 hysterectomies. Obstet Gynecol. 1976; 47:665–670.
5. Poulsen HK, Borel J, Olsen H. Prophylactic metronidazole or suction drainage in abdominal hysterectomy. Obstet Gynecol. 1984; 63:291–294.
6. Galle PC, Urban RB, Homesley HD, et al. Single dose carbenicillin versus T-tube drainage in patient undergoing vaginal hysterectomy. Surg Gynecol Obstet. 1981; 153:351–352.
7. Gray LA: Open cuff method of abdominal hysterectomy. Obstet Gynecol. 1975; 46:42–46.
8. Hemsell DL, Cunningham FG, Kapus S, et al. Cefoxitin for prophylaxis in premenopausal women undergoing vaginal hysterectomy. Obstet Gynecol. 1980; 56:629–634.
9. Falk HC, Bunkin IA. A study of 500 vaginal hysterectomies. Am J Obstet Gynecol. 1946; 52:623–630.
10. Ledger WJ, Child MA. The hospital care of patients undergoing hysterectomy: an analysis of 12,026 patients from the Professional Activity Study. Am J Obstet Gynecol. 1973; 117:423–433.
11. Jacobson JA, Hebertson R, Kasworm E. Comparison of ceforanide and cephalothin prophylaxis for vaginal hysterectomies. Antimicrob Agents Chemother. 1982; 22:643–647.
12. Pratt JH, Galloway JR. Vaginal hysterectomy in patients less than 36 or more than 60 years of age. Am J Obstet Gynecol. 1965; 93:812–821.
13. Ledger WJ, Sweet RL, Headington JT. Prophylactic cephaloridine in the prevention of postoperative pelvic infections in premenopausal women undergoing vaginal hysterectomy. Am J Obstet Gynecol. 1973; 115:766–774.
14. Polk BF, Tager IB, Shapiro M, et al. Randomised clinical trial of perioperative cefazolin in preventing infection after hysterectomy. Lancet. 1980; 1:437–441.
15. Jackson C, Amstey MS. Prophylactic ampicillin therapy for vaginal hysterectomy. Surg Gynecol Obstet. 1975; 141:755–757.
16. Swartz WH. Prophylaxis of minor febrile and major infectious morbidity following hysterectomy. Obstet Gynecol. 1979; 54:284–288.
17. Ohm MJ, Galask RP. The effect of antibiotic prophylaxis on patients undergoing vaginal operations. I. The effect on morbidity. Am J Obstet Gynecol. 1975; 123:590–596.
18. Ohm MJ, Galask RP: The effect of antibiotic prophylaxis on patients undergoing total abdominal hysterectomy. I. Effect on morbidity. Am J Obstet Gynecol. 1976; 125:442–447.
19. Grossman JH III, Greco TP, Minkin MJ, et al. Prophylactic antibiotics in gynecologic surgery. Obstet Gynecol. 1979; 53:537–544.
20. Stage AH, Glover DD, Vaughan JE. Low-dose cephradine prophylaxis in obstetric and gynecologic surgery. J Reprod Med. 1982; 27:113–119.
21. Breeden JT, Mayo JE. Low dose prophylactic antibiotics in vaginal hysterectomy. Obstet Gynecol. 1974; 43:379–385.
22. Allen JL, Rampone JF, Wheeless CR. Use of a prophylactic antibiotic in elective major gynologic operation. Obstet Gynecol. 1972; 39:218–224.
23. Bivens MD, Neufeld J, McCarty WD. The prophylactic use of Keflex and Keflin in vaginal hysterectomy. Am J Obstet Gynecol. 1975; 122:169–175.
24. Rosenheim GE. Prophylactic antibiotics in elective abdominal hysterectomy. Am J Obstet Gynecol. 1974; 119:335–340.
25. Wheeless CR Jr, Dorsey JH, Wharton LR Jr. An evaluation of prophylactic doxycycline in hysterectomy patients. J Reprod Med. 1978; 21:146–150.
26. Ledger WJ, Campbell C, Taylor D, et al. Adnexal abscess as a late complication of pelvic operations. Surg Gynecol Obstet. 1969; 129:973–978.

27. Cron RS, Stauffer J, Paegel H Jr. Mobidity studies on one thousand consecutive hysterectomies. Am J Obstet Gynecol. 1952; 63:344–350.
28. Osborne NG, Wright RC. Effect of preoperative scrub on the bacterial flora of the endocervix and vagina. Obstet Gynecol. 1977; 50:148–151.
29. Amstey MS, Jones AP. Preparation of the vagina for surgery. A comparison of povidone-iodine and saline solution. JAMA. 1981; 245:839–841.
30. Osborne NG, Wright RC, Dubay M. Preoperative hot conization of the cervix: a possible method to reduce postoperative febrile morbidity following vaginal hysterectomy. Am J Obstet Gynecol. 1979; 133:374–378.
31. Gorbach SL, Menda KB, Thadepalli H, et al. Anaerobic microflora of the cervix in healthy women. Am J Obstet Gynecol. 1973; 117:1053–1055.
32. Levison ME, Corman LC, Carrington ER, et al. Quantitative microflora of the vagina. Am J Obstet Gynecol. 1977; 127:80–85.
33. Onderdonk AB, Polk BF, Moon NE, et al. Methods for quantitative vaginal flora studies. Am J Obstet Gynecol. 1977; 128:777–781.
34. Bartlett JG, Moon NE, Goldstein PR, et al. Cervical and vaginal bacterial flora: ecologic niches in the female lower genital tract. Am J Obstet Gynecol. 1978; 130:658–661.
35. Ohm MJ, Galask RP. The effect of antibiotic prophylaxis on patients undergoing vaginal operations. II. Alterations of microbial flora. Am J Obstet Gynecol. 1975; 123:597–604.
36. Ohm MJ, Galask RP. The effect of antibiotic prophylaxis on patients undergoing total abdominal hysterectomy. II. Alterations of microbial flora. Am J Obstet Gynecol. 1976; 125:448–454.
37. Grossman JH III, Adams RL. Vaginal flora in women undergoing hysterectomy with antibiotic prophylaxis. Obstet Gynecol. 1979; 53:23–26.
38. Hemsell D, Hemsell P, Nobles B, et al. Moxalactam versus cefazolin prophylaxis for vaginal hysterectomy. Am J Obstet Gynecol. 1983; 147:379–385.
39. Hemsell DL, Heard ML, Nobles BJ, et al. Single-dose cefoxitin prophylaxis for premenopausal women undergoing vaginal hysterectomy. Obstet Gynecol. 1984; 63:285–290.
40. Hemsell DL, Johnson ER, Bawdon RE, et al. Cefoperazone and cefoxitin prophylaxis for abdominal hysterectomy. Obstet Gynecol. 1984; 63:467–472.
41. Swenson RM, Michaelson TC, Daly MJ, et al. Anaerobic bacterial infections of the female genital tract. Obstet Gynecol. 1973; 42:538–541.
42. Thadepalli H, Gorbach SL, Keith L. Anaerobic infections of the female genital tract: bacteriologic and therapeutic aspects. Am J Obstet Gynecol. 1973; 117:1034–1040.
43. Sweet RL. Anaerobic infections of the female genital tract. Am J Obstet Gynecol. 1975; 122:891–901.
44. Bartlett JG. Anaerobic infections of the pelvis. Clin Obstet Gynecol. 1979; 22:351–360.
45. Ledger WJ, Kriewall TJ, Sweet RL, et al. The use of parenteral clindamycin in the treatment of obstetric-gynecologic patients with severe infections. A comparison of a clindamycin-kanamycin combination with penicillin-kanamycin. Obstet Gynecol. 1974; 43:490–497.
46. diZerega G, Yonekura L, Roy S, et al. A comparison of clindamycin-gentamicin and penicillin-gentamicin in the treatment of post-cesarean section endomyometritis. Am J Obstet Gynecol. 1979; 134:238–242.
47. Harding GK, Buckwold FJ, Ronald AR, et al. Prospective, randomized comparative study of clindamycin, chloramphenicol, and ticarcillin, each in combination with gentamicin, in therapy for intraabdominal and female genital tract sepsis. J Infect Dis. 1980; 142: 384–393.
48. Gall SA, Kohan AP, Ayers OM, et al. Intravenous metronidazole or clindamycin with tobramycin for therapy of pelvic infections. Obstet Gynecol. 1981; 57:51–58.
49. Sweet RL, Ledger WJ. Cefoxitin: single-agent treatment of mixed aerobic-anaerobic pelvic infections. Obstet Gynecol. 1979; 54:193–198.
50. Cunningham FG, Hemsell DL, DePalma RT, et al. Moxalactam for obstetric and gynecologic infections. In vitro and dose-finding studies. Am J Obstet Gynecol. 1981; 139:915–921.
51. Faro S, Sanders CV, Aldridge KE. Use of single-agent antimicrobial therapy in the treatment of polymicrobial female pelvic infections. Obstet Gynecol. 1982; 60:232–236.
52. Hemsell DL, Cunningham FG, DePalma RT, et al. Cefotaxime sodium therapy for endomyometritis following cesarean section: dose-finding and comparative studies. Obstet Gynecol. 1983; 62:489–497.
53. Gilstrap LC III, Maier RC, Gibbs RS, et al. Piperacillin versus clindamycin plus gentamicin for pelvic infections. Obstet Gynecol. 1984; 64:762–766.
54. Rapp RP, Bannon CL, Bivins BA. The influence of dose frequency and agent toxicity on the cost of parenteral antibiotic therapy. Drug Intell Clin Pharm. 1982; 16:935–938.
55. Cohen MB, Pernoll ML, Gevirtz CM, et al. Septic pelvic thrombophlebitis: an update. Obstet Gynecol. 1983; 62:83–89.

56. Collins CG, MacCallum EA, Nelson EW, et al. Suppurative pelvic thromboplhlebitis. I. Incidence, pathology, and etiology; a study of 70 patients treated by ligation of the inferior vena cava and ovarian vessels. Surgery. 1951; 30:298–310.
57. Collins CG, Nelson EW, Collins JH, et al. Suppurative pelvic thrombophlebitis. II. Symptomatology and diagnosis; a study of 70 patients treated by ligation of the inferior vena cava and ovarian vessels. Surgery. 1951; 30:311–318.
58. Collins CG, Ayers WB. Suppurative pelvic thrombophlebitis. III. Surgical technique; a study of 70 patients treated by ligation of the inferior vena cava and ovarian vessels. Surgery. 1951; 30:319–328.
59. Schulman H, Zatuchni G. Pelvic thrombophlebitis in the puerperal and postoperative gynecologic patient. Obscure fever as an indication for anticoagulant therapy. Am J Obstet Gynecol. 1964; 90:1293–1296.
60. Josey WE, Cook CC. Septic pelvic thrombophlebiti. Report of 17 patients treated with heparin. Obstet Gynecol. 1970; 35:891–897.
61. Silva J Jr, Batts DH, Fekety R, et al. Treatment of *Clostridium difficile* colitis and diarrhea with vancomycin. Am J Med. 1981; 71:815–822.
62. Zaske DE, Cipolle RJ, Strate RG, et al. Increased gentamicin dosage requirements: rapid elimination in 249 gynecology patients. Am J Obstet Gynecol. 1981; 139:896–900.

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